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CLINICAL METHODS

Clinical Methods

A GUIDE TO THE PRACTICAL STUDY OF MEDICINE

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PREFACE TO THE ELEVENTH EDITION

IN the present edition the whole of this Manual has been thoroughly revised. The chapter on Bacteriological Investigations has been entirely rewritten by Dr. A. W. Downie. We are indebted also to Dr. William Evans for help in the revision of the chapter on the Circulatory System, to Dr. Kenneth Tallerman for help with the Clinical Examination of Children, and to Dr. George Hadley for help with the Alimentary System. Modifications and additions have been made in the chapters on Case-Taking, General Condition, Respiratory System, and Examination of Pathological Fluids. The frontispiece is new and a number of clinical photographs and diagrams have been added. By discarding a great deal that is out-of-date it has been possible to make the book somewhat smaller than formerly.

R. H.
D. H.

January, 1940.

PREFACE TO THE ORIGINAL EDITION

THE title "Clinical Methods" probably describes the scope of this book better than any other. It is not intended as a treatise upon medical diagnosis. On that subject there is already a sufficiency of good works in existence. It aims rather at describing those methods of clinical investigation by the proper application of which a correct diagnosis can alone be arrived at. To every student when he first begins work in a medical ward the question presents itself: How shall I investigate this case? To that question the present work is intended to provide an answer. The first chapter deals, therefore, with the methods of case-taking in general, and includes a general scheme for the investigation of medical cases. The rest of the book is really an expansion of that scheme, each system being taken up separately, and the methods of investigating it described in detail.

A special chapter has been devoted to the clinical methods of examining children, as these differ in many respects from those employed in the case of adults. Chapters have also been added on the examination of Pathological Fluids and on Clinical Bacteriology, subjects which are daily growing in importance. The methods employed in the investigation of surgical, gynæcological, or obstetric cases do not fall within the scope of the work.

No effort has been spared to make the book thoroughly up to date, and it is hoped, therefore, that it will be found useful by those practitioners who may wish to make themselves acquainted with the latest

methods of clinical investigation. While the whole book has passed through the hands of both of us, yet each has made himself specially responsible for certain parts. Thus Dr. Rainy has written Chapters II, IV, VI, and XIV, the sections on the electrical examination of muscles and nerves, on the parasites of the alimentary tract, and on the microscopical examination of the urine. The rest of the work is from the pen of Dr. Hutchison.

In order to avoid burdening the text, but few references have been given to authorities and original sources. We should like, however, to take this opportunity of acknowledging the help which we have received from various friends. Amongst these are Drs. Alex. Bruce, R. W. Philip, G. Lovell Gulland, and John Thomson, who have helped us with criticism and advice in the preparation of Chapters IV and IX, VI, V, and XII respectively. We have also to thank Dr. Patrick Manson, Dr. Byrom Bramwell, Dr. J. Purves Stewart, and Prof. Symington for the use of specimens and illustrations, and Dr. T. F. Milroy for assistance in the revision of proofs. To Dr. R. J. M. Buchanan we are specially indebted for preparing the drawings illustrating the microscopical examination of the blood.

R. H.
H. R.

September, 1897.

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CLINICAL METHODS

CHAPTER I

CASE-TAKING

THERE can be no question of the value of accurate and systematic case-taking. It trains the beginner in habits of thoroughness and exactness at the bedside, and ensures that no point of importance in the case is missed. To the more experienced clinician the systematic record of cases is of no less value. It gives to his experience a concrete embodiment, so that he can draw upon it at any future time by the comparison of new cases with old, and so enables him gradually to build up his clinical knowledge upon a sure foundation. When we come to the *method* to be pursued in taking a case, however, there is much divergence of opinion, and almost every clinical teacher has his own special plan. Nor is it of so much importance what particular method one adopts, provided one adheres to it. Every good method of case-taking should be both comprehensive and concise. It should be comprehensive, so as to be capable of being applied to every case and of covering all the points in it; it should be concise, so as to present all the important features of a case in as small a compass as possible. The question of conciseness is of very great importance. Nothing is more annoying than to be obliged to wade through a mass of verbiage in order to get at the chief facts of a particular case. The student should practise the art of focusing a case in such a way as to present its leading features in a few sentences. For this purpose the writing of résumés of cases will be found a useful exercise. He should also avoid lengthy verbal descriptions as far as possible,

especially where the facts admit of graphic representation. The use of outline diagrams will be found very helpful in this respect. Physical signs can be filled in on them by means of conventional symbols.

We have appended to this chapter a scheme of case-taking which meets all necessary requirements. At the same time, it must be used, like all such schemes, with some judgment and elasticity. All the points mentioned need not be minutely inquired into in each individual case. For example, if a patient is suffering from advanced cardiac disease, there is no use in writing a minute description of the state of his teeth. Yet that is the kind of error into which beginners not infrequently fall. Of course, it demands some experience to enable one to say what the points are which it is of importance to inquire into in any particular case, and at first one may sometimes be at fault ; but the application of a little common-sense will ensure the avoidance of any gross blunders.

The " taking " of any case consists of two parts—

I. The interrogation of the patient.
II. The physical examination.

Clinical clerks will find it best to make rough notes of both of these, and afterwards to write out the whole case in detail.

I. The Interrogation of the Patient

The object of the interrogation of the patient is to elicit information regarding his present illness and the state of his previous health and that of his family. The interrogation must be pursued with patience, the patient being allowed, as far as possible, to tell his story in his own words. One patient is a good witness and another poor. For example, the cockney patient usually gives an excellent history. His answers are logical and to the point. His evidence is

direct and given in a helpful forthright manner. By contrast, to take another example, the countryman, especially if he works on the land, often has to have the history of his illness dragged out of him by methods of slow extortion, and even then a great deal of what he says may be based on superstition and therefore prove irrelevant. To sort out what is relevant in a history and to do it well is an art which comes only by experience. The manner of one's questions may make all the difference in the world to a nervous or suspicious patient. One may defeat one's own ends by wounding the sentiments or conscience of the patient long before the physical examination begins. It is important to avoid asking the same question twice, because to do so looks careless, and conveys to the patient the impression of taking but a languid interest in his case.

All symptoms are not of equal diagnostic importance. There is usually one symptom which troubles the patient more than any other, and that is the *presenting symptom*. Special attention should always be given to it. It is a good rule not to ask leading questions when taking the history, but once a tentative diagnosis has been made on the presenting symptom it is well to ask for corroborating symptoms which the patient may not have specially observed or which he may have omitted to mention. The experienced doctor shows great skill in the choice and wording of his leading questions. The methodical student will learn this art by experience, but he must be careful always to keep his mind open to learn.

We may proceed now to go more into detail regarding the questions which should be asked. In doing so, we shall consider first the questions which one has to put in every case—what one may call the *general interrogation;* and then we shall take up the questions which have to be put in examining cases of disease

affecting the different systems or organs—this may be called the *special interrogation.*

1. **General interrogation.**—Begin by ascertaining the patient's name, age, occupation, and whether he is married or single. It is also of importance to note his exact postal address for purposes of future communication.

Two important questions then follow—(1) Of what does he complain? It is a mistake to ask "What is the matter?" as this lays one open to the retort that that is what the patient came to find out. (2) How long have the symptoms been present? The patient usually dates the start of his illness from some impressive event or from the onset of some severe symptom. Questioning will sometimes reveal earlier symptoms which belong to the history of the present illness. The real date of onset can often be more accurately defined by asking such questions as "When were you last quite well?" Sometimes it is useful to ask "Did you ever have a pain like this before?" Having thus defined his complaint and its duration, proceed to ascertain the chief facts in his history.

The most logical plan is to take the **family history** first, but in practice it is perhaps more convenient to begin with the history of the present illness, to pass from that to an inquiry into the patient's previous health, and thence to the family history. It is usually sufficient to inquire regarding the state of health or cause of death of the immediate relatives only—the parents, brothers and sisters, and, if the patient is married, of his own children, if any. These facts may tell us whether he is predisposed by heredity to any particular disease.

One may then pass to the **personal history,** which includes the patient's mental attitude to his life and his work. Here it is well to begin with what

may be grouped together as the patient's physical and emotional environment, his surroundings, both at home and at work, and his habits. One should endeavour to visualize the life of one's patient, sharing his emotions and viewing step by step his daily habits, his diet, and his work. It will often be found advantageous to ask the patient to give a brief account of a typical day.

Inquire into (*a*) the exact nature of his occupation (not merely the name of his trade but what precisely his work involves) and whether or not it exposes him to injurious influences ; former occupations should also be noted. Sometimes one should inquire into a patient's business affairs, his ambitions, anxieties, quarrels and, in general, his attitude to his work. (*b*) His home surroundings their sanitary conditions and the possible existence of overcrowding (*c*) His domestic relationships, his psychological make-up. his interests, his hobbies, his hopes, his fears, the holidays he gets and whether he enjoys them, the amount of exercise he takes, the games he plays and, in general, the sort of life he leads. (*d*) The nature of his food, and the extent of his indulgence in such articles as alcohol and tobacco. (*e*) Whether or not he has lived abroad, if so, in what part of the world and whether he was ill there.

One should take up next the question of the patient's **previous health.** Ascertain what illnesses he has had, when he had them, and their duration. Be careful not to accept uncritically a diagnosis of a previous illness. Ascertain by whom the diagnosis was made and confirm this if possible by a few questions as to the symptoms experienced. Patients repeatedly state that they have suffered from influenza, an expression used loosely for a large number of febrile illnesses. In all cases the patient should be questioned specifically for a history of rheumatic

fever, chorea, scarlet fever, tonsillitis, pneumonia, diphtheria, and exposure to tropical disease. Adult males should be questioned for a history of gonorrhœa or syphilis and their treatment. It is not sufficient to ascertain that he has had a sore ; inquiry must be made into the question of secondary symptoms, such as a rash. If the patient denies venereal disease, it may be necessary to ask whether or not he has ever been exposed to the risk of it. In the case of female patients, information regarding venereal disease should, as far as possible, be obtained indirectly, and in these circumstances questions should be put as delicately as possible.

We are now ready to obtain the history of the present disorder. Ask how and when it began, getting dates of the chief events if possible, and whether suddenly or gradually ; what was the first thing he noticed wrong ; what has been the order of appearance of his symptoms, and which are those that chiefly trouble him at the present time. Ascertain whether or not he has already been under treatment, and, if so, what has been done for him.

This exhausts the general interrogation, and includes the chief facts that have to be inquired into in every case.

2. The **special interrogation,** to which we have already referred, must be modified according to the particular organ which one believes to be affected and the nature of the disease of which it is suspected to be the seat. It is here that the student has most difficulty. It is only by experience that one can tell what it is essential to ask in each individual case. In order to help the beginner, however, we have drawn up for his guidance a scheme of interrogation which he can pursue when he has reason to suppose that the patient's general symptoms point to an affection of any particular system or organ. Such a scheme is necessarily very

far from complete, and may require to be supplemented in individual cases. Nor is one able in such a work as this to explain *why* such and such questions should be put in affections of this or that organ or system. The reasons for the questions the student will find out for himself in due time. Our present object is merely to help him in the interrogation of his earlier cases, so that he may not miss any important facts. The questions are to a considerable extent concerned with eliciting what are sometimes spoken of as *subjective symptoms*—i.e. the morbid sensations experienced by a patient as the result of the disease of some organ or system.

In making the notes, these, along with the other replies, should be entered under the special system to which they refer.

1. Alimentary system and abdomen.

(*a*) Symptoms point to an affection of the *stomach*. Inquire regarding—

Pain.—For how long has he been subject to it? Has he had intervals of complete freedom? If so, for how long? What is its relation to meals (if any)? Does it wake him at night? If so, at what time? What factors afford relief, e.g. food, alkaline powders, vomiting? What is the exact site of the pain? Is it localized or diffuse? Does it radiate to any other part of the body? Does it make him vomit?

Appetite.—Is it excessive, diminished, or capricious? Does it increase on eating? Does he suffer from thirst?

Meals.—Arrangement of these; the nature of the food. Does he eat between meals?

Sensations referred to stomach.—Their nature, and where exactly they are felt. Their relation to the taking of food; are they produced or relieved by it? How long after food do they come on? Are they specially influenced by different kinds of food? Distinguish especially between "pain" and mere "sense of discomfort" or "fullness."

Vomiting.—Frequency; by day or by night; in the morning or in the evening. Its relation to food; is it only

after food, or does it occur at other times? Its relation to pain; does it relieve pain or not? Does patient strain and retch much, or does the vomited matter come up quite easily?

General characters of vomited matter.—Its amount and colour. Is there ever "coffee-grounds" vomiting; is it ever sour and frothy? Does it contain cellulose residues of food taken days before?

Eructations.—Do they burn; have they any taste?

Flatulence.—Relation to meals; after food only or between meals? Relation to particular articles of food. Does the flatus tend to escape downwards or upwards? Is it accompanied by pain or discomfort?

Water brash.—Does he ever experience excessive secretion of saliva into the mouth, with regurgitation of mouthfuls of clear, tasteless fluid?

Heartburn.—Does he suffer from a burning sensation behind the lower end of the sternum?

State of the bowels —How often are they opened? Any special characters of the motions

Menstrual periods.—Having inquired regarding the regularity of the bowels, one may ask if the patient's periods are regular or if she is "regular in her own health" or "regular in her unwell times" In the majority of cases menstruation occurs every 28 days, but the intervals may be longer or shorter according to the patient's habit. If menstruation has ceased, one must inquire how long it has been absent. Normally the cessation of menstruation, or menopause, should not occur until about the 45th year or later. It is also necessary to inquire whether the patient is losing more or less blood than usual. The menstrual flow is to be regarded as abnormal if it lasts for less than two or more than eight days. The presence or absence of pain at the periods must also be noted.

(*b*) Symptoms point to an affection of the *intestines.* Inquire regarding—

Diarrhœa.—Number and time of occurrence of motions during the day; their relation to meals or to special articles of food. Colour of the motions; are they formed, unformed or frankly watery? Has he ever passed any blood or slime? Is there any straining (tenesmus) during defæcation? Is there any flatulence? Is there abdominal pain or pain at the anus during defæcation?

Constipation.—What is his usual habit? are the bowels

opened regularly, and if so, how often? Has there been recent change in bowel habit? How long since the last motion? Has he ever noticed any grooving or flattening of the motions? Does the constipation alternate with diarrhœa? Has he any griping pain? Has he had any vomiting?

Pain—Character; persistent or intermittent Where is it felt worst? Is it relieved or aggravated by pressure?

(*c*) Symptoms point to an affection of the *liver* or *gall-bladder*—e.g. patient is jaundiced, or has pain in region of liver. Inquire regarding—

Pain.—Its site. Has he ever any attacks of very severe pain, coming on suddenly and lasting for a few hours? If so, did the pain radiate, and in what direction? Was there vomiting with it? Was he yellow after it subsided? Has he ever pain in the tip of the shoulder?

Does he suffer from piles?

Does he ever vomit blood?

Has he noticed any change in the colour of the urine or fæces?

If he is jaundiced does his skin itch? Inquire also regarding his digestion on the lines of the interrogation already laid down for affections of the stomach.

2. The symptoms point to an affection of the **circulatory system.** Inquire regarding—

A *family history* of rheumatism, angina, apoplexy, or heart disease.

A personal history of rheumatic fever, chorea, scarlet fever, or diphtheria. (If a child, ask also about sore throats and "growing pains.")

The following subjective sensations :—

Dyspnœa.—Has he to sit up in bed, or can he sleep lying down? When does it come on? *Præcordial pain* or distress; its exact site and character; does it radiate or not? If so, in what direction? *Palpitation;* its relation to meals, and to exertion. Does the heart give an occasional thump now and then? *Sleep*, good or bad; does he dream? *Giddiness*, is it ever present, and when? Is there any sense of undue exhaustion after bodily or mental work?

Ask also for signs indicative of general venous distension —e.g. do the feet ever swell? Has he any cough? What is the state of the digestion? Does his nose ever bleed?

3. The symptoms and appearances point to an affection of the **blood.** Inquire regarding—

Family history of bleeders Has he had any loss of blood ? Has he bleeding piles ? (If a woman—is menstruation excessive or diminished ?) What is the state of the bowels ?

Any possibility of lead-poisoning or malaria ?

Such subjective sensations as breathlessness on exertion; headache; giddiness

Do the feet ever swell ?

4. The symptoms point to an affection of the **respiratory organs.** Inquire regarding—

Family history of bronchitis, asthma, or phthisis. The patient's occupation; does it expose him to the inhalation of irritating fumes or particles ? Has he ever had large glands in the neck ? Does he sweat at night ?. Is he getting thinner ?

Cough —Its character and frequency; when is it worst ? Does it cause pain or not ? Does he ever vomit with it ?

Expectoration.—Its amount and general characters; yellow or not ? Ever blood in it ? If so, is it only after severe coughing ? Is the blood bright and frothy or dark in colour ?

Pain in chest.—Is it aggravated by taking a breath ? Is it constant or not ? Where is it seated ?

Dyspnœa.—When is it felt ? If spasmodic, ask him to describe an attack.

5. The symptoms point to an affection of the **kidneys**—e.g. general dropsy—or **urinary passages**—e.g. pain in micturition. Inquire regarding—

Family history of Bright's disease or apoplexy.

Personal history of scarlet fever, lead-poisoning, prolonged suppuration, gravel or gout, and previous renal disease.

Has he any pain in the lumbar region or any attacks of acute pain shooting down into the groin ?

The following remote symptoms :—

Headache, vomiting, drowsiness, paralysis or fits, dimness of sight, dyspnœa.

Does the face ever look puffy in the morning ?

What is the state of the bowels ?

Inquire regarding micturition as follows :—

Urine.—Is it altered in amount ? Has he to rise in the night to pass it ?

Is it altered in colour? Is it clear or turbid when passed? Ever any blood in it? If so, at what period of micturition is it present?

Is there any increased frequency of micturition? Is the increase by day or by night?

Is there any pain during micturition? Is it before, during, or after the act? What is its character, and where is it felt? Is it aggravated by movement?

6. In **skin diseases.**

Inquire carefully into the patient's personal habits as regards diet, clothing, and washing. What is his occupation? Does he handle chemical substances or other irritants? Ask if he has been taking any drugs recently. It may be necessary to inquire carefully regarding syphilis. Does the eruption itch? If so, when is the itching worst? Did the eruption appear all at once or in crops?

7. The symptoms point to an affection of the **nervous system.** Inquire regarding—

A *family* history of mental disease, chorea, paralysis or fits.

The nature of the patient's work; is he exposed to any poisons—e g. lead, mercury, manganese, carbon bisulphide, or other volatile substances? Syphilis and alcohol should be inquired about with special care.

In cerebral cases it is important to inquire regarding discharge from the ear.

Should he complain of *fits*, the following questions should be asked :—

Age at first fit? Any assigned cause? Describe the first fit. When did the second occur? What has been shortest and longest interval between the fits? Are they more or less frequent now? Do they occur in sleep or not? Has he any premonition or aura? What is its character? How long before the loss of consciousness does it occur? Is the onset sudden or gradual? Are convulsions present? Are they general or local? Where do they begin and end? Does he fall? Has he ever hurt himself? Does he bite his tongue, micturate, or defæcate during the fit? Are there any after-symptoms, such as sleep, headache, automatism, or paralysis? Is there any subsequent mental disturbance?

If he complains of *paralysis*, inquire regarding—

Symptoms of heart disease, or chronic renal disease (*see*

Circulatory and Urinary Systems) Had he any premonitory symptoms before the onset? Has he any headache or vomiting? Where is the headache situated? Has he any giddiness or difficulty in walking? (The method of eliciting other subjective symptoms of nervous disease is considered along with the investigation of the cranial nerves in Chap. IX., p. 392.)

8. The symptoms point to an affection of the **bones** or **joints.**

Inquire specially, in the family history, for tuberculous disease, rheumatism, gout, or syphilis, and in the personal history for tuberculous disease, previous manifestations of gout or rheumatism, for syphilis or gonorrhœa, and for any remote or recent injury (and in a woman for leucorrhœa).

If there is pain referred to a bone, ask whether it is worse during the day or during the night. If the pain is in a joint, ask whether it is present constantly or only when the joint is moved Are there any starting-pains at night? Is the pain affected by weather? Does the pain shift from one joint to another?

If the patient is a young **child,** the following special questions should be put to the mother or other responsible person :—

How many other children are there? Any dead, and of what? Where does patient come in the family? Have there been any miscarriages? If so, when? Health of father's and mother's family? Mother's health during pregnancy?

Was this a full-time child? Was the labour normal? Was the child breast-fed; if so, how long? If not, how was it fed? When did mixed feeding begin? What food does it get now? Had it any rash after birth, or any snuffles? When did it begin to get its teeth, to walk, and to talk?

What is the usual state of the digestion and bowels?

Inquire regarding previous illnesses: Infectious diseases and ages at which they occurred (measles, whooping-cough, chicken-pox, scarlet fever, etc.). Fits (number and dates), attacks of diarrhœa, vomiting, sore throat or bronchitis. Has there been any running from the ears? Have there been severe persistent limb or joint pains? If the child has a cough, inquire whether it has ever whooped, when the attacks are worst, and whether the cough is ever followed by vomiting.

The interrogation of the patient being completed, proceed to—

II. The Physical Examination

Investigate first, in every case, the patient's general state. This includes the general condition of his nutrition, the presence of any obviously morbid appearances, and the other points considered in detail in Chap. II. One proceeds after that either to the investigation of each system by itself, or to an examination geographically, proceeding from above downwards, afterwards writing down the findings under the various systems ; this plan should *always* be followed in the case of children. The results yielded by inspection, palpation, percussion, and auscultation respectively, should always be stated in that order. If, however, it is decided to investigate each system separately, one may either take up the systems in one and the same order in every case, beginning, say, with the Alimentary, or one may examine first the system which is most affected. The latter is, on the whole, the better plan, provided always that one is able to tell which system it really is that is most diseased. The advantage of this method is that it gives prominence to the most important part of the physical examination. Whichever plan the student elects to adopt, he may now proceed to the physical examination of the different systems in accordance with the instructions laid down in the following chapters, the results being noted in the order given in the scheme below.

Only one more point regarding case-taking remains to be emphasized, and that is the importance of noting negative as well as positive facts. It is often quite as essential, for example, to state that such a symptom as

dyspnœa is absent as to record the fact of its presence. This is a point the importance of which is apt not to be fully appreciated by the beginner.

In conclusion, it need hardly be said that the examination should be carried out as gently as possible, all unnecessary exposure, exhaustion, or chilling of the patient being carefully avoided. If the patient is suffering from severe or acute disease, it may be advisable to postpone all physical examination other than that which is absolutely necessary for the diagnosis of his condition, or for guidance in treatment. It should also be borne in mind that when a patient is much exhausted, or suffering from serious disease of the lungs or heart, very dangerous and even fatal results may ensue if he is thoughtlessly made to sit up in bed in order to have his chest examined.

CASE-TAKING SCHEME

I. INTERROGATION

Name. Age Occupation Married or single. Address Date of coming under observation

Complaint.

Duration.

Family history.

Inquire regarding parents, brothers and sisters, and patient's own children. Note state of their health, or the cause of their death, with age at which they died.

Personal history.

Environment—Nature of work and its surroundings. Hygienic conditions at home, habits as to exercise, food, tea, alcohol, and tobacco.

Previous illnesses or accidents (if any), with their time of occurrence, duration, and results.

Present illness.—Time and mode of its origin, the order in which symptoms appeared, and the chief symptoms which trouble patient now; treatment (if any) already employed.

II. Physical Examination

1. Present state.

General condition.—General state of consciousness and intelligence Decubitus (if in bed), or attitude and gait (if up) (pp 20 and 23) General state of development and nutrition. Expression of face, presence or absence of pallor, jaundice, cyanosis, dropsy, or trophic changes. Presence or absence of any special characters of the hands (p 32). Glandular enlargements Character of the respiration, and the presence or absence of cough. Take the temperature.

2. Alimentary system.

Subjective symptoms (*see* Special Interrogation, p. 6). Examine the *mouth* (including the teeth, gums, and tongue), the *pharynx*, and *fauces* (pp. 46-50), and the *œsophagus* (p. 51).

General inspection, palpation and percussion of the *abdomen* (pp. 51-62).

Stomach.—Palpation and percussion (pp 62-63). Examination of test-breakfast, fractional test-meal, or vomit (pp. 74-88).

Intestines.—Investigation of (p. 71). Rectal examination, if necessary (p. 72) Examination of fæces (p. 88

Liver and *gall-bladder.*—Examination of, by palpation and percussion (pp. 63-67).

Spleen —Examination of (p. 67).

3. Circulatory system.

Heart.—Subjective symptoms (Special Interrogation, p. 9).

Pulse.—Describe its rate and its rhythm. Compare the force of successive beats. Ascertain the state of the vessel-walls. Note the blood-pressure during and between the beats. Observe the amplitude of the pulse-waves. Analyse a complete beat of the pulse regarding rise, maintenance and fall of pressure, and determine the presence or absence of secondary waves. Take tracings if the pulse is abnormal.

Inspection and palpation of *præcordia*, noting position and character of apex-beat, presence or absence of epigastric pulsation or præcordial thrills, or of pulsation in the neck or at the base of heart.

Percussion of *heart* (p. 120).
(*a*) Upper border
(*b*) Right border } superficial, deep.
(*c*) Left border

Auscultation of *heart* (p. 125).
(*a*) At apex and a little internal to it.
(*b*) Tricuspid area at lower end of sternum.
(*c*) Aortic area.
(*d*) Pulmonary area and a little outside it
(*e*) Between base and apex (3rd and 4th left costal cartilages).
(*f*) Veins and arteries of neck.

If a bruit is heard, note—
(*a*) Its time
(*b*) Its character (musical, harsh, etc.).
(*c*) Its point of maximum intensity.
(*d*) Its direction of propagation.

4. The blood.

Count the red and white corpuscles (pp. 187-199). Estimate the hæmoglobin (pp. 199-204). Examine the blood microscopically, making films if necessary (pp. 204-218).

5. Respiratory system.

Subjective symptoms (*see* Special Interrogation, p. 10).
Count the respirations and describe their character.
Inspection of chest, noting its shape, power of expansion, etc. (p. 235)
Mensuration of the two sides of the chest.
Palpation of chest (expansion and vocal fremitus) (p. 246).
Percussion of lungs anteriorly, laterally, and posteriorly (p. 251).
Auscultation of lungs in same order (p. 265), noting—
(*a*) Type of breath-sounds.
(*b*) Character of vocal resonance.
(*c*) Presence or absence of accompaniments.
Sputum.—Note its naked-eye and microscopic characters (p. 279).

6. Urinary system.

Palpate the *kidneys* (p. 69).
Examine the *urine*—physically (p. 284), chemically (p. 295), microscopically (p. 333), making a note in *every* case of the following points :—
Quantity in twenty-four hours, colour, specific gravity, reaction, odour, general character of deposit.

Presence or absence of albumin, blood, sugar, and bile. Microscopic characters of deposits

7. **Skin.**

General colour; presence or absence of pigmentation or eruption; nature of "primary lesion" in eruption and of "secondary lesions," if present (p. 348)

Palpate the skin; dryness, smoothness, thickness, elasticity. Character of subcutaneous tissues.

8. **Nervous system.**

Inquire regarding subjective symptoms (*see* Special Interrogation, p. 11).

Investigate state of—

(1) Intellectual functions (intelligence, memory, sleep, coma, delirium, speech, etc.) (p. 383)

(2) Cranial nerve functions (testing them in order) (p. 392).

(3) Motor functions (noting presence or absence of paralysis, or of abnormal muscular movements, and state of muscular nutrition) (pp. 433-451). Electrical reactions of muscles and nerves, if necessary (pp. 471-475).

(4) Sensory functions (including condition of sensibility to touch, weight, temperature, and pain, and the muscle-sense) (pp. 451-458), presence or absence of abnormal sensations (p. 458)

(5) Reflexes:—
Superficial reflexes (p. 459).
Deep reflexes (p. 463).
Organic reflexes and sphincters (p. 469).

(6) Vaso-motor and trophic changes. Tache or abnormal flushing. Localized pallor or blueness. Sweating (presence or absence in any locality). Joint changes. Changes in the nails, hair, or skin (abnormal pigmentation, eruptions, atrophies, etc.) (p. 471).

9. **The eye.**

Appearances seen on ordinary inspection of lids, conjunctiva, cornea, iris, etc. (p. 476).

Use oblique illumination and ophthalmoscopy, noting state of media, refraction, and characters of fundus (pp. 479-494).

N.B.—The fundus of the eye should be reported on in all cases of nervous disease.

The **ear.**—Examine pinna, meatus, and membrane (using speculum and inflation if necessary) (p. 494).

The **throat, nose, and larynx.**—Examine larynx (laryngoscopy) and anterior and posterior nares (posterior rhinoscopy) (pp. 499-508), noting any abnormalities.

10. **Locomotory system.**

Describe any changes in the bones or joints (p 509).

Diagnosis
(Prognosis)

Notes of Treatment and Progress

(Daily notes in acute cases; in others make a note of progress every three days.)

State on dismissal.

If patient died, add notes of post-mortem (if held).

The following special scheme for cases presenting **mental symptoms** will often be found useful in medical wards:—

1. **General.**

The aspect of the patient as modified by the mental disturbance. General attitude and behaviour.

Any peculiarity in clothing. Does patient tend to strip himself or behave indecently? Can he dress himself? How does he take his food?

General standard of intelligence. Can he read? Can he write? Can he amuse himself with pictures?

Speech as modified by the mental state.

Is he destructive?

Is he dirty in his habits? If so, is it from inattention, or is he actively dirty?

Masturbation, alcoholism, etc.

Is he cataleptic or rigid? Does he make any rhythmical movements or sounds? Restlessness or tremor of the hands? Over-action of muscles of face?

Does he sleep?

Does he tend to wander about the room or house at night?

2. **Sensory.**

Delusions of sight, hearing, smell, taste. Subjective sensations of touch based upon wrong interpretation of some actual sensation.

Hallucinations.

3. Emotional.

Exaltation.—Chattering, shouting, singing Excessive sense of well-being. Restlessness or violence.

Depression.—Crying, sighing, moaning. Miserable feeling, either in attacks or continuous. Fear. Is the patient suicidal?

Erotism.—Are the patient's statements coloured by an erotic tone? Give examples.

Religion.—Is the patient's mental state coloured by an extravagantly religious tone?

4. Memory.

Memory of intention, i.e. does the patient wish to say or do something and immediately forget the intention? Does the patient misplace things?

Memory of recent events.

Memory of remote events (e g. events of childhood).

[If recent memory only is lost, try to find out when the break in memory occurs]

5. Ideation.

Orientation.—Sense of time and space Delusions of identity (i e. does patient mistake those around him for his friends and associates before he entered hospital, or does he imagine they are famous or legendary persons?) Does he appreciate his surroundings, or does he imagine himself elsewhere than he really is? Does he invest the acts of those around him with a secondary or symbolic meaning? Does he describe actions he has performed, in themselves not impossible or improbable, which, however, did not actually occur? (e g. when in bed with alcoholic paralysis does he describe the walk he took in the morning, the people he met, etc. ?).

Coherence or incoherence of ideas.

Delusions of suspicion.—Continuous or only in attacks.

Delusions of persecution.—Action of unseen agencies, etc. (especially at night).

Delusions of grandeur.—Riches, power, bodily strength.

Delusions concerning his health or bodily state

Fears (*unfounded*) in neurasthenia.

CHAPTER II

GENERAL CONDITION AND APPEARANCES

BEFORE beginning the physical examination, the physician may gather invaluable information from a more general survey of his patient. During the time occupied in asking questions, and even before it, the skilled eye and ear may detect much that has an important bearing on the case. Experience in actual clinical practice can alone educate to this, but some lines may be indicated along which to work.

One of the first things to observe is the **attitude of the patient as he lies in bed** (decubitus). In health a person lies in any manner in which he feels comfortable. He changes his position without much difficulty from time to time, and has no hesitation in altering his attitude if he slips from his pillows or feels otherwise uncomfortable. But the stress of disease will often confine his activity in narrow bounds. When fever has run high, or when some other cause has reduced the patient to extreme weakness and dulled his consciousness, he no longer makes any effort to secure a position of comfort, but *passively slips downwards* from his pillows in obedience to the law of gravity, and lies listless, flaccid, and silent even when the resulting attitude is such as to render the act of breathing unnecessarily exhausting.

Almost equally characteristic is the *lateral position* necessitated by some diseases of the viscera, and especially by those of the lungs and pleura. The two main factors in compelling this attitude are, first, the greater ease with which respiration can be performed on one side than on the other ; and, second, the fact that in

certain positions the pain is rendered less acute, whilst in others it is aggravated. When these factors co-operate, it is easy to say which side the patient will choose Thus, in pleurisy with much effusion, where the chief difficulty is the mechanical one of providing sufficient expansion for the uninjured lung, and where pain is slight or absent altogether, the patient will be found lying on the diseased side. If, however, pain is the prominent element, as occurs in the earlier stage of pleurisy, he will best secure easy respiration by lying in the position of least suffering. What this position will be it is not easy to predict, for the pain depends both on the amount of movement and the pressure exerted by the inflamed surfaces on each other. When the inflamed pleura is uppermost its movement is greatest, but its pressure against the chest-wall is least; when it is lowermost the opposite is true; and so when movement is the chief cause of pain the patient will lie on the affected side, but when pressure exerts the greater influence, on the sound one. In either case, however, he confines himself to the selected side, and any change indicates an alteration in the state of the disease.

Another class of patients who prefer one side are those who have a cavity in the lung. When this cavity lies with its aperture below, the secretion flowing from it enters healthy bronchi, and by irritating them maintains a perpetual and most distressing cough. If, however, such a patient turns over, so that the cavity fills before its contents escape, a period of tranquillity is obtained, and though the cough eventually recurs, a larger quantity of secretion is promptly got rid of, and another period of rest secured. When, as frequently happens in pulmonary tuberculosis, the secretion is tough and scanty, this symptom is inconspicuous.

Even in health many persons feel more comfortable on one side than on the other, and when ill will often continue to prefer the accustomed attitude; hence the fact that the patient is repeatedly found on one side, although it suggests the propriety of being on the outlook for disease, does not always indicate its presence.

In cases where great demands are made upon the respiratory system, and especially when it fails to respond fully to such demands, the sufferer can rarely lie down in bed, but sits more or less erect and propped up with pillows. To this condition the name of **orthopnœa** has been given. It is common in advanced stages of heart, lung, and kidney disease, and its rationale is found in the fact that this attitude permits of freer use of the accessory respiratory muscles, whilst it leaves the diaphragm less impeded by intra-abdominal pressure, and perhaps, also, acts favourably on the intracranial venous pressure. When the abdomen is greatly distended the sufferer cannot flex his thighs without raising the abdominal pressure; at the same time he prefers to sit up rather than to remain in bed, in order that the weight of the fluid may not hinder the descent of the diaphragm. In sitting up he tries to avoid bending his thighs, and therefore he keeps well forward in his arm-chair, sometimes almost in a kneeling attitude, whilst he rests his head on a table placed before him. In such cases it is obvious that the removal of the ascitic effusion may afford unspeakable relief to the patient.

In **abdominal disease,** especially when the peritoneum is involved, the aspect is frequently characteristic. The patient lies on his back with a rigidity of attitude and shallow respiration which betoken the pain that any movement produces, whilst one or both legs are drawn up, according as the inflammation is limited to one side or has become more general.

In **colic** and **dysmenorrhœa** there is often great restlessness, which contrasts vividly with the fixed attitude of serious inflammation ; in **renal colic** the patient tosses about and tries one position after another in futile search for a posture free from pain.

Patients who are attacked by **acute rheumatism** have a peculiar aspect of helplessness, the limbs lying motionless, the joints being swollen, stiff, and painful.

Various **diseases of the nervous system** produce characteristic attitudes; peculiarly important is that of meningitis, where the neck is bent backwards so that the head seems to bore into the pillow.

When possible, the physician should not only study his patient in bed, but should also see him up and walking. Many very characteristic attitudes, which are of the greatest value in forming a diagnosis, can only be observed when the patient is in the erect posture. Thus the forward stoop, the stiff neck, the tremor, and the fingers flexed at the metacarpal joints and working against the thumb as though engaged in making cigarettes, are as characteristic of paralysis agitans as is the festinant gait.

When the patient is **standing,** observe (1) the pose of the head; (2) the set of the shoulders; (3) the inclination at which the trunk is carried on the pelvis —thrown back in hypertrophic muscular paralysis, in pregnancy, and in massive abdominal tumour, often bent forward when abdominal pain is present; (4) the position of the arms and hands; (5) the outline of the lower limbs.

When the patient walks, any peculiarity in his **gait** must be observed. The more important types of gait are described in Chap. XI, but the student must remember not only that alterations may be due to diseases of the muscular and nervous systems, but that the pain of a gouty toe, or of a blistered heel, or

surgical conditions in the ankle-, knee-, and hip-joints, likewise produce characteristic effects.

At least a passing glance should be bestowed on the **dress.** Apart from insanity, where the patient's clothing is frequently dishevelled or grotesque, one may discover indications of a local or general change in his bulk, or his boots may wear unevenly in consequence of some abnormality of gait.

The **general development and nutrition** of the patient demand careful examination. In different types of men very considerable variations must be looked for, and various races differ greatly in breadth of chest. Age also is a factor which cannot be left out of the reckoning, and a proportion between height, girth, and weight that would be natural enough at 50 may be quite abnormal at 21. Recognizing, however, that variations must be expected in individual cases, there is still a certain general ratio between height, weight, and chest circumference which has been found to represent the average of a very large number of cases, and may therefore be taken as a rough standard, any wide divergence from which would call for special explanation. The table on the following page is one of several that have been compiled from very extensive statistics. A deviation of more than 20 per cent., either in excess or defect, from the figures tabulated would, for insurance purposes, be regarded as incompatible with a first-class life.

Various attempts have been made, with partial success, to produce a formula which would enable the weight to be estimated when the height and girth are known. One of these, cited by H. Vierordt, is $W = \frac{H\,G}{240}$ kilogrm., where W stands for weight, H for height in centimetres, and G for girth in centimetres.

TABLE OF STANDARD HEIGHTS AND WEIGHTS

Height	Male				Female				Height
	Age 20	Age 30	Age 40	Age 55	Age 20	Age 30	Age 40	Age 55	
ft. in.	lb	lb	lb	lb	lb	lb	lb	lb	ft in
5 0	114	121	125	128	110	115	120	128	5 0
5 1	117	124	128	132	113	118	124	132	5 1
5 2	121	128	133	136	116	122	128	136	5 2
5 3	125	132	137	141	119	126	132	140	5 3
5 4	128	136	141	145	123	130	136	144	5 4
5 5	132	140	145	149	127	134	140	148	5 5
5 6	136	144	149	154	131	138	144	153	5 6
5 7	140	148	154	158	135	142	148	158	5 7
5 8	144	153	158	163	139	146	152	163	5 8
5 9	148	158	163	168	143	150	157	168	5 9
5 10	153	163	168	173	147	154	162	173	5 10
5 11	158	168	173	178	151	159	167	178	5 11
6 0	163	172	178	183	156	164	172	183	6 0
6 1	168	177	184	188	161	169	178	188	6 1
6 2	173	182	189	194	—	—	—	—	6 2
6 3	178	188	195	200	—	—	—	—	6 3

If one translates the metric into the more usual British system, estimating the weight in pounds and the height and girth in inches, the formula becomes

$$W = \frac{H\,G}{17}\ \text{lb.}$$

It is important to compare the chest girth taken at the level of the nipples (in male subjects) with that of the abdomen. If in a man below middle age the latter measurement is the larger, it either indicates an undue tendency to fat-formation, which may at a

later period impair his vitality, or it is due to intra-abdominal disease.

When these measurements have been made, the **nutrition** of the patient is observed. Under this head one notes whether the patient is too stout, is well nourished, or is emaciated. In health there is a fair quantity of subcutaneous fat, the muscles are of moderate size and firm texture, whilst those which have been called into special exercise in the ordinary occupation of the individual under examination may be markedly prominent, and the skin is elastic and neither very moist nor very dry. When nutrition is perverted, the muscles become flabby, and the subcutaneous fat is increased so as eventually to become burdensome to its possessor; or emaciation sets in, owing to the balance between ingestion and excretion becoming deranged, and the waste of tissue exceeding its repair. Emaciation is thus an important indication of many diseases, especially those which are accompanied with fever.

In estimating the state of nutrition the observer will take into account the general build of the patient —some are naturally small and slight, others are large and raw-boned; and one also meets with persons who, though possessed of little subcutaneous fat, have well-nourished muscles, whilst others, whose muscles are weak and soft, have an abundant supply of fat in the subcutaneous tissues.

To the trained observer the **expression** of the patient yields information of the very highest importance, and amongst the factors which determine expression the **eye** holds the foremost place. Some patients cannot look their doctor in the face, and this tendency to avoid catching his eye is important, as indicating a probability that the information they are about to give lacks truthfulness, and also that they

are not to be trusted to obey the instructions which they receive. Sometimes the eyes are restless, following every movement of the attendant, as often occurs in phthisis; at other times they stare vacantly into space, regardless of all that is passing around them—a condition well seen when the consciousness is growing dull. In exophthalmos the eyes are prominent, and show a ring of sclerotic above the cornea; or the prominence may be due to a high degree of myopia. In wasting disease or in profound collapse, such as is found in cholera, the sunken eyes and half-closed eyelids cannot fail to command attention. There are racial differences in the "set" and obliquity of the eyes, and by noting this feature something may at times be learned either of the heredity of a patient or of the tendency to reversion towards a lower type.

More detailed reference is made in a subsequent chapter to important abnormalities in the different structures of the eye, where the student will learn how the conjunctiva and sclerotic tell of Bright's disease, of anæmia and rheumatism, of jaundice and of intemperance; and how the cornea foretells an early onset of senile changes in other organs by the appearance of an arcus senilis, or gives indications of syphilis; how the size and mobility of the pupils indicate the existence of disease in the nervous system, or the presence of aneurysm, or it may be only of synechiæ from an old iritis; and how the iris may contain a tuberculous nodule, or be muddy and discoloured from iritis.

The **lower eyelids** are puffy and œdematous, especially in the morning, when the patient is suffering from Bright's disease; and a like appearance is often to be noted in patients who are suffering from very severe paroxysms of cough. It is very characteristically present in children affected with whooping-cough.

The eyelids may also be swollen and inflamed as the result of bug-bites.

People look "dark under the eyes" when their digestion is out of order, or when fatigued, especially from want of sleep; and often women are darker under the eyes during menstruation than at other times.

The **nose** has a sunken bridge in congenital syphilis; the tip is red in some cases of mitral stenosis, in habitual drunkards, in females with chronic indigestion, and sometimes in purely local conditions. Undue mobility of the alæ nasi may be due to neurosis, or it may indicate obstruction to inspiration, and is in this respect very important to look for in infants. Young persons who suffer from adenoids, and to a lesser extent those afflicted with enlarged tonsils or chronic bronchitis, have pinched noses and open, fishy mouths. The pinching of the nose is due to falling-in of the alæ nasi where they lose the support of the nasal bones, whilst the mouth is kept open to reduce the resistance to the entrance of air.

The **lips** are pale in anæmia; livid and blue in congestive heart failure. Thin, mobile lips occur in persons of a neurotic temperament. The vesicles of herpes febrilis on the lip are very often associated with inflammation of the respiratory tract, and their presence should lead to a close search for pneumonia.

The **ears** are often ill developed in idiots, and sometimes in the insane develop hæmatomata. Of greater frequency is the occurrence of tophi in persons of gouty habit.

The **cheeks** give valuable information regarding the patient's health. In anæmia and aortic disease they are pale; in hectic fever there is a bright circumscribed blush over the malar bones; in many persons

who lead an open-air life they are ruddy and high-coloured; in congestive heart-failure they are also high-coloured, but the colour is of a bluish tint which cannot be mistaken for the rubicund cheeks of weather-beaten people. In unilateral chest inflammations, and particularly in pneumonia, the cheek corresponding to the affected lung may be flushed, but if the patient has been lying for some time on one side there is often a difference between the two cheeks, resulting from the pressure of the lower one upon the pillow, quite apart from the presence of disease.

The **form of the cranium** may also indicate some points of importance, to which reference is made in Chap. XI.

In addition to the appearance of individual features, the **general expression of the patient** must be noted. Is it animated, apathetic, or has it the absolute vacancy of unconsciousness? Are there wrinkles on the face, or is it smooth; or is one side smooth and the other wrinkled, as one sees it in unilateral paralysis of the seventh nerve? Is the mouth drawn over to one side, and is there any other lack of symmetry between the two halves? The expression may be characteristic of pain, or there may be a placidity resting on the features which gainsays the assertion of a patient that his agony is most severe. A look of anxiety on a patient's face often presages serious illness at a time prior to the appearance of any other signs or symptoms which would suggest the gravity of the situation. Twitching of the face sometimes results from a nervous habit, at other times it is a symptom of definite disease, of which chorea affords a good example. (Plates 1, 24.)

When **pain** is present, the various features are differently affected, according to its situation. Pain

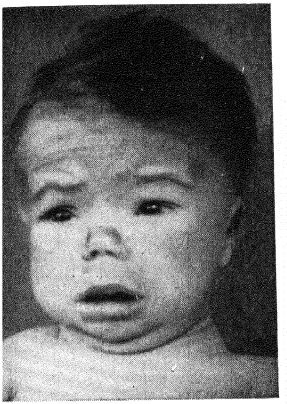
in the head, whether simple headache or of organic origin, causes the sufferer to frown; painful diseases of the chest and abdomen tend rather to affect the expression of the lower part of the face. These signs are of peculiar importance in the case of children who cannot describe their sufferings.

The physiognomy of insanity is often highly characteristic, but descriptions of it must be obtained from special textbooks. In serious illness the nose often looks pinched, the eyes sunken and lustreless, and the chin and malar bones sharp and prominent.

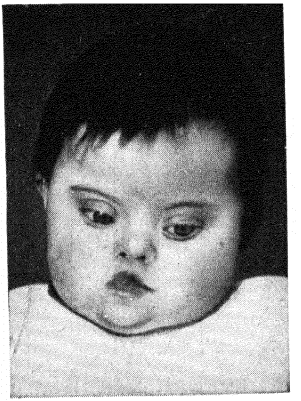
Several types of expression have received special names. Of these the most important are the facies Hippocratica and the typhoid facies.

In the **facies Hippocratica** the skin is livid or pale, and opaque, the eyes are dull and sunken but remain open, the nose is sharpened, the temples are hollow, the chin is sharp, the mouth is open through dropping of the lower jaw, the ears are cold and shrunken, and the cheeks drawn in. When this facies is associated with abdominal disease there is a red or livid ring around the eyes. The Hippocratic facies is a presage of death. The **typhoid facies** is characterized by dull, lustreless eyes, tremor of the lips (with muttering delirium), and a blank, expressionless countenance. Associated with this are found a brown, dry tongue, a rapid pulse, a tendency to sink low in the bed, twitching of the tendons (*subsultus tendinum*), and a constant, purposeless picking of the bed-clothes.

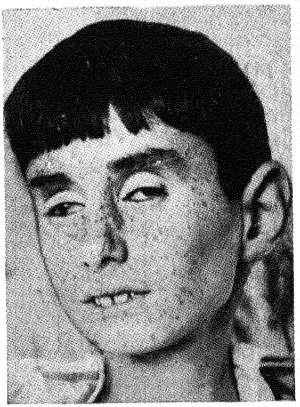
The **state of the skin** where it is exposed must be carefully investigated. In the face we notice especially the **complexion.** This is dependent on two factors—the colour and the transparency of the skin. The most important abnormalities are pallor, yellowness, bronzing, an earthy tint, and a dusky bluish-red



Cretinism



Mongolism



Dehydration

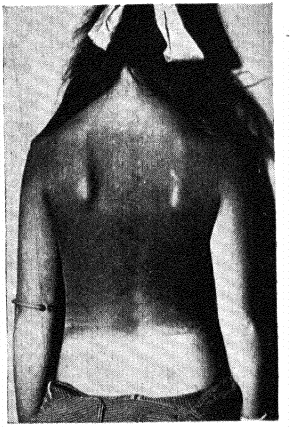


Tetanus

Plate 1.—FACIES.



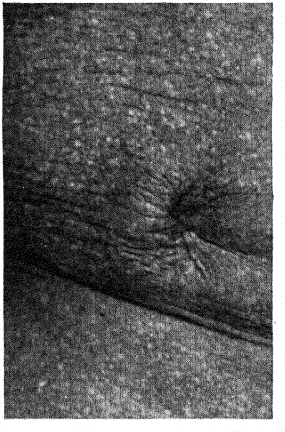
Leucodermia



Ultraviolet Irradiation



Livedo Reticularis



Arsenic Pigmentation

Plate 2.—PIGMENTATION OF SKIN.

hue. **Pallor** occurs in various anæmic states, and also when the action of the heart is greatly enfeebled, as in fainting or severe nausea. **Yellowness** may be due to hæmolytic jaundice, when the tint is pale lemon-yellow; or to obstructive jaundice, when it may be of a dark yellow or orange colour. In obstructive jaundice there may be excoriations from the scratching that results from the intense itching which the bile-acids evoke. **Bronzing** is found in Addison's disease, and affects both the skin and the buccal mucosa. An **earthy tint** is common in states of serious ill-health. It sometimes indicates chronic malaria; and in other instances it is the result of syphilis or of cancer. The **dusky tint** of embarrassed breathing and of heart failure does not demand further notice here. (Plate 2.)

It is also important to search for **cutaneous eruptions**, some of which—measles and syphilitic rashes, for example—frequently appear first about the roots of the hair, whilst others have equally distinctive situations. Ulcers and scars should also be looked for. The colour and nutrition of the hair, and the dryness or moisture of the skin, must be noted; and if perspiration is present, its amount and situation. The perspiring brow of a rachitic child is very characteristic.

Reference has already been made to the **panniculus adiposus** (p. 26); but, in addition to the presence or absence of fat, morbid conditions may lead to abnormal states of the cellular tissues. The chief of these is the presence of fluid or of air, the former being by far the commoner.

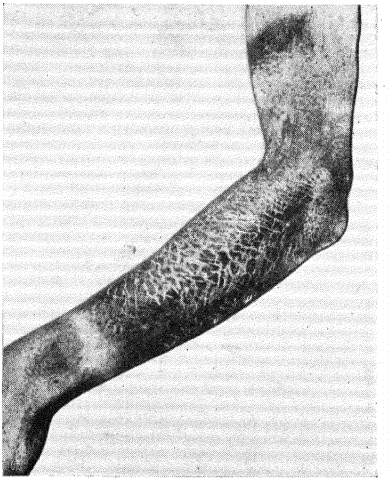
When fluid is present, the condition is that known as **dropsy**, and there are two varieties of this, which are sometimes described as "hydræmic" and "passive." In hydræmic dropsy, typical examples of which occur in sufferers from Bright's disease, the transudation

does not first show itself in the most dependent parts of the body, but in other sites where laxity of the tissues favours its accumulation. Thus in chronic nephritis an early symptom is the œdema of the face, especially below the eyes, which comes and goes, being most noticeable when the patient first rises in the morning. In passive dropsy, however, which is typically present in congestive heart failure, the swelling first appears at the ankles and over the dorsum of the foot, and only gradually mounts to the legs, thighs, and trunk. In local venous obstruction, the dropsy is confined to the parts from which the return of blood is impeded. In this way one finds ascites resulting from cirrhosis of the liver, or œdema of an arm when the axillary glands are cancerous and constrict the axillary vein. Œdema of the whole upper part of the body may result from intrathoracic tumours, or more rarely from compression of the superior vena cava by an aneurysm. Dropsy may be recognized by the pallid and glossy appearance of the skin over the swollen part, by its doughy feel, and by the fact that it pits on pressure.

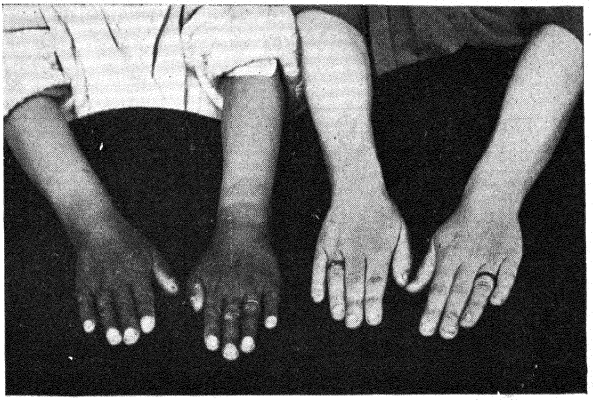
Localized œdema may be due to local changes in capillary permeability, as in angio-neurotic œdema and giant urticaria.

Subcutaneous emphysema is not common, but when present it can be readily recognized by the crackling sensation which is detected on pinching the part affected.

The **hands** of the patient merit careful observation (Plate 4). Notice the strength of grip as he shakes hands; this often indicates improvement or the reverse with considerable accuracy. Their general shape should then be noted, along with the state of the joints, the character of the nails and the presence or absence of finger-clubbing. In osteo-arthritis



Pellagra



Addison's Disease

Plate 3.—PIGMENTATION OF SKIN.



Rheumatoid Arthritis



Heberden's Nodes



Koilonychia



Clubbing

Plate 4.—HAND.

the finger-joints are often implicated, and nodules, known as **Heberden's nodes,** are formed at the terminal joints. In nerve disease the skin of the hand may undergo **trophic changes**, becoming thin and glossy; or the vessels may be influenced by vaso-motor disorders, and lead to redness or to a pallid and dead-looking state of the fingers. Nerve diseases also produce very characteristic movements or attitudes of the hand, as may be seen in athetosis, tetany, and lead palsy. **Tremor** of the hands is a frequent indication of disease. Among the conditions which produce it may be instanced paralysis agitans, disseminated sclerosis, certain traumatic neuroses, Graves's disease, uræmia, insomnia, mercurial poisoning, alcoholism, abuse of tobacco, and senile degenerative changes. The methods of studying this symptom are detailed at p. 449. In ulnar paralysis the hand becomes deformed by over-extension of the first phalanges, combined with excessive flexion of the rest, so that a claw-like attitude is produced. This is known as the **"main en griffe."** When the muscles of the thenar and hypothenar eminences have undergone atrophy the hand becomes flattened, and thus somewhat resembles that of an ape. In acromegaly the hands are massive, the fingers being spatulate with square tips, the knuckles enlarged and the skin thickened. In **clubbing of the fingers** the tissues at the base of the nail are thickened, whilst the nail itself loses its longitudinal ridges and becomes convex from above down as well as from side to side. In extreme cases the terminal segment of the finger is bulbous like the end of a drumstick. The condition may be seen in pulmonary tuberculosis, bronchiectasis, empyema, subphrenic abscess, infective endocarditis and congenital heart disease. In **hypertrophic pulmonary osteo-arthropathy** there

is, besides clubbing of the fingers, thickening of the periosteum of radius, ulna, tibia, and fibula. This gives rise to swelling above the wrist and ankle. In rare cases these joints themselves are swollen. A transverse furrow in the nail is the record of some former interference with its nutrition and, in the absence of a local cause, points to some severe illness in the recent past. **Koilonychia** occurs in idiopathic hypochromic anæmia. The nails are soft, thin and brittle. The normal convexity is entirely lost and replaced by hollowing so great that in some cases eight drops of water from a drop-bottle can be run into the concavity of the nail without overflowing. In infants the movements or position of the hands and fingers will often direct an acute observer to the seat of disease.

The **neck** should always be inspected, and special note taken of any of the conditions described in the paragraphs that follow.

1. The state of the **lymphatic glands.** In syphilis the glands under the upper part of the trapezius are often palpable. In infected conditions of the tonsils the glands at the angles of the jaw are enlarged, and those below the jaw in cases of malignant disease in the mouth. Enlarged tuberculous glands may occur in groups or in long chains beside the sterno-mastoid, and scars will mark the points where they have suppurated. In lymphadenoma the glands are enlarged and discrete. If enlarged glands are found either in the neck or elsewhere, it is important to observe whether they remain firm and distinct, or become fused together, or whether fluctuation can be detected.

2. The **thyroid gland.** The existence of any swelling of this gland is important. Note whether the enlargement is uniform or nodular, hard

or soft. Sometimes such enlargements exercise considerable pressure on the trachea; at other times, particularly if the disease is malignant, the recurrent laryngeal nerves may become implicated. In cases where there is difficulty in determining whether a tumour is connected with the thyroid, much assistance may be obtained from the fact that the gland and any tumour which is connected with it move up and down with the larynx during deglutition.

3. **Pulsations in the vessels** of the neck must be recorded. In aortic incompetence the carotid arteries are seen to pulsate forcibly. Women patients with hyperpiesia sometimes show kinking of the right common carotid artery which simulates aneurysm. In syphilis aneurysm of the innominate artery or of the carotids may occur but is rare. In exophthalmic goitre the superior thyroid arteries can often be felt pulsating excessively. The jugular veins may be distended in congestive heart failure. In retro-sternal goitre and malignant neoplasm of the mediastinum, distended veins may be seen all over the neck: cyanosis and œdema may accompany this sign.

4. **Unusual prominence of any muscle** or group of muscles in the neck should be described. Such prominence may be bilateral, as of both sterno-mastoids in emphysema, or unilateral, as in tonic wry-neck. A congenital sterno-mastoid tumour may be present, and, if unrecognized, may lead to much perplexity; whilst various cysts, abscesses, or developmental abnormalities may be encountered. Their recognition, however, is rather a question for surgical diagnosis.

5. **Rigidity of the neck** may be due to fibrositis, to rheumatism, to disease of the spinal column,

or to various nervous diseases, whilst spasmodic movements occur in clonic torticollis.

6. Any **bulging of the apices of the lungs** during a fit of coughing, or pulsations seen in the vessels, must be recorded.

7. **Boils and carbuncles** are very frequently situated on the back of the neck. As they are not infrequently present in cases of diabetes, the urine should be tested for sugar.

The character of a patient's **respiration** is often of great service in reaching a diagnosis and a prognosis. Under the name of *extra-auscultation* may be grouped together the various phenomena connected with respiration which are to be heard, apart from those revealed by the stethoscope, when standing at the bedside of the patient.

CLASSIFICATION OF THE PHENOMENA OF EXTRA-AUSCULTATION

I. Obstructive noises in the respiratory passages.

1. In the nose
   - *a.* Thickened mucosa, or accumulated secretion
   - *b.* Paralysis of alæ nasi.

2. In the back of the throat
   - *a* Nasal stertor. The soft palate strikes the back of the pharynx.
   - *b.* Oral stertor. The soft palate strikes the tongue, which has fallen back.

3. In the larynx
   - *a.* Swelling of cords.
   - *b* Paralysis or spasm of glottis.

   (Laryngeal stridor (almost invariably inspiratory).)

4. In the trachea
   - *a* Tracheal stridor (leopard growl).
   - *b.* Tracheal rattle (death-rattle).

   (Occur during both inspiration and expiration).

5. In the bronchi
   - *a.* Musical sounds (wheezing).
   - *b.* Crepitant sounds.

II. Cough.

1. Duration. Single coughs, repeated coughs, paroxysms.
2. Quality. Resonant or toneless, moist or dry, suppressed or free.

III. Hiccough

IV. Voice

1. Volume. 2. Quantity

When the **respiratory passages are obstructed** the normal quiet respiratory sound is replaced by more or less noisy breathing. When the obstruction occurs in the nose, either from mucus in the meatus, or from thickening of the mucosa which covers the turbinated bones, or from paralysis of the alæ nasi, the breathing is sniffing or bubbling in character. When the soft palate is relaxed, and especially when it is paralysed, it prevents the free passage of air between the mouth and thorax, and produces a snoring or stertorous sound. When the rima glottidis is obstructed from any cause, such as spasm or paralysis of the vocal cords or œdema of the larynx, stridulous breathing results. If a polypus or other tumour lies between the cords, there may either be stridor or simply noisy breathing. The trachea may have its airway narrowed by pressure from the outside, as in cases of tumour and especially of aneurysm, when the breathing becomes growling; or mucus may obstruct the lumen, producing a rattling sound. The "death-rattle," which occurs when weakness and insensitiveness combine to prevent any effort at expectoration, is a typical example of the condition. Obstruction in the bronchi gives rise to wheezing and crackling sounds. An important division of dyspnœic conditions may be made according as the difficulty in respiration is felt during the inspiratory or the expiratory period. Most cases of obstruction of the air-passages are

characterized by *inspiratory dyspnœa*, whilst many of the pulmonary causes of dyspnœa produce *expiratory difficulty*. As a common example of the latter one may cite the prolonged expiration in a case of bronchitis with emphysema. The breathing may be characteristic of diseases quite distinct from those of the respiratory system. Examples of this are found in the stertorous breathing of apoplexy, the hissing expiration of uræmia, and the dyspnœa or "air-hunger" of commencing diabetic coma, which affects both inspiration and expiration.

If **cough** is present, its character must be carefully noted. The first thing to observe in this connexion is whether the cough consists of independent explosive expirations, or is paroxysmal. The former occurs in early pulmonary tuberculosis, in granular pharyngitis, and in some forms of nervous irritation; the latter is often found in severe bronchitis, and is very typical in pertussis. One should also notice whether the cough induces pain or nausea, and whether its tone is resonant, or suppressed, or husky. In *common colds* the cough is at first short and dry, but as the quantity of secretion increases, the type becomes more paroxysmal, and the fit of coughing continues till the mucus is expectorated. In *bronchitis* the condition resembles that found in the last affection, but the paroxysms are more severe, and wheezing is often present. When due to *early tuberculosis*, the cough is frequent, short, and sharp, and is described as *dry* because there is no rattling of mucus associated with it. Later, when the caseous masses are breaking down, secretion is much more copious, and the cough becomes moist and paroxysmal. In severe cases actual vomiting may be induced. A *nervous cough* generally has the character of single, short, dry explosions, repeated at intervals, and a similar type

is produced by irritation of the peripheral nerves, whether the source of the irritation is found in a disordered stomach, or threadworms in the rectum, or is due to disease in the ear or to the discomforts of teething, or takes origin in the nerves of the pregnant uterus. Local conditions in the *throat* may be the cause of most troublesome and persistent coughing, and a careful observer will not fail to look for granular pharyngitis when the patient complains of constant hawking, or for a relaxed and trailing uvula, more particularly when the cough starts the instant the patient lies down.

In *pleurisy* and *pneumonia* (associated as it often is with more or less pleurisy) the cough consists of solitary dry, hacking, expulsive efforts, suppressed as much as possible to prevent unnecessary pain, but repeated frequently. In *laryngitis* and *croup* the cough may be simply noisy, but more often is either husky or stridulous. When the lumen of the trachea is encroached upon by a *mediastinal tumour* or an *aneurysm* there is generally a very resonant, brassy cough, aptly compared to the cry of a gander. When once heard, this is almost sufficient to clinch the diagnosis without further examination.

In *hysteria* the cough is often loud and barking, and gives the impression of being produced with the view of attracting attention. Such a cough is sometimes associated with hysterical aphonia. *Pertussis*, when it is fully developed, is dinstinguished by a most characteristic cough. There is first a long-drawn, almost stridulous inspiration, then a series of short, sharp, expiratory coughs, which follow each other with extreme rapidity. The face turns dark and the veins grow prominent, the child clings firmly to any support it can find, so as to give full play to the accessory muscles of respiration, and when at last the fit of

coughing ends it is followed by a long-drawn whooping inspiration. The severity of the paroxysm induces vomiting, and sometimes causes evacuation of the bladder and bowel.

Hiccough, which results from spasmodic contraction of the diaphragm, is a common enough disorder. It may be due to trivial causes, such as an attack of indigestion ; but it also occurs, and that most persistently, in many serious illnesses, when the symptom may become one of considerable gravity.

The **voice,** as well as the cough, should be studied. The chief points to observe are its strength, whether it is clear or husky, or whether aphonia exists. The voice may be nasal either through habit or in consequence of obstruction in the upper airways. A distinction should be made between open and stopped nasal tones, the former resembling the sound produced when the mouth is kept shut during phonation, the latter that heard when one speaks whilst holding the nose.

Temperature.—The hand laid upon the skin gives a certain amount of information as to the temperature, especially if there is no perspiration; but a far more accurate guide is found in the **thermometer,** the use of which should never be omitted, although just because the hand takes account of moisture as well as of the actual temperature, it may convey information which the thermometer fails to impart —e.g. the " pungent " dry heat of early pneumonia is most characteristic. In taking the temperature, the following practical points must be attended to :—

1. The thermometer must be accurate and of good quality. To ensure accuracy, it should be compared with a standard instrument. In Britain this is done at Kew, and certificates are issued which state the error of each individual instrument. In process of

time, however, and particularly if the thermometer has been recently made, molecular changes occur in the glass which tend to make the reading too high. Such changes are slight, and seldom attain a value of any clinical importance, though if great accuracy is necessary a fresh comparison should be made every two or three years. Also, if the bulb of the thermometer is made too thin, the glass will yield to pressure, and the patient may either purposely or accidentally compress it so much as to make the mercury reach to four or six degrees above the actual temperature.

2. The thermometer must be kept in position long enough to allow the mercury to reach the body temperature. Generally it is well to exceed the period which the instrument professes to require. In the case of the ordinary "half-minute" thermometer, it should be left in position for from two to three minutes if an accurate reading is desired.

3. In adults the temperature is taken in the mouth or in the axilla; in young children the thermometer should be placed in the fold of the groin, and the thigh flexed on the abdomen, or it may be inserted into the rectum. The temperature of the mouth and rectum is generally at least half a degree higher than that of the groin or axilla, but in old people the mouth temperature is often too low, and less trustworthy than that of the axilla. When it is taken in the latter situation, care must be exercised to keep the part as free as possible from perspiration, both during the observation and for a few minutes before it. Moreover, the arm should be drawn to the side for a short time before the thermometer is inserted, that the skin may not have been chilled by exposure to the air. When the temperature is taken in the mouth, the patient must breathe through the nose and keep

the lips firmly closed during the whole time of the observation.

4. Before inserting the thermometer, make an invariable rule of washing it in lotion or in cold water, and see that the mercury is well shaken down ; wash it again before replacing it in its case. In Great Britain the Fahrenheit scale is used, on the Continent the Centigrade.*

The temperature *should be taken at fixed times*, twice daily when possible, and at shorter intervals when fever fluctuates or runs high. Times that are convenient, and that fairly represent the daily conditions, are 9 a.m. and 7 p.m. In health the temperature has a daily range of from one to two degrees Fahrenheit, being lowest in the small hours of the morning, and gradually rising to attain its principal maximum somewhere about 5 or 6 p.m. Age exercises a rather marked influence on the temperature. In children it varies greatly with their time of life, and trivial causes produce great fluctuations. On the average, it is about half a degree higher than in adults. In the very old it is also slightly higher than in middle life, unless the circulation is weak, when the temperature may be considerably lower.

In diseased conditions marked **deviations from the normal temperature** are often present. Temperatures may be classified as follows :—

Normal † .	98°–99° F. or 36·6°–37·2° C
Subnormal .	Below 98° F. or below 36·6° C
Collapse . .	„ 96° F. „ 35·5° C.
Febrile . .	Above 99° F. or above 37·2° C
Hyperpyrexia .	„ 107° F. „ 41·6° C

* For a comparison of the two scales, *see* Appendix 1. p. 573.

† D. M. Lyon and A. L. Wallace point out that 98·1° is the mean axillary temperature of active, healthy persons, but that the mean temperature of patients at rest in bed, who are not suffering from fever, is a degree lower than this (*Brit. Med. Journ.* 1932, i. 980.)

By consecutive observations, taken at suitable intervals, it is easy to determine whether an abnormal temperature is constantly present, or only occurs at intervals. When the temperature rises quickly, the patient feels chilly in consequence of the incomplete response of the vaso-motor mechanism to the new conditions, and in marked cases **rigors** occur. If, however, the temperature remains continuously high, the rigor gives place to a feeling of heat, coupled with thirst, headache, and a rapid pulse. This is known as **pyrexia,** or fever.

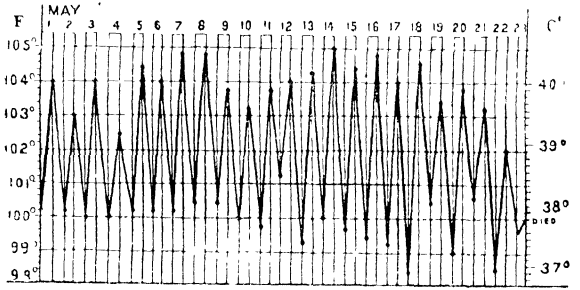


Fig. 1.— Remittent fever (hectic). Case of pulmonary tuberculosis.

If after fever the temperature falls rapidly, or if during the fever the extremities are chilled, the patient suffers from **collapse,** when the pulse is small, the features are pinched, the skin is moist with a clammy sweat, and the patient suffers from a sinking sensation and from nausea.

There are three principal **types of fever**—the continued, the remittent, and the intermittent. When fever does not fluctuate more than about a degree and a half (Fahrenheit) during the twenty-four hours, and at no time touches the normal, it is described as **continued.** When the daily fluctuations exceed two

degrees, it is known as **remittent** (Fig. 1); and when fever is only present for several hours during the day it is called **intermittent.** In remittent fever the evening temperature is usually higher than the morning one, but in some cases, not infrequently in pulmonary

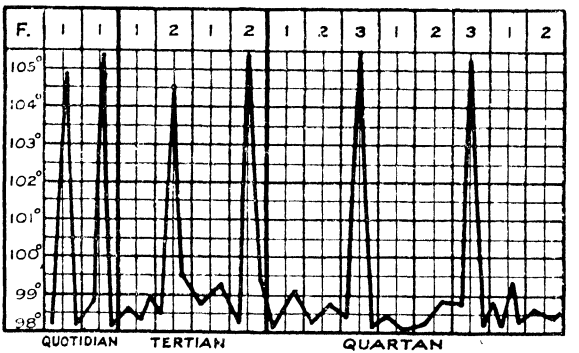


Fig. 2.—Intermittent fevers.

tuberculosis, this type is *inverted*, and the "remission" occurs in the evening, whilst there is a morning "exacerbation." When a paroxysm of intermittent fever occurs daily, the type is "**quotidian**"; when on alternate days, "**tertian**"; when two days intervene between consecutive attacks, "**quartan**" (Fig. 2). A "**double tertian**" is the name given to a daily fever when the paroxysms occurring on the first, third, fifth, and following odd days differ from those of the second, fourth, sixth, and following even days in hour of appearance, in severity, or in character.

The **course of a fever** is divided into three stages—the initial or pyrogenetic, "*stadium incrementi*"; the stage of full development, or "*fastigium*";

and the stage of termination, or "*stadium decrementi.*" When the fever ends rapidly it is said to resolve by **crisis** (Fig. 3); when gradually, by **lysis** (Fig. 4). Not seldom crisis is preceded by a short but marked rise of temperature, accompanied in many cases by delirium; it is sometimes followed by collapse.

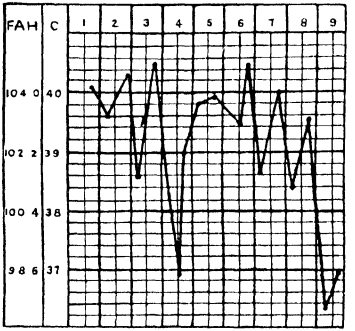


Fig. 3.—Crisis. Case of lobar pneumonia.

In the study of any case of fever the points which require to be observed are whether the type is one of apathy and indifference, or of restlessness and twitching; whether, and if so how far, the sensorium has been involved; what the height of the temperature is, and what its course has been; what are the rate and character of the pulse; whether the skin is moist or dry, or exhibits any eruption; and which of the viscera or secretions are characteristically affected. The explanation of these points may be found in works on medicine, but their true significance can only be learned at the bedside.

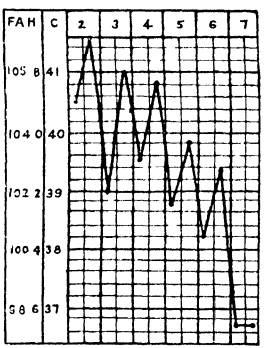


Fig. 4.—Lysis. Case of broncho-pneumonia.

CHAPTER III

THE ALIMENTARY SYSTEM AND ABDOMEN

I. THE MOUTH, THROAT AND ŒSOPHAGUS

The Mouth and Throat

The mouth.—For the examination of the mouth the patient should be placed facing a good light. If artificial light is used it should be thrown into the mouth by means of a reflector; or a pocket torch may be used for the illumination.

The lips.—Note the colour of the lips. They are blue in cyanosis, pale in anæmia. Note the presence of any crusts, fissures, or ulcers. The lips should be everted in order to permit of an examination of their inner surfaces. Herpes febrilis of the lips is often seen in inflammatory conditions of the air-passages and lungs, especially in lobar pneumonia.

The teeth.—The **temporary teeth** are cut in the following order :—

First.—The two lower central incisors, sixth to eighth month.
Second.—The four upper incisors, eighth to tenth month
Third.—The lower lateral incisors and all the front molars, twelfth to fourteenth month.
Fourth.—The canines (upper first), eighteenth to twentieth month.
Fifth.—Posterior molars, at two to two and a half years.

The **permanent teeth** appear as follows:—

First molars at six years.
Central incisors at seven years.
Lateral incisors at eight years.

Bicuspid (anterior) at nine years
Bicuspid (posterior) at ten years.
Canines at eleven to twelve years
Second molars at twelve to thirteen years.
Third molars at seventeen to twenty-five years

The following table shows the relations of the permanent and the temporary teeth :—

		M	C	I	I.	C	M	
TEMPORARY	Upper	2	1	2	2	1	2	20
	Lower	2	1	2	2	1	2	

		M.	Bi.	C.	I.	I.	C.	Bi.	M	
PERMANENT	Upper	3	2	1	2	2	1	2	3	32
	Lower	3	2	1	2	2	1	2	3	

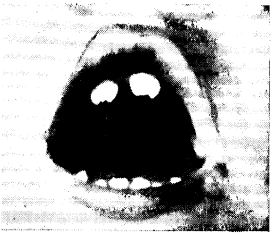
The presence of any irregularity or defect or carious disease in the teeth should be noted. It should be observed whether there is any exposure of their roots, or whether they are surrounded with tartar. Grinding of the teeth leads to bevelling of their edges; this is especially found in young children. The presence of "Hutchinson's teeth" affords (Plate 5) evidence of congenital syphilis. In this condition the two central upper *permanent* incisors are at a higher level than the adjoining teeth; they are rounded in section and slope inwards below, they are broader nearer the gum than at the crown, so as to be peg-shaped, and they present a semilunar notch at their ends. They are usually discoloured as well. In the same condition the molars tend to be dome-shaped.

The gums.—Their colour should first be noted. In patients exposed to lead compounds a blue line can often be observed running along the edge of the gum. It is seen especially opposite those teeth showing pyorrhœa. It may be distinguished from mere staining of the gum by the fact that, when examined with the aid of a lens, it is seen to be a stippled line made up of a large number of minute

blue spots, whereas staining is continuous. A similar stippled blue line may be seen after a course of some bismuth preparation given by the intramuscular route. The gums may be swollen and spongy in scurvy. Hæmorrhages may be observed in the buccal mucous membrane in thrombopenic purpura and acute leukæmia. In *pyorrhœa* the gums are retracted, frequently bleed easily, and pus may be observed exuding from between the gums and the teeth.

The tongue.—Ask the patient to protrude it. Note if it is put out in a straight line. Observe its size and shape and whether one half of it is wasted and deeply grooved—**lingual hemiatrophy.** Look for tremulousness of the whole tongue and for fibrillary twitching of it. Note in the dorsum (1) its colour: is it pale, red, or discoloured? (2) Is it dry or moist? (3) The presence or absence of fur; the colour and distribution of the latter should be noted. (4) The character of the papillæ: are they atrophied, leaving smooth bald patches, a condition seen in some types of anæmia? (5) The under-surface of the tongue—a small ulcer on the frænum is sometimes seen in persistent coughing, and particularly in whooping-cough. Lastly, (6) observe the edges of the tongue. Look for ulcers, indentations of the teeth, areas of leukoplakia and bald patches. (Plate 5).

The presence of **thrush** may sometimes be observed on the surface of the buccal mucous membrane, especially in children. It presents the appearance of small white points or patches raised somewhat above the surrounding surface, which is sometimes redder than normal. Patches of thrush are very apt to be mistaken for small milk curds. They may be distinguished by the fact that milk curds can be easily detached, while thrush patches can only be removed with difficulty, and when removed are apt to leave behind



Hutchinson's Teeth



Gums in Scurvy



Lingual Hemiatrophy



Congenital Fissures

Plate 5.—MOUTH.



Collateral Venous Anastomosis



Tophi of Gout



Rickety Rosary

Plate 6.

a raw surface. To search for the fungus (*Saccharomyces albicans*) a small piece of the patch should be scraped off and examined in a drop of glycerin. A quantity of epithelial debris, along with bacteria and leucocytes, will be seen, and mixed up with these the filaments of the fungus. These consist of long but unequal segments, each usually possessing a refractile nucleus at each end.

The palate, fauces, and pharynx.—Introduce a tongue depressor, and note first the general colour of the soft palate, fauces, and pharynx, observe any abnormal degree of pallor or redness. Remember that great insensitivity of the palate and pharynx is common in hysterical patients. The yellow tinge of jaundice often lingers long on the soft palate, and in commencing measles a patchy redness can be made out very early in the same situation. Note the presence of any ulcers or mucous patches on the palate, fauces, or tonsils. Instruct the patient to make the sound "Ah". This raises the soft palate and increases the visibility. Look carefully at the **tonsils,** noting any enlargement. Yellowish or greyish points or patches may sometimes be seen on their surface. Try whether these can be wiped off, leaving a sound surface, as is the case with accumulated follicular secretion, or whether removal leaves behind a raw surface, as happens with the false membrane of diphtheria. Note always whether or not the soft palate and uvula show any similar spots or patches. Next look at the **pharynx.** The presence upon its surface of a number of flat adenoid swellings, somewhat resembling sago grains, is so common as to be almost a normal appearance. In granular pharyngitis these are much increased. A few dilated venules can also be frequently observed. Note the presence of any pus or excess of mucus on

the surface and the existence of any ulceration. In retropharyngeal abscess the posterior wall of the pharynx is bulged inwards. Sometimes this can be more easily made out by palpation.

The breath.—The character of the breath, may be noted at this stage. If it is offensive, ask the patient to breathe out first through the nose only, and then through the mouth, and observe whether the odour is present on both occasions or not. This affords an indication as to whether the source of the odour is in the nose or mouth only, or whether it is lower down than either. If the odour proceeds from the nose, make a rhinoscopic examination (p. 505), looking especially for the presence of a foreign body or for evidence of atrophic rhinitis or other local disease. Bad teeth, ulcerations of the gums or mucous membrane, and enlarged tonsils, accompanied by retention and decomposition of secretion in their follicles, are the commonest sources of offensiveness in the mouth.

In gangrene of the lung the breath often has a putrid smell, resembling that of the sputum. In bronchiectasis, also, it sometimes has a peculiarly offensive odour only to be recognized by experience. Fetor due to pulmonary conditions is best brought out by asking the patient to cough.

Slighter degrees of offensiveness may be due to gastric disorder or to prolonged constipation.

In uræmia the breath may have a urinous or ammoniacal odour. In cases where diabetic coma is impending, the odour of acetone may be present. Various drugs—e.g. turpentine, creosote, paraldehyde, etc.—impart their characteristic odours to the breath, while in the case of patients who are taking bismuth a garlicky odour can sometimes be observed. Iodides produce a peculiar fetor.

The Œsophagus

Special anatomy.—The œsophagus is from 9 in. to 10 in. long. It begins opposite the cricoid cartilage, and ends opposite the 9th thoracic spine. It is crossed by the left bronchus between the 4th and 5th thoracic vertebræ.

In cases of **difficulty in swallowing** the radiologist must be asked to screen the patient and to report upon the course down the œsophagus of an emulsion opaque to X-rays.

II. THE ABDOMEN

Anatomy.—The natural lines on the surface of the abdomen are (1) the linea alba; (2) the lineæ semilunares; (3) the lineæ transversæ.

The **linea alba** is often selected as the site of puncture in tapping the abdomen. The structures lying behind it, from above downwards, are (*a*) the left lobe of the liver, extending to about three fingers' breadth below the ensiform; (*b*) part of the stomach, unless when empty; (*c*) the transverse colon, reaching as low as the umbilicus; (*d*) coils of intestine covered by omentum; (*e*) the bladder when distended, and the uterus when pregnant.

The **linea semilunaris** runs from the lowest part of the 7th rib to the spine of the pubes. It is about 3 in. from the umbilicus, but lies farther out when the abdomen is distended. The gall-bladder lies just to the outer side of the linea semilunaris of the right side.

Of the **lineæ transversæ,** one is opposite the umbilicus, another at the ensiform, and a third midway between these points.

In addition to these markings, the abdomen has been artificially divided into **regions** by means of vertical and horizontal lines. The vertical lines are

drawn upwards from the mid-point of Poupart's [the inguinal] ligament on each side. The transverse lines are (1) the subcostal or infracostal, drawn across horizontally at the level of the lowest points of the 10th costal arches, and (2) the intertubercular or bi-iliac, between the tubercles marking the most prominent points of each iliac crest. Nine regions are thus marked off in three vertical rows. Those in the middle row are, from above downwards, the epigastric, umbilical, and hypogastric, and in each lateral row we have the (right or left) hypochondriac, lumbar, and iliac regions. The contents of these nine regions are exhibited in the table on the next page.

The *umbilicus* is $1\frac{1}{4}$ in. to $1\frac{1}{2}$ in. above the level of the bi-iliac line, and lies opposite the upper part of the 4th lumbar vertebra. Its position is far too variable for it to be a trustworthy landmark.

The *aorta* bifurcates about $\frac{3}{4}$ in. below and slightly to the left of the umbilicus, the *iliac arteries* running in a line drawn from that point to a point midway between the anterior superior spine and the symphysis pubis.

The *cœliac axis* arises at a point $4\frac{1}{2}$ in. to 5 in. above the umbilicus, and the *renal arteries* about an inch lower than the cœliac axis.

The *transpyloric plane* is often used as a guide in the examination of the abdomen. It is defined as lying midway between the suprasternal notch and the upper border of the symphysis pubis. It usually lies about halfway between the xiphisternal junction or apex of the infrasternal fossa and the umbilicus, and it corresponds posteriorly with the lower border of the 1st lumbar vertebra.

General Examination of the Abdomen

The patient should be lying on his back in a good light. The abdomen is exposed by turning down all

Contents of the Abdominal Regions

Right Hypochondriac	Epigastric	Left Hypochondriac
Most of R. lobe of liver.	Part of R. lobe of liver.	
	Whole of L. lobe of liver (usually).	Part of L. lobe of liver (sometimes)
	Gall-bladder.	
	Part of stomach, including orifices.	Part of stomach.
Hepatic flexure of colon.		Splenic flexure of colon.
	1st and 2nd parts of duodenum	
	Pancreas and upper end of spleen	Tail of pancreas and most of spleen
	Parts of the kidneys.	
Part of R. kidney.	Suprarenals.	Part of L. kidney

Right Lumbar	Umbilical	Left Lumbar
Part of R. kidney.	Part of R. and sometimes both kidneys.	Part of L. kidney (sometimes).
Ascending colon	Most of transverse colon.	Descending colon
	3rd part of duodenum	
Part of ileum (sometimes).	Coils of jejunum and ileum.	Part of jejunum.
	Part of mesentery and great omentum	
	Part of stomach	

Right Iliac	Hypogastric	Left Iliac
End of ileum.	Coils of ileum.	Coils of jejunum and ileum
Cæcum and vermiform appendix.	Upper part of rectum and sigmoid loop	Sigmoid flexure.
	Bladder in children and (if distended)	
	Gravid uterus [in adults	

the bedclothes except the inner sheet. The clothing should then be drawn up, and, lastly, the sheet folded down a little above the level of the pubes. These details are of especial importance in the case of female patients. Before beginning the examination of the abdomen, make sure that the bladder is empty. Flexion of the hips is of no help. Better relaxation is sometimes obtained in a semi-recumbent position, but access to the abdomen is naturally less since the costal margin is then made to approximate to the pelvis. Nearly always the abdomen is best examined with the patient fully recumbent.

Inspection of abdomen.—Look first at the general contour of the abdomen. Is it of normal fullness, is it swollen or protuberant, or is it sunken or retracted? If there is any bulging, note if it is general or local. General fullness, it has been remarked, may be due to "fat, fluid, flatus, or fæces." If one were to venture to improve upon this, it would be to add "fœtus" as a possibility in the case of women. It must further be remembered that a *new growth* may also be a cause of general abdominal tumidity. The mode of distinguishing these conditions will be considered when we come to the consideration of ascites. In general bulging it should be noted whether the distension is most marked in the antero-posterior or in the transverse diameter. In cases of general abdominal swelling measurement should never be omitted as it affords a valuable index of the progress of the case. The circumference may be taken at the level of the umbilicus or at the point of maximum distension.

If the bulging is merely local, observe in which zone it is situated. Is it above or below the level of the umbilicus, and in which of the abdominal regions is it most marked? Lastly, note if there is any move-

ment to be seen in the swelling, either along with or independently of respiration.

Pulsation in the epigastric region is a phenomenon which may be noticed on abdominal inspection. The causes of it are in order of frequency:—(1) Aortic pulsation. This may be visible in any slightly-built patient with a thin abdominal wall. (2) Transmitted pulsation from a tumour (often a carcinoma of the stomach) overlying the aorta. (3) Distension of the right ventricle. (4) Venous pulsation of the liver. (5) Aneurysmal; the pulsation in this case is expansile.

The **movements of the abdominal walls** should be studied. Normally, they bulge during inspiration, and fall in again during expiration. In paralysis of the diaphragm the reverse holds true; sometimes the paralysis is unilateral, in which case one side of the abdomen will move naturally. Cessation of movement of the abdominal walls is a valuable sign of peritonitis.

Sometimes **peristaltic waves** are visible through the abdominal wall. This is especially apt to be the case in chronic intestinal obstruction. The coils of intestine above the constricted part then stand out prominently. From this a definite "pattern" of abdominal tumidity results, depending on the site of the obstruction. If, for example, there is a constriction at the ileo-cæcal valve, the distended coils of small intestine may often be observed standing out in the centre of the abdomen one above the other, so as to form a "ladder pattern." On the other hand, if the obstruction is low down, say in the sigmoid flexure, the pattern of tumidity is one in which the periphery of the abdomen is chiefly affected. A dilated stomach may also stand out as a prominent tumour in which peristaltic waves are visible. The direction of such waves should always be noted. If

absent, they can often be elicited by flicking the surface with a wet towel, or by merely sharply tapping it with the finger. Peristaltic waves in the stomach run from left to right; those in a distended transverse colon, from right to left. This may sometimes be of diagnostic value. The viscus undergoing peristalsis should always be palpated to determine whether it hardens under the hand.

Visible peristalsis is an important sign. It will not be observed if inspection is restricted to a casual glance. Frequently it may be necessary to sit and observe the abdomen carefully for several minutes. In the case of congenital pyloric stenosis of infants visible peristalsis may be the all-important diagnostic sign, and the minutes thus spent are never wasted.

Attention should next be paid to the **surface of the abdomen.** In great distension the surface is smooth and glossy *Striæ* (white or lilac lines in the epidermis) should be looked for; they indicate former distension Note any *distension of the surface veins*, and endeavour to ascertain in what direction the blood in them is flowing. In obstruction of the inferior vena cava the inferior epigastric veins are full from the establishment of a collateral circulation. In such cases also a large *lateral vein* can be seen running up about the midaxillary line, and thus establishing a communication with the tributaries of the superior vena cava. In portal obstruction a number of distended veins may sometimes be seen radiating from the umbilicus. They may be brought out only on coughing. To this appearance the term "caput Medusæ" has been applied. It is due to establishment of a connexion between the portal and parietal veins by means of the round ligament. *Pigmentation* of the abdominal wall is sometimes important. Along the middle line it forms the linea nigra—one of the

signs of pregnancy. Note the appearance of the *umbilicus*. Is it depressed, level with the surface, or bulging? Is there any excoriation around it? Lastly, one should never omit to look at the usual sites for any evidence of hernia

Palpation of the abdomen. – The patient should be on his back, with the shoulders a little raised. He should be told to keep the mouth open and to breathe quietly, or his attention may be diverted by conversation. The observer must sit or kneel beside the patient in order to get his hand into the horizontal position. Ordinary palpation is performed with one hand only. The hand must be warm. In order to gain the confidence of the patient the hand should be allowed to rest for a moment on the surface of the abdomen before palpation is actually begun. Each region should be palpated systematically. Poking with the finger-tips should be avoided, the best movement being a gentle one from the metacarpo-phalangeal joints During expiration the receding abdominal wall should be followed by the fingers, and a gentle rotatory motion of the finger-tips may then be carried out. It often enables one to feel the deeper structures better than one can do by simple pressure. In examining the lateral regions of the abdomen, bimanual palpation is often of service. The physician should sit or kneel by the bedside. One hand is placed posteriorly in the interspace between the last rib and the crest of the ilium. The other is placed over the abdominal wall in front. The posterior wall is then pushed up against the hand in front, so that any structure lying between the two hands can be distinctly felt. The secret of the method consists in keeping the front hand as still as possible. This procedure is of special value in the examination of the kidneys.

The first thing to notice in palpation of the abdomen is the degree of tension of the walls and of *resistance* experienced. Begin always by a systematic very light palpation of the whole abdomen, noting any local or general resistance and any marked tenderness. In this way the patient's confidence is gained and the later deep palpation rendered easier. Normally, the abdomen has an elastic or doughy feeling, only to be learnt by experience. In disease the resistance may be increased. It should be observed whether this increase is general or local. General peritonitis produces a great increase in the resistance from a reflex contraction of the muscles of the abdominal wall. Local increase in resistance is very frequently due to localized peritonitis, and is often of great diagnostic value. Palpation of the normal abdomen is painless. If tenderness is elicited its exact extent and point of maximum intensity should be noted. Anything in the nature of a tumour should be carefully felt for. In doing this, confusion is apt to be brought about by the recti. The thickening produced by parts of these may easily simulate a tumour. If this source of fallacy is suspected, try if the fingers can be got under the edge of the muscle, and feel if it thickens as the patient raises himself in bed.

If it is decided that a tumour is really present, one has first to determine whether it is situated inside the abdomen or in the abdominal wall. Try, therefore, to move the abdominal wall from side to side over the tumour. If the growth is intra-abdominal, this can usually be done without difficulty, unless it has contracted adhesions to the parietal peritoneum. Try also to grasp the tumour and to make the fingers meet, as it were, under it. This can usually be accomplished in the case of tumours situated wholly in the abdominal wall.

Supposing the tumour to be intra-abdominal, the first question to be settled is—Where is it growing from? and, especially, is it coming up out of the pelvis, or is it truly abdominal? To decide this the edge of the hand should be pushed back about an inch below the umbilicus, and in the direction of the prominence of the sacrum. One can then feel whether the tumour is passing down into the pelvis or not. The size and shape of the tumour should next be noted, and the nature of its surface—whether smooth or nodular. The presence or absence of fluctuation should then be investigated.

The *mobility* of a tumour is a very important point to determine. The directions in which it can be moved should be noted, and whether it is influenced by respiration. The latter is a point of special value. Tumours connected with the liver and spleen move freely with respiration, and so may those of the stomach. Tumours of the kidney may be slightly movable. Those connected with the other abdominal organs do not move with respiration at all unless they have contracted adhesions.

In palpating the abdomen, the existence of *splashing or gurgling* at any points should be noticed. Splashing is often found over a dilated stomach, but is only of diagnostic value if it can be elicited some hours after the swallowing of food. Gurgling is produced by the passage of gas and fluid through constricted parts of the alimentary tract. It may thus be felt at the pylorus, especially if stenosed, or over strictures of the intestines.

Percussion of the abdomen.—This should be carried out in the same manner as will be described for the chest, but particular care should be taken to percuss *lightly;* students usually tend to use too heavy a stroke. Percussion of the normal abdomen

yields a tympanitic note throughout, except in the regions of liver and splenic dullness, or over a full bladder. The percussion pitch of the hollow viscera depends on two chief factors—(*a*) the depth of the air-space; (*b*) the tension of the containing wall.

As these two factors are of almost equal importance, and as each of them varies greatly in the same viscus at different times, the reader will readily understand that it is a mistake to dogmatize about the relative pitch of the note yielded by the various hollow viscera. The presence of free gas in the peritoneal cavity causes the normal liver and spleen dullness to disappear.

If any abnormal dullness is detected, the chief point to be determined regarding it is whether it is constant in position or shifts with alterations in the position of the patient. This will be more fully discussed when we come to speak of ascites.

In about 50 per cent. of cases hydatid cysts yield on percussion a special kind of vibration called the "*hydatid thrill*." To elicit it, three fingers should be placed over the cyst, and the middle one firmly percussed, the percussing finger being allowed to rest for a moment after each stroke. An "after thrill" will then be experienced in the two adjacent fingers.

The examination of cases which are believed to have fluid in the peritoneal cavity, or **ascites**, calls for special consideration. In cases in which the fluid is sufficient to cause general distension, the conditions for which one is apt to mistake it are, as we have seen, fat in the abdomen and abdominal wall, gas in the intestines or free in the peritoneum, and new growths. Fluid gives, of course, a dull note on light percussion. The dullness is not always absolute, however, owing to the transmitted resonance of subjacent bowel.

Free fluid is also distinguished by the fact that it shifts its position with that of the patient. If he is turned over on his side and time given for the intestines to float up, it will be found that the uppermost flank is now resonant, while the height of the dullness on the lower side has risen. If the fluid is very small in amount, it is a good plan to turn the patient on to his hands and knees. A dull area then appears around the umbilicus from accumulation of fluid there.

The "*transmitted thrill*" is another important physical sign of fluid in the peritoneum. It is elicited by placing one hand over the lumbar region of one side, the patient being on his back, whilst the opposite lumbar region is sharply tapped with the fingers of the other hand. A distinct impact will be felt to pass from one hand to the other. As a not dissimilar impulse is apt to be transmitted through the abdominal wall, especially if fat, it is necessary to get an assistant to place the edge of his hand firmly in the middle line of the abdomen while percussion is being made. This damps down any vibrations transmitted by the wall. Where the amount of fluid is large, the vibrations are visible as well as palpable. On the whole, we consider that the results of simple percussion afford the most trustworthy evidence of the presence of ascites. A fluid thrill can only be expected when the amount of fluid is large and under tension.

Fat is to be distinguished by taking the abdominal wall between the hands and pinching it up. *Gas* is distinguished by the results of percussion. Of *new growths*, ovarian cyst is, perhaps, most liable to be mistaken for ascites. An ovarian tumour, however, causes an antero-posterior bulging of the abdomen, while in ascites the bulging is mainly lateral. In ovarian tumours, also, the dullness is central, and does not change with the position of the patient; in

ascites the chief dullness is in the flanks, and it shifts, as we have seen, when the patient is moved. Lastly, in ascites the umbilicus is flat or bulges out, while in ovarian tumours it is drawn upwards.

Auscultation of the abdomen.—Normally on auscultation of the abdomen numerous borborygmi are audible. Sometimes, however, in cases of general peritonitis or of intestinal ileus these sounds may be entirely absent and the "silent abdomen" is then a valuable physical sign.

III. THE ABDOMINAL VISCERA

One may now pass to the examination of the viscera in the abdomen, beginning with the stomach.

The Stomach

Special anatomy (Plates 7, 10).—The normal stomach in the living subject is shaped like the letter **J**, and, when empty, is situated within the limits of the left hypochondriac and the left half of the epigastric region. The cardiac orifice lies, as a rule, on the left side of the 11th thoracic vertebra, and 4 in. behind the 7th left costal cartilage, 1 in. from the sternum. The position of the pylorus is usually at, or just to the right of, the middle line and midway between the infrasternal notch and the umbilicus. It is normally under cover of the liver. The fundus of the stomach reaches as high as the 5th interspace in the midclavicular line, and rises a little above and behind the apex beat of the heart. Only a small part of the body of the stomach and of the pyloric region is in contact with the anterior abdominal wall. The exact position of the greater curvature varies greatly according to the degree of distension of the stomach and the posture of the patient.

Inspection of the stomach region is included in the general examination of the abdomen (p. 52).

Palpation of the stomach.—Note any *tenderness*, and define its point of greatest intensity. Examine for *tumours*. The commonest of these is a carcinoma of the pyloric end of the stomach. Tumours of this region are characterized by their great mobility. They may be felt in, or pushed into, any region of the abdomen. Lastly, try for *splashing*. To make this out, sit at the left side of the patient with one hand over the left lower ribs behind ; with the other placed over the front of the stomach, make short, sudden dipping movements. If " splashing " is elicited it will be partly heard and partly felt.

Distinct splashing elicited three hours after a meal, especially if it can be made out below the level of the umbilicus, is very suggestive of a dilated stomach. It should be remembered, however, that a splash may be elicited over even a normal stomach shortly after a meal containing much fluid, especially if the abdominal wall is thin; and care should be taken not to mistake a splash produced in the transverse colon for a stomach splash.

The Liver

Special anatomy (Plates 7, 9).—The liver lies chiefly in the right hypochondrium. Its left lobe extends across the epigastric region, but does not pass more than 2 in. to the left of the sternum. Above, the liver reaches almost to the nipple; below, it extends to the costal margin. The lower border passes obliquely upwards from the 9th right to the 8th left costal cartilage, crossing the middle line somewhat above the midpoint between the base of the xiphoid and the umbilicus.

The gall-bladder is situated just internally to

the 9th right costal cartilage, and immediately to the outer side of the right rectus muscle.

Inspection of the liver is of little value. Any visible swelling, fullness, or pulsation should be noted. The edge of the liver can sometimes be seen when the organ is enlarged. It forms a sharp line which moves up and down with respiration.

Palpation of the liver.—The lower edge should first be felt for. In order to do this, place the hand flat on the abdomen, with its edge towards the costal margin and just to the outer side of the rectus muscle, the reason for going so far out being to avoid the upper septum of the rectus sheath, which is apt to be mistaken for the lower edge of the liver. Then depress the edge of the hand slightly so as to push up a fold of skin, and ask the patient to take a long breath. If the edge of the liver is palpable, it will be felt to ride over the edge of the hand. Trial, of course, must be made at different levels before it is decided that the edge cannot be felt. The edge of the liver cannot, or can only very rarely, be felt in health. It moves down from two-fifths to three-fifths of an inch with inspiration. The character of the edge should also be noted —whether it is smooth or irregular, thickened or sharp. If in doubt whether what is felt is really the liver edge, feel for the fissure of the gall-bladder, and, towards the middle line, for that produced by the round ligament.

The **surface of the liver** in the epigastrium should then be felt in the usual way. Any tenderness should be noted, and whether it is localized or general. The character of the surface should be made out. Is it smooth, as in amyloid disease, or nodular, as in carcinoma? In the latter condition the centres of the nodules will sometimes be found to be umbilicated. Care must be taken not to confound little irregularities which are frequently present in the upper parts of the

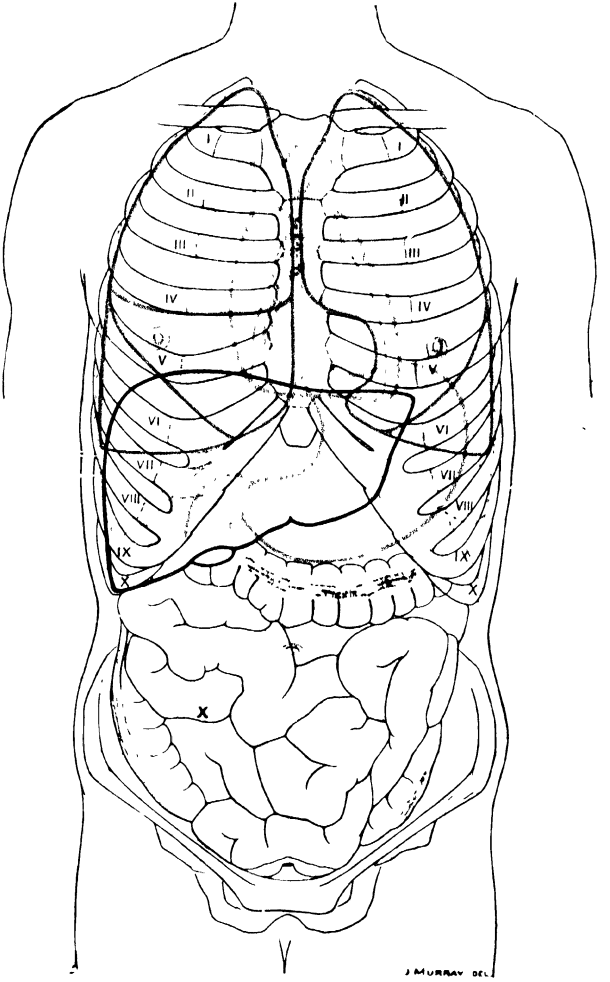


Plate 7.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE FRONT IN THE CADAVER. Scale: 1 = 5.6.

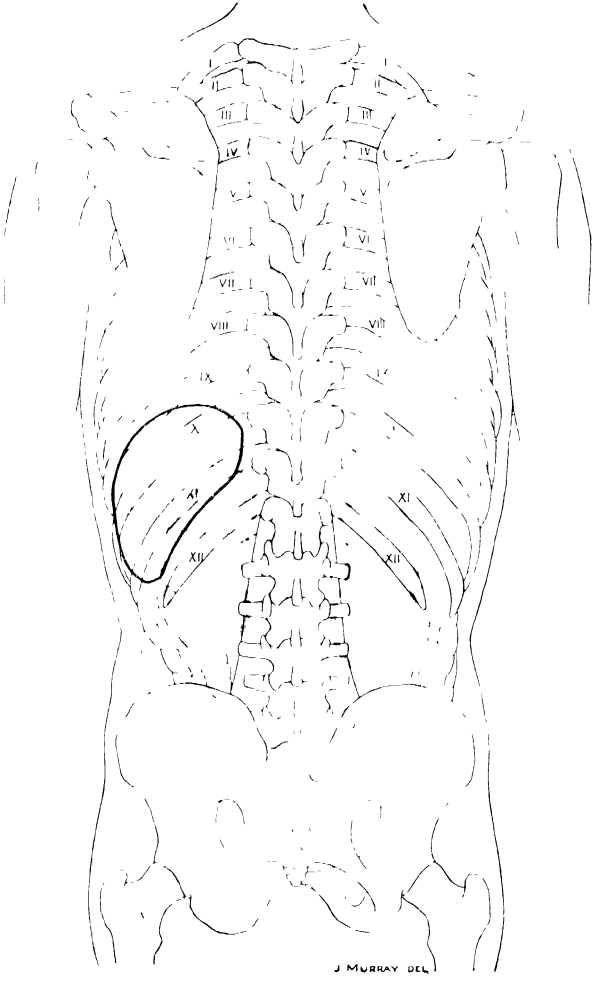


Plate 8.—Viscera of Thorax and Abdomen, as seen from Behind in the Cadaver. Scale 1 = 5·6

recti with irregularities on the surface of the liver. Liver friction (due to perihepatitis) can sometimes be felt. It is usually best made out over the posterior surface of the organ between the vertebræ and the midaxillary line.

Heaving pulsation of the whole organ can best be appreciated by placing one hand over the lower right ribs and the other over the organ in front.

Percussion of the liver.—The patient should be lying down for percussion of the anterior and lateral aspects; sitting up or standing for the posterior aspect.

To make out the dullness, use fairly heavy percussion. Begin high up—say about the 2nd rib—so as to get a good lung note, and percuss down from rib to rib till impairment is detected. Then repeat the process, going from space to space instead of from rib to rib. Percuss in this way down the mammary, midaxillary, and scapular lines.

The upper limit of liver dullness in the middle line cannot be distinguished from the heart dullness. To map it out, draw a straight line from the apex beat to the angle where the right edge of the heart and the deep liver dullness meet. The upper limit of liver dullness forms an almost horizontal line around the chest.

In defining the lower edge of the liver, use very light percussion, and pass upwards.

The exact position of the lower edge of the liver is extremely variable. Usually it coincides with the costal margin in the mammary line. It may be considerably above or below this, however, without there being any pathological change in the organ. Its position in the middle line is also very variable. As a rule, it is situated about a hand's breadth below the base of the xiphoid.

In percussing the surface of the liver where it is

not covered by lung, it should be observed that the organ has a certain degree of *resistance* or *resilience*. The normal amount of this can only be learnt by practice. If the organ is enlarged or congested, its resistance to percussion is increased owing to its being more firmly pressed against the chest-wall.

The liver may be displaced, enlarged, or diminished.

Displacement may be either upwards or downwards. Upward displacement may occur from tumours, etc., in the abdomen pushing the liver up. Downward displacement may be brought about by right pleural effusion, by emphysema of the lungs, or, more rarely, by effusion below the diaphragm, for example, subphrenic abscess. When the liver is dislocated downwards, the rounded upper surface of the left, and part of the right lobe, can usually be made out crossing the epigastrium. A displaced liver, also, does not move freely with respiration, while a liver which is merely enlarged does.

One must distinguish between real enlargements and diminutions of the liver and those which are apparent only.

Thus, *enlargement* of the liver may be simulated by consolidation of the base of the right lung, or by effusion into the right pleura. Downward enlargement may be simulated by accumulation of fæces in the transverse colon.

An hydatid cyst in the liver often produces an enlargement of the organ upwards rather than downwards.

Diminution of the liver may be simulated by the organ being covered up by an emphysematous lung, by free gas in the peritoneal cavity, or by the colon passing up between it and the abdominal wall. The latter is a rare condition. It should be suspected if the lower limit of liver dullness varies very much at different points.

The **gall-bladder** is examined by palpation and percussion. It cannot be *felt* unless distended. It may then form a smooth, pear-shaped swelling, situated just to the outer edge of the right rectus muscle. It can be moved freely from side to side round a point opposite to the 9th costal cartilage. It also moves with respiration.

The gall bladder should always be felt carefully for *tenderness.* In order to elicit this the patient should sit up and lean forward whilst the examiner, standing behind and to the right of him, tucks his fingers in under the costal margin just outside the right rectus muscle. The patient is then told to take a deep breath. As the diaphragm descends the gall-bladder is driven against the fingers, and if it is tender the breath is at once arrested with a "catch." This is spoken of as **Murphy's sign.**

On *percussion,* a distended gall-bladder forms a dull area, projecting from the liver dullness towards the umbilicus, but usually continuous with it. Sometimes, however, the transverse colon comes to lie across the neck of the gall-bladder, so as to separate it from the liver. When this occurs, diagnosis of the tumour is apt to give trouble. To this point we shall recur when we come to the examination of the right kidney (p. 69).

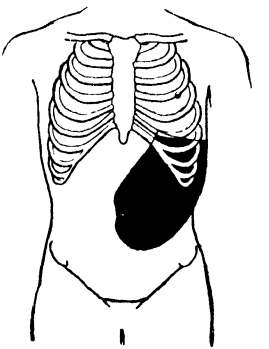


Fig. 5.—Enlargement of spleen.

THE SPLEEN

Special anatomy (Plates 8, 10).—The spleen lies in the left hypochondrium. It is bounded above by lung, elsewhere by

stomach and intestine. Its lower end rests upon the costo-colic fold of peritoneum. It lies along the 9th, 10th, and 11th ribs, being partially separated from them by the diaphragm and lower edge of the left lung. Its upper end is opposite the 9th thoracic spine, and reaches to about 1½ in. from the middle line. Its lower end comes as far forward as the midaxillary line.

Inspection of the spleen.—If much enlarged, the spleen may form a visible tumour in the left side of the abdomen, which moves with respiration (Fig. 5).

Palpation of the spleen.—This is really the most important method of investigating the spleen. If one can exclude dislocation, then a spleen which is palpable may safely be pronounced to be enlarged; and it is never safe to diagnose enlargement of the spleen unless it *is* palpable.

The spleen should be felt for in the following manner: Go to the right side of the patient. Place the fingers of one hand behind in the space between the ends of the 10th and 11th ribs. Place the other hand over the left hypochondrium, with the fingers slightly tucked in under the edge of the costal arch. With the posterior hand tilt the spleen forwards while the patient inspires. The edge of the organ will then be felt against the fingers of the other hand. Start well down towards the right iliac fossa and work up. Large spleens have been missed by starting palpation too near the costal margin. Sometimes in the case of minor degrees of splenomegaly the organ is best felt if the patient rolls over, half on to his right side towards the examiner.

The edge of the spleen is sharp and usually quite smooth. Notches can often be felt in it, but by no means invariably. It is important to note (1) that the anterior border of an enlarged spleen is always directed

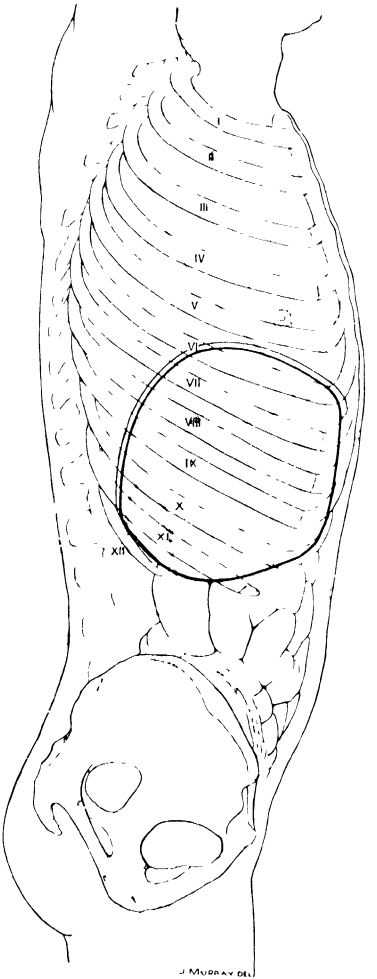


Plate 9.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE RIGHT SIDE IN THE CADAVER. Scale 1 = 5·6

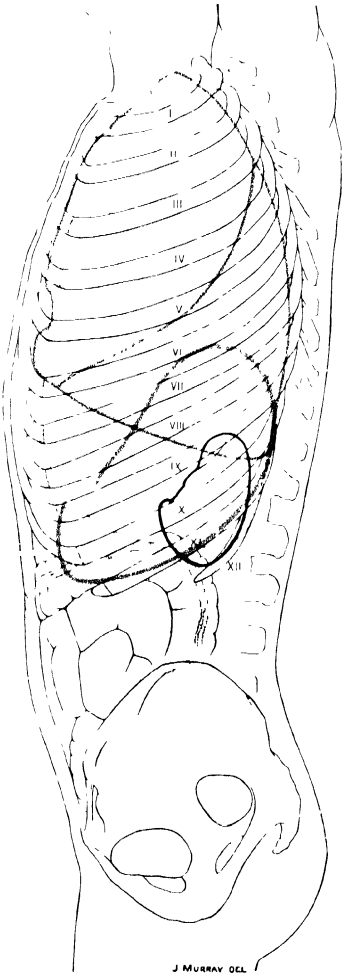


Plate 10.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE LEFT SIDE IN THE CADAVER. Scale 1 = 5.6.

downwards and inwards, and (2) that there is always a slight space between the posterior edge of the spleen and the erector spinæ, into which the fingers can be dipped. Occasionally the spleen enlarges upwards only. This may happen where the costo-colic fold is abnormally well developed, and keeps the organ up. For the detection of such a condition one must have recourse to percussion

Sometimes, on the other hand, when the spleen gets very large it pushes down the costo-colic fold and then drops down itself. A spleen which at one time crossed the middle line may cease to do so by dropping down in this way, though the organ is really larger than ever.

Auscultation over the spleen may be practised to detect the existence of friction. The latter occurs in perisplenitis and over the surface of splenic infarcts.

The Kidneys

Special anatomy (Plates 8, 9).—Each kidney lies partly in the epigastric, partly in the hypochondriac region. The right kidney lies partly in the lumbar region as well. As regards their relation to the anterior abdominal wall, the kidneys are higher up than one is apt to suppose. The lower end of the right kidney is fully 1 in. above the umbilicus, the left is about ½ in. higher. The lower end of each is about 3 in. from the middle line.

As regards their posterior relations, about one-third of each kidney lies above the last rib. The upper end of the right kidney is at the level of the 11th thoracic spine, whilst its lower end reaches to about 1 in. above the iliac crest. The left kidney is about ½ in. higher.

Palpation of the kidneys.—The patient must be on his back with the knees slightly flexed, and the shoulders raised on a firm pillow. The lumbar

region must be quite flat on the couch, not arched forward. Sit or kneel beside the patient on the side to be examined. Place one hand upon and below the last rib behind, the other immediately below the costal margin in front. The posterior hand should press the loin forwards, while the other hand pushes the anterior abdominal wall backwards, upwards, and outwards. The kidney will then be felt between the two hands if it is at all enlarged or displaced. Even in health (provided the patient is not too fat) the lower part of the organ can often be felt.

Should this manipulation not succeed, the patient must be asked to take a long sighing inspiration. If the front hand follows up the receding abdominal wall as the air leaves the chest, the kidneys will be caught between the two hands.

The kidney moves very slightly with respiration. An exaggeration of this normal mobility, so that the organ slips up and down like a pea in a pod, constitutes *movable kidney*. This must be distinguished from *floating kidney*, in which the organ moves freely forward so as to " float " towards the anterior abdominal wall, as well as moving vertically and laterally. The palpability of the kidneys varies greatly with the build of the patient. In health the right kidney is frequently just palpable in persons of spare build, the left very infrequently so.

A floating right kidney is very apt to be mistaken for a distended gall-bladder, and vice versa. The shape, size, and consistence of the tumour may be apparently identical in the two cases. One point of distinction is that while a distended gall-bladder can be temporarily pushed back from the abdominal wall, yet it always tends to spring forward again; it is therefore always in evidence. It is not so with a floating kidney; the latter often disappears for a

time, and can only with difficulty be got hold of again. Another point of distinction is that a kidney can be pushed down towards the pelvis and held there even during forcible expiration, whilst the gall-bladder moves upwards again during the expiratory act. The different relation of the colon to the kidney and to the gall-bladder should also be remembered.

An enlarged left kidney may be mistaken for the spleen. The points of distinction are : (1) The spleen has a sharp edge in which a notch can often be felt. The edge of the kidney is *always* rounded and has no notch. (2) There is no space between the posterior border of the kidney and the erector spinæ, as there is in the case of the spleen. That is, one can " get behind " a renal tumour but not in the case of an enlarged spleen. (3) The fingers can usually be passed between the upper end of a kidney tumour and the ribs but not between the ribs and a splenic tumour. (4) The colon lies between the kidney and the anterior abdominal wall, but not over the spleen.

An enlarged kidney tends to bulge forwards. Perinephric abscesses, etc., bulge backwards.

The Intestines

Special anatomy.—The small intestine occupies chiefly the umbilical and hypogastric regions ; the large intestine, the peripheral zone of the abdomen. The ileum joins the colon at a point 2 in. internal to, and somewhat above, the right anterior superior iliac spine. The apex of the cæcum corresponds to a point a little to the inner side of the middle of Poupart's ligament. The base of the vermiform appendix usually lies opposite a point $1\frac{1}{2}$ to 2 in. from the anterior superior spine along a line drawn from that spine to the umbilicus. This is sometimes called McBurney's point, because he showed that in the majority of

cases of appendicitis that is the point of maximum tenderness as determined by the pressure of one finger.

The splenic flexure of the colon lies behind the stomach, the hepatic lies under cover of the liver. The former is at a somewhat higher level than the latter. The transverse colon passes across the abdomen in a slightly curved direction, the lower part of the curve reaching to about the umbilicus.

Examination of the intestines by **inspection** and **palpation** has been described under the general examination of the abdomen (p. 52).

Percussion of the intestines.—The notes yielded by the small and the large intestine cannot be satisfactorily discriminated.

Rectal examination.—Place the patient in a good light and in a semi-prone position—i.e. resting on the left breast with the right thigh and knee well drawn up, the inner aspect of the right knee resting on the couch. Draw aside the glutei and inspect the region of the anus, noting the presence of any eruption, of external hæmorrhoids, etc. Fit a finger-stall to the right forefinger and smear it with vaseline. If no finger-stall is available fill the nail with soap and smear the finger with vaseline. Pass the finger slowly and gently through the anus, directing it slightly forwards at first. Note the degree of resistance offered by the sphincter; this shows whether the latter is normal, spasmodic, or relaxed.

Once the anal canal is passed, direct the finger slightly backwards and upwards, asking the patient to bear down a little at the same time. The finger can then be swept round and the whole inner surface of the rectum explored.

The prostate will be felt projecting into the rectum in the male, and above it is the trigone of the bladder flanked by the seminal vesicles; below

it is the membraneous urethra. In the female the cervix will be felt projecting back in the form of a firm rounded swelling. The mucous membrane must be examined for polypi, ulcers, and malignant neoplasm.

It must be remembered that hæmorrhoids are not palpable. The presence of scybala or foreign bodies can be determined. If the lymphatic glands which lie in the hollow of the sacrum are enlarged, they can be felt. If secondary malignant deposits or an abscess be present in the pouch of Douglas the corresponding mass will be palpable through the wall of the rectum. On withdrawing the finger examine the fingerstall for the presence of mucus, blood, or melæna.

Proctoscopy and sigmoidoscopy.—If rectal examination is negative and there is reason to suspect abnormality close to the anus, the anal canal and lower three inches of the rectum should always be examined by means of the proctoscope. With the patient in the position previously described for rectal examination, the instrument, after warming and lubricating, is passed carefully to its full depth. The obturator is removed and the mucous membrane inspected as the instrument is slowly withdrawn. In this manner hæmorrhoids may be seen or the nature of a dubiously palpable abnormality directly ascertained.

It is frequently necessary to examine the rectum and colon more fully than is possible by proctoscopy, and in such cases the sigmoidoscope is employed. Sigmoidoscopy requires a modicum of skill and experience and the technique will not be described here. It is worth while to remember, however, that the sigmoidoscope in skilled hands can be passed for 16 cm. and that a further 4 cm. of the colon is visible beyond this—20 cm. in all. The procedure causes very little discomfort, and anæsthesia is unnecessary and undesirable.

Sigmoidoscopy is particularly useful in the differential diagnosis of diarrhœa of colonic origin. It serves to distinguish carcinoma of the colon, the ulcerated bleeding mucosa of ulcerative colitis, the polypi in polyposis coli, and the red granular surface in granular proctitis.

IV. INVESTIGATION OF THE GASTRIC FUNCTIONS

The object of the investigations now to be described is to test the digestive and motor power of the stomach.

The patient has a light supper and a charcoal biscuit on going to bed, the tube is passed the first thing next morning, and the stomach completely emptied. The contents of the fasting stomach thus obtained furnish useful information. The presence of *food* or particles of charcoal indicates some degree of pyloric obstruction ; if there is over 5 oz. of *clear gastric juice* without food, hypersecretion exists; if on washing out the stomach flakes of *mucus* are obtained which sink in the wash-water, one may diagnose gastritis. A foul smell implies either gross pyloric obstruction or extensive carcinoma. The presence of blood supports the latter diagnosis.

1. The Ewald Test-meal

The next step is to give a test-breakfast consisting of two slices of toast and two cups of weak tea. It is withdrawn an hour afterwards.

Method of withdrawal.—Pass the stomach-tube, which should be connected with a glass or vulcanite funnel. The patient should sit on a chair placed on a square of waterproof sheeting. He should be wrapped in a mackintosh and sit with his legs apart, so that anything spilt on the mackintosh may run into a pail placed between them.

When the tube has reached the stomach, ask the patient to retch or cough, the end of the funnel being depressed at the same time. In this way a sample of the contents can usually be obtained and caught in a clean vessel. In difficult cases, removal of the contents is greatly facilitated by the use of *Senoran's stomach-aspirator*.

When the stomach contents have been obtained they should be allowed to settle in a tall jar, after which one can proceed to examine them. If necessary, they may be decolorized by shaking them up with animal charcoal.

The points that have to be investigated in the product are as follows :—

(1) Physical characters.
(2) Acidity.
(3) Microscopic characters.

(1) **Physical characters.**—A very small result from the test-meal, containing little fluid and imperfectly dissolved masses of food, indicates defective secretion or hypermotility. An abundant and very liquid yield points to excessive secretion or diminished motility, or both. When the contents consist of a large quantity of greenish fluid with a deposit of starchy material at the bottom, hypersecretion is present. A froth on the top of the fluid, with a yeasty odour, indicates fermentation from pyloric obstruction. A sour, acrid smell points to the presence of organic acids. If the contents are viscid and filter slowly, mucus is present in excess (gastritis).

The product of the test-meal should now be filtered through a folded filter-paper, and the filtrate used for the following tests.

(2) **Acidity.** (*a*) **Are the contents acid ?**—Test with litmus.

(*b*) **Is free HCl present?**—*Congo-red paper* (Appendix, 8) is turned blue. An approximate idea of the amount of acid present may be obtained from the depth of tint produced. Normal gastric juice turns the paper a sky-blue.

Gunzburg's test for free HCl.—Place 10 drops of the stomach contents in a porcelain capsule, add an equal quantity of the phloroglucin and vanillin solution, which must be freshly prepared (Appendix, 5). Heat gently, taking care to avoid charring. When almost dry, complete the evaporation by blowing on the surface of the fluid. If free hydrochloric acid is present a pink colour appears, usually at the periphery of the dried fluid.

The test may also be carried out by simply dissolving a few small crystals of vanillin and resorcin in a drop of the test-meal filtrate and evaporating to dryness (Panton).

The reaction is only given by *free* hydrochloric acid. The combined acid and organic acids do not yield it.

Boas's resorcin reagent (Appendix, 6) may be used similarly. It gives a purplish colour. This method is less sensitive but more economical than Gunzburg's.

Methyl-orange gives a red reaction with free hydrochloric acid, but is not such a delicate test as either of the above. It is best used in the form of test-papers soaked in a 0·5-per-cent. alcoholic solution of the dye.

(*c*) **Are organic acids present?**—These need only be tested for if free HCl is absent, or if there are indications of stagnation in the contents.

The reader is referred to a text-book of biochemistry for details of the technique.

(*d*) **Total acidity.**—Take 10 c.c. of the filtered gastric contents (in a beaker), add a few drops of Topfer's reagent, and cautiously run in decinormal caustic soda solution until the pink colour is discharged. Read the burette (first

reading). Now add a few drops of an alcoholic solution of phenol-phthalein. Again run in the soda solution, this time until the least trace of persistent pink colour appears in the beaker. This can best be appreciated by holding against a white surface. Read the burette (second reading). The first reading gives the amount of the "free acidity"; the second reading shows both the "total" and the "combined" acidity, the "total" by the number of cubic centimetres run in from the beginning of the titration, the "combined" by the number added after the first reading was made. The term "combined," as used here, includes acid combined with proteins, with enzymes, and with mineral bases in the form of acid salts. Acidity due to free organic acids will also be included in the second reading of the burette.

Should there be no free acid, the "deficit" may be determined, i.e. $\frac{N}{10}$ HCl is added to 10 c.c. of the filtered contents until free acid is present. Then, after adding phenol-phthalein, the acidity is determined with $\frac{N}{10}$ NaOH. In this case the "total" acidity is obviously the number of cubic centimetres of alkali used minus the number of cubic centimetres of $\frac{N}{10}$ acid previously added.

The results of these titrations may be stated in one of two ways: (1) Directly, from the number of cubic centimetres of $\frac{N}{10}$ NaOH required to neutralize 100 c.c. of the stomach contents; e.g. if to neutralize the 10 c.c. of contents 5 c.c. of $\frac{N}{10}$ NaOH were necessary, then for 100 c.c. of contents 50 c.c. of soda would be required, and the "total" acidity would be 50. If of this 5 c.c. of soda 3 c.c. were required to neutralize the free acid and 2 c.c. to neutralize the combined acid, the "free" and "combined" acidities would be respectively 30 and 20. Normally the total acidity varies from 40 to 70.

(2) Indirectly, in terms of HCl. Thus, one litre of decinormal soda is required to neutralize 3·65 grm. of HCl. If, therefore, 100 c.c. of stomach contents require 50 c.c. of soda to neutralize them, then the acidity of the 100 c.c. is equal to that of 0·18 grm. HCl—that is to say, the acidity is 0·18 per cent. The normal total acidity in terms of HCl is about 0·2 per cent. The necessity for calculation may be avoided

one remembers that, provided 10 c c. of contents have been taken and that decinormal soda is used for titration, the number of c.c. of soda required × 0·365 = HCl per 1,000 parts. In order to get the percentage of HCl, one has merely to shift the decimal point one place to the left. For example, if 10 c.c. of decinormal soda solution were required to neutralize 10 c c. of gastric contents, the amount of HCl present is 3·65 per 1,000 or 0·365 per cent.

(3) **Microscopic characters.**—Films are made in the usual way and stained with dilute gentian violet. Oppler-Boas bacilli, yeasts and sarcinæ are commonly seen.

Interpretation of test-meal.—If a test-meal is given to a healthy person and the contents are removed one hour afterwards, it will be found that 20 c.c. to 40 c.c. of fluid are obtained. This is transparent, straw-coloured, of an acidity equal to that of 0·2 per cent. or so HCl (40–70, Ewald scale) and contains free hydrochloric acid.

In some disorders of the stomach the total acidity of the contents is increased (*hyperchlorhydria*). Sometimes no free HCl is found (*achlorhydria*). In cases of carcinoma of the stomach the absence of free HCl is of sufficient constancy to be of diagnostic value. In pernicious anæmia, not only HCl but also pepsin, rennin and intrinsic factor are absent; this is spoken of as *achylia*.

2. The Fractional Test-meal

Additional information may be furnished by the fractional test-meal, which is not difficult to carry out, and, though lasting three hours, is not too exacting for the patient.

The apparatus required is a special narrow-bore stomach-tube, a 20 c.c. Record syringe, and a rack of twelve numbered test-tubes. The most suitable stomach-tube is that devised by Ryle (Fig. 6). It

is supplied by Messrs. Down Bros. It is of thin rubber, of about 8 mm. external circumference, and is marked by one transverse line at 40 cm. to indicate

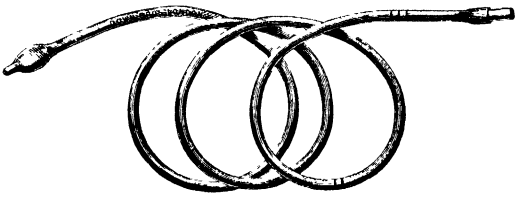


Fig. 6.—Ryle's stomach-tube.

the cardiac orifice and by three transverse lines at 57 cm. to indicate the pylorus. It has a small

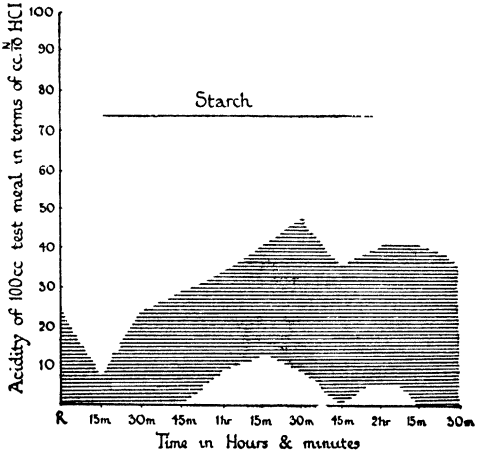


Fig. 7.—Normal gastric function.

From observations by Bennett and Ryle on 100 healthy males (*Guy's Hosp. Repts.* 1921, lxxi. 317.)

The shaded area represents the *limits of free HCl* in 80 per cent. of healthy males.

blind, bulbous extremity weighted with metal, and, at a distance 2 cm. from the tip, it is perforated by a number of small holes, 2 mm. in diameter. The resilient edges of these perforations obviate the risk of damage to the gastric mucosa when strong suction is exerted on an almost empty stomach.

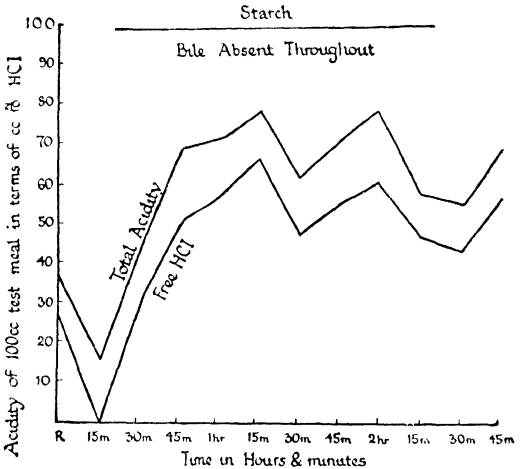


Fig. 8.—Fractional test-meal : chronic ulcer of duodenum.

Specimens withdrawn with ease. All deposits very clean. Clear, colourless supernatant fluid. No mucus. Fluid like water in last three specimens.

The test-breakfast consists of thin gruel, made by adding a quart of water to two tablespoonfuls of fine oatmeal, boiling down slowly to a pint, and straining through muslin. The patient is given a light supper, all drugs are suspended, and he is starved from midnight. At 9 A.M. the tube is boiled and placed in warm water, whilst the patient sits up in bed or in an arm-chair, a towel being arranged across his

chest. The tube is not lubricated, but is placed on the back of the patient's tongue, and he is asked to close his mouth and to swallow the bulbous end just like a pill. The act of swallowing is continued until the pyloric mark almost reaches the teeth, the patient meanwhile being reassured and told to breathe gently through his nose.

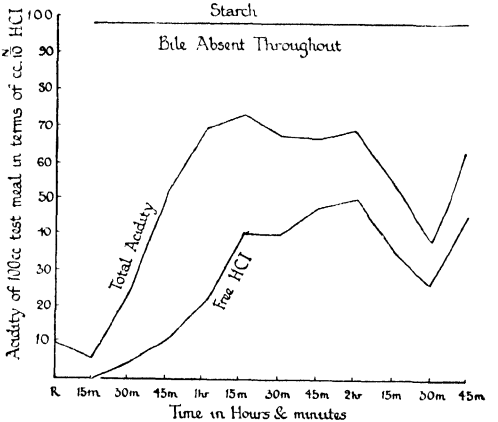


Fig. 9.—Fractional test-meal: chronic ulcer of pyloric end of stomach. Specimens withdrawn with ease. Clean deposits, full volume of porridge to one hour. Supernatant fluid opalescent. No mucus

A sample of about 15 c.c. of the fasting-stomach content is withdrawn by gentle aspiration with the syringe, though it is sometimes an advantage to withdraw and to measure the whole of the resting fluid. No funnel is used, neither is the patient asked to retch or to cough. Without withdrawing the tube, the patient drinks the pint of warm gruel; after which, at fifteen-minute intervals, samples of stomach content (10–15 c.c.) are aspirated, the tube remaining

in situ for three hours. The patient can talk, read, or devote his attention toward any minor employment to pass the time. After the withdrawal of each specimen, air is injected to empty the tube, and a small stopper made of thin glass rod is used to close the end of the tube, thus preventing leakage. Blocking of the tube seldom occurs, but when it

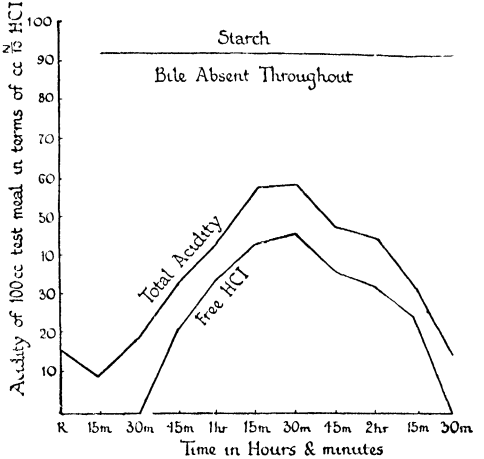


Fig. 10.—Fractional test-meal: chronic ulcer of body of stomach. Specimens withdrawn with ease. Clean deposits, porridge visible up to two hours. Transparent supernatant fluid with mucoid flecks.

does, it is easily overcome by air-pressure. In the majority of cases there is no difficulty in obtaining twelve specimens, each of 15 c.c. In cases of marked hyposecretion, however, great difficulty is encountered in the withdrawal of specimens, and each may measure only 3 c.c. If difficulty is experienced in withdrawing the specimens, the tube should be withdrawn or swallowed a few centimetres or the patient may be tilted well towards the left. At the end of

three hours the tube is gently withdrawn, the patient being asked to swallow as it passes the level of the cricoid.

In the laboratory, the naked-eye appearance of each specimen and the amount and colour of both sediment and supernatant fluid are noted. A rough estimate of the amount of each specimen is made,

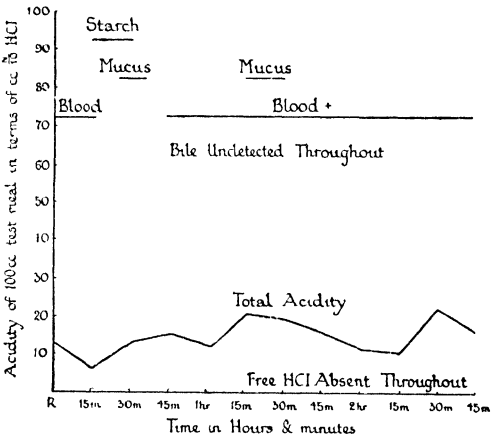


Fig. 11.—Fractional test-meal: carcinoma of stomach.

Specimens withdrawn with ease. All dirty and turbid, and majority stained bright red with blood. Porridge visible up to 30 minutes. Floating blood-stained mucus in most specimens

and the presence or absence of bile, blood, and mucus is noted. Every specimen is then examined for free HCl and total acidity by the method previously described. The emptying time of the stomach is estimated by the addition of a few drops of iodine solution to each tube, the presence of starch being shown by an intense blue coloration. The curves of free HCl and total acidity are then plotted as shown in Figs. 7–12, the first reading being that of

the fasting gastric content. The rate of emptying of the stomach, and the presence of bile, blood, and mucus, are plotted horizontally as black lines.

Typical findings in chronic duodenal ulcer, chronic gastric ulcer, carcinoma of the stomach, and per-

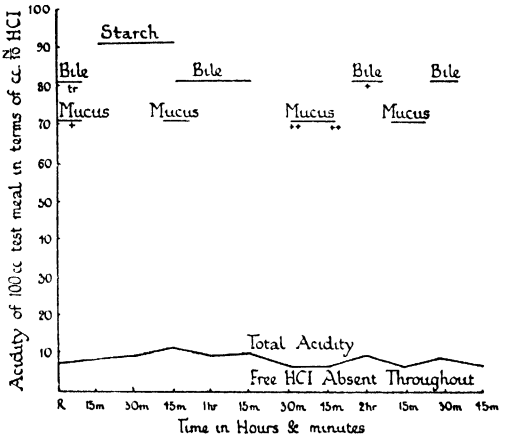


Fig. 12.—Fractional test-meal : pernicious anæmia.

Specimens withdrawn with great difficulty, most of them being only 5 c.c. Much mucus. Porridge visible to one hour. Then dirty, turbid fluid.

nicious anæmia are shown in Figs. 8–12. By reference to Fig. 7, which depicts the behaviour of the normal stomach in response to the fractional test-meal (Bennett and Ryle), the abnormality of gastric function in each of these conditions can at once be appreciated.

3. The Histamine Test-Meal.

The discovery that histamine is a powerful stimulant to gastric secretion has resulted in the introduction of the histamine test-meal. There is

no standard method of carrying out this test, but for most purposes it is convenient to combine it with the fractional test-meal. This is done in the following manner :

The patient is prepared for and given a gruel meal after swallowing a Ryle's stomach tube as described previously. Specimens are withdrawn at fifteen-minute intervals up to one hour. At this stage the specimen withdrawn is tested for free hydrochloric acid by means of Congo-red paper. If this is not present an injection of 0·75 mg. of histamine acid phosphate is administered subcutaneously and further specimens are withdrawn up to three hours from the start of the meal. Analysis of the specimens is carried out in a manner similar to that already described for the gruel test-meal. Following the injection of histamine the majority of patients experience slight palpitation, flushing of the skin and headache, but these symptoms are always transitory.

The chief importance of the histamine test-meal lies in its ability to demonstrate without any uncertainty the presence or absence of achylia. This can never be regarded as proven by the result of an Ewald meal or a fractional gruel meal alone, and in many patients it is necessary to be certain on this point. If the Congo-red test is positive for hydrochloric acid at one hour in the course of the fractional test-meal, then the absence of achylia is conclusively demonstrated and there is little object in administering histamine.

4. Gastroscopy

The introduction by Schindler of the flexible gastroscope has added greatly to our knowledge both of the normal and of the pathological stomach. Gastroscopy is a specialized method of investigation

demanding considerable experience. The technical procedure will not be described here.

It may be noted, however, that by means of this instrument, in a large majority of cases, almost the whole of the stomach from cardia to pylorus may be inspected. The procedure is safe provided that no œsophageal abnormality is present. It is carried out under local anæsthesia without any great disturbance to the patient.

By means of the gastroscope doubtful radiological appearances may be confirmed or refuted and it may be possible to express an opinion as to the malignancy or otherwise of a gastric ulcer seen on X-ray examination. The stages of healing of a simple gastric ulcer may be followed, and it is usually found that an ulcer crater can be seen gastroscopically for a considerable time after radiological criteria have pronounced the ulcer healed. The most important contribution of the gastroscope to gastric diagnosis lies, however, in the recognition of the various types of gastritis, and it should be borne in mind that a diagnosis of gastritis is almost impossible without the aid of this instrument.

V. EXAMINATION OF VOMIT

1. **Naked-eye characters.**—The general character of the vomit varies greatly, of course, with the nature of the food which has been taken. In pyloric stenosis the vomit is apt to be very copious, sour-smelling, and after standing exhibits a froth on the surface. *Bilious vomit* is yellow or green in colour; *fæcal vomit* presents a very similar appearance, but is distinguished by its fæcal odour and by its neutral or alkaline reaction. The presence of much *mucus* gives to the vomit a viscid consistence. The appearance of the vomit in hæmatemesis varies. If the bleeding is very

copious, the vomit may present the appearance of pure blood and may contain clots. Such bleeding may proceed from a gastric ulcer or from the œsophageal varices of portal obstruction. More commonly the blood is altered in colour by being retained for some time in contact with the gastric juice; thus it may be blackish in colour, or dark brown. The latter appearance is due to the conversion of hæmoglobin into hæmatin. The altered blood gives to the vomit an appearance often compared to that of *coffee-grounds* or hare soup. It should be borne in mind that the taking of preparations of iron or red wines may produce a very similar appearance in the vomit. Vomit which contains dark-green bile may resemble very closely vomit which contains blood. On diluting with water, however, the green colour of the bile becomes more apparent, while blood remains dark.

2. **Chemical examination.**—The vomit should be filtered through fine muslin. The filtrate can then be examined, if desired, in the manner already described for the stomach contents.

Bile can be detected by Gmelin's test (p. 320). For the chemical detection of **blood** in the vomit the guaiac test is not satisfactory. It is better to take up some of the brown deposit with a pipette, place it in a porcelain capsule, and add a pinch of powdered potassium chlorate and a few drops of strong hydrochloric acid. Heat till dissolved. Cool and add a few drops of potassium ferrocyanide solution. A blue colour indicates that blood is present. The reaction is due to the iron contained in the blood pigment. If the patient has been taking iron the test is, of course, inapplicable. In such a case some of the deposit should be digested with potassium hydroxide, filtered, and the solution examined for the spectrum of alkaline hæmatin (Plate 17—*6*, facing p. 224), or

the deposit may be subjected to Teichmann's test (Appendix, 17). To confirm the test, add to the alkaline hæmatin solution a few drops of ammonium sulphide, which converts it into hæmochromogen. The spectrum of the latter is identified by its possessing two bands—one, narrow and dark, in the yellow between E and *b*; the other, broader and less dark, at the junction of the yellow and green between the lines D and E (Plate 17—*8*, facing p. 224).

3. **Microscopical examination.**—Take up some of the deposit which adheres to the muslin, spread it out on a slide, and examine either directly or in a drop of salt solution.

Various particles derived from the food may be recognized—*muscle-fibres* by their transverse striæ; *starch granules* by their concentric lines and the fact that a drop of very dilute iodine solution turns them blue; *elastic fibres* by their double contour and bold curves; *fatty particles* by their high refractility.

Various **vegetable parasites** may be present. The most important are the **sarcina ventriculi** (a large micrococcus) and the **yeast fungi.** The former can be recognized by their forming small cubical packets of cells resembling miniature bales of wool; the latter consist of round or oval cells in chains or clusters. They are usually about the size of white blood-corpuscles.

The addition of a very little dilute iodine solution to the vomited matter may render the detection of sarcinæ more easy. The iodine stains them a deep mahogany brown.

VI. EXAMINATION OF FÆCES

Examination of the fæces is an investigation of great importance too frequently omitted. No patient with bowel disturbance has been properly examined

until the stools have been inspected. The white surface of a bedpan makes an ideal background for the detection of blood, pus and mucus.

It is often desirable to examine the fæces of a patient upon a fixed diet. This may be satisfactorily accomplished by giving 0·2 grm. of powdered charcoal in a gelatin capsule, or two charcoal biscuits, at the beginning and at the end of the diet, and collecting the fæces between the two black stools thus produced. The same plan may be followed using a gelatin capsule containing 0·3 grm. of carmine alum lake and watching for a red stool.

1. **Naked-eye inspection.** The following points should be attended to :—

(*a*) Amount of the daily stools.
(*b*) Their colour.
(*c*) Their odour.
(*d*) Their consistence and form.
(*e*) The presence of any abnormal constituents.

As regards **amount,** it is usually sufficient to state whether the stools are copious or scanty. The average daily amount of fæces in health is 120-180 grm. (about 4 oz.).

The **colour** of normal fæces is partly due to stercobilin, partly to chlorophyll and other pigments. A mixed diet yields stools varying in colour from light to dark brown—being darker with an exclusive meat diet, lighter with a milk diet. The presence of unaltered bile-pigment is always abnormal, and may be due to increased rapidity of peristalsis. **Black** stools may be produced by the administration of iron, bismuth, or manganese. In hæmorrhage high up in the intestine the altered blood makes the stools dark, tarry-looking, and very offensive. The blackness due to blood may be distinguished from that produced by drugs by mixing part of the stool with twice its

volume of water and allowing it to stand in a glass jar. If blood is present the water becomes reddish; under other conditions it remains dark or greenish.

Pallor of the stools may be due to an obstruction to the entrance of bile into the intestine, as in obstructive jaundice, to dilution and rapid passage of the stool through the intestine as in diarrhœa, or to an abnormally high fat content.

The **odour** of the fæces is due to the presence of indol and skatol. The intensity of the odour depends to a large extent upon the amount of meat ingested. The absence of bile favours putrefaction; hence the stools in jaundice are often very offensive. Cholera stools, on the other hand, contain very little organic matter, and are almost free from odour. In fermentative processes in the intestine the stools may have a sour smell.

The **form** and **consistence** of the stools is of importance. In obstinate constipation they may be much drier and harder than normal, and even friable. In all forms of diarrhœa they are more fluid than normal, and may even be watery. Slimy stools are due to the presence of an excess of mucus.

It is important to note whether the stools are formed or fluid. If formed, any abnormality in the shape should be noted. The stools of constipation have often the form of round balls, frequently coated with mucus. In obstruction in the large intestine the stools may be ribbon-like. The presence of a rectal polypus may produce a groove or furrow along the fæcal mass.

In order to facilitate the detection of **abnormal ingredients,** the stool should be placed on a fine sieve, and a large quantity of water added. The whole is shaken and stirred up till the soluble parts are all washed away. The residue is then examined.

For the detection of finer particles it is better to take a portion of the fæces of about the size of a walnut and to rub it up in a mortar with some distilled water to the consistence of pea soup. It should then be poured into a shallow glass dish with a black background, against which particles of mucus or of food residues are easily seen.

Gall-stones are easily recognized. It is important to note whether they are faceted or not, for if they are, then the stones are multiple. Particles of undigested food, fruit-stones, foreign bodies, concretions—e.g those produced by magnesia—and parasites should all be looked for.

The full consideration of the parasites which may be found in the stools is undertaken later. We would only mention here that one has often to search stools for the **head of a tapeworm.** The best method of procedure in such a case is to add to the stool a considerable quantity of water containing a little carbolic acid, and to shake the mixture gently for a few moments. It is then allowed to stand for about ten minutes. The parasite sinks to the bottom, the supernatant fluid is poured off, and more water added till the residue is nearly colourless. The parasite will then be readily found. The head is only about as large as that of a large pin, and the neck about as thick as a stout thread.

Special terms are applied in clinical medicine to some particular **varieties of stool.**

The **bilious** stool is well illustrated in the typical stool of typhoid fever. Its characters are described by the term " pea-soup " stool, usually applied to it.

Watery stools are found in all cases of colliquative diarrhœa, and after the administration of hydragogue cathartics. To the stools of cholera the special name

of *rice-water* stools is applied. Such a stool is colourless, almost devoid of odour, alkaline in reaction, and contains a number of small flocculi consisting of shreds of epithelium and particles of mucus. The name is applied to it from its resemblance to the water in which rice has been boiled. **Purulent** or pus-containing stools are found in severe dysentery or intestinal ulceration, or in cases where an abscess has found its way into the intestines. **Slimy** stools are due to the presence of an excess of mucus, and point to an affection of the large bowel. The mucus may envelop the fæcal masses, or may be intimately mixed with them. **Bloody** stools vary in appearance according to the site of the hæmorrhage. If the bleeding takes place high up, the stools look like tar, as has been already mentioned. In an ordinary intussusception they may look like red-currant jelly. In those rare cases in which the intussusception occurs in the jejunum the appearance of the material passed per anum has been compared to that of a melted strawberry ice. If the hæmorrhage is from the large intestine, the blood is less intimately mixed with the fæcal matter, and may even be of a bright colour. In hæmorrhage from the rectum or anus it may merely streak the fæcal masses.

In muco-membranous colitis the motions contain **casts of the bowel,** which are mainly composed of **mucin.** The individual casts vary considerably in size, being commonly from 1 in. to 6 in. long, but in exceptional cases attaining a length of 1 ft. or more. They vary in width from narrow strips to tubes more than 1 in. in diameter; they are sometimes grey and transparent like ordinary mucus, at other times they are more opaque and resemble the casts formed in the air-passages in membranous croup. Sometimes they are stained of a brownish-yellow colour from adherent or incorporated fæcal matter; rarely they are red

from the presence of blood. They are often very abundant, and may become agglomerated into irregular masses which, when floated on water, can be unravelled into their component parts. As a rule the cast is tubular, but the wall varies much in thickness, and the lumen may be obliterated. If teased out in water, one can sometimes separate the cast into membraneous layers between which small particles of fæcal matter may be observed.

These casts, when small, may, on casual observation, be mistaken for segments of tapeworm. On the other hand, cases are recorded in which the remains of indigestible substances in the food or firm clots of curdled milk have been regarded as casts.

The stools on rare occasions contain **intestinal sand,** which may be of either mineral or vegetable origin. The former, which is sometimes called " true sand," chiefly consists of calcium phosphate, associated with smaller amounts of calcium carbonate and of silica, around an organic nucleus of animal origin. When it is washed and dried it usually presents the appearance of ordinary fine sand, some of the particles being yellowish-brown and others almost colourless. On microscopic examination the grains are found to be very varied in shape; some are oval and smooth, others irregular and rough, whilst in structure they are granular rather than crystalline.

When the sand is of vegetable origin it is known as " false sand," and consists of sclerenchymatous particles such as those that are present in pears, bananas, and some other fruits. Under the microscope it is easy to recognize the nature of the grains, as, after the removal of any inorganic incrustation by an acid, the thick transparent walls of the woody cells, traversed by channels passing between the surface and the narrow cell-cavities, are clearly visible.

2. **Chemical examination.** i. **Reaction.**—If fluid the stool should be well mixed, and if solid a small portion rubbed in a mortar with distilled water to a soup-like consistency. A drop should then be applied with a glass rod to a piece of blue or red litmus paper previously moistened. The reaction can easily be seen on the other side of the paper. A normal stool should be nearly neutral. Marked acidity indicates fermentation; marked alkalinity, putrefaction.

ii. **Test for bile-pigment.**—Mix some of the stool with a concentrated solution of corrosive sublimate and allow it to stand twenty-four hours. A normal stool is turned red from the presence of urobilin; a green colour shows unaltered bile pigment. A complete absence of either green or red colouring shows the absence of bile altogether.

The tests for bile-pigments and urobilin described under Urine (pp. 288, 320) may also be performed with a semi-fluid mass of fæces and water; indeed, this is preferable. Bile-acids, if present, may be obtained by extracting the fæces with alcohol, filtering, evaporating off the alcohol, and dissolving the residue in dilute alkali. Test with a few drops of cane-sugar solution and sulphuric acid; a purple colour indicates the presence of cholalic acid (Pettenkofer's test). *See also* the tests for bile-acids in urine (p. 322).

iii. **Test for occult hæmorrhage.** *Benzidin test* (Gregersen).—This test is the simplest and most suitable for routine use. It is carried out as follows :—

Powders are made up containing 0 2 grm. of barium peroxide and 0 025 grm. of pure benzidin, if put up in wax papers they will keep indefinitely. Just before the test one powder is dissolved in 5 c c. of freshly-prepared 50-per-cent. acetic acid solution A "button" of fæces is taken by means of a glass rod from the centre of the stool and smeared on to a clean glass slide. A few drops of the solution are then run on to the smear. A blue or blue-green colour develops

within a minute if the test is positive, and the reaction is graded according to the depth of the colour and the time it takes to develop. Gregersen recognizes three grades: 1 +, a pale blue or green colour within 60 seconds; 2 +, a definite pale blue in 12 to 15 seconds; 3 +, deep blue within 3 seconds.

It is usual, prior to carrying out this test, to exclude red meats and green vegetables from the diet for 2 or 3 days. It should be noted, however, that if a blue or blue-green reaction appears *within 30 seconds*, the presence of occult blood may be inferred no matter what the diet has been.

Guaiac test (Ryffel).—A portion of fæces about the size of a walnut is thoroughly stirred with 8 to 10 c.c. of glacial acetic acid. To the emulsion add rather more than an equal quantity of ether and stir again. To about 2 c.c. of the ether-acid extract add 5 drops of fresh tincture of guaiacum and then, slowly, 10 per cent. ozonic alcohol[1] until a blue or green colour develops or an excess has been used.

This test does not appear to be affected by iron-containing drugs.

The fæces may also be examined for blood by suitably diluting and examining spectroscopically (*see* p. 311).

3. **Microscopical examination.**—It is best to use a low-power objective, preferably about $1\frac{1}{2}$ *in*.

Prepare a film as follows: Remove a portion of the fæces, about the size of a split pea, with the end of a match. Place it in the centre of a slide and lay another *slide* on the top, and press the two together. If the stool is very hard, one may soak the selected portion in water for a few minutes before preparing the film. If liquid, a drop of the sediment may be taken up with a pipette, placed on a slide, and covered with a *cover-slip*.

The following are the chief constituents of fæces to be looked for: (1) Muscle-fibres, (2) connective-tissue and elastic fibres, (3) starch granules, (4) vegetable detritus, (5) fat, fatty-acid crystals, and soaps (6) triple phosphate, oxalate, and cholesterol crystals, (7) mucus, (8) blood, (9) yeasts and other fungi.

[1] **10 c.c. of 20-vol. H_2O_2, alcohol to 100 c.c.**

Muscle-fibres are easily recognized by their cross-striation. If present in large numbers they indicate defective intestinal digestion.

Connective tissue may somewhat resemble mucus, but is distinguished by its striation, which disappears on the addition of acetic acid. If masses of it are seen, defective gastric digestion may be inferred. Elastic fibres have no significance.

Starch granules are readily detected if a drop of iodine solution is added. Their presence in excess is pathological, and such a stool is usually markedly acid and often shows signs of fermentation (gas bubbles) and the presence of yeasts.

Detritus derived from vegetables and fruits is easily identified by its areolar tissue, spiral ducts, vascular bundles, and pigment cells.

Neutral fat occurs as colourless, highly refractile droplets, or as bile-stained irregular masses which stain with Sudan III and are soluble in ether.

Fatty acids occur as sheaves of colourless acicular crystals which dissolve on warming or in ether.

Soaps occur as greasy-looking amorphous masses, or as needles which are shorter and thicker than those of fatty acids. They dissolve on warming, but not in ether. On heating the slide with a drop of acetic acid, crystals of fatty acid will be seen to separate out.

A simple way of distinguishing fats from mucus or vegetable material is to press down the cover-slip. If the material be fat, the slip remains down; if vegetable detritus or mucus, it springs back when the pressure is released, and air rushes in from all around.

Normally, fat in the fæces is almost entirely in the form of amorphous masses of soap; less often as crystals. Neutral fat should be practically absent.

Triple phosphate crystals (Fig. 74, p. 341) are always present in normal fæces and never coloured by bile. Oxalate crystals are generally found as well, especially when much vegetable food is taken. Cholesterol crystals may also be found in normal fæces.

Mucus occurs as transparent blobs or shreds, sometimes bile-stained. It may contain numerous leucocytes or epithelial cells.

VII. INTESTINAL PARASITES

The parasites which occur in the intestinal tract include worms and protozoa. Some of the nematode and cestode worms will be described.

A. NEMATODA

1. Perhaps the commonest of all internal parasites is the thread-worm, **Oxyuris vermicularis,** whose presence is associated with considerable itching about the anus. It inhabits the large intestines, cæcum, and appendix, and specimens can often be seen wriggling about in the recently passed motion of their host. To the naked eye they look like small white threads, 0·5 to 1 cm. in length. Under the microscope the female may be distinguished by the large uterus filled with ova, and the pointed posterior end, whence its name is derived.

2. **Ascaris lumbricoides,** or round worm, has a general resemblance to an earth-worm. It measures from 6 to 8 in. or even more. Not infrequently its presence in children is associated

with nervous disorders. The ova, which can occasionally be found in the dejecta, have brownish-yellow granular contents, and in many cases the shell is surrounded by an irregular albuminous sheath.

3. **Ankylostoma duodenale** is a parasite whose presence is fraught with much greater risk to the host than that of those already mentioned, as it causes profound anæmia by drawing blood from the wall of the bowel. It lives for the most part in the upper part of the jejunum, and its presence there is rendered probable when, in an infested district, severe anæmia, otherwise inexplicable, sets in. The diagnosis is confirmed by the discovery of ova in the motions. They exhibit a segmented yolk enclosed in a thin shell, and are sufficiently numerous to be readily detected. The adult worm, which is but rarely seen before therapeutic agents have been employed, is about half an inch long and the mouth is provided with four claw-like teeth. It is no longer to be found in Britain.

4. **Trichina spiralis** gains access to the body as the result of eating infested pork. Trichiniasis is rare except in Germany. When man ingests the muscle trichinellas of the pig, larvæ are set free in the small intestine giving rise to the symptoms of the first stage of the illness—abdominal pain, vomiting and diarrhœa. The adult female, 3 mm. long, penetrates the intestinal wall and discharges embryos into lymph spaces whence they migrate into muscles. In this second stage of the illness the patient has fever and the muscles swell and become hard and tender. Death may occur at the height of the myositis. Otherwise the embryo undergoes no further development and its capsule becomes calcified.

B. Cestoda

Many different kinds of tapeworm have been found as parasites in man, but the most important are *Tænia saginata*, *T. solium*, and *T. echinococcus*. Besides its occurrence in the fully developed state, *T. solium* may be present in the tissues in the form of a cysticercus; *T. saginata* is never found in this condition in man; whilst *T. echinococcus* always occurs in the cystic stage, and has never been found in the mature condition in the human intestinal tract.

The presence of an adult tapeworm in the bowel is generally revealed by the passage of ripe proglottides in the stools, and after the administration of anthelmintics the head may be detected by the methods previously described.

1. **Tænia saginata (mediocanellata),** the English tapeworm, occurs as a result of consuming insufficiently cooked beef infested with the embryo of the worm. The adult parasite reaches an average length of 15 metres and consists of about 2,000 segments. The ripe proglottides measure 16 by 15 mm. The head is quadrate, measures 2 mm. in diameter and has four suckers but is devoid of hooklets. The terminal gravid segments of the worm from time to time become separated and the ova are then ingested by the ox, in the muscles of which the larva develops. It becomes a bladder worm, *Cysticercus bovis*, measuring 10 by 6 mm. and containing an invaginated head which possesses in miniature the characteristics of the adult scolex. *Cysticercus bovis* is never found in human muscle tissue or brain.

2. **Tænia solium,** the pork tapeworm, is no- encountered in Britain but is endemic wherever in fested pork is eaten raw or insufficiently cooked. I measures 2 or 3 metres in length; a ripe proglott is 10 by 6 mm. The head mea ures 1 mm. in diamete

and in addition to four suckers has a rostellum with 32 hooklets. The ova in the terminal proglottides are ingested by the pig, in the muscles of which the bladder worm *Cysticercus cellulosæ* develops. Occasionally soldiers serving in India become infested with *Cysticercus cellulosæ* from partaking of food contaminated with the ova of the parasite. The muscles of the human host are then infested by cysticerci which are palpable through the skin as tense ovoid swellings 10 by 5 mm. and of almost cartilaginous hardness. About four years after infestation they become calcified and may then be demonstrated radiologically. Unfortunately the cysticerci come to reside also in the motor cortex and in this situation they give rise to **cysticercus epilepsy** which often proves fatal.

3. **Tænia echinococcus.**—The adult worm, which consists of a head and three segments, and whose length is only 4 or 5 mm., need not be fully described, since it is not found in man. The *cystic stage* is very important, as it gives rise to serious disease in many of the viscera, and especially in the liver. The cysts of this tænia are not simple, but produce from their inner surface one or two generations of secondary vesicles, on which the brood-capsules, containing the cestode heads, are formed. During the period in which this process is going on, the primary vesicle dilates to accommodate its increasing contents, and may eventually reach the size of a coco-nut. The vesicles may rupture spontaneously and their contents may escape by the lungs, by the bowel, or by the urinary passages, or specimens may be obtained by aspiration, or after surgical interference. In a case of suspected hydatid disease one may require to found the diagnosis either on the chemical nature of the fluid withdrawn, or on the recognition of hooklets or scolices, or on the appearance of the ectocyst, portions of which are sometimes discharged,

especially when the cyst has opened into the lungs and bronchi.

The *fluid* is clear, alkaline, devoid of albumin, and contains abundance of sodium chloride and traces of glucose. Its density is low, being generally under 1010. The *scolex*, if it is obtained in a perfect condition, is about 1 to 1·5 mm. in diameter, and a number of them often spring in a group from one brood-capsule. They have four suckers and a crown of hooklets. Portions of the *ectocyst* appear as whitish-yellow shreds which can be recognized under the microscope by their lamination, and by the pectinate markings on the laminæ.

4. **Diphyllobothrium latum (Dibothriocephalus latus),** the fish tapeworm, is encountered in Sweden, Finland, and in Michigan. The adult worm measures from 3 to 10 metres or more and has a total of 3,000 segments. The scolex is small, spatula-shaped and possesses two deep suctorial grooves. The first larval host is a water-flea and the second the pike, perch, or salmon trout. Human infestation takes place from eating raw or uncooked fish. Most patients infested complain only of the passage of segments in the stools and are otherwise perfectly well. In 0·5 per cent. of those infested an anæmia practically identical with pernicious anæmia develops. The anæmia is controlled by injections of liver extract, but if the worms remain this treatment has to be continued indefinitely. Where they are expelled rapid and permanent recovery from the anæmia occurs.

C. Protozoa

A number of protozoa, many of them non-pathogenic, have been found in the fæces. Of these the most important clinically is *Entamœba histolytica* (Fig. 13), which causes amœbic dysentery (as opposed to bacillary dysentery) and sometimes tropical abscess of the liver. *Entamœba coli* (Fig. 13) in non-pathogenic.

When amœbic dysentery is suspected, a specimen of the fæces should be examined in the fresh state for living motile forms. Entamœbæ are found in two states, (*a*) the vegetative or motile stage and (*b*) the encysted stage (Fig. 13).

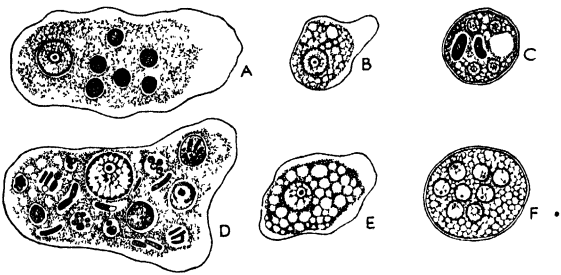


Fig. 13.—The intestinal amœbæ of man (magnified 1,250 times).
(*Dr. C. M. Wenyon, Wellcome Bureau of Scientific Research.*)

A-C: Entamœba histolytica, the cause of amœbic dysentery. A, the tissue-invading form with one nucleus and six ingested red blood-corpuscles: B, the small (precystic) form, which will protect itself by a cyst. C, the cyst containing the amœba with four nuclei and chromatoid bodies. D-F: Entamœba coli, the large non-pathogenic amœba of man, which lives in the lumen and on the surface of the intestine. D, the large multiplying form with one nucleus and various ingested food bodies; E, the small (precystic) form which will become encysted; F, the cyst containing the amœba with eight nuclei.

In examining fæces it is undesirable to take a specimen passed after the administration of a purgative as in these circumstances immature cystic forms are often evacuated and it becomes difficult to differentiate between the species. Amœbæ are most easily detected in the little masses of mucus which occur in the stool. When the motion is pultaceous the addition of warm normal saline solution will facilitate the preparation of a suitable film, in which, if examined on a warm stage, the protozoon may be seen moving about.

The demonstration of the structure of entamœbæ is best accomplished by "fixing" a moist smear of mucus from the fæces with Schaudinn's sublimate

alcohol solution (saturated watery solution of corrosive sublimate 2 parts, absolute alcohol 1 part) to which from 3 to 5 per cent. of glacial acetic acid has been added, and staining with Haidenhain's iron-hæmatoxylin and eosin.

For rougher clinical purposes a fairly satisfactory method of examining the fæces is to emulsify a fragment with Gram's iodine solution, using it in about double the strength of the solution recommended for bacteriology. This brings both the vegetative and the encysted forms into prominence.

The following table may facilitate the differentiation of the two amœbæ (see Fig. 13):—

	Entamœba hlstolytica	*Entamœba coli*
1. Occurrence	Fairly abundant, when present, in dysenteric stools.	Not abundant. Only occasional in dysenteric stools.
2. Size	Variable, average 20μ to 30μ.	Less variable, on the average rather larger than *E. h.*
3. Motility	Active, fine pseudopodia. These become blunter as the activity diminishes before death.	Sluggish. Blunt pseudopodia.
4. Cytoplasm	Homogeneous and "ground-glass-like" (apart from food granules). When activity is diminishing, the differentiation of ectoplasm and endoplasm may be seen at root of pseudopodia, otherwise the distinction is inconspicuous. Red blood-corpuscles often seen in cytoplasm.	Appearance "porcellanous." Ectoplasm less differentiated and line of demarcation between it and endoplasm inconspicuous. Endoplasm granular, abundant food vacuoles with inclusions. Red blood-corpuscles or tissue elements never present.

	Entamœba histolytica	*Entamœba coli*
5. NUCLEUS	Fainter than in *E. c.* Karyosome *central* with clear "halo" round it. Periphery marked by ring-like layer of chromatin granules.	Distinct. Karyosome *nearly always eccentric*, "halo" more definite. Ring-like layer of peripheral granules more pronounced.
6. CYSTS	When mature, four nuclei, with nuclear karyosomes central. Size slightly smaller on average than *E. c.* cysts. Average size, 10μ to 20μ. Glycogen less abundant. Refractility moderate. "Chromidial bodies" usual. Cyst-wall rather thinner.	When mature, eight nuclei, with nuclear karyosomes usually eccentric. Size slightly larger than *F. h.* cysts. Vary from 10μ to over 20μ. Glycogen more abundant. Refractility considerable. "Chromidial bodies" absent in mature cysts. Cyst-wall rather thicker.

CHAPTER IV

THE CIRCULATORY SYSTEM

I. ANATOMY

THIS system is composed of two main elements, the heart and the blood-vessels, and these are for the most part dealt with separately, although, when the chest is exposed for the examination of the heart, the vessels in the thorax and at the root of the neck are more conveniently examined along with it. (*See* Plate 7, facing p. 64).

The **heart** lies obliquely in the thorax, being inclined from above downwards, forwards, and to the left. Two-thirds of it lie to the left of the middle line. The part which reaches highest in the thorax is the **left auricular appendix [left auricle]**, which in the cadaver extends as far up as the 2nd left costal cartilage. During life it is usually opposite the 2nd interspace or lower border of the 2nd cartilage, as the diaphragm then occupies a lower level. The greater portion, however, of the **left auricle [atrium]** lies posteriorly, and constitutes the hindmost cavity of the heart.

The **right auricle [atrium]** is the chamber that lies most to the right. It extends somewhat beyond the right margin of the sternum, and its border may be traced by a curved line joining the 3rd and 7th right chondro-sternal [sterno-costal] articulations, and reaching about 1 in. to the right of the sternum.

The **right ventricle** occupies the great portion of the front of the heart. Its inferior margin extends from the 7th right chondro-sternal [sterno-costal]

articulation to the apex, and constitutes the lower border of the heart.

The **left ventricle** only appears in front as a narrow strip, scarcely $\frac{1}{2}$ in. broad, and its outline completes that of the heart on the left, where its border forms a curved line, ascending from the apex to the lower margin of the 2nd left interspace at a point just internal to the parasternal line. The topographical anatomy of the valves of the heart and of the great vessels will be discussed in connexion with auscultation, as it is in this department that a knowledge of their situation is most necessary (pp. 129-32).

The most important organs which come into relation with the heart are the lungs on either side, the liver below, and the great vessels above. A small portion of the anterior surface is only separated from the thoracic wall by the anterior mediastinum, whilst, behind, the heart is in relation with the structures that occupy the posterior mediastinum.

That portion of the anterior aspect of the chest which overlies the heart is known clinically as the **præcordial region.** It is an area of ill-defined extent.

It is often necessary to define the exact situation of a point on the front of the thorax, and certain landmarks, some natural and some artificial, are commonly made use of for this purpose.

The **ribs** and **interspaces** on either side form convenient horizontal landmarks. In order to count them, one must feel for the ridge which marks the junction of the manubrium with the body of the sternum, known as the angle of Louis, or sternal angle. When this has been found, by running the finger outwards it reaches the 2nd costal cartilage, which articulates with the sternum at this level. It is then easy to reckon upwards or downwards to the other ribs. The

determination of the 1st rib directly is neither so easy nor so certain, since it is overlapped by the clavicle.

In order to define the distance of any given point from the mesial sagittal plane of the body, a series of vertical lines may be drawn on the chest. These are the **midsternal** and **lateral sternal lines,** down the middle and either border of the sternum; the **mammary line,** best defined, since the situation of the nipple is inconstant, as the vertical line dropped from the centre of the clavicle, or, what amounts to the same thing, the line midway between the middle of the suprasternal notch [incisura jugularis] and the tip of the acromion; the **parasternal line,** midway between the lateral sternal and mammary lines; the **anterior, mid-,** and **posterior axillary lines,** descending from the anterior border, the centre, and the posterior border, respectively, of the axilla; and the **scapular line,** which is defined as the vertical line drawn through the inferior angle of the scapula.

The methods commonly employed in the examination of the heart are inspection, palpation, percussion, and auscultation. These will be taken up consecutively, although in practice inspection and palpation are often advantageously combined.

II. INSPECTION

Inspection determines

(A) Form—

1. Of præcordia { Bulging. / Flattening.
2. Of surrounding parts (especially bulging).

(B) Movements—

1. In præcordial region { Apex-beat. / Diffuse pulsation. / Pulsation at base of heart. / Local indrawing.

2. Outside præcordial region	Pulsations at root of neck.	
	,,	in thorax.
	,,	in epigastrium.

(C) Dilated veins and venules.

For inspection of the chest, the patient should be stripped to the waist, set in a good light, and examined first standing or sitting up, and then lying on his back. The observer should directly face him, but must be careful not to obstruct the light. In some cases the observer may with advantage take up a position at the top of the bed, and lower his head until he looks along the chest tangentially. By this manœuvre he will be able to study various pulsations with great facility.

The following points must then be systematically noted:—

1. The shape of the præcordia.
1. Pulsations in the præcordial region.
3. Bulging or pulsation outside the præcordia, either at the root of the neck, or the front of the chest, or the epigastrium.
4. The presence or absence of distended veins on the chest-wall or in the neck.

1. **The shape of the præcordia.**—In health the chest is bilaterally symmetrical, and there is no greater prominence on the left side than on the corresponding area of the right.

In cases where the **præcordial area is prominent,** it must be remembered that other conditions than disease of the heart may have caused it. Again, it should be remembered that serious disease of the heart is comparatively seldom accompanied by bulging of the præcordia unless it had already manifested itself when the patient was young and the bones were incompletely ossified.

Should prominence be observed, note whether the ribs are involved, or whether the intercostal spaces alone bulge. The latter condition occurs in pericarditis with effusion. Prominence of the præcordia may be due to disease in the framework of the thorax, such as scoliosis, parietal tumour, or abscess, or to a diseased condition of the thoracic contents, such as cancer of the lung or effusion into the pleural cavity, mediastinal tumour, fluid in the pericardium, enlargement of the heart, especially if it occur in early life, and aortic aneurysm behind or above the heart.

Flattening of the præcordia may be congenital; it may mark the former occurrence of pericarditis; it may be due to retraction of the lung; and in some instances, particularly in certain trades, it may be the result of pressure.

2. **Pulsations in the præcordial region.**—Besides the movement of respiration, which affects the præcordia with the rest of the chest, an impulse which occurs three or four times to each respiration is generally to be seen in the lowest and outermost part of that region.

This pulsation is called the **apex-beat** of the heart, and in health usually exhibits the following characters:—

(1) It is found in the 5th left intercostal space.

(2) It is limited to an area less than an inch in breadth, and is only visible in one interspace.

(3) It is situated outside the left parasternal line, and inside the left mammary line.

(4) It is due to the impact on the chest-wall of the apical segment of the heart and to its hardening during systole; and for clinical purposes the actual apex of the heart may be assumed to be situated at the lowest and outermost part of the above area

of direct pulsation, although it may really be slightly lower down and farther out, under cover of a rib.

The apex-beat may be abnormal in **force,** in **position,** or in **extent.** Even in perfect health, if the chest-wall is thick, or if the apex lies behind a rib, it may be quite invisible. *Absence*, therefore, of the apex-beat is not to be regarded as necessarily indicative of disease, though it must not be forgotten that in cases of weak action of the heart it may be absent or diminished in force. When abolished, its place may be taken by a more *diffuse impulse* over the lower part of the præcordial area, in cases where the apex is pushed away from the chest-wall by a dilated right ventricle, or when pericardial effusion separates the heart from the front of the thorax. On the other hand, the apex-beat may appear to be *more forcible* than usual in cases where the heart's action is excited, where the chest-wall is thin, or where the left ventricle is hypertrophied. Such changes are more accurately observed by palpation, and will be discussed under that head.

The **position of the apex-beat** may be altered in three classes of cases. The cause may be (*a*) *congenital*, where the heart is reversed so that the apex lies to the right (*dextrocardia*), or where in coarctation of the aorta the left ventricle is enlarged, or where in pulmonary stenosis and patent ductus arteriosus a distended right heart displaces the left ventricle. The displacement of the apex-beat may be due to (*b*) *extrinsic causes*, where the heart is displaced by diseased conditions of surrounding viscera which push or pull it from its usual site. Instances of this are found in pleural effusion, pneumothorax, pulmonary fibrosis, collapse of the lung, and a raised diaphragm from any cause. Scoliosis should be remembered as a common cause of outward displacement of the apex-beat.

Where the heart is pushed over to the right by a left pneumothorax, or pleuritic effusion, the pulsation which may be conspicuous to the right-hand side of the sternum is not that of the apex, which is usually lying somewhere behind the bone, but is due to pulsation of the right ventricle and auricle [atrium].

Again, the displacement of the apex-beat may result from (*c*) *disease of the heart*, as in hypertension, aortic incompetence and aortic stenosis.

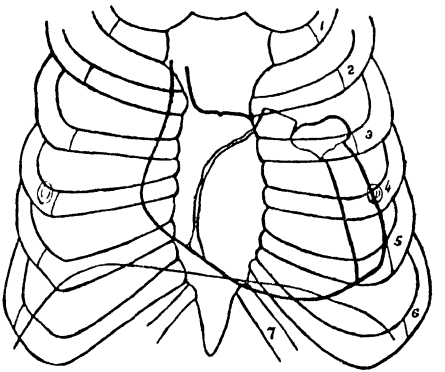


Fig. 14.—The heart in an infant. (*After Symington.*)

In addition to these causes, it should be remembered that the position of the apex-beat varies considerably with the patient's age; *in children* it is usually as high as the 4th interspace, *in the aged* it descends as low as the 6th.

In certain cases the apex-beat is replaced by an *indrawing* of the same area during cardiac systole. This, when it is marked in degree, and when it appears over an extensive area of the lower segment of the præcordia, suggests adherent pericardium. However, in lesser degree, it is commonly present in cardiac

enlargement from any cause, and especially in young subjects.

The consideration of **other pulsations** which may be visible in the præcordial region must next be undertaken. Allusion has already been made to the diffuse pulsation which occurs when the right ventricle is enlarged, and which in these circumstances is visible over the lower part of the area in several of the intercostal spaces, and nearer the middle line than the normal apex-beat.

Pulsation is also seen at times in the 2nd left intercostal space. It may arise either in the pulmonary artery, which lies half under cover of the left side of the sternum, and half under the inner end of this interspace, or it may also be due to aneurysm.

In chests which have thin parietes, and especially when, in addition, the left lung is retracted from fibrosis, pulsation of a diffuse nature can be observed over most of the interspaces of the præcordial region, as well as at the apex. In these cases the apex-beat still causes a limited area of the chest-wall between the left parasternal and mammary lines to bulge forward with each beat of the heart, whilst the diffuse pulsation which is caused by the systole of the right ventricle is associated with indrawings of some portion of the intercostal spaces.

3. **Pulsations outside the præcordia.**—In addition to the pulsations already described, movements should be looked for at the root of the neck, the front of the chest, and the epigastrium.

At the root of the neck pulsation may occur either in the episternal [jugular] notch or externally to the sterno-mastoid.

In the episternal [jugular] notch the pulsation is usually systolic in time, and when well marked

may be an indication of raised aortic arch in hypertension or of aneurysm of the arch of the aorta. Less commonly it is due to an abnormal origin of the right subclavian from a point to the left of the middle line. Palpation generally enables these conditions to be discriminated.

Outside the sterno-mastoid various pulsations may be observed. These may be either arterial or venous. The carotids pulsate visibly on exertion; from mental excitement; in diseases which cause excitement of the circulatory system, such as exophthalmic goitre; in cases of aortic incompetence, hypertension, and aneurysm of the artery. In high blood-pressure the right carotid sometimes shows abnormal pulsation due to kinking, which must not be mistaken for aneurysm.

The jugular veins show slight pulsation normally, but if it is excessive and the veins are distended one may assume overfilling or enlargement of the right auricle, or, rarely, actual regurgitation through an incompetent tricuspid valve. It will be discussed under the head of Venous Pulse (p. 165).

In the thorax, besides the pulsations referred to as occurring in the præcordial region, a *diastolic pulsation* may rarely be observed in the 2nd right intercostal space, and results from the closure of the aortic valves. An important source of pulsation in unusual parts of the thorax is *aneurysm of the aorta.* Such aneurysmal pulsations always manifest themselves at first above the level of the 4th rib, though at a later period they may affect a very considerable portion of the chest-wall. The position of the impulse varies according to the part of the aorta which is diseased. If the *ascending aorta* is affected, the pulsation is chiefly to the right of the sternum, whilst the *transverse*

aorta gives rise to less distinct pulsation under the manubrium sterni, and the *descending aorta* still more to the left. Aneurysm of the *innominate* may project far into the neck. The time of this pulsation is systolic, following immediately on the apex-beat, and it may be observed to be expansile in character.

Pulsating empyema is a rare event; it occupies the præcordial area from which the heart is more or less displaced, and malignant tumours with a large blood supply may also give rise to pulsation in the part of the chest-wall that overlies them.

Sir W. Broadbent pointed out that marked *systolic retraction* of some of the lower ribs on the lateral and posterior aspects of the thorax may occur as a result of extensive pericarditic adhesion, involving not only the central tendon, but also the muscular part of the diaphragm on the one hand, and the interior wall of the thorax on the other. It usually occurs on the left side, and is known as *Broadbent's sign.* It should be noted, however, that this sign may be met with in cases of great enlargement of the heart without adherent pericardium.

In the epigastrium there may be several kinds of pulsation. The first thing to be determined is whether it is *strictly systolic*, coinciding exactly with the apex-beat, or whether the pulsation is *slightly delayed*, so as to appear just after the apex-beat has occurred.

In the former case the pulsation is caused by a dilated and hypertrophied right ventricle, which either conveys its impact directly to the parietes, or does so indirectly by exercising a thrust upon the liver, or else it is due to the apex-beat of a heart displaced to the right by some diseased condition, of which the most important are left-sided pleurisy and pneumothorax.

In the case of delayed pulsation, the cause may be arterial. The existence of an aneurysm of the abdo-

minal aorta would produce such an effect. More commonly, however, the condition is simply neurotic; whilst in other instances the pulsation of a normal abdominal aorta is conveyed to the surface either by the liver or by an abdominal tumour, such as pyloric cancer, which lies in front of it. (*See also* p. 55.)

In cases of regurgitation from the right side of the heart, pulsation also occurs just after the apex-beat, and is due to a distensile pulsation of the liver itself from the back flow of blood into the hepatic veins. It should be observed, however, that distensile pulsation of the liver is common only in gross heart-failure associated with tricuspid incompetence.

In order to observe with greater facility the characters and time relations of these various pulsations, one can employ small flags, made as light as possible and attached to the various areas of the chest-wall. To determine whether a pulsation is expansile, place one of these flags on either side of the tumour. If it is expansile their free extremities will recede from each other as the tumour fills. If it is desired to time the occurrence of two pulsations, after fixing a flag on the point where each occurs, one may take up a position in which they are as nearly in line as possible. It is then quite easy to determine which of them begins to move first.

Flags can readily be improvised by taking a piece of straw or a bristle about 3 in. long, fixing a fragment of gummed paper to one end, and surrounding the other with a pellet of modeller's wax or stiff ointment which will adhere with sufficient tenacity to the skin. Other and more primitive methods may also be used, such as passing a pin through a piece of adhesive plaster, with the head to the sticky side, and fixing it on the chest, or affixing little cones of

cotton-wool to the points in question by means of vaseline.

4. **Conspicuous veins.**—The veins of the thoracic wall may be unduly conspicuous. This occurs (*a*) when the patient's skin is unusually transparent; (*b*) when the patient has been undergoing considerable exertion, especially when the effort is of such a kind (e.g. playing a wind instrument) as to throw a strain on the respiratory system; (*c*) when an intrathoracic growth or aneurysm impedes the return of blood to the heart; (*d*) when the action of the right side of the heart is laboured; (*e*) when, in consequence of portal obstruction or of blockage of the inferior caval system, the blood returning from the abdominal viscera or lower limbs is forced to find its way through collateral channels.

III. PALPATION

Palpation determines—

(A) **Form of præcordia, etc.** [Confirms or modifies results of inspection.]

(B) **Movements**—

(*a*) Apex-beat. { Position. / Character.

(*b*) Other præcordial pulsations.

(*c*) Pulsations outside præcordia. { Heaving. / Expansile.

(C) **Vibrations**—

(*a*) Originating within the heart and blood-vessels. (Thrills.)

(*b*) Originating exocardially. (Friction.)

By palpation the observer not only confirms the facts determined by inspection and adds to their pre-

cision, but is also able to detect movements which are too slight to be noted by the eye alone. For palpation the patient should be placed in an attitude which he finds easy to maintain, since the exertion which a constrained position demands is certain to increase the observer's difficulties. If the patient is lying down, care must be taken to keep him on his back. By turning to his left side he will produce a very material alteration in the position of the apex-beat, which is thereby displaced outwards towards the axilla; whilst if he lies on his right side the apex of the heart may recede from the chest-wall, and an impulse, which in the more favourable dorsal attitude would be easily felt, may entirely disappear.

The position of the observer is almost as important as that of the patient. For the examination of the præcordia he should stand or sit, on the right-hand side. He should then place his right hand, which must be thoroughly warm, on the patient's chest. To begin with, the whole palm of the hand should be in contact with the chest-wall, and care must be taken not to dig the finger-tips into the intercostal spaces, as this causes discomfort, and may thereby interfere with the subsequent observations.

When pulsation is detected over any part of the region under examination, its exact localization is best determined by the pulp of the fingers.

The first pulsation to attract attention is that due to the **apex-beat.** Not infrequently the fingers will determine that this is really farther from the middle line than inspection would have led one to suppose. In such a case that point is to be taken as the cardiac apex which is the outermost and lowest where the finger is distinctly forced up with each beat of the heart. The sensation of a thrust from below raising the finger is important, because in not a few cases where

the heart is acting forcibly and frequently it communicates some vibration to portions of the chest-wall considerably beyond those which actually lie above it.

The observer, having thus determined the site of the apex-beat, must study the **extent** and **character** of the impulse. As has been previously stated (p. 109), it lies in health well outside the left parasternal line, but never beyond the left mammary line, is as a rule confined to one interspace, and seldom can be seen over an area of more than 1 in. in diameter. These points will now be carefully examined by palpation, and any deviation from them noted. In addition, however, an estimate must be made of the *energy with which the heart is acting*, and the apex-beat may be found to differ from the normal, in possessing a forcible or "*heaving*" *character*, or a *feeble* impulse.

When the pulsation of the apex is so feeble as to be imperceptible when the patient is lying down, it often becomes distinct if he sits up, and still more so if he leans forward. If, however, these postures are uncomfortable for a patient who is seriously ill, it is better to forgo such advantages than to tire one whose strength is already taxed. The chief causes of **impalpable apex-beat** are (*a*) a thick chest-wall, (*b*) emphysema of the lungs, and, less often, (*c*) a feeble heart.

When analysed, the varying characters of the beat will be found, after due allowance has been made for the thickness of the chest-wall and intervening lung, to depend upon the force with which the palpating finger is driven upwards, and upon the celerity and amplitude of the movement of the cardiac apex as it approaches the front of the thorax at each ventricular systole. *A shock* or "*jog*" is sometimes felt at the

apex in consequence of sharp closure of the pulmonary and aortic cusps.

In addition to pulsation, vibrations may sometimes be observed at or near the cardiac apex. Such vibrations are termed **thrills.**

The time of their occurrence in relation to the apex-beat or to the carotid must be determined. When they commence with the apex-beat and continue during the period of ventricular contraction, they are termed **systolic;** if they are felt whilst the ventricles are relaxed, they are termed **diastolic;** if they occur near the close of diastole, they are termed **presystolic.**

These thrills may be due either to valvular disease, to pericardial friction, or to friction resulting from pleurisy over that part of the left lung which lies in front of the heart. The thrills due to valvular disease will exhibit a more definite relation to the apex-beat, both in point of time and in situation of maximum intensity, than those whose origin is exocardial. A systolic, diastolic or presystolic thrill felt at the apex indicates mitral stenosis. A thrill in the pulmonary area is invariably systolic in time and indicates either pulmonary stenosis or patent ductus arteriosus. A thrill in the aortic area is usually systolic in time signifying aortic stenosis; less commonly it is diastolic and due to aortic incompetence.

The true presystolic thrill has been aptly compared to the sensation produced when the hand is placed on the back of a purring cat.

Pericardial or **pleural thrills** will be readily recognized as such when the patient is auscultated (*see* p. 148). They are generally to-and-fro in character, and are always audible as well as palpable.

When the **liver exhibits expansile pulsation,** owing to backward pressure in the veins due to tricuspid incompetence, the whole organ will be found

to be affected, and in most cases the expansile character of the movement can be distinctly made out. The pulsation is most readily recognized by placing one hand over the anterior surface of the enlarged liver, and the other at the back. In cases of doubt as to the nature of epigastric pulsation, a change in the patient's posture, particularly if he is made to assume a knee-elbow position, sometimes clears up the difficulty.

IV. PERCUSSION

Percussion is used to try to determine the boundaries of the heart and surrounding viscera, and to detect the presence of pericardial effusion and thoracic aneurysm. It often gives fallacious results and for this reason those who crave for machine-made diagnosis have sought to throw it into disrepute. (For methods which purport to measure the size of the heart by X-rays, *see* p. 179.)

For the most part the heart is surrounded by resonant lung, but does not lie so deeply as to be out of reach of a firm percussion stroke. The method used is to note, as one percusses towards the cardiac region, the points at which the lung resonance begins to grow emptier. In two areas this cannot be achieved. At the base of the heart the roots of the great vessels produce a dulling of the lung resonance which cannot be distinguished from that caused by the heart, while the lower border of the viscus is in relation to non-resonant liver which yields on percussion the same dull thud that the heart itself does.

By percussing in the fourth interspace from the left lung towards the heart one is able to define the left border more or less precisely. It is found about half an inch internal to the mammary line. If it is per-

cussed at a higher level it will be found to curve round so as to merge insensibly with the upper border. To ascertain the upper border one should percuss downwards between the left lateral sternal and left parasternal lines. This border is found at the third rib or upper border of the third interspace in the left parasternal line. The right border of the heart is just

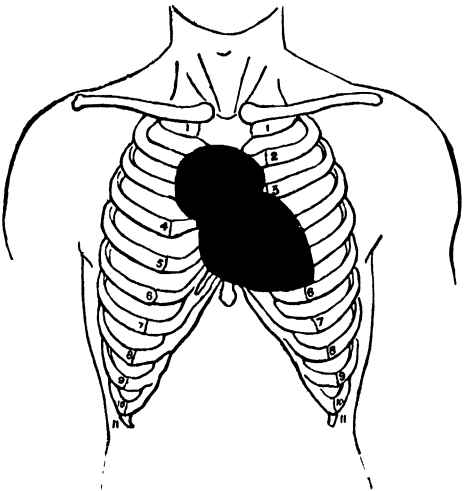


Fig. 15.—Aortic aneurysm.

to the right of the right lateral sternal line at the level of the fourth rib. It is difficult to define since the sternum acts as a sounding-board.

Lying as they do largely behind the sternum, the dullness due to the great vessels can seldom be made out by percussion. If, however, there is aneurysmal dilatation of the ascending aorta, a dull area can often be mapped out. It is continuous below with that of

the heart, above it bulges outwards to the right of the sternum at the level of the 2nd interspace and adjacent ribs; whilst the sound produced by percussion of the manubrium sterni is also rendered much less resonant, or even, in cases where the aneurysm is large, absolutely dull (Fig. 15). Aneurysm of the descending part of the aortic arch will sometimes cause

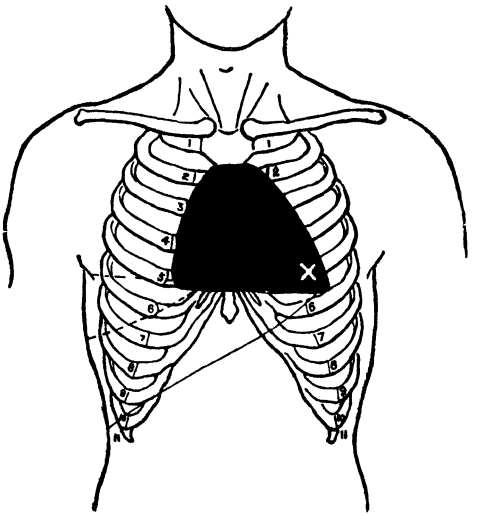


Fig. 16.—Pericardial effusion.

dullness on percussion in the 2nd interspace on the left, but in this circumstance the vascular swelling can usually both be seen and felt.

If the relative dullness extends to the **left of the apex-beat,** provided the lung and pleura are healthy, we have probably to do with pericardial effusion; and in this case the right border may be found at a considerable distance to the right of the sternum—it

may be as far as the right parasternal line. If the cardiac dullness extends to the left of the mammary line, but does not reach beyond the situation of the apex-beat, the condition is probably due to dilatation and hypertrophy of the left ventricle, unless the heart is bodily dislocated to the left by some such cause as massive pleural effusion on the right side. If the dull-

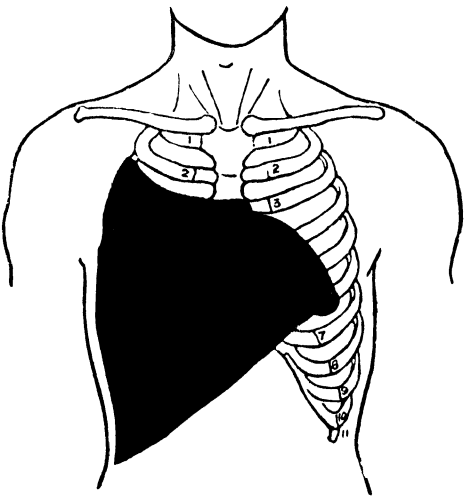


Fig. 17.—Displacement of heart in right-sided pleural effusion.

ness extends, in the absence of lung disease, pleurisy, or pericardial effusion, to **the right of the sternum**, one is justified in concluding that the right auricle is enlarged.

The dullness which one finds in *pericarditis with effusion*, or in *hydropericardium*, varies with the amount of fluid which is present, but in well-marked cases is pear-shaped, with the broader end

downwards and the upper end higher and broader than the ordinary upper limit of dullness (Fig. 16).

The chief causes outside the heart and pericardial sac that produce an increase in the area of cardiac dullness are consolidation or tumour of the lung, or pleural effusion.

The situation of the area of cardiac dullness is

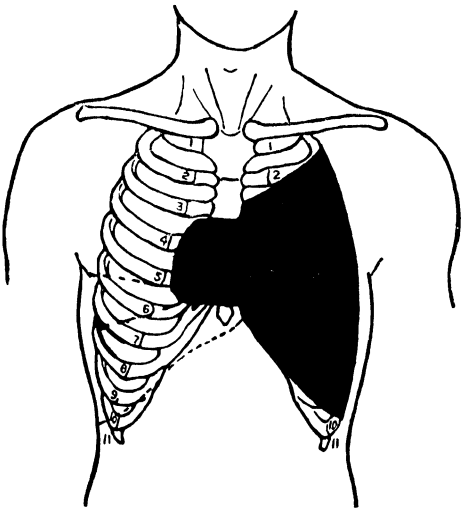


Fig. 18.— Displacement of heart in left-sided pleural effusion.

naturally altered by **changes in the position of the heart.** These result from its displacement by the pressure or traction of other organs, or from developmental anomalies. Thus in dextrocardia the heart is placed with its apex to the right, and the area of dullness is then the mirror image of what is usually found.

In some cases of pericarditis a dull area can be found posteriorly near the inferior angle of the left scapula (Bamberger's sign). It is believed to be due to pressure on the lung by the distended pericardium, and is said to occur also sometimes in cases of great enlargement of the heart without pericarditis.

V. AUSCULTATION OF THE HEART AND VESSELS

Auscultation determines—

(A) **Character of the heart-sounds** with respect to —
1. Intensity.
2. Rhythm.
3. Quality.

(B) **Abnormal sounds associated with the heart-sounds**—

(*a*) Over præcordia	Endocardial murmurs. Pericardial friction.
(*b*) Over vessels	Clear sounds. Murmurs or bruits.

The stethoscope.—Auscultation, though sometimes performed with great advantage by the direct application of the ear to the chest-wall (covered with a thin towel) is generally conducted by means of a stethoscope and the student cannot take too great pains in choosing a good one.

Stethoscopes are of two types, single and binaural.

In the choice of a *binaural* stethoscope, one should avoid instruments with unnecessary joints and loose parts, or with woven tubes. The chest-piece should not be very large, nor made of metal; vulcanite is not so chilly, and is easily cleaned. Unless the ear fittings are suitably shaped, much discomfort will be produced. They should be comfortable and only press lightly into the ears.

The cardiac cycle and surface anatomy of the valves and vessels.—In order to understand the various sounds which can be heard by listening to the heart through the chest-wall, a clear conception of the events which occur during a cardiac cycle is essential.

After the completion of a beat the auricles and ventricles are both relaxed. Thereafter the auricles con-

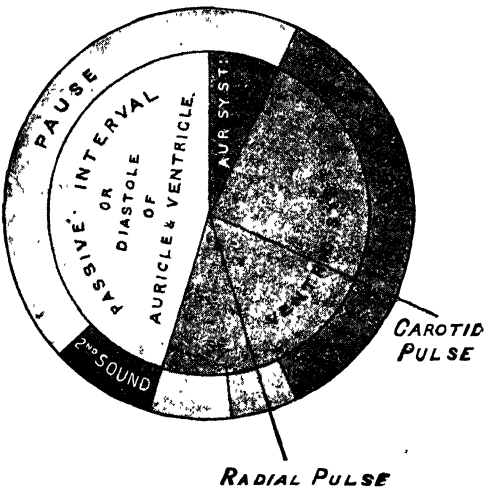


Fig. 19.—Cardiac cycle.

tract, forcing their contents through the cuspid valves into the ventricles, and filling them. The ventricles then contract in turn, expelling the blood into the vessels, whilst the auricles [atria] begin to relax and become refilled with blood; finally, the ventricles relax also, and so the cycle is completed. We have thus in rapid succession auricular systole, ventricular

systole, and ventricular diastole; auricular diastole commencing during ventricular systole, and ventricular diastole continuing through auricular systole.

The beginning of ventricular systole is marked by the closure of the mitral and tricuspid valves, which had remained open during the systole of the auricles [atria], and by the occurrence of the apex-beat; the beginning of ventricular diastole is marked by the closure of the aortic and pulmonary valves, which remain closed until the beginning of the following ventricular systole. The pulse in the carotid occurs a short time after the commencement of ventricular systole; in the radial artery it is decidedly later in its appearance, and therefore the radial pulse must never be taken as an index of the commencement of ventricular systole. As an index of the commencement of systole one should take as a guide the carotid pulse or the apex-beat.

Various authors have constructed diagrams to represent the sequence of events in a cardiac cycle. The accompanying one (Fig. 19) may be taken as representing these in an ordinary case, though the relative duration of the successive events will be found in practice to vary within fairly wide limits. The most important variation is when the heart acts with unusual rapidity, the duration of diastole is curtailed to a greater degree than that of systole, and hence a shorter interval elapses between the time of closure of the semilunar valves and the commencement of ventricular systole than one would infer from an examination of the diagram. For some purposes it is found more convenient to unroll the above diagram, so that the sequence is represented along a straight line instead of round a circle. Fig. 20 shows the details of the cardiac cycle, and especially their exact time relations, with greater accuracy than the simpler diagram on

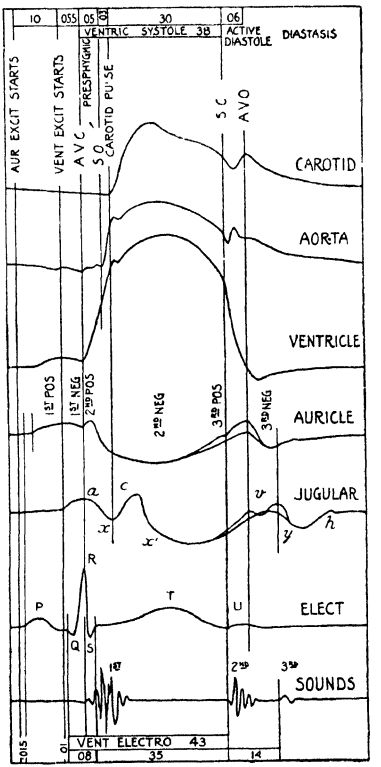


Fig. 20.—The cardiac cycle in relation to the pressure in the arteries, veins, and chambers of the heart, and to the electrocardiographic curve. (*From Lewis's "Mechanism of the Heart Beat."*)

The times at which the excitation wave begins in auricle and ventricle are represented by vertical lines, the movements of the valves are also represented in a similar way. The scale of abscissæ is 1 mm.=0·015 sec.

A.V.C. and A.V.O. = closure and opening of auriculo-ventricular valves respectively: S.O. and S.C. = opening and closure of semilunar valves respectively. Other verticals are drawn at convenient points, and the chief time-intervals are marked above and below in seconds.

In the jugular curve, *a*, *c*, and *v* represent the auricular, carotid, and venous-stasis waves: *x*, *x'*, and *y* the troughs which follow them.

In the electrocardiographic curve, P represents auricular systole, Q, R, S are associated with the initial stages, and T with the final stage of ventricular systole.

p. 126. It will be found of special value when the student arrives at the consideration of the venous pulse.

It must be recollected that in clinical language the words systolic and diastolic are used with reference to the state of the ventricles, events which take place during the auricular contraction being described as diastolic (or presystolic). On the other hand, physiologists generally regard the period of auricular contraction as included in the systolic period of the cardiac cycle. Clinically, then, the systolic phase of the cycle begins with the apex-beat and commencement of the first sound. It terminates immediately before the second sound, whose commencement marks the beginning of the diastolic period.

In addition to a knowledge of the cardiac cycle, auscultation presupposes acquaintance with the situation of the valves of the heart and of the course of the principal arteries, as well as of the areas where sounds produced at the valves are best heard. For full particulars the student must consult works on regional anatomy. The following summary merely recapitulates the most important facts:—

The **pulmonary valve** lies horizontally at the level of the upper border of the 3rd left costal cartilage; the right half of the valve lies under cover of the sternum, the remainder passes outwards behind the costal cartilage (Fig. 21).

The **aortic valve** lies farther from the surface, and at a slightly lower level. Its situation may be indicated on the front of the chest by a line drawn obliquely across the left half of the sternum on the level of the lower border of the 3rd costal cartilage.

The **mitral valve** lies slightly obliquely behind the inner end of the 4th left costal cartilage and adjoining

part of the sternum. The **tricuspid valve** is placed much more obliquely; its upper end is opposite the 4th cartilage or interspace, and its lower near the lower border of the 5th right sterno-costal articulation. It marks the line of junction between the right auricle [atrium] and right ventricle.

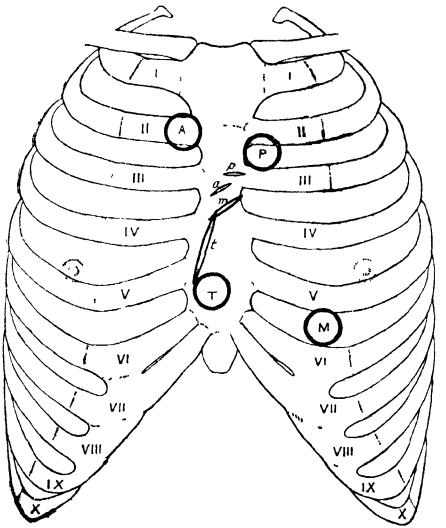


Fig. 21.—Position of the cardiac valves and auscultatory areas.

The **pulmonary artery** is situated at the inner edge of the 2nd left interspace, and behind the adjacent part of the sternum. At the lower border of the 2nd cartilage it divides into its branches to the right and left lungs. The **ductus arteriosus** passes upwards from the left branch to join the aorta.

The aorta arises behind and slightly lower down than the pulmonary artery, and, passing upwards and

to the right, approaches the surface of the chest most closely at the inner end of the 2nd right costal cartilage, arching backwards and to the left from that point. The **innominate artery** passes in a direction represented by a line drawn from the middle of the manubrium to the right sterno-clavicular junction.

A stethoscope placed over the valves of the heart would fail to distinguish at which of them a given sound takes origin, because they lie so near each other that the sounds from all of them would reach the chest-piece. Besides, in the case of the valves that lie more deeply the sounds would have to pass through the chambers of the heart which are situated between them and the surface, and thereby their clearness would be impaired. To avoid these inconveniences, the sounds produced by each valve are listened for over that part of the chest where the cavity in which the valve lies most closely approaches the surface, and is most remote from the other cavities of the heart.

Hence one listens to the mitral valve at the cardiac apex, to the tricuspid at the lower end of the sternum, to the aortic over the aorta at the 2nd right costal cartilage, and to the pulmonary over the artery in the 2nd left intercostal space. It is found practically that in these regions the sounds of the respective valves are heard with a maximum of loudness and distinctness. They are therefore called the **mitral, tricuspid, aortic,** and **pulmonary areas**, although they do not lie immediately over the valves from which they derive their names. Auscultation should be performed systematically over these areas. In ordinary cases the student may begin with the mitral area, making certain of the time at which the sounds that he hears occur in the cardiac cycle by feeling the carotid pulse whilst he listens. He may then pass to the tricuspid area, thereafter to the aortic, and lastly to the pulmonary. When

necessary, auscultation may also be performed along a diagonal line joining the mitral and aortic areas. This is often of service, as, for instance, when a mitral systolic murmur is associated with an aortic one.

In health two sounds are often heard over each of these areas, the first corresponding with the beginning of ventricular systole, the second with the commencement of ventricular diastole. The first sound depends, from the clinical standpoint, on the closure of the mitral and tricuspid valves, and on the muscle-tone of ventricular contraction. The second sound is due to the closure of the aortic and pulmonary valves, and also, but very subordinately, to tension of the vessel-walls. This sound is sharper and shorter than the first, which continues through an appreciable period of systole, but not until its termination. At and to the left of the apex, only the aortic element of the second sound is audible. The observer must remember that it is always important to note the character of both the first and second sounds in each of the areas.

Deviations from the Normal in Disease

In disease the following deviations from the normal may occur:—

1. The sounds may have a different intensity, both absolutely and relatively to each other, from that which they possess in health. In estimating this, allowance must be made for the thickness of the chest-wall and the volume of the lungs.
2. The sounds may be doubled.
3. The rhythm or spacing of the sounds may be altered.
4. Adventitious sounds may be heard, either replacing or occurring along with the heart-sounds.

I. ALTERATIONS IN INTENSITY

(a) **The first sound may be weaker than usual.** Decided shortening or weakness of the first sound during the course of an illness, still more its disappearance, may portend cardiac failure. In acute febrile disease this change may occur rapidly, and should always be looked for.

(b) **The first sound may be louder than usual.** It is then said to be **accentuated.** A well-known example is the sudden loud first sound at the apex in mitral stenosis. Accentuation of a different quality often accompanies a simple tachycardia and disappears with fall in the heart-rate. In hypertrophy of the left ventricle the sound is accentuated, but dull, prolonged, and thudding.

(*c*) **If the second sound is more distinct** in the mitral or tricuspid areas than the first, we have to do with either a weakened first sound or an accentuated second; whilst if the first sound is louder than the second in the aortic and pulmonary areas, the first sound is accentuated.

(*d*) The **relative loudness** of the second sound in the aortic and pulmonary areas varies somewhat, and is a good deal influenced by the patient's age. The pulmonary sound is rather more accentuated than the aortic in youth; in old age the reverse is the case when the subject is in good health.

Accentuation of the second sound means that the valve where the accentuated sound is produced is closed with unusual force. The force of closure depends on the momentum of the column of blood that effects it, and the momentum depends equally on the mass of moving blood and on the velocity of its recoil against the valve. In the aorta the mass of blood is increased when the vessel is dilated

near its origin; the velocity of recoil when, in consequence of contracted arterioles or other obstruction to the outflow of blood, the arterial blood-pressure is increased. When the aortic accentuation is due to the former cause, the sound often assumes a peculiar resonance suggestive of the echo produced when a cork is drawn from an empty bottle. Over the pulmonary artery an accentuation of the second sound generally indicates increased blood-pressure in the pulmonary circulation, due to disease either of the lungs or of the left side of the heart, as in mitral stenosis. In *pneumopericardium* the sounds are singularly clear and resonant, in *pericardial effusion* they are faint and muffled.

2. REDUPLICATIONS

Under certain conditions the first or the second sounds may be **doubled** or again, extra heart-sounds may appear. Clinically, **reduplication of the first sound** is known as the **"bruit de galop,"** where the rhythm is that of a galloping horse's hoof-beats; two sounds in rapid sequence and a third after a definite interval, the accent being on the second element of the triplet. It sometimes proves difficult to distinguish a short presystolic murmur from reduplication of the first sound. The presence or absence of other signs of mitral stenosis will usually decide, and if not the diagnosis may become clear at a subsequent examination. Reduplication of the first sound is common in hypertension and heart failure. Sometimes it may be caused by partial heart-block where the P—R interval in the electrocardiogram is prolonged. In rare cases it is a clinical indication of a lesion of one branch of the bundle of His, as shown by an electrocardiogram.

Reduplication of the second sound indicates, in a large proportion of the instances in which it is heard, an increase of pressure in the pulmonary circulation. It occurs, therefore, in the majority of cases of mitral stenosis and in congenital heart disease. It is also found where the right and left ventricles fail to contract simultaneously, whether the failure be due to an increase of work thrown upon one of them, or to the presence of structural changes in the heart-muscle. It also occurs physiologically. Reduplication of the second sound gives rise to a cantering rhythm (**bruit de rappel**) like that of the hoof-beats of a cantering horse: one sound, an interval, and then two sounds in rapid succession. It must be distinguished from the gallop rhythm already described.

3. ALTERATIONS IN RHYTHM OR SPACING

Alterations in the rhythm or spacing of the sounds deserve attention. The usual rhythm is that of triple time in music, with the accent on the first beat in the mitral and tricuspid areas, and on the second in the aortic and pulmonary, whilst the third beat is silent. This is slightly modified by the fact that a quickly acting heart gains time chiefly in the period of ventricular diastole, but the relation of the sounds is less modified by this than are the phases of the cycle. When, however, the vitality of the heart has been seriously impaired by long-continued high blood-pressure, such as is seen in chronic nephritis, and especially if fever or some such cause assists in weakening the myocardium, the sounds become almost equidistant, the period of ventricular systole being unduly lengthened.

This *deliberate pendulum-like sequence* of the sounds should always be regarded with considerable anxiety, as it points to serious involvement of the cardiac muscle.

4. ADVENTITIOUS SOUNDS

Adventitious sounds may be of three kinds: endocardial, vascular, or exocardial. Abnormal endocardial sounds are called **murmurs** or bruits. They are due to disease either of, or close to, the valve where they occur, when they are often known as organic; or to some alteration in the state of the blood which, by affecting its viscosity on the one hand and the nutrition of the tissues of the heart and vessels on the other, produces the conditions necessary for the development of a murmur.

The physical explanation of murmurs is by no means simple. The following are some of the factors concerned in their production :—

i. The viscosity of the blood.
ii. The velocity of the bloodstream.
iii. The passage of the stream from a narrower into a wider channel.

The third condition is equally well produced when a narrowed orifice leads to a normal cavity beyond it, or when a normal orifice opens into a dilated cavity.

Endocardial murmurs always have a definite relation to the events occurring in the course of the cardiac cycle, their time and import varying with their point of origin.

In the so-called " organic " cases, where the valves or their surroundings are implicated, a murmur may result either from obstruction to the onward flow of the blood, or from leakage backwards through a closed but incompetent valve. The former are known as obstructive murmurs, the latter as regurgitant. In examining a murmur the following points must be noted:—

(*a*) Its time of occurrence.
(*b*) Its point of maximum intensity.
(*c*) Its direction of selective propagation beyond the præcordial area.
(*d*) Its character.

The **time of its occurrence** is noted with reference to the sounds of the heart, and these by comparison with the time of occurrence of the apex-beat.

The **maximum loudness of a murmur** which has been produced at a given valve usually occurs at the point where the valve-sound would be best heard in health. To this rule, however, there are some exceptions.

Experience shows that valvular murmurs are not equally well heard at all points of the chest-wall which are equidistant from the point of their greatest intensity, but that each is much more distinctly audible at a distance in some directions than in others; this fact is expressed by saying that such murmurs have **directions of selective propagation.**

The **character** of the murmur also helps to decide a doubtful case. Obstructive murmurs are apt to be rough; regurgitant, to be soft and blowing.

The pitch and general quality of murmurs vary greatly; some have quite a distinct musical character, others are harsh and sawing. The loudness of a murmur has no relation to the amount of damage which causes it. A very loud murmur is often far less serious than one so soft as to be nearly inaudible.

Murmurs due to disease of postnatal origin are very much oftener found to proceed from the valves of the left side of the heart than from those of the right, and in adult life murmurs at the pulmonary area, due to morbid processes arising in this valve, are rare. The following is a short epitome of the chief murmurs which may be heard at the various valves, and of exocardial sounds: the diagrams illustrate the position of the more common murmurs in the cardiac cycle.

(1) *Mitral Murmurs*

Mitral murmurs may be either (*a*) obstructive or (*b*) regurgitant. (Fig. 22.)

(*a*) **Obstructive murmurs** occur during ventricular diastole, and are invariably of organic origin. They sometimes follow immediately on the second sound, when they are known simply as *diastolic*. At other times the murmur is separated from the second sound by a brief interval, but terminates before the occurrence of the first sound; it is then called *mid-diastolic;* in yet other instances the murmur only begins with the advent of auricular contraction, when it is designated a *presystolic* murmur. In each case

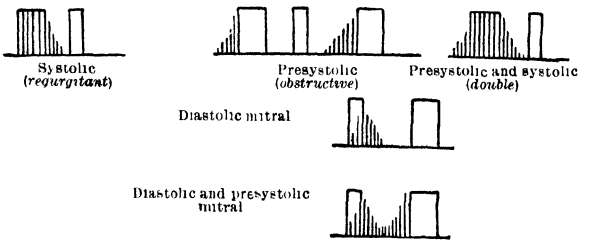


Fig. 22.—Mitral murmurs.

the murmur is due to the onward rush of the blood through the deformed or narrowed mitral valve into the wider cavity of the left ventricle. During the earlier part of diastole this is effected by the aspiration of the relaxing ventricle, which is ordinarily strongest near the beginning of diastole—at the end of the period the contracting auricle is the main agent in producing the flow. Sometimes an early diastolic murmur is followed by a moment of silence, which is then succeeded by a presystolic murmur.

Best heard at the apex, or rather nearer the

sternum, these murmurs have no direction of selective propagation; they are harsh and rumbling in character, more particularly when of the presystolic variety, and very often are associated with a distinct thrill. In the majority of cases the second sound is reduplicated, so that the murmur and accompanying

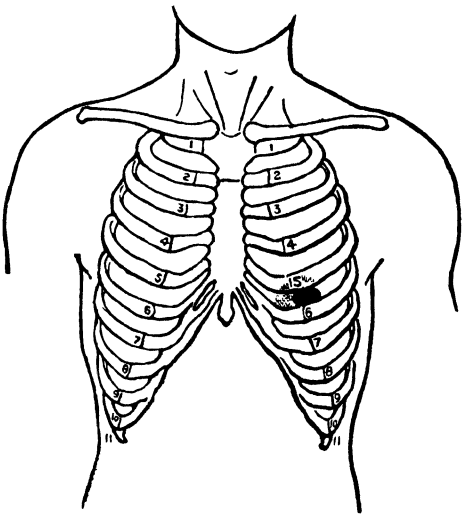


Fig. 23.—Presystolic mitral murmur.

sounds may be phonetically represented by "*rr*úp ti-ti"; or, where the heart's action is rapid and the murmur occupies a considerable portion of the diastolic period, by "ti-ti *rr*úp." Occasionally the presystolic murmur is accompanied by a mitral obstructive murmur occurring at the beginning of diastole, when the phonetic representation would become "*rr*úp ti tiff, *rr*úp ti tiff," or, if the murmur

occur a shade later in diastole, by "*rr*úp titi iff, *rr*úp titi iff." (Fig. 23.)

The exact significance of the reduplication of the second sound in mitral stenosis is a matter of doubt. It is not improbable that one element of the double sound under discussion may originate in the

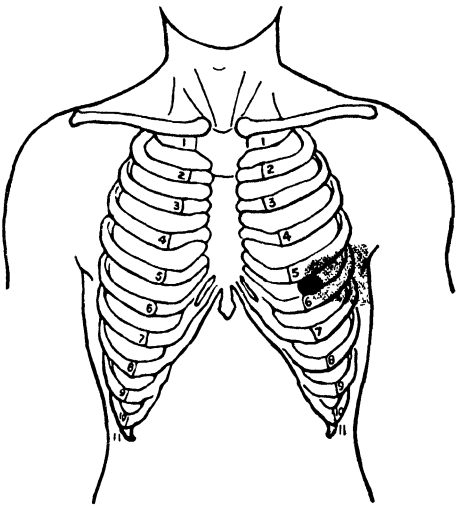


Fig. 24.—Mitral systolic murmur—propagation in front.

mitral valve, whose segments, partially adherent along their adjacent margins, are no longer free to fall backwards before the blood-stream as it again passes from the auricle to the ventricle at the beginning of diastole, but are suddenly arrested in their progress, and, bulging into the ventricular cavity, become tense, and emit a sharp sound like that produced by a sail suddenly bellied by a gust of wind.

(*b*) **Regurgitant murmurs** occur during ventricular systole, and may be either organic or simply due to enlargement or to change in the blood. They begin with the apex-beat and replace more or less completely the first sound in the mitral area. Their point of maximum intensity is at the apex, their

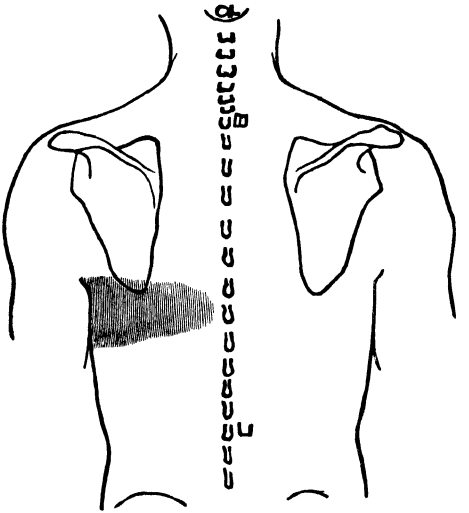


Fig. 25.—Mitral systolic murmur—propagation behind.

direction of selective propagation is outward towards the axilla and angle of the left scapula, and they are generally soft and blowing in character. Slight mitral systolic murmurs, especially those due to dilation of the ventricle and not to disease of the valve curtains, frequently lack any selective propagation backwards.

(2) *Aortic Murmurs* (Fig. 26)

(*a*) **Obstructive murmurs** occur during ventricular systole; they are due either to obstruction of



Fig. 26.—Aortic murmurs. (*After Wyllie.*)

the ostium aortæ from valve disease or to aortic dilatation beyond a normally sized ostium. They are rough in character; have their area of greatest intensity at

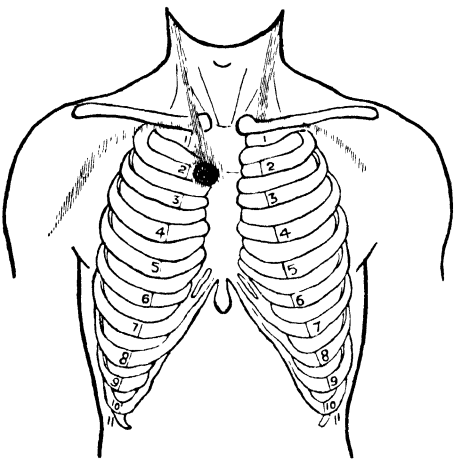


Fig. 27.—Aortic systolic murmur.

the second right costal cartilage near the sternum; are propagated with the bloodstream into the arteries; and may, in most instances, be readily heard over the carotids—sometimes at a much greater distance. (Fig. 27.)

(*b*) **Regurgitant murmurs** occur during ventricular diastole; they begin with the closure of the

semilunar valves, and may replace in part or completely the normal second sound in the affected region. They may be heard in the aortic area: not infrequently, however, they are better heard over the left half of the sternum, at the level of the 3rd rib and interspace and lower. Their direction of selective propagation is towards the lower end

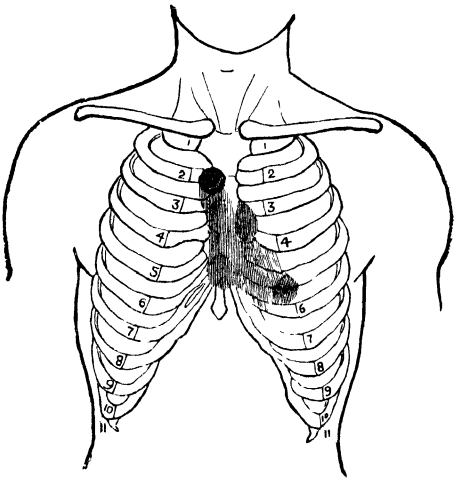


Fig. 28.—Aortic diastolic murmur.

of the sternum, though occasionally they are almost equally well heard near the apex (Fig. 28); their character is less harsh than that of systolic aortic murmurs. Their intensity is greatest at first, and gradually diminishes during the diastolic period.

In many instances one finds that a **double murmur** is present at the aortic orifice, the systolic element of which is not caused by real stenosis of the ostium, but by roughening and deformation of the

valve segments, the diastolic murmur being due to the backward leakage through the misshapen cusps, This double murmur often possesses a very distinctive " sawing " or " bellows " character.

Austin Flint has directed attention to the presence of a presystolic murmur at or near the apex of the heart in certain cases of aortic disease where, post mortem, no change was

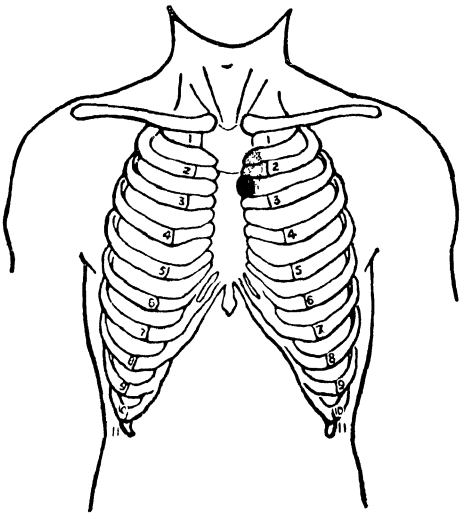


Fig. 29.—Pulmonary systolic murmur

found to be present in the mitral valve. Most probably this murmur indicates a dilatation of the left ventricle.

(3) *Tricuspid Murmurs*

These are comparatively rare.

(*a*) **Obstructive murmurs** have their maximum intensity at the lower end of the sternum, and are systolic in time. They have no selective propagation.

(*b*) **Regurgitant murmurs** have a similar character to mitral systolic murmurs, are best heard in the tricuspid area, and are associated with an exaggeration of the venous pulse (*see* p. 165). They are a sequel to disease of the tricuspid valve.

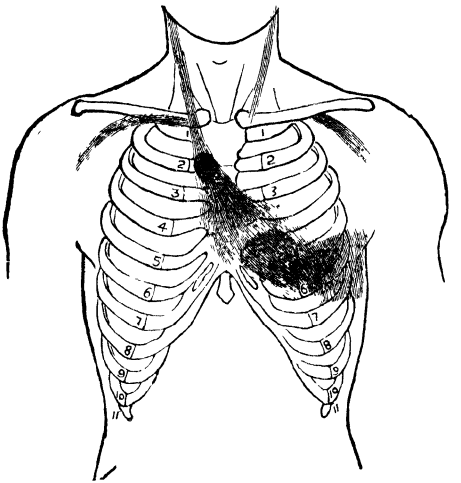


Fig. 30.—Combined aortic and mitral systolic murmurs.

(4) *Pulmonary Murmurs* (Fig. 29)

These are best heard in the pulmonary area, have no direction of selective propagation, though occasionally they are well heard as high as the 1st rib, are usually systolic, and are rarely due in postnatal life to disease of the valve. They are common in fevers and anæmias, and are well heard in many cases of exophthalmic goitre. A diastolic pulmonary murmur of organic origin is excessively rare.

(5) *Multiple Murmurs*

In a large number of cases **more than one murmur** is present during the cardiac cycle. When they occur at different epochs, it is easy to study each separately; when two or more occur together, each will be found to possess its own position of maximum loudness, its characteristic direction of selective propagation, and its peculiar quality of sound. (Fig. 30.) Each lesion, moreover, will produce more or less definite effects on the general circulation, and by observing these by the other methods at our disposal, a diagnosis can usually be arrived at. Some murmurs vary very much with the posture of the patient; it is therefore important to examine the heart in both the erect and supine positions.

(6) *Congenital Murmurs*

(*a*) In a child with cyanosis from congenital heart disease, a harsh systolic murmur (often with thrill) in the second left interspace, with faint or absent pulmonary second sound, indicates **congenital pulmonary stenosis.** It is usually combined with a ventricular septal defect.

(*b*) In a child or adult without cyanosis, and even without symptoms, a peculiar loud continuous murmur (systolic-diastolic) in the second left interspace, often transmitted towards the neck, is characteristic of a **patent ductus arteriosus.** A thrill is usually associated, and the pulmonary second sound accentuated.

(*c*) A harsh systolic murmur at the base of the heart, with enlargement of the ascending aorta, large arterial pulsation and high pressure in the upper limb and the opposite in the lower, and a collateral circulation, may occasionally be produced by **coarctation of the aorta.**

A patent foramen ovale, like certain other developmental defects, need not give rise to a murmur.

The history, cyanosis when present, site of the murmur nearer the base than the apex, and its usually harsh character, in general distinguish congenital from acquired lesions. Radioscopy and electrocardiography will help to make a diagnosis.

(7) *Cardio-Pulmonary Murmurs*

Murmurs may be closely simulated by sounds due to diseased conditions in the neighbourhood of the heart, for in such circumstances the heart may be displaced, or it may be pressed upon, or its movements may through adhesions be communicated in an abnormal way to the lung, and induce abrupt movements of the air contained in its tissue and in the bronchi. It is also possible that a slight degree of pleural friction may become audible during cardiac systole if the roughened surfaces are situated near the apical region of the heart.

Cardio-pulmonary murmurs occur at the time of cardiac systole. They generally begin about the middle or near the end of that period. They are short in duration and are best heard during inspiration, but a very full inspiration may render them faint or inaudible.

Their commonest situation is just outside the apex-beat. In a smaller number of cases they are only heard at the base of the heart, at or near the 2nd left intercostal space. Changes in the posture of the patient often cause them to disappear entirely. In arriving at a diagnosis the observer must take into account the condition of the lungs, pleura, and abdominal viscera, the phenomena observed on auscultation over the trachea, where true mitral murmurs are practically never heard, and the character of the pulse, which is dealt with in a subsequent section.

(8) *Hæmic and Vascular Murmurs*

In **anæmia** hæmic murmurs frequently occur over the heart and vessels. They are usually heard in the 2nd left intercostal space over or just external to the pulmonary area, but they are also heard at times in the mitral, and much less frequently in the

tricuspid and aortic areas, the last being particularly uncommon. *In all cases such murmurs are systolic in time and become less distinct or even inaudible when the patient assumes an upright posture.*

Hæmic murmurs may arise **in the larger arteries,** and are present independently of the pressure of the stethoscope.

There are, moreover, other sounds which may become audible in the arteries, and which are the result of changes in the pressure of the bloodstream. The most notable instance of this is found where relaxed arteries are so rapidly distended by a large blood-wave that their walls are thrown into vibration by the sudden strain, and a sound is produced which corresponds with the advent of the pulse-wave. In cases of aortic regurgitation, where these conditions are most fully developed, we have also a second sound which occurs at the instant when the pressure once more falls off. This double sound, when heard in the femoral, is very characteristic of aortic regurgitation. Pressure produced by an ill-applied stethoscope often converts these sounds into murmurs.

When there is an **aneurysmal dilatation** of the aorta, murmurs are absent except in the presence of aortic incompetence. The aortic second sound may be accentuated over the sac, but no definite rule holds for such cases. When an aneurysm opens into another large vessel—e.g. the superior vena cava—the murmurs produced may be very loud, and are heard in unusual situations while the pulse becomes collapsing in character.

(9) *Exocardial Sounds*

Exocardial sounds may be due either to pericardial friction or to a localized pleurisy near the heart.

When **pericardial friction** occurs over an area uncovered by lung, it has a singularly superficial

character, and thus can often enough be readily recognized.

Unlike the murmurs already described, pericardial friction does not correspond definitely with the events of the cardiac cycle. It is generally more distinct in systole than in diastole, but tends to exhibit a to-and-fro character, the first element occurring during systole and the second during diastole, but not necessarily commencing at the beginning of either phase. Sometimes the sound occupies the latter part of systole and the early part of diastole without exhibiting any pause between its first and second elements; sometimes it remains audible during the whole of the cardiac cycle. Further, its position of greatest intensity does not correspond with any of the areas in which valvular murmurs are best heard, and it is not propagated to a distance, but remains confined within narrow limits. Its position may be observed to vary from day to day. As a rule, it appears first near the base of the heart



Fig. 31.—Pericardial friction. (*After Wyllie.*)
A. Ordinary type, one rub in systole and one in diastole.
B. Type with triple rhythm, one rub in systole and two in diastole.

on the left side, but when the condition has become general it is best heard near the left nipple, and is sometimes associated with a distant fremitus. The intensity is often considerably modified by the posture of the patient. When the inflammatory process involves the auricle as well as the ventricle, the to-and-fro rub may be replaced by a triple friction sound (Fig. 31).

When sufficient fluid is present, the cardiac sounds become faint and distant.

When air and fluid are both present in the peri-

cardial sac—an event of very rare occurrence—a churning or "water-wheel" sound can be heard on auscultation.

To distinguish between the rub of pericarditis and that of pleurisy over a neighbouring portion of lung the patient should be instructed to hold his breath. Pericardial friction is unchanged by this, but if it is of pleural origin it will either be much reduced in intensity or will wholly cease. On the other hand, deep respiration will increase the pleural sound, but will not influence the pericardial.

The possible coexistence of both pleuritic and pericardial friction must not be overlooked.

VI. THE PULSE

The examination of the pulse gives us direct information regarding two things, namely, the condition of the vessel-walls and the amount and variations of pressure of the contained blood. By intelligent observation of these facts we can obtain very valuable information regarding the state of the heart and circulation, as well as the general state of the patient.

When any observation is to be made on the pulse, the patient should be resting; and, except for special purposes, should not have been making any effort for some little time previous to the examination. The pulse is most readily felt when the patient's forearm is pronated and the wrist slightly flexed. In cases of aortic regurgitation the peculiar character of the pulse (*see* p. 164) is more distinctly brought out when the patient's arm is elevated.

To feel the pulse, place three fingers of the right hand on the patient's radial artery at the wrist. It is immaterial whether the observer's index finger is nearer the elbow or the hand of the patient, but for

beginners it is best to select the same position in all cases. If it is made a habit to examine both radials in every case, errors in diagnosis, such as failing to detect the presence of aortic aneurysm or an abnormal position of the vessel, will sometimes be avoided.

When the artery is beneath the finger, the following observations should be systematically made:—

(A) **Factors which depend upon the heart.**
1. Rate of pulse.
2. Rhythm of pulse (irregularity, inequality).

(B) **Factors which depend upon the vessel.**
3. Size of the artery (calibre).
4. Condition of the vessel-wall.

(C) **Factors which depend on both heart and vessel.**
5. Amount of movement during a pulse-wave (volume).
6. The blood-pressure in the vessel during the beat (systolic pressure ; " force ").
7. The blood-pressure in the vessel between the beats (diastolic pressure; " tension ").
8. The form of the individual pulse-wave (rise, maintenance, and fall of pressure; dicrotism).

1. The **rate** of the pulse is stated as so many beats a minute. It is well to count the rate not when the fingers are first laid upon the pulse, but when any quickening due to agitation of the patient has subsided and the pulse has resumed its normal rate. Count the beats for not less than half a minute and give the results in beats per minute. The beat at which the observation commences should not be counted.

2. The successive beats of the pulse may recur at equal or unequal intervals, giving a regular or irregular **rhythm.** The latter may consist of nothing more than an occasional intermission in the pulse, or the intermission may recur, as in pulsus bigeminus, after two beats. The individual beats may be not only irregular in their time-relation to each other, but unequal in volume. This inequality in volume

is often found in association with the irregularity in rhythm. When the irregularities of the pulse have been studied as recorded on a pulse-tracing, it becomes easier to recognize them on palpation of the pulse (*see* p. 162).

3. The **size** or calibre is determined by emptying the vessel of blood and stopping the pulse, by firm pressure, and then gauging the size of the vessel in the flattened state. When it is contracted the calibre is small; when the muscular coat is fully relaxed it is distinctly larger.

4. To discover the condition of the **walls**, flatten the vessel and cause the skin of the patient's wrist to slip up and down along the length of it. In health the vessel-wall can often be felt, especially in those engaged in strenuous occupations. In disease one may feel thickening, calcification, and tortuosity. It is a good custom to feel both radial arteries and both brachial arteries for thickening.

5. The **volume** of the pulse is the amplitude of movement of the vessel-wall during the passage of the pulse-wave. Apply just sufficient pressure to flatten the vessel between the beats; with each pulse-wave the amplitude will then be perceived—"observing the expansion," as it is sometimes called. As the elastic stretching of the vessel is never great, the movement chiefly depends on the resumption by the flattened artery of its cylindrical shape, and the amount of such movement is therefore greater the more dilated the vessel is. The force of the heart's action also exercises a certain influence on the amplitude, but to a much less degree than the relaxation or contraction of the wall of the artery. In other words, the size or calibre of the artery (*see* 3, above) at the time largely determines the volume of the pulse.

6. The next point is the determination of the **systolic blood-pressure or force,** which, of course, occurs during the beat. In this case three fingers must be placed on the artery, so that it may be compressed both above and below the point where the pulse is being felt. Place the finger next the wrist firmly on the vessel to prevent any pulse from the ulnar artery reaching the middle finger through the palmar arch; let the middle finger rest on the vessel with such pressure as will render the pulse most distinct, and then gradually compress the artery above this point with the remaining finger, noting the pressure employed when the pulse ceases to be felt by the middle finger. This pressure, being just sufficient to prevent the blood from lifting the finger during the beat, corresponds to the maximum blood-pressure.

Even by careful practice and comparison with the readings obtained by a sphygmomanometer (*see* Section VII) the student will seldom be able to determine whether the pressure is normal, excessive, or diminished.

7. It is still more difficult to estimate the **diastolic blood-pressure or tension.** A method which might be tried is to feel the pulse first with light, then with moderate, and finally with considerable pressure of the fingers on the artery. A pulse of low tension (i.e. with a low diastolic pressure) is best felt in the first case, for the light pressure is sufficient to flatten the vessel between the beats, whilst it allows the artery to resume its cylindrical shape without much resistance during the beat, and is thus favourable to the development of the greatest possible amplitude of movement; whilst on the contrary, where the tension is high, considerable pressure is required to flatten the vessel between the

beats. But one obtains the greatest amplitude of movement precisely when the vessel is thus flattened, and so in a high-tension pulse the more firmly one presses, the more forcible does the pulse appear to grow. A normal pulse is best developed when moderate pressure is applied.

8. Study the general character or **form of the individual pulse-wave.** This is divided into three periods: first, the period during which the blood-pressure is rising; second, the period at which the blood-pressure continues near its maximum; and last, the period during which the blood-pressure is falling.

The rise or impact may be abrupt, or it may be slow. When abrupt, the pulse is of low tension ; when slow, it is of high tension, or aortic stenosis is present.

In the second period, while the pressure remains near the maximum, observe whether the pressure is well sustained or whether, on the contrary, it has no sooner attained its highest value than it begins to fall off again rapidly, i.e. is ill-sustained.

In the third period we notice whether the fall of pressure is swift or gradual. The fall is naturally interrupted by the *dicrotic wave*, as shown on a sphygmogram, and this may become quite perceptible to the finger as a distinct impact following the primary stroke of the pulse. It is best felt in a pulse of low tension and when the finger is very lightly applied to the vessel.

The **typical pulse** of a healthy adult man should be described in some such terms as the following. The rate is 70 per minute. The beats are regular in rhythm and equal in volume. The vessel is of medium size; it is not tortuous, its walls are not thickened, and between the beats it is just

possible to feel it. The volume of the pulse is moderate. The passage of the pulse-wave can be arrested by moderately firm pressure, and the beat is best felt when a medium degree of pressure is applied. With regard to the general character of the pulse-wave, the impact is neither abrupt nor very gradual; it is fairly well sustained, and its fall is gradual but not very tardy. Dicrotism is not noticeable.

VII. THE BLOOD-PRESSURE

When a preliminary estimation of the blood-pressure has been attempted by palpation of the pulse (Section VI, 6, 7), it may be controlled and extended by the more exact instrumental method, the use of the sphygmomanometer. This provides us not merely with the information that the pressure is high, moderate, or low, but with figures which represent the actual pressure in the arteries.

The best form of apparatus for clinical use is one of the modifications of **Riva-Rocci's sphygmomanometer.** This consists of a mercurial manometer, an armlet for compressing the upper arm, and a rubber bulb or metal pump for inflating the armlet with air. The manometer is graduated in millimetres of mercury, and in the best models the mercury cannot be spilled, though it can be removed for cleaning. The armlet is a long flat rubber bag enclosed and reinforced on the outer side by an unyielding sheath. It should be about 12 cm. broad, as narrow armlets introduce serious errors, especially in the case of high-tension pulses. The three parts of the apparatus are connected by means of stout rubber tubing. In the circuit is a valve permitting escape of air at the will of the observer, so that the pressure in the air-system may be very gradually reduced.

Method of use.—The patient should be sitting or lying at ease. The manometer is placed so as to be at the same level as the observer's eye. Fix the empty armlet closely to the upper arm, which should be thoroughly relaxed. The estimation may now be made by one of two methods, or by one and then the other.

(i) **Palpatory method,** for systolic pressure. Steadily inflate the armlet until the pulse is no longer felt at the wrist. By means of the valve, allow the air to escape very gradually so that the pressure falls, watching the manometer but concentrating attention upon the return of the pulse at the wrist. Immediately the pulse returns, read the scale; this reading is the *systolic* blood-pressure in millimetres of mercury.

(ii) **Auditory method** for systolic and diastolic pressures. Raise the pressure by pumping to 30 mm. or so above the systolic blood-pressure as determined by the palpation method. Instead of feeling the pulse, place the chest-piece of a binaural stethoscope on the arm immediately below the armlet and auscultate the brachial artery. Open the valve and so reduce the compression gradually until the faint tapping sounds produced by successive pulse-waves are first heard, and immediately take a reading. This is the *systolic* blood-pressure. Continue to listen while the mercury falls; the sounds persist, often changing in character from a tap to a loud knock, then to a murmur. Ignore these changes and still allow the air to escape until the sounds suddenly become soft and almost inaudible. Take a reading at this point also; it is the *diastolic* pressure.

The figures for systolic pressure as obtained by the palpatory and the auditory method are much the same; by the latter method the reading is usually a trifle higher (5–10 mm. Hg.). The auditory method may be recommended for general use because by the same technique both systolic and diastolic pressures are obtained.

Several types of sphygmomanometer are now obtainable with an aneroid manometer, bearing a dial, in place of a column of mercury. These have

the advantage of compactness and convenience for general practice, but the mechanism is hidden and liable to give false readings, especially with the lapse of time. It is therefore important to connect it with a mercurial manometer occasionally to make sure that it is accurate.

Precautions.—Arterial pressure shows temporary variations with change of posture, after meals, on exertion, and notably a rise on excitement. Hence it should be observed only after the patient has been reassured and when he is quietly resting, free from excitement and with the arm relaxed. In nervous patients the first reading is often too high and should be rejected; a second reading will more closely represent the true pressure. The pulse-rate at the time should be noted, for blood-pressure varies to some extent with the rate of the heart. It is essential to work as quickly as is compatible with accuracy, for compression of a limb itself induces a rise in blood-pressure. To reduce this source of error when successive estimations are to be made, the air-pressure in the armlet should always be allowed to fall to zero as soon as each reading has been taken.

Occasionally the sounds heard on auscultatory estimation of blood-pressure disappear at a point below 200 mm. for a period and then reappear, finally disappearing at the point of diastolic pressure. Thus the sounds may first appear when the mercury falls to 210 (systolic pressure), disappear from 180 to 160 (silent gap), reappear and finally disappear at 120 (diastolic pressure). This phenomenon of a **silent gap** is found in certain patients with hypertension; its significance is unknown, but its occurrence makes it important that the armlet pressure should always be well raised at the beginning of an estimation of blood-pressure, as advised above.

It has been shown by experiment that pressures obtained by compression of a limb, as here described, closely correspond with the direct readings obtained in animals by a manometer connected directly with a cannula inserted in an artery. In man, the error introduced by rigidity of the arterial wall does not in ordinary cases exceed 10 mm., though with very hard or contracted arteries the error may be considerable. If there is gross œdema of the arm, or if the muscles are held contracted as in tetanus or in fright, the readings are so inaccurate as to be valueless.

Occasionally it becomes necessary to compare the systolic blood-pressure in the arm with that in the leg. The patient lies face downwards and the cuff is applied above the knee and auscultation carried out over the popliteal artery. In health it is found that the systolic pressure in arm and leg is approximately the same provided that the subject is horizontal. Under the same conditions, patients with aortic incompetence may show a systolic pressure in the leg far higher than that in the arm, while in coarctation of the aorta the systolic blood-pressure is low in the leg.

Normal blood-pressure.—The average systolic pressure in healthy adults is 100–140 mm. Hg, the average diastolic pressure, 60–90 mm. In children it approximates to the lower figure in each case, and in the elderly it reaches or even exceeds the higher figure. The difference between the systolic and the diastolic pressures—the pulse-pressure—is 30–60 mm.

Abnormal blood-pressure.—An abnormally high pressure (*hypertension*) is found in chronic nephritis and in a large group of patients in whom hypertension may be the leading sign (hyperpiesia). Too great importance need not be attached to a single

reading which does not greatly exceed the normal limits. On the other hand, a high systolic pressure such as 200, especially if persistent and accompanied by a high diastolic pressure such as 130, has always great significance.

An abnormally low pressure (*hypotension*) is not nearly so commonly encountered. It may be found temporarily, as in hæmorrhage or shock, or persistently, as in Addison's disease. It must be remembered, however, that a low pressure is natural to some persons and is not necessarily a sign of disease.

VIII. CARDIOGRAPHIC METHODS

To analyse the form of the individual pulse-wave, and, what is now recognized as more important, to observe the time-relations or rhythm of successive pulse-waves, we make use of an instrument which will give a graphic record of the pulse (sphygmogram). For this purpose it has long been the custom to use a **sphygmograph**, such as that of Marey or of Dudgeon. A sphygmograph is a small instrument, fixed upon the wrist, with which the pulse-wave is recorded by a style, deriving its movement, mechanically amplified, from a metal button lying on the radial artery. The style scratches the record on a strip of smoked paper which is passed through by a clockwork mechanism.

Since the introduction of the **polygraph** into clinical medicine, however, this instrument has superseded the sphygmograph. The fundamental improvement in technique lies in the provision of *two* writing tambours, one for the arterial pulse giving a sphygmogram, and another for the venous pulse giving a jugular tracing or phlebogram. It is by a comparison of the arterial with the venous tracing,

taken simultaneously, that the advance in our knowledge of cardiac rhythm was first made possible. For the first time the auricular contraction in health and disease came into the field of clinical observation. Other improvements were the introduction of a time-marker, the use of a special pen writing in ink upon a long roll of paper, and the convenience of air-transmission.

A. The Polygraph

Mackenzie's ink polygraph consists of a metal case containing two independent pieces of clockwork, one of which actuates a time-marker indicating fifths of a second, the other drives a long roll of paper beneath the writing pens at a rate which can be controlled by the observer. The movements of the radial pulse are transmitted to the polygraph through a metal button attached to a spring which is applied to the wrist by a small splint. The excursions of the button are directly transmitted to a radial tambour which rests on the wrist-splint, and thence by means of rubber tubing to a recording tambour fixed to the polygraph, and provided with a lever and writing pen. The venous pulse is received by a small shallow cup or receiver and conducted by tubing to a similar recording tambour. Thus are inscribed simultaneously on the strip of paper a time record and two tracings, one of the arterial and the other of the venous pulse.

Method of use.—The patient should lie on his back with the head on a single pillow. The polygraph is assembled on a firm table placed on the right, close to the patient's shoulder. It should be made ready for immediate use before the patient is connected. Sit down facing the apparatus and apply the wrist-splint firmly with the button exactly on the radial artery. This is the chief difficulty for beginners, and the splint should be applied and re-applied if necessary until it is seen that the button moves with the pulse. The hand should lie dorsiflexed and with some ulnar deviation over a small

cushion or rolled towel. Now apply the wrist tambour, already connected by tubing with the recording tambour next the supporting rod, and the pen should move with the pulse. Adjust it to the paper, which is then allowed to run through so that a radial tracing or sphygmogram is obtained. Next place the neck-receiver over the jugular bulb in the neck close to the sterno-clavicular joint. The pen of the second tambour will now move with the jugular pulse. Apply both pens to the paper, let the paper pass through, and thus obtain a polygram. When

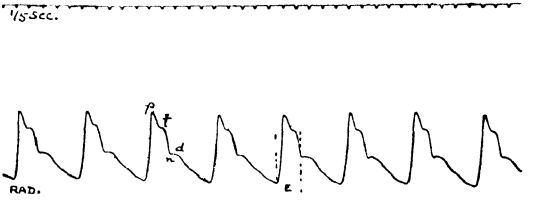


Fig. 32.—Normal sphygmogram.
p, percussion waves (partly instrumental), *t*, tidal, or main pulse-wave, *n*, aortic notch, *d*, dicrotic wave, E, the period of ventricular systole (aortic valves open).

a satisfactory record has been gained, stop the mechanism, and move both pens lightly up and down so that ordinates are drawn.

If required, the neck-receiver may be applied to other pulsations, such as the apex of the heart, a pulsating liver, or an aneurysm, or it may be replaced by a small rubber bag inserted under the patient's clothing to record respiration, e.g. Cheyne-Stokes respiration.

A modified form of the Mackenzie polygraph has been introduced by Sir Thomas Lewis (*Mackenzie-Lewis polygraph*). It can be brought more quickly into use and the radial splint and tambour is replaced by a glycerin pelotte which is easy to apply to the brachial or to the radial artery. It is apt not to give such a clear tracing as the original form.

Another type of polygraph is in use on the Continent, the *Jacquet polygraph.* It is rather heavier and more costly, but it gives excellent records. Some types carry three or more recording tambours, but the control of more than two presents great difficulty.

THE SPHYGMOGRAM

In a pulse-tracing, rise of blood-pressure will be represented by an upstroke, and fall by a downstroke. Bearing this in mind, the student will readily understand the main outlines of a normal radial sphygmogram. (Fig. 32.) The pressure rises fairly rapidly; therefore the upstroke, when the paper is driven forward at the usual speed, is nearly but not quite perpendicular. The percussion wave is quickly followed by what is known as the tidal wave; these are not separately distinguishable by the finger in health, and the second wave (the tidal) represents the natural summit rather than the first (the percussion wave), which is largely due to instrumental fling. Thereafter the pressure begins to fall off, but at the moment when the aortic valves close the decrease of pressure is arrested, and a positive (dicrotic) wave is propagated into the vessels; this event is recorded as a small break in the downstroke of the tracing. The foot of the notch immediately *before* the dicrotic upstroke or wave indicates the point of time when the aortic valves close (aortic notch). After the dicrotic wave the line again curves downwards until a new upstroke marks the arrival of the next pulse-wave. Ordinarily the blood-pressure requires much longer to fall than to rise, hence the downstroke is much less vertical than the upstroke.

The upstroke is longer and steeper than usual when the ventricle discharges a larger volume of

blood than normal into the arteries, and also when the arterioles are dilated (low tension).

The upstroke is shorter and less steep when the heart acts feebly or when there is stenosis at the

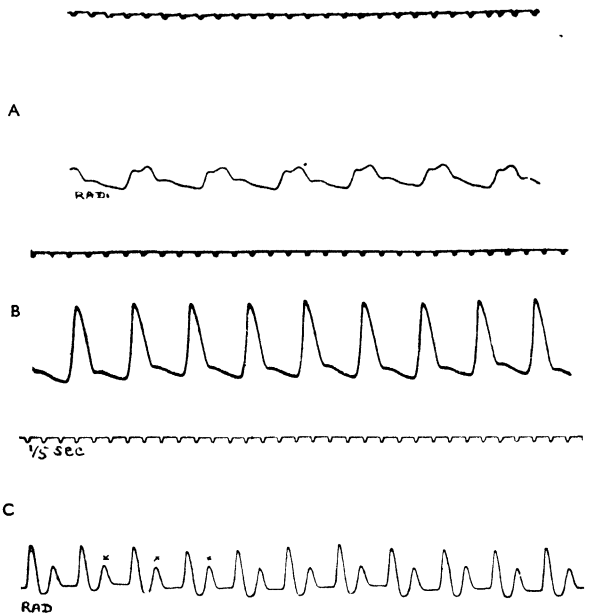


Fig. 33.—Sphygmograms showing typical form of pulse-wave in (A) aortic stenosis, (B) aortic incompetence, and (C) dicrotism.

aortic valve, so that less blood than usual is delivered in a given time, and also when the blood-pressure is high and undue opposition is thereby offered to the outflow from the heart.

If the diastolic pressure is low, the dicrotic wave is well marked; if it is high, the wave is small.

The following conditions often present characteristic tracings:—

(*a*) **Aortic stenosis.**—Small amplitude, sloping upstroke (pulsus tardus); tidal wave well developed and often higher than the percussion wave (anacrotic pulse) (Fig. 33, A).

(*b*) **Aortic incompetence.**—Great amplitude, abrupt upstroke, rapid fall, little or no dicrotic wave. This is known as the water-hammer, collapsing, or Corrigan pulse (Fig. 33, B).

(*c*) **Mitral stenosis.**—Small amplitude if the lesion is advanced. Complete irregularity (auricular fibrillation) is often present.

(*d*) **Aneurysm** of the arch of the aorta occasionally leads to a diminution in amplitude in the pulse on one side, usually the right, according to the part of the aorta implicated. Other signs of aneurysm would be present, and it must be remembered that in health there may be a natural difference between the pulse on one side and on the other.

Special types of pulse:

(i) **Pulsus frequens.**—The pulse is rapid, due to very various cardiac conditions, or to extracardiac causes such as an infection.

(ii) **Pulsus rarus.**—The pulse is infrequent or "slow." This may be a natural occurrence in certain people, and infrequency is sometimes noted during convalescence. If extreme (e.g. 30–40 a minute), it is probably due to heart-block.

(iii) **Pulsus celer.**—The upstroke is abrupt, and the down-stroke sudden, i.e. ill-sustained.

(iv) **Pulsus tardus.**—The upstroke is gradual, the summit is well-sustained, even plateau-like, and the downstroke is gradual.

(v) **Pulsus anacroticus.**—The tidal wave is higher than the percussion wave. It may be seen where the blood-pressure is high and, like the pulsus tardus, in aortic stenosis (Fig. 33, A).

(vi) **Pulsus dicroticus.**—The dicrotic wave is exaggerated (Fig. 33, C). Occasionally, as in high fever, the dicrotic wave may be distinctly felt after the main pulse-wave.

(vii) **Pulsus paradoxus.**—The pulse becomes smaller or even disappears at the end of inspiration when the patient breathes deeply. This occurs most commonly in disease of the pericardium.

Pulsus alternans has a special significance, and is described on p. 178.

THE VENOUS PULSE (PHLEBOGRAM)

By means of the polygraph we may obtain, simultaneously with the sphygmogram, a phlebogram, i.e. a record of the pulsations in the jugular vein, or the venous pulse.

Pulsation in the jugular vein is physiological, and a jugular tracing is obtainable from most people in health as well as in disease. Visibility of the pulsation in health varies in different individuals; it may not be seen at all when the neck is fat or when the person is standing.

Venous pulsation at the root of the neck is best observed when the patient is lying down with the head only slightly, if at all, raised on a pillow. In some cases, as in aortic incompetence, the pulsation in the neck seems exclusively arterial (carotid). Given the necessary conditions, of which the chief is

an interference with return of the blood to the right auricle, the jugular veins become distended and prominent. The venous pulsation then becomes obvious on inspection, and occasionally it is practicable to make direct observations on its character. In complete heart-block, for instance, waves may be seen while the ventricle is in diastole, from which it may be inferred that they are auricular waves, dissociated from the far less frequent ventricular waves. Where relative tricuspid incompetence occurs,

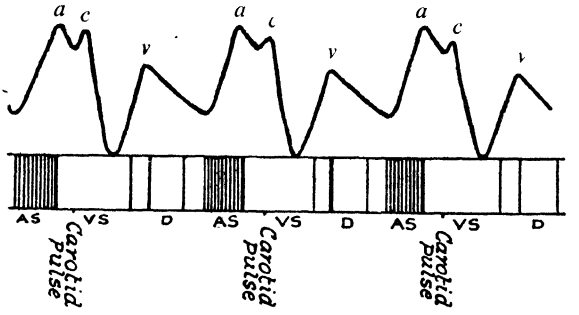


Fig. 34.—Diagram of venous pulse.
A S, auricular systole, V S, ventricular systole, D, diastole (ventricular)

a frequent event in advanced stages of heart disease, the distended jugular vein may show pulsation of great amplitude.

When the neck-receiver of a polygraph is lightly applied to the root of the neck (*see* p. 161), we may register the pulsation or changes of pressure occurring in the jugular vein, and so obtain exact information in permanent form of the action of the right auricle. If we analyse the venous tracing obtained (Fig. 34), it will be seen that there are three main waves, *a*, *c*, and *v*. The first wave, *a*, is due to auricular systole, in part directly from an impulse sent back

to the vein, and in part from swelling of the vein while the contracting auricle holds back the blood in the neck. The second wave, *c*, is due to the carotid pulse, and therefore indicates the beginning of ventricular systole. It is true that there is a systolic wave in the vein as well as in the artery at this moment, and both vessels may contribute to the wave *c*, but for practical purposes we may accept *c* as a carotid wave. Immediately after the carotid wave there is a fall in pressure, as shown

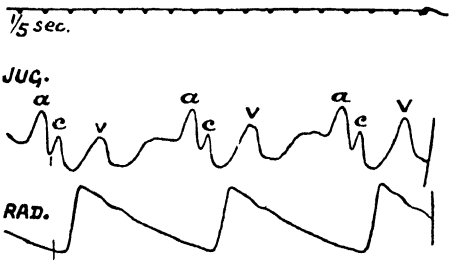


Fig. 35.—Normal polygram, showing three heart-cycles.
a, auricular systole, *c*, carotid wave, the beginning of ventricular systole; *v*, venous-stasis wave at the end of ventricular contraction. The radial beat occurs 1-10th sec. after the carotid wave.

by a depression, which is soon followed by the third wave, *v*. The *v* or "venous-stasis" wave is due to rising pressure and stasis in the jugular vein while ventricular systole still continues, so preventing the flow of venous blood from the neck into its cavity through the auricle. Immediately ventricular systole ends and the ventricle relaxes, the blood from the vein rushes into it, hence the *v* wave falls and a final depression in the curve is seen. To repeat, the jugulo-carotid or venous pulse corresponding to a single heart-beat consists of an auricular wave, *a*, a carotid (or ventricular) wave,

c, and a "venous-stasis" wave, *v*. The *v* wave is bound up with a *c*, and follows it in a fixed relation. The *c-v* period is the period of ventricular systole. Normally *a* precedes *c* by not more than $\frac{1}{5}$ sec., for this represents the time taken for an impulse from the auricle to reach and affect the ventricle (*a-c* interval).

The **interpretation of a polygram** is seldom difficult. (Fig. 35.) A pair of dividers is used for measuring. Much can be learned from inspection of the radial curve alone. The rate is counted, and the regularity or irregularity of consecutive beats is determined, taking as a standard the dominant rhythm or succession of normal regular beats if such is anywhere present. Then place one foot of the dividers on the ordinate at the end of the radial curve, and the other $\frac{1}{10}$ sec. in front of a chosen radial beat. Transfer the dividers, thus separated, to the jugular curve with one foot on its ordinate; the other foot will mark the beginning of a wave which is the *c* wave. The carotid precedes each radial wave by $\frac{1}{10}$ sec., which is therefore allowed. When several *c* waves have been marked, it will be found that in a normal record each is preceded by an *a* wave, and succeeded at a fixed distance by its *v* wave.

The commonest alterations in the venous pulse, as the result of disease, are—

(i) Absence of the auricular wave (*a*)—characteristic of auricular fibrillation and associated with complete irregularity of the *c* wave and of the corresponding radial pulse.

(ii) Prolongation of the *a-c* interval, i.e. heart-block. If the block is more severe, *a* waves will occur without a consecutive carotid wave *c* (dropped beat); if the block is complete, *a* and *c* waves are dissociated entirely.

B. The Electrocardiograph

While detailed instructions for using this instrument are outside the scope of this book, it is essential for the comprehension of disordered heart action that the general principles of the method should be grasped and the normal electrocardiogram understood.

When part of a muscle contracts, the contraction is associated with a difference in potential between the part in contraction and the part not in contraction. If the two parts are connected it may be shown that a current is produced. This is true when the heart contracts, and if for example the right arm and the left leg are put into circuit with the electrocardiograph, this will indicate the current produced by a difference of potential between the basal and the apical portions of the heart. Broadly speaking, an electrical current passes when auricular systole occurs, and another during ventricular systole. We have therefore at our disposal not only a mechanical (or kinetic) means of recording auricular as well as ventricular systole in the polygraph, but an even more exact and informative electrical method in the electrocardiograph.

The essential part of one type of instrument is the Einthoven string galvanometer. The tiny current from the heart, amounting to a millivolt or so, is led through a fine conducting string of silvered glass which hangs vertically between the poles of an electro-magnet. During the passage of the current the fibre moves in response, changing in direction and amount with every change in the direction and strength of the current. The movements of the fibre are rendered visible by enlargement of its shadow through a microscope and by means of a strong lamp. The moving shadow is photographed upon a falling plate or a film in a special camera. The photograph displays and records the movement, and also bears the shadows cast by a time-marker usually indicating fifths of a second. Owing to the small inertia of the almost microscopic fibre, this instrument is practically free from instrumental error. It is customary to

receive the current from the patient's limbs by non-polarizable electrodes. The right and left arms and the left leg are so connected. The three principal derivations or leads are then obtainable by means of a special switch. For Lead I the current is taken from the right arm and left arm; for Lead II, the most generally useful if one lead only is required, the right arm and left leg; and for Lead III, the left arm and the left leg. Though Lead II usually gives direct information as to rhythm and the proper sequence of auricular and ventricular contraction, a comparison of the three leads gives more detail, especially as to the comparative preponderance of the right and the left ventricle in contraction. Lead IV constitutes a chest lead in which the proximal electrode is placed upon the apex-beat. When this is paired with the right arm it is called Lead IVR, and when it is paired with the left leg it is called Lead IVF.

A schematic outline of the normal electrocardiogram is given in Fig. 20, an actual tracing in Fig. 36, and examples of the commonest abnormalities are represented in Figs. 38, 39, 41, 42.

THE ELECTROCARDIOGRAM

The several deflections produced by changes in electrical potential which accompany a normal heartbeat are scarcely comparable with the waves of changing pressure in the auricle as recorded in the venous pulse. Yet in a simple way we may look upon the *P* deflection of the electrocardiogram as representing auricular systole and the *a* wave, and the *R-T* deflections as representing the period of ventricular systole and the *c-v* waves. This relation will be appreciated by looking carefully at Fig. 36.

There are wide variations in the form of the normal electrocardiogram, indeed each individual may be said to have his own particular form. It is imperative, therefore, to know the normal variations before assuming that an unusual record indicates disease. Abnormal rhythms do, however, alter the

electrocardiogram in unmistakable fashion. Thus, in auricular fibrillation, *P* waves are absent, and in their place may often be seen finer and variable deflections, "f" waves, produced when the auricle is fibrillating and not beating. At the same time the

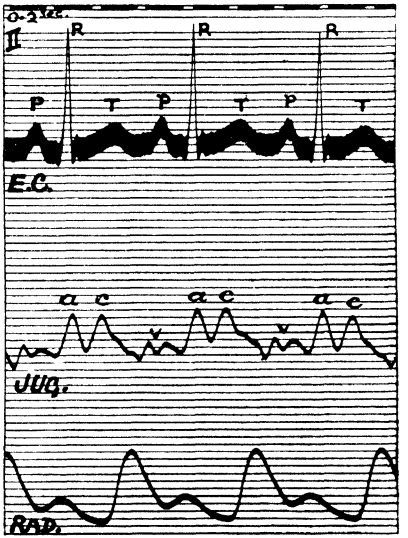


Fig. 36.—Normal electrocardiogram, with simultaneous jugular and radial curves.

P and *a* represent auricular systole, *R*, *c*, and the radial beat indicate the beginning of ventricular systole.

R waves (onset of ventricular systole) are irregularly spaced. Again, in complete heart-block, it is easy to recognize the *P* waves occurring quite independently of the *R* waves, and much more frequently (complete dissociation).

Information is often obtainable from a patient's electrocardiogram even when the rate and the

rhythm are normal. A comparison of the three leads may indicate relative hypertrophy of the right or of the left ventricle. Inversion or widening of certain deflections may give definite evidence of myocardial changes as in cases of cardiac infarction (coronary thrombosis). An excellent example of cardiac localization is the curve characteristic of a lesion of one branch of the auriculo-ventricular bundle of His.

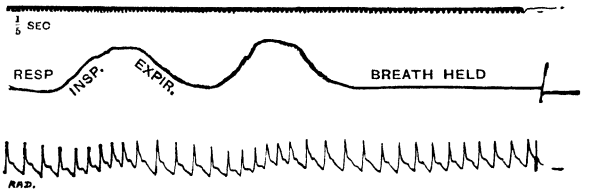


Fig. 37.—Sinus arrhythmia.

Respiratory and radial polygram showing the quickening of the pulse during inspiration, and the slowing during expiration. When the breath is held, this physiological arrhythmia disappears.

Common Disorders of Rate and Rhythm

By graphic methods the common disorders of the rate and rhythm of the heart have now been clearly differentiated. As they can all be produced experimentally in animals, physiology and clinical research have together been able to effect a remarkable advance in this field. All the varieties commonly encountered in clinical medicine are included in the following brief summary.

(1) **Sinus arrhythmia** (Fig. 37) is due to irregular stimulus production at the sino-auricular node, consequent upon variations in vagal control of that node. The variation is most often associated with respiration, the beats being frequent during inspiration and infrequent during expiration. When the

breath is held the irregularity ceases; with deep breathing the irregularity is accentuated. It usually disappears with any form of tachycardia, including the simple tachycardia induced by test exercise. It is the commonest irregularity of early life, and is clinically important only because it may be mistaken for some serious form.

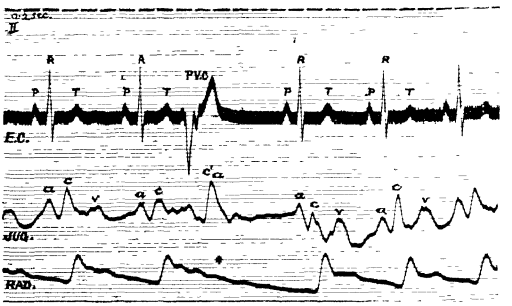


Fig. 38.—Premature ventricular contraction, or extrasystole. Simultaneous electrocardiographic, jugular, and radial tracings.

The rhythm is normal until the premature ventricular contraction (*P.V.C.*) which in this instance produces no radial beat, but only an intermission at the wrist.

(2) **Premature systoles,** or extra systoles, arise in some part of the heart-muscle which is not a normal site for a stimulus to arise in. With a premature *ventricular* contraction, the radial tracing shows either a small premature beat followed by a long pause, or the abnormal contraction fails to produce a radial beat and there is an intermission only. In either case the space between the preceding and the succeeding normal beats is generally equal to two normal pulse-periods, for the auricular rhythm has not been disturbed. The jugular tracing shows a premature carotid wave, *c'*, corresponding with the small radial beat if one is produced.

The electrocardiogram shows a large diphasic deflection indicating an abnormal impulse traversing the ventricular muscle. (Fig. 38.)

Less common are premature *auricular* contractions, which arise in the wall of the auricle away from the sino-auricular node. A small premature radial beat is nearly always produced, but the pause is not so long as that after a premature ventricular contraction, so that the space between the preceding and succeeding normal beats is *less* than two normal pulse-periods. On the jugular curve is seen a premature auricular wave, followed by the carotid wave of the premature beat. On the electrocardiogram, similarly, there is a premature *P* of abnormal form, commonly inverted, followed by a ventricular complex of normal outline.

On auscultation, a premature beat is recognized as a feeble *lubb-dupp* or *lubb* occurring quickly after a normal beat and followed by a pause. This method is only applicable when the heart rhythm is otherwise regular.

If a premature beat occurs after each normal beat, and it is felt at the wrist, the pulse is said to be coupled (a form of *pulsus bigeminus*). Digitalis coupling is of this nature.

Premature beats are not a sign of disease, though they may occur along with other signs in disease of the heart.

(3) **Heart-block.**—In the slight degrees of heart-block there is merely a delay in the transmission of the normal impulse from the auricle to the ventricle through the auriculo-ventricular (A-V) bundle. The pulse is unaffected, but the jugular curve shows a prolonged *a-c* interval (over $\frac{1}{5}$ sec.), and the electrocardiogram a prolonged *P-R* interval. A further stage of block results in a

dropped beat at the wrist because one of the auricular impulses has failed to reach the ventricle. If auscultation is practised, a dead silence is noted during this pause (compare the sounds heard when an intermission of the pulse is the result of an extrasystole). If every second ventricular beat is dropped, the pulse-rate will be halved (2:1 block); if every third beat (3 : 2 block), each pair of radial beats will be followed by a pause (another form of pulsus bigeminus).

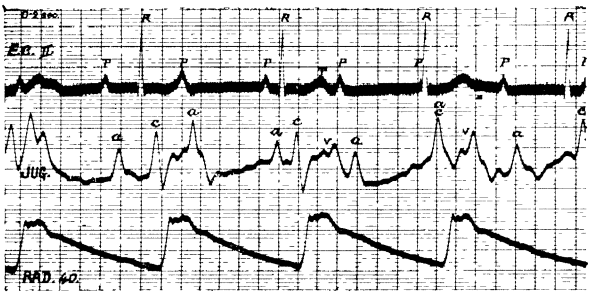


Fig. 39.—Complete heart-block. Triple record.

The auricular contractions, represented by *P* on the electrocardiogram and *a* on the jugular tracing, are completely dissociated from the ventricular contractions, which are indicated by *R* (or *R-T*) on the electrocardiogram, *c* on the jugular tracing, and by the radial beats at the rate of 40 a minute.

Complete heart-block (Fig. 39) results in complete dissociation of the auricular from the ventricular beats; no impulses reach the ventricle, which beats at its own low rate of 30–40 a minute. On the jugular curve the *a* waves occur regularly at their normal rate (e.g. 70) and the *c* waves at the low ventricular rate. Similarly, on the electrocardiogram *P* deflections are seen at the normal rate, and *R-T* deflections at a rate of 30-40 independently.

Heart-block is a sign of myocardial disease, though not an index of its extent or severity. In a certain proportion of cases of heart-block, standstill of the ventricle determines the occurrence of Stokes-Adams attacks.

(4) **Paroxysmal tachycardia** (Fig. 40).—When an abnormal centre of stimulus-production

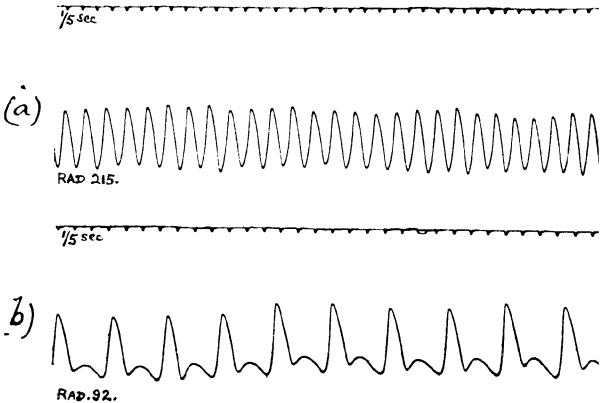


Fig. 40.—Paroxysmal tachycardia.

Radial tracings taken (*a*) during a paroxysm at a rate of 215 beats a minute, and (*b*) immediately after the attack had stopped, the rate being now 92 a minute.

controls the heart, it evokes a paroxysm of rapid contractions. These may be looked upon as a long run of premature beats and they may arise in the auricle, the A-V node, or in the ventricle. The rate is usually 120-220 a minute, and, unlike ordinary simple tachycardia, it is fixed so that it does not vary with posture or exertion. The attack begins and ends abruptly, lasting a few minutes, days, or even weeks. The electrocardiogram always shows deflections of an abnormal contour, depending upon the site of origin of the impulses.

(5) **Auricular flutter** (Fig. 41).—Auricular flutter is a special form of tachycardia in which the auricular rate is about 300 a minute. The ventricle usually responds to every second beat, so that the pulse is regular and about 150 to the minute. Less often, the ventricle responds irregularly and the pulse is rapid and irregular. By blocking more auricular

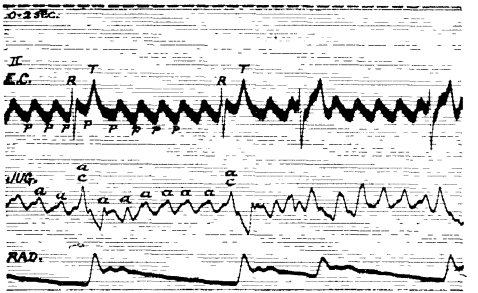


Fig. 41.—Auricular flutter. Triple record of electrocardiogram with jugular and radial tracings.

The auricular contractions (*P* and *a*) are regular at 240 a minute. The ventricular contractions (*R* and *c* and radial beats) are irregular and slow, the result of varying block induced by digitalis.

impulses, as by means of digitalis, the form of the auricular tachycardia on the electrocardiogram becomes evident (*see* Fig. 41).

Auricular flutter may occur in paroxysms, but more often it lasts for months or years, apart from treatment; clinically it has more in common with auricular fibrillation than with paroxysmal tachycardia.

(6) **Auricular fibrillation** (Fig. 42).—This condition is characterized by a completely irregular pulse, one in which a succession of normal beats, evenly spaced, is nowhere to be found. The auricle is fibrillating and does not contract as a whole;

the ventricle beats in a rapid, irregular fashion. The *a* wave is of course absent from the jugular tracing, and the *P* wave absent from the electrocardiogram. Instead, there may be seen fine fibrillary waves (*f*, *f*). The *c* waves and the *R-T* waves are irregularly spaced, corresponding with the ventricle and the pulse.

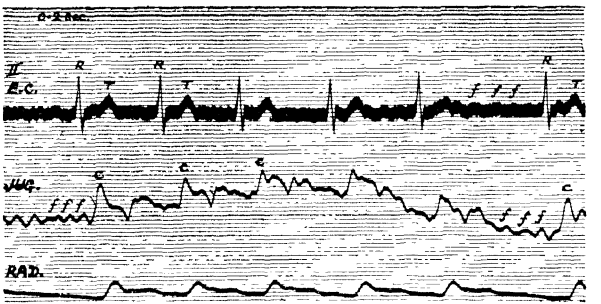


Fig. 42.—Auricular fibrillation. Triple record of electrocardiogram with jugular and radial tracings.

The *P* and *a* waves of auricular contraction are absent, and small fibrillation waves (*f*) are visible. The ventricular contractions (*R*, *c*, and radial beats) are irregular, and at a slow rate (60-70 a minute) under the influence of digitalis

Auricular fibrillation may occur in paroxysms, but it is usually a permanent condition and an indication of myocardial disease. If it supervenes in a patient with mitral stenosis, the murmur can no longer be presystolic in the sense of being due to auricular systole. It now should be termed a diastolic murmur, equally characteristic of mitral stenosis, and not to be mistaken for an aortic diastolic murmur.

(7) **Pulsus alternans** (Fig. 43).—When the ventricle beats first strongly, then weakly, in successive beats of normal rhythm, alternation is

present. In the radial tracing are seen alternate large and small beats, which are, however, *equidistant*. The polygram and the electrocardiogram are of normal form, though the latter does sometimes show alternation of the *R* summits.

When this condition is discovered, provided the heart-rate is moderate and no abnormal rhythm is present, it may be inferred that the heart-muscle is damaged.

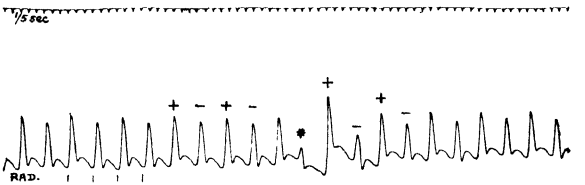


Fig. 43.—Pulsus alternans. Radial tracing which shows equidistant beats, alternately large and small. The alternation is accentuated after the single premature contraction.

IX. X-RAY EXAMINATION OF THE HEART AND AORTA

With the rapid extension of the use of X-rays in practical medicine, it is increasingly recognized that they provide a valuable additional method of investigating diseases of the heart and aorta. The diagnosis of thoracic aneurysm should always be confirmed by radioscopy, which may, moreover, reveal a deep-lying aneurysm unsuspected on ordinary clinical examination. By palpation of the apex-beat and by percussion much may be learned of the size of the heart. Radiology permits greater accuracy, and gives greater detail of the shape and size of the heart, especially of the base and its vascular stem, where the method of percussion is least

applicable. Radioscopy is taking its place as a modern development of the classical method of inspection, for it is nothing less than inspection of organs otherwise hidden to the eye.

Methods.—(1) **Radioscopy,** or screening, is the best single method for clinical purposes, and any ordinary screening stand may be used. It is convenient to fix a small strip of lead on the chest with adhesive plaster immediately above the left nipple or, in women, in the midclavicular line. The patient stands facing the observer, care being taken that the shoulders are parallel with the screen. The relation of the apex of the heart to the lead strip can be decided at once. In addition, the chief points to note are (*a*) the position of the heart in the chest, (*b*) the shape of the heart, (*c*) its size, and (*d*) the pulsation of the heart and aorta, and any abnormal pulsation.

Further information may be gained, especially as to the size and shape of the aorta, by radioscopy with the patient half-turned to the left (1st or right oblique position) and half-turned to the right (2nd or left oblique position).

A radiogram or X-ray film may be taken subsequently if a permanent record is desired.

(2) **Orthodiagraphy.**—Ordinary radioscopy has the disadvantage that, owing to the nearness of the tube to the patient, the image seen on the screen (or photographed) is enlarged, and even distorted, owing to divergence of the X-rays as they traverse the chest. To overcome this difficulty, a special form of screening apparatus, the orthodiagraph, has been devised for recording the exact outline of the heart by means of *parallel* X-rays. Such are obtained as a narrow beam from the X-ray tube,

which, though behind the patient, is so fixed to a small fluorescent screen in front that tube and screen always move together. A lead spot in the centre of the screen, illuminated by the beam, is moved until it is in line with the edge of the heart shadow. When this is seen to occur, a corresponding mark is made by a mechanical device, also in alinement, on a large sheet of paper fixed behind the patient. By moving the beam (and so the lead spot and the marker) methodically round the limits of the heart, a silhouette of this organ is obtained—an *ortho-diagram.* Similarly, the outline of the thoracic wall is also dotted out and, by suitable accessories, such landmarks as the nipple line and the sternum.

This is the most exact method we possess for determining the size of the heart in cases where this has great importance.

(3) **Telerontgenography,** or radiography with the X-ray tube two metres distant from the patient, also obviates the distortion inseparable from divergent rays, for at this distance they may be considered parallel. The radiogram thus obtained gives an accurate record of the size and shape of the heart, but a powerful installation is necessary to obtain it.

The Normal Cardiac Outline

The heart is seen as a flask-shaped shadow, lying between the translucent lungs, about one-third of its area to the right, and two-thirds to the left, of the midline. The apex of the heart is internal to the mid-clavicular line. (Fig. 44.)

The *right border* of the cardiac shadow is formed, from above downwards, by two curves:—

(i) A slightly curved portion, the outer edge of the superior vena cava with the ascending arch of the aorta.

(ii) A more convex portion, the outer border of the right auricle, which ends at the diaphragm.

On the *left border* are four step-like convexities, of which the second and third may be difficult to distinguish.

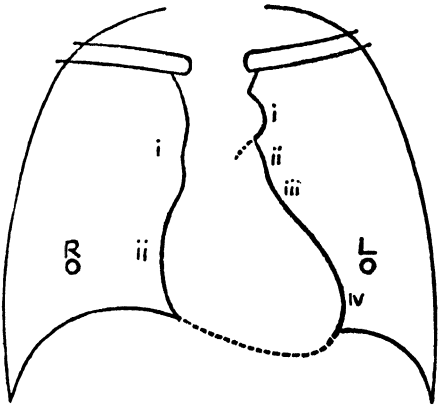


Fig. 44.—Normal cardiac outline (*see* text).

(i) Above, the prominent knuckle produced by the arch of the aorta as it passes backwards, slightly to the left, then downwards.

(ii) The straighter line of the pulmonary artery.

(iii) The conus of the right ventricle.

(iv) Below, the wide sweep of the left ventricle, ending at the apex, where it rests on the diaphragm.

COMMON ALTERATIONS IN DISEASE

1. **Position of the heart in the chest.**—Displacement of the heart as a whole is well seen in pleural effusion and pneumothorax, and in fibroid lung. In distension of the stomach and in obesity, the heart is raised with the diaphragm and the apex tilted upwards. The common type of scoliosis (convexity of the curve to the right) is a frequent cause of displacement of the heart to the left. In narrow chests the heart often hangs mesially and seems small and slender or, in some cases, drop-like.

2. **Shape and size of the heart.**—(*a*) Prominence and undue convexity of the left ventricle is best seen in aortic incompetence and in cases with high blood-pressure. In its typical form the outline may be described as boot-shaped. (Fig. 45, A.)

(*b*) The right auricle is prominent in mitral stenosis, and where there is obstruction in the lesser circulation, as in congenital heart disease and, more rarely, in emphysema. In both conditions the lung areas are commonly darker than normal, owing to the congestion, and there is undue prominence of the pulmonary artery, so that the step-like appearance of the left border of the cardiac outline is exaggerated (Fig. 45, B). A characteristic prominence in the same region is often seen in congenital heart disease.

(*c*) The outline is large and often globular in form where all the chambers of the heart are enlarged. It is seen, therefore, in advanced mitral stenosis with auricular fibrillation, and in combined aortic and mitral disease. (Fig. 45, C.)

3. **Shape and size of the aorta and superior vena cava.**—Aortic enlargement occurs in (*a*) syphilitic aortitis, (*b*) atheroma of the aorta, and (*c*) high blood-pressure. The shadow of

the superior vena cava is widened in congestive heart-failure.

4. **Pulsation of the heart and aorta.**—An abnormal degree of aortic pulsation is a feature in the radioscopic examination of cases of aortic incompetence. General dilatation of the aorta is shown

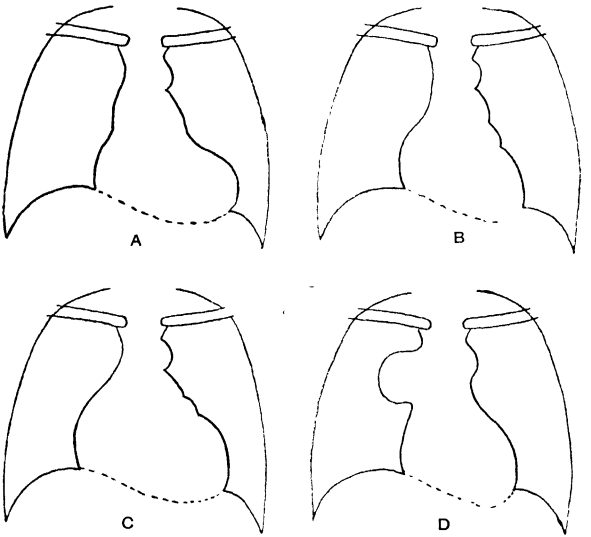


Fig. 45.—Cardiac outline in (A) aortic incompetence, (B) mitral stenosis (C) general cardiac enlargement, (D) aortic aneurysm.

by prominence of the ascending aorta with enlargement of the aortic knuckle and widening of the descending aorta. It is confirmed by radioscopy in the oblique positions. A saccular aneurysm appears as a rounded outgrowth from some part of the aorta, which itself will often be dilated (Fig. 45, D). Its relation to the course of the aorta and its

pulsation help to distinguish it from an intrathoracic tumour.

Watching the progress of a thick barium paste down the **œsophagus** is a valuable aid in the diagnosis of disease of the heart and greater vessels. In its thoracic course the œsophagus is impressed by four structures, namely the aorta arch, left bronchus, left auricle, and descending aorta, and abnormality of these causes alteration of the natural impressions. Of particular importance is the deepening and displacement to the right in the right oblique position of the left auricle impression. Aortic aneurysm is another important cause of œsophageal displacement.

X. EXERCISE TOLERANCE TEST

The best test of the efficiency of the heart is the effect of exercise. In its simplest and safest form the test is applied by the patient's walking briskly up a flight of 40 steps, or he may hop 20 times on each foot, raising the shoulders 6 inches at each hop. The test may also be carried out by his stepping on and off an 18-inch stool twenty times.

As a result of such a test, there should, if the heart is healthy, be little disturbance or respiration and the pulse-rate should not rise by more than 10 to 20 beats per minute and should reach the normal again after about one minute.

XI. CIRCULATORY RATE

When dyspnœa due to heart failure is present the rate of the circulation becomes lowered, and many methods have been devised for measuring the rate of the blood-flow. These all depend on noting the time taken for a substance injected into a vein in the arm to reach the head. The *cyanide* method gives

the *arm-carotid* time (normal, 9 to 21 seconds); 5 minims of a 2-per-cent. aqueous solution of sodium cyanide is used and its arrival at the carotid sinus is marked by a sudden change in the depth and rate of respiration.

The *histamine* method gives the *arm-face* time (normal, 16 to 25 seconds); 4 minims of histamine solution (0.125 mg. in 10 minims of normal saline) causes a blush of the face.

The *sodium dihydrocholate* method gives the *arm-tongue* time (normal, 8 to 14 seconds); 5 cc. of 20-per-cent. solution causes a bitter taste.

The *ether* method gives the *arm-lung* time (normal 3 to 8 seconds); 5 minims causes a sweet taste in the mouth and an odour of ether in the breath.

CHAPTER V

CLINICAL EXAMINATION OF THE BLOOD

Enumeration of Red Blood-Corpuscles

This may be done by means of either a Thoma-Zeiss hæmocytometer or by Strong's method. The former is the method more commonly used; we shall describe it first.

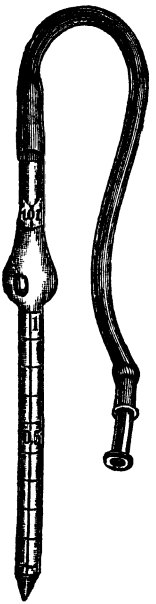


Fig. 46.—Hæmocytometer pipette. (*Thoma-Zeiss.*)

1. **Thoma-Zeiss hæmocytometer.**—The instrument consists of a mixing pipette (Fig. 46), suitably graduated, and a counting-slide. Cleanse the lobe of the patient's ear with ether and dry it. Make a puncture on the lower border of the lobe by means of an ordinary surgical needle. The needle should be inserted with a sudden stab—not slowly—and the blood must flow freely. On no account must the blood be *squeezed* out, as it is then always diluted by lymph squeezed out of the tissues. Slowly suck up blood by means of the pipette till either the mark 0·5 or 1 is reached. If one should happen to go a little beyond the 0·5 mark, the column of blood should be gently blown down to the proper point. If blood has been sucked past the mark 1, it has reached the mixing chamber, and the process must be begun over

again. Having charged the pipette, *wipe the end of it* on a clean cloth, and plunge it at once into the diluting fluid (Appendix, 13), which should be standing ready in a small, wide-necked bottle with the stopper out. Suck up the diluting fluid as far as the mark 101. While this is being done, the pipette should be gently rotated so as to start the mixing. Seize the pipette firmly by its ends between the forefinger and thumb, and shake thoroughly for about one minute. This induces a thorough mixing of the blood with the fluid. It must be remembered that the column of diluting fluid which occupies the capillary part of the pipette does not enter into the mixture. Hence, if blood is sucked up to 0·5, the solution produced is in the proportion of 1 in 200,



Fig. 47.—Thoma-Zeiss counting-slide.
s, Slide: *m*, platform; *c*, wall of trench.

whereas if blood is taken up to the mark 1 the dilution is only 1 in 100. The former degree of dilution is to be preferred in most cases. The finger should now be removed from the pipette, and the diluting fluid in the capillary tube blown out. After a few drops of the diluted blood have been shaken out, a small drop is transferred to the counting-slide (Fig. 47).* The latter consists of a small platform (*m*) bounded by a trench which is surrounded by a glass slab (*c*). On the surface of the platform microscopic squares are ruled, each having an area of $\frac{1}{400}$ sq. mm. Special cover-glasses,† carefully ground, are supplied, which rest upon the glass

* The counting-slide is now made by Hawksley and Sons of London and known as the Thoma (Hawksley). The English slide differs from that made by Zeiss in the absence of the glass slab, *c*, the platform, *m*, being sunk below the general surface of the slide to a depth of $\frac{1}{10}$ mm. The counting-slide is otherwise marked and used in the same manner.

† Ordinary cover-glasses must not be used.

slab, a space being left between the under-surface of the cover and the surface of the platform, which space is exactly $\frac{1}{10}$ mm. in depth.

The drop of diluted blood should be placed in the centre of the platform, and should be of such a size that when the cover-glass is in position the drop is flattened out so as to cover most of the surface of the platform, *but yet without any of it flowing over the edge into the trench.* It requires a little experience to enable one to take just the proper size of drop. It is important that the cover-glass should lie quite flat upon the glass slab. This can best be achieved by previously washing both it and the slab with caustic potash, so as to remove all grease, and then rubbing them with soft chamois leather. The cover must be lowered into position by means of a needle. One recognizes that the cover-glass is lying properly by the appearance of concentric colour (Newtonian) rings between it and the slab; they should be produced by gently stroking down the cover-glass with the handles of two mounted needles. The rings should remain when the cover is simply *lying* on the slab without any pressure being exerted; they are best seen by looking horizontally along the surface of the cover. Unless the rings are seen, one cannot be sure that the space between the cover and the platform is exactly $\frac{1}{10}$ mm. in depth. Having placed the drop in position, and the rings being visible, one should set the preparation aside for two minutes or so, to enable the corpuscles to settle. It should then be examined with the low power to see whether any air-bubbles or foreign bodies are present, and whether the corpuscles are distributed with fair uniformity throughout the field, after which the high power (No. 2 eye-piece and $\frac{1}{6}$-in. objective) is used for counting. The microscope must be vertical and should be provided with a condenser and a diaphragm.

The light should be gradually cut off until the red cells become clearly visible. The little squares will be seen to be marked off into sets of sixteen by double ruling (Fig. 48). Should the lines marking off the squares be only dimly seen, it may be necessary to intensify them. This is best done by rubbing the surface of the platform with a little finely powdered graphite—e.g. the scrapings from a very soft lead pencil—and then polishing it with soft chamois leather.

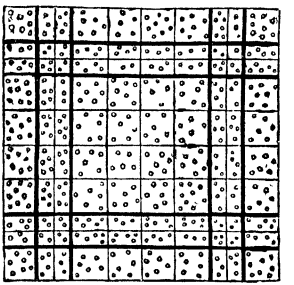


Fig. 48.—Microscopic view of Thoma-Zeiss counting-slide.

For enumeration of the red cells, at least four sets of sixteen squares should be counted. The squares in each set should be gone over systematically in horizontal rows of four at a time. Of the corpuscles which lie *upon* the lines bounding the row, only those on the upper and on the left-hand lines should be counted. The number of corpuscles in each of the four sets should be approximately equal.

Calculation.—Count the corpuscles in each of the four horizontal rows from above downwards. The total is the number of corpuscles in sixteen squares. Count in this way four sets of sixteen, and divide the total by sixty-four, which gives the average of corpuscles in one square. But the dimensions of this square are $\frac{1}{400} \times \frac{1}{10} = \frac{1}{4000}$ c.mm. Therefore, if there be x corpuscles in this dimension, there will be 4,000 x in 1 c.mm. But the blood was diluted 200 (or 100) times. Therefore, in 1 c.mm. of blood there will be 4,000 $x \times 200$ (or 100) corpuscles.

Suppose, for example, that one finds a total of 384 corpuscles in the sixty-four squares. This gives an average of six corpuscles per square, or $6 \times 4{,}000$; i.e. 24,000 per c.mm. of *diluted* blood,

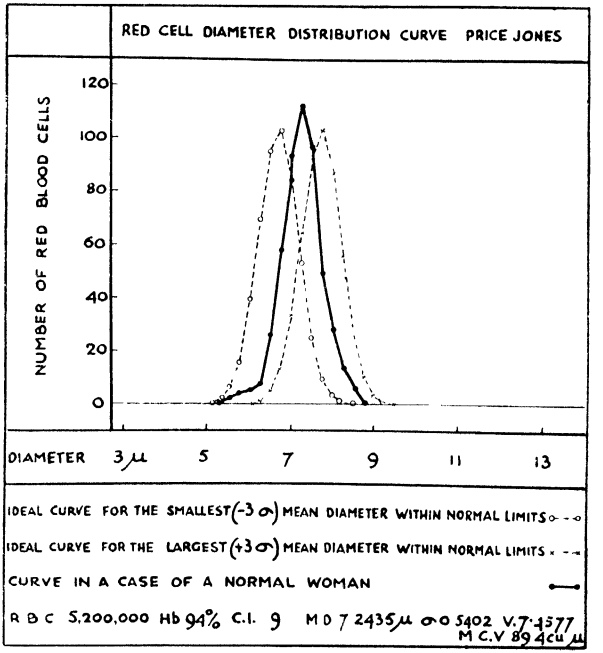


Fig. 49.—Price-Jones curve in a normal woman.

or 4,800,000 per c.mm. of pure blood, if the dilution was 1 in 200.

2. **Strong's method (modified).**—The necessary instruments consist of a capillary tube provided with two marks placed close together (the tube at the

lower mark contains 5 c.mm.; from the upper mark it delivers 5 c.mm.), a pipette graduated for 995 c.mm., and a small bottle provided with a well-fitting stopper. Take up 995 c.mm. of diluting fluid (Appendix, 14), and transfer it to the bottle. Draw up 5 c.mm. of

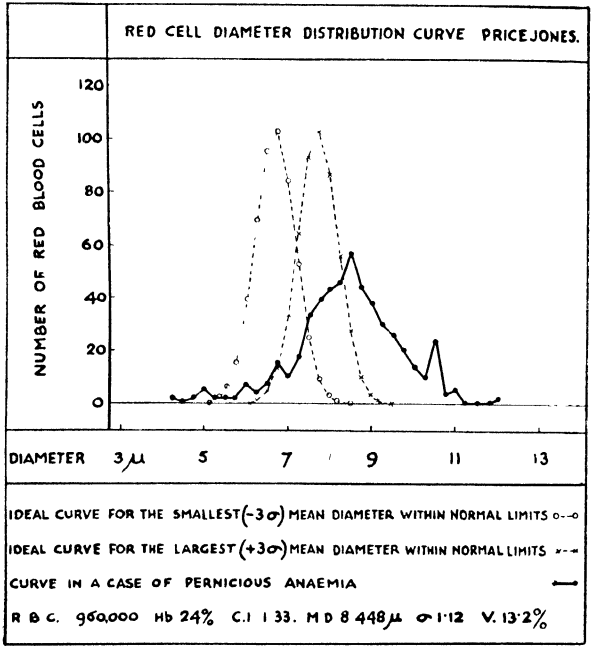


Fig. 50.—Price-Jones curve in a case of pernicious anæmia.

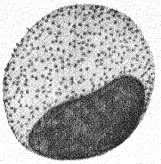
blood to the lower mark in the capillary tube, wipe the outside of the tube free from blood, blow out the blood into the diluting fluid, and wash out the inside of the tube by sucking the mixture of blood and fluid several times up and down the tube. The blood is now

PLATE 11

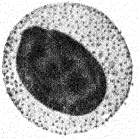
BLOOD CELLS, NORMAL AND ABNORMAL

The left-hand column represents cells produced in the bone-marrow, and present in the blood in disease only. The central column represents normal and abnormal varieties of red cells. The right-hand column illustrates the normal leucocytes of the blood.

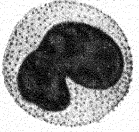
(Leishman's stain.)



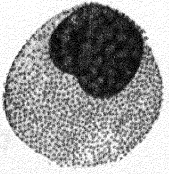
Neutrophil myelocyte (large type).



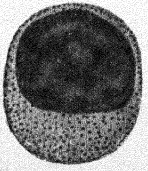
Neutrophil myelocyte (small type).



Transitional neutrophil.



Eosinophil myelocyte.



Basophil myelocyte.



Normal red cell.



Poikilocyte.



Polychromatophilia.



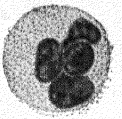
Normoblast.



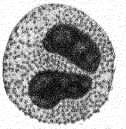
Megaloblast.



Granular degeneration.



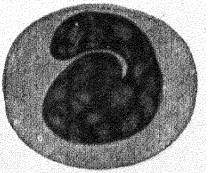
Polynuclear neutrophil.



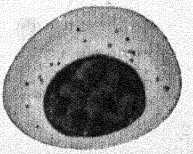
Eosinophil.



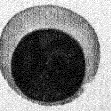
Mast cell.



Large hyaline.



Large lymphocyte.



Small lymphocyte.

Plate 11

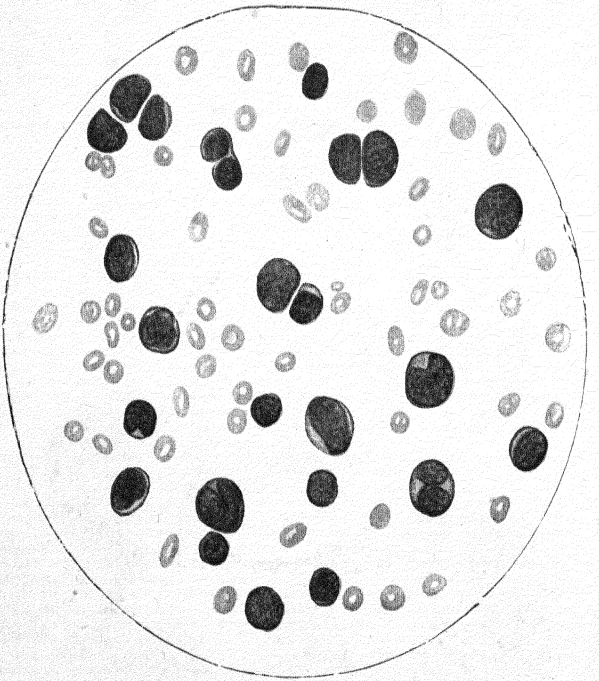


Plate 12.—THE BLOOD IN LYMPHATIC LEUKÆMIA.

The lymphocytes are abnormal in size and in shape of nucleus; they are of the small type. (*Leishman's stain.*)

diluted in the proportion of 1 in 200. Shake the bottle thoroughly and transfer a drop of the mixture to the Thoma-Zeiss counting-slide. Proceed as described under the Thoma-Zeiss hæmocytometer.

The advantages of this modification are that the

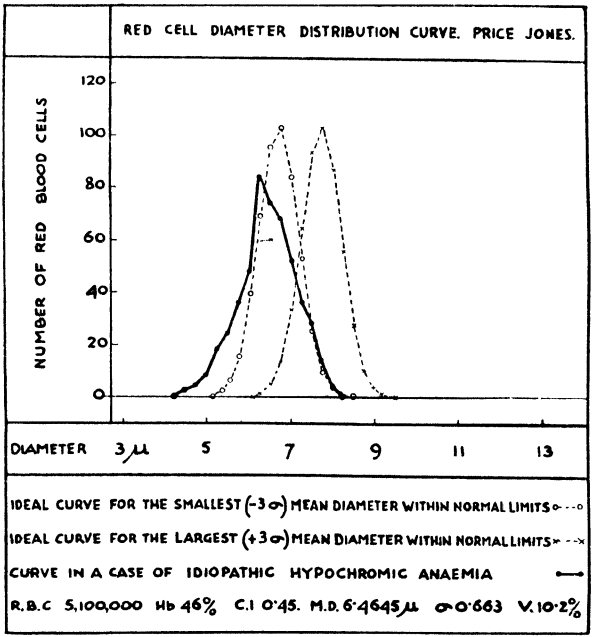


Fig. 51.—Price-Jones curve in a case of idiopathic hypochromic anæmia.

mixture of blood and diluting fluid is more intimately made, that in the stoppered bottle the mixture can be readily transported and counted at leisure, and that the same mixture can be used for an accurate estimation of the leucocytes.

The normal **number of red corpuscles** varies within considerable limits. At birth it is from 5,200,000 to 5,600,000 per c.mm., and falls during the first week of life to between 4,000,000 and 5,000,000 per c.mm. In adult life it is different in the two sexes. In men the average figure is found to be 5,428,000 per c.mm. The range or limit of health (calculated from the standard deviation of the distribution of a large series of observations) is from 5,100,000 to 6,350,000 per c.mm. In women the average figure is 5,012,000 per c.mm. and the limit of health 3,940,000 to 6,080,000 per c.mm. For purposes of estimating the colour index the number five million is adopted for the sake of convenience.

RED CELL DIAMETER DISTRIBUTION CURVE (PRICE-JONES)

Variations in the diameters of red blood cells are observed and measured in dried films These are dried in air without heat, fixed and stained with Jenner stain for two minutes, and after washing with distilled water and drying, are super-stained with a weak aqueous solution of eosin for two minutes.

A projection apparatus adjusted for a magnification of 1,000 diameters is then arranged to project the microscope field on to a sheet of paper. Five hundred red cells are then outlined in pencil. Two diameters, maximum and minimum of each of these cells, are then measured to 0·5 mm. with a glass millimetre-scale and can be expressed directly in terms of μ. Calculations are then made from mathematical formulæ, and, from the figures thus obtained, a distribution curve is plotted.

The normal range of mean diameter is 6 686 μ to 7·718 μ with a normal mean of 7 20 μ (Price-Jones).

Fig. 49 shows the normal mean diameter of 500 red cells in a healthy woman to be 7 24 μ. To the right of this is the ideal curve for the largest mean diameter within normal limits (7·718 μ); to the left the ideal curve for the smallest mean diameter within normal limits (6 686 μ). A mean diameter outside these limits is abnormal. By counting the cells that fall outside these limits it is possible to calculate the degree of megalocytosis or microcytosis. The healthy range of the coefficient of variability which gives mathematical expression to the degree of anisocytosis present is from

5.33 per cent. to 7 32 per cent , with a mean of 6.33 per cent. (Price-Jones)

In pernicious anæmia the average mean diameter is high (8 31 μ). So also is the average coefficient of variability (13·0 per cent.). Price-Jones concludes that a high variability is almost more constant, though less characteristic, than a high mean diameter. In the case illustrated here the mean diameter is 8 45 μ, and the coefficient of variability is 13·2 per cent. The high variability is to be seen in the wide base of the curve (Fig 50). It is a mathematical expression of a high degree of anisocytosis. The megalocytosis is to be seen in the fact that the peak of the curve falls outside the right-hand boundary of the calculated curve.

In idiopathic hypochromic anæmia the mean diameter may be less than normal, but often falls within normal limits. In a series of eight cases Price-Jones found the mean diameter ranged from 6·2 μ to 6·7 μ. In all there was a microcytosis, 6 to 37 per cent. of the cells lying outside the left-hand boundary of the calculated curve. In the case illustrated here (Fig. 51) the mean diameter is 6·46 μ.

Enumeration of Leucocytes

1. **By the Thoma-Zeiss hæmocytometer.**—A special pipette is supplied for this purpose with the Thoma-Zeiss instrument. It is used in precisely the same manner as the red-corpuscle pipette, but permits of a lesser degree of dilution of the blood. The best diluting fluid to employ is one containing 1 c.c. of glacial acetic acid in 100 c.c. of water, to which enough of a watery solution of methyl green or gentian violet has been added to give the mixture a decided colour. The advantage of this mixture is that it dissolves all the red cells while it stains the nuclei of the white. One can thus easily count the whites, and at the same time note roughly the relative numbers of the unipartite and multipartite nucleated varieties.

It is important that a large drop of blood should be allowed to exude before one begins to fill the pipette. The blood should be sucked up to the mark 0·5, the

end of the pipette wiped, and diluting fluid taken up to the mark 11.

Owing to the relatively large calibre of the pipette, the blood is apt to run out of it. It is well, therefore, to keep the pipette in a horizontal position as soon as one has filled it with blood.

The blood and the fluid are mixed as already described. This produces a dilution of 1 in 20. A drop is then placed on the counting-slide with the same precautions as were observed in the case of the red cells.

In this case the whole sixteen sets of sixteen squares should be counted, or 256 squares in all. Instead of going over the squares in rows of four, a whole set of sixteen can easily be counted at one time. A movable stage greatly facilitates the enumeration.

One should note on a piece of paper the number of leucocytes with multipartite and with rounded nuclei respectively in each set of sixteen squares. In this way one gets a rough idea of the proportion of each variety present, and by adding them together the total number of white corpuscles is obtained. The *calculation* is made in the same way as that of the red corpuscles, it being borne in mind that each of the 256 squares counted represents $\frac{1}{4000}$ c.mm. of diluted blood, and that the dilution is much less than in the enumeration of the reds (1 in 10, or 1 in 20). For example, if there be 20 leucocytes in the 256 squares, this represents an average of $\frac{20}{256}$ per square, or $\frac{20}{256} \times 4{,}000$ per c.mm. of diluted blood, or 6,250 per c.mm. of pure blood if the dilution is 1 in 20. This is about the normal number.

In leukæmia, where a very large excess of leucocytes is present, one can easily count the red and the white cells in the same drop. For this purpose a 3-per-cent. solution of common salt just coloured with gentian violet is to be preferred for diluting the blood.

This stains the nuclei of the whites, and at the same time preserves the reds. Toisson's solution (Appendix, 15) may be used similarly. The dilution and calculation are the same as for the red cells.

2. **By Strong's method.**—The same mixture and the same capillary tube are used as for the enumeration of the red cells. The stoppered bottle is well shaken, and 5 c.mm. of the mixture is drawn to the upper of the two marks in the capillary tube. The end of the tube is wiped and held lightly against the centre of a clean slide, the tube being at right angles to the slide. The contents of the tube are gently blown out on to the slide in the form of a drop. The drop is allowed to dry, is then stained for five minutes in filtered hæmalum (or in any simple nuclear stain), washed in tap-water, again dried, and mounted in cedar-wood oil. The leucocytes are stained blue, the red cells being unstained. All the leucocytes in the drop are then counted in the following manner: A metal disc with a central square aperture is placed in the eye-piece of the microscope. The metal disc may be replaced by a paper or cardboard one of home manufacture, all that is necessary being to obtain a square field. The $\frac{1}{6}$-in. objective is used, and the microscope should be fitted with a mechanical stage. The edge of the drop is easily defined, and by moving the stage the leucocytes in the top segment of the drop are counted from left to right. By marking a red cell the drop can be moved down exactly one square field, and by proceeding backwards and forwards across the drop no leucocytes need be omitted. The total number of leucocytes thus counted comprises those present in 5 c.mm. of blood diluted 1 in 200. To arrive at the number of leucocytes per c.mm. of undiluted blood, the number counted should therefore be multiplied by

40. In cases of leukæmia a further dilution is necessary, and this may be made by means of an ordinary Wright's pipette. After shaking the bottle, one volume of the original dilution is taken, followed by 9 volumes of diluting fluid, giving a blood dilution of 1 in 2,000; 5 c.mm. of this mixture are put up and counted, and the number found is multiplied by 400.

After use, the diluting pipettes should be thoroughly cleaned. A little trouble in this is repaid by saving of time and annoyance when next they come to be used. They should be washed out (1) with distilled water, (2) with absolute alcohol, and (3) with ether. A stream of air should then be blown through till one is sure that the glass ball in the chamber moves freely without tending to adhere to the sides. To save time in these manipulations, the rubber tube may be taken off and the fluid blown out through the wide end of the pipette. If a few drops of antiformin are sucked into the pipette it quickly disintegrates any organic matter left behind. Coagulated blood may be removed from the capillary tube by means of a horse-hair. If the blood adheres firmly to the pipette, it may be removed by repeated rinsing with strong alkali or acid, or it may even require to be digested away with pepsin.

The **number of leucocytes in normal blood** is about 6,000 per c.mm. in the adult. The number varies, however, within considerable limits even in health. In early childhood much higher numbers are reached (*see* p. 526). The normal proportion of leucocytes with divided nuclei to those with rounded nuclei is about 2 to 1.

Leucocytosis is often pathological, the increase affecting chiefly the polymorphonuclear neutrophil cells, hence the name *neutrophilia*. It occurs in suppurative infections, tonsilitis, lobar pneumonia, and also following acute hæmorrhage, and in rapidly

forming malignant neoplasms. The condition of the leucocytes in *leukæmia* will be referred to later (p. 211).

Estimation of Platelets

The **blood-platelets** or "thrombocytes" are definite elliptical or circular bodies with basic cytoplasm and azurophil granules. The normal platelet-count may be stated as 150,000 to 300,000 per c.mm. and the normal "platelet ratio," that is the ratio of platelets to red cells, is 1 in 18. **Thrombocytopenia,** or diminution of the platelets, is associated with bleeding into the skin and from the mucous membranes. It is important in certain forms of purpura. **Thrombocythæmia,** or increase of the blood-platelets, may be accompanied by vascular thromboses. It is sometimes encountered after splenectomy.

To count the platelets the skin of the ear is cleaned up with ether and on the clean surface is deposited a large drop of diluent (2 per cent. sodium citrate in normal saline). The skin is then stabbed by means of a sharp sterile needle so that the blood oozes directly into the diluent This prevents clumping of the platelets. With a platinum loop of 3 mm. diameter some of the diluted blood is transferred to a slide and carefully covered with a cover-slip The amount taken should be sufficient to spread out evenly between the slide and cover-slip without causing the latter to float. The preparation is ringed with vaseline Using a microscope fitted with moving stage, square eye-piece, and $\frac{1}{12}$-in. oil immersion objective, the number of platelets and red cells is counted in several fields, thus determining the ratio of platelets to red cells. With a knowledge of the red-cell count the actual number of platelets per c mm. is easily calculated.

Estimation of Hæmoglobin

For the estimation of the amounts of hæmoglobin one has the choice of several instruments:—

1. **Gowers' hæmoglobinometer.**—Place a couple of drops or so of distilled water in the little

graduated test-tube supplied with the instrument. Get a large drop of blood from the ear, and fill the pipette with it up to the mark. Then dip the end of the pipette into the distilled water in the tube and gently blow out the contained blood. Mix, and go on adding water drop by drop, comparing the colour from time to time with that of the standard tube. The latter is filled with tinted gelatin, and represents the colour of blood which contains a normal amount of hæmoglobin when diluted in the proportions effected by the instrument. The comparison should be made both by transmitted light, care being taken to hold both tubes level with the eye, and by reflected light, the tubes being held side by side against a sheet of paper. Good daylight is indispensable. Stop adding water when the tint in the two tubes is the same, and read off the level at which the mixture stands in the graduated tube. If this be at (say) 60, then the blood contains 60 per cent. of hæmoglobin. The mean of the dilution which is just too much and that which is just too little is the correct point.

2. **Haldane's hæmoglobinometer.** — Haldane has modified Gowers' instrument by using as a standard of comparison, instead of gelatin tinted with picrocarmine, a 1-per-cent. solution of blood containing the average percentage of hæmoglobin found in the blood of healthy men, and saturated with carbonic oxide. It has an oxygen capacity of 18·5 per cent. as determined by the ferricyanide method, and is both definite and permanent.

The instrument (Fig. 52) is used as follows:—

Sufficient water is first placed in the graduated tube to dilute the blood as far as safely possible. A puncture is then made in a finger or the lobe of an ear, and the capillary pipette (which must be clean and dry) at once filled to a little beyond the mark 20 from the drop of blood obtained. The point of the pipette is wiped, and dabbed on any convenient surface

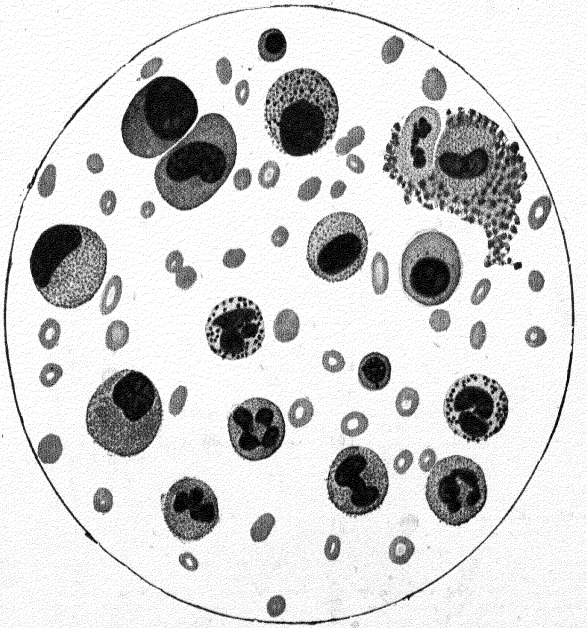


Plate 13.—THE BLOOD IN MYELOID LEUKÆMIA

(*Leishman's stain.*)

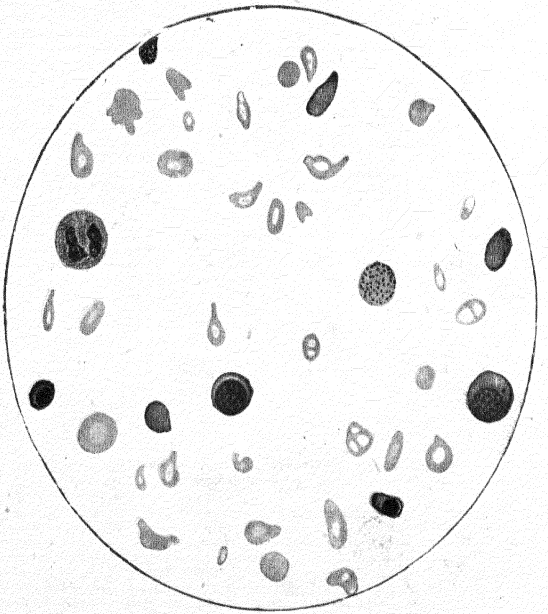


Plate 14.—THE BLOOD IN PERNICIOUS ANÆMIA.

(Leishman's stain.)

until the contained blood stands exactly at the mark. The blood is then gently blown out into the graduated tube, where it sinks; the pipette is rinsed with the water in the graduated tube and withdrawn. The piece of rubber tube attached to a gas-burner, or preferably to a CO generating apparatus, is now introduced into the graduated tube to near the level of the water, and gas allowed to pass for a few seconds. As the

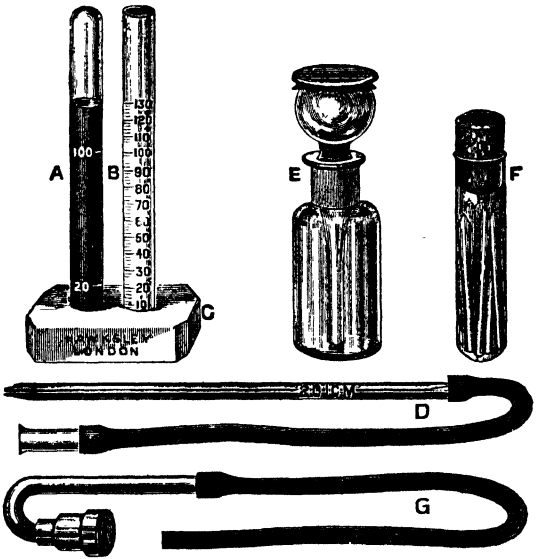


Fig. 52.—Haldane's Hæmoglobinometer.

A, Standard blood solution ; B, graduated mixing tube ; C, rubber stand, D, pipette, E, bottle for distilled water, F, lancets, G, tube and cap for fixation to gas-burner.

tube is withdrawn (with the gas still passing) the end is closed with the forefinger, and the liquid made to pass up and down in the tube—not violently shaken—at least a dozen times, so as to saturate the hæmoglobin with CO. During this manipulation the tube is held in a handkerchief, otherwise it will become heated and liquid will spurt out when the finger is withdrawn. Water is now added drop by drop with the pipette topper, the tube after each addition being inverted, until the

point is reached at which the tints of the liquids in the two tubes are just equal. In judging of the equality the tubes should be held against the light from the sky, or, if artificial light be used, from an opal glass shade It is also absolutely necessary to transpose the tubes repeatedly, otherwise serious errors may arise The level is read off on the graduated tube after half a minute has elapsed since the last drop added was mixed with the rest of the liquid by inverting the tube. The observation is repeated after the addition of another drop of water, and if necessary another, until the point is reached when the tints are again unequal The true result is the mean of the readings giving equality. The error in any single determination ought not to exceed 1 per cent.

The result obtained is the percentage actually present of the average proportion of hæmoglobin in the blood of healthy adult men The blood of healthy men, however, contains more hæmoglobin on an average than the blood of healthy women and children Women give an average of 89 per cent , and children of 87 per cent , of the proportion in men. The results may be expressed in terms of oxygen capacity (the number of volumes of oxygen taken up in combination from air by 100 volumes of blood), if it is borne in mind that 100 per cent on the scale corresponds to an oxygen capacity of 18 5.

The advantages of the modifications introduced into the original method of Gowers are · (1) That the standard solution is a definite one, so that an instrument can be verified at any time by making a determination with ox-blood of which the oxygen capacity has been determined by the ferricyanide method; (2) that the standard solution is permanent; (3) that the apparatus can be used with equal correctness by daylight and by artificial light.

If the capillary pipette becomes blocked or soiled by coagulated fibrin, use the brass wire in the case from the wide end of the pipette, or a horse-tail hair, to remove it. (On no account use iron or steel wire) To dry the pipette, suck air through it. Always examine the pointed end of the pipette before using it: it should be smooth and rounded; if sharp or angular it is broken.

3. **Oliver's hæmoglobinometer.**—The apparatus adopted by Oliver is founded on the colorimetric principle. But the applications of that principle are modified in these ways:—

(1) Double transmission of light (or reflected light) is used instead of single transmission.

(2) A standard white background is selected on which the solution of the blood and the standard colours rest.

(3) The standard is presented as a series of definite gradations.

(4) The colour of the blood solution is compared with that of the standard in camera.

The **apparatus** has been considerably modified of recent years. A full set of directions is supplied with the modern instrument, which makes a detailed description here unnecessary.

Another hæmoglobinometer in common use is Sahli's modification of Gowers' instrument, in which the blood to be examined is mixed with decinormal hydrochloric acid and compared with a standard solution of blood and acid. The Tallqvist method gives results which are only roughly approximate and therefore cannot be recommended.

The normal **percentage of hæmoglobin** varies within considerable limits and differs in the two sexes. The mean concentration of hæmoglobin in the capillary blood of healthy men is 105 per cent. on the Haldane hæmoglobinometer scale, which is equivalent to 14·5 grm. of hæmoglobin per 100 c.c. of blood. The observed limits are 96 per cent. to 116 per cent. In the case of healthy women the mean concentration in the capillary blood is 98·3 per cent. which is equivalent to 13·6 grm. of hæmoglobin per 100 c.c. of blood. The observed limits are 90 per cent. to 110 per cent.

One can also state the percentage of hæmoglobin in terms of the average amount contained in each corpuscle, this being known as the **colour index.** Thus, if the number of red cells be 20 per cent. of the normal and the hæmoglobin 10 per cent., then the hæmoglobin value of each corpuscle is $\frac{1}{2}^{0}/_{0}$ or half normal. In calculating the percentage of red cells, 5,000,000 is for convenience commonly reckoned as the normal number of red cells per c.mm. or 100 per cent. The importance of this method of expressing

the facts is seen when one recollects that the total amount of hæmoglobin in the blood may be diminished while the average amount in each corpuscle is really above the normal. This happens in some forms of anæmia.

The last point to be remembered in making blood estimations is that, as far as possible, all observations on the same individual should be carried out under the same conditions as regards time of day, taking of food, etc. This is important, as it is found that the composition of the blood is temporarily altered by the taking of food, or by the occurrence of profuse sweating, diarrhœa, etc.

Microscopical Examinations of Blood

Blood may be examined (1) fresh, (2) stained.

1. **Blood examined fresh.**—This simple procedure should never be omitted, since much valuable information may be derived from it. To obtain the blood, hold a cover-slip lightly on a slide, one edge of the cover-slip being exactly flush with one edge of the slide. Hold these edges against a drop of blood; the blood will then flow between slide and cover-slip. Examine at once with the diaphragm of the microscope partly shut down.

In the case of normal blood, the red corpuscles will be observed to range themselves in rouleaux as one watches, clear spaces being left between in which the white cells and little clumps of aggregated platelets may be seen. Any abnormality in the shape or size of the red cells or in the formation of rouleaux should be noted. Distorted red cells—pear-shaped, indented, budded, etc.—are known as poikilocytes; red cells larger than normal are called megalocytes; smaller than normal, microcytes. All these abnormal shapes and sizes may occur in any form of severe anæmia.

One can also see if any large excess of white corpuscles is present. The presence of abnormal elements should be noted. Among these are abnormal varieties of white cells, more easily recognized, however, in stained specimens.

Sometimes particles of pigment can be noticed amongst the corpuscles. This condition, known as **melanæmia,** is found occasionally in chronic malaria.

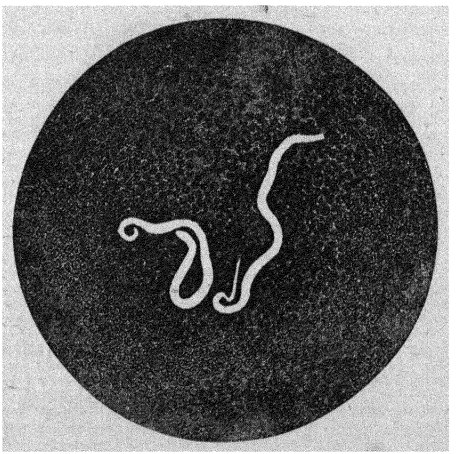


Fig. 53.—Filaria bancrofti ; × 160.

During a malarial attack the parasites can readily be seen and the activity of their pigment granules noted.

The **spirillum** of relapsing fever and the **trypanosoma** of sleeping sickness can be recognized by this method, as also the **Filaria sanguinis hominis (F. bancrofti).** This last can be seen, with a low power, moving about among the red cells. The embryos average about $\frac{1}{75}$ in. in length, and are about as broad as a red blood-corpuscle. (Figs. 53, 54.) They remain

alive for a surprisingly long time even at ordinary temperatures, especially if the preparation is kept from drying by being sealed with a little vaseline. The following is the method for their demonstration:—

Spread out a thick drop of blood on a slide by means of a needle, and allow it to dry. It may then be preserved indefinitely. When the parasites are to

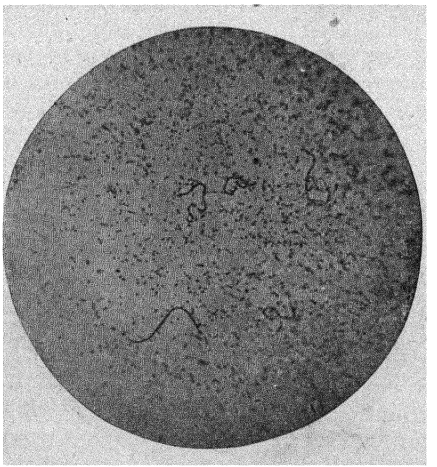


Fig. 54.—Embryos of Filaria bancrofti in blood ; × 5.
(From an original photomicrograph.)

be demonstrated, immerse the slide in a solution of 1 drop of saturated alcoholic fuchsin in 1 oz. of water. Stain in this for one or two hours. If, on examining the film, it is found that the blood is very deeply stained, one must decolorize by means of dilute acetic acid (4 drops of acetic acid to 1 oz. of water). The specimen may be examined either wet or dry, and with or without a cover. On searching it with a low power, the filariæ will be recognized by their being

very deeply stained. The preparation is apt to fade after a few days.

A more rapid result is obtained by staining the film for half a minute in a 2-per-cent solution of methylene blue. It is then decolorized a little with dilute acetic acid as above described, and examined with a low power while wet. If a permanent preparation is desired, the film is allowed to dry and a drop of balsam and a cover-glass are applied.

2. **Examination of blood in films.**—Films may be made either on slides or on cover-glasses. The former have the advantage of being more easily cleaned and manipulated, but cover-glasses give the best results in skilled hands. The slides should be of colourless glass, thin, and with ground edges. The cover-glasses should be $\frac{3}{4}$ in. square, and as thin and flexible as possible. It is important that both slides and cover-glasses should be entirely free from grease. To ensure this the cover-glasses should be dropped one by one into an enamelled iron dish containing 10-per-cent. chromic acid and boiled for 20 minutes. They should then be tipped into a shallow basin, and water allowed to run on them till the washings are colourless. After this they are covered with spirit, and finally transferred with forceps to a wide-necked stoppered bottle containing absolute alcohol. When required for use they should be picked out with forceps, excess of alcohol drained off, and the remainder got rid of by passing through a flame. They should finally be rubbed with a clean handkerchief. Slides may be cleaned in the same manner, but it is sufficient to polish them with the finest emery paper and then to place them in absolute alcohol till required. The alcohol is best removed by wiping dry with a clean cloth.

If ordinary slides or cover-slips have to be cleaned

in a hurry, glacial acetic acid, followed by water and alcohol, gives good results.

How to make films. (1) *On cover slips.*—The surface of the cover-slips must on no account be touched by the fingers. They may be held by their corners between the thumb, middle and index fingers, but it is preferable to use forceps—a clamp forceps for the lower cover-slip and a fine-pointed forceps for the upper. Clean, dry, and prick the lobe of the ear; wipe away the first drop of blood, and when another about the size of a large pin's head has appeared, touch its apex with the upper cover-slip and lightly drop it diagonally on to the surface of the lower. Directly the blood has spread, separate the slips with the utmost rapidity, avoiding any pressure or lifting (Fig. 55). If the separation is delayed, the slips tend to stick and the films are useless; if made too soon, the resulting films are small and thick.

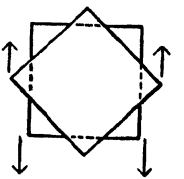


Fig. 55.—Method of making films on cover-slips. (*After Daniels.*)

(2) *On slides.*—Apply one end of a slide to a drop of blood, place the slide on some firm, smooth surface, such as a polished table without a cloth, holding it in position with the thumb and index finger of the left hand. The narrow edge of a second slide is placed in the drop and held there till the blood has spread across it; it is then drawn slowly over the whole length of the first slide. The inclination of the second slide to the first should be at an angle of 45°, and there should be no pressure whatever between the two surfaces; this can be ensured by holding the second slide between the thumb on one side and the index and middle fingers on the other, allowing the tips of the thumb and index

finger to rest on the table. The more slowly one slide is drawn over the other the thinner is the resulting film. Smooth spreading of the film is aided by warming the first slide in the flame of a spirit-lamp immediately before applying it to the drop of blood. After the blood is spread it should be dried by being waved rapidly in the air to prevent undue shrinkage of the cells.

Fixation of the film.—Films may be fixed either while still wet or after drying. They may be fixed wet by exposure to formol vapour for twenty minutes; after drying, by immersing in absolute alcohol for fifteen minutes. Fixation of the films when dry is provided for by the methods of Jenner and Leishman, since the stains are dissolved in methyl alcohol.

How to stain the film.—Either of the two following methods gives excellent results:—

1. *Jenner's stain.*—The stain consists of a 0·5-per-cent. solution of a specially prepared crystalline compound of methylene blue and eosin in pure methyl alcohol. Films are made on cover-slips in the usual way. So soon as they are dry a few drops of the solution are poured on, and they are covered with watch-glasses to prevent evaporation and precipitation of the stain. Pour off in one to four minutes. Rinse in *distilled* water till pink (this takes five to ten seconds). Dry rapidly high over a flame or by waving in the air. Mount in xylol balsam. In a successful film the red corpuscles are terra-cotta-coloured; nuclei are blue, platelets mauve, the granules of polynuclear cells and myelocytes red, mast cells dark violet, bacteria, filarial and malaria parasites blue.

2. *Leishman's stain.*—This is a simplification of the method of staining first introduced by Romanowsky. The stain consists of a compound of alkaline medicinal methylene blue and eosin, extra B.A. (Grubler),

dissolved in pure methyl alcohol in the proportion of 0·5 per cent. The dry film is well covered with the stain, which should be evenly distributed over the entire slide or cover-glass. At the end of one minute, double the quantity of distilled water is carefully added and mixed with the stain by means of a clean glass pipette. At the end of seven minutes the mixture is poured off, and the film covered with distilled water for two minutes. The water is then washed off with fresh distilled water, and the film gently blotted dry with clean blotting-paper. When dry it can be mounted in xylol balsam.

Examination of the film.—In a good film the corpuscles should be spread out evenly, no rouleaux being seen. Even with the low power the white cells can be recognized by their stained nuclei, and some idea of their relative numbers gained. For the minute examination of the white cells a high power, and preferably an immersion lens, is requisite. In many cases it is important to make a **"differential count"** in order to ascertain the relative numbers of the different varieties of leucocyte. For this purpose 200-500 cells must be counted, which, with a little practice, can be done in a quarter of an hour.

It is often necessary also to calculate the *absolute* number of each kind of white cell per c.mm. of blood, as otherwise a relative increase or diminution of one kind may be mistaken for an absolute increase or reduction. Throughout *adult* life the absolute number of polynuclears per c.mm. is about 4,000, whilst that of the lymphocytes is about 2,000.

The following are the varieties of leucocytes found in normal blood (Plate 11), with their relative proportions:—

1. *Finely granular oxyphils* (or polynuclear neutrophils). Cells with multipartite nucleus and

fine neutrophil or faintly oxyphil granules: 60 to 65 per cent.

2. *Coarsely granular oxyphils* (or eosinophils). Cells with multipartite nucleus and coarse, strongly oxyphil granules: 2 to 3 per cent.

3. *Coarsely granular basophils* (or mast cells). Cells with very pale cytoplasm, a nucleus usually bilobed, and coarse basophil granules: 0·5 per cent.

4. *Large hyalines* (or large mononuclears). Cells with a characteristic notched or kidney-shaped nucleus and a slightly basophilic, faintly reticular cytoplasm: 3 to 5 per cent.

5. *Large lymphocytes* with round nucleus and clear basophilic cytoplasm: 5 to 10 per cent.

6. *Small lymphocytes* with round, deeply staining nucleus which almost fills the cell, leaving a rim of strongly basophilic cytoplasm: 20 to 25 per cent.

Some of the alterations which occur in the relative proportions of these in **leucocytosis** have already been mentioned (p. 198).

In the lymphatic form of **leukæmia** an enormous increase occurs in the number of the lymphocytes (Plate 12).

In the myeloid form of the disease the neutrophils, eosinophils, and mast cells are all increased, and in addition bone-marrow cells—myelocytes—appear in the blood. These are often of large size, with a single round nucleus, and contain granules which may be either neutrophilic or eosinophilic in reaction (Plate 13).

A relative diminution of the leucocytes is spoken of as **leucopenia.** As a rule in leucopenia the diminution affects chiefly the granulocytes, hence the

name **granulocytopenia.** When this occurs there is, of course, a relative lymphocytosis. Leucopenia occurs in:—

(1) Infections such as typhoid fever, undulant fever and measles.

(2) Exhaustion of the bone-marrow, as in aplastic anæmia and chronic benzene poisoning.

(3) Crowding out of the leucopoietic tissues by abnormal erythropoiesis, as in pernicious anæmia and acholuric jaundice.

The **red cells** may present various alterations in disease (Plate 11). The alterations may affect—

(*a*) Their *size* and *shape*. Instead of the normal-sized erythrocytes, small cells may appear, devoid of the usual central indentation (microcytes), or unusually large forms may be met with (megalocytes), particularly in pernicious anæmia. Instead of being rounded the corpuscles may become oval, pear-shaped, etc. These changes are spoken of collectively as *poikilocytosis*.

(*b*) The *staining power* of the cells may be altered. Thus, instead of taking up eosin in the normal manner, they may stain with the basic dye, and have a violet or even bluish tinge. This is spoken of as *polychromatophilia*. It is seen in various kinds of anæmia, and is believed to indicate either a degeneration of the red cells or, more probably, an immature condition of them.

(*c*) *Nucleated forms* may appear. If these are of the same size as ordinary red corpuscles they are spoken of as *normoblasts*. They can be distinguished from lymphocytes (for which at the first glance they are apt to be mistaken) by (1) the more homogeneous and intense staining of the nucleus, (2) the presence

round the nucleus of a cell-body which stains red, (3) their smoother contour.

Megaloblasts are large nucleated red corpuscles. They may be even four times as large as an ordinary red cell. They have a relatively small and characteristically stippled nucleus, and a large cell-body, which always exhibits polychromatophilia. Megaloblasts are a characteristic feature of the blood in pernicious anæmia (Plate 14).

Microblasts—nucleated forms smaller than an ordinary red corpuscle—are found in traumatic anæmias, and are of no great significance.

Reticulated red corpuscles or "reticulocytes" can be demonstrated by *supravital staining*, that is by the application to fresh blood of a special dye before the use of a fixative. The reticulocytes are the youngest red cells in the circulation, and, in the normal blood they rarely exceed 1 per cent. of the total red cells. In circumstances demanding active regeneration of blood they may reach 15 to 30 per cent. The reticulocyte (*see* Plate 15) is of slightly larger diameter than the red cell and contains a delicate granulo-filamentous cytoplasmic network which later disintegrates and disappears as the cell matures into a red blood-corpuscle.

The best dye to use for supravital staining of the blood is brilliant cresyl blue. Saturate 0·85-per-cent. sodium chloride solution with the dye, filter through a double filter-paper and centrifugalize. Pour off the supernatant dye solution and keep it in a stock bottle. For use dilute a small quantity with four volumes of 2-per-cent sodium citrate in physiological saline. Puncture the ear and draw a large drop of blood into a Wright's pipette. Follow up the blood by an equal volume of dye. Blow out on to a slide, mix thoroughly, take up again into the Wright's pipette, seal off and incubate at body temperature for 20 minutes. Then make films by the same technique as for blood-films, fix in methyl alcohol and counterstain with Giemsa (1 in 20) for

20 minutes. In counting the cells the use of an Ehrlich eye-piece is of great assistance.

Punctate basophilia of the red cells, "granular degeneration" or "stippling," is a condition in which the reticulum of the reticulocyte is broken up into a number of basophil granules by the action of some toxic substance. It is occasionally seen in the various anæmias and is especially important in lead-poisoning.

It is best demonstrated by staining blood-films with Unna's alkaline methylene blue solution (one part of methylene blue and one part of potassium carbonate per hundred parts of water). A blood-film is fixed in methyl alcohol, dried, flooded with a 1-in-15 dilution of the stain and allowed to stand for 15 minutes The stain is then washed off with water until only a faint bluish-green tinge remains.

Fragility of the red corpuscles.—The fragility of the red blood-corpuscles is shown by their inability to resist hæmolysis in diminishing strengths of salt solution. Normal salt solution is usually taken as 0·85 per cent. of NaCl, but normal red cells do not hæmolyse until a dilution of about 0·4 per cent. is reached. In most forms of chronic jaundice the red cells are less fragile than normal cells, and do not hæmolyse in a salt solution appreciably less than 0·4 per cent. In hæmolytic icterus the fragility of the red cells is greatly increased, and hæmolysis takes place in strengths of salt approaching that of normal saline. Not uncommonly hæmolysis begins at 0·6 per cent. of salt, or even higher. The undue fragility of the red cells is the most constant and characteristic sign of this disorder, and such wide deviation from the normal is found in no other condition.

The estimation of the fragility of the red blood-corpuscles is simply made, all that is necessary being to put up two series of test-tubes each containing about 5 c.c. of a range

of dilutions of NaCl in water, and to add a drop of blood from the patient under investigation to each of one series of tubes and a drop of normal blood to each of the other series. It is not necessary to wash and centrifuge the red cells; the whole blood may be taken. The normal control series should never be omitted. The range of dilutions should extend from 0·8 per cent. to 0·2 per cent., and there should be a difference of not less than 0·05 per cent. between each dilution. The solutions can be rapidly made by placing in one burette distilled water, and in a second burette a 1-per-cent. solution of NaCl in water. Then into the first tube run 4 c.c. of the salt solution and 1 c.c. of water, giving 0 8 per cent. NaCl; into the following test-tube put $\frac{1}{2}$ c.c. less of salt solution and $\frac{1}{2}$ c.c. more of water, and so on until a strength of 0·2 per cent. NaCl is reached. Each tube should be thoroughly shaken after the dilution is made, and again after the blood has been added. Finally, the tubes are allowed to stand at room temperature until all the intact corpuscles have settled to the bottom.

Parasites in the blood.—We have already described the way to look for **filaræ** in the blood (p. 205). Of this parasite there are several species which are the embryos of corresponding parental forms. The embryos live free in the blood (Fig. 54); the parental forms are found in the tissues and lymphatics. In one species the embryos are present in the blood during the night only (*F. bancrofti*, Fig. 53), in another only by day (*F. diurna*). In yet another form (*F. perstans*) they are always present. The blood in suspected cases should therefore be examined both during the day and during the night. For the diagnosis of the different species special works must be consulted,

but the chief points to attend to are (1) the time when the parasites are present in the blood: (2) the nature of their movements: (3) whether or not they possess a sheath; (4) the shape and character of their extremities.

To recognise the **parasite of malaria**, proceed as follows:—

Prepare a film of fresh blood in the manner already described (p. 204). Examine with a $\frac{1}{12}$ immersion lens and rather feeble illumination. Look in the red corpuscles for the presence of small black specks, often rod-like and showing slow movements of translation. These are surrounded by clear areas One may also see in the centre of some of the red cells clear amœboid areas which show no pigment. Rosette forms may be visible. These forms of the parasite are always present in cases of malaria which have not had quinine. Other varieties are only met with in some chronic or malignant cases. Of these there are two chief forms—(1) the crescentic, which are not found except in cases of malignant infection, and (2) the flagellated (Plate 16, A, Figs. 6, 7, 8). These are easily recognized. The crescentic bodies are highly refractile, rather longer than a red blood-corpuscle, and about 2μ in diameter. Particles of pigment may be recognized in the parasite and also in some of the ordinary leucocytes.

Parasites are most abundant in the blood about eight to twelve hours after the rigor, and examination for them demands some care and patience. The quartan form of the parasite (Plate 16) is distinguished from the tertian (1) by being smaller in size, (2) by its pigment granules being darker, (3) by its showing fewer segmenting forms.

In addition, stained preparations of the blood should invariably be made, preferably with

Leishman's stain in the manner described above (p. 209). The appearances of the stained parasite are shown in Plate 16.

Trypanosomata may be looked for in fresh blood, or they may be fixed and stained in blood-films. As the parasites are often few, it is important, in doubtful cases, to centrifuge the blood before examining it, when most of the trypanosomes will be found collected along with the leucocytes in a thin, pale-coloured layer. It is not easy in all cases to obtain a sufficient quantity of blood to fill an ordinary centrifuge tube, therefore one may employ a small tube similar to Widal's pipette. The chamber is half filled with blood, and into the remaining space is drawn a solution made by dissolving 1 grm. of sodium citrate in 100 c.c. of normal saline. The chamber is thereafter sealed off in the flame of a spirit-lamp and placed in the hæmatokrit arm of the centrifuge. After revolving for ten or fifteen minutes a distinct white ring is formed. The chamber is then scratched with a file about ½ cm above the white ring, the clear fluid removed, and the white ring itself transferred, with the aid of a capillary pipette, to a cover-slip, and examined on an ordinary slide The examination should be made with a $\frac{1}{6}$-in. objective and an eyepiece with a fairly wide field; and before a negative conclusion is arrived at, not less than a quarter of an hour should be spent in searching the specimen. The parasite is more readily recognised by its movements than by its form. In doubtful cases it is important to centrifuge the blood imperfectly, so as to precipitate most of the cells, thereafter to remove the supernatant fluid with a pipette and to centrifuge this fluid a second time, when the deposit that subsides will contain nearly all the trypanosomes.

Fixed preparations can be made by staining blood-films, prepared in the usual way, with Leishman's stain (p. 209) For the study of finer structural details the following more elaborate method has been devised by Dutton and Todd :—

Fix the blood-film by exposure, for five to fifteen seconds, to the vapour of a mixture of equal parts of glacial acetic acid and 2-per-cent. solution of osmic acid. Thereafter stain with one or other modification of Romanowsky's stain (Appendix, 25) for three to six minutes, the slide to be stained being placed face downwards in the solution. When the staining is complete, wash with water, dry in the air, and mount. Preparations should be kept unmounted, as they are apt to fade

quickly in balsam. In such preparations the trypanosome appears as a spindle-shaped body, along one border of which a narrow and delicate "undulating membrane" may be observed. Within the body two masses of chromatic substance are visible. Of these the larger, which is the nucleus, lies more anteriorly. The smaller, known as the micronucleus, or blepharoplast, or centrosome, is generally situated near the posterior or rounded end of the trypanosome, and in its neighbourhood a vacuole is often found. From the centrosome a fine filament emerges, and, passing along the free border of the undulating membrane, is continued anteriorly as a flagellum. On an average, the length of the human trypanosome may be reckoned at 17 to 25 μ (including the flagellum, which averages 6 or 7 μ); the breadth is from 1·5 to 2 μ.

Trypanosomes can also be found, in the later stages of the disease, in the cerebro-spinal fluid. The latter is easily obtained by lumbar puncture (p. 536), and is often more turbid than usual in consequence of the presence of an excess of leucocytes. The specimen is best centrifuged and examined fresh, as the organism does not stain so satisfactorily in cerebro-spinal fluid as it does in blood. In cases where there are enlarged glands these may be punctured, and a drop of fluid withdrawn and examined for trypanosomes, which may be fairly abundant In many cases where no trypanosomes can be found in the blood they may be readily demonstrated in fluid from glands. In most cases of the disease the organisms are never numerous either in the blood or the spinal fluid, and a negative diagnosis in such cases may only be justifiable after inoculation experiments have failed.

Leishman-Donovan bodies are small round or oval bodies with a diameter of about 3 μ, which are found in splenic blood in certain fevers. They develop, outside the body, into large flagellated forms. They can be stained by Leishman's or Romanowsky's methods, and are then found to possess two chromatic masses, a large and a small, the latter of which is often associated with a vacuole. The parasites appear to occupy the interior of endothelial cells or of leucocytes. By taking 10 c.c. of blood from a vein into citrated Locke's solution and centrifuging it according to the method of Young and Sant the parasites can be demonstrated in a high proportion of cases in films and obtained in culture in a still higher proportion. It should never be necessary to resort to spleen puncture, an operation which is by no means devoid of risk.

Spectroscopic Examination

(Plate 17)

In cases of suspected carbon monoxide poisoning, the diagnosis may be established by spectroscopic examination. Some blood is obtained by pricking the thumb and squeezing two or three drops into several c.c. of distilled water. The solution has a cherry-red colour. Place some of it in a thin, flat glass tube, and examine with a hand spectroscope. Direct the instrument, as in all such examinations, towards a white cloud, and not towards the sun. Two bands (Plate 17) are seen (bands of carboxy-hæmoglobin) occupying very much the position of the oxyhæmoglobin bands. They are distinguished from the latter by the fact that the addition of a few drops of ammonium sulphide produces no alteration in them.

Estimation of Coagulation Time

The **coagulability** of the blood can be estimated with a fair degree of accuracy by means of Wright's coagulometer. The instrument consists of a series of fine tubes of equal calibre which are kept immersed in water at body temperature. Blood is drawn into each of the tubes at definite intervals, and, after the lapse of varying periods of time, one blows down the tubes in succession. If the blood can no longer be blown out, coagulation has occurred. The interval between the filling of the tube and the occurrence of coagulation is known as the *coagulation time*.

At a temperature of 37° C. the coagulation time of a healthy individual is about four minutes.

Estimation of Bleeding Time

Bleeding time is determined by stabbing the ear with a sharp sterile needle and blotting off the drop of blood every 30 seconds until it ceases to ooze from the puncture. The blots are recorded in series along a strip of blotting-paper and subsequently counted. The normal bleeding time is 4 to 5 minutes. It is increased in some forms of purpura.

Blood Grouping

With the increasing recognition of the importance of blood transfusion, the determination of the compatibility of two bloods becomes of the greatest importance. The sera of some persons agglutinate the red cells of others, forming clumps which are readily seen under the microscope and may be obvious to the naked eye. This agglutination of red cells may progress in vivo to actual hæmolysis, so that a patient transfused with blood of a wrong group may show dyspnœa, præcordial pain, and hæmoglobinuria, and may even die if sufficient blood be given.

Such ill-effects can be avoided by careful grouping. Individuals can be separated into four groups, according to the interaction of the sera of the one and the red corpuscles of the other. The interaction may be expressed as follows (Moss):—

Group I cells are agglutinated by sera of II, III and IV
" II " " " " " " III " IV
" III " " " " " " II " IV
" IV " " not agglutinated by any sera.

In Jansky's classification, which is widely used on the Continent, Groups II and III are the same as in the Moss classification, but Groups I and IV are transposed.

A third method of classification employs letters instead of numbers to designate the blood groups.

Group IV (Moss) is Group O, Group III is B, Group II is A, and Group I is AB. The letters correspond to the two cell agglutinogens A and B.

For purposes of grouping, stock specimens of sera II and III are necessary. A drop of serum II is placed at one end of a clean slide and a drop of serum III at the other end. These two drops are diluted with one drop of citrated saline, and a drop of the blood to be tested is added to each and mixed with a platinum loop. Should agglutination take place the corpuscles can be seen to clump with the naked eye. The observations should be confirmed by placing a cover-slip over each drop and examining under a microscope, using the $\frac{2}{3}$-in. objective. If agglutination occurs with serum III alone, the blood is group II, if with II alone it is group III. If no agglutination occur at all, the unknown blood belongs to group IV, and if with both to group I

It is preferable and, in cases of long standing grave anæmia, essential, that both donor and recipient should be of the same group. In cases of emergency IV, being a universal donor, may be used for any case.

In grouping a patient a sample of the serum should always be taken as well as the red cells. Having obtained the patient's blood group from the red cell examination and sent for the appropriate group donor, any possibility of error should be excluded by cross-grouping, and two preparations should be put up, one of the patient's serum with the donor's red cells, the other of the patient's cells with the donor's serum. There must be no agglutination in either preparation.

For **sedimentation rate** and **sternal puncture**, see p. 571.

SPECIAL CHEMICAL METHODS OF BLOOD INVESTIGATION

1. **Van den Bergh's reaction.**—The diazo reaction of bilirubin has been introduced by A. A. Hijmans van den Bergh as a test for this substance

in serum. It appears that the bilirubin exists in serum in a different state when its presence is due to excessive breakdown of red corpuscles from that when its presence is due to obstruction. In the first case the diazo reaction is only given after the proteins have been precipitated with alcohol (indirect reaction). This difference may be used to distinguish hæmolytic from obstructive jaundice. Van den Bergh's original method is as follows:—

For the test, as ordinarily carried out, about 3 c c. of serum may be required, although less will suffice after some practice has been obtained The blood is taken from a vein in the usual way into a dry test-tube, allowed to clot, and the separated serum is then removed by a pipette. It is best to begin to practise the test on a case of fairly intense icterus.

Apparatus and Reagents Required

(1) A few test-tubes of ordinary size.

(2) Freshly prepared Ehrlich's diazo reagent. This consists of two solutions, each of which keeps well, but the mixture of the two must only be made immediately prior to the test. The two solutions are made up in the following proportions :—

A.	Sulphanilic acid . . .	1 grm.
	Concentrated HCl . . .	15 c c.
	Distilled water . . .	1,000 c c.
B.	Sodium nitrite . . .	0 5 grm.
	Distilled water . . .	100 c.c.

The diazo reagent consists of a mixture of these two solutions in the proportion of 25 c.c. of solution A to 0·75 c c. of solution B.

(3) A graduated 1 c c. pipette.

(4) Absolute alcohol (96-per-cent.).

(5) A centrifuge and centrifuge tubes.

The test is then carried out as follows : To 1 c c. of the serum, in a small test-tube, van den Bergh adds 0·25 c.c. of freshly prepared diazo reagent (better results are frequently obtained by adding 1 c c of the reagent). One of three events may now occur :—

1. *An immediate (direct) reaction.*—This begins instantly and is *maximal in* 10-30 *seconds.* The colour reaction

obtained is a bluish-violet, of intensity depending on the amount of bilirubin present.

2. *A delayed reaction.*—This begins only after 1–15 minutes, or even longer, and consists in the development of a reddish coloration, which gradually deepens and becomes more violet.

3. *A bi-phasic reaction.*—In this a slight reddish colour appears immediately (10–30 seconds), which after a minute or much longer time is seen to deepen gradually and become more violet.

The modified method of Thannhauser and Andersen gives perhaps more satisfactory results.

Reagents Required

A. Sulphanilic acid	1 grm
Concentrated hydrochloric acid .	10 c.c
Distilled water . . .	to 200 c.c
B. Sodium nitrite	0·5%

When required for use, mix 25 c.c. of A with 0·5 c.c. of B, making the diazo reagent.

Direct reaction (without precipitation of proteins).—In a centrifuge tube place 2 c.c. of clear serum, 1 c.c. diazo reagent, mix, and allow to stand for 5 minutes. Add 5·8 c.c. absolute alcohol and 2·0 c.c. saturated ammonium sulphate solution. Shake well and centrifuge. To 0·9 c.c. of the clear supernatant fluid add 0 1 c.c. of concentrated hydrochloric acid. If the test is positive a blue colour develops.

Indirect reaction (after precipitation of proteins).—In a centrifuge tube place 2 c.c. of serum, add 4 c.c. of absolute alcohol, shake, and centrifuge. To 1 c.c. of the supernatant fluid add 0·25 c.c. of the diazo reagent, and stand for 5 minutes; then add 0·55 c.c. of absolute alcohol and 0·2 c.c. of concentrated hydrochloric acid. If the test is positive a blue colour slowly develops.

Normal serum gives a faint indirect reaction.

2. Estimation of urea in blood.—The *reagents required* are:—

0 6% acid potassium phosphate.
Coarsely ground soya bean.
Caprylic alcohol.
Anhydrous potassium carbonate
$\frac{1}{100}$ normal hydrochloric acid
$\frac{1}{100}$ normal sodium hydroxide.

Methods.—In tube B (Fig 56) place 2 c c. of blood, 2 c c. of the acid potassium phosphate solution, a knife-point of the soya bean powder ground to an emulsion with about 1 c.c. of water, and 3 drops of caprylic alcohol Incubate this tube for 20 minutes at about 40°.

Into tube C place 10 c.c. $\frac{N}{100}$ hydrochloric acid.

Tube A contains about 20 c.c of dilute sulphuric acid.

When the incubation of B is complete, connect the tubes as shown and connect the outlet tube of C to a strong water-pump. Start the current of air gently, and pour about 4 grm anhydrous potassium carbonate into B, reinserting the stopper at once. Run a gentle current for about 10 minutes,

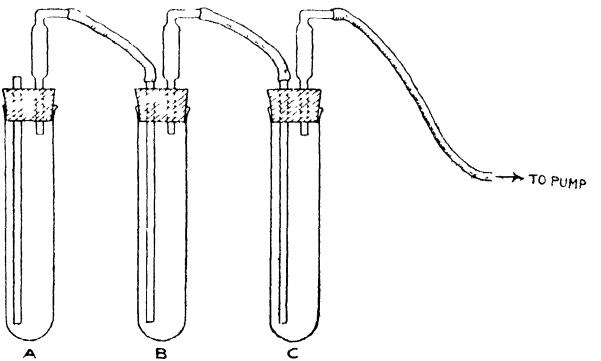


Fig. 56.—Apparatus for estimation of urea in blood.
(For references, *see* text.)

and then run for 50 minutes as fast as possible without frothing over. The urea in the blood is converted into ammonium salts by an enzyme in the soya bean; this ammonia does not escape, as the solution is kept acid by the phosphate. When the fluid in B is made strongly alkaline the ammonia is set free and carried by the air-current to be caught by the acid in C. The acid in A traps any ammonia in the air. After the current of air has run for an hour in all, disconnect from the pump, wash down the entry tube of C with distilled water inside and out, and titrate the remaining acid with $\frac{N}{100}$ sodium hydroxide, using methyl red as indicator.

If a c.c. of alkali are used $(10-a)$ c.c. of acid have been neutralized.

Since 1 c.c. of $\frac{N}{100}$ acid is neutralized by 0 14 mg. of ammonia nitrogen,

and 0·30 mg. of urea contain 0 14 mg. of nitrogen,

Then 2 c.c. of blood used contain $(10-a) \times 0{\cdot}30$ mg. of urea.

100 c.c. „ „ „ $(10-a) \times 15$ mg. of urea.

It is preferable that the ends of the entry tubes should be blown into bulbs perforated with about 6 holes of the size that would be made by a needle.

If it is suspected that the blood contains excessive amounts of urea, 1 c.c. only should be used.

This method may be used for the estimation of urea in urine; it is more accurate than the hypobromite method. 0 5 or 1·0 c.c. of urine is used, 5 c.c. of acid phosphate solution and $\frac{N}{10}$ acid and alkali instead of $\frac{N}{100}$.

Normal blood contains from 0·02 to 0·05 per cent. of urea. With moderate reduction of renal efficiency normal values are usually found. With more severe degrees of reduction the blood-urea begins to rise, and may reach 0·6 per cent. before death. Patients with blood-urea above 0·1 per cent., not due to a temporary condition which can be relieved, rarely live more than a year.

3. **Estimation of sugar in blood.** (1) **Mac-Lean's method.**—*Solutions required:*

(*a*) Sodium sulphate 15-per-cent. To this add 0·1 c.c. of acetic acid per 100 c.c. immediately before use.

(*b*) Dialysed iron. That supplied by the British Drug Houses is satisfactory.

(*c*) Copper solution. Dissolve 12 grm. of potassium bicarbonate with gentle heat in 70 c.c. of distilled water; add 8 grm. of anhydrous potassium carbonate. Dissolve 0·35 grm. of copper sulphate crystals in a few c.c. of water in another vessel and add to the bicarbonate solution without waiting for the potassium carbonate to dissolve completely. When effervescence has finished, complete the solution of the carbonate by heating. Add 0 05 grm. of potassium

iodate and 0 5 grm. of potassium iodide, shake, make up to 100 c.c., filter. This solution should not be used for two days, after this it keeps indefinitely. It must be standardized by taking 2 c.c. in 10 cc. of acid-sodium-sulphate solution and adding 2 c.c. of 75-per-cent. hydrochloric acid. One minute after effervescence has subsided shake and titrate with $\frac{N}{400}$ sodium thiosulphate as described later. 2 c c. should require about 11 c.c. of thiosulphate. This standardization should be repeated from time to time.

(*d*) $\frac{N}{400}$ sodium thiosulphate solution made by diluting 5 c.c. of $\frac{N}{10}$ thiosulphate to 200 c.c. The thiosulphate solution should be kept in blue bottles in the dark and the $\frac{N}{10}$ solution standardized from time to time.

(*e*) 1-per-cent. solution of soluble starch.

(*f*) 75-per-cent. hydrochloric acid solution. (75 c c. concentrated hydrochloric acid diluted to 100 c.c.).

Rubber tubing is wound round the patient's finger to produce congestion, a little powdered potassium oxalate is spread on the place to be punctured, and a needle stab made in the middle of the oxalate patch just above the root of the nail. 0 2 c.c. of blood are allowed to run into a 0 2 c.c. pipette,* which is placed in contact with the side of the drop and held horizontally. If too much blood is obtained it may be brought down to the mark by tapping on the finger-nail. When the blood reaches the mark, any that remains on the point is wiped away.† The blood is added to 23·8 c.c. of acid-sodium-sulphate solution which has been run from a burette into a Duro glass flask, and the pipette washed out by sucking up and blowing out the solution. The mixture is heated until it begins to boil, the flask being fitted with a rubber stopper through which passes a glass tube

* Obtainable, with the rest of the apparatus, from Messrs. Hawksley & Sons.

† It is very important that the pipette should be entirely free from grease; it should therefore be washed out, after each experiment, with a hot mixture of sulphuric acid and potassium bichromate. After exposure to this mixture, it is washed out with distilled water, followed by alcohol and ether, and then dried with the aid of a blowpipe and gentle heat.

gradually drawn out to a capillary point. This allows the escape of air and practically prevents the loss of fluid. The flask is cooled under the tap, and 1 c.c. of dialysed iron is added with constant shaking. The fluid is filtered and 20 c.c. of filtrate used for sugar estimation, or if a high blood-sugar is expected, 10 c.c. of filtrate made up to 20 c.c. with acid sodium sulphate.

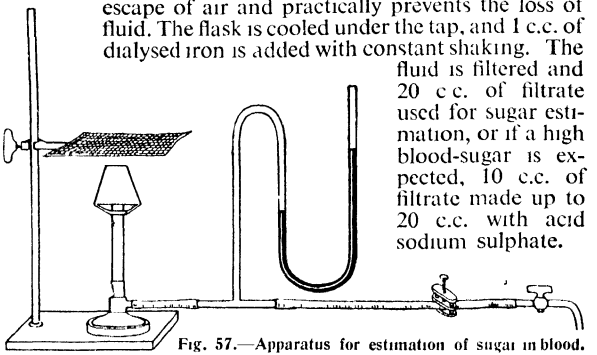


Fig. 57.—Apparatus for estimation of sugar in blood.

Two c.c. of solution No. 3 are added to the filtrate taken, and it is heated over a flame which will bring the mixture to brisk boiling in 100 secs. In order to ensure a flame of the right height, the arrangement in Fig. 57 is used. By previous trials it is found what pressure of gas, as shown by the manometer, will bring 20 c.c. of acid-sulphate solution to a brisk boil in 100 secs, and in subsequent work the screw clip is adjusted until the manometer records this pressure; the same flask, gauze, tripod, and burner must always be used.

Boiling is continued for 6 minutes after the liquid begins to boil vigorously all over. The flask is cooled under a tap, 2 c.c. of 75-per-cent. HCl are added, and it is shaken gently until effervescence has finished, and at intervals for a minute after. The fluid is titrated with $\frac{N}{400}$ sodium thiosulphate until the yellowish colour of iodine has almost disappeared, 2 or 3 drops of soluble starch solution added, and the titration continued until the blue colour disappears.

The number of c.c. of thiosulphate used is subtracted from the number used by 2 c.c. of solution 3 alone, and the percentage of sugar in the blood is calculated from the table. Thus, if the figure obtained on titration was 8·98 c.c. and 2 c.c. of solution 3 alone required 11·05 c.c., the difference, 2·07 c.c., represents the sugar present. From the table

below, 2.07 c.c. of thiosulphate = 0·18 mg. of sugar; therefore $\frac{20}{25} \times 0.2$ c.c. of blood contain 0·18 mg. of sugar; therefore 100 c.c. of blood contain 0 112 grm

In all methods of blood-sugar estimation the filter-papers must be free from carbohydrate. Whatman's No. 1 are suitable.

Table showing amount of glucose (in mg.) and its percentage equivalent to $\frac{N}{400}$ *sodium thiosulphate solution when 20 c.c of blood filtrate are used*

N/400 Thiosulphate c.c.	Glucose mg.	%	N/400 Thiosulphate c.c.	Glucose mg.	%
0 12	0 03	0 018	2 61	0 22	0 137
0 25	0 04	0 025	2 74	0 23	0 143
0 38	0 05	0 031	2 86	0 24	0 150
0 50	0 06	0 037	2 99	0·25	0 156
0 62	0 07	0 043	3 11	0·26	0 162
0·73	0 08	0 050	3 24	0 27	0 168
0 86	0·09	0 056	3 36	0 28	0 175
0 99	0 10	0 062	3 49	0 29	0 181
1 13	0 11	0 068	3 61	0 30	0 187
1 26	0 12	0 075	3 74	0 31	0 193
1·39	0 13	0 081	3 87	0 32	0 200
1 53	0·14	0·086	3·99	0 33	0·206
1 67	0·15	0 093	4 12	0 34	0 212
1 80	0 16	0 100	4·24	0 35	0 218
1 94	0 17	0 106	4 37	0 36	0 225
2 07	0 18	0 112	4 49	0·37	0 231
2 22	0 19	0 118	4 62	0·38	0 237
2·35	0 20	0·125	4·74	0·39	0·243
2 49	0 21	0·131	4·87	0 40	0·250

(2) **Method of Folin and Wu.**—This is the simplest and most rapid method if a colorimeter is available. The *solutions required* are:—

10-per-cent. sodium tungstate; $\frac{2}{3}$ normal sulphuric acid. Alkaline copper solution:—

Dissolve 40 grm. of anhydrous sodium carbonate in 400 c.c. of water. Add 7·5 grm. of tartaric acid and dissolve; then add 4·5 grm. of copper sulphate, weighed accurately, and dissolve. The solution of

each solid should be complete before the addition of the next. Make up to 1,000 c.c. Test for absence of cuprous salts by mixing 2 c c. with 2 c.c. phosphomolybdic acid reagent; the deep blue colour should almost vanish.

Phosphomolybdic acid reagent :—

Dissolve 35 grm. of molybdic acid and 5 grm. of sodium tungstate in a large flask, add 200 c.c. of 10-per-cent. sodium hydroxide, 200 c.c of water, and boil until ammonia no longer comes off. Cool, dilute to 350 c.c , and add 125 c.c. of 85-per-cent. phosphoric acid. Make up to 500 c.c.

Stock glucose solution :—

1-per-cent. pure glucose dissolved in 2 5-per-cent. benzoic acid.

Weak glucose standard :—

Make 1 c.c. of the stock glucose up to 100 c c. with 2·5-per-cent. benzoic acid solution. Contains 1 mg. of glucose in 10 c.c.

Strong glucose standard :—

Make 2 c.c. of the stock glucose up to 100 c.c. with benzoic acid solution. Contains 2 mg. of glucose in 10 c.c.

Precipitation of proteins.—Place 2 c.c. of blood, which has been prevented from clotting by the addition of a small amount of potassium oxalate, in a small flask. Add 14 c.c. of distilled water, shake gently to cause hæmolysis. Then add 2 c.c. of sodium tungstate solution and 2 c c. of $\frac{1}{3}$ normal sulphuric acid, shaking during the addition. Cork the flask and shake violently. After 15 secs., filter.

Reduction of the copper solution.—In a tube which has a bulb holding just under 4 c.c. at its end, and above this a neck about $\frac{1}{4}$ in. in diameter, place 2 c.c. of the filtrate and 2 c.c. of the copper solution. In two other tubes (I) and (II) place 2 c.c. of the weak (I) and strong (II) glucose solutions; add 2 c.c. of copper solution to each. Place the tubes in a boiling water bath for 6 minutes; cuprous oxide is formed.

Reduction of the phosphomolybdic acid reagent by the cuprous oxide and comparing the depths of colour produced.—Cool the tubes in water for two minutes. Add to all three 2 c.c. of the phosphomolybdic acid reagent. Stand two minutes for the solution of the cuprous oxide to be completed; a deep blue colour develops. Dilute to 25 c.c. (it is convenient to have the tubes graduated at 25 c.c.). Mix well and compare

the depths of colour in the tube containing the blood-filtrate with that in tube (I) or (II), which it most nearly resembles in depth. If tube (I) is used as standard and the reading of the standard in the colorimeter is 20, and if the unknown is a: Since the depth of colour is proportional to the amount of glucose present,

2 c.c. of filtrate contain $\frac{20}{a} \times \frac{2}{10}$ mg.

$\therefore$ 20 c.c. „ „ $\frac{20}{a} \times 2$ mg.

$\therefore$ 2 c.c. of blood „ $\frac{20}{a} \times 2$ mg

$\therefore$ 100 c.c. „ „ $\frac{20}{a} \times 100$ mg.

If tube (II) was used for the comparison, 100 c.c. of blood contain $\frac{40}{a} \times 100$ mg.

The precipitation of the blood should be done within a few minutes after it is drawn, as the glucose is destroyed on standing.

The blood of normal persons, taken when they have fasted for some hours, contains 0·09 to 0·12 per cent. of glucose. After a meal containing carbohydrate this may rise to 0·15 per cent. Usually only traces of glucose appear in the urine when the blood-sugar is below 0·17 per cent., the "renal threshold."

In diabetes mellitus the blood may contain much larger quantities of glucose, up to 0·6 per cent.

CHAPTER VI

THE RESPIRATORY SYSTEM

I. ANATOMY

(Plates 7, 8, 9, 10)

THE following anatomical facts must be borne in mind when the lungs are examined:—

1. **Borders of the lungs. Right lung.**—The anterior border passes forwards, downwards, and towards the middle line from the apex, which, situated at the level of the neck of the 1st rib, corresponds posteriorly with the 7th cervical spine. Behind the sternum, at the level of the 2nd rib, it has nearly reached the middle line, and passes directly downwards to the level of the junction of the 6th costal cartilage with the sternum, where it turns rather abruptly to the right, to pass outwards as the lower border. The lower border meets the right parasternal line at the level of the upper border of the 6th rib, the mammary line also at the level of the 6th rib, the axillary lines at the 7th and 8th ribs, the scapular line at the 10th rib, and at the side of the vertebral column reaches as far as the 10th interspace or 11th rib.

Left lung.—From the apex to the level of the 4th costal cartilage the anterior border passes in a direction which corresponds with that of the right lung. At this point it bends rather suddenly outwards, thereby leaving part of the anterior surface of the heart exposed, and passes in an arched line outwards and downwards, to reach the 6th rib a little externally to the parasternal line. From this

point the lower border passes backwards along a line corresponding to, but a little lower than, that of the lower border of the right lung. The lower borders of both lungs are convex towards the abdomen. In forced respiration they may vary in level to the extent of 2 or even 3 in., according to the phase of the respiratory cycle. In quiet respiration the difference between the extremes is only about 1 cm.

2. **Lobes of the lungs.**—It is often important to know the limits of the individual lobes of the lungs. This may be done by drawing a line from the 2nd thoracic spine to the 6th rib in the mammary line; this corresponds to the upper border of the lower lobe. A second line, drawn forwards on the right side from the centre of this line to meet the sternum at the level of the 4th costal cartilage, will mark the boundary between the upper and middle lobes.

Obviously, therefore, the greater part of each lung, as seen from behind, is composed of the lower lobe, only the apex belonging to the upper lobe; while the middle and upper lobes on the right side, and the upper lobe on the left, occupy most of the area in front. In the axillary regions, parts of all the lobes are accessible.

The **bifurcation of the trachea** corresponds in front with the lower border of the manubrium sterni; behind, with the disc between the 4th and 5th thoracic vertebræ.

The **reflected pleural sacs** reach decidedly lower than the inferior borders of the lungs, whose limits they overstep for about 2 in. in the mammary, nearly 4 in. in the midaxillary, and 1½ in. in the scapular lines. The sinus thus formed lies on the left side above the resonant stomach cavity, and therefore, should it become distended with fluid, as

in cases of hydrothorax, a dull area will be discovered at a part where the healthy percussion note is tympanitic. The anterior reflection of the left pleura below the 4th rib is considerably nearer the middle line than the anterior border of the left lung; hence in emphysema, when the lung presses forwards into this available space, the area of absolute cardiac dullness is greatly encroached upon.

With reference to the correspondence of points in front and at the back, Quain gives the following relations as existing during expiration:—

"The upper margin of the sternum is on a level with the disc between the 2nd and 3rd dorsal vertebræ; the junction of the manubrium and body is opposite the 5th dorsal vertebra; and the xiphi-sternal articulation generally corresponds to the lower part of the 9th dorsal vertebra."

The **scapula** is a useful landmark posteriorly. Its median angle, when the arms hang by the side, is generally on a level with the disc between the 1st and 2nd thoracic vertebræ, the root of the spine with that between the 3rd and 4th thoracic vertebræ, and its inferior angle with the body of the 8th thoracic vertebra.

In reference to the ribs, the median angle of the scapula just covers the 2nd rib; the inferior angle reaches as low as the 7th interspace or 8th rib.

The 12th rib cannot always be felt. It is not safe, therefore, to count the ribs from below upwards.

For convenience in description, the thorax is mapped out into regions, as follows:—

1. **Three central regions anteriorly.**

Suprasternal, from the cricoid to the upper border of the manubrium.

Superior sternal, from the upper border of the manubrium to the level of the 3rd sterno-costal articulation.

Inferior sternal, from the 3rd sterno-costal articulation to the lower end of the sternum.

These three regions are bounded laterally by the lateral sternal lines and their upward continuations.*

2. **Five antero-lateral regions** on each side.

Supraclavicular, bounded above by an oblique line from the side of the cricoid to the outer end of the clavicle, below by the clavicle.

Clavicular, composed of the area occupied by the clavicle.

Infraclavicular, bounded above by the clavicle, below by a horizontal line at the level of the 3rd sterno-costal articulation.

Mammary, from the lower edge of the infraclavicular area to the level of the 6th sterno-costal junction.

Inframammary, below that level.

These regions extend outwards to the anterior axillary line.

3. **Two lateral areas on either side.**

Axillary, } meeting each other at the
Infra-axillary, } level of the 6th rib.

4. **Four regions at the back on either side of the spine.**

Suprascapular.

Scapular, subdivided into supra- and infra-spinous.

Infrascapular, and

Interscapular.

The positions of the dorsal regions are sufficiently defined by their names.

* Sometimes the sternal regions are classified as "episternal" and 'xiphisternal."

II. INSPECTION

Inspection determines—

A. Form of chest.

1. Healthy.
2. Symmetrical chests with features indicating proclivity to disease. — The alar chest. The flat chest.
3. Symmetrical chests with features indicating past disease. — The rachitic chest. The pigeon breast. Harrison's sulcus.
4. Symmetrical chests with features indicating present disease. — The barrel-shaped chest. Bilateral retraction.
5. Unilateral changes. — Enlargement. Diminution.
6. Local changes. — Bulging. Retraction. Funnel-shaped depression.

B. Movements of chest.

1. Respiratory.
   (1) Rate.
   (2) Rhythm.
   (3) Type.
   Character (*see also* Chap. II). — Amount of expansion. Unilateral fixation. Local lagging. Local indrawing and bulging.
2. Non-respiratory. Pulsations (Chap. IV).

A. Form of the Chest

This depends partly on the curvature and obliquity of the ribs, partly on the curves of the spinal column. The curvature of the sternum results from the relations of these factors.

When the ribs are normally curved, the more horizontally they lie the more nearly does a cross-section of the chest approach the form of a circle, the wider are the intercostal spaces, and the more obtuse does the subcostal angle become; whilst, on the contrary, increasing obliquity of the ribs leads to

narrowing of the intercostal spaces, to increasing ellipticity of the cross-section of the chest, the major axis lying transversely and the minor axis in an anteroposterior direction, and at the same time the subcostal angle becomes more acute. In a healthy male the angle is about 70°, in a female about 75°. The variations may amount to 10° above or below these averages. When there is lateral curvature of the spine, the chest is rendered asymmetrical; when the spine is unduly concave forward, other changes are produced, which will be dealt with subsequently.

1. The **ideal healthy chest** will conform to the following description: It is bilaterally symmetrical, its contours are smooth, it has no deep hollows, and at most shows only a slight recession below the clavicles. In cross-section it is an ellipse, broader from side to side than from front to back in the proportion of about 7 to 5; its general shape is ellipsoidal, with the longest axis vertical. In children the cross-section is more nearly circular.

The sternum, which is convex from above downwards when viewed from the front, lies at the bottom of a shallow groove known as the sternal furrow, formed by the pectoral muscles on each side. The junction of the manubrium with the body of the sternum exhibits a slight angular projection (the sternal angle, or angle of Louis), sometimes visible, almost always palpable. The sternal furrow ends below, at the level of the 7th costal cartilage, in the infrasternal depression. A slight hollow below the clavicle marks the separation between the divisions of the pectoralis major; it should not be deep, and ought only to be distinct when the muscle is made to contract. A second hollow, which is much more distinct, separates the pectoralis from the deltoid. This fossa lies farther from the middle line, and is

known as the infraclavicular (or Mohrenheim's) fossa. It becomes very marked in many cases of pulmonary tuberculosis.

The shape in the mammary regions depends greatly on the degree of development of the mammary gland and on the amount of subcutaneous fat. In the adult male the nipple is usually about 4 in. from the middle line, in the 4th intercostal space.

In actual practice it is very rare to find a chest which is perfectly symmetrical. Generally the right side is rather more capacious than the left, the right clavicle is tilted more than the left, and the spinal column almost always has a slight degree of lateral curvature. In inspection of the chest the examiner should first look at it from the front, then from the side, thereafter from the back, and, finally, he should look over the shoulders from behind and above, so as to see the profile of a horizontal section of the thorax. The last method is very useful in detecting lack of symmetry or unequal expansion on the two sides. The neck, especially as regards the manner in which it is set on the chest, and the epigastrium should be inspected at the same time as the thorax. In examining the chest from behind, it is important to note whether the vertebral borders of the scapulæ are unduly prominent, whether they are equidistant from the middle line, and whether their lower angles lie at the same level on either side.

Deviation from the normal form may affect either the whole of the thorax or localized parts of it. The **abnormal shape** of the chest as a whole may be grouped in three classes, according as it indicates merely a proclivity to lung disease, a history of former disease, or the existence of present disease. The first class contains the alar and flat chests; the second the rickety chest, the pigeon breast, and the

chest with Harrison's sulcus; the third the barrel-shaped chest, and the hollow or retracted chest. In these groups the changes affect both sides of the thorax, and so the symmetry remains undisturbed. In other instances the morbid conditions at work may lead to unilateral changes in the shape of the chest, one side having its volume either increased or diminished, and being otherwise deformed. Lastly, the chest may exhibit local deviations from the normal form, due generally to local disease.

2. **Symmetrical chests with features indicating proclivity to lung diseases ("phthinoid" chests).**—The two forms which belong to this class are the alar and the flat chest.

i. The **alar chest** is one where the vertebral borders of the scapulæ project unduly, and the shoulders droop. The cause of this appearance is to be found in the obliquity of the ribs, which makes the projection of their curves and angles in the horizontal plane more sharp, leads to a long and rather shallow thorax, and is associated with a long neck and prominent throat.

ii. The **flat chest** is due to a loss of the forward convexity of the costal cartilages, which become more or less straight. As a result, the sternum is less distant from the vertebral column than usual. The flat chest is often, but not always, associated with the alar form.

3. **Symmetrical chests with features indicating past disease** (and not seldom predisposing to pulmonary disease).

This group contains a number of forms, but only a few need be considered here.

i. **The rachitic chest.**—In rickets the bones are softer than normal, and so are more readily deformed by any applied force. From the nature of the disease

the part which yields most readily is that where the bone and cartilage meet, and therefore, when any cause prevents the free access of air to the lungs during inspiration, this part bends inwards before the pressure of the external air. A vertical groove is thus formed in this region, and persists even after the cause which first led to its production has disappeared. The section of a rachitic chest is shown in the accompanying figure, where the depressions situated at a little distance from either side of the sternum are easily recognized. When the rachitic condition is severe the line of least resistance becomes so weak that no unusual obstruction to inspiration is necessary in order to produce the grooves; the slightly lower air-pressure within the thorax, which is necessarily present during inspiration, being sufficient to lead to its formation (Fig. 58).

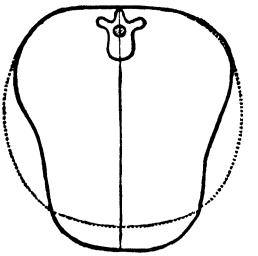


Fig. 58.—Cross-section of rachitic chest. (*Gee.*) The dotted line represents the normal outline for the same age.

ii. **The pigeon breast.**—Here, in consequence of some obstruction (often quite trivial) to inspiration at a time of life when the ribs are soft, they become straightened in front of their angles, where, owing to their smaller degree of curvature, they are most readily deformed by external pressure. The result is that the sternum becomes unduly prominent and projects beyond the plane of the front of the abdomen, so that there is a sharp angle at its lower end. At the same time the cross-section of the chest ceases to be elliptical, and approaches a triangular form, the angles being situated at the sternum in front, and at the costal angles behind (Fig. 59).

iii. **Harrison's sulcus.**—This is a transverse constriction which, beginning at the level of the xiphisternum, passes outwards and slightly downwards. It seldom reaches as far as the midaxillary line. This deformity is due to the same cause as the last, but either the obstruction has been slighter, or the bones have been more fully hardened.

The depression is therefore limited to the most yielding part of the chest, and this corresponds to the region where the cavity is widest. Lower down than the sulcus, the liver and other abdominal viscera had supported the chest-wall and so prevented it from being drawn inwards; whilst, higher up, the greater curvature of the ribs had enabled them to withstand the external pressure.

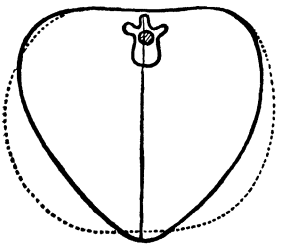


Fig. 59.—Cross-section of pigeon breast. (*Gee*) The dotted line represents the normal outline.

The three deformities just described are frequently found together in one individual, and cases of pigeon breast almost invariably exhibit a well-marked Harrison's sulcus.

4. Symmetrical chests with features indicating present disease.

i. In **emphysema** the increased volume of the lungs demands increased space for their accommodation. To provide this, the ribs are less obliquely set than usual, the spine becomes unduly concave forwards, and the sternum is much more arched than under normal conditions, while the angle of Louis becomes extremely prominent.

Since this increase of volume is accomplished by the mechanism which is provided for the expansion of

the chest in inspiration, the latter can only be effected by movements of the chest as a whole, and so the accessory muscles of respiration have to take the place of the intercostals, thereby becoming abnormally conspicuous, whilst the diaphragm has a considerable excess of work imposed upon it. The chest of emphysema is described as *barrel-shaped.*

Kyphosis may produce a form of chest which closely simulates that of emphysema.

ii. **Bilateral hollowing** is an extreme case of the flat chest already described, and is caused by the existence of phthisis.

5. **Unilateral changes in the shape of the chest which affect the whole of one side.** —These may result either in unusual bulging or in retraction.

Unilateral enlargement is due either to the presence of fluid or of gas in the pleura, or to increase in volume of one lung due to a tumour or to compensatory hypertrophy. The presence of fluid does not always cause enlargement of the affected side of the chest, although it often does so. Sometimes, indeed, that side is smaller. The explanation is not very obvious, but it must be recollected that the fluid is primarily accommodated by displacement of the lung, and only later, after the elasticity of the lung has been exhausted, will the contour of the thorax be altered.

Diminution of volume results from shrinkage of the lung. This may be caused by tuberculosis, or it may be the result of adhesions formed during an attack of pleurisy with effusion. Collapse of a lung from obstruction of the bronchi may produce a similar result.

Before connecting these changes with disease of the lungs or pleura, the observer must ascertain that

no *scoliosis* exists; for the rotation of the vertebræ in this condition leads to a deformity which, when inspected only from the front, it is almost impossible to discriminate from those just described. Scoliosis may, however, be induced by the retraction of a lung in a young subject. Abdominal disease must also be excluded.

6. **Local changes** affecting only part of either side.

i. **Bulging.**—In emphysema the apices may produce an unusual fullness above the clavicles, and in pleural effusion, especially when of a purulent nature, the interspaces which lie in the area of effusion often bulge considerably; these phenomena may accompany a general enlargement, or occur without it.

Tumours of various kinds, and disease of the heart, may be the cause of localized bulging.

ii. **Shrinking.**—In phthisis one or both apices are often contracted, and thereby a hollowing is produced above the clavicles. A similar condition occurs also in the infraclavicular regions, which may exhibit marked hollowing. This is particularly noticeable in Morenheim's fossa.

To detect either bulging or flattening (as well as diminished expansion) it is important to look tangentially along the chest either from above the shoulders or upwards from below. Both in phthisis and in other wasting diseases the interspaces are very sunken, and the ribs prominent, in consequence of the malnutrition of the muscles and subcutaneous tissues.

Not infrequently a local shrinking is due to adhesions of the pleura resulting from a former attack of pleurisy.

iii. A **funnel-shaped depression** is sometimes found in the lower part of the middle line of the thorax

in front. Sometimes it is congenital, or it may be developed in infancy with or without any obstruction to respiration being present. It extends in some cases as high as to the 3rd rib. A similar depression—though seldom of such magnitude—is found as a trade deformity in shoemakers.

B. Movements of the Chest

The movements of the chest during respiration also demand attention, and the rate of movement, its rhythm, its type, and its amount, must be noted.

1. The **rate** for an adult in health is about 18 or 20 respirations per minute, but there is a wide margin on either side of these figures. Increased rapidity may result from exertion, nervous excitement, fever, or defective aeration of the blood, whether this be due primarily to cardiac, pulmonary, bronchial, or laryngeal causes, or to some alteration in its oxygen-carrying power. It may also arise from the association of pain with all attempts at respiration, as in pleurisy and peritonitis, when the breathing becomes shallow, and must therefore be more frequent to make up for the slighter expansion.

The **ratio between respiration and the pulse** is important. In health it is about 1 to 4; in pneumonia respiration may occur almost as frequently as the pulse; in certain cases of narcotic poisoning the ratio may become 1 to 6 or 7.

2. The **rhythm** varies very considerably even in health, and if the act is performed consciously it may become very irregular. Hence it is important to study it when the patient is off his guard, as only then can accurate observations be made. Either inspiration or expiration may be unduly prolonged; the former being commonly associated with laryngeal or tracheal, the latter with bronchial or pulmonary diseases. A

peculiar type, where successive respirations gradually get deeper and deeper till a maximum is attained, and then fall off again until a pause of complete apnœa occurs, to be followed by another wave of gradually deepening and then diminishing respiration, is known as **Cheyne-Stokes breathing.** The pause may last for fully half a minute, though it is often shorter, and the whole cycle is usually completed in less than two minutes. It is very conspicuous when the patient who exhibits it is asleep, or is unconscious; but is apt to be overlooked if the patient is awake, and particularly if he is talking. Apart from completely typical Cheyne-Stokes respiration, various modifications, more or less nearly approaching it, occur.

There is another form of respiration which is often mistaken for Cheyne-Stokes breathing, but which is really different. In this form, instead of a gradual increase in the depth of respiration from the apnœic pause to the middle of the cycle, the deep breathing begins suddenly, and gradually diminishes, until the apnœic pause is reached, thereafter to recommence once more with full vigour. It is often observed in cases of meningitis.

3. **Type.**—Breathing may be more evidently performed by the upper part of the thorax; this is known as the **thoracic type of respiration.** It is found to a certain degree in women, but in its full development is either associated with paralysis of the diaphragm, or else is a result of its fixation from inflammatory causes or from increased abdominal pressure.

In men and young children the **diaphragm** and **abdominal muscles** play the most important part in respiration; and in cases where the intercostal muscles are paralysed, or where some inflammatory and painful condition, such as pleurisy or pleurodynia, exists

in the thorax, the breathing may be wholly abdominal in type.

In health the male type of respiration may be described as **abdomino-thoracic,** and the female as **thoracico-abdominal,** or almost purely thoracic.

The presence of **pain** or **dyspnœa** should always be inquired for, and its exact nature noted. (*See* Chap. II, p. 37.)

4. Regarding "**movement**" during respiration, the points to be noted are its amount, whether it is expansive in character, and whether it is similar, or different, on the two sides and over corresponding areas.

Amount of movement and expansion are by no means interchangeable terms; in emphysema the chest may move considerably, but there is little expansion.

In comparing the two sides it will often be found that deficient or absent movement betokens pleurisy with effusion, or non-expansion of the lung from consolidation or rigidity of its structure.

Local deficiency in expansion is frequently a very important indication of tuberculosis, or it may be due to lobar pneumonia, the former especially at the apices, the latter at the apex or base according to the situation of the disease.

Sometimes one part of the chest-wall lags behind the rest during inspiration. Any such lagging is important as suggestive of disease. The existence of any **indrawing** of the chest-wall or of the interspaces during inspiration, or of any **bulging** during expiration, must be noted. Both may occur physiologically, in which case the conditions are present over the whole chest, and are not very conspicuous, or they may result from pathological conditions, when they sometimes affect the whole thorax, at other times one side,

and yet at others only appear locally. Examples of inspiratory indrawing are found in obstruction of the larynx (general), or in blocking of some of the smaller bronchi (local). One of the best instances of localized expiratory bulging is seen at the apices of the lungs in advanced emphysema.

III. PALPATION

Palpation determines—

A. **Form of chest** (confirms or modifies the results of inspection, q.v.).

B. **Movements of chest.**
   1. Respiratory (*see also* Inspection).
   2. Pulsations (Chap. IV).

C. **Vibrations.**
   - Palpable pleuritic friction.
   - Palpable râles.
   - Vocal fremitus: Increased. / Diminished / Absent

D. **Tenderness.**

E. **Fluctuation.**

F. **Resistance of chest-walls to compression.**

Palpation takes note, first, of the form and movements of the thorax; second, of vibrations or tremors which are communicated to the hand ; and third, of the behaviour under pressure of any pain of which the patient complains. Under the first head inspection is supplemented; under the second, one learns something of the accompaniments—e.g. friction, or rhonchi—which interrupt the smoothness of the respiratory movements, and also of vocal fremitus, which serves to indicate the condition of the conducting media. The third enables one to detect the cause of many thoracic pains.

Before making a systematic examination, it is well to lay the hand on any part of the chest which presents an obvious swelling, or where the patient complains of

pain. In doing so the observer should remember to look at the patient's face rather than at the part under examination, as he thus most quickly learns whether he is causing any avoidable suffering. Pain may be due to inflammatory conditions in the chest-wall; to intercostal neuralgia, where, as a rule, specially painful spots can be discovered corresponding to the points where the branches of the affected nerves escape through the fascia; to intercostal myalgia, where the pain is aggravated by pinching the affected muscles; or to pleurisy. In the case of pleurisy, pressure may considerably increase the pain by bringing the opposed surfaces of the inflamed pleura more firmly into contact. At the same time the nature of any swelling should be investigated. The hand will also supplement the information derived from inspection with regard to prominence of the intercostal spaces, and may occasionally detect fluctuation in them when there is pleuritic effusion. Fluctuation also occurs, and is much more distinct, when an abscess has formed in the chest-wall. Such an abscess may be due to disease of the bones or soft parts forming the parieties of the thorax, or to pus which has broken through from the pleural cavity (*empyema necessitatis*). In the latter case the pus may often be driven back by gentle pressure, to reappear when the patient coughs.

When these preliminary observations have been completed, the observer should direct his attention to the **form of the thorax.** Here the hand is best aided by mechanical appliances, such as the cyrtometer, and by simple measurements. Tracings and measurements should be taken at the periods of full expiration and inspiration. In a well-formed adult male the girth of the chest at the level of the nipples should be 34 in. at the end of expiration, and should measure at least 2 in. more when a deep inspiration has

been taken. Height, age, and build of course greatly modify these measurements, and insurance returns indicate that different races vary very considerably in chest girth. It is generally far more important to ascertain the increase of girth between expiration and inspiration, both full and ordinary, than to determine the exact circumference of the chest at either phase.

If the shape of a cross-section of the chest is required, a tolerably efficient cyrtometer can be improvised by connecting two pieces of flattened composition gas-pipe, each about 2 ft. long, by a hinge of elastic tube. The hinge should be placed over the spine, and the metal pipe moulded to the surface of the chest. It can them be opened at the hinge and closed again over a piece of paper, to which the outline should be transferred if a permanent record is desired. To prevent any risk of subsequent confusion, the back and right side of the tracing should at once be marked as such, and a line should be drawn from the position of the hinge to the point in front which corresponds with the middle of the sternum. The length of this line may be checked by a pair of callipers, as a precaution against accidental bending of the cyrtometer during its transference from the patient to the sheet of paper. For convenience in preservation, a reduced copy of the tracing may be made by means of a pantograph; if this is done, a note of the exact proportion between the original and the copy should be recorded on the latter. There is no difficulty in applying the same simple instrument so as to obtain the outline of the chest in other planes than the horizontal. Thus, by placing the hinge above the shoulder, the two pieces of pipe may be carried down the parasternal line in front and in a corresponding line behind, whilst by sharply bending their lower ends outwards they may be made to cross each other in the axillary line and the point of

intersection marked. The instrument is then opened at the hinge and readjusted over the paper so as to yield the desired tracing.

The **nature of the respiratory movements** must next be studied. It is important to make certain that the two sides of the chest move to approximately the same extent. This is done by fixing the finger-tips of either hand at the patient's sides, and making the radial borders of the thumbs meet in the middle line in front of the chest. The hands being kept rigid, the patient is directed to take a full inspiration, when the distance of departure of the thumbs from the middle line indicates the extent of expansion of either half of the chest.

Sometimes one half of the thorax lags behind the other; this is readily detected by the hands no longer moving synchronously.

The movements at the apices may be similarly observed. In this case the physician stands behind the patient, and, fixing his thumbs on the vertebræ, lets his fingers lie over the right and left lung apices reaching towards the clavicles whilst the patient breathes deeply. Thereafter one hand should be placed on the front of the chest, and the other on the epigastrium. In health, as the chest expands, the epigastrium is also raised to a greater or less degree. If the epigastrium falls in with each expansion of the chest, there is reason to suspect paralysis or flaccidity of the diaphragm. Fixation of the diaphragm with immobility of the epigastrium during respiration is generally due to abdominal disease (*see* p. 55).

Vibrations may be detected by palpation. *For this purpose the palm of the hand should be applied flat on the chest,* and, since the sensitiveness of the two hands is often unequal, *the same one should be employed on both sides.* In addition to the vibrations already

referred to in Chap. IV. (p. 119), fremitus may be due to pleural friction, to catarrhal changes in the mucosa of the bronchi, leading to local constrictions, or to fluid in the bronchi or in pulmonary cavities. After the presence or absence of these forms of fremitus has been determined, the observer should study the **vocal fremitus,** or vibrations which the voice communicates to the chest-wall. These are conducted from the larynx by the trachea and bronchi to the smaller tubes within the lungs, and thence through the lung tissue to the surface. Anything which affects the conducting power of the air-passages or lung tissue, or the interposition of additional materials through which the vibration must pass to reach the palpating hand, will obviously affect the intensity of the fremitus. To test the vocal fremitus, the patient is told to repeat " one, one, one," or " ninety-nine," in a clear voice. The hand placed on the thorax detects distinct vibration whilst this is done, and it must be determined whether the vibrations in corresponding areas on the two sides of the chest are approximately equal in intensity—not, however, forgetting that where the heart encroaches on the left lung the fremitus is necessarily much diminished—and also whether they correspond to what former experience has led the observer to recognize as normal for the region under examination, for a similar chest, and like pitch and loudness of voice. **Vocal fremitus is increased** when the voice is of a deep pitch, when the chest-wall is rigid, and often when it is thin, as also when the lung is consolidated, or contains a cavity near its surface. Since the right bronchus is wider and shorter than the left, whilst the septum separating the two bronchi occupies a position to the left of the centre of the trachea, the laryngeal sounds pass more freely along the right than they do along the left bronchus, and therefore the vocal fre-

mitus is normally somewhat greater over the right lung than over the left. **Vocal fremitus is diminished** when the pitch of the voice is high, when the chest-wall is thick, and especially when there is much thickening of the pleura. It is greatly diminished, or totally absent, when the lung is separated from the chest-wall by pleuritic effusion. The cause in this case is not that fluid is a bad conductor of sound or of vibration —the reverse is the case—but that the relaxed lung itself fails to convey the vocal fremitus, and so the vibrations never reach the fluid. In young persons and in female subjects the vocal resonance is different both in character and intensity from that which occurs in male adults. The differences are due to the different conformation and degree of rigidity of the thorax, and to the distinctive pitch and quality of the voice in each instance. The resistance of the chest to compression is best estimated by placing the hand over the sternum whilst the patient is lying down, and attempting to press it backwards toward the vertebral column. The rigidity increases with advancing age, and also in certain diseases (e.g. in tuberculosis and in emphysema). Where this is so the prognosis is less favourable, as free expansion of the lung is hindered.

IV. PERCUSSION

Percussion determines—

A. **The boundaries of the lungs** (topographical percussion).

B. **The resonance of the lungs.**

(*a*) Normal variations in different parts.

(*b*) Abnormal alterations.

1. Quantitative { *Increase* (*hyper-resonance*). *Diminution*, in varying degrees, from slight impairment to absolute dullness.

2. Qualitative: Tympanitic { High-pitched. Medium-pitched. Low-pitched.

Skodaic.
Boxy.
Cracked-pot
Bell sound (coin percussion).
Amphoric.

Methods of percussion.—When percussion was first introduced, the tap was delivered directly on the patient's skin without the interposition of any substance over the point struck. This method, known as *direct percussion*, is now seldom used, except on the clavicles, which in examination of the lungs are lightly tapped by the observer's finger-tip. In order to obtain better resonance, as well as with a view to the patient's comfort, various materials were subsequently interposed between his skin and the percussing finger. A flat plate of bone or ivory, of such a size and shape as to be readily applied and closely adapted to the surface of the chest, is sometimes employed, and is called a *pleximeter*. Most physicians, however, prefer to make use of the middle or forefinger of the left hand as a pleximeter, and the preference is due not only to the fact that it can be readily adapted to almost any surface, but also that it often conveys information additional to that obtained by the percussion sound, as it takes cognizance of the different *degrees of resistance* which the tissues offer to the percussion stroke.

Sometimes a small rubber-tipped hammer, known as a *plessor*, takes the place of the percussing finger, and is occasionally of service; but as a rule the finger should be preferred.

The ordinary method, then, of percussion is conducted in the following manner: The middle finger of the left hand is placed *firmly* on the part which is to

be percussed, and is adapted to any inequalities of surface, so that no air-space is interposed between it and the skin. The back of its middle phalanx is then struck with the tip of the middle finger of the right hand. The stroke should be delivered from the wrist and finger-joints, not from the elbow, and the percussing finger should be so bent that when the blow is delivered its terminal phalanx is at right angles to the metacarpal bones, and strikes the pleximeter perpendicularly. As soon as the blow has been given, the striking finger must be raised, lest it should impair the vibrations it has excited, just as the hammers of a piano fall back from the wires as soon as these have been struck. In cases where the percussion requires to be firmer, several fingers may be used; but it is better, whenever possible, to employ only one percussing finger. In some cases a modification, known as flicking percussion, is useful, and this is particularly valuable in the examination of the abdomen.

There are **three cardinal rules** which should always be remembered when percussion is being carried out. The **first** is that in defining the boundaries between contiguous organs the percussion should invariably be performed from the resonant towards the less resonant. The **second** is that the longer axis of the pleximeter should be parallel to the edge of the organ whose delimitation is being attempted, and the line of percussion should be at right angles to that edge. The **third** is that the pleximeter finger must be kept in firm contact with the chest-wall.

It is seldom necessary to deliver more than two or three strokes at any one situation; repeated blows cause much discomfort to a sensitive patient. The points to be noted on percussion are the *volume* and *pitch* of the resonance elicited, and the sense of *resistance* experienced by the finger.

It must be recollected that it is a most difficult task to give even a partial explanation of the phenomena observed, from the standpoint of physics, and in practice it is rarely necessary to appeal to theory, as a long experience has enabled physicians to attach certain meanings, more or less empirically, to the results of percussion.

It may, however, help the student to appreciate the various sounds when he hears them, if a few of the main factors in their causation are recapitulated.

First, we have to consider the materials which produce the sound. These are the pleximeter, the chest-wall beneath it, and the subjacent viscus so far as it comes within the range of action of the percussion stroke. The pleximeter sound, by the choice of a suitable material, may either be rendered insignificant, or, in consequence of its special qualities, immaterial in its effect on the resonance. The chest-wall yields a sound varying with the part struck, and depending for its quality on whether sternum, clavicles, ribs, or soft parts underlie the pleximeter. The sound due to the wall is, however, subordinate to that of the organ beneath when this contains air and the percussion stroke is firm enough.

The **character of the sound produced** varies quantitatively and qualitatively, the quantitative variations depending on the force of the blow delivered, and on the capacity of the part struck to resound to the blow. The **quality of the sound** depends on the particular vibrations which are elicited, and on the selective reinforcement of some of them by the resonance of the organs involved.

When the air in a cavity of sufficient size and appropriate shape is set into vibrations which are not modified by excessive tension of the containing walls

of the space, the sound heard has a tympanitic character; but when the cavity is subdivided into a number of small loculi by numerous septa, more or less tense, a characteristic resonance, no longer tympanitic, is produced. Such conditions prevail in the healthy lung, and the observer must learn by assiduous practice to recognize its distinctive quality. In general terms, this pulmonary resonance may be said to be low in pitch and clear in character.

In percussion over the lung we endeavour to ascertain three sets of facts: first, the position of the apices and lower border of the lungs, and also of that portion of the anterior border of the left lung which lies over the heart; second, the state of the lungs in regard to the quantity of air contained in their various parts, and the tension of their elastic framework; and, third, whether they are unusually remote from the surface of the chest, the separation being due to thickened parietes, or to fluid or gas in the pleural cavity.

The apices and borders.—Resonance can usually be observed in health for $1\frac{1}{2}$ to 2 in. above the level of the clavicle. The **apices** are either equally high above the clavicles, or the right may reach a shade higher than the left; if the right is a little lower than the left, or the left decidedly lower than the right, there is a probability of past or present disease in the lung whose apex fails to attain the normal limits. Should both apices be very low in level, there may be disease of both lungs. In emphysema both apices are generally found considerably higher than in health. When the examination is made the patient must look straight before him, not turning the head to the side away from the examiner, as this alters the tension of the muscles over the lung. The percussion stroke should not be too strong, and care should be taken that it is delivered quite perpendicularly to the surface.

Whenever there is any doubt of the apices of the lungs being normal, the whole course of their upper borders should be determined. Beginning posteriorly at the level of the spine of the vertebra prominens, the limit of lung resonance in health passes outwards along a line which curves gradually upwards to reach the anterior border of the trapezius, about $1\frac{1}{2}$ in. above the level of the clavicle. Thence it passes obliquely downwards and forwards until it approaches the outer border of the sterno-mastoid, when it inclines more directly downwards towards the clavicle. Sometimes it hardly reaches so far forwards as the sterno-mastoid, at other times the line runs along the surface of the muscle. In cases where the tracheal resonance interferes with the precise delimitation of the lung, the difficulty may be avoided by making the patient open his mouth, thus altering the pitch of the tracheal percussion note.

If disease of the upper lobe of the lung is suspected, it is advisable to percuss along the top of the shoulder from the acromio-clavicular articulation inwards, noting the points at which the pulmonary resonance begins and ends. The distance between these points can then be accurately measured on each side and any difference recorded. The record is valuable for subsequent reference.

The **lower border of the right lung** lies over the liver, and is thin; therefore its exact situation is best made out by light percussion. Posteriorly, however, the muffling due to the thick muscles and fat of the back makes it necessary to percuss more firmly. When the patient is obese, very heavy percussion with several fingers may be necessary in order to penetrate the parietes and bring the lung tissue within the sphere of influence of the blow. In quiet respiration the lower border is found to lie in the mammary line at the 6th rib, in the midaxillary line at the 8th rib,

in the scapular line at the 10th rib, and nearer the vertebral column, as low as the 10th space.

On the left side the lower border overlaps the stomach, and so the transition is not from lung resonance to dullness, but to tympanitic stomach resonance. Posteriorly, however, the splenic dullness and the dullness of the various solid structures which lie below the lung near the spine are interposed, so that the conditions resemble those found on the right.

The position of the lower border corresponds pretty closely with that on the right side; it may, however, be found a trifle farther down.

In old people the lower borders of both lungs extend beyond these limits by about a rib's breadth; in children they do not reach them by about the same degree.

The **anterior border of the left lung** emerges from behind the sternum at the level of the 4th costal cartilage, and forms the upper and left limits of the area of superficial cardiac dullness.

The limits described are exceeded in very deep inspiration, and in diseases such as emphysema, where the volume of the air-containing lung is increased. In pneumothorax the lower border of resonance is often considerably below the limits assigned, and the character of the sound is different (p. 261).

The limits are not attained when the lungs are shrunken or consolidated, when increased abdominal pressure interferes with the normal level of the diaphragm, or when there is effusion in the cavity of the pleura. In this case, should the effusion be left-sided, instead of passing in the anterior axillary line from lung resonance to tympanitic stomach resonance, a band of dullness will be found between the two resonant areas; and since the lower limit of the pleural sac reaches nearly 4 in. lower at this point than the inferior border of the lung, the dullness will pass

downwards to a lower level than the normal lung resonance does, and Traube's area will be encroached upon (Fig. 60). In consolidation of the lung, on the contrary, this area will not be diminished.

Since, in health, the borders of the lungs have a considerable range of movement during deep respiration, whilst in the presence of disease the range is often much restricted, it is important to percuss the apices

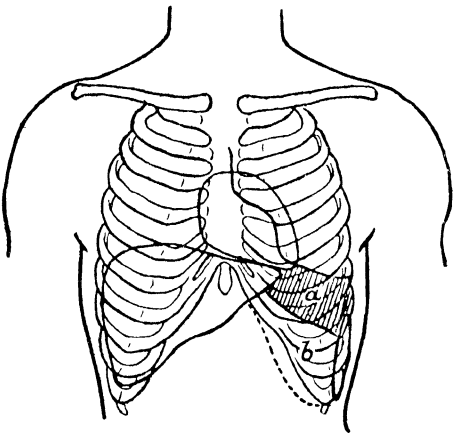


Fig. 60.—Traube's area in pleuritic effusion.
a, Portion rendered dull, *b*, portion remaining resonant

and lower borders of the lungs during both expiration and full inspiration. A unilateral diminution of the range of movement, and still more a unilateral absence of movement either at the apex or base, is an important sign of early infiltration of the lung. Where the range of excursion is deficient at both apices the defect may be due either to bilateral disease, or to imperfect use of the lungs such as is often observed in sedentary

persons. To this procedure Sir Robert Philip has applied the name "*tidal percussion.*"

The lungs having been outlined, the **character of the percussion sound over the various parts** must be studied. Beginning in front, the examiner should tap lightly on the most prominent point of each clavicle—being careful to ascertain that the points examined correspond exactly with each other—and should observe the quality of the sound, and particularly determine whether under like conditions of percussion the effects on the two sides are identical. Thereafter the other corresponding areas on either side should be carefully compared, many points being systematically percussed in each area, and especially in the supraclavicular triangles. The presence of the heart will obviously interfere, in certain parts of the left side, with the development of a sound resembling that on the corresponding point on the right.

When the front has been fully examined, the observer should percuss in both axillary and infra-axillary regions—the patient meanwhile holding his hands joined above his head; lastly, the various areas posteriorly should be worked out; the patient, if able to sit up, being instructed to fold his arms and bend slightly forwards.

It is most essential at all parts of this examination that the patient's attitude is a comfortable one, and that his arms and shoulders are placed symmetrically. The head must not be inclined to either side.

If any of the regions are unusually hollowed, so that the finger cannot be readily adapted to them, a small cork will make a good pillar pleximeter.

Should the patient's chest be unsymmetrical, equal resonance on the two sides is not to be expected.

In a healthy individual the resonance in the various regions will exhibit certain characteristics:—

Apices.—Clear, not very intense, as the vibrating mass is small, and tending to have a slight tympanitic quality added as the trachea is approached. The right apex is usually rather less resonant than the left, and somewhat higher pitched.

The reasons for the difference are that the right apex is less voluminous, as it is encroached upon anteriorly by the great vessels, and that its inner aspect is in contact with the resonant trachea, whereas on the left side the inner aspect of the lung is in relation to non-resonant structures

Clavicular regions. Sternal end.—Clear, moderately intense, with tympanitic element due to trachea. *Centre.*—Clear, more intense than in supraclavicular or outer clavicular regions. Devoid of tympanicity. *Outer end.*—As centre, but less intense.

Infraclavicular regions.—Clear and intense. Slightly tympanitic near sternum.

Mammary regions.—Here there is naturally a difference between the two sides: on the right, the lung is encroached on in the lower part of this area by the liver; on the left, the heart occupies a good deal of the space, and the stomach note is elicited through the thin lung at the lower part. In general, however, the pulmonary resonance is clear and fairly intense, except where the neighbouring organs come within the range of vibration. The chest-wall here is thicker from the presence both of the pectoral muscles and of the mammary gland, and the sounds elicited are consequently more muffled.

In the **inframammary regions** the sounds are greatly influenced by the neighbourhood of the liver, the colon, and the stomach. The lung sound, however, is clear, though not intense, the thin layer of lung becoming rapidly emptier of resonance as its lower border is approached.

In the **axillary regions** the sound is more intense and clearer than elsewhere, diminishing however, in intensity at the lower part of each lateral area.

Posteriorly, the great masses of muscle which clothe the back muffle the resonance and make it feebler; and therefore firmer percussion, often with several fingers, is required. The scapular region is most muffled, the infrascapular least so. The interscapular and suprascapular regions are intermediate in quality.

In disease the resonance may be affected (1) quantitatively and (2) qualitatively.

1. **Quantitative.—Resonance is increased** in emphysema (slightly), but at the same time the pitch is raised by the greater tension of the chest-wall, and this in some cases not only prevents the increased resonance from being observed, but almost suggests dullness.

When the lung tissue is relaxed, but still contains air, the effect of the septa which subdivide the air columns is for the most part abolished, and the sound becomes distinctly tympanitic. At the same time the resonance is increased in intensity. This is sometimes called **skodaic resonance,** and occurs above the level of a pleural effusion, or in the upper portion of a lung whose lower lobe is affected by pneumonic consolidation. When air has found its way into the pleural cavity the sound is, as a rule, intensely *tympanitic*, unless the air is under considerable pressure. A characteristic form of high-pitched tympanitic resonance (**"bruit d'airain,"*** also known as the "bell sound" or "coin sound") may be heard, in pneumothorax, by percussion over the front of the chest with a couple of coins—one being used as a plessor and the other as a plexímeter—whilst the

* Airain = brass.

observer listens at the back of the patient. In very marked cases the sound is soft and musical, and has been compared to the chiming of a distant church bell; in cases that are less pronounced it approximates rather to the stroke of a hammer on an anvil when heard a long way off.

Instead of making use of coins, the chest-wall may be flicked by the finger and thumb whilst the physician auscultates It will be found that the "flick," which is heard through the stethoscope over the *normal* chest as a dull thud, is converted into a ringing or chiming sound as soon as the area of pneumothorax is reached. The alteration in note is more striking than when coins are used, for it is no longer simply a difference in intensity that is observed, but an entire change in the character of the sound.

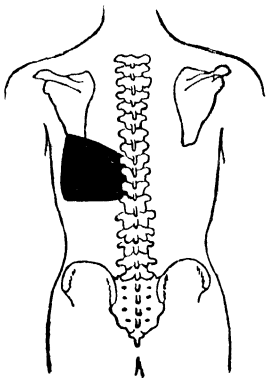


Fig. 61.—Pleurisy, with effusion, seen from behind. (Case I.)

It must be noted, however, that failure to elicit the "bruit d'airain" does not mean that a pneumothorax is not present. Cavities in the lung, or the presence of large or medium bronchi within range of the percussion stroke, likewise cause the sound to become tympanitic. A tympanitic sound, which may closely resemble that caused by the presence of a vomica, is heard when the portion of lung which lies between the trachea or primary bronchi and the surface becomes consolidated or retracted. This sound is sometimes called "Williams's tracheal resonance," and is most frequently discovered in the 1st or 2nd intercostal spaces near the sternum.

Resonance is diminished in cases where the pleura is thickened, or where there is consolidation of the lung—either of a whole lobe, as occurs in pneumonia, or of small patches, as in early tuberculosis. In the latter instance a particular strength of percussion stroke will in each case be found to develop the dullness to the best advantage, according to the size of the solid patch and its distance from the surface. When fluid is present, as in hydrothorax or pleurisy with effusion, the *dullness is absolute*, and an unusual sense of resistance is experienced by the plexımeter finger. In pleurisy with effusion the upper limit of the fluid generally follows a curved line, as is shown in the accompanying figures (Figs. 61, 62, 63).

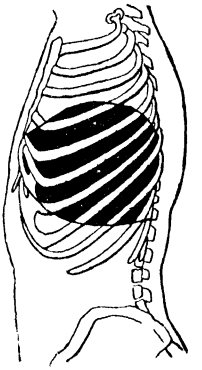


Fig. 62.—Pleurisy, with effusion, seen from the side. (Case 2.)

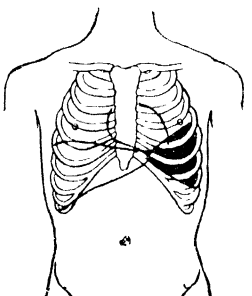


Fig. 63.—Pleurisy, with effusion, seen from the front. (Case 1.)

In the case of patches of solid lung substance the airless portion is often surrounded by a shell of tissue in which the septa are relaxed. The result of percussion over this composite arrangement is to produce a sound whose resonance is less intense and emptier than the healthy lung would yield, whilst what is left of it assumes a

subtympanitic quality. The effect is described as a **boxy or wooden sound.**

2. **Qualitative.**—Several peculiar sounds which are produced by percussion in pathological conditions remain to be noted.

Cracked-pot sound.—This is due to a sudden expulsion of air through a constricted orifice. It occurs in cases where percussion is practised over a cavity which communicates with a bronchus of moderate size, and is most distinct when the mouth is opened. It has a hissing character, combined with a chinking sound like that produced by shaking coins together. It is also heard in certain cases of thoracic fistula, and occasionally in pneumothorax, as well as in the relaxed lung above the level of fluid in pleurisy, and near the consolidated area in pneumonia. If healthy children are percussed while they are crying, a cracked-pot sound is often produced.

Amphoric resonance.—This phenomenon is due to the selective reinforcement of certain vibrations by a large cavity; by this means the overtones are accentuated and die out more slowly.

The following alterations in percussion sounds may be observed under certain pathological conditions, their explanation is for the most part simple from a physical standpoint :—

(*a*) Tympanitic resonance, when due to the presence of a pulmonary cavity which communicates with a bronchus, is raised in pitch when the patient opens his mouth.

(*b*) The pitch of the percussion sound over a cavity varies with the position of the patient. The most obvious explanation is that, if the cavity is partly filled with fluid, this varies in position, and so alters the shape of the cavity when the patient changes his attitude Other factors, however, often enter into the interpretation of this change.

(*c*) The resonance over a cavity becomes higher in pitch during inspiration, and lower during expiration The phenomenon depends on the tension of the wall of the cavity

(*d*) In pneumothorax the metallic resonance is higher in pitch when the patient is lying down than when he sits up

In certain conditions of malnutrition the muscles on the front of the thorax are unduly irritable In these circumstances a light tap over the sternum produces fibrillary contractions, at some distance off, in the pectoral muscles. This phenomenon often occurs in tuberculosis, and is known as *myotatic irritability* or *myoidema.*

Attempts have been made, with some measure of success to map out not only the limits of the lungs but their several lobes, by placing a vibrating tuning-fork, whose stem ends in a small plate, upon an interspace in front of the chest, and at the same time auscultating posteriorly with an ordinary stethoscope A marked difference can sometimes be observed in the note which is heard when the stethoscope passes from one lobe to another, the tone being clearer and louder when the lobe against which the tuning-fork is placed is the one to which the stethoscope is applied.

V. AUSCULTATION

Auscultation determines—

A Character of respiratory sounds.

1. Vesicular breathing (rustling in character)
   - Normal
   - Puerile
   - Harsh
   - Jerky or cog-wheel
   - Feeble or absent
   - With prolonged expiratory murmur.
2. Bronchial breathing (guttural [*ch*] or aspirate [*ha*] in character)
   - I. Ordinary
     - Low-pitched (cavernous)
     - Medium-pitched
     - High-pitched (tubular).
   - II. Amphoric (with an echoing quality added).
     - Low-pitched
     - Medium-pitched
     - High-pitched
3. Indeterminate or broncho-vesicular breathing

B. Vocal resonance.

1 Quantitative changes
- *Increase*
  - Slight.
  - Marked, Bronchophony.
  - Extreme, Pectoriloquy.
- *Decrease*
  - Slight.
  - Marked
  - Entire absence.

2. Qualitative changes
- Ægophony.
- Amphoric resonance.

C. **Accompaniments.**

1. Râles
- Dry (Rhonchi)
  - Sibilant or high-pitched.
  - Medium-pitched
  - Sonorous or low-pitched.
- Moist (Crepitations)
  - *Non-resonant* or toneless
    - Fine.
    - Medium.
    - Coarse
  - *Resonant* (metallic) or consonant
    - Medium.
    - Coarse

2 Friction sounds—Fine, medium, coarse.

3 Splashing sounds—Hippocratic succussion.

In auscultation three observations must be made at each point examined: First, the character of the breath-sounds; second, the character of the vocal resonance; and third, the presence or absence of other sounds.

In order to make these observations with facility, the examiner should attend to the attitude of the patient, which must be as symmetrical and as unconstrained as circumstances will permit. This is easily attained when the patient can sit up; but if he is unable to do this he should be rolled round first to one side and then to the other, in order that the back, and especially the bases of the lungs, may be thoroughly examined. The student must remember that in serious cases great injury may be done to a patient by too prolonged an examination. Care must be taken, especially when an ordinary single stethoscope is used, that the chest-piece is accurately applied, and that no undue pressure is exerted. The patient must be

directed to breathe through the nose, regularly and fairly deeply, but not noisily.

A. Character of Respiratory Sounds

There are two typical varieties of breath-sound, both of which are audible in health at certain parts of the chest, and these must be carefully studied. The first is known as vesicular breathing, the second as bronchial. The former is heard over healthy lung tissue, the latter over the trachea and main bronchi.

In **vesicular breathing**, which can be heard typically in the axillary and infrascapular regions of a healthy individual, the following facts will be noted:—

The inspiratory sound is fairly intense, and is audible during the whole of the act. The pitch is low, and the quality is characteristic, being somewhat rustling. It is this quality which is especially described as vesicular.

The expiratory sound follows that of inspiration without a distinct pause—unless, as not infrequently happens, the patient holds his breath for a second at the end of inspiration; it is less intense than the inspiratory sound, is lower in pitch, and lacks the vesicular quality, being more of a simple blowing sound. It only remains audible during the earlier part of the expiratory phase, and under normal conditions the inspiratory sound is heard for at least twice as long as the expiratory.

To learn to recognize **bronchial breathing**, the student should listen over the trachea, though he must not expect to hear so intense a type of bronchial respiration when he subsequently examines a diseased lung.

The inspiration sound is moderately intense. It becomes inaudible shortly before the end of inspiration. Its pitch is much higher than that of vesicular breathing, and the quality is blowing or hollow, with a guttural or aspirate intonation.

The expiration sound is generally more intense than the inspiratory; the pitch is often higher; the duration extends through the greater part of expiration, being as long as, or even longer than, the inspiratory sound, from which it is divided by the silent period that marks the end of inspiration. In quality it exactly resembles the inspiratory sound, being aspirate or guttural in character. This quality is sometimes described as "tubular," but the same name is also applied to one of the *varieties* of bronchial breathing, and so is better avoided.

1. The principal variations which can be detected in **vesicular breathing** are as follows:—

i. **Puerile.**—The sounds are harsher than in the adult, but have a similar duration.

ii. **Harsh,** with prolongation of expiration, the character, however, remaining vesicular. This frequently indicates loss of elasticity of lung tissue; hence it often occurs in early pulmonary tuberculosis, but may occur in bronchitis.

iii. **Jerky, interrupted, or "cog-wheel" inspiration.**—Here the sound is not continuous, but occurs in waves or sharp jerks. This indicates irregular expansion of the alveoli, due to unequal elasticity in various parts of the lobules, and is therefore not infrequently present in early tuberculosis. It may also result, however, simply from nervousness, and to carry any weight as a physical sign it must be well marked even on deep inspiration. Even then, however, it may mean little or nothing, and should only take a very secondary place.

iv. **The respiratory murmur may be feeble,** or even inaudible. In quiet breathing the expiratory sound is often quite absent. By making the patient breathe more deeply the murmur may be rendered audible. When marked, this condition may indicate defective expansion.

Total disappearance of the breath-sounds usually occurs below the level of fluid in pleuritic exudation, because the relaxed lung does not conduct sounds well, and hence they are not conveyed to the fluid, which is itself a comparatively good conductor. If, however, there is only a small quantity of fluid present, the sounds may be faintly heard, as the relaxation of the lung tissue is less pronounced. Occasionally it happens that when a considerable quantity of fluid has accumulated, the breath-sounds, instead of disappearing, become loud, and possess a marked bronchial character. In such cases the vocal resonance also is loud, but is usually more or less ægophonic. This exceptional state of matters is most commonly observed posteriorly over the lower lobe of the lung, and may be due to collapse of part of the lung enabling the stronger vibrations which are present in a bronchus to be transmitted to the fluid with less loss of intensity than if they had first required to pass through air-containing lung.

With regard to *prolongation* of the expiratory sound, it must be recollected that in certain diseases, such as emphysema and asthma, the expiratory act is performed much more slowly than in health. In consequence of this, the respiratory sound may also be prolonged; hence in these diseases prolongation of expiration conveys a meaning different from the usual one.

Some patients are habitually shallow breathers, whilst others naturally breathe deeply. The ear can

detect these variations, partly by the duration of the respiratory sounds, and partly by their intensity. The depth of breathing as estimated by auscultation is sometimes known as the "*respiratory excursion.*"

2. **Bronchial breathing** is subdivisible into three varieties, according as the laryngeal respiratory sound is conveyed to the ear through consolidated lung from the larger, medium, or smaller air-passages, each of which, by reinforcing certain elements of that sound, gives it a distinctive character.

In the first case we have **low-pitched** bronchial breathing, the more capacious tubes responding best to the deeper-toned elements of the laryngeal murmur ; in the second case the pitch is **medium** ; in the last it is **high.** Low-pitched bronchial breathing is heard pathologically over moderately large cavities in the lungs, and is hence sometimes called *cavernous*; high-pitched bronchial breathing is heard when consolidation has occurred round the smaller tubes, as in pneumonia, where the most perfect examples of bronchial breathing may often be found. Here the character is aspirate rather than guttural. This variety is often known as *tubular breathing.*

A special variety of bronchial breathing exists under diseased conditions, and is known as **amphoric respiration.** It resembles the sound produced by blowing across the mouth of a bottle or the muzzle of a gun. The sound, when analysed, is found to consist of one or more low-pitched fundamental tones and a number of high-pitched overtones. It is characteristic of a direct communication between the bronchus and either a considerable cavity with fairly smooth walls or a pneumothorax. The latter condition yields the best examples.

In cases where the resonance of a bronchus is within earshot of the observer, but where at the same time

air-containing lung intervenes between the bronchus and the chest-wall, the sound of the breathing combines both vesicular and bronchial elements, one or other type predominating according to the exact relations in each case. This variety of breath-sound is known as **broncho-vesicular or indeterminate.** In such cases it is usually the expiratory sound which has more or less of a bronchial character. It occurs in health in certain regions where anatomical causes favour its production, especially near the roots of the lungs behind, and in the upper portions near the middle line in front. The resonance of bronchi which lie so deeply in the chest as to be completely muffled by the thick layer of lung tissue that separates them from the ear may become audible when the tissue around them becomes solidified, and thus conducts the sounds more effectively. If the consolidation reaches to the surface of the lung, the breathing will be bronchial, but if it fails to extend so far, bronchial breathing will be heard through the vesicular breathing which is still being produced between it and the surface.

The breath-sounds must be auscultated in the various regions that have already been examined by percussion, their character in each noted, and similar regions on the two sides of the chest compared, care being taken that the points examined correspond accurately to one another.

B Vocal Resonance

The second series of observations is directed to **the intensity and character of the vocal resonance.** It varies in intensity even in health on the two sides and over different areas of the lung, being louder on the right side, and more intense the nearer the stethoscope is to the larger bronchi. When

the patient repeats the words "one, one, one," or "ninety-nine," the ear receives from the chest no distinct impression of the syllables pronounced, but only a buzzing sound, whose intensity depends on the loudness and depth of the patient's voice and on the conductivity of his lungs. Other words or sentences may be used instead of the above, but on the whole these are well adapted to produce satisfactory and uniform vibration of the chest, and are therefore suitable for the purpose of comparing different points with one another.

An easy way of keeping a standard of intensity in the mind when examining is to conceive of the sound arising at different distances from the observing ear. In some cases the sound is very distant. This is equivalent to "marked decrease" in vocal resonance. Sometimes the sound appears to be produced at a little distance from the chest-piece of the stethoscope. In this case the resonance is slightly decreased, and, to make certain of this, a comparison should at once be made with the corresponding point over the other lung.

In fact, as in percussion and palpation, so in estimating resonance—each point examined on one side of the chest should be at once compared with the corresponding point on the other side. Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest-piece of a single stethoscope. If it seems to be nearer the ear than this, the resonance is increased. When it is near the ear-piece of the stethoscope the increase is marked, and the condition is often described as **bronchophony.**

If the words become articulate and seem to be spoken right into the auscultator's ear, it will generally be found that even whispered words are clearly heard.

This condition is called **pectoriloquy.** Increased resonance occurs when, through any cause, the lung substance conducts the sound-waves set up by the voice more clearly than usual from the bronchi. Consolidation is the commonest cause of increased lung-conductivity. Bronchophony occurs when a moderately large bronchus is surrounded by a layer of solid lung reaching to the chest-wall. Pectoriloquy is fairly characteristic of a cavity of some size communicating with a bronchus. In some cases, however, a certain degree of pectoriloquy is heard over the front of the upper lobe of the lung when the lower lobe is compressed, as, for instance, by pleuritic effusion. Care must be taken that the articulate sounds do not reach the observer either through the other ear or by the patient's lips being directed towards the stem or ear-piece of the stethoscope.

The presence of the whispered pectoriloquy over the spinous processes of the fourth, fifth and sixth thoracic vertebræ in adults and children, and over the first two or three thoracic spines in infants is spoken of as **D'Espine's sign.** It is believed to indicate the existence of enlarged mediastinal glands.

For reasons already explained, vocal resonance is either entirely abolished or much diminished where a layer of fluid separates the lung from the chest-wall (*see* p. 269). It is also diminished in cases of thickened pleura, and of emphysema.

In certain conditions the **quality** of the vocal resonance undergoes modification. Pectoriloquy has already offered us an example of such a modification, but a noticeable change also occurs in pneumothorax, when an **amphoric** or metallic **echoing resonance** is imparted to the voice, as well as to the breath- and heart-sounds. Another alteration in the quality of the vocal resonance is observed in some cases of pleurisy.

When the quantity of effusion is rather scanty, so that the lung is only separated from the chest-wall by a thin layer of fluid, a nasal or bleating character may be imparted to the voice. This bleating tone is observed much more frequently at the back, near the lower angle of the scapula, or between that point and the axillary line, than it is over other regions of the thorax. It is known as **ægophony**, and it is probable that the peculiar quality of the voice is due to the fundamental tone being intercepted by the effusion to a much greater degree than the overtones.

C. Accompaniments

The last series of observations is directed to the detection and recognition of various **adventitious sounds.**

These may arise either in the lung or in the pleura, and it must never be forgotten that sounds by no means very dissimilar may be produced by the friction of the stethoscope on a hairy chest-wall; but the latter can usually be suppressed by moistening the skin. The accompaniments arising in the *lung and bronchi* themselves first demand attention.

Such accompaniments are collectively known as **râles**, and are subdivided into dry râles and moist râles.* **Dry sounds,** known also as **rhonchi,** are produced in the air-passages, and are due to partial obstruction of their lumen either by swelling of the mucosa or by the presence of tough secretion. The mechanism of their production is thus comparable with that to which cardiac murmurs owe their existence.

They vary in pitch, the variations being in a great measure due to the size of the tubes where they take

* The term "râles" is really superfluous and might with advantage be dropped, all accompaniments being subdivided into dry sounds (rhonchi), moist sounds (crepitations), and friction sounds.

origin. The smaller tubes are the seat of high-pitched or **sibilant rhonchi,** and these are most abundant during the latter part of inspiration; the medium-sized tubes yield medium-pitched rhonchi, and the larger bronchi produce the deep-toned or **sonorous rhonchi,** which are heard early in inspiration, and may be almost continuous. Dry sounds are characteristic of bronchitis, but are also found quite apart from any definite bronchitis in certain other diseases of the respiratory system, such as cases of tuberculosis when the bronchial tubes get plugged.

Moist râles, called also **crepitations,** are discontinuous sounds, and are produced either in the alveoli or in the bronchioles and bronchi. They produce on the ear a noise like the bursting of smaller or larger air-bubbles, and indicate the presence of fluid secretions in the air-cells or tubes. They are classified as fine, medium, and coarse or bubbling. The term "crepitation" is sometimes restricted to the first variety, the others being called fine and coarse bubbling râles.

Fine crepitations are caused by the opening up of collapsed alveoli whose walls have been agglutinated by the exudation of a little fluid secretion. This at first causes them to adhere, but, as the air-pressure gradually increases during the movement of inspiration, the adhesion at last gives way suddenly, and allows air to enter. The separation of the walls is accompanied by a cracking sound, which can be imitated by separating the moistened forefinger and thumb near the ear. When this condition occurs in a number of alveoli, the combined effect is to produce a sound of fine crepitation. It occurs only near the end of inspiration, as is to be anticipated from its mode of production, and indicates the presence of exudation in the alveoli of the affected part of the lung.

Fine crepitations are very characteristically present during the first stage of pneumonia, and in acute congestion from any cause; they are also met with in early miliary tuberculosis. After atelectasis they are occasionally heard, and in œdema of the lung they are found in association with bubbling râles caused by the simultaneous presence of fluid in the bronchi.

Medium crepitations occur chiefly in the smaller bronchi, and are audible at the end of inspiration and the beginning of expiration. They are caused by the air bubbling through fluid secretion which has been poured out into the lumen of the bronchi.

Coarse bubbling crepitations occur in the larger divisions of the bronchi, and may be heard at almost any phase of respiration; they may be quite continuous in their occurrence. Coarse crepitations may also originate in pulmonary cavities.

Sometimes the sounds are **non-resonant or toneless.** In this case they occur, as a rule, in spongy lung-tissue; but in other cases they are quite **resonant,** and convey an impression to the ear of being all possessed of a definite pitch. There are only two conditions in which resonant râles are present—either consolidation exists, or there is a cavity of sufficient size to act as a resonator for râles which are produced either in itself or in a neighbouring bronchus.

The highest degrees of resonance are known as **metallic** and **tinkling consonances.** Here the sounds have a very distinct high pitch, and give the impression of a shower of drops falling into a metallic vessel, which reverberates the sound of their fall. This is associated with amphoric breathing, and, like it, suggests either a large cavity or pneumothorax.

The position where râles are heard greatly influences the importance to be attached to their presence. If heard at the apex, they at once suggest tubercu-

losis; whilst medium and coarse crepitation at the bases may be due merely to a transient exudation which will rapidly disappear. When the patient has been breathing quietly for some hours, and especially if he has been lying in bed, a few crepitations, even if heard at the apex, may be due to temporary causes, though they should always be regarded with a degree of suspicion.

The commonest accompaniment arising in the *pleural cavity* is a **friction sound** characteristic of pleurisy at the stage where exudation is not abundant enough to separate the inflamed and roughened surfaces. It possesses a creaking or rubbing character, often quite characteristic; but sometimes, when less well marked, rather hard to distinguish from a râle. The friction sound may be fine, medium, or coarse. In some instances it is palpable, but, since coarse râles may be so too, this does not serve to distinguish them.

The chief features of difference are that friction sounds occur during that part of inspiration when the roughened surfaces are rubbing against each other, to reappear at a corresponding period of expiration. They are, moreover, unchanged after the patient has coughed, whilst râles may alter under these conditions because of changes in the disposition of the secretion which causes them. The fact that friction is sometimes more localized than crepitation may also be of service. Sometimes friction is markedly intensified by increasing the pressure with which the stethoscope is applied. This acts by causing the roughened surfaces to rub against each other more firmly. Pressure does not affect the intensity of râles. The situation of the doubtful sound, or the presence of pain, or some point in the history of the case, may assist the observer in arriving at the diagnosis.

It must never be forgotten that the presence of one form of accompaniment does not exclude the others. Any two or three may be found coexisting in one case. When pleuritic friction is developed along the anterior edge of the left lung, and especially when that part of it which is in relation to the apical segment of the heart is affected, the friction sounds often assume the rhythm of the heart-beat rather than that of the respiratory movements. Hence the sound is liable to be mistaken for pericardial friction. To distinguish between this so-called *pleuro-pericardial friction* and that of true pericarditis will rarely be very difficult if it is recollected that the former, depending as it does on the apposition of two roughened patches of pleura, is only heard during those phases of respiration when the patches are in contact. Hence a deep inspiration, by removing one of them from the other, may prevent the production of the sound, whilst in other cases holding the breath, or emptying the lungs as completely as possible, may lead to a like result. In short, pleuro-pericardial friction is much more dependent than true pericardial friction on the movements of respiration.

Hippocratic succussion is the name given to a splashing sound which can be heard when a patient who has both gas and fluid (usually pus) in the pleural cavity is shaken or moves suddenly.

Post-tussive suction is the term applied to a sucking noise, resembling that produced by an india-rubber ball that has been compressed and is springing open again, which is sometimes heard immediately after a cough. It occurs over a cavity in the lung when its walls are not too rigid, and is caused by the re-entry of the air. When distinctly heard it is of considerable diagnostic value, as it can only occur when a cavity is present.

VI. X-RAY EXAMINATION

In all doubtful cases of pulmonary disease, it is impossible to exaggerate the importance of a careful X-ray examination of the chest. The carrying out of such an examination, however, is a matter requiring special skill and experience and its description does not fall within the scope of such a book as this.

VII. THE SPUTUM

The characters of the cough have already been discussed in a previous chapter (Chap. II, p. 38). It remains to add a few notes on the appearance and examination of the sputum in different diseases.

Naked-Eye Inspection of Sputum

The following are the principal points to be observed with the naked eye:—

1. Quantity.
2. Consistency.
3. Whether homogeneous or in layers of different appearance.
4. Whether frothy or airless.
5. Colour and transparency.
6. Odour.

The above qualities depend on the character of the material which is coughed up. The main varieties are mucous sputum, serous sputum, fibrinous sputum, purulent sputum, and blood. In many instances transition types between them are observed.

Mucous sputum is characteristically present in early bronchitis. It is clear, tough, and sticky. As a rule, the amount is not great. At a later stage of bronchitis the mucus is mixed with pus cells. The sputum is then less tough, more copious, and has a greenish-yellow colour.

Muco-purulent sputum occurs in many diseases of the lung. In tuberculosis with cavity formation one often finds small ragged lumps of muco-pus, surrounded by mucus, which are heavier than the other constituents since they are airless. They therefore sink to the bottom and become more or less flat and button-like. This constitutes the "**nummular**" sputum of phthisis. If there is a fair amount of serous or watery fluid mixed with such sputum it gradually settles into three layers, the lowest being purulent, the next serous, and the uppermost composed of frothy mucus.

Sputum composed of pus alone comes from an abscess or an interlobar empyema which has been ruptured into the air-passages.

Serous sputum occurs apart from mucous expectoration as a thin, watery fluid, generally blood-stained. It indicates œdema of the lung. Pulmonary œdema without extravasation of blood yields a white frothy sputum like soapy water.

Blood may be coughed up alone, or the sputum may be more or less blood-stained. It must be distinguished from blood brought into the mouth from epistaxis, gastric hæmorrhage, or bleeding from varicose veins in the walls of the œsophagus. Its brighter colour and its frothy appearance often make the discrimination perfectly simple. When it comes from the lungs its presence may result either from pulmonary or cardiac disease, or from aneurysm.

Several diseases cause a **characteristic coloration of the sputum.** Thus, in pneumonia it is **rusty,** and so viscid that it often will not fall out of an inverted spittoon; it is **bright-yellow or green** when a liver abscess has ruptured into the lung, and the latter colour also appears in some cases of pneumonia. Sometimes, when an amœbic hepatic abscess has discharged by the lung, the sputum has the appearance

of **anchovy sauce. Black sputum** is common with coal miners, whilst red-streaked sputum is suggestive of tuberculosis. **Prune-juice sputum** occurs when blood lingers in a lung which has become œdematous. Thus it is found in cases of chronic pneumonia that are going on to disintegration of the lung tissue. **Red-currant-jelly sputum** is said to be characteristic of malignant disease in the lung.

The **quantity** of sputum coughed up in twenty-four hours is important; and still more so whether large quantities are rapidly got rid of at considerable intervals, or whether it comes away in small amounts and frequently.

Occasionally small **casts of bronchi** are to be found in the sputum, but the examination for formed elements is best conducted with the aid of a microscope.

The **odour of the sputum** is seldom very characteristic. Ordinarily it has a "stale" smell, but in cases of gangrene of the lung, of fetid bronchitis, and of bronchiectasis it may develop an exceedingly penetrating putrid odour. An unpleasant odour may also be acquired during its transit through the mouth.

Microscopical Examination of Sputum

Generally it is well first to examine an unstained and fresh specimen. Thereafter special methods are used for the recognition of bacteria (*see* p. 549). To select a suitable piece, place the sputum in a flat glass vessel, which can be laid on either a white or a black background as is found convenient. Mixed with the amorphous mucous exudation which forms the basis of the sputum may be seen various organized structures as follows:—

1. **Cellular structures.**—i. **Pus cells** in various stages of granular degeneration.

ii. **Epithelium** from the mouth, air-passages, and alveoli. The latter may contain pigment which has reached them from the air, or they may exhibit a very characteristic iron-containing pigment, which is unusually abundant in cases of heart disease with pulmonary congestion, and indicates brown induration of the lung. This pigment yields the hæmosiderin reaction on the addition of hydrochloric acid and potassium ferrocyanide.

iii. **Red blood cells.**—A few are of no importance. Large numbers occur in hæmoptysis.

iv. **Eosinophil cells** occur in asthma.

2. **Elastic fibres** indicate destruction of lung tissue, whether from phthisis, gangrene, or abscess. In gangrene only a few fibres escape the destructive process. They are found in the small tough lumps of the sputum, and are best demonstrated by a rapid heating with an equal quantity of 10-per-cent. solution of caustic soda. After boiling, a gelatinous mass is left, to which a considerable quantity of water should be added, and the mixture left in a conical glass till the elastic fibres settle to the bottom. Thus they may be isolated, and in well-marked cases exhibit the alveolar arrangement of the lung tissue.

3. **Hair** may be coughed up in a case where a teratoma communicates with a bronchus. Trichoptysis visible to the naked eye is very rare.

4. **Fibrin casts,** often large enough to attract the unaided eye, are still more frequently visible under a low power of the microscope.

5. **Parasites.**—Hydatid disease of the lung may be indicated by the presence of hooklets, and still oftener of fragments of the laminated ectocyst of *Echinococcus hydatidosus.*

6. **Asbestosis bodies.**—Workers in asbestos may suffer from asbestos pneumonokoniosis. In the diagnosis of this condition the sputum should

be searched for highly characteristic golden-yellow bodies (Plate 18). They vary in size and shape, but characteristically they have bulbous enlargements at the extremities with a regularly or irregularly segmented body resembling dumb-bells. The appearance of fully formed bodies has been aptly compared to beads on a necklace. The beads vary in size and represent the irregularly segmented body. These bodies have been found by various observers to vary in length from 20 to over 200 microns. An asbestos fibre forms the central core of each body, and can frequently be detected. They are best seen with an oil immersion lens, and show up clearly, without staining, as golden-yellow structures. They can, however, be stained by hæmatoxylin. Further, the golden-yellow material covering each fibre contains an iron substance which gives the Prussian blue reaction when potassium ferrocyanide and hydrochloric acid are used.

CHAPTER VII

THE URINE

THE method of interrogating a patient whose symptoms point to an affection of the urinary system has already been described (p. 10), and the physical examination of the kidneys has been considered along with that of the other abdominal organs (p. 69).

In this chapter we propose to take up the examination of the renal secretion.

Collection of samples.—Owing to the variations in the composition of the urine at different times of the day, the sample examined should, if possible, be taken from the total urine of the twenty-four hours. If only one sample can be obtained, it should be that which is passed about three hours after taking a meal, as abnormal ingredients are then more likely to be present. The sample should be poured into a tall conical glass, covered, and allowed to stand for some hours in a cool place. If it is desired to preserve the urine for some time, toluol should be shaken up with it in sufficient quantity to form, after separation, a thin film over the surface, or the urine may be acidified by adding 15 c.c. of HCl to $1\frac{1}{2}$ litres.

Any suspended matters soon settle to the bottom of the glass, and the examination of the sample may then be proceeded with. This should be conducted (1) physically, (2) chemically, (3) microscopically.

I. PHYSICAL EXAMINATION

Attention should be paid to the following points, viz.: (1) quantity, (2) colour and transparency, (3) consistence, (4) odour, (5) density, (6) naked-eye characters of the deposit.

1. **Quantity.**—The amount of urine passed during the day should be measured separately from that passed during the night. The sum of the two gives the total for twenty-four hours. The bladder should be emptied at a fixed hour—say 8.30 a.m.—and the product discarded. All the urine passed during the day is collected, and the bladder emptied again at 8.30 p.m., the product being added to the day's secretion. This is the amount of the "*day urine.*"

The bladder is again emptied at 8.30 next morning, and the product added to that which has been passed during the night. The total quantity is the "*night urine.*" This added to the day urine gives the total for twenty-four hours.

It is often difficult to collect all the urine that is passed, some being lost with the motions. This is especially the case with children, female patients, and those who pass their evacuations involuntarily. Where great accuracy is required, recourse must be had to the catheter.

A healthy adult male passes on an average 50 oz. (1,450 c.c.) of urine in twenty-four hours; women, a few ounces less.

The following table represents the amount of urine passed daily by children of different ages (Holt):—

Age	*Quantity*
First twenty-four hours	0 to 2 oz.
Second twenty-four hours	½ „ 3 „
Three to six days	3 „ 8 „
One week to two months	5 „ 13 „
Two to six months	7 „ 16 „
Six months to two years	8 „ 20 „
Two to five years	16 „ 26 „
Five to eight years	29 „ 40 „
Eight to fourteen years	32 „ 48 „

Churchill asserts, as the result of his own observations, that the amount of urine passed by children is less than is usually supposed. He gives the following averages of quantity and specific gravity at different ages —

Age	*Quantity*	*Sp. gr.*
3 years	358 c.c.	1024
4 ,,	299 ,,	1027
5 ,,	392 ,,	1024
6 ,,	405 ,,	1023
7 ,,	564 ,,	1018
8 ,,	628 ,,	1021
9 ,,	731 ,,	1020
10 ,,	768 ,,	1023
11 ,,	716 ,,	1018
12 ,,	829 ,,	1021

Above the age of 15 the quantity passed is about up to the adult standard.

It will be observed that, relatively to their weight, children pass more urine than adults. This is to be attributed to the relatively greater activity of the metabolic processes in children and to the more fluid nature of their diet. The above quantities, however, are only roughly approximate, and in many cases one will find that the amount of urine excreted by a child of given age is smaller than the quantity tabulated.

Normally, very much more urine is secreted during the day than during the night, the average volume of the night urine in a healthy adult being less than 15 oz. with a specific gravity of about 1032. The normal proportion of day urine to night urine is 100: 25-60. Approximation of the night quantity to that of the day is always abnormal, and is especially apt to occur in chronic renal disease, of which it may constitute one of the earliest signs. Thus the proportion of day to night urine may become 100: 100

or even 200. The solids are increased in proportion to the water.

An *increased secretion* of urine occurs physiologically after increased consumption of food or drink, and after exposure to cold. Conversely, one finds the *secretion diminished* when little food or drink has been taken, and after exposure to heat—especially if followed by sweating.

A **pathological increase** in the urine occurs in diseases associated with an increased arterial pressure—e.g. interstitial nephritis; also in both forms of diabetes, during the absorption of exudates, and in some neurotic conditions—e.g. hysteria. **Abnormal diminution** of urine is found where the arterial pressure is lowered or the intravenous pressure in the kidney increased—e.g. in acute nephritis and in advanced mitral disease; also in all fevers, in diarrhœa and vomiting, and in cerebral irritation—e g. concussion.

2. **Colour and transparency.**—Normal urine is said to have the colour of amber or pale sherry. The exact tint fluctuates widely even in health, depending upon the degree of dilution and upon the reaction. An acid urine is always darker than one which is alkaline, even when they are equally concentrated. The colour of normal urine is mainly due to a yellow pigment, to which the name of urochrome has been given. The pigments uroerythrin and urobilin only occur in very small quantity in the urine under normal conditions. Where there is excessive blood destruction or impaired liver-function, however, a large quantity of urobilin may appear in the urine. The latter has then a warm orange colour, and usually shows a dull pink tint at the apex of a conical glass. Urobilin is not present in freshly voided normal urine, its place being taken by its precursor, urobilinogen.

On standing, this becomes converted into urobilin, giving the urine a darker colour. The change takes place in a few minutes if hydrochloric acid is added to the urine, being particularly noticeable when excess of urobilinogen is present. Excess of urobilinogen

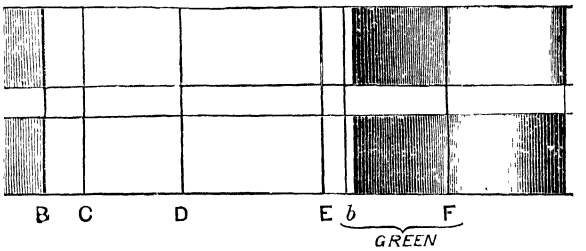


Fig. 64.—1, Spectrum of urobilin; 2, spectrum of urobilin masked by other pigments. (See text.)

may be tested for with the following reagent: Dimethyl-paramino-benzaldehyde 2 grm., hydrochloric acid 5-per-cent., 100 c.c. If 20 drops are added to an inch of urine in a test-tube, excess of urobilinogen is shown by a bright red colour, which appears immediately *in the cold.*

If urobilin urine is examined spectroscopically* in a thin layer the urobilin band will be seen in the green between *b* and F (Fig. 64). Such a urine is often

* The following directions for the spectroscopic examination of urine are given by A. E. Garrod:—

1. Use a small direct-vision spectroscope.
2. Examine the urine in a 6-oz. conical glass. This permits of the inspection of layers of different thickness.
3. Hold the slit 1 in. from the glass, and move it up and down the entire length of the cone.
4. Either daylight or artificial light may be employed.
5. If in doubt as to the bands, shake up 400 c.c. of the urine with 50 or 60 c.c. pure amyl alcohol and a few drops of acetic acid. Collect the layer which floats. Clear it, if need be, by the addition of a little ethyl alcohol filter, and examine.

dichroic—looking red by transmitted and green by reflected light. The presence of excess of urobilin may be confirmed by the following test: To 5 c.c. of urine add 3 drops of tincture of iodine. Into another test-tube place 0·5 grm. of zinc acetate and 5 c.c. of absolute alcohol. Mix the two solutions and repeatedly pour from one tube to another until most of the zinc acetate is dissolved. Filter. Examine the filtrate against a dark background. If urobilin or urobilinogen is present in excess, a green fluorescence appears.

The table on p. 290 shows the chief varieties of alteration in colour of the urine, with their causes.

Normally, when freshly passed, urine is quite transparent, but it may be **opalescent** from the presence of various substances in suspension. If the opalescence persists after filtration, it is due to the presence of bacteria.

A slight opalescence which causes the urine to look smoky is produced by the presence of small quantities of blood (*see* p. 311).

Alkaptonuria.—This is a condition in which the urine is natural-looking when passed, but, when exposed to the air, becomes gradually darker from the surface downwards; ultimately it may be dark brown or black. It is due to the presence in the urine of dihydroxyphenyl acetic (homogentisinic) acid.

The addition of an alkali causes the urine to become dark at once. Such urine reduces alkaline solution of cupric oxide. With Millon's reagent it gives a yellow precipitate, and the addition of dilute ferric chloride drop by drop causes a passing deep-blue colour. The urine, moreover, will not ferment with yeast nor turn the plane of polarized light, nor form an osazone.

The condition is a rare anomaly, attended by no

Alterations in Colour of Urine

Colour	*Cause*	*Condition or Remarks*
Nearly colourless.	i Large amount of urine excreted	Much drinking Nervous conditions, diabetes insipidus, etc
	ii Diminution of pigment	
Orange-coloured.	i Small amount of concentrated urine.	Hard muscular work Fevers
	ii Increased pigment	Fevers.
	iii Occasionally bile-pigment	Jaundice.
Orange-coloured. Reddish-brown.	Administration of rhubarb, senna, chrysophanic acid	(These turn yellow with acid, red with alkali, a normal colour going red with alkali = phenolphthalein)
Dark-brown.	Methæmoglobin.	(P 312.)
Red or Pink	i Blood	(P. 311.)
	ii Aniline dyes in sweets, etc.	
Cherry Red	Pyramidon (amidopyrin)	(Colour may become darker on standing)
Port-wine	Hæmatoporphyrin	(P 313)
Brownish-black	i. Melanin	Melanotic sarcoma (Darkens on standing, does not reduce Fehling's solution, gives greenish-brown precipitate with ferric chloride, blackens with HNO_3)
	ii Much hæmoglobin	(Yields chocolate deposit.)
	iii Alkaptonuria.	(P. 289)
Greenish-black	i. Hydroquinone, carbolic acid, salol, guaiacol, resorcin, naphthalin, etc.	
	ii Bile.	In old-standing cases of jaundice.
Yellowish-Green Green.	i Bile	Jaundice (p. 320)
	ii. Santonin	(Turns red with alkali.)
Yellowish and Milky.	i Pus.	(P. 322)
	ii Fat as (*a*) emulsion, (*b*) droplets	Chyluria. Lipuria in advanced renal disease.
Greenish-blue Blue.	i. Excess of indigo-forming bodies.	Typhus
	ii. Administration of methylene blue.	(Violet with alkali; cuts off red and yellow in spectrum.)

symptoms beyond sometimes a slight dysuria and frequency at night. It is due to an inborn error affecting the breaking down of the tyrosin linkage in the process of protein metabolism.

Urines containing melanin also become darker on exposure to the air, owing to oxidation of the pigment, but they do not reduce cupric oxide.

Carbolic-acid urine, too, becomes darker on exposure to air, owing to the oxidation of the hydroquinone which it contains into pigments similar to those in alkaptonuria.

In alkaline urines an **iridescent pellicle** frequently appears on the surface. When the urine has cooled this can be skimmed off like a thin brittle film. It is composed of calcium phosphate. The idea formerly entertained that such a pellicle occurs especially in the urine of pregnancy is groundless.

3. **Consistence of urine.**—In health the urine is quite watery in consistence. If much sugar or bile is present it is less mobile, and in the presence of bile or of much albumin the froth which forms on shaking is more persistent than is usual. Alkaline urine containing pus may be quite ropy.

4. **Odour.**—Normal urine has a characteristic "aromatic" odour. When the urine has stood for some time the odour becomes ammoniacal. In cases where there is an abnormal communication between some part of the urinary tract and the intestine the odour may become fæcal. In acetonuria the odour is fruity. After the administration of turpentine the urine has an odour like violets. Cubebs, santonin, and some other drugs also impart to it their peculiar smells.

5. **Density.**—Clinically the specific gravity of urine is always taken with the instrument known as a **urinometer.** An ordinary urinometer is graduated

for a temperature of 15° C., and will record variations in a specific gravity from 1000 up to 1060.

How to use the urinometer.—The urine should be allowed to cool, and should be placed in a tall jar, wide enough to allow the urinometer to float freely without touching the sides. All bubbles must be removed from the surface by means of bibulous paper. The urinometer should be wiped clean and placed floating in the centre of the jar. The eye is then placed level with the surface of the urine, and the division of the scale to which the latter reaches read off. Care must be taken to read the level of the true surface of the urine, not the edge of the rim which heaps up around the shaft of the urinometer.

If only a small specimen of the urine is obtainable it may be necessary either to use "specific gravity beads," or else to add water to it in order to get enough fluid to float the urinometer. The specific gravity found is then multiplied by the necessary figure according to the degree of dilution.

Normal urine has a specific gravity varying from 1015 to 1025. If very concentrated, the specific gravity may rise to 1035 even in health. During the first month of life the specific gravity varies between 1001 and 1005, but by the second year it has reached 1026 or 1030, and in older children the urine tends to be rather more concentrated than that of adults.

The gravity is greatly increased by cooling. If, for example, it is 1020 when passed, it will rise to about 1025 when the urine has cooled to the temperature of the room. This should be especially borne in mind in insurance work.

In normal urine the specific gravity is in direct proportion to the amount of urea present. An abundant urine of *low* specific gravity is suggestive either of diabetes insipidus or of chronic renal disease. An abundant urine of *high* specific gravity is characteristic of diabetes mellitus. In the latter condition the specific gravity may reach 1075; in most cases, however, it is between 1040 and 1045. In diabetes insipidus, on the other hand, the specific gravity may fall to nearly that of distilled water. The presence

of albumin in the urine does not materially affect its specific gravity.

Estimation of the amount of solids.—This may be done roughly by multiplying the last two figures of the specific gravity taken at 15° C. by 2·33. The result is the number of grammes of solids in 1 litre of the urine; e.g. if the specific gravity of a urine is 1020, it contains $20 \times 2{\cdot}33 = 46{\cdot}6$ grm. of solids in every litre, or 4·6 per cent. The average daily output of solids in the urine is about 60 to 70 grm. (2–2½ oz.). The above mode of calculation is not applicable to urine containing abnormal ingredients, e.g. sugar or albumin.

6. **Naked-eye characters of the deposit.**—When voided, normal urine is perfectly clear and transparent. After it has stood for some time there appears in it a deposit of "**mucus.**" This forms a woolly-looking cloud which usually settles to the bottom of the glass, but, if the urine is of high specific gravity, may be in the middle of the glass, or even at the top. Opinion differs as to whether this " mucus " is a gluco-protein (mucin) or a nucleo-protein. It is possibly both, the latter being in excess.

If traces of blood are present in the urine the cloud of " mucus " has often a brownish tint.

The normal urinary ingredients, which may separate out in the form of a deposit visible to the naked eye, are—earthy phosphates, urates, and free uric acid.

Phosphates.—The phosphates of calcium and magnesium separate out if the urine is neutral or alkaline. They form a colourless deposit. It can be recognized by the fact that if a little of it is transferred by a pipette to a test-tube, and some dilute acetic acid added, the deposit dissolves. A deposit of pus is apt to be mistaken for one of phosphates, but the former is not dissolved by acetic acid. Deposits of pus and

phosphates often occur together, and the certain recognition of pus is often of the greatest importance. Such recognition can only be made under the microscope, and no other method should ever be relied upon.

Urates.—The urates of sodium, potassium, and ammonium may form a deposit if the urine is concentrated or highly acid. They may appear, even in health, when the urine cools. Owing to their affinity for the urinary pigments the deposit is usually coloured, being commonly red, or like terra-cotta, forming what is known as the "*brick-dust*" *deposit*. If the urinary pigment is scanty, however, the deposit may be merely yellowish, or even colourless. Deposits of urates can always be recognized by the fact that they disappear rapidly on heating the urine. The heating ought to be accomplished gradually, because the urine may also contain albumin, which, if the urine is rapidly heated, may be coagulated before the deposit of urates has all had time to clear up, and thus confusion may arise. Acetic acid does not dissolve a deposit of urates. On the other hand, strong mineral acids, such as nitric acid, dissolve the deposit at once, with the production of effervescence.

Acid sodium urate is a rare deposit. It occurs in acid urines. It forms a yellowish, granular, sandy-looking sediment. It does not dissolve readily on heating.

Acid ammonium urate forms a very similar deposit, but it occurs in ammoniacal urines, and is therefore usually mixed up with a deposit of phosphates.

Uric acid.—This may form a scanty deposit visible to the naked eye. The deposit occurs in the form of crystalline grains of a darkish-brown colour, and is therefore known as the "*cayenne-pepper*" *deposit*. When in doubt use the microscope.

The sulphates practically never form urinary deposits. **Oxalates** do, but the deposit is generally scanty and impossible to recognize with the naked eye. We have already spoken of the occurrence of fibrin, and the other abnormal ingredients which may be deposited will be described in the section on the microscopical examination of the urine.

We would warn the reader against the common mistake of supposing that a substance is necessarily being excreted in excess when it appears in the urine in the form of a deposit. This, of course, is not necessarily the case at all. Thus the occurrence of a "cayenne-pepper" deposit does not necessarily mean that the patient is excreting an excess of uric acid. It may merely be due to the fact that the conditions which normally cause the uric acid to be in solution have become modified. The urine may be abnormally acid, for example, or it may be deficient in colouring matter or in salts, all of which conditions tend to lessen the solubility of uric acid, and to favour its deposition in the form of crystals. Similarly in the case of a deposit of phosphates. That does not mean that more phosphoric acid is being eliminated; it merely indicates that the urine has become alkaline.

II. CHEMICAL EXAMINATION OF THE URINE

1. Reaction

This is taken with litmus paper. The urine is usually acid in reaction, but it may be normally alkaline after meals. This is sometimes known as the *alkaline tide*. It reaches its height three hours after the taking of a meal. Alkalinity of the urine may be due to ammonia. This can be detected by its smell, also by the fact that if the red litmus paper which has been turned blue is heated, the

red colour is restored, owing to the ammonia being driven off.

For clinical purposes **the total acidity and the "ammonia"** in the urine may be determined as follows:—

Place in a flask about 15 grm. of powdered potassium oxalate (neutral to phenol-phthalein) (This is to precipitate the calcium in the urine, otherwise the formation of calcium phosphate interferes with the end-point of the reaction) Add 25 c c of urine, an equal quantity of distilled water, and 10 drops of 1-per-cent alcoholic phenol-phthalein Mix well. After one minute run in $\frac{N}{10}$NaOH from a burette until a faint pink colour results. Read the burette; this gives the total acidity in terms of $\frac{N}{10}$NaOH. Now to 5 c.c. of formalin in a beaker add 5 c.c. of water and a few drops of phenol-phthalein; then run in $\frac{N}{10}$NaoH till a faint pink colour again appears Add this mixture to the neutralized urine in the flask. The pink colour disappears. Run in $\frac{N}{10}$NaOH until it returns, and read burette. This reading gives the amount of ammonia in terms of $\frac{N}{10}$NaOH. By the addition of the neutral formalin the ammonia in the urine is combined with it to form a neutral compound, hexamine. The previously neutralized acid is thus liberated and determined by the second titration. The amount (in grammes) of N present as ammonia is determined by multiplying this reading by 0 0014. This reading is in reality a trifle high, but is sufficiently accurate for clinical purposes. The error is due to the fact that by this means the amount of the amino-acids present in the urine is also determined. Except in cases such as cystinuria, the amount of these acids is so small as to be negligible. For very accurate work, Folin's method may be employed; an advanced physiological-chemistry manual should be consulted.

Sellard's test for acidity. — This is the simplest method of arriving at an idea of the excess of acid retained in the body. It depends upon the fact that a normal individual will secrete an alkaline urine after taking from 5 to 10 grm. of sodium bicarbonate, but when there is a condition of acidosis much larger quantities are required to render the urine alkaline.

In carrying out the test, 5 grm. of sodium bicarbonate should be given every hour until an alkaline reaction is obtained. The successive specimens of urine should be boiled, and tested with litmus paper after cooling.

The test is not of much value in nephritis, but in cases of diabetes it enables one to form a fairly accurate idea of the degree of acidosis.

2. Examination of the Urine for its Normal Non-nitrogenous Constituents

(1) **Chlorides.**—Sodium chloride is the chief inorganic constituent of normal urine. Small quantities of the potassium salt also occur.

Qualitative test for their presence.—Filter the urine if not already clear. If albumin is present, remove it by boiling. Add to ½ in. of the urine in a test-tube a few drops of nitric acid (be sure that the acid used is quite pure and free from HCl), and then as much of a 3-per-cent. solution of nitrate of silver as there is of urine. If the normal amount of chlorides is present, an abundant curdy precipitate appears at once. If the chlorides are diminished, the solution merely becomes milky If a mere trace of them is present, the solution is opalescent; and if they are altogether absent, it remains quite clear.

The use of the nitric acid is to prevent the precipitation of phosphate of silver.

About 12 grm. represents the average daily excretion of chlorides in health. The chief cause of physiological variation is the nature of the diet. Pathologically, chlorides are found to be diminished in all febrile affections with the exception of malaria. In the latter disease the chlorides are increased during the febrile period, diminished in the apyrexial intervals. In lobar pneumonia the chlorides are markedly diminished and may indeed disappear entirely.

(2) **Phosphates.**—The salts of phosphoric acid with calcium and magnesium (earthy phosphates) are insoluble in an alkaline medium, hence they are precipitated when the urine loses its acid reaction

This precipitation is aided by the action of heat. The heat probably acts by driving off carbonic acid. Hence if a urine, the reaction of which is not acid, is heated, a cloud of earthy phosphates may appear. This is distinguished from albumin by its ready disappearance on adding a few drops of acetic acid.

Qualitative tests for phosphoric acid in urine.—(i.) Add ammonia. A white crystalline precipitate, increasing on standing, shows the presence of the phosphates of the alkaline earths (Ca, Mg), the so-called *earthy* phosphates. The *alkaline* phosphates of Na and K still remain in solution.

(ii.) To 10 c.c. of urine add half its volume of nitric acid, 3 c c of a 10-per-cent. solution of ammonium molybdate, and boil. A yellow precipitate is given by both forms of phosphates.

Normally, 1-1 5 grm. of phosphorus is excreted daily in the form of inorganic phosphates. Physiological variations depend chiefly upon the food. The phosphates are often considerably diminished in renal disease, but not, apparently, out of proportion to the other solids of the urine. Their behaviour in fever is inconstant.

(3) **Sulphates.**—Sulphuric acid occurs in the urine as inorganic salts (inorganic sulphates), and in combination with cresol, phenol, indol, skatol, pyrocatechin, etc. (organic sulphates). The former are ten to twenty times more abundant than the latter.

Sulphur is also present as "neutral sulphur," that is, the sulphur present in organic bodies of which it is an integral part of the molecule, e.g. cystin.

About 1-1·5 grm. of sulphur is excreted daily as sulphates. The exact determination of the total sulphates, and of the proportion of inorganic to organic, is a gravimetric process unsuited for ordinary clinical work.

The total sulphates are increased by an increase in the diet, and in fever. The amount of sulphur excreted as organic sulphates is increased when a

larger quantity than usual of the aromatic substances with which it is combined enters the circulation. This occurs when phenol and allied substances are given as drugs, or when the production of such substances in the body is increased, as it is whenever putrefactive processes are going on. The ethereal sulphates of the urine are not, as has been supposed, a direct measure of such putrefactive processes, since the proportion of inorganic and organic sulphates and neutral sulphur varies with the amount of nitrogen in the diet.

(4) **Oxalates.**—A precipitate of calcium oxalate does not necessarily mean that the excretion of oxalic acid is increased, although it is true that the more oxalic acid there is present, the greater is the tendency for it to be precipitated. About 0·017 grm. is the average amount of oxalic acid excreted daily. It is mainly derived from the food. It is increased after the taking of certain vegetables, especially cabbage, spinach, and rhubarb.

3. Examination of the Urine for its Normal Nitrogenous Constituents

Of the total amount of nitrogen in the urine—

84–87% is in the form of urea;
2–5% " " ammonia compounds;
3% " " creatinine;
1–3% " " uric acid;
4–7% undetermined (including purin bases).

These vary in percentage according to the intake of nitrogen in the diet, the relative percentage of urea falling markedly when the nitrogen intake is much reduced.

About 15–20 grm. of nitrogen is excreted daily in the urine of a healthy adult on ordinary diet. A knowledge of the quantity excreted in disease is not of much value *unless one has some idea of the amount*

of nitrogen in the diet. It must be remembered also that normally 1–2 grm. of nitrogen appear in the fæces.

Urea ($CO(NH_2)_2$). **Qualitative test.**—Place a drop or two of the suspected fluid on a slide and add one drop of nitric acid; warm gently. On evaporation, rhombic or hexagonal crystals of nitrate of urea will be found if the latter body be present (Fig. 65).

Quantitative estimation.—*From the amount of nitrogen given off on treating the urine with hypobromite of soda.*—This method, sufficiently accurate for clinical purposes, depends upon the fact that urea is decomposed by hypobromite of soda according to the following equation: $CO(NH_2)_2 + 3NaBrO = 3NaBr + N_2 + 2H_2O + CO_2$. It is found that under ordinary conditions 1 grm. of urea yields 371 c.c. of nitrogen; all that is necessary, therefore, is to remove the CO_2 by means of an alkali (caustic soda), and to measure the remaining volume of gas, and to calculate from it the amount of urea which was contained in the quantity of urine taken. The various forms of apparatus employed in carrying out the process differ chiefly in the method adopted for catching and measuring the nitrogen given off.

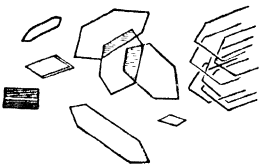


Fig. 65.—Urea nitrate.

The number of c.c. of nitrogen given off (from 5 c.c. of urine) multiplied by 0·056 = grammes of urea in 100 c.c. of urine, i.e. the percentage, and this multiplied by 4·375 = grains of urea in 1 oz. of urine.

The following is the chief form of apparatus employed :—

Gerrard's ureometer (Fig. 66).—This consists of a graduated glass cylinder closed at the upper end by a rubber stopper. Through the stopper there passes a T-tube. One limb of this tube is closed by a clip or stopcock, the other is connected to a piece of rubber tubing. The other end of the rubber tubing terminates in a piece of glass tube, which is inserted into the rubber stopper of a wide-mouthed flask of about 6 oz. capacity. From the lower end of the graduated cylinder another rubber tube passes to a short wide glass tube open at its upper end. The object of this tube is to act as a reservoir of water. It can be slipped up and down upon the cylinder by means of a metal ring.

How to use the apparatus —Place in the glass flask 25 c.c. of hypobromite solution (Appendix, 9). An excess of hypobromite does no harm—one must merely be sure that enough is taken to decompose all the urea likely to be found in the urine. Measure 5 c.c. of urine into the small glass tube pro-

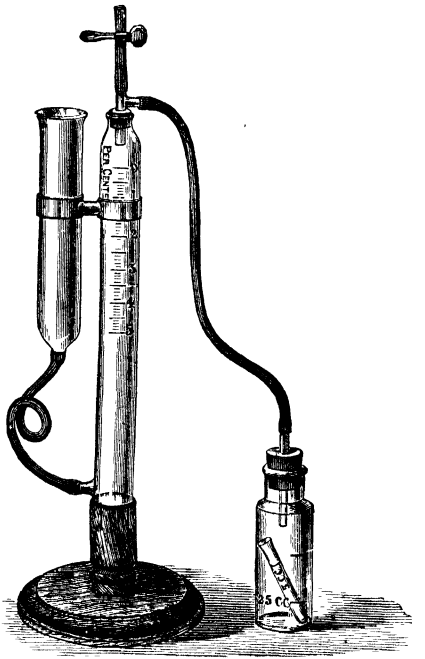


Fig. 66.—Gerrard's ureometer.

vided for the purpose. If the urine contains albumin, the latter must be first removed. This is best done by taking a definite quantity of urine—say 50 c.c.—adding to it a drop or two of acetic acid, and boiling for a couple of minutes. Filter and make up to its original volume.

The small tube containing the urine must now be lowered into the flask. This is best done by inserting the point of the

little finger—not too tightly—into the mouth of the tube. The latter must then be propped up against the upper surface of the flask so as to prevent the hypobromite solution from mixing with the urine. The reservoir of the graduated cylinder must now be filled with water. The stopper is then tightly inserted into the mouth of the flask The clip or stopcock must now be opened, and the reservoir raised until the water inside the cylinder stands at the zero mark and is level with that in the reservoir. The water must also be very low down in the latter, else there will be an overflow subsequently. Now close the stopcock and gently tilt the flask so as to allow the urine and hypobromite solution to mix. Great effervescence ensues, and the nitrogen liberated enters the cylinder and drives water out of it up into the reservoir. Wait for ten minutes to allow cooling to take place. Then lower the reservoir until the water in it and the cylinder are again level, and read off the amount of gas in the latter The cylinder is graduated in percentages of urea. To get the number of grains per ounce, multiply this by 4 375.

In normal urine only 92 per cent. of the nitrogen of the urea is given off. If sugar is present the yield is for some reason much larger, amounting to about 99 per cent. In Gerrard's instrument the scale is constructed for normal urine. In cases of diabetes, therefore, it is necessary to correct one's result by multiplying the figure obtained by $\frac{92}{99}$, i.e. by 0 93

About 20–35 grm. of urea is excreted daily in health. This is about 2 per cent. It is increased when much protein is taken; also in fevers, in diabetes, and in poisoning by phosphorus or arsenic. It diminishes under diminished diet, and in some conditions of the kidney. It must be repeated that *no calculation of the amount of urea which a patient is passing is of much use unless the amount of nitrogen in his diet is also taken into account.*

Ammonia. Quantitative estimation.—Folin's method is the most exact, but it requires a special apparatus. The formaldehyde method is convenient and rapid, and sufficiently accurate for ordinary clinical purposes. (*See* p. 296.)

On an ordinary diet the average daily excretion of ammonia is about ½ to 1 grm., and the ammonia nitrogen is from 2 to 5 per cent. of the total daily nitrogen. The excretion of ammonia is increased by the ingestion of acids or by their production in excess of the body. Thus the ammonia nitrogen rises greatly in all conditions leading to "acidosis," such as advanced diabetes, delayed chloroform-poisoning, and the malignant type of the vomiting of pregnancy.

Uric acid occurs in the urine in combination with alkalis. Being a dibasic acid, it forms two classes of salts, the normal urates and the acid urates. Under certain conditions uric acid becomes free in the urine and separates out as a crystalline deposit ("cayenne-pepper" deposit). Acid urates may also separate out in a crystalline form. Both forms of separation, when occurring inside the urinary passages, lead to the disease known as "gravel" or to stone-formation. The microscopic characters of these deposits are described in another section (p. 334). The conditions which favour the separation of uric acid and acid urates are (1) the presence of a large amount of uric acid in the urine; (2) a high degree of acidity; (3) the presence of little salts and of a small amount of pigment; (4) cold.

If present in large amounts, uric acid will reduce Fehling's solution.

Quantitative estimation.—For clinical purposes this is best accompanied by Hopkins's method as modified by Folin. It is based upon the insolubility of acid urate of ammonia in a saturated solution of ammonium sulphate. By saturating a given quantity of urine with sulphate of ammonium, all the uric acid separates out as acid urate of ammonium, which is collected; the uric acid is then split off from it, and

estimated either by weighing or by titration with permanganate of potash. Proceed as follows:—

(1) Saturate 100 c c. of urine (preferably warmed to 45° C.) with powdered ammonium sulphate. About 30 grm. will be required. Saturation is complete so soon as a few crystals remain undissolved after vigorous stirring at short intervals. Even if these should redissolve as the temperature of the mixture rises again after its initial depression, it does not matter.

(2) Render alkaline by adding a little ammonia.

(3) After it has stood for ½-1 hour, filter and wash the precipitate several times with saturated solution of ammonium sulphate.

(4) Wash the precipitate off the filter with a jet of hot water, add a pinch of carbonate of soda, and heat till the precipitate dissolves.

(5) Add distilled water to 100 c.c.

(6) Add 20 c.c. strong sulphuric acid.

(7) While hot, titrate with a $\frac{1}{20}$th normal solution of potass. permanganate—i e. 1 578 grm. per litre.

(8) Stop when a pink colour lasting a few seconds has appeared. Subsequent disappearance of the colour is to be disregarded; it is sufficient that it should be visible for a second or two after stirring.

(9) Every c.c. of the permanganate used = 0 00375 grm. of uric acid.

The presence of bile-pigment interferes with the titration with permanganate. In that case the uric acid must be estimated by weighing. The trouble in the above method is that the solution is so dense that it passes very slowly through the filter-paper. To obviate this difficulty, glass-wool may be employed. (A little experience is necessary to enable one to pack the glass-wool firmly enough to keep back the precipitate, but yet not so tightly as to render filtration very slow.)

The presence of albumin does not affect these methods. If there is a deposit of uric acid or of urates in the sample of urine, the whole should be thoroughly shaken up, the amount to be operated with measured off, and saturated as usual. Or a few

drops of ammonia may be added and the urine warmed till the deposit dissolves. A deposit of phosphates may be neglected.

From 0·4 to 0 7 grm. (7–10 gr.) of uric acid is excreted daily, the amount depending largely upon the amount of nucleo-proteins in the food. It is increased whenever a large destruction of nuclein is going on; thus in myeloid leukæmia as much as 4 grm. may be excreted daily. It is also increased in acute fevers. It is diminished in chronic gout and after the administration of quinine.

We would again warn the reader against the common error of assuming that a deposit of urates or uric acid necessarily indicates an increased excretion of the latter.

Purin bases.—Among the chief products of the disintegration of nuclein are uric acid and some basic bodies which may be termed the "nuclein" or purin bases. These substances differ from uric acid in being basic. They are hypoxanthin, xanthin, adenin and guanin. They contain between them from 1 to 5 per cent. of the total nitrogen in the urine. The purin bases are increased, just as uric acid is, in conditions associated with increased destruction of nuclein—in leukæmia. There is no constancy in their behaviour in gout.

The only other nitrogenous constituents of normal urine which call for mention are creatinin and hippuric acid.

Creatinin is creatin minus water. The creatinin excretion of normal persons is remarkably constant and very little is really known as to the clinical significance of increased or decreased creatinin content of the urine. It is said to be increased in pneumonia, typhoid, and tetanus; decreased in leukæmia, anæmia, advanced degeneration of the kidneys and liver,

muscular atrophy and paralysis. About 1 grm. of it is excreted daily.

Hippuric acid occurs in the urine as hippurate of sodium. About ½ grm. of it is excreted daily. This amount is increased by the taking of benzoic acid as a drug, or of fruits—e.g. mulberries and cranberries—which contain aromatic acids.

4. Abnormal Chemical Constituents of Urine

(1) Proteins

Any or all of the proteins of blood-plasma—serum-albumin, serum-globulin, and fibrinogen—may occur in the urine. In addition, one meets with the compound proteins—mucin and nucleo-protein—and with proteoses, both primary and secondary. It is doubtful whether true peptone ever occurs in the urine. It is true that two of these—mucin and nucleo-protein—are to be regarded as normal urinary constituents, being added to the renal secretion as it passes along the urinary passages, but in health they are present in such small amount that they may be neglected. It is also true that albumin occurs in the urine in perfectly healthy persons; the urine of healthy infants, for instance, often exhibits traces of it to the ordinary tests; generally, however, the amount present is so small as not to be detected by the tests used for clinical purposes. Into the possible causes of this, and into the distinction between "functional" and "organic" albuminurias, we do not propose to enter. Chemical examination of the urine can merely show the presence in the urine of a protein; it cannot tell us to what its presence is due.

i. **Serum-albumin and serum-globulin in the urine ("albuminuria").**—These proteins may be found in the urine either together or separately.

The former condition is the usual one, and constitutes what is ordinarily spoken of as "albuminuria." The relative proportion of each protein varies greatly in different cases, but usually serum-albumin is present in considerably larger amount than serum-globulin. The variations in their relative amounts have no clinical significance. In what follows, the term "albumin" will be held to include also globulin, unless stated otherwise.

Before proceeding to apply **tests for albumin** it is essential that the urine should be *absolutely clear*. It may therefore be necessary to filter it. If, after filtering more than once, the urine remains turbid, bacteria are probably present, and can be removed by long centrifugation, or by shaking up the urine with powdered barium carbonate and filtering. The following test should then be proceeded with:—

Pour the urine into a test-tube which is clean both inside and outside, until it is about two-thirds full. Boil the top inch or so in a Bunsen flame, without shaking Compare against a dark background the boiled portion with the clear column below. If no turbidity appears, add a few drops of 3 per cent. acetic acid If the boiled portion still remains clear, albumin is absent. If a turbidity appears after boiling it may be due to albumin or to earthy phosphates, and a few drops of acetic acid must be added. If the turbidity remains, albumin is present. Whatever the original reaction of the urine may be, always boil before adding the acid Never omit to add the acid after boiling, whether a turbidity appears or not.

Another test, probably the most sensitive of all tests for albumin, is a saturated solution of pure *salicyl-sulphonic acid*. This has the advantage of being non-corrosive, and therefore easily carried about. To perform the test, add a few drops of the saturated acid solution to 3 c.c. of urine. A white precipitate indicates albumin, nucleo-protein, or proteoses. In the case of proteoses it disappears on heating the solution, reappearing on cooling.

Quantitative estimation of albumin.—This can be done with sufficient accuracy for clinical purposes by means of Esbach's albuminimeter. The principle of the method consists in measuring the depth of the coagulum produced in the urine by the addition of picric acid. The instrument consists of a thick glass test-tube, with graduations on it from 0 up to 7.

Method.—Filter the urine if not already clear, and if alkaline render slightly acid with acetic acid. If the specific gravity be 1010 or more, dilute the urine sufficiently to bring the density below that level (to 1008). This is important, and is often overlooked. Fill the tube with the urine up to the mark U. Pour in the reagent (Appendix, 10) up to the mark R. Close the tube with a rubber stopper, and gently invert it a few times to allow the fluids to mix. Set aside for twenty-four hours. At the end of that time read off the level of the surface of the precipitate. The figures on the scale represent grammes of dried albumin per litre of urine.

Divide by 10 to get the percentage. If the urine requires to be diluted, the result must, of course, be multiplied the requisite number of times.

The method yields only approximate results, since the precipitates obtained in different urines vary in compactness and in the length of time they take to settle

Very small quantities of albumin cannot be estimated by Esbach's method, as the instrument does not record less than 0 1 per cent. If after the first trial the level of the precipitate is found to be above the mark 4, the urine must be diluted and a fresh estimation made.

ii. **Proteosuria.**—This is a more correct term than " peptonuria," formerly in use. It is very doubtful, as before remarked, if true peptone ever occurs in the urine. The clinical significance of the presence of proteoses in the urine is not yet finally determined. Recent investigations tend to show that they may occur in any " infective " disease—i.e. wherever disintegration of tissue is going on under the action of micro-organisms. Thus they are not uncommonly met with in the urine in pneumonia. They are most constant, however, in cases where a large collection of

pus has formed in the body—e.g. in empyema or large abscess-formation. They have also been found in considerable quantity in some cases of nephritis. The proteose in these cases is usually transient and of little importance. Bence Jones protein is not a proteose.

Detection of proteoses.—There are two classes of proteoses—primary and secondary—the latter standing nearest to the peptones. From a clinical point of view the differentiation of the two is of no importance. We will assume first that the urine to be examined is free from albumin. Proceed as follows :—

(*a*) Add to the urine (filtered and acidified if necessary) a few drops of a saturated solution of salicyl-sulphonic acid. Boil, and filter whilst hot. If the filtrate becomes cloudy on cooling, proteose is present.

The presence of antipyrin, quinine, and certain resins in the urine is apt to give a similar reaction.

(*b*) Add to the urine an equal volume of a saturated solution of common salt, and then drop in acetic acid so long as a cloud forms. If this disappears on heating and reappears on cooling, proteoses are present. Both forms of proteose give this reaction.

If the urine is already albuminous the albumin should be removed before testing for proteose. To do this, bring the urine to boiling-point, add a drop or two of acetic acid, and boil for two minutes. Filter and test filtrate as above.

iii. **Nucleo-proteinuria and mucinuria.**—We have already mentioned that both a nucleo-protein (or a substance very closely resembling one) and mucin occur normally in the urine, and it is probable that the so-called "mucus" of the urine consists mainly of the former. In catarrhal conditions of the urinary passages, however, and especially of the bladder, an excess of what is perhaps true mucus may appear in the urine, and to this the term "**mucinuria**" has been applied. So long as the urine is acid, mucin is insoluble and forms a deposit at the bottom of the vessel. Such a deposit may be distinguished from pus by the absence of pus cells on microscopical examination. If the urine is alkaline the mucin goes partially or

entirely into solution. It may then be detected by adding to the urine a few drops of acetic acid. A white cloud, insoluble in excess and increased on boiling, indicates mucin. This will often succeed better if the urine is previously diluted with its own bulk of water, as the presence of a large quantity of salts tends to prevent the precipitation.* There is no simple clinical test for distinguishing true mucin from nucleo-protein in the urine.

iv. **Bence Jones protein** and allied proteins are usually, but not invariably, found in the urine of patients with multiple myelomatosis, a fatal disease involving the hæmatogenous elements of the bone-marrow. This protein is also found, very rarely, in the urine of patients with leukæmia. It coagulates at a lower temperature (55° C. and under) than the usual coagulable proteins of urine. Typical Bence Jones protein has also the remarkable property of going into solution again at about 100° C., if, as is usually the case, the concentration of salts in the urine is suitable. Typical Bence Jones protein may be detected, even in the presence of considerable albumin, by boiling the urine, which has been made slightly acid with acetic acid, and filtering hot, using a funnel with a hot-water jacket. If typical Bence Jones protein is present, the filtrate will become cloudy as it cools.

Allied proteins, instead of redissolving, form a rubber-like mass when the urine is brought to the boil, very different from the coagulum formed by albumin and globulin. To test the urine for these proteins take 10 c.c. of urine, made slightly acid

* Previous dilution of the urine has also the advantage that it prevents one from being deceived by a precipitate of urates which may be thrown down if acetic acid is added to a concentrated urine.

with acetic acid: add 1 c.c. of 7-per-cent. calcium chloride, place a thermometer in the test-tube, and stand in a beaker of water. Heat the water slowly and note the temperature at which the protein begins to coagulate. If this is under 60° C. the protein is probably one of the Bence Jones group. This may be confirmed by the solution of the protein or the formation of a rubbery mass when the water is brought to the boil.

(2) BLOOD AND ITS DERIVATIVES IN URINE

Blood may appear in the urine as a whole (hæmaturia), or blood-pigment may appear without corpuscles (hæmoglobinuria). These two conditions can only be differentiated by examining the deposit for blood-cells. The detection of small numbers of red cells, such as may be found for several days after an attack of renal colic, can only be achieved by a careful microscopic examination. (*See* p. 341).

If urine contains only a small amount of blood or blood-pigment it has a peculiar opaque appearance, to which the term "smoky" is applied. Large quantities of blood give to the urine a red colour varying in intensity with the amount of blood present. The blood-corpuscles are apt to settle at the bottom, producing a flocculent deposit, which is brown or red according to the amount of the blood and the degree of its alteration.

The following tests depend upon the presence of blood-pigment, and therefore give a positive reaction both in **hæmaturia** and in **hæmoglobinuria**:—

i. **The spectroscopic test.**—The urine, if very dark in colour, should first be diluted, and it should always be filtered. It should then be examined in a layer of 5 cm. thick—a small flat glass bottle does well enough, or, failing that, a test-tube. The spectrum of oxyhæmoglobin is readily

detected The spectroscopic is a certain test for hæmoglobin if positive, but unless that substance is present in fair amount it may not be possible to identify the spectrum with the ordinary direct-vision spectroscope. (Plate 17.)

ii. **Guaiac test.**—Take 1 in. of urine in a test-tube, add to it two drops of tincture of guaiac. A white precipitate forms, owing to partial precipitation of guaiac resin. Now add 1 in. of ozonic ether without shaking. If blood-pigment is present a blue colour appears at the line of junction of the fluids *

The blue colour is due to oxidation of the guaiac by oxygen derived from the ozonic ether, the blood-pigment acting as the carrier. Ozonic ether is a solution of peroxide of hydrogen in ether.

Fallacies.—If iodides are present in the urine a blue colour is produced on applying the test. It is distinguished from that due to blood by the fact that it appears much more slowly, and by its appearing simultaneously all through the fluid, not at the junction of the ether and the urine.

Pus gives with guaiac alone a greenish-blue colour, which disappears on heating.

The presence of much saliva in the urine (e.g. from the patient spitting into it) is also a possible source of fallacy, as it gives the guaiac test owing to the presence of an *oxidase*. As oxidases are present sometimes in urine, it is better to boil the urine before performing the test. The coagulum in this case is turned blue when blood is present.

Methæmoglobinuria.—Methæmoglobin may be formed from hæmoglobin in any acid urine after it has stood for some time. Not infrequently, however,

* The tincture of guaiac must be prepared from fresh—i.e. unoxidized—resin, and the ozonic ether must contain in solution peroxide of hydrogen of 30-volume strength It should give off bubbles of gas when poured into the test tube If these points are not attended to, the test may fail Sanitas is a very good substitute for ozonic ether in the above test, and is less expensive

methæmoglobin is present in the urine when passed. It has been said to indicate that the hæmorrhage has its origin in the kidney. The characteristic smoky tint of the urine in hæmaturia of renal origin is largely due to methæmoglobin, and the pigment present in "paroxysmal hæmoglobunuria" consists mainly of it also. Spectroscopic examination is the only satisfactory test for methæmoglobin. It gives a band visible in the red, in addition to two bands nearly in the position of those due to oxyhæmoglobin (*see* Plate 17, facing p. 224).

Hæmatoporphyrinuria. — Hæmatoporphyrin (iron-free hæmatin) occurs normally in the urine in very small amount, and may be considerably increased without affecting its colour. When present in large quantities the urine has a dark port-wine colour. Such a urine does not give the guaiac reaction. If examined with the spectroscope in a thin layer it may possibly show the characteristic spectrum of so-called alkaline hæmatoporphyrin, that being the form met with even in acid urines. Often, however, no distinct spectrum can be obtained on direct examination of the urine. In such a case the pigment can be extracted by shaking up the urine with a little amyl alcohol or acetic ether, after the addition of a few drops of acetic acid. The extract so obtained shows the bands of alkaline hæmatoporphyrin, viz. four bands, one at the junction of the red and yellow, a second in the yellow, a third in the green, and a fourth (the broadest) between the green and the blue. On adding a drop or two of hydrochloric acid the bands of acid hæmatoporphyrin are obtained, viz. two bands, one in the orange (narrow) and one at the junction of the yellow and green (broader) (*see* Plate 17, facing p. 224). The latter is the characteristic band, and consists really

of two halves—a lighter half on the side next the narrow band, and a very dark half on the side away from it.

Hæmatoporphyrin sometimes appears in large amount in the urine of patients who are taking sulphonal, but much more commonly in females than in males. It is a sign of very grave significance, as such cases often terminate fatally. The excretion of port-wine-coloured urine by a patient who is taking sulphonal is always an indication for the immediate stopping of the drug and for the free administration of alkalis.

Urine which contains blood or hæmoglobin contains also, of course, some albumin, and it is often difficult to say whether the blood is sufficient to account for all the albumin present or whether true albuminuria exists as well. We have found that if human blood is added to normal urine in an amount sufficient to produce distinct smokiness, the quantity of albumin amounts to merely a trace. Even when the quantity added is sufficient to render the urine distinctly red, the amount of albumin, as shown by Esbach's method, is only $\frac{1}{2}$ per 1,000.

(3) SUGARS IN THE URINE

The sugars which are of most practical importance in the examination of the urine are glucose and lactose. Lævulose may sometimes occur along with glucose. Cane sugar and maltose may conceivably appear in the urine if excessive quantities of either are ingested. Under rare conditions pentoses may also occur.

Glucose in the urine.—Glucose (dextrose or grape sugar), $C_6H_{12}O_6$, is by far the commonest variety of sugar met with in the urine. The condition is spoken of generally as "glycosuria." This must be distinguished from "diabetes." Diabetes—or, more

correctly, diabetes mellitus—is a disease of which glycosuria is the chief symptom, but every patient with glycosuria has not necessarily got diabetes. Traces of glucose occur in normal urine, but not in an amount capable of detection by the reagents usually employed. If, therefore, glucose is detected by any of the tests we are about to describe, its presence may be regarded as pathological.

i. **Fehling's test.**—As a preliminary to carrying out the test, one must always make sure that the reagent is good. This is necessitated by the fact that Fehling's solution alters on keeping, with the result that on boiling it deposits a precipitate of cuprous oxide. The exact nature of the alteration is not fully understood. To test the Fehling's solution, add to it an equal volume of water, and boil for two minutes. If the solution remains clear, it is to be regarded as safe. Should a precipitate occur, a little more caustic soda should be added and the liquid filtered. It is then ready for use Add to 1 in. of Fehling's solution in a test-tube a few drops of the urine (freed from albumin), and boil. If any considerable quantity of glucose is present, a yellow or red precipitate will appear. Should none be evident, add as much urine as there was Fehling's solution and boil for two minutes. Set aside If after standing the solution still remains quite clear, there cannot be more than a mere trace of sugar present.

The test may be made more sensitive by pouring the urine from a pipette on to the surface of the just-boiled Fehling's solution and allowing the tube to stand for a few minutes. Reduction occurs at the interface and in the urine where there is little caustic alkali or tartrate to dissolve the oxide of copper.

If the urine contains 1 per cent. or more of glucose, a reddish or yellow precipitate appears at once on mixing the boiling urine and Fehling's solution. If under 0·5 per cent. is present nothing but a greenish deposit may appear on standing.

Certain *precautions and fallacies* in the use of Fehling's test have still to be mentioned.

In the first place, the urine must be free from albumin. If necessary, add a drop or two of acetic acid to the urine, boil and filter.

Fehling's test cannot be applied to strongly ammoniacal urine, as the free ammonia would prevent precipitation of cuprous oxide.

If the amount of glucose present is more than is required for reduction of all the cupric oxide, some of it is apt to be caramelized, especially on prolonged boiling. The whole liquid and precipitate then becomes of a dark-brownish colour.

The fallacies attendant upon the use of Fehling's test are due to the fact that other substances in the urine besides glucose can reduce cupric oxide. The chief of these among the normal ingredients is uric acid; of the abnormal constituents, the chief are lactose, glycuronic acid, and the products of certain drugs—e.g. chloral, chloroform, salicylates, carbolic acid, etc. "Alkapton" urines, and those to which formalin has been added, also reduce Fehling's solution. In a doubtful case, if the specific gravity of the urine is high, it should be reduced by the addition of water to about 1015. Any reduction of Fehling's solution then obtained after boiling for ten seconds, either immediately or on standing for a minute or two, almost certainly indicates the presence of sugar in pathological amount, provided the patient is taking no drugs.

ii. **Benedict's test.**—To about 5 c.c. of the reagent (Appendix, 12) add about 8 drops of the urine and boil for two minutes. When the solution has cooled, the original blue colour will have turned brown if a reducing sugar is present. Opalescence or a greenish colour indicates various degrees of reduction.

Apart from homogentisinic acid, Benedict's solution is only reduced by glucose, lactose, or pentose.

iii. **Phenyl-hydrazine test.**—Place 60 c.c. of urine in a 100-c.c. beaker. Add 1 grm. of sodium acetate and rather less pure phenyl-hydrazine hydrochloride (the colourless crystals are best). Place in a water-bath, stirring from time

to time, and scraping off any deposit which forms on the sides. Continue till the bulk is reduced to 10 or 15 c.c. Allow to cool, and examine the deposit after two hours. If there are no crystals, sugar is absent.

The following is a simpler form of the test for clinical purposes :—

To 10 c.c. of the protein-free urine in a test-tube add 6 drops of glacial acetic acid, enough solid phenyl-hydrazine hydrochloride to cover a shilling, and twice this amount of solid sodium acetate. Heat to dissolve, and filter into another test-tube. Immerse this in a boiling water bath for forty minutes. Turn out the flame and allow the tube to cool in the bath for an hour.

When glucose is present, bright, sulphur-yellow needle-shaped crystals will be found arranged in tufts, sheaves, or rosettes. If sugar is absent, only brown or yellowish globules or granules are seen, and in such a case the reduction of the Fehling's solution cannot have been due to glucose. Glycuronic acid, lactose, and the pentoses, however, yield crystals which might be mistaken for those given by glucose. If the nature of the reducing substance is in doubt, one should proceed to the fermentation test.

iv. **Fermentation test.**—This is really the one absolutely certain test for glucose, that being the only fermentable substance which is ever found in the urine.

The following precautions must be observed in carrying out the test: (*a*) The urine must be acid. Alkaline urine would putrefy; therefore render it acid, if necessary, by adding tartaric acid. (*b*) Boil the urine for ten minutes, to drive off any air it may contain; cool. Use bakers' yeast. Shake the urine up with a small piece of it, so as to form an emulsion free from lumps, then place the urine so prepared in a tube. Special fermentation tubes are manufactured. If one of these is not obtainable, an ordinary test-tube inverted in a bath of mercury will do. A Doremus (Southall's) ureometer tube does extremely well. The long limb of it should be filled with the urine completely, no air-bubbles being left. Set aside the tube in a warm place, and examine after a few hours. If a distinct bubble has appeared at the top of the tube, the urine is fermentable, and contains at least 0·5 per cent. of glucose.

Care must be taken to ascertain that the yeast is active. It should be tested with a dilute solution of glucose. It is also well to have a control tube full of normal urine to which yeast has been added, as the yeast is apt to give off a little gas.

If these precautions are observed, the test is trustworthy and delicate.

The only drawback to the fermentation test is that it may not give any reaction if the urine contains less than 0·1 per cent. of sugar. If, then, no gas is produced on fermentation, one should apply Benedict's test to the urine after it has been submitted to the action of yeast for twenty-four hours. If no reduction is now obtained, then the urine has contained a trace of glucose, though not enough to ferment appreciably. Should the Benedict test be positive and the fermentation test negative, it will be necessary to test for lactose and pentose (p. 319).

Quantitative estimation of sugar. Fehling's method consists in titrating the urine with a known quantity of Fehling's solution at boiling temperature, and observing when all the cupric oxide has been reduced to cuprous oxide, as evidenced by the discharge of all the blue colour from the solution. Owing to the formation of the red precipitate, it is not an easy matter to determine accurately when the blue colour has disappeared, and for this reason Fehling's method is not recommended for quantitative work, the following modification being preferable:—

Gerrard's cyano-cupric method.—This method depends upon the fact that the colourless double cyanide of potash and copper is capable of holding cuprous oxide in solution. If, therefore, Fehling's solution is titrated with a sugar solution in the presence of this cyanide, the blue colour fades gradually, no precipitate being thrown down. The end-point is thus very sharp, and, as there is no tendency to re-oxidation, the process may be safely conducted in an open porcelain basin.

To prepare the double cyanide.—Dilute 100 c.c. of

Fehling's solution with about 200 c.c. of water, boil, and run in cautiously an approximately 5-per-cent. solution of cyanide of potash. When the colour is just gone, dilute to exactly 500 c.c., and keep in a well-stoppered bottle.

Method of titration.—To 30 c.c. of the above solution add 10 c.c. of Fehling's solution, and boil in a porcelain basin. Run in the diluted urine cautiously till the colour of the solution has just gone. As the double cyanide undergoes no reduction, one has only to reckon with the 10 c.c. of Fehling's solution added. This process is rapid and accurate enough for clinical use. As a mixture of the double cyanide and Fehling's solution will keep quite well for a number of weeks, it is easy to prepare it in bulk and to measure out each time such an amount as will correspond to 10 c.c. of Fehling's solution.

Calculation.—10 c.c. Fehling's solution = 0·05 grm. glucose. Suppose 10 c.c. of the diluted urine has been used, and 5,000 c.c. is the amount of urine passed in twenty-four hours: then

$$\text{Sugar in 24 hours} = \frac{5{,}000 \times 0\ 05}{10} = 25 \text{ grm.}$$

but the urine was diluted 1 in 20,

$\therefore$ sugar in 24 hours = $25 \times 20 = 500$ grm.

The result must be multiplied according to the degree of dilution.

Lactosuria.—Lactose is sometimes found in appreciable quantity in the urine of women who are nursing. It reduces Benedict's solution, and gives with the phenyl-hydrazine test yellow rosettes of phenyl-lactosazone which are smaller than the sheaves yielded by glucose, but it gives no reaction with the fermentation test. It may be estimated by titration in the same way as glucose, it being remembered that the reducing power of lactose is to that of glucose as 7 is to 10; i.e. if 7 parts of glucose reduce a given quantity of solution, it will require 10 of lactose to effect the same result.

Pentosuria.—This rare condition consists in the presence in the urine of pentoses, i.e. carbohydrates containing only 5 atoms of carbon.

The best test for pentoses is with Bial's reagent (1 grm. of orcin, 500 c c. of HCl, specific gravity 1151, 25 drops of 10-per-cent. solution of ferric chloride). Boil 5 c.c. of this reagent in a test-tube, *remove from flame*, add 5 drops of urine. A green ring at the junction is diagnostic of pentoses. Glycuronic acid and compound glycuronates (p. 325) give the tests for pentoses, including Bial's, but there is no risk of confusion if the test-tube is first *removed from the flame*.

Pentoses occasionally occur in diabetic (hexose) urine. They are also present after the ingestion of certain fruits (cherries, grapes, plums). But their chief interest is in connexion with so-called "pentosuria," a rare anomaly of metabolism not necessarily attended by morbid symptoms, probably harmless and needing no treatment.

(4) BILE IN THE URINE

Both bile-pigment and bile-acids may be present. Usually they occur together, but the pigment much more abundantly than the acids. The usual cause of the entrance of the bile constituents into the urine is some obstruction in the bile-passages. As long as the urine is fresh, bilirubin is the form of bile-pigment always found in it. After it has stood for some time, biliverdin is apt to be formed as the result of oxidation.

Urine which contains bile is greenish or brownish-yellow in colour, and somewhat more viscid than normal, so that the froth which forms on the top after shaking is usually permanent. Salol urine may closely resemble urine which contains bile, but the froth in the latter case is also greenish; in salol urine it is not.

Test for bile-pigment. Gmelin's test.——Place some of the urine in a conical glass, and run a little yellow nitric acid, containing nitrous acid*, down the side so as to form a layer at the bottom. Oxidation of the bile-pigment

* Nitric acid which has turned yellow after standing in the light is suitable; or a little sodium nitrite may be added to the nitric acid.

occurs, the most highly oxidized product (choletelin) forming a yellowish-red ring nearest the acid. Above this is a reddish ring, then violet (bilicyanin), and highest of all, green (biliverdin) Of these rings the green is alone characteristic of bile; all the others may be yielded by urinary indigogens.

The test, as thus carried out, is not very sensitive, and may fail even when 5 per cent. of the bile is present. The sensitiveness of the reaction can be increased by filtering the urine repeatedly through an ordinary filter-paper. The latter becomes impregnated with the bile-pigment, and if a drop of yellow nitric acid is placed upon it a play of colours can easily be seen.

The following modifications of it are much more delicate, and should always be employed in doubtful cases. They will reveal the presence of 0·2 per cent. of bile.

i. To 50 c.c. of urine add 5 c.c. of 10-per-cent. barium chloride solution and 5 c.c. of chloroform. Shake for several minutes. Set aside for ten minutes. The chloroform and precipitate of phosphates fall down, carrying with them any bile-pigment. If there is still any of the precipitate suspended, move the jar gently to and fro for a little, when it will settle down. Now draw off the chloroform and precipitate with a pipette; if some urine is removed at the same time, no matter. Place in a flat dish, and set the latter over a basin of hot water till all the chloroform has evaporated. Allow to cool, and pour off any fluid from the precipitate. The latter will be yellowish. Place yellow nitric acid in drops here and there on the surface of the precipitate. If bile-pigment is present, a play of colours appears round each drop.

ii. Render 10 c c. of the urine alkaline with caustic soda, and add a little 10-per-cent. calcium chloride solution. Collect and wash the precipitate, and place it along with the filter-paper in a small porcelain dish. Pour on it 10 c.c. of acid alcohol (5 c.c. of strong HCl to 100 c.c. of alcohol). Heat the yellowish solution in a test-tube. If bile is present, it becomes green or bluish.

Iodine test.—If a 10-per-cent. alcoholic solution of iodine is poured on the top of the urine in a test-tube, an emerald-green layer appears where the two fluids join, if bile is present.

Tests for bile-acids.—Pettenkofer's test for bile-acids is inapplicable in the case of urine, for even normal urine gives, with strong sulphuric acid, a purplish colour which might be mistaken for a positive reaction.

The simplest test is **Hay's sulphur test.**

Sprinkle some powdered sulphur upon the surface of the urine. If bile-salts are present it will sink; with normal urine it floats. This test depends upon the fact that bile-salts lower the surface tension of fluids in which they are dissolved.

(5) PUS IN THE URINE (PYURIA)

The naked-eye characters of a urine which contains pus have already been described (p. 293). On chemical examination such a urine is, of course, always albuminous. It is often difficult to decide, just as it is in hæmaturia, whether all the albumin is accounted for by the pus alone, or whether there is true albuminuria in addition. Reinecke has proposed the following method for enabling one to form a conclusion in this matter. He shakes up the urine of twenty-four hours thoroughly, so as to diffuse the pus evenly through it. He then counts the pus cells present, by means of a hæmocytometer, just as in the method for estimating the red blood-corpuscles in the blood, only without previous dilution. He finds that 100,000 pus cells per cubic millimetre should correspond to 1 per cent. of albumin (Esbach). If there is more albumin than this with that number of corpuscles, then albuminuria is present in addition to pyuria. Obviously, the method can only afford approximate indications. Moreover, it is inapplicable if the urine is ammoniacal, or if it contains much mucus. It should be added that if the number of pus cells exceeds 3,000 per cubic millimetre, the urine should be diluted with 1-per-cent. salt solution prior to counting.

Tests for pus.—As already mentioned, urines which contain pus give, on the addition of guaiac alone, a green colour, which, however, disappears upon heating.

If caustic potash is added to the deposit of pus, a ropy, gelatinous mass results.

Neither of these commonly used tests, however, is satisfactory. The recognition of pus must always be made under the microscope. (*See* p. 341.)

(6) SOME RARER ABNORMAL CONSTITUENTS OF URINE

i. **Urinary indigogens.**—We have seen (p. 298) that indol is excreted in the urine in the form of indoxyl hydrogen sulphate—the so-called "indican." Small quantities of skatoxyl hydrogen sulphate, derived from skatol, are also to be found in human urine. On oxidation these compounds yield coloured substances, indigo blue and indigo red. Hence they are called urinary indigogens. To detect their presence one may use the following test:—

Mix 5 c c. of urine with an equal volume of strong HCl coloured faintly yellow by the addition of ferric chloride solution. Stand for half an hour. Add 3 c.c. of chloroform and shake well. On settling, the chloroform becomes tinged blue if indican is present

This test may be used roughly as a quantitative one, judging the amount of indican by the intensity of the blue colour produced. It is always to be borne in mind, however, that this is liable to the fallacy that indigo red may also be produced, and thus the intensity of the colour be misleading; also, allowance must be made for the concentration of the urine.

Traces of the indigogens are normally present in the urine. The reddish-yellow transparent ring which appears above a layer of nitric acid when the latter is added to the urine is due to their partial oxidation.*

* In part also the reddish colour so obtained is due to the pigment uroiosein, which is produced from its chromogen by the action of mineral acids. Its clinical significance is unknown.

The amount of indican in the urine is greatly reduced when the protein in the food is limited.

ii. **Acetone bodies.** — Hydroxybutyric acid, aceto-acetic acid, and acetone may all occur in the urine in conditions of ketosis. The relationship between the three may be seen from the formulæ:

β-hydroxybutyric acid = $CH_3CH(OH)CH_2COOH$.
Aceto-acetic acid (di-acetic acid) = CH_3CO, CH_2COOH.
Acetone = CH_3, CO, CH_3.

β-hydroxybutyric acid is formed first; it then becomes oxidized, yielding aceto-acetic acid,

$$CH_3CH(OH)CH_2COOH + O = CH_3COCH_2COOH + H_2O$$

The aceto-acetic acid is very easily decomposed into acetone and CO_2,

$$CH_3COCH_2COOH = CH_3COCH_3 + CO_2.$$

It is even asserted that acetone only appears after the urine has been passed.

Test for acetone bodies.—The urine must be fresh and unboiled, as aceto-acetic acid readily decomposes. **Rothera's nitro-prusside test** is performed first, as follows :—

Ten c.c. of the urine are saturated with ammonium sulphate by adding 5 grm. of the crystals; 3 drops of 5-per-cent. sodium nitro-prusside and 2 c.c. of strong ammonia are then added. A fine permanganate colour is produced. This test is given both by acetone and aceto-acetic acid.

If Rothera's test is negative, acetone bodies are absent; if positive, aceto-acetic acid may be tested for by the **ferric chloride reaction (Gerhardt's test),** as follows :—

Take some urine in a test-tube, drop in a solution of perchloride of iron, diluted until it is of a pale sherry colour, as long as a precipitate of phosphate of iron falls. Filter, and add to the filtrate another drop or two of the iron solution. The solution becomes brownish-red if aceto-acetic acid is present.

Antipyrin, salol, salicylates, carbolic acid, and some other

drugs give a similar colour with ferric chloride. Prolonged boiling (before adding the ferric chloride) destroys aceto-acetic acid while the other substances which give a colour with ferric chloride are unaffected. If, therefore, urine which has been subjected to prolonged boiling still gives the ferric chloride reaction, it may be inferred that the reaction was not due to aceto-acetic acid. Boiling *after* adding ferric chloride destroys the colour, whether this is due to aceto-acetic acid or to other substances. The colours produced by solutions of salicylates in water with ferric chloride do not resemble that given by aceto-acetic acid. But in urine the colours may be indistinguishable.

A positive ferric chloride reaction is only obtained if aceto-acetic acid is present in considerable amount. If, therefore, the urine reacts to Rothera's test but not to ferric chloride, it may be inferred that only small quantities of acetone bodies are present.

iii. **Glycuronic acid** is probably derived in the body from dextrose. Traces of it exist in combination in normal urine. It is very prone to form ethereal or glucosidal compounds if suitable substances are introduced into the circulation. Hence it appears in the urine in considerable quantity, in paired combination with aromatic substances, etc., after the administration of such drugs as chloral, benzoic acid, chloroform, morphia, etc. This circumstance gave rise to the old belief that such drugs produce glycosuria; in reality, the substance which is excreted after their use is glycuronic acid, not glucose.

Occasionally glycuronic acid occurs spontaneously in the urine and is then apt to be mistaken for glucose. Glycuronic acid reduces Fehling's solution but not Benedict's and gives a yellow crystalline precipitate with the phenyl-hydrazine test. It can also be distinguished from glucose in the following ways:—

(*a*) It does not ferment with yeast.

(*b*) It gives a deep-red colour with phloroglucin and HCl.

(*c*) It does not reduce Bial's reagent when the urine is added to the boiling reagent (cf. Pentoses p. 320).

(*d*) It gives the *naphtho-resorcinol reaction*. To 5 c.c. of urine in a wide test-tube add 0·5 c.c. of a 1-per-cent. solution of naphtho-resorcinol, and 5 c.c. of strong hydrochloric acid. Heat slowly to boiling, keep boiling for one minute, shaking meanwhile. Let stand for four minutes; then cool under a tap. Shake with an equal volume of ether. If glycuronic acid is present the ether will be coloured violet to red, and will show two absorption bands, one on the D line and one further towards the green.

iv. **Cystin** is sometimes found as a deposit in acid urines. It is recognized by its characteristic crystals (*see* Fig. 71, *b*, p. 338). It is soluble in alkalis; hence the deposit disappears when the urine putrefies, an odour of sulphuretted hydrogen being evolved. If urine which contains it is boiled with a little caustic potash and acetate of lead, a black precipitate of sulphide of lead appears.

Cystin is a product of protein metabolism, and is probably derived from cystein, the normal oxidation of which has somehow been interfered with. It is often associated with the appearance in the urine of diamines, such as cadaverin and putrescin. It is about twice as common in males as in females, and has rarely been observed after 50 years of age. It is apt to occur in several members of the same family and may appear either persistently or intermittently. It is of interest as being due to an inborn error of metabolism, but is of little pathological significance, except from its tendency to form calculi.

(7) DRUGS IN THE URINE

Antipyrin.—After its use the urine may be red and dichroic, leading to the suspicion that blood is present. On adding a little dilute perchloride of iron a purplish-red colour develops, which persists on boiling, but disappears on adding an acid. Urines containing antipyrin produce a partial reduction of Fehling's solution on boiling.

Bromides.—Add a little hydrochloric acid and a few drops of a weak solution of bleaching powder. Shake with chloroform, and the latter becomes brownish-red from the solution of the free bromine.

Carbolic acid (*see also* section on Colour of Urine, p. 287).—The best test for it is to add a little bromine water. The appearance of a whitish precipitate (tribromophenol) indicates the presence of phenol.

Chloral, chloroform, etc., may lead to the appearance of glycuronic acid (p. 325).

Iodides.—Acidify the urine with a little pure nitric acid, and shake up with chloroform. The latter becomes of a rose-red colour.

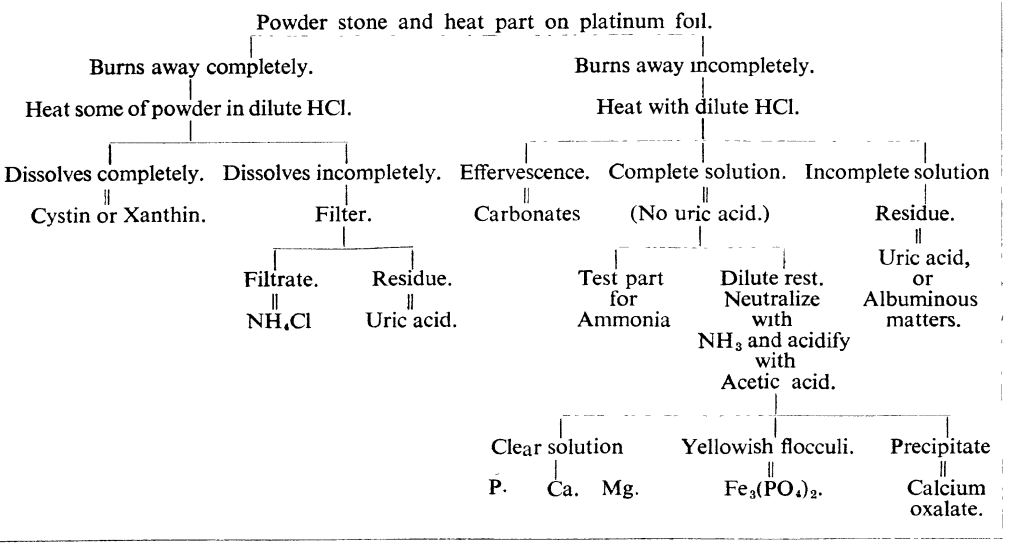
Iron.—Add a few drops of nitric acid. Boil, cool, and add a little 10-per-cent. ferrocyanide of potash. A precipitate of Prussian blue forms if iron is present.

Rhubarb and **santonin** have been referred to under Alterations in Colour of Urine (p. 290).

Salicylates and **salol** appear in the urine as salicyluric acid. Such urines give a bluish-violet colour on the addition of a little ferric chloride if large amounts are present; if small amounts only are present the colour may be reddish-brown, indistinguishable from that given by aceto-acetic acid. These urines also partially reduce Fehling's solution.

Tannin gives a bluish-black colour with ferric chloride.

Scheme for Analysis of Urinary Calculi. (*After Salkowski*)



(8) ESTIMATION OF DIASTASE IN THE URINE

Prepare a 0.1-per-cent. solution of soluble starch, a 1.0-per-cent. solution of NaCl, and a $\frac{1}{50}$N iodine solution in distilled water.

About 3 c.c. of urine are required, preferably taken from a twenty-four hours' specimen.

Label a series of ten clean test-tubes from 1 to 10. With a 1-c.c. pipette graded in hundredths, measure 0.6 c.c. of urine into tube 1, 0·5 c.c. into tube 2, 0·4 c.c. into tube 3, and so on, down to tube 10, which will contain 0 06 c.c. of urine. The measurements of urine after 0·1 c.c. are best made from a 1-in-10 dilution of the urine with the 1-per-cent. salt solution. The amount of fluid in each tube is then made up to 1 c.c. with the salt solution.

Add 2 c.c. of the starch solution to each tube and shake.

Place the tubes in a water-bath at 38° C. for half an hour, then in cold water for three minutes. Add one drop of the iodine solution to each tube and observe which tube in the series is the first to show a blue tint, or in other words contains a trace of undigested starch.

Normally, tube 6, containing 0.1 c.c. of urine, would be the first tube to show a blue colour, in which case 0·2 c.c. of urine can digest 2 c.c. of 0·1-per-cent. starch in 30 minutes at 38° C.

$$\therefore \text{ 1 c.c. of urine can digest } \frac{20}{2} = 10 \text{ c.c.}$$

which can be expressed as the figure $d\,\frac{38^\circ}{30} = 10$

$$\text{or } d = 10 \text{ units.}$$

In health the d value varies from 6·6 to 33·3 units; in acute pancreatitis it may reach 400 units. It is low in nephritis with much impaired renal efficiency.

(9) THE PHENOL-SULPHONE-PHTHALEIN TEST FOR RENAL EFFICIENCY

The principle of the test is to estimate the amount of the dye phenol-sulphone-phthalein excreted in two hours after the injection of 6 mg.

In order to secure a good flow of urine, 400 c.c. of water are given to drink. After about 15 minutes the patient's bladder is emptied, if necessary by catheter, and 6 mg. of the dye dissolved in 1 c.c. of water are injected intramuscularly.

After exactly one hour, and again after two hours, the bladder is emptied, and the whole of these two specimens reserved for estimation of the dye colorimetrically in each.

The urine, being usually acid, will not be coloured red by the dye until alkali is added. Each specimen is measured, made strongly alkaline with 40-per-cent. NaOH, made up to 500 c.c. if the red colour developed is deep, to 250 c.c. if not deep, and filtered to remove the precipitate of phosphates.

To serve as a standard with which to compare the urines, 1·5 c.c. of a 0·02-per-cent. solution of phenol-sulphonephthalein is made alkaline and brought up to 50 c.c.; this gives a concentration equal to 6 mg. in 1,000 c.c. This standard has to be matched against a solution in urine, and, in order to allow for the yellow colour of the urine, urine should be used in making up the standard. The most convenient way is to make up two solutions, A and B.

A. Add 1·5 c.c. of 0·02-per-cent. solution to 40 c.c. of distilled water, add 1 c.c. of 40-per-cent. NaOH, and make up to 50 c.c. with distilled water.

B. Add 1·5 c.c. of 0·02-per-cent. solution to 40 c.c. of normal urine, less if highly coloured, 1 c.c. of 40-per-cent. NaOH, make up to 50 c.c. with distilled water, and filter.

These solutions contain the same amount of the red dye, and by mixing them in various proportions it is possible to obtain a standard containing the right amount of yellow pigment to match the urines to be investigated.

Such a mixture of A and B having been made suitable for the first-hour urine, the standard and urine are put in the two pots of a colorimeter, the standard set at 20 mm., and the depth of the unknown required to give an equal depth of colour read.

Supposing this urine has been diluted to 250 c.c. and the reading observed is Y:

Then,
Depth of colour in unknown $\times \frac{Y}{20}$ = depth of colour in standard.

$\therefore$ amount of dye in 250 c.c. unknown $\times \frac{Y}{20}$ = amount of dye in 250 c.c. of standard.

$= \frac{250}{1000} \times 6$ mg., or 25 per cent. of the original dose.

$\therefore$ amount of dye in the unknown $= \frac{20}{Y} \times 25$ per cent. of original dose; if the urine was diluted to 500 c.c. the amount of dye it contained would be $\frac{20}{Y} \times 50$ per cent. of the original dose.

The percentage of the original dose in the second-hour specimen is estimated in the same way.

If a colorimeter is not available, the estimation may be performed by dilution, as follows:—

In two similar test-tubes of equal bore are placed 10 c c. of the unknown and of the standard, and distilled water is added from a burette till the solutions match.

Suppose Y c.c. are added to the standard:

Then

depth of colour in unknown $\times \frac{10 + Y}{10}$ = depth of colour in standard.

$\therefore$ dye in 250 c c. unknown $\times \frac{10 + Y}{10}$ = dye in 250 c.c. standard.

= 25 per cent. of original dose.

$\therefore$ dye in 250 c.c. unknown $= \frac{10}{10+Y} \times 25$ per cent. of original dose.

The amount of the original dose excreted in the first hour by a normal adult is usually over 40 per cent., and in two hours together over 60 per cent.

(10) UREA-CONCENTRATION TEST

The concentration of urea in the urine, taken under ordinary conditions, is of little value as an index of the efficiency of the kidneys, since a normal person may excrete a urine of low urea-concentration

when the amount of urea formed is small or the volume of water passed is large.

To make a test of more value, MacLean recommends giving 15 grm. of urea in 100 c.c. of water flavoured with a little tincture of orange, by the mouth, when the patient has had nothing to drink for some hours, and after emptying the bladder; the urine passed in each of the subsequent three hours is collected and the urea-concentration estimated preferably in the specimens from the second and third hours. The urease method is more satisfactory than the hypobromite, but the latter may be used.

If the kidneys are healthy the concentration in the second and third hours is usually over 2·5 per cent. and almost invariably over 2·0 per cent. unless the volume is large (over 150 c.c. per hour). With moderate damage to the kidney, concentrations from 1·5 to 2·5 per cent. may be encountered, and with severe damage under 1·5 per cent. A certain amount of judgment is required in interpreting the result; such high concentrations cannot be expected when the initial blood-urea is low (e.g. about 0·020 per cent.) as when it is comparatively high (e.g. about 0·045 per cent.); nor such high concentrations when the volume passed is comparatively large (e.g. 130 c.c.) as when it is small (e.g. 50 c.c.).

This test is a useful complement to the phenol-sulphone-phthalein test, as the latter sometimes gives low figures when the kidneys are little affected. If low figures for phenol-sulphone-phthalein are accompanied by low figures for urea-concentration, there is little doubt that the kidneys are deficient. On the other hand, the phenol-sulphone-phthalein test rarely gives normal figures when the real efficiency is reduced, and high figures for phenol-sulphone-phthalein confirm the urea-concentration test in its doubtful zone between 2·0 and 2·5 per cent.

The two functions, of excreting urea and similar substances on the one hand, and water and chlorides on the other, can apparently be reduced independently. In chronic interstitial nephritis the first func-

tion, which is that estimated by the tests given above, is the one mainly affected (azotæmic type). In some cases of parenchymatous nephritis the second function alone is reduced (hydræmic type); but in most cases clinically diagnosed as parenchymatous nephritis the first function is impaired also. In acute nephritis both functions are affected.

The following are typical results of these tests.

	Phenol-sulphone-phthalein	*Urea-concentration*	*Blood-urea*
CHRONIC "INTERSTITIAL" NEPHRITIS:			
MODERATE	30	1.6 %	0.045 %
SEVERE	0	1.1 %	0.140 %
CHRONIC "PARENCHYMATOUS" NEPHRITIS:			
HYDRÆMIC	50	3.5 %	0.040 %
MIXED	33	1.8 %	0.051 %
ACUTE NEPHRITIS	16	1.4 %	0.095 %

III. MICROSCOPICAL EXAMINATION OF URINARY DEPOSITS

The examination of deposits in urine and other fluids liable to decomposition is greatly facilitated by

using a centrifuge. For ordinary clinical purposes, Beck's is perhaps as convenient as any; where a large number of examinations are undertaken it is better to employ one driven by electricity or water power.

A drop of the deposit is placed on the centre of a slide and covered with a cover-glass. The preparation is then examined with both the low ($\frac{1}{2}$ in. or $\frac{2}{3}$ in.) and high ($\frac{1}{6}$ in.) objectives, the microscope being vertical and the diaphragm partly closed.

Unorganized Deposits

The first group of urinary deposits includes the various salts and crystalline substances that are found in urine, either when freshly voided, or more often

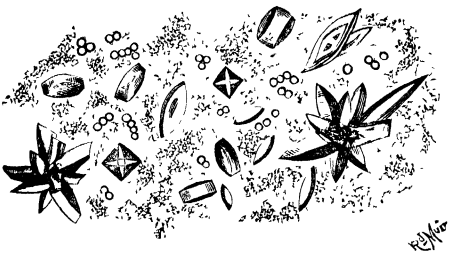


Fig. 67.—Deposit in acid urine.

when it has stood for some time. The following occur in **acid** urine (Fig. 67) :—

1. **Uric acid.**—This appears under a variety of forms, and, unless the urine is almost devoid of colouring matter, assumes a reddish-brown colour in consequence of its absorbing a considerable amount of pigment. To the naked eye the appearance resembles that of a shower of grains of cayenne pepper collected at the bottom of the specimen. Under the microscope the crystals are either rhombic prisms or some

modification of that form. Often the more obtuse angles are rounded off and the edges continued in curved lines, so that pointed oval shapes result. Numerous crystals may be joined together to produce rosettes and other composite forms. Some of the more common are represented in the accompanying figure (Fig. 68).

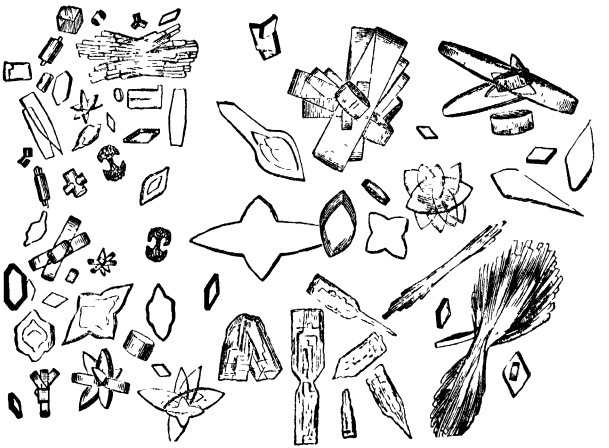


Fig. 68.—Uric acid.

2. **Urates.**—These are urates of potassium, sodium, and ammonium. They have a considerable affinity for the urinary pigments, and hence are generally more or less pink or brick-coloured. In very pale urines they are colourless, and resemble rather closely a deposit of phosphates. Microscopically they consist of small granular particles, arranged in moss-like clumps. Ammonium urate may form spheres, either solitary or in clusters, having a more or less crystalline structure, with numerous spines

radiating from their surface (Fig. 69). On heating a urine from which they have separated out, they will be found to redissolve before the boiling-point has been reached.

Uric acid and urates can be preserved in Canada balsam—the water being got rid of by passing them through alcohol, then letting a drop dry on the slide, and adding balsam in xylol.

3. **Hippuric acid** appears in human urine chiefly after the administration of benzoic acid or its salts. It occurs as colourless four-sided prisms, insoluble in hydrochloric acid, but soluble in ammonia.

4 **Calcium oxalate.**—This deposit is rarely abundant. The small colourless crystals lying on the top of the numerous deposit that settles at the bottom of the urine glass give the impression of an undulating snowy surface. They also adhere to irregularities on the surface of the glass, producing the appearance of scratches. Two forms are found under the microscope. The first, which is by far the commoner, consists of small octahedral crystals. When, as is generally the case, they are slightly flattened along one axis, they appear like squares crossed by two diagonal lines, or like long octahedra, according as the short axis lies in or perpendicular to the line of sight. The other form in which oxalates occur is that of minute dumb-bells or oval biscuit-shaped discs. Some writers consider that this form is not really due to calcium oxalate but



Fig. 69.—Ammonium urate.

to calcium carbonate; yet, though carbonates frequently enough assume this shape, there can be little doubt that, under certain conditions, oxalates do so too. For permanent specimens octahedral oxalates are best mounted in glycerin jelly, dumb-bell oxalates in balsam (Fig. 70).

5. **Cystin** is a rare deposit in human urine, but when it occurs the precipitate is often copious, and is



Fig. 70.—Calcium oxalate.

not unlike a sediment of fawn-coloured urates. It is soluble in alkalis and in strong acids (such as hydrochloric), but insoluble in acetic acid. It does not separate out from alkaline urine, but the addition of a few drops of acetic acid may determine its precipitation. From urine it is deposited as hexagonal tablets, soluble in ammonia, and, when the ammonia evaporates, recrystallizing as hexagons or prisms (Fig. 71, *b*).

6. **Xanthin** is of extremely rare occurrence; the crystals are said to be similar to " whetstone " crystals of uric acid, but are soluble in ammonia, in warm hydrochloric acid, and in nitric acid.

7. **Tyrosin** is generally found associated with leucin, but occurs independently also. It forms colourless sheaves of fine needle-like crystals. A similar appearance may be presented by several other deposits; therefore, if there is any doubt as to the nature of the sediment, a chemical analysis may be necessary (Fig. 71, *a*).

8. **Leucin** occurs in urine as yellow spherical masses without obvious crystalline structure. Leucin

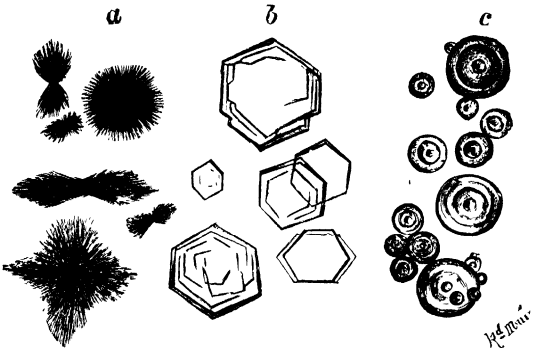


Fig. 71.—*a*, Tyrosin crystals ; *b*, cystin ; *c*, leucin.

and tyrosin are found together in acute yellow atrophy of the liver (Fig. 71, *c*).

In **alkaline** urine the following occur:—

1. **Phosphates.**—These may be salts of phosphoric acid and calcium, or of phosphoric acid with ammonium and magnesium.

(a) **Phosphate of lime** is found either in an amorphous or in a crystalline form, the latter being also known as *stellar phosphates* (Fig. 72).

Amorphous phosphate of lime occurs in small white granules as a deposit at the bottom of alkaline urine.

To the naked eye the sediment is white and flocculent; unlike urates, it has no affinity for uninary pigment. The deposit is increased on heating.

Stellar phosphates are rather uncommon. They consist of colourless prismatic crystals, which occur either singly or more often in radiating clusters. They are found in very faintly acid as well as in neutral and alkaline urine.

(b) **Ammonium magnesium,** or **"triple" phosphate,** is deposited in ammoniacal states of the urine.

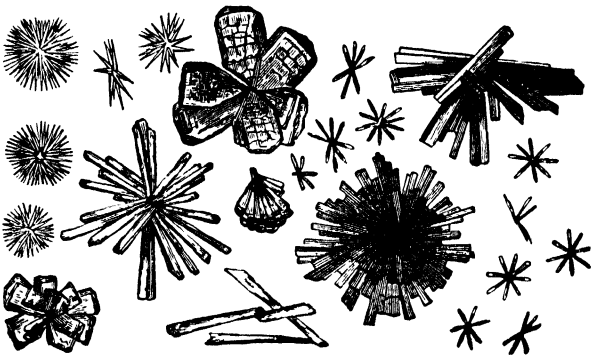


Fig. 72.—Stellar phosphates.

To the naked eye the sediment appears very white, and when the crystals are large they may be visible as bright points. Sometimes the deposit also clings to the sides of the glass and forms a film on the surface of the urine.

The crystals are incomplete, triangular, colourless prisms, which may offer considerable variations in appearance, according to their length and degree of perfection. Often they are described as "knife-rest" or "coffin-lid" crystals. If the ammoniacal

change is well marked, and still more if excess of ammonia is added to healthy urine, the deposit takes the form of feathery stars, and is then known as a precipitate of "feathery" phosphates (Fig. 73). All the phosphate deposits are dissolved on adding acid.

It is difficult to keep these crystals permanently, but they may be preserved fairly well in a solution of ammonium chloride.

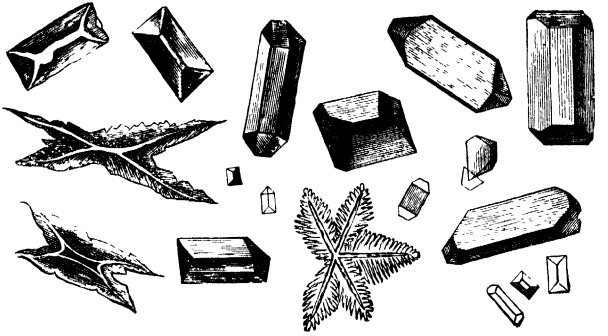


Fig. 73.—Triple phosphates.

2. **Ammonium urate** occurs in alkaline urine, and is very commonly present in cases of cystitis. Microscopically, it occurs in small spherical masses, which may have smooth surfaces, or they may be beset with innumerable spiny processes (Figs. 69 and 74).

3. **Carbonates** generally occur in human urine as granular particles, which dissolve in acetic acid with evolution of CO_2. As phosphates give off no gas on solution in acetic acid, it is quite easy to distinguish between them. On rare occasions in

human urine, and commonly in horses' urine, calcium carbonate appears in the form of dumb-bells or of spheres with a radiating crystalline structure.

4. **Cholesterol** has occasionally been found in the urine; it occurs in characteristic thin, rhomboidal, colourless plates, with a notch at one of the corners.

Other sediments, such as indigo, lime and magnesia, soap crystals, and hæmatoidin, have been observed, but are of little importance.

Organized Deposits

1. **Red blood-corpuscles** are best examined for in recently voided urine. Usually of normal size, they may, according to the density of the urine, appear either swollen or shrunken and crenated. They are to be recognized by their shape, colour, and biconcavity, and in cases of doubt a dried preparation should be made and stained by one of the blood stains, such as Leishman's. Droplets of oil from a catheter are frequently mistaken for red blood-corpuscles. The droplets are readily differentiated by their higher refractive index, more circular shape, and variable size.



Fig. 74.—Deposit in alkaline fermentation of urine, showing ammonium urate, triple phosphates, and bacteria. × 200.

2. **Leucocytes** and **pus-corpuscles** occur where there is irritation and suppuration in the urinary tract. According to the length of time which has elapsed, the

cells may be indistinguishable from ordinary leucocytes, or they may be very granular and fatty. The addition of acetic acid clears up the cell body and brings two or three nuclei into view. Where pus is present, examine carefully for pathogenic microbes, especially for gonococci and tubercle bacilli.

3. In cases of **chyluria** the urine contains nucleated granular corpuscles similar to leucocytes, and very finely divided fatty material, which appears simply granular under the microscope. A few red blood-corpuscles are often present. The urine and blood should be carefully examined for the presence of filariæ, particularly if the patient comes from a district where these parasites exist.

In **lipuria** the fat may occur in larger globules which refract light strongly, and which are sometimes free in the fluid, at other times enclosed in cells or tube-casts.

It must not be forgotten that fatty matter may reach the urine unintentionally from an oiled catheter, or may be added purposely in the form of milk by a patient who wishes to deceive the physician.

4. **Epithelium** from various parts of the urinary tract may be found in the urine. The following varieties are readily recognized:—

(*a*) *Renal epithelium.* This is polygonal, nucleated, and rather larger than a leucocyte. It may present fatty degeneration, or be more or less disintegrated.

(*b*) *Epithelium from the bladder and urinary passages* presents various appearances, according to whether it is derived from the more superficial or the deeper layers. Formerly, tailed cells were thought to indicate implication of the pelvis of the kidney; this is, however, inaccurate. They may equally well proceed from the deeper layers of the bladder epithelium.

(*c*) *Vaginal epithelium* is very commonly present in the urine of women. It is squamous, and the large cells appear sometimes singly, at other times in groups.

5. **Spermatozoa** occur at times in the urine, where their characteristic appearance makes it easy to recognize them.

6. **Prostatic threads** are found when there is chronic inflammation of the prostate, especially after gonorrhœa. They consist of mucus with entangled leucocytes and epithelial cells, and are mostly voided with the first portions of the urine. They are much larger than tube-casts, being visible readily enough to the naked eye as they float in the urine or on its surface.

7. **Tube-casts.**—The following classification may be adopted:—

i. Casts wholly or mostly composed of cellular structures.
ii. Granular casts.
iii. Amorphous casts, having a homogeneous structure, and occasionally striated on the surface.
iv. Lipoid casts, containing fatty material.

i. **Cellular.**—The cells may be epithelial or composed of red blood-corpuscles or leucocytes.

(*a*) *Epithelial.*—The casts may be completely covered with epithelial cells, as though the whole epithelium had scaled off a tubule, or the cells may have been separately detached and subsequently moulded. The cells may or may not show a nucleus, and they may appear fresh, or affected by granular or fatty degeneration.

(*b*) The *red blood-corpuscle* casts exhibit a surface thickly covered with the minute round corpuscles.

(*c*) *Leucocytes* rarely form casts by themselves, but are fairly often found adhering to the surface of other casts.

ii. **Granular.**—The granules are sometimes coarse, at other times fine. They are sometimes fatty, at other times they result from granular degeneration of protoplasm. They represent in some instance the relics of broken-down epithelium, in other cases they result from a granular change occurring in old amorphous tube-casts.

iii. **Amorphous.**—This group contains two varieties, the hyaline and the colloid.

(*a*) *Hyaline* tube-casts are pale, transparent, and homogenous. Occasionally the surface is striated. They may be almost invisible, but are rendered more prominent by the addition of iodine solution.

(*b*) *Colloid* casts are broader and more highly refractile than hyaline. Often they are more or less fissured. Possibly they may be formed from other casts which have remained long in the urinary tubules; they are *not* symptomatic of *amyloid* disease of the kidney.

iv. **Lipoid.**—These are found when the tubular cells are undergoing lipoid degeneration. Clear drops of lipoid material may be seen in the casts.

Transition forms between the various groups are not uncommon; often, for instance, a cast is partly epithelial and partly hyaline.

Casts dissolve in alkaline urine and may easily be missed in a stale specimen of urine. A tube-cast frequently picks up adventitious elements from the urine, and thus comes to contain bacteria, or crystals such as calcium oxalate.

In length, tube-casts are very variable; occasionally they approach 1 mm. in length. One end may

be spirally twisted, or in rare cases bifurcated. Formerly tube-casts were called "fibrin cylinders," but now it is proved that fibrin rarely or never enters into their composition, the only exception being that, in the case of red blood-corpuscle casts, the blood discs may be bound together by a little fibrin.

It appears that tube-casts, including their matrix, are essentially products of the cells which line the kidney tubules, and that the relative proportion of epithelial, granular, and hyaline casts is greatly influenced by the reaction and constitution of the fluid which bathes them. An acid urine yields, as a rule, granular casts, but, if the acidity is very high, casts which appear hyaline. An alkaline urine renders the majority of the casts hyaline. On the other hand, the salts of the urine, apparently by precipitating colloid material, tend to preserve the granular aspect of the casts, and to make them smaller by causing the colloid material to lose water.

Structures called **cylindroids** have been described by Thomas and others. They resemble extremely long and narrow tube-casts, but are usually considerably flattened. Some observers regard their presence as quite immaterial; others look on them with considerable suspicion as being nearly related, both in origin and clinical import, to tube-casts.

Not to be confounded with either tube-casts or cylindroids are the little strings of mucus which are occasionally present in a urinary sediment. Small clumps of micrococci and short so-called "prostatic threads" are also liable to be misinterpreted by an inexperienced observer. When there is reasonable cause for doubt, the addition of acetic acid or some other reagent will often settle the question.

8. Tumours of the bladder, especially when villous, may very rarely be detected by the presence of **fragments of the growth** in the urine. These show a core of connective tissue with its blood-vessels, coated with several layers of nucleated epithelial cells.

9. **Elastic fibres** may be present in cases of ulceration of the bladder. They may be detected either without special treatment, or after the use of caustic soda (as is described on p. 282), preceded by a preliminary filtration of the yet acid urine to remove the phosphates, which would otherwise be precipitated copiously when an alkali was added.

The following **parasites** may occur in the urinary tract: Echinococcus, *Cysticercus cellulosæ Eustrongylus gigas*, *Schistosoma hæmatobium*, *Filaria bancrofti*, *Nephrophages sanguinarius*, and certain psorosperms. Several of these are so rare as to be of no practical importance; echinococcus, cysticercus, and filaria having been described elsewhere, *Schistosoma hæmatobium* alone needs to be referred to here.

S. hæmatobium—The ova measure 0·12 mm. by 0·04. A spine projects at one pole or at a little distance from it. In urine the spine is usually situated at the pole; the form with a lateral spine (*S. mansoni*) predominates in ova obtained from the rectum.

The adult male parasite is thicker and shorter than the female, and is provided on the ventral surface with a gynæcophoric canal. The female is cylindrical and worm-like. The male measures 12 mm., the female about 16 mm., in length. Their habitat is in the blood-vessels of the portal system and in the venous plexuses of the bladder and rectum. The ova escape from the blood-vessels into the tissues of the body. Those which reach the rectum and bladder are discharged, and enable a diagnosis to be made. The parasite is very common in Egypt.

After the urine has been voided for some time, it becomes contaminated by numerous non-pathogenic fungi and infusoria; but several pathogenic *bacteria* occur in the urinary tract, and in cases of doubt should

always be sought for. The chief of these are the gonococcus, the tubercle bacillus, which must not be mistaken for the morphologically similar smegma bacillus, and the Bacillus coli communis.

In cases of cystitis a great variety of bacteria may occur. Actinomyces, though rarely present in the kidney, has been found there.

Foreign bodies often occur in urine which has been set aside for examination. Besides hairs, feathers, moth-wing scales, cotton, woollen, and silk fibres, starch grains derived from dusting powders (readily recognized by their turning blue on the addition of a little dilute tincture of iodine), and, more confusing than any of these, pinewood dust swept from the floor, one occasionally finds fragments of the contents of dermoid tumours or abscesses that have opened into the bladder or ureter.

It may happen also that the patient has vomited, and sputum or vomited matter may be more or less abundantly mixed with the urine.

CHAPTER VIII

THE SKIN

FOR the examination of the skin and its appendages the patient should be stripped as completely as circumstances permit and placed in a good light.

One should first note the **colour** of the skin as a whole. In anæmia the skin is pale; in chlorosis it has a greenish tint; in pernicious anæmia it is lemon-yellow. In order to distinguish the yellowness of pernicious anæmia from jaundice, look at the conjunctiva. The best way to do that is to place one hand on the patient's forehead, ask him to look at the ground, and then raise the upper eyelid with the thumb. In jaundice the conjunctiva is seen to be yellow where it covers the sclerotic; in anæmia it is white. In judging of the degree of anæmia, one should be guided more by the colour of the mucous membranes than by that of the skin itself. The conjunctiva lining the lower eyelid is usually taken as an index. It is easily seen by getting the patient to look up while one depresses the lower lid with one finger. Instead of being pale, the skin may be abnormally red or *flushed*. The flushing may be general or local. Its exact extent should always be noted, and whether or not it fades on pressure. The best way of telling whether any redness of the skin fades on pressure, or not, is to place a lens on the skin and press it down. It will then be seen whether or not the skin becomes pale under the lens.

The term **tache cérébrale** is applied to the red flush which appears in some cases of intracranial

disease when the skin is stimulated. To elicit its presence, draw the finger-nail firmly across the patient's forehead. A red line soon develops along the track of the nail, and persists for some time. It is due to a disordered vaso-motor supply, but is found in other conditions besides those of cerebral irritation, and is therefore not of much diagnostic value.

Taches bleuâtres are steel-blue spots, which may occur in large numbers, usually on the trunk and thighs. They are deeply placed in the skin, irregular in outline, and without diagnostic significance. They are probably always associated with the presence of pediculi.

Rarer alterations in colour of the skin are those due to the taking of nitrate of silver and those which occur in Addison's disease. The former constitutes what is known as **argyria.** It consists in a leaden-grey hue of the whole skin, which is unaffected by pressure. The pigmentation of **Addison's disease** consists in a bronzing, which appears first on parts in contact with the air, and next on those which are exposed to pressure. It is made up of small brownish spots, which fade off at their margins into healthy skin. The lips and buccal mucous membrane should always be examined in cases of supposed bronzing. In Addison's disease they often exhibit marks of pigmentation of a dark bluish-black colour, which have been compared to the stains produced by sucking a pen. Diffuse pigmentation of the skin is also a common occurrence in pregnancy (chloasma), and in many cases of pulmonary tuberculosis, and also after the prolonged administration of arsenic.

Having noted any alteration in the colour of the skin, one should look for the presence of any **eruption.** If any such is observed, the patient should be questioned about it on the lines laid down on p. 11.

The exact situation and extent of the eruption should be noted, and whether it is symmetrical or confined to one side only. These general facts having been noted, one should pass to a description of the minute characters of the eruption. In order to do this, it must be borne in mind that every cutaneous eruption consists of a **primary lesion,** to which secondary lesions may or may not be superadded. The following is a description of the different primary lesions which may be met with:—

1. **Macules** (spots).—Any abnormal change in the colour of the skin confined to a limited area. Always note whether or not they fade on pressure. The spots of typhoid fever, for example, fade on pressure, whilst those due to hæmorrhages into the skin, as in the bites of fleas, do not.

2. **Papules.**—Solid projections above the surface which are not larger than a pea. The term **tubercle** or **nodule** is applied to any solid projection from the skin which is larger than a pea, but not larger than a cherry. Anything larger than that is called a **tumour.** Always note whether the top of a papule is rounded as in some forms of eczema, pointed as in acne, or flattened as in lichen. As regards the base, observe whether it infiltrates the skin widely or not. The wider the infiltration, the more extensive and severe the inflammation.

3. **Vesicles.**—Elevations of the horny layer of the epidermis by transparent or milky fluid which are not larger than a pea. If larger than that they should be described as **bullæ** or **blebs.** Always note whether or not there is an area of redness around the base of a vesicle, for such redness indicates that the vesicle is planted upon an inflamed base—a fact which may be of diagnostic value.

4. **Pustules.**—Small elevations of the skin

containing pus. Always observe whether there is much infiltration around them or not.

5. **Wheals.**—Slightly elevated portions of skin, the centre of which is paler than the periphery.

Having stated which of these primary lesions it is that composes the eruption, one should next note whether the lesions are isolated (discrete), or whether they run together. It must also be remembered that an eruption may be made up of more than one kind of primary lesion. Thus, papules may be mingled with pustules, or pustules with vesicles, and so on.

Next look for **secondary lesions.** These are either produced mechanically, or are the result of changes which take place in the primary lesion in the course of its growth or decline. The commonest secondary lesions of mechanical production are **excoriations** due to scratching, and **fissures** (rhagades)—deep cracks going down to or through the corium, and produced by the stretching of the skin after it has become inelastic owing to infiltration. Fissures are often very painful.

The following are the secondary lesions produced by changes in those which are primary:—

i. **Desquamation.**—If the primary lesion is a dry one (macules or papules), a mere scaling off of epidermic cells occurs, and the eruption is then said to be "*scaly.*"

In moist lesions (vesicles, pustules, bullæ) the epidermic cells become glued together by the dried fluid, and a **scab** or **crust** forms. The scab may be serous, purulent, hæmorrhagic, or sebaceous, according to the nature of the contents of the primary lesion.

ii. **Infiltration** may occur around the primary lesions, leading to the production of a leathery feeling

in the skin. This is usually the result of prolonged chronic inflammation.

iii. **Pigmentation** may occur around the primary lesions. This also is usually due to prolonged inflammation.

iv. **Ulceration.**—Due to breaking down of the primary lesions and destruction of a part of the true skin.

The points to note in describing an ulcer are (1) the nature of the floor of the ulcer and the granulations covering it; (2) the character of the edge—smooth, raised, undermined, etc.; (3) the discharge, whether serous, purulent, watery, fetid, etc.; (4) the character of the surrounding skin, whether indurated, pigmented, etc.

v. **Scar-formation.**—This only occurs where the true skin has been involved, i.e. where there has been an ulcer. Describe the scar, noting especially whether it is thin or thick, freely movable or adherent to the deeper tissues, pale or livid, pitted or not, surrounded by a zone of pigmentation or not.

Proceed now to the **palpation** of the skin. Pass the hand gently over it, pinching it up between the forefinger and thumb, and note the following points:—

Is it smooth or rough, thin or thick, dry or moist? If there is any visible sweating, note whether it is general or local; whether it is attended or not by any flushing of the skin; and whether the sweat has any particular odour.

The **elasticity** of the skin should be investigated. If a fold of healthy skin is pinched up, it immediately flattens itself out again when released. Sometimes, however, it only does so very slowly, remaining for a considerable time in a creased condition. This indicates a diminished elasticity. It occurs not infrequently

in debilitated and in old persons. It may also be often observed in babies exhausted from diarrhœa.

The condition of the **subcutaneous tissue** should also be investigated. It may be infiltrated with fluid (œdema), with solid material (as in myxœdema), or with air (subcutaneous or surgical emphysema). The presence of **œdema** is usually recognized by the fact that if the skin is pressed with the finger, especially over a hard body such as a bone, a pit is left which persists for some little time. It must be borne in mind, however, that this is not an invariable guide. In some cases of œdema no pitting can be produced. This is especially apt to be the case where the œdema is of very long standing. The best place to look for slight degrees of œdema in cardiac disease is behind the malleoli of the tibia and fibula. In chronic renal disease, œdema can often be earliest detected beneath the conjunctiva. This subconjunctival œdema is seen by pushing up the lower lid over the sclerotic. A little drop of fluid resembling a tear is then squeezed up underneath the conjunctiva over the sclerotic.

Subcutaneous emphysema gives rise on palpation to a crackling sensation, which has been compared to that which is experienced in handling a bag of feathers. It starts in, and is usually confined to, the neighbourhood of the air-passages or air-containing organs, and is due to the establishment of an abnormal communication between such a passage or organ and the subcutaneous tissue. In rare cases it may be of bacterial origin.

Microscopical examination of the skin and its appendages is confined to the diagnosis of some **parasitic diseases,** of which the following are the chief:—

1. **Scabies or itch.**—This is due to the *Acarus*

(*Sarcoptes*) *scabiei*. The female acarus is larger than the male, and forms burrows in the skin, in which the eggs are deposited. These burrows should be looked for between the fingers and on the inner aspects of the wrists. They are recognized with the naked eye as little short dark lines terminating in a sort of shining spot of skin. The eggs lie in the dark line, the insect in the shining spot. It may be picked out by means of a flat surgical needle passed along the black line to the clear spot. The use of a lens aids the operation—which is by no means invariably successful—and permits of the recognition of the insect. The latter may be placed on a slide under the microscope for more minute inspection.

2. **Pediculosis.**—Three varieties of pediculus occur—*Pediculus capitis* on the head, *P. corporis* on the trunk, *P. pubis* on the pubic and axillary hairs. The eggs or "nits" of *P. capitis* are stuck on the hairs. From their position on the hairs one can judge roughly of the duration of the condition, for they are fixed on at first near the root of the hair, and are then carried up with the latter in its growth. The higher up the nits are, therefore, the longer have pediculi been present. *P. corporis* should be looked for in the seams of the clothes, especially where the latter come into close contact with the skin—e.g. over the shoulders. The bites of the parasite produce hæmorrhagic spots, each with a dark centre and a paler areola. Marks of scratching should always be looked for on parts accessible to the patient's nails.

The three pediculi require no verbal description, except to state that *P. corporis* is the longest of the three, *P. pubis* is shortest, and *P. capitis* is between the two in size. *P. pubis* is also distinguished from the others by being yellowish-brown in colour. *P. capitis* and *P. corporis* are both

greyish in colour, though the latter varies considerably with the colour of the skin of its host. The shape of the thorax and abdomen forms a distinguishing character between these varieties and *P. pubis*.

3. **Ringworm.**—Two distinct classes of parasites are capable of producing the appearances which are included under the name "ringworm." One of these is not a trichophyton. It goes by the name of *Microsporon audouini*, and is the cause of the commonest and most contagious and intractable form of the disease. Its operations are chiefly confined to the scalp, but 90 per cent. of all cases of ringworm in that situation are due to its presence. The other parasite belongs to the trichophyton family, but it is probable that there are several varieties of it, as there are also of the microsporon. It is the commonest producer of ringworm of the beard and skin (tinea barbæ and tinea circinata), but only occurs in about 10 per cent. of the cases of ringworm of the scalp. It succumbs much more readily under treatment than does the microsporon. The trichophyton may be situated mostly outside the hair (*ectothrix* or *endo-ectothrix*), or mainly in the hair substance (*endothrix*). Intermediate forms are also described. It is characterized microscopically by having, as a rule, fairly large so-called spores, which are arranged in chains, and the joints of its mycelium are placed at regular intervals. The microsporon has, on the whole, small spores, which are scattered irregularly, and the joints of its mycelium are at unequal intervals.

A useful method of detecting hairs which are affected by ringworm consists in dabbing over the diseased patch with a piece of wool soaked in chloroform. On evaporation of the latter, the affected hairs are whitened, and look as if covered with hoar-frost. They

can thus readily be distinguished from healthy hairs of the same size by the aid of a lens. Hairs affected by favus are not similarly whitened by chloroform.

Microscopical examination.—If one is dealing with a patch of ringworm of the skin, it is sufficient to scrape off some of the scales with a blunt penknife, to place them in a drop of 10-per-cent. liquor potassæ, and cover. The mycelium of the fungus will be recognized as branching, refractile threads, amid which the spores are scattered in groups or rows.

If a hair is similarly examined, it will be found to be broken up and full of spores. No mycelium can be seen. For diagnostic purposes, in simple cases a broken hair should be extracted by broad-pointed forceps, traction being made in the axis of growth, so that the bulb may be obtained intact. It should be washed in ether, soaked for a quarter of an hour in liquor potassæ, a drop of glycerin then run under the cover-slip, and the edge sealed down with melted paraffin. The spores will be seen in the substance of the hair and in its sheath. Fatty particles are the only thing likely to be mistaken for them. A drop of ether will dissolve fat particles, but leaves the spores unaffected. Liquor potassæ, however, causes the spores to swell. Its use should therefore be avoided if one wishes to distinguish the two varieties of fungus. For that purpose staining is of great help. It should be carried out as follows:—

(1) Soak the hair for some minutes in ozonic ether to bleach it and to remove grease, and allow to dry on a slide.

(2) Steep for ten to thirty minutes in a mixture of 10 parts of a 5-per-cent. alcoholic solution of gentian violet and 30 of aniline water.

(3) Dry between blotting-paper, and steep in Gram's solution for five minutes. This fixes the stain.

(4) Dry again, and soak for from ten minutes to some hours if necessary in pure aniline to which enough iodine has

been added to give it a distinctly brown colour. This decolourizes everything except the fungus, but the process should be controlled under the microscope

(5) Wash in pure aniline for a few seconds, then in xylol, and mount in balsam.

On examination with the high power the two varieties of fungus can be differentiated by the characters already described. It should be noted that the difference in size of the spores of the two varieties is really not very great after all. The arrangement of the spores and the character of the mycelium are the points to which attention should be directed.

4. **Favus.**—This disease, which is characterized to the naked eye by the production of yellow cup-shaped crusts or *scutula*, is caused by a parasite named, after its discoverer, the *Achorion schonleinii*. The hair and accompanying crust may be examined in liquor potassæ, some pressure being exerted on the cover-glass in order to flatten out the mass of epidermic scales by which the parasite is apt to be obscured. The hair will be found completely filled with mycelium, and the medullary canal obliterated. The joints of the mycelium are branched, and often present a resemblance in shape to metacarpal and metatarsal bones. The spores are rather large, and arranged in rows or groups.

5. **Tinea versicolor.**—This is produced by the *Microsporon furfur*. A scraping should be examined in caustic potash. The fungus shows a refractile mycelium which interlaces freely, and includes bunches of round spores in its meshes. The spores may be stained with saffranin, differentiated by weak acetic acid, and the mycelium counterstained with methylene blue.

6. **Demodex folliculorum** is a minute acarus, about $\frac{1}{120}$ in. in length, which is sometimes found in the sebaceous contents of comedones. It has a dis-

proportionately large abdomen marked with transverse rings which give it at first sight the appearance of a minute worm. It possesses a suctorial proboscis and styliform jaws, and from the thoracic portion of the body four pairs of stunted legs project. It is simply a parasite living in sebaceous matter, and is of no pathological importance. It stains an intense red by the Ziehl-Neelsen method, whilst the sebaceous debris of the follicle stains blue.

7. **Erythrasma.**—This disease, which consists of brownish-yellow patches with slightly desquamating surface and well-defined margins, usually situated in the axillary or genital regions, is produced by the *Microsporon minutissimum*, a fungus which is made up of a network of extremely fine filaments irregularly segmented. It may be examined by the same method as the *Microsporon furfur*.

CHAPTER IX

THE NERVOUS SYSTEM

I. ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM

IF the student wishes to investigate a case of nervous disease intelligently he must first have a clear grasp of some well-established facts in the anatomy and

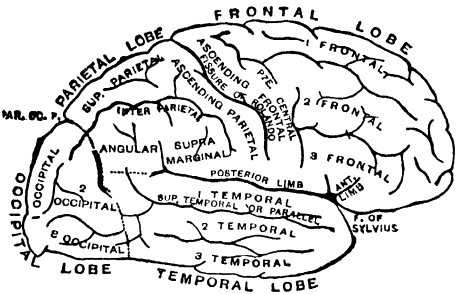


Fig. 75.—Outer aspect of right hemisphere, showing convolutions.

physiology of the brain and spinal cord. A few paragraphs devoted to these subjects will therefore not be out of place.

1. Anatomy and physiology of the motor and sensory paths.—The reader will remember that the **motor area** of the brain is situated in front of the fissure of Rolando (Figs. 75, 76), the leg centre being highest up, the arm centre next to it, and the centres for the face, lips, and tongue

being lowest. For the more exact localization of the various centres the figures in Plate 19 should be consulted.

The **motor fibres** start from the pyramidal cells in the above convolutions, and pass in the white matter of the hemispheres to the internal capsule (i.e. the knee-shaped band of white matter which is bounded on its outer side by the lenticular nucleus, and on its inner side by the optic thalamus and caudate nucleus).

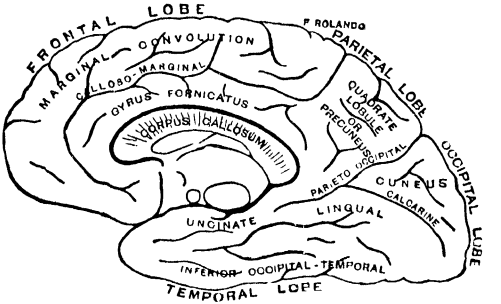


Fig. 76.—Mesial aspect of right hemisphere, showing convolutions.

The motor fibres occupy the anterior two-thirds of the posterior limb, the genu and the posterior part of the anterior limb of the internal capsule, the fibres for the face being farthest forward, those for the leg farthest back, the fibres for the arm being between (Fig. 77). It is in the internal capsule that hæmorrhage most frequently occurs, and, owing to the close approximation of all the fibres at this point, a comparatively small lesion is able to produce a widespread paralysis. From the internal capsule the motor fibres descend to the crus cerebri, occupying the middle third of its ventral aspect. As they descend in the crus the fibres for the leg are to the outer side, the fibres for the face are nearest the middle

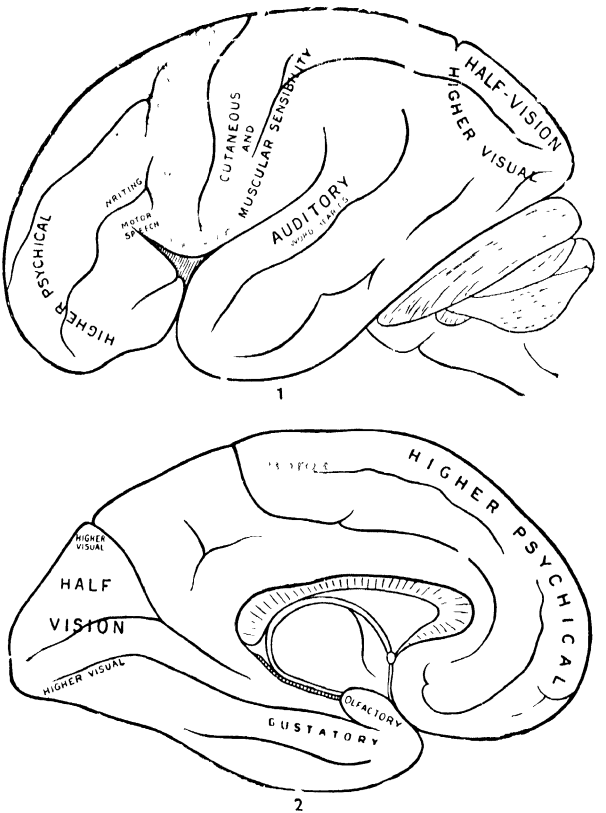


Plate 19.—(1) **Outer.** (2) **Mesial Aspects of Left Hemisphere, showing Functional Areas.**

(*After Purves-Stewart, "Diagnosis of Nervous Diseases"*)

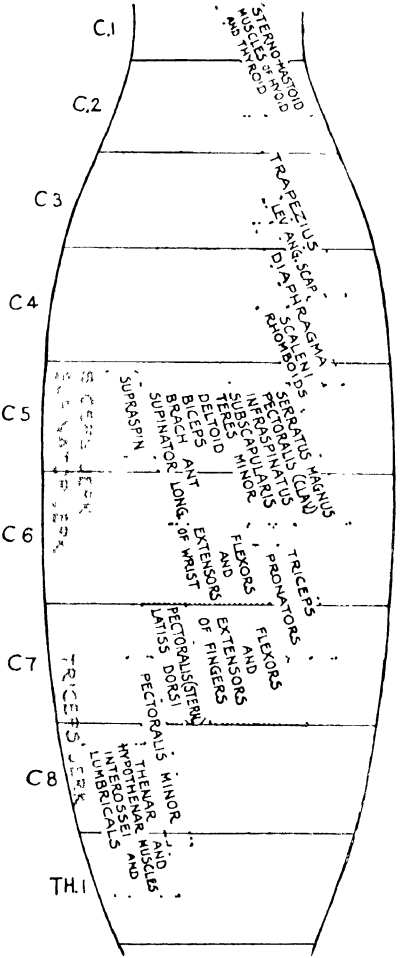


Plate 20.—Motor Segmental Functions of the Cervical Enlargement.

line, and those for the arm are between the two. Below this level the fibres for the arm and leg no longer run separate, but are diffusely scattered over the cross-section of the pyramidal tract; consequently a lesion cannot produce a monoplegia, or paralysis of a single

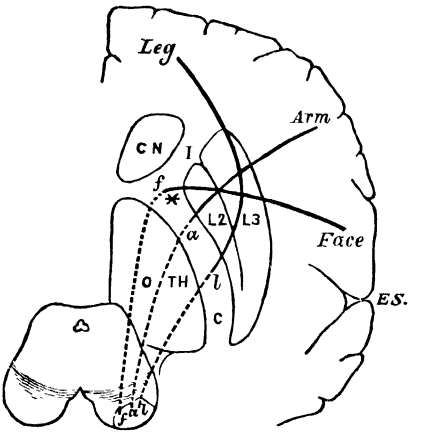


Fig. 77.—Diagram to show relative positions of the face, arm, and leg fibres in their course from cortex to crus. The section through the cortex and crus is vertical; through the internal capsule it is horizontal; *indicates the elbow of the internal capsule.

f, face, *a*, arm, *l*, leg fibres

limb. Entering the pons, the fibres are no longer quite on the surface, but are intermingled with a layer of transversely-placed fibres. In the upper part of the medulla they form a well-marked bundle, the anterior pyramid, lying quite on the surface. At the lower part of the medulla the greater number of the fibres cross to the opposite side, forming what is known as the *decussation of the pyramids*, and run down in

the crossed pyramidal tract in the lateral column of the cord, to end at different levels in the grey matter of the anterior cornu (Fig. 78). The motor impulses are here transferred to the cells of the anterior cornu and, through the anterior roots, which arise from them, to the motor nerves and muscles. A small number of fibres which do not decussate in the medulla are continued down in the anterior or direct pyramidal tract, but they eventually decussate in the anterior commissure of the spinal cord, and then end in the opposite anterior horn. Owing to this decussation of the pyramidal tracts, the impulses which take origin in one hemisphere of the brain pass to the opposite side of the spinal cord, and innervate the opposite limbs and the muscles of this side of the body. A small number of fibres also descend in the crossed pyramidal tract of the same side. This small number of motor fibres which do not decussate at all, but end in the anterior cornu of the same side as that on which they took origin in the brain, may be concerned in the innervation of voluntary movements which are normally bilateral and symmetrical, as those of the thorax; these are never completely paralysed by a unilateral cerebral lesion, as they are excited by motor impulses from

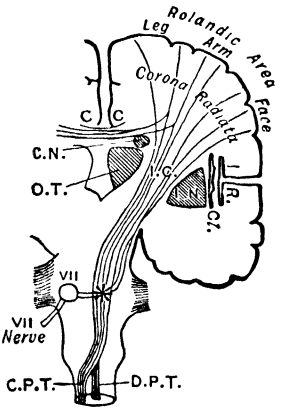


Fig. 78.—Diagram showing the course of the motor fibres from the cortex to the cord.

C C, corpus callosum, C N, caudate nucleus; I R, island of Reil, Cl, claustrum, *, site of facial decussation; C P T, crossed pyramidal tract, DPT, direct pyramidal tract.

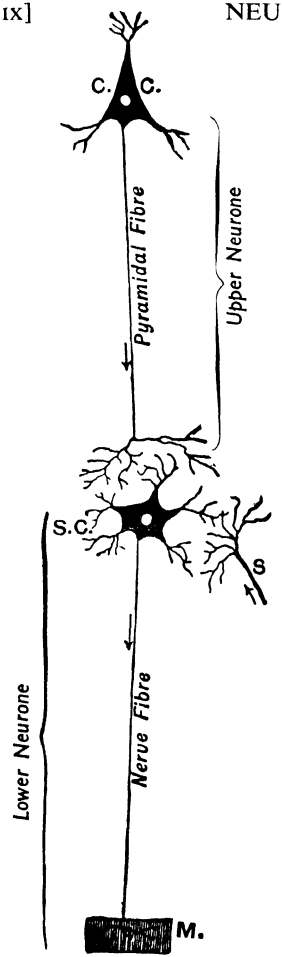


Fig. 79.—Upper and lower neurones of motor path.
C, cerebral cell; S C, spinal cell; S, afferent fibre; M, muscle.

both hemispheres of the brain. The pyramidal fibres to the motor cranial nerves decussate in the brain-stem at a short distance above the nuclei in which they terminate.

In thus tracing the course of a motor impulse, we have spoken of nerve-cells and nerve-fibres. It would be better, however, to discard these names in favour of the modern terminology, which describes a nerve-cell, its dendritic processes, and the fibre connected with it (axis-cylinder process) as a **neurone.** Thus, anatomically, the motor impulses are conveyed by means of two neurones. One of these consists of the pyramidal cell in the cerebral cortex and the motor fibre arising from it (i.e. its axis cylinder process) and ending in the anterior cornu. The other is the anterior cornual cell and the fibre arising from it and ending in the muscle. There is apparently no direct anatomical continuity between these two neurones,

but the nerve impulse is able to pass from the one to the other by contact (Fig. 79).

This conception has also the advantage of making clearer some well-known physiological and pathological facts. Thus, it can easily be understood that if one part of a neurone is injured, the health of the whole neurone suffers. If, for example, what one may call the body of the upper neurone (i.e. the cortical cell) is injured, the axis-cylinder process of the neurone (i.e. the pyramidal fibre) is also affected, and ultimately undergoes the process spoken of as degeneration. Conversely, if a motor fibre is cut across—say in the spinal cord—the health of the cortical nerve-cell, of which it is a process, becomes secondarily impaired.

We have spoken of the motor path as consisting of an upper neurone (the cortical cell and its motor fibre) and a lower neurone (the anterior cornual cell and its motor fibre). It must be realized that these are not entirely independent of one another. Not only do the upper neurones excite the lower, but they also exercise an important controlling influence, or "inhibition" effect, upon them. Under normal conditions, for instance, they check or restrain muscle-tonus, which is kept up by the activity of reflex arcs passing through the brain-stem and spinal cord and influencing, by way of descending extrapyramidal tracts, the lower motor neurone. Of these descending extrapyramidal motor tracts, the ponto-spinal, and especially the vestibulo-spinal, from Deiter's nucleus, seem to be the most important. Tonic reflexes are excited by the otolith organs in the internal ear and contractions of the muscles of the limbs and trunk, particularly those of the neck. The result of this is, that if the upper neurone suffers damage in any of its parts, the restraint on the lower

neurone is diminished and the tone of the muscles is raised; as a consequence the muscles become rigid or spastic, and what are known as their " tendon-reflexes " become exaggerated (*see* p. 463). On the other hand, the lower motor neurones exert an important nutritive influence on the muscles, and when this is cut off, as it may be by injury of the peripheral nerves or disease of the grey matter of the anterior cornua, e.g. in infantile paralysis, the muscles atrophy and degenerate, and in addition lose their tone.

Consequently, though paralysis may result from lesions of both the upper and the lower motor neurones, considerable differences exist between the types produced. In *upper motor neurone paralysis* the muscles do not waste; their tone is increased, and the limb is consequently rigid; the electrical reactions are normal; the tendon-reflexes are exaggerated, and the paralysis, which is of movements and not of individual muscles, is usually widespread, as of one side of the body (hemiplegia) or of a whole limb (monoplegia). In *lower motor neurone paralysis* the muscles atrophy and lose their tone, and the paralysis is consequently flaccid; their electrical excitability is lost, or the reaction of degeneration can be obtained (*see* p. 474); the tendon-jerks are abolished, and the palsy is usually restricted and involves only individual muscles or groups of muscles.

The anatomical paths for **sensation** are less accurately known than the motor paths. All afferent impulses from the periphery are conducted to the spinal cord by the afferent nerves, and through the spinal ganglia and the posterior spinal roots. These constitute *the peripheral or primary afferent neurones.* But only a small proportion of these impulses ever reach consciousness as sensations. The rest are

concerned in the spinal-reflex functions, in the maintenance of the tone of the muscles, or they terminate in the portions of the spinal cord, or in higher centres such as the cerebellum, which control the co-ordination of muscular activity.

Sensory impressions come not only from the skin and superficial tissues, but also from muscles, tendons, and joints. Disease of the peripheral afferent neurones may consequently affect (1) cutaneous sensibility, and abolish or disturb the perception and localization of tactile, painful, and thermal stimuli, or (2) deep sensibility, which is conveyed by the afferent fibres that come from the muscles, tendons, bones and joints. This system underlies the recognition of position and movement, and, when cutaneous sensibility is lost, heavy touches or the pain produced by pressure can be appreciated through it. Owing to the wide anastomosis and distribution of the fibres of this system, deep sensibility frequently escapes in areas in which cutaneous sensibility is lost; then firm touches, as those produced by a finger or the point of a pencil, may be felt, though light touches cannot be appreciated.

After they have entered the spinal cord, the various sensory impulses are rearranged and grouped into other systems. The majority of the peripheral neurones that have carried them hither terminate in the grey matter of the posterior horn at or near the level at which they enter, and from this grey matter the secondary sensory tracts take origin. These cross immediately, or within a few segments, to the opposite antero-lateral column of the cord, and in its ascend to the brain-stem (Fig. 80). Touch, pain, and temperature are carried in this crossed secondary path. Other peripheral fibres, however, do not terminate in the grey matter of the spinal cord, but run cerebral-

wards in the posterior column of the same side as that on which they entered the cord; these posterior column fibres carry the impulses on which depends the appreciation of position, of movement, and of size and shape. Vibration and the power of discriminating

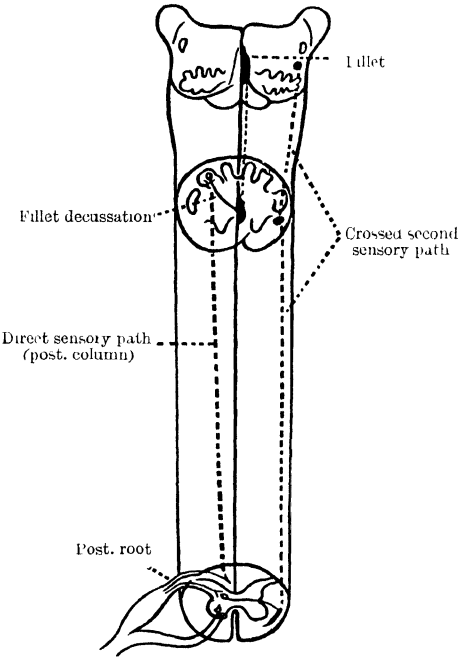


Fig. 80.—Diagram of the sensory paths in the spinal cord and the medulla oblongata.

Weber's compass points are also conveyed in the posterior column, and this contains, too, a path for touch. Consequently, at any level of the spinal cord we have in each half of it two sensory paths conveying sensory impressions cerebralwards: one, in the antero-lateral

column, carries touch, pain, and temperature from the opposite half of the body, and a second, in the posterior column, conveys the appreciation of posture, weight, size, shape, and other qualities of sensation from the same side of the body (Fig. 80). A unilateral lesion of the spinal cord, therefore, produces the Brown-Séquard phenomenon, in which pin and thermal sensibility are lost below the level of the lesion on the opposite side of the body, while on the side of the lesion there is, in addition to spastic paralysis, disturbance of the sense of position and of movement, and loss of the recognition of weight, size, shape, of vibration and of the compass touch. As touch has a double path, one on the same and another on the opposite side of the spinal cord, it is rarely much affected by unilateral spinal lesions.

At the upper end of the spinal cord the posterior-column fibres terminate in the nuclei of Goll and Burdach, and the impulses they carry are taken up by **secondary sensory fibres,** which immediately cross to the opposite side of the medulla in the fillet decussation. Consequently, in the medulla oblongata all sensory impressions are carried in secondary tracts which lie on that side of the nervous system opposite to the half of the body from which they come. But even here all do not run in a single path, for pain and thermal impressions pass through the lateral part of the bulb, while those conducted by the posterior columns enter the mesial fillet (Fig. 80). Higher in the brain-stem, however, all sensory elements probably run closely together in the mesial fillet, and those which have come from the spinal cord are joined by the secondary fibres from the nuclei of the sensory cranial nerves. Finally, the fibres of the fillet terminate in the optic thalamus, no secondary sensory fibres passing uninterrupted beyond it, and from this a tertiary system of sensory

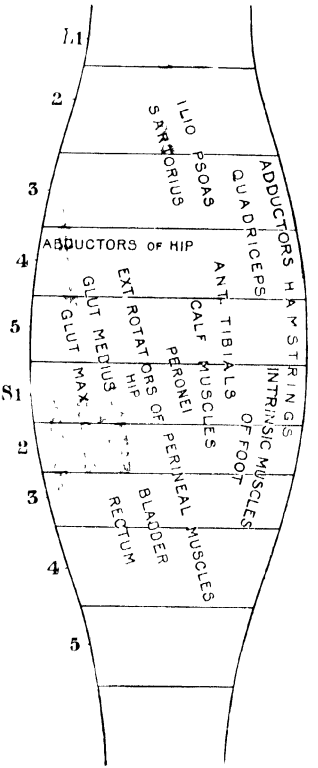


Plate 21.—Motor Segmental Functions of the Lumbar Enlargement.

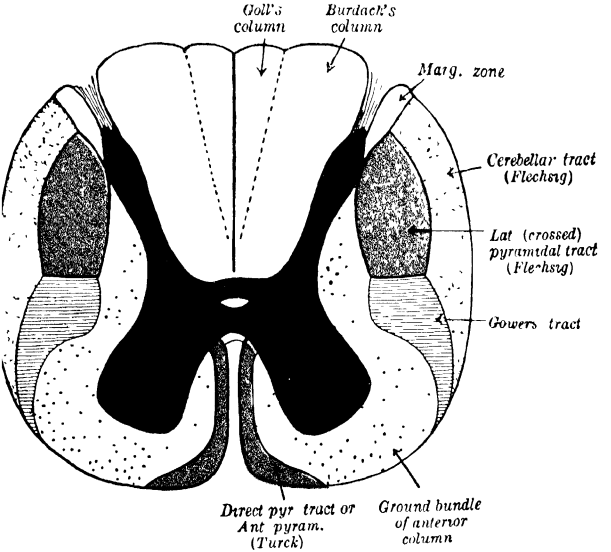


Plate 22.—TRACTS OF THE SPINAL CORD. (*Morat*)

Points of Origin and Exit of Nerve-Roots from Cord and Spinal Canal

Roots	Level of Surface Origin	Point of Exit
C.1	Just above arch of atlas	Between atlas and occiput.
2	Ranges from just above to just below spine of atlas	Above axis
3	At or just above spine of axis	Above 3rd C.
4	From spine of axis to spine of 3rd cervical	Above 4th C.
5	Ranges from lower edge of spine of axis to to that of 4th cervical.	Above 5th C.
6	Ranges from lower edge of spine of 3rd to that of 5th cervical	Above 6th C.
7	Ranges between top of 4th to bottom of 6th spine.	Above 7th C.
8	Ranges between top of 5th to top of 7th spine.	Above 1st Th.
Th.1	Ranges between above 6th and below 7th spine	Between 1st and 2nd Th
2	Ranges from lower edge of 6th cervical spine to that of 1st thoracic.	Between 2nd and 3rd Th
3	Ranges from upper edge of 7th cervical spine to lower of 2nd thoracic	Between 3rd and 4th Th
4	Ranges from top of 1st thoracic spine to that of 3rd thoracic	Between 4th and 5th Th.
5	Ranges from top of 2nd to top of 4th thoracic spine.	Between 5th and 6th Th
6	Ranges from lower edge of 2nd thoracic spine to upper of 5th	Between 6th and 7th Th
7	Ranges from top of 4th to bottom of 5th thoracic spine.	Between 7th and 8th Th.
8	Ranges from top of 5th to top of 6th spine.	Between 8th and 9th Th.
9	Ranges from between 5th and 6th spines to top of 7th	Between 9th and 10th Th.
10	Ranges from lower edge of 6th to upper of 8th.	Between 10th and 11th Th
11	Ranges from top of 7th to top of 8th spine.	Between 11th and 12th Th
12	Ranges between top of 8th and bottom of 9th spine	Between 12th Th and 1st L.
L.1	Ranges between top of 9th spine and bottom of 10th.	Between 1st and 2nd L.
2	Ranges between 9th and 11th thoracic spines.	Between 2nd and 3rd L.
3	Ranges between top of 10th and bottom of 11th spine.	Between 3rd and 4th L.
4	Ranges between bottom of 10th and top of 12th spine	Between 4th and 5th L.
5	Ranges between top of 11th and top of 12th spine.	Between 5th L. and 1st S.
S.1	Ranges between lower border of 11th thoracic spine and top of 1st lumbar.	Between 1st and 2nd S.
2	Usually between 12th thoracic and 1st lumbar spines.	Between 2nd and 3rd S.
3	Usually between 12th thoracic and 1st lumbar spines.	Between 3rd and 4th S
4	Usually between 12th thoracic and 1st lumbar spines	Between 4th and 5th S.

fibres conveys sensory impressions to the cerebral cortex.

The exact extent of the **cerebral cortex** concerned in reception of sensation is still doubtful; it certainly lies mainly in the parietal lobes behind the fissures of Rolando. It seems very probable, however, that certain sensory qualities, such as pain, never reach the cortex, but affect consciousness through subcortical centres, probably through the optic thalamus. The courses of the fibres and the position of the centres for the special senses are described in the section dealing with the cranial nerves (p. 392). The speech centres and their connexions are described at p. 386.

2. **The spinal cord.**—The cord extends as far down as the interspace between the 12th thoracic and 1st lumbar spines; the membranes are continued down as far as the body of the 2nd sacral vertebra.

The cervical enlargement reaches to the 7th cervical spine. Its largest part is opposite the disc between the 5th and 6th cervical vertebræ.

The *lumbar segments* lie opposite the 10th and 11th thoracic spines and the next interspinous space.

Physiologically, the cord is to be regarded as made up of a series of superimposed segments, from each of which a pair of nerve-roots arises. To enable us to localize focal lesions of the cord it is necessary to be acquainted with the functions of each segment, and therefore with the area of supply of the pair of nerve-roots arising from it.

The table on p. 369 shows the points of origin and emergence of the nerve-roots from the cord and spinal canal. Plates 20 and 21 exhibit in a diagrammatic way the motor functions of the cervical and lumbar segments (or anterior roots) and the reflexes over which each segment presides. Figs. 81 and 82 show the distribution of the cervico-brachial and

lumbo-sacral plexuses. The sensory functions of the cord are exhibited in Figs. 83, 84, and 85.

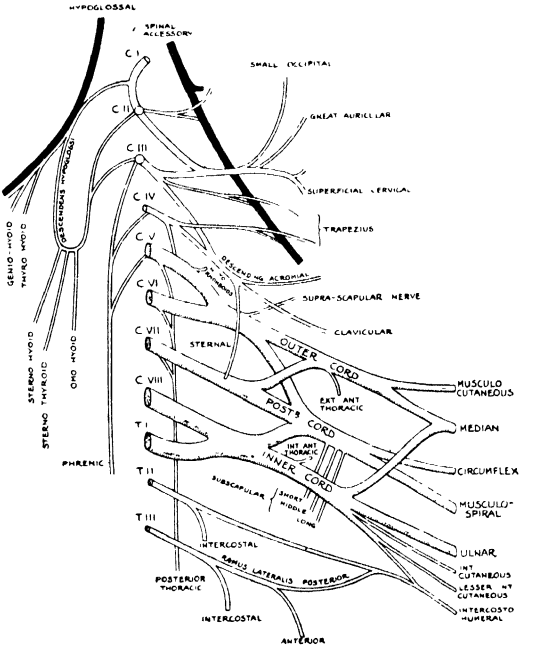


Fig. 81.—Cervico-brachial plexus and its branches.
(After Purves-Stewart, " Diagnosis of Nervous Diseases ")

Figs. 84 and 85 show the sensory distribution of the **posterior nerve-roots** (" root areas ").

Plate 22 shows the position of the different **tracts of the cord** on transverse section.

The list on p. 376 shows the **nerve supply of the**

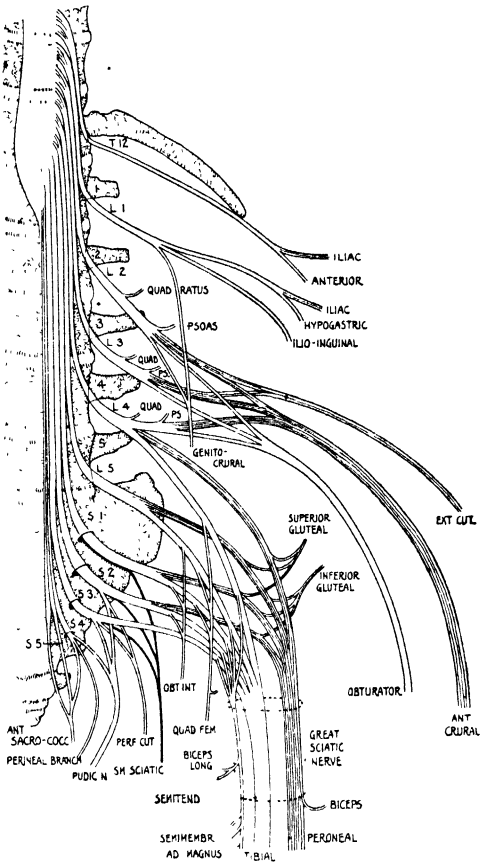


Fig. 82.—Lumbo-sacral plexus and its branches.
(*After Purves-Stewart, "Diagnosis of Nervous Diseases"*)

muscles of the trunk and limbs. It may be found convenient for reference in the study of cases of

peripheral paralysis. The nerve supply of the head is considered along with the cranial nerves (p. 392).

The peripheral distribution of the chief **sensory nerves** is sufficiently indicated in Figs. 86, 87, 88, and 89.

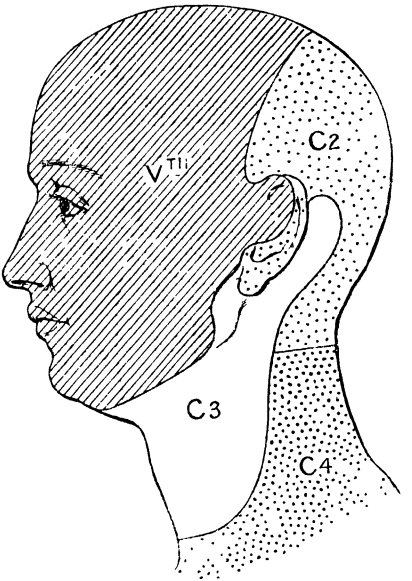


Fig. 83.—Lateral view of the skin areas supplied by the 2nd, 3rd, and 4th cervical segments.

Vascular supply of the brain and spinal cord.—The **brain** is supplied by the internal carotid and vertebral arteries. Owing to the position of origin of the left common carotid, an embolus can enter it more easily than it can the artery of the opposite side. Embolic lesions are therefore more frequent in the left than in the right cerebral hemisphere.

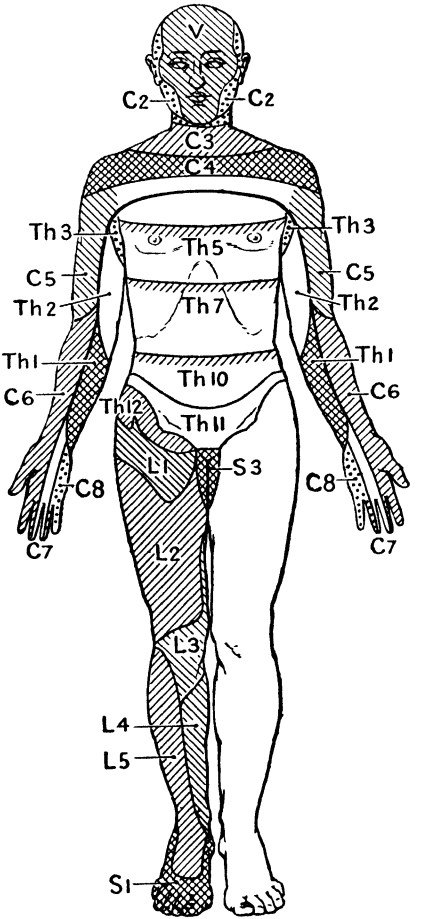


Fig. 84.—Segmental sensory areas of the cord.

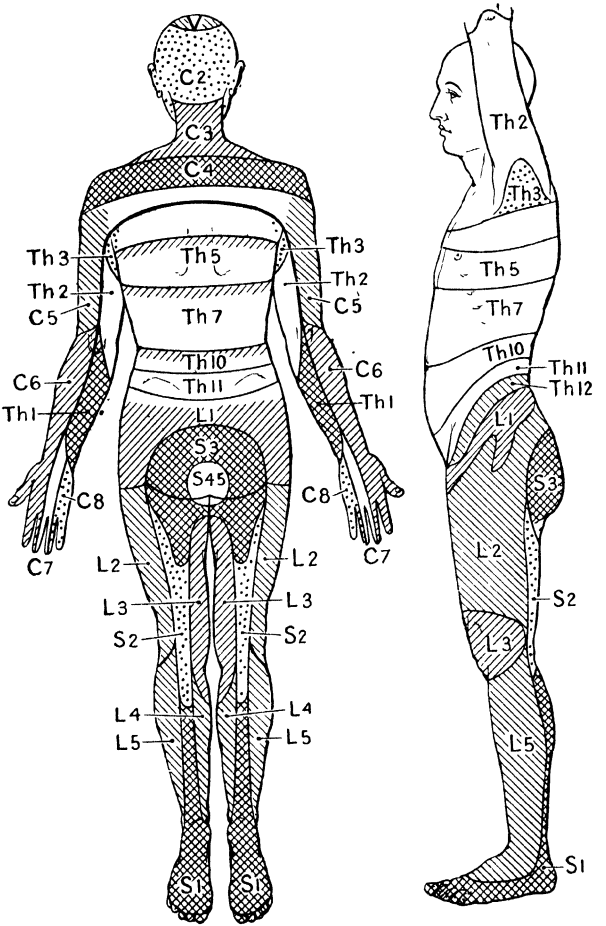


Fig. 85.—Segmental sensory areas of the cord.

Nerve Supply of Trunk and Limb Muscles

Upper Limb		Trunk and Lower Limb	
Post. Thoracic	Serratus magnus.	*Intercostals*	Intercostals.
Suprascapulars	Supraspinatus.		Rectus abdominis.
	Infraspinatus.		External oblique.
Ext. Ant. Thoracic	Pectoralis major (upper part).	*Branches of Lumbar Nerves*	Erector spinæ.
Int. Ant. Thoracic	Pectoralis major (lower part).		Quadratus lumborum
	Pectoralis minor	*Genito-Crural*	Cremaster.
Musculo-Cutaneous	Coraco-brachialis	*Anterior Crural*	Sartorius.
	Biceps		Pectineus.
	Brachialis anticus		Rectus femoris
	Subscapularis.		Vastus externus.
Subscapular	Teres major		Vastus internus
	Latiss. dorsi		Crureus.
Circumflex	Deltoid.		Psoas and iliacus.
	Teres minor.	*Obturator*	Gracilis.
Musculo-Spiral	Triceps.		Obturator externus.
	Ext. carp. rad. long		Adductor longus
	Supinator long		Adductor brevis.
	B. anticus.		Adductor magnus (with sciatic).
	Anconeus.	*Infer. Gluteal*	Gluteus maximus
	Supinator brevis	*Sup. Gluteal*	Gluteus medius and minimus.
Post. Interosseous	Ext. carp. rad. brev.		Tens. vag-femoris
	Ext. carp. uln.	*Great Sciatic*	Biceps femoris.
	Ext. comm. digit		Semitendinosus.
	Ext. ossis metac. poll		Semimembranosus.
	Ext. primi intern. poll.		Adductor magnus (with obturator)
	Ext. secund. intern. poll.	*Int. Popliteal*	Gastrocnemius.
	Ext. indicis.		Soleus
	Ext. minim. digiti.		Tibialis posticus.
Median	Pronator radii teres.		Flex. comm. digit.
	Pronator quadratus.		Flex. long hallucis
	Palmaris longus	*Int. Plantar*	Flex. brev. hallucis
	Flexor carpi radialis.		Flex. brev. digit.
	Flexor sublim. digit		Abductor hallucis.
	Flexor longus pollicis.	*Ext. Plantar*	Adductor hallucis.
	Opponens pollicis		Interossei.
	Abductor pollicis		Flex. brev. min. dig.
	Two outer lumbricals		Abductor min. dig.
Median and Ulnar (jointly)	Flex. profund. dig.		Tibialis anticus.
	Flexor brevis pollicis.	*Ext. Popliteal*	Ext. prop. hallucis.
Ulnar	Flexor carpi ulnaris.		Ext. digit. longus.
	Adductor pollicis		Peroneus longus.
	Muscles of little finger.		Peroneus brevis.
	Interossei.		Extens. brev. digit.
	Two inner lumbricals.		

The two *vertebral arteries* unite at the lower border of the pons to form the *basilar*, which runs up the middle of the anterior surface of the pons, and ends by dividing into the two posterior cerebrals. It gives off lateral branches which run out transversely over the pons, and vertical branches which pass into its substance. The latter not infrequently become thrombosed.

The *posterior cerebrals* supply the occipital lobes, the lower part of the temporal lobes, with the uncinate gyrus, the inner part of the crus and the corpora quadrigemina, and the posterior part of the posterior limb of the internal capsule. Blocking of one of these arteries at its origin will therefore involve the visual centre and the sensory fibres, but thrombosis often involves the calcarine branch and hence the visual centre alone.

The basilar artery supplies the upper surface of the cerebellum ; the vertebrals supply its lower surface, as well as the greater part of the medulla oblongata.

The *internal carotid* gives off the *anterior cerebral* artery, which curves round the anterior end of the corpus callosum, and is chiefly distributed to the inner surface of the cerebral hemisphere as far back as the parieto-occipital fissure. It also supplies the superior frontal convolution.

The internal carotid is practically continued on to the brain as the *middle cerebral*, which lies in the Sylvian fissure. An embolus which has found its way into the internal carotid, therefore, usually ends in the middle cerebral or one of its branches. The middle cerebral gives off *cortical branches*, which supply the motor area and the upper part of the parietal and temporal lobes. These branches anastomose freely with those of adjoining arteries, hence blocking of one of them may be largely compensated by the

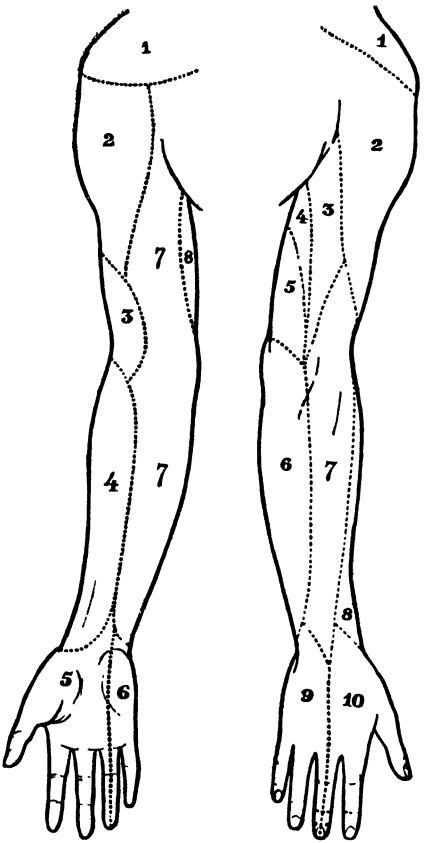


Fig. 86.—Cutaneous nerve supply of upper limb.

Anterior aspect; 1, cervical plexus; 2, circumflex; 3, ext. cut of musc.-spiral; 4, ext. cutaneous; 5, median; 6, ulnar; 7, int. cutaneous; 8, nerve of Wrisberg. *Posterior aspect;* 1, cervical plexus; 2, circumflex; 3, int. cut. of musc.-spiral; 4, intercosto-humeral; 5, nerve of Wrisberg; 6, int. cutaneous; 7, ext. cut. of musc.-spiral, 8, musc.-cutaneous; 9, ulnar; 10, radial.

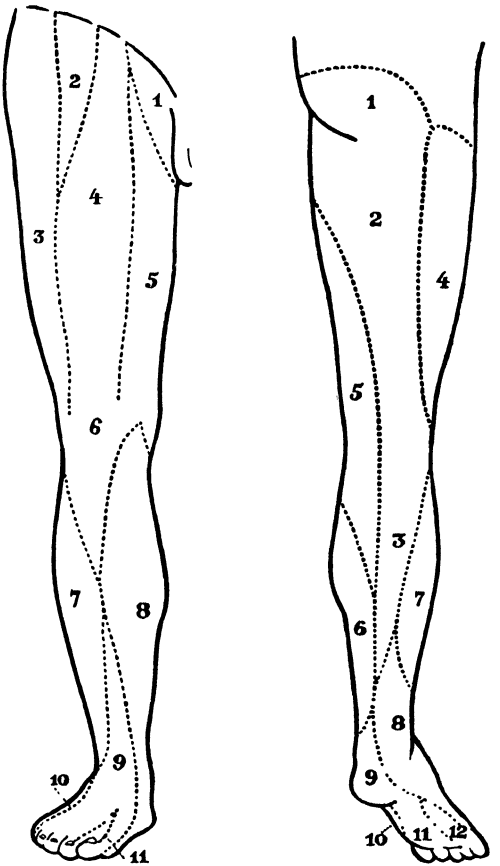


Fig. 87.—Cutaneous nerve supply of lower limb.

Anterior aspect; 1, ilio-inguinal; 2, genito-crutal; 3, ext. cutaneous; 4, middle cutaneous; 5, int. cutaneous; 6, patellar plexus; 7, branches of ext. popliteal; 8, int. saphenous; 9, musc.-cutaneous; 10, ext. saphenous; 11, ant. tibial. *Posterior aspect;* 1, 2, 3, small sciatic; 4, ext. cutaneous; 5, int. cutaneous; 6. int. saphenous: 7, branches of ext. popliteal; 8, short saphenous; 9, post. tibial · 10, int. saphenous 11, int. plantar; 12, ext. plantar.

establishment of a collateral circulation. It also gives off *central branches*, which penetrate into the brain substance and supply the white matter and the basal ganglia. There are two chief groups of these cen-

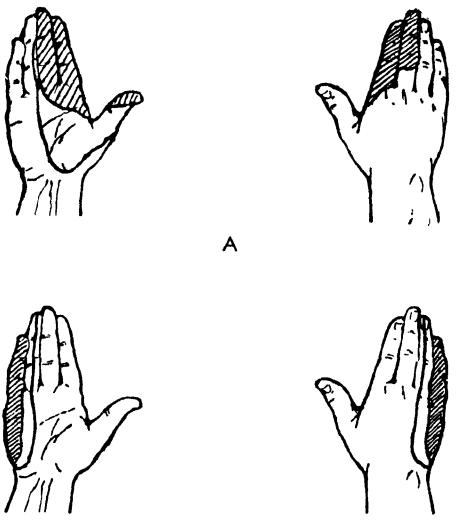


Fig. 88.—A, Area of cutaneous sensory loss after division of the median nerve in the arm. Area of loss to light touch is bounded by the continuous line, and that to pin-prick is indicated by the oblique lines. B, Area of cutaneous sensory loss after division of the ulnar nerve above the elbow. Anæsthetic area (light touch) is limited by the continuous line, and analgesic area (pin-prick) by the cross-shading.

tral arteries—an anterior group called the *lenticulo-striate*, and a posterior group, the *lenticulo-optic*. As the lenticulo-striate are more directly exposed to the force of the wave of arterial blood they are more frequently ruptured than are the lenticulo-optic. These central arteries do not anastomose

with one another. They are, therefore, to be regarded as end-arteries. Hence it is that a lesion of one of them is much less likely to be compensated than is a lesion of a cortical branch.

The venous blood from the brain is poured into the *venous sinuses*. Owing to the slow current in these,

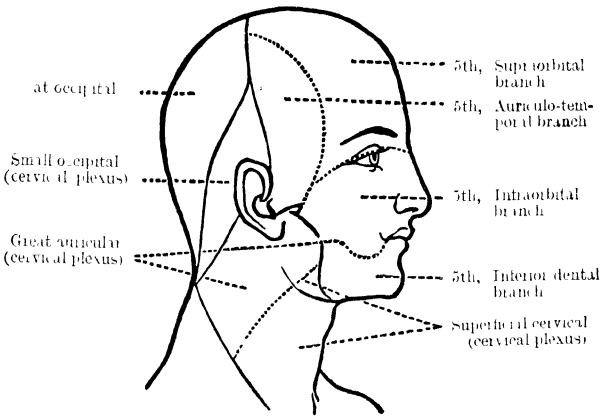


Fig. 89.—Distribution of the sensory nerves of the head. Compare with it the segmental distribution as shown in Fig. 83.

thrombosis readily occurs in them. The blood from the interior of the lateral ventricles is chiefly returned by the veins of Galen, which end in the straight sinus. Owing to their long course, these veins are frequently exposed to pressure by tumours, etc. If the pressure is on the anterior portion of the veins it is apt to lead to increased exudation of fluid into the lateral ventricles.

Spinal arteries.—The *anterior and posterior spinal arteries* arise from the vertebrals and travel downwards in the pia mater, the former in the

antero-median fissure and the two latter alongside the posterior nerve-roots. Although they have a long and tortuous course, they do not diminish in size, being reinforced by radicular tributaries from the intercostal and lumbar arteries. The anterior spinal artery supplies the greater part of the grey matter, namely, the anterior horns and the base of the posterior horns, including Clarke's column of cells. The posterior spinal arteries form a vasa-coronal system and irrigate the whole of the white matter of nerve-tracts of the cord, along with part of the posterior horns.

The chief **veins of the spinal cord** are situated dorsally and ventrally in the middle line. Like the arteries, they communicate by radicular branches with the lumbar and intercostal veins, and empty into the vertebral veins. The blood in them flows upwards; hence in compression of the spinal cord, as by tumour or tuberculous abscess, there is venous engorgement below the level of pressure. This results in asphyxia of the spinal cord with paralysis and loss of sensibility, which frequently disappears if the cause of compression is removed. It also leads to increase in the albumin content of the cerebro-spinal fluid below the site of compression, the main feature of Froin's syndrome.

The student may now pass to the method of examining a patient with nervous disease, as described in the subsequent sections. We would recommend him to begin by ascertaining the state of the intellectual faculties of the patient, including speech (Section II). He should then test the condition of the cranial nerves in their order. How this is to be done is described in Section III (p. 392). By proceeding thus, valuable information is gained at the outset, which

may guide one in the subsequent investigations. The motor, sensory, reflex, and trophic functions should then be examined in order, following the methods described in Sections IV (p. 433), V. (p. 451), VI (p. 458), VII (p. 471). Lastly, the electrical reactions of the muscles and nerves should be tested in those cases in which it may seem necessary (Section VIII, p. 471).

II. INTELLECTUAL FUNCTIONS

It is important to arrive at some idea of the patient's intellectual state early in the taking of a nervous case, as it affords indications that are of help in the subsequent investigation of his symptoms. For example, if one finds that his memory is deficient, one attaches only a limited value to the account that he gives of the onset of his illness or the state of his previous health. Or if one discovers that he is comatose, or unable to understand speech, it is evident that one cannot expect to make much of any attempt to investigate the state of his sensory functions. This section will, therefore, be devoted to methods of investigating a patient's mental condition, including the functions concerned in producing and interpreting speech.

The first thing to be determined is whether we are dealing with **a right-handed or a left-handed patient.** The importance of this depends upon the fact that in right-handed people speech functions are localized in the left cerebral hemisphere, and vice versa. Ask the patient, if a male, which hand he uses to throw a stone or to pull a cork; if a female, which hand is employed in combing the hair. It is of comparatively little use to ask which hand he writes with, as most children are taught to write with the right hand.

The state of the **memory** next calls for investigation. It may be tested by asking the patient what day of the week it is, what he ate at breakfast, and so on. It is important to distinguish between (*a*) the memory of recent events and (*b*) the memory of older occurrences. Both should be tested in every case, as one or the other may be lost alone; in the psychosis which often accompanies alcoholic neuritis, for instance, the patient may retain his memory of events of his childhood, but may remember nothing that has happened during his illness. Inquire as to his **sleep,** and whether or not he is troubled with dreams.

Note whether or not he is more or less **emotional** than is normal. An abnormal emotional state may be evidenced by the patient's bursting into laughter or into tears on very slight provocation, or by his giving way easily to fits of anger.

In the course of taking his case, one will already have arrived at a general notion of the degree of the patient's intelligence. Sometimes it is necessary to ascertain whether he is the subject of **hallucinations** or **delusions.** An hallucination consists essentially in an imaginary sense-impression. A delusion is an erroneous idea which would be incredible to the patient's equals, and which is unshaken by facts. If the patient says he hears voices when no one is present, or if he sees persons or forms which do not exist, he is the subject of an hallucination—in the former case auditory, in the latter visual. If he declares that he is the Emperor of Russia, he is the victim of a delusion. The existence of hallucinations and delusions is often difficult to ascertain. Sometimes they are discovered by chance; in other cases they can be elicited by skilful questioning; often they are reported by the friends.

Delirium or **coma** may be present; in such a

case the investigation of the intellectual faculties already described is futile.

One should next proceed to the investigation of the **speech functions.** In considering speech it is essential to distinguish between defects of articulation and enunciation, and those disturbances of speech, due to diseases of its cerebral mechanism, which we speak of as aphasia.

Supposing that the patient is able to speak, one should note whether there is any peculiarity in his **articulation.** The following are the chief abnormalities which may be present:—

1. **Stammering.**—This requires no special description.

2. **Lalling, or baby speech.**—Ask the patient to read something aloud. If he lalls, one will recognize that all the difficult consonants are dropped; he speaks like a baby, and, if a child, may perhaps make use of words of his own invention—**idioglossia.** Lalling and idioglossia are usually the result of a congenital defect in the appreciation of the meaning of sounds—congenital auditory interception.

3. **Scanning, or staccato speech.**—The patient speaks slowly and deliberately, syllable by syllable, as if scanning a line of poetry. Ask him to say "artillery"; he will pronounce it "ar-til-ler-y." This is the kind of speech found in some cases of disseminated sclerosis.

4. **Slurring speech.**—The syllables are slurred together as in a state of intoxication. Thus, "British Constitution" becomes "Brizh Conshishushon." This kind of speech is met with very typically in general paralysis of the insane.

5. **Dysarthria** or disorder of articulation is due to paresis or inco-ordination of the peripheral

mechanism of speech, either of the larynx, tongue, or lips, though often all three are affected together. When it is severe **(anarthria),** sounds can be no longer emitted, as is the case in advanced bulbar or pseudo-bulbar paralysis.

If the patient's defect consists not so much in a perversion of articulation as in an inability to produce speech, or to understand it when spoken or when written, then his condition is described as one of **aphasia.**

In order to understand the method of investigating a case of aphasia, it must be remembered that for purposes of speech we have (1) a producing mechanism. This consists of two parts—one concerned in the production of spoken speech, the other in the production of written speech. (2) A receiving mechanism. This also consists of two parts—one for the reception of spoken speech, the other for the reception of written speech.

We may thus classify cases of aphasia as follows:—

1. Lesions of productive mechanism (motor aphasia)	Motor aphasia (loss of power of talking). Agraphia (loss of power of writing).
2. Lesions of receptive mechanism (sensory aphasia).	Auditory (word deafness). Visual (word blindness).

It must be borne in mind, however, that it is the exception to meet with a case of aphasia of a pure type. Thus, a patient may have both motor aphasia and also word-deafness; he may be both unable to read as well as unable to write; and so on.

The *cortical centres* for the production and reception of speech are situated in the left cerebral hemisphere in right-handed persons, in the right hemisphere in the case of those who are left-handed. Hence the

importance of ascertaining early in the investigation of a nervous case whether the patient is right- or left-handed.

The centre for spoken speech occupies the posterior extremity of the 3rd frontal convolution (Broca's convolution) and the lower end of the ascending frontal.

The centre for the production of written speech is believed to be in the posterior end of the 2nd frontal convolution.

The centre for the reception of spoken speech is in the posterior half of the superior temporo-sphenoidal convolution, and that for the reception of written speech (visual speech centre) extends from the posterior part of this convolution into the angular gyrus (*see* Plate 19, facing p. 360).

The visual speech centre is connected by special fibres with the primary visual centre in each occipital lobe. Hence, a lesion in the left occipital lobe does not produce word-blindness unless it is so situated as to cut off also the fibres which connect the visual centre in the right occipital lobe with the left angular gyrus (Fig. 90).

For practical purposes it is best to proceed with the investigation of aphasia in this order:—

I. Spoken Speech

1. *How is it received and interpreted?*—Find out, first, whether the patient's hearing is good. If so, ask him to put out his tongue, shut his eyes, etc. If he does so, test him as to his understanding of nouns by asking him to touch his nose, ear, chin, forehead, etc., in turn. Then test his verbs by asking him to smile, to whistle, etc. Finally, put to him longer questions, or give him more complicated orders, as when the

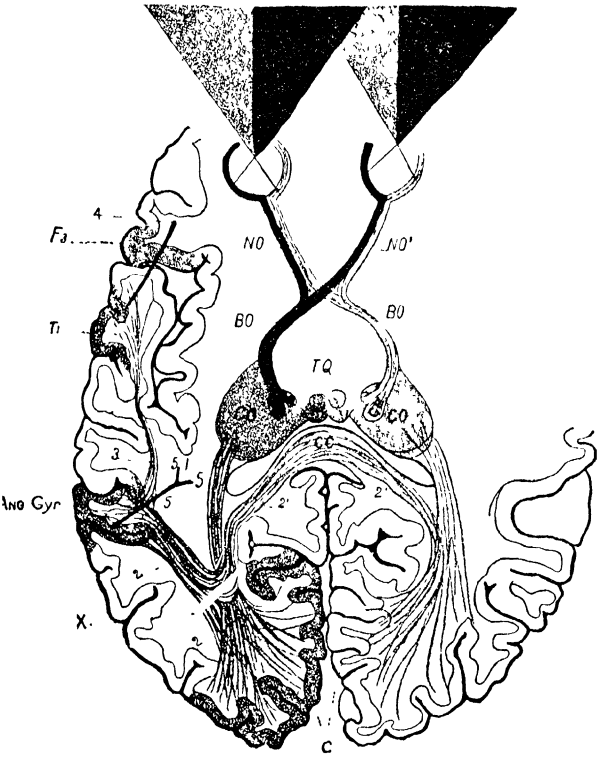


Fig. 90.—Schematic figure, showing the course of the optic fibres.

C, Cuneus, CC, posterior extremity of corpus callosum, F 3, Broca's convolution (speech centre); T 1, superior temporo-sphenoidal, or Wernicke's convolution (auditory word centre): 1, left optic radiation, 2, fibres connecting left angular gyrus with left cuneus, and through the corpus callosum (2′ and 2″) with the right cuneus, a lesion at x cuts these fibres as well as the optic radiation, and therefore causes right lateral homonymous hemianopsia—word-blindness—but no agraphia, 3, fibres connecting angular gyrus with Wernicke's convolution. The straight black line (4) represents the connexion between Broca's and Wernicke's convolutions; the black line bifurcating in front (5) represents the connexions of the angular gyrus with the motor region of the left (5′) and right (5″) hemispheres

disturbance of speech is only slight he may be able to understand simple questions and commands but not more complicated ones. If the patient responds satisfactorily to these tests, he has evidently no difficulty in interpreting the meaning of words heard—i.e. there is no *word-deafness.*

2. *How is it produced?*

i. If the patient can use only a few words, make a note of what these are. If he repeats any word or phrase again and again (**"recurring utterance"**), note what it is.

ii. If he has a considerable vocabulary, (*a*) make a note of any examples of lalling, slurring, etc., as described at p. 385. This affords an indication of his *power of articulation.*

Test him with such words and phrases as "British Constitution," "West Register Street," "Biblical criticism," "artillery."

(*b*) Show him common objects—a knife, a pen, a matchbox, etc.—and ask him to name them; or, if he is dumb, to indicate with his fingers the number of syllables in the name of each. If he is unable to fulfil these tests, he has evidently got some forgetfulness of words (**amnesia verbalis**). Sometimes the patient has a general idea of the word he wants to use, but forgets exactly how to pronounce it; he omits some syllables, or substitutes others for them, so that the listener may hardly be able to make out what word it is he wishes to use. This has been termed by Wyllie *articulative amnesia.*

(*c*) If he makes mistakes in his use of words, calling the knife a pen, or vice versa, he is suffering from **paraphasia.** In that case, one should note whether or not the patient shows that he is aware of his error by trying to correct himself, or whether he goes on talking gibberish.

3. *How is it repeated or echoed?*—Ask him to repeat words after you. If he is word-deaf, try to make clear your request by the aid of pantomime, repeating the word or phrase over and over again. If he is able to repeat what you say, endeavour to find out whether or not he understands what he is saying.

II. Written Speech

1. *How is it received or interpreted?*—Ascertain whether or not his sight is good. If so, write on a piece of paper such questions or commands as, How old are you? Put out your tongue; etc. If he does not respond satisfactorily, there is some word-blindness present—i.e. the patient has **visual aphasia.**

2. *How is it produced?*—Ask him to write his name. (This can often be done when all other power of writing is lost.) If he is able to do so, ask him some simple question—e.g. How many do two and two make?—and get him to write a reply. If he has word-deafness, put your question in writing. If his right hand is paralysed, make him write or print with his left. If he writes pretty well, get him to write an account of his illness, and note whether he makes use of the wrong word at times (**paragraphia**), or whether there is repeated use of any particular word.

3. *Can he write to dictation or copy?*—Try, using some simple book. If he succeeds, endeavour to ascertain whether or not he understands the meaning of what he writes.

III. Phenomena associated with Speech

1. *Does he understand pantomime?*—Does he nod his head for "yes," shake it for "no," and can he indicate numbers with his fingers? Loss of gesture language is termed **amimia.** Mistakes in the use of

gestures—e.g. nodding for "no," or shaking the head for "yes" are termed **paramimia.**

2. *Does he understand symbols*—e.g. numerals?—Thus, one may write down—

2	2	2
2	2	2
—	—	—
4	5	6

and ask him to point out which is right. If he can read music, test him with musical notes.

3. *Can he recognize common objects?*—Place beside him a pencil, a coin, and a match. Ask him to strike a light, or to write something down. If he is unable to select the proper article for the purpose, he is suffering from **mind-blindness** or **visual agnosia.** Inability to recognise his friends is another proof of the same condition.

It occasionally happens that a patient who has neither motor nor sensory paralysis, nor ataxia, cannot perform certain acts, though he can easily execute their component movements. He is consequently unable to make use of objects though he can recognize their use. This condition is known as **apraxia.** It results from destruction of the left hemisphere, or of its connexions, through the corpus callosum, with the right hemisphere. It affects only the left limbs when the callosal fibres are injured, but it is usually bilateral. It may be tested for by asking the patient to use certain objects, or make or imitate certain movements. For instance, he may be given a box of matches and a candle, and asked to light the latter. If there is apraxia he may fail to open the box, or to take a match from it, or to strike the match, or even to light the candle with the match if he has succeeded in striking it. It is, of course,

important to make sure that the patient understands the order.

III. CRANIAL NERVE FUNCTIONS

In this section we propose to give a brief résumé of the essential points in the anatomy of each cranial nerve, to indicate its functions, and, in some cases, the chief symptoms which result from its paralysis, and then to describe the method in which one investigates the state of the nerve at the bedside.

First or Olfactory Nerve

Anatomy.—The nerve-fibres which arise from the olfactory bulb are distributed to the Schneiderian membrane, at the upper part of the nasal fossæ. The cortical centre for smell is believed to lie in the uncinate gyrus. The exact course of the fibres between the cortex and the bulb is unknown, but it is probable that some of them do not decussate.

Test.—Have three small bottles containing some oil of cloves, some oil of peppermint, and some tincture of asafœtida. Apply these to each nostril separately, and ask the patient if he recognizes them. In testing, avoid the use of such irritating substances as ammonia, for these act, partly at least, through the 5th nerve. The sense of smell may be abolished. This is known as **anosmia.** Before concluding that the nerve is at fault, take care to exclude local changes in the nose itself—e.g. catarrh. **Parosmia** is the name applied to that condition in which the sense of smell is perverted, so that, for instance, offensive substances seem to have a pleasant odour, and vice versa.

Inquire also regarding **hallucinations of smell.** These sometimes constitute the aura of an epileptic fit.

Second or Optic Nerve

Anatomy.—From the retina, which is the end-organ of the sense of sight, the fibres of the optic nerve pass back to the optic chiasma. Here the fibres from the inner half of

each retina decussate, whilst those from the outer half remain on the same side. Each optic tract, therefore, consists of fibres from the outer half of the retina on the same side and the inner half of the retina on the opposite side. Each tract passes back to the anterior corpus quadrigeminum and to the external geniculate body and the pulvinar of the optic thalamus of the same side In these, which are known as the primary optic centres, all the fibres of the optic tracts terminate. But another system of fibres, which is known as the optic radiation, takes origin in the external geniculate body and passes through the posterior limb of the internal capsule and then backwards to the cortex around the calcarine fissure (*see* Fig. 90, p. 388). This, therefore, constitutes the chief visual centre, and represents the opposite half of the field of vision, the left half of the field of vision being represented in the cortex of the right hemisphere, and vice versa.

Test.—In testing the optic nerve, one has to investigate three functions: (1) Acuity of vision; (2) field of vision; (3) colour sense. We shall consider *seriatim* the methods of testing these.

Certain preliminaries must always be attended to. One of these is to see that any error of refraction in the patient's eye is first corrected, and that there is no opacity of his media; another is to take care to examine each eye separately.

1. **Acuity of vision.**—If this is very much diminished, it may be doubtful whether the patient is able to tell light from darkness. To investigate this, place the patient in a darkened room opposite to a lamp, alternately cover and uncover his eye, or, what is perhaps a better plan, concentrate the light upon his eye by means of a mirror or lens, and ask him to say when it is light and when it is dark.

In lesser degrees of impairment, ask the patient to count fingers. This is done by placing him with his back to the light while the observer, standing facing the patient, holds up a varying number of fingers of one hand, and asks the patient to say how many there are. The test should be applied at varying distances.

For the detection of slight degrees of impairment of visual acuity Snellen's types will be found useful. These consist of letters of different sizes, each of which should be capable of being read at a definite distance—the largest at 60 metres, the smallest at 6. In using the types, the patient is placed with his back to the light, while the types are placed level with the eye at a distance of 6 metres (about 20 ft.). He is then asked to read the letters from above downwards. For the purpose of recording the result, the following symbols are employed:—

V = visual acuity.
d = distance of eye from type (i.e. 6 metres).
D = distance at which type should be capable of being read.

Suppose that at 6 metres the patient is able to read the smallest type—that is to say, that which should be readable at 6 metres off. Then his visual acuity (V) = $\frac{d \text{ (i.e. 6 metres)}}{\text{D (i.e. 6 metres)}}$ or normal.

But if at that distance he can only read the largest size of type—that which one should be able to read at 60 metres—then V = $\frac{6}{60}$.

The term **amblyopia** (literally "blunt-eyedness") is often used to mean defective vision. The term **amaurosis** (literally "darkness") is sometimes used to signify complete blindness.

2. **Field of vision.**—When we fix the eye upon an object, we not only see that object, but also a number of objects in the neighbourhood more or less distinctly. The sum of the objects that form images upon the retina whilst the eye is gazing in one particular direction is called the field of vision. It should be noted that although the field of vision differs

with each different act of fixation, the peripheral limits are the same and are largely determined by the margins of the orbit, nose and cheek. A rough estimate of the extent of the field of vision for large objects may be obtained in the following way:—

Seat yourself opposite to the patient and at a distance of about half a yard from him. If his right eye is to be tested, ask him to place his hand upon his left, and to look steadily at your own *left* eye. Look steadily yourself at the patient's right eye, your own right being closed, and hold up your left hand in a plane midway between his face and your own, and at first at almost full arm's length off. Keep moving the fingers of the hand, and bring it nearer until you can just yourself "with the tail of your eye" catch the movement of the fingers. Then ask the patient whether he sees them, telling him meanwhile to be sure not to take his own eye off yours. If he fails to see the fingers, keep bringing the hand nearer until he does see them. Test the field in this fashion in every direction —upwards, downwards, to right, and to left—using the extent of your own field always for purpose of comparison.

This gives the outline of his field for appreciation of a moving object, which may, however, be relatively intact when the fields for other forms of stimulation are seriously constricted. In consequence, his field for appreciation of a stationary object must also be investigated in a similar manner, but by asking the patient to indicate when he sees the observer's fingers held at rest.

Considering the field of vision in more detail we appreciate that whereas the objects which cause images to fall upon the central part of the retina (the macula) are seen in minute detail and bright colouring, objects further and further from the point

of fixation are seen with less and less distinction and colour until at the periphery of the field we can only appreciate the presence of an object of considerable size without being able to judge its form. We may look upon the field as an island of vision surrounded by a sea of blindness, with a coast-line roughly oval in shape with precipitous cliffs upon the nasal side and more sloping upon the temporal. From these cliffs there arises gradually a plateau that culminates in an eccentrically placed peak with steep sides. To the temporal side of this peak is a pit with sharply sloping sides that rapidly become vertical and extend to the sea level (the blind spot). The shores of the island may become invaded by the sea, or the surface of the island altered by weathering or subsidence, and in this way depressions may form which remain circumscribed, or may actually extend to the shore. It is the object of modern perimetry, as it were, to survey this mountainous island, marking out differences of level by contour lines similar to an ordnance map. The centre-point of the chart corresponds to the visual axis; the point of fixation is therefore the point of most distinct vision. Around this point are arranged a series of more or less concentric lines, each of which denotes equal visual acuity, and is called an *isopter*. For purposes of investigation we divide the visual field into three parts:—

(1) An area surrounding the point of fixation to 20°.

(2) An intermediate area between 20° and 50°.

(3) An outer area from 50° to the periphery.

As stated above, the fixation point is not exactly central, so that the outer and inner part of the field is unequally divided. Further, the boundary is delimited inwards and upwards by the nose and brow. Testing with a large object we find the

outermost limit of the field of vision reaches 104° outwards, 50° upwards, 70° inwards and upwards, whilst down and in (owing to the obstruction of the

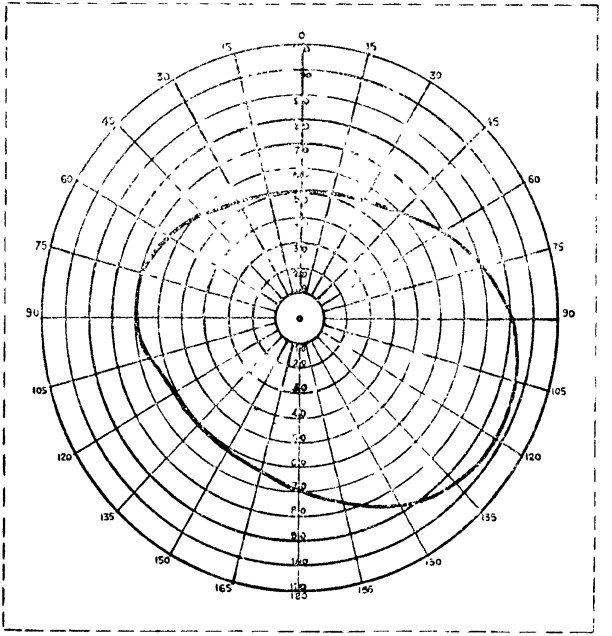


Fig. 91.—Extent of the right field of vision with a white target 20 mm. in diameter mapped on a perimeter at a distance of 33 cm.

most prominent part of the nose) it reaches from 45° to 50° (Fig. 91).

The binocular field extends 200° or more laterally and about 140° vertically, in the middle of which is a circular portion common to each eye with a diameter of about 120°.

On each side of this paired area is a semilunar

area which is unpaired and which accounts for the remainder of the field.

Perimetry is concerned with an investigation of the uniocular field of vision. We have noted that the acuity of perception is very much lower at the periphery and gradually increases as we pass to the point of fixation. We may test this acuity with objects of different size, and we shall find that

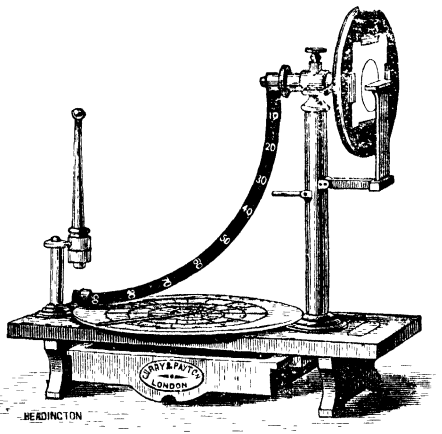


Fig. 92.—Priestley Smith's perimeter.

whereas a very small object is visible at and near the fixation point it fades from view as it is withdrawn towards the periphery. By using a graduated series of objects we are able to plot out a series of isopters each of which corresponds to a known size of object used at a known distance from the eye.

That part of the field between the periphery and the 30° circle is investigated by means of a perimeter, of which the Priestley Smith model is a useful form. (Fig. 92.) It is used as follows:—

i. The patient rests his cheek against the wooden pillar, so that the eye is about 1½ in. above the knob and vertically over it. The height of the instrument is regulated by movable blocks.

ii. The quadrant, which is a flat strip of metal engraved upon its two sides, is rotated by a wooden hand-wheel attached to the axis; it is balanced by a weight upon the hand-wheel, so that it will stand in any position without being fixed.

iii. The test object is a square of paper gummed upon a light vulcanite wand which the operator holds in the left hand With the right hand he rotates the hand-wheel and pricks the chart.

iv. The chart is placed upon the hinder surface of the hand-wheel, and rotates with it There is a mark on the hand-wheel to show which way the chart is to be placed. This mark is brought to the top, and the chart is then slipped in from above downwards and in the upright position.

v. Immediately behind the hand-wheel is fixed a horizontal scale, the divisions of which correspond with the circles on the chart. As the quadrant rotates, the chart rotates with it, and, in whatever position the quadrant stands, the corresponding meridian of the chart stands against the scale. This arrangement enables the operator to prick off his observations with the greatest ease, and has the further advantage that the chart is constantly under inspection, so that any portion of the field can be immediately brought under examination at any time

vi. The charts are of two kinds, A and B The A charts correspond to the entire field and are divided by circles from 0° to 90°, the limits of the average normal field being shown by a dotted line. The B charts are for mapping the central part of the field on a larger scale, and are divided from 0° to 45°. The scale of the perimeter is graduated accordingly on its two sides; the A side is to be used with the A charts, the B side with the B charts.

vii. There are many cases in which it is better to sweep the field, or parts of it, in circles rather than in meridians, e.g. hemiopic and sector-like defects in which the boundary line of the field runs in a meridianal direction. In cases of this kind the test objects may be placed in the clip upon the quadrant, and carried round the field in successive circles.

The first observations are taken with a white object 3–5 mm. in diameter. This is moved up from the periphery and the patient indicates when it first comes into view, and an appropriate mark is made upon the chart.

The object is then carried along the arc up to the 30° circle and the patient asked to indicate if it disappears from view at any point. The arc is now moved through 30° and the examination repeated: if a defective area is encountered the observations must be repeated each 10°, the test object being given centripetal and centrifugal movements and moved in a circular direction in conjunction with the arc so as to map out precisely the edge of the defect. When the field of a 3–5 mm. white object has been recorded the observations are repeated with a 1 mm. white object, which should yield a field (in normal circumstances) not differing appreciably from that of the larger object, showing only a constriction of 5–10° on the temporal side, above and below, whilst actually coinciding with the field for the larger object down and in.

The area within the 30° circle is examined by means of test objects upon a black screen—*Bjerrum's screen*—at a distance from the patient of 1,000 to 2,000 mm.

The patient is seated comfortably at this distance with the head steadied by a chin and head rest, and a grey object 1 cm. in diameter with a black centre is fixed to the screen on a level with the patient's eye.

The blind spot is first of all mapped out with a white object 20–30 mm. in diameter. The peripheral field is next mapped out with a 1 mm. object, and at a distance of 2,000 mm. it should be circular and extend to about 26°, that is to the edge of the 2 metre square screen. With the small object areas of blindness or defective perception should be sought around the blind spot, especially between this area and the macula, the centrocæcal area, and in the horizontal meridian on the nasal side of the fixation spot. The findings are marked upon the screen with black pins, and subsequently transferred to a chart.

Changes in the field of vision.—It may be contracted all round its periphery. This is spoken of as **concentric diminution** of the field of vision. It occurs in hysteria, some forms of optic atrophy, bilateral lesions of the anterior part of the cortical visual centres, and various affections of the retina.

Sometimes the loss of vision is confined to the centre of the field. This is spoken of as a **central scotoma** or as **central amblyopia.** It is frequently

due to toxic causes (e.g. excess in tobacco or alcohol), and is then generally bilateral. Sometimes it is due to local disease of the choroid or of the retina in the neighbourhood of the macula. In that case it may affect only one eye. A unilateral central scotoma is also produced by an acute inflammatory lesion of the optic nerve—optic or retrobulbar neuritis, which in most cases is a symptom of disseminated sclerosis. It may also result from a lesion of the posterior part of the cortical visual centres, and is then bilateral.

The term **hemianopsia** (also written hemianopia and hemiopia) means loss of sight in one-half of the field of vision in both eyes from causes other than disease in the retina. Right lateral hemianopsia means abolition of the right half of the field of vision; left lateral hemianopsia, abolition of the left half. When the same half of both fields of vision is lost, the hemianopsia is described as homonymous, e.g. right homonymous hemianopsia when the blindness occupies the right half of both the right and left fields.

Superior and *inferior hemianopsia* means loss of the upper and lower halves of the visual field respectively. They are of rarer occurrence than the lateral variety, and are sometimes spoken of as *altitudinal hemianopsia*. Hemianopsia limited to one quadrant of the field is described as quadrantic hemianopsia or quadrantanopsia.

Bitemporal hemianopsia means loss of vision in the temporal or outer halves of both fields, and is due, therefore, to loss of visual power in the nasal half of each retina. It can only be produced by a lesion of the optic chiasma, involving those fibres of the optic nerves which decussate, and is accordingly rare. It is commonly due to a tumour of the pituitary body

but may be produced by inflammatory lesions of the optic chiasma.

Binasal hemianopsia signifies a loss of the nasal or inner half of each field, and indicates a diminution of visual power in the temporal half of each retina. It can only be produced by a bilateral lesion confined to the uncrossed optic fibres on each side of the chiasma. It occurs with excessive rarity.

Temporal and nasal hemianopsia are sometimes spoken of as *heteronymous*, in contradistinction to the *homonymous* variety.

3. **Colour sense.**—This is tested by means of Holmgren's wools. Throw all the skeins together on a table in good daylight, keeping the test skein separate. Explain to the patient that he is to match the colour, not to name it, and that he is to select all those skeins which are *like* it, whether they are of a darker or lighter shade or not. Show him first a pure pale-green skein, and ask him to match it. If he does so correctly, his colour vision is normal. If, on the other hand, he selects one of the "confusion colours" (grey, straw colour, etc.), he is to be regarded as colour-blind.

Total colour-blindness is rare. Red-green blindness is the commonest form. Yellow-blue blindness is not nearly so common. If the patient is totally colour-blind he confuses with the test skein all those of equal brightness, no matter what their tint may be. If red-green blindness is suspected, show him a purple skein, and he will select blue as a match for it—indicating that he fails to see the red element in the purple. If he is blue-blind, he will select red or orange.

Colour field.—The field for colour is investigated in the same manner as is the field for white. For the same size of object the fields for colour are less in extent than those for white, so that with an

object of similar size the field for blue is smaller than that for white, and then follow yellow and red, whilst the field for green is the smallest.

The use of coloured test objects is mainly restricted to an examination of the field within the 30° circle with red and green objects, where important information (as shown by the character and shape of the scotoma) is gained by investigating cases of tobacco amblyopia and retrobulbar neuritis. It is, of course, essential that the patient be not colour-blind.

The exact extent of the field for each colour is best tested by means of the perimeter.

Subjective visual sensations may be present. Among the commonest of these for which one may have to inquire is the occurrence of what are known as *muscæ volitantes*—little specks or motes seen floating before the eyes, especially on looking at a white surface or up to the sky. They are not infrequent in anæmic and debilitated persons. In migraine, peculiar zigzag lines, known as "fortification figures," are often seen at the beginning of the attack, and in the investigation of such a case should always be inquired for. The term *teichopsia* is applied to this condition. Hallucinations of sight occur in some cases, notably in delirium tremens; they may also form part of the aura in epilepsy.

Third, Fourth, and Sixth Nerves

It is convenient to take these together, as conjointly they serve to innervate the muscles which move the eyeball.

Anatomy.—The fibres of these nerves take their origin from a series of nuclei which begin in the floor of the aqueduct of Sylvius below the anterior corpora quadrigemina, and extend down as far as the eminentia teres in the floor of the 4th ventricle. The nucleus for the 3rd nerve is highest up; its

most anterior cells supply the ciliary muscle and iris, those for the ocular muscles being farther back. Behind that comes the nucleus of the 4th, and, most posteriorly of all, that of the 6th. The 3rd nerve emerges on the inner aspect of the crus, and is therefore apt to be involved in lesions implicating that part of the brain.

The 4th pair emerge on the anterior part of the roof of the 4th ventricle. They are peculiar in that they are the only cranial nerves which decussate between their nuclei and their point of emergence.

The 6th emerges between the medulla and pons, and runs forward beneath the latter for a considerable distance before leaving the skull. This long course renders it particularly liable to the effects of pressure.

Functions.—The 6th nerve supplies the external rectus, the 4th supplies the superior oblique. All the other ocular muscles, along with the sphincter pupillæ, the muscle of accommodation, and the levator palpebræ superioris, are supplied by the 3rd.

Symptoms of paralysis. *Sixth nerve.* — Inability to move the eye outwards, and diplopia on looking in that direction. Possibly internal squint. In nuclear lesions there is also loss of the power of conjugate deviation of both eyes horizontally to the side of the lesion.

Fourth nerve.—Impaired power of downward movement, and on the attempt to look downwards the eyeball is rotated inwards by the inferior rectus. Diplopia only below the horizontal plane, with the images uncrossed, but the false one tilted. There is rarely a visible squint.

Third nerve.—Ptosis; the eye can only be moved outwards and a little downwards and outwards; pupil usually dilated and unable to contract; loss of power of accommodation.

Paralyses of the 3rd nerve are not infrequently partial—only one or a few of these functions being lost. Thus the levator palpebræ superioris is often alone

affected, producing **ptosis,** while the other muscles retain their normal power. In order to estimate the degree of ptosis, one must eliminate the action of the occipito-frontalis. This is done by pushing down upon the latter muscle so that the eyebrows are kept level, and then asking the patient to look up. The extent to which the lids are raised indicates the strength of the levator. It must be remembered that a smooth muscle in the upper lid, innervated by the cervical sympathetic, exerts a tonic elevating action. Slight ptosis therefore occurs after a lesion of the cervical sympathetic (*see* p. 432).

Any **retraction of the upper lid,** from over-action of the levator, is to be noted by observing the relation of the edge of the lid to the upper margin of the cornea when the patient is looking straight forward.

How to test these nerves.— As will be gathered from the above résumé, the signs of a lesion involving any of these nerves may be—1, defective power of movement of the eye; 2, the presence of a squint; 3, the presence of diplopia. Of these signs the last is really the most trustworthy of all, for paralysis of the muscles supplied by the nerve may be so slight as to lead to no appreciable squint and to no visible defect in mobility.

STRABISMUS

By *squint* or *strabismus* is meant a condition in which the visual axes do not meet at the point of regard. Of this there are two varieties, *paralytic* and *concomitant*, and it is necessary that the two be carefully distinguished.

Paralytic strabismus.—The following are the characters of a paralytic squint:—

(1) *Limitation of movement.*—As its name implies paralytic strabismus is due to loss of power in one or more of the extra-ocular muscles; a prominent

feature, therefore, is lack of ability to move the eye in the direction of the physiological action of the muscle affected. Although this lack of power is usually very apparent, nevertheless sometimes the loss of muscular power is so slight, or the unaffected muscles mask the loss of action of the affected muscle so much, that the defective movement of the eye is hardly visible.

Movements in the so-called *cardinal directions* are tested by fixing the patient's chin with one hand and moving the forefinger of the other in the direction indicated. The eyes move normally 50° outwards, 50° inwards, 33° upwards and 50° downwards.

If an eye fails to move at all, or fails to move throughout the normal angular excursion, the deviation of the eye in a direction opposite to the physiological action of the muscle is called the *primary deviation or squint*, and it is measured by the angle which a line from the object to the nodal point of the eye makes with the visual axis (Fig. 93). If now we cover the unaffected eye and so cause the patient to take on fixation with the affected eye, we shall find that the eye that is covered will deviate still more than the primary deviation of the affected eye (Fig. 94). This deviation of the healthy eye is the so-called *secondary deviation or squint*, and it is this difference in amount between the primary and secondary

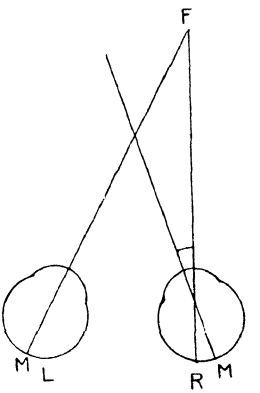


Fig. 93.—Diagram to illustrate *primary* deviation in a case of paralysis of the right external rectus muscle.

With fixation by the left eye, the deviation of the right eye is indicated. F=point of fixation, M=macula.

deviation which is the most important distinguishing feature between paralytic and concomitant strabismus.

(2) *False orientation of the field of vision.*—This is an erroneous judgment by the patient of the position of an object in that portion of the field of vision towards which the paralysed muscle should normally move the eye. Take the case in which a patient has paralysis of the right external rectus muscle. If such a patient closes the left eye and is asked to touch suddenly an object held in the horizontal direction on his right side, he will fail and will strike wide of the object on his right-hand side.

The explanation of the phenomenon of secondary deviation being greater than primary is, that when fixation is assumed by the paralysed eye the same amount of nervous energy passes to the associated muscles of the two eyes, in this example to the right external rectus and left internal rectus As, however, the right external rectus is paralysed an unusually great impulse is needed to stimulate it, the result is that its associated muscle in the left eye is over-stimulated and its movement too great

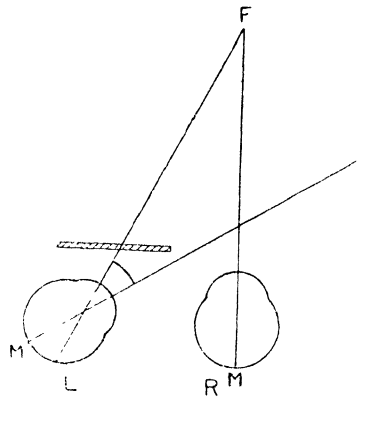


Fig. 94.—Diagram to illustrate *secondary* deviation in a case of paralysis of the right external rectus muscle.

With fixation by the right eye, the left eye when covered by a screen deviates inwards far more than did the right eye during fixation by the left eye (*see* Fig 93).

This same reasoning may be applied to explain the phenomenon of false orientation in that an object is projected into space largely according to the amount of nervous energy expended in moving the eye so as to fix that object, and as in the case of paralysis the amount of nervous energy

expended is in excess of movement, the object is projected too far in the direction of the physiological action of the paralysed muscle.

(3) *Vertigo* is occasionally a symptom of paralytic strabismus when both eyes are opened. It is due partly to the confusion of double sight, and partly to false orientation.

(4) In order to overcome double vision, the patient *turns the head in the direction of the action of the paralysed muscle.* Information may therefore be obtained as to which muscle is involved by noting the way in which the head is held.

(5) *Diplopia.*—Patients with paralytic squint complain of double vision, which is due to the fact that owing to the lack of movement of one eye in a particular direction the images of external objects do not fall upon "identical points" of the two retinæ; this double vision or *diplopia* is therefore in that part of the field of vision into which the paralysed muscle should move the eye were it unaffected.

In health, when fixing an object, the image formed in each eye falls upon the macula, so that not only are the two images of equal intensity and definition, but since they fall upon identical points of the two retinæ they produce but a single image. In paralytic strabismus the image of the object fixed falls upon the macula of the healthy eye and is seen with distinctness and detail, and is called the *true image*, whereas in the affected eye the image falls upon the retina outside the macula, and as in consequence it is indistinct and blurred, it is called the *false image*.

The investigation of a case of paralytic strabismus and the diagnostic value of diplopia.—First of all make certain that the diplopia is *binocular*, since certain conditions, astigmatism, lens opacities, etc., may produce *monocular* diplopia.

Movements in *cardinal* directions have already been mentioned, and they consist of movements up, down, in and out. The main action of each individual muscle is in a cardinal direction, and so they are spoken of as lateral turners, elevators or depressors.

All the rectus muscles arise around the apex of the orbit and pass forwards to be inserted into the sclera, a varying distance behind the cornea. With the eyes in the primitive position the external and internal recti turn the eyes to right and left only. Owing to the direction in which the superior and inferior recti pass to be inserted, although they act mainly as elevators and depressors of the eye they have a second component which causes them to act also as adductors.

The superior and inferior oblique muscles also act mainly as depressors and elevators respectively, but also act in a subsidiary way as abductors.

By turning the eye outwards 27° the superior and inferior recti may be made into almost pure elevators and depressors. Similarly by turning the eye inwards the superior and inferior oblique muscles may be made into almost pure elevators and depressors.

In this way we are able to resolve diplopia into horizontal and vertical (which, we shall see, much simplifies our investigation), and find the field of maximum diplopia in one of the cardinal directions.

When the images in diplopia are separated laterally, so that the right image belongs to the right eye and the left to the left eye, the condition is spoken of as *homonymous diplopia*. If, however, the left image belongs to the right eye and the right to the left, it is called *heteronymous or crossed diplopia*. It will also be found that the real image belongs to the healthy eye, whereas the false image belongs to the paralysed eye.

The production of homonymous diplopia.—If, as the result of paralysis of an abductor muscle (external rectus muscle), there is deviation of the eye inwards (convergent strabismus), the image in this eye will fall upon a point of the retina internal to the macula. Two things will result: (1) the image will not be so sharp as the image in the healthy eye,

proving that the *false* image belongs to the affected eye; (2) since images that fall upon the retina on the nasal side of the macula are projected in space to the temporal side of the eye, it follows that *paralysis of an abductor producing convergent strabismus causes homonymous* diplopia and also that the false image is projected in the direction of the physiological action of the paralysed muscle. (Fig. 95.)

The production of heteronymous or crossed diplopia.—If, as the result of paralysis of an adductor muscle (internal rectus) there is a deviation of the eye outwards (divergent strabismus), the image in this eye will fall upon a point of the retina external to the macula. As seen on p. 408, the false image is produced in the affected eye, and since images that fall upon the retina to the temporal side of the macula are projected into space to the nasal side of the eye, it follows that *paralysis of an adductor producing divergent strabismus causes heteronymous or crossed diplopia* (Fig. 96).

In a similar way it may be shown that in paralysis of an elevator muscle the false image (which belongs to the affected eye) lies on a higher level than the true image, and in paralysis of a depressor muscle the false image lies below the true image (Figs. 97, 98).

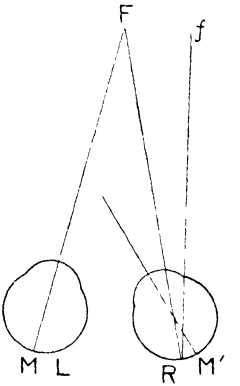


Fig. 95.—In the case of paralysis of an *abductor* muscle (here the right external rectus) the result is *homonymous* diplopia.

F is the point of fixation which produces an image on M, the macula of the left eye, the true image. As the image falls internal to the macula M' in the right eye, it is projected to f, the false image. Thus the false image belongs to the affected eye and is projected in the direction of the physiological action of the paralysed muscle.

Actions of the muscles.—The external and internal recti muscles move the eyes in a horizontal direction, consequently maximum diplopia is produced when the eyes turn horizontally in the direction towards which the paralysed muscle normally turns the eye.

We have seen that the superior and inferior recti muscles become simple elevators and depressors when the eyes are turned outwards, consequently maximum vertical diplopia is produced when the eyes look up and out and down and out respectively.

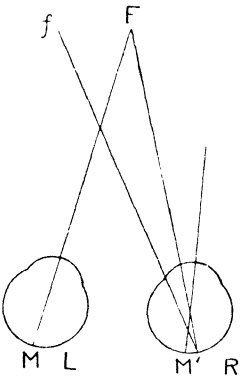


Fig. 96 —In the case of paralysis of an *adductor* muscle (here the right internal rectus muscle) the result is *heteronymous*, or *crossed* diplopia. F is the point of fixation which produces an image on M, the macula of the left eye, the true image. As the image falls external to the macula, M′ in the right eye, it is projected to f, the false image Thus the false image belongs to the affected eye and is projected in the direction of the physiological action of the paralysed muscle

The inferior and superior oblique muscles become simple elevators and depressors when the eyes are turned strongly inwards, consequently maximum vertical diplopia is produced when the eyes look up and in and down and in respectively.

In conjugate movements the muscles of the two eyes act in pairs (for instance, in conjugate movements in the horizontal direction to the right, the right external rectus muscle is linked in action with the left internal rectus muscle) and the table below shows a series of six pairs, each pair representing "true associates."

1. *Muscles moving the eyes laterally.*

(a) To the right:

Right external rectus,
Left internal rectus.

(b) To the left:
Left external rectus,
Right internal rectus.

2. *Muscles moving the eyes upwards.*
(a) With the eyes turned to the right:
Right superior rectus,
Left inferior oblique.
(b) With the eyes turned to the left:
Left superior rectus,
Right inferior oblique.

3. *Muscles moving the eyes downwards.*
(a) With the eyes turned to the right:
Right inferior rectus,
Left superior oblique.
(b) With the eyes turned to the left:
Left inferior rectus,
Right superior oblique.

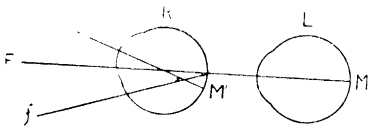


Fig. 97.—In the case of paralysis of a *depressor* of the right eye it will be seen that the object F causes an image to fall above M', the macula of the right eye; the result is that the false image is projected to f, below F, the true image. Thus the *lower* image belongs to the affected eye.

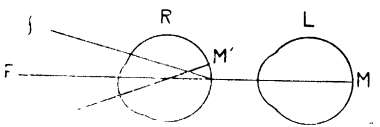


Fig. 98.—In the case of paralysis of an *elevator* of the right eye it will be seen that the object F causes an image to fall below M', the macula of the right eye: the result is that the false image is projected to f, above F, the true image. Thus the *higher* image belongs to the affected eye.

Method of finding the direction of maximum diplopia.—The patient is seated with the head fixed in position (preferably in a head-rest) with a red glass before the right eye, and a green glass before the left. At a distance of about fifteen feet the observer moves a lighted candle in the direction indicated in the subjoined diagram, each of the lateral squares corresponding to a pair of true associated muscles; thus a maximum vertical diplopia produced when the patient looks up and to the right into the right superior square shows that either the right superior rectus or the left inferior oblique muscle is the one affected. It is only necessary to find to which eye the false image belongs to decide which muscle is paralysed. The higher of the two images indicates the affected eye.

L **The patient looks** **R**

Upwards to the left	Upwards	Upwards to the right
LEFT SUPERIOR AREA	SUPERIOR MEDIAN AREA	RIGHT SUPERIOR AREA
To the left	Straight ahead	To the right
LEFT EXTERNAL AREA	PRIMARY AREA	RIGHT EXTERNAL AREA
Downwards to the left	Downwards	Downwards to the right
LEFT INFERIOR AREA	INFERIOR MEDIAN AREA	RIGHT INFERIOR AREA

Table devised by Maddox for charting the field of diplopia in a case of paralytic strabismus.

Maddox points out that in using this chart the area of greatest vertical diplopia is the namesake

either of the affected muscle or its true associate in the other eye, further, that the true associates always bear names which are the most contrary possible, thus *left inferior oblique* is in every term opposite to *right superior rectus*.

Concomitant strabismus.—It has been explained when dealing with paralytic squint that the amount of angular deviation of the two visual axes varies with different positions of the two eyes, and also that secondary deviation is always greater than primary deviation.

In concomitant strabismus, as its name implies, the angular deviation of the visual axes is the same in whatever position the eyes may be, in other words, the primary and secondary deviation are always equal.

As there are certain conditions which simulate squint it is important to make certain that the case under investigation is truly one of squint.

Screen test.—Steadying the patient's head by fixing the chin with one hand causes the patient to fix an object immediately in front of him. Suddenly cover the apparently fixing eye and ask the patient to fix the object with the uncovered eye. If this eye makes any movement in taking up fixation, it must have been previously deviating. If now the eye behind the screen (which was previously fixing) be observed it will be seen to deviate in the same relative direction as was the other eye, and *to the same angular amount*, that is, the primary and secondary deviation are equal.

The clinical features of concomitant strabismus are :—

(1) It always begins in early childhood, over 70 per cent. before the 5th year and the great majority before three years of age.

(2) The movements of the eyes are good in all directions.
(3) Diplopia is practically never a symptom.
(4) The primary and secondary deviation are equal.
(5) The deviating eye often has defective vision.

A squint may be *periodic* or *constant*, and if constant, *monocular* when the same eye deviates whilst the other usually fixes, or *alternating* when either eye fixes indifferently.

Abnormal movements of the eye.—Involuntary rhythmical contractions of the muscles of the eyeball not infrequently occur. The movements usually occur equally in both eyes. To these movements the term **nystagmus** is applied.

It may exist during rest, but is usually more pronounced on voluntary movement, and is then greatest in the plane of the movement that is made, that is, it is horizontal on lateral movement of the eyes, and chiefly vertical on upward or downward movement.

In examining for nystagmus, ask the patient to look straight in front of him, and observe whether the eyes remain steady. Then ask him to look to his extreme right, then to the left, and then upwards and downwards. Observe the rate, amplitude, and rhythm of the nystagmoid movements in each direction.

Conjugate ocular palsies.—In addition to the defects of movement due to paralysis of the individual ocular muscles, weakness or paralysis of the movement of both eyes in one direction frequently occurs. Thus the patient may be unable to look to either side, or upwards or downwards; or the power of convergence alone may be lost. Palsy of lateral conjugate movement indicates most probably a lesion in the neighbourhood of the sixth nucleus of the side to which the movement is weak. The conjugate

vertical palsies are always associated with disease of the corpora quadrigemina, or in the neighbourhood of the oculo-motor nuclei.

If both eyes are kept persistently turned in one direction, the condition is spoken of as **conjugate peviation** of the eyes. It is usually either to the right or to the left. Conjugate deviation of the eyes may be brought about either by a lesion which produces paralysis or by one which causes irritation or spasm. In the former case the eyes (and usually, also, the head) are turned towards the side of the lesion, provided the latter is in the cerebral hemisphere. The patient, in fact, is said "to look towards his lesion." An irritative lesion in a similar situation causes the deviation to be towards the healthy side. If, however, the lesion has its seat in the pons, these rules are just reversed, the deviation being towards the sound side in a paralytic lesion, and towards the affected side in one which is irritative.

Skew deviation of the eyes—in which, for example, one is directed upwards and the other downwards—occurs in certain lesions of the labyrinth, 8th nerve, and cerebellum.

EXAMINATION OF THE PUPILS

This important part of the investigation of a nervous case may be conveniently considered at this stage. The following points must be noted about the pupils in every case:—

1. **Size.**—Compare the size of the two pupils, first in a bright light and then in a dim light. Note whether the pupils are large or small, and whether any irregularity is present. It must be remembered that the size of the pupil in health is subject to great variations. As a rule, the pupils are larger in dark eyes than in light. A much-dilated pupil is often a

sign of nervous exhaustion or instability. Slight inequality of the pupils may also be present in perfectly healthy subjects. We are inclined to think that in such cases the left pupil is usually the larger.

If one pupil is larger than the other, the question arises, Which is the normal? This question is not always very easily answered, but, as a rule, the pupil which exhibits the less mobility is to be regarded as the abnormal one.

2. **Shape.**—Note whether the pupil is circular in outline, as it should be, or whether its contour is irregular. Such irregularities may be due to adhesion of the iris to the lens or to the effects of an old iritis (*see* p. 479). Irregularity in shape of the pupil is often an early symptom in general paralysis of the insane.

3. **Mobility.** (a) **Reaction to light.**—This is a reflex action. The afferent fibres involved are contained in the optic nerve, the intermediate station is in the corpora quadrigemina, and the efferent fibres pass by the 3rd nerve, through the ciliary ganglion, to the pupil-sphincter.

Test.—Examine each eye separately. Place the patient opposite a bright light, be sure his accommodation is relaxed, and cover the eye with the hand. Leave it covered for about half a minute, then withdraw the hand and watch the pupil. It should contract almost immediately, then dilate again a little, and, after undergoing slight oscillations, settle down to its normal size.

The test may also be carried out by concentrating light upon the pupil by means of a mirror or lens, just as one does in testing the light perception.*

* A convenient method is to throw the light on the pupil by the mirror of an ophthalmoscope with a + 8 lens behind. Looking through the lens, one gets a magnified view of the pupil, and small changes in it can be more easily observed.

Owing to the decussation of some of the fibres of the optic nerves at the chiasma, light acting upon one eye affects the centre for pupil contraction of the other eye as well as that of its own side. It is probable also that fibres pass directly between the centres for the two 3rd nerves which aid in bringing about this result. As a consequence, one finds that if light is shut off from one eye both pupils dilate, and if bright light is made to enter one eye both pupils contract. This is known as the *consensual reaction* of the pupils. It should be tested by keeping one eye in the shade while light is thrown into the other. The effect on the pupil of the shaded eye is then observed.

Lesions of the optic nerve, of the mid-brain, or of the oculo-motor nerve or its nucleus, interfere with this reflex contraction of the pupil to light.

Wernicke's hemiopic pupil reaction may be mentioned here Hemianopia, as we have seen, may be due to a lesion of the optic tract between the chiasma and the corpora quadrigemina, or it may be produced by destruction of the optic fibres between the corpora quadrigemina and the occipital cortex, or it may be due to a lesion in the cortical visual centres themselves.

If the lesion is in front of the corpora quadrigemina—i.e. in front of the pupil centre—the reflex contraction of the pupil to light coming from the blind portion of the visual field is lost, whereas, if it is at any point behind that, the contraction of the pupil to light is retained. It is upon this fact that Wernicke's reaction is based. In carrying out the test the light must, of course, be concentrated on the blind halves of the retinæ. Proceed as follows: Place the patient in a dark room with a light beside his head. Hold a large plane mirror in the left hand, and by means of it illuminate both pupils and observe their size Then take an ordinary ophthalmoscopic mirror in the right hand and direct a strong beam of light on to the blind side of the retinæ. If the lesion is in front of the corpora quadrigemina no contraction of the pupils should result; if behind that, they become smaller. Lately, considerable doubt has been thrown on the reliability of Wernicke's reaction for the localization of a lesion involving the visual fibres.

(b) **Reaction to accommodation.**—As is well

known, the pupils become smaller on accommodating for a near object. Convergence of the eyes, accommodation and contraction of the pupils are due to associated muscular contractions. The contraction of the pupils is thus also described as the reaction on convergence and the reaction on accommodation-convergence.

Test.—Hold up one finger close to the patient's nose. Ask him to look away at a distant object. Then suddenly tell him to look at your finger. As the eyes converge to accomplish this the pupils should become decidedly smaller.

If the patient is unable to see, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face, and then asking him to direct his eyes to it.

Argyll-Robertson pupil.—This is the term applied to the condition of pupil usually observed in locomotor ataxy, but also found very frequently in general paralysis and other degenerative diseases of the nervous system, especially syphilitic. It reacts to accommodation, but not to light. Sometimes the reaction to light is not entirely absent, but takes place in a very sluggish fashion. The Argyll-Robertson pupil is usually small, and dilates slowly and imperfectly to mydriatics.

(c) **Cilio-spinal reflex.**—Dilation of the pupil can often be observed to follow irritation of the skin of the neck either by pinching or by the action of a faradic current. It is due to reflex excitation of the pupil-dilating fibres in the cervical sympathetic (p. 432), and is abolished in lesions of that nerve.

Abnormal movements of the pupil.—The term *hippus* is applied to the alternate contraction and dilatation of the pupil, which can sometimes be observed going on rhythmically (*see* p. 479).

Fifth Nerve

Anatomy.—1. The **sensory root** takes origin from the cells of the Gasserian ganglion and enters the lateral surface of the pons at about its middle. The fibres which conduct impulses for light touch and postural sensibility terminate in a large nucleus in the pons, situated near the floor of the 4th ventricle and lying externally to the motor nucleus, while the fibres for pain and thermal sensibility terminate in the "descending" or bulbo-spinal root, which extends as low down as the 2nd cervical segment of the cord Immediately beyond the Gasserian ganglion the nerve separates into its three divisions

The **first or ophthalmic division** supplies the eyeball conjunctiva (except that of the lower lid), and lachrymal gland, the mesial part of the skin of the nose as far as the tip, the upper eyelids, the forehead, and the scalp as far as the vertex.

Paralysis of this division results in loss of sensibility in the area of skin and mucous membrane supplied, and may cause trophic changes in the eyeball, *neuropathic keratitis*. The corneal reflex is abolished.

The **second or superior maxillary division** supplies the cheek, the front of the temple, the lower eyelid and its conjunctiva, the side of the nose, the upper lip, the upper teeth, the lining membrane of the nose, the upper part of the pharynx, the roof of the mouth, the soft palate, and the tonsils.

Paralysis of this division leads to abolition of sensibility in the above area, and loss of the palate reflex.

The **third or inferior maxillary division** is joined by the motor root. It supplies sensation to the lower part of the face, the lower lip, the side of the head, the ear, the tongue, and the lower teeth. It supplies also the salivary glands and, through the motor division, the muscles of mastication, the tensor tympani, and also, perhaps, the tensor palati, although many believe that this muscle is innervated by the spinal accessory.

2. **Motor root.**—This takes origin in a small nucleus lying internally to the chief sensory nucleus, and partly also from the mesencephalic root, which arises in nerve-cells scattered around the aqueduct of Sylvius. It emerges at the

side of the pons, just in front of the sensory division, passes underneath the Gasserian ganglion, and joins the inferior maxillary division, to which it gives its motor fibres.

Paralysis of the whole 5th nerve leads to loss of sensation in the areas of skin and mucous membrane above mentioned, and to defective power of chewing. (Fig. 99.) Trophic lesions may be present, and the salivary, buccal, and lachrymal secretions much diminished; and the sense of taste is occasionally abolished on the anterior two-thirds of the same side of the tongue.

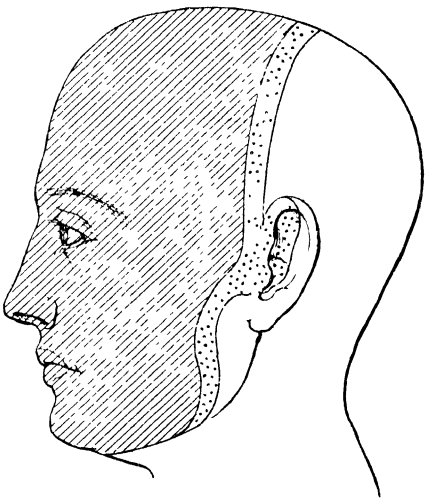


Fig. 99.—Distribution of sensory loss in complete paralysis of the 5th nerve. The shaded area represents complete anæsthesia, the dotted area partial, and chiefly loss to light touch.

One curious result of the sensory paralysis is that the patient, when drinking, imagines that the cup is broken, as he only feels it on one side of his mouth.

How to test the fifth nerve. 1. **Motor functions.**—Ask the patient to clench his teeth while the observer keeps his hands on the temporal and then on the masseter muscles. These should stand out with equal prominence on each side. If there is paralysis on one side, the muscles on that side will fail

to become prominent. On opening the mouth the jaw deviates towards the paralysed side, being pushed over by the healthy external pterygoid muscles. The condition of the tensor tympani muscle cannot be satisfactorily examined except by noting whether there is any difficulty in hearing notes of a particular pitch—i.e. a diminution in the "field of hearing."

2. **Sensory functions.**—The common sensibility of the area supplied is tested in the usual way (p. 452).

Taste.—In suspected lesions of the 5th nerve the sense of taste should always be examined, as it seems probable that, in certain cases at least, taste fibres from the anterior two-thirds of the tongue reach the brain through the 5th nerve As a rule, however, they pass from the lingual nerve into the chorda tympani, and thence through the geniculate ganglion and the nervus intermedius of Wrisberg into the medulla oblongata The taste fibres from the posterior third of the tongue enter by the glosso-pharyngeal nerve, and probably terminate in the same centre in the medulla as those that enter by the nervus intermedius.

The higher connexions of this primary gustatory centre are not accurately known in man, but it is probable that a cortical centre exists in the tip of the temporo-sphenoidal lobe.

How to test the sense of taste.—Have some sugar, some quinine, and some salt, all in powder. Ask the patient to put out his tongue and to keep it out until the conclusion of the test. Many men, especially smokers, are unable to taste on the protruded tongue. The tongue should be then drawn in, but the mouth kept open in order to avoid spread of the test substances. Place some sugar on the tongue, rub it gently in, and ask him, "Is that salt?" If taste is normal he will shake his head. In this way all the substances are tried, first on the anterior part of the tongue and then at the back. A weak galvanic current is also a useful test. It should produce a sort of metallic taste.

Another plan is to use strong solutions of sugar and common salt, and weak solutions of citric acid

and quinine. These are applied by a glass rod to the surface of the protruded tongue, and if the taste is recognized the patient writes down "sweet," "salt," "sour," or "bitter," as the case may be, without withdrawing the tongue. After each test the mouth must be rinsed. The quinine test should be applied last, as its effect is more permanent than that of the others.

Loss of taste may, of course, arise from lesions of the taste fibres in any part of their course above stated.

In addition to loss of taste, one should always ask the patient whether he has any abnormal taste sensations.

Seventh Nerve

Anatomy.—The course of the fibres from the cortical centre to the nucleus of this nerve has already been described (p. 360) The nucleus is situated in the pons externally to that of the 6th nerve On leaving the nucleus the fibres wind round the nucleus of the 6th, and finally emerge mesially to the 8th nerve, between the olive and restiform bodies

The nerve lies in close contact with the 8th, so that a lesion of the one at this part can hardly avoid injuring the other, and enters the internal auditory meatus along with it. During its course in the aqueduct of Fallopius it gives off a branch to the stapedius muscle, and is joined by the chorda tympani, which contains taste fibres from the anterior two-thirds of the tongue In this part of its course the nerve is exposed to the effects of pressure, owing to its being enclosed in a bony tube. It emerges at a point opposite the junction of the anterior border of the mastoid with the ear, and spreads out on the side of the face to supply its muscles In this part of its course it seems to be peculiarly liable to the effects of exposure to cold

Functions.—The 7th is a purely motor nerve. It supplies all the muscles of the face and scalp, except the levator palpebræ superioris. It also supplies the platysma.

Effects of paralysis.—These are usually at once seen on looking at the patient. The affected side of the face has lost its expression. The naso-labial fold is less pronounced than on the other side. The furrows of the brow are smoothed out, the eye is more widely open than the other, and the mouth is somewhat drawn to the healthy side. The patient is unable to whistle, food is apt to collect between his teeth and his gums, and saliva and any fluid he drinks may escape from the affected angle of the mouth.

How to test the seventh nerve.—1. Ask the patient to shut his eyes as tightly as ever he can. Note that the affected eye is either not closed at all—in which case the eyeball rolls upwards to make up for the failure of the lid to descend—or, if the eye is closed, the eyelashes are not so far rolled in as on the healthy side. Try also forcibly to open the eyes while the patient attempts to keep them closed. If the orbicularis is acting normally, it should be almost impossible to open the eye against the patient's wish. If the muscle is partially paralysed, however, the exertion of very little force may suffice to open it.

The effect made in screwing the eyes tightly shut causes the corners of the mouth to be drawn upwards. In paralysis of the lower part of the face the corner on the affected side is either not drawn up at all, or at all events not so much as on the healthy side.

2. Ask the patient to whistle. He is unable to do so.

3. Ask him to smile or show his upper teeth. The mouth is then drawn to the healthy side.

Signs of paralysis of the facial nerve in different parts of its course.—Paralysis of the face presents different symptoms according as the lesion is situated above the nucleus, or either at the nucleus or below it. The former constitutes what is known as cerebral or supranuclear facial paralysis

the latter produces peripheral or infranuclear paralysis.

The chief difference between the two forms is that in *supranuclear paralysis* the lower part of the face is chiefly affected; in *infranuclear paralysis* both the upper and lower parts are equally involved. The probable explanation of this is that the two orbicularis palpebrarum muscles are so often required to act together that each is supplied from both sides of the brain, and consequently a unilateral lesion only partially cuts off the nerve impulses to one side. We describe at page 438 what is meant by "crossed paralysis," and the part which the facial plays in it.

Infranuclear facial paralysis may be produced by a lesion of the nucleus itself, of the nerve-trunk inside the aqueduct, or of the nerve-trunk either after its emergence from the aqueduct or before it has entered it.

A lesion inside the aqueduct—unless it is towards the outer end of the latter—involves the fibres of the chorda tympani, and therefore produces loss of taste sensation in the anterior two-thirds of the tongue. A lesion in any of the other situations produces a typical complete facial paralysis (Bell's paralysis).

A lesion of the nerve before it has entered the aqueduct can be distinguished from a lesion below the stylo-mastoid foramen by the fact that in the former condition the stapedius muscle is paralysed (causing excessive sensitiveness to loud sounds, or "hyperacusis"), while in the latter it escapes. Never omit, therefore, in a case of facial paralysis to inquire regarding the patient's sensitiveness to loud sounds.

Lesions of the nucleus or the nerve below it will result in atrophy of the facial muscles and the

appearance in them of the reaction of degeneration (p. 473). Supranuclear lesions do not produce this effect.

Abnormal facial movements.—The muscles supplied by the facial nerve are frequently affected by spasm or spasmodic movements. These may involve all the facial muscles, or groups of them only. The spasm may be of either the clonic or tonic variety (p. 445). The nature of the movements, if present, their extent, and the muscles affected by them, should always be carefully noted.

Eighth Nerve (Auditory)

Anatomy.—This nerve consists of two sets of fibres. One set supplies the cochlea, and subserves the function of hearing; the other part supplies the vestibule and semicircular canals, and is the nerve of equilibration. The *auditory* fibres, which arise from the cochlear ganglion, enter the brain laterally to the corpus restiforme and form the dorsal root of the 8th nerve; they terminate in the ventral cochlear nucleus and in the tuberculum acusticum. The *vestibular* fibres take origin from the vestibular ganglion, and terminate in a nucleus placed laterally in the floor of the 4th ventricle; they enter the medulla mesially to the corpus restiforme, and therefore form the ventral root of the 8th nerve.

The secondary auditory tracts, after partial decussation, terminate in the posterior corpora quadrigemina and the median geniculate bodies, and another system that takes origin from these passes through the internal capsule to the cortical centre for hearing, in the 1st and 2nd temporo-sphenoidal convolutions. Sounds received in one ear probably reach the opposite hemisphere of the brain predominantly, but owing to the partial decussation of the secondary auditory tracts a unilateral cerebral lesion cannot produce deafness in one ear.

The vestibular nerve is closely connected with the cerebellum. Nothing is known of its cerebral connexions.

Tests. 1. **Hearing.**—Before testing a patient's power of hearing, it is well to exclude the presence of wax in the ear (*see* p. 495). This being disposed of, one can test the hearing power by means of a watch.

Stand behind the patient and ask him to shut his eyes. Begin outside the probable range of hearing power, and bring the watch gradually nearer the ear, asking the patient to speak whenever he hears the tick. One requires, of course, to know at what distance the tick should be audible to a healthy ear. It is necessary to test each ear separately, one being closed whilst the other is being examined.

If impairment of hearing is detected, one must next try to ascertain whether it is really due to disease of the auditory nerve or merely to some affection of the middle ear. In order to settle this point, the **tuning-fork** test may be employed. When the fork is beating strongly, hold it opposite the ear; if it can be heard, then place its base on the mastoid process in order to determine if its vibrations can be heard when conducted through bone. If the patient succeeds in this, ask him to compare the relative loudness of the fork when heard through air and through bone, or to determine which can be heard the longer as the vibrations die out. This is **Rinne's test.** Normally, aerially-conducted sounds are louder to the patient than those conducted through bone. In middle-ear disease, aerial conduction is diminished or lost, while bone conduction remains more or less normal. When the auditory nerve is affected, both air and bone conduction are diminished or lost.

Weber's test, though less reliable than Rinne's, should also be used. Strike a tuning-fork and place the end of it against the centre of the patient's forehead. If the deafness discovered by the watch is due to an affection of the middle ear, the patient will hear the tuning-fork *louder* on that side than on the healthy one. On the other hand, if the deafness is due to disease of the auditory nerve, the tuning-fork will only be heard on the healthy side. The test may also be

carried out by means of the watch. In affections of the nerve, the watch is not heard even when pressed against the ear; in disease of the middle ear, it is heard even more loudly than when similarly applied to the healthy side. The explanation of these facts is not yet clearly made out, nor are they invariably trustworthy. They hold good, however, for a majority of cases. Other points in favour of the deafness being due to a lesion of the nerve and not to disease of the middle ear are, (*a*) if the hearing is better in a quiet place, (*b*) if conversation is heard better than the watch, (*c*) if inflation of the middle ear renders the hearing worse.

Abnormal auditory sensations.—The patient may complain of "ringing in the ears," or **tinnitus.** The precise character of the sound varies in different cases. It may be of a humming, buzzing, hammering, or whistling character. The presence or absence of this symptom should always be inquired for, and whether it is constantly present or in what circumstances it comes on.

Hyperæsthesia of the auditory nerve **(hyperacusis),** by which even slight sounds are heard with painful intensity, sometimes occurs, especially in hysteria and in lesions of the facial nerve above or in the aqueduct, owing to paralysis of the stapedius muscle.

Hallucinations of sound may also be present, the patient fancying that he hears voices, bells, etc. These occur chiefly in states of mental disturbance, but they have been occasionally observed with lesions of the cerebral auditory centre.

2. **Vertigo.**—The patient will describe this as giddiness or dizziness. In order to constitute true vertigo, external objects should seem to move round him. Ask if this is so, and, if it is, in what

direction the objects seem to move. Ask also whether the vertigo ever causes him to fall to the ground.

Vertigo may be due to paralysis of one of the ocular muscles, or to an affection of the ear or vestibular nerve. When a patient complains of vertigo as his chief symptom, one should therefore examine carefully for squint, for disease of the outer and middle ear, and for signs of disease of the 8th nerve.

Ninth (Glosso-Pharyngeal), Tenth (Vagus), and Eleventh (Spinal Accessory) Nerves

Anatomy.—These arise in order from above downwards from an elongated nucleus in the floor of the 4th ventricle They emerge by several roots along the lateral aspect of the medulla, beginning above in the groove between the olive and restiform bodies. The spinal part of the 11th emerges from the lateral column of the cord, beginning as low as the 6th cervical nerve; it passes up through the foramen magnum to join the medullary (accessory) part, and emerges with it through the jugular foramen After its emergence the two divisions of it again part company, the medullary or accessory portion joining the vagus.

The **ninth** (glosso-pharyngeal) is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle constrictor of the pharynx and for the stylo-pharyngeus. It contains the taste fibres for the posterior part of the tongue (*see* p. 422).

How to test the glosso-pharyngeal.—The 9th nerve is rarely paralysed alone. Paralysis of it can best be diagnosed by examining its sensory and reflex functions. Examine the power of taste in the posterior part of the tongue (p. 422). Loss of it *may* mean paralysis of the trunk of the glosso-pharyngeal nerve.

Tickle the back of the pharynx, and note if reflex contraction occurs.

The **tenth** (vagus) is motor for the soft palate (with the exception of the tensor palati), pharynx, and larynx. It is also sensory and motor for the respiratory passages, the heart, and (through the sympathetic ganglia) for most of the abdominal viscera.

The fibres for the soft palate and larynx take origin in the nucleus ambiguus, emerge in the upper roots of the 11th, reach the pharyngeal plexus, and thence pass to the muscles of the palate, the constrictors of the pharynx, and to the larynx.

The viscero-motor and the cardio-inhibitory fibres are derived from the dorsal vagus nucleus in the floor of the 4th ventricle.

How to test the vagus.—Paralysis of the vagus is chiefly evidenced in its palatine and laryngeal branches.

1. **The palate.**—Ask the patient whether he is troubled with the regurgitation of fluids through his nose when he tries to swallow. This is a common occurrence in total paralysis of the soft palate, owing to defective elevation of it during swallowing. For a similar reason the patient is unable to pronounce words which require complete closure of the naso-pharynx. Thus "egg" is sounded as "eng," "rub" becomes "rum," and so on. In unilateral paralysis these symptoms are not observed.

For direct examination of the soft palate, place the patient facing the light with his mouth open, and introduce a tongue depressor. The position of the uvula is quite unreliable as a guide to the state of the soft palate, as deviation of it is not uncommon even in health. One must watch the movements of the palate during phonation. Ask the patient, therefore, to say

Ah, and observe whether both sides of the palate arch upwards; in health, elevation of the palate will occur when the patient says *Ah*. If one side is paralysed, that side will remain flat and immobile, and the median raphe will be pulled towards the other side. The manner in which the palate rises in such a case has been compared to the ascent of a curtain of which one string is broken. In bilateral paralysis the whole palate remains motionless.

2. **The larynx.**—The superior laryngeal branch of the vagus is sensory for the larynx above the level of the true cords, and is motor for the crico-thyroid muscle. Unilateral paralysis of the nerve does not produce any symptoms. Bilateral paralysis causes the vocal cords to be relaxed. The voice is therefore hoarse and deep, and the utterance of high notes impossible.

The recurrent laryngeal branch supplies sensation to the larynx below the level of the cords, and motor fibres to all the laryngeal muscles except the crico-thyroid. Paralysis of it leads to appearances which are recognized by the laryngoscope, and are described at p. 504.

The eleventh nerve.—*Anatomy* —The accessory part of this nerve gives to the vagus motor fibres for the larynx and pharynx. The spinal part of the nerve dips beneath the sterno-mastoid muscle about one inch below the tip of the mastoid process, and emerges from underneath that muscle again at about the middle of its posterior border. It supplies the sterno-mastoid and trapezius, which are also supplied by twigs from the cervical plexus. Lesions of the 11th nerve, therefore, lead to paralysis of these muscles.

How to test the spinal accessory.—Paralysis of the upper part of the trapezius is evinced by asking the patient to shrug his shoulders while the observer offers passive resistance by pressing on the shoulders from behind. Paralysis of the sterno-

mastoid causes weakness in rotation of the chin towards the opposite side.

Twelfth or Hypoglossal Nerve

Anatomy.—The 12th nerve arises from a nucleus in the lower part of the floor of the 4th ventricle, close to the middle line. It emerges between the anterior pyramid and the olive. It is a purely motor nerve, supplying the tongue and the depressors of the hyoid bone. Its cortical centre is in the lower part of the ascending frontal convolution.

Test.—Ask the patient to put out his tongue as far as possible. If the hypoglossal is paralysed, the tongue, instead of being protruded straight, is pushed over to the paralysed side. Be careful not to mistake an apparent deviation of the tongue, really due to the mouth being twisted to one side, for a real deviation of it. Such an apparent deviation occurs in facial paralysis. Ask him also to move his tongue from side to side, and to lick each cheek with it; observe whether he can do so freely. Note whether there is any wasting of the tongue, and whether there is any tremor or fibrillary twitching in it. The presence of wasting indicates that the lesion is either nuclear or infranuclear.

Paralysis of the cervical sympathetic may be conveniently considered here. A complete description of the functions and distribution of the nerve, however, is not necessary in such a work as this. For purposes of diagnosis the fibres supplied to the eyeball are alone of importance. These take origin in the lower cervical and upper thoracic regions of the spinal cord (cilio-spinal centre), from which the fibres emerge in the first thoracic nerve-roots and pass to the sympathetic cord by the rami communicantes. From the cervical sympathetic cord the fibres pass along the internal carotid to the cavernous plexus, and thence via the ophthalmic division of the 5th to

the eyeball. They convey the impulses which cause dilation of the pupil, and supply also the unstriped muscle in the insertion of the levator palpebræ into the upper lid. Paralysis of the cervical sympathetic is recognized by the following signs: Some recession of the eyeball, so that the eye looks smaller than its fellow; slight drooping of the upper lid, due to paralysis of the unstriped muscle-fibres contained in it; contraction of the pupil with absence of dilatation on shading the eye or on instillation of cocaine; abolition of the cilio-spinal reflex; less commonly, absence of sweating, even after the use of pilocarpin, on the corresponding half of the head and neck, both in front and behind, extending as low as the 3rd rib and 3rd thoracic spine, and over the whole of the upper limb on the same side. Sweating of the face can best be induced by making the patient smell mustard.

IV. MOTOR FUNCTIONS

In investigating the motor functions of a patient, one has to satisfy oneself on five separate points:—

1. Is there any muscular paralysis or weakness?
2. Can the patient co-ordinate his actions normally?
3. What is the state of nutrition of his muscles?
4. Is muscle tone altered?
5. Is there any abnormal muscular movement?

1. Investigation of Motor Power

The first thing to be noted as regards the patient's voluntary power is whether or not he is capable of performing gross muscular movements. Can he

walk? Can he sit up in bed? Can he move each of his limbs as a whole?

These main points having been determined, it may be necessary to investigate the range of the movements that the patient can make, and the strength of the principal muscles and groups of muscles separately.

The general rule for one's guidance in this investigation is to ask the patient to throw into action the particular muscle or group of muscles which one wishes to test, whilst the observer offers to that action a greater or less degree of passive resistance. The following is the method of procedure:—

i. **Upper limb.** *Flexors of fingers.*—Ask the patient to squeeze your hand. If a record of the power of grasp is desired, which can be compared with the result yielded in similar circumstances on another occasion, one should make use of the dynamometer.

Interossei and lumbricales.—Paralysis of these muscles gives rise in cases of some standing to a peculiar position of the hand known as "**main en griffe**" or claw-hand. The above-mentioned muscles produce flexion of the first phalanges on the metacarpals and extension of the other two phalanges. Paralysis of them produces, by over-action of the long flexors and extensors of the fingers, over-extension of the first phalanges and flexion of the other two. The fingers are also slightly separated from one another. Claw-hand occurs in some cases of progressive muscular atrophy; and, in a partial form, in paralysis of the ulnar nerve. Claw-foot is an analogous condition.

Opponens pollicis.—Ask the patient to touch the tip of his little finger with the point of his thumb.

Adductor of thumb.—Ask the patient to grasp a book between the forefinger and thumb, keeping the thumb and fingers in the same plane.

Flexors of wrist.—The hand being held with the palm upwards, ask him to bring the points of his fingers towards the front of the forearm.

Extensors of wrist.—The hand being held with the palm downwards, the observer grasps the patient's wrist and asks him to bend the hand up backwards as far as possible. The fingers should be at the same time held flexed, as the wrist can be extended by contraction of the long extensors of the fingers. If he is unable to produce dorsiflexion of the wrist, some weakness or paralysis of the extensors is present.

Slight weakness of the extensors of the wrist may be elicited by asking the patient to grasp something firmly in his hand. If the extensors are weak the wrist becomes flexed as he does so, owing to the flexor muscles getting the better of the extensors.

Weakness or paralysis of the extensors of the wrist leads to the condition known as **wrist-drop.**

Supinator longus.—Place the arm midway between the prone and supine positions; then ask the patient to bend up the forearm whilst the observer offers opposition to the act by grasping the hand. If the muscle is healthy, it will be seen and felt to stand out prominently at its upper part.

Biceps.—The patient's elbow being held against his side, ask him to bend up the forearm while opposition is offered by grasping the hand or wrist. If the biceps is healthy, it will be observed to stand out prominently as it contracts.

The *triceps* is tested by asking the patient to straighten out his forearm whilst the observer endeavours to keep it flexed by means of passive resistance.

Deltoid.—Ask the patient to lift his arms straight out at right angles to the trunk. In paralysis of the deltoid he is unable to do so.

Pectorals.—Ask the patient to stretch his arms out in front of him, and then to clap his hands while the observer endeavours to hold them apart. Note whether both heads of the muscle are thrown into contraction or not.

Serratus magnus.—Ask the patient to push against resistance. In a healthy condition of the muscle its various digitations will be seen to stand out in contraction, whilst the scapula will remain in close apposition to the chest-wall. If the muscle is paralysed, the posterior border and inferior angle of the scapula will come to project more or less when the patient pushes.

Latissimus dorsi.—Ask the patient to clasp his hands behind his back while the observer, standing behind the patient, offers passive resistance to the downward and backward movement; or grasp the two posterior axillary folds and ask the patient to cough. In health the latissimus can be felt to contract.

ii. **Trunk muscles.**—Weakness of the muscles of the abdomen is indicated by the patient being unable to raise himself in bed without the aid of his arms. *Babinski's "rising-up sign"* consists in making the patient lie on his back with the legs extended and rise up without using his hands. In organic spastic paralysis of a leg the affected limb will rise first, owing to the rigidity; but in functional paralysis this does not occur. Paralysis of a portion of the anterior abdominal wall can be detected by the displacement of the umbilicus that occurs when the patient attempts to lift up his head from the pillow against resistance. With paralysis of the lower segment the umbilicus moves upwards, but when the upper segment is affected the umbilicus is pulled downwards. So also with unilateral paralysis the umbilicus is displaced by contraction of the unaffected

muscle (*Beevor's sign*). To test the *erector spinæ* and muscles of the back, make the patient lie on his face and try to raise his head from the bed by extending the neck and back. If the back muscles are healthy, they will be seen to stand out prominently during this effort.

The method of detecting paralysis of the *diaphragm* has already been described (p. 244).

The *trapezius* is tested in its upper part by asking the patient to shrug his shoulders while the observer tries to press them down from behind. In its lower part it can be tested by asking him to approximate the shoulder-blades.

iii. **The head muscles.**—For the methods of detecting weakness or paralysis in the muscles of the head, the reader is referred to the section dealing with the investigation of the Cranial Nerves (p. 392).

iv. **The lower limb.**—The muscles of the foot are tested on the same lines as the corresponding muscles of the hand—passive resistance being offered to their action in each case.

Extensors of knee.—Bend up the patient's knee, and then, pressing with your hand on the sole of his foot, ask him to try to straighten it out again.

Flexors of knee.—Raise his limb from the bed, supporting his thigh with your left hand and his ankle with your right. Then ask him to try to bend his knee.

Extensors of thigh.—The knee being extended, lift the patient's foot off the bed, and ask him to depress it against resistance. If the extensors of the hip are paralysed he will be unable to do so.

Flexors of thigh.—The knee being extended, ask the patient to raise his leg off the bed.

The *adductors of the thigh* are tested by abducting the limb and then asking the patient to bring it back

to the middle line while passive opposition is offered to the act. In a similar way the *abductors* are tested by bringing the limb across the middle line and then asking the patient to move it outwards again.

Rotators of thigh.—With this lower limb extended on the bed, ask the patient to roll it outwards or inwards, whilst passive resistance is offered by grasping his foot.

If, on carrying out any of these tests, a muscle or group of muscles is found to have only a feeble power of contraction, **paresis** of it is said to be present. If no contraction is elicited at all, the condition is one of **paralysis.**

The term **hemiplegia** is applied to a condition in which there is paralysis of one side of the face, and of the arm and leg on the same side. If the paralysis of the arm and leg is on one side, and that of some of the muscles supplied by the motor cranial nerves on the other, the condition is one of **crossed paralysis.** The term **paraplegia** is applied to a paralysis of the lower part of the body; the term **monoplegia** to a paralysis of one arm (which is therefore characterized as a *brachial* monoplegia), one leg (*crural* monoplegia), or one side of the face (*facial* monoplegia).

The detection of hemiplegia in a patient who is comatose is often a very difficult matter. It is to be observed, however, that, if the paralysis is of recent onset, one can usually detect in such a patient a greater degree of *limpness* in the paralysed limbs. If his arm, for example, is raised from the patient's side and allowed to drop, it falls, if it is paralysed, just as if it did not belong to him; the sound arm also falls, but not in such an utterly limp fashion. The face is asymmetrical, the angle of the mouth more open on the paralysed side, and the affected cheek moves loosely outwards and inwards

on respiration. The abdominal and tendon reflexes may be abolished on both sides, but an extensor plantar response is often obtained on the hemiplegic side.

2. Investigation of Muscular Co-ordination

By muscular co-ordination is meant the co-operation of separate muscles, or groups of muscles, in order to accomplish a definite act. If such co-operation is absent or imperfect, the performance of certain acts becomes difficult or impossible, and the condition is then said to be one of **inco-ordination.** The term **ataxia** or **ataxy** has a similar meaning.

The co-ordination or harmonious action of groups of muscles is the product of various factors, among the chief of which are the afferent impulses coming from the muscles that never reach consciousness and those on which the sense of position of the limbs depends; the state of tone of the muscles, and in some acts, perhaps, cutaneous sensibility. When inco-ordination is present it is not always easy to say which of these factors is at fault. The movements that constitute an act can be controlled and directed by vision, but sight itself is not concerned in the co-ordination of movements. When, however, there is loss of the sense of position, the sensory defect may be compensated by vision, and the disturbance of movement may become apparent only when the eyes are closed or bandaged, or in the dark. Such ataxia occurs typically in tabes dorsalis, when sense of position is diminished or lost in the lower limbs.

How to test co-ordination. 1. *In the upper limbs.*—Ask the patient to touch the point of his nose first with one forefinger and then with the other; or ask him to bring the points of the two forefingers together. If he is able to succeed in these tests

naturally and without making random shots, no inco-ordination is present. He may then be asked to perform the same actions with his eyes closed; any additional irregularity of the movements can be due only to disturbance of the sense of position.

Another good test of co-ordination in the upper limb is to ask the patient to thread a needle. In this case, of course, the eyes must be left uncovered.

2. *In the lower limbs.*—If the patient is able to walk, a good test for co-ordination in the lower limbs consists in asking him to walk along a straight line—e.g. the edge of a carpet. If inco-ordination is present he will soon deviate to one side or the other.

If he cannot walk, ask him, as he lies in bed, to place one heel on the opposite knee, first with his eyes open and then when they are closed.

Another method is to leave the eyes open, and then ask him to follow with his toe one's forefinger as it describes circles in the air. If he is able to describe the circles accurately his power of co-ordination is good.

Romberg's sign is often regarded as a special test for the co-ordination of the lower limbs, but though its presence is often evidence that the sense of position in these limbs is defective, it may also be elicitable when the patient's instability is due to some other cause, e.g. aural vertigo or a lesion of the cerebellum. The patient is made to stand with his feet close together, and if he can do so he then closes his eyes. If the sign is present, he begins at once to sway about or may even fall. To elicit slight degrees of the phenomenon, it may be necessary to make the patient stand on tiptoe with his knees bent. The essential feature of the sign is that the patient is more unsteady standing with his eyes closed than when they are open. In tabes dorsalis

this is due to the fact that, owing to deficient sensory impressions from the lower extremities, the patient is unable to maintain his attitude without the aid of vision.

Babinski has described a special sign for cerebellar ataxia under the name **adiadokokinesia**; it consists in inability to execute rapidly-repeated movements. In order to test for it the patient is asked to flex his elbows to a right angle and then supinate and pronate his forearms as rapidly as possible. All normal persons can do this at approximately the same rate, but, as a rule, slightly less rapidly with the left than with the right arm. When, however, adiadokokinesia is present the movements are slow, awkward, and incomplete, and often become impossible after a few attempts.

3. Nutrition of Muscles

It is important to determine if there is atrophy or wasting of any muscles, and if so, which muscles or groups of muscles are affected. This can often be seen by inspection alone, but it is usually necessary to examine them more minutely. The size of the muscles can be most easily ascertained when they are firmly contracted, and consequently the methods that have been recommended in testing their strength should be employed; by this means the absolute and relative bulk of the muscles and the distribution of any atrophy that is present can be made out. The size of the muscles can also be gauged by palpation, and sometimes, as in children, we have to rely on this alone; the wasted muscles are not only smaller but also softer and more flabby than the normal. In some of the primary muscle diseases, as in pseudo-hypertrophic paralysis, some

of the muscles, as those of the calves and the infraspinati, are abnormally firm and large. Such increase in size and firmness must not be mistaken for increased muscular development; it is due to an overgrowth of the interstitial tissue of the muscle at the expense of the muscle-fibres, which undergo degeneration. The information on the state of the muscles that is afforded by the use of electrical stimulation is described in the section on the Electrical Examination of Muscles and Nerves (p. 471).

4. Muscle Tone

By **tone** we mean that slight degree of tension or contraction that is always found in healthy muscles. An increase of tone is spoken of as **hypertonia,** and a diminution as **hypotonia.** We can estimate the amount of tone by handling the limbs and moving them passively. A hypertonic or spastic limb offers a resistance to passive movement, which may be greater in movement in one or other direction according to the distribution of the hypertonia. Sometimes the resistance disappears suddenly, as in flexing the knee of a hemiplegic patient, so that further movement occurs with the expenditure of little or no force, the so-called "clasp-knife rigidity." In other cases it is jerky throughout the whole range of movement; this is, for instance, the rule in the "cog-wheel" rigidity that occurs in paralysis agitans. On the other hand, in hysterical rigidity the muscular resistance increases in proportion to the effort made by the observer to move the limb. In spastic conditions the muscles also, as a rule, feel firmer than normal.

When the muscles are hypotonic, passive movement encounters little or no resistance, and when the

limb is handled or shaken the unsupported segment flops about inertly. The range of movement, too, is generally increased, as it is in tabes dorsalis, though here it is partly due to relaxation of the articular ligaments. Hypotonic muscles are softer to palpation than the normal.

The outstretched upper limb, when hypotonic, as in chorea or cerebellar disorders, usually shows an abnormal posture. It is hyper-extended at the elbow, the forearm is overpronated, the wrist unusually flexed and the fingers at the metacarpo-phalangeal joints are over-extended.

In spastic disorders, due to lesions of the pyramidal tracts, the postures of the limbs and trunk vary according to the situation of the lesion and its severity, but are always determined by the unequal distribution of the hypertonus to opposing muscles. Thus in hemiplegia, the trunk is extended, the upper limb adducted at the shoulder and flexed at the other joints, whilst the lower limb is extended. (It should be understood that the calf muscles are physiologically extensors and not flexors.)

When spastic paralysis is due to lesions of the spinal cord, the lower limbs tend to adopt either an extended posture-paraplegia in extension, or a flexed posture-paraplegia in flexion.

In muscular rigidity in extrapyramidal motor disorders, such as paralysis agitans and Parkinsonism following encephalitis lethargica, the posture of the body is also characteristic. It is one of general flexion, so that the neck and trunk are bent forwards, the upper limbs adducted and flexed with the thumb lying along the palmar aspect of the index finger and abnormally straightened at the metacarpo-phalangeal joint, whilst the lower limbs are also bent. When the patient is at rest, as in sitting or lying, there is a striking immobility or absence of

normal fidgetiness of the body, apart from tremor of the limbs and heads which may be present.

The term **contracture** is applied to a permanent shortening of the muscles, that cannot be overcome by passive movement. It is often seen in long-standing cases of spastic paralysis due to spinal or cerebral lesions, and it may develop in muscles

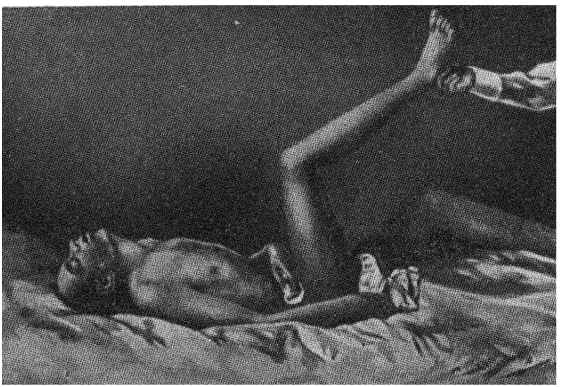


Fig. 100.—Head retraction and Kernig's sign.

that are paralysed and wasted owing to disease of their motor nerves. It is due to secondary fibrous changes in the muscles.

Kernig's sign is often associated with increased muscle-tone. In order to elicit it as originally described, place the patient on his back with the legs relaxed and extended at the knees. On raising him to a sitting posture the knees become flexed, and cannot be straightened when he is again laid upon his back, owing to a strong contraction of the ham-

strings. The sign is more commonly elicited now by flexing the thigh to almost a right angle and then trying to extend the knee (Fig. 100), while the other leg is kept flat upon the bed. If a positive result is obtained the hamstrings are thrown into contraction. Another plan is first to extend the knee fully, then flex the thigh on the pelvis, and measure the angle at the hip. (Rudolf.)

Kernig's sign is present in about 85 per cent. of all cases of meningitis, and is also frequently present in subarachnoid hæmorrhage; also in diseases of the upper motor neurones, after disuse of the lower limbs for some days, as in recumbency; and in local conditions such as sciatica. It commonly coexists with cervical opisthotonos.

5. Abnormal Muscular Movements

These consist of involuntary muscular contractions of various sorts. The first thing to note is whether the movements are widespread or localized.

If they are confined to one part of the body, note the joints at which the movements occur, and the muscles or groups of muscles involved. The term **spasm** is often applied to any exaggerated and involuntary muscular contraction. The contraction may either be continuous, in which case it is said to be **tonic**; or there may be a series of short contractions with complete or partial relaxation of the muscle in the intervals, and in that case they are spoken of as **clonic.**

Tetanic spasm is observed in its completest form in tetanus, strychnine-poisoning, hydrophobia, and some kinds of hysterical fits. It may lead to a bending of the whole body backwards (*opisthotonos*), or sideways (*pleurothotonos*), or forwards (*emprosthotonos*). The jaws may also be firmly clenched (*trismus*).

The term **tetany** is applied to a symptom-complex occurring under widely varying circumstances. The underlying disturbance is in some cases a diminution of serum calcium, and in others an alkalosis. In both groups there is great hyper-excitability of the neuro-muscular apparatus which manifests itself by intermitting spasms of the muscles.

These spasms may affect any muscles of the body, but they most commonly occur in the periphery of the limbs. They are usually bilateral, in severe cases painful, and they may last for ten to twenty minutes and recur over many weeks.

An attack begins with a sensation of tingling and stiffness in the fingers. The thumb is forcibly adducted, the fingers pressed closely together, being flexed at the metacarpo-phalangeal joints and extended at the interphalangeal joints, sometimes the index finger is more powerfully flexed than the other fingers, the palm of the hand is made hollow by the approximation of its outer and inner margins, the whole hand assuming a conical shape—" accoucheur hand." In severe cases the wrists and elbows may be flexed and the shoulders adducted. When the lower limbs are affected the toes and ankles are plantar-flexed, the soles of the feet hollowed out, and the knees and hips extended.

Laryngospasm may occur in tetany. In rickets it is especially common and often appears without the occurrence of tetany. It is commonly termed " *laryngismus stridulus*." At any time in the night or day the child affected will hold its breath until the face is cyanosed. Then the momentary spasm of the glottis relaxes, and as it does so air is drawn past the still closely approximated vocal cords with a high-pitched crowing sound.

In tetany the muscles of the trunk are involved

only rarely, those of the face are sometimes affected, the lips being pursed up—"carp mouth"—and spasm of the oculo-motor muscles sufficient to cause diplopia is occasionally seen.

The following physical signs peculiar to tetany are important, the more so since they often persist between attacks:—

Trousseau's sign.—Pressure upon the vessels or nerves of the limb, for example by a tourniquet or

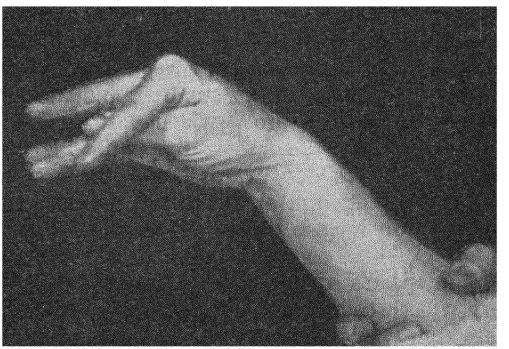


Fig. 101.—Trousseau's sign.

sphygmomanometer bag, will produce the typical spasm (Fig. 101) if absent or augment it if present.

Chvostek's sign.—Tapping over muscles and over nerves superficially situated induces spasm. For example the light tap of a patellar hammer over the facial nerve in front of the lobe of the ear causes muscular twitchings over the whole of that side of the face.

Erb's sign.—Owing to increased electrical excitability of the motor nerves, fibrillation and spasm may

be induced in the muscles by cathodal stimulation with currents subminimal to the normal subject.

Hoffmann's sign.—This comprises marked tenderness of the sensory nerves to pressure and hyperexcitability to galvanism.

Occupational cramps may occur in persons whose occupations involve complicated movements of the fingers for long periods of time, for example, telegraphists and violinists. They consist of irregular tonic spasms and are occasionally associated with paresis, pain, tremor, and even hypertrophy of the muscles concerned.

Conjugate ocular spasm or "oculo-gyral spasm" sometimes occurs as a sequel of encephalitis lethargica. It consists of attacks of spasmodic conjugate ocular deviation usually upwards, which last about half an hour. During the attack, the patient is unable to deviate the eyes downwards below the horizontal plane, and any attempt at downward displacement is associated with intense tremor of the lids (Brain and Strauss).

Clonic spasms are of various degrees of severity. If very widespread they are spoken of as **convulsions,** and are seen typically in epilepsy. If the patient gives a history of fits, their character should be inquired into, following the lines laid down on p. 11. Should the observer be fortunate enough to witness an attack, he should note—

i. *The nature and distribution of the movements.*—Are they general, or confined to one limb or part of a limb? What part is first and what last affected? Are the convulsions tonic or clonic? Is there any struggling, arching of the back, or attitudinizing? Are the abdominal muscles involved or not?

ii. Is there any *involuntary evacuation* of the bladder or rectum? Is there any blood or froth about the mouth? Does the patient change in colour?

iii. *The state of the eyes.*—Is the corneal reflex present or abolished? Do the pupils react to light? Is there any inco-ordinate movement of the eyeballs?

iv. How does the patient behave *after the fit?*

If one group of muscles is first affected, the spasm spreading to others by degrees, it indicates a spread of the discharge along the cortex cerebri. This occurs typically in Jacksonian epilepsy.

Myoclonus is the term given to shock-like contractions occurring in individual muscles. They have been observed in certain epidemics of encephalitis lethargica, the abdominal muscles and the quadriceps femoris being the sites most commonly affected. Their frequency varies from 10 to 80 contractions a minute, but they usually have no definite rhythm.

Tremor consists of more or less rhythmical oscillations of a part or parts of a limb, and is due to the alternate contractions of a group of muscles and its antagonists. Tremor may be either *fine* or *coarse*. Fine tremor is usually more easily felt than seen. It occurs in exophthalmic goitre, alcoholism, and in some forms of metallic poisoning. All forms of tremor are most easily seen by increasing the leverage at which the affected muscles act. Thus, tremor of the upper limbs is often brought out by getting the patient to extend his arms in front of him. In describing tremor, always note whether it is constantly present, or if it is affected in any way by voluntary muscular action. Also observe its rate, the amplitude of the movements, and whether they are regular or irregular. Ask the patient to lift a glass of water to his lips, and note whether the tremor is increased thereby (as it is, for example, in cases of disseminated sclerosis), or whether it is diminished or altogether abolished.

Tremor which only comes on when the patient attempts to use the affected muscles is described as *intention tremor.*

Clonic contraction of individual fibres or bundles of fibres in a muscle is termed **fibrillary twitching.** It is seen in many cases of progressive muscular atrophy, and indicates an abnormal state of nutrition in the spinal cells connected with the affected fibres.

To the transient flickering of a few muscle-fibres (commonly known as " live-flesh " or " live-blood ") the term **myokymia** is applied. It is most often seen in the orbiculares palpebrarum, and is usually an indication of fatigue or debility. It also occurs as an independent condition, and is then more or less general.

The term **choreic** is applied to involuntary movements of a purpose-like character occurring in individual muscles or groups of muscles. Such movements are seen most typically in chorea minor or St. Vitus's dance. They consist of abrupt involuntary twitchings or contractions which cause the patient (usually a child) to seem fidgety and unsettled. They are increased by mental agitation, but are often diminished by voluntary muscular effort. If the movements are limited to one side of the body the term *hemichorea* is applied.

Choreic movements, if slight, can be elicited in two ways. First, one may ask the patient to hold both hands straight up above the head; or, second, one may ask him to spread out his hands, palms downwards, on the extended hands of the observer. In the former case it may be observed that the patient is unable to hold up his hands steadily for any length of time; in the latter, one may notice that little twitchy movements soon become evident in the patient's fingers.

If the patient is able to write, one may get him to scrawl his name with the affected hand, and keep the result for purposes of comparison later. In this way one is able to estimate any increase or diminution in the choreic movements.

Choreiform movements also occur as a result of cerebral disease; they are then usually limited to one side of the body, and as they appear after local lesions they are sometimes described as *post-hemiplegic chorea.*

Tics are co-ordinated, repetitive, purposive acts which are started in the first place by some external cause, or by an idea. By repetition they become habitual and finally involuntary, without any relation to the cause that first excited them. They may assume various forms; perhaps the most common are blinking of the eyes, smacking the lips, or rotation or nodding of the head. They can be distinguished from other involuntary movements by their complexity, and by their always retaining their purposive character.

The term **athetosis** is used to describe slow muscular contractions which lead to continuous and deliberate twisting movements specially affecting the hands and feet.

The last point to be noted regarding any abnormal muscular movement is whether or not it persists during sleep.

V. SENSORY FUNCTIONS

In investigating the sensory functions of a patient, we have to test the acuteness of the following forms of sensibility:—

1. Tactile sensibility. This includes the powers of appreciating light touch and pressure.

The ability to localize the stimulus should be observed at the same time.

2. Sensibility to pain.
3. Thermal sensibility.
4. The sense of position and the appreciation of passive movement.
5. The recognition of the size, shape, and form of objects.
6. The power of appreciating weight.
7. The appreciation of vibration.

In addition, one has to note the presence or absence of any abnormal sensations.

At the outset it is well to explain to the patient the nature of the tests to be performed, so as to secure, as far as possible, his intelligent co-operation. The eyes should then be closed, or the part under examination screened from sight, and the different forms of sensibility tested as follows:—

1. **Tactile sensibility.**—The feather end of a quill pen may be used as a stimulus, but for carefully mapping out areas of altered sensibility a small wisp of cotton-wool, preferably the untreated cotton-wool used by jewellers, is best. It is so light that the element of pressure is entirely eliminated. A fine camel-hair brush also answers the purpose very well. If it is desired to test the sensibility of the skin to light touch over a hairy part, it is essential to shave it first, as the sensibility of the hairs themselves is so acute.

Tell the patient to say " Now " so soon as he feels a touch. Compare corresponding points on opposite sides of the body, and employ every now and then a negative test, asking the patient if he feels you touch him, in order to prevent his making random replies. The appreciation of *pressure touch* should then be

tested; this may be done by touching him with the point of a finger or any blunt object. It is important that its temperature should not differ much from that of the skin, and the pressure must not be so heavy as to give pain or discomfort. Ask him also to localize the stimulus by describing or in other way indicating the exact position of the spot touched. This is important, as a patient may be able to feel the stimulus and yet not be able to localize it.

Sensibility to touch may be altered in various ways. (1) It may be entirely abolished. This constitutes **anæsthesia.** If the abolition affects the whole of one side of the body, it is termed *hemianæsthesia.* If the existence of anæsthesia is discovered, one must at once proceed to mark out its exact extent and boundaries. (2) Sensibility may be so altered that what should in health be felt as a mere touch produces a painful impression resembling pricking or burning. This is generally called **hyperæsthesia.** If hyperæsthesia is discovered, its extent should be carefully mapped out. Hyperæsthetic spots are sometimes met with, especially in hysterical patients. The commonest sites for these are over the brim of the pelvis, in the inframammary region, along the vertebral column, and on the scalp. Pressure on such spots may sometimes induce hysterical fits. If that occurs, the spots are spoken of as " hysterogenetic." (3) Sensation may be appreciated well enough, but there may be great delay in its conduction, an appreciable interval occurring between the application of the stimulus and the response of the patient. This **delayed conduction** exists not infrequently in cases of alcoholic neuritis and tabes. (4) The stimulus may be badly localized, the patient believing, for example, that the outer side of a limb was touched when the

stimulus was really applied to its inner aspect. Sometimes a touch on one side of the body is referred to a corresponding point on the opposite side; this is termed **allocheiria**.

2. **Sensibility to pain.**—Pain may be evoked either by a cutaneous stimulus, as the prick of a pin, or by pressure on the deeper structures, as the muscles or bones. Sensibility to superficial and to pressure pain should be tested separately.

(*a*) **Superficial pain.**—The point of a steel pin or needle may be used as the stimulus. Care must be taken that the patient distinguishes between the sharpness of the point (that is, its relative size) and the pain which the prick evokes; it often happens that even when sensibility to pain is abolished he can recognize that the stimulus is pointed, and thus confuse the observer by calling it "sharp." The relative sensibility to pain may be measured by an algesimeter, by which the observer can determine the amount of pressure on a needle that is necessary to evoke pain.

The application of a faradic current is also an excellent method of testing sensibility to pain. It enables one to estimate the degree of sensibility by noting what strength of current is necessary to cause pain, and then comparing the result with the corresponding area on the opposite side.

(*b*) **Pressure pain** may be examined by pressing firmly on the part with a blunt object, as the end of a pencil, or by squeezing the muscles. The degree of sensibility may be measured by such an instrument as Cattell's algometer, by which the amount of pressure necessary to evoke pain may be determined. Abolition of pressure pain is often the most prominent sensory disturbance in tabes dorsalis.

Absence of sensibility to pain is termed **analgesia ;** and an exaggerated sensibility, so that even a mild stimulus causes an unnatural degree of suffering, is known as **hyperalgesia.**

3. **Thermal sensibility** is most conveniently examined by using test-tubes containing hot and cold water. The part to be tested is touched with each in turn, and the patient says whether each tube feels hot or cold. It is often important to determine the thresholds for heat and cold, i.e. the lowest temperature that feels warm and the highest that is cold. This can be done by noting the temperatures of the water in the tubes on thermometers contained in them; but in attempting this it is desirable to use large copper or glass test-tubes. Note also the reactions evoked by high and low temperatures and the sensations they produce in the patient. It frequently happens that such temperatures evoke only pain, and may be called indiscriminately hot or cold.

The different forms of sensibility already mentioned may have to be tested on mucous membranes as well as on the skin surfaces. The sensibility of some viscera is also important. Thus the absence of pain on squeezing the testicle may be an early sign of tabes.

4. **Sense of position.**—The patient's eyes being carefully shut, take hold of one of his limbs and move it about in various directions through the air, finally leaving it in some definite position, say semiflexed and slightly elevated; then ask him to put the corresponding limb in a similar position. If there is no paralysis of the latter, and yet the patient is unable to imitate with it the position of the other, then there is reason to believe that the sense of position is impaired.

In the case of the hand the patient may be told that

the fingers of one hand will be moved, and that he must imitate with the other the position in which they have been placed. In the case of the foot he may be told that the great toe will be placed pointing upwards or downwards, and that he must try to tell which it is.

In testing a patient's sense of position in this manner, be careful not to allow the part tested to touch any other skin surface; otherwise the patient will be able to appreciate its position by the information derived from his ordinary sense of touch.

A very delicate test for the sense of position in the upper limbs consists in shutting the patient's eyes and then making him hold his arms straight out in front of him with the fingers in a horizontal row. After a moment or two, if the muscular sense is defective, the fingers cease to remain in an even line. Some will rise a little, others fall, or even become twisted in below the rest.

The **appreciation of movement** is closely related to the sense of position, and should be tested at the same time. Grasp any segment of a limb firmly, and then move it gradually into another position; ask the patient to say "Now" so soon as he recognises the movement, and note the angle through which the limb was moved. If the appreciation of movement is diminished this angle is many times greater than that which is necessary in a normal limb, but if the defect is slight it may be necessary to measure the range of the movement accurately for comparison. Movements of, at most, 3° can be appreciated at all normal joints. Finally, test if the patient can recognize the direction of the movement, that is, whether the joint is flexed or extended. It often happens that the patient can recognize the occurrence of a movement though he is ignorant of its direction.

5. The **recognition of size, shape and form.**—These faculties can be tested most accurately in the hands. To test size, place in the patient's palm objects of the same shape, but of different sizes, as small rods or matches of different length. Two objects should be applied consecutively, and he is asked to say which is the larger.

To test the power of recognizing form, familiar objects, as coins, a pencil, a penknife, scissors, etc., are placed in the hand, and the patient is asked to identify them or to describe their form. Loss of this power is generally known as **astereognosis.**

6. **Appreciation of weight.**—Place in the patient's hand substances which resemble one another as far as possible in every respect except as regards weight. Metal balls covered with leather, some being solid and others hollow, are often used for the purpose. In their absence one may use two match-boxes, one full, the other empty, or some other extemporized device. A solid ball and a hollow one may be placed one in the patient's right hand, the other in his left, and he is then asked to state which is the heavier; or one hand may be tested at a time, the balls being lifted one immediately after the other. If the leg is being investigated, the weights should be placed in a handkerchief and slung round the patient's ankle.

7. **Appreciation of vibration.**—If the foot of a vibrating tuning-fork is placed on the surface of the body the vibrations can be felt, provided they are sufficiently strong. This is a valuable test, as the ability to appreciate vibration may be lost in various diseases, as in tabes dorsalis and peripheral neuritis, and in conditions that involve the posterior columns of the cord. Williamson has pointed out that it is frequently absent in diabetes, even though there is

little evidence of neuritis. A heavy tuning-fork of 128 vibrations a second (C.o) is generally employed. Make the fork vibrate by striking its prongs gently on a firm object, and place its foot immediately on the part to be tested; ascertain if the patient perceives the vibrations, and if so, ask him to say at once when he ceases to feel them. If the fork is then transferred to the observer, it can be seen if the sensibility to vibration is diminished or not, and the amount of diminution can be measured by the further time that the vibration can be perceived by the normal parts. The appreciation of vibration is sometimes spoken of as **pallæsthesia.**

Are there any abnormal sensations present ?—These are termed **paræsthesiæ,** and consist in various sensations experienced by the patient in the absence of any outward stimulus. The commonest of these are a feeling of " needles and pins," of numbness, of heats or chills, of pressure or tightness (a good example of the latter being the " girdle pain " of tabes dorsalis), of itching—sometimes termed pruritus —or a feeling as if insects were crawling over the body (formication).

The term **aura** is applied to the curious paræsthesiæ which frequently precede an epileptic fit and serve as a warning of its approach.

VI. REFLEXES

There are three classes of reflexes which one has to test—

1. The superficial reflexes.
2. The deep or tendon reflexes.
3. The organic reflexes (including the action of the sphincters).

We shall consider these separately.

1. Superficial Reflexes

In these the simplest form of reflex action is concerned. On stimulation of a certain part of skin or mucous membrane, contraction of certain muscles results. The path of the impulse is by the sensory nerve-fibres to the grey matter of the cord or to a higher centre in the brain-stem or forebrain, thence by motor nerve-fibres to the muscle. A lesion in any part of this path causes the reflex to disappear. Thus, anæsthesia of the skin, disease of the sensory fibres or posterior nerve-roots, changes in the grey matter of the cord, lesions of the motor nerve-fibres or of the fibres of the muscles, may all cause abolition of the superficial reflexes. In addition to this, it must be borne in mind that the reflex excitability of some individuals is normally very much greater than that of others, and this makes it difficult for one to estimate the value of slight alteration in the reflexes unless the lesion is unilateral, in which case the healthy side can be taken as a standard of comparison. The investigation of the superficial reflexes is of value both as affording information regarding the health or otherwise of the reflex arc concerned and as a guide to the presence or absence of disease elsewhere. In hemiplegia the superficial reflexes are disturbed on the paralysed side.

The chief superficial reflexes of spinal origin, their nature, the mode of obtaining them, and the level of the cord concerned in their production, are given in the table on p. 464.

The **plantar reflex** demands special consideration. In order to elicit it the muscles of the lower limb should be relaxed and care should be taken that the sole of the foot is warm. The outer edge of the

sole of the foot is stimulated by gentle scratching with the finger-nail or a pin. In healthy adults a minimal stimulus produces a contraction of the tensor vaginæ femoris, often accompanied by a slighter contraction of the adductors of the thigh and sartorius. With a slightly stronger stimulus, flexion of the four outer toes appears, which increases with the strength of the stimulus till all the toes are flexed on the metatarsus and drawn together, the ankle being dorsiflexed and inverted. With still stronger stimuli, violent regular movements of the limb occur, which spread to the lower part of the trunk and to the opposite side. The position of the foot at the height of a response to a moderate stimulus is shown above in Fig. 102 (" normal flexor response ").

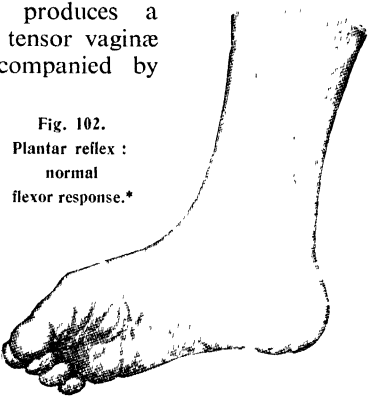


Fig. 102.
Plantar reflex : normal flexor response.*

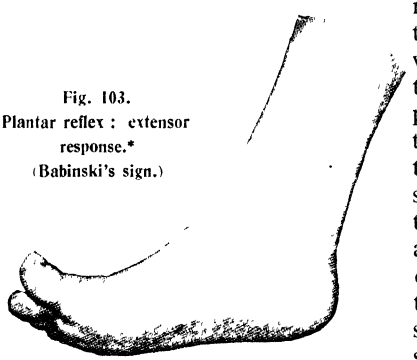


Fig. 103.
Plantar reflex : extensor response.*
(Babinski's sign.)

* After an instantaneous photograph by Dr. James Collier.

It is doubtful whether the plantar reflex is ever constantly and completely absent in healthy subjects.

In infants up to the age of six to twelve months the reflex is very brisk, and differs markedly from that in adults. The earliest response is in the great toe, which is drawn back. This is followed by extension and spreading out of all the toes, with eversion of the foot or dorsiflexion of the ankle, and subsequently by flexion of the hip and knee ("infantile response").

During sleep the plantar reflexes are diminished and the infantile and adult forms preserved, save in some children up to the age of 12 years, in whom in deep sleep the infantile form of reflex returns. In pathological conditions the reflex varies, and may be of great diagnostic importance. In *lesions of the pyramidal systems* an alteration in the type of response was first described by Babinski. In this, which is spoken of as **Babinski's sign,** or the "extensor response," the reflex closely resembles that obtained in infants, but differs in a few points. The whole response is more deliberate than is that obtained either in adults or infants, and appears with much more certainty than does the flexor response to each stimulation. Extension of the great toe precedes all other movement. It is followed by extension of the other toes, dorsiflexion of the ankle, and flexion of hip and knee. The tensor vaginæ femoris does not contract early. The small amount of movement at the ankle is conspicuous, and contrasts with the brisk movement at the ankle in the ordinary response. The extensor response is almost always most easily elicited by stimulation of the outer part of the sole, and, with slight pyramidal lesions, may be evoked from this region alone

when a normal flexor response is obtained by stimulating the inner part. If the lesion is progressive the area in which the extensor plantar reflex can be excited (receptive field) increases, and spreads first inwards over the sole of the foot, and then upwards along the leg to the knee or even the groin. For this reason extension of the great toe, generally associated with some dorsiflexion of the foot, can often be obtained by squeezing the calf or pressing heavily along the inner border of the tibia (**Oppenheim's sign**), or by pinching the tendo Achillis (**Gordon's reflex**), when the upper motor neurones are injured or diseased. Fig. 103 shows the position of the foot at the height of a moderate extensor response.

The extensor response is met with in adults only in cases of organic disease involving the pyramidal tract. In functional cases, if the plantar reflex can be elicited at all, the flexor response is obtained. In tabes and peripheral neuritis the same is true. In neurasthenia, chorea, myopathies, poliomyelitis, and paralysis agitans the flexor response is found; also in intracranial tumours, provided the pyramidal systems are not involved.

The **epigastric and abdominal reflexes** (*see* p. 464) are also extremely valuable signs, as they disappear when the pyramidal tract of the same side is in any way affected. The lower abdominal reflex is then abolished earlier than the epigastric. It is often impossible to obtain them in old or obese people, or in women who have borne many children.

The following superficial reflexes are dependent on cranial nerves:—

i. **Conjunctival.**—Elicited by touching the conjunctiva, resulting in contraction of the orbiculares palpebrarum. The nerves concerned are the 5th (sensory) and the 7th (motor).

ii. **Pupil reflexes.**—(*See* pp. 417 and 479.)

iii. **Corneal reflex.**—Consists in rapid closure of the eyelids on touching the cornea (e.g. with cotton-wool). It depends upon the integrity of the first division of the fifth cranial nerve on the afferent side and upon that of the seventh cranial nerve on the efferent.

iv. **Palate reflex.**—Elevation of the palate on touching the mucous membrane covering it. The nerves concerned are the glosso-pharyngeal and the vagus (or spinal accessory ?).

2. Deep or Tendon-Reflexes

If a muscle is put upon the stretch and its tendon is then sharply struck, the muscle immediately contracts. This is spoken of as a *deep* or tendon-reflex.

The tendon-jerks were at one time regarded not as reflexes but as direct muscular contractions excited by a sudden stretching of the muscles by a blow on their tendons, and it was assumed that they were merely a measure of muscle-tone. But more recent investigations have shown that they are true reflexes and are due to the activity of special reflex arcs. There is, however, a close relation between the state of these reflexes and the condition of muscle-tone, as both disappear when the peripheral neurones are injured; and since the upper motor neurones exert an inhibitory influence both on the reflex arcs and on the ponto-spinal arcs that maintain muscle-tone, when this influence is cut off the muscles become hypertonic and the tendon-reflexes are at the same time increased or exaggerated.

Exaggeration of the tendon-reflexes, unless some other disease coexists, is always associated with lesions of the upper motor neurones—i.e. with lesions affecting either the motor cortex or the fibres passing from

Chief Superficial Reflexes of Spinal Origin

Reflex	How excited	Result	Level of Cord concerned
Anal	Stroking or scratching the skin near the anus.	Contraction of the anal sphincter.	3rd and 4th sacral segments
Bulbo-cavernosus	Pinching dorsum of glans penis.	Contraction of bulbo-cavernosus.	3rd and 4th sacral segments.
Plantar	Stroking sole of foot.	Movements of toes, of toes and foot, or leg.	Lower part of lumbar enlargement (5th lumbar and 1st sacral segments).
Cremasteric	Stroking skin at upper and inner part of thigh.*	Drawing upwards of testicle.	1st and 2nd lumbar segments.
Abdominal	Stroking abdominal wall from costal margin to nipple line.	Contraction of abdominal muscles	11th thoracic to 1st lumbar segment.
Epigastric	Stroking side of chest downwards from nipple	Drawing in of epigastrium on same side.	7th to 9th thoracic segments.
Scapular	Stroking skin in interscapular region.	Contraction of scapular muscles.	5th cervical to 1st thoracic segment.

* The cremasteric reflex can often be most easily elicited by pressing over the sartorius in the low third of Hunter's canal.

it to the anterior horns of the cord. A similar exaggeration may be brought about by anything that stimulates the reflex arc, as strychnine, the toxin of tetanus, exposure to cold, and other factors. Attention or expectation can also induce a state of greater excitability, and the reflex response is greater if the muscles concerned are not fully relaxed; this is probably the explanation of the frequent exaggeration of these reflexes in hysteria and other functional conditions. Increase of the tendon-reflexes is consequently not invariably a sign of organic disease.

On the other hand, anything that impairs the activity of the reflex arc makes it correspondingly difficult to elicit the tendon-reflexes. Their diminution or abolition is therefore always associated with disease of the lower afferent and efferent neurones, or of the reflex centres in the grey matter of the spinal cord. Hence it is that in tabes dorsalis, in which the posterior roots are involved, and in peripheral neuritis, in which both motor and sensory fibres are generally affected, the deep reflexes are absent.

In these conditions the tone of the muscles, too, is generally diminished, and we therefore find absence or reduction of these reflexes usually associated with hypotonia. But the reflexes are not always absent when the muscles are atonic; in cerebellar disease, for instance, they may be brisk or even exaggerated though there is extreme hypotonia.

In a lesion—e.g. a fracture-dislocation—which produced complete transverse destruction of the cord at any level, one might expect that, owing to the cerebral influences being cut off, all the deep reflexes below that level would be exaggerated; but for the first few weeks, at least, all the reflexes are totally abolished. This seems to be due to a state of spinal shock in which the activities of the isolated portion

of the cord are depressed as a result of its severance from the rest of the central nervous system. Later, however, if the general condition of the patient is satisfactory, the reflexes reappear, and a condition may develop in which a single stimulus is capable of producing a widespread effect. Thus, if the spinal cord is divided in the midthoracic region, stimulation of the abdomen or lower limbs may evoke a bilateral flexor spasm of these parts, along with reflex evacuation of urine and perhaps fæces, and an outburst of sweat from the lower limbs and trunk. This reaction has been called a "mass-reflex." But if bedsores, cystitis, or serious nutritional disturbances develop, the reflexes may remain absent or disappear again.

The **knee-jerk** or **patellar tendon-reflex** is the best-known of the deep reflexes. It consists in a contraction of the quadriceps extensor when the patellar tendon is tapped. The spinal segments concerned are the 3rd and 4th lumbar.

How to elicit the knee-jerk.—If the patient is able to sit up, get him to sit on a chair or on the edge of the bed and cross one knee over the other. If he is unable to effect the latter movement, pass your wrist under the knee to be tested, resting your hand on the opposite knee and allowing the patient's leg to swing suspended, as it were, on the back of your wrist. If he cannot sit up, bend the knee up a little as he lies on his back, and support it by allowing it to rest on your hand or the back of your wrist.

The result of this disposition of the limb is slightly to stretch the quadriceps extensor, and so reflexly to increase its tone. The next thing to do is to try to divert the patient's attention. This may be done by asking him to "let the leg hang as if it did not belong to him," or by engaging him in conversation. The patellar tendon must then be struck a sharp blow

midway between the patella and its insertion. The edge of the hand may be used for the purpose, or the edge of a thin book; or the ear-piece of a stethoscope, especially if it is surrounded by a rim of solid india-rubber; or, best of all, a percussion hammer.

Immediately after the blow the foot will be observed to be jerked up, from the sudden contraction of the muscle.

The briskness of the knee-jerk varies greatly in different individuals. In health, however, it is hardly ever entirely absent. Sometimes one is unable to elicit it without having recourse to what is known as **reinforcement** of the knee-jerk. This consists in asking the patient to make some strong voluntary muscular effort with the upper limbs. One may ask him, for example, to hook the fingers of the two hands together and then to pull them against one another as hard as possible. Whilst he is doing so, one tries to elicit the knee-jerk, and one usually gets it more readily than under ordinary conditions. Reinforcement acts by increasing the muscular tone throughout the body.

The following tendon-reflexes are similar in nature to the knee-jerk, but they are connected with the spinal cord, through anterior and posterior roots, at different levels, and so are valuable in diagnosis.

Ankle-jerk.—Place the lower limb on the bed so that it lies everted and slightly flexed. Then with one hand slightly dorsiflex the foot so as to put the tendo Achillis on the stretch, and with the other hand sharply flick the latter on its posterior surface. A sharp contraction of the calf muscles results. This reflex can also be conveniently elicited when the patient is kneeling on a chair. It depends upon the first and second sacral segments.

Adductor-jerk.—This is produced by abducting the thigh and tapping the tendon of the adductor

magnus. Contraction of the adductors results. Sometimes in patients who have very exaggerated reflexes one finds that on tapping the patellar tendon a sudden contraction occurs in the adductor muscles of the opposite thigh. This is termed the **crossed adductor-jerk.** Its explanation is still rather obscure, but it is apparently a truly reflex phenomenon.

Triceps- or elbow-jerk.—Flex the elbow to more than a right angle, then tap just above the olecranon. The triceps contracts. The reflex depends upon the 7th and 8th cervical segments.

Biceps- or flexor-jerk.—Flex the elbow to a right angle and place the forearm in a semipronated position; then strike the lower end of the radius, and the elbow flexes owing to contraction of the biceps and supinator longus. The 5th and 6th cervical segments of the cord are concerned.

Wrist-jerk.—Produced by letting the hand hang down, and then striking the extensor tendons just above the wrist. The hand is jerked up. This reflex depends upon the 6th cervical segment.

Jaw-jerk.—Ask the patient to open his mouth, but not too widely. Place one finger firmly on his chin and then tap it suddenly with the other hand as in percussion. A contraction of the muscles that close the jaw results. This jerk is sometimes absent in health. The motor nucleus of the 5th nerve is the centre involved.

The term *clonus* is applied to the following tendon-reflexes:—

Ankle-clonus.—To elicit this phenomenon, bend the patient's knee slightly and support it with one hand, grasp the fore part of the foot with the other hand, and suddenly dorsiflex the foot. The sudden strain put upon the soleus muscle causes it to contract. The pressure of the hand upon the sole of the foot is meanwhile continued and, when the contraction ceases

causes the muscle again to become tense, and so produces another contraction in the latter. In this way a whole series of contractions—i.e. a clonus—results.

The relative tendency to the development of ankle-clonus on the two sides is best estimated by slowly dorsiflexing the foot and observing the exact point at which the movements first begin. The less the degree of dorsiflexion required to produce the clonus, the greater is the tendency to the development of the latter.

In cases of functional paralysis a spurious clonus may be elicited. It is usually ill sustained and irregular in rhythm, and can be recognized by the feeling of voluntary contraction in the muscles, especially at the beginning of the clonus.

Ankle-clonus is nearly always a sign of disease. The spinal segments concerned in it are the 1st to the 3rd sacral.

Knee-clonus.—In cases where the knee-jerk is exaggerated, one can sometimes elicit a knee-clonus by extending the patient's leg and then suddenly pushing down the patella towards the foot. If the pressure on the latter is continued, a series of clonic contractions of the quadriceps can in many cases be produced.

3. Organic Reflexes and Sphincters

This term includes such processes as respiration, deglutition, micturition, and defæcation. They depend upon complex muscular movements excited by increased tension in the wall of the viscus concerned, or, in the case of respiration, partly by stimulation of a centre in the medulla.

One should always ascertain from the patient whether he has any difficulty in swallowing, noting especially whether there is any regurgitation of food through the nose. The function of **deglutition** does not usually require to be specially tested beyond the

examination necessary to exclude the existence of an obstruction.

Defæcation.—The patient should be questioned as to any difficulty in the act, and as to the presence or absence of tenesmus. Note also the occurrence or not of incontinence of fæces.

The reflex action of the *anal sphincter* may be tested by introducing the oiled finger into the anus, and noting whether contraction of the sphincter occurs with normal force, whether it is weak or altogether inactive, or whether any spasm is excited.

The activity of the anal-sphincter reflex may also be tested by pricking the skin in the neighbourhood of the anus. If the conditions are normal, a brisk contraction of the sphincter should immediately be visible. This depends upon the 4th and 5th sacral segments.

Micturition.—The patient should be questioned as to difficulty or pain in the act (*see* p. 10). One should then note whether there is either *retention* of urine or *incontinence* of it. If there is incontinence, ascertain by the use of the catheter whether it is due to the *overflow* from a distended bladder, or whether it is a *reflex incontinence*—i.e. whether the bladder merely fills up and then empties itself completely by reflex action. In another group of cases the patient feels the desire to micturate, and is unable to restrain the act, which takes place at once. This is spoken of as *precipitate micturition.*

The bladder is connected with two portions of the spinal cord: (1) the 11th and 12th thoracic and 1st lumbar segments, by way of the inferior hypogastric nerves; and (2) the 2nd, 3rd, and 4th sacral segments, by way of the pelvic nerves. The rectum, along with the whole of the large intestine, is also innervated by the pelvic nerves from the 2nd, 3rd, and 4th sacral segments.

VII. TROPHIC FUNCTIONS

In disease of the nervous system the nutrition of different tissues or organs may be impaired. The *bones* may become more brittle from interstitial absorption, or may exhibit spontaneous fracture (osteopathies), or the *joints* may be the seat of painless effusion with or without atrophy or enlargement of the articular ends of the bones (arthropathies). In other cases the bones and joints are involved together (osteo-arthropathies). More commonly the *skin* is the seat of change. It may exhibit an erythema, which may pass on to ulceration and the formation of bedsores at points of pressure; pigmentary changes may develop in it, or it may be the seat of various eruptions—urticarial, vesicular, pemphigoid, or herpetic—or it may be simply glossy. Perforating ulcers may appear, usually on the toes or soles of the feet, as in tabes, or there may be actual gangrene, or the development of painless whitlows. In other cases it is the epidermic appendages which especially suffer change, the hair falling out, or the nails becoming dry and brittle. Atrophy of the *muscles* is a common phenomenon, and may be marked by the appearance of the reaction of degeneration (p. 473). More rarely, certain glandular organs, such as the testis, may be the seat of atrophic change.

In taking a case of nervous disease it is necessary to look for such changes, and to note their situation and extent.

VIII. ELECTRICAL EXAMINATION OF MUSCLES AND NERVES

The principal uses of electricity in the diagnosis of nervous diseases are to afford confirmatory evidence (1) when a lesion of the lower motor neurones

is suspected, and (2) in certain rare diseases of the muscles, especially myasthenia gravis. The increase in the accuracy of clinical methods of diagnosis has, however, led to electrical methods being required much less frequently than was formerly the case. Two forms of electricity yield valuable information —the faradic (interrupted) and galvanic (constant) currents. In health, faradism causes a muscular contraction which persists as long as the current is passing. Galvanism causes a contraction only when the current is made or broken, but not when it is passing.

The cathodal closing contraction is the first to appear: in other words, the muscle responds more readily when the pole applied to it is the cathode, and when the electric circuit is completed; a decidedly stronger current is required to elicit the anodal closing and opening contractions, and the cathodal opening contraction appears last of all. This sequence may be represented by the formula C.C.C.>A.C.C.>A.O.C.>C.O.C.or by the following expansion of it:—

Weak current	C C C.	—	—	—
Medium current	C.C.C ′	A.C.C.	—	—
Fairly strong current	C.C C.″	A C C.′	A.O.C.	—
Very strong current	C.C Tetanus	A.C.C″	A.O C.′	C.O.C.

The contractions in health are abrupt and sharp. Except when the current is very strong, contractions only occur when the circuit is made or broken, not during the passage of a uniform current.

In **disease** the response to electrical stimulation may be altered either quantitatively or qualitatively. By "**quantitative alterations**" one understands that a given current produces a greater or less contraction than it could were the nerves and muscles in

a normal state. "**Qualitative alterations**" involve either or both of two changes—namely, first, the character of the contraction, instead of being abrupt, becomes "sluggish"; second, the cathodal closing contraction is less readily elicited than the anodal contractions.

Such qualitative changes depend on the separation of the nerve and muscle from their nutritive centre. After a brief period the separation induces degeneration, and whilst the degeneration proceeds the nerve first fails to respond to electrical stimuli, and after a longer interval the muscle also becomes insusceptible.

It is during the time that elapses between the insensitiveness of the nerve and of the muscle that the most characteristic electrical changes are observed, and these are together known as the **reaction of degeneration.** They may be summarized as follows:

(1) *Faradic current.*—No response can be elicited, even when a very strong current is employed.

The reason for this is that the faradic stimulus, in consequence of its brief duration, acts only on the nerve, and, since the degenerated nerve can no longer transmit stimuli to the muscle, the latter remains unaffected.

(2) *Galvanic current.* i. *Quantitative change.*—The muscular excitability is increased, contraction following the application of a weaker current than is necessary to produce it in health (the "irritability of weakness").

ii. *Qualitative changes.*—(*a*) The contraction is no longer sharp, but "sluggish."

(*b*) In most cases anodal closing contraction is elicited with a weaker current than cathodal closing contraction. This phenomenon is less constant and less important than the sluggish character of the contraction.

The quantitative change depends partly on alterations in the nutrition of the muscle, and partly on the removal of inhibitory influences; the qualitative changes are produced partly because the nerve no longer regulates the character of the contraction, and partly also as a result of changes in the muscle itself.

The foregoing description applies to a fully developed reaction of degeneration. This is not manifested till more than a week after the trophic influence has been cut off. The nerves, however, begin to lose their sensitiveness about three days after the injury, and gradually become less and less responsive. The muscles behave as do the nerves to faradic stimulation. To the galvanic current they at first exhibit diminished excitability. After eight or ten days the excitability again increases, and eventually exceeds that found in health. At the same time as the increase appears, the sluggishness of contraction and the abnormal sensitiveness to anodal closure become manifest. When the cause which has led to the severance of the nerve and muscle from their trophic centre cannot be removed, the muscular response once more gradually diminishes, and after the lapse of a considerable period, which may extend to two years or even longer, disappear entirely. When the trophic influence is re-established, the reactions of nerve and muscle progressively return to the normal. In such cases ordinary muscular power usually begins to return some days before the nerves respond to electrical stimuli.

It will be readily perceived that, from the standpoint of diagnosis, electricity yields far more valuable information when the disease is situated in the lower motor neurone, thereby severing the nerve-endings and muscles from their trophic centres, than when the morbid process occupies a more central position. Serious disorder may be present in the higher trophic realms without revealing itself by any change in the

electrical reactions—at most there will only be a quantitative change whose detection is often difficult and whose value is uncertain.

Stress has been laid upon the "myasthenic reaction," a mode of response to electrical stimulation which is sometimes found in myasthenia gravis. When this is present, it is found that during faradization with a tetanizing current the muscle gradually loses its power to respond, though it is still capable of some voluntary movement. This exhaustion is not produced by the galvanic current. The myasthenic reaction, however, is by no means constantly present in myasthenia gravis, nor is its occurrence pathognomonic of this disease.

In the sensory system, electro-diagnosis is of less value, and is chiefly of service in cases where an hysterical element is present.

For the technique of electro-diagnosis in nervous diseases the reader is referred to text-books on medical electricity.

CHAPTER X

EXAMINATION OF THE EYE, EAR, THROAT, AND NOSE

I. THE EYE

NOTE first any obvious peculiarity about the eye. Observe whether there is any undue prominence of one or both eyes. **Prominence** of the eyes occurs in exophthalmic goitre. It is associated in that disease with the presence of what is known as *von Graefe's sign.* Ask the patient to look down. If von Graefe's sign is present, the upper lid seems to lag behind the eyeball in its descent, so that a large part of the upper portion of the sclerotic becomes visible.

In paralysis of the cervical sympathetic the eyeball recedes so as to look more sunken than normal (enophthalmos).

Note also whether the winking movements are increased or diminished in frequency. Infrequency of the movements along with an increased size of the palpebral fissure constitutes *Stellwag's sign* of exophthalmic goitre.

The occurrence of squint, ptosis, retraction of the upper lid, and alterations in the pupil have already been considered. The presence of any inflammation along the margins of the lids (marginal blepharitis) should always be noted.

Next turn your attention to the **conjunctiva.** It may be necessary to examine the conjunctiva lining the eyelids. In order to do this in the case of the lower lid,

it is sufficient to depress the latter firmly with the thumb. To expose the inner surface of the upper lid, place the right thumb at the upper part of the upper lid and push upwards, so as to make the eyelashes stand out prominently. Grasp the lashes between the forefinger and thumb of the other hand, and evert the lid by rotating it round the thumb of the right hand. Note the colour of the conjunctiva—whether it is pale, injected, or jaundiced. The method of detecting œdema of it has already been described (p. 353).

Look next at the **cornea**. Note the presence of any ulceration or opacity of it. Small opacities are described as *nebulæ*, larger opacities are spoken of as *leucomata*. Try to make out whether the opacity is really on the surface of the cornea or deeper down in its substance. This can best be determined by looking along the surface of the cornea, as it were, and observing whether the light is reflected from it over the opaque spot, or whether it is dull. If the former is the case, the opacity is deep-seated; if the latter, it is superficial. Such superficial opacities point to former ulceration. Deep-seated opacities are due to a previous keratitis, often the result of a kerato-iritis peculiar to congenital syphilis.

The term *arcus senilis* is applied to a crescentic opacity which is sometimes observed towards the margin of the cornea. It usually appears at the upper part of the cornea first, and then gradually extends round. It occurs normally in old people, and is sometimes seen also in the eyes of younger persons who have sclerosed arterial walls and other signs of premature senility. True arcus senilis can be recognized by its leaving a small strip of clear cornea between the arc and the sclera; a crescentic opacity

extending inwards from the sclera, which is sometimes met with, leaves no such clear strip. If arcus senilis is observed, its presence should always be noted.

It is often of importance in medical cases to be able to say whether a patient is suffering from **iritis** or merely from **conjunctivitis.** In each case the eye looks red and injected, but the characters of the injection are different in the two conditions. They are contrasted in the following table:—

Conjunctival Injection	*Ciliary Injection* (Iritis)
Colour, brick red.	Colour, pink.
Vessels very tortuous.	Vessels straight.
Vessels can be moved on sclera.	Vessels cannot be moved.
Injection greatest on lids and in cul-de-sac, diminishes round cornea.	Injection greatest round cornea, diminishes towards periphery.
Pressure on lid leaves no anæmia.	Pressure on lid leaves temporary anæmic spot.

In taking the case, note specially which of these sets of characters is present.

The **tension** of the eyeball should next be tested. This is done by placing the two forefingers on the upper part of the sclera outside the upper lid while the patient looks downwards, the other fingers resting on his forehead. Then try for fluctuation. The normal tension must be learnt by practice, and any increase or diminution of it noted. An increased tension contra-indicates the use of atropine. Having observed these different points, one should next proceed to what is termed **oblique focal illumination** of the eyeball. For this purpose it is best to have the patient in a dark room, a lamp being placed in front and to one side

of him. By means of a convex lens—the ophthalmoscope lens does very well—the light is focused on the surface of the eyes. If necessary, one may hold another lens in the left hand, and so magnify the view. Study in this way the surface of the cornea. The nature of the opacities already referred to can now be observed more minutely. Look at the iris. The outline of the pupil, its contractility to light, the existence of synechiæ and the presence or absence of hippus, can all be very well observed by this method. Note whether any opacity can be detected behind the pupil, and, if so, try to estimate the depth at which it is situated.

Then proceed to **retinoscopy.** Use for the purpose a plane or slightly concave mirror, with an aperture in the centre. An ordinary ophthalmoscope mirror does very well, but it is preferable to use one of larger size, say about 2 in. in diameter. The patient should be in a dark room with a light just above his head, and it is well to have his eyes atropinized. Sit fully a yard from him, and ask him to look far away over the top of your head. Then throw the light into his eye by means of the mirror. In a normal eye a red reflection from the retina will be seen through the pupil. If there is any opacity of the refractile media the red reflection will be obscured. In this way cataract may be detected. When commencing, it usually takes the form of opaque bands passing in towards the centre of the pupil like the spokes of a wheel. Now tilt the mirror, first upwards and downwards, then from side to side. As one does so, a black shadow is observed to flit across the pupil. Watch the edge of this shadow. From the direction in which it moves information is obtained as to the state of refraction of the eye. If the eye is emmetropic, or if it is hypermetropic, or has less than one dioptre of myopia, the

edge of the shadow moves in the opposite direction to that in which the mirror is tilted if it is concave, but in the same direction as that in which it is tilted if the mirror is plane. In myopia of more than one dioptre the edge of the shadow moves in the same direction as a concave mirror, but in the opposite direction to one which is plane. In a normal eye the shadow moves very rapidly, and has a straight and sharply defined edge. The more abnormal the patient's refraction, the more slowly does the shadow move, and the more crescentic and the less well defined is its margin.

If the edge of the shadow moves differently in opposite meridians, the eye is **astigmatic.** If one meridian is normal the astigmatism is *simple*, and may either be of the myopic or of the hypermetropic variety, according to the nature of the refraction in the abnormal meridian. If both meridians are abnormal, the error of refraction being the same in kind in each, but unequal in degree, *compound astigmatism* is present. It may also be either of the myopic or of the hypermetropic variety, according to the nature of the refraction. If one meridian is myopic, the other hypermetropic, the condition is one of *mixed astigmatism.*

In *regular astigmatism* the directions of greatest and least refraction are at right angles to each other, and usually fall exactly in the vertical and horizontal meridians, the meridian of greatest curvature being most frequently the vertical. Sometimes, however, the directions of greatest and least refraction are in the oblique meridians.

In *irregular astigmatism* the directions of greatest and least refraction are not at right angles. This occurs comparatively rarely.

For the optical explanation of these facts, and for

the more detailed description of their significance, the reader is referred to special works on ophthalmology. We would only remark here that retinoscopy affords the simplest and readiest method of arriving at an idea of the state of a patient's refraction.* In examining many medical cases such information is well worth obtaining, as errors of refraction have been found to be the reflex cause of many nervous symptoms, e.g. of headache, vomiting, etc.

The *fundus oculi* (*see* Plate 23) remains still to be examined, and for this one must have recourse to the use of the **ophthalmoscope.** Many patterns of this instrument are sold. The essential points are that there should be two mirrors—a larger one for use in the indirect method of examination, and a smaller-angled one for direct examination of the fundus. It is also important to be able to bring a series of small lenses of different refraction behind the eye-hole in the mirror.

There are two methods of using the ophthalmoscope—the indirect and the direct. We shall describe the former first.

* Sometimes a patient comes before one wearing glasses, and it may be important to know what their refraction is. In order to discover this, hold the glass in front of the eye and look at an object through it. Then move the glass from side to side, and watch the object. If the latter seems to move in the opposite direction to the glass, the latter is convex; if in the same direction, it is concave.

The strength of the glass may be approximately determined by bringing the small lenses of the ophthalmoscope behind it, until one finds that which abolishes the apparent movement of the object looked at.

In order to tell whether the glass is spherical or cylindrical, look at a straight object, e.g. a window bar, through the glass, and then slowly twist the latter round. If the glass is cylindrical the object looked at will appear to take up an oblique position. Patients who are astigmatic use cylindrical glasses.

1. **Indirect ophthalmoscopy.**—It is best to have the room darkened; at any rate, bright sunlight should be excluded. If the patient is in bed, this may be effected by placing an umbrella over him. The patient (if he is able) sits in a chair with his head *slightly* inclined forwards, and the observer in another about 2 ft. from that of the patient and directly opposite to the latter. A light, usually an electric focus filament lamp with a frosted globe, is placed close to the patient and on a level with his left shoulder. The observer should sit rather obliquely, his left shoulder being turned well round towards the patient. The ophthalmoscope is held in the right hand, the larger mirror being opposite the eye-hole. If the observer is not emmetropic, he corrects his own error of refraction by means of the small lenses behind the mirror; light is then reflected into the patient's eye from the lamp. A red reflection from the retina should fill up the entire pupil unless there is any opacity of the media, as already mentioned. No details of the fundus should yet be visible. If any blood-vessels are seen, one can be sure that the patient's refraction is abnormal. If these seem to move in the same direction as the head of the observer, the patient is hypermetropic; if in the opposite direction, he is myopic. Having illuminated the fundus, one proceeds to interpose the convex lens of the ophthalmoscope. Hold it between the finger and thumb of the left hand, so that it rests opposite the joints between the first and second phalanges of the index. The finger should not be at all flexed. The forearm should be kept vertical, the hand drooping from the wrist, and the little finger supported on the patient's forehead. This is the position demanding the least muscular effort, and therefore the least fatiguing to the observer. The ordinary ophthalmoscopic lens is of about + 13 dioptres

strength, and should be held about $2\frac{1}{2}$ in. from the patient's eye.*

The exact point at which it should be held is arrived at by moving the lens backwards and forwards till a clear image is obtained.

A larger image of the fundus can be obtained by using a convex lens of about + 10 D (4 in. focus) and magnifying the image by means of a + 2 D lens placed behind the mirror. In many ways this is preferable to the ordinary method.

Having focused the fundus, examine its various parts in the following order: (1) the optic disc, (2) the blood-vessels, (3) the macular region, (4) the periphery of the fundus. In order to bring the disc into view, the patient must be made to turn his eye somewhat inwards. If the left eye is being examined, ask him to look at the tip of your left ear; if the right is being examined, ask him to look at the tip of the little finger of the right hand, which is stretched out for the purpose beyond the handle of the ophthalmoscope.

In order to see the macular region, ask the patient to bring the eye slowly back from the above position towards the centre of your forehead. The macular region is reached at about two discs' breadth from the

* A lens of 1 dioptre (1 D) strength has a focal distance of 1 metre (40 in.); a lens of 2 D has a focal distance of 20 in.; and so on. To signify a convex lens the sign + is used, thus: + 2 D means a convex lens of 2 dioptres; — 2 D means a concave lens of 2 dioptres. A lens which is curved equally in every direction—i.e., which is part of a sphere—is called *spherical*. It may be either convex or concave. A concave spherical lens of 1 dioptre strength is indicated thus: — 1 D spher.

A *cylindrical* lens is part of a cylinder, and is therefore curved in one direction only. The direction corresponding to the axis of the cylinder is uncurved, and is spoken of as the axis of the lens. A convex cylindrical lens of 1 dioptre strength is written + 1 D cyl. As mentioned on p. 481 (footnote), cylindrical lenses are required in astigmatism.

margin of the disc. Attempts to look straight at the macula in this way are sometimes baffled by the great contraction of the pupil which results, and by the reflection of light from the surface of the cornea. When this is the case it is better to ask the patient to look, not straight at the forehead, but a little to one side. The observer must then move his head till a view of the macular region is obtained.

The periphery of the fundus is seen by asking the patient to look first to his extreme right and left, then up to the ceiling, then downwards.

If, on gradually withdrawing the lens, the image of the fundus appears to become larger, there is *myopia* present; if it becomes smaller, there is *hypermetropia.* If it alters in one direction and not in others, there is *simple astigmatism;* if it alters in one direction more than others, the astigmatism is *compound.*

If, on moving the lens from side to side, one part of the fundus seems to move over the rest ("parallactic movement"), that part is at a higher level than its surroundings. Thus, if the disc is excavated, its margin will appear to move over the deeper part.

The examination of the fundus, and especially of the peripheral region, is greatly facilitated by a preliminary dilatation of the pupil. The best way of effecting this is to apply a 2-per-cent. aqueous solution of homatropine and cocaine to the eye about half an hour before examining it. The effect must be afterwards counteracted by instilling a drop of eserine solution (1-per-cent.).

If the patient is unable to leave bed, indirect ophthalmoscopy may easily be carried out by the above method, provided he is able to be propped up. If that is impossible, place the lamp on the pillow

above his head, and carry out the rest of the procedure as above.

2. **Direct ophthalmoscopy.**—The patient is placed as before, but with the light a little behind and above the shoulder corresponding to the eye under examination. The observer sits so that his eye can be advanced to within 2 in. of that of the patient. In examining the left eye of the patient, use your own left eye, and for his right use your right. Tilt the patient's head and your own in opposite directions, so as to avoid breathing one another's breath. Arrange the ophthalmoscope with the small oblique mirror opposite the eye-hole, and its surface directed towards the light. The apex of the wedge formed by the tilt of the mirror should be directed towards the root of the observer's nose, when the instrument is held flat against his cheek. If there is difficulty in getting proper illumination of the fundus, move the source of light about until the bright-red reflection is seen through the pupil. Ask the patient to look over your shoulder at a distant object, and try to relax your own accommodation entirely. This is the real point of difficulty in the direct method. As one is desirous of seeing clearly the fundus of the patient, and as that is so near, one almost instinctively accommodates one's own eye for a near object. With practice, however, this difficulty can be overcome. It sometimes helps one to achieve the desired result if one tries to think in a dreamy way of some distant object, and to picture it to oneself. If it is found impossible at first to relax one's accommodation completely, it may be nullified by the use of a -2 or 3 D lens behind the mirror.

When the fundus has been brought into clear focus, its different regions must be systematically studied, just as in the use of the indirect method. In order to

see the disc by the direct method, look backwards and inwards obliquely into the eye, telling the patient meanwhile to look straight in front of him.

In the case of a patient who is unable to rise from bed, the direct method is most easily carried out by means of an electric ophthalmoscope. If this is not available, the ordinary instrument may be used in one of the following ways:—

(1) The observer kneels beside the bed at right angles to the patient, the light being placed on the pillow at the opposite side of the head of the latter. (2) The observer places himself at the top of the bed, so as to look down, as it were, on the patient's eyes, the light being placed on the opposite side of the head from the eye to be examined. (3) If the patient is a child, place him across the bed, and kneel at the patient's head, the light being held at the opposite side from the eye under examination.

The images furnished by the two methods of ophthalmoscopy differ. In the indirect method the image is inverted, so that what seems to be the upper part of the fundus is really the lower, and the inner (nasal) side appears to be the outer, and vice versa. The image, however, embraces a large part of the fundus at one time, so that one gets, as it were, a bird's-eye view of it. This method is, therefore, well suited for ordinary diagnostic purposes in medical cases.

The image obtained by the direct method is an upright image; consequently the different parts of the fundus are seen in their proper positions. The image embraces only a small part of the fundus at one time, but gives a magnified view of it. It is, therefore, well suited for the minute study of pathological changes in the fundus.

The image obtained by the modification of the

indirect method already described (i.e. by interposing a weak convex lens, and magnifying the image by a + 2 D lens behind the mirror) is intermediate between the images obtained by the ordinary indirect and by the direct method. It is an inverted image, pretty highly magnified, and shows also a fairly large part of the fundus at one time.

The optic disc, the blood-vessels, the macular region, and the periphery of the fundus must be studied in detail in each case.

1. **The optic disc.**—Note—

(1) Its **shape.** Normally this is circular. Sometimes it is oval. If there is astigmatism present, the disc will appear to be oval, although it is really circular. This apparent oval shape may be distinguished from that which is real by moving the lens backwards and forwards. If the disc is really oval, it remains unaltered; if it is only apparently oval, its shape will be found to vary with the position of the lens.

(2) Its **colour.** The normal disc is of a rosy tint, but distinctly paler than the rest of the fundus. The nasal side is normally rather redder than the other.

In atrophy of the optic nerve the disc becomes very pale, and may even be dead-white or greyish in tint. In active hyperæmia of the disc its colour approaches in intensity to that of the rest of the fundus. Such active hyperæmia is often present in high degrees of hypermetropia. In passive hyperæmia of the disc the veins are alone affected, and the general tint is not altered.

(3) The presence or absence of a **physiological cup** (*see* Plate 23), and its size. Do not mistake the pallor produced by a very large cup for the pallor of optic atrophy.

(4) The **edge of the disc.** It should be clear and well defined—especially at its outer side. As the

vessels run across it, they should not be observed to tumble over at all. This "tumbling over," if present, is best evinced by the sudden disappearance of the central light stripe on the vessel.

(5) The **surroundings of the disc.** This part of the fundus should be carefully searched if one is looking for hæmorrhages or miliary tubercles, as both of these are more often encountered in the immediate neighbourhood of the disc than at other parts of the fundus.

2. The **blood-vessels.**—The arteries are normally distinguished from the veins by the following characters: They are only two-thirds to three-quarters the breadth of the veins, and they are not so dark in colour. They have a broader, better defined, and more continuous light stripe along their centres.

Normally, the arteries do not pulsate. They may be observed to do so in cases of aortic regurgitation and in increased intra-ocular tension. The veins sometimes pulsate, even in normal eyes.

A point where an artery crosses a vein should be chosen for observation. In health it should be possible to see the vein walls through the artery, neither artery nor vein should be altered in direction at such a crossing, and neither should the calibre of the vein be reduced.

3. The **macular region** is situated about two discs' breadth from the outer edge of the disc. It is recognized by being rather darker in colour than the rest of the fundus, by being very devoid of blood-vessels, and frequently by being surrounded with a halo of reflected light, producing a shot-silk appearance. The macula itself is in the centre of the region, and is rather pale in colour, and often glitters somewhat.



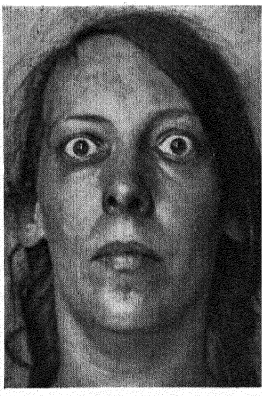
(*Hamblin*)

1. Normal fundus oculi.
2. Primary optic atrophy.
3. Papillœdema.
4. Opaque nerve fibres.
5. Hypertensive retinitis.
6. Syphilitic choroido-retinitis.

Plate 23—THE FUNDUS OCULI.



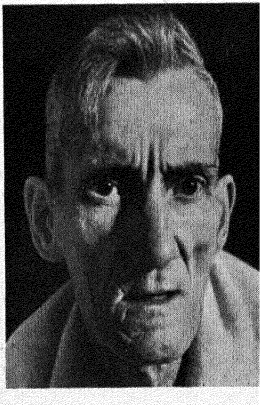
Myxœdema



Hyperthyroidism



Nephrotic Œdema



Dyspeptic Facies

Plate 24.—FACIES.

Changes in the macular region are important, in that they interfere more with vision than similar changes in any other part of the fundus. In cases of hypertensive retinitis a circle of white spots may often be observed arranged around the macula (*see* Plate 23).

4. **Periphery.**—Inspection of the periphery of the retina is important, as it is here that some changes—such, for example, as disseminated choroiditis (*see* Frontispiece) and retinitis pigmentosa—are first to be detected.

The following is a brief description of the chief changes met with in the fundus which are of importance from a medical point of view:—

Papillœdema was at one time known as optic neuritis. It arises as a result of pressure and not of inflammation. It is usually bilateral and begins as a mere passive congestion of the disc, with slight œdema. At this stage the veins are fuller than normal; on indirect examination the edge of the disc seems clear enough, but on closer inspection by the direct method it is seen to be slightly fluffy-looking. The change in the edge of the disc usually begins at its upper and lower margins. These parts should therefore always be most carefully inspected.

In cases of raised intracranial pressure intense œdema of the optic disc may result in a swelling of 8—9 D, a condition known as "choked disc," an expression that well describes the condition seen. The veins are distended and tortuous and here and there the path of the vein is hidden by exudation, and hæmorrhages occur on the surface of the swollen and distorted disc.

As the process progresses, the œdema increases and the disc becomes definitely swollen (*see* Plate 23). This is best recognized by the fact that, on direct examination, one requires (provided one's

accommodation is fully relaxed) the aid of a convex lens behind the mirror in order to bring the vessels on the disc clearly into focus. The veins are still larger than before, and distinctly tortuous. Pathological tortuosity of the veins occurs at right angles to the plane of the retina. Tortuosity in the same plane as the retina may be quite normal. Often the veins can be observed to tumble, as it were, over the edge of the swollen disc. The arteries are smaller than normal, and may be partly obscured by the presence of exudation. The edge of the disc is no longer clear, even on indirect examination, but fades off into the surrounding retina. Small hæmorrhages may be observed on or near the disc.

It is often important to decide whether the papill-œdema is advancing or not. One ought not to form an opinion on this point unless one has already examined the disc on a previous occasion. The best criterion is the degree of swelling of the disc. In order to estimate this, use the direct method, and be sure that your own accommodation is thoroughly relaxed. Notice first whether the retina can be seen quite clearly without the aid of a lens. If the eye is emmetropic, one ought to be able to do so. If the refraction is abnormal, place behind the mirror the lens which is required to bring the vessels on the retina clearly into focus. Then look at the vessels on the disc. Owing to the swelling of the latter, the vessels are nearer the observer's eye than they should be, and a + lens must therefore be brought behind the mirror in order to enable one to focus them clearly. The strength of the lens required is the measure of the amount of swelling which is present in the disc. Suppose, for example, that one requires to use + 1 D in order to focus the retina clearly (i.e. the patient has 1 D of hypermetropia), but that in

order to focus the vessels on the disc one requires to make use of a + 6 D, then there is obviously + 5 D of swelling. Roughly speaking, every 3 D = 1 mm. of swelling. In this way one can estimate the amount of swelling from day to day, and so determine whether the condition is advancing or receding.

The above method, it is obvious, necessitates a considerable amount of practice, and it is absolutely essential for success that the observer should be able thoroughly to relax his own accommodation.

Those who are unable to do this may use the following method instead of the above: Bring behind the mirror the weakest — or strongest + lens with which (*a*) the vessels on the fundus can still be clearly defined; do the same for (*b*) the vessels on the top of the disc. Then

$$\frac{b - a}{3} = \text{the height of the disc in millimetres.}$$

Papillœdema is present in most cases of cerebral tumour at some period of the disease. It occurs also in about 50 per cent. of cases of tuberculous meningitis, although it is often late in making its appearance. It is very uncommon to meet with it in the meningococcal meningitis of infants, and in ordinary acute meningitis. It is also unusual in cerebral abscess, and is not met with in cases of cerebral hæmorrhage or thrombosis.

On the other hand, swelling of the optic disc is not infrequently seen in other than intracranial diseases, especially in chronic nephritis, malignant hypertension, and in some cases of anæmia.

Particular attention should be paid to the condition of the blood-vessels, as this may not infrequently help in deciding the cause of the swelling of the optic disc. Examine carefully the crossings of arteries and veins (*see* p. 488).

Papillitis (intra-ocular optic neuritis) is an inflammatory swelling of the optic disc which occurs as part of a neuro-retinitis. It should not be confused with papillœdema, for the pathology of the two conditions is quite different, although the ophthalmoscopic appearances may be very similar. Generally speaking, in the optic neuritis of a neuro-retinitis the swelling is moderate—2 to 3 D—gradually fading into the surrounding retina, which shows signs of inflammation (exudates, hæmorrhages and so on). The distension of the veins is less marked than in papillœdema.

In those cases in which an optic neuritis has arisen in the retrobulbar part of the nerve, there may be a true descending neuritis which will give the appearances mentioned with little or no signs of inflammation of the retina.

Optic atrophy. The most striking change in the fundus in this condition is the pallor of the disc and the smallness of the arteries on it. The atrophy may be primary, secondary, or consecutive. It is not always easy to say from a mere inspection of the fundus which variety it is that one has to deal with, and the longer the process has gone on the more difficult does the diagnosis become.

(*a*) **Primary or simple atrophy.**—The thinning of the nerve fibres renders very visible the structure of the lamina cribrosa, and the disc acquires a mottled appearance (*see* Plate 23).

(*b*) **Secondary or post-neuritic atrophy.**—Here the atrophy is secondary to papillœdema. The edges of the disc are indistinct and white streaks can be seen radiating along the vessels into the retina.

(*c*) **Consecutive atrophy.**—Here the atrophy is consecutive to changes in the retina, the disc looks like a bit of dirty parchment and pigmentary changes will be seen in the retina.

Retinal hæmorrhages may be observed in hypertension, chronic nephritis, aplastic anæmia, acute leukæmia, and thrombopenic purpura. When superficial, the hæmorrhages are elongated with so-called flame-shaped edges. When deep they occur as dark red blotches or as minute discrete rounded spots.

Embolism of the central artery of the retina.—The appearance of the fundus is characteristic. Look at the macular region for a peculiar round cherry-red spot. The disc itself is pale and its arteries are empty. The retina as a whole is somewhat milky-looking from the presence of œdema.

Retinal arterio-sclerosis occurs either as part of generalized decrescent arterio-sclerosis or as a complication of hyperpiesia. The principal changes are: (1) Tortuosity and irregularity in the lumen of the arteries; (2) nipping, indentation or deflection of the veins where they are crossed by the arteries; (3) small flame-shaped hæmorrhages and (4) small sharply-defined white patches of exudate in the region of the macula.

Hypertensive retinitis.—This condition, met with in some cases of Bright's disease, was originally called albuminuric retinitis. However it is not found except in the presence of high blood-pressure and therefore it is more appropriate to call it hypertensive. The changes consist in (1) the presence of papillœdema with marked fullness of the veins; (2) the occurrence of hæmorrhages on or near the disc; (3) the development of white shining spots around the disc at a distance of about three discs' breadth from it, and of similar but much smaller spots arranged in a stellate form around the macular region (*see* Plate 23), and (4) retinal arterio-sclerosis. Any one of these sets of changes may be present without the others.

Diabetic retinitis may bear a close resemblance to hypertensive retinitis, but distinguishing character-

istics are often present. The hæmorrhages tend to occur in the deeper layers of the retina and are therefore punctate or blot-like. The white masses of exudate are discrete and waxy, and there is no star figure at the macula. Retinal arterio-sclerosis is commonly present.

In leukæmic retinitis the appearances vary. The red reflex and blood-vessels are pale, the veins enlarged, and round white spots up to 2 mm. diameter occur, sometimes fringed with blood.

Disseminated choroiditis.—This is frequently an important sign of previous syphilis. There are small white patches of various shapes and sizes surrounded by heaped-up black pigment (*see* Plate 23). That the lesions are situated in the choroid can be recognized by the fact that the retinal vessels pass over them.

Choroidal miliary tubercles may be looked for in cases of suspected acute miliary tuberculosis and tuberculous meningitis. They will be recognized as ill-defined, rounded, dull yellow lesions, usually about half a disc's breadth in diameter. As, however, they may only appear late in the disease they are usually of little diagnostic importance.

Opaque or medullated nerve fibres occur in the form of one or more broad streaks of brilliant white, radiating for a short distance from the disc and showing a characteristic feathered edge (*see* Plate 23). The condition is a harmless congenital abnormality.

II. THE EAR

Examine first the **external ear.** Note any peculiarity of shape, observe the presence of any tophi, tumour, or swelling, or the existence of any skin eruption upon it. Observe whether there is any discharge from the meatus; if so, make a note of its character,

colour and odour. Sometimes *Aspergillus niger* grows in the external auditory meatus and then the inspissated discharge is black. Note also whether there is any redness, tenderness, or swelling over the mastoid.

The **meatus** and **membrane** must next be inspected. Daylight can be used for the purpose. Place the patient with the ear to be examined turned away from the light. Use a slightly concave mirror of about 3 in. diameter and a focal distance of 6 in., with an elliptical slit in the centre. In order to catch the light properly the surface of the mirror should be turned slightly upwards. By this means the external auditory meatus can be inspected. Note the presence of any foreign body, of an accumulation of wax, of eczematous eruption or furuncles, or any other abnormality, as the presence of such may contraindicate the use of the speculum.

In order to see the membrane a **speculum** must be employed. A metal speculum is best, unless one wishes to make use of caustic applications, in which case it is better to use a vulcanite instrument. Choose a size of speculum appropriate to the ear under examination, warm it slightly, and introduce it so that the long diameter of its smaller end is placed almost vertically, but with a slight inclination from above downwards and forwards. Take care not to introduce it beyond the cartilaginous part of the meatus.

Having introduced the speculum, hold it in position by means of the forefinger and thumb of one hand, while the pinna is grasped between the ring and middle finger of the same hand, the mirror being held in the other. The pinna should be pulled gently upwards and backwards, so as to straighten the meatus as much as possible. The mirror is held with its surface looking slightly upwards as before, and the

membrane can then usually be seen on looking down the speculum. If the view is obstructed by the presence of impacted wax, the latter should be removed by being first softened with warm almond or olive oil or soda solution (two teaspoonfuls of the bicarbonate to one pint), and then syringed out.

The student will find that it is not easy at first to hold the speculum properly and at the same time to pull the pinna upwards and backwards. It is therefore better to have a head-mirror attached to the forehead by means of an adjustable tape. This leaves both hands free to manipulate the ear and speculum, or to make applications by means of instruments.

If an ear mirror is not at hand, the large mirror of an ophthalmoscope can be made to serve the purpose. The lens of the ophthalmoscope can also be held close to the speculum, and so a magnified view of the membrane be obtained. This is especially serviceable in examining the ears of children.

A better magnified view of the membrane can be obtained by aid of the electric **auriscope.** This instrument consists of a metal cylinder, connected at one end with an ear speculum, and at the other with an adjustable magnifying lens. Light from a tiny bulb operated by a dry battery is admitted through a tube in the side of the instrument. The auricle is drawn upwards and backwards with the left hand and the speculum gently introduced with the right. It is advisable to use the largest speculum which is practicable.

The first thing noticeable about the *normal membrane* (Plate 25, Fig. 1) is its bluish-grey colour and translucency. A small white knuckle-like prominence may be observed towards the middle of its upper part. That is the short process of the malleus (Fig. 104, *h*). Passing downwards and backwards from it may be

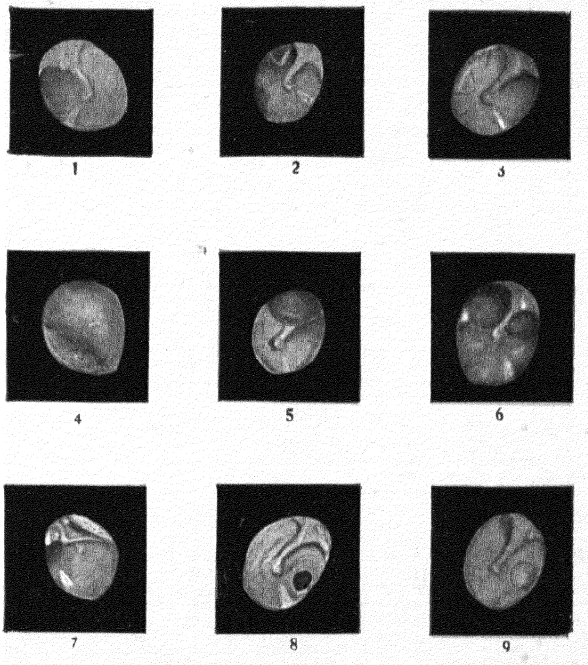


Plate 25.—THE TYMPANIC MEMBRANE IN HEALTH AND IN DISEASE.

Fig. 1.—The normal drum.
Fig. 2.—Healthy but transparent membrane.
Fig. 3.—Acute inflammation, early stage (radial injection of vessels).
Fig. 4.—Acute inflammation (bulged membrane).
Fig. 5.—Acute inflammation limited to attic (bulging of Shrapnell membrane).
Fig. 6.—Catarrhal exudate in middle ear.
Fig. 7.—Indrawing and catarrhal exudate in a case of adenoids.
Fig. 8.—Perforation in chronic otitis media.
Fig. 9.—Cicatrix in thickened membrane.

After Porter, "Diseases of the Throat, Nose and Ear."

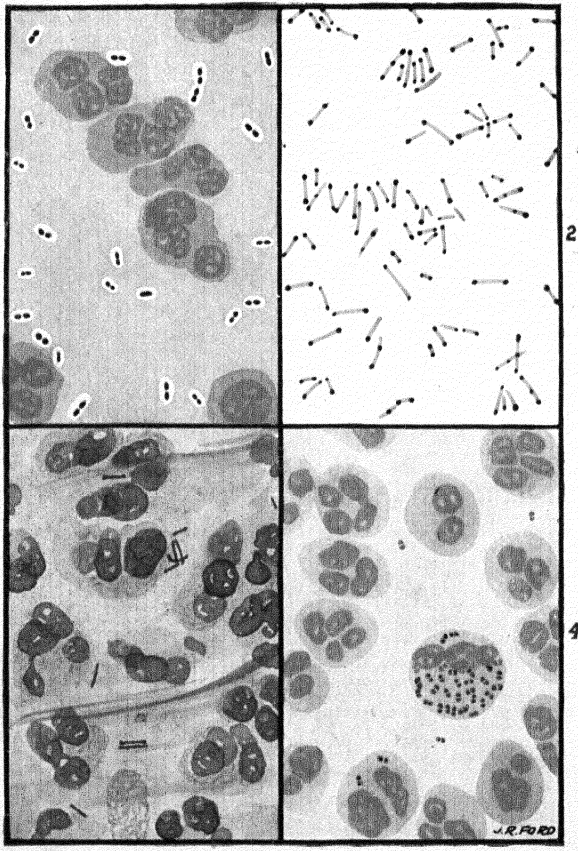


Plate 26.—BACTERIA.

1. Pneumococcus in sputum. Stained by Jensen's modification of Gram's method.
2. Bacillus diphtheriæ grown on Loeffler's blood-serum. Stained by Neisser's method.
3. Bacillus tuberculosis in sputum. Stained by Ziehl-Neelsen's method.
4. Gonococci in pus. Stained by Gram's method.

noticed the long process of the malleus, which ends in the umbo near the centre of the membrane. Passing forwards and backwards from the short process of the malleus will be seen the anterior and posterior folds of the membrane. A triangular light portion of the membrane usually catches the eye, the apex of which meets

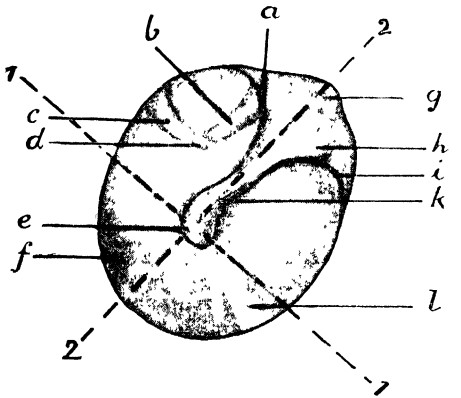


Fig. 104.—The drum membrane, enlarged. *(From Potter's "Diseases of the Throat, Nose and, Ear")*

a, Posterior fold, *b*, long process of incus, *c*, tendon of stapedius muscle; *d*, head of stapes; *e*, umbo; *f*, shadow of niche to fenestra rotunda, *g*, Shrapnell's membrane (membrana flaccida); *h*, short process of malleus (processus brevis); *i*, anterior fold. *k*, handle of malleus (manubrium mallei), *l*, light reflex; 1, 1, and 2, 2, imaginary lines dividing the drum membrane into four quadrants.

the lower end of the handle of the malleus at an obtuse angle which opens forwards. This bright spot is due to the reflection of light. Its presence may usually be accepted as proof of a healthy state of the membrane. Immediately above the short process of the malleus a notch may be observed in the ring of bone which bounds the tympanic membrane. The part of the

membrane which fills in this notch is called the membrana flaccida, or Shrapnell's membrane. It would be beyond the scope of this work to enter into a description of the various abnormal appearances which may be met with in the tympanic membrane (Plate 25). For the purpose of describing the situation of any abnormality which may be observed, it is customary to divide the membrane into an anterior part, which is in front of the handle of the malleus, and a posterior part, which lies behind it. Each of these is then divided, by an imaginary line drawn through the tip of the handle, into a superior and an inferior portion. Four quadrants are thus obtained (Fig. 104), and in making notes one should say that a perforation (e.g.) is seen in the anterior superior quadrant, and so on.

Inflation of the middle ear.—It is sometimes desirable to inflate the middle ear with air. This is best effected by aid of a Politzer's bag. The bag should have a piece of rubber tubing, about 1½ in. in length, attached to its nozzle. Give the patient a mouthful of water, and bid him keep it in his mouth until he is told to swallow it. Introduce the rubber tubing into the lower part of one nostril, pinch firmly the other nostril and the upper part of the one into which the tubing has been introduced, and, holding the bag in the palm of the right hand, tell the patient to swallow. The moment the larynx rises, squeeze the bag firmly, and the air will enter the middle ear.

To ensure the entry of the air into the ear, or to direct it into one ear only, the **passage of the Eustachian catheter** may be necessary. Hold the instrument lightly near the broad end with the thumb and two fingers of the right hand. With the thumb of the other hand push the point of the patient's nose gently upwards. Pass the end of the catheter into the nostril

with the curve of the instrument looking downwards, and the handle somewhat lower than the point. Pass it backwards along the floor of the inferior meatus, and as soon as the curved part has entered the nostril raise the handle of the instrument until it is level, and continue to push it backwards until it comes in contact with the posterior wall of the pharynx. Then rotate the instrument till its point is directed towards the middle line, and withdraw it until the curve hooks against the posterior end of the nasal septum. Now rotate the instrument, so that the point sweeps downwards and then upwards and outwards, the handle being kept pressed towards the nasal septum, and stop when the ring of the instrument is directed towards the outer canthus of the eye of the same side. The point of the instrument can then usually be felt to be arrested by the cartilaginous rim of the tube. The nozzle of a Politzer's bag may now be introduced into the outer end of the instrument, and the inflation accomplished.

If one end of a rubber tube, with an ear-piece at each extremity, is inserted into the ear of the patient, and the other end into that of the observer, the latter can hear the sound which the air makes as it impinges against the membrane. If a whistling sound is heard, it indicates the existence of a dry perforation. A bubbling sound betrays the presence of fluid.

III. THE THROAT

The methods of examining the fauces and the pharynx have already been considered (p. 49). In order to obtain a view of the larynx, one must have recourse to laryngoscopy. In performing **laryngoscopy** the patient and observer should be seated opposite to one another in a darkened room, and about a foot apart. A light should be placed a little to the

right (or left) of the patient's head and on a level with his mouth. An ordinary lamp will serve, but it is better to have a frosted electric bulb, and it is also an advantage to have the light fitted with a bull's-eye condenser. The observer adjusts the reflector to his head by means of a forehead band or spectacle frame. If the former is used, the two knobs on the band should go against the root of the nose. It is then rotated on its ball-and-socket joint until the hole in the centre is directly opposite the right eye. This is ascertained by closing the left eye and observing whether one has a clear view through the aperture. One can also arrange the reflector so that it is in the centre of the forehead, and one then looks under its lower edge. This requires a little practice, but has the advantage of allowing one to make use of both eyes. It is also of advantage in the former method to have the aperture in the centre of the reflector in the form of an elongated slit rather than of a round hole, as a better view is thus obtained.

The observer should next manipulate the reflector with both hands until the light is directed on to the patient's mouth. He then selects a mirror, and warms it face downwards over the lamp until the moisture, which at first condenses on the surface, has all dried off. He should also touch his cheek with the back of the mirror before inserting it, in order to make sure that it is not too hot. Having warmed the mirror, he should hold it in such a way that it can be readily introduced and manipulated. On the whole, it is more convenient to hold the mirror like a pen than in any other way. It should also be held rather short, so that the hand of the observer can be steadied by resting the little finger on the patient's cheek.

The mirror being ready, the patient is told to crane out his neck a little, and to open his mouth and put

out his tongue. The observer then throws a clean dry cloth over the anterior part of the latter, and grasps it firmly but gently between the forefinger and thumb of the left hand. It must be held firmly but without any squeezing, and should then be, as it were, rolled out, as if round an imaginary axis situated near the hyoid. This manœuvre has the advantage of causing a better elevation of the epiglottis, whilst it prevents any risk of injuring the tongue against the lower incisor teeth. Before introducing the mirror, make sure that the light from the reflector is concentrated on the back of the patient's throat. This having been ascertained, the mirror should be introduced with its surface turned almost directly downwards, and passed rapidly back, care being taken to avoid touching either the tongue or the palate. The patient should be told to be sure to breathe regularly and through the nose. This serves to engross his attention. As the soft palate rises during an inspiration, the back of the mirror is placed gently against it, opposite the base of the uvula. The soft palate is then gently pushed upwards and backwards, and the handle of the instrument lowered or raised until the back of the epiglottis comes into view. The patient is then told to say *Eh*, and that usually causes the vocal cords to become visible.

If the reflex excitability of the patient's pharynx is very great, so that any attempt to introduce the mirror induces retching, the application of a 5-per-cent. solution of cocaine previous to beginning the examination will be found of great assistance.

It must be remembered also that one sometimes meets with a patient whose larynx baffles all attempts at inspection, owing to the position and shape of the epiglottis.

In studying the **view obtained,** the true cords

usually first attract attention owing to their gleaming white colour. They should move freely on phonation. Tracing them forwards, they are seen to converge and disappear behind the cushion of the epiglottis. Posteriorly they diverge and terminate in knob-like prominences, which are the apices of the arytenoid cartilages. Immediately external to each of these is a smaller knob—the cartilage of Wrisberg. Passing backwards from each side of the epiglottis to the arytenoid cartilages are seen the ary-epiglottic folds. In favourable circumstances the observer can see down the trachea, and even as far as its bifurcation. This is facilitated by the observer placing himself at a lower level than the patient, and holding the mirror in such a way that is surface looks almost directly downwards.

In observing any abnormal condition of the larynx, the chief points to be attended to are—

1. The colour of the cords and mucous membrane.
2. The presence of any swelling or ulceration.
3. The mobility of the vocal cords.

1. The normal colour of the vocal cords is a pearly white. In laryngitis they become red. Any increase or diminution in the redness of the laryngeal mucous membrane should be noted. In tuberculous affections the mucous membrane is abnormally pale; in syphilitic conditions it is unusually red.

2. The position and character of the swelling should be noted. Tumefaction of the ary-epiglottic folds and of the epiglottis should be looked for in suspected laryngeal tuberculosis. Tumours of various sorts on the true cords are occasionally met with. As regards ulcers, note their number, their position, and the character of their floor. Tuberculous ulcers are usually multiple, and are met with very frequently on

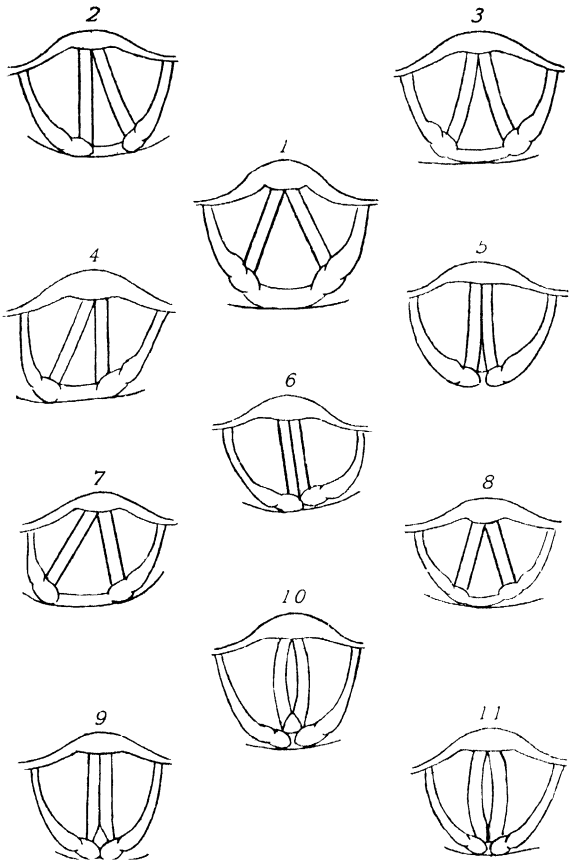


Fig. 105.—Diagram of larynx.

1, Normal larynx, respiration. PARALYSES: 2, adductor, left, phonation, 3, adductor, right and left, phonation; 4, abductor, left, respiration; 5, abductor, right and left, respiration; 6, recurrent, left, phonation; 7, recurrent, left, respiration; 8, recurrent, right and left, respiration and phonation, 9, arytenoid, phonation. 10, arytenoid and thyro-arytenoids, right and left, phonation, 11, thyro-arytenoids, right and left, phonation. All drawn as seen in the mirror, i e. patient's left hand to observer's right.

the interarytenoid fold. Syphilitic ulcers are usually single, and have a yellow, sloughy floor.

3. Observe whether the cords come together normally on phonation and open widely during inspiration. In **adductor paralysis** the affected cord fails to move inwards on phonation, or the cord makes a sudden movement inwards and then goes back, the position being unsustained (Fig. 105—*2*, *3*). In **abductor paralyis** the cord looks normal on phonation, but fails to move outwards again on inspiration (Fig. 105—*4*, *5*).

In paralysis of both abductors and adductors (paralysis of the whole recurrent laryngeal nerve, or **recurrent paralysis**) the cord is fixed in the cadaveric position—i.e. midway between complete adduction and abduction. This is much more common on the left than on the right side, owing to the greater liability of the left recurrent laryngeal nerve to be pressed upon by aneurysms (Fig. 105—*6*, *7*, *8*).

Adductor paralysis is usually the result of functional disease. Abductor paralysis, on the other hand, is the form of paralysis characteristic of an organic lesion of the nervous system. Bilateral adductor paralysis or paresis is the cause of the condition known as hysterical aphonia.

If on phonation the cords come together incompletely, leaving an elliptical space between them, there is **paralysis of the internal thyro-arytenoids** present (Fig. 105—*11*). If the anterior two-thirds of the cords come together, but leave a triangular cleft behind, the **interarytenoid muscle** is affected (Fig. 105—*9*). For further details regarding these forms of paralysis special works must be consulted.

IV. THE NOSE

Anterior rhinoscopy.—The position of the patient and of the observer, and the arrangement of

the light and reflector, are the same as for laryngoscopy (p. 499).

The anterior nares should first be inspected without the aid of a speculum. Tilt the tip of the nose upwards with the finger, and note the presence of any eczematous or ulcerated condition of the mucous membrane or skin. Observe whether any dried secretion or blood can be seen. Look for any swelling, ulceration, or perforation of the cartilaginous part of the septum.

Having noted these points, take a solid two-bladed speculum, warm, and introduce it. Hold it in position with the left hand, and gently screw the blades apart with the right. The first object to be observed is usually the anterior end of the inferior turbinated body. Note whether it is larger than normal, or not. If it is enlarged, touch it with a probe, so as to ascertain whether the enlargement is osseous or merely due to swelling of the mucous membrane. Then depress the patient's chin somewhat, so as to bring the inferior meatus into view, and ask him to hold his head a little back, so as to obtain a view of the middle meatus and middle turbinated body. The latter is considerably lighter in colour than the inferior turbinated. The superior meatus can never be seen, and the superior turbinated only very rarely.

The presence of polypi should be specially looked for in these parts. Their recognition is facilitated by the use of the probe. Lastly, turn the patient's head a little, so that the septum can be inspected. Note any deviation of it, or the presence of any prominence or spine, or the existence of any ulceration or perforation.

Swelling of the inferior turbinated body sometimes obstructs the view of the rest of the nasal cavity. The application of a little 10-per-cent. cocaine on a pledget of wool will usually cause the swelling to disappear.

Posterior rhinoscopy.—This is the only method of obtaining a view of the posterior nares. In carrying it out, the position of the observer, the patient, the reflector, and the light should be the same as for laryngoscopy. The patient, however, should have the chin rather depressed, the neck not being craned out as in the examination of the larynx.

Select the smallest laryngeal mirror, warm it, and

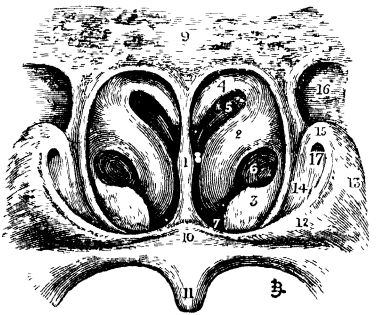


Fig. 106.—Posterior nares and surrounding parts.

1, Septum, 2, middle turbinated bone, 3, inferior turbinated bone; 4, superior turbinated bone; 5, superior meatus, 6, middle meatus, 7, inferior meatus, 8, main passage of nostrils, 9, vault of pharynx, 10, cushion of soft palate, 11, posterior surface of uvula, 12, ridge formed by levator palati, 13, salpingo-pharyngeal fold, 14, salpingo-palatine fold, 15, Eustachian cushion, 16, fossa of Rosenmuller, 17, Eustachian orifice.

ask the patient to open his mouth. It is sometimes an advantage to have the shaft of the mirror bent back a little, about 1½ in. above the reflecting surface. Introduce a right-angled tongue-depressor, and hold down the tongue with the left hand. Take the mirror in the right hand, and pass it in with the surface looking upwards. Introduce it behind the soft palate, passing it along between the uvula and the left anterior pillar of the fauces. Now turn the mirror a little, so that its surface looks upwards and forwards. The posterior nares will then come into view. What one

usually sees first is the posterior end of the nasal septum. It looks narrow, sharp, and pale in colour below, but expands a little and looks reddish above; a slight cushion-like swelling can also be often seen about the middle of it on each side (Fig. 106).

On the outer wall the posterior end of the middle turbinated bone can usually be easily seen as a large bluish-red swelling. Above it, one can see the superior meatus and the end of the superior turbinated bone; below it are the middle meatus and the upper part of the inferior turbinate. Observe the presence of any increase in size of any of these objects. Note also the general character and colour of the mucous membrane, or whether any mucus or pus can be seen adhering to it. Next turn the mirror a little upwards and to one side, keeping it rather low down and with its back against the tonsil, and look for the cushion of the opposite Eustachian tube, which can usually be made out. It forms a bright-red rounded projection, bounding a depression which leads to the orifice of the Eustachian tube. Observe whether there is any secretion at the mouth of the latter, or any adenoid swelling of the mucous membrane.

Lastly, turn the surface of the mirror upwards, and examine the vault of the naso-pharynx, noting especially the presence of any adenoid swelling or tumour in that region. Normally the roof should present an appearance not unlike that of the surface of the tonsil (Luschka's, or the pharyngeal tonsil).

Posterior rhinoscopy is often very difficult to perform. The difficulty may be due to there being very little room between the posterior wall of the pharynx and the soft palate. If this cannot be overcome by inducing the patient to breathe quietly through the nose, or to sniff, it may be necessary to introduce a palate retractor; but for a description of this process

special works must be consulted. Even after the mirror is properly introduced, it is not always easy to recognize what one sees. This is largely due to the fact that only a small portion is seen at one time, and the mirror has to be turned about till every part has been viewed separately. Experience alone can overcome these difficulties.

CHAPTER XI

LOCOMOTORY SYSTEM (BONES, JOINTS, GAIT)

THE locomotory system includes the muscles, bones, and joints. The examination of the muscles is most conveniently considered along with that of the nervous system (Chap. IX.). There remain for consideration the bones and joints.

BONES AND JOINTS

In examining the long bones of the limbs, look (1) at the condition of the shaft, (2) at the articular ends.

As regards the **shaft,** note any distension or bending of the bone and any signs of a former fracture. Pass the hand along the bone, noting the presence of any tenderness or thickening of it. Thickening is most likely to be detected on the exposed surfaces of bones, e.g. over the anterior surface of the tibia and at the lower ends of the radius and ulna. Such thickening often affords valuable evidence of old periostitis, especially of the syphilitic form.

As regards the **ends of the bones,** note the presence of any general enlargement, such as occurs in rickets, or of any nodulation at the margins, such as one finds in rheumatoid arthritis.

In examining a **joint,** note first the points which can be made out by simple inspection. Observe the position in which the patient keeps the joint; note any alterations in its contour, such as local or general swelling, and the presence or absence of any redness. Then pass to palpation, noting whether or not there is any increase of local heat in the joint, whether it is tender to the touch, and whether one can make out

the presence of any fluctuation in the joint cavity. Then try to move the joint, observing the degree of mobility in each direction, and whether or not attempts at movement produce pain. If the joint is movable, note whether any sensation of grating is produced on movement. If the mobility is limited in one or every direction, try to form a conclusion as to the cause of the limitation, and especially whether it is due to changes in the components of the joint itself. e.g. contraction of ligaments, or fibrous or bony ankylosis, or whether it is due to changes in the structures surrounding the joint, e.g. shortening of tendons. Next turn your attention to the synovial membrane. Try to make out whether there is any thickening or bogginess of it. Lastly, examine the articular surfaces of the bones, moving the joint (if possible) so that the edges of the articular surfaces can be examined with the fingers. Note the existence of any irregularity of thickening of these, and the presence of any outgrowth or " lipping " of them.

The **vertebral column and skull** demand special attention. Observe in the former the presence of any local projection of the vertebral spines. If such there be, state which are the vertebræ involved, and at which the projection is most prominent. In counting the vertebræ for this or any other purpose, one can take as landmarks either the spine of the vertebra prominens or the last rib, tracing the latter back to the 12th thoracic vertebra.

In many cases, however, the last rib cannot be distinctly felt. It is therefore rather untrustworthy as a guide.

Note also the presence of any curvature of the spinal column as a whole, or of one part of it, distinguishing carefully such general curvature from the local projections above referred to.

The curvature may be in an anterior or a posterior direction, or laterally. Anterior curvature is termed *lordosis*, and is commonest in the lumbar region. General posterior curvature is spoken of as *kyphosis*. It occurs most typically in the thoracic region in old persons, and must be distinguished from the localized angular curvature of spinal caries. Lateral curvature is termed *scoliosis*, and may be towards either the right or the left side. It is always accompanied by a rotation of the bodies of the vertebræ in such a way that the spines come to point towards the concavity of the curve. An absence of the normal curves of the spine may be an indication of commencing vertebral disease.

Ask the patient to stoop down, and notice the degree of mobility of the vertebral column, and the occurrence of any pain during stooping, noting the exact site of the latter if present. Then pass the hand down the vertebral column, and observe whether any tender spots can be made out. Such tender spots are not infrequently met with in hysteria and in cases of irritation of the posterior nerve-roots. Their presence can often be more easily elicited by drawing a sponge wrung out of hot water down the vertebral column: the patient complains of pain as soon as the hyperæsthetic area is reached. To elicit more deep-seated tenderness of the vertebræ, it may be necessary to "punch" the spines gently with the fist from above downwards, observing the point at which the patient complains of pain, and verifying the observation by repeating the process from below upwards.

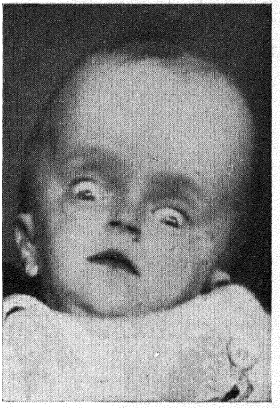
In studying the **skull**, note first its **size.** For this purpose it may be necessary to take **measurements.** This should be done in three directions: (1) antero-posteriorly from the root of the nose to the external occipital protuberance; (2) circumferentially

at the level of a line drawn horizontally round the skull from the supraorbital ridges in front to the external occipital protuberance behind; (3) coronally from one auditory meatus to the other. If the skull is abnormally small, the patient is microcephalic. This is frequent in some forms of idiocy. Abnormal enlargements of the skull occur in hydrocephalus, in osteitis deformans, and in acromegaly.

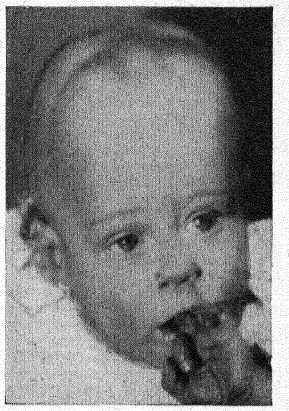
The average normal circumference of the skull at different ages is as follows:—

Birth	13 inches.	Seven years ..	20 inches.
Six months ..	16 ,,	Fifteen ,, ..	21 ,,
One year .	18 ,,	Adult .	22 ,,
Three years ..	19 ,,		

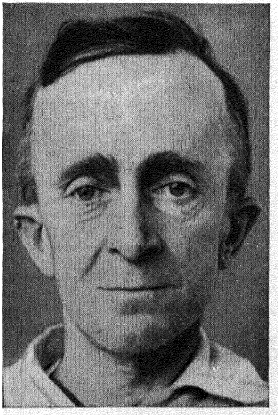
Next observe the **shape** of the skull. Is it of the dolichocephalic (long-headed) or the brachycephalic (bullet-headed) type? Are the two sides of the head symmetrical? Certain well-recognized types of abnormal skull are met with. In *hydrocephalus* the skull tends to assume a globular form. The forehead is overhanging and the eyes are pushed down so that the upper part of the sclerotic is exposed. The lateral aspects of the skull (above the ears) project outwards. If the patient is a child, as is usually the case, the fontanelle is wide and bulging, and often fluctuates very distinctly. The sutures may be opened up, and imperfectly ossified areas (*craniotabes*) may be detected in the bones. In *rickets* the skull tends to be square or oblong and box-shaped. The frontal and parietal bones often show central thickening ("bossing"). The forehead, however, does not overhang, nor are the eyes depressed, and although the fontanelle is usually widely open, it does not bulge as it does in hydrocephalus, nor are the sutures opened up. In *congenital syphilis* the forehead is vertical, the frontal



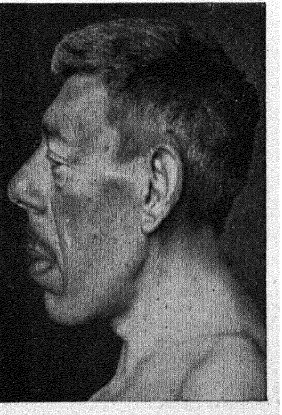
Hydrocephalus



Rickets



Osteitis Deformans



Acromegaly

Plate 27.—SKULL.



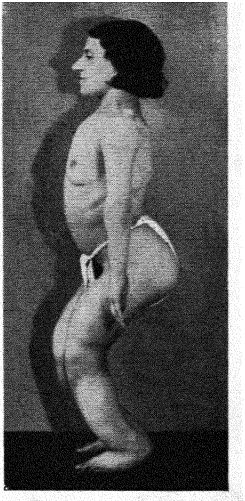
Spastic Diplegia



Parkinsonism



Ankylosing Spondylitis



Rickets

Plate 28.—ATTITUDE.

eminences are often exaggerated, and the bridge of the nose is depressed.

Having noted the general shape of the skull, ask the patient to open his mouth, so that one may see the **hard palate.** Observe its width and degree of arching. A high, much-arched, and narrow palate is often one of the minor signs of mental deficiency.

Next proceed to the **palpation of the skull.** Note first the thickness of the scalp, and whether it moves freely, as it ought, upon the subjacent bone. Atrophy and adherence of the scalp are apt to be associated with skin diseases in this region, and are often a bar to successful treatment. Observe the presence or absence of inequalities in the bones, such as may indicate the site of former injury or fracture. If a swelling is detected, pay special attention to its margins, noting whether a hard rim can be made out, and whether or not the rim disappears on firm pressure steadily applied by the finger for a minute or two. In blood extravasations the rim disappears, in a depressed fracture it persists. Note also whether the swelling can be moved as a whole upon the skull, or not. If the patient is a child, note the condition of the fontanelles and sutures, and look for the presence of unossified areas in the bones (*craniotabes*). The best place to look for these is in the neighbourhood of the lambdoidal suture. They feel like little spots which are covered only by parchment. Observe the presence of any tender spots or areas on the skull. For this purpose it may be necessary to tap the skull gently all over with the forefinger. If tenderness is detected, note carefully its maximum point. Such tender points are sometimes met with in inflammatory affections of the cranial bones or membranes, and in cases of superficially situated intracranial tumours, but they may also be present in neuralgic affections of the scalp.

THE GAIT

The character of a patient's gait is often an important indication of the nature of the affection from which he is suffering. It is especially important in cases of nervous disease.

In studying the gait, it is well, if possible, to have the legs fully exposed. For this purpose the patient should have on only a night-shirt or dressing-gown, which should be brought through between the legs from behind, and pinned up in front. The feet should be bare. The patient is told to walk away from the observer, to turn round at a given point, and then to come towards him again.

In studying the gait, the **points to be noted** are—(1) Can the patient walk at all? This being decided, one has to ask oneself—(2) Does he pursue a straight line, or does he tend to deviate to one side or the other, or to both alternately? To bring out this point, it is well to ask him to walk along a straight line—e.g. a crack in the floor. (3) Does he tend to fall, and, if so, in what direction? These questions being settled, the next point to be decided is whether the gait conforms to any of the well-recognized abnormal types. Before one tries to make up one's mind in this matter, however, it is well to be quite sure that the peculiarity in the patient's gait is not due to some surgical cause or to local disease of a joint—e.g. osteo-arthritis of the hip. A previous examination of the bones and joints will eliminate such sources of fallacy.

The three chief types of abnormal gait due to nervous affections are—

1. The spastic.
2. The ataxic.
3. The reeling.

In taking a patient's case, it is usually sufficient to state that the gait belongs to one or other of these types, or to two or more combined. The chief peculiarities of each variety are as follows:—

1. The **spastic** may be described as a "sticky" gait. The patient has difficulty in bending his knees, and drags his feet along as if they were glued to the floor, the toes scraping the ground at each step. The foot is raised from the ground by tilting the pelvis, and the leg is then swung forwards so that the foot tends to describe an arc.

This gait is seen most characteristically in patients with lateral sclerosis of the cord. The **hemiplegic gait** is essentially a spastic gait in which only one leg is affected.

2. The **ataxic** may be described as a "stamping" gait. The patient raises his feet very suddenly, often abnormally high, and then jerks them forward, bringing them to the ground again with a stamp, and often heel first. He seems to exhibit, also, an indefiniteness of purpose in the place chosen to put down the foot; and the feet while in the air do not move in one plane, but are waved about, as it were, before being set down. By adopting a "broad base" the patient tries to counteract the unsteadying effects of his style of progression. This gait is best seen in cases of locomotor ataxy.

3. The **reeling** gait may be described as a "drunken" gait, and, therefore, requires no further description. It will be observed that patients with this gait walk "on a broad base," the feet being planted widely apart. It is important to notice whether supporting the patient by his axillæ abolishes the reeling tendency. In some cases of cerebellar disease, such support has been observed to abolish the patient's vertigo for the time during which he is supported.

This gait occurs most typically in cases of cerebellar lesion. It is, therefore, sometimes referred to as a "cerebellar gait."

Some rarer varieties of abnormal gait may be briefly referred to. These are—

The **festinant** gait. This is the form of gait met with in typical cases of paralysis agitans. The patient is bent forwards, and advances with rapid short shuffling steps, so that, as has been said, "he looks as if he were trying to catch his centre of gravity." In some cases, if one suddenly pulls the patient backwards, he begins to walk backwards, and is unable to stop himself, though he is leaning forwards all the time. This peculiar phenomenon is spoken of as *retropulsion.*

The **waddling** or oscillating gait is like the gait of a duck. The body is usually tilted backwards, there being a degree of lordosis present; the feet are planted rather widely apart; and the body sways more or less from side to side as each step is taken. The heels and the toes tend to be brought down simultaneously. The chief peculiarities of this gait are due to a difficulty in maintaining the centre of gravity of the body owing to weakness of the muscles of the back. It is met with in pseudo-hypertrophic paralysis and in congenital dislocation of the hip.

The **high-stepping** or prancing gait is a device adopted by the patient to avoid tripping from his toes catching the ground. It is therefore, met with in cases where the toes tend to droop from weakness of the extensor muscles, e.g. in peripheral neuritis affecting the anterior tibial nerve. The name sufficiently describes its characters.

CHAPTER XII

CLINICAL EXAMINATION OF CHILDREN

THE clinical examination of young children is a matter full of difficulty to the inexperienced, for it has to be carried out, not merely without the help of the patient, but often in spite of his strenuous opposition. In this chapter we propose to describe the best methods of ascertaining the necessary facts, and also the chief points in which the child differs from the adult in a clinical sense.

The history of the patient and his illness must, of course, in the case of young children, be ascertained from the mother or friends. The best **scheme of interrogation** to employ will be found on p. 12. Whilst the history is being elicited, opportunity may be taken to cultivate the friendship of the child or, at all events, to get him accustomed to one's presence. The history having been ascertained, one proceeds to an examination of the child. This requires gentleness and deliberation, combined with infinite patience and good temper. If one is at all hurried or rough, the child begins to cry at once, and the subsequent examination is rendered a thousandfold more difficult. We would emphasize the fact that it is often not possible to be really systematic in one's examination of children. Certain things must always be looked for, but no definite order can be observed in looking for them. One has to seize the opportunity of ascertaining a fact as it presents itself, and a rigorous adherence to " systems " is often out of the question. In the first place, a number of points can be ascertained before

the child is undressed. One can study the **facies** of the child, note its complexion, the colour of its lips, and whether or not the alæ nasi are acting. One should also at this period of the examination count the respiration- and pulse-rate; it is very important to get these noted while the child is still undisturbed.

The **respirations** can usually be counted by merely watching the movements of the child's abdomen, that being very much more affected by respiration in young children than is the chest. The normal rate of a new-born child is 40 or so respirations per minute, by the second year they have fallen to 30 or so, at the fifth year they are about 25, and by the fifteenth they have sunk to 20. Much more important than the absolute number of respirations is the ratio of respiration to pulse. Normally this should be as 1 : $3\frac{1}{2}$ or 4.

The **pulse** is best counted by allowing the mother to hold the child's hand in hers; the fingers of the physician are then quietly slipped over the mother's hand on to the child's wrist, and the pulse counted. If the child has begun to cry, it is useless to take the pulse-rate, as it may be at least 20 beats above the normal rate. The pulse-rate at birth should be 130, by the second year it is 110, by the fifth 100, by the eighth 90, and by the twelfth 80; after this it gradually sinks to the normal adult rate. During sleep the pulse-rate always falls about 10-20 beats. As a matter of fact, the examination of the pulse in infancy is of comparatively little clinical value. It is of little use as an index of the vital powers, the fontanelle replacing it in that respect. The vessel being extremely small, the characters of the pulse-wave can hardly be ascertained; irregularity by itself is of comparatively little significance, being very common even

in healthy infants, and being almost the rule in sleep. A pulse that is continuously *slow* and irregular is, however, of great significance.

These preliminary facts having been noted, the child should be stripped and placed in a blanket on the knee of the mother or nurse; examination must then be proceeded with by the usual methods of inspection, palpation, auscultation, and percussion. In the clinical investigation of children it must be noted that the two former methods are of much the greatest assistance.

It is well to begin by looking and feeling the child all over. One notes the general state of development and nutrition; the state of the skin, whether dry and fevered, or moist, and the presence or absence of any rash or skin eruptions, and whether or not the normal degree of elasticity is present. The shape of the chest and the degree of prominence of the abdomen should be noted, it being borne in mind that the rickety and pigeon-breasted types of chest are very common in diseased children, and that a rather protuberant abdomen is to be regarded as normal. The hand should then be passed lightly over the head. The state of the **anterior fontanelle** should first be investigated. The fontanelle closes normally between the fifteenth month and the second year. If it remains patent after the second year, it is often a sign of disease—usually of rickets. Too early closure of the fontanelle occurs in some forms of microcephaly and idiocy.

The degree of tension of the fontanelle is of great importance. In health it pulsates distinctly, and is neither sunken nor unduly elevated. A depressed fontanelle is an important sign of exhaustion and of dehydration; a tense fontanelle indicates increased intracranial pressure. It must be borne in mind, of

course, that the fontanelle is normally tense when the child is crying. The systolic bruit heard over the fontanelle is of no clinical importance.

The **shape of the head** and of its bones must be investigated. The development of "**bosses**" on the frontal and parietal bones is a common occurrence in rickets. One should also look for evidence of craniotabes (in young babies) and of rheumatic nodules (in older children). The general shape of the head as a whole should always be noted; it may be box-shaped as in rickets, or globular as in hydrocephalus; it may be abnormally small or large, or it may be asymmetrical.

Passing from the head, one may examine the limbs. In children this is of extreme importance; many of the commonest and most serious diseases of infancy affect the long bones more prominently than any other part of the body. Look for thickening or tenderness along the shafts of the bones. This may be due to scurvy, to syphilitic or to suppurative periostitis, or to tumours. Examine carefully the **epiphyses.** In rickets these become enlarged. This is most easily seen where the ribs join their cartilages, the thickening there forming a row of bead-like prominences ("*rickety rosary*"). It is also easily seen at the wrists. The frequency of inflammatory affections of the epiphyses should be borne in mind. The presence or absence of "*rheumatic nodules*" should also be noted. These are little fibrous bodies varying in size from that of a large pin's head to a pea, or even bigger. They occur not in the periosteum, but in the deep fascia where it covers superficial bones, and also in the sheaths of tendons. They should be looked for especially over the olecranon, patella and occiput. They are usually movable, but not tender. If found, they are pathognomonic of rheumatism. The **vertebral**

column should always be examined for signs of tuberculous disease or curvature.

At this point, if not earlier, the child's **temperature** should be taken. In young children the thermometer should be inserted into the rectum, or placed on the groin or axilla; in older children it may be placed in the mouth. It should be remembered that the temperature in children is much more variable than in adults, and that it often rises on very little provocation. The rectal temperature, which is often taken, is moreover somewhat higher normally than the mouth or axillary temperature.

One must now proceed to the examination of the **thorax and abdomen.** The front of the chest and abdomen may be examined together, and either after or before the posterior aspect of the chest. The order adopted should be, first, inspection and palpation, then auscultation, and, last of all, percussion. Percussion is left to the last owing to the fact that it frequently makes the child cry.

In **palpation,** be sure that the hand is quite warm; this is even more important in examining a child than in the case of an adult. It is also important to watch the child's face while this examination is being carried out, as wincing will provide evidence of tenderness and pain. In **auscultation** it is important to warm the chest-piece of the stethoscope, if it is made of metal, before applying it to the chest. There is only one point to be observed in the **percussion** of a child, and that is, that the stroke should be *light.* This is not merely in order to avoid frightening the little patient, but also to escape the confusion that is apt to arise from the excessive resonance of the child's chest.

When the abdomen and front of the chest have been gone over in this way, one should turn one's

attention to the posterior aspect of the lungs. For the examination of these, the child should not be laid on his face, as that interferes with respiration and causes the abdominal viscera to push up the diaphragm, but he should be held against the mother's breast with his head looking over her shoulder. In this way the whole of the back of the chest can be gone over. The presence of enlarged glands should be sought for, particular attention being paid to the neck, which should be palpated from behind.

Last, but by no means least, comes the examination of the **mouth and throat.** It is impossible to exaggerate the importance of systematically examining the mouth and throat in all cases of illness in children. At the same time, it is just this part of the clinical examination in which we are most likely to meet with opposition; and for that reason it is left to the last, as it may be necessary to employ coercion in order to get it carried out.

Begin by looking at the **tongue**. Sometimes the child will put out the tongue when asked. In little babies gentle pressure on the chin will often cause the mouth to be opened, when a view of the tongue can be obtained. Or, if a drop of milk or a little sugar is placed just outside the lip, the child will often put out its tongue in order to lick it off. In more refractory children it may be necessary to push the lower lip over the teeth, and then to press the lip down against the lower incisors. The child then opens the mouth in order to avoid biting the lip. With very obstinate children one may be obliged to compress the nostrils until the mouth is opened to get breath.

Once the child has been induced, either voluntarily or by aid of one of the above devices, to open the mouth, one should note the state of the **buccal mucous membrane,** remembering the frequency of thrush,

stomatitis, and ulcerations in children. In cases of suspected measles, *Koplik's spots* should be carefully looked for. They consist of irregularly stellate or round rose-red spots, with a bluish-white speck in the centre of each. They are to be found on the inside of the lips and on the buccal mucous membrane, especially opposite the upper molars. At first they are very sparse, but later on become more numerous, and the red parts may then coalesce into large areas dotted with the bluish-white specks. They should always be looked for in strong sunlight if possible, and never by artificial light. They are of considerable diagnostic importance, for they may precede the appearance of the skin eruption by three or four days. The number and character of the teeth should be observed (*see also* p. 46), and the finger should be run along the gum to feel for any teeth that may be about to come through.

One must then proceed to an examination of the **throat**. The child should be wrapped in a towel to restrain the movements of its arms. The mother or nurse sits down opposite a good light and takes the child on her lap. Another assistant steadies the head from behind. The child having then been induced or compelled to open its mouth, one introduces a small-sized tongue-depressor and holds down the tongue, thus exposing the pharynx. The finger will often serve instead of an instrument, and has the advantage of frightening the child less. Look for any enlargement of the tonsils, for any redness of the mucous membrane, and especially for the presence on it of any membranous patches.

Palpation of the pharynx must also be carried out in some cases. To do this one must stand behind the child and, when the mouth is open, push in the cheek from one side between the molar teeth. This serves as a gag, and effectually prevents the child from

attempting to bite. The forefinger is then passed to the back of the pharynx and up behind the soft palate. Note the presence of any adenoids, or any bulging into the posterior wall of the pharynx, which may be the indication of the presence of a retropharyngeal abscess. This particular examination causes extreme discomfort and should always be avoided unless absolutely necessary.

The following is the general routine method to be employed in examining a child:—

The following summary of the chief facts to be noted in the general inspection and palpation of a child may be of service:—

Facies and expression—general appearance (if healthy or otherwise)—nutrition, muscle tone, dentition and development—complexion (anæmia, cyanosis, jaundice, etc.)—state of skin (dryness, moisture, eruptions, desquamation, pigmentation, œdema)—posture, demeanour, temper—pain on being handled.

Shape of head and state of its ossification (fontanelle, cranio-tabes)—facial irritability—hair—eyes, nose, and ears (formation of, and if any discharge from)—shape of thorax, abdomen, back, and limbs (especially the hands)—enlarged glands—evidence of rickets, syphilis, and tuberculosis.

Character of voice, cry, and cough—rate and character of respiration; if noisy, dyspnœic, or painful—movements of alæ nasi—rate and character of pulse—temperature.

Palpation of abdomen (tenderness, resistance, fluid, size of liver and spleen, tumours, etc.).

Certain special points which remain will be considered briefly under the different systems:—

1. **General condition.**—Special importance attaches to the regular weighing of children. Alterations in **weight** from time to time are of much help in prognosis and treatment. It should be remembered that a healthy child should weigh at birth about 7 lb. This should be doubled during the sixth month and trebled in the first year. By the sixth year it is again doubled, so that a healthy child of six should weigh about 3 stones. This is again doubled when the fourteenth year is reached. (Fig. 107.)

Measurement of the head is often of importance. Two measurements are usually sufficient—a coronal measurement from one auditory meatus to the other, and a circumferential measurement at the level of the root of the nose and external occipital protuberance.

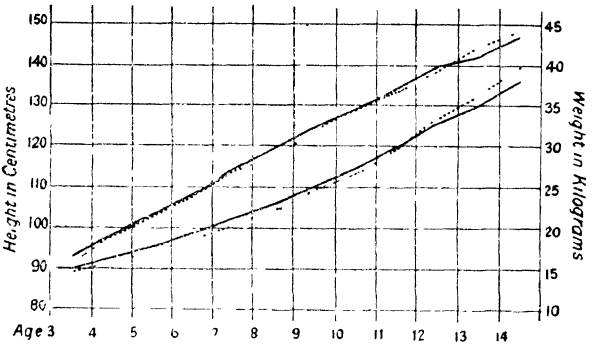


Fig. 107.—Average height and weight of English school-children.

(Tuxford and Gegg.)

The two upper lines represent heights, the two lower lines weights. The continuous lines relate to boys, the dotted lines to girls. (1 centimetre = 0·39 in.; 1 kilo = 2 lb. 3 oz.)

(By permission of the "British Medical Journal")

2. **Alimentary system.**—Note that the **liver** is normally rather large in children, and often reaches at least ½in. below the coastal margin. Enlargement of the **spleen** is frequent in infantile diseases. It is best made out by palpation, the hand being passed across the child's abdomen from right to left. By depressing the finger-tips opposite the 11th interspace the edge of the spleen, if the organ is enlarged, may be felt as it descends during inspiration. When indicated, a rectal examination must not be omitted.

Inspection of the **stools** should be carried out. The healthy infant, on the breast or bottle only, has generally two or three stools daily, though there

are wide variations. These should be of the colour and consistence of beaten-up eggs. Any alterations in frequency, colour, or consistence, or the presence of worms, should be taken into consideration.

3. **Circulatory system.**—Note that the **apex-beat** of the child is normally rather higher than in the adult. It is usually outside the mammary line up to the third year, in the mammary line from the third to the tenth year; after that age it gradually assumes the adult position. It should also be observed that alterations in the general contour of the **præcordia** are much more frequent results of cardiac disease in children than in adults. As regards **auscultation,** it should be remembered that the pulmonary second sound in a young child is normally rather louder than the aortic. The pulmonary second is accentuated if it is permanently louder than the first. The aortic second is accentuated if it is as loud as the pulmonary.

Remember also that hæmic bruits are rare in babies, while congenital bruits are relatively frequent. In this connexion it is important to look out for cyanosis and the presence of clubbing of fingers or toes. We have already mentioned that the cardiac rhythm in the child is not infrequently irregular even in health.

4. **The blood.**—It is sometimes difficult to get a large enough drop of blood from the ear of a child. In that case a piece of woollen thread should be twisted round the great toe—not too tightly—and the latter punctured with a triangular needle at the root of the nail. The number of red cells is usually (after the second week of life) slightly lower than in the adult. The leucocytes are more numerous in the child than in the adult; 12,000 per c.mm. is about the average number throughout infancy. The *lymphocytes* and granular *leucocytes* are both relatively and absolutely

more abundant than in the adult, amounting to about 45 per cent. of the total leucocytes in the first three years. In new-born babies the percentage of hæmoglobin is often very high, but throughout the rest of infancy it is lower than in the adult.

5. **Respiratory system.**—A child uses the diaphragm much more than the intercostals in breathing. Hence the movements are chiefly abdominal, and there is little real chest expansion. Indrawing of the lower interspaces on inspiration should always be looked for. It occurs whenever there is obstruction to the entrance of air (e.g. diphtheria), but may also be present when there is collapse of the lower parts of the lungs, and also in pneumonia. In "extra-auscultation" one should be on the look-out for any stridor, and for the existence of a short, grunting expiration and working of the alæ nasi. The latter is a frequent sign of severe respiratory disease. In the adult the normal cycle of respiration is, of course, inspiration, expiration, pause. In the child this is often reversed, so that one gets first a short expiration, succeeded by a longer inspiration, and then by a pause. This reversal is specially frequent in respiratory disease or embarrassment. The respiratory pauses are often very prolonged in the child, so that one has to wait a long time if one is auscultating before the next inspiration is heard. The normal breath-sound in the child is, after the age of six months or so, **puerile** in type. **Vocal resonance** is often difficult to estimate and hence is an unreliable sign in childhood. In babies one may make use of the cry as a producer of vocal resonance; in older children one may ask them their name, get them to count, etc. It should be remembered as a general rule that if the breath-sounds are distinctly harsher on one side than the other, then the harsh side is probably

the normal. Children's chests conduct sound very readily. Hence abnormal sounds, especially crepitations, are very apt to be heard on both sides, although they are really only being produced on one. The great frequency of collapse of part of the lung should be borne in mind in diagnosing pulmonary disease in infancy. In percussing the lungs in children one must, as already mentioned, *use a very light stroke.* One should also take care only to percuss when the chest has been filled by an inspiration, otherwise one may be led into thinking that there is dullness present.

It should also be remembered that the chest-wall of a young child is so elastic that one can often obtain the "cracked-pot" sound on heavy percussion, even although the lung be perfectly healthy. This is especially apt to occur if the child is crying.

6. **Urinary system.**—It is difficult to collect the total quantity of urine passed by a child per diem. A rough table of the average quantity for each child will be found at pp. 285-6. Sugar is only rarely present in the urine of children, but protein is often met with, even in healthy babies.

7. **Nervous system.**—**Motor paralysis** is to be made out by watching whether the child ever moves the suspected limb. One cannot estimate the paralysis as one does in adults, by means of passive resistance. Remember that inability to walk is not necessarily a sign of paralysis of the legs. One must note whether the legs are moved when the child is sitting or lying. Thus, a rickety child may not be able to walk, but moves his legs freely if one tickles the soles. A child with infantile paralysis of the legs cannot move the limbs in any circumstances.

The **knee-jerks** in little children are best elicited by placing the child's foot on one's hand as a stirrup, and then gently percussing the tendon. The latter

lies rather to the outer side in the child, and is comparatively narrow, so that one may easily miss it.

The **superficial reflexes** are usually more brisk in healthy children than in adults. The exact localization of **sensory paralysis** is extremely difficult in children, but sensory lesions occur only rarely in infancy.

In examining the **eyes** with the ophthalmoscope, the direct method is the best to employ. An electric ophthalmoscope should always be used for choice. It may be necessary to hold open the lids; but as far as possible avoid touching the child at all. One must often be satisfied with mere fleeting glimpses of the disc.

In testing light-perception in little children, it is best to hold a light in front of the eyes, and see if they attempt to follow its movements. One may also threaten the cornea by suddenly bringing the finger near it, and observing whether the child winks before the eye is touched.

In examining the **ears,** one must remember the shortness of the auditory meatus in the child, and the great obliquity of the drum membrane. The magnification of the view by means of an ophthalmoscopic lens is a useful aid in these cases.

It is often difficult to gauge the **intellectual capacity** of a young child. Early signs of mental deficiency are—inability to support the head, which often rolls about helplessly; causeless screaming; inability to notice things; and backwardness in grasping.

In older children we can inquire as to progress at school, etc., or ask the patient questions; get him to count, multiply, and so on. The position of the child in the school is also a rough guide to the development of the intelligence.

Most normal infants start to walk between

twelve and fifteen months of age. They begin to say words about this time, although the speaking of sentences comes rather later and the age at which a child actually talks is very variable. The distinction drawn by West between children that are defective and those that are merely backward may also be of help in the investigation. A backward child would be normal for a younger age; a defective one would be abnormal at any age.

CHAPTER XIII

EXAMINATION OF PATHOLOGICAL FLUIDS

In this chapter we propose to deal with the methods of examining fluids obtained from one of the body cavities or from abnormal growths, for diagnostic purposes.

The fluid is obtained by means of **exploration.** An ordinary hypodermic needle may be employed, but special exploring needles—which are really merely large and strong hypodermics—are also sold. The needle should be of such calibre as to be capable of sucking up oil. If it can do that, it will be able to suck up any fluid likely to be met with in exploring. Before being used, the needle should be sterilized by boiling for three minutes, and the patient's skin should be cleansed with some 1-in-20 carbolic acid, or with iodine solution, at the spot selected for puncture. As a rule, it is not necessary to employ any local anæsthetic. In very nervous patients a small spot of skin may be frozen by means of the ethyl-chloride spray. It should be remembered, however, that the local reaction after freezing often causes more pain than the original puncture. The needle should be held short, with the forefinger of the operator resting on it near the point. It should be introduced rapidly and steadily, but without any "stab." When the needle has been fully entered, the piston is withdrawn. Should no fluid be obtained, the needle is drawn slowly outwards, whilst a negative pressure is maintained in the syringe. It may then be found that fluid is obtained nearer the surface.

Where to Puncture

In the case of the **pleural cavity** the puncture is best made in the 9th or 10th space midway between the posterior axillary and scapular lines, this being the point at which fluid that is lying free in the cavity is most likely to be obtained. In cases of localized dullness, one must be guided, of course, by circumstances. Usually, one selects that point where the dullness, as estimated by the feeling of " resistance " on percussion, is most absolute and the breath-sounds are faintest.

Puncture of the **peritoneal cavity** may be performed either in the middle line through the linea alba, or laterally, about a point on a line with, but rather above, the anterior superior spine. The former position ensures that no large blood-vessel will be injured; but by lateral puncture one is more certain of entering fluid, especially if the patient is turned over somewhat on to the side of operation. Before puncturing in the middle line, be sure the bladder is empty, and never insert a needle at any point unless it yields a dull note on moderately heavy percussion.

In puncturing the **pericardium** it is probably safest to select a spot between the apex-beat and the outer limit of the pericardial dullness. A fine needle should be used.

In the exploration of **cysts,** etc., one must be guided by local circumstances, the rule being to select for puncture that part of the tumour which is nearest the surface, and where one is not likely to injure important structures.

Examination of the Fluid

The fluid, having been obtained, should be transferred to a conical glass and allowed to settle.

Note first its **physical characters.** The chief of these are the colour, consistence, specific gravity, odour, and the appearance of the deposit (if any).

As regards the **colour** of the fluid, one of the most important points to note is whether it is blood-stained or not. It must be borne in mind, however, that a small amount of blood is apt to get into the fluid in the process of exploring. Observe also, whether the fluid is transparent, opaque, or opalescent.

Opacity is usually due to the presence of cellular elements; opalescence to fatty particles or large numbers of micro-organisms.

Opacity or opalescence due to fat may be removed by adding to the fluid some caustic potash solution, then shaking up with ether. The fat is dissolved out, and, if the ether is sprinkled on to blotting-paper, leaves a stain. Fluid which is opaque from the presence of much fat is usually spoken of as "chylous," and is derived from the thoracic duct. It may be simulated very closely by a "pseudo-chylous" fluid in which the milkiness is due to a lecithin-globulin complex which is held in suspension by the inorganic salts present. Removal of these by dialysis causes the precipitation of the lecithin-globulin body and the disappearance of the opalescence.

Pathological fluids are usually of a more or less watery **consistence.** Viscidity usually indicates the presence of mucin. It should be carefully noted whether or not the consistence of the fluid alters on standing. Many pathological fluids clot after standing for some time. The clot consists of fibrin.

The **specific gravity** is taken with a urinometer, the same precautions being used as in the case of urine (p. 291).

Most fluids are devoid of **odour** ; sometimes, however, they are extremely fetid.

The amount and colour of the **deposit** should be noted. If red, it probably consists of red blood-corpuscles; if white, it may be made up of leucocytes, cancer-cells, etc.

For **chemical investigation** the fluid should first be filtered. In the examination of the filtrate the following points must be attended to:—

1. The **reaction.** This is almost invariably alkaline. Sometimes it is neutral.

2. The presence of **serum-albumin** and **serum-globulin.** This is ascertained by means of the same tests as have already been described for the urine (p. 306). If these proteins are present in large amount, the fluid is coagulated on boiling, even although the reaction is alkaline. If proteins are scanty, the fluid should first be rendered slightly acid by means of dilute acetic acid.

As in the case of the urine, nothing is gained by testing for albumin and globulin separately. **Proteoses** and **peptone** are almost never found in the fluids under consideration.

The quantitative estimation of albumin and globulin cannot be accurately carried out in ordinary clinical work. Approximate results may be obtained by the use of Esbach's tube (p. 308). The fluid must first be very freely diluted, so as to bring the specific gravity down to 1008, and should then be rendered acid by means of acetic acid.

3. The presence of **mucin** or **nucleo-protein** is proved by the appearance of a precipitate on the addition of acetic acid insoluble in excess.

4. **Sugar** should be tested for by rendering the fluid slightly acid, boiling, and filtering. The filtrate is then evaporated down to a small bulk, and the tests for glucose described at p. 314 are applied.

5. **Urea** is not often present, except in traces, in

ordinary pathological fluids. In fluids derived from the urinary organs it may be more abundant, and should be tested for by removing all proteins by heat, evaporating the filtrate to a small bulk, and then testing for urea as described on p. 300.

Microscopical Examination of the Sediment

Some of the deposit is taken up with a pipette, and a drop of it placed on a slide, covered, and examined. For cytological examination it is best to centrifugalize some of the fluid and make films from the deposit. The films may be stained with carbol thionin or Leishman's stain.

One may recognize under the microscope (1) elements derived from the **blood**—altered red and white corpuscles. The recognition of altered white corpuscles or pus-cells is facilitated by mixing with a drop of the deposit a small quantity of a 1-per-cent. solution of acetic acid to which a little methyl green has been added. The nuclei are then more easily recognized. In acute inflammatory exudates polymorphonuclear cells predominate; in tuberculosis and syphilitic infections lymphocytes are in excess. (2) Endothelial cells.—These are derived from the lining of the cavity and are the only cells present in dropsical transudates. Cancer-cells may be present in malignant cases, but it is almost impossible to distinguish them with certainty from ordinary cells. In fluid derived from hydatid cysts, scoleces and *hooklets* may be found. (3) **Crystals** of cholesterol or of fatty acids, and fragments of muscular tissue are sometimes seen.

The **bacteriological examination** of pathological fluids is described in Chapter XIV.

Inflammatory and Dropsical Effusions

Inflammatory effusions are often spoken of as exudates, and dropsical effusions as transudates. They present the same general appearances, being clear fluids of a yellowish-green colour, and containing much albumin and globulin. It is very difficult to tell a dropsical from an inflammatory fluid by chemical examination; one must rely for that on cytology. It would appear that the amount of proteins in an effusion depends much more upon site than upon cause. Pleural fluids contain the highest percentage of protein, peritoneal fluids rather less, and subcutaneous fluids very little. The fluid in cardiac dropsy is more highly albuminous than in dropsy of renal origin. From a diagnostic point of view, all that one can say is that a fluid with a specific gravity of more than 1018, which contains more than 4 per cent. of protein, is almost certainly inflammatory; while one with a specific gravity of less than 1015, and a protein percentage of less than 2.5, is certainly dropsical. Between these limits one must be in doubt. Nor is the occurrence of coagulation in the fluid after tapping of much help. If the coagulation is very rapid and complete, the fluid is probably inflammatory, but considerable coagula may form even in purely dropsical fluids after standing for some time.

Cerebro-spinal Fluid

The fluid is obtained by **lumbar puncture,** which is performed as follows:—

Draw a line with a swab dipped in alcoholic solution of iodine vertically down the vertebral spines and another horizontally at the level of the highest

Characters of various Pathological Fluids

	Hydatids	Hydro-nephrosis	Distended Gall-Bladder	Ovarian Cysts	Parovarian Cysts	Pancreatic Cysts	Amniotic Fluid
Colour	Colourless or slightly opalescent	Colourless or yellowish	Colourless or bile-stained	Varies — brown, green, yellow, etc.	Colourless	Colourless or yellowish and turbid	Greenish-yellow
Consistence	Watery	Watery	Slightly viscid	Viscous	Watery	Watery	Watery
Specific gravity ..	1006–10	1008–20	Low	1002–50	Low	Low but variable	1006–11
Coagulable proteins ..	Very scanty	Vary — may be fairly abundant	Usually scanty	Vary	Scanty	Variable	Scanty
Special characters ..	Presence of scoleces or hooklets	*May* contain urea or uric acid	May contain bile. Mucin usually present	Presence of pseudo-mucin (gives a white precipitate with alcohol after other proteins have been removed by boiling)	—	Contains cholesterol, and (if recent) will digest egg-albumin in alkaline medium, and may convert starch	Heavy animal odour Contains some urea (at least, in later months)

points of the iliac crests. The lines intersect at the space between the 3rd and 4th lumbar spines or sometimes at the tip of the 4th lumbar. The puncture may be made through either the 3rd or 4th interspace. The patient should be lying on his side on a firm couch, with the knees and chin approximated. Local anæsthesia may be produced by the ethyl chloride spray or by the injection at the site of puncture of 4-per-cent. sterile novocain, first raising a bleb under the skin, and, when this is insensitive, thrusting the needle in towards the centre of the intervertebral space, injecting the solution as one does so. A special platinum-iridium or nickel needle about 8 cm. in length should be employed (steel is too brittle); it should be of fine calibre and provided with a bevelled end and a stylet. It may be mounted for convenience (but not for suction) on an all-glass syringe of 10 c.c. capacity. It is sterilized by boiling in distilled water.

Push the needle firmly through the skin in the middle line or just to one side of it and press it forwards and slightly upwards, the bevel pointing towards the side on which the patient is lying. When the needle is felt to enter the spinal cavity the stylet is withdrawn and the fluid which escapes collected in strong sterilized test-tubes stoppered with glass or rubber. The puncture is sealed with collodion, and the patient should rest for 24 hours after the operation.

It is an advantage to have a manometer connected with the needle so that the pressure of the fluid can be measured at the time of puncture. If this is done the patient's head must be on the same level as the sacrum and he must be breathing quietly and with his muscles relaxed. The normal pressure is from 100 to 200 mm. of fluid.

EXAMINATION OF THE FLUID

1. **Physical characters.**—Normal cerebro-spinal fluid is clear and colourless like distilled water, with a specific gravity of 1006. Any yellowness of tint is pathological and indicates either old hæmorrhage or excess of protein In *Froin's syndrome* a pronounced yellow colour (xanthochromia) is associated with great excess of protein and massive coagulation of the fluid. Formation of a clot in a colourless fluid indicates meningitis or high protein from other cause, e.g. tumour or polyneuritis. In tuberculous meningitis, and sometimes in poliomyelitis, the clot resembles a cobweb.

Turbidity of the fluid may be due to pus or to red blood-corpuscles. If it does not clear on standing it is due to micro-organisms.

The presence of *blood* may be due to injury to a vessel by the needle or to subarachnoid hæmorrhage. In the latter case the blood is more uniformly mixed with the fluid, and the supernatant fluid, after the corpuscles have settled, is yellow.

2. **Cytology**—If the fluid is turbid 5 c.c. should be centrifugalized and films made from the deposit and stained with Leishman's stain. Examination of the film will give a rough idea of the number and character of the cells present. More than three cells in a field may be regarded as pathological.

To carry out a **cell-count,** take a capillary pipette and make a mark on it with a grease pencil about 3 cm. from its distal end. Using this marked-off portion as a unit of volume, take 4 volumes of cerebro-spinal fluid and one volume of any simple stain (Loeffler's methylene blue or carbol thionin). Mix well in a watch-glass or clean test-tube and place a suitably sized drop in a Thoma-Zeiss counting-chamber.

Adjust the draw-tube of the microscope so that the diameter of the field with the $\frac{1}{6}$-in. objective is 8 small squares. Count the cells in 100 fields, refilling the chamber when necessary. Then the number of cells in 100 fields = the number in 1 c.mm.

For the area of one field = 50 small squares (approximately).

$$50 \text{ small squares} = \frac{50}{4{,}000} \text{ c.mm.} = \frac{1}{80} \text{ c.mm.}$$

but the fluid is diluted to four-fifths strength,

$$\therefore 100 \text{ fields} = \frac{100}{80} \times \frac{4}{5} \text{ c.mm.} = 1 \text{ c.mm.}$$

A special counting-chamber (e.g. the Neubauer) may also be used.

It should be noted that a cell-count must be done immediately the fluid has been collected. Counts done some hours later give very inaccurate results owing to the fact that pus cells stick together and to the sides of the tube, while endothelial cells break up in a short time. If any clot has formed an accurate cell-count cannot be obtained.

An excess of cells ("pleocytosis") is described as being of the polymorphonuclear type if these cells are above 75 per cent. of the total, and of the lymphocyte type if more than 90 per cent. are lymphocytes. A mixed type also occurs in which the polymorphs amount to from 15 to 70 per cent. of the total. The coccal forms of meningitis are associated with the polymorphonuclear type, syphilis with the lymphocytic type, and tuberculous meningitis and poliomyelitis with either a lymphocytic or a mixed type.

3. **Chemical examination.**—(a) *Proteins.* Normal cerebro-spinal fluid contains only a trace of albumin and hardly any globulin, the total protein being not more than 35 mg. per 100 c.c.

The protein content can be roughly estimated by placing 2 c.c. of fluid in a test-tube and carefully running in an equal quantity of absolute alcohol down the side. In normal fluid the line of junction is just visible; if protein is present in excess there is a turbid ring.

For accurate estimation of the total protein an Aufrecht's albuminometer must be used.

In a few instances, as in G.P.I., globulin is increased as much as the albumin. It is best tested for by the Nonne-Apelt reaction. To 1 c.c. of the fluid add an equal quantity of a saturated solution of pure neutral ammonium sulphate and shake. Any definite opalescence after standing for 3 minutes indicates globulin excess.

(b) *Glucose.* Normal cerebro-spinal fluid contains from 50 to 75 mg. glucose per 100 c.c., which is less than the amount of sugar in the blood.

If 1 c.c. of fluid is boiled with 0·25 c.c. of Fehling's solution the latter should be almost decolorized; if much blue is left it is an indication of reduced sugar content. Accurate estimation of the sugar is carried out in the same way as in blood (p. 225). The amount of sugar is diminished in cases of acute meningitis by the action of the glycolytic ferments set free from dead cells and bacteria.

(c) *Chlorides.* Normal fluid contains from 0·72 to 0·75 per cent. of sodium chloride. Amounts below this are met with in cases of meningitis and above it in renal inefficiency. The chlorides are estimated by adding 2 c.c. of fluid to 20 c.c. of distilled water and titrating with standard silver solution, chromate of potash being used as indicator.

(d) *Urea.* The amount of urea in cerebro-spinal fluid is always the same as the amount in the blood. Its separate estimation is, therefore, usually unnecessary.

The **Wassermann** reaction and **Lange's** colloidal gold test are of great value in many cases, but for these special books must be consulted. The bacteriological examination is described in Chapter XIV.

For a table showing the typical changes in the cerebro-spinal fluid in various diseases *see* the opposite page.

Table showing the Typical Changes in the Cerebro-spinal Fluid in various Diseases (*modified from Greenfield*).

—	Normal.	Meningitis.		Disseminated sclerosis	G P I.	Tabes	Meningeal syphilis	Acute anterior poliomyelitis.
		Meningo-coccal.	Tuberculous					
Physical characters	Clear: colourless, no coagulum.	Ginger-beer turbidity; coagulum.	Colourless, with cobweb coagulum	Clear, colourless	Colourless; sometimes fine coagulum	Clear, colourless	Clear or turbid, sometimes fine coagulum	Usually clear, sometimes cobweb coagulum.
Cells—								
Polymorphs	0 (Cells	200 to 2000	0 to 100	0	0 to 5	0	10 to 50	10 to 100 or higher
Lymphocytes	0 3 / per c mm	5 to 50	100 to 300	5 to 100	5 to 100	5 to 100	50 to 500	mixed count or chiefly polymorphs at first Lymphocytes later
Total protein	20 to 35	50 to 200	50 to 200	30 to 60 rarely higher	40 to 100	30 to 60	50 to 200	30 to 60 in early days rising to 100-200, and remaining high for 8 to 10 weeks
Globulin—								
Nonne-Apelt	0	± to +	± to +	− to +	± to + +	± to +	± to +	0 to +, later maybe ± +
Glucose[1]	50 to 75	0 to 15	15 to 50	Normal	45 to 60	15 to 70	Normal.	100
Chlorides[1]	720 to 750	650 to 680	580 to 650	,,	Normal	Normal	,,	Normal
W.R. . .	—	—	—	—	+	± in 80 % − in 20 %	±	—
Culture . .	Sterile	Meningo-cocci.	Sterile T B in films	Sterile	Sterile	Sterile	Sterile.	Sterile.

The characters of other pathological fluids are described in the Table on p. 537.

[1] mg. per 100 c.cm.

CHAPTER XIV

BACTERIOLOGICAL INVESTIGATIONS

In this chapter it is intended to indicate briefly the methods by which material may be obtained for bacteriological investigation and the value of such investigations in those cases where bacterial infection is suspected. It is not proposed that it should in any way replace the textbook of bacteriology, which should be consulted for further details of technique and for descriptions of the various bacteria and the reactions by which they may be identified. Many of the examinations, particularly those involving cultivation of material on special media, serological technique and injection of laboratory animals, will be carried out in the bacteriological laboratory. In such instances it is essential that the specimen sent to the laboratory should be labelled with the patient's name, age, and sex. The duration of the illness, the tentative diagnosis, and a few notes on the patient's clinical condition should be supplied. In this way the examination of the specimen in the laboratory may be greatly facilitated and the bacteriologist will be in a better position to indicate the possible significance of his findings. The result of bacteriological investigations must finally be assessed in relation to the clinical condition by the physician in charge of the patient, but it will often be helpful to have the opinion of the bacteriologist in the interpretation of the laboratory findings.

The bacteriological laboratory may be of assistance in the examination of a case by finding either an infecting organism or evidence of a specific infection in various serum and other reactions.

Collection of Specimens

All specimens should be taken with such precautions as will reduce to a minimum the chance of contamination from external sources, but admixture with antiseptics must be avoided. Specimens should be received directly into sterile vessels which are at once closed with suitable stoppers. In cases where cultural methods or animal inoculations are to be employed the examinations should be carried out as soon as possible after withdrawal of the material from the body, as many pathogenic organisms soon die out or, if present in mixtures, are overgrown. Where fluid specimens are to be sent by post, cotton-wool plugs are obviously useless and should be replaced by sterile rubber stoppers; the containers should be suitably packed in absorbent material and the package labelled "pathological specimen, with care."

Blood

A specimen of blood may be taken for examination by cultural methods or to have the serum examined for specific antibody by the methods described later under serum reactions.

Blood culture is indicated when bacteriæmia is suspected. By this is meant a condition in which bacteria are present in the circulating blood, reaching it from an improperly localized focus of infection. Bacteriæmia may be suspected in local pyogenic infections where the temperature and pulse rate fail to settle down. Typhoid or paratyphoid bacilli

are always present in the blood in the early stages of enteric fever, and the infecting organisms may be isolated by blood culture in cases of undulant fever, infective endocarditis and lobar pneumonia. In cases of pyrexia of unknown origin the isolation of an organism of the typhoid-paratyphoid group, or other bacterium, may serve to establish the true nature of the condition.

METHOD OF OBTAINING BLOOD FOR CULTIVATION

The blood should be withdrawn from a vein by means of a 10-c.c. syringe of "Record" or "all glass" type. The separate parts of the syringe should be sterilized by boiling in water for 10 minutes before use. When cool the parts should be put together by means of a sterile forceps and care should be taken that no water remains in the syringe as this would tend to produce laking of the blood.

A piece of rubber tubing should be used as a tourniquet and applied round the upper arm over the middle of the biceps so as to impede the venous but not the arterial flow. The skin at the bend of the elbow is "painted" with iodine in spirit. The skin is rendered tense by the operator's left hand; the syringe with needle attached is held in the right hand and almost parallel with the patient's arm; then the needle, with the bevel upwards, is inserted into a prominent vein. The median basilic is usually selected and the needle is pointed in the direction of the blood flow. Then 5 to 10 c.c. of blood is drawn up into the syringe and the tourniquet is removed before the needle is withdrawn as otherwise a hæmatoma tends to form. The blood is at once distributed into suitable tubes or flasks of medium. In certain cases it may be desirable to ascertain the number of living bacteria in the blood, and for this purpose the

specimen should be mixed with an equal volume of a sterile solution of 0·3 per cent. sodium citrate in 0·6 per cent. sodium chloride. Coagulation is thus prevented and known quantities of blood can then be incorporated in a suitable medium.

Success in the demonstration of bacteria which are present in the blood-stream depends on the number present, the amount of blood taken for culture, the use of suitable media, and the conditions of cultivation. For most purposes the volume of medium used should be at least ten times the amount of blood added, e.g. 10 c.c. of blood to 100 c c. of culture medium. The medium may with advantage contain trypsin or some other substance to prevent the clotting of the added blood and to destroy the antibacterial properties. Where bacteria are few in number several days may be required before they are detected in culture.

In suspected cases of Weil's disease the specimen of blood should be added to citrate to prevent clotting. The causal organism (*Leptospira icterohæmorrhagiæ*) will not grow in ordinary culture media, but may be detected by special cultural methods or by animal inoculation.

Bacteriæmia is usually present in infections with *Bact. typhosum*, *Bact. paratyphosum A and B Brucella abortus*, *Brucella melitensis* and the leptospira of Weil's disease, and may be a complication of infections due to streptococci, staphylococci, pneumococci and meningococci. Other organisms such as *B. coli*, the gonococcus, *Cl. welchii*, and the anthrax bacillus, are occasionally found in the blood-stream in unusually severe infections caused by them.

The identification of an organism isolated by blood culture may serve to determine the nature of an infective condition and also as a guide for specific

treatment. The result of the examination is frequently of value in prognosis. The isolation of typhoid bacilli from the blood in the early stage of typhoid fever is the rule and has no grave prognostic significance, whereas the isolation of the pneumococcus from the blood in a case of lobar pneumonia, or of a hæmolytic streptococcus in a case of puerperal infection, is of more serious import. In severe infections accompanied by bacteriæmia the determination of the number of the bacteria in the blood by repeated blood culture will serve to indicate the severity of the blood infection and the possible effect of treatment.

CEREBRO-SPINAL FLUID

Examination of the cerebro-spinal fluid by bacteriological methods should be made in any patient in whom meningitis is suspected.

The technique of lumbar puncture has already been described. In cases of meningitis the number of cells is always increased and, except in meningitis of tuberculous or syphilitic origin and in the rare cases of meningitis due to virus infection, the fluid is usually turbid and may be frankly purulent. Films should be made from the fluid, after centrifugation if necessary, and stained by Gram's and by Ziehl-Neelsen's methods. Bacteria may be recognized in films, and the results of this examination will indicate what cultural methods are to be adopted for the isolation and identification of the infecting organism.

In cases of tuberculous meningitis the bacilli are usually scanty and may be found in films of the centrifuge deposit only after prolonged search. Better results may be obtained if the fluid is allowed to stand at room temperature for some hours, when a

delicate cobweb clot of characteristic appearance frequently forms. A film made from the clot and stained by Ziehl-Neelsen's method will usually show acid-fast bacilli if careful search be made. The organism requires several weeks before visible growth appears on suitable media and, as the test by animal inoculation also takes 3 to 6 weeks, these two methods are seldom of practical clinical value in cases of tuberculous meningitis.

Meningitis may also be due to the meningococcus, pneumococcus, streptococcus, staphylococcus, *Hæmophilus influenzæ* and, less commonly, to other organisms. In these instances cultural methods should always be used to confirm and supplement the results obtained by microscopic examination of stained films of the fluid.

Sputum

Bacteriological examination of the sputum may be indicated in any inflammatory condition involving the trachea, bronchi or lungs.

The specimen is best collected first thing in the morning. The mouth should be washed out with warm water so as to avoid, as far as possible, oral contamination and excessive mixture with saliva. The specimen should be collected in sterile wide-mouthed metal or glass containers.

The bacteriological examination of sputum may be considered under two headings: (i) for the tubercle bacillus, and (ii) for other bacteria.

If the tubercle bacillus alone is to be sought for, a specimen may be satisfactory even if it is two or three days old. When, however, it is desired to obtain a true picture of the bacterial flora the specimen should reach the laboratory with as little delay as possible.

1. **Examination for tubercle bacillus.**—The tubercle bacillus is not readily culturable direct from specimens, but on the other hand it has characteristic staining qualities and is therefore sought for in film preparations.

A mucopurulent portion may be selected and spread fairly thickly on a microscope slide. Only new slides should be used. More satisfactory results are obtained if the sample of sputum be mixed with 5 volumes of 1-in-20 carbolic acid in a stoppered container, which is then thoroughly shaken. The specimen is then allowed to settle overnight and the supernatant fluid is discarded. The remainder is spun in the centrifuge for 5–10 minutes and films made from the deposit. The film should be allowed to dry, fixed by heat, and stained by Ziehl-Neelsen's method. The tubercle bacillus, being acid-fast, retains the fuchsin and shows up red against the blue background. (*See* Plate 26, facing p. 497.)

In young children and infants who habitually swallow their sputum, stomach washings may be examined.

A single negative examination for tubercle bacilli should not carry great weight, but the examination should be repeated several times in any case of suspected respiratory tuberculosis.

If present in very small numbers, tubercle bacilli may be demonstrated by animal inoculation. This examination, however, entails a delay of 3–6 weeks before a final report can be made.

2. **Examination for other bacteria.**—For identification of other bacteria films of the sputum should be stained by Gram's method and cultures prepared on suitable media. In cases of lobar pneumonia due to the pneumococcus, where specific treatment with anti-serum is contemplated, the

pneumococcal type should be determined as the therapeutic sera are only type-specific in their efficacy. The specimen of sputum should be examined as soon as possible and direct typing may be made on the specimen of sputum supplied, or the type may be determined by the indirect method involving the inoculation of the specimen into a mouse.

Lung puncture.—In cases of pneumonic consolidation where no sputum is available, material for bacteriological examination may be obtained by lung puncture, using a sterile syringe and needle as for pleural fluid. Puncture should be made over the consolidated area and the material obtained examined in the same way as sputum.

In the bacteriological examination of a child suspected of suffering from whooping-cough the cough plate method gives the best results. A Petri dish containing a special potato extract blood agar is held in front of the patient's mouth during a paroxysm and is placed in an incubator at 37°C. immediately thereafter. The examination of the plate within the next few days may reveal the presence of *H. pertussis.*

Sputum from normal individuals may contain various bacteria such as streptococci, pneumococci, diphtheroid bacilli, *Micrococcus catarrhalis*, and *H. influenzæ*. Except in cases of respiratory tuberculosis, acid-fast bacilli are very rarely found in the sputum, so that the findings of such organisms in a suspected case of pulmonary tuberculosis is of great diagnostic importance. In attempting to assess the significance of other bacteria found in the sputum of patients suffering from infections of the respiratory tract, one may be guided by the presence of pus cells and the relative numbers of various bacteria present.

THE THROAT AND NASO-PHARYNX

Bacteriological examination of the throat and naso-pharynx may be desirable (*a*) in inflammatory conditions for identification of the infecting organisms and (*b*) in healthy individuals to determine the presence of pathogenic bacteria, for example in searching for carriers of diphtheria bacilli, hæmolytic streptococci or meningococci.

Specimens should be obtained by rubbing the surface with a swab of cotton-wool wrapped around the end of a strong wire. The swab is kept in a narrow test-tube of stout glass and the other end of the wire may be fixed in the cork stopper. The whole apparatus is sterilized by dry heat and kept ready for use. In taking specimens it is important that no antiseptic should have been used (e.g. as a gargle) for some hours before. Where inflammatory exudate is present the swab should be rubbed firmly over the affected area and passed under the edge of the membrane in suspected cases of diphtheria.

The swab used for the naso-pharynx is longer than that used for the throat and the wire is bent near the extremity. A tongue depressor is used, the wire is passed into the mouth and up behind the soft palate, and then brought into contact with the posterior pharyngeal wall. Care must be taken not to touch any part of the mucous membrane of the mouth.

The method of examination of the material obtained varies according to the nature of the infection.

In suspected cases of diphtheria the swab is used to inoculate a slope of Loeffler's serum and, if desired, other special media containing tellurite. The inoculated media are kept at 37° C. and examined for the presence of diphtheria bacilli in suitably stained films after 6 to 18 hours. The bacteriological findings

must be evaluated in conjunction with the clinical condition and the final verdict must rest with the clinician. In a small percentage of cases of diphtheria the bacteriological report may be negative; when the clinical picture is that of a diphtheritic infection it is wiser not to wait for the bacteriological report before administering antitoxin.

In patients presenting the features of acute tonsillitis a Gram-stained film of the material on the swab may indicate the nature of the infecting organism and confirmation should be sought by cultural methods using blood agar and Loeffler's serum.

Vincent's angina may present superficial ulceration of the throat and gums and may simulate diphtheria when the lesion is confined to the region of the tonsils. Identification of the causal organisms should be made by the examination of stained films of the exudate. The organisms associated with the condition, a large Gram-negative bacillus and a spirochæte, do not grow on ordinary culture media. For their demonstration, Gram's stain may be used but polychrome methylene blue or Leishman's stain give better results.

The identification of meningococci in swabs from the naso-pharynx involves culture on suitable media such as serum or blood agar and the isolation and identification of the Gram-negative cocci by suitable tests. These organisms tend to die out rapidly in material kept at room temperature so that it is essential that naso-pharyngeal swabs should be received and examined in the laboratory as soon as possible after they have been taken.

For the detection of carriers the same general methods are used. It should be noted, however, that for the identification of diphtheria bacilli in such instances microscopical examination of films

from cultures must be confirmed by virulence tests in the guinea-pig before organisms which resemble diphtheria bacilli can be reported as such. The carrying out of a virulence test involves a delay of several days before a final report can be received.

A variety of bacteria may be present in the normal throat and naso-pharynx including diphtheroid bacilli, pneumococci, staphylococci, streptococci, influenza bacilli and Gram-negative cocci. Apart from the infections due to diphtheria bacilli and the organisms of Vincent's angina, acute inflammation of the throat and tonsils is most commonly due to hæmolytic streptococci, although staphylococcus aureus, pneumococcus and, occasionally, actinomyces and *Monilia albicans* may be responsible.

Nose and Nasal Sinuses

Examination of material from the nose may be carried out, as in the case of the throat, to identify the organisms responsible for inflammatory conditions or to detect carriers of pathogenic bacteria among apparently healthy individuals.

Material from the nose may be obtained by means of a throat swab. In cases of sinus infection, where there is free discharge, a specimen may be blown into a wide-mouthed bottle, or, if the discharge is not free, the sinus may be washed out with sterile saline and the washings sent for examination. The identification of diphtheria bacilli in material obtained from the nose cannot be made by microscopic examination of films from cultures alone and must be confirmed where necessary by a virulence test. For the identification of other bacteria in a nasal discharge, films of the material should be stained by Gram's method and the results of the examination of such films should be supplemented by culture on appropriate media.

In swabs taken from the healthy nose, diphtheroid bacilli, staphylococci, pneumococci and Gram-negative cocci may be present. Inflammation of the nose and accessory sinuses is most frequently due to the pneumococcus, hæmolytic streptococcus and staphylococcus aureus.

Fæces

In any intestinal infection, bacteriological investigation of the fæces may reveal the causal organism. In suspected cases of enteric fever and dysentery this examination should always be made, and is the best method for the detection of typhoid or dysentery carriers.

A loose motion should be obtained and in the case of suspected carriers it may be necessary to administer a purgative. Of a loose motion 1 c.c. is sufficient, and any abnormal portion, e.g. containing mucus or pus, should be selected. The usual sterilized throat swab soaked in fæces and replaced in its test-tube is satisfactory when there is no delay in making the examination. In other cases the specimen should be placed in a wide-mouthed vessel by means of a small metal spoon fitted into the stopper. In suspected cases of bacillary dysentery a satisfactory specimen may be obtained by means of a rectal swab; from material taken from the mucous membrane of the rectum in this way dysentery bacilli may be recovered more readily than from a sample of stool.

Where the making of cultures for organisms of the enteric and dysentery groups must be delayed for more than a few hours after the specimen has been taken, it is advisable to add to one volume of the fæces two volumes of 30-per-cent. neutral glycerol in 0·6-per-cent. sodium chloride solution and to make a thorough mixture. The presence of the glycerol prevents

the suppression of the specific organisms by *B. coli* which would otherwise occur.

In the laboratory, films of the specimen may be stained by Ziehl-Neelsen's method if the presence of tubercle bacilli is suspected. When the examination of such films shows the presence of acid-fast bacilli this result must be interpreted with caution, for acid-fast organisms other than the tubercle bacilli are sometimes found in the fæces. The identification of tubercle bacilli in fæces will finally depend on the inoculation of animals or suitable media with a portion of the specimen treated by one of the methods devised to destroy other bacteria without killing tubercle bacilli.

For the detection of bacilli of the enteric and dysentery groups, a suspension of the fæces should be sown in a selective liquid medium or on a solid medium such as MacConkey's lactose bile-salt agar. The identification of these bacteria depends on their isolation in pure culture and the results of fermentation and agglutination tests.

Isolation of the infecting organism in cases of enteric fever or dysentery is not always successful. The results will vary according to the relative number of these bacteria in the fæces, the interval elapsing after the specimen has been obtained and before cultures have been set up, the cultural methods used, and other factors. In dysentery, for example, the specific bacteria may be difficult to isolate after the first few days of an attack, especially if delay between the collection and cultural examination of the specimen occurs. In such cases further specimens should be examined and after the first week of the disease indirect evidence of the nature of the infection may be obtained by examination of the serum for specific antibodies (*see* below).

In the detection of carriers it may be noted that typhoid or dysentery bacilli may appear in the fæces only at irregular intervals and in small numbers. It may, therefore, be necessary to have many specimens examined at weekly intervals before a carrier is detected.

The fæces of normal individuals contain enormous numbers of various types of bacteria including lactose-fermenting and late lactose-fermenting coliform bacilli, non-hæmolytic streptococci, Gram-positive bacilli of the acidophilus type, and various ærobic and anærobic spore-bearing bacilli. The finding of the bacteria of these groups in pathological conditions affecting the alimentary tract has, therefore, no ætiological significance.

URINE

In any suspected case of bacterial infection of the urinary tract, examination of the urine should be made. It may be noted that in cases of obstruction due to any cause such as calculus, enlargement of the prostate, or urethral stricture, infection is liable to occur, although the symptoms of such infection may be masked by those of obstruction.

Urine affords a suitable medium for the growth of many bacteria, hence it is essential to prevent contamination of the specimen by extraneous organisms, and it is advisable that bacteriological examination should be carried out as soon as possible after the urine has been collected. In the male it is sufficient to wash thoroughly the glans penis and the meatus with 1-in-1,000 corrosive sublimate; the urine is then passed into two sterile flasks the first of which is rejected in case contamination has occurred. In the female, after similar precautions as regards cleansing, a sterile catheter must be used. The bacteriologist

should be notified whether or not the specimen is a catheter one and also of the date and hour of collection.

If organisms are scanty, the specimen should be centrifuged and films and cultures made from the sediment. Films should be stained by Ziehl-Neelsen's method for tubercle bacilli and by Gram's method for other bacteria. In staining by Ziehl-Neelsen's method the film should be treated for 1 minute with alcohol after decolorizing with acid. By this means the acid-fast smegma bacillus, which is frequently present on the external genitals and may contaminate the specimen, is decolorized. Tubercle bacilli in the urine occur either in small clumps or singly. They are morphologically very characteristic, but are often scanty. In such cases better results will be obtained if the sediment of a twenty-four hour sample is concentrated in the centrifuge and examined. In cases of suspected renal tuberculosis where pus cells are present in the urine, but no tubercle bacilli can be demonstrated microscopically, the inoculation of the urinary sediment into a guinea-pig may provide evidence of tuberculous infection.

In searching for other bacteria it is essential that the results of the examination of the Gram-stained films should be amplified by culture, the medium to be used being chosen in accordance with the findings in films. The bacteria can then be identified by their cultural character, fermentation reactions, and, if necessary, serological tests.

In some instances it may be desirable to ascertain the number of bacteria present in a sample of urine. This can be conveniently done by plating known quantities of the fresh specimen (diluted if necessary) on a solid medium.

Bacterial infection of the kidneys may be due to the tubercle bacillus, *Staphylococcus aureus*, or

members of the colon-typhoid-paratyphoid group of bacteria. Pyelitis is most frequently due to coliform bacilli, while cystitis may be due to any of these bacteria, or streptococci, or possibly the gonococcus. In cases of gonococcal urethritis, gonococci are generally to be found in the urinary sediment. In undulant fever due to *Brucella abortus* or in Weil's disease the causative organisms may be found in the urine but, as in the case of typhoid and paratyphoid infections, their excretion may be confined to a certain stage of the disease, or may be intermittent.

Pus and Purulent Exudates

In the examination of pus and purulent exudates one should be guided in some degree by the clinical course of the infection, the tissue or organ affected and, as in the case of actinomycosis (*see* below), the naked-eye appearance of the fluid may suggest a particular causal organism.

Pus may be collected by means of a sterile syringe and should be sent to the laboratory in a suitably plugged tube. Only in exceptional circumstances should a swab be used to collect the material.

In general the examination of pus should be by films and culture. Film preparations should be stained by Ziehl-Neelsen's method if there is any possibility of tuberculous infection, and by Gram's method for other organisms. The preliminary examination of films will be a guide in the planting of cultures. Cultures should in general be made on agar and blood agar. If there is reason to suspect an anærobic infection, such as tetanus or gas gangrene, a duplicate set of cultures on appropriate media will be set up under anærobic conditions.

The bacteria most commonly found in pus are staphylococci, streptococci, pneumococci and tubercle

bacilli. The gonococcus, meningococcus, coliform bacilli and actinomyces also cause suppurative lesions, while pus formation may occasionally be associated with localized infections by bacteria which do not usually produce suppuration, such as typhoid and paratyphoid bacilli, *Brucella abortus* and *Hæmophilus influenzæ*. In suppurative lesions following wounds and in those opening into a mucous surface more than one type of bacterium is frequently present.

In gonorrhœa the ease with which the organism may be detected varies with the stage of the disease and the effect of treatment. In the urethral discharge of acute gonorrhœa in the male the appearance of Gram-negative intracellular diplococci is sufficiently characteristic for diagnostic purposes. In chronic cases where microscopical examination of the discharge or urinary sediment is negative, the organism may be grown on a suitable medium such as agar containing blood or serum. Gonococci die out rapidly at room-temperature, and inoculated culture media should therefore be incubated at 37° C. as soon as possible. In the female adult patient, material for examination should be obtained from the urethra or cervix—not from the vagina. In cases of vulvovaginitis in children the vaginal discharge should be examined. In chronic cases of gonorrhœa where microscopic and cultural methods have given negative results the complement-fixation test may yield evidence of infection (*see* below).

Actinomycosis may be suspected if small granules are present in the pus. These granules can be readily seen if a few drops of pus are added to a tube of sterile saline or distilled water, when the granules rapidly sink to the bottom. If one of the granules is pressed between a slide and cover-slip it will be seen

to be composed of a central mass of filaments, and homogeneous club-shaped bodies may be seen at the periphery. Films stained by Gram's method show Gram-positive filaments of varying length, some of which show branching. The organism can be cultivated only under anærobic conditions.

In suspected cases of "malignant pustule" the anthrax bacillus may be demonstrated in films and cultures made with the exudate from the lesion or the fluid obtained from the blisters which are usually present.

Serous exudates which have any considerable cellular content should be examined in the same way as pus. If the cellular content is scanty centrifugation may be necessary as a preliminary.

Examination for Spirochæta Pallida

In untreated syphilis in the primary stage, careful examination of fluid from the chancre will usually reveal the causal spirochæte. The sore should be cleaned with a sterile swab and saline, and then is squeezed gently until a little serous fluid exudes. If the fluid is blood-stained this should be wiped away until a specimen free from blood can be obtained. The fluid should be taken up in a piece of capillary glass tubing, the ends of which are sealed with a flame and the specimen dispatched to the laboratory. The fluid should be examined in a wet preparation under dark-ground illumination. The spirochæte is recognized by its size and close spirals. Where the equipment necessary for dark-ground illumination is not available the spirochæte may be seen in films stained by Giemsa or Fontana's method, but identification of the spirochæte in such films is not always easy. In the less common extragenital chancres, as for example on the lips or tongue, the

examination may be complicated by the fact that other spirochætes resembling the *Spirochæta pallida* may be present. As the Wassermann reaction does not usually become positive until one to three weeks after the appearance of the primary sore, the detection of the spirochæte is the most certain laboratory aid to diagnosis during this period.

Serum Reactions in Diagnosis

When a patient becomes infected with bacteria his tissues usually respond by the formation of antibodies. These antibodies are usually specific for the type of infecting bacterium and, in consequence, the detection of antibodies against a specific organism may afford evidence as to the exact nature of the infection. It should, however, be emphasized that the evidence obtained by the examination of a patient's serum for antibodies can in most cases only be presumptive and is never of the same value as the detection of the infecting organism. Further, as the production of specific antibodies is a response to the infection these will not usually appear during the first week or so of the disease, so that negative serum tests at this time cannot be taken as evidence against the presence of a suspected infection. It may be noted that, in general, the infecting organism is most readily detected before the appearance of specific antibody.

The presence of antibody in a patient's serum may be revealed by the agglutination reaction or by the complement-fixation test. Blood is obtained by venepuncture as for culture, except that the blood (5.0 c.c.) is placed in a sterile tube and allowed to clot. The serum is later separated from the clot and freed from blood-cells by centrifugation if necessary.

Agglutination Reactions

For these tests, increasing dilutions of the patient's serum are mixed in small tubes with suspensions of the type of organisms with which the patient is believed to be infected. The tubes are then placed in a water bath at 55°C. for a few hours before being examined. The presence of specific agglutination will be indicated by the appearance of clumps in the bacterial suspension. The agglutinating titre of the serum is the highest dilution which produces visible clumping of the bacteria.

Standard suspensions of various organisms suitable for use in agglutination tests may be obtained from the Department of Pathology, University Museum, Oxford.

Agglutination tests with the patient's serum may be of value in the diagnosis of infections due to the typhoid and paratyphoid bacilli, the dysentery bacilli, *Brucella abortus*, and the spirochæte of Weil's disease.

In assessing the results of agglutination tests in suspected cases of enteric fever, the patient's history as regards prophylactic inoculation with typhoid vaccine or previous enteric infection is important. The agglutination titre of normal human serum for organisms of the typhoid-paratyphoid group is low, but a higher titre than normal may be present in individuals who have previously been inoculated with typhoid vaccine, or suffered from typhoid fever. The duration of illness is also important; agglutinins in the serum are strong towards the end of the third week of enteric infection so that, if a result of doubtful significance is obtained by the agglutination test in the second week of a patient's illness, a second examination made a week later will be helpful.

In bacillary dysentery the acute stage of the illness frequently lasts only a few days and a significant increase in serum agglutinins above normal does not occur in all cases. A negative agglutination reaction against the various types of dysentery bacilli does not, therefore, exclude the possibility of infection with one of the types.

In suspected cases of undulant fever the agglutination test is of great value as most cases of infection with *Brucella abortus* show a high titre of agglutinins after the first week of the disease.

In testing for agglutinins against the spirochæte of Weil's disease, dilutions of the serum are mixed with a living culture of the organism and the mixtures are examined microscopically by dark-ground illumination. A positive reaction is indicated by the cessation of motility and clumping of the organisms. Practically all cases of Weil's disease give a well-marked positive reaction after the first week of illness.

Complement-Fixation Tests

These tests are based on the fact that serum containing antibody when mixed with a suitable antigen has the power of fixing a measurable amount of complement. Serum to be tested is heated for 30 minutes at 56° C. to destroy its own complement. In doing the test, a known dose of complement is allowed to act for a given time with a known amount of antigen and the serum to be tested. At the end of this time the presence or absence of free complement is tested for by adding a known amount of red cells which have been sensitized with a hæmolytic antibody. If free complement is still present the red cells are laked and the reaction is negative. If the complement has been fixed no hæmolysis takes place and the reaction is positive.

The complement fixation test in the diagnosis of syphilis is usually known as the Wassermann reaction. A positive reaction may be expected from 5 to 7 weeks from the date of infection, or from one to three weeks after the appearance of a primary sore. The reaction is positive in all cases of secondary syphilis and practically all cases of untreated tertiary lesions. Further, a positive reaction will be obtained with the cerebro-spinal fluid in all cases of general paralysis, in most cases of cerebral syphilis and, less constantly, in tabes dorsalis.

It will be seen, therefore, that, although a negative reaction does not exclude a diagnosis of syphilis, the reliability of the test is such that, in the presence of a negative test, strong evidence will be necessary to uphold such a diagnosis. It must also be borne in mind that a positive reaction may be present in latent cases and, therefore, may be met with in patients suffering from some other disease.

In the complement-fixation test as applied in the diagnosis of gonococcal infection a suspension or extract of gonococci is used as antigen. A positive reaction is not to be expected in the acute stage o gonorrhœa and may never be obtained in simple cases of urethritis. The reaction is generally positive in cases with complications and is of especial value in chronic pelvic disease and in cases of arthritis and tenosynovitis.

In cases of whooping-cough the serum obtained in the third week of the disease and later usually fixes complement in the presence of an antigen made from a suspension of *Hæmophilus pertussis;* so that the test is most useful at that stage of the disease when cultural methods are likely to fail.

In cases of hydatid disease the serum usually gives a positive complement-fixation test in the

presence of a hydatid antigen. The reaction may, therefore, be of value in suspected cases of this infection.

PAUL-BUNNELL TEST

This test is diagnostic of glandular fever. The patient's serum is inactivated at 56° C. for half an hour. Dilutions are made with normal saline from 1 in 2 to 1 in 128. An equal volume of 2 per cent. washed sheep cells is added, giving final dilutions of 1 in 4 to 1 in 256. The tubes are left over night at room temperature and next morning each tube is gently inverted to look for agglutination. A positive result is one in which agglutination occurs in dilutions from 1 in 16 onwards.

VIRUS DISEASES

The laboratory may be of help in the diagnosis of certain virus diseases by (1) the detection of a particular virus, or (2) by determining the presence of specific antibodies in a patient's serum. The success of the first procedure will depend on whether the virus to be looked for is capable of infecting laboratory animals or can be grown by the special methods used for the cultivation of viruses in living tissues *in vitro*. The success of the second procedure will depend on whether a suitable antigen is available. These conditions and the fact that the carrying out of such tests requires a specialized knowledge has imposed a limit on their use in routine laboratory diagnosis. Laboratory investigations along the lines indicated may be useful in the following diseases:

Psittacosis.—Where the history and clinical picture suggests this disease, sputum may be examined for the presence of virus. After the first week the blood serum of the majority of cases gives a positive complement-fixation test with a psittacosis antigen.

Lymphocytic choriomeningitis.—In the acute stage

of this condition the virus may be isolated from the cerebro-spinal fluid by inoculation of animals and in convalescence the blood-serum may be examined for the presence of specific antibodies.

Lymphogranuloma inguinale.—Virus may be demonstrated in smears from the bubo by special staining methods or by inoculation of animals. A skin-test is also of value in later stages of the disease (*see* below).

Smallpox.—In doubtful or atypical cases of smallpox the nature of the infection may be established by sending crusts of the lesions to a suitable laboratory. Complement-fixation tests with appropriate sera, using extracts of the crusts as antigen, may serve to elucidate the nature of such cases.

Skin-Tests

Various skin-tests are used as a measure of susceptibility of an individual's tissues to the test substances. This susceptibility is indicated by an inflammatory reaction at the point of application or injection of the test substance; it may indicate a normal susceptibility to toxic material, e.g. bacterial toxin as in the Schick or Dick tests, or it may indicate a state of hypersensitiveness or allergy to a bacterial or other protein as in the tuberculin test. In the first instance a positive reaction may be expected in persons who have had no previous contact with the toxic substance whereas the second (allergic) type of response may be the result of sensitization through previous experience of the test substance.

The Schick Test

This is a test of susceptibility or immunity ot diphtheria toxin. The toxin is so diluted that the test dose is contained in 0.2 c.c., and similarly diluted heated toxin is used as control. Glass syringes,

graduated in tenths of a cubic centimetre, should be sterilized by boiling and allowed to dry before use; a No. 18 or 20 needle with short bevel should be used. The skin of both forearms should be washed with soap and swabbed with alcohol or ether; when dry the skin is stretched by holding the forearm firmly with the left hand, the injection being made with the right. Introduce the needle almost parallel with the skin and bevel upwards; when the bevel has disappeared inject 0.2 c.c. The control heated toxin is injected into one forearm and the unheated toxin into the other. In individuals who are not immune to the toxin a positive reaction will usually appear within 24 hours and will reach its maximum on the 4th or 5th day. Any non-specific reaction due to the protein in the medium from which the toxin was prepared will show within 24 hours on the control arm, but will usually have disappeared by the 4th day when the final reading should be made. By this test it is possible to divide people into two groups, immune and susceptible. By inoculating the susceptibles with toxoid-antitoxin mixtures or floccules it is possible to render them immune. Where this practice has been widely adopted in young children it has been found possible to reduce the incidence of diphtheria to negligible proportions.

The Dick Test

This test is similar to the Schick test but applies to scarlet-fever, and the toxin used is derived from scarlatinal streptococci. The test is, however, of doubtful value because of the difficuty of immunizing against streptococcal infection.

The Tuberculin Test

This test is based on the fact that the tissues of a person infected with the tubercle bacillus become

sensitized to tuberculin. The older cutaneous test of Von Pirquet has been largely replaced by the more delicate intracutaneous method of Mantoux and the recently introduced patch test.

Von Pirquet's cutaneous test.—This test consists of placing a drop of "Old Tuberculin" on the forearm and a drop of 50 per cent. glycerin broth adjacent to it as a control; with a small lancet or needle a scratch is made through each of the drops. A positive reaction is denoted by the appearance within 24 to 48 hours of a bright-red papule, at least 5 mm. in diameter, at the site of scarification through the tuberculin.

The Mantoux test.—In applying this test 0.1 c.c. of a 1-in-10,000 dilution of "Old Tuberculin" is injected intradermally into the skin of one forearm, a similar dilution of glycerin broth being injected from another syringe into the other arm as control. A positive reaction is characterized by the development within 48 to 72 hours of an area of erythema and infiltration, 10 mm. or more in diameter, at the site of the injection of the tuberculin. If the reaction is negative after 48 hours a dilution of 1 in 1,000 may then be injected and, if this is negative, a further test with 1 in 100 dilution may be made.

The Patch test method.—This method consists in applying to the skin, by means of adhesive plaster, tuberculin in the form of a powder or paste. The necessary preparations have recently been placed on the market by various firms. The skin is thoroughly cleansed with alcohol and ether and, when dry, the plaster bearing the tuberculin is applied. The material is removed after 48 hours, when the result is read.

The value of the tuberculin test is limited owing to the fact that as a person gets older the less likely is he or she to have escaped previous clinical or sub-

clinical tuberculous infection. At the age of 1 year, approximately 5 per cent.; at 5 years, 20 per cent.; at 10 years, 40 per cent.; at 15 years, 60 per cent.; and, at 20 years or over about 90 per cent. of individuals in an urban population give a positive reaction to tuberculin. In rural communities the percentage of positive reactions is rather less. In young children a positive reaction will have greater significance, while at all ages a negative reaction will be of value as evidence against a diagnosis of tuberculosis. It must be remembered, however, that in the earliest stages of the disease the reaction may be negative, and in very acute cases or in the last stages, when no response from the tissues can be expected, a negative reaction may be obtained.

The Frei Test

This test is widely used in the diagnosis of lymphogranuloma inguinale. The antigen consists of pus from a bubo, or brain suspension from an experimentally-infected mouse, heated to 60° C. for 2 hours. Intracutaneous injection of 0.1 c.c. gives rise in an infected subject to an infiltrated inflammatory area, at least 5 mm. in diameter, with a central zone of necrosis. The reaction reaches its height in 48 or 72 hours.

The Casoni Test

This test is useful in suspected hydatid disease. The antigen is prepared from the fluid or walls of cysts and on injection intradermally gives rise in positive cases to a reaction of the wheal and erythema type. The reaction appears in 5 or 10 minutes and reaches its maximum within about an hour.

Tests for Sensitivity to Various Animal or Vegetable Proteins

In the investigation of patients suffering from allergic states, such as asthma or hay fever, the

sensitiveness to various animal or plant proteins may be determined by skin tests. Sterile watery extracts of such materials as horse-hair, feathers, various pollens, fish muscle, fruit, eggs, etc. are on the market in a form ready for use. A single line of scarification, about 1 cm. long, is made through a drop of fluid and various solutions may be tested at one time. Sensitivity to a particular extract is indicated by a reaction of the wheal and erythema type, which appears within 10 minutes and attains its greatest size in from 30 minutes to an hour. The examination of the responses to these skin tests in a patient suffering from hay fever or asthma may provide information of value in the prevention of further attacks.

Sedimentation Rate

In health the red cells of the blood agglutinate very little on standing; the resultant clumps of corpuscles are small and sediment slowly. On the other hand, in certain diseases the red cells agglutinate into larger clumps which sediment more rapidly. As a sick person improves, the clumps become smaller and the sedimentation rate slower. There is no standard method for estimating the erythrocyte sedimentation rate. The Westergren method is simplest, and the one most generally in use. In this technique 0.2 c.c. of 3.8 per cent. sodium citrate is sucked into a 1 c.c. syringe and blood is then drawn by venepuncture up to the 1 c.c. mark. The mixture is shaken so that it is evenly mixed. The blood is then sucked into a pipette 2.5 mm. in diameter and graduated to 200 mm. This is fixed in a stand and sealed by being clipped on to a cork at the bottom. The distance settled in millimetres in one hour is the sedimentation rate. The normal is 1 to 7 mm., the slightly abnormal range is 8–15 mm. and the grossly abnormal 15–110 mm.

The erythrocyte sedimentation rate serves as a

rough index of the concentration of plasma fibrinogen. It fails, however, in the presence of abnormally elevated levels of serum globulins or of lipids, and when there is advanced microcytic or macrocytic anæmia. Other methods have been devised in an attempt to correct these factors. They include the Rourke-Ernstene, Wintrobe-Landsberg, Linzenmeier and Cutler methods. The Rourke-Ernstene is the most reliable of them but it is time-consuming and complex. The other methods are less reliable than the Westergren.

The sedimentation rate is useful in following the progress of patients suffering from chronic diseases. It is especially useful in chronic pulmonary tuberculosis, rheumatoid arthritis and acute rheumatism. So long as the sedimentation rate is accelerated, there is an active disease process, and this is frequently found a considerable time after the temperature has returned to normal. It is convenient to estimate the sedimentation rate at weekly intervals. A trend towards acceleration of the rate is an indication that the disease process is active; whereas a trend towards slowing of the rate indicates that it is subsiding.

The *sedimentin index* is the logarithm of the maximum velocity of sedimentation expressed in millimetres per hundred minutes. It is obtained by noting the amount of fall every five minutes in the Westergren method. The series of figures so obtained is plotted on a time-distance graph. The steepest part of the curve so obtained is produced in both directions, and from it the maximal velocity is calculated in mm. per hundred minutes.

STERNAL PUNCTURE

In the investigation of certain anæmias sternal puncture is employed. It is a biopsy method, and as such it produces results inferior to the older studies made from histological sections obtained by necopsy

methods. However, it is arguable that the cells obtained by sternal puncture in life are less degenerate than those seen in necropsy material.

The apparatus used for puncturing the sternum is a stout trochar and cannula, fitted with an adjustable stop, and so made that a hypodermic syringe fits into the top of the cannula. The skin and tissues down to the periosteum of the sternum are anæsthetized with 2 per cent. novocaine without adrenalin. A small incision is made in the skin with a tenotome, slightly lateral to the mid point of the manubrium. The stop on the trochar is unscrewed and adjusted according to the build of the patient; the adjustment may well be 1 cm. at first. The corticalis of the sternum is pierced, the stop being unscrewed to lengthen the projecting portion of the trochar if necessary. Considerable force may be required, but it is easy to recognise the entry of the tip of the trochar into the marrow cavity. The trochar is withdrawn, the hypodermic syringe inserted, and as few drops as possible of thin red fluid aspirated. Without disconnecting the syringe, the trochar is removed and a swab put over the incision. The plunger of the syringe is pushed, and as quickly as possible films are made on slides from the fluid expressed from the trochar. The syringe being disconnected from the trochar, a drop of thin red fluid is expressed on to a slide, and with a hæmocytometer pipette a preparation is made as for a red cell count.

APPENDIX

Weights and Measures

1. Imperial weights and measures.

1 grain, gr.
1 ounce, oz. = 437 5 grains.
1 pound, lb. = 16 ounces = 7,000 grains.
1 minim = 0 91146 grain.
1 fluid drachm = 60 minims.
1 fluid ounce = 8 fluid drachms.
1 pint = 20 fluid ounces.
1 gallon = 8 pints.

2. Relation between Imperial and metric systems.

1 grain = 64 8 milligrammes (mg.)
1 ounce = 28 3 grammes (grm)
1 lb. = 453 6 grammes.
1 gramme = 15 432 grains.
1 kilo = 2 lb. 3 oz.
1 minim = 0 059 cubic centimetres (c.c)
1 fluid drachm = 3 5 c c
1 fluid ounce = 28.39 c.c.
1 pint = 567 9 c.c.
1 c.c. = 16 9 minims.
1 litre = 35 2 fluid ounces.
1 inch = 2 54 centimetres (cm.).
1 foot = 30 48 cm.
1 yard = 91 44 cm.
1 cm. = 0 39 in.
1 metre = 39 37 in.

3. Conversions.

To convert grammes per 100 c.c. into grains per ounce, multiply by 4·375.

To convert grammes into ounces avoirdupois, multiply by 10 and divide by 283.

To convert litres into pints, multiply by 88 and divide by 50.

To convert kilos into pounds, multiply by 1,000 and divide by 454.

4. Centigrade and Fahrenheit scales.

To convert Fahrenheit into Centigrade, subtract 32, multiply the remainder by 5, and divide the result by 9

To convert Centigrade into Fahrenheit, multiply by 9, divide by 5, and add 32.

The following table and figure show the relation of degrees Fahrenheit to Centigrade, as far as is likely to be required in clinical work :—

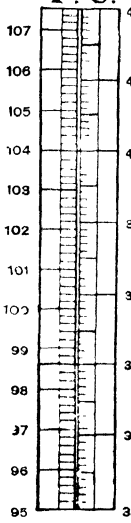


Fig 141
Fahrenheit and Centigrade scales compared

Centigrade.	Fahrenheit	Centigrade	Fahrenheit.
110°	230°	37°	98 6°
100	212	36 5	97 7
95	203	36	96 8
90	194	35 5	95 9
85	185	35	95 0
80	176	34	93 2
75	167	33	91 4
70	158	32	89 6
65	149	31	87 8
60	140	30	86
55	131	25	77
50	122	20	68
45	113	15	59
44	111 2	10	50
43	109 4	+ 5	41
42	107 6	0	32
41	105 8	− 5	23
40 5	104 9	− 10	14
40	104 0	− 15	+5
39 5	103 1	− 20	− 4
39	102 2	0 54°	1°
38 5	101·3	1	1 8
38	100 4	2	3 6
37 5	99·5	2·5	4 5

Solutions required for Examination of Gastric Contents

5. Phloroglucin and vanillin solution.

Dissolve 2 grm. of phloroglucin and 1 grm. of vanillin in 30 c c. of absolute alcohol. Keep the solution in the dark, and use it economically.

6. Boas's resorcin reagent.

Resorcin	75 gr.
White sugar	45 gr.
Dilute spirit	3½ oz.

Dissolve.

7. Uffelmann's reagent.

Carbolic acid (1 in 20)	10 c.c.
Distilled water	20 c.c.

Mix.

Add one or two drops of liq. ferri perchlor. An amethyst-blue solution results. It should be prepared fresh each time, as it does not keep. Lactic acid turns it yellow. Hydrochloric acid simply discharges the blue colour. Acetic and *combined* hydrochloric acid turn it somewhat brownish.

8. Congo red test papers.

These are made by soaking bibulous paper in a solution of Congo red, of the strength of 1 decigramme to 100 c.c. of water, or in a saturated alcoholic solution. They are allowed to dry, and are then ready for use.

Solutions required for Urinary Testing

9. Hypobromite solution.

Dissolve 100 grm. of caustic soda in 250 c.c. of water; cool, then add 25 c.c. of bromine. The solution is apt to undergo the following decomposition:

$$3NaBrO = 2NaBr + NaBrO_3.$$

It is therefore better to prepare it as required by adding 2·5 c.c. of bromine to 25 c.c. of the caustic soda solution.

The bromine is supplied in small tubes, which readily break when shaken up smartly with the soda solution in a stout stoppered bottle.

10. Esbach's reagent.

Dissolve 10 grm. of picric acid and 20 grm. of citric acid in about 900 c.c. of boiling water; cool, and add water to 1 litre.

11. Fehling's solution.

(*a*) Take 34.64 grm. of pure sulphate of copper which has been powdered and pressed between bibulous paper, dissolve in 200 c.c. of warm distilled water; cool, and fill up to 500 c.c.

(*b*) Dissolve 180 grm. of crystallized Rochelle salt in 300 c c. of hot water, filter, and add 70 grm. of pure caustic soda, or 100 grm. of potash; cool, fill up to 500 c.c.

When required, mix equal volumes of (*a*) and (*b*). The result is an alkaline solution of potassic cupric tartrate, of which 1 c.c. is exactly reduced by 5 mg. of pure glucose

12. Benedict's reagent.

Copper sulphate	17·3 grm.
Sodium citrate	173 grm.
Anhydrous sodium carbonate	100 grm.
(or crystalline sodium carbonate	200 grm.).

Dissolve the copper sulphate in 100–150 c c of distilled water, and add slowly with constant stirring the other ingredients dissolved in distilled water and filtered ; this should amount to about 800 c.c. Finally, make up to 1 litre with distilled water.

Solutions required in the Examination of Blood

13. Diluting fluid for hæmocytometer.

Sulphate of soda	104 gr.
Acetic acid	1 dr.
Distilled water	6 oz.

14. Diluting fluid for hæmocytometer (by Strong's method).

Sodium chloride	0·95 grm.
Sodium citrate	0 85 grm.
Commercial formalin	0 5 c.c.
Distilled water	100 c c.

15. Toisson's solution has the following formula:

Methyl violet, 5B	0 025 grm.
Sod. chlor.	1·000 grm.
Sod. sulph.	8·000 grm.
Neut. glycerin	30 000 grm.
Aq. destill.	160·000 grm.

Should be filtered just before use.

16. Hayem's solution.

Sodium chloride	1 grm.
Sulphate of soda	5 grm.
Corrosive sublimate	0·5 grm.
Distilled water	200 c.c.

17. Teichmann's test for blood (hæmin test).

Take up in a pipette some of the deposit to be examined. Rub it up with a small amount of common salt, and evaporate a little of the mixture to dryness on a slide. Moisten the residue with glacial acetic acid, and put on a cover-glass. Gently heat this over a very small flame for several minutes, avoiding boiling. Let a little glacial acetic acid run in from the side of the cover-glass from time to time during the process. Allow to cool, and examine for hæmin crystals with a high power.

Some Staining Methods

18. Gram's method (Lillie's modification).

The original Gram's method involves the use of aniline water gentian violet. This is an unstable solution and requires to be made up fresh at frequent intervals. It is recommended, therefore, that Lillie's modification should be used.

To stain by Lillie's modification of Gram's method:—

(1) Stain for half a minute with ammonium oxalate crystal violet solution (2 grammes crystal violet, 20 c.c. absolute alcohol, 80 c.c. of 1-per-cent. aqueous solution of ammonium oxalate).

(2) Pour off the crystal violet solution and pour on strong Lugol's solution (iodine 1 part, potassium iodide 2 parts, distilled water 100 parts). Pour off and add a fresh quantity of iodine solution. Leave for half a minute.

(3) Wash off with water and remove excess water with filter paper. Decolorize with acetone 1 to 5 seconds.

(4) Wash off with water and counterstain with 1-per-cent. watery solution of safranin for half a minute.

(5) Wash with water, blot and dry.

19. Ziehl-Neelsen's method of staining for tubercle bacilli.

(1) Carbol fuchsin:

Basic fuchsin	1 grm.
Absolute alcohol	10 c.c.
5-per-cent. aqueous carbolic acid..	90 c.c.

(2) 25-per-cent. aqueous solution of sulphuric acid.
(3) Loeffler's methylene blue:

Saturated alcoholic solution of methylene blue	30 c.c.
Distilled water	100 c.c.
1-per-cent. solution of caustic potash	1 c.c.

Pour on hot carbol fuchsin and leave for three minutes. Wash off and place in the acid until on washing in water not more than a faint pink colour returns. Pour on the methylene blue solution and leave for one minute. Wash in water, blot and dry.

20. **Loeffler's stain.** (This should be freshly prepared.)

Concentrated alcoholic solution of methylene blue	1 c.c.
Caustic potash in 0 1-per-cent. aqueous solution	3 c.c.

Specimens are stained in five to thirty minutes. Excess of stain is discharged by rapid washing in water acidulated with acetic acid (2 drops of acid in a small bowl of water), and all traces of acid are well washed out. The specimen is then dried and mounted.

21. **Carbol thionin blue.** (Prepared freshly.)

Saturated solution of thionin blue in 50-per-cent. alcohol ..	10 cc..
1-in-40 solution of phenol in water	100 c.c.

This stain is one of the best for film preparations. After staining, which is rapidly effected, wash the specimen in water, then dry and mount. Sections should, after washing, be passed through alcohol containing a trace of ammonia, thereafter dehydrated by absolute alcohol, cleared with xylol, and mounted in balsam.

22. **Neisser's method of staining B. diphtheriæ.**

Stain No. I	Methylene blue	1 grm.
	Absolute alcohol	20 c.c.
	Glacial acetic acid	50 c.c.
	Distilled water	1,000 c.c.
Stain No. II.	Crystal violet	1 grm.
	Absolute alcohol	10 grm.
	Distilled water	300 c.c.

Stain No. III.	Chrysoidin	1 grm.
	Distilled water	300 c.c.
	Heat to dissolve, and filter	

The film should be fixed by heat.

Two parts of No. I stain should be added to one part of No. II. Stain with this mixture for 5 minutes. Wash with water and then stain with No. III for 10 seconds. Wash in water and blot.

The mixture of I and II must be made up fresh daily.

23. Hiss's method of capsule staining.

This is very useful for the demonstration of pneumococci in the sputum or in pneumococcal exudates. The stain consists of 1 part of a saturated alcoholic solution of fuchsin and 19 parts of distilled water. A film of the material to be examined, having been dried and fixed, has a few drops of the stain placed upon it, and is heated for a few seconds until steam rises. The stain is then washed off with a 20-per-cent. solution of copper sulphate; without being washed in water, the preparation is dried between filter-papers and mounted in balsam.

24. Ehrlich's triacid stain.

Prepare saturated watery solutions of *chemically pure* and crystalline orange-G, acid fuchsin, and methyl green. Then make the following mixture:—

Orange-G solution	13–14	c.c.
Acid fuchsin solution	6–7	c.c.
Distilled water	15	c.c.
Alcohol	15	c.c.
Methyl green solution	12.5	c.c.
Alcohol	10	c.c.
Glycerin	10	c.c.

The fluids must be measured out in the above order, in the same glass, and from the addition of the methyl green onwards the mixture is thoroughly shaken. The solution can be used at once, and keeps indefinitely.

Blood-films stain in it in one to five minutes, depending upon the particular blood under examination and the mode of its fixation. The exact time required can therefore only be found out by experiment.

25. The Romanowsky stains.

These stains depend for their action on the compounds formed by the interaction of methylene blue and eosin, as

originally introduced by Romanowsky. They are used for staining blood-films, the cells in pathological fluids, films or tissues containing bacteria, and material containing protozoal parasites, e.g. malaria.

The chief combinations in use are those of Leishman, Jenner, Giemsa, and J. H. Wright. " Soloids " for the preparation of these stains can be had from Burroughs Wellcome & Co., through any dealer.

Leishman's preparation is perhaps that most used, and is prepared by dissolving one " soloid " in 10 c.c. of *pure* methyl alcohol. It is of great importance that the methyl alcohol should be " acetone-free."

The instructions sent with the various soloids should be strictly followed, and the bottle in which the stain is stored must be well stoppered. If the preparation is too blue, this may be corrected by careful washing with acetic acid, 1-in-1,500; if the eosin tint is too strong, it can be lightened by the use of 1-in-7,000 solution of caustic soda. The application of the stain is described in Chap. V (p. 209).

With this stain the red blood-corpuscles are coloured pink, the nuclei of leucocytes a reddish purple, and any acidophil or basophil granules pink and blue respectively. The nuclear substance of the malarial parasite is stained a reddish purple. In trypanosomes the two nuclei have a purplish stain, the protoplasm is blue, and the edge of the undulatory membrane is stained pink.

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