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CHARLES R. BOX, M.D.

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POST-MORTEM MANUAL

A HANDBOOK OF MORBID ANATOMY AND POST-MORTEM TECHNIQUE

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CLINICAL APPLIED ANATOMY

BY

C. R. BOX, M.D., B.S., B.Sc., F.R.C.P., F.R.C.S.

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POST-MORTEM MANUAL

A HANDBOOK OF MORBID ANATOMY AND POST-MORTEM TECHNIQUE

BY

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PREFACE

This manual is intended as a guide for students engaged in post-mortem work, and for those medical men who may, in the course of practice, be called upon to conduct autopsies.

The procedures described are based upon a long experience of post-mortem work at St. Thomas's Hospital, and are such as have been found to be most satisfactory in actual practice.

Care has been taken to explain in detail those manipulations which are apt to present special difficulties.

The naked-eye appearances which are characteristic of disease of the different viscera have been summarised, and will be found appended to the sections descriptive of the removal and examination of the viscera in question, an arrangement which, it is hoped, will prove convenient.

As the book is essentially a manual for the post-mortem room, no attempt has been made to enter into details of microscopical examination or bacteriological identification; but, at the same time, notes have been added to indicate the circumstances under which such examinations may be desirable.

For many of the details in the section on embalming I desire to record my indebtedness to the writings of Dr. J. G. Garson.

For the illustrations I wish to express my thanks to my friend, Mr. D. C. Bluett.

CHARLES R. BOX

LONDON,

June 1910.

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POST-MORTEM MANUAL

A HANDBOOK OF MORBID ANATOMY AND POST-MORTEM TECHNIQUE

CHAPTER I

PRELIMINARY CONSIDERATIONS

The instruments which are actually needed for the performance of a post-mortem examination are neither many nor complicated.

The body is opened and the viscera are removed by means of a short, broad-bladed knife generally known as a 'post-mortem knife.' The shape of this knife is shown in Fig. 1, whilst beside it, for contrast, is placed the pattern known as a cartilage knife, which is not so generally useful. A Swedish knife, which can be bought at any cutler's, is a very efficient substitute for a post-mortem knife on emergency, but because of its wooden handle cannot be sterilised by boiling.

A second knife, with a broad blade at least seven inches long, and technically known as a 'brain knife' should be used for slicing the brain or other viscera.

Long-bladed scissors are used to open the stomach, bowels, œsophagus, air passages, and cavities of the heart. These scissors, which are known as 'intestine scissors,' should be without the hook on the long blade with which they are usually provided. The two patterns are shown in Fig. 2.

Stout surgical scissors, sharp scalpels, dissecting forceps, a thin grooved director, and a probe are required for various purposes.

A medium-sized bone forceps and a periosteal elevator

or rugine are also useful. For opening the skull and removing the laminæ of the vertebræ, the small back-saw, such as is used by carpenters, has no equal. A chisel and mallet are also needed. If a 'coronet' is used to steady the skull cap during removal, the common and faulty pattern should be discarded in favour of one in which the vertical bow is large enough to admit its being firmly grasped by the hand when properly applied to the skull.

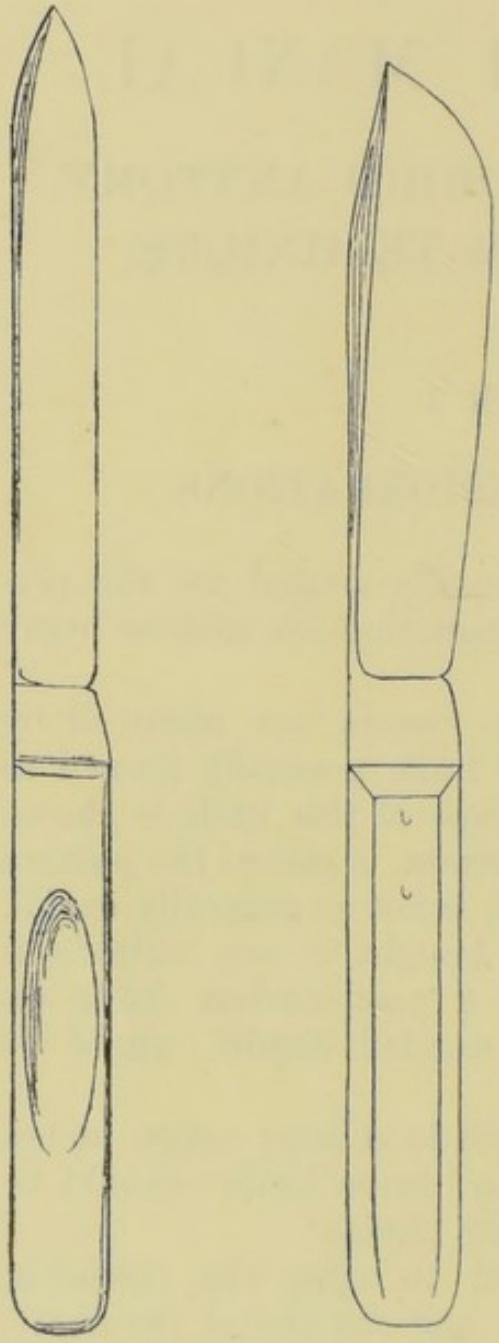

Fig. 1.—Post-mortem Knife and Cartilage Knife.

The use of rib shears facilitates expeditious opening of the chest. The form devised for use at St. Thomas's Hospital is shown in Fig. 3.

Scales and weights, graduated vessels, cones for measuring the diameters of orifices, a tape measure, and a metal catheter or bladder sound should be handy.

Capacious glass bottles provided with stoppers are useful for the preservation of gastric contents, portions of viscera, urine, etc., in cases of suspected poisoning. The specimens should be sealed up as soon as they are obtained.

Needles, twine, sponges, sawdust, and tow are required for the proper restitution of the body.

In certain cases where bacteriological examination is necessary the proper outfit must be provided (lamp, searing iron, platinum wires, scalpel, sterile pipettes, sterile test-tubes, culture tubes, etc.).

Gloves should always be worn by those engaged in post-mortem work. They protect the hands from infection, and have the additional advantage of preventing clinging odours which are so objectionable. Rubber composition gloves,

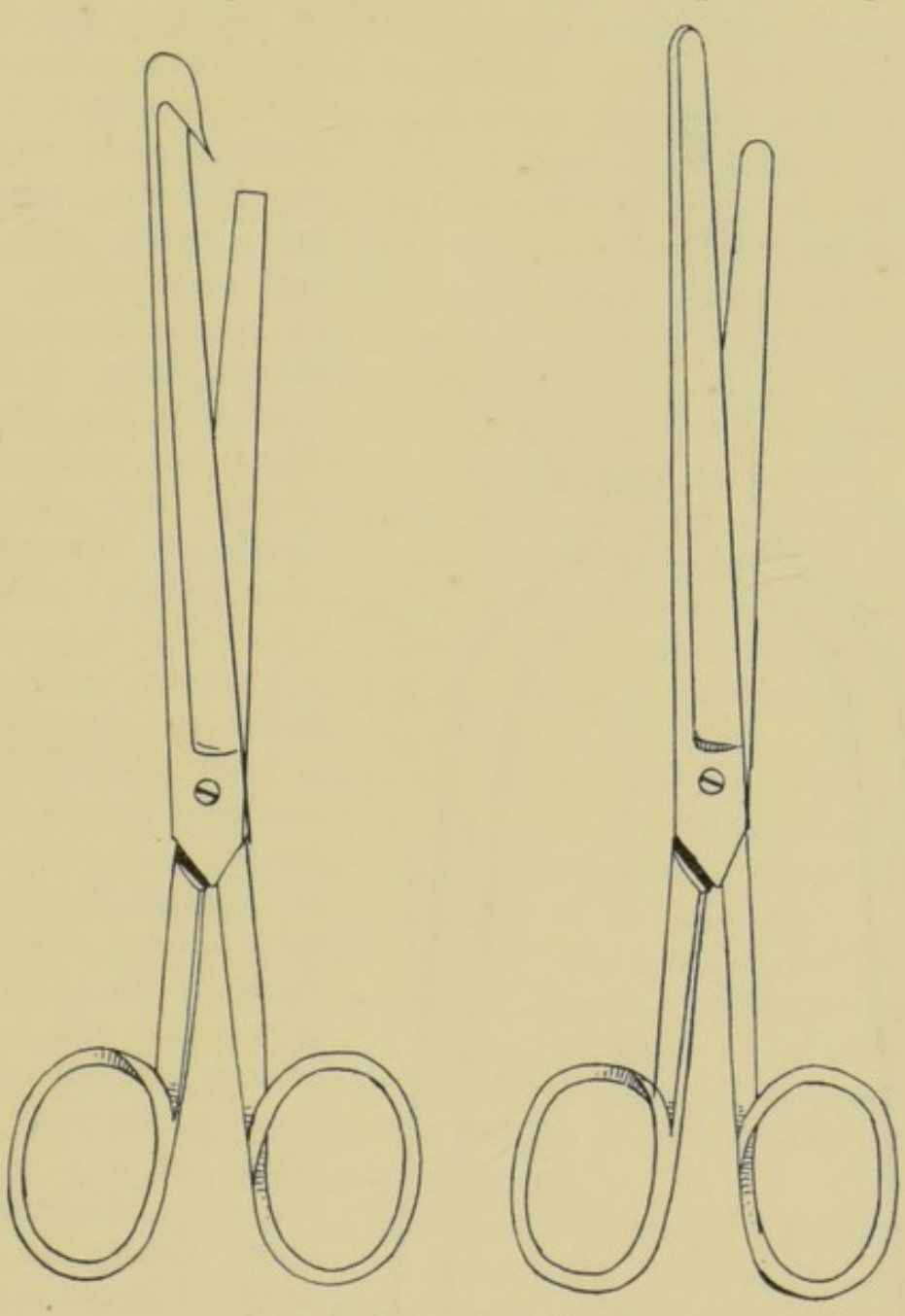

Fig. 2.—Intestine Scissors.

which will stand repeated boiling and are not too thin to stand hard wear, can now be obtained at small cost. A glove which cannot be washed and boiled should be avoided.

In cases where it can be obtained, a Clinical Abstract is most useful as a guide in the performance of a post-mortem

examination. The work of the pathologist is facilitated by such an abstract, and the risk of omitting important parts of the necropsy is reduced to a minimum.

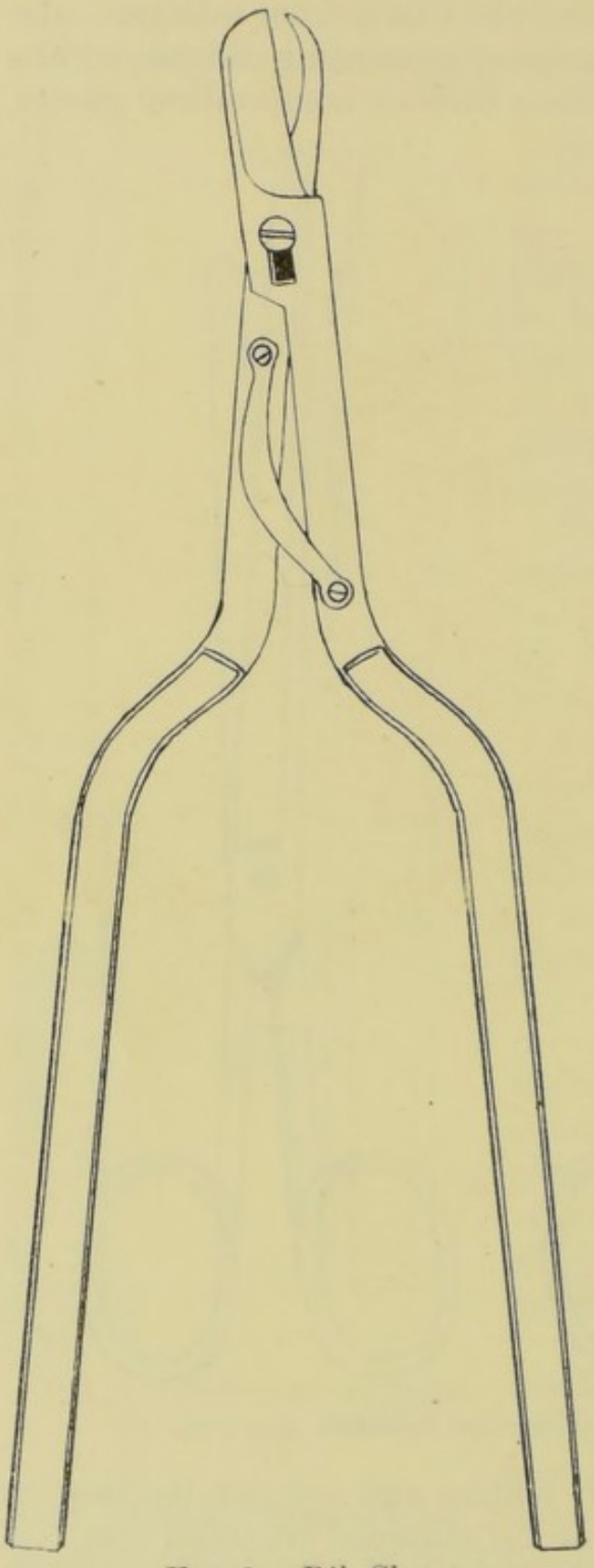

Fig. 3.—Rib Shears.

The abstract, to be useful, should contain the following particulars: name, age, sex, and occupation; date of commencement of illness, date of death—so that the interval which has elapsed between the time of death and time of the examination may be known—names of physicians and surgeons in attendance; a brief outline of the essential points of the history; an account of all abnormal physical signs present, especially those of doubtful import; date and nature of any surgical operations performed; account of any pathological observations made during life, such as blood examinations, investigations of body fluids, expectoration or urine, bacteriological examinations, or examinations of tissues or parts removed; clinical diagnosis; mode of death.

When no abstract of history is available, care must be taken to make the post-mortem examination in the most complete and thorough manner possible.

It is often necessary to make bacteriological examinations after death. For a successful result it is important that the body should be examined with as little delay as possible, and that contamination and exposure of the parts to be thus examined should be carefully avoided. It is best in such cases to arrange for the presence of a skilled bacteriologist, who will direct the necessary proceedings.

In cases which involve medico-legal proceedings the examination of the body must not be made until the formal order of the coroner has been obtained, and any medical men concerned in the case should be given due notice of the autopsy and afforded an opportunity to attend.

For many reasons it is important to carry out the post-mortem examination as soon after death as possible. This is particularly desirable in hot weather, in cases of death from infectious processes, and in cases where lesions of the brain or spinal cord are likely to be present. An interval of twelve hours is ample in all cases. In cases of cord lesion the body should be kept in the prone position after death, and the injection of a formalin solution into the spinal theca is often advantageous, especially in cases where examination is likely to be delayed.

Notes written at the time of the autopsy are much more accurate than those written afterwards from memory, when the opportunity of filling in gaps or confirming details has passed. In medico-legal cases all the medical men present should sign the report, provided they are in agreement as to the facts observed.

Whenever possible the examination should be conducted early in the day, and in a good light. Artificial light is undesirable, and its effects upon colours are very misleading; in particular, jaundice is likely to be overlooked.

Valuable information may be obtained by post-mortem examination even though the body be very much decomposed. Hair resists decomposition, and identity may sometimes be established by its colour or appearance; or by the presence of malformations, and the characters of the teeth.

Abnormalities and injuries of the bones may be detected. It is possible to determine the existence of pregnancy, and often possible to prove poisoning. Foreign bodies, such as bullets, may be discovered, and substances which have entered the body by the various orifices may be found.

EXAMINATION OF THE EXTERIOR OF THE BODY

Before proceeding with the examination, the body should, if possible, be weighed. The height should be ascertained by measuring from the crown of the head to the centre of the external arch of the instep, the foot being at right angles to the leg. It is well to measure, in addition, the breadth of the shoulders and the maximum circumference of the head.

The body must be inspected systematically. For this purpose it should lie fully exposed on a flat table in a good light. Inspection whilst in the coffin is useless. By a rapid general survey determine the configuration, state of nutrition, sex and apparent age. At the same time the occurrence of any abnormal odours should be noticed (acetone, prussic acid, etc.). The presence of parasites on the body may be detected. Then proceed to a more minute and systematic examination of the body from head to foot. First inspect the anterior aspect, not forgetting the inner surfaces of the limbs; then the lateral aspects, including the axillæ and sides of the chest, for which inspection the arms must be abducted; lastly the back, the body being rolled over for this purpose and the trunk and limbs scrutinised from this aspect. Carefully inspect the joints of the limbs and test their mobility. Determine the degree of post-mortem rigidity by manipulation of the extremities, but do not mistake ankylosis for rigor mortis. The necessary manipulations may reveal the presence of fractures or of abnormal mobility from destructive joint changes. Do not overlook deformities

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such as club foot, etc. In cases of death from unknown causes examination of the hands, fingers and nails for cuts, powder marks and foreign material is very important.

The natural orifices and depressions with their contents are to be specially examined. Note the condition of the eyelids, conjunctivæ, corneæ, and irides. Inspect the nostrils; the mouth, including gums, lips, and tongue; the auditory canals; the urinary orifice; the anus and, in the female, the vagina and hymen. The condition of the umbilicus and of the various hernial orifices should be investigated.

The common signs of death, such as cessation of the pulse and heart beat, absence of respiration, loss of the pupil reflex to light, glazing of the cornea, and flaccidity of the globe of the eye, need no comment. In doubtful cases the application of a moderately tight ligature to a finger will lead to congestion if a feeble circulation is still going on, whilst the opening of a superficial artery, such as the temporal, will remove all doubt.

The phenomena of cooling, post-mortem staining, rigor mortis and decomposition, call for more detailed consideration.

Cooling of the Body.—Other things being equal, dead bodies are said to cool at the rate of 1° F. per hour, but the rate is not uniform, being more rapid at first and slower as the external temperature is approached. As a general rule post-mortem cooling requires twenty-three hours for completion. Clothing and fat delay the process; exposure and free hæmorrhage accelerate it. Naturally, cooling is more rapid in bodies immersed in water or kept in a refrigerator. During the period of rigor mortis an elevation of temperature may be noticed, and in consequence of some diseases the temperature of the body may rise considerably after death. Post-mortem pyrexia of this kind has been noticed in cholera, rheumatic fever, strychnine poisoning, tetanus, cerebro-spinal meningitis, and certain cases of apoplexy or of injury to the brain. The temperature should be determined by the use of a thermometer in the axilla and in the rectum—not by touch.

The rectal temperature is ordinarily the same as that of the room in which the body lies at the expiration of forty-two hours from death.

Post-mortem Staining, Cadaveric Lividity, or Hypostasis.—This appears as sharply defined, dull red or purplish patches in dependent parts of the body. Those points upon which the corpse actually rests will not become discoloured, neither will the characteristic lividity appear beneath tight bands or wrappings, although it may become apparent between the folds. Post-mortem stains should not be mistaken for signs of violence. The colour is due to gravitation of blood into the capillaries, and to slight transudation of blood-stained serum. The stains may also appear in internal organs, and be recognisable on the posterior part of the stomach, in parts of the intestines, on the posterior aspects of the lungs, and even of the kidneys, presuming the body to have lain on the back after death.

The more fluid the blood the more marked is the staining. In carbon monoxide poisoning, which may result from the inhalation of coal gas, water gas, or the fumes of burning charcoal or coke, post-mortem stains have a bright rosy tint and are very extensive. During the interval in which the blood remains fluid after death the position of post-mortem stains may be made to alter by changing the position of the body. When bruising and staining coexist in the same regions, advantage may be taken of this fact, for by turning the bruise uppermost the staining may disappear in a few hours.

The following points should be noticed in making a distinction between bruising and post-mortem staining :

1. The position of the stain, whether in a dependent part of the body or not. Post-mortem staining is due to gravitation of blood ; bruises are due to extravasation.

2. The colour changes. Variegation of colour is characteristic of bruises ; colour zones do not surround post-mortem stains.

3. The presence or absence of elevation of the discoloured area above the surrounding surface. Bruises are usually elevated more or less ; post-mortem stains are not.

4. The appearances on incision. In post-mortem staining the blood has not left the vessels, although these may bleed when incised. In bruises blood is effused into the tissues, where it may be found either in a clotted condition or still fluid. Little or no oozing occurs on incision of a bruise.

Rigor Mortis.—As a rule this commences three or four hours after death, and is complete in another three hours. Its average duration is from twenty-four to thirty-six hours in summer, and from thirty-six to forty-eight hours in winter. Of the external muscles those belonging to the neck and lower jaw are first involved, then in turn the muscles of the face, trunk, arms and legs. The presence of rigor mortis in the facial muscles induces a facies hippocratica; the fixation of the mandible may give rise to difficulty in opening the mouth for examination; the presence of rigor mortis in the arms may be concealed by forcible straightening in removing the body to the post-mortem table, but even then it may still be noticeable in the firmly flexed fingers; rigor mortis in the lower limbs produces rigid extension and some degree of pes cavus.

The involuntary muscles enter into rigor mortis before the voluntary ones. The eyelids and heart are primarily affected. The heart usually contracts from rigor mortis an hour after death, and the ventricles may thus empty themselves; it remains contracted from twelve to thirty-six hours. The finding, therefore, of ventricles contracted in systole is no proof that this was their condition at the moment of death.

Dryness and cold (winter) as a rule delay and certainly prolong rigor mortis. Warmth and moisture accelerate its appearance and shorten its duration. (Note the analogy to ferment action.) Whatever produces exhaustion of muscles, such as violent exertion or violent and prolonged convulsions, causes rigor mortis to come on suddenly, and usually, but not invariably, to pass off quickly. After death from lingering disease the onset is also very quick, and the disappearance speedy.

Instantaneous rigor mortis occurs occasionally as

the result of sudden and violent death. This accounts for the firm gripping of weapons in the hands of suicides.

Decomposition.—The occurrence of decomposition is a positive sign of death. The first external evidence is a greenish discoloration of the lower parts of the abdomen, which makes its appearance about the third day of death, and has been mistaken for an indication of violence. After the abdomen, the chest and finally the whole body become involved. The superficial veins may be mapped out on the surface as a network of dark lines. After death from drowning the discoloration is said to travel from the head and neck downwards.

Decomposition is accelerated by moist warmth and free access of air. It is particularly rapid in fat bodies, and after death from acute infective diseases.

In the latter stages of decomposition the development of the gases of putrefaction lead to deceptive appearances. Eyes which were sunken may become prominent; the face may become bloated and the tongue swollen and protruded as in suffocation; the uterus may empty itself, and blood be forced out from any open wound. The body may actually change its position in the coffin. In infants the general swelling of the body may lead to an erroneous estimate of age.

A knowledge that putrefactive changes have begun may modify the interpretation of appearances observed in the internal organs.

Discoloration of the mucous membranes of the larynx, trachea, stomach and intestines appears early, and is easily mistaken for inflammation by those not familiar with the appearances produced. The endocardium and intima of the arteries are much stained; the lungs appear unduly crepitant, and the veins contain gas bubbles.

Wounds.—Any wounds found on the body must be carefully examined with regard to the following points:

1. The exact situation of the wound.
2. The direction and extent (length, breadth, and depth).
3. The presence of effused blood and its condition, whether coagulated or liquid.

4. The presence of swelling, bruising, or powder marks in the neighbourhood.

5. The presence of lymph or pus around or in the wound.

6. The condition of the edges of the wound, whether inverted, everted or showing signs of cicatrisation or of necrosis.

7. The presence of foreign substances of any kind.

The extent of wounds must be determined by actual dissection, and not by probing, but a soft bougie may be used as a guide to the investigation.

Although there is but little difference between wounds made immediately before death and those inflicted shortly after, the following characteristics will serve to determine whether a particular wound was inflicted some time before or some time after death occurred:

Wounds inflicted during life.	*Wounds inflicted after death.*
Edges everted. Wound gaping.	Edges in contact.
Free hæmorrhage and effusion of blood in tissues around.	Little or no hæmorrhage, and no surrounding effusion.
Presence of clots in divided or injured vessels.	No clots.
Signs of inflammation, cicatrisation, ulceration or necrosis.	All these signs wanting.

Lacerated or contused wounds inflicted during life do not necessarily lead to much loss of blood.

Bruises.—The distinctions between bruises and hypostases or post-mortem stains have already been given. There is no practical difference between a bruise inflicted immediately before death and one inflicted immediately after.

It is not possible to discuss all the variations from the normal which may be detected on external examination of the body, but the following points are worthy of mention:

The nutrition of the body, as indicated by the presence of corpulence or wasting; the presence of degenerative changes, such as arcus senilis, loss of teeth, greyness or loss of hair.

The presence of marked anæmia, which is very obvious in deaths from blood disease or hæmorrhage, but may also occur in cases of chronic metallic poisoning (lead,

arsenic, mercury), in certain chronic diseases like granular kidney and carcinoma of the digestive tract, or as the result of the presence of certain intestinal parasites (*Ankylostomum duodenale, Bothriocephalus latus*).

The occurrence of marked dusky pigmentation of the skin, which should suggest Addison's disease, pernicious anæmia, Graves' disease, poisoning by arsenic or silver, or even bronzed diabetes.

The mucous membrane of the mouth and gums should be inspected when anæmia or pigmentation of the surface of the body exists. Pigmentation of the mucous membrane of the lips and cheeks may be found in Addison's disease, and sometimes also in pernicious anæmia. The presence of a lead line on the gums may throw much light on diseased conditions. The cutaneous pigmentation of jaundice, or the lemon-yellow tinge of pernicious anæmia, or of acholuric icterus may be overlooked if the examination is conducted by artificial light.

Discoloration of the face, navel, and areolæ of the breasts may be very marked in pregnancy.

Cyanosis of the body, often most marked in the extremities, may suggest lung or heart disease. In cases of marked cyanosis without causative lesions in these organs bear in mind the possibility of sulph-hæmoglobinæmia, meth-hæmoglobinæmia, or poisoning by coal-tar products.

It is necessary to remember the situations and characters of scars due to formal surgical operations on various regions of the body. The detection of vaccination scars may be important, both thighs and arms should be examined. The triradiate scars of leech-bites, the scars (not often seen) of cross-cupping, the segmental scars of shingles, and the pitted scars of small-pox, are very distinctive. The corona veneris of syphilis, and the destructive ulceration of the nose due to lupus or syphilis, will attract due attention.

Tattoo marks may be valuable aids to identification. Hypodermic punctures from self-medication are usually situated on the flexor aspects of the arm or thigh ; they may appear as indurated papules or even abscesses.

Lineæ albicantes on the abdomen are evidence of

previous abdominal distension, often, but not necessarily always, due to past pregnancy. Similar striæ may occur on the thighs or breasts after absorption of excessive fat.

The presence of subcutaneous œdema and its distribution should be carefully noted, as also should the presence or absence of pressure sores.

In corrosive poisoning burns may be present on the face and lips, also in some cases on the hands. Notice the colour of the burns, this may indicate the nature of the corrosive.

The rashes of the specific fevers and skin eruptions of the erythematous type may completely disappear after death, but vesicles, bullæ, pustules, punctate hæmorrhages, pigment spots, exfoliating lesions, and ulcerations persist.

Congenital malformations should always be noted if present. The more common ones are small dermoids near the outer end of the eyebrow, hare lip, cleft palate, deformities of the external ear, supernumerary digits, club foot, and malformation of the external genitals.

Hairy moles and nævi should not escape attention.

Herniæ and enlarged glands may be obvious.

The presence of parasites on the surface of the body should be noted. Head lice forsake the hair and run down over the body after death.

Abnormal odours, such as those of acetone or prussic acid, may lead to important conclusions.

CHAPTER II

METHOD OF OPENING THE BODY

EXAMINATION OF THE MOUTH, NECK, AND THORAX

Take up a position on the right side of the body. The head of the corpse should be extended and the chin steadied with the left hand. Grasping the post-mortem knife firmly in the palm of the right hand and cutting with the belly, not with the point, of the knife, make a median incision from the chin to the pubes (Fig. 4). This incision should commence an inch below the symphysis menti and be carried down the mid-line of the neck, dividing the skin and subcutaneous tissues. From the manubrium to the ensiform cartilage the incision should at once be carried down to the bone, but from the ensiform cartilage to the pubic symphysis again only the skin and subcutaneous fat should be divided. The incision should deviate slightly to the left of the umbilicus but regain the mid-line below it, the object being to avoid injury to the round ligament of the liver.

The upper part of the abdominal incision, for the extent of two inches, is cautiously carried deeper through the whole thickness of the abdominal wall. If the presence of gas within the peritoneal cavity is suspected, water may be poured into the small pouch thus made, before the peritoneum is incised,then on puncturing the latter at the bottom of the water, gas will be seen to escape in bubbles. Even apart from this precaution the hiss of the escaping gases may be noticeable when the body cavity is opened.

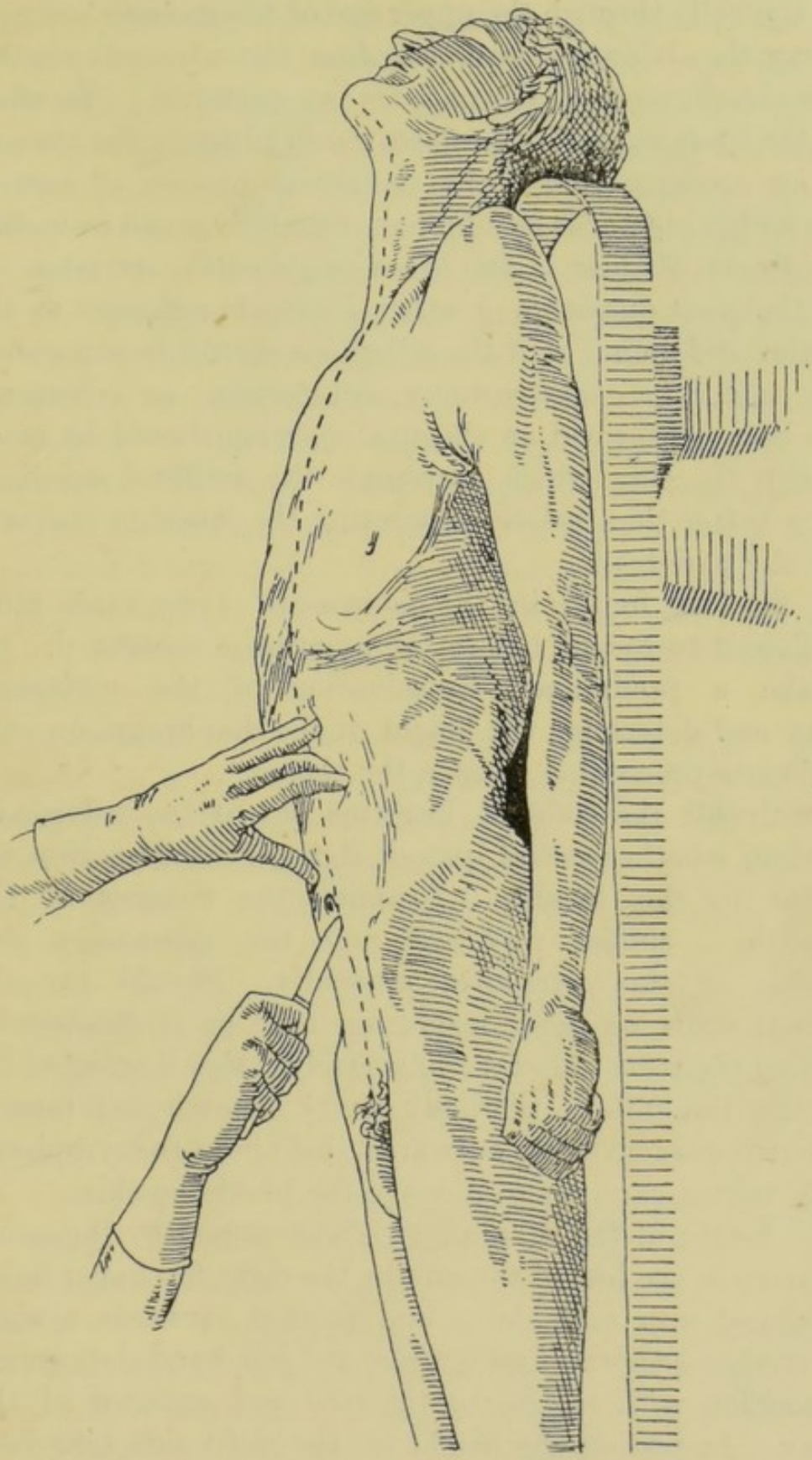

FIG. 4.—Preliminary Incision.

The index and middle fingers should now be inserted into the belly through the upper end of the incision and used to draw the abdominal wall away from the subjacent viscera, while the division of the parietes is completed. To effect this the knife is used edge downwards towards the viscera. On no account should the dangerous process of cutting from within outwards be adopted. Carefully avoid wounding the urinary bladder in the lower angle of the incision.

If the great omentum or other parts are adherent to the anterior abdominal wall the adhesions should be separated.

In cases where gastrostomy, enterostomy or colostomy have been performed an elliptical incision should be made through the skin which surrounds the artificial opening; this is better than merely separating the bowel or stomach from the parietes.

If fluid is found in the peritoneal cavity none must be allowed to escape before the amount is measured.

Make a preliminary examination of the peritoneal cavity and determine the height of the diaphragm on each side before proceeding to open the thorax.

Gently lift the coils of intestine out of the pelvis and ascertain whether serum, pus or signs of inflammation are present in this locality. Examine the recesses of the flanks in a similar way and note the appearance and position of the vermiform appendix. Should the abdominal walls be rigid, inspection may be facilitated by dividing the recti muscles transversely; this is effected by inserting the knife in the body cavity beneath each muscle and cutting transversely outwards, just above the symphysis pubis, taking care not to damage the overlying skin.

The height of the diaphragm is ascertained by bimanual palpation in the line of the nipple, the right forefinger being introduced into each dome and pressed forwards against the overlying thoracic wall whilst the left hand determines its position with regard to the ribs and exterior of the thorax. In testing the height on the right side take care to pass the fingers to the right of the falciform ligament.

The normal upward extent of the diaphragm is to the fourth rib on the right side and the fifth rib on the left.

The soft parts are now to be reflected from the front of the chest. In stripping the right side the operator should stand on the right side of the abdomen, facing the head; in stripping the left he stands to the left side of the thorax, facing the feet. Forcibly evert the upper part of the abdominal wall of the right side by hooking the left thumb into the upper part of the incision, grasping the wall between thumb and forefinger and pulling it upwards and outwards. The rest of the left hand should be firmly pressed against the outside of the lower part of the thorax during this manœuvre. The lower costal margin being thus rendered prominent beneath the peritoneum in the subcostal angle, the knife is drawn along it from the lower end of the sternum for several inches, fully exposing the bone and cartilages (Fig. 5). The costal margin on the left side is exposed in a similar manner.

Commencing over the cartilages and sternum thus exposed, the front of the chest is rapidly and completely cleared of its soft parts by bold sweeps of the knife. The tissues should be forcibly pulled away from the chest wall so that the layer of areolar tissue which connects them to the latter comes into view. It is in this layer that the separation is effected. No muscle must remain on the cartilages or bones, and some care is necessary to cleanly reflect the origins of the pectoral muscles. The denudation of the thorax should be carried as far outwards as the mid-axillary lines.

If necessary, the test for pneumothorax is applied at this stage. The soft coverings are supported at the side of the thorax so that a pouch is formed between them and the bony wall. The pouch is filled with water and one of the upper intercostal spaces, below the water level, punctured with a scalpel or trocar. Pressure is made from within the belly on the under surface of the proper half of the diaphragm, and if air is present in the pleural sac a stream of

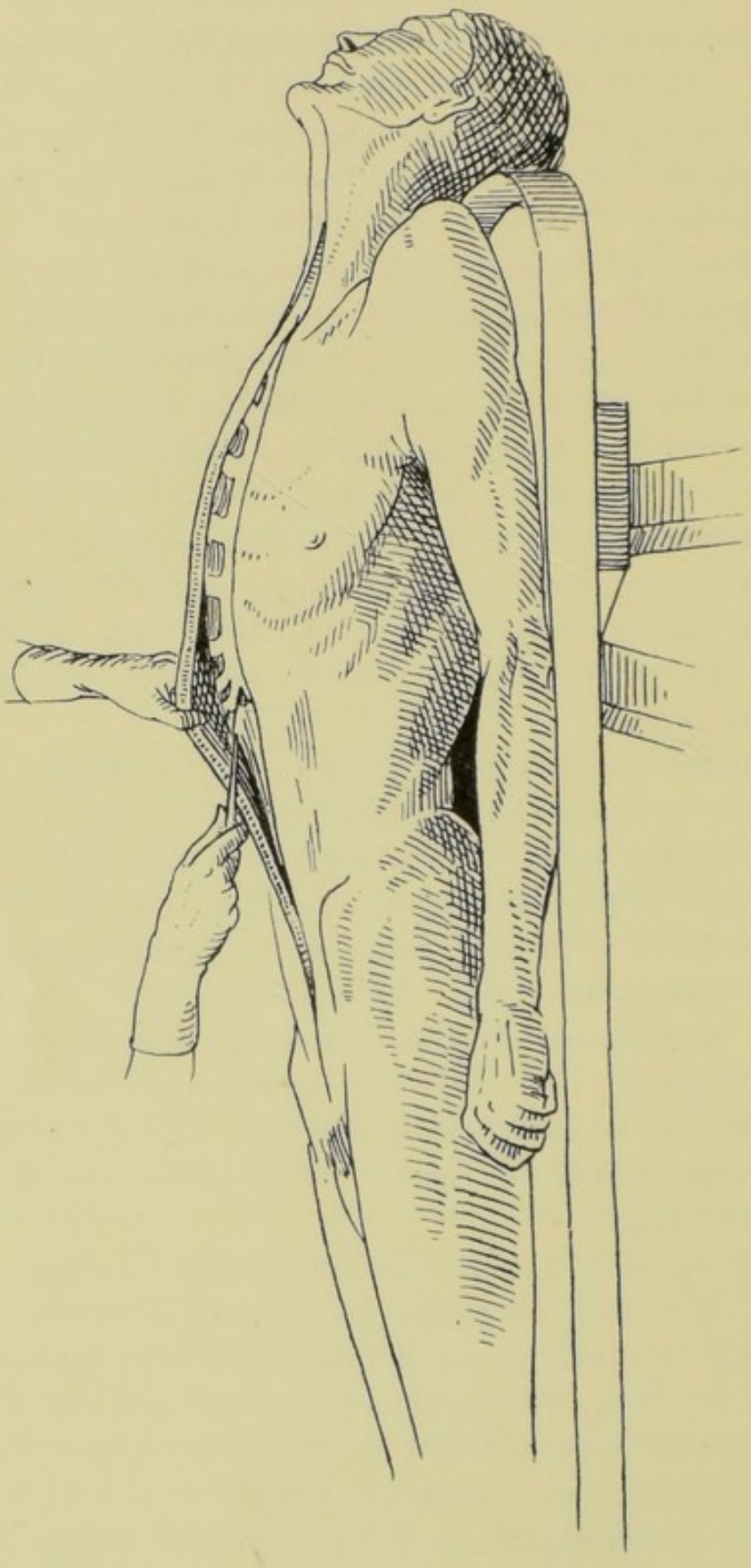

FIG. 5.—Reflexion of Thoracic Coverings.

bubbles will rise from the puncture. If the air pressure in the thorax is greater than that of the atmosphere no counter-pressure on the diaphragm is necessary.

EXAMINATION OF THE STERNUM AND COSTAL CARTILAGES

Before the thorax is opened the exposed costal cartilages and sternum should be inspected.

The angle of Ludwig—at the union of the manubrium with the body of the sternum—may be unduly prominent in emphysema.

Subjacent aneurysms or growths may cause irregular projections of the upper costal cartilages, or flattening may be noticeable as the result of chronic phthisis.

Gummatous infiltration of the periosteum of the sternum sometimes occurs.

Fractures of the sternum are uncommon.

There is an imperfect joint between the manubrium and the body of the sternum in which suppuration may occur in pyæmia, and which is sometimes separated by aneurysms on their way to the surface.

Congenital malformations of the sternum in the shape of fissures and perforations may be present.

Abnormalities of the rib cartilages in the form of fissures and bifurcations have no pathological importance.

The costal cartilages are often calcified in old subjects, but not necessarily so. The calcification may be limited to the cartilages of the first ribs, it is said to be common in phthisis.

Dark pigmentation (inky staining) of the cartilages has been noticed in ochronosis, and in such cases the urine may also darken on exposure to the air, indicating alkaptonuria.

There is normally a slight swelling at the junction of rib with its cartilage, and this must not be taken as evidence of rickets. In rickets the beading is well marked and most prominent, as a rule, on the inner aspect. The changes may be demonstrated by removing a segment of rib with its costal cartilage attached, and splitting the whole

longitudinally. The irregular ossification of the epiphyseal cartilage is then evident. Confirmatory evidence may be sought in the epiphyses at the wrists and ankles. Delayed dentition and undue persistence of the cranial fontanelles point in the same direction (see p. 275).

Fractures at the junctions of the ribs and cartilages with periosteal hæmorrhages may occur in scurvy.

EXAMINATION OF THE MAMMARY GLANDS

Both glands should always be examined. First inspect them, noticing their contour, size, and superficial appearances.

The nipples should be scrutinised for the presence of retraction, fissuring, ulceration or eczema. The colour and appearance of the areolæ demand attention.

Examine the skin for the presence of dilated veins, inflammatory changes (lymphangitis), or the thickening and dimpling (pig-skin appearance) which is evidence of subjacent malignant disease. The presence of fistulæ and abscesses should not be overlooked.

Carefully palpate each breast, first by pressing it backwards against the ribs and then by compressing it between the thumb and fingers. The presence of indurations, growths or cysts may thus be detected.

Squeeze each gland towards the nipple and note the character of any exudation which may occur, whether opalescent, milky, purulent or blood-stained. Human milk is a clear, bluish liquid; colostrum is viscid and may have a yellowish tinge, but is easily distinguished from pus by its microscopical appearances.

The exposed surfaces of the breasts should be as little mutilated as possible, but if incisions have been made during life these may be opened up and utilised for the purpose of further examination.

The proper incision for post-mortem examination of the breast is made from the deep surface of the reflected thoracic flap and carried through the base of the gland towards

the nipple without implicating the skin. Other parallel incisions should expose any indurated areas or swellings.

The breast having no capsule, peripheral portions of the secreting substance extend widely in the para-mammary fat. The arrangement and extent of the parenchyma can be investigated macroscopically by treating the tissue with a 5 per cent. solution of nitric acid. If slices of the fresh gland be placed in this solution and then washed under running water the lobules or clusters of acini will appear as small opaque masses (Stiles).

After incising the breast, attention should be directed to the axillary, clavicular, and retrosternal lymphatic glands. The axillary glands may be examined and, if necessary, removed, by fully reflecting the thoracic flap towards the axilla ; an external incision can thus be avoided.

Quite apart from disease the appearance of the breast varies with its functional activity (lactation), and with age.

In the quiescent, non-lactating condition the fully developed mammary gland presents, on section, a whitish corpus mammæ, which consists of a firm stroma in which a few inactive acini can be recognised as greyish red or brownish points. There is often an abundance of yellow fat around the gland, and in the stroma.

In the mid-months of pregnancy the corpus mammæ has a softer, somewhat œdematous appearance, and the scattered acini can be more clearly recognised. These acini rapidly increase in size, and an opalescent fluid can be squeezed from the nipple.

During lactation the fully developed acini form the bulk of the corpus mammæ, so that the latter assumes a reddish, granular appearance. A glairy or milky fluid exudes from the cut surfaces on compression.

In old age the breasts atrophy. The glandular substance disappears and may be replaced by fat. The stroma of the corpus mammæ become harder, and some of the milk ducts may form involution cysts. The condition is bilateral, and a microscopical examination will serve to exclude the presence of carcinoma.

Developmental Anomalies of the breasts, such as polymastia, are of no great pathological importance.

Hypertrophy of the Breasts is a rare affection. The increase in size may be due to diffuse fibromatosis only, or the parenchyma may share in the overgrowth.

Acute Mastitis.—Acute inflammation of the breasts is mostly associated with pregnancy and lactation, but sometimes it occurs in the newly born. Abscess formation is a common result, and it may be possible to express pus from the nipples. Pus may be distinguished from milk by its viscidity and yellow colour. Look for fissures or excoriations of the nipples, lesions which may serve as portals for infection.

If an abscess is found in the breast or its neighbourhood determine the exact site. Suppuration may occur in the superficial structures (lymphangitis), in the glandular tissue, between the breast and the pectoral muscle, or beneath the latter. The deep-seated abscesses may be independent of disease of the breast, and arise in connexion with the ribs or other structures in the neighbourhood.

Chronic abscesses in the breast tissue may complicate lactation or be tuberculous in origin. The contents of some mammary cysts, being greenish and viscid, may simulate pus.

Chronic Mastitis may occur in a diffuse or a localised form.

Diffuse chronic mastitis is a rare condition. There is widespread induration of a considerable area of the gland and, when incised, the breast proper is found to be represented by a compact mass of dull-white, tough, fibrous tissue, which lacks the inelastic hardness of carcinoma. The axillary glands are normal, or, at most, slightly enlarged. A microscopical examination is requisite to confirm the distinction from carcinoma.

Disseminated lobular mastitis appears in the form of ill-defined, indurated sectors. Small tense cysts are scattered throughout these, and the inflammatory changes may also lead to the formation of small fibro-adenomatous nodules. Some of the cysts may contain delicate papillary projections.

Tuberculosis of the Breast.—Tuberculous disease of the mamma assumes the form of a chronic mastitis, which often ends in suppuration. On section, disseminated, nodular, cheesy masses or chronic abscesses in the midst of caseous indurations may be found. Tuberculous sinuses may have formed, and the axillary glands may be infected.

Tuberculosis of the breast is generally preceded or accompanied by tuberculous disease of the lungs, pleuræ, glands, bones, or joints.

Actinomycosis of the Breast is rare. Its distinguishing characteristics are the presence of chronic inflammatory induration, with involvement of the skin and sinus formation. The characteristic organism must be detected by microscopical examination and culture.

Fibro-adenomata of the Breast occur as circumscribed, encapsuled tumours which show no signs of invading the breast tissues, and give rise to no metastases.

Sarcoma of the Breast.—True mammary sarcoma is uncommon. The tumour is well circumscribed and does not give rise to adhesion of the skin or retraction of the nipple. Over large, rapidly growing tumours, however, the skin sometimes gives way, and the sarcoma fungates. Infection of the axillary glands is late.

Spindle-celled sarcoma is the harder variety and on section may present a pinkish fibrous appearance. Round-celled sarcoma is much softer, and the centre of the tumour tends to degenerate into a cyst from hæmorrhage and softening.

A microscopical examination is necessary to confirm the nature of the growth.

Carcinoma of the Breast.—This, the most common malignant disease of the mamma, may occur in a circumscribed or a diffuse form.

The circumscribed carcinomatous tumour is extremely hard (scirrhus) and creaks under the knife. The cut surface retracts and becomes slightly concave. The central part of the neoplasm is white and mottled with yellow fatty points, thus presenting the 'unripe pear' appearance; the peripheral part is more translucent, often softer, and may

be of a greenish or pinkish colour. On scraping the surface of the growth a milky fluid, known as cancer juice, exudes. The tumour is never encapsuled, and the growth sooner or later becomes adherent to the adjacent structures. Adhesion to the skin produces puckering and induration; superficial carcinomatous ulceration may ensue. Retraction of the nipple depends on proximity to it of the growth.

Carcinoma may also occur in a more rapidly growing form with superficial redness and œdema, which simulates acute inflammation. The new growth in such a case is softer and often pinkish in colour. Rapidly growing cancer of this type may occur in association with pregnancy and lactation.

Carcinoma of the breast tends to invade the axillary, clavicular, and retrosternal glands. Tracks of malignant infiltration occur in the skin and connective tissues about the breast, and in this way the growth may extend towards the pectoral muscles, the epigastrium and peritoneum, or elsewhere.

Secondary growths may occur in the liver, pleuræ, lungs, bones, brain, ovaries, or opposite breast. Of the bones, those of the cranium, spine and pelvis may be involved; also the femur, the humerus, and the ribs.

Duct Papillomata appear as small, pink, raspberry-like growths with slender pedicles. The small tumours lie in smooth, cystic spaces derived from dilated ducts. Papillomata are often multiple, but one may exceed the others in size. There is no evidence of invasion of the breast tissue or of the lymphatic glands.

Duct Carcinoma.—The papillary tumour which constitutes this affection may be hard or soft, the former variety being firm and greyish, the latter pink and friable. Invasion of the cyst wall and breast tissue with implication of the lymphatic glands are the criteria of malignancy.

Cysts of the Breast.—These are of various kinds, and may be classified as (*a*) Simple serous cysts; (*b*) Involution cysts; (*c*) Multiple cysts, the result of chronic lobular mastitis; (*d*) Cysts containing intra-cystic growths of the nature of papillomata or carcinomata. (*e*) Galactoceles or milk cysts. (*f*) Hydatid cysts. The last are rare. The

contents of cysts, if greenish and viscid, may be mistaken for pus. Microscopical examination of the cyst wall may show a true epithelial lining, and so differentiate the cyst from an abscess.

REMOVAL OF THE STERNUM AND COSTAL CARTILAGES

The thorax is now to be opened by cutting through the costal cartilages just internal to their junctions with the ribs. A small cap of cartilage should be left on each rib to obviate risk of scratching the wrist or arm during the subsequent stages of the autopsy. The cartilages should be divided by cartilage shears, the division being commenced below, near the nipple line, and carried upwards. The beak of the shears should be passed under each cartilage from below and the division effected by a sharp snap of the handles. The cartilage of the first rib should be divided far outwards, well beyond the edge of the manubrium.

In children the cartilages may be cut with stout scissors or even with a post-mortem knife, the edge of which should be directed obliquely outwards and backwards.

Having completed the cutting of the cartilages, disarticulate the sterno-clavicular joints. The subjacent innominate veins are liable to be wounded during disarticulation, so it is better done after division of the cartilages, when the pleuræ can at once be inspected. Blood from the punctured veins runs into the pleural sacs, and delay may render it difficult to say whether the blood in the sacs is derived from the veins or due to antecedent injury or disease, such as, for instance, fractures of the ribs, tears of the lungs, rupture of aneurysms, or perforating wounds.

Disarticulation of the sterno-clavicular joint is at times difficult owing to the angular shape of the joint surfaces

and the expansion of the inner end of the clavicle. A knife with a stiff blade and not too broad should be chosen. The line of the upper border of the joint is determined by the finger, and the knife, gripped firmly in the hand, is carried vertically downwards through the joint towards its lower end; the knife is then turned sharply outwards and carried a short way along the under surface of the clavicle, to divide the rhomboid ligament. The first part of the incision should include in its sweep the sternal tendon of the sterno-mastoid muscle. When the joint is satisfactorily disarticulated the head of the clavicle springs forward and away from the manubrium. The interior of the joint must be inspected. It is occasionally the seat of suppuration in pyæmia or may show changes due to osteo-arthritis.

The sternum and costal cartilages may now be lifted and removed. Proceed as follows:

The attachments of the diaphragm to the lower costal cartilages are first divided. The knife blade should be inserted on the flat beneath the cut surfaces of the lower cartilages and pushed on, parallel with the chest wall until its point appears in the epigastric angle. The knife is then carried downwards close to the back of the cartilages until the insertions of the diaphragm are cut through.

Whilst carrying out this manœuvre on the left side care should be taken to avoid wounding the pericardium.

The lower end of the sternum must next be lifted and, by transverse sweeps of the knife, separated from the tissues behind it. The edge of the knife should be turned towards the back of the bone and the blade in its upward passage carried through the gaps already made in the costal cartilages by the shears. There is a great tendency to cut the cartilages a second time or run the knife off into the interchondral spaces towards the sternum. Finally the sternum is drawn upwards and slightly twisted laterally so that it may be completely detached by a touch or two of the knife

at the back of the sterno-clavicular joints and by division of the sterno-hyoid and sterno-thyroid muscles.

If the first cartilage has not been completely divided before the sternum is turned upwards the latter is apt to be fractured through the sterno-manubrial joint.

EXAMINATION OF THE ANTERIOR MEDIASTINUM

Inspect the structures exposed by removal of the sternum and costal cartilages.

Mediastinal Emphysema appears in the form of bubbles of air or of gas in the mediastinal connective tissue. It is often seen in cases of diphtheria, which have proved fatal after tracheotomy. Sometimes it occurs as the result of decomposition, and, more rarely, is associated with pneumothorax, or a communication between the interior of the lung and the mediastinal spaces, such as may result from gangrene or abscess. Perforation of the trachea or rupture of the œsophagus may also give rise to emphysema of this kind. Emphysema of the superior mediastinum may accompany emphysema of the neck or of the superficial tissues of the body.

Acute Inflammation of the Mediastinum, terminating in abscess, is rare. Occasionally it is found in association with pneumonia, pleurisy, or pericarditis. It may also occur in certain infective fevers, or in consequence of injury. Sometimes it has extended from the neck, as a sequel to inflammation around a tracheotomy wound or to submaxillary angina.

Chronic Inflammation of the Mediastinum shows itself as an adhesion of the pericardium to the back of the sternum and the costal cartilages. It may be associated with adhesion of the pericardial sac to the heart, and of the lungs to the chest wall.

Anterior Mediastinal Glands.—The glands which lie alongside the internal mammary arteries are occasionally caseous as the result of tuberculosis. They may

be infiltrated with white growth, secondary to cancer of the breast. (For further examination see p. 92.)

EXAMINATION OF THE PLEURÆ

The pleuræ should be examined as soon as exposed. Samples for bacteriological or cytological examination should be collected at once, or taken by aseptic puncture before the chest is opened.

Delay in inspection allows blood to trickle in from wounded veins and pus or ascitic fluid to run in from the abdominal cavity. Or a pleural effusion may overflow into the abdomen and part be lost. The practical rule for examination of the great serous cavities is : First inspect the peritoneum, this should be done as soon as the abdomen is opened ; next examine the pleuræ, without delay, as soon as the chest is open ; lastly, examine the pericardium.

The examination for air in the pleura must be carried out before the thorax is opened. The method has already been described (p. 17).

If the sternum and costal cartilages have been removed carefully, the costo-mediastinal reflexions of the pleuræ may be still intact, but are easily torn open.

The flaps of integument having been folded over the cut edges of the costal cartilages to protect the wrist from scratches, the hand should be swept over the whole of the lung. If the pleural sac is free from adhesions the lung can next be drawn forward towards the mid-line, turning on its root as on a hinge, and the deeper parts of each cavity fully inspected, attention being directed to the appearance of the serous membrane and the condition of the ribs and of the thoracic aspect of the diaphragm.

If fluid is present its characters should be noted and its quantity determined. The presence of loculi or of collections in the lung fissures should not escape attention.

Adhesions between the parietal and visceral layers of

the pleura are very common. Their standing is roughly gauged by their strength. Very recent adhesions are soft and friable, older adhesions are firmer and tougher, very old adhesions may have a consistence which is almost cartilaginous.

In cases of universal and firm adhesion, the lung is extricated with the greatest difficulty and is very likely to be lacerated in the process. It is only advisable to use the knife for the separation of adhesions as a last resource. Often it is possible to break down the adhesions to the thoracic wall from before backwards and to peel the lung off the diaphragm by defining the thin anterior border of its base and starting from this point. Attempts to separate the base from behind forwards nearly always lead to lacerations.

Another method of dealing with universal adhesions is to peel the parietal pleura from the wall of the thorax, thus removing the whole pleura with the lung. The separation should be commenced in front, close to the cut surfaces of the costal cartilages, and the adherent portion of the diaphragm removed with the lung. The knife is necessary for this.

The final step in the examination of the pleuræ is to search for growths, abscesses or fractures of the subjacent ribs. The site of a fracture is often indicated by an effusion of blood beneath the costal layer of the sac. First palpate the suspected rib, then pass a knife along its borders to free it from its intercostal attachments. The rib thus becomes movable and is easily examined. Fractures close to the costal cartilages and also those close to the vertebræ are apt to be overlooked. Costal new growths may be examined by removal and section of the rib.

Subpleural Ecchymoses and Extravasations of Blood.—Where death has occurred from asphyxia, or where an asphyxial condition has resulted from convulsions, small hæmorrhagic spots and splashes may be found

scattered over the parietal and also the visceral pleuræ. Similar petechiæ occur in certain blood-diseases such as scurvy, purpura, leukæmia, and septicæmia.

Larger effusions of blood, showing darkly through the visceral pleura, may be seen in cases of hæmoptysis where the alveoli are full of aspirated blood, or may indicate the presence of pulmonary infarcts.

Blood may be effused beneath the mediastinal pleura in large quantity in consequence of rupture of an aneurysm or perforation of the aorta by a foreign body.

Blood beneath the costal pleura is often an indication of the presence of rib fractures.

Acute Pleurisy.—If the surface of the pleura, instead of shining, is dull and sticky, and especially if it is coated with a delicate pellicle which is easily stripped off, acute pleurisy is present. Sometimes, in association with pneumonia, the plastic layer is of considerable thickness.

Miliary Tubercles, if present, may be recognised as minute glistening bodies the size of millet seeds or smaller. If of some standing they may become opaque and be surrounded by a zone of pigment. They are plentiful in cases of general tuberculosis, and are to be seen in small clusters near tuberculous deposits in the lung. Small plaques of new growth, which sometimes occur in disseminated carcinoma, are larger, flatter, and more irregular than tubercles, for which they may be mistaken. These growths favour the upper aspect of the diaphragm and the pleura which covers the ribs and chest wall. A fringe of minute capillaries is sometimes present in the nodules of new growth, never in tuberculous deposits.

Serous Effusions.—A quantity of clear yellow serum with no evidence of pleural inflammation may be part of the general œdema of cardiac or renal disease. As a rule the effusion of cardiac disease is more copious in the right sac than in the left. Serous effusions may be due to tuberculosis.

A small quantity of fluid is often found in the pleuræ when intravenous saline infusion has been practised shortly before death. Quite apart from any of these causes a few drachms of fluid are often found, the effusion being, in all probability, the outcome of slow cardiac failure.

Blood-stained Effusions.—Wound of a large vein during removal of the sternum may allow blood to trickle into the pleural sacs. Slightly blood-stained effusions, small in quantity, are quite common in patients who have died from septic or infectious disease, and also in bodies which are decomposing. Chronic tuberculous pleurisy or malignant disease of the pleura may also give rise to bloody effusions. Similar effusions may occur in chronic renal disease.

Pure Blood in the pleural sac is derived from wound of the chest wall, rupture of a thoracic aneurysm, laceration of the lung, or other injury.

Empyemata.—It is important to determine whether purulent effusions are free, encysted, or loculated. Interlobar empyemata may lie concealed in the pulmonary fissures. Empyemata may be primary, secondary to lung disease, or due to extension of infection from contiguous structures. All the boundaries of the pleural sac, including the diaphragm and visceral pleura, should be inspected carefully in a case where pus is found in the pleural cavity. A bacteriological examination should be made.

Pleural Adhesions.—Recent acute pleurisy may be accompanied by soft, buttery adhesions. Old adhesions at the apices, due to tuberculosis, are exceedingly common, and even in their absence an apical scarring of the visceral pleura, due to the same cause, may often be found. Adhesions limited to the lower lobes may result from past pleurisy or pneumonia, or be associated with bronchiectasis or pulmonary fibrosis. Basal adhesions may be due to an inflammatory condition which has extended through the diaphragm ; they may accompany cirrhosis of the liver, or result from sub-diaphragmatic abscess.

Adhesion of the mediastinal to the visceral pleura may occur as the result of aneurysm of the aorta, disease of the œsophagus, or inflammation of the mediastinal glands : inflammation of the pericardium may also give rise to pleural adhesions. Universal pleural adhesion may be associated with chronic interstitial pneumonia, chronic tuberculosis, widespread saccular bronchiectasis, old empyema, or chronic inflammation and induration of all the

mediastinal tissues, together with adhesion of the pericardium (**chronic mediastinitis**).

When pleural adhesions are dense and thick a careful examination may possibly reveal evidence of chronic tuberculosis in the form of caseous masses. Calcareous plates or loculi of inspissated pus are sometimes found in the midst of old adhesions.

EXAMINATION OF THE PERICARDIUM

The pericardium should be examined immediately after the pleuræ.

Adhesions between the pericardium and the chest wall should not be overlooked, they will have been cut through when removing the sternum and costal cartilages. The sterno-pericardiac ligaments and the mediastinal tissues in front of the pericardium, being lax and of no great strength, are unlikely to be mistaken for pathological adhesions. The latter when present are usually close and firm. External adhesions may involve the whole anterior surface of the sac, the lungs being retracted, or may be limited to that part of the pericardium which is normally uncovered by lung and lies in relation with the lower part of the sternum and the cartilages of the fourth, fifth and sixth ribs of the left side.

The presence of adhesions between the pericardium and the apposed surfaces of the lungs should next be investigated. The phrenic nerve may be involved in adhesions in this situation.

If the anterior margins of the lungs have become adherent to the front of the pericardial sac, the lung edges must be defined and the adhesions separated before the sac is incised.

The pericardium should not be opened before the examination of the pleural cavities is completed, or the latter may be contaminated with fluid which escapes from the former. Where a bacteriological examination of the pericardial contents is contemplated the sac should

be well seared, punctured through the seared area with a sterile knife, and the platinum wire loop carefully introduced to obtain the requisite material.

At the same time it may be desirable to collect a sample of fluid from the most dependent part of the sac for cytological investigation.

To open the pericardium pinch up a fold of its anterior wall, near the right border, and cut into this with knife or scissors. Prolong the incision upwards along the tubular sheath which invests the great vessels. Make a second cut from the lower end of the first, towards the heart's apex. If a knife is used to make these incisions the sac should be hooked away from the surface of the heart by introducing the fingers, and the knife used with its cutting edge directed towards the interior, so that the sac is cut from without inwards.

No pericardial fluid should be allowed to escape before it is measured.

If pure blood is present in the sac, the aorta, sinuses of Valsalva and heart walls must be examined for perforations or rents. If pus is present the surfaces of the heart should be inspected for small abscesses which may have ruptured; any evidence of infection of the sac from without should not escape attention.

If a culture from the blood of the heart is desired it should be made at this stage, before the heart is handled. The surface of the right ventricle should be widely seared, punctured with a sterile knife, and the platinum wire loop introduced.

When internal pericardial adhesions are present there is a danger of incising the heart in the attempt to open the pericardial sac. Difficulty in picking up a fold of pericardium should arouse suspicion, which the exposure of reddish muscular tissue by incision will verify. A plane of cleavage between the adherent layers can usually be found and separation effected by the hand, but in shelling out the heart there is always a risk of tearing its substance, particularly the walls of the auricles,

The thickened pericardial layers should always be examined for evidence of tuberculosis either in the form of caseous deposits or miliary tubercles; the latter may be concealed beneath fibrinous exudation.

If healthy, the inner aspect of the parietal pericardium is smooth, glistening, slightly moist and of a bluish-white colour.

To permit inspection of the back of the sac the heart should be lifted up by its apex. Attention should be directed to the space between the entry of the pulmonary veins (sinus obliquus) and to the tubular investment of the aorta and pulmonary artery. Where pulmonary embolism or pulmonary thrombosis is suspected, and in all cases of death from unknown cause, the right ventricle and pulmonary artery should be examined *in situ* before the thoracic viscera are disturbed. Further details will be found on pages 67 and 68.

Milk Patches.—Patches of milky white opacity on the visceral pericardium are due to localised thickenings. Their usual situation is the anterior surface of the right ventricle, subjacent to the sternum. They are due to pressure or friction, and are common in hearts which are enlarged.

Hæmorrhages in the form of spots or splashes occur beneath the pericardium under circumstances similar to those which determine their appearance in the pleura.

Miliary Tubercles are sometimes seen in cases of general tuberculosis. They may be minute and translucent or aggregated into larger patches, and opaque or caseous.

New Growths, either sarcomatous or carcinomatous, may appear as nodular deposits in the walls of the sac. Both varieties are secondary. The sarcomata may spread from mediastinal growths and reach the pericardium along the sheaths of the great vessels or extend to it from contiguous glands. Carcinomatous growths may be secondary to cancer of the œsophagus or of other parts.

Serous Effusion into the Pericardium. The presence of a drachm or two of serum, apart from any evidence of inflammation, is very common and of no pathological importance. A larger effusion may occur as part of general œdema due to cardiac or renal disease. In a case where profuse diarrhœa or free hæmorrhage has preceded death, the pericardium and other serous sacs may be abnormally dry.

Pericarditis.—In the early stages of inflammation the inner surface of the pericardium becomes dull and sticky; its vessels are more or less injected. An inflammatory exudate soon forms which, from the rubbing together of the apposed surfaces, assumes a pitted, granular, or shaggy appearance. An effusion appears which is serous blood-stained, or purulent. Although simple pericarditis usually is of rheumatic origin, or occurs as a terminal event in chronic renal disease, the possibility of tuberculous infection must not be overlooked during the examination.

Pericarditis may also complicate gout, acute renal disease, and cirrhosis of the liver.

Incipient pericarditis may be limited to the posterior recess of the sac (sinus obliquus).

The lymphatic glands adjacent to the sac are enlarged and soft when pericarditis is present.

Purulent Pericarditis is, in most instances, the result of systemic infection, and associated with pneumonia or various forms of pyæmia. Pyæmic abscesses of the heart wall may burst into and infect the sac. Inflammation may spread to the pericardium from tuberculous, pneumonic or gangrenous foci in the lung. Adjacent empyemata may also give rise to pericarditis. Malignant growths invading the sac may excite suppuration. Subphrenic abscesses or gastric ulcers have been known to open into the base of the pericardium. Perforation of the sac by foreign bodies in the œsophagus is rare.

Pneumo-pericardium or gas in the pericardial sac is exceptional, and nearly always the result of advanced decomposition. It may also be caused by communication between the pericardium on the one hand, and the lung, stomach, or œsophagus on the other.

Blood-stained Effusions often occur in pericarditis and are attributed to the rupture of delicate vessels in the newly formed tissues by the churning movements of the heart. Bloody effusions may be seen in rheumatism, renal disease, certain malignant fevers, in septic cases, in blood-diseases, and as the result of decomposition.

The presence of pure blood or of blood clot in the pericardium, apart from external wounds or other injuries, should lead to a search for rupture of an aneurysm of a sinus of Valsalva, or of the first part of the aorta. Sometimes the hæmorrhage is due to rupture of the heart itself in consequence of abscess or of fibroid disease of its walls. Fibroid disease is usually syphilitic, and the rupture is in the left ventricular wall. Very rarely the intrapericardial portion of one of the great veins is torn.

The hæmorrhage in certain blood diseases, such as purpura, scurvy, or leukæmia, may be very free. Vascular new growths may be sources of hæmorrhage and, rarely, as the result of the impaction of foreign bodies in the retropericardial segment of the œsophagus, inflammatory changes leading to intrapericardial hæmorrhage may occur.

Hæmorrhage into the pericardium may cause sudden death. Under ten ounces of blood, if rapidly effused, are sufficient to stop the heart's action.

Pericardial Adhesions.—The sac may be wholly or partially obliterated by adhesions which are the result of inflammation. Localised adhesions are sometimes found between the front of the left ventricle, near its apex, and the anterior wall of the sac. Irregular adhesions of the pericardial sheath of the great vessels are not uncommon. Recent adhesions are soft and friable. Old adhesions may be exceedingly tough, and it may be quite impossible to separate them without lacerating the heart. When combined external and internal adhesions are present, or when internal adhesions are associated with myocarditis, the condition is much more serious than when internal adhesions are present alone. The condition of the heart wall should always be investigated. Interstitial myocarditis may be found.

The possibility of tuberculosis as a cause of universal adhesion should be remembered and search made for

caseous masses in the thickened pericardial layers. In many cases the adhesions are due to rheumatism. Endocarditis often coexists, and may be either of simple or malignant character. The coexistence of extensive pericardial and pleural adhesions with induration of the mediastinal connective tissues constitutes **chronic mediastinitis.**

REMOVAL OF THE THORACIC VISCERA, TOGETHER WITH THE TONGUE, FAUCES, UPPER AIR AND FOOD PASSAGES

Commencing at the median incision already made, the skin and superficial tissues on each side of the neck should rapidly be dissected backwards until the anterior margin of the sterno-mastoid muscle comes into view. The dissection is to be continued outwards in the plane beneath this muscle until the great vessels are fully exposed without injury. The only muscle divided is the anterior belly of the omo-hyoid which crosses them.

The head and neck being allowed to drop well backwards over the edge of the table, the skin is dissected back on each side from the underlying mylo-hyoid muscle until the submaxillary gland is seen ; this gland should not be cut through, but raised so that it can be turned over with the skin flap. The margin of the mandible should now be identified.

The floor of the mouth is transfixed in the mid-line, immediately behind the symphysis of the jaw, the point of the knife being carried into the mouth in a forward direction so that it may pass between the incisors and the tip of the tongue without mutilating the latter. Taking the inner surface of the mandible as a guide, the knife is carried backwards on each side with a to-and-fro movement, separating from the bone all the structures which form the floor of the mouth.

The tongue should next be hooked down through the wound and drawn forwards so that a view of the palate and fauces may be obtained. By sounding in the mid-line with the point of the knife, the junction of the soft palate with the hard may now be defined and the former transfixed at this point. The knife should then be carried outwards on each side along the posterior edge of the hard palate and the incisions continued downwards external to the pillars of the fauces and the tonsils so that these structures are detached in continuity with the soft palate.

The tissues which lie on the front of the spine, forming the posterior pharyngeal wall, are divided by inserting the blade of the knife from each side, in the gap between the hard and soft portions of the palate, and cutting backwards against the front of the subjacent vertebra.

By hooking the finger over the root of the tongue, the soft parts may be drawn forwards, allowing the knife to be carried downwards in the retropharyngeal space as far as the upper aperture of the thorax. During this manœuvre the knife must be kept internal to the great vessels, leaving these structures behind in the neck and undamaged. The parts liberated and drawn forwards are the floor of the mouth and tongue; the soft palate, tonsils and pillars of the fauces; the epiglottis, larynx and pharynx; the trachea, cervical portion of the œsophagus and thyroid gland.

Attention may now be turned to the structures which pass beneath the clavicles. The sterno-clavicular joints having been disarticulated already, the inner end of each clavicle should be drawn forwards, the blade of the knife introduced behind it and carried outwards in close contact with its posterior surface. It is then possible to see and isolate the bundle of vessels and nerves which passes towards each axilla. These structures should be divided as far outwards as possible, the insertion of the scalenus anticus into the first rib also being severed. By dividing the neuro-vascular bundles in this way the terminal portions

of the thoracic and right lymphatic ducts which enter near the points of junction of the subclavian and internal jugular veins, are preserved and removed with the other parts.

At this stage the condition of the cervical lymphatic glands should be ascertained and attention also turned to the state of the carotid arteries and jugular veins.

Examination of the veins is effected by opening them up in continuity with the great veins of the thorax and continuing the incision up to the base of the skull.

When disease of the carotid artery is suspected the vessel may be opened up in its whole length, starting at its origin from the aorta. In obscure cases of hemiplegia this examination should never be neglected.

The great vessels should now be divided just above the root of the neck by cutting across them on to the spine.

Then, in part by traction on the trachea and œsophagus, but in the main by means of the left hand introduced behind it from above, the mass of thoracic viscera is raised out of the thorax and turned over the right costal margin. A few touches with the knife, in front of the upper dorsal vertebræ, may be necessary to facilitate the liberation.

The aorta is easily defined and cut transversely just before it pierces the diaphragm. The œsophagus then comes into view and may be divided at a rather higher level. The attachment of the base of the pericardium to the diaphragm is next severed, and finally the inferior caval vein is divided as it lies stretched over the edges of the ribs. The cervical and thoracic viscera are now entirely free and can be placed aside for subsequent examination.

Where poisoning is suspected and it is important to preserve the contents of the stomach for chemical examination, the œsophagus should be ligatured below the point at which it is to be divided in the thorax.

In cases of corrosive poisoning, stricture, or malignant growth at its lower end, the œsophagus should not be cut across but the stomach removed in continuity with it.

EXAMINATION OF THE NASOPHARYNX, HARD PALATE, JAWS, AND MOUTH

Before proceeding to dissect the mass of viscera removed, complete the inspection of the nasopharynx, hard palate, jaws and mouth.

Cleanse the nasopharynx with a sponge, or a stream of water. Ascertain the condition of the mucous membrane, the Eustachian orifices, and the posterior openings of the nasal fossæ. Notice any adenoid vegetations and their extent.

Examine the hard palate, looking for malformations, injuries, ulcerations, perforations, new growths or other abnormalities.

By cutting through the hard palate round its line of junction with the alveolar margin of the maxilla with chisel or bone forceps, the inferior meatuses of the nose may be exposed from below and access also gained to the maxillary antra. (For examination of the nasal cavities see p. 289.)

The teeth and gums of both upper and lower jaws should next be inspected. It may be necessary to extract suspicious stumps with forceps to determine the presence or absence of suppuration round the fangs.

If it is necessary to remove portions of the jaws for further examination or on account of the presence of tumours, care should be taken not to injure the overlying cheeks or lips.

The inner aspects of the cheeks and lips and also the gums should be inspected for ulceration or pigmentation (Addison's disease, pernicious anæmia, lead poisoning, etc.).

The orifice of each parotid duct may be identified opposite the second upper molar tooth.

The submaxillary salivary glands and adjacent lymph glands will be found close to the inner aspect of the mandible on each side. The salivary glands may be dissected out and examined by a series of sections.

EXAMINATION OF THE FAUCES, TONSILS, AND TONGUE

The mass of viscera removed from the neck and thorax should now be placed, with its posterior aspect upwards, upon a table, the tongue being directed towards the examiner. Divide the soft palate vertically with scissors, on one side of the uvula. Cleanse by gentle sponging and examine the anterior and posterior surfaces. At the same time inspect the surfaces of the tonsils and carry a couple of deep incisions across them. The supratonsillar fossæ, above and behind the tonsils and under cover of the posterior pillars of the fauces, should be pulled open and examined. The tonsils can be enucleated with the scissors should a microscopical examination be necessary.

Œdema of the Fauces is recognised by the swollen translucent appearance of the tissues. It may be due to acute inflammation, to scalds, to the action of irritant fumes or corrosive liquids, or to renal disease.

Membranous Exudation on the Fauces is usually diphtheritic, but may be due to scalds or the effect of corrosive poisons. The extent and distribution of the membrane should be noted. In bad cases of diphtheria the membrane may be discoloured and putrid.

Ulcerations of the Palate and Fauces may be due to acute inflammatory processes, to syphilis, to tuberculosis, or, rarely, to chronic glanders. Shallow grey ulcers may occur in various infections. An extremely destructive, gangrenous inflammation may occur as a sequel to measles (noma).

Syphilitic Ulceration may give rise to widespread destruction of the soft palate, and terminate in the formation of adhesions between the velum and the posterior pharyngeal wall. Perforations of the palate may be produced.

Tuberculosis of the Palate is rare. The miliary nodules and caseating foci are characteristic, and the

condition is usually associated with tuberculosis of the tongue, pharynx, larynx, or with lupus of the face.

Cleft Palate is a common malformation.

The Tonsils may be the seats of acute or chronic inflammation.

Acute Tonsillitis.—The tonsils are swollen and rather soft. Shallow grey ulcers may be present, or the follicles may be plugged with masses of exudation.

In some cases of **Tonsillar Diphtheria** the membrane may be so thin and so similar in colour to the grey tonsils that careful scrutiny is necessary to determine its presence. It may be picked up with dissecting forceps, but, owing to its prolongations into the tonsillar lacunæ, comes off piecemeal.

Chronic Tonsillitis.—The tonsils are enlarged and fibrous. On section lacunæ plugged with cheesy or calcareous débris may be laid bare.

Suppurative Tonsillitis.—Determine whether the abscess is tonsillar or peritonsillar.

Malignant Disease of the Tonsil involves neighbouring structures as well. Microscopical examination is necessary for the detection of its precise nature.

Examine the tongue. Its surfaces must be scrutinised, not forgetting its edges and under aspect. Palpation will determine its consistence and the presence of any localised indurations or swellings. The foramen cæcum, which is the termination of the thyroglossal duct, should be inspected. Attention should be directed to the condition of the frænum and glosso-epiglottic folds. Examine the orifices of the lingual and submaxillary ducts alongside the frænum. Finally the tongue should be freely divided by transverse incisions to expose its substance.

The lymphoid follicles at the base of the tongue vary much in prominence. A general enlargement of these follicles, and also of the tonsils, thymus and other lymphoid structures in the body, constitutes the condition known as **Lymphatism** (see p. 88).

Bitten Tongue may be found in those who have died from epileptic, uræmic, or other convulsive conditions.

Acute Glossitis is manifested by swelling and œdema. The condition may be due to infection through wounds or abrasions of the organ, or be associated with a deep-seated abscess in the submaxillary region (Ludwig's Angina).

Lymphangiectasis gives rise to enlargement of the tongue. The condition is due to lymphatic obstruction.

Atrophy of the Tongue, usually unilateral, is caused by lesions of the hypoglossal nerve or of the muscles.

Leucoplakia presents itself as thickened whitish patches on the edges or dorsum of the organ.

Gummata may cause localised swellings on the dorsum, usually near the central region (see p. 286).

Epitheliomatous Growths give rise to indurated ulcers at the margin which tend to invade surrounding parts, fixing the tongue and causing swellings of the submental, submaxillary, or cervical lymph glands. Visceral metastases are rare.

Tuberculous Ulcers are rare and usually associated with tubercle elsewhere. They are grey and sinuous.

Cysts may be found in connexion with the thyro-glossal duct. **Dermoids** may lie in the mid-line, usually near the under surface of the mylo-hyoid muscle. Tumours having the structure of **thyroid tissue** sometimes occur.

Ulcer of the Frænum Linguæ.—A shallow ulcer on the frænum linguæ should lead to a suspicion of whooping-cough, but may also occur in other conditions where cough is intractable.

Laxity of the Frænum may allow the tongue to fall back into the pharynx and so cause suffocation (p. 304).

EXAMINATION OF THE THORACIC DUCT

The thoracic duct is easily found in the furrow between the œsophagus and the upper end of the thoracic aorta. The mass of viscera lying with the posterior aspect

upwards, the duct should be exposed, in the situation indicated, by blunt dissection. It should be gently picked up with the fingers, not by forceps or it will be lacerated, and freed by the scissors in its whole length. Its opening in the termination of the left internal jugular vein or, less commonly, the point of union of the latter with the subclavian, must be found by opening up the veins from the front ; a probe may be passed from the vein into the duct. The opening of the duct is usually guarded by a valve, which may be double. The terminal portion of the duct often bifurcates and reunites before entering the vein. Pay particular attention to the presence of enlarged glands around the upper end of the duct. The lower end of the duct will have been cut across, above the receptaculum chyli, in removing the thoracic viscera. This mutilation can be avoided and the structures examined in continuity if the contents of the thorax and abdomen are removed entire as described on page 176.

Obstruction of the Thoracic Duct.—Although the duct is frequently subjected to pressure, definite dilatation as a rule occurs only when the terminal part, near its entrance into the vein, is involved. Obstruction may be due to malignant, tuberculous, or lymphadenomatous enlargement of lymphatic glands, especially of those at the root of the neck on the left side. Thrombosis of the innominate vein or its tributaries may also give rise to obstruction.

Intra-abdominal malignant disease may secondarily involve the receptaculum chyli and walls of the duct, or travel up in the connective tissue planes which surround it, or implicate the posterior mediastinal glands in the neighbourhood, and so exercise pressure on it. Abdominal tuberculosis may also infect it.

In filarial disease the parent worms may possibly occlude the lumen.

EXAMINATION OF THE PHARYNX AND ŒSOPHAGUS

The pharynx and œsophagus should be slit up in the mid-line behind with bowel scissors. The interior should be cleansed from mucus with a damp sponge.

Normally the walls of the œsophagus are quite supple and its mucous membrane is thrown into slight longitudinal folds. Both œsophagus and pharynx may contain some gastric contents which have been regurgitated at the time of death, and the mucous membrane of the lower end of the œsophagus is sometimes bile-stained from the same cause.

The pharynx rarely presents evidence of gross disease.

Impaction of the Tongue in the Pharynx.—In infants, sometimes, and in some cases of death by hanging, the tongue may be found rolled back into, or otherwise obstructing, the upper end of the oro-pharynx.

Foreign Bodies are occasionally found impacted in the pharynx. Small particles of food or of bone may lodge in the pyriform fossæ or between the tongue and the epiglottis.

Diverticula of the Pharynx occur at its lower end. They project between two parts of the inferior constrictor muscle.

Acute Pharyngitis is generally associated with tonsillitis or œdematous inflammation of the fauces. The inflammatory process may terminate in suppuration or gangrene.

Retropharyngeal Abscesses, if present, will be recognised during removal of the pharynx from the neck. They may be median or lateral. Ascertain whether the pus is superficial to the anterior common ligament of the spine or beneath it. In the latter case suspect caries of the vertebral bodies. A superficial abscess may complicate tonsillitis or arise from a suppurating gland.

Chronic Pharyngitis may be indicated by undue tenuity and smoothness of the mucous membrane or by

the presence of nodules of lymphoid tissue. The nose and accessory cavities should be examined for pus.

Syphilitic Pharyngitis.—Evidence of old or of active syphilis may be found in the pharynx : the former in the form of scars and palatine adhesions, the latter as serpiginous ulcerations or gummata.

Actinomycotic Pharyngitis may occur by direct infection. The condition must be diagnosed by detection of the ray-fungus microscopically.

Varicose Œsophageal Veins may be found at the lower end of the gullet in cases of portal obstruction, such as occurs in cirrhosis of the liver. Pore-like erosions may sometimes be found in these veins when death has been due to hæmorrhage from them.

Simple Ulcers are sometimes found at or near the termination of the œsophagus.

Post-mortem Digestion of the Œsophagus is uncommon, but occasionally the lower inch or so of the tube is soft, gelatinous, and easily lacerated if not already dissolved. In such cases gastric contents may be found in the pleural sac without signs of pleurisy (see p. 156).

Acute Inflammation of the Œsophagus occurs as the result of corrosive poisoning : the whole of the mucous membrane may sometimes be found thickened and detached in the form of a cast. Later on strictures are apt to occur. Sometimes, quite apart from poisoning, the epithelial lining is sodden and loose and can be detached in the form of a wrinkled pellicle. This condition is not pathological.

Diphtheria of the Œsophagus is rare. The nature of the membrane is determined by the presence of diphtheria in the fauces or larynx.

Foreign Bodies may be found impacted in the œsophagus. The point of impaction and the amount of damage to surrounding structures call for investigation.

Aneurysm of the Aorta may give rise to a localised bulging into the lumen of the gullet, or may even perforate into the tube.

Malignant Disease of the Œsophagus is usually a squamous-celled carcinoma, but spheroidal-celled growths

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occasionally occur. The site of the growth may be at the commencement, the end, or the middle of the tube. As a rule the growth encircles the lumen; is ulcerated and septic. If the growth is high up in the œsophagus, examine the conditions of the larynx, trachea, laryngeal nerves, and thyroid gland, as well as the adjacent lymph glands. With a growth midway in the tube, examine the trachea and left bronchus for invasion, also the mediastinal glands. A growth at the lower end may involve the pleuræ, the lung bases, and the lumbar, as well as the mediastinal glands.

Congenital Malformations and Pouches of the œsophagus are not at all common. Complete interruption of the tube a short distance above the tracheal bifurcation may occur. Congenital strictures may be found at the same spot. Pressure Pouches are found in the mid-line posteriorly, at the junction of the œsophagus and pharynx (see p. 45); they are at first lodged in the retropharyngeal space, but later may be present at the left side of the neck. Dimples or Traction Diverticula may be found near the tracheal bifurcation; in such cases search should be made for inflamed or adherent lymphatic glands, which are supposed to cause the pouching. Sometimes the diverticulum appears to originate in the floor of a punched-out ulcer.

Rupture of the Œsophagus is rare and is practically limited to the lower third of the tube.

Idiopathic Dilatation is an uncommon affection of unknown origin.

Œsophageal Warts are occasionally met with. They should be submitted to microscopical examination to determine their nature.

EXAMINATION OF THE LARYNX, TRACHEA, AND MAIN BRONCHI

First clear away any mucus from the upper aperture of the larynx and inspect the epiglottis, the arytænoepiglottidean folds, the arytænoid cartilages, and the false cords. Determine the position of the true cords and the condition of the ventricles of the larynx.

Remove the œsophagus by dissecting it away from the pericardium and subjacent tissues, commencing the separation at the lower end. Note the condition of the bronchial glands exposed in the angle of bifurcation of the trachea.

Divide the larynx and trachea along their posterior aspects in the mid-line and continue the incision along the main bronchi towards the lungs (Fig. 6). Avoid injury to the vocal cords when introducing the scissors into the larynx to commence the incision. Grasping each ala of the thyroid cartilage between the thumb and forefinger, forcibly open out the larynx for inspection. Where there is any possibility of a fracture of the laryngeal cartilages having occurred before death, they must be carefully examined before the two halves are forced apart. Do not overlook fractures of the hyoid bone.

Sometimes it is advantageous to open the larynx and trachea in the anterior median line, as for instance where the trachea and œsophagus are involved in the same lesion or it is desirable to keep intact a lesion of the posterior wall of the larynx.

The posterior incision has the advantage of dividing only the membranous portions of the trachea and bronchi. It also lays bare the layers of retrolaryngeal connective tissue and affords easy access to the branches of the recurrent laryngeal nerves.

When the interior of the larynx, trachea, and bronchi is fully exposed, note the presence and character of any abnormal contents; remove these, if present, by sponging and inspect the mucous lining of the passages.

The normal colour of the vocal cords is a pearly white. They should be smooth and sharp-edged. The presence of small ulcers at the points of junction of the true cords and the arytænoid cartilages is apt to be overlooked.

After a thorough examination of the interior of the larynx it may be necessary to ascertain the condition of

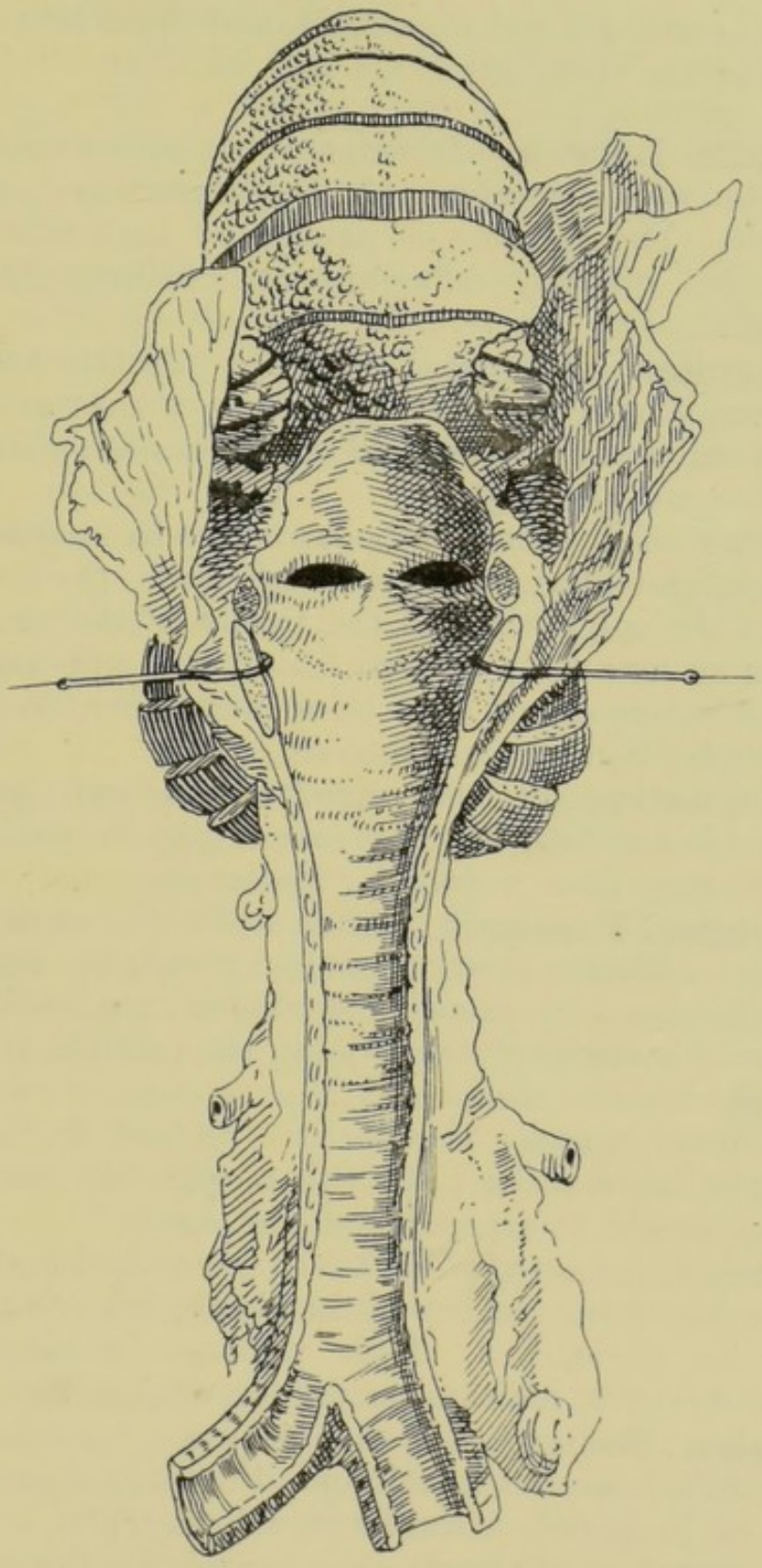

FIG. 6.—Examination of Tongue, Tonsils, Larynx, Trachea and Main Bronchi.

the laryngeal cartilages, also of the joints between the arytænoid cartilages and the cricoid, and those between the cricoid and the thyroid.

Infantile Larynx.—In infants the structures which bound the upper aperture of the larynx are soft and collapsible. The aperture is narrow, so that it is easily blocked by œdema, inflammatory swelling, or false membrane.

Congenital Laryngeal Stridor.—In this condition the laryngeal aperture may be merely a narrow antero-posterior slit, and the arytænoid cartilages show a tendency to prolapse into the larynx.

Œdema easily occurs in the lax connective tissue of the arytæno-epiglottidean folds, the false cords, the laryngeal aspect of the epiglottis, and the tissues of the ventricles. It causes an extremely swollen translucent appearance of the parts involved. It may be inflammatory, traumatic, or a complication of renal disease.

Inflammation of the same tissues also produces considerable swelling, but the colour may be redder and the parts may have a granular appearance.

Laryngeal Paralysis.—After death the cords should lie in the cadaveric position, mid-way between adduction and abduction. In cases of laryngeal paralysis or of ankylosis of a crico-arytænoid joint, the position of one or both cords may be abnormal: in such cases the abductor and adductor muscles should be examined for signs of wasting or degeneration; for this purpose the muscles of two sides should be exposed and compared.

Gastric Contents, which have regurgitated just before death or during the removal of the viscera, may be found in the larynx or trachea. Their presence in the smaller bronchi, however, indicates aspiration during life.

Foreign Bodies, such as fish bones, may be found sticking in the mucous membrane of the upper part of the larynx, or impacted in the laryngeal ventricles.

Laryngeal Diphtheria is indicated by the presence of membrane which is pearly or opaque and easily stripped. Its position and extensions should be noted. Membrane

may be well marked in the laryngeal sinuses. Examine also the epiglottis and tonsils.

Papillomata of the Larynx, small cauliflower-like growths, which are usually pedunculated, spring from the anterior commissure or the anterior thirds of the true vocal cords. Sometimes they are much more extensive in their distribution.

Malignant Growths of a warty or infiltrating character spring from the posterior thirds of the true cords, or from the inter-arytænoid fold. They are squamous-celled carcinomata, and may spread widely, tending to extend to the opposite side of the larynx or to the subglottic region rather than upwards.

Malignant disease may also arise on the posterior aspect of the cricoid cartilage, near the arytænoids, or from the arytæno-epiglottidean folds, or the posterior surface of the epiglottis. Growths in these situations tend to spread to the upper part of the larynx, and the adjacent pharynx.

Pachydermia Laryngis is a rough and thickened condition of the mucous membrane of the upper subdivision of the laryngeal cavity, together with small nodular thickenings of the true vocal cords (singers' nodes).

Tuberculosis of the Larynx is most likely to involve the structures which bound the upper laryngeal aperture, but no parts are exempt. The tissues involved are swollen, infiltrated and anæmic. Ulceration is most often seen over the processus vocales, but the epiglottis may be destroyed and perichondritis and necrosis of the laryngeal cartilages set up. The ulcers have a worm-eaten appearance, and cicatrisation is absent. Pulmonary tuberculosis is almost invariably associated and aids the recognition of the condition.

Syphilitic Laryngitis is characterised by inflammatory infiltrations, ulcerations, or cicatrices with contractions. Deep undermined ulcers may be found in the later stages. The lung apices are free from disease, and ulcerations of the palate and pharynx may indicate the nature of the disease.

Perichondritis of the Laryngeal Cartilages may occur as the sequel of syphilitic, tuberculous or malignant

disease, or may result from injury due to the impaction of foreign bodies. Perichondritis may also arise as a complication of certain specific fevers, especially typhoid. The perichondrium may be thickened, pulpy, or separated ; abscesses and ultimately necrosis of the cartilages may result. The arytænoid cartilage is particularly apt to suffer in tuberculosis, whilst in typhoid it is usually the posterior aspect of the cricoid which is involved.

Ossification of the Laryngeal Cartilages.—This begins at the age of twenty or thereabouts. It is first seen in the thyroid and cricoid cartilages near the crico-thyroid joint. A few years later the arytænoid cartilages begin to ossify from below upwards. Ossification begins earlier and proceeds more rapidly in male subjects.

Malformations of the Trachea are rare. In certain cases of congenital occlusion of the œsophagus the trachea communicates with the latter below the occlusion.

Bronchitis may be indicated by the presence of tenacious mucus or pus which wells up from the bronchi.

In Drowning a mucous froth may be found in the trachea and upper air passages.

Blood in the Air Passages may be the result of lung disease, or of the rupture of an aneurysm into the air tubes. It is sometimes aspirated into the larynx from some other source. The origin should carefully be sought.

Abscesses may burst into the trachea, flooding the air passages.

Discoloration of the mucous membrane of the lower part of the trachea need be of no pathological importance. Considerable diffuse and purplish staining is a sign of decomposition.

Tracheitis is indicated by the presence of tenacious mucus and a granular appearance of the mucous membrane, in addition to vascular injection.

Extensive Ulceration of the Trachea is seen in some cases of pulmonary and laryngeal tuberculosis. In rare cases the tracheal cartilages may be laid bare and recent miliary tubercles seen in the tracheal mucous membrane.

Diphtheritic Membrane sometimes forms a complete cast of the trachea. It tends to become softer and less coherent towards the bronchi.

Syphilitic Stenosis of the trachea is exceedingly rare.

Foreign Bodies are occasionally found in the trachea, and even caseous bronchial glands which have ulcerated through its wall.

Lateral compression of the trachea may be present in cases of enlargement of the thyroid gland. Aortic aneurysms and new growths of the œsophagus may encroach on the lumen of the trachea, and exceptionally, narrowing may be caused by an enlarged thymus gland (see p. 87).

Tracheotomy may have been performed. Note the exact position of the incision, and also whether it involves any of the cartilages of the larynx. Sometimes ulcers are found, which are evidently due to pressure of the lower end of the tracheotomy tube.

EXAMINATION OF THE LUNGS

Weight.—The lungs should be weighed before and after incision. Their combined weight varies within wide limits, from 30 to 48 oz. The average weight in a healthy male is R. Lung 24 oz. (680 grms.), L. Lung 21 oz. (595 grms.); in the female R. Lung 21 oz. (595 grms.), L. Lung 15 oz. (425 grms.). It is worthy of remark that the lungs often appear to be unduly small in very fat subjects.

Commencing with the right, all parts of the exterior of the lungs should be inspected, not omitting the diaphragmatic and mediastinal aspects. The interlobar fissures should be opened up and explored. Any abnormality in the shape of the lung or increase in the number of its lobes should be noticed.

The lungs should next be carefully and systematically palpated, attention being paid first to the superficial and then to the deeper portions by pinching them up between

the fingers and thumb. By this means an idea is gained as to the amount of air in the lung, the elasticity of its substance, and the presence or absence of consolidation, whilst localised lesions such as scars, infarcts and tuberculous deposits are less likely to be overlooked.

When palpating the lung the resistance offered by the bronchi may simulate the presence of a pathological condition. In case of doubt an incision will soon settle the point.

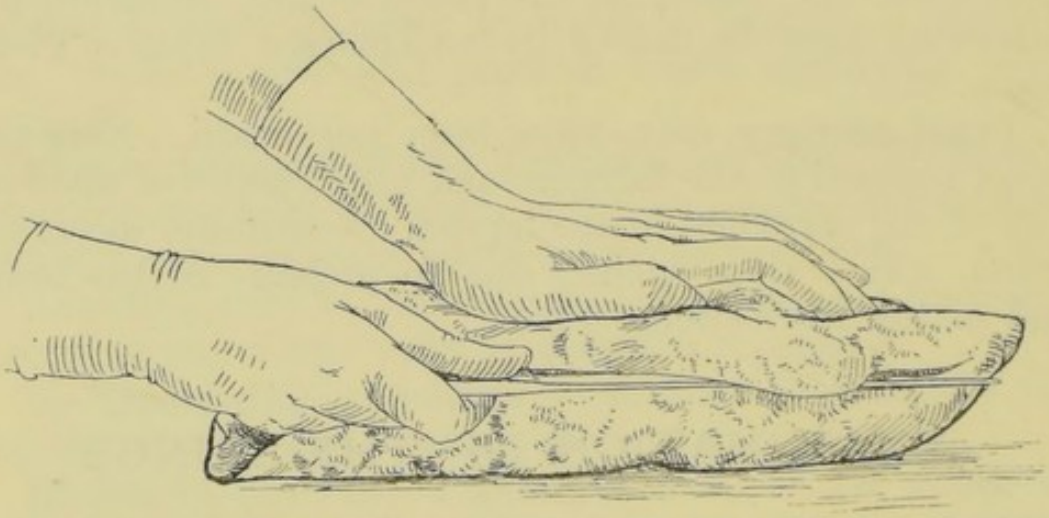

Fig. 7.—Incision of the Lung.

The lungs should be incised before the structures which form their roots are divided. The right lung should be arranged so that it lies with its anterior surface on the table and its base looking away from the operator. Firm downward pressure must be made on it by the palm and fingers of the left hand whilst an incision is made with the brain knife from its convex border to its root. The knife must be sharp and used with a slight sawing movement which facilitates the incision. The left lung is incised in a similar manner, being also placed on its anterior surface, but base towards the operator (Fig. 7).

The lung substance may further be exposed by means of incisions at right angles to each of the cut surfaces, still

following the long axis of the organ. In addition to these certain special incisions should be made from the outside (*a*) into the apex, (*b*) into each lobe parallel to the interlobar fissures, (*c*) from the basal aspect vertically upwards.

With lesions near the root it may be an advantage to make two transverse incisions into the lung, one immediately above and the other immediately below the hilum.

None of the incisions described should be carried so deeply as actually to separate any part of the lung from the rest.

Each cut surface should be lightly scraped with the knife to remove fluid, carefully inspected and finally palpated. The inspection must include an investigation of—(*a*) the bronchi; (*b*) the peribronchial glands; (*c*) the thick-walled pulmonary artery; (*d*) the thin-walled pulmonary veins; (*e*) the lung parenchyma itself. Palpation, conducted by gently squeezing the lung tissue, gives information as to the presence or absence of air (crepitation), the contents of the bronchi, and the consistence of the lung. The friability is tested by making firm pressure with the pad of the thumb —a healthy lung is not easily lacerated by moderate force applied in this manner. Next apply the hydrostatic test. Remove a small cube of the part to be examined, and, without squeezing, throw it into water. Air-containing lung will float, and will continue to do so after gentle squeezing. Collapsed or pneumonic lung will sink at once.

A special examination of the smaller bronchi may be necessary in bronchiectasis and certain other conditions. This examination should be carried out before incisions are made into the lung. Commencing at the root the bronchi are followed out and laid open by scissors, cutting through the whole thickness of lung between the tubes and the surface. The bronchi of all the lobes should be examined in this way.

Investigation of the branches of the pulmonary artery may be effected in a similar manner. It is called for where mitral stenosis, thrombosis or infarction are present. This

dissection is better carried out before the heart is detached from the lungs (see p. 67).

In cases of hæmoptysis the source of bleeding may sometimes be identified by injecting the pulmonary artery with water. The fluid may be seen to issue from a bronchus which, when slit up, exposes the site of hæmorrhage.

Bronchitis.—Congestion and dulling of the mucous membrane of the bronchi, coupled with the presence of tenacious mucus, indicate bronchitis. Mucus can also be squeezed from the smaller tubes exposed by section of the lung. Mere injection of the larger bronchi, without secretion cannot be considered pathological. The diffuse purplish staining of the interior of the air passages which is seen in commencing decomposition should not be mistaken for evidence of bronchitis.

Bronchiectasis.—This is usually associated with chronic inflammatory lesions of the lung. Adhesions of the pleuræ may or may not be present.

Bronchiolectasis is really a combination of dilatation of the small bronchioles with an emphysematous condition of the air sacs. A cystic condition of the lung in childhood, called honeycomb lung, is of similar nature, although sometimes it is held to be a congenital malformation. Bronchiolectasis and honeycomb lung are probably the results of bronchopneumonia, with or without abscess formation.

Saccular Bronchiectasis is similar in appearance to honeycomb lung, but on a larger scale. The condition is mostly basal, but may be widespread.

Fusiform or Cylindrical Bronchiectasis involves the larger bronchi, usually those of the lower lobe. It may be the outcome of chronic bronchitis. Bronchiectasis, such as results from pressure on the bronchi, involves the air passages below the point of obstruction.

Foreign bodies may give rise to bronchiectasis, which is then frequently associated with a suppurative broncho-pneumonia.

In all cases of bronchiectasis the bronchi should be slit up from the lung root, and the fact of their communication

with the cavities established. Note any indication of active or chronic inflammation. Determine whether the tubes are compressed near the root of the lung, or contain a foreign body. Observe the condition of the surrounding lung tissue, whether indurated, pneumonic, or gangrenous. The presence or absence of pleural adhesions and the condition of the cavities of the right heart should also be ascertained. Bronchiectasis may give rise to brain abscess or be a cause of pyæmia.

Emphysema occurs in several forms.

Interstitial Emphysema appears as air-containing streaks, corresponding to the septa of the lung and visible beneath the visceral pleura. It is due to rupture of air cells into the supporting tissue or to extension of emphysema from the mediastinum into the root of the lung.

Hypertrophic Emphysema gives rise to pale and voluminous lungs which often have large air-containing bullæ at their margins. The emphysematous tissue is inelastic and hence doughy in consistence. It is pale both externally and on section. The usual distribution of the emphysema is along the anterior margin and around the basal edges; it is often extensive near the apex. It is frequently associated with chronic bronchitis, arterio-sclerosis, and chronic renal disease. The lung bases in emphysema are often in a condition of hypostatic congestion.

Atrophic Emphysema is emphysema occurring in a wasted lung. It may be seen in the aged.

Compensatory Emphysema will be found surrounding lung cicatrices or intermingled with collapsed areas in bronchopneumonia.

The whole of one lung may undergo over-distension to compensate for crippling of the opposite one, or if one lobe of a lung for any reason becomes completely collapsed, the neighbouring lobe or lobes may present general emphysematous dilatation. If the condition is established suddenly it is known as Acute Emphysema.

Congestion or Engorgement of the Lung may be active and inflammatory, or passive. Acute congestion is the earliest stage of pneumonia; the lung is over-full of

arterial blood, and at the same time is unduly friable in consequence of inflammatory softening.

Chronic Passive Congestion or Brown Induration of the Lungs occurs in old-standing heart disease. Owing to long-continued back-pressure in the pulmonary circuit the lungs become firm and short of air. On section they present a reddish-brown colour. They are not readily lacerated by pressure with the finger, and still float in water.

Another form of engorgement of the lung is found in those dead from head injury, cerebral hæmorrhage, or narcotic poisoning. In such cases the lungs may appear saturated with venous blood, so that they are of a very deep red or almost black colour.

Hypostatic Congestion of the Lungs is a mixed condition. It occurs in feeble, bed-ridden patients, and those dying from acute fevers or infections. The posterior parts of the lungs, and particularly the lower lobes, are congested, œdematous, in part collapsed, and often broncho-pneumonic.

Post-mortem Hypostases may cause the posterior dependent parts of the lungs to assume a deep bluish tint.

Decomposition of the Lungs should not be confounded with congestion. Decomposing lungs are sodden and friable, often over-crepitant and black or greenish in colour. The interior of the air passages is much stained. The presence of such signs does not of necessity indicate that the lungs were inflamed during life.

Œdema of the Lung.—An œdematous lung is bulky and heavy. An enormous amount of watery or blood-stained fluid can be expressed from its cut surfaces. In cases of jaundice this fluid may be yellowish. The degree of frothiness of the expressed fluid will depend on the amount of air still present in the lung. Before the fluid is expressed, such lungs may even tend to sink in water.

A marked increase in the friability of an œdematous lung may be taken as an indication that the condition is of inflammatory origin. Œdema of this kind is found, though in varying degree, in association with all acute pulmonary inflammations.

A slight degree of pulmonary œdema is exceedingly common, and appears to be due to slow failure of the circulation.

Passive œdema of the lung is often a sequel of the chronic venous congestion which results from valvular disease of the heart, but the tough, red lungs due to back-pressure do not become very greatly waterlogged.

A more pronounced passive œdema may occur in renal disease, or as the result of certain acute lesions of the brain. It is sometimes seen after paracentesis of the thorax, the lung on the side punctured being bulky and sodden.

Collapse of the Lung.—Bluish, airless patches which are depressed below the surface of the surrounding lung tissue are due to collapse. The patches are congested, airless and solid. They sink in water. Collapse may appear in the form of extensive tracks, particularly on the posterior, dependent portions of the lung; it is more common in young children than at other ages. Unlike patches of pneumonia, collapsed areas can be reinflated, provided that this is attempted before the bronchi are incised. Patchy collapse is usually associated with broncho-pneumonia.

Massive collapse and retraction of the lung may occur as the result of pleural effusion or of pressure on the root. In such cases the lung is darker, firmer and smaller than normal; the pleural envelope shows wrinkles, and parts, if not all, of the organ will sink in water. Massive collapse may also be due to the presence of foreign bodies in the bronchi, or to pressure exerted on the neighbouring part of the lung by a greatly distended pericardium or an enlarged heart. Collapse of the lower lobes may occur when the diaphragm is paralysed or ceases to act owing to abdominal inflammation or distension. Intercostal paralysis affecting the upper part of the thorax may induce massive collapse of the upper lobes of the lungs. The main bronchial tubes in a collapsed lung appear unduly large by contrast; this condition must not be confounded with bronchiectasis.

Atelectasis is a congenital condition in which the lungs of the infant are completely or partially unexpanded.

In the complete form the fleshy, airless lungs lie far back in the thorax. Such lungs sink in water, but can be inflated. Presence of atelectasis does not necessarily prove that a child has not breathed at all.

Lobar Pneumonia or Croupous Pneumonia may come under observation at various stages. In the early stage, that of acute congestion, the lung is much injected, exudes bloody serum when squeezed, and is rather more lacerable than normal. It is, however, still crepitant, and floats in water. This stage may be seen where pneumonia is advancing at the time of death, and is usually associated with consolidation of other parts of the lung. More rarely it is the only lesion present.

In the second stage, that of red hepatisation, the affected part, usually a whole lobe or more, is solid. The lung is bulky and heavy ; it maintains its contour when removed from the body, and is often marked by indentations corresponding to the ribs. The visceral pleura is dull, sticky, and often coated with inflammatory exudation. On section the lung is deep red, granular and dry. Casts of fibrin are found in the pulmonary arteries, and, more rarely, in the bronchi. The lung parenchyma is friable, airless, and sinks at once in water.

In the third stage, grey hepatisation, the cut surface of the solid portion has undergone transition from red to grey, and the friability is more marked.

Sometimes a still more advanced stage, that of purulent infiltration, is met with. The colour is still grey, but the consistence of the exudation has so diminished that purulent matter exudes from the cut surface.

In the lobar pneumonia of infants the consolidated area tends to be less well defined and has not the finely granular appearance which it presents in the adult : the lung parenchyma is more moist, and the surface rather glistening instead of dull and opaque looking.

Bronchopneumonia or Catarrhal Pneumonia.—The appearances presented by bronchopneumonic lung show great variations. The slighter forms are only differentiated from the bronchitis with which they are always associated by the mottled appearance of the lung on section. The

mottling is due to the intermixture of small areas of emphysema, which are paler, with areas of collapse and consolidation which are darker than the normal lung tissue. The lung on the whole is denser and decidedly less crepitant than normal. Muco-purulent fluid exudes from the air tubes on pressure. It is only in the later stages that the appearances resemble those described in the next paragraph.

Bronchopneumonia, particularly that which occurs as a complication of measles or whooping-cough, presents a more distinctive appearance. Small islets of consolidation, each of which surrounds an inflamed bronchiole, can be recognised. The islets are reddish or greyish in colour, and project slightly from the surface of the section. When they become confluent, large portions of the lung, even whole lobes, may become solid, but the circular outlines of the consolidated lobules may still be distinguished. Apart from aspiration and deglutition bronchopneumonia, the only form which is at all frequent in the adult is that which occurs as a sequel to influenza. Here the condition is widespread, and the surface on careful inspection suggests a consolidation by confluence; it is less granular and less uniform than in lobar pneumonia and the bronchial tubes are acutely inflamed.

Bronchopneumonic lung may become the site of abscess formation and of bronchiolectasis.

Infarction of the Lung.—Hæmorrhagic infarctions of the lung are conical masses of extravasated blood, the bases of which abut upon the pleura. They are usually found in the lower lobes and vary much in size. Sometimes they are buried in the interior of the lung, and then may be rounded or oval instead of triangular on section. Recent infarcts have a dark red and dry appearance; older ones are much paler. Absorption without scar formation occurs, provided the patient lives long enough. Infarcts are easily felt when the lung is handled, and the pleura immediately over them may show slight signs of inflammation. The subjacent infarct can be seen as a dark purplish mass of consolidation.

The pulmonary arteries which supply an infarcted area should be carefully opened up and search made for an

embolus or a thrombus : the latter may itself be merely a result of infarction. The embolus which produces a pulmonary infarct must be derived from the recesses of the right ventricle or right auricle since it cannot reach the lung from the left side of the heart. It is possible that some infarctions are due to simple pulmonary hæmorrhage and not to embolism ; in such cases the conical shape of the infarcts would be determined by the shape of the lung lobules.

Septic infarction of the lung leads to suppuration and is described under Pulmonary Abscess below.

Solitary Abscess of the lung is uncommon. It is usually a sequel of lobar pneumonia, but may depend upon the presence of a foreign body in the bronchus, or of a hydatid cyst.

Multiple Embolic Abscesses of the Lung occur in pyæmia. They mostly lie at the surface of the organ, and at first resemble small white infarcts, but are surrounded by a zone of intense congestion. They rapidly become converted into abscesses, and infect or perforate the pleura.

Multiple abscesses may also form in aspiration bronchopneumonia, a condition in which bronchopneumonic islets are large and the pulmonary inflammation very acute.

Bronchopneumonia, such as follows measles, may cause small abscess cavities. This event is not common, and care must be taken to distinguish the exudation of pus from small bronchioles for evidence of true abscess formation.

In **Glanders** multiple lung abscesses may occur ; the zone of hyperæmia around these is peculiarly intense. Look for ulceration of the nasal mucous membrane, or fauces; a papular suppurating skin eruption; subcutaneous and intramuscular abscesses.

Gangrene of the Lung gives rise to irregular cavities with fœtid contents consisting of shreddy, sodden lung tissue, altered blood and pus. The surrounding lung is in a condition of acute inflammation and much discoloured. Extensive pulmonary gangrene is rare, its chief cause being pneumonic inflammation in diabetic subjects. Invasion of the lung by a malignant growth of the œsophagus also causes gangrene owing to infection. Gangrene sometimes

occurs during the course of pulmonary tuberculosis and in acute infective fevers.

Multiple Gangrenous Abscesses of small size may result from aspiration bronchopneumonia, particularly from that variety which complicates malignant disease of the mouth or fauces. Infective embolisms such as occur in lateral sinus pyæmia, or from extensive bedsores, may also give rise to similar abscesses.

Miliary Tuberculosis of the Lung is recognised by the presence of numerous granules of grey, translucent appearance, and about the size of millet seeds. In their later stages the tubercles are rather larger, and may be yellowish and opaque owing to caseation. In the more chronic cases a zone of pigmentation and fibrosis may surround them. Tubercles coexist in the visceral pleura, and here the pigmentation may be more marked.

A very copious growth of tubercles is found in the lung when the tubercle bacilli have gained access to the circulation through the systemic veins or thoracic duct. In some instances of this kind the lung feels almost solid.

Sometimes the miliary tubercles in the lung are scanty and the brunt of the infection falls upon the spleen, liver and kidneys; a pulmonary vein may then be suspected as the portal of general infection.

If the miliary tubercles spread in lines along the septa and in the fibrous sheaths of the bronchi and vessels of the lungs, and if they are limited in their distribution, extension along the lymphatics from the lung root is indicated.

In miliary tuberculosis all the deposits need not be of the same age and appearance; deposits of different ages point to successive blood infections.

In all cases the focus of infection should be found if possible. In children it is commonly a caseous bronchial gland, but mesenteric glands may infect the mesenteric veins; cervical glands, the jugular veins; or the infection may be derived from tuberculous disease of the joints, bones, ear or lung.

Chronic Pulmonary Tuberculosis affords an opportunity of observing all stages of the tuberculous process. The changes are more advanced at the apices of the lobes,

and of older standing at or near the middle of the upper lobe than in the lower lobe of the same side. The rigth middle lobe may sometimes escape.

Near the apices of the lobes involved, excavation and fibrosis may be found; lower down will be caseous areas undergoing softening, and below these smaller islets of tuberculous aspiration bronchopneumonia. The older caseous areas may present a racemose appearance owing to the spread of tubercles into the surrounding connective tissue and lymphatics. If an artery supplying the lung has been penetrated a dense crop of miliary tubercles will be found in the wedge of territory which it feeds.

Occasionally, with an advanced lesion in one lung, a recent tuberculous bronchopneumonia may be found near the root of the opposite lung, pointing to secondary infection by aspiration across the tracheal bifurcation.

Firm pleural adhesions overlie the old lesions. In the neighbourhood of active lesions, small grey tubercles may often be recognised in the visceral pleura; these lie in the pleural lymphatics.

In cases of phthisis the presence of pneumothorax, due to perforation of the visceral pleura, is indicated by considerable retraction of the lung, and intense inflammation of the pleural sac. The test for pneumothorax has already been described (p. 17).

Recent phthisical cavities are small and their walls are ragged and eroded; the condition is best demonstrated by examination under a stream of water. Patches of caseous material may still be present. Old cavities have smooth walls surrounded by dense fibrous tissue. They may be empty or full of pus, or obviously undergoing contraction. Trabeculæ of connective tissue may traverse the cavities or project as tags from their walls. Aneurysms may occur in these septa, but are more common in the walls of the cavities. Rupture of an aneurysm is the cause of profuse hæmoptysis. In such cases injection of the pulmonary artery from the root of the lung before the latter is incised may lead to escape of the fluid from a bronchus; if the bronchus is opened up carefully the source of the hæmorrhage may be traced.

The bronchial glands should always be examined in cases of phthisis.

The usual concomitants of advanced phthisis are a fatty liver and wasted heart. Search should be made for laryngeal and intestinal ulceration, and also for lardaceous disease.

Obsolete Pulmonary Tuberculosis.—Adhesions of the posterior aspects of the apices and of other parts of the lungs occur with great frequency in adults. In such cases careful examination of the underlying lung tissue frequently reveals the presence of scars, fibrous knots, or encysted gritty masses, the residues of past tuberculosis. Sometimes the scars are concealed by localised compensatory emphysema. Discovery of calcareous or caseous masses in the visceral pleura of the fissures or lower lobes of the lungs may also indicate the nature of old adhesions.

Caseous Pneumonia.—A process of rapid pneumonic caseation may occur in the lung, but is uncommon. The greater part of one lobe or sometimes the whole lung is found to be solid, and presents a more or less uniform greyish or yellow colour. Light may be thrown on the nature of the process by the presence of caseous bronchial glands in the case of a child, or older foci of phthisis in the adult.

The process is rapid and acute. Any adhesions present are soft and friable. Occasionally the caseous matter may soften with great rapidity, giving rise to large, irregular cavities.

Tuberculous Bronchopneumonia.—Disseminated bronchopneumonia of this nature occurs in the form of nodules scattered throughout the lung, the intervening tissue being congested but crepitant. The nodules are about the size of peas and can readily be felt on palpation before section. They tend to caseate, but excavation is uncommon. This form of tuberculosis is best seen in childhood, but is not limited to that period of life.

Syphilitic Disease of the Lung is very rare. In the adult gummata may occur anywhere in the lung tissue, but chiefly favour the region of the root, where they may

form irregular caseous masses, encapsuled by dense fibrous tissue which radiates into the lung. Stenosis of the trachea or of a bronchus, with secondary bronchiectatic changes, may result. The positive recognition of the disease turns upon the recognition of gummata in other organs, such as the liver and testis, and the elimination of tuberculosis by microscopical examination.

In children, still-born or dying shortly after birth, a syphilitic inflammation of the lung may occur in the form either of a white pneumonia or a diffuse interstitial inflammation. The two conditions may coexist. In the former the lung is bulky, solid, and, on section, dry, white and smooth. In the interstitial form the lung is firm, and presents to the naked eye a coarse reticular appearance. A microscopical examination is necessary to eliminate tuberculosis and confirm the character of the lesion.

Pneumonokonioses or Dust Diseases of the Lung.—The inhalation of irritant particles gives rise to chronic inflammatory foci. As a rule the lungs are firm and bulky, but they may be shrunken. Pleural adhesions are present and the lymphatics in the deeper layers of the visceral envelope contain gritty particles. The lung tissue varies in colour according to the nature of the irritant, the coal-miner's lung being black, the knife-grinder's red, and so on. Owing to the spread of irritation from the small bronchioles, the interstitial tissue of the lung is increased. Small gritty nodules of fibrous tissue containing the foreign particles can both be felt and seen. Tuberculosis may complicate these cases. The bronchial glands will be found affected in the same manner as the lung.

Carbonaceous deposits in the lungs and bronchial glands are very characteristic of town-dwellers. The lungs of adults are rarely found to retain their original pink colour.

Chronic Interstitial Pneumonia.—This chronic process is a rare sequel of acute inflammation, either pneumonic or bronchopneumonic. One or more lobes of the lung become densely fibrous and shrunken. The lower lobes are mainly affected; firm pleural adhesions are present, and more or less dilatation of the bronchi may coexist.

EXAMINATION OF THE HEART AND GREAT VESSELS

The pericardium having been opened and examined in the manner already indicated and cultures made from the heart's blood if necessary, the heart and the origins of the great vessels should be inspected *in situ* before the thoracic viscera are in any way disturbed.

Where **Pulmonary Embolism** or **Pulmonary Thrombosis** is suspected or where the cause of death is unknown, the condition of the pulmonary artery and the nature of its contents should be specially examined before the heart is removed from the body. The method of examination is as follows :

Incise the wall of the right ventricle well to the right of the septum and near the apex of the heart. Carefully insert a blade of the long scissors into the ventricle and slit up its anterior wall towards the base of the pulmonary artery, continue the incision into the artery well towards its left border and extend the cut into the right and left branches of the vessel. For a thorough exposure of the right branch it is necessary to divide the ascending aorta transversely.

The contents of the right ventricle and of the main branches of the pulmonary artery can now be examined without undue disturbance. The characters of the clots present should be determined (p. 77), and any obstruction of the lumen by embolisms or coiled clots can be detected.

If an embolus or thrombus is discovered in the vessel the examination may be continued in the following manner :

The ventricular incision should be prolonged through the tricuspid ring and the wall of the right auricle into the superior vena cava and thence into the innominate veins and their main tributaries. The intestines should then be removed (see p. 103) and the liver split vertically to expose the inferior vena cava in its whole course. This

vein must then be laid open from its cardiac orifice to its bifurcation and the incision carried along the internal iliac trunks and also along the external iliac and femoral veins into the legs.

Even though the main venous trunks appear free from thrombosis, clots may be found adherent to the wall of the profunda vein of the thigh or to some of the pelvic tributary veins, indicating the possibility that a continuation of the clot has been bodily detached and carried into the right side of the heart.

Proceed with the examination of the heart :

Inspect the anterior surface, paying attention to the following points: the relations of the various cavities to the anterior chest wall ; the position of the apex ; the relative degree to which the ventricles occupy the anterior surface ; the condition of distension of the right auricle ; the position of the conus arteriosus of the pulmonary artery ; the relations and relative sizes of the aortic and pulmonary stems. Note also whether the heart appears flaccid, or is firm and contracted.

Grasping the apex, lift the heart out of the pericardial sac and inspect its posterior surface together with the sinus obliquus of the pericardium and the points of entry of the veins into the auricles.

The preliminary inspection should also include observation of the degree to which the heart is encased in subendocardial fat and of the presence of any petechiæ or milk-spots on its surface.

Where congenital disease of the heart is suspected the condition of the ductus arteriosus and the course taken by the pulmonary veins should be determined before separation is effected. The condition of the ductus is investigated by opening the pulmonary artery along its anterior aspect and looking for an orifice behind and between the two main branches. Its patency should be tested with a probe. The course taken by the pulmonary veins may be ascertained by lifting up the heart and noting if these veins have their

normal relations to the lungs on the one hand and the left auricle on the other.

In cases of pulmonary atresia enlarged bronchial arteries may alone carry blood to the lungs.

The heart should now be separated from its connexions. To effect this the blade of the long scissors must be passed through the sinus transversus, behind the aortic and pulmonary trunks. The two arteries should be cut through one inch above their points of origin.

Lift up the apex of the heart to put on the stretch the veins which enter the posterior aspects of the auricles. With short scissors cut across, from below, first the pulmonary veins of the left lung, then the inferior vena cava, then the pulmonary veins of the right lung, and, finally, the superior vena cava. The veins should be cut as long as possible to avoid damage to the walls of the auricles.

The heart is now free, and a detailed examination should be carried out.

In some cases the pericardium is so firmly adherent that separation is impossible; both heart and pericardium must then be removed together, but no alteration is made in the subsequent procedure.

METHOD OF OPENING THE CAVITIES OF THE HEART AND OF EXAMINING THE VALVULAR ORIFICES, THE MYOCARDIUM, THE CORONARY ARTERIES AND THE CORONARY SINUS

Place the heart on the table with its anterior surface downwards. Open the right auricle by passing one blade of the scissors into the inferior vena cava and out of the superior, dividing the bridge of tissue between. From

the middle point of this incision another should be carried into the right auricular appendix, laying it open right to its tip (Figs. 8 and 9). The clot in the auricle is inspected and turned out, particular attention being directed to the contents of the appendix. Small globular thrombi may often be found in this recess and, by passing into the pulmonary artery, are believed to be the common cause of infarction of the lungs.

The condition of the right side of the interauricular septum and of the foramen ovale should be ascertained. A small valvular communication of the auricles through the foramen has no significance. Inspect the orifice of the coronary sinus and open up the coronary vein.

Having completed the examination of the right auricle, open the left in a somewhat similar manner. First define the orifices of the pulmonary veins. With scissors passed into the left upper and out of the left lower orifice make an incision connecting the two. Join the orifices of the two right veins in the same way, and finally cut transversely from the middle of one incision to the middle of the other. Complete the opening by laying open the auricular appendix.

The interior of the left auricle is then examined in the same way as the right. The endocardium of the left auricle is naturally thicker and more opaque than that of the other.

When the auricles are freely opened and all clots removed from their cavities it is possible to inspect the mitral and tricuspid orifices from above. A rough estimate of the size of the orifices can be formed by inserting the fingers. The mitral orifice should easily admit two fingers, as far as the first joint; the tricuspid, three (one finger for each cusp). The presence of stenosis is readily recognised, and the character of the orifices, whether buttonhole, funnel-shaped or irregular, should be noted. Care must be taken not to disturb any recent vegetations which may be present on the valves.

Further dissection of the heart should be postponed until the WATER TEST has been applied to the valves. Extract any clot which may be present and see that the aorta has been cut sufficiently short to admit a good view of its three valve segments from above. Grasp the inter-auricular septum between the forefingers and thumb of the left hand, close to the root of the aorta, to support the heart. Seize the anterior margin of the aorta with forceps to hold the vessel open. A gentle stream of water should now be allowed to fall on the valves either from a tap or a jug. The accuracy with which the cusps meet should be noticed ; if the valve is competent the water will remain supported in the stem of the aorta. In applying the test three fallacies must be guarded against : (1) incompetent valves may appear competent if the ventricle below them is allowed to become filled with water ; (2) the presence of clot in the orifice may prevent closure of the valves ; (3) if a large branch of the coronary artery has been cut through before the valve is tested a slow leakage may occur through that vessel.

The pulmonary valve may be tested in a similar fashion.

The mitral valve is tested in a different way. For this purpose an elongated conical nozzle connected with the water-supply is requisite. The heart resting by its anterior surface on the table, the nozzle is carefully inserted through the aortic orifice and the root of the aorta firmly grasped around it. The mitral valve being brought into full view from the left auricle, water pressure is applied. Any particles of clot in the mitral orifice must be removed, and by gradually increasing the pressure the competence of the valve may be gauged. Since the competence of the auriculo-ventricular valves during life depends in great degree on the active contraction of the surrounding muscle, the water test is only a rough one, but still it yields useful information.

The tricuspid valve can be tested by inserting the nozzle into the pulmonary artery and inspecting the valve from

the right auricle. The tricuspid valve normally leaks under high pressure, hence the test is not often applied to it.

The ventricles should now be examined. By palpation of the front of the heart determine the position of the ventricular septum. Incise the anterior wall of the right ventricle with a scalpel. Keep to the right of the septum and commence the incision mid-way between the base and apex of the ventricle, cutting downwards towards the apex. If the incision is begun too near the base a flap of the tricuspid valve may be injured. A blade of the long scissors should now be introduced into the incision and passed up close to the anterior wall and ventricular septum of the heart until it emerges from the cut end of the pulmonary artery. The scissors' blade may be passed through the tricuspid orifice by mistake unless it is kept close to the anterior wall of the ventricular cavity. Keep well towards the left margin of the pulmonary artery and cut upwards, taking care to pass between two of the pulmonary valve segments.

The interior of the right ventricle can now be inspected: clots should be carefully disentangled and removed. Determine their characters. Soft globular thrombi may be found entangled among the columnæ carneæ. Any undue opacity or thickening of the endocardium should not escape attention. Inspect the pulmonary valves and allow a stream of water to trickle on them from above so that they bulge away from their attachments: a good view of the bodies of the cusps is thus obtained.

The method of opening the left ventricle is similar to that adopted for the right. The long blade of the scissors is this time brought out of the aortic orifice, which also should be incised between its valve segments. The interior of the left ventricle is examined and the aortic valves scrutinised in the same manner as the pulmonary. Pay particular attention to the condition of the septal wall adjacent to the auriculo-ventricular bundle of His, near the undefended space. In cases where it is desirable to

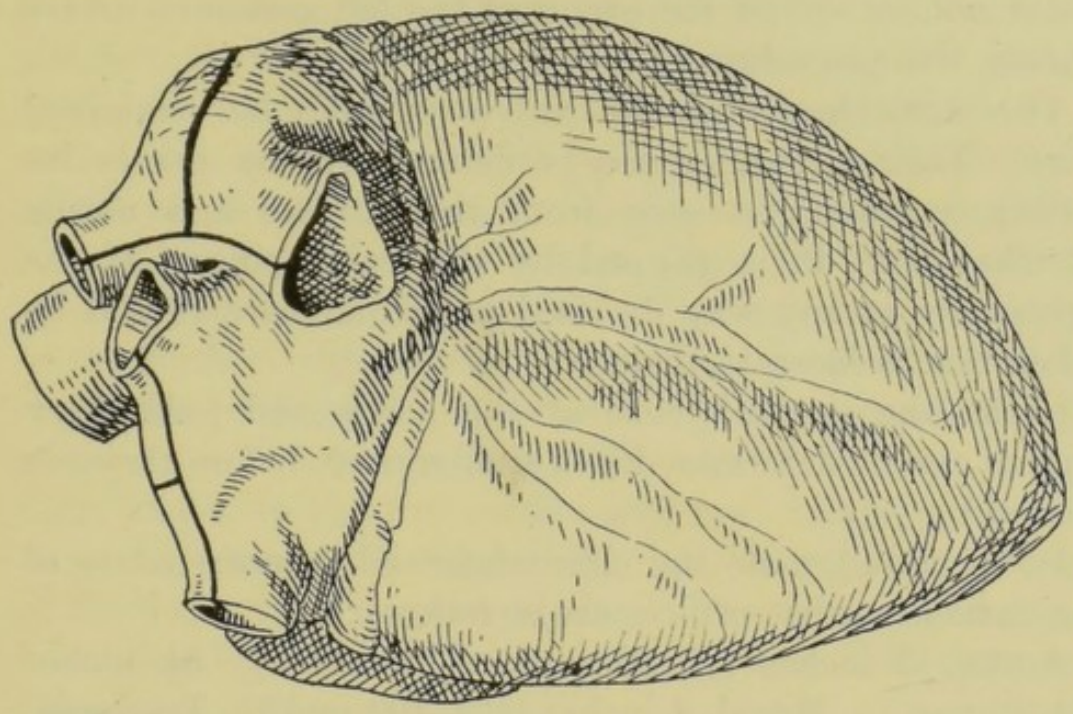

FIG. 9.—Incisions for opening the Auricles.

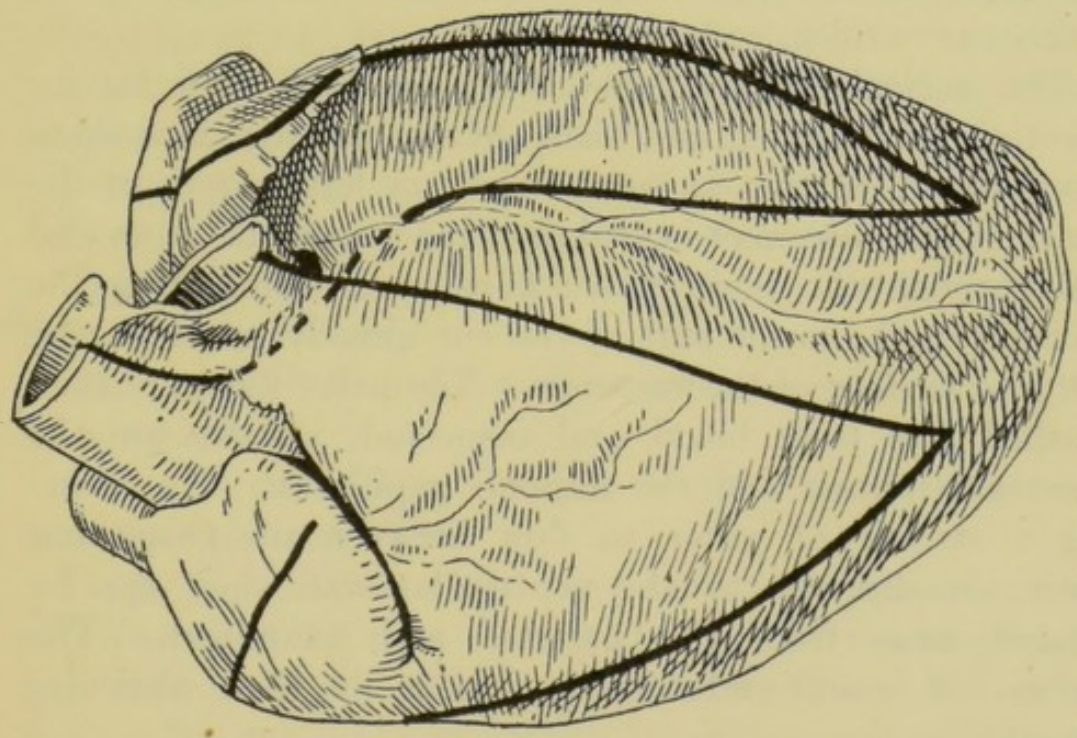

FIG. 8.—Incisions for opening the Ventricles.

preserve the pulmonary artery it can be dissected away from the aorta by a few snips of the scissors, care being taken not to wound the origin of the left coronary artery during the procedure.

The auriculo-ventricular valves should be examined next. Their circumference is determined by gently inserting a graduated cone from the auricles and noting at what level it is gripped by the valve rings. If the valves are in any way diseased particular care should be taken not to lacerate them.

(*Note*, the circumference of the aortic and pulmonary orifices can be determined in a similar way, before they are incised.)

In the adult male the circumferential measurements of the various cardiac orifices are as follows :

Aortic, 3 inches (77–80 mm.) ; Pulmonary, 3½ inches (89–92 mm.) ; Mitral, 4 inches (104–109 mm.) ; Tricuspid, 4½ inches (120–127 mm.). The measurements in the female are slightly less. As age advances stretching of the aortic orifice may cause it to measure more than the pulmonary orifice.

The auriculo-ventricular orifices are laid open for inspection by inserting one blade of the scissors into them from above, cutting along the right or left margin of the heart as the case may be, taking care to pass between and not through the main valve flaps, and terminating the incision in the lower part of the cut already made on the anterior surface of the ventricle. The valve flaps are then washed free from blood and inspected, as also are the chordæ tendineæ and the musculi papillares. By allowing a stream of water to flow between the cusps and their attachments to the ventricular wall they can be floated away from the parietes and examined. The lesions of recent endocarditis appear on their auricular aspects.

Examination of the Myocardium.—The condition of the muscular walls of the heart and also of the papillary

muscles is determined first by careful inspection of both inner and outer surfaces, not forgetting the cardiac septa, next by testing the resistance offered by the walls to pressure by the finger, remembering that cardiac muscle should be tough and not easily lacerable, and finally by a series of transverse incisions passing deeply into the walls of the heart and allowing an inspection of their condition. Any variations from the normal deep red colour of the myocardium should be noticed. The maximum thickness of the walls of the various cavities, but particularly of the ventricles, should be ascertained by measurements. To avoid error the section measured must have been cut at right angles to the free surface and not obliquely, and the muscular columns or ridges which project into the interior of the heart must not be included. The cavities also must be in diastole, for post-mortem contraction leads to apparent thickening of the walls.

The thickness of the walls of the various cavities is as follows :

That of the Left Ventricle varies from $\frac{1}{4}$ inch at the apex to $\frac{1}{2}$ or $\frac{3}{4}$ inch at auriculo-ventricular furrow.

That of the Right Ventricle varies from $\frac{1}{8}$ inch at the apex to $\frac{1}{4}$ inch at auriculo-ventricular furrow.

That of the Left Auricle measures about $\frac{1}{8}$ inch.

That of the Right Auricle measures about $\frac{1}{12}$ inch.

The capacity of each adult ventricle in diastole is about four ounces of blood.

The capacity of each adult auricle in diastole is said to be a little less.

Lastly, examine the condition of the Coronary Arteries and the Coronary Sinus.

Inspect the orifices of the coronary arteries in the sinuses of Valsalva at the root of the aorta. There are frequently two orifices for the right artery, one small and the other large. By passing a probe into its orifice each vessel may be cut down upon and opened just beyond the aorta, or the incisions may be carried onwards from the orifices

themselves. The arteries should be opened up with fine scissors, and on arriving at a point where the smallness of the vessels renders further opening difficult, a series of transverse incisions into the heart muscle at right angles to the vessel's course will enable its condition to be determined (Fig. 10). It is well to commence with the

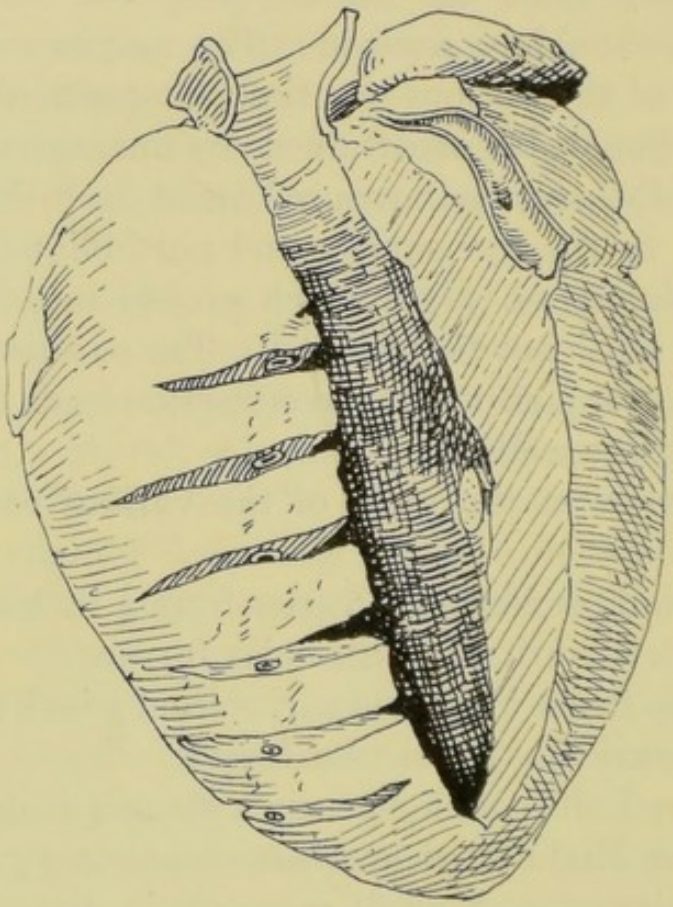

Fig. 10.—Examination of Coronary Artery.

examination of the left coronary artery, which runs to the left in the auriculo-ventricular groove and terminates on the posterior surface of the heart; it supplies a large branch to the anterior interventricular furrow. The right coronary artery runs in the right auriculo-ventricular groove and supplies branches to the right margin of the heart and the posterior interventricular furrow.

The coronary arteries may be obstructed at their orifices

by disease of the aorta or their lumina encroached upon by endarteritis.

If not already examined, the coronary sinus should be laid open in its whole length. It is often much dilated in cases of venous back-pressure from heart disease.

Weight of the Heart.—After it has been opened and all clots have been removed from its cavities, the heart should be weighed. The average weight in the adult male is 10½ oz. (297 grms.); in the adult female 9½ oz. (269 grms.). The heart increases somewhat in weight with increase in years.

DISTINCTIVE CHARACTERS OF ANTE-MORTEM AND POST-MORTEM BLOOD CLOTS

There are certain characteristics which may conclusively prove that a clot was formed *some time before death*. These are:

1. Firm adhesion of the clot to the wall of the vessel or the cavity of the aneurysm in which it was found. Adhesion of this kind is common in veins and venous sinuses, and not unusual in aneurysms, but is rarely a characteristic of clots formed in the arteries or in the heart. Entanglement of a clot among the fleshy columns of the heart, or in the tendons of the papillary muscles, must not be confounded with true adhesion.
2. Lamination of the clot. On transverse section laminated clot shows an alternation of red layers with others which are white and grey. If the clot be hardened in formalin the difference is accentuated. The presence of lamination indicates that the clot was formed while the blood was still circulating. Occasionally the outer layers of the clot form a pale, dry, tubular sheath, which contains soft red clot in its lumen; the outer layers are old, and the inner red layers recent. Conversely a pale dry clot of some standing may be encased in layers of recent coagulum.

3. Decolorisation and softening of the clot. A red thrombus becomes brownish in a week or ten days owing to the formation of hæmosiderin from the blood pigment. The clot finally assumes a dirty grey hue, and the central part becomes softened. Softening may occur rapidly in infected clots.

Clots which are formed *after death* from the stagnating blood are loose and gelatinous in consistence, and red or purple in colour. Thrombi of this character are often found in the auricles of the heart. The uppermost layers of such clots may be white owing to the separation of a buffy coat. It is said that, by noticing the relation of the two portions of the clot *in situ*, the attitude in which the body has lain after death may be inferred.

There is yet another variety of clot which presumably is formed slowly *a short time before death actually occurs*. These clots are firm, tenacious, and of a pale yellowish colour ; they have been likened to chicken fat. They are known as *paulo ante-mortem* clots, or agony clots. Thrombi of this nature may be found in the right cavities of the heart in pneumonia and septic diseases. They extend into the pulmonary artery, from which they can be drawn out, and if floated in water present a dendritic mould of the ramifications of that vessel. The impress of the pulmonary valves may be found stamped on a clot which was formed in contact with them.

When ante-mortem clot is found in the pulmonary artery the question always arises whether it is due to thrombosis of that vessel or to embolism. The characters of the clot may be sufficient to determine that it was formed ante-mortem, but may not indicate its seat of origin. The clot may be of such length that it obviously came from the veins, or it may present an impress which fits the pulmonary valves, and indicates its formation *in situ*. It is well to bear in mind that there also are comparatively large valves in the femoral veins. Sometimes examination of the clot may disclose an embolus encapsuled by a recent thrombosis. Occasionally a microscopical examination of the lung affords evidence that thrombosis originated in the small pulmonary arteries, and presumably spread backwards.

Atrophy of the Heart is usually part of general emaciation of the body, such as occurs in starvation, phthisis, malignant disease or diabetes. The epicardial fat disappears ; the heart is shrunken, and, in advanced cases, is of a dark brown colour, hence the term 'brown atrophy.' The coronary arteries are strikingly prominent and tortuous. They do not waste, and so appear large in comparison with the size of the heart. In extreme cases the heart may be reduced to half its normal weight.

Hypertrophy of the Heart is indicated by increase in its weight. The thickness of the walls of the affected cavity or cavities is greater than normal. The walls of a hypertrophied heart become tougher and stiffer, and consequently the cavities when laid open retain a somewhat hollow shape. This toughness is due to chronic venous congestion of the heart, and is usually the result of valvular lesions.

Hypertrophy is frequently associated with more or less dilatation. In such cases the cavities are markedly large and globular.

Hypertrophy without increase in size of the cardiac chambers is rare.

Hypertrophy with diminution of the cardiac chambers is apparent only, and due to post-mortem contraction of the heart.

Increase in mural thickness is rarely limited to one side of the heart, but the degree to which it predominates in the left or right ventricle should be determined. Considerable hypertrophy of the auricular wall is uncommon, but a moderate degree may be seen in the left auricle when mitral stenosis is present, and in the right auricle in cases of tricuspid stenosis.

Valve lesions are not the only causes of hypertrophy ; it may be found with adherent pericardium ; in athletes or those following laborious occupations ; and, in moderate degree, accompanies exophthalmic goitre and pregnancy. Preponderating left-sided hypertrophy may be the result of chronic renal disease or of arterio-sclerosis.

Hypertrophy chiefly marked on the right side may be

due to obstructive disease in the pulmonary circuit such as emphysema, chronic bronchitis, or fibrosis of the lungs.

In the absence of valve lesions, arterio-sclerosis or chronic renal disease, a combination of hypertrophied and dilated heart with passive congestion of the kidneys ('renal' heart and 'cardiac' kidney) is sometimes met with. It indicates chronic alcoholism or chronic alcoholism combined with syphilis.

In young people the contrast between the small aorta and pulmonary artery, and the size of the hypertrophied heart may be very striking, and to a certain degree indicate the duration of the disease.

Dilatation of the Heart.—Some degree of dilatation usually accompanies hypertrophy, and owes its origin to similar causes.

Acute dilatation occurs as a primary condition in acute infectious fevers and in rheumatism. It may also arise in simple or pernicious anæmia, and as the result of overstrain.

In pure dilatation, and in some cases of dilatation with hypertrophy, the myocardium is less firm than normal. When it is pale, easily lacerated by pressure with the finger, and the opened cavities fail to retain their normal contour owing to flabbiness of the walls, myocardial degeneration is present.

Globular thrombi tend to occur in the recesses of dilated hearts, especially in the cavities of the right side. The auricular appendages, the apices of the ventricles, and the recesses among the fleshy columns of the heart are favourite positions for such thrombi. The older clots appear as white masses which have liquefied internally, so that they form sacs of puriform fluid.

Fatty Infiltration of the Heart is an extension of the epicardial fat into the connective tissue between the muscular fasciculi. In this way the superficial layers of the myocardium may become replaced by fat. The right ventricle is more often affected than the left.

In obese subjects the auriculo-ventricular groove, the tracks of the coronary arteries, and the inferior border of the right ventricle may be heavily loaded with fat; in conjunction with this fatty infiltration may occur. A fatty

infiltration may sometimes be found in old and debilitated persons without general obesity.

Fatty Degeneration of the Heart is a grave condition. The minor forms may show no naked-eye evidence of the change, the severe forms alone can thus be recognised. The heart is usually dilated and the myocardium flabby, pale, and easily lacerable. In advanced cases the muscular tissue, when viewed from the interior, is seen to be flecked with yellowish bands or patches producing the so-called 'thrush-breast' appearance.

The most pronounced form of fatty degeneration occurs in poisoning with phosphorus or arsenic, but it is also well marked in grave anæmias, leukæmia, septic poisoning, and in various specific fevers. In diphtheria fatty deposit in the muscle fibres is extremely common, but needs microscopical examination and special staining for its demonstration.

Acute Simple Endocarditis.—In recent endocarditis the characteristic vegetations appear as rows of minute, glistening granules. These lie on the auricular aspects of the auriculo-ventricular valves; on the ventricular aspects of the aortic or pulmonary valves. Their occurrence on the tricuspid valve is exceptional, and on the pulmonary valve rare.

The vegetations do not arise in the first place on the free edges of the valves, but a little away from them. It is only when endocarditis attacks a valve already thickened and deformed that the new vegetations are found on the free margins of the cusps.

Chronic Simple Endocarditis.—In chronic endocarditis the valve segments become opaque, thickened, stiffer than normal, and are often distorted. In the case of the semilunar cusps of the aortic orifice the lunulæ may no longer be recognisable, owing to thickening and contraction. The margins of contiguous cusps may become adherent, giving rise to stenosis of the orifice. Segments may become parted from their attachments and prolapse, causing serious incompetence. Calcareous masses may be deposited in the cusps, usually near their attached margins. The mitral orifice may be contracted

in a buttonhole, or, more rarely, a funnel-shaped form. Stenosis of the tricuspid orifice is not common.

The sclerotic process is not limited to the valve flaps. It may be evident in the chordæ tendineæ, causing thickening and shortening; or in the apices of the papillary muscles, causing fibrosis.

An opaque yellowish patch of atheroma in the base of the anterior mitral flap is frequently seen in old people, and does not necessarily give rise to incompetence.

In valvular incompetence the endocardium of the ventricles or auricles may show patches of thickening, which indicate the point of impact of a regurgitant stream. Such areas may be seen on the left side of the ventricular septum in certain cases of aortic incompetence, and on the posterior wall of the left auricle in consequence of mitral incompetence.

In mitral stenosis it is not uncommon to find the whole of the endocardium of the dilated left auricle more opaque than normal and the pulmonary arteries atheromatous.

Chronic endocarditis, limited to the aortic cusps, is generally associated with inflammatory changes at the root of the aorta, and often has a syphilitic origin (page 91). Examination of the coronary arteries should not be omitted in such cases.

The condition of the various chambers of the heart as to hypertrophy and dilatation should be investigated. With aortic incompetence there is always more or less dilatation of the left ventricle in addition to hypertrophy. In pure mitral stenosis the left ventricle escapes, although all the cavities behind it in the circulation suffer. But mitral stenosis is often associated with incompetence, and then the left ventricle will be enlarged. As the result of back-pressure the viscera are congested and valves not actually inflamed, e.g. the tricuspid, may be found incomplete.

Malignant Endocarditis or Ulcerative Endocarditis.—There is no sharp line of demarcation between simple and malignant endocarditis. Like the simple form, the latter attacks the mitral and aortic valves more commonly than those of the right side of the heart, but both sides may be involved. Endocarditis limited to the valves

of the right side is nearly always malignant in character. Malignant endocarditis mostly attacks valves already the sites of chronic inflammation. The character of the vegetations varies greatly, sometimes they are quite small, but as a general rule the malignant process is characterised by exuberant growth. There is a great tendency for the vegetations to extend to adjacent structures, such as the chordæ tendineæ, and the posterior wall of the left auricle in the case of the mitral valve; the aortic septum, the contiguous portion of the anterior segment of the mitral valve, or even the root of the aorta in the case of the aortic valves.

Extensive ulceration of the valve segments may occur; the softening of the cusps may lead to the formation of valvular aneurysms, or to perforations. A flapping valve may infect the endocardium against which it strikes. Chordæ tendineæ may rupture.

Widespread infective embolisms may occur, and the infarcted areas may suppurate. Cerebral infarction may lead to softening, but frequently, in young persons, causes hæmorrhage.

In malignant endocarditis the kidneys, as a rule, are swollen owing to tubal inflammation, and the spleen enlarged.

In some cases the focus of endocardial infection can be discovered: it may lie in the uterus (after childbirth), in a bone, such as, for example, the petrous bone or the femur, or in some viscus drained by the portal vein (portal pyæmia). The endocarditis may be the result of pneumonic, influenzal or other acute infection. In the majority of the cases no such focus or antecedents can be traced, and it is believed that in such instances a malignant 'rheumatic' infection may be the sole cause of the disease.

In all cases of malignant endocarditis it is important to make a bacteriological examination of the blood from the heart, the vegetations, the spleen, and from any secondary lesions.

Infarction of the Heart is due to obstructive disease of the coronary arteries. These may be found thickened, atheromatous, or thrombosed. The condition is rarely due to embolism. The infarcted area may be hæmorrhagic, white, or in a transitional condition. Infarction of the

heart may cause a localised bulging of the wall, known as cardiac aneurysm, or a rupture. Septic embolisms of the coronary arteries may produce multiple abscesses in the heart wall. These also may lead to rupture.

Fibroid Disease of the Heart.—This is rare. It may be a terminal result of obstruction of the coronary arteries, or a sequel of gummatous inflammation of the myocardium. Patches of fibrous tissue replace the muscular substance. The left ventricle is almost exclusively affected, generally near its apex. Aneurysms or ruptures of the heart may result, but sudden death may occur in fibroid disease, apart from these accidents.

Syphilitic Disease of the Heart may take the form of syphilitic arteritis with aortic endocarditis ; gummatous deposits ; or diffuse chronic myocarditis. Sudden death is apt to occur in these affections. Gummatous disease is especially prone to attack the interventricular septum in the neighbourhood of the auriculo-ventricular bundle of His. The Stokes-Adams syndrome may result.

(The bundle of His originates in the annular and septal fibres of the right auricle. It passes below the foramen ovale in the substance of the interauricular septum. After piercing the fibrous septum of the heart, it runs along the lower part of the pars membranacea or undefended spot, and plunges into the interventricular septum, where it divides into two branches for the ventricles. The right branch continues buried for a time, the left is more superficial.

The bundle is likely to be injured by a lesion at the lower edge of the pars membranacea, by extension of lesions from the adjacent parts of the auriculo-ventricular valves, by lesions of the central fibrous body of the heart, or of the branch of the right coronary artery which supplies the latter.)

Angina Pectoris.—Lesions of the root of the aorta (p. 91) or of the coronary arteries are usually found. Aortitis may involve the coronary orifices or aortic cusps. The coronary arteries may be thick, calcified, or thrombosed. The heart may be fibroid or infarcted.

Malformations of the Heart are rarely found in persons who attain adult life. The common malformation

is stenosis of the pulmonary artery or adjacent part of the right ventricle. Defects in the cardiac septa are not infrequent. It is advisable to seek for a patent ductus arteriosus by opening the stem of the pulmonary artery before the heart is removed. The heart may be examined in the ordinary way, but special attention should be paid to its venous connexions. Marked passive congestion of the lungs and viscera is usually present. The extremities may be clubbed and other malformations may coexist.

EXAMINATION OF THE THYROID GLAND

Dissect the sterno-hyoid and sterno-thyroid muscles from the front of the thyroid gland with scissors. Bear in mind that the right lobe is larger than the left, an inequality which is maintained in general enlargement. Expose the pyramidal lobe which passes upwards in front of the larynx. In cases of goitre the sterno-hyoid and sterno-thyroid muscles may be flattened out over the gland or actually occupy grooves in its surface. Notice the relation of the internal jugular vein and the carotid artery to the lateral lobe; also ascertain whether pressure appears to be exercised on the recurrent laryngeal nerves which lie in the sulcus between the trachea and œsophagus. The trachea may be compressed laterally.

Accessory thyroids may be found in the neighbourhood of the pyramidal lobe or thyro-glossal duct, or even at the root of the tongue near the foramen cæcum. They also may occur near the upper, lower or posterior borders of the lateral lobes.

The Parathyroid Glands are small and difficult to find. The microscope is necessary for their decisive recognition. Two may occur amongst the terminal branches of the inferior thyroid arteries and two are usually situated near the lower extremities of the lateral lobes. Parathyroids may be embedded in the thyroid itself.

The thyroid gland is examined by making a transverse incision in the isthmus, an incision in the long axis of each lateral lobe, and another in the long axis of the pyramid.

The **Weight of the Thyroid Gland** is about an ounce (25–30 grms.). It is generally rather heavier in females than in males.

Congenital Absence of the Thyroid may occur. Cretinism is well marked in such cases.

Atrophy of the Thyroid is characteristic of myxœdema and of cretinism, the gland consisting merely of capsule and connective tissue. When goitre is present in these diseases the gland is functionless. A certain degree of atrophy may occur as a senile change.

Parenchymatous Goitres are in part due to increase in the solid elements and in part to excess of colloid.

Cystic Goitres should be inspected for the presence of adenomatous tissue, since large cavities may be due to the liquefaction of adenomata. Sometimes the cysts contain blood, and in old cases calcification of the cyst wall may occur. Small cysts may be due to retention of colloid.

Adenomata of the Thyroid occur as definitely encapsuled masses, but even where there is no naked-eye evidence of invasion the tumours may be malignant.

Exophthalmic Goitre. — The gland is uniformly enlarged ; the surface smooth and rather pale ; the interior firm, dense, and uniformly fleshy on section. The enlargement is due to epithelial proliferation ; there is little or no colloid. The vessels are not engorged. Exceptionally the thyroid is adenomatous or cystic in this disease. Enlargement of the thymus gland is present also, and sometimes a general enlargement of the lymphatic glands. The body is wasted, and a brownish pigmentation of the skin is often present. The heart is dilated.

Malignant Goitres are sarcomatous or carcinomatous. The latter form is usually glandular, although squamous-celled tumours do sometimes occur. Malignant goitres become adherent to the deeper structures of the neck and invade them. The air passages may be perforated, or

œdema of the glottis set up. Secondary growths in the bones may occur with carcinomata. Sarcomata may invade the veins of the neck. In all cases the condition of the adjacent lymphatic glands demands attention.

Lardaceous Disease of the Thyroid may be found in association with lardaceous disease elsewhere.

Gummatous and **Tuberculous** deposits in the thyroid gland are exceptional.

EXAMINATION OF THE THYMUS GLAND

The thymus gland should be inspected as soon as the thorax is opened. It is easily recognised in children, but in the adult, even though enlarged, it is apt to be overlooked owing to its being spread like a thin apron over the superior mediastinum and front of the pericardium.

Dissection of the gland can be carried out and its interior examined after removal with the rest of the thoracic viscera.

In infants and young children the gland forms a prominent, pink, fleshy mass in the superior mediastinum. It may extend as low as the fourth costal cartilage below and upwards into the root of the neck.

The **Average Weight** from birth to the age of two years is from $\frac{1}{4}$ oz. to $\frac{1}{2}$ oz. (7–14 grms. or more). A rapid diminution of the gland accompanied by degenerative changes occurs at puberty, and in the adult it may be difficult to differentiate the residue from the mediastinal connective tissue.

If any question of compression of the trachea by the thymus has arisen during life, it is well to dissect out the cervical part of the trachea and examine its interior from above before disarticulating the clavicles or disturbing the sternum. The effect of gently retracting the head should be investigated, as in some cases of enlarged thymus it is said that the lumen of the trachea may be completely occluded by this movement.

Enlargement of the Thymus in Children (*Lymphatism* or *status lymphaticus*).—There is a state met with in children where the thymus is enlarged in conjunction with other lymphoid structures, such as the lymphoid nodules on the base of the tongue, the tonsils, the pharyngeal tonsil, the lymphatic glands generally, the lymphoid follicles of the spleen, and the agminate glands (Peyer's patches) of the small intestine. This constitutes the condition known as **Lymphatism,** which is looked upon by some authorities as tending to induce glottic spasm or sudden death from syncope. Fatty degeneration or brown atrophy of the heart may coexist with the lymphoid changes. Hypoplasia of the aorta is also described.

Enlargement of the Thymus in Adults may occur as part of exophthalmic goitre, or in myxœdema, acromegaly, leukæmia, lymphadenoma, and some other conditions.

Inflammation of the Thymus may spread to it from the surrounding connective tissue.

Abscesses have been found in the gland.

Hæmorrhage has been known to result from severe thoracic injuries.

Tuberculosis of the Thymus may be simulated by caseation of the superior mediastinal glands, which are in intimate relation to the thymus.

Tumours of various kinds may occur.

Syphilitic Disease of the Thymus.—Under any circumstances the parenchyma of the thymus tends to break down into a fluid containing lymphocytes. This condition must not be mistaken for suppuration, and although looked upon by some as evidence of congenital syphilis, there appears to be no basis for the assumption.

EXAMINATION OF THE GREAT VEINS OF THE THORAX

The Great Veins of the Thorax, if they have not already been opened, should now be examined. Commencing at its cut end open up the superior vena cava with scissors

and continue the incision into the innominate and subclavian veins.

The intrathoracic portion of the inferior vena cava should also be inspected, and the condition of the pulmonary veins as they pass through the mediastinal tissues to the back of the right auricle should be ascertained.

In extensive venous thromboses it is important to determine, if possible, the point of origin of the thrombus. The actual site of commencement may sometimes be detected by the discovery that a portion of the thrombus is grey or reddish grey and adherent to the vessel wall; it may even be undergoing softening. The rest of the thrombus may be redder and fresher, which is evidence of its being a secondary formation.

Thrombosis of the Intrathoracic Veins may be due to pressure exerted on them by mediastinal tumours, aneurysms or enlarged glands.

Acute mediastinal inflammation or cardiac insufficiency from valvular lesions may also give rise to thrombosis in the veins of the thorax and arms.

Intrathoracic new growths often invade the lumina of the veins.

Sometimes the caval or pulmonary veins may be found obstructed by cicatrisation of the mediastinal tissues in which they lie.

An aneurysm of the aorta may establish a communication with the superior vena cava, causing great congestion of the head, neck, and arms.

EXAMINATION OF THE THORACIC AORTA

Inspect the exterior of the aorta for dilatations, bulgings or irregularities. By palpation ascertain its consistence and the presence of any thickenings in its walls. Test its elasticity by traction. Do not mistake the great sinus for a pathological condition. Open the ascending portion

from below with scissors and carry the incision upwards and backwards along the arch, in front of the origins of the great vessels. Turn the mass of thoracic viscera over and open the descending portion of the aorta from below by an incision in the posterior mid-line between the points of origin of the intercostal arteries. On arriving at the posterior part of the arch the two incisions may be made to meet or the posterior incision may be carried on into the left subclavian or left carotid trunk, but this should not be done if the thoracic duct has not yet been examined, or the termination of the latter will be injured.

The first incision in the ascending and transverse portions involves damage to the thymus gland and, if carried too far, to the left vagus nerve, where it gives off its recurrent laryngeal branch. These structures should therefore be examined before the aorta is opened.

Sponge the interior of the vessel, inspect its inner lining and also the orifices of the principal branches. Open these branches up.

A cicatrix at the top of the descending portion, immediately beyond the origin of the left subclavian artery, indicates the position of the ductus arteriosus.

If an aneurysm is present, its position and its relations to surrounding structures must be determined before the aorta is incised. In such case it is better to open the aorta on the side away from the origin of the aneurysm. Small aneurysms in the vicinity of the root of the left lung may not be discovered until the artery is opened up.

Diffuse Staining of the interior of the aorta may be present in fevers, septic conditions, and decomposition. It is due to the liberation of blood pigment which then permeates the intima. Yellow staining may be marked in cases of jaundice, and a pale yellow tinge may sometimes be seen in pernicious anæmia.

Recent Atheroma is very common. It occurs as soft, slightly prominent, dull yellow patches of various sizes.

Acute Infective Arteritis in the form of ulcerated patches with adherent fibrin sometimes attacks in the aorta or its large branches. Thrombosis of the latter may result.

Old Atheromatous Patches may become fibrous or calcified. The interior of the aorta sometimes shows great irregularity, owing to the presence of these patches. Calcified plates may lie exposed to the blood stream.

Syphilitic Disease of the Aorta is not rare. It is the common precursor of aneurysm. If soft, raised, gelatinous-looking swellings of considerable extent are found at the commencement of the vessel, syphilis should be suspected as a cause. Suspicion is also justified when there is an area of extensive scarring and puckering of the lining membrane of the vessel with small bluish patches of localised atrophy, which may be combined with thickening and rigidity of the vessel wall. Scarring of this nature may be limited to the ascending and transverse portions of the arch. The aortic valves and the orifices of the coronary arteries may be involved in the sclerotic process.

General Arterio-sclerosis, manifested by a thickening and stiffness of the walls of the arteries of all sizes, may be accompanied by some degree of atheroma, but this is not always so. Examine the kidneys and adrenals.

A slight dilatation of the ascending aorta is common as age advances, particularly in those who have followed laborious occupations.

Aneurysms of the Aorta may be fusiform, saccular or dissecting. The characters of the clots they contain, the structure of their walls, and their relations to surrounding parts should all be ascertained. Sometimes communication will be found to have occurred between an aneurysm and the pulmonary artery or the superior vena cava. The relations of important nerves (the recurrent laryngeal, the phrenic, the intercostal, or the thoracic sympathetic) to the sac must be ascertained. The recurrent laryngeal nerves may be recognised by the fact that the left turns under the arch of the aorta distal to the ductus arteriosus and the right turns under the subclavian artery. There may be evidence of pressure on the œsophagus, the air passages, or the roots of the lungs.

Rupture of the sac may have occurred. Aneurysms of the commencement of the aorta may burst into the pericardium, and cause sudden death. Rupture may take place into the pleura, œsophagus or mediastinal tissues. Sometimes the sac opens on the surface of the body.

In aneurysm near the spine determine the condition of the vertebral bodies and discs. The elastic intervertebral discs resist destruction in a remarkable way.

In the case of a dissecting aneurysm a rent may be found in the inner coats of the aorta. The extent of the extravasation in the walls of the artery should be determined (see p. 174).

Always look for evidence of antecedent arterial disease in cases of aneurysm. Syphilis is the most important factor.

If hypertrophy of the heart is present ascertain whether it is due to incompetence of the aortic valves or to pre-existing arterial disease. Aneurysm may occur without hypertrophy. The heart may be displaced downwards by aneurysm of the ascending or transverse portions of the aorta.

EXAMINATION OF THE MEDIASTINAL CONNECTIVE TISSUE AND MEDIASTINAL LYMPHATIC GLANDS

The mediastinal tissues exposed on removal of the anterior chest wall have already been inspected. A swollen and œdematous condition indicates the presence of acute inflammation which may be associated with disease of the lungs and serous sacs.

Induration of the connective tissues in front of the spinal column (**chronic mediastinitis**) will cause considerable resistance to the stripping out of the mass of thoracic viscera. Extensive chronic mediastinitis may be combined with obliteration of the pleural and pericardial sacs. The intrathoracic veins should be examined for signs of constriction in such cases. Evidence of tuberculosis should not be overlooked.

The condition of the glands which form the bronchial

group should be investigated. Those which lie in the bifurcation of the trachea may be exposed by dissecting off the œsophagus. The lungs should be drawn away from the sides of the trachea to expose the glands above each main bronchus. Incisions through the lung, carried well into the root, will lay bare glands embedded in the pulmonary tissue in this neighbourhood. All the glands should be freely incised.

Pathological changes may also be present in the glands of the superior mediastinum; these lie above and in front of the transverse arch of the aorta. The posterior mediastinal glands will be found alongside the aorta and œsophagus and at the back of the pericardium.

When new growths are found in the mediastinum their sites of origin should be determined if possible. The relations which neoplasms bear to the different glandular groups and also to the bronchi, œsophagus, thoracic nerves, arteries and veins are important. The veins in particular should be freely opened, since growth may sprout into their lumina. The glands at the root of the neck, in the axillæ and in the groins should be examined. The nature of an intrathoracic tumour is only satisfactorily determined by microscopical examination.

Acute Mediastinitis may be associated with inflammation of the lungs, pleuræ, pericardium, or other intrathoracic viscera. Inflammation may also extend from the neck. A swollen and sodden appearance of the connective tissue is indicative of the condition.

Mediastinal Abscess may originate in some of the structures which lie in the mediastinum or may spread from neighbouring parts. Abscesses in the retropharyngeal space of the neck may track downwards into the thorax. Small mediastinal abscesses sometimes occur in pneumonia.

Chronic Mediastinal Inflammation is indicated by induration of the connective tissue, and obliteration of the serous sacs of the thorax. Occasionally there is evidence that it is tuberculous. The large systemic veins

or the pulmonary veins at the back of the pericardium may be strangled by the contracting connective tissue. Occasionally the phrenic, vagus, or recurrent laryngeal nerves show signs of compression. Chronic peritonitis may be present.

Tuberculosis of the Bronchial Glands is common in childhood. The glands are usually caseous, and may infect the lung from its root or give rise to general miliary tuberculosis. Occasionally they ulcerate into the air or food passage, and should not be mistaken for pieces of cheese, which they resemble. Collapse of lung may result from pressure on the bronchi. The rest of the body should be examined for primary tuberculous foci. The lungs should be carefully inspected, attention being particularly directed to their lower lobes. Occasionally the mesenteric glands show more advanced changes than the bronchial. The intestines should be examined for tuberculous ulcers.

Hodgkin's Disease (*Lymphadenoma*).—In this disease the glands are greatly enlarged, forming conglomerate tumours which, however, on dissection, can be resolved into a number of discrete smooth glands. Matting or adhesion is rare. The glands vary much in consistence, but never suppurate. On section they appear grey and semitranslucent. The larger ones may be lobulated, owing to the presence of bands of connective tissue, which have a yellowish appearance. The disease is distinct from tuberculosis, but tuberculous infection of lymphadenomatous glands may occur. (See also pp. 122 and 136.)

Mediastinal Growths may be sarcomatous or carcinomatous. The structures from which the growths are most likely to arise are the mediastinal lymphatic glands, the œsophagus, or the bronchi. Growths in the lung may invade the mediastinum secondarily or vice versa. Sometimes the primary growth is in the mamma or an abdominal viscus. Growths in the brain may be secondary to growths in the mediastinum.

Dermoid Cysts containing hair and teeth may occur in the mediastinum. They sometimes suppurate and open into the pleura, causing empyema ; or into the lung.

CHAPTER III

EXAMINATION OF THE ABDOMINAL CAVITY

The abdomen having been opened (see p. 14) a systematic inspection of the abdominal contents should be carried out.

Notice the disposition of the great omentum and investigate any adhesions which it may have contracted in the neighbourhood of the hernial orifices, the vermiform appendix or elsewhere. Observe the amount and the colour of the fat which it contains. Having determined the position and appearance of the transverse colon, turn the omentum upwards over the thorax and inspect the central part of the abdominal cavity in which most of the coils of small intestine lie. The amount of distension of these coils, their colour, the appearance of their peritoneal coats and of the terminal vascular twigs should be observed. Note the presence of collapsed intestine, strictures, adhesions or localised inflammation of the bases of the Peyer's patches. Inflammatory exudation, pus, blood or even fæcal matter may be found between the coils. Ruptures or perforations may be discovered.

If obstruction of the small bowel is present, its nature and site should be determined, as far as possible, before any part is unduly disturbed or removal attempted. Finally, commencing at the duodeno-jejunal flexure, the whole of the small intestine should be palpated, down to its point of entrance into the cæcum. In certain cases the coils of small intestine may have prolapsed into the pelvis, and if so, note whether traction on the mesenteric

vessels has obstructed the duodenum and caused dilatation of the stomach.

The large intestine should be inspected next. Find the cæcum, which in some instances may lie in the pelvis, or may have failed to descend from its original position under cover of the liver. By tracing downwards the longitudinal bands of the colon, which converge towards the base of the appendix, find the latter; trace its whole length and define its exact position.

Trace the ascending colon to the hepatic flexure, carefully palpate the flexure, trace the transverse colon in the omentum across the abdomen, noting the presence of any acute flexures in it, palpate the splenic flexure and follow the descending colon thence to the crest of the ilium.

The iliac colon, which extends from the iliac crest to the brim of the true pelvis, should be examined, and the pelvic colon with its fan-shaped mesentery should be inspected in turn. Lastly, pass the hand into the recto-vesical pouch and investigate the condition of the rectum.

In the female inspect the uterus, ovaries and Fallopian tubes.

The transverse colon, pylorus, and sometimes also coils of small intestine in the neighbourhood of the gall-bladder, may be stained yellow by bile—this is not abnormal. Multiple invaginations of the small bowel, without signs of inflammation, may occur just before death ; they are known as 'agony intussusceptions' and have no pathological significance. The descending colon is frequently found contracted to a very small size; this is a natural state, and sometimes the transverse colon is in similar condition.

Dependent coils of the small intestine may show post-mortem lividity, which is distinguished from inflammation by the fact that the peritoneal coat retains its natural polish.

The stomach should be inspected *in situ*. Note its position, shape and size. If enlarged or prolapsed, the level of its lower border should be ascertained with relation to some fixed point such as the line joining the two anterior

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superior iliac spines. If the stomach is prolapsed, it may be possible to feel the pancreas above its greater curvature. Pass the right hand between the stomach and the diaphragm, noting the presence of any adhesions. Separate the lower border of the stomach from the colon by cutting through the great omentum, and inspect the interior of the lesser sac for adhesions or inflammation. Do not mistake the pancreatico-gastric and pancreatico-duodenal folds for inflammatory formations. The positions of perforations or new growths of the stomach should be ascertained before the viscus is removed. If the stomach is much dilated ascertain whether the duodenum also shares in the dilatation and the point at which the condition ceases. This may be where the mesenteric vessels cross, or farther on by the left side of the spine where the superincumbent stomach presses backwards.

The condition of the lymphatic glands in the vicinity of the stomach and also of those which lie in the mesentery of the small intestine should be ascertained, incisions being made into them where necessary.

Attention should next be directed to the liver. Any increase in size or irregularity of outline is at once obvious. Notice any alteration in its colour or smoothness. Pass the right hand over its convexity to determine the presence of any abnormal adhesions or projections. Lift up its lower border and inspect its under surface, noting the condition of the gall-bladder. Pass the left forefinger into the foramen of Winslow to determine the condition of that opening. By pinching the liver margin between the finger and thumb some idea of its consistence may be obtained.

The surface of the spleen should be investigated by passing the hand over it, between the fundus of the stomach and the diaphragm. In cases of suspected rupture the spleen should be inspected before removal. The gastro-splenic and lieno-renal folds of the peritoneum must not be mistaken for adhesions, neither should the tail of the

pancreas, which lies near the hilum of the spleen, be taken for a new growth on account of its hardness.

The kidneys should be palpated and their mobility ascertained before the colon is removed.

In Enteroptosis or Glénard's Disease, the stomach, transverse colon, small intestine and the right kidney occupy an abnormally low position. Exceptionally the left kidney, the spleen and sometimes even the liver are also prolapsed.

The Omentum.—The omentum may be heavily loaded with fat, but in those dead from chronic starvation, or from wasting diseases, fat may be entirely absent. The omentum is then represented by a peritoneal fold of extreme tenuity, mainly recognisable by the vessels it contains.

Fat Necrosis may occur in the omentum and other peritoneal folds; it may also be seen beneath the parietal peritoneum, and sometimes even in the pleuræ and pericardium. The areas of necrosis are usually small and widely scattered; the fat lobules present a dull white appearance not unlike suet. The discovery of fat necrosis should always lead to a careful examination of the pancreas.

Malignant Infiltration of the Omentum is generally secondary to carcinoma of the stomach or of the transverse colon. It may also occur in sarcomatous disease of the kidney, retroperitoneal glands, or other structures.

Tuberculous Disease of the Omentum may lead to considerable infiltration and thickening, and ultimately to caseation.

Acute Inflammation of the Omentum is indicated by a hard and injected condition with a dull, sticky surface. The condition is usually a local one, and depends upon inflammation of a viscus in the neighbourhood. Appendicitis or perforations of the stomach, bowel, or gall-bladder may cause it. Inflamed omentum may be found in a hernial sac or adherent to the back of a recent abdominal incision. Bands of omentum which have contracted adhesions may cause intestinal obstruction.

Lacerations, twists, or bruises of the omentum are sometimes found.

Hydatid Cysts may grow in the omentum and other peritoneal folds.

Hæmorrhagic Effusion into the Peritoneal Cavity.—A small quantity of bloody fluid with no evidence of peritonitis may often be found in cases where abdominal exploration has been recently performed for intestinal obstruction or other disease.

A blood-stained exudation may also occur in advanced decomposition.

Hæmorrhage into the peritoneum may be the consequence of rupture of the liver or spleen. Sometimes aneurysms burst into the sac, and tubal pregnancy should not be overlooked as a cause of bleeding. In some cases of tubal pregnancy the blood is encysted in the lower part of the abdomen.

A deeply blood-stained effusion into the lesser sac may be due to acute pancreatitis.

Acute Peritonitis is indicated in its early stages by loss of the normal peritoneal gloss, slight stickiness of the surface of the membrane, and congestion of the intestinal coils. Those portions of the coils which press against each other may be free from injection.

In more advanced peritonitis the coils become distended with gas and liquid; soft, sticky adhesions occur, and a peritoneal exudate is poured out which rapidly becomes purulent.

Although acute peritonitis may occur as the result of certain acute general infections, including pneumonia, or as a terminal result in renal disease, yet its origin is commonly due to a lesion of some viscus which lies in direct relation with the serous sac. Thorough search must always be made for the focus of infection, particular attention being paid to the vermiform appendix, the stomach, duodenum, gall-bladder, pelvic viscera, and pancreas.

Localised Abscesses may occur in the peritoneal cavity. The exact positions and boundaries of these should be determined before the adhesions are disturbed. The point of origin of the infection, and the track followed in the peritoneum, should be ascertained if possible. Sometimes the abscess is at some distance from the primary

lesion; for instance, a right subphrenic abscess may be the result of appendicitis, or a pelvic abscess of the rupture of a gastric ulcer.

Gas in the Peritoneum may indicate its presence by the noise of its escape through the incision made when the abdomen is opened, or its existence may be inferred from the discovery that the liver has retreated considerably from the anterior abdominal wall, and bears evidence of acute inflammation of its peritoneal coat. When free gas is present, perforation of a hollow viscus should be suspected and sought for.

Simple Chronic Peritonitis is indicated by a thickening and milky opacity of the serous membrane. The mesenteries and omenta may become contracted as well as thickened, the bowel is then closely bound down to the posterior abdominal wall, and its length may be diminished. The liver may be distorted and curled up by contraction of its capsule. In some cases the peritoneum is much pigmented. Chronic general peritonitis is usually accompanied by ascites. It may be associated with chronic renal disease.

Chronic Local Peritonitis may present itself in the form of tough adhesions or localised patches of peritoneal thickening. It may depend upon cirrhosis or syphilitic disease of the liver, or on chronic infective processes in the neighbourhood of the spleen. When the vault of the diaphragm is involved pleural adhesions often coexist. Inflammation of the gall-bladder, chronic ulceration of the stomach or duodenum, repeated inflammation in the region of the vermiform appendix, or infections of the Fallopian tubes may any of them cause a chronic local peritonitis.

Patches and bands of chronic peritoneal thickening on the anterior surface of the liver may result from long continued constriction of the waist by belts or corsets.

Tuberculous Peritonitis may occur in an acute or a chronic form. It may or may not be accompanied by the effusion of fluid.

In acute miliary tuberculosis the tubercles may be scanty, grey, and small, or larger and yellow.

In chronic cases the tubercles become surrounded by

pigmented fibrous zones. Caseous masses may sometimes be found in the omentum, mesenteries and retroperitoneal lymphatic glands. Very tough adhesions may be present, and loculi of greenish pus or even of fæcal matter may lie between the intestinal coils.

Encysted collections of clear fluid sometimes result from tuberculous peritonitis and may be of considerable size. As the result of contraction of the mesentery and of peritoneal adhesions the small intestine may be bound into a coherent mass which defies all attempts to unravel it. An attempt should always be made to determine whether intestinal ulceration coexists with tuberculous peritonitis. The infection may sometimes be traced to the mesenteric glands or to the genital organs.

Extensive black pigmentation of the omentum, the mesenteries and the parietal peritoneum may be found in some cases of obsolete tuberculosis. Calcareous glands may be present in such cases and give a clue to the nature of the disease.

Disseminated Malignant Disease of the Peritoneum is secondary to malignant disease of the stomach, colon or some other part. The nodules of growth may, at first sight, be mistaken for tubercles. In cases of doubt a microscopical examination is necessary. When malignant nodules are widely spread in the peritoneum, attention should be directed to the condition of the thoracic duct, in or around which the growth may spread, also to its glandular chain and to the glands at the root of the neck.

Distended Subperitoneal Lymphatics show as white or yellow nodules or streaks beneath the peritoneum of the small intestine. They are apt to be mistaken for tubercles, new growths or areas of fat necrosis. On incision a milky fluid exudes. Obstruction of the lymphatics may be present.

EXAMINATION OF THE PORTAL VEIN

In cases of pylephlebitis the portal vein must be examined before any of the abdominal viscera are removed. (See also p. 178.)

Divide the duodenum immediately beyond the pylorus between two ligatures. Carefully separate the great omentum from the greater curvature of the stomach, noticing at the same time the condition of such veins as are divided. Pull the stomach over to the left and the omentum downwards. Recognise and isolate the portal trunk in front of the foramen of Winslow. Trace it down to the neck of the pancreas. Carefully sever the latter without wounding the vein. Reflect the body and tail of the pancreas to the left. The portal trunk, together with the splenic, superior mesenteric and inferior mesenteric tributaries, will now be exposed, and the course of the veins can be rapidly followed out by the use of the scalpel and scissors.

In some cases of portal pyæmia and liver abscesses no clots are found in the main portal branches, but some of the terminal venules, as for instance those in the region of the appendix and ascending colon, may be thrombosed.

Remove the Abdominal Viscera in this order:

(1) Small Intestine; (2) Large Intestine; (3) Spleen; (4) Kidneys and Adrenals; (5) Liver; (6) Stomach, Duodenum and Pancreas; (7) Pelvic Viscera.

The order in which the viscera are removed and the method of removal require special modification in certain cases. For instance, in dealing with lesions of the head of the pancreas, or of the bile-ducts, or cases of intestinal obstruction by gall-stones—the liver, stomach, duodenum and pancreas must be removed together without separation. In cases where there is reason to suspect disease of the urinary bladder or ureters and in cases of hydronephrosis or pyonephrosis—the kidneys, ureters and pelvic viscera should be removed in continuity.

In cases of Addison's disease, abdominal aneurysm or large adherent abdominal tumour it is often convenient to remove the abdominal viscera *en masse* and to study their relations from behind as well as from the front (p. 176).

EXAMINATION OF THE SMALL AND LARGE INTESTINES WITH THE VERMIFORM APPENDIX

Remove the small intestine first. Lift up the great omentum and find the point at which the jejunum emerges from behind the peritoneum close to the left side of the spinal column. Incise the mesentery for a short distance to allow the placing of a couple of ligatures on the gut. These should be at least two inches apart and tied tightly, or they may slip off when the bowel is divided. Divide the bowel between the ligatures. Draw the jejunum away from the spine with the left hand, to put its mesentery on the stretch. Holding the post-mortem knife like a fiddle-bow and drawing it to and fro across the mesentery, divide the latter as close to the bowel wall as possible. Shifting the grip of the left hand downwards as separation is effected, continue the division until a point three inches above the ileo-cæcal valve is reached. Again apply double ligatures at this point and divide the bowel between them, A sharp knife is essential, and it is often an advantage at this stage of the post-mortem examination to use the part of the blade near the handle, as it is less likely to have lost its keenness. If the mesentery is cut too long the bowel will fall into loops and cannot easily be opened ; if cut too close the bowel may be wounded, or may burst under water pressure when being washed out subsequently. A little extra care during the removal of the intestine will save much annoyance and delay later on.

Whilst the small intestine is being removed various points may be observed which have escaped attention at the preliminary inspection. For instance, 'agony intussusceptions' may be found, collapsed coils or internal herniæ may come into view, coils which at some time or other have been incarcerated may be detected by the presence of thickenings or adhesions, and a Meckel's diverticulum, if present, can hardly be missed.

WASHING, OPENING, AND EXAMINATION OF THE INTERIOR OF THE SMALL INTESTINE

With few exceptions the intestine should be thoroughly washed out before it is opened. The exceptions are when parasites, especially *Ankylostomata*, are suspected; when membranous casts may be present; when it is desirable to locate the source of some obscure hæmorrhage; or when portions of the gut are so gangrenous that water pressure is liable to cause rupture. When anastomoses have been effected they should always be carefully tested by water pressure before incising the bowel.

To facilitate washing, the intestine should be arranged in regular coils, longitudinal twists and sharp bends being avoided. It is convenient to run the water into the upper end of the intestine by means of a nozzle connected with the water supply. The pressure should be moderate. The characters of the stream issuing from the lower end should be observed and the irrigation persevered with until the washings are quite clear.

Then with a pair of blunt-ended bowel scissors lay open the intestine along the line of attachment of the mesentery (to avoid the Peyer's patches). The incision is effected by drawing the bowel on to the blade of the scissors with the left hand, rather than by pushing the scissors on with the right. If the mesentery has been properly divided in removing the bowel, the process of opening is extremely simple. If the bowel has been carelessly separated, the operation is both tedious and difficult.

The interior of the small bowel is examined by stretching it laterally whilst it is passed under a stream of water and inspected (Fig. 11). By careful palpation of its thickness between the finger and the thumb small infiltrations and incipient ulcerations, which might otherwise be overlooked, are easily detected.

Attention should be directed to the thickness, consistence

and colour of the bowel wall and to the condition of the mucous membrane, with especial reference to the lymphoid follicles and the Peyer's patches. The healthy mucous membrane is soft and pliable; its colour is pale and its surface somewhat shiny. It is thickest in the upper part of the bowel where the valvulæ conniventes are most developed. The lymphoid tissue is especially abundant

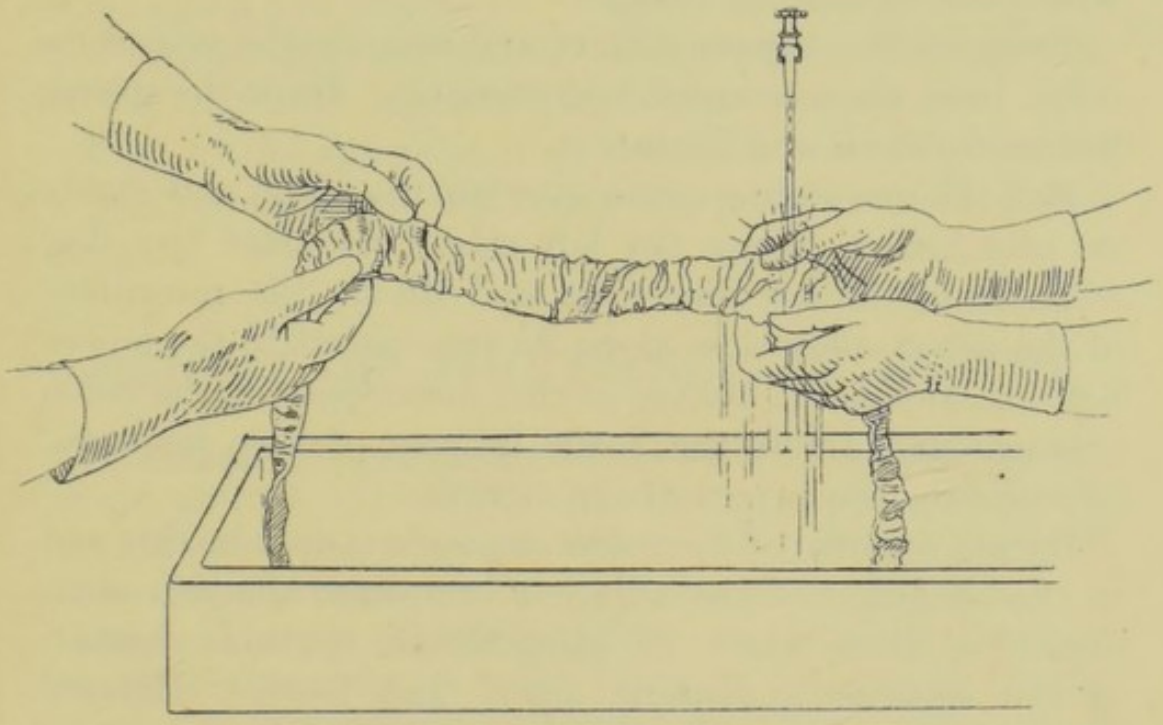

Fig. 11.—Examination of Intestine.

just above the ileo-cæcal valve; it is more prominent in children than in adults, and tends to atrophy as age advances. If the intestine has been carefully washed the normal mucous membrane is unstained by fæcal matter, but around recent ulcerations some staining of this character may often be observed.

REMOVAL OF THE LARGE INTESTINE AND VERMIFORM APPENDIX

Draw down the great omentum, spread it out and remove it by cutting it away from the lower border of the transverse colon.

Find the cæcum and define the position of the vermiform appendix, or the latter may unwittingly be severed and lost. Drawing the cæcum towards the mid-line, divide the peritoneum and connective tissues which bind it to the abdominal wall. Working upwards along the outer side, liberate the ascending colon in the same way. Be careful not to wound the descending part of the duodenum which lies close to the colon.

Extricate the hepatic flexure and separate the transverse colon from the mesocolon and stomach. Draw the splenic flexure forwards and liberate it.

Pull the descending colon over to the right and divide its attachments along the left side. Separate the iliac colon from the left iliac fossa and divide the mesentery of the pelvic colon close along its attachment to the bowel. Squeeze any fæcal matter in the lower part of the colon upwards and divide the bowel between double ligatures, opposite the upper part of the sacrum.

During removal of the colon attention should be directed to the condition of the adjacent lymphatic glands, particularly of those which lie alongside the ileo-colic branch of the superior mesenteric artery and receive afferents from the cæcum and vermiform appendix.

The veins passing from the appendix and colon should also be scrutinised for evidence of thrombosis or suppuration.

Wash out the cæcum and colon from above. Examine the ileo-cæcal valve by picking up a fold of the right aspect of the cæcum, incising it and continuing the incision down to the root of the appendix. Insert a blade of the scissors into the cut end of the ileum and slit up the latter along its mesenteric attachment as far as the valve. The valve itself should not be divided until both aspects have been inspected. Then pass the scissors through the valve and divide its anterior commissure ; continue the incision into the opening already made in the cæcum and thence along the whole colon, in the line of one of the longitudinal bands, since by following one of these the point of the

scissors is unlikely to be caught in the sacculi of the colon which lie between the tæniæ.

Cleanse and examine the mucous membrane. Carefully open the vermiform appendix in its whole length, along the side remote from the mesenteriolum. Notice the presence of strictures, obliterations of the lumen, cystic dilatations or foreign bodies. Cleanse the interior by sponging and inspect it. Palpate the wall to determine its thickness and consistence. The artery of the appendix and the adjacent lymphatic glands must be inspected. The position of the appendix in the abdomen and its relations to the peritoneum will have been determined before removal.

LESIONS OF THE SMALL INTESTINE

Intestinal Hypostases occur as widely separated, well-defined patches of lividity, without any evidence of peritoneal inflammation, which involve the dependent or posterior parts of the intestine.

Dark Purple Discoloration of the coils may be due to the presence of much blood in the interior of the intestine. The coloration disappears when the bowel is washed out.

Perforation of the Ileum.—The only common cause of perforation is typhoid ulceration. Tuberculous ulceration and damage by strangulation are occasional causes. The position of the perforation, the presence of adhesions, and the direction in which peritoneal infection has spread must all be noticed before the intestine is disturbed. The distance of the perforation from the duodeno-jejunal flexure and from the ileo-cæcal valve should be measured.

Obstruction of the Small Intestine.—The position of the obstruction should be ascertained. The coils of intestine below it will be collapsed, whilst those above will be distended or even hypertrophied if the obstruction is of some standing.

If a **Volvulus** of the small intestine is found, the direction of the twist and the number of turns should be noted, also

the distance of the obstruction from the commencement or end of the small bowel.

Strangulation by a Band is often due to a Meckel's Diverticulum. The arrangement of the parts involved may be very complicated. The site of the diverticulum, the attachment of its distal end, the way in which the obstructed coil is ensnared, and the condition of the intestinal wall at the site of construction must all be ascertained.

Adventitious Bands, which cause obstruction, are generally the results of past inflammation or of operations. They are most common in the lower part of the abdominal cavity and may arise in connexion with the vermiform appendix, the hernial apertures, the Fallopian tubes, or the mesenteric lymph glands.

Acute Intussusceptions occur in early childhood. They are often compound, but usually commence near the lower end of the ileum. The exact construction of the invagination can only be elucidated by the closest attention during reduction.

Chronic Intussusceptions may occur in the adult. They are often caused by an intestinal polyp.

Agony Intussusceptions are caused by irregular peristalsis at the time of death. They differ from true intussusceptions in that they are multiple, short, and may be retrograde as well as direct. They show no trace of peritoneal inflammation, and are said never to occur at the junction of the ileum and the cæcum, being restricted to the higher parts of the small bowel. They have no pathological importance.

Obstruction by a Gall-stone is uncommon. The position of the stone and the route by which it reached the bowel must be determined. Its presence in the intestine may cause a volvulus or set up local spasm which relaxes after death, leaving the stone loose.

Localised Gangrene or Necrosis of the Bowel may be the result of the various forms of obstruction. The damaged bowel is swollen, pulpy, and much discoloured. The tissues become friable, and the peritoneal coat loses its natural sheen.

Mesenteric Thrombosis may give rise to intense

purple discoloration of a segment of the intestine, going on to gangrene. The mesenteric arteries and veins must be dissected and examined for blood clots, emboli or evidence of obstructive inflammation. If the clots in the vessels are old they may be adherent, discoloured, and undergoing central softening.

Internal Hernia may occur near the duodeno-jejunal flexure to the left of the spine; near the transverse piece of the duodenum to the right of the spine; in a fossa near the cæcum; in a pouch near the root of the mesentery of the pelvic colon, or into the foramen of Winslow. In every case it is well to define the relations of named blood-vessels to the mouth of the sac, so that the hernia may be classified correctly.

Atrophy of the Intestine is indicated by a thin and translucent appearance of its wall. This condition is found in death from chronic starvation, and in infants who have long suffered from vomiting and diarrhœa. The intestines may waste in old age.

Hypertrophy of the muscular coats of the bowel may occur in consequence of long-continued partial obstruction. In Diabetes Mellitus the mucous membrane of the jejunum may appear unusually thick and heavy. (In this disease the liver is large and dark, the kidneys swollen and pale, the pancreas possibly sclerosed.)

Œdema of the Bowel may occur as a part of general œdema of the body and may simulate hypertrophy.

Shrunken and Thickened Intestine may be observed in some cases of chronic peritonitis where the mesenteries and peritoneal coats are thickened and contracted. A similar appearance may be presented when the parts are extensively infiltrated with malignant growth.

Hæmorrhages into the Bowel Wall.—Submucous petechiæ may be seen in ulcerative endocarditis or other forms of acute microbic infection. They may also occur in the various blood diseases and in chronic renal disease.

Large extravasations of the blood into the walls of the bowel may occur as the result of very acute enteritis, mesenteric thrombosis, strangulation or bruising of the bowel.

Passive Congestion of the mucous membrane may

be due to heart disease or portal obstruction. The mucous membrane is swollen and engorged, and an excess of mucus may be present.

Swelling of the Lymphoid Aggregations of the mucous membrane may occur in various infective conditions, including scarlet fever and diphtheria. It is very marked in typhoid fever, and may occur in zymotic diarrhœa, and in the condition known as *status lymphaticus*.

In Lymphadenoma the follicles and Peyer's patches undergo a notable enlargement, especially in the region of the ileo-cæcal valve, where the tissue is very abundant. Lymphadenomatous tissue may invade the intestine from adjacent lymph glands, encasing and infiltrating the bowel.

Lardaceous Disease of the mucous membrane causes it to appear pale and smooth, at the same time it feels thicker and stiffer than normal. Its appearance has been likened to wet wash-leather. The use of the iodine test causes the surface to appear stippled with brown spots, which correspond to the arteries of the villi, whilst the lymphoid follicles and Peyer's patches remain unaffected. (A watery solution of iodine with iodide of potassium should be employed for this test, and the intestine washed free from blood before it is immersed in the fluid.)

Catarrhal Enteritis is indicated by the presence of an excess of mucus which is tenacious and may be adherent. Vascular injection is inconstant, and not a certain sign of inflammation. In most cases of zymotic diarrhœa in childhood, and so-called *ptomaine poisoning* in adults, the naked-eye changes in the intestines are slight, the condition being due to acute microbic infection. The most that may be found is swelling of the lymphoid tissue and of the mesenteric glands.

Severe Forms of Enteritis occur as the result of irritant poisoning or in consequence of injury to the bowel wall. Acute pneumonia and chronic renal disease are sometimes complicated by acute inflammation of the intestine.

Acute enteritis may be limited in extent. The mucous membrane may show various degrees of congestion or necrosis ; the other coats of the bowel may be œdematous

or hæmorrhagic, and the inflammation may have spread to the peritoneal investment.

Follicular Ulcers are small erosions of the lymphoid follicles.

Tuberculous Ulcers may occur in the ileum, especially in its lower part. They are sometimes found in the cæcum and the vermiform appendix. The ulcers are due to the necrosis of caseous foci which form in the solitary follicles and the Peyer's patches. Even before ulceration has occurred the infiltrations may be felt as small nodular thickenings in the mucous membrane. Owing to their mode of formation the ulcers do not present large sloughs such as occur in typhoid fever. At first they are more or less circular in outline, but later tend to spread transversely, following the course of the encircling lymphatics and blood-vessels. The edges of the ulcers are infiltrated, raised, and firm ; the ulcerated surfaces have an eroded appearance and the bases are thickened. Minute yellow tubercles often occur in the peritoneum over areas corresponding to the sites of the ulcers within.

Tuberculous ulcers which have cicatrised may produce annular strictures. The chronicity of the tuberculous process allows time for the formation of protective external adhesions, so tuberculous ulcers rarely perforate the bowel during life.

General tuberculosis may cause small caseous tubercles to form in the mucous membrane, and these may even go on to the formation of small superficial ulcers.

Typhoid Ulcers occur in the Peyer's patches and solitary follicles of the ileum. They are most numerous and extensive just above the ileo-cæcal valve, but may also occur in the cæcum, appendix or colon. The infected lymphoid patches are at first greatly swollen; later on, ulceration and sloughing ensue. It is not uncommon for the greater part of a Peyer's patch to slough completely, but the separation may take place piecemeal. As a rule the sloughs are deeply bile-stained. The edges of the ulcers which result are thin and undermined, not infiltrated as in tubercle. The bases are smooth or, if the muscular coat is exposed, slightly ridged. If healing is in process the edges may be

rounded or shelving instead of overhanging. The ulcers are mainly longitudinal, corresponding to the arrangement of the Peyer's patches, but circular ulcers may occur. Owing to the abundance of lymphoid tissue just above the ileo-cæcal valve the ulceration may appear in irregular sinuous tracks. Here the sloughs may be detached as early as the fourteenth day of the disease, whilst higher up the ileum they may remain adherent for another week or even longer. Perforation is liable to take place. Cicatrisation ensues in the same order and may be complete by the fifth week. When death has occurred during a relapse healing ulcers formed during the first attack may coexist with the more recent lesions of the relapse.

White specks, possibly lymphatics, may often be seen on the peritoneal aspects of typhoid ulcers. They should not be mistaken for tubercles, which they resemble.

The lymphatic glands, near the angle of junction of the ileum and colon, should always be examined when any kind of ulceration is present.

Ulcers of Vascular Origin which tend to encircle the bowel may be met with as the results of embolism, thrombosis, syphilis, or lardaceous disease of the mesenteric vessels. They are uncommon.

Traumatic Ulceration of the small intestine may be due to strangulation of the bowel in a hernial orifice, or under a band.

Malignant Disease of the small intestine is rare. If a distinct tumour is present it is generally sarcomatous.

Congenital Strictures and **Sacculations** are occasionally met with.

LESIONS OF THE LARGE INTESTINE

Spasm of the Colon.—Considerable lengths of the colon may be firmly contracted and pale after death. This condition is more common in the descending colon than elsewhere.

Distension of the Colon.—Gaseous distension of the cæcum or colon may be the result of peritonitis or of

post-mortem decomposition. The extreme thinness of the walls serves to distinguish the condition from distension with hypertrophy, which may be the consequence of malignant strictures lower down the bowel, or a congenital condition (idiopathic hypertrophy of the colon, p. 116).

Volvulus of the Cæcum or of the ascending colon may also give rise to considerable distension of the bowel, due to obstruction. Determine the nature of the volvulus. The cæcum may be bent forwards and upwards on itself or there may be a twist of the bowel on its long axis. Abnormalities of the mesocolon may be present.

Pigmentation of the mucous membrane may result from chronic poisoning with lead, mercury, or bismuth. The pigment may be limited to the solitary follicles.

Acute Colitis.—In this condition the mucous membrane is congested, œdematous and soft. Sometimes there are foci of suppuration or superficial ulcerations. The inflammation may involve the whole length of the colon, or be limited to the pelvic portion.

In cases of poisoning with corrosive sublimate, arsenic, or antimony, intense inflammation and even ulceration of the colon may occur although the small intestine is normal. In poisoning with mineral acids the transverse colon may be involved by direct extension from the stomach.

Acute colitis sometimes occurs in acute pneumonia, chronic renal disease, or pyæmic conditions.

Chronic Simple Colitis is sometimes found in children. The mucous membrane is thickened, stiff, and granular; its surface may resemble morocco leather.

Membranous Colitis rarely proves fatal, and it is exceptional to find the tubular membranous casts of this disease post-mortem.

Ulcerative Colitis.—Ulceration may involve the whole colon or be limited to the pelvic colon, the descending colon, or the cæcum. The ulceration when extensive is often serpiginous. The islets of undermined and congested mucous membrane which remain may be mistaken for polypoid growths, and the denuded surface for normal mucous membrane. The absence of any signs of repair may be a marked feature. When the disease is very chronic

the colon becomes contracted and brittle; the surrounding subperitoneal tissue is indurated and adhesions may form.

Dysentery.—There is nothing in the naked-eye appearances of dysentery to distinguish it from acute and ulcerative colitis.

Typhoid Ulcers may occur in the colon. In this situation they are more or less circular, and in all, save very exceptional instances, the typical ulceration of the ileum coexists.

Tuberculous Ulcers are sometimes found in the cæcum. They present the same appearance as tuberculous ulcers in the small bowel (p. 111).

Hyperplastic Tuberculous Infiltration may occur in the cæcum or ascending colon. It is a rare variety of tuberculosis in which the intestinal wall is greatly thickened and the lumen of the bowel narrowed. The mucous membrane may be ulcerated, papillomatous, or intact. The condition closely simulates malignant disease, and in some instances can only be differentiated by microscopical examination.

Follicular Ulcers are superficial and of small size. Their edges are sharply cut. The ulcers occupy the lymphoid follicles and may be very numerous.

Hæmorrhagic Ulcers are small, shallow and discrete. They are not common, and their mode of origin is indicated by their name. They may occur in the chronic blood diseases, in chronic renal disease, or other conditions in which submucous hæmorrhages appear.

Ulcers of Vascular Origin, similar to those which occur in the small intestine, may sometimes be found in the colon (see p. 112).

Traumatic Ulcers due to strangulation or constriction of the bowel are also met with.

Trophic Ulceration.—Ulceration of the colon is sometimes associated with disease of the spinal cord. Whether the ulcers are really trophic is open to question.

Sacculation of the Pelvic Colon.—The terminal part of the colon may present small sacculations, due to hernial protrusions of the mucous membrane and atrophy of the muscular coat. The saccules correspond in position

with appendices epiploicæ, and their orifices are small and thin-lipped. Sometimes the pouches are large and arranged in series between the two longitudinal bands of the terminal part of the bowel. These little diverticula are prone to undergo inflammatory changes which lead to ulceration, pericolic adhesions, and even perforation.

Solitary Ulcers of uncertain pathology may be found in the cæcum or other parts of the large intestine. In cases of obstruction low down in the colon these ulcers may be limited to the cæcum; occasionally they rupture.

Actinomycosis of the Cæcum and Vermiform Appendix is a rare condition. The early lesions consist of small abscesses in the submucous coat; the sulphur grains of the fungus may sometimes be recognised. Extensive infiltration and ulceration of the mucous membrane may occur later, with the formation of fistulous tracks and the occurrence of infection in the liver (p. 136).

Polypoid Growths may be found in the colon; sometimes they are exceedingly numerous. Occasionally they are associated with carcinomatous strictures.

Carcinoma of the Colon generally occurs as an annular constriction, but sometimes it assumes a tubular form. In the annular form the exterior of the bowel is constricted, puckered, and often adherent to adjacent structures. The interior shows ulceration, with raised everted edges and a basis of white growth. Leakage may occur.

The junction of the iliac and pelvic portions of the colon is a favourite site for carcinomatous stricture; the left ureter, which lies behind the growth, may be obstructed. Other common sites for malignant growth are the flexures of the pelvic colon, the point where the descending colon crosses the bony prominence of the iliac crest, the splenic flexure, the hepatic flexure, and the cæcum.

In all cases the amount of obstruction should be determined before the stricture is incised. Foreign bodies such as plum-stones may lodge above the stricture. The condition of the lymphatic glands in connexion should be examined. Secondary growths may occur in the liver and rarely in the bones. The bowel above the obstruction is hypertrophied and dilated.

Idiopathic Dilatation of the Colon is a term applied to dilatation with hypertrophy of the pelvic colon when there is no mechanical obstruction. The mucous membrane may be ulcerated. The pathology of the condition is not understood.

LESIONS OF THE VERMIFORM APPENDIX

The Length and Position of the Vermiform Appendix vary considerably in different bodies; they have an important influence in determining the position and extension of peritoneal infection caused by appendicitis.

Foreign Bodies may be found in the appendix. Small masses of moulded fæces are common. Pins, fish-bones, minute metallic fragments, small shot, and parasites are found occasionally. Appendicitis may or may not coexist.

Acute Appendicitis is indicated by swelling of the whole or of part of the appendix, which is due to inflammatory infiltration, and is not limited to the mucous membrane only. Ulceration or gangrene and perforation may occur. The investing peritoneum may be acutely inflamed and recent adhesions present.

Chronic Appendicitis is a persistent inflammatory infiltration of the appendix, causing it to appear and feel stiff and thickened. The condition is usually more or less general.

Strictures, Obliterations and Cysts may result from past attacks of inflammation.

Typhoid and Tuberculous Ulcers may occur in the appendix. Their nature is evident from the lesions of the ileum or cæcum which accompany them.

Actinomycosis of the appendix presents features identical with those already described as characteristic of actinomycosis of the cæcum (p. 115).

Intussusception.—Although the appendix is often drawn into the sheath of an advancing intussusception, actual inversion of the appendix itself is exceptional.

Neoplasms rarely occur in the appendix.

EXAMINATION OF THE SPLEEN

Removal of the Spleen.—Having passed the right hand over the surface of the spleen to determine the presence or absence of adhesions, grasp the organ in the hollow of the left hand and carefully draw it forwards for inspection. Next cut through the lieno-renal and gastro-splenic folds, close to the hilum, taking care not to damage the tail of the pancreas, which is adjacent. Observe the condition of the lymphatic glands, which are close to the point of entry of the vessels, and also the condition of the vessels themselves. Small accessory spleens are often present in the neighbourhood.

If adhesions are present between the spleen and the diaphragm care must be taken to break them down without lacerating the splenic substance. The presence of any adhesions at the base of the left lung will already have been noticed.

When dealing with considerable enlargements of the spleen of unknown origin it is always advisable to remove the organ together with the pancreas, stomach, and splenic vessels, so that the artery and vein may be opened (see p. 155).

After removal, the weight, size, shape and consistence of the spleen should be determined. The condition of the capsule and of the parenchyma, including the Malpighian bodies and fibrous trabeculæ, should be investigated. The splenic vessels and adjacent lymphatic glands should be examined.

An abnormally firm spleen tends to retain its contour and surface markings after removal from the body. A soft spleen flattens out and loses its original shape. The surface, being covered by peritoneum, should be firm and polished. The dark red tint of the spleen pulp shows faintly through the capsule. The part of the spleen which lies in contact with the colon, and sometimes also the

gastric surface, may be stained a bluish-black colour by the influence of intestinal gases after death.

The interior of the spleen should be exposed by an incision made in the long axis of the organ (Fig. 12), but not quite through its whole thickness. Incisions should also be planned to pass through any depressions or swellings which may be detected on examination of the exterior. The cut surfaces should be inspected at once, for they

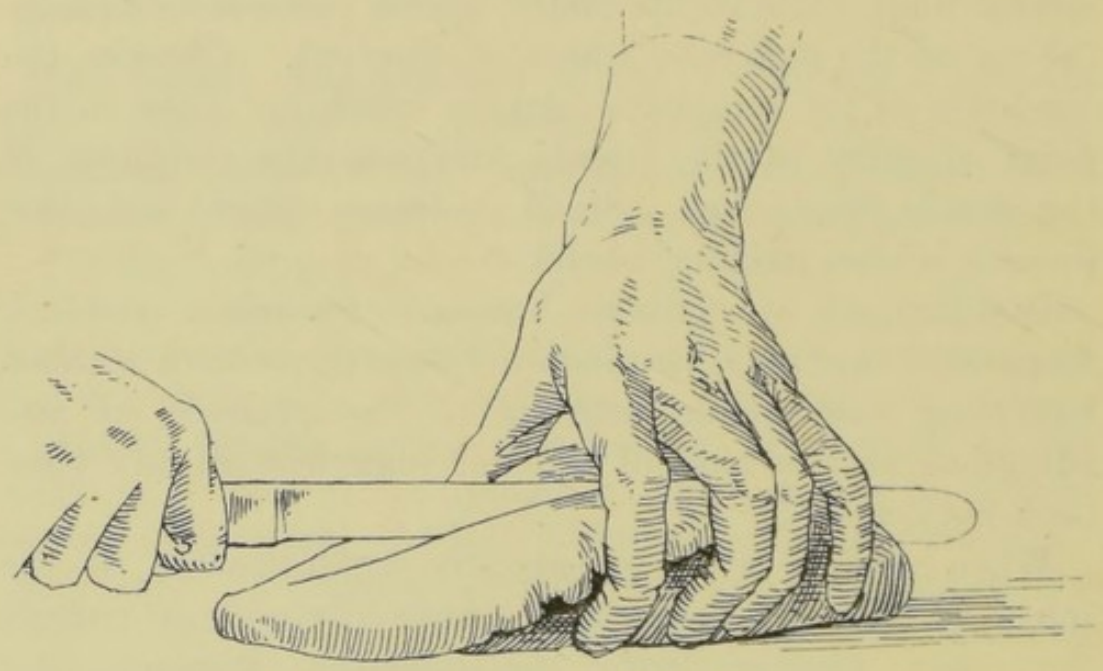

Fig. 12.—Incision of the Spleen.

readily oxidise, becoming much lighter in tint. The normal tint is a dull reddish brown.

A bacteriological examination will throw light on infective conditions.

Weight of the Spleen.—The weight of the healthy spleen varies considerably, being greatest during the full tide of digestion, a few hours after a meal. The average weight in the healthy adult may be taken as from six to seven ounces (170 to 198 grammes), but it varies from five to eight ounces.

Atrophy of the Spleen is indicated by diminution in size, wrinkling of the capsule, wasting of the pulp, and atrophy of the Malpighian bodies. The trabeculæ are

unduly prominent, and the vessels may be thickened. Atrophy is a senile change, but may sometimes occur in starvation.

Shrinkage of the Spleen may result from severe loss of blood or diminution of body fluids in consequence of severe diarrhœa and vomiting. The organ shrinks; its capsule appears thickened owing to contraction, and may be wrinkled. If hæmorrhage caused the shrinkage the parenchyma is anæmic.

Enlargement of the Spleen occurs in many diverse conditions. The greatest enlargement occurs in leukæmia, splenic anæmia, lymphadenoma, cirrhosis of the liver, malaria, typhoid fever, and ulcerative endocarditis. There are certain obscure tropical fevers, other than malarial, in which the spleen is enlarged. Moderate enlargement is common in specific fevers and other infective conditions. In early childhood enlargement may be due to congenital syphilis, rickets, or the splenic anæmia of infants.

Perisplenitis or Capsulitis.—This may be either acute or chronic, and each variety may be diffuse or localised.

Acute diffuse perisplenitis may occur when peritoneal inflammation involves the left subdiaphragmatic region. The inflammation is recent, and the surface of the spleen is coated with a yellowish inflammatory pellicle.

Chronic diffuse perisplenitis may be associated with chronic proliferative peritonitis in the upper half of the abdomen. The capsule is opaque, uniformly thickened, and sometimes of almost cartilaginous hardness. The surface may be pitted with shallow depressions, the results of unequal contraction of the fibrous tissue.

Acute localised perisplenitis may overlie recent infarcts or abscesses.

Chronic localised perisplenitis is common. It may appear in the form of 'lamellar fibromata,' which are hard, semi-cartilaginous plates in the capsule, or may overlie the bases of old infarcts, in which case the thickening is depressed below the general surface, and often sharply demarcated by a groove.

Acute Congestion of the Spleen.—As a result of acute congestion the spleen becomes enlarged and soft.

The parenchyma bulges above the cut surface, and may be almost diffluent. The cut surface is of uniform deep red tint. An acutely congested spleen indicates profound septic infection. The spleen is stuffed with cellular elements undergoing phagocytosis, and the proper splenic structures undergo proliferation, especially the Malpighian bodies which appear to be enlarged.

Chronic Venous Congestion of the Spleen occurs as the result of venous back-pressure in chronic heart disease or obstruction of the pulmonary circulation by lung disease. The passively congested spleen is enlarged and firm, so that instead of flattening out on removal, it retains its normal contour. The cut surface is of a dark plum colour, and the slightly thickened trabeculæ show up as white points or bands. Infarcts may be present. Cirrhosis of the liver also causes chronic venous congestion, but as a rule the spleen is larger and not so firm as in chronic heart or lung disease. The characters of a passively congested spleen may be much modified by the presence of malignant endocarditis or other acute septic congestion, the swelling increasing and the consistence diminishing.

Infarction of the Spleen occurs from embolism in endocarditis, aneurysm of the aorta, or disease of the splenic artery. In certain other conditions thrombotic infarcts may occur.

Simple infarcts may be either red or white, the difference in colour depending on the amount of blood which they contain. White infarcts are more common than red, but even in the recent state, may not be so bloodless as white infarcts of the kidney. Infarcts of some standing may be yellow in colour and their bases depressed below the surface of the spleen owing to shrinkage. The infarcted areas are more or less triangular on section, with their bases towards the capsule; sometimes a strip of normal tissue intervenes between the infarct and the surface. The wedge shape is due to the mode of distribution of the splenic arteries; the subcapsular strips of normal tissue are nourished by vessels from the capsule.

Occasionally the infarcted areas present an irregular

edge like the pattern of a dissected map. A recent infarct is swollen and surrounded by a zone of inflammatory reaction and congestion. An old infarct is firm and shrunken ; its surface is yellowish and dry, or fibrous. Old infarcts may give rise to depressed scars on the peritoneal surface. When infarction is present the left cavities of the heart, the aorta, and the splenic artery should be examined for possible sources of emboli. The infarcts which occur in certain infective fevers and in blood diseases may be due to thrombosis of the smaller splenic vessels. Portal thrombosis (splenic vein) may also lead to infarction, or a wandering spleen may become infarcted in consequence of torsion of its pedicle.

Abscess of the Spleen.—Embolic abscesses are preceded by infarctions ; they occur in ulcerative endocarditis and in pyæmia. Abscess of the spleen may also result from pylephlebitis or from direct infection of the spleen by an ulcer of the stomach or colon which has become adherent to it. Hydatids of the spleen also may suppurate.

Perisplenic Abscess.—In the majority of cases this is secondary to perforation of a gastric ulcer, but may occur in other infective conditions, or as a consequence of injury. The collection of pus is bounded above by the diaphragm and the left lobe of the liver, and below by the costo-colic fold. It is limited externally by the diaphragm covering the ribs, whilst adhesions between the great omentum or stomach and anterior abdominal wall seal it off in front. These abscesses may erode the splenic substance or set up portal pyæmia.

Tuberculosis of the Spleen.—Recent miliary tubercles in the parenchyma may occur in general tuberculosis ; they closely resemble the Malpighian bodies, and are only certainly distinguished when exceedingly numerous or assuming a yellow tint from caseation. The presence of tubercles on the peritoneal surface may throw light on the condition. Little tags of adhesion may accompany the peritoneal tubercles. Primary tuberculosis of the spleen is rare and may be associated with polycythæmia. Small caseous nodules are easily recognised.

Syphilitic Disease of the Spleen.—There is nothing specially characteristic of the enlarged spleen of the earlier stages of syphilitic infection. In congenital syphilis the spleen may be enlarged and firm.

Gummata of the spleen are rare. Owing to their yellow colour they somewhat resemble infarcts or tuberculous masses undergoing caseation. They may be distinguished from the former by their shape, and from the latter by the presence of a capsule of glistening fibrous tissue and intersection of the caseous substance by irregular bands of fibrosis. Deep scars may result from the absorption of gummata.

Lardaceous Disease of the Spleen (Waxy or Amyloid Disease).—The spleen is affected by lardaceous disease more frequently than any other organ. Two varieties of the affection are described, but they may occur in combination :

1. The sago spleen, in which the organ is pale, firm, and moderately swollen. The Malpighian bodies resemble sago grains, being enlarged and glistening owing to lardaceous change in their capillaries.
2. The diffuse waxy spleen, which is larger than the sago spleen, pale, firm, and dry. The walls of the large splenic blood sinuses and of the small arteries of the parenchyma are involved in this form. The Malpighian bodies may escape.

The iodine test should be applied (p. 110).

Lymphadenoma (Hodgkin's Disease).—In this disease the splenic enlargement is moderate as a rule, the weight seldom exceeding thirty ounces. In some cases the spleen is exceedingly large, whilst in a few its weight is not appreciably altered. The consistence is firm owing to chronic hyperplasia, and the Malpighian bodies may show more or less enlargement, giving rise to lymphadenomatous nodules. These nodules vary in size from that of a pea upwards, and, if large, may have a lobulated appearance owing to the fusion of adjacent masses ; on section they are white like suet rather than yellow and opaque ; definite nodules are not, however, invariably present. Lymphadenomatous spleens may be infarcted

from obstruction of splenic vessels; perisplenitis and adhesions may also be present, and the lymphatic glands of the hilum may show changes similar to those in the lymphatic glands of other parts of the body (p. 94).

Leukæmia (Myelæmia and Lymphocythæmia).—The splenic enlargement in leukæmia may be very great. It is more marked in myelæmia than in lymphocythæmia, and greater in the chronic cases than in those of short duration. In myelæmia the organ may attain a weight of 15 lbs. or more. The increase in size is uniform, and perisplenitis with adhesion is usually present. On section the surface of the spleen appears dry; its red colour varies in depth in different cases. Extensive infarctions of various ages may be present. As a rule the Malpighian bodies are indistinct, but sometimes they stand out as greyish swollen masses.

In myelæmia the liver is usually enlarged from leukæmic infiltration, sometimes to an enormous size. The kidneys also are large and pale from a similar cause. Sometimes leukæmic nodules may be recognised in the liver, kidneys, and spleen. Hæmorrhages may occur in the serous membranes and the various viscera. The yellow, fatty marrow in the shafts of the long bones is replaced by a pinkish or rarely by a puriform tissue.

In lymphocythæmia the glands throughout the body are enlarged. The tonsils, lymphoid tissue of the gastro-intestinal tract, and the thymus may be hyperplastic. Leucocytic infiltrations of the viscera may occur, and hæmorrhages, both petechial and massive, may b met with. The bone marrow is implicated in this form also.

Pernicious Anæmia.—The spleen may be slightly enlarged. Its consistence is variable, sometimes it is unduly firm. The parenchyma may give a blue coloration with potassium ferrocyanide and hydrochloric acid, indicating the presence of free iron, but this reaction, being sometimes obtained in cases of leukæmia and splenic anæmia of infants, is not absolutely distinctive of pernicious anæmia. (See also 'Liver,' p. 132.)

Splenic Anæmia.—In this disease the spleen is greatly enlarged and may attain a weight of sixty ounces

or more. It is red, tough, and sometimes infarcted. There is a general increase in the fibrous tissue and shrinkage of the Malpighian bodies. Perisplenitis is often present. Gastro-intestinal hæmorrhages may occur.

Banti's Disease.—This may be an advanced stage of splenic anæmia. In addition to fibro-adenitis of the spleen there is an interstitial hepatitis which tends to assume the form of multilobular cirrhosis. The disease is often characterised by a tendency to hæmorrhage in the skin and mucous membranes, and by ascites.

Splenomegalic Polycythæmia or Erythræmia.—The enlargement of the spleen in polycythæmia is variable. In some cases it is excessive, the enlarged organ reaching to the level of the umbilicus; in a few it is insignificant. The condition is one of chronic passive congestion with hyperplasia of the spleen elements. Red marrow may replace the yellow in the shafts of the long bones.

Malaria.—In recent acute cases the spleen is greatly enlarged, congested, and semi-diffluent. In cases of old standing the organ is large and firm with well-defined edges. The capsule and fibrous trabeculæ are thickened. A condition of melanæmia is also present, leading to the deposit of dark pigment in the organs and peritoneum.

Malignant Disease of the Spleen.—Neoplasms of the spleen may be carcinomatous or sarcomatous. Primary splenic new growths are uncommon. The viscus is more often invaded by contiguity than by metastatic deposits. Some chronically enlarged spleens reveal, on microscopical examination, a hyperplastic condition of the endothelium of the blood sinuses. This has been termed endothelioma, but is possibly only a stage in fibrous transformation.

EXAMINATION OF THE LIVER AND GALL-BLADDER

As a general rule the liver may be removed by itself, but when lesions of the gall-bladder, bile-ducts, or pancreas

are suspected the continuity of the biliary system should be preserved by removing the liver, bile-ducts, stomach, duodenum, and pancreas together (see p. 154).

Before touching the liver inspect the round ligament which still connects it with the umbilicus. If the ligament is healthy it may at once be divided, but if it shows signs of suppuration or is infiltrated with growth the condition of the navel and umbilical vessels must be ascertained.

Then raise the anterior edge of the liver and insert the forefinger into the foramen of Winslow. By palpation between the finger and thumb determine the position of the portal vein, hepatic artery, and common bile-duct, all of which lie in the hepato-duodenal ligament, which forms the anterior boundary of the foramen. Forcibly separate the liver from the duodenum by the thumb and forefingers of the left hand so that the hepato-duodenal ligament is rendered tense. Divide the ligament transversely, noting the condition of the common bile-duct, which lies to the right, of the hepatic artery, which lies to the left, and of the portal vein, which is posterior. Bile may be made to exude from the common duct by gently squeezing the gall-bladder: this indicates the patency of the upper bile passages.

If the portal vein is thrombosed it should not be divided, but a special dissection of the main trunk and its tributaries carried out (see pp. 101 and 178).

Divide the falciform ligament close to the liver in its whole extent. By pulling down the left lobe of the liver make tense the left lateral ligament and divide it. By traction on the right lobe define the right lateral ligament and divide this also. Finally, lift the liver from the abdomen in both hands and turn it over the cut edges of the ribs on the right side. Hold it in this position with the left hand whilst the remaining attachments, which include the lesser omentum and the inferior vena cava, are divided close to its under surface. Notice whether the vein is thrombosed. The viscus may then be removed.

Weight of the Liver.—Weigh the liver before incising it and again at the completion of the examination. Where the liver is greatly engorged with venous blood or with retained bile, or where large abscesses are present, the two weights may differ considerably.

The average weight of the liver in the adult is 50 to 60 ounces (male, 1579 grms; female, 1526 grms., Vierordt); it varies from 40 to 50 ounces in the female and from 48 to 58 ounces in the male. It is $\frac{1}{40}$th of the body weight in the adult, but at birth it is relatively twice as large, being equal to $\frac{1}{20}$th of the body weight. In the early fœtus it is relatively larger still.

An increase in the weight of the liver usually indicates enlargement. Slight and uniform enlargement occurs in cloudy swelling. More decided uniform enlargement is met with in consequence of fatty infiltration, chronic venous engorgement (nutmeg liver), lardaceous disease, monolobular cirrhosis, multilobular cirrhosis with fatty changes, leukæmia, lymphadenoma, obstruction of the bile ducts, infection of the portal or biliary channels, and diffuse cancerous infiltration.

Enlargement which is irregular in its distribution occurs as the result of hydatid disease, large abscesses, extensive gummatous infiltration with cirrhosis, and secondary malignant growths.

Diminution of the weight of the liver occurs in old age, in wasting diseases such as carcinoma of the œsophagus or stomach (provided no secondary growths are present in the liver itself), in starvation, and in acute yellow atrophy. Although there may be considerable shrinkage of the liver in advanced multilobular cirrhosis (hob-nailed liver), yet, owing to the density of the infiltrating fibrous tissue, the weight is not diminished proportionately to the diminution in size.

Having weighed the liver, make a preliminary inspection of all surfaces of the organ, noting the general appearance, size, colour, and configuration. The surface should be smooth and glistening save in the parts which have no

peritoneal covering. The capsule should be thin and semi-translucent, allowing the reddish brown colour of the liver substance to show uniformly through it. The liver, when removed from the body, tends to flatten out a little, and this tendency is increased when the substance is unduly soft. When the consistence of the organ is increased, the natural configuration is retained in a remarkable way.

After the preliminary inspection examine the gall-bladder and cystic duct, the portal vein, the hepatic veins and the lymphatic glands which lie near the portal fissure. Turn the under surface of the liver uppermost for this purpose.

The Gall-bladder may be distended or shrunken. Observe any evidence of acute inflammation in its neighbourhood or of chronic inflammation in the form of opacities, adhesions or contractions. If a bacteriological examination of the bile is desirable the surface of the gall-bladder should be thoroughly seared and an opening made into it with aseptic precautions. Culture tubes should be inoculated from the bile before it escapes. In an ordinary case the bladder is incised by snipping its fundus with scissors. The bile which escapes should be received into a suitable receptacle and the presence of gall-stones should not be overlooked. Small dark stones easily escape notice when surrounded by dark and inspissated bile. Completely slit up the gall-bladder towards its stalk, cleanse its mucous membrane, and examine it by inspection and palpation. The healthy mucous membrane is smooth and pliant. It presents a reticulated surface if it is not unduly stretched by distension. Do not overlook localised thickenings at the fundus of the gall-bladder; these sometimes prove to be malignant. Carry transverse incisions through the gall-bladder into the adjacent liver substance and inspect the cut surfaces.

The Portal Veins are next to be opened up with scissors and traced as far as possible into the liver. Observe the nature of their contents.

The Hepatic Arteries should be inspected and opened up if necessary.

Any enlarged glands in the portal fissure should be incised and examined. Ascertain whether they exert pressure on the other structures in the fissure.

The Hepatic Veins are exposed by opening up that portion

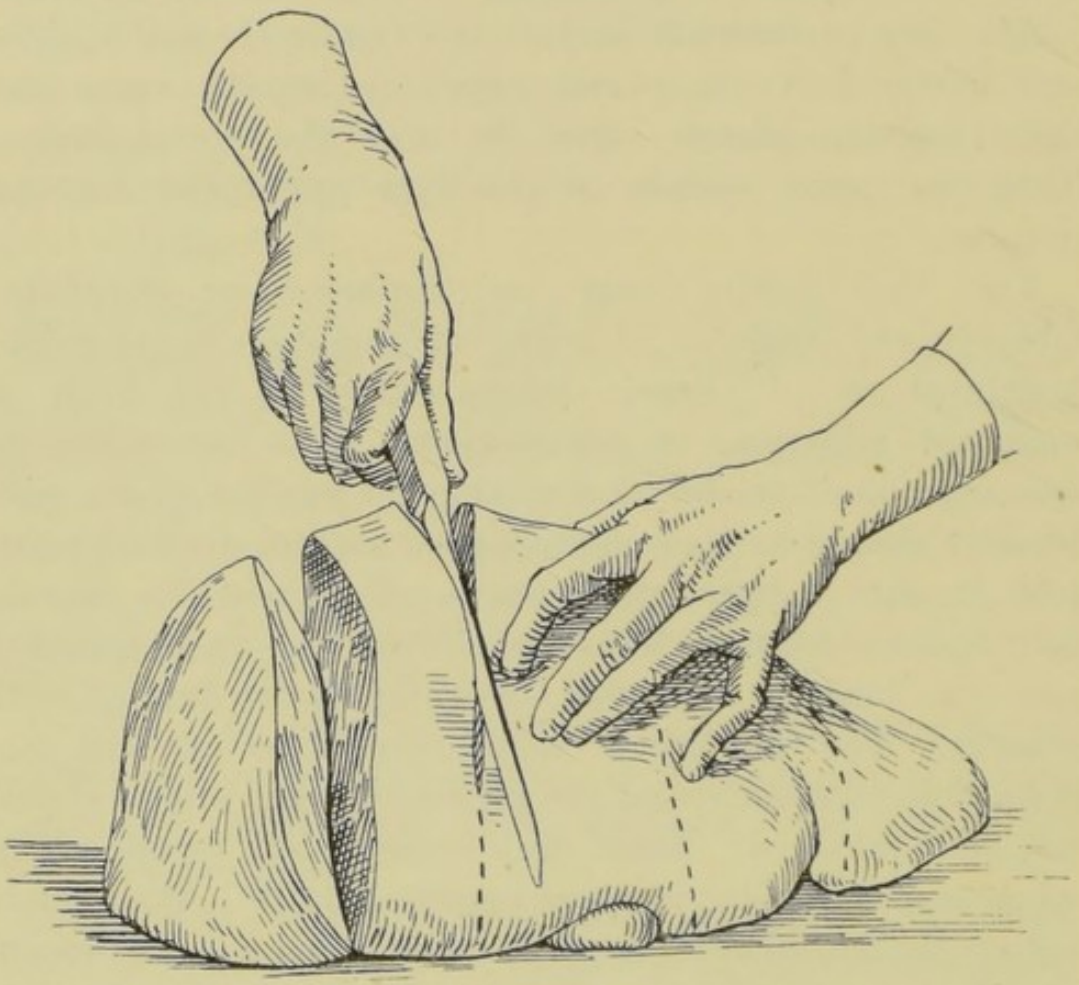

Fig. 13.—Incision of the Liver.

of the inferior vena cava which is attached to the posterior border of the liver. The hepatic trunks should be followed into the liver substance with the scissors. The portal and hepatic systems of veins cannot both be examined satisfactorily in the same liver.

Examine the falciform and coronary ligaments for enlarged veins. Their presence indicates obstruction to the blood flow through the portal system in the liver

and the establishment of a collateral circulation between the portal and general venous systems.

Incise the liver and examine its interior. Place the organ with its convex surface upwards ; steady it with the left hand ; with a sharp, long-bladed knife make a series of incisions parallel to the falciform ligament (Fig. 13). The slices should be about an inch thick and the incisions should not quite extend through the whole thickness of the liver substance, but should be deep enough to allow free inspection of the cut surfaces. The examination is rendered more complete by a second series of parallel incisions at right angles to the first. The incisions should be planned to pass through any abnormal depressions, elevations or patches of discoloration on the surface of the liver.

Gently scrape the blood from the cut surfaces with the knife and make a detailed examination. Pay particular attention to the characters of the liver substance, the condition of the portal canals which carry the portal veins, hepatic arteries and bile-ducts, and the gaping hepatic veins which run separately.

The interior of the liver should present a uniform, brownish-red colour. The colour is deeper in the aged and in wasted livers, but is still uniform.

Test the consistence of the liver by pressure on it with the pad of the thumb: normal liver substance is not very easily lacerated by pressure applied in this way.

Deformities of the Liver.—Extra fissures and lobes may occur as congenital deformities. A linguiform projection from the anterior edge of the right lobe, or more rarely of the left, may be acquired in consequence of tight lacing, but it is sometimes congenital. The gall-bladder may lie under a linguiform lobe or to the left of it. Oblique impressions, which correspond to the ribs, are sometimes seen low down on the right lobe. Radial grooves, which lodge ridges of the diaphragm, may occur on the upper surface. Like the linguiform lobes, these grooves and

impressions may be due to pressure or constriction. The liver may be deformed by disease such as syphilitic cirrhosis of congenital origin, advanced multilobular cirrhosis, malignant tumours or hydatids.

Discolorations of the Liver.—Irregularly shaped, superficial patches of a yellow colour may occur in various infective conditions: they indicate degenerative changes in the cells of the area involved. Anæmic areas may also be caused by pressure of the costal margin. Bile-stained areas may be found in the immediate vicinity of the gall-bladder. Permeation of the liver by intestinal gases may cause extensive superficial discoloration of a bluish tint, owing to the formation of iron sulphide. The discoloured areas are those which have lain in contact with the colon, and sometimes also with the stomach and duodenum. In advanced decomposition the staining may involve the greater part of the liver. A uniform yellow discoloration of the liver which is not merely superficial but involves the whole liver substance, indicates extreme fatty change. Flecks of hæmorrhage may appear on the surface in septic conditions and blood diseases.

Perihepatitis.—Inflammation of the capsule of the liver may be acute or chronic. Either variety may be diffuse or localised.

Acute perihepatitis is indicated by a sticky appearance of the capsule or the presence of a definite layer of soft, inflammatory exudation which may vary in thickness from a thin pellicle to a thick, spongy deposit. Extensive acute perihepatitis may be a part of a localised or a general peritonitis. Localised acute perihepatitis may overlie superficial inflammatory lesions of the liver.

Diffuse chronic perihepatitis gives rise to considerable fibrous thickening of the liver capsule and peritoneal covering. The liver is compressed or distorted by its shroud of contracting fibrous tissue. Chronic diffuse perihepatitis may be part of a general chronic peritonitis or be associated with cirrhosis of the liver. It may cause firm peritoneal adhesions; adhesions between the diaphragm and the base of the right lung may also be present.

Circumscribed areas of chronic perihepatitis may result

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from friction or pressure caused by tight belts or corsets. The position of the areas of thickening in such cases is generally sufficient to indicate their cause. Chronic localised perihepatitis may also overlie old gummata, hydatid cysts, etc.; it may occur in the neighbourhood of a chronically inflamed gall-bladder, or be due to adherent ulcers of the stomach or duodenum.

Atrophy of the Liver occurs in wasting diseases. It may be found in obstruction of the œsophagus or pylorus, or be the result of chronic starvation.

Acute Atrophy of the Liver is rare. The organ is much diminished in size and may be overridden by the intestines. Its capsule is wrinkled and its consistence flabby. The surface is greenish-yellow and often speckled with small subserous hæmorrhages. On section the cut surface is partly yellow and partly red, the red areas being those in which the atrophy is most advanced. (The body is jaundiced; the kidneys are swollen and may be the seats of petechial hæmorrhages; the spleen is enlarged and soft; the myocardium is pale and friable; subcutaneous, submucous, and subserous petechiæ may be present, and the intima of the large vessels is deeply blood-stained. Leucin and tyrosin may be found in the urine.)

Cloudy Swelling.—There is moderate, uniform enlargement of the liver. The colour is paler than normal, and the section has a lustreless, clouded appearance. The consistence is somewhat diminished. Cloudy swelling results from febrile affections of a toxic nature.

Fatty Liver.—The viscus is moderately enlarged, soft and its colour light brown, or, in advanced cases, yellow. The sections have a greasy appearance, and the lobules are outlined by pale, fatty zones which contrast with the darker central portions. Where the contrast is well marked the liver may present a speckled appearance to which the term 'fatty-nutmeg' is applied, but this must be distinguished from the much more pronounced mottling of the true nutmeg liver.

With extreme fatty change the colour of the liver may become uniformly yellow and its consistence almost pasty.

The liver may become extremely fatty in phthisis, some forms of cirrhosis, poisoning by phosphorus, arsenic, or antimony, and as the result of chronic alcoholism.

A marked fatty change in the liver together with an odour of acetone in the body is sometimes caused by delayed chloroform poisoning and by diabetes.

Cloudy swelling passes on to fatty degeneration, so fatty livers may be found in diphtheria and other infectious conditions.

Passive Congestion of the Liver.—The appearance of a liver which has suddenly become engorged with blood differs from that of a liver subject to prolonged venous back-pressure.

An acutely engorged liver is large, smooth on the surface, and purplish in colour, the hepatic veins being charged with large quantities of venous blood. The condition is due to recent heart failure. Livers presenting these characters may be found after deaths from cerebral hæmorrhage, narcotic poisoning, diphtheria, pneumonia, and some other causes.

The Nutmeg Liver is the result of chronic passive congestion. The liver is decidedly enlarged and firm. The capsule is but slightly thickened, and indications of the nutmeg mottling may be seen through it. Numerous dark red or purple spots and striæ are visible on the surfaces of the sections; they correspond to the distended sublobular and intralobular branches of the hepatic veins. The rest of the liver substance is pale or bile-stained. Nutmeg livers may be fatty, the fat lying in the central congested areas of the lobules and not, as usually stated, in their periphery.

Anæmia of the Liver is indicated by pallor. When death has resulted from profuse hæmorrhage the pallor may be extreme, and the bulk diminished somewhat by shrinkage.

Pernicious Anæmia leads to enlargement and more or less fatty change in the liver. If the cut surface is treated with ferrocyanide of potassium, and then with hydrochloric acid, the lobules become outlined by rings of Prussian blue, owing to the presence of iron in their

middle and outer zones. A slighter iron reaction may exceptionally be obtained in leukæmia and the anæmias of infancy; it is also present in cases of bronzed diabetes.

(Other indications of pernicious anæmia are: an anæmic condition of the body generally; a slight icteric tinge; the presence of subcutaneous and subserous petechiæ; abundance of bright yellow fat in the subcutaneous and subserous layers; dilatation of the heart owing to fatty changes; the presence of thrush-breast markings on the interior of the ventricles; replacement of the yellow marrow in the shafts of the long bones by red marrow, rich in megaloblasts; and prominence of the hæmolymph glands. Occasionally the iron reaction occurs in the spleen and kidneys as well as in the liver. Atrophy or sclerosis of the gastric mucosa may be present, and sometimes degeneration of the posterior, and also of the lateral, columns of the spinal cord. The presence or absence of suppuration in the tooth sockets should be ascertained.)

Lardaceous Liver.—The lardaceous, waxy, or amyloid liver is decidedly enlarged and firm. It retains its shape on removal from the body; is smooth, dense, and paler than normal. The cut surfaces have a glistening, semi-translucent appearance. The lobules are swollen, and the intermediate zone of each is marked out as a greyish, waxy-looking ring. The peripheral portions of the lobules may be yellowish. A weak solution of iodine and iodide of potassium in water stains the lardaceous portions dark brown. The body should be examined for signs of past syphilis, chronic suppurative bone disease, or chronic tuberculosis of lungs.

Multilobular Cirrhosis.—In advanced multilobular cirrhosis the size of the liver is much diminished. Although the shrinkage may be considerable there is not a proportional loss of weight, owing to the density of the infiltrating tissue. The surface is beset with nodular projections, which vary in diameter up to a third of an inch or more (hobnail liver). Depressions between and around the nodules are formed by shrinkage of fibrous tissue which permeates the portal canals. The liver capsule often presents a fine injection, and adhesions may be present.

The liver is difficult to cut and its tissue grates under the knife. On the cut surface a grey fibrous network is visible which separates and encloses islets of liver tissue. The islets consist of small groups of lobules; they vary in size and also in colour, being usually bright yellow, but sometimes reddish-brown and quite pasty from degenerative changes. Thrombi may sometimes be found in the portal trunks or the hepatic veins.

Sometimes multilobular cirrhosis is of such slight severity that the surface of the liver is merely granular in certain situations, and microscopical examination is necessary to confirm the diagnosis. Some cirrhotic livers are greatly increased in size owing to fatty infiltration.

The anastomoses between the portal and general venous systems may be enlarged, especially the veins at the lower end of the œsophagus, in the ligaments of the liver, and in the rectum. The spleen is enlarged; ascites is common; chronic peritonitis may occur.

Monolobular Cirrhosis or Biliary Cirrhosis.—In this form of cirrhosis the liver is much enlarged. The surface is smooth or at most slightly granular. The consistence is increased. The network of fibrous tissue is with difficulty recognised on the surface of sections, the strands being finer and the meshes, which envelop individual lobules, being smaller than in multilobular cirrhosis. The colour of the liver may be deep olive green in cases of some standing, owing to jaundice. The spleen is enlarged. In childhood monolobular cirrhosis may be associated with congenital obliteration of some part of the bile passages.

Pericellular Cirrhosis.—In congenital syphilis a diffuse form of cirrhosis may occur which is intercellular as well as interlobular in its distribution. The liver in such cases in enlarged and tough. On section it is paler than normal, and may present a mottled, marbled, or even flint-like appearance. Minute grey syphilomata, which resemble miliary tubercles, may sometimes be recognised on the cut surfaces; gummata are rare. The exact distribution of the cirrhosis can only be determined by microscopical examination. The spleen is enlarged.

Bronzed Diabetes.—In this disease the liver is enlarged and shows multilobular cirrhosis: its colour is deep red, and a blue coloration is obtained by the action of potassium ferrocyanide and hydrochloric acid. The hepatic artery is the site of obliterative endarteritis. The spleen is enlarged, firm, and contains pigment like the liver. The pancreas, too, is pigmented and enlarged (chronic interstitial fibrosis). Dark pigmentation of the skin is present.

Gummata of the Liver are but rarely seen in the post-mortem room, but the scars they leave are occasionally recognisable as irregular depressions of various sizes on the anterior surface of the organ. The nature of these depressions may sometimes be established by the discovery of syphilitic lesions elsewhere.

Gummata in their early stages are greyish-pink in colour without central necrosis. Later they become fibrous and irregular in shape. Small foci of caseation may occur in them, but are not invariably present. A fibrous capsule forms around old gummata and may extend irregularly into the surrounding liver substance. Livers which contain gummata should always be tested with iodine solution for the lardaceous reaction.

A combination of gummatous formations with cirrhosis may cause extreme irregularity in the shape of the liver. The surface becomes nodular and is deeply seamed; the interior is permeated by broad tracks of fibrous tissue enclosing gummata which may be undergoing various degenerative changes.

In some subjects of congenital syphilis the liver is so distorted in the later stages of the disease that the term 'botyroid' has been applied to it, from a fancied resemblance to a cluster of grapes.

Leukæmia.—In this disease the enlargement of the liver may be considerable, particularly in the lymphatic variety (lymphocythæmia). In the spleno-medullary form (myelæmia) enlargement may be absent. When present the increase in size is uniform, and the surface smooth. The consistence of the liver is diminished or, at most, slightly increased, since cirrhosis does not occur as

a result of leukæmia. The colour is paler than normal, and small petechial hæmorrhages may be present in the capsule. On section pallor may be uniform or limited to zones which outline and separate the lobules. Small lymphoid nodules, somewhat resembling miliary tubercles, may sometimes be recognised in the vicinity of the portal vessels, especially in the lymphatic form of the disease. (See 'Spleen,' p. 123 and also p. 275.)

Lymphadenoma.—The liver is uniformly enlarged. It may be the seat of lymphadenomatous growths which arise in the portal spaces and on section resemble suet. These growths vary in diameter up to half an inch or more. Old-standing lymphadenomatous nodules are large, and being definitely circumscribed, resemble neoplasms. Extensive fatty degeneration and sometimes passive congestion of the liver coexist. A perilobular cirrhosis may also occur. In some places a lardaceous reaction with iodine may be obtained. The portal obstruction may be sufficient to cause ascites. (See also pp. 94 and 122.)

Miliary Tuberculosis of the Liver is part of a generalised tuberculous infection. The viscus is enlarged and may be fatty. Grey tubercles are to be recognised in the peritoneal coat, but are inconspicuous or invisible in the interior.

Tuberculous Pericholangitis.—The liver is somewhat enlarged. On section localised tuberculous infiltrations, which vary in diameter up to a quarter of an inch or more, may be found in the portal spaces. The nodules are pale in colour at first, but later they erode the bile-ducts, becoming excavated and bile-stained. The lymphatic glands in the portal fissure may be caseous. The infection appears to reach the liver by the portal vein, the infection of the bile-ducts being a secondary process.

Actinomycosis.—An actinomycotic abscess of the liver consists of an aggregation of suppurating foci embedded in a reticulum of glistening fibrous tissue ; a characteristic honeycombed appearance being thus produced. The edges of the abscess present a crenate outline. Sulphur-coloured granules may sometimes be seen in the pus. The abscesses vary much in size, and enlargement of the liver depends

on the number and extent of the purulent collections. Infection of the liver is secondary to actinomycosis of some part of the alimentary tract (see p. 115).

Hydatid Cysts of the Liver may be solitary or multiple. They are recognised by their opaque white walls which, when incised, tend to curl inwards. The cysts, which vary much in size, are surrounded by adventitious capsules of fibrous tissue. The nature of their contents should be noticed: these may consist of daughter cysts, scolices, hydatid fluid merely (sterile cysts), or pus. Dead cysts may be bile-stained, shrunken, or even calcified. The discovery of hooklets affords valuable confirmatory evidence in doubtful cases.

An alveolar form of hydatid occurs sometimes. This appears as a congeries of small vesicles embedded in a gelatinous matrix and surrounded by a fibrous capsule. It may be mistaken for a neoplasm.

Multiple Abscesses of the Liver are due to infection carried by the portal vein (portal pyæmia), by the bile-ducts (suppurative cholangitis), or, more rarely, by the hepatic artery (general pyæmia). Abscesses, which vary in size, are scattered through the organ; small superficial abscesses are often visible beneath the capsule in the form of yellow, rosette-shaped areas. Where a section happens to follow the course of the infected vessels or ducts, racemose areas of suppuration may be seen (suppurative pylephlebitis). Attention must be directed to the portal vein, which should be thoroughly examined (p. 101): its gastric and intestinal drainage areas should be inspected, not forgetting the gall-bladder. The condition of the bile passages likewise calls for investigation. Rarely the focus of infection is in some distant part of the body, and the abscesses are due to arterial pyæmia. Portal pyæmia may itself infect the hepatic vein and cause endocarditis on the right side of the heart.

Solitary Abscesses of the Liver may be associated with dysentery or ulcerative colitis. Here again the portal veins and alimentary tract should be thoroughly examined.

Obstruction of the Common Bile-Duct causes swelling and greenish discoloration of the liver. In such

cases the liver, pancreas, and duodenum should be removed together, to ensure discovery of the point of obstruction and its cause. On section of the liver, dilated bile-ducts may be recognised. In old-standing cases their contents may be inspissated. If the gall-bladder has been drained by operation the liver may recede considerably.

Cystic Disease of the Liver is often associated with a cystic condition of the kidneys. The cysts are multiple and vary in size; they do not communicate with the bile-ducts. Their contents may be clear or blood-stained.

In infants, cystic disease of the liver may appear to the naked eye as a diffuse cirrhosis, but on microscopical examination the presence of cystic spaces in the fibrous tissue is revealed.

As rarities, small cysts of the liver which contain parasites may be mentioned ; they arise in connexion with the portal vein or bile-ducts.

Gas-containing cavities may arise in the liver owing to decomposition. Sometimes they are so numerous that the viscus will float in water. The term ' foaming liver ' has been applied to the condition ; it is frequently due to the presence of bacillus ærogenes capsulatus. By compressing the viscus under water bubbles of gas can be seen to exude from it.

Malignant Disease of the Liver is, as a general rule, secondary to disease elsewhere.

Carcinomatous growths are usually multiple and often project as nodules on the surface. The liver may attain an enormous size. The consistence of the growths varies in different cases. The projecting nodules are umbilicated and often surmounted by a fringe of injected vessels. Localised perihepatitis is frequent. On section the growths are of a white colour, but the central portions may be hæmorrhagic or yellow and necrotic. Suppuration is uncommon. Diffuse bile-staining may be present owing to obstructive jaundice. The primary growth should be sought in the portal area (stomach, colon, rectum, pancreas, etc.), but it may be elsewhere (œsophagus, breast, genital organs). Carcinomata of the gall-bladder or stomach may invade the liver by direct extension.

Primary Carcinoma of the Liver is rare. It may appear as a large, circumscribed tumour, with outlying nodules which represent metastases or, more rarely still, may present itself as an infiltrating fibrosis which is liable to be mistaken for cirrhosis. The presence of enlarged glands in the portal fissure may indicate its true nature. In all doubtful cases the microscope must decide.

Sarcomatous growths are uncommon in the liver.

Melanotic Tumours of the Liver are secondary. Search should be made for the primary growth in the eye or the skin.

Angiomata of the Liver occur as deep red, vascular tumours, which are usually solitary. They are more prone to appear beneath the capsule of the anterior surface of the right lobe than elsewhere. On section they appear smooth and somewhat depressed below the surface. They are rarely encapsuled.

Infarcts of the Liver are rare. They are usually hæmorrhagic and resemble angiomata. When an infarct is suspected, determine the condition of the main portal and hepatic veins, and also of the hepatic artery.

EXAMINATION OF THE KIDNEYS AND ADRENAL BODIES

Feel for the left kidney in the loin. Having determined its position, test its mobility by sliding it upwards and downwards beneath the peritoneum, noting the extent to which dislocation can be produced. The range of mobility of the kidney should always be tested before removal of the large intestine from the front of it. Incise the peritoneum vertically over the kidney and carefully enucleate the latter from its fatty capsule, taking care not to separate the true renal capsule by mistake. Next identify the adrenal body which lies near the upper extremity of the kidney. Determine the condition of the renal arteries and veins. Grasping the kidney in such a way

that the renal vessels lie in a cleft between two of the fingers of the left hand, draw it forwards. Divide the renal vessels, noticing the characters of their contents. Next isolate and inspect the ureter where it becomes continuous with the renal pelvis ; cut it across if it is healthy, but if thickened, dilated, or otherwise abnormal, the kidneys, ureters and urinary bladder should be removed in continuity (see p. 187). Urine which is turbid from precipitated salts may escape from the pelvis of the kidney when the ureter is cut across ; this should not be mistaken for a gush of pus.

The right kidney is removed in a similar way with the same precautions.

To facilitate a rapid recognition of the side to which the kidney belongs, make a distinctive cut in the left organ as soon as it is removed, or cut its ureter long.

One of the kidneys may be absent. Before concluding that this is the case search should be made for remnants of the organ by tracing the corresponding ureter upwards and the renal artery and vein outwards. The presence of fibrous remnants in the proper position of the kidney, together with a normally placed ureter, indicate that the atrophy is an acquired lesion. In such cases the opposite kidney is hypertrophied.

Sometimes one of the kidneys is misplaced and may be found in the iliac fossa, or on the brim of the true pelvis, or even below this point. In such a case the origin of the vascular supply of the gland should be determined.

Horseshoe kidney is characterised by the junction of the lower poles across the front of the spinal column ; the ureters pass over the front of the bridge of renal tissue which is thus formed.

As a general rule the adrenal bodies can be examined sufficiently by a series of transverse incisions as they lie *in situ*. It is important to recognise them at the time of removal of the kidneys, as their identification may be difficult later on. If the right adrenal has been overlooked

it may sometimes be found attached to the liver, being more adherent to this organ than it is to the kidney.

The left adrenal is easily dissected from its connective tissue bed, behind the peritoneum, care being taken to avoid injury to the tail of the pancreas which lies across it.

The right adrenal may be removed in similar fashion. It is closely attached to the adrenal impression of the liver and should be identified and separated before the latter organ is removed. To obtain a full view it may be necessary to divide the hepato-duodenal ligament, turn the liver upwards towards the thoracic cavity, and reflect the vena cava inferior after dividing it as close to the liver as possible. When the adrenals are the seats of tumours it is well to remove them still attached to the kidneys.

In cases of Addison's disease, where it is necessary to inspect contiguous structures, such as the solar plexus and the semilunar ganglia as well as the adjacent portion of the spinal column, removal of the abdominal viscera in mass and examination of the adrenals by posterior dissection is the ideal method (see p. 176).

EXAMINATION OF THE KIDNEYS

Abnormalities in mobility, position or blood supply of the kidneys have already been noticed during removal of the organs.

Difficulty in enucleation of the kidneys from their fatty capsules is the result of perirenal inflammation. This may be the sequel of inflammatory diseases of the kidney itself or the result of adjacent inflammation such as may arise from appendicitis or other conditions.

Weight of the Kidneys.—The kidneys must be weighed before they are incised, all extraneous fat being first removed. The weight of the two kidneys is about 11 oz. (312 grms.) in the adult male and somewhat less than this in the female. The left kidney of the adult is a little ($\frac{1}{2}$ oz.) heavier than the right. In infants the kidneys

are often equal in weight. When drawing deductions from the size and weight of the kidneys the degree of development of the body from which they were taken should be borne in mind, and also inequalities in the bulk of the two organs.

Next inspect the exterior of the kidneys, noticing any discolorations or depressions. After the preliminary inspection incise each one. To effect this the viscus should either be pressed flat against the table and steadied with the left hand, or grasped in the left hand with the hilum towards the palm and the convex surface outwards. With one sweep of a long-bladed knife divide the organ in its long axis from convex border to hilum, leaving the two halves still attached near the pelvis (Fig. 14). Oblique

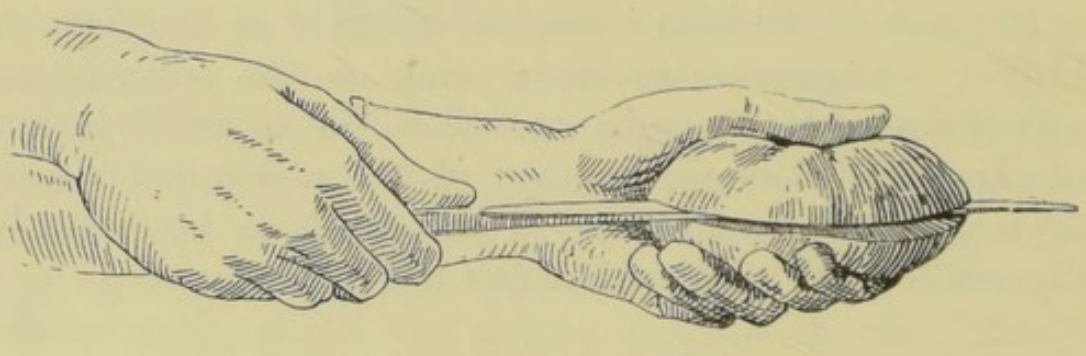

Fig. 14.—Incision of the Kidney.

incisions give erroneous impressions as to the thickness of the cortex and should be avoided. Gently scrape the cut surfaces with the blade of the knife or wash them under a stream of water, then make a systematic inspection. Attention should be directed in turn to the cortex, both peripheral and interpyramidal, the pyramids with their boundary zones and papillæ, the vessels visible on the cut surface, the condition of the pelvis, the amount of fat which surrounds it, also the appearance of the upper end of the ureter.

Finally the capsule should be stripped off. To effect this grasp its cut edge with forceps or obtain hold of it between the finger and thumb by nipping up a little of the cortex and tearing this away with it.

In a healthy kidney the cortex should form a little less than one-third and the pyramid a little more than two-thirds of the distance from the apex of the pyramid to the free surface of the organ. The colour of the cortex is a dark brownish red, whilst the pyramids have a purplish hue, and are definitely striated in their boundary zones ; the papillæ are naturally pale. The capsule is thin and glistening and allows the coloration of the kidney to be seen through it. The surface exposed by stripping off the capsule should be smooth and of uniform colour. Primitive lobulation is evident in childhood and sometimes persists in the adult. The cortex of a healthy kidney is easily lacerated in directions at right angles to the surface of the organ. Firm adhesion and thickening of the true renal capsule indicate the presence of interstitial inflammation of the kidney. This condition is best marked in the contracted granular kidney, but may coexist with hydronephrosis, chronic tuberculosis, chronic ascending nephritis and other chronic inflammations which involve the interstitial tissue.

On the other hand, when the capsule can be stripped off with undue ease, parenchymatous inflammation or recent acute swelling from congestion is present.

Marked increase in the thickness of the cortex, with patchy pallor, indicate parenchymatous inflammation (tubal nephritis). A slighter swelling is seen in fevers.

Atrophy of the cortex indicates chronic interstitial inflammation.

Interstitial and tubal inflammation often coexist.

Abnormal firmness of the renal substance occurs in passive congestion, chronic interstitial nephritis (granular kidney), and old-standing cases of ascending nephritis.

Chronic Passive Congestion of the Kidneys (Cyanotic Induration : Cardiac Kidneys). — The kidneys are moderately enlarged and much darker in colour than normal. They feel weighty, and their substance is tough and resilient. The capsules are thin and come off

cleanly, leaving smooth surfaces which may be marked by prominent stellate veins. On section the organs are purplish, the degree of cyanosis varying with the amount of congestion present. With slight congestion the bases of the pyramids are chiefly discoloured, but in cases of old standing all parts of the cut surface present a dark purple colour which, however, is still most marked at the boundary zones of the pyramids. The slight increase in size involves both cortex and medulla. The cut surface of a passively congested kidney soon brightens by oxidation when exposed to the air.

Passive congestion of the kidneys occurs in valvular disease of the heart and is rapidly produced in cases of death from suffocation or from coma. An extreme form, culminating in necrosis, may result from twist of a movable kidney or from thrombosis of the vessels. In these cases the kidney is greatly enlarged and of a deep chocolate or purple colour. Buff-coloured areas of necrosis may be present.

Cloudy Swelling of the Kidneys is extremely common. In fact, save in cases of sudden death from injury, it is exceptional to find an absolutely normal kidney at a post-mortem examination. In cloudy swelling the kidneys are slightly swollen and rather paler than normal. The capsules can be stripped off with ease. The details of the renal structure as exposed by section have a blurred or clouded appearance. The swollen kidneys are rather softer than in health.

Acute Nephritis. — The appearances presented by the kidneys in acute nephritis vary much, and are in a measure dependent upon the duration of the disease. In some cases, and especially in early stages, the kidneys are but slightly swollen. They are dark in colour, and on section the cortices appear broadened and congested. The glomeruli may sometimes be recognised as minute red dots in the cortex (acute glomerular nephritis). The edges of the swollen cortex tend to become everted when tension is relieved by incision of the capsule. The capsules can be removed with great ease, leaving a smooth surface.

In other cases the size of the kidneys is considerable,

and is due to congestion with cortical swelling. In such cases the cortex is soft, and hyperæmic, but its colour is not uniform, red spots and striæ of hæmorrhage alternating with yellow streaks of anæmia and fatty change. The glomeruli may possibly be recognised as greyish-red or pale granules.

Another variety of acutely inflamed kidney is much paler than either of the preceding, the organ being anæmic and of a yellowish colour. The capsule, as in the other forms, is easily removed, and the edges of the swollen cortex become everted when the kidney is split open. The cortex has a greyish, blurred appearance, and all the finer details are lost. The pyramids are purplish in colour. Kidneys which present these characteristics are distinguished from large white kidneys (chronic tubal nephritis) by the clinical history of the case.

Acute nephritis may supervene in kidneys which are already the seats of chronic disease; the appearances are then modified according to the type of the pre-existing lesions.

Chronic Tubal Nephritis (Large White Kidney).—In this variety of nephritis the kidneys are greatly enlarged and their weight increased. Their shape is more globular than in health, and the colour yellowish. As a rule the capsules come off easily, leaving a pale, smooth surface with conspicuous stellate veins. Adhesion of the capsules and granularity of surface indicate the coexistence of chronic interstitial inflammation. On section the cortex, both peripheral and interpyramidal, is swollen and of a white or a pale yellow colour. It may be mottled or streaked radially by patches of a more decided yellow hue. The swollen cortices both look and feel soft. The pyramids present a marked contrast to the cortices, being hyperæmic and bluish. There is no great marked thickening of the coats of the small renal vessels. Lardaceous changes are frequent.

Chronic Interstitial Nephritis (Red Granular Kidney: Contracted Granular Kidney).—This condition, like other forms of nephritis, is bilateral. Unilateral contraction is due to local causes, such as complete obstruction of the ureter.

Granular kidneys are much reduced in size; their capsules are thickened, and are usually, but not invariably, very adherent. The supervention of acute terminal infection may in some instances account for lack of adhesion. The subcapsular surface is red and finely or coarsely granular, the granules consisting of glandular elements; the intervening depressions are caused by contracting fibrous tissue. Cortical cysts, which vary in size from a pin's head upwards, are sometimes very numerous; they are frequently ruptured during removal of the capsule, allowing their transparent, fluid contents to spurt out. On section the cortex appears thin and tough. Its colour is deep red, approximating to that of the pyramids. The rays from the boundary layers of the pyramids are distorted by the contraction of the cortex. The walls of the renal artery and its small branches are much thickened, small, thick-walled vessels being evident near the bases of the pyramids. The pelvis of the kidney appears relatively large, but this is due to the atrophy of the renal parenchyma. A large amount of perirenal fat is found in the renal sinus, around the pelvis, and so appears to be invading the interior of the kidney.

The White Granular Kidney only differs from the red in the fact that the viscus is yellowish white (anæmic and fatty) instead of red in colour.

Associated with chronic interstitial nephritis expect hypertrophy of the heart, which preponderates in the left ventricle, and thickening of the walls of the arteries. The adrenals may be enlarged. Examine the great-toe joints for gouty deposits in the cartilages, the gums for a lead line, the pelvic organs for indications of pregnancy, present or past, and the bladder and ureters for indications of ascending infection.

Acute Miliary Tuberculosis of the Kidneys.—In this affection minute miliary tubercles are scattered through the kidneys. They may be seen beneath the capsules as minute grey or yellow spots. When yellow they resemble small points of suppuration, but, unlike abscesses, are never surrounded by a zone of congestion; in addition, their nature is revealed by the appearances presented by

the kidney on section, tubercles being recognised in the interior and not striæ of suppuration. Recent tubercles are pearly looking and translucent, older ones become more opaque and finally caseous.

Chronic Generalised Tuberculosis of the Kidneys is occasionally met with, usually in children. Yellow caseous masses of some size are distributed through the kidneys, and may undergo central softening. Similar caseous deposits may coexist in the lungs, brain, and other organs.

Chronic Localised Tuberculosis of the Kidney.—In this form one kidney only may be involved, or both may suffer. When the fellow kidney is free from tuberculosis, it may be hypertrophied or lardaceous. The tuberculous kidney, if the disease is advanced, is enlarged, and may be converted into a pyonephrosis. The perirenal tissues and peritoneum are adherent to the inflamed and thickened capsule; the cortex and pyramids are converted into masses of cheesy tubercle or into tuberculous abscess cavities with rough, thickened walls. The pelvis is thickened, and its interior may present a characteristic mouse-nibbled appearance from ulceration. The upper part of the ureter is often converted into a rigid, thick-walled tube.

Chronic renal tuberculosis is sometimes more localised. If the focus has not ulcerated into the pelvis of the kidney the appearance is that of a cyst with caseous contents. In other cases, ascending tuberculosis induces ulceration of the lower part of the pelvis and the lower calyces without involving the rest of the organ. But even then extension of the infection is not long delayed.

(In all cases of renal tuberculosis, the urinary bladder, prostate, testicles, vasa deferentia, and vesiculæ seminales should be examined for evidence of tuberculous infection. It is also necessary to examine remote parts of the body, such as the lungs, bones, and lymphatic glands, for tuberculous foci.

Lardaceous Kidneys (Waxy or Amyloid Disease).—Lardaceous disease may be present in kidneys which appear normal to the naked eye, but it is frequently associated with nephritis, usually in the form of a large white

lardaceous kidney, less commonly as a contracted lardaceous kidney.

In the large white form the thickened cortex is hard, but friable, and pale. It may be possible to recognise the glomeruli as greyish, translucent specks. The lardaceous contracted kidney is paler than the red granular kidney, and its surface on section is more glistening.

The consistence of a lardaceous kidney is largely determined by the form of nephritis with which it is associated. In all cases of nephritis, and in all conditions in which lardaceous disease is possible (prolonged suppuration from any cause, especially in bone or lung, tertiary syphilis, chronic ulcerative colitis, etc.), the iodine test should be applied. A thin slice of the kidney should be washed free from blood and immersed for some time in a watery iodine solution. The lardaceous Malpighian bodies will assume a dark brown colour, and the arterioles, if involved, appear as brown striæ.

Infarction of the Kidney.—Infarcts of the kidney show superficially as pale, yellow areas. On section they appear as wedge-shaped masses of a dry, yellowish appearance, with their bases directed towards the surface of the organ. Recent infarcts are surrounded by zones of congestion. The infarcted kidney is not necessarily enlarged. The presence of a large number of cicatrised infarcts, such as sometimes occur in old-standing heart disease, may cause both kidneys to become shrunken and coarsely granular (embolic contracted kidney). The condition may be distinguished from chronic interstitial nephritis by the greater size of the kidney and the large size and irregular distribution of the cicatricial depressions on its surface.

Pyæmic Infarcts show decided signs of inflammation. They are visible on the surface as small, deeply congested spots which later become the sites of abscesses. On section, in their earlier stages, only a triangular leash of hæmorrhagic striæ, radiating towards the periphery, with intervening streaks of pale or yellow renal substance, may be seen. Later the infarcted areas become more anæmic and soften. Ascending infections of the kidney may

produce lesions in the pyramids and medullary rays which somewhat resemble infarcts; they are distinguished by their definite relations to the pyramids and by the condition of the lower urinary tract.

Uric Acid Infarcts.—These occupy the renal papillæ. They appear as buff-coloured streaks at the apices of the pyramids, and correspond in direction to the excretory ducts, in which they lie. Uric acid infarcts occur in the kidneys of newly born children, and have no pathological significance.

Cystic Conditions of the Kidney: Retention Cysts of small size, the largest rarely exceeding a pea in diameter, occur in the cortices of contracted granular kidneys. It is exceptional for them to be present in great numbers.

Larger Solitary Cysts, which may correspond to one or more renal lobules, are sometimes seen in kidneys which are otherwise healthy.

Tuberculous Cysts.—Caseous deposits in the kidney sometimes assume a cystic appearance, but these may be distinguished by the character of their contents.

Cystic Adenomata of the kidney are rare. They are encapsuled.

Congenital Cystic Kidney.—One, or more commonly both kidneys, may be converted into a congeries of cysts with watery or viscid contents. The cysts vary considerably in size, and the residual kidney substance is in a condition of chronic interstitial nephritis. Cystic kidneys may attain a great size. The heart is usually hypertrophied.

Pyelitis.—Acute inflammation causes the mucous membrane of the renal pelvis to become swollen and hyperæmic. The surface may lose its glossy appearance, become speckled with hæmorrhages, or coated with a muco-purulent deposit.

Chronic inflammation leads to greater thickening and a greyish discoloration of the mucous membrane. If not velvety and hyperæmic the mucosa may be stiff and granular. Sometimes ulcerations and patches of phosphatic deposit are present.

The presence of a calculus may set up pyelitis, or the inflammation may be due to ascending inflammation from the bladder. Certain drugs and systemic infections also produce pyelitis.

Tuberculous pyelitis has already been described.

Note, care must be taken not to mistake urine, which has become turbid by deposit of its salts, for pus in the pelvis of the kidney. The pelvis shows no inflammation in such a case.

Hydronephrosis is a dilatation of the renal pelvis and its calyces, with gradual recession and ultimate destruction of the pyramids. The calyces may appear as depressions in the wall of the pelvis or as diverticula communicating with the main cavity by short tubular channels. In advanced cases the residue of the secreting substance of the kidneys may be found flattened out against one side of the hydronephrotic cyst, and much indurated from interstitial inflammation.

If hydronephrosis is present its cause must be sought. The urinary passages should be examined for evidence of stone or other obstruction, external or internal; abnormal vessels which cross the ureter should be investigated, and the possibility of movable kidney borne in mind. In some cases of hydronephrosis no cause is apparent.

Pyonephrosis.—In pyonephrosis the contents of the dilated pelvis and calyces are purulent. The walls of the sac are thickened and stiff from inflammation; they may be roughened by the deposit of urinary salts on their interior. Ulcerations are not uncommon. Owing to ammoniacal decomposition of the urine the pus in a pyonephrosis is often slimy and gelatinous. Tuberculous pyonephroses are distinguished by the presence of caseous material.

Pyelonephritis (Surgical Kidney: Consecutive or Ascending Nephritis).—As the result of ascending infection the kidney becomes swollen. On section necrotic striæ may be traced from the papillæ through the pyramids towards the cortex. The striæ are often surrounded by hyperæmic zones similar to those produced by hæmatogenous infarction. Yellow spots may be visible on the

surface when the capsule is stripped; they correspond to the bases of the striæ in the interior. The necrotic areas tend to suppurate, and thus a diffuse suppurative nephritis may be produced. Ascending nephritis sometimes infects the cortex alone, the pyramids escaping. In such cases the infection is presumably transported by lymphatics direct from the pelvis to the periphery of the organ. Sometimes one kidney only, usually the right, is involved by ascending infection.

In consequence of chronic pyelonephritis the kidney may become firm and indurated. The capsule is then unduly adherent owing to chronic interstitial inflammatory changes.

In ascending nephritis the whole of the lower urinary tract, including the urethra, must be examined for evidence of infection or obstruction.

Fibromata occur in the pyramids or cortex of the kidney as small, fibrous nodules of a greyish colour. They are unimportant.

Sarcoma is the commonest renal growth in children under five years of age. The growth is yellowish white on section, and small cavities may be present in it. The secreting portion of the kidney may be stretched over the tumour, giving rise to an appearance of encapsulation. The ureter may escape invasion, but the growth has a great tendency to fungate into the renal veins and vena cava. (Microscopically these growths are mixed-celled sarcomata; many contain striated muscle fibres, and sometimes nodules of cartilage are found.) The growths may be bilateral. Sarcomata of a similar character may arise in the kidney of the adult.

Carcinoma of the Kidney may spring from the cortex or from the pelvis. In the latter situation it is rare, but may be associated with calculus. The kidney is gradually infiltrated and the pelvis invaded. The growth is unilateral.

Hypernephroma is a tumour derived from an adrenal 'rest.' Fragments of adrenal tissue may occur in the cortex of the kidney, just below the capsule, as pale, fatty-looking deposits, which vary in size from a pin's head upwards. Malignant growths arising from them are very vascular and often have a yellowish-red colour. As with

tumours of the thyroid, secondary growths, repeating the adrenal structure, may occur in other parts of the body.

EXAMINATION OF THE ADRENAL BODIES AFTER REMOVAL

The Weight of an Adrenal Body is about 1½ drs. (6–7 grms.).

The bodies must be handled with care during removal, because the central parts tend to soften and become cystic after death, so that lacerations are easily produced. The exterior of the adrenal is of a dull yellow colour. The surface may present small yellow nodules which should not be mistaken for caseous tubercles ; they are adenomata which have undergone fatty changes.

On section there is, in the healthy organ, a sharp contrast between cortex and medulla. The cortex appears as a thin reddish or purple streak within a buff-coloured layer ; the medulla of the adrenal presents a brownish or a glistening greyish appearance. Most pathological changes commence in the medulla of the glands.

Malformations of the Adrenals are rare, but Accessory Adrenals may occur in the form of small yellowish masses near the main glands or along the course of the spermatic or ovarian arteries. They have also been found implanted in the kidney or the liver, in the epididymis, the spermatic cord, or the broad ligaments. Tumours may arise from these detached portions.

In **Nephroptosis** the adrenal bodies are not displaced, but maintain their normal positions in the abdomen.

Hypertrophy of the Adrenals.—In cases of chronic nephritis with cardiac hypertrophy and arterio-sclerosis the adrenals are usually increased in size.

A Pseudo-cystic condition of the Adrenal is often found. It is due to post-mortem softening of the interior.

Adrenal Cysts.—Cysts arising in adenomatous or lymphomatous tumours, blood cysts, and parasitic (hydatid) cysts sometimes occur.

Hæmorrhage into an Adrenal may only be evident when the gland is incised or may cause it to appear swollen and purple. Hæmorrhage may occur in asphyxia, especially of the new-born infant, in diseases with a hæmorrhagic tendency, in acute bacterial infections, in certain specific fevers, or in consequence of severe abdominal injuries.

Although easily produced experimentally by the injection of diphtheria toxin, or overdosage with thyroid gland, hæmorrhage is rarely found in patients who have died from diphtheria and is absent in Graves' Disease.

Lardaceous Disease of the Adrenals attacks the vessels of the cortex. When subjected to the action of watery solutions of iodine the affected cortex becomes dark brown, whilst the medulla may appear pale by contrast.

Tuberculosis of the Adrenal commences in the medulla, in the form of discrete tubercles which soon coalesce and become caseous.

In Addison's Disease the adrenals may be converted into fibro-caseous masses, or be shrunken and fibrosed; in exceptional cases they are merely atrophic. The pathological change in most, if not all, instances is tuberculous. The semilunar ganglia and the nerves of the solar plexus may be enveloped in dense, fibrous adhesions. The skin and mucous membranes should be carefully examined for the characteristic dusky pigmentation.

Syphilitic Inflammations of the Adrenal is exceptional; gummatous and fibrous changes may occur.

Abscess of the Adrenal may be simulated by the softening of caseous material. Small pyæmic abscesses sometimes occur.

Simple Tumours of the Adrenals are, in most instances, adenomatous. They arise in the cortex. There is a hæmorrhagic form of adenoma which simulates sarcoma.

Malignant Disease of the Adrenals, as a rule, is secondary to disease elsewhere. The growth commences in the medulla; its progress is rapid, and the symptoms of Addison's Disease are not necessarily present.

Primary Malignant Disease is rare. It, too, may be either sarcomatous or carcinomatous. There is a tendency to rapid growth with hæmorrhage and cyst formation. Secondary growths may occur in the liver, the cœliac glands, the lungs and kidneys.

EXAMINATION OF THE STOMACH AND DUODENUM

Removal of the Stomach and Duodenum, together with the Pancreas.—Detach the great omentum and transverse colon from the greater curvature of the stomach. Divide the upper part of the jejunum between double ligatures. Dissect the root of the mesentery away from the front of the spine, completely exposing the transverse portion of the duodenum. Liberate the termination of the œsophagus from its aperture in the diaphragm and pull the cardiac end of the stomach forwards. Raise the tail of the pancreas from the posterior wall of the abdomen, avoiding injury to the subjacent suprarenal body if that has not already been removed. Continue to raise the pancreas until the aorta comes into view behind it, then cut across the branches of the cœliac axis and also the superior mesenteric artery. Lastly, dissect the descending portion of the duodenum free from its posterior attachments, commencing at its right border, and remove the stomach, duodenum, and pancreas together.

Where corrosive poisoning is suspected, the proper procedure is to remove the œsophagus in continuity with the stomach and duodenum, otherwise a ligature should be placed around the lower end of the gullet before it is divided, to prevent loss of the gastric contents.

Removal of the Liver, Stomach, and Pancreas together.—When lesions of the liver and stomach coexist, and also in all cases of chronic jaundice, disease of the bile passages or of the pancreas, the integrity of the bile passages must be preserved. This is effected as follows :

Divide the falciform, coronary and lateral ligaments of the liver, thus separating that viscus from the diaphragm Draw it forwards as far as possible so as to isolate and ligature the œsophagus below the diaphragm. Then proceed, as already described above, to remove the stomach, pancreas, and duodenum, taking with them the liver, and, if necessary, the spleen; the structures in the hepato-duodenal ligament (anterior boundary of the foramen of Winslow) being preserved intact.

EXAMINATION OF THE STOMACH

The external appearance of the stomach, its size, shape, position, and any external adhesions have already been investigated (p. 96). If the stomach has been opened by surgical operation, or an ulcer has been sewn up, or a short circuiting operation, such as gastro-enterostomy, has been performed, the site of operation should be examined before removal of the organ, for evidence of peritonitis, leakage, or kinking of the attached bowel. The stomach should also be tested under moderate water pressure before it is laid open. A water nozzle should be introduced into the œsophageal opening, the area of operation brought into full view, and the interior of the organ subjected to gradually increasing pressure. In this way the mechanical efficiency of any repair which has been effected can be tested and the patency of any artificial communications demonstrated.

Proceed to open the stomach with bowel scissors. Introduce the long blade into the œsophageal opening and cut along the fundus and greater curvature. If strictures or obstructions are present inspect their inner aspects before dividing them. On approaching the pylorus incline the scissors towards the front so that the pyloric ring is divided anteriorly. Continue the incision along the convex border of the duodenum in order to avoid injury to the

biliary papilla. The incision along the greater curvature of the stomach admits a better view of the interior, and is less likely to divide any ulcers which may be present than one along the lesser curvature.

In medico-legal cases it may be necessary to preserve the gastric contents for analysis, so the stomach should be opened over a perfectly clean basin into which its contents are received. They are then transferred to a suitable stoppered bottle, which should be sealed and placed in safe keeping.

In the ordinary case, after inspection, the contents of the stomach and duodenum may be removed by a gentle stream of water or by light sponging. Note should be made of the nature of the contents, the stage of digestion, the odour, and the presence of any abnormal constituents such as blood, pus, or excess of mucus. If blood is found in the stomach its source should be ascertained. Small arterial perforations are easily overlooked. The bleeding may have occurred from the nasopharynx, lung or gullet.

The healthy mucous membrane has a pale greyish colour and a slightly glairy appearance. It should be quite soft and supple.

When the stomach or other parts of the bowel have been subjected to water pressure some of the water may be driven beneath the cut edge of the mucous membrane, producing a bullous appearance. This is not pathological.

Cloudy Swelling and **Fatty Degeneration** of the cells of the gastric mucous membrane occur under the same conditions as those which induce cloudy swelling in the other organs of the body. If the change is very marked, the mucous membrane assumes a turbid, greyish or yellowish tint.

Post-mortem Digestion of the Stomach, as the name denotes, is a post-mortem phenomenon. The gastric wall, usually near the cardiac end, becomes soft and pulpy, ultimately melting away. The macerated tissue may be white in colour, or, from diffusion of dissolved blood pigment

and the action of gases of decomposition, it may assume a grey or greenish-black colour. Post-mortem softening is easily distinguished from ulceration by the absence of inflammatory induration and adhesions, and also by the fact that, although the stomach wall may be perforated and the gastric contents extravasated into the peritoneum, yet there is no peritonitis. Occasionally the diaphragm and other structures adjacent to the stomach also show signs of the digestive process.

Post-mortem Lividity occurs in the stomach, just as it does in other parts of the body. The hypostases are found on the posterior or dependent parts of the mucous membrane near the cardia. The portion of stomach involved becomes sodden and presents a purplish discoloration which may also be visible through the peritoneal coat.

Passive Congestion of the Stomach is a result of portal congestion and may be due to cirrhosis of the liver or to venous stasis caused by heart disease. The veins of the mucous membrane may be prominent, and small ecchymoses occur; there is usually an excess of gastric mucus. In cirrhosis of the liver, where the obstruction to the circulation is considerable, the œsophageal veins may be varicose or perforated. Perforation may lead to fatal hæmorrhage, and should the vessels be empty after death, the site of the bleeding is easily overlooked. The hæmorrhage is sometimes from a gastric vein.

Hæmorrhagic Petechiæ in the gastric mucous membrane may result from various blood diseases, from septicæmia, malignant endocarditis or jaundice. They are sometimes induced by violent retching, and may accompany passive congestion or gastritis. The effused blood rapidly becomes brown or black under the action of the acid gastric juice.

Gastritis is indicated by the presence of a thick layer of tenacious mucus on the inner surface of the stomach. The mucous membrane is swollen, and its rugæ are prominent; its colour may be dark red, but as a rule any accompanying hyperæmia is inconspicuous after death. Petechial hæmorrhages may be present, and the submucous tissues may be œdematous.

Chronic gastritis may lead to a slaty discoloration of the mucous membrane, with atrophy of the glandular elements, and more or less fibrous hyperplasia. Sometimes a warty or polypoid condition results.

The pyogenic and phlegmonous forms of gastritis only exceptionally occur. They primarily involve the submucosa. Membranous gastritis is also rare; it is characterised by the formation of superficial membranous pellicles on the mucous membrane. It may be due to diphtheritic, pneumococcal, or other infections.

Corrosive Poisons give rise to a very intense gastritis, with a degree of destruction of the stomach wall which depends on the nature and quantity of the poison taken. The jejunum may also be inflamed.

Alkalies tend to produce moist, gelatinous sloughs. The necrosis due to the action of carbolic acid, corrosive sublimate, or mild doses of oxalic acid is greyish white, superficial and satin-like in appearance. Extensive yellow sloughs indicate the action of nitric acid, whilst actual charring of the mucous membrane is due to the action of sulphuric acid. Hydrochloric acid also darkens and destroys the mucous membrane. The tissues subjacent to the corroded areas may be finely injected. The action of the acids on the blood in the tissues may cause a diffuse darkening quite apart from any charring action. Corrosion by strong mineral acids more frequently causes perforation than does poisoning with other substances. Sulphuric acid is particularly apt to produce this result.

Quite apart from actual perforation, the transverse colon and other parts adjacent to the stomach may become inflamed owing to the diffusion of strong acids through the stomach walls. If the pyloric segment of the stomach remains firmly contracted in cases of poisoning, the gastric inflammation may be limited to the cardiac portion. In other cases a relaxed and dependent pyloric segment suffers most, and may become the site of cicatricial contraction should the patient survive. Combined strictures of the lower end of the gullet and of the pyloric canal are not uncommon sequels of corrosive poisoning.

Atrophy of the Stomach may occur in marasmic

infants, in starvation and in old age. In such cases the walls are attenuated and more or less translucent. As the result of œsophageal obstruction the stomach may be found contracted and somewhat wasted. Atrophy of the mucous membrane may accompany chronic gastritis or dilatation.

Hypertrophy of the Stomach is rarely met with apart from pyloric obstruction. It is most marked in the pyloric region, and is usually associated with dilatation. In diabetes the stomach may be found enlarged and its mucosa thickened.

Hypertrophic Stenosis of the Pylorus is a congenital condition which involves the muscular coats of the whole pyloric canal in infants, causing firm cylindrical thickening. The stenosis is rendered more complete by prolapse of redundant gastric mucous membrane. Similar stenosis of the pylorus is sometimes found in adults.

Acute Dilatation of the Stomach is a rare condition. The stomach is enormously dilated and secretes a great quantity of fluid. The proximal parts of the duodenum are dilated as well. The superior mesenteric arteries may be found dragged tightly across the transverse portion of the duodenum owing to prolapse of the small intestine, or the duodenum may be forcibly compressed between the dilated stomach and the spinal column.

A considerable degree of gaseous distension of both stomach and intestines may accompany acute peritonitis.

Follicular Ulcers of the Stomach are minute circular excavations, sharply defined and shallow. They are often multiple.

Hæmorrhagic Erosions are minute ulcers which arise from small hæmorrhages in the mucous membrane.

Erosions of Gastric Arteries.—Minute ulcers of the mucous membrane may cause lateral erosion of a subjacent vascular arch and thus bring about death from hæmorrhage. Careful inspection will sometimes show that the ulcers are multiple, although only one may be so situated as to perforate a small artery or vein.

Gastric Ulcer.—An acute gastric ulcer is from a third to half an inch in diameter, and has a sharply punched

out, circular outline. The edges are not indurated, and the surrounding mucous membrane appears healthy. Sometimes a slough is present. The base of the ulcer consists of the muscular coat of the stomach or the investing peritoneum. From retraction of the mucous membrane the ulcer may assume a terraced appearance.

Chronic Ulcers are usually larger and more irregular in shape. The edges are indurated and their shelving character may give the ulcer a 'funnel-shaped' appearance. Chronic inflammatory changes are evident around the ulcer, and the base is often adherent to some adjacent structure. Eroded arteries are often present in the floor.

Gastric Ulcers may be solitary or multiple. Active ulcers may coexist with the scars of healed ones. Both in the stomach and duodenum, ulcers which lie apposed to each other, although not necessarily of the same age, are not uncommon. The vicinity of the lesser curvature is the usual site for gastric ulceration.

In cases of perforation the neighbouring viscera should be inspected for tags of adhesion, the tearing away of which has often led to the rupture of the base of the ulcer. 'Perforation' of a gastric ulcer is practically always due to tear of adhesions and not to an acute destructive process.

Fatal hæmorrhage from a gastric ulcer usually occurs from an artery of the lesser curvature or from the splenic trunk. Its source should always be determined if possible.

If portal pyæmia has occurred determine the route of infection. Localised intraperitoneal abscesses in connexion with the ulcer often coexist in such cases.

Bilocular or Hour-Glass Stomach may be due to cicatricial processes following ulceration, or to perigastric adhesions. Localised muscular contraction in a healthy stomach may simulate the condition. The contraction which follows ulceration is often due to muscular spasm and not to a complete cicatricial ring, a very small cicatrix on the lesser curvature leading to an annular constriction.

Malignant Disease of the Stomach.—Carcinoma is common, sarcoma comparatively rare. Carcinomatous

growths when present generally involve the pylorus and the lesser curvature, but sometimes are limited to the cardiac region. They may arise in old ulcers.

A carcinoma presents itself in the interior of the stomach as an induration with a bossy, uneven surface, and firm, well-defined edges. It constricts the pyloric canal, and extends towards the lesser curvature, but, with rare exceptions, leaves the duodenal mucosa intact. Destruction of the surface of the growth gives rise to an ulcer with rampart edges and oozing surface. On section the growth appears as a white infiltration in the stomach walls, more widespread in the submucosa than the induration of the mucous membrane appears to indicate. Microscopically, growth presenting these characters is usually a cylindrical-celled carcinoma with excess of fibrous stroma. Firm, circumscribed, almost globular tumours may also belong to the same category.

Fungating growths which are soft, readily ulcerate and tend to bleed. They are usually spheroidal-celled, with but little stroma. Growths of this nature may be limited to the cardia or involve the whole stomach.

A diffusely infiltrated, thickened and contracted stomach (leather-bottle stomach) is generally the seat of a spheroidal-celled growth with much fibrosis. The mucous membrane may escape ulceration in this variety.

The malignant nature of a case of fibrosis of the stomach should always be suspected, and both the stomach wall and adjacent lymphatic glands should be subjected to microscopical examination.

Squamous-celled carcinomata occur near the cardia, having extended from the lower end of the œsophagus.

Colloid degeneration may occur in gastric carcinomata. It is more common in the spheroidal than the other varieties. The colloid substance has a transparent, jelly-like appearance and often infiltrates the peritoneum.

Sarcoma of the Stomach is infrequent. It may appear as a diffuse infiltration or a prominent globular tumour with a tendency to ulceration and superficial necrosis. Histologically, it is usually a lympho-sarcoma. The distinction from carcinoma is made by the microscope.

When investigating a case of gastric new growth, attention should be directed to the condition of the perigastric, cœliac, and other glands. Secondary growths may be found in these glands, and may also invade the receptaculum chyli, the walls of the thoracic duct, and the lymphatic glands of the left supraclavicular region. The portal territory, especially the liver, is a favourite site for metastatic growths. Adhesions surrounding the gastric tumour, and the planes of subperitoneal connective tissue in the neighbourhood, may be permeated by cancer cells.

Tuberculous and **Syphilitic Lesions** of the stomach are very rare. Their nature must be determined by concomitant lesions and microscopical examination.

LESIONS OF THE DUODENUM

Rupture of the Duodenum may occur as the result of violence applied to the abdomen. The intestinal contents escape into the peritoneal cavity or into the retroperitoneal tissues according to the situation of the rent.

Malformations of the Duodenum.—Atresia of this portion of the bowel is rare. It may be due to contraction or to the presence of a septum in the neighbourhood of the biliary papilla or at the junction of the duodenum and pylorus. Duodenal Pouches may occur near the papilla.

Hypertrophy of the Mucous Membrane of the duodenum and jejunum is common in cases of diabetes mellitus.

Inflammation of the Duodenum, whether from infections or from the action of poisons, produces appearances similar to those already described as occurring in the stomach.

Duodenal Ulcer. — Circular, punched-out ulcers, in every respect similar to those which may be found in the stomach, sometimes occur in the duodenum. They usually involve the first part, and, like gastric ulcers, may be paired and apposed to each other. The ulcer may give rise to a pouching of the gut. In cases of hæmorrhage the vessel eroded should be recognised if possible ; it will usually

be a branch of the hepatic artery. When perforation occurs it is mostly by rupture of adhesions; it is anterior, into the greater sac. The duodenal contents may be directed, by the configuration of the abdomen, into the right kidney pouch and the appendix region, or a right subdiaphragmatic abscess may form.

In severe burns an ulcer of the duodenum may occur. It is uncommon, and its exact pathology is obscure. It may possibly arise as a hæmorrhagic erosion.

Carcinoma of the Duodenum as a primary disease is rare. The growth tends to encircle the bowel, and may be spheroidal or columnar-celled, usually the latter.

The duodenum may be invaded by a carcinoma which arises in the head of the pancreas or from the biliary papilla. The growth in the former situation may be spheroidal-celled, in the latter columnar-celled.

Sarcoma, usually of the round-celled type, may implicate the duodenum. It tends to become diffuse and nodular. It is uncommon.

EXAMINATION OF THE BILIARY PAPILLA, BILE-DUCTS, AND GALL-BLADDER

The liver, bile passages, duodenum, stomach and pancreas are removed together (p. 154). If necessary, cultures are at once made from the gall-bladder and bile passages (p. 127). Open up the stomach and duodenum in the manner already described. Identify the biliary papilla, which appears as a nipple-like projection on the inner side of the second part of the duodenum, $3\frac{1}{2}$ or 4 inches below the pyloric ring; if the bile-ducts are pervious, compression of the gall-bladder may cause a little bile to exude from the biliary orifice and facilitate its recognition. A second small papilla, belonging to the accessory duct of the pancreas, lies about an inch above and slightly ventral to the main papilla. The orifice of the bile-duct, when recognised, is seized and dragged upon by the forceps, whilst a

probe is introduced into the bile passages, care being taken to avoid passing it in the direction of the pancreas. The common bile-duct is carefully opened up with fine scissors, the incision being carried along the hepatic ducts right up the portal fissure of the liver. The orifice of the cystic duct should be identified where it opens into the main passage about an inch below the portal fissure. The valvular plication of its mucous membrane gives rise to considerable difficulty in opening up this small duct.

Lay open the gall-bladder by snipping its fundus with the scissors, carrying the incision onwards to the commencement of the cystic duct. Receive the contents of the gall-bladder into a suitable receptacle, and do not allow them to run over and soil the parts to be examined. Note their colour and consistence. If the cystic duct is obstructed the contents of the gall-bladder may be purulent or consist of colourless mucus. The consistence of bile in the gall-bladder may be thin or almost treacly. Small dark gall-stones may be concealed in tenacious bile.

When the lower end of the common duct is obstructed it may be easier to incise the duct in the mid part of its course, and then, guided by a probe, to lay it completely open.

After noting the character of their contents the gall-bladder and bile passages should be cleansed with a stream of water or by gentle sponging. The common bile-duct is often deeply bile-stained; this does not indicate disease.

The gall-bladder should be inspected, the thickness of its walls noticed, and their consistence tested between the fingers. If healthy the mucous membrane is thin and pliant. Localised thickenings at the fundus of the gall-bladder, which may prove to be malignant, should not be overlooked. The gall-bladder presents a reticulated inner surface if not unduly distended. Small flakes of bright yellow pigment (? cholesterin) may often be seen on its mucous membrane. The examination is concluded by carrying a series of transverse incisions through the walls of the viscus into the adjacent liver substance.

The width of the common bile-duct when flattened out is about $\frac{1}{4}$ inch. The cystic duct admits a No. 5 catheter (English gauge) save at its junction with the hepatic duct, which is rather narrower. The common duct should take a No. 7 catheter. Strictures or impacted gall-stones may be found in the common or cystic ducts.

Examine the lymphatic glands which lie alongside the bile passage, and are known as the 'satellite chain' of the bile-duct.

LESIONS OF THE GALL-BLADDER

Rupture of the Gall-bladder is an uncommon accident. Signs of antecedent disease, such as distension, inflammation, or ulceration, should not be overlooked.

Gall-stones vary much in their number and size. Small dark stones, lying in the midst of inspissated bile, are apt to be overlooked. The colour of the stones ranges from almost pure white to dark olive-green, according to the amount of biliary pigment which is present. Glistening yellow crystals indicate the presence of cholesterin; they are sometimes seen when stones are split open. The stones may be faceted by mutual pressure, but two or more rounded stones may lie together. A gall-bladder which contains gall-stones may be shrunken and thickened, or of normal size; exceptionally it is distended. The interior may be the site of inflammation, ulceration, or new growth. Cholecystitis and portal pyæmia are sometimes present as complications. Acute or chronic pancreatitis may be found.

When gall-stones are present in the gall-bladder, the biliary passages should be examined in their whole extent, and the condition of the pancreas also investigated.

Fistulous Communications between the gall-bladder and the colon or duodenum are usually due to ulceration, set up by gall-stones. Large biliary calculi may enter the bowel through fistulæ formed in this way, and give rise to intestinal obstruction.

Acute Cholecystitis.—The gall-bladder is inflamed

and distended with muco-purulent fluid. The walls are œdematous and soft. In the phlegmonous form of cholecystitis patches of necrosis may appear. Peritonitis, limited to the neighbourhood of the gall-bladder and right kidney pouch, or more widespread, may complicate the condition.

Empyema of the Gall-bladder.—The gall-bladder is inflamed, thickened, and full of pus. The condition of the cystic duct should be investigated. It will probably be found obstructed by a gall-stone, by inflammatory adhesions, or perhaps by malignant disease.

Adhesions, the result of past attacks of cholecystitis, may closely bind the gall-bladder to surrounding structures, such as the pylorus, duodenum, transverse colon, and abdominal wall. These adhesions may cause dilatation of the stomach or intestinal obstruction. The presence of fistulous communications between the gall-bladder and the bowel should not be overlooked when dense adhesions are present.

Ulceration of the mucous membrane of the gall-bladder is generally due to gall-stones, but may occur in other conditions, such as typhoid fever, tuberculosis, and cancer.

LESIONS OF THE BILE-DUCTS

Congenital Obliteration of the Bile Passages is of rare occurrence. The lesion may involve the ducts at any part of their course. The liver may show monolobular cirrhosis. The body is jaundiced, and the spleen may be enlarged. Petechial hæmorrhages may be found in the skin and mucous membranes. Infants may survive several months with complete biliary obstruction. The affection is sometimes syphilitic in origin.

Dilatation of the Bile-ducts indicates obstruction, but sometimes ducts are found much thickened by chronic inflammation and dilated without obvious obstruction. When obstruction is present, it may be due to the presence of gall-stones, strictures, adhesions, disease of the head of the pancreas, or the pressure of tumours or enlarged

glands. When the common duct is completely obstructed colourless mucus will be found in the passages above ; when the obstruction is incomplete the mucus is bile-stained. When obstructed, the common duct may be so dilated as to simulate the gall-bladder itelf.

Impacted Gall-stones tend to lie near the upper end of the common duct or else at the biliary papilla. They may cause partial obstruction when impacted at the former point, but in the latter position obstruction is complete, and the pancreatic duct may be blocked also. In all such cases the condition of the pancreas calls for careful investigation.

Suppurative Cholangitis is usually the result of obstruction from some cause or other. The ducts, which are thickened and dilated, are full of purulent mucus or thick pus. The intrahepatic ducts share in the infection.

Simple Strictures of the Bile-ducts are uncommon. They may be sequels of the ulceration caused by gall-stones. On microscopical examination strictures, apparently simple, often prove to be carcinomatous.

Malignant Disease of the Ducts is more frequent in the common duct than elsewhere. Most strictures of the ducts are cancerous in character, and the growth is usually a cylindrical-celled carcinoma. The condition is often complicated by the presence of suppurative cholangitis.

Pressure by Enlarged Glands may induce biliary obstruction. The glands in the portal fissure, the gland which lies on the cystic duct, or the glands which form the satellite chain of the common duct, when enlarged may cause compression. The enlargement may be simple or malignant. Glands which are chronically inflamed may feel so hard as to simulate malignant disease or even gall-stones. They should be incised, and also examined microscopically if there is any doubt as to their condition.

Disease of the Pancreas, either inflammatory or malignant, may obstruct the terminal part of the common bile-duct. In inflammatory conditions it may be possible to pass a probe along the duct after death, but dilatation of the duct above, and sudden narrowing below, with absence of bile-staining in the lower portion, afford evidence of the obstruction which existed.

EXAMINATION OF THE PANCREAS

The stomach and duodenum having been examined and the bile passages laid open as already described, the examination of the pancreas, which is still attached by its head to the duodenum, is proceeded with (Fig. 15).

Turn up the stomach to expose the anterior surface of the gland. Inspect this and palpate it carefully all over. Then turn up its tail towards the duodenum, and, as far as possible, examine the posterior surface. The positions of the biliary papilla and of the accessory pancreatic

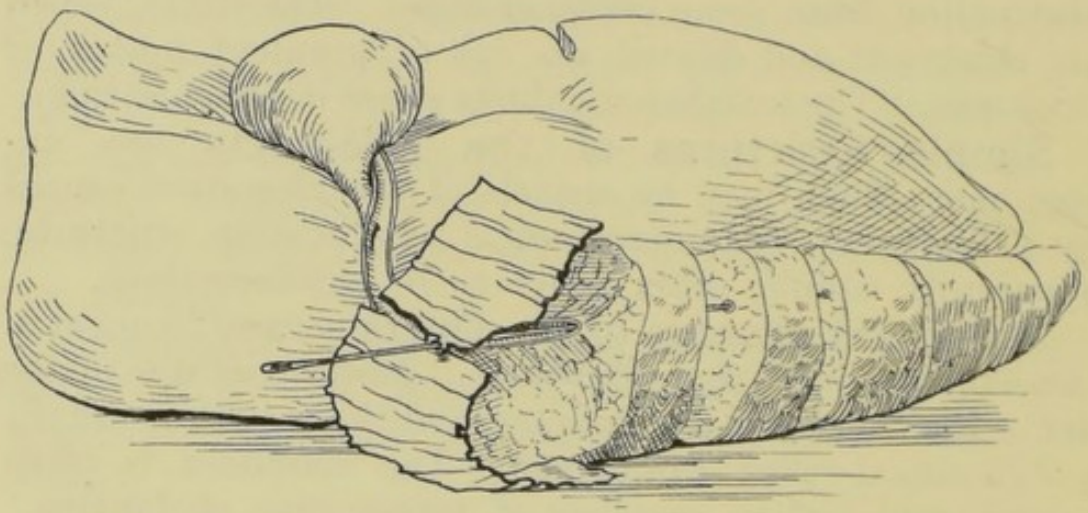

Fig. 15.—Examination of the Pancreas and its Duct.

papilla in the duodenum have already been determined when examining the bile-ducts. A series of incisions should be made into the gland, at right angles to its long axis, commencing at the tail. The slices are held in position by the retroperitoneal tissue at the back. On arriving at the neck of the gland the small, white-walled pancreatic duct, lying near the middle of the section, should be recognised. The wall of the duct should be seized with dissecting forceps, and a flexible probe inserted and coaxed on until it emerges from the biliary papilla. By following up the probe with scissors the duct can be laid open from the anterior aspect of the organ. The accessory duct, which leaves the main duct

in the head of the gland and passes to the accessory papilla, should be recognised if possible.

Another method of recognising the pancreatic orifice on the papilla is to squeeze the head of the gland after slitting up the papilla and bile-duct. A little fluid will then exude from the pancreatic orifice, and a probe can be introduced into the latter and passed along the duct.

The splenic vein and artery should also be opened up and examined at this stage.

The healthy pancreas is of a uniform dull pale-yellow colour. Its consistence is firm, almost hard, which renders the detection of cirrhosis difficult apart from microscopical examination. A pancreas which is much infiltrated with interstitial fat may appear soft.

It is well to bear in mind that a chain of lymphatic glands lies immediately above the pancreas. These glands, when infiltrated with tubercle or new growth, may be found adherent to, or almost embedded in, the pancreas itself. Disease of the glands may thus simulate disease of the pancreas.

Weight of the Pancreas.—The weight of the gland is from $2\frac{1}{4}$ to $3\frac{1}{2}$ or 4 ounces (66–114 grammes).

Cloudy Swelling of the Pancreas may occur in fevers and other infective diseases. It causes the gland to appear soft and greasy.

Post-mortem Decomposition gives rise to a reddish staining of the gland with diminution of its consistence. In advanced decomposition the interstitial tissue becomes infiltrated with bloody serum. These changes must not be taken as indications of the existence of disease of the organ during life.

The Pancreas in Corrosive Poisoning.—In cases of death from poisoning with hydrochloric acid the pancreas is often greatly swollen and hyperæmic. A similar condition may be induced by poisoning with other mineral acids.

Acute Pancreatitis.—Both hæmorrhagic and gangrenous varieties occur, and the two may coexist. The

disease may be general or localised. The gland is swollen, purplish from hæmorrhage, or actually grey from extensive necrosis. In the hæmorrhagic form the interstitial tissue is infiltrated with blood, and bleeding may also take place into the retroperitoneal tissues.

On section an acutely inflamed pancreas presents a variegated appearance, patches of red, grey, and yellow being intermixed. Opaque white areas of fat necrosis may also be present, both in the subperitoneal tissues and in the pancreas itself. The pancreatic duct usually contains a thick, sometimes blood-stained, fluid.

The lesser sac of the peritoneum and adjacent parts of the greater sac may be inflamed.

The condition of the bile-ducts and of the splenic vessels which pass along the back of the pancreas should be investigated. Gall-stones may be present in the gall-bladder or ducts and venous thrombosis may have occurred.

Suppurative Pancreatitis may occur as multiple abscesses in connexion with the ducts or as larger localised collections of pus. The former condition is sometimes associated with gall-stones or with pancreatic calculi. The larger abscesses may be complicated by pancreatic hæmorrhage, or by inflammation of the retroperitoneal tissues or of the peritoneum itself.

Chronic Pancreatitis.—Chronic inflammation, leading to sclerosis, may be limited to the head of the pancreas or involve the whole of the gland. The process usually leads to shrinkage of the organ, but sometimes to such an increase in size as to simulate malignant disease. The surface is either smooth or nodular, and the substance presents varying degrees of hardness. The cut surface appears homogeneous if the fibrous infiltration is diffuse, but in other cases may be granular owing to the projection of compressed islets of pancreatic tissue. Areas of fatty degeneration or of fat necrosis will appear as yellowish or dull white specks respectively. A distorted or sacculated appearance of the duct indicates the presence of obstruction.

The common bile-duct may be constricted by the contraction of fibrous tissue in the head of the gland. Gall-stones or catarrh of the bile-ducts and duodenum may co-exist with chronic pancreatitis. A microscopical examination is

advisable in all cases to determine the amount of sclerosis and, in cases where the process is circumscribed, to distinguish it from malignant disease (scirrhous carcinoma).

Atrophy of the Pancreas.—This may be part of a general wasting of the whole body; it occurs in a few cases of diabetes mellitus, but it is by no means constant in this disease. Atrophy may also be associated with chronic pancreatitis or result from localised pressure on the gland. The atrophied organ is flabby, and on section the fibrous framework becomes unduly conspicuous by contrast with the wasted glandular tissue.

Pancreatic Calculi.—Small white calculi, consisting of carbonate or phosphate of lime, are occasionally found in the pancreatic ducts. They may be very numerous. Sometimes only a mortary material is present. If the pancreas is carefully palpated before incision, calculi are not likely to be overlooked. The pancreatic ducts may be dilated and show signs of chronic catarrh; interstitial pancreatitis may also be present. Cancer of the pancreas rarely accompanies calculi.

Pancreatic Cysts.—True retention cysts, the result of obstruction of the pancreatic ducts by calculi or other agencies, sometimes occur. Cysts may also arise as the result of injuries to the gland or of hæmorrhage into it. They must be distinguished from encysted collections in the lesser sac of the peritoneum, which also may arise as the result of injuries to the pancreas. Cystic disease of the pancreas, apart from injury or obstruction of the ducts, is rare.

Malignant Disease of the Pancreas usually occurs as a diffuse carcinomatous induration of the head of the gland. Sometimes the disease is circumscribed. The relations of the new growth to the bile-duct, the pancreatic duct, and the adjacent part of the duodenum, should be carefully investigated. The growth is usually white and firm, but it may show discoloration due to bile-staining, to necrosis, or, more rarely, to hæmorrhage. If invasion is present, or secondary growths are found in the adjacent lymphatic glands or liver, the malignant nature of a diffuse induration of the head of the pancreas is evident, otherwise microscopical examination may be necessary to distinguish the condition from a simple chronic pancreatitis.

Sarcomata sometimes arise in the pancreas. They appear as nodular tumours in the body of the gland, and may undergo widespread dissemination. Disease of adjacent and adherent lymphatic glands should not be mistaken for disease of the pancreas itself.

Fat Necrosis.—Irregular white patches of various sizes may occur in the subperitoneal fat, in the omenta, the mesenteries, on the under aspect of the diaphragm, and sometimes in the subpleural and subpericardial tissues as well. These are areas of fat necrosis. They are most marked in the immediate neighbourhood of the pancreas, and often occur in that gland itself. They are usually, if not invariably, associated with pancreatic disease. They must not be confounded with peritoneal tubercles, distended lymphatics, or carcinomatous growths.

The Pancreas in Diabetes.—As a rule naked-eye lesions are absent in this disease. Sometimes the organ is sclerotic or wasted. A microscopical examination should be made.

EXAMINATION OF THE INFERIOR VENA CAVA, THE ABDOMINAL AORTA, THE RETROPERITONEAL LYMPHATIC GLANDS, AND OTHER STRUCTURES ON THE POSTERIOR WALL OF THE ABDOMINAL CAVITY

The Inferior Vena Cava has already been divided just below the liver. Lay open the vein with blunt-pointed scissors, prolonging the incision into the iliac veins of each side. The renal, ovarian and spermatic veins are also to be opened and examined if necessary. (For a method of examining the veins in cases of pulmonary embolism or thrombosis, see p. 67.) In cases where swelling of the lower extremities may have resulted from thrombosis (as in typhoid fever) it will be necessary to examine the femoral and popliteal veins and also the veins of the calves.

Thrombosis of the Inferior Vena Cava may be due to the pressure of abdominal tumours or abdominal aneurysm; it sometimes occurs in cirrhosis of the liver. More commonly the occlusion is caused by extension of clot from the pelvic or the femoral veins.

The Abdominal Aorta should be laid open from the front, the incision being prolonged into the iliac arteries. Blunt-pointed scissors should be used for this purpose, as the intima is easily lacerated. In some cases it is advisable to prolong the incisions along the femoral arteries into the thighs; to do this a preliminary exposure of each femoral artery is necessary.

The aorta, if healthy, is supple and easily cut.

Atheroma of the Abdominal Aorta may occur, especially in the aged. It appears in the form of thickened, subintimal plates. The patches may become calcified, and the calcareous laminæ denuded of their coverings and exposed to the blood-stream. As a rule atheromatous changes of the abdominal aorta are most marked just above the terminal bifurcation of the vessel; if thrombosis is present, this is its most likely site. Atheromatous changes are not necessarily uniform in their distribution. The thoracic aorta may be atheromatous and the abdominal portion healthy or vice versa. The peripheral arteries of the body may present atheromatous changes when the aorta is free, or the latter may be extensively diseased and the former healthy.

Arterio-sclerosis.—In cases of generalised arterio-sclerosis the walls of the aorta may share in the universal vascular thickening and rigidity.

Syphilitic Aortitis.—Extensive scarring, due to syphilis, such as is often met with in the vicinity of the aortic valves and the aortic arch, is uncommon in the abdominal portion of the vessel (see p. 91).

Aneurysm of the Abdominal Aorta.—Saccular aneurysm, when present, may lead to extensive matting of the surrounding structures and may rupture into the abdominal planes of connective tissue. Removal of the

abdominal contents *en masse* (p. 176) is the best way of examining these aneurysms.

Dissecting Aneurysm of the Abdominal Aorta may extend from the thoracic portion of the vessel. It causes the aorta to assume the appearance of a double tube. Close examination will show that one chamber represents the true lumen of the aorta, whilst the other is formed by a cleavage of the substance of the vessel wall.

Thrombosis of the Abdominal Aorta generally depends upon disease of the vessel wall, and is more likely to occur at the lower end than elsewhere. The occasional presence of an embolus as a cause must not be overlooked. In all cases the relations of the upper and lower limits of the clot to the different branches arising from the aorta should be ascertained.

In cases of sudden paraplegia or of gangrene of the feet, the aorta should be examined as well as the arteries of the lower limbs and the spinal cord.

(See also remarks on *Examination of the Thoracic Aorta*, p. 91.)

THE RETROPERITONEAL LYMPHATIC GLANDS

The normal colour of the lymphatic glands is rosy white, but those near the liver are yellowish, and those attached to the spleen are darker still. Certain glands, lying alongside the abdominal aorta, are of a decided brownish-red hue, these are 'hæmolymph glands'; their sinuses contain blood instead of lymph. Similar glands lie along the great vessels of the thorax and neck. Hæmolymph glands become conspicuous in severe anæmias, and infectious processes.

The iliac constituents of the abdominal glandular chain not only receive efferent vessels from the pelvic viscera, but are also connected with the lymphatics of the glans penis, the testis, the clitoris, those of the inguinal regions, and those of the lower part of the abdominal wall.

The lumbar glands receive lymphatics from the abdominal

viscera. and from the body of the uterus and its adnexa. The lymphatic trunks from the ovary and testicle also pass to the lumbar group.

The retroperitoneal glands when enlarged may exercise pressure on surrounding parts. The bile-duct may be obstructed near the duodenum, the inferior vena cava compressed or invaded. The intimate relation of the adjacent lymphatic glands to the pancreas has already been pointed out.

Inflammatory Swelling of the Retroperitoneal Glands is very marked in some cases of typhoid fever, but may arise under other conditions.

Tuberculosis of the Retroperitoneal Glands is recognised by the extensive caseation produced, the tendency to matting and adhesion, and the frequent occurrence of miliary tubercles in other parts.

Hodgkin's Disease (*Lymphadenoma*) is characterised by the formation of conglomerate tumours which consist of discrete glands (see p. 94).

Carcinoma of the Glands is characterised by an invasive and infiltrating tendency, and is secondary to carcinoma somewhere in the drainage area of the glands involved.

Sarcoma of the Glands also shows an invasive tendency and very rapid growth. It is secondary as a rule, but may be primary. A microscopical examination is necessary in all cases of doubtful nature.

The Receptaculum Chyli lies between the right side of the aorta and the right crus of the diaphragm. It is variable in size and shape, appearing sometimes as a spindle-shaped dilatation, sometimes as a network of anastomosing trunks. To expose it the crus must be divided and drawn aside.

In all cases of tuberculous or malignant disease of the abdomen, and especially where an infiltrated gland lies just above the left clavicle, the condition of the receptaculum chyli and thoracic duct, together with that of the

ensheathing connective tissue, calls for close examination. In such cases tubercle or growth may be found extending along the outside of the duct or implicating its lumen.

REMOVAL OF ALL THE ABDOMINAL VISCERA TOGETHER

Under certain conditions it is advisable to remove the abdominal viscera in one mass and not to enucleate the organs one by one in the manner already described.

Simultaneous removal is advantageously adopted (1) in cases of Addison's disease, so that the adrenal bodies, semilunar ganglia, and nerve plexuses may be dissected; (2) in cases of retroperitoneal growth, since the ramifications and relations of the growth can better be appreciated by this method; (3) in aneurysms of the abdominal aorta; and (4) in cases of extreme and extensive malignant or tuberculous disease of the peritoneum and abdominal viscera, where the tissues are often so matted together that separate extraction of the viscera, and especially of the intestines, is extremely difficult.

After the preliminary inspection of the abdominal contents the thoracic viscera may be completely removed in the ordinary way, or their diaphragmatic attachments may be left intact so that the abdominal and thoracic contents are removed together.

The abdomen is eviscerated in the following manner:

Pull taut the left wing of the diaphragm and divide its attachments quite close to the ribs, commencing in front and continuing until the vertebral column is reached. Avoid injuring the spleen, the fundus of the stomach, or the left kidney. As the diaphragm becomes free the mass of viscera should be forcibly drawn over to the right by an assistant until the left crus comes into view. The tendinous insertion of the crus must be divided quite close to the vertebræ, the knife being carried from above

downwards and towards the mid-line. The right wing of the diaphragm is detached in a similar way.

Then, commencing on the left, the parietal peritoneum is stripped back on each side towards the spine. Anteriorly detachment of the serous layer is rather difficult, but it can be raised with ease from the ilio-psoas and the vertebral column. The separation is continued, using fingers only, downwards as far as the promontory of the sacrum and the brim of the true pelvis. The iliac arteries and veins should at the same time be lifted from their beds with the peritoneum.

The separation may, if desired, be carried into the planes of pelvic connective tissue and the contents of the pelvic basin also extracted (see p. 186), but it is generally convenient to remove the pelvic viscera separately. In the latter case a knife is carried through the soft tissues on the promontory and pelvis just below the brim, dividing also the round ligament or vas deferens near its point of passage through the internal abdominal ring. A double ligature having been placed on the pelvic colon, the latter is divided and the abdominal mass freed.

EXAMINATION OF THE VISCERA REMOVED

The posterior surface should be examined first.

Expose the suprarenals. To do this determine the position of the kidneys by palpation and incise the diaphragm vertically near their inner borders. Turn aside the flaps and expose the adrenals by blunt dissection in the adjacent connective tissue. The adrenal vessels and nerve plexuses can be traced out and the semilunar ganglia identified.

The kidneys, renal vessels and ureters are next exposed in a similar manner.

The abdominal aorta is slit up posteriorly and the vena

cava inferior is also laid open. The prevertebral lymph glands are inspected and their relations to surrounding structures investigated.

The portal vein may now be examined from behind, but this entails division of the abdominal aorta, the vena cava, the cœliac axis, the superior mesenteric and the renal vessels. Pick up the aorta and the vena cava, divide them between the origins of the renal and superior mesenteric arteries. With a few snips of the scissors expose the subjacent portal vein without wounding it. By blunt dissection trace the main trunk to the portal fissure; trace it also in the reverse direction, exposing the splenic vein as it runs along the back of the pancreas, and also its mesenteric tributaries.

The bile passages may next be identified and dissected.

The pancreas, being exposed, can be examined next, but should not be detached from the duodenum.

The remaining viscera may be examined from the front in the ordinary way.

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CHAPTER IV

EXAMINATION OF THE PELVIC VISCERA AND ORGANS OF GENERATION

EXAMINATION OF THE SCROTUM, TESTES, AND SPERMATIC CORDS

Make a preliminary inspection of the scrotum and penis. Note any abnormalities or external signs of disease. Ascertain the position of the testes. When diffuse inflammatory conditions of the scrotum are present the urethra should be explored with a sound in order to determine the presence or absence of a stricture. If the scrotum is ulcerated, ascertain whether the testis is adherent to the base of the ulcer.

Œdema of the Scrotum.—Simple œdema of the scrotum may be part of a generalised dropsy due to renal or cardiac disease, or resulting from obstruction of the inferior vena cava.

Œdema restricted to the scrotum and its neighbourhood is generally of inflammatory origin; it may be due to extravasation of urine. In the latter case the œdema involves the anterior part of the perineum as well as the whole scrotum, and also tends to spread to the integuments of the penis and of the lower part of the abdominal wall.

Gangrene of the Scrotum is most commonly due to extravasation of urine, but may be the result of acute inflammation from other causes, as, for instance, extension from a periproctitis due to perforation of the lower end of the rectum or anal canal.

Hernia Testis.—If the testis is adherent to the scrotal wall and testicular substance is protruding, the nature of the disease is determined by further examination of the testis (p. 184). The latter may be the seat of tuberculous or of syphilitic inflammation.

Simple inflammation rarely leads to abscess and hernia testis; occasionally, malignant disease produces this result.

Epithelioma of the Scrotum or Chimney Sweep's Cancer may present itself as a warty growth covered by a scab, the removal of the latter leaving a red, granular surface. If the disease is more advanced it may appear as a malignant ulcer with indurated edges and base. The adjacent inguinal glands should be examined for secondary growth.

Elephantiasis gives rise to a warty thickening of the scrotum owing to the formation of dense white fibrous tissue. The lymphatics are dilated; they may rupture and discharge on the surface (lymph-scrotum).

REMOVAL AND EXAMINATION OF THE TESTES AND SPERMATIC CORDS

This part of the examination should be carried out before the pelvic viscera are extracted from the body.

Define the position of the internal abdominal ring by tracing the course of the spermatic vein or of the vas deferens towards it; a small peritoneal depression may indicate the spot. Isolate the spermatic cord near the internal ring by incision of the peritoneum over it, then, grasping the cord with the left hand, make gentle traction on it and by this means draw up the testis towards the abdomen. The process may be facilitated by making pressure from below on the scrotum and by incising the margins of the abdominal rings with the knife. The spermatic cord is easily ruptured by forcible traction; this should be avoided. As the testis passes from the

abdomen the gubernaculum at its lower pole will invaginate the bottom of the scrotum to which it is attached. This attachment must be divided, keeping the knife close to the testis, or the scrotum will be buttonholed. Where inflammation or new growth has united the testis to the skin, the two must be dissected apart or the adherent portion of skin removed with the gland.

If there is a distinct orifice in the situation of the internal ring, the mouth of the processus vaginalis is patent or a hernial sac is present. By incising the peritoneum around the orifice, at some distance from the margin of the latter, the sac may be dissected out and removed together with the cord and testis.

When extricated from the scrotum the testis may be detached by dividing the cord, but where the gland or its coverings are diseased it is preferable to leave the cord attached, so that the testis, cord, prostate and other pelvic viscera may be removed together.

In cases where considerable swellings exist along the course of the spermatic cord or where, on account of the great size of the testicle, or distension of the tunica vaginalis, extraction through the abdominal rings is impracticable, the cord and testis should be exposed by a free incision carried along the inguinal canal into the scrotum.

Attention should always be paid to the degree of obliteration of the processus vaginalis, particularly when hernial sacs or hydroceles are present, either in the scrotum or in the inguinal canal.

Varicocele. — The dilated and tortuous condition of the veins of the pampiniform plexus which constitutes a varicocele is not nearly so obvious post-mortem as it is during life. Examine the veins for the presence of thrombosis or of phleboliths. Varicocele is usually left-sided, and the corresponding testicle may be abnormally small.

Hydrocele of the Cord.—An elongated hydrocele may occur in the funicular process of the peritoneum. It

may or may not extend into the scrotum, and is dependent upon imperfect obliteration of the peritoneal extension.

Hæmatoma of the Cord.—An extravasation of blood into the tissues of the cord may be the result of injury.

Polycystic Tumours are of fœtal origin and rare. They lie along the track of the spermatic cord and may be extensive.

Torsion of the Cord may lead to strangulation of the testis and of the tissues of the cord below the twist. Ascertain in such a case if the testis has fully descended.

Tuberculosis of the Vas Deferens may occur in the course of genito-urinary tuberculosis. The infected segments appear as moniliform thickenings, less commonly the infiltration is uniform. The lesion may be limited to the testicular end of the cord or to the immediate neighbourhood of the vesicula seminalis.

Syphilitic Disease of the Spermatic Cord is extremely rare. When present it occurs as a gummatous infiltration.

Lay open the tunica vaginalis with scissors, making the incision along the front border of the testis. If healthy, the interior of the sac is smooth and glistening like other serous membranes. It should be free from adhesions.

Acute Hydrocele.—The tunica vaginalis may present signs of recent inflammation and contain a collection of serous fluid. In such a case the testis and epididymis should be carefully examined for evidence of recent injury or of acute inflammation.

Miliary Tubercles are sometimes found in the tunica vaginalis, and localised tuberculous foci may overlie tuberculous disease of the epididymis or testis.

Chronic Hydrocele is characterised by thickening, and sometimes by calcification, of the tunica vaginalis, as well as the presence of a serous effusion. Glistening scales of cholesterin may float in the fluid. The walls of the sac may be rough or polypoid. Like acute hydrocele, the condition may be primary or depend upon disease of the testis or epididymis.

Hæmatocele.—A collection of blood in the tunica vaginalis may be inflammatory or traumatic in origin. In old-standing cases the walls of the sac may be greatly thickened, and the contents consist of dirty brown blood clot or thick treacly fluid.

Abscess in the Tunica Vaginalis is rare. The purulent collection may occur as a sequel to urethritis (not invariably gonorrhœal), or as a complication of tuberculosis of the testis.

Adhesions in the Tunica Vaginalis may arise as sequels to epididymitis, or complicate tuberculous or gummatous disease of the testis and epididymis. They may also overlie new growths.

Loose Bodies in the form of small white formations may arise in connexion with inflammation of the sac walls, or possibly, in some instances, be derived from fœtal remnants.

The Epididymis should be examined by a series of incisions transverse to its long axis.

Acute Epididymitis, leading to considerable swelling, may be the result of infective urethritis or of certain specific fevers, particularly mumps. Examine the urethra.

Gonorrhœal Epididymitis of a chronic or recurrent form tends to involve the tail of the epididymis to a greater extent than the head. The connective tissue which surrounds and permeates the organ is indurated; when the tail is incised, small cavities filled with purulent fluid are found. The cavities are localised dilatations of the infected tube. The overlying layers of the tunica vaginalis may show signs of acute or chronic inflammation. The walls of the vas deferens are thickened and pus may exude from its lumen.

Tuberculous Epididymitis usually commences near the head of the organ, but is not restricted to this situation. In advanced cases the epididymis forms a nodular, crescentic swelling containing caseous nodules which have a great tendency to soften. The tunica vaginalis shows signs of chronic inflammation, and the epididymis may become

adherent to the skin with formation of sinuses. The testis itself may be implicated. The vas deferens undergoes tuberculous infiltration. The vesiculæ seminales and the prostate should always be examined, since tuberculous deposits are apt to occur in these structures also.

EXAMINATION OF THE TESTES

The testis should be inspected after opening the tunica vaginalis and divided longitudinally by an incision through its anterior border. The incision should be carried backwards, splitting the globus major of the epididymis and terminating at the hilum of the gland. The lateral halves, still attached by the tissues of the hilum, are allowed to fall apart and the cut surfaces inspected. If the gland is healthy they are of a light-brown colour, soft, remarkably free from fibrous tissue, and bulge somewhat owing to the relief of tension by division of the unyielding tunica albuginea.

The Weight of each Testis, together with its epididymis, varies from ¾ oz. to a little under 1 oz. (15–24·5 grms.).

Malposition of the Testis. — The testis, having failed to complete its descent, may be found in the abdomen, the inguinal canal, or just outside the external inguinal ring, in the fold between scrotum and thigh. An incompletely descended testis is often accompanied by hernia. Ectopia testis is a term applied in those instances where the gland descends into an abnormal position such as the perineum or the front of the thigh.

Atrophy of the Testis.—This may be sclerotic and the sequel of inflammation or, more rarely, a fatty degeneration of the glandular tissue due to interference with the blood supply of the organ. In the former the gland is shrunken and nodular; the normal buff-coloured surface seen on section is replaced by bands of fibrous tissue. In the latter, and also in old age, the testis is small, soft, and flabby; on section it has an anæmic fatty appearance.

Diffuse Syphilitic Orchitis.—The testis is swollen. On section its tissue is harder than normal, and pinkish rather than buff-coloured. In advanced cases bands of connective tissue permeate the organ in the direction of its septa, and are especially evident in the rete testis, near the hilum. Both the tunica albuginea and the tunica vaginalis show signs of inflammation; some yellow fluid may be present in the cavity of the latter.

Gummatous Orchitis.—Gummata of the testis are usually multiple. They appear as dry, yellowish-white or grey masses surrounded by fibrous capsules. Their centres become caseous. The condition is bilateral as a rule, and the epididymis and cord usually escape.

Tuberculous Orchitis. — This is associated with advanced tuberculosis of the epididymis. The deposits in the testicle may be miliary or caseous. Caseous masses may soften and give rise to hernia testis.

Malignant Disease of the Testicle.—Neoplasms of the testis are soft, vascular, and usually of rapid growth. Carcinomata, sarcomata, endotheliomata, and embryomata (teratomata) all occur: some of the latter contain trophoblast, and so resemble chorion-epitheliomata.

Cystic tumours (embryomata ?) of the testis which arise in the rete tend to flatten out the body of the testis and reduce it to a narrow stratum intervening between the tunica vaginalis and the tumour.

These cystic tumours may contain cartilage. Metastases may occur.

The glands which become involved secondarily to tumours of the testis belong to the iliac and, particularly, to the lumbar groups.

REMOVAL OF THE PELVIC VISCERA OF THE MALE

Before removal a preliminary inspection of the pelvic cavity should be made. Determine the condition of the peritoneal lining of the recto-vesical pouch. A dull, sticky or injected appearance of the serous surfaces denotes

recent inflammation. If adhesions are present ascertain their consistence ; recent adhesions are sticky and friable, old adhesions may be very tough. Be careful to explore the lowest recess of the pouch, since this is sometimes quite sealed off, and the presence of inflammation in this part may be overlooked. Pus in the recto-vesical pouch may be the result of inflammation in the vicinity or may have gravitated from some other part of the peritoneal cavity. A mass of gelatinous fibrin is sometimes found lying in the pouch in cases of ascites without there being any signs of peritoneal inflammation. Extravasation of fæcal matter indicates a perforation of the bowel wall.

Inspect the rectum ; it may be contracted or distended. Palpate it ; fæcal masses may be recognised by their plastic character whilst malignant growths feel hard and resistant. Foreign bodies are sometimes found. The rectum is commonly plugged with absorbent wool after death ; the plug should be extracted from the anus before proceeding further with removal of the viscera.

Next direct attention to the urinary bladder. This, as a rule, is found contracted after death ; if it is greatly distended suspect an obstruction to the urethra or some disease of brain or spinal cord leading to retention.

When the bladder is found distended it may be advantageous to incise its fundus *in situ* before proceeding further. After removing the urine the interior of the bladder can be inspected before the neighbouring parts are disturbed. This method of inspection is useful as a preliminary measure in cases where the prostate is enlarged.

Now proceed to extract the pelvic viscera in one mass as follows : Standing on the left side of the body, draw the bladder upwards and backwards ; incise the peritoneum and loose connective tissue uniting the viscus to the back of the symphysis pubis, but do not, at this stage, divide the urethra. Continue the dissection round the right side of the pelvis in the loose connective tissue, avoiding injury to the external iliac vein by keeping below and internal

to it. The separation should be carried right down the side of the pelvis to the pelvic outlet and is facilitated by drawing the bladder well over to the left.

The left side of the pelvis is dealt with in a similar manner, the bladder being drawn over to the right.

Continue the incision of the peritoneum across the promontory of the sacrum, behind the rectum which, owing to the previous division of the pelvic colon, can be drawn forwards out of the way of the knife. Carry the dissection down in the connective tissue, following the hollow of the sacrum as far as the point where the rectum passes through the pelvic floor. Then, dragging the rectum forwards, divide it transversely above the anus and continue the dissection onwards for a short distance.

It is essential to make a preliminary examination of the anus before the rectum is divided, since the presence of new growths or of other lesions at the anal margin necessitates the removal of the anus in continuity with the bowel above by means of an elliptical incision in the pelvic floor.

Finally, draw the bladder well back from the pubic arch, to avoid damage to the prostate, and divide the urethra close to the triangular ligament. A few additional touches of the knife will now allow the whole mass of pelvic viscera to be lifted out for detailed examination.

METHOD OF REMOVING THE KIDNEYS AND URETERS IN CONTINUITY WITH THE LOWER URINARY PASSAGES

This method of removal should be adopted when disease of the bladder or lower urinary passages is suspected, when the ureters are dilated, or when hydronephrosis or pyonephrosis is present.

First isolate the kidneys in the manner already described (p. 140), but, instead of dividing the ureters, dissect them

out from the retroperitoneal connective tissue in which they lie, following them down over the pelvic brim.

The presence of abnormal vessels crossing and compressing the ureters, of sharp twists or other obstructions, should be noticed before the ureters are lifted from their beds. Then, having drawn the kidneys and ureters forwards out of harm's way, extract the pelvic viscera in the manner already described. The continuity of the kidneys and urinary passages is thus maintained.

The presence of urethral stricture, cystitis, or extravasation of urine will necessitate the removal of the urethra in continuity with the urinary bladder. In such cases the pelvic viscera should be separated from their connexions in the ordinary way, but the urethra must not be cut across behind the symphysis. The median abdominal incision is continued over the front of the pubes and along the dorsum of the penis as far as the corona glandis. The body of the penis is shelled out from its integuments and divided transversely close behind the glans, the latter being left attached to the skin. The denuded stump is then pulled upwards and its under surface carefully liberated from the superficial structures of the perineum, keeping the knife well above the scrotum. As soon as the attachments of the crura come into view they should be separated from the ischial rami. The pubic arch is then cleared and sawn out, the horizontal rami being divided an inch away from the symphysis and the descending rami at their junctions with the ischia. The pubic bones are thus removed with the soft parts of the pelvis, and can be dissected from the urethra without injury to the latter.

A less trustworthy method is to omit the removal of the pubic arch, the body of the penis being dissected free from it and passed underneath into the pelvis, for removal with the bladder. A metal sound should be passed into the urethra as a guide whilst this dissection is carried out, but even with this precaution there is great danger of mutilating the canal.

The pelvic viscera, having been removed, should be placed on a table and examined in the following order: first the rectum and lower end of the colon, then the urinary passages, and finally the vesiculæ seminales and the prostate.

EXAMINATION OF THE RECTUM AND LOWER END OF THE COLON

Cleanse the bowel thoroughly by passing a stream of water through it from above. Hard fæcal masses may need to be removed by gentle expression. Open up the rectum in the posterior median line with bowel scissors, commencing at the lower end. Cleanse the mucous membrane by gentle sponging. Recognise the small anal valves which lie at the junction of the contracted anal canal with the rectum proper (muco-cutaneous junction). Do not overlook the presence of dilated hæmorrhoidal veins.

Hæmorrhoids.—Internal hæmorrhoids project beneath the mucous membrane at the upper part of the anal canal. They may not be very conspicuous after death. External hæmorrhoids lie beneath the skin at the anal orifice; they appear as small tags of skin, unless thrombosed.

Anal Fissure.—Fissures of the anus tend to lie posteriorly. In most cases they are due to the tearing down of an anal valve which, in a swollen and œdematous condition, forms the so-called sentinel pile at the lower end of the fissure.

Proctitis.—Acute or chronic inflammations of the rectum are rarely found post-mortem. They do not differ in appearance from similar inflammations of other parts of the bowel.

Ulceration of the Rectum.—A simple chronic ulcer is sometimes found posteriorly, just above the anal canal. The rectum may participate in simple, dysenteric or tuberculous ulceration of the colon: the nature of this ulceration is revealed by its associations. A widespread ulceration of the rectum which appears to commence at the anus and

spread upwards, leading to extensive cicatrisation, is sometimes present. It is said to be more common in women than men, and is attributed either to syphilitic or gonorrhœal inflammation.

Stricture of the Rectum.—If a stricture is present, determine whether it is due to changes outside the bowel or in the wall itself. Strictures due to pelvic cellulitis lie outside the bowel, a short distance above the anal canal. Syphilitic strictures involve the lower part of the rectum; it is exceptional to find submucous gummata or serpiginous ulcers in connexion with these, but should either occur the nature of the stricture becomes evident. Strictures due to other forms of chronic inflammation or ulceration may be met with.

Fistulæ.—The openings of fistulæ may be found just within the anus, sometimes between the internal and external sphincters, but usually superficial to the latter. They may be felt as small dimples and indurations. They should be explored with a probe and their connexions ascertained. Tuberculous fistulæ often have ragged and irregular orifices, surrounded by blue and undermined skin: they occur in the subjects of tuberculous disease. Fistulous openings may communicate with ischio-rectal abscesses.

Fistulous Communications may be present between the Rectum and the Bladder or Vagina. These communications may be of traumatic origin (such as childbirth); due to the ulceration of malignant disease; or, more rarely, to rupture of tuberculous abscesses of the vesiculæ seminales into both bladder and rectum.

Rectal Polyps.—These as a rule occur low down and posteriorly. They are of adenomatous structure, and have an appearance which resembles a raspberry.

Malignant Disease of the Rectum.—This is usually a columnar-celled carcinoma. It may produce an annular stricture, the bowel wall being infiltrated with firm, white growth, or may project as a definite tumour into the lumen. Cancerous ulcers have the usual indurated, rampart edges. Secondary growths may occur in the rectal, iliac, or lumbar glands, and in the liver.

Growths which originate near the anal margin are squamous carcinomata. Secondary growths may appear in the inguinal glands when the primary disease occupies the anal region.

Prolapse and Intussusception of the Rectum sometimes occur. The intussusception may or may not depend on the presence of polyp or growth.

Malformations of the Rectum include congenital strictures; imperforate conditions; absence of anus or rectum; and openings of the rectum in abnormal situations such as the urinary bladder, vagina, urethra, or vulva. They call for careful dissection.

EXAMINATION OF THE URINARY BLADDER

If the bladder contains urine it may, after it has been removed from the pelvis, be partially emptied by expression. In this way a specimen may be obtained for chemical examination. If a bacteriological examination is necessary some urine should be removed by a sterilised catheter with strict aseptic precautions, or by incision through the seared bladder wall.

It may be possible to detect the presence of certain poisons or of sugar in the urine which is obtained after death. The presence of a trace of albumen may be of no significance, but a larger quantity is of importance. Ammoniacal decomposition is present in some cases of cystitis, but since some organisms have not the power of decomposing urea, it is possible for certain forms of cystitis (*coli-cystitis*, for example) to coexist with acid urine. Blood, pus, or mucus may be found in the urine after death, but before any weight is attached to the presence of these substances, the possibility of contamination of the urine during removal must be excluded. Contamination with blood will necessarily lead to the presence of albumen. An empty condition of the bladder is no proof of suppression of urine, but, on the other hand, the presence of urine indicates that the kidneys were active. The

occurrence of a much distended bladder without obstruction of the urinary passages should always lead to a careful examination of the central nervous system. Cystitis, which may lead to ascending infection of the kidneys, is common in the terminal stages of certain diseases of the spinal cord and brain.

In cases of suspected poisoning the urine should at once be transferred to a clean stoppered bottle and sealed up pending chemical examination.

Open the bladder with scissors, introducing one blade into the urethra and cutting upwards in the anterior median line towards the fundus of the organ. The use of blunt-pointed scissors will obviate any risk of damage to the lax mucous membrane.

Inspect the interior of the bladder as soon as it is exposed ; gently stretch the mucous membrane with the fingers to obliterate the rugæ and determine the condition of the mucous membrane between the ridges. Note the position and relations of any sacculations which may be present and also the nature of their contents, such as calculi, decomposing urine, or pus.

Examine the trigone. If any projections are present in the region of the prostate ascertain whether they raise or in any way obstruct the urethral orifice ; notice also any sagging down of the bladder wall behind the prostate in the form of a pouch. Next recognise the inter-ureteric bar, and at its two extremities locate the ureteric orifices. Explore the latter with a fine probe.

When drawing conclusions as to the presence of hypertrophy of the urinary bladder it is essential to determine whether the viscus was distended or contracted when removed. A contracted condition leads to thickening of the walls, which may be mistaken for hypertrophy. If the interior of the bladder appears fasciculated, owing to the prominence of the muscular ridges, hypertrophy is undoubtedly present.

Injuries to the Bladder.—If a rent is present in the wall of the bladder ascertain whether it is intra- or extra-peritoneal or both. Examine the wall of the bladder for evidence of pre-existing disease and the bony walls of the pelvis for fractures.

Acute Cystitis gives rise to swelling and injection of the mucous membrane, which is most marked in cases where the urine has become ammoniacal. Petechial hæmorrhages and adherent exudation may also be present.

Chronic Cystitis.—The mucous membrane is thickened and may even have a leathery consistence. Its veins are injected, its surface discoloured and granular or superficially ulcerated; a coating of phosphates is often present. Hypertrophy and sacculation may coexist with cystitis in the male, when the condition is due to urethral obstruction. Cystitis in the female is often independent of obstruction.

Tuberculosis of the Bladder.—In the early stages grey or yellow tubercles are found in the neighbourhood of the ureteric orifices. Ulceration ensues, and by coalescence the ulcers may form sharp-edged, sinuous tracts. Cystitis coexists, and the bladder becomes shrunken and thickened. In advanced cases, fistulous communications with neighbouring viscera may occur.

Note, the kidneys, ureters, prostate, vasa deferentia, seminal vesicles, and testes should all be examined in cases of bladder tuberculosis.

Ulceration of the Bladder may be the result of injury, inflammation, tuberculosis, malignant disease, or the presence of foreign bodies. Occasionally a simple ulcer is found in the trigone or on the posterior wall.

Carcinoma of the Bladder is usually of the squamous-celled type and commences near the trigone, where it gives rise to an ulcer with infiltrated and indurated edges.

Secondary invasion from the rectum, vagina, uterus or prostate is, however, more common than primary disease, and then the microscopical appearances will depend on the nature of the primary growth.

A general thickening of the bladder wall, without the

formation of a distinct tumour, and without ulceration of the mucous membrane, is sometimes due to a diffuse infiltration with glandular carcinoma.

Sarcoma of the Bladder tends to form prominent intravesical masses, pedunculated or sessile, which spring from the base or posterior wall. The bladder walls are infiltrated. The growth may be firm and fleshy or soft and villous, and, in the latter case, microscopical examination may be necessary to distinguish it from a papilloma. Vesical growths in young subjects are usually sarcomatous; they may be multiple.

Papillomata of the Bladder appear as villous tufts or shaggy, flocculent transformations of the mucous membrane. Microscopical examination of the bases of such tumours may reveal evidence of carcinomatous or sarcomatous transformation.

EXAMINATION OF THE URETERS

If the ureters have become twisted on their long axes during removal, untwist them. Find the ureteric slits at the lateral angles of the trigone. Inspect the orifices for thickening, retraction, obstruction, or other abnormalities, and then pass a probe into them and along the ureters. Using the probe as a guide, and commencing at the bladder, lay open the whole length of each ureter up to the pelvis of the kidney.

Inspect the interior for inflammation, constriction, or ulceration, and palpate the wall to detect localised thickenings.

Malformations of the Ureter. — The ureter of one side may be double in part or the whole of its course.

Inflammation of the Ureter may extend upwards from a cystitis or downwards from a pyelitis. The condition may be acute or chronic, and the changes produced in the mucous membrane do not differ from those already described in connexion with the mucous membrane of the renal pelvis (p. 149). The inflamed ureter is often dilated.

Calculi, often elongated in shape and resembling date-stones, may be found impacted in the ureter near its vesical orifice. Stones may also lie near the point where the ureter turns over the brim of the pelvis or just above the narrow orifice of communication between the renal pelvis and commencement of the ureter.

Dilatation of one or both Ureters may be due to obstruction within or without the tubes. Obstructions by kinks or abnormal vessels will have been noticed already during removal of the ureters from the body. In the male obstruction at the base of the bladder or in the urethra should not be overlooked (see p. 197). In the female pelvic cellulitis, malignant disease of the cervix uteri or uterine prolapse may act as special causes. A moderate dilatation of the ureters and renal pelves may be found in women who have died in, or shortly after, labour at full term.

In some cases of dilatation of both ureters, no obstruction is found; such cases are commonly supposed to be of congenital origin, but possibly arise from inflammation.

EXAMINATION OF THE URETHRA OF THE MALE

The urethra should be laid open along its dorsal aspect with scissors, and the incision continued along the anterior aspect of the bladder. The scissors should be blunt-pointed to avoid lacerations of the urethral mucous membrane. It may be necessary to guide the scissors through strictures with a fine probe or a director, or even to cut down upon the stricture from outside the urethra. When the urethra has been laid open it should be inspected for irregularities of calibre and palpated between the fingers in order to detect any localised indurations of its walls.

Strictures of the Urethra may appear as irregular tracks of induration or as fibrous rings or bridles. In some cases they are rather difficult to detect after the tube has

been laid open, but a difference in the size of the canal above and below the point of obstruction may draw attention to their position. The tissues surrounding a stricture may be indurated and the mucous membrane above may be ulcerated, pouched, or the site of periurethral suppuration. Any false passages should be carefully probed and their connexions traced. Strictures are more common in the bulbous portion of the urethra than elsewhere. Extravasation of urine into the tissues of the scrotum and its neighbourhood may result from the giving way of the urethra behind a stricture, rupture of a urinary abscess, or injuries inflicted on the perineum (see p. 179).

Calculi are sometimes lodged in the urethra. They may have been formed *in situ*, and consist mainly of phosphates, or have passed into the canal from above, when they are usually fawn coloured and consist of urates.

Malformations, such as hypospadias or epispadias, and openings of the urethra in abnormal situations may occur. Malformations of the urethra may be associated with extroversion of the urinary bladder.

Malignant Disease of the Penis. — Squamous carcinoma may arise in the prepuce or on the glans. Tumours of the erectile tissue are Sarcomatous. The inguinal glands are enlarged.

Malignant Disease of the Urethra is rare.

EXAMINATION OF THE VESICULÆ SEMINALES

Divide the peritoneum of the recto-vesical pouch at the point where it is reflected on to the urinary bladder. Lift up the cut edges, and snip the subjacent connective tissue with the scissors until each seminal vesicle comes into view. Continue the dissection until the vesicles are fully exposed as far as the base of the prostate. The vasa deferentia should also be recognised as they pass downwards in the interval between the two sacs.

The vesiculæ, when healthy, appear as bluish-grey, firm, convoluted sacs. They should be examined by making a

series of sections across them, exposing the thin-walled honeycombed spaces of which they consist and the viscid, yellowish fluid which they contain.

Distension of the Vesiculæ Seminales.—In consequence of obstruction of the ejaculatory ducts by prostatic disease, the vesicles may become distended with retained secretion.

Inflammation of the Vesiculæ Seminales may be acute, leading to abscess formation, or chronic, resulting in thickening and induration of their walls. In all such cases the urethra should be examined for evidence of inflammation or stricture, especially in the neighbourhood of the prostate gland.

Tuberculous Disease of the Vesiculæ Seminales, leading to caseation or softening, may occur in connexion with similar disease of the kidney, ureter, trigone of the bladder, prostate, epididymis, or vas deferens.

Concretions of small size and white colour may sometimes occur in the seminal vesicles. They consist of phosphate of lime.

Malignant Disease of the Vesiculæ Seminales is, as a rule, secondary to carcinoma of the prostate, urinary bladder, or rectum.

EXAMINATION OF THE PROSTATE GLAND

The vesical aspect of the prostate is examined from the interior of the bladder. Notice the shape and position of the internal orifice of the urethra. It may have been raised above the level of the base of the bladder. Nodular projections or irregularities in the trigone may be due to prostatic adenomata. Infiltration or ulceration of the overlying vesical mucous membrane will indicate the presence of tuberculous or malignant disease.

The rectal aspect of the prostate is seen when the bowel has been laid open. Dissect off the rectum. See if the vasa deferentia have, by traction, obstructed the ureters.

As a general rule the prostatic urethra may be laid open from the front as already described, and the examination of the gland completed by a series of transverse sections carried from before backwards. In cases of great enlargement it is preferable to omit the preliminary opening of the urethra and carry out the examination by a series of transverse sections only, as the relations of the prostatic urethra are better determined by this method. The prostatic utricle and the ejaculatory ducts should be recognised in the sections.

The Weight of a Normal Adult Prostate is $4\frac{1}{2}$ drachms (20·5 grms.). Its measurements are : base to apex, $1\frac{1}{4}$ inches (30 mm.), greatest transverse measurement, $1\frac{1}{2}$ inches (36 mm.), maximum antero-posterior measurement, $\frac{3}{4}$ inch (18 mm.).

Acute Prostatitis.—The gland is swollen and the surrounding venous plexus may be engorged. On section a reddish turbid fluid exudes, which in the later stages may become purulent. The brunt of the inflammation may fall either on the prostate itself or the periprostatic tissues. Abscess formation may occur. The urethra should be examined for evidence of past or present inflammation or stricture.

Tuberculosis of the Prostate may be secondary to tuberculous disease of other parts of the genital or urinary tract, or to tuberculosis elsewhere. The tendency is to caseation and softening. Perforation may occur into the bladder, bowel, ischio-rectal fossa or peritoneal cavity. The abscess cavities tend to become septic.

Prostatic Adenomata.—These first appear as ill-defined, opaque, white masses in the gland. Later they assume the form of distinct but conglomerate tumours of a yellowish colour and surrounded by concentric laminæ of prostatic stroma. The tumours consist of glandular elements.

In some cases a diffuse change, probably adenomatous without encapsulation, occurs. On section the substance of the prostate then appears to be flecked with white or yellow areas.

Prostatic Carcinoma may arise as a primary disease or in a prostate which is already adenomatous. In the former case the gland is not necessarily much enlarged, but it is hard, and on section appears tough and fibrous. A microscopic examination is often necessary to determine the true nature of the change, but there are certain characteristics which indicate malignancy. These are, extension to the trigone of the bladder in the form of submucous nodules which ulcerate; extension to the neighbourhood of the vesiculæ seminales or to the lateral wall of the pelvis; infiltration of the pelvic, iliac and lumbar glands and appearance of secondary growths in the liver.

EXAMINATION OF THE EXTERNAL GENITALS OF THE FEMALE

The External Genital Organs should be inspected before the contents of the pelvis are removed.

Separate the thighs widely and examine the parts in a good light. First inspect the Labia Majora; then separate them and expose the Labia Minora; in infantile and wasted conditions the latter may project beyond the labia majora. Investigate the condition of the Vaginal Orifice and of the Hymen, noticing particularly any lacerations or discharges which may be present. Finally, pay attention to the condition of the Perineal Body. Note any tendency to prolapse of the pelvic floor. If the perineum is torn, determine the extent of the tear and find out whether it extends into the rectum.

Bruising, Laceration or Hæmatoma of the Labium Majus may be the result of injury or of childbirth.

Varicocele of the Labium is generally associated with pregnancy.

Inflammatory Infiltrations and sometimes **Ulcerations** of various kinds may occur.

The Gland of Bartholin may be the site of an

Abscess or a Cyst. In such cases an ovoid swelling is found in the posterior part of the labium and should be examined by incision. The duct of the gland lies within the labium minus, close to the attachment of the hymen.

An Inguinal Hernia may give rise to a swelling in the anterior part of the labium, running in the direction of the inguinal canal.

A Hydrocele of the Canal of Nuck occupies a similar position to that in which an inguinal hernia appears.

Carcinoma of the Vulva generally arises in the clitoris or one of the labia majora. It is of the squamous-celled variety.

Purulent Urethritis, allowing pus to be squeezed from the urethral orifice by pressure of the forefinger on the anterior vaginal wall, indicates gonorrhœal infection even though the vagina may show little or no signs of inflammation. Detection of gonococci will confirm the diagnosis.

REMOVAL OF THE PELVIC VISCERA OF THE FEMALE

The preliminary inspection is carried out as in the male, attention being directed to the following additional points: the position of the uterus with regard to flexions, versions and lateral displacement; also its size; the appearance and positions of the Fallopian tubes and ovaries; the condition of the femoral, iliac and ovarian veins with regard to thrombosis. The veins in particular should be inspected, and, if necessary, opened up before the viscera of the pelvis are disturbed. If thrombosed the ovarian veins should be traced through the broad ligaments.

Commence the removal of the pelvic viscera by drawing the bladder backwards from the pubes, dividing the peritoneum and areolar tissue with the knife. Carry the incision round at the level of the pelvic brim, deepening it in the planes of connective tissue until the pelvic outlet is reached. The ovaries and broad ligaments must be drawn inwards during the process. Posteriorly the incision

is made across the promontory of the sacrum and carried downwards in the hollow of the bone, so that the rectum is separated from it. Finally the rectum and vagina are divided transversely close to the pelvic floor and the urethra is severed behind the pubic arch.

During the separation of the planes of pelvic connective tissue, the presence and distribution of any pelvic cellulitis and the position of any abscesses which may lie in the connective tissue must be noticed.

It may be desirable to remove the vagina and labia minora intact, instead of dividing the former at the pelvic outlet. To effect removal in this way the thighs should be widely separated and an incision traced with a large scalpel in the perineum. This incision should commence close under the pubic symphysis well above the clitoris and be carried downwards and backwards on each side just external to the labium minus, and then across, behind the posterior border of the vagina. Proceed to remove the viscera from above as already described, dividing the rectum above the pelvic floor, but freeing the vagina and labia by carrying the point of the knife through the incision already traced in the perineum.

Having removed the pelvic contents proceed with the examination of the rectum as already described (p. 189).

EXAMINATION OF THE URETHRA, BLADDER, AND VAGINA

Open up and examine the urethra and bladder.

Vascular Caruncles occur as small red growths near the urinary meatus.

Slight Prolapse of the mucous membrane of the female urethra is not uncommon.

Urethritis may be the only evidence of gonorrhœa.

Distortion and Stretching of the Urethra may be found when the uterus is prolapsed, retroverted, or otherwise displaced.

For lesions of the bladder see p. 193.

Dissect the bladder from the vagina and then proceed to the examination of the vagina, uterus, Fallopian tubes, and ovaries.

The vagina should be opened with scissors in the anterior mid-line. Its mucous lining can then be inspected and the condition of the vaginal fornices and cervix uteri ascertained. Notice the amount of projection of the cervix uteri into the vagina and the shape of the os externum. Examine both cervix and fornices for lacerations, or for scars of fissures which have healed.

Developmental Abnormalities of the Vagina.—The vagina may be absent, rudimentary or malformed. An imperforate hymen or a transverse vaginal septum above the hymen may give rise to retention of menstrual products in the vagina (hæmatocolpos), or in the uterus (hæmatometra).

Cysts of the Vaginal Wall appear to arise in unobliterated remnants of Gärtner's duct (Wolffian duct). Sometimes they can be traced upwards in the broad ligament at the side of the uterus.

Vaginitis.—Injection of the vaginal mucous membrane is not very evident after death, but in consequence of very acute inflammation, œdema and petechial hæmorrhages may be found. Vaginitis, associated with purulent urethritis, usually is of gonorrhœal origin.

Chronic Vaginitis may give rise to small hard granulations in the mucous membrane.

Pus in the Vagina may be due to vaginitis or be derived from a pelvic abscess which has opened into the canal. Sometimes the pus will be found to exude from the uterus.

Ulceration of the Vagina.—Superficial erosions may be the result of vaginitis. Deep, infiltrated ulcers may be due to retention of pessaries or other foreign bodies. When the uterus is prolapsed, pressure ulcers may occur on the anterior vaginal wall and near the attachment of the cervix. Ulceration of the vagina may also be the result of injuries during childbirth. In consequence of severe

laceration or ulceration the paravaginal connective tissue may become infected and pelvic cellulitis ensue.

Syphilitic Inflammation of the Vagina may occur in the form of mucous tubercles, or, in advanced cases, as deep gummatous ulcerations with sinuous outlines.

Tuberculosis of the Vagina, either primary or secondary, is very rare. The lesions do not differ in appearance from those produced by tuberculous disease of other mucous membranes.

Fistulous Communications between the vagina on the one hand and the rectum, urethra, bladder, or ureter on the other, may be the results of injuries sustained during parturition, of operative interference, or of deep ulcerations.

Carcinoma of the Vagina as a rule is secondary and due to extension from the uterus, bladder or rectum. Less commonly it occurs as a primary growth on the upper part of the posterior vaginal wall. Carcinoma usually appears in the form of an ulcer with infiltrated walls and indurated base, but occasionally a large mass of friable growth is found.

Sarcoma of the Vagina.—Sarcoma may occur in childhood as a polypoid tumour of the anterior vaginal wall with a tendency to invade adjacent parts. In the adult it may appear either as a polypoid mass or as a diffuse growth.

The lymphatics of the vagina terminate in the iliac and hypogastric glands of the pelvis and also in glands situated on the promontory. There is a free communication between the lower vaginal and the vulval lymphatics, the latter passing to the superficial inguinal glands. All these glands should be examined if new growths are present.

A Cervical Polyp or the **Fundus of the Inverted Uterus** may project into the vagina. A polyp is likely to be smooth and glistening, but the inverted uterus will have a reddish, shaggy appearance. The nature of the protrusion will be cleared up by examination of the uterus.

EXAMINATION OF THE UTERUS

After examination of the exterior of the uterus and also of the vaginal portion of the cervix, carefully introduce a probe or director into its interior, and measure the distance

to which it enters. Open the uterus by partially dividing the anterior wall in a longitudinal direction on a director, and then completing the incision with the scissors introduced into the cervix. At its upper end the incision should be prolonged towards the orifice of each Fallopian tube at the lateral angles of the uterine cavity. The interior of the uterus should be closely inspected and the nature of any contents determined. Ascertain the consistence of the uterine walls by palpation, and notice the presence of any localised indurations, such as may be caused by fibromyomata.

That portion of each Fallopian tube which is embedded in the uterine muscle must be examined by incisions made at right angles to its long axis. Sections made in this way not only expose the lumen of the tube, but also lay bare certain important vessels and lymphatics in the immediate neighbourhood. The bases of the broad ligaments should be examined by vertical incisions across them.

Size and Weight of the Uterus.—The length of the adult uterus is 3 inches, the width 2 inches, and the thickness 1 inch. The cavity, measured from the external os to the fundus, is $2\frac{1}{2}$ inches. The weight is $1\frac{1}{2}$ to $1\frac{3}{4}$ oz. (32–50 grms.). These measurements are somewhat greater in the case of a woman who has borne a child, and the weight is then $1\frac{3}{4}$ to $2\frac{1}{2}$ ozs. (48–70 grms.).

In old age the uterus becomes atrophic, sometimes to a very considerable degree. Its muscular tissue is paler and denser. The ostium internum is frequently, and the ostium externum occasionally, obliterated.

Malpositions of the Cervix.—If the cervix lies low in the vagina the uterus may be found prolapsed, or the vaginal (or supravaginal) portion of the cervix may be hypertrophied.

Pin-hole Os.—A minute, circular os externum is usually associated with a conical cervix. The uterus in these cases is undersized.

Parous Os.—The parous os is larger and more elongated transversely than in the virgin condition. It may be irregular in outline owing to the presence of lacerations. The tears may be recent and raw or old and scarred. (For the appearances presented soon after delivery, see below.)

Cervical Erosions or Adenomata appear as soft, red, velvety masses which are not necessarily ulcerated. They should be examined by incision, and must not be mistaken for malignant growths.

Retention Cysts of small size with clear contents may be found in the cervical mucous membrane near the os externum or in the neighbourhood of erosions. They arise in the glands of the cervix and are sometimes known as Nabothian follicles.

Squamous-celled Carcinoma may arise from the vaginal aspect of the cervix (see p. 208).

Characters of the Adult Nulliparous Uterus.—The contour of the body is more or less triangular. The external os is circular or very slightly elongated and not fissured. The cervical canal is nearly equal in length to the cavity of the body, and its arbor vitæ is well marked. The walls of the body bulge inwards towards the lumen.

Characters of the Uterus which has been Parous.—The contour tends to be globular. The external os is transverse, irregular, or fissured. The cervical canal is shorter and smoother than in the nulliparous organ. The cavity of the uterus is wider and longer and its walls are not convex inwards. The fundus rises higher between the points of attachment of the Fallopian tubes than it does in the virgin. (See also size and weight, p. 204.)

Characters of the Uterus shortly after Childbirth.—The cervix is thick and short with partly everted lips. The body forms a large, flattened sac. The length of the cavity, immediately after delivery, is from 9–12 inches, but a day or two later it measures 7 inches. The wall, in the flaccid condition, has a thickness of from a $\frac{1}{2}$ to $\frac{3}{4}$ inch. The weight is about $3\frac{1}{4}$ lbs. The cavity, when laid open, is pear-shaped. A ragged, purplish patch on the anterior

or posterior wall, near the fundus, marks the placental site; the openings of the uterine sinuses, which are usually thrombosed, may be recognised in the discoloured area. The interior of the cervix is bluish and ecchymosed. The cervical canal is smooth, but the rest of the interior is covered by shreddy, reddish decidua and coated with lochial discharge: these appearances should not be mistaken for signs of inflammation. The Fallopian tubes, round ligaments, and ovaries have a purplish, congested appearance.

Within fourteen days of childbirth the cervical canal narrows down, and the internal os again becomes distinguishable. The lining of mucous membrane is regenerated by the fourth week; at first it is thin and pale, and for some time the placental site may be recognised by its discoloured appearance.

At the end of the sixth week the cervix, except for any lacerations, appears quite normal. Complete involution requires from six to eight weeks.

After childbirth the vagina is capacious and the perineum relaxed. Bruising and lacerations may be present. In medico-legal cases particular attention should be paid to any punctures or lacerations in the cervix or vaginal fornices, such as may have been caused by instrumental attempts to induce abortion.

Rupture of the cervix or vagina may occur during childbirth. The cervix and adjacent part of the lower segment of the uterus may be found thinned out, but may be distinguished from the vaginal walls by a slight zone of thickening and pallor at the junction with the latter. The relations of the rupture to the peritoneal cavity and to the pelvic connective tissue planes should be determined.

Characters of the Uterus after Abortion.—The length of the uterus should be ascertained, and if a fœtus has been found, the probable age of the latter. The placental site can usually be made out after the third week of gestation. Note if any uterine mucous membrane is present. Careful search should be made for punctures, incisions, or lacerations in the vagina, uterus, and neighbourhood. The cervical ecchymoses of recent delivery should not be mistaken for evidence of criminal violence.

Notice any signs of inflammation of the uterus or its connective tissue and peritoneal investments.

After the lapse of three or four weeks, especially if the pregnancy were early, all signs of abortion may have disappeared.

Characters of the Uterus during Menstruation.—The uterus is slightly swollen; its mucous membrane is tumid and dark red. The external os pouts slightly and is patulous; menstrual secretion may be seen exuding.

Character of the Uterus in Puerperal Infection.—The interior of the uterus and the surfaces of any vulval, vaginal, or cervical lacerations may be sloughy and have an offensive odour. The uterine wall is œdematous and sometimes even gangrenous. The muscular coat may be completely exposed by disintegration of the mucous membrane. The inflammatory changes are particularly marked at the placental site, and fragments of placenta may be still adherent. The uterine lymphatics may be prominent and contain pus, less commonly pus is found in the uterine veins. Purulent infection of the Fallopian tubes may also be present. Inflammation and abscess formation may occur in the connective tissue basis of the broad ligaments. When infection spreads into the broad ligaments from lacerations of the cervix, the bases of the ligaments are chiefly involved. Inflammation of these ligaments may spread in various directions in the pelvic connective tissue, constituting pelvic cellulitis. Suppuration may ensue. The pelvic peritoneum is usually inflamed, infection being carried to it by the uterine lymphatics and the Fallopian tubes.

The most virulent forms of puerperal infection may cause death before the local changes become at all pronounced.

Chronic Metritis (Fibrous Uterus).—The uterus is enlarged. On section its walls are thick, pinkish, and firm, and the arteries may be decidedly thickened (arteriosclerosis). In addition fibromyomata often coexist. The endometrium shows no thickening.

Fibromyomata of the Uterus.—These occur as dense, more or less spherical tumours, which may be multiple, and in some instances attain a very great size. They tend to

become encapsuled by condensed uterine tissue and can often be shelled out. Sections of the tumours present a pale and characteristically whorled appearance. The masses are softer and pinker when muscular tissue is in excess. The softer fibroids may possess no capsule. Subperitoneal or submucous fibroids may become pedunculated, the latter then form one variety of uterine polyp. Degenerative processes leading to necrosis or calcification are not uncommon; malignant transformation also may occur. Fibromyomata may cause death by repeated hæmorrhage; by pressure on the bowel, urethra or ureters; by degenerations leading to peritonitis; or by complications ensuing on pregnancy.

Diffuse Adenomyomata of the Uterus differ from fibromyomata in being diffuse and not encapsuled. The new tissue, which is spongy-looking, can, on close inspection, be differentiated from the investing muscular coat of the uterus. The growth contains glandular elements, derived from the endometrium, lying in a richly cellular connective tissue, which is itself embedded in a fibromyomatous matrix.

Polypi of the Uterus.—Any pedunculated mass projecting into the cavity of the uterus is known as a polyp. Fibroid polypi are submucous fibroids in process of extrusion. Mucous polypi are adenomatous projections from the mucous membrane, usually of the cervix. They may be multiple, and sometimes are cystic. Placental polypi consist of portions of retained placenta coated with fibrin.

Tuberculous Infection of the Uterus is uncommon. The disease may occur in miliary, polypoid, caseous, or ulcerating forms. A papillary form of tuberculosis which affects the arbor vitæ of the cervix may attain a considerable size and mimic carcinoma.

Carcinoma of the Uterus.—Cancer arising on the vaginal aspect of the cervix is squamous-celled; it may appear as a typical carcinomatous ulcer with indurated edges, or as a warty mass. It tends to invade contiguous parts.

Carcinoma of the cervical canal (adeno-carcinoma) may appear as a deeply eroding ulcer or as a fungating growth. It tends to spread to the body of the uterus on the one hand

and the bases of the broad ligaments or the vaginal walls on the other. The bladder, rectum, ureters and uterine vessels may one or all be implicated. Purulent infection of the body of the uterus and of the Fallopian tubes may coexist with cervical carcinoma.

Carcinoma of the body of the uterus may appear as an eroding ulcer or a vascular polypoid growth (the so-called villous endometritis). It is glandular in structure. In all cases of uterine cancer the lumbar as well as the iliac glands should be examined for secondary growths. Other parts of the body may also be infected.

Sarcoma of the Uterus is rare and must be distinguished from other tumours by microscopical examination.

Deciduoma Malignum (*Chorion-epithelioma*).—This is a form of malignant disease which may follow abortion or the expulsion of a hydatidiform mole. Exceptionally it occurs after labour at term. The uterus is the site of growths which vary in size and colour, but usually are dark purplish-red. The growth is soft and closely resembles placental tissue in its irregularly fissured surface, its fibrous appearance on section, its extreme vascularity and its tendency to blood extravasation. Secondary growths are generally found in the vagina, lungs and other parts of the body. The microscopical appearances are distinctive.

EXAMINATION OF THE FALLOPIAN TUBES

The position of the tubes should be determined as far as possible before the pelvic viscera are in any way disturbed. Inspect both of them for discolorations, swellings, or rents, and carefully palpate their whole length for abnormal thickenings. If the tubes are distended ascertain whether the fimbriated ends are patent or sealed. Leakage of pus from the fimbriated ends may give rise to acute spreading peritonitis or to local abscess. Slow leakage of blood may cause a pelvic hæmatocele. Firm adhesions around the tubes are indicative of past peritonitis.

With the aid of a fine probe and sharp-pointed scissors each tube should be opened along its free margin, commencing at the fimbriated extremity. When the narrow isthmus near the uterine end is reached it is often a matter of great difficulty to pass the probe, and so this part may have to be examined by a series of transverse incisions at right angles to the long axis of the tube. A similar method is adopted for the examination of that part of the tube which traverses the uterine wall (p. 204).

Adhesion of the ovarian fimbria of the tube to the ovary is not pathological, neither is the presence of a small stalked cyst (Hydatid of Morgagni) attached to the fimbriated end. The ampulla or widest part of the tube, which intervenes between the isthmus and the ostium, is normally flexuous and irregularly dilated, its diameter being about $\frac{1}{5}$ inch. The fimbriated end often presents a purplish passive congestion which is not necessarily an indication of disease.

When the pouch of Douglas is the site of acute inflammation or abscess the free end of the tube may become hyperæmic or swollen. If no signs of antecedent disease are found in the uterus or proximal ends of the tubes, the tubal changes are secondary to the pelvic inflammation, for which some other cause than tubal infection must be sought.

If the tubes are distended the fimbriated ends will probably be found to have become sealed up. The tubal contents may be watery (hydrosalpynx), purulent (pyosalpynx), or bloody (hæmatosalpynx). In every case of hæmatosalpynx or of pelvic hæmatocele the suspicion of tubal pregnancy should be strongly entertained.

Hydrosalpynx.—The fimbriated extremity of the tube is sealed as the result of peritonitis originating in some other part, sometimes from the tube of the opposite side. The sealed tube becomes distended with clear, watery fluid, its walls are stretched and thin and the longitudinal plicæ

of its membrane obliterated. The sealed end usually forms a retort-shaped expansion, whilst the middle and proximal portions are convoluted in a complicated manner.

Acute Salpingitis.—The tube is swollen, its mucous membrane congested and œdematous, its lumen filled with purulent or mucopurulent secretion. Acute salpingitis is due to ascending infection. It sometimes coexists with purulent infection of the uterus as a complication of carcinoma of the cervix. Cultures are desirable.

Pyosalpynx.—The fimbriated end is occluded and the tube is distended, lengthened and tortuous. The wall is thickened and may be indurated. The interior, which has a loculated appearance, is filled with pus. The ovary may be suppurating and is nearly always involved, with the tube, in peritoneal adhesions. When pyosalpynx is not a sequel of parturition or abortion it is usually of gonorrhœal origin. Pyosalpynx in the virgin may be tuberculous.

Tuberculous Salpingitis is the most common form of genital tuberculosis in the female. The condition, when chronic, is recognised by the presence of caseous pus in the tube, ulceration of the mucous membrane, and often by a deposit of miliary tubercles in the adjacent peritoneum. Widespread pelvic adhesions may be present. A recent miliary tuberculosis of the tubes is sometimes found.

Hæmatosalpynx.—Distension of a tube with blood almost invariably indicates a tubal pregnancy. An impregnated ovum, arrested in the Fallopian tube, is liable to become the seat of hæmorrhage. The 'tubal mole' thus formed may remain in the tube, or may escape with blood through the ostium into the peritoneal cavity, and the tube remain distended with blood as a hydrosalpynx. The diagnosis of tubal pregnancy may be confirmed by the detection of chorionic villi with the microscope.

When hæmorrhage into the ovum occurs after closure of the tubal ostium, i.e. after the sixth or seventh week, the tube itself may rupture.

Pelvic Hæmatocele.—Blood which escapes slowly from the tube may become encysted and form a pelvic hæmatocele. In such cases the blood is usually extravasated from

the abdominal ostium of the tube. When the escape of blood is considerable and rapid, as occurs when the tube has ruptured, the blood becomes widely diffused in the peritoneal cavity.

Malignant Disease of the Fallopian Tube (*Carcinoma; Sarcoma; Deciduoma or Chorion-epithelioma*) is very rare.

Parasitic Disease of the Fallopian Tube (*Actinomycosis; Hydatid Disease*) also is exceptional.

EXAMINATION OF THE OVARIES

The ovary may occupy its normal position in the ovarian fossa, a peritoneal depression in the angle of bifurcation of the common iliac artery; or may be prolapsed towards the pouch of Douglas. Occasionally it is found in a hernia, which is then, usually, of the inguinal variety and irreducible. (The gland in such cases may be a testis.)

The gland may be buried in adhesions in consequence of disease of the Fallopian tube or of other structures in the neighbourhood.

The healthy ovary of the adult is ovoid in shape and flattened from side to side. Its average dimensions are $1\frac{1}{3}$ inch in length, $\frac{3}{4}$ inch in breadth, and $\frac{1}{2}$ inch in thickness.

The average weight in the adult is nearly $\frac{1}{4}$ oz. (7 grms.).

In childhood the ovary is small and elongated; its surface is smooth and yellow. The smoothness is retained until the onset of puberty and commencement of rupture of the Graafian follicles.

In the adult the ovary has a pearly hue. Darker spots mark the position of follicles containing ova, whilst old and ruptured follicles are indicated by yellowish discolorations or scars.

In old age the ovary is hard, atrophic and much puckered by cicatricial tissue.

The size of the ovary varies, as already indicated, with

age, and also, physiologically, with menstruation and pregnancy. The maximum size of the gland is said to be attained about six weeks after parturition.

Pathological enlargement of the ovary may be due to inflammation or abscess formation, tuberculous disease, cystic transformation or malignant disease.

The consistence of a healthy adult ovary is remarkably hard ; this is still more pronounced after the menopause ; the hardness should not be taken as an indication of chronic ovaritis.

Small superficial cysts of the ovary have no pathological importance.

After inspection, the ovary should be incised longitudinally from the convex border towards the mesovarium. It may be examined equally well by a series of transverse incisions carried from its convex border inwards.

Recognise the two portions of which the ovary consists, i.e. the medullary, vascular territory or paroöphoron and the cortical, parenchymatous zone or oöphoron. The former is pinkish and, although firm, is softer than the latter, which is pale and dense. The cortical zone contains the Graafian follicles, which, from their great numbers, may impart to it a granular appearance. The smallest follicles are superficial, the mature follicles are more deeply seated, but, owing to their larger size, form decided superficial projections. They contain a clear fluid.

In cases of malformation of the genital organs it is unsafe to hazard an opinion as to the nature of the genital gland without making a microscopical examination, for a fimbriated tube may coexist with a genital gland which is a testicle and not an ovary.

If an ovarian cyst is present, the following points require investigation :

(1) The presence or absence of a pedicle and the structures which enter into its formation, i.e. broad ligament, ovarian ligament, etc. ; (2) The exact relation of the cyst to the ovary, Fallopian tube and broad ligament ;

(3) The character of the cyst, whether unilocular or multilocular; (4) The presence or absence of intracystic growths or other intracystic structures; (5) The presence of adhesions; (6) Evidence of axial rotation of the cyst, and if present, the extent and direction of the twist; (7) The occurrence of hæmorrhage into the cyst or of purulent infection of the walls and contents; (8) Evidence of malignancy in the form of infiltration of the lumbar glands, or of secondary growths in the liver or other viscera.

Corpora Lutea.—A corpus luteum is formed by the ingrowth and plication of the vascularised wall of a Graafian follicle after the latter has ruptured. The folded wall assumes a bright yellow colour and imprisons the central blood clot. A contracted, white cicatrix ultimately results.

There is no essential difference in appearance between the corpus luteum of pregnancy and the corpus luteum of menstruation, but the latter passes through its stages much more quickly than the former. The corpus luteum of menstruation is visible for a few months only. The corpus luteum of pregnancy remains of considerable size up to the time of delivery, its diameter being greatest at the third month of gestation when it is about $\frac{2}{3}$ inch; at full term it measures $\frac{2}{5}$ inch or more, and does not entirely disappear until two months later.

Inflammation of the Ovary.—An acutely inflamed ovary is moderately swollen, rather softer than normal, and presents an œdematous, sometimes a reddish, appearance on section. Acute ovaritis occurs in connexion with, and is consequent to, acute tubal inflammation, pelvic peritonitis or general peritonitis. Small ovarian abscesses may coexist.

Chronic Ovaritis may lead to sclerosis and cyst formation. The cysts are small. If the ovary is adherent to neighbouring structures this is good evidence of an inflammatory process, but small, hard, cystic ovaries may be the result of retrograde changes apart from pathological processes. As with acute ovaritis, salpingitis is also a causal accompaniment of chronic inflammation.

Tuberculosis of the Ovary is rare as a primary disease; as a rule it is secondary to tuberculosis of the peritoneum, tubes or uterus. Extensive peritoneal adhesions are usually present. The disease may occur in the miliary or the caseous form, or tuberculous abscesses may be present.

Fibromyoma of the Ovary.—Encapsuled fibromyomata resembling those of the uterus, are uncommon in the ovary. They are unilateral and innocent.

Carcinoma of the Ovary.—Primary carcinoma is rare, but secondary carcinoma, arising in connexion with cancer of other parts, is more frequent and usually bilateral. In all cases of bilateral ovarian growths a careful examination of the breasts, stomach, intestines, gall-bladder and uterus is called for.

Sarcoma of the Ovary may occur in early life and also in the adult. In children bilateral growths, which rapidly attain a great size, may be found. Ascites accompanies ovarian sarcomata as a rule, but may also complicate other ovarian growths and cysts.

Cysts of the Ovary and Broad Ligament.—Cysts may arise from the germinal portion of the ovary or oöphoron; from the tissue of the medulla and hilum, or paroöphoron; and from those developmental residua in the broad ligaments which constitute the parovarium.

Cysts which originate in the Germinal Portion of the Ovary (Oöphoritic Cysts).—Minute cysts of the ovary are very common and of but small importance. They may arise from Graafian follicles or from corpora lutea. Cysts derived from the latter are known as lutein cysts owing to the bright yellow colour of their walls. There is some obscure connexion between large lutein cysts and chorion-epithelioma of the uterus.

Large Cysts arising from the germinal portion of the ovary may be recognised by the relation they bear to the Fallopian tube; on raising the latter from the outside of the cyst, on which it lies curled, a distinct mesosalpynx is found to persist between the tube and the tumour.

These large cysts may be (1) Simple and Unilocular; (2) Adenomatous and Multilocular, with masses of honeycombed tissue projecting from the walls of some of the

loculi; or (3) Teratomatous, the so-called Ovarian Dermoids, which may be recognised from the fact that they contain piliferous projections, sebaceous material, cartilage, teeth, nervous tissue or other embryonic products. Teratomatous cysts may undergo sarcomatous or carcinomatous transformation.

Cysts which arise in the Paroöphoron: Papillomatous Cysts of the Ovary.—These cysts are usually sessile, burrowing into the mesometrium and also into the mesosalpynx. In consequence no mesosalpynx can be demonstrated between the tube and the tumour. Papillomatous cysts are often bilateral, one being larger than the other. Their walls are thin and they comprise but few loculi. The interior of the cysts is beset with soft branching processes or cauliflower masses which may erode the walls, and so appear on the exterior, or the cysts may rupture, in which case the papillomata are exposed. The papillomatous elements have a local malignancy, and when disseminated over the peritoneal cavity become engrafted on the serous membrane. Ascites may occur.

Papillomatous cysts are believed to arise in the tissue of the ovarian hilum (paroöphoron). It is possible that similar cysts may spring from the remains of the parovarium which lie between the layers of the mesovarium, or even from parts of Muller's duct (from which the Fallopian tube is developed), or from Gärtner's duct (Wolffian duct).

Parovarian Cysts.—These are large, thin-walled, unilocular cysts which contain a limpid or opalescent fluid of low specific gravity (1·002–1·007). Cholesterin is sometimes present. The cysts arise from the parovarium and are necessarily sessile, extending between the layers of the broad ligament. The Fallopian tube is, in consequence, tightly stretched over the surface of the cyst and the ovary sessile on the cyst wall.

CHAPTER V

EXAMINATION OF THE HEAD

As a general rule the brain and spinal cord should be removed before the thoracic and abdominal viscera, the reasons being, first, that removal of the thoracic viscera entails escape of blood from the cerebral sinuses and veins. Secondly, it is cleaner and more convenient to turn the body on its face for removal of the cord before the abdominal cavities are opened.

EXAMINATION OF THE SCALP AND PAROTID GLAND

A stout block should be placed under the neck to raise the head from the table. The hair, if long, must be parted along a line joining the two mastoid processes across the vertex.

The face of the subject is turned towards the right shoulder and the left ear firmly grasped by the operator's left hand and drawn forwards.

Insert the point of the post-mortem knife, edge outwards, under the skin which covers the left mastoid process, and carry an incision across the vertex to the mastoid process of the opposite side, turning the head towards the left as far as may be necessary to facilitate the procedure. The incision should divide all the tissues of the scalp from within outwards; unnecessary injury to the hair is thus avoided.

Strip the anterior part of the scalp forwards to the

supraciliary ridges, assisting its liberation by a few touches of the knife. Strip the posterior flap backwards to below the occipital protuberance. The hair, if long, may conveniently be rolled up and tucked away beneath the scalp flaps.

Whilst reflecting the scalp, take particular notice of any bruises, lacerations, or punctures which may come into view. Ascertain also the exact position, with regard to the layers of the scalp, of any hæmorrhage or suppuration which may be present, i.e. whether above or below the aponeurosis of the occipito-frontalis muscle or between the pericranium and the bone.

Pus beneath the aponeurosis tends to spread widely. Blood beneath the pericranium is limited by the sutures to the surface of one bone (cephalhæmatoma neonatorum). Boggy infiltration of the scalp with blood and serum constitutes the caput succedaneum seen in newly-born infants.

The **Parotid Gland** is exposed by continuing the coronal incision downwards, almost to the angle of the jaw, and dissecting the flaps from the surface of the gland. To complete the raising of the anterior flap it is necessary to separate the cartilaginous meatus from the bony margins of the auditory canal. The fascial covering of the gland must also be removed.

Pathological changes in the small lymphatic glands which lie on and in the parotid should not be mistaken for lesions of the salivary gland itself.

The presence of foci of suppuration in the parotid is easily ascertained by making a series of incisions at right angles to the surface. Suppuration may be the result of ascending infection from the mouth.

If a parotid tumour is present its relation to the facial nerve should be determined before the parts are disturbed. Roughly, the nerve may be said to pass downwards and forwards in the lower third of the gland. The parotid gland itself passes deeply towards the side of the pharynx, the carotid artery and deep-lying nerve trunks; above, it reaches to the base of the skull.

The parotid duct may be explored from the interior of the mouth by using a fine probe. The orifice will be found opposite the second upper molar tooth.

METHODS OF OPENING THE SKULL

The skull cap should be removed by a circular incision. The saw cut should pass just above the supraciliary ridges

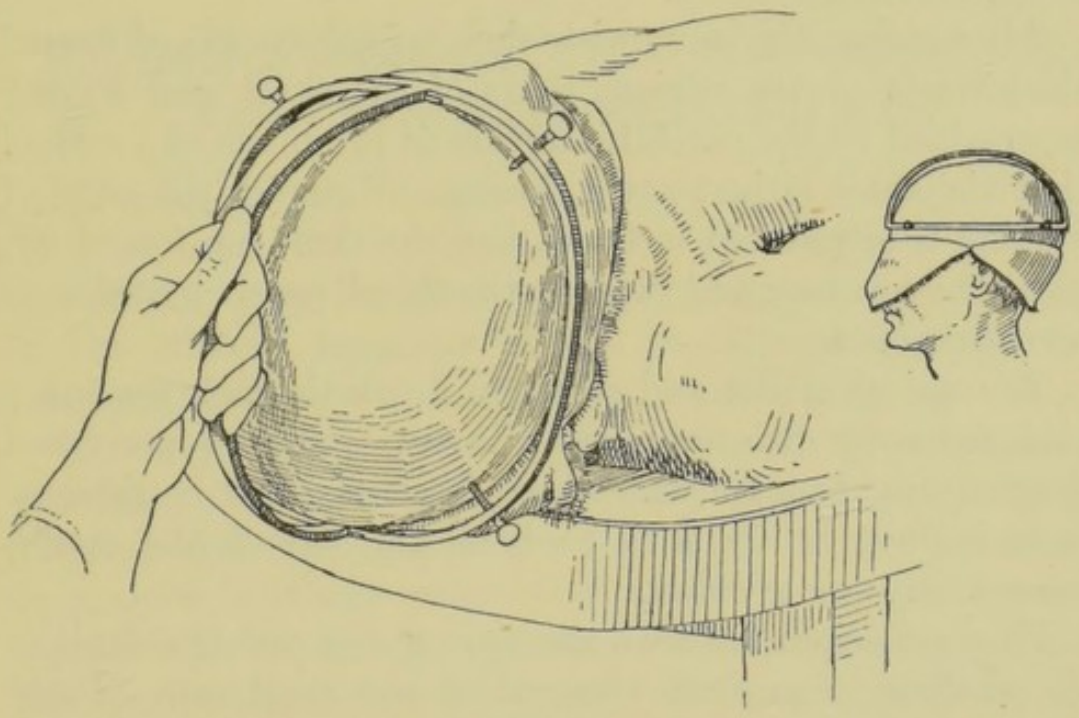

Fig. 16.—Coronet applied to Skull and steadied by Left Hand. Faulty Pattern, which cannot be grasped, shown in Small Sketch.

in front and through the occipital protuberance behind. A preliminary incision should be traced through the periosteum with a sharp scalpel, and the temporal muscles divided or turned down before the saw is applied.

For cutting the bone a small, sharp back-saw is most convenient, the skull being steadied and the saw guided by the use of a steel coronet which can be firmly fixed to the dome of the skull by adjusting screws (Fig. 16). In the absence of a coronet, the left hand must be protected in the folds of a thick towel and used to steady the head.

Great care is requisite in the management of the saw. If the temporal muscles are not thoroughly detached and the pericranium incised, the saw teeth become clogged; if the cut is carried too low across the forehead the saw will pass into the roof of the orbit instead of well above it; unless the saw is used with the greatest care at the sides of the head, where the temporal bones are extremely thin, the brain is damaged, a common fault. If the saw cut is made too high the resulting opening in the skull is too small to permit extraction of the brain.

After using the saw with due regard to the different thicknesses of the various parts of the skull, and having ascertained by the careful insertion of the blade of a scalpel that the skull is just sawn through all round, the edge of a chisel is introduced in front, and the skull cap forced off, partly by leverage and twisting the chisel, partly by traction on the coronet.

The use of mallet and chisel to break through the inner table is hardly ever necessary if the saw cut is properly and carefully made. In cases where fractures may be present, or an inquest is likely to be held, the use of the mallet must always be avoided.

Firm adhesions between the dura mater and the vault of the skull may prevent removal of the skull cap. These adhesions are constant in children and not uncommon in old age. In such cases traction on the skull cap may pull the brain out of its bed and cause considerable damage. The proper procedure is to prize up the skull cap sufficiently to admit a circular division of the dura mater and section of the falx cerebri just above the crista galli. The calvarium will then come away with the adherent portion of the membrane still attached to it.

In infants under six months it is not necessary to saw through the bones of the skull. With a sharp pair of scissors the sutures and adherent dura should be divided from the vertex. First cut downwards between the two halves of the frontal bone, next separate the frontal and

parietal bones in a similar way, then cut backwards along the sagittal suture between the two parietal bones, to the left of the superior longitudinal sinus, and finally cut down on each side between the parietal and occipital bones. The skull is partly opened and the falx cerebri divided from the left side in its whole length just below the superior longitudinal sinus.

The bone flaps and dura can then be forced open like the petals of a flower and the brain exposed.

EXAMINATION OF THE FORAMINA OF THE FOURTH VENTRICLE

In cases of cerebro-spinal or post-basic meningitis, of hydrocephalus, or of tumour in the subtentorial region of the skull, it may be necessary to investigate the condition of the efferent foramina of the fourth ventricle and the relations of the cerebellum to the foramen magnum.

The skull is opened in the manner already described and the dura mater reflected in the usual manner (see p. 226). The cerebral hemispheres are either left *in situ* or raised from their beds and removed by cutting across the mesencephalon just above the opening in the tentorium cerebelli. The dura mater is now separated as far as possible from the inner aspect of the occipital bone, using a rugine or the handle of a scalpel for this purpose. The separation must be continued downwards to the margin of the foramen magnum. A broad, V-shaped sector is next sawn out of the occipital bone, its blunt apex comprising the posterior half of the circumference of the foramen magnum. The wedge is then carefully removed, the dura, which is closely adherent to it in the neighbourhood of the foramen magnum, being separated with the scalpel, the edge of which should be kept towards the bone. It is an advantage if the spinal canal has been previously opened by removal of the vertebral laminæ.

(When the brain and spinal cord are to be removed

together the whole procedure may be carried out with the body in the prone position, it being unnecessary to turn the corpse over for the application of the coronet to the head.)

The occipital aspect of the exposed dura is sponged and incised vertically, the two flaps being drawn aside. The reflexion of the arachnoid from the under surface of the cerebellum on to the spinal cord is now seen. If healthy, it forms a delicate, translucent septum. This should be divided transversely. Then, by raising the cerebellum carefully from the subjacent fourth ventricle, it is possible to investigate the central foramen of Majendie and also the foramina at the lateral recesses of the ventricle. By means of these foramina the ventricles of the brain communicate with the subarachnoid space, their contents passing downwards around the cord.

Before raising the cerebellum it is necessary to determine the extent to which it enters and plugs the foramen magnum, since in some cases it is forced into this aperture by increased intraventricular pressure.

In consequence of meningitis the foramina of the fourth ventricle may be sealed, or, what is equally effectual in occluding them, the cerebellum may become closely adherent to the subjacent surface of the ventricle. Occasionally the arachnoid sheath is found adherent in tubular fashion to the upper cervical cord, although the ventricular apertures are patent.

EXAMINATION OF THE SKULL CAP

The skull cap should be examined as soon as removed. The outer surface is still covered by periosteum, the inner is usually bare, the dura mater having been stripped off. If the latter membrane is still adherent it should be separated. The periosteum may also need removal; this is effected as follows: make a large crucial incision through it with a scalpel and place the calvarium under the stream from a hot-water tap; in a few minutes the softened covering may be stripped off with a rugine.

Make a careful search for fractures, and trace their continuity with any fractures of the skull base. Notice whether they are recent or show signs of repair.

Inspect the cut edges of the bones and notice any evidence of thickening, thinning, condensation, or rarefaction; determine also to what extent the outer table, the diplöe, and the inner table of the bone are involved.

By holding the skull cap up to the light a good idea of the relative thickness of its various parts can be obtained, and the presence of atrophic, carious, or necrotic patches will become evident. The Pacchionian depressions alongside the groove for the lateral sinus must not be mistaken for pathological changes in the bone.

Some idea of the age of the child can be obtained by an inspection of the fontanelles and sutures. The anterior fontanelle should be closed at eighteen months or thereabouts; if it is still open at twenty months evidence of rickets or of hydrocephalus will probably be found.

To determine age in the adult the sutures on the inner surface of the skull must be inspected, those on the outer aspect affording no sure criteria. For all practical purposes absence of obliteration of any parts of the internal sutures fixes the age at less than thirty ears. Over thirty there is always a fair amount of obliteration of the coronal and sagittal sutures on their inner aspects. The neighbourhood of the obelion (between the parietal foramina) and of the stephanion (intersection of the coronal suture and temporal crest) are the localities where signs of commencing fusion should be looked for. Over fifty usually, and over sixty always, all the entocranial sutures are obliterated.

Generalised Thickening of the Skull with condensation of the diplöe and sclerosis may indicate diffuse chronic osteitis and periostitis of syphilitic origin, either acquired or congenital. A moderate degree of general sclerosis is also met with in aged persons. In osteitis deformans the bone is greatly thickened but is porous; in the early stages of this disease the bone is soft, but by the time the skull is available for examination a certain amount of sclerosis has usually occurred. A general thickening of the skull cap may also occur in cases of extreme

rickets, but here the newly formed bone is not sclerosed. Owing to deficient calcification the convex surface of the skull cap in rickets is soft, resilient, and easily cut. A combination of rachitic softening with syphilitic sclerosis may coexist (syphilo-rachitis).

Localised Thickening of the Skull.—Frontal and parietal bosses may occur in children the subjects of congenital syphilis or rickets. The differences in the consistence of the bone in these diseases has just been indicated.

Localised thickenings or nodes may also be found in the adult, and are the result of gummatous affections, rarely of periostitis due to causes other than syphilis.

Generalised Attenuation of the Skull occurs in hydrocephalus.

Localised Attenuation of the Skull.—Cranio-tabes, which occurs in infants, is a patchy atrophy of the parietal bones, sometimes also of the occipitals or squamous portions of the temporals. The atrophic patches are rounded or oval, and usually lie in the posterior parts of the parietal bones. They vary in diameter up to an inch or more, and are easily detected on holding the bone up to the light. Their consistence is that of stiff parchment. Cranio-tabes occurs in congenital syphilis and also in rickets.

Localised patches of attenuation, due to caries or necrosis, may occur in the adult in consequence of syphilis, usually acquired but sometimes congenital.

The bone lying over a cerebral tumour is sometimes thinned. The depressions which lodge the Pacchionian bodies have already been referred to.

Malignant Tumours of the Skull are uncommon. Primary sarcomata may occur. Secondary tumours are sarcomatous or carcinomatous; the microscope will reveal their nature. Secondary tumours, which repeat the tissue of the thyroid gland, may arise in connexion with certain thyroid growths.

Adrenal sarcomata in young children may give rise to secondary growths in the soft tissue of the orbit and skull.

EXAMINATION OF THE DURA MATER

The outer surface of the dura mater should be cleansed with a damp sponge and inspected. This surface is naturally rough and dull, having been detached from the bones, of which it is the 'internal periosteum.'

In the young, while the bones are still growing, the dura is always firmly adherent to them, and a certain degree of adhesion is also common in aged subjects. Firm adhesion in the young adult is always pathological.

General Adhesion may be the result of syphilis or of chronic alcoholism. Localised adhesions may be due to fractures, or to caries, necrosis, and other inflammatory processes.

If **acutely inflamed** the outer surface of the dura mater becomes red and œdematous.

Localised Inflammation of some standing gives rise to circumscribed masses of granulation tissue. Inflammation of the outer surface of the membrane is almost invariably due to injury or adjacent disease.

Extra-dural Suppuration, between the membrane and the bone, may be due to perforating wounds or fractures. Erysipelas may give rise to it, probably through the agency of the diploic veins. At the base of the skull extra-dural suppuration may extend from the petrous bone, the accessory air sinuses or the nose. When suppuration takes place the dura becomes discoloured, assuming a greyish-yellow appearance.

Hæmorrhages between the Dura Mater and the Bone are usually due to injury. They may be extensive when the middle meningeal artery or the outer wall of a large blood sinus has been torn in consequence of fracture of the skull.

Fibro-sarcomata and Dermoid Tumours are sometimes found on the outer surface of the dura mater. The superjacent bone may be thinned or perforated.

EXAMINATION OF THE SUPERIOR LONGITUDINAL SINUS

Before reflecting the dura mater, slit up the superior longitudinal sinus in its whole length. In its fore part the sinus is narrow, but it is easily found behind, where it is broad and lies rather to the right of the mid-line owing to the greater size of the left hemisphere of the brain.

The blood in the sinus is usually dark and liquid, at most an attenuated loose clot is found. Mere quantity of blood has no pathological significance apart from that which attaches to engorgement of the venous system generally. The amount in the sinus is larger if the jugular veins or great veins of the thorax have not been incised during the earlier stages of the post-mortem examination. Before deciding that thrombosis is absent the parasinoidal recesses and superior meningeal veins should be examined.

Firm, partly decolorised ante-mortem Clots, adherent to the sinus walls, may occur in infants, chlorotic girls, or old people. These 'marantic thrombi' are supposed to originate as the outcome of extreme debility.

Puriform Thrombi may be found in infective conditions of the sinus walls and of the parts adjacent.

REFLEXION OF THE DURA MATER AND EXAMINATION OF THE SUB-DURAL SPACE

Notice whether the outlines of the subjacent convolutions and sulci can be recognised indistinctly through the dura before it is reflected. If they are apparent the dura is certainly not thickened. The membrane should be picked up over one frontal lobe with forceps and incised, the incision is then continued backwards along the cut edge of the skull with knife or blunt-pointed scissors almost as far as the occipital protuberance. The procedure is repeated on

the other side. Great care is necessary to avoid injury to the subjacent leptomeninges and convolutions. Next cut across the falx cerebri, just above the crista galli, with knife or scissors introduced between the front parts of the cerebral hemispheres.

Reflect the dura towards the occiput. The large veins of the vertex will need division where they enter the superior longitudinal sinus. Adhesion of the Pacchionian bodies can be overcome by gentle traction, whereby the bodies are dragged out from the invaginations which lodge them.

Inspect the inner surface of the reflected membrane; if healthy it is smooth and polished. If inflamed it will be dull and vascular, whilst suppuration gives rise to yellowish discoloration.

Fibrous Thickening of the Dura Mater, general or localised, may occur as a result of syphilis, and the same disease may give rise to localised bony deposits in the dura or between the folds of the falx cerebri or tentorium cerebelli. These deposits originate in gummatous infiltrations.

Purulent Effusions in the Subdural Space are associated with acute inflammation of the inner surface of the dura mater and subjacent pia-arachnoid. They are quite rare. In some cases the space is obliterated, the dura and arachnoid being glued together by fibrinous exudation.

Hæmorrhage into the Subdural Space is commonly the result of injury. It is rather exceptional for intracerebral hæmorrhages to rupture into this space, and the bleeding from a ruptured aneurysm at the base of the brain is primarily into the space between pia and arachnoid.

Hæmatoma of the Dura Mater.—A membranous and highly vascular adventitious tissue is sometimes found between the inner aspect of the dura and the outer surface of the arachnoid. Blood cysts may lie between layers of this tissue. The condition is known as hæmorrhagic pachymeningitis, or hæmatoma of the dura mater. Whether it is primarily hæmorrhagic or primarily inflammatory is

open to question. It may be the result of head injuries or various hæmorrhagic states, and is closely associated with chronic insanity and chronic alcoholism.

REMOVAL OF THE BRAIN

After the dura mater has been reflected, and before the brain is disturbed, a preliminary inspection of the convexity should be made and the following points observed : (1) The appearance of the arachnoid membrane, whether healthy and translucent or inflamed and opaque ; (2) The amount of fluid beneath the arachnoid, in the sulci between the convolutions ; (3) The degree of injection of the arterioles of the pia mater which clothes the convolutions ; (4) The appearance of the convolutions, whether wasted and separated or flattened and compressed ; (5) The condition of the veins of the convexity ; these may be gorged with fluid blood, contain loose and gelatinous clots, or be occluded by firmly adherent and partly decolorised ante-mortem thrombi.

Proceed to remove the Brain :—

Inserting the fingers of the left hand between the frontal poles and the dura mater raise the hemispheres and the olfactory lobes. Then divide, as close to the bone as possible, the carotid arteries, the stalk of the pituitary body, and the various cranial nerves as they come into view. Expose the lateral margins of the tentorium cerebelli by lifting the corresponding temporo-sphenoidal lobe towards the mid-line and divide the membrane along its attachments to the bones. Carefully avoid injuring the subjacent cerebellum and lateral sinuses. The fifth nerves will probably be severed at the same time as the tentorium. The cranial nerves of the posterior fossa should now be recognised and cut across. Tilt the brain backwards into the palm of the left hand to support it, but avoid undue traction on the mesencephalon. Pass the blade of a scalpel into the front part of the foramen magnum and divide the vertebral arteries and the spinal cord as low in the spinal canal as

possible. The cerebellum and medulla can then be hooked out from the posterior fossa by the fingers of the right hand, and the extraction of the brain thus completed.

Weigh the Brain. This should be done both before and after its dissection.

The **Weight of the Brain** in the adult male varies between 46 ozs. and 53 ozs. (1,301–1,502 grms.). In the adult female the weight varies between 40 ozs. and 47 ozs. (1,133–1,332 grms.).

The average weights in infancy and childhood are (Holt) :

At three months 21 ozs. (602 grms.). At six months, 25½ ozs. (712 grms.). At twelve months, 32½ ozs. (916 grms.). At two years, 35 ozs. (990 grms.). For the weights at different ages and for the relative weights of different parts of the brain the tables given in Quain's ' Anatomy ' should be consulted.

Examine the inner aspect of the Base of the Skull.

Pass in review the nerves, arteries, venous sinuses, dura mater, and pituitary body.

Inspect the severed ends of the cranial nerves ; small tumours are sometimes present on these nerve trunks. Attenuation with grey discoloration of the nerves indicates the presence of grey atrophy, such as occurs in disseminated sclerosis or syphilis. The Gasserian ganglion should be exposed by slitting up the dura mater along the trunk of the fifth nerve. In suppurative meningitis the ganglion may be surrounded by pus which lies in the cavum Meckelii, a condition which should not be mistaken for suppuration in the cavernous sinus.

Pus may sometimes be seen around the seventh and eighth nerves in the internal auditory meatus, infection having spread either from the internal ear inwards or from the cerebral meninges outwards.

Examine each Internal Carotid Artery and its ophthalmic branch at the base of the skull. If adherent clot is present the vessel must be exposed and examined in its course through the carotid canal, by chipping away the bony wall,

and also in the neck in order to determine the extent of the clot and condition of the interior of the vessel (arteritis).

The Middle Meningeal Artery, which lies on the outer aspect of the dura mater, should be identified. Its trunk and both branches should be inspected.

Complete the Examination of the Great Venous Sinuses.

The Superior Longitudinal Sinus has already been opened and inspected.

Lay open the Cavernous Sinuses at the sides of the sella turcica. The interior of these channels is spongelike, the blood circulating in the interstices. Thrombosis is uncommon, when present it may be due to disease of the nasal cavity, naso-pharynx, face or jaws. Clotting may also extend forwards from the other large venous sinuses.

Open the Petrosal Sinuses. The upper runs along the ridge of the petrous bone and connects the cavernous sinus with the lateral sinus ; the lower runs more directly between the cavernous sinus and the jugular bulb in the jugular fossa.

The Lateral Sinus should be laid open in its whole extent. Thrombosis of the sinus is nearly always secondary to disease of the middle or internal ear. Extra-dural abscess, cerebellar abscess or temporo-sphenoidal abscess, may coexist.

Extra-dural abscesses in the groove for the lateral sinus are exposed by stripping up the dura mater of the posterior fossa.

The Straight Sinus may be found in the mid-line, where it runs from before backwards at the junction of the falx cerebri with the tentorium.

Non-adherent clots, red and gelatinous, or pale, soft and attenuated, are of no pathological importance.

Adherent, granular, or puriform clots are of ante-mortem origin and pathological. If thrombosis is present in a sinus, determine whether it is traumatic (secondary to fracture, etc.) ; inflammatory (secondary to disease elsewhere, as already indicated) ; or marantic (such as occurs in debilitated conditions at the two extremes of life).

Inspect the Dura Mater of the Base of the Skull.

Localised yellowish discolorations may indicate disease of the subjacent petrous bone or of the walls of the accessory nasal air sinuses.

The position of a basic fracture may be indicated by signs of subdural hæmorrhage or basic inflammation, but these are not necessary concomitants.

EXAMINATION OF THE BONES AT THE BASE OF THE SKULL

This is carried out by stripping off the dura mater with a rugine, taking care not to abraid the hands against the sawn surface of the bones.

In association with fractures certain sutures may be 'started.' The sutures between the orbital plates of the frontal bone and the ethmoid or sphenoid bones are naturally irregular, and should not be mistaken for fractures. Pay particular attention to the relations of basic fractures to the tympanic cavity, the canal of the facial nerve, the pituitary fossa and clinoid processes, the cribriform plate of the ethmoid (roof of naso-pharynx and nose), and to the foramina at the base of the skull.

Most cranial fractures radiate from the point struck.

EXAMINATION OF THE PITUITARY BODY

This body should always be inspected; it is easily removed by cutting through the base of the dorsum sellæ with a chisel. If much enlarged the bony boundaries of the fossa may be undergoing absorption. The relations of pituitary tumours to the base of the brain, to the cavernous sinuses, and to the adjacent nerves, particularly the optic nerves with their commissures and tracts, should be ascertained. These tumours may also implicate the tip of the petrous bone, the Gasserian ganglion, and the roof of the pharynx. Aneurysm of the internal carotid artery may encroach on the pituitary fossa and simulate a tumour.

The **Weight of the Pituitary Body** is 77 grains (5 grms.) in the adult.

Acromegaly. — This condition may be associated with benign or malignant enlargement of the pituitary body. Of malignant tumours sarcomata are most common, but gliomata are also described. Benign enlargement may be a mere hypertrophy (' pituitary goitre ') or accompanied by adenomatous and cystic changes. Gummata are rare.

Look for symmetrical enlargement of the hands and feet, mainly due to thickening of the skin and subcutaneous tissues; hypertrophy of the nose, lips, tongue and gums; enlargement of the malar prominences and alveolar processes; great overgrowth of the lower jaw; increase in thickness and weight of skull; more or less symmetrical enlargement of certain bones of the extremities and of the clavicles; cervico-dorsal kyphosis with compensating lumbar curve; cardiac hypertrophy and thickened arteries; enlarged thyroid in many cases and sometimes a persistent thymus. Occasionally enlargement of one or more of the abdominal viscera occurs.

EXAMINATION OF THE ARTERIAL TRUNKS AND MENINGES OF THE BRAIN

The surface of the brain should be examined thoroughly before dissection is proceeded with. Attention must first be directed to the structures at the base. Notice the condition of the leptomeninges which form the walls of the great basal cisterns. In health they are glistening and translucent. Distension with clear fluid indicates œdema, whilst opacity and thickening are due to chronic meningitis. In suppurative meningitis purulent infiltration of the membranes occurs. Sometimes the latter are infiltrated with blood or blood-stained fluid, the origin of which should be sought. They may also be the sites of miliary tuberculosis.

Inspect in sequence the exposed aspects of the medulla, pons, crura cerebri, temporal and frontal lobes, the olfactory

bulbs and tracts, the interpeduncular space, the optic chiasma, the commencements of the Sylvian fissures and the perforated spots. Examine the under surface of the cerebellum, and, by gently raising the medulla from it, determine the condition of the inferior vermis and also of the fourth ventricle and its roof. Then raise the cerebellum itself from the brain to inspect the upper aspects of its lobes and the superior vermis. Pay particular attention to the condition of the meninges over the fore parts of these structures, since they are apt to be implicated by extension in various forms of basal meningitis.

Whilst inspecting the under surface of the brain in this way look out for any asymmetry, localised bulging, depression, area of discoloration or softening. Depressions may be due to pressure exerted by tumours arising from the base of the cranial cavity or by blood effused between the brain and the skull.

Next pass in review the various cranial nerves.

The arteries at the base of the brain should be examined in their turn. First the vertebral trunks, which are often unequal in size, then in succession, the basilar, the vessels forming the Circle of Willis, and the stems of the internal carotids. To expose the middle cerebral arteries, lift up the tip of each temporo-sphenoidal lobe and slit up with the scissors the meninges which bridge over the Sylvian fissures. Separate the lips of each fissure until the Island of Reil and the terminal branches of the artery on its surface are in full view.

Trace the anterior cerebral arteries forwards round the anterior extremity of the corpus callosum on to its upper surface.

The posterior cerebral trunks are to be traced backwards around the crura cerebri. Irregularities in the origins and distributions of the various vessels should not be overlooked, as they may afford an explanation of abnormal symptoms produced by the disease of a particular artery.

After death the arterial trunks should appear flattened

and empty. Localised swellings and opacities or a failure to collapse are indications of disease.

The examination of the arteries is completed by slitting them up with fine scissors to detect the presence in them of emboli, thrombi, or disease of the intima.

The presence of ante-mortem clots in the internal carotid, where that lies in the cavernous sinus, petrous bone, and neck, should not be overlooked. Some cases of hemiplegia without obtrusive brain lesion, can be traced to occlusion of the carotid trunk in one of the situations named. Aneurysms of the carotid artery in the cavernous sinus may bulge towards the sella turcica and be overlooked, or mistaken for enlargements of the pituitary body.

The brain should now be turned over and the convexity of the hemispheres again inspected. In health the pia-arachnoid, here as elsewhere, presents a glistening, translucent appearance; the cerebral veins contain fluid blood unless this has already drained away, the arterioles of the pia are inconspicuous, the sulci between the convolutions are narrow, and the convolutional ridges are slightly rounded off.

LESIONS OF THE SUPERFICIAL BLOOD VESSELS OF THE BRAIN

Arterio-sclerosis of the basal arteries is indicated by uniform thickening of the vessels, failure of their walls to collapse after death, and a tendency to gape when cut across.

Atheroma (Endarteritis Deformans) is a patchy disease occurring in the form of opaque yellow thickenings, which are sometimes calcified. The calibre of the vessels may be considerably increased or, in some cases, diminished. Thrombosis may occur. Atheroma of the basal arteries is often a senile change.

Syphilitic Arteritis gives rise to localised nodular thickenings of the vessels, rendering their walls less translucent and diminishing their lumina. After anti-

syphilitic treatment the vessel walls are less nodular and more opaque; the disease is then not to be distinguished from atheroma save by taking into consideration the age and the history, or evidence of previous syphilitic infection.

Small Saccular Aneurysms of the large basal arteries are usually, if not invariably, of syphilitic origin. They tend to rupture into the leptomeninges or into that part of the brain to which they have become adherent. Localised yellowish discolorations in the neighbourhood of such aneurysms, if present, indicate the occurrence of leakage at a former period. Ascertain the relations of aneurysms to the cranial nerves at the base of the brain.

Emboli in Cerebral Arteries may be derived from the left cavities of the heart (particularly in mitral stenosis), the aorta or the large arteries of the neck. Rarely they are derived from the pulmonary veins. An embolus may be distinguished by its lighter colour and greater firmness from the secondary red clot which forms around it. The artery distal to the embolus may be found contracted.

Thrombosis of Cerebral Arteries may be due to atheroma, to syphilitic arteritis, to implication of the arterial wall by adjacent disease, or may occur in vessels apparently healthy, when it is attributed to changes in the coagulability of the blood. Spontaneous arterial thrombosis may occur after childbirth, in chlorosis, or during the course of various infective and wasting disorders.

When thrombosis is present the older portion of the clot will probably be adherent to the vessel wall, pale, and showing signs of lamination or softening.

Thrombosis of the Veins of the Convexity is uncommon. It may be combined with thrombosis of the sinuses or occur independently. Two forms are recognised, infective and simple. The former can be traced to an infective focus such as purulent otitis, suppuration on the face or in the orbit, etc. The latter may supervene in debilitated conditions, consequent upon severe and exhausting illnesses; it also occurs occasionally in severe chlorosis or after childbirth. Thrombosis of the veins causes congestion of the cerebral capillaries and petechial

hæmorrhages into the brain substance. Localised œdema may also result.

Intradural Effusions of Blood may lie on the surface or in the substance of the pia-arachnoid. If free on the surface the blood can be removed by light sponging, but subarachnoid effusions cannot be dislodged in this way. Bloody effusion may be due to bruising and laceration of the brain substance, either by direct injury or by *contre-coup*. It may also be caused by rupture of an aneurysm of one of the large basal arteries, or have escaped from the interior of the ventricles. (See also p. 227.)

LESIONS OF THE CEREBRAL MENINGES

Hyperæmia of the Pia Mater may be of little significance or indicate incipient meningitis. The small, brush-like arterioles of the pia, even in the normal brain, become very evident when the hemispheres have been exposed for some time, owing to oxidation.

Anæmia of the Pia Mater, with stickiness and flattening of the convolutions, point to ventricular distension, usually originating in meningitis.

Œdematous Infiltration of the Leptomeninges is easily recognised by the watery appearance it produces. The condition appears as a primary change in uræmia, but it may also be secondary to slow cardiac failure in valvular disease of the heart, or compensatory to wasting of the subjacent cerebral convolutions.

Thickening of the Leptomeninges, giving rise to spots and streaks of white opacity, is very common in mental disease, particularly in general paralysis of the insane and senile insanity. The appearance is most marked over the convexity near the large veins. Similar patchy opacities may be met with in chronic renal disease, chronic alcoholism, cases of long continued back pressure due to heart disease, or as sequels to chronic meningitis in children. The change is usually looked upon as a chronic inflammation.

Tuberculous Meningitis is distinguished by the presence of miliary tubercles in the pia mater at the base of the brain. The tubercles are chiefly aggregated in the

region of the chiasma and within the lips of the Sylvian fissures, where they may be so numerous as to give the membranes a granular appearance. They may also be seen on the under surfaces of the temporo-occipital lobes and the adjacent surface of the cerebellum. The basal cisterns of the arachnoid may present a milky opacity or be infiltrated with a greenish gelatinous exudate. The ventricles are distended and the convolutions flattened. Turbid serum often escapes from the interior during removal of the brain. The walls of the ventricular cavities may be in a condition of creamy softening, the change being particularly evident around the posterior and descending cornua. Tubercles may sometimes be recognised in the choroid plexuses. Tubercles are also conspicuous on the convexity in exceptional cases, and, by confluence, may form caseous masses. The inflammation of the meninges at the base may, in some cases, strangle small perforating arteries and lead to localised softening in the basal ganglia. Tubercles may also be present on the spinal membranes.

The Pacchionian bodies, which lie in contact with the superior longitudinal sinus and with the parasinoidal blood recesses, appear as clusters of dull white projections alongside the great longitudinal fissure; these bodies should not be mistaken for tubercles.

Tuberculous meningitis is, as a rule, part of a widespread miliary tuberculosis. Search should be made for the focus of infection, which may be found in a mediastinal or mesenteric gland, in the lungs, bones, joints, generative organs, or elsewhere.

Cerebro-spinal Meningitis.—In this affection the leptomeninges are infiltrated with a purulent exudate which is widespread, involving the base of the brain extensively, and spreading also on to the convexity and along the membranes of the spinal cord. The ventricles are distended with purulent exudation and the convolutions of the vertex become flattened and sticky. In some instances petechiæ may be recognised in the skin and meninges. The parenchymatous organs of the body show cloudy swelling, and the lymphoid structures of the intestine are prominent.

In cerebro-spinal meningitis of long duration the post-mortem appearances are those of hydrocephalus. In cases of this sort the presence of some obstruction to the drainage of the ventricular system should be sought, such as sealing of the foramina of the fourth ventricle, glueing of the cerebellum to the surface of the medulla, or adhesion of the dura mater to the arachnoid in the cervical region of the cord. (The proper method of examination is described on p. 221.)

Post-basic Meningitis of Children.—The appearances in the earlier stages are those of cerebro-spinal meningitis. In the later stages hydrocephalus may be present.

Suppurative Meningitis.—This, which may appear as a local or a diffused leptomeningitis, can only be distinguished from epidemic cerebro-spinal meningitis by its bacteriology, or by the presence of an obvious focus of infection. At the base the large subarachnoid cisterns show purulent infiltration; on the surface are streaks of purulent matter, which may at first be limited to the tracks of the superficial veins and the sulci between the convolutions, but which later become widespread. Superficial encephalitis and infection of the ventricles may also occur.

Local causes should always be sought when a purulent meningitis is discovered. Examine for fractures of vault or base, purulent infection of the ear, nose, air sinuses or orbit, infective thrombosis of the great venous sinuses, or extension of infection from pyæmic brain abscesses. The condition may also be associated with lobar pneumonia or with pyæmia from remote lesions, such as osteomyelitis.

Syphilitic Meningitis.—The early stages, in which the leptomeninges, usually at the base near the optic chiasma, but sometimes of the convexity, are infiltrated with a viscid, gummy material which resembles thick collodion or has a pinkish tinge, are rarely seen post-mortem. Gummatous and fibroid transformation of the infiltration occurs. Firm adhesions may form between the dura, the leptomeninges, and the surface of the brain, matting the whole together. Dry, yellowish, gummatous masses simulating tubercle may lie in the adhesions. The process is really a gummatous meningo-encephalitis. The arteries

at the base of the brain usually present opaque inflammatory thickenings (syphilitic endarteritis).

General Paralysis of the Insane.—In this disease the dura mater is often thickened and adherent to the skull, particularly along the sagittal suture. There is an excess of fluid in the subdural space and œdema of the pia-arachnoid. The leptomeninges present a more or less diffused or a patchy thickening and opacity, particularly in the prefrontal regions. Firm adhesion of the pia to the subjacent convolutions, so that it cannot be raised without causing superficial laceration of the cortex, is very characteristic. The convolutions are wasted, especially those of the frontal regions where the membranes are most adherent. The ventricles are dilated and their ependyma presents a granular frosted aspect which is particularly well marked in the floor of the fourth ventricle. The brain is wasted, being several ounces under its normal weight (see p. 229). The optic nerves are often atrophied. The spinal cord may present the lesions of tabes or show degeneration in the lateral columns. The skull cap is often thickened and the spongy diploe condensed. Syphilitic degenerative lesions may be found elsewhere in the body.

Atrophy of the Convolutions gives the appearance of a walnut-kernel to the surface of the brain. The wasting may be local or general. A certain degree of general atrophy may occur as a senile change. Its occurrence in general paralysis has been mentioned. In the young brain atrophy may be the result of ante-natal disease, of injuries inflicted during birth, or of cerebral lesions occurring during the early years of life. In all cases of localised atrophy attention should be directed to the condition of the main arterial trunks, since occlusion of one of these might be a causal factor.

Localised Bulgings of the Convolutions may indicate the presence within the brain of tumours, abscesses, hæmorrhages or cysts.

Localised Depressions may be caused by pressure outside the brain or indicate a defect in the brain substance, such as occurs in porencephaly.

EXAMINATION OF THE INTERIOR OF THE BRAIN

In cases of suspected hæmorrhage, abscess, or tumour, it is often an advantage to harden the brain before it is dissected. For ordinary purposes this can be effected in ten days by immersion in 10 per cent. formalin solution. Where special microscopical examinations are to be carried out the use of other hardening fluids may be preferable. As a general rule, however, the brain is dissected as soon as it is removed from the body, but after its weight has been ascertained.

Method of dissection :

Examine the ventricular system first. If cultures have to be taken from the cavities the media must be inoculated, with proper precautions, before the dissection is proceeded with.

Then place the brain, base downwards, on the table, draw the hemispheres apart to expose the corpus callosum, and by further gentle outward traction open up the space between this structure and the callosal convolution under which it passes. Pass the blade of a scalpel into the narrow space exposed and divide the corpus callosum longitudinally through its whole thickness, but without injury to the subjacent structures. The incision will expose the body of the *lateral ventricle* and should be continued both backwards and forwards until the anterior and posterior cornua are fully opened up. Repeat the dissection in the opposite hemisphere.

Having exposed both lateral ventricles in this way, pass the blade of the scalpel transversely through the foramen of Monro and divide the corpus callosum by cutting forwards and upwards.

The corpus callosum and the subjacent fornix can now be turned backwards, exposing the fold of pia mater which forms the velum interpositum and contains the veins of

Galen and choroid plexuses. Turn back the velum, carefully liberating the pineal body, which, being entangled, is apt to be torn from its stalk.

The *third ventricle* and the corpora quadrigemina will now come into view. Examine the interior of this ventricle and then divide the posterior attachments of the reflected corpus callosum and fornix on the right side, turning these structures over to the left. The velum interpositum should be turned aside at the same time.

Split the superior vermis of the cerebellum longitudinally, separate the two halves, and thus fully expose the *fourth ventricle.*

The iter, connecting the third and fourth ventricles, can be opened by a longitudinal incision between the lateral pairs of corpora quadrigemina.

When examining the cerebral ventricles note their size; the character of any fluid contents, such as serum, serum with suspended purulent flocculi, pus or blood; also the appearance of the ependymal lining, whether transparent, dull from recent inflammation, or thickened and discoloured from chronic inflammatory changes.

The condition of the fringes which contain the choroid plexuses of the various cavities should be ascertained. They may show signs of inflammation or may be the sites of tumours or of deposits of tubercle. In doubtful cases, before assuming that minute choroidal granulations are tubercles, a microscopical examination is necessary. The presence of small, translucent, berry-like cysts in the plexuses is of no pathological importance. Small gritty tumours are usually psammomata (peritheliomata). Aneurysms of the choroidal arteries which supply the plexuses are occasional sources of hæmorrhage.

The Veins of Galen, which receive the blood from the plexuses and walls of the ventricles, conveying it to the straight sinus, should be examined for evidence of obstruction or thrombosis. They may be compressed by sub-tentorial tumours or by adhesive inflammation of

the transverse fissure; the latter lies below the posterior extremity of the corpus callosum.

An undue projection of the basal ganglia into the lateral ventricles may indicate the presence of tumours or hæmorrhages. Softening or shrinkage of the ganglia may be the result of occlusion of the perforating arteries, and call for a careful examination of the vessels in the vicinity of the perforated spots at the base of the brain.

Acute Internal Hydrocephalus is a complication of meningitis, generally of the tuberculous, posterior-basic or cerebro-spinal varieties.

Chronic Internal Hydrocephalus may be ante-natal or post-natal in origin. The ventricles are dilated and the cerebral substance correspondingly stretched and thinned.

In all cases search should be made for some obstruction to the exit of the ventricular contents. The foramina in the roof of the fourth ventricle may be found sealed by adhesions; subtentorial tumours or abscesses may exercise pressure on the iter or obstruct the veins of Galen; the downward drainage of the ventricles may be interfered with by firm adhesions between the membranes which encase the upper part of the spinal cord; obstruction may be caused by the cerebellum being forced down into the foramen magnum like a plug in consequence of increased pressure above.

In some cases of hydrocephalus no obstruction is evident, but the presence of discoloration with granular thickening of the ependyma of the ventricles, or of similar changes on the surfaces of the choroid plexuses, indicates the existence of **Chronic Ventricular Meningitis.**

Localised Dilatations of one or more of the ventricles may be due to obstruction situated at the foramen of Monro or in the iter.

Dilatation of the ventricles may also be present, and the surrounding brain substance sclerosed, in certain cases of birth palsy.

Porencephaly. — Defects in the substance of the

hemispheres may allow the ventricular cavities to open on the surface of the brain. Such defects are attributed to local softening and absorption of the brain substance. Occlusion of cerebral vessels is one factor which may produce this result. In some cases syphilis may be suspected as a cause.

Hæmorrhage into the Ventricles.—An effusion of blood into the ventricles is often secondary to the rupture of a cerebral hæmorrhage which has primarily involved the basal ganglia.

Primary ventricular hæmorrhage is rare, but may be derived from the arteries or veins of the choroid plexuses or of the ventricular walls.

It is usually stated that ventricular hæmorrhage may be induced by hanging or strangling, but its occurrence in death from these causes is exceptional. It may occur in certain blood states characterised by a tendency to spontaneous bleeding. After protracted and difficult labours ventricular hæmorrhage may be found in the infant, and be associated with meningeal hæmorrhage.

Concussion is also alleged as an occasional cause of hæmorrhage into the ventricles.

A Collection of Pus is sometimes found in the ventricles. This may be due to suppurative internal meningitis, the result of a general meningeal infection. But the pus may have escaped from a ruptured cerebral abscess. An abscess in the temporo-sphenoidal lobe may burst into the descending cornu of the corresponding lateral ventricle.

Remove the Cerebellum, together with the Pons and Medulla, by cutting across the crura cerebri. This is easily effected by turning the base of the brain uppermost and lifting the parts to be removed, so that slight traction is exercised on the crura whilst they are divided. Inspect the cut surfaces and place aside the portions removed.

It is now possible to complete the examination of the descending cornu of each lateral ventricle by completely opening up the lips of the dentate fissures. The cornua are thus exposed from their inner aspects.

The degree of adhesion between the hemispheres and the leptomeninges can be tested, if necessary, by stripping off the latter. The separation should be commenced on the inner aspect, immediately above the corpus callosum, the pia-arachnoid being picked up with forceps in this situation and gradually raised from the surface, first of the inner and then of the outer aspect of each hemisphere. Great care should be exercised, the convolutions being pressed away from the meninges rather than the meninges torn from the convolutions.

Where a microscopical examination of the cortex is to be carried out it is not advisable to remove the leptomeninges at all.

Next examine the inner parts of the hemispheres, the basal ganglia, and the internal capsules. For this purpose let the brain rest on its base, the hemispheres will then fall apart by their own weight, exposing their inner faces. Commencing on these exposed faces, slice each hemisphere longitudinally. Two incisions are requisite in each hemisphere, the inner (lower) being just above the level of the corpus callosum. The incisions should not quite traverse the whole thickness of the brain, in order that the different slices may still remain attached to each other. By these incisions the centrum ovale minus and the centrum ovale majus are exposed and examined.

The parts of the brain as yet untouched lie between the two inner incisions, and comprise portions of the frontal, occipital, and temporo-sphenoidal lobes, together with the basal ganglia. The condition of these parts must be examined by a series of parallel transverse incisions which should not be carried deeply enough to effect complete separation. Scrutinise the surfaces of the various slices as they come into view. Identify the caudate and lenticular nuclei of the corpus striatum, the optic thalamus, and the dense white band of the internal capsule between the thalamus and the nuclei first mentioned.

If the dissection has been carried out properly no part of

the hemisphere is completely severed from the rest, and the whole can be reconstructed for the purpose of localising any lesions which may have been discovered.

The temporo-sphenoidal lobes should not be overlooked. In cases of temporo-sphenoidal abscess these lobes should be sliced transversely from their under surfaces before proceeding to incise the other parts of the brain.

During the process of removal and dissection of the brain a general idea of its consistence will have been gained. In children the brain is softer and less opaque than in adults, whilst in infants it is still more translucent and almost gelatinous. In alcoholics and those who have suffered from long continued venous back-pressure due to cardiac disease, the brain substance is appreciably firmer than normal.

The more common lesions of the cerebral hemispheres are hæmorrhage, softening, tumour, abscess, and sclerosis.

Cerebral Hæmorrhage is usually derived from one of the striate group of perforating arteries, even large extravasations of blood in the centrum ovale will often be found to have extended from the corpus striatum.

The exact position of the extravasation with regard to the basal ganglia, the internal capsule, the ventricular cavities, and the adjacent cortical areas, should be determined. The brain matter around a hæmorrhagic focus is softened and may exhibit a yellowish discoloration.

By washing away the clot with a gentle stream of water it may be possible to pick out from the débris of brain substance portions of vessels bearing minute aneurysms.

The possibility of the occurrence of hæmorrhage into vascular brain tumours or into areas of acute embolic softening should not be overlooked.

Hæmorrhage in unusual situations near the surface of the brain should lead to suspicion of rupture of a large aneurysm of one of the basal arteries. These aneurysms, if adherent to the brain substance, are apt to be overlooked.

The residua of old hæmorrhages may be discovered accidentally; they occur as apoplectic cysts or cicatrices with yellowish pigmentary deposits (hæmatoidin crystals) in

their neighbourhood. In more recent cases shrunken reddish-brown blood clot may be found.

Cerebral Softening is recognised by marked diminution in the consistence of the brain substance. It may be due to vascular occlusion, acute infective inflammation, or bruising. General softening of the brain may also be due to decomposition, and in such cases small gas-containing cavities may be apparent.

The naturally soft consistence of the infant's brain should not be mistaken for a pathological condition.

Areas of cerebral softening may be white, red, or yellow. The redness is due to hyperæmia and, later, is replaced by yellow staining.

Brain matter which has undergone white softening is of a creamy consistence; softening of this character is a common accompaniment of tuberculous meningitis, being particularly evident around the posterior cornua of the ventricles, and also in the fornix and corpus callosum.

Red softening is mostly seen in the cortex and basal ganglia; the softened area may be flecked with hæmorrhages.

Yellow softening is most conspicuous in inflammatory lesions. It is often seen around cerebral abscesses. As above mentioned, it is a sequel of red softening.

In case of cerebral softening, the large arterial trunks at the base of the brain, together with their perforating branches, should be examined for obstruction in the form of embolism; thrombosis; strangulation by inflammation of the surrounding membranes, such as sometimes occurs in tuberculous meningitis; or compression by tumours.

The territory of the middle cerebral artery is a common site of cortical softening.

Softening of the basal ganglia results from lesions of the middle or posterior cerebral arteries blocking the small vessels which enter at the perforated spots.

In head injuries, bruising and softening of the superficial parts of the brain may occur on the opposite side to that on which the injury was inflicted; the brain lesion is then attributed to *contre-coup*. (See also p. 252.)

If a brain is examined soon after the onset of embolism

or thrombosis, softening may not have had time to become evident in the ischæmic territory.

The red softening caused by infective embolism, such as may occur in ulcerative endocarditis, is often complicated by free hæmorrhage from the damaged vessel.

Cerebral Tumours.—The exact position, size, and relations of a brain tumour should be carefully noted. Any secondary results of its presence, such as hydrocephalus, meningitis, hæmorrhage, softening, or pressure, should not be overlooked. It may be possible to make out whether the tumour originates in the brain substance itself, in folds of the pia mater, or in the choroid plexuses and ependymal lining of the ventricles (internal meninges). Gliomata originate in the brain substance, but tumours springing from the internal meninges are, for the most part, sarcomata or endotheliomata. To ensure accuracy a microscopical examination should always be carried out.

Tuberculous Tumours are firm and circumscribed. They are uniformly opaque or cheesy on section, and mostly surrounded by a translucent grey rim. They may undergo softening or calcification. The membranes in contact with them are matted, and may contain miliary tubercles. A terminal tuberculous meningitis is often present. Tuberculous tumours may occur in the cerebellum, the mesencephalon, or the hemispheres. They are fairly common in childhood.

Gummata may occur within the brain substance or in the meninges as a gummatous meningo-encephalitis. Intracerebral gummata are hard, irregular in shape, but circumscribed. Caseous areas may be evident on section, but the presence of broad intersecting bands of grey, semi-translucent connective tissue affords a distinction from cerebral tubercle. After treatment gummata may appear shrunken and fibroid. The meninges are often adherent in their neighbourhood, and endarteritis of the cerebral vessels may coexist. The cerebral hemispheres and the pons are likely sites for gummata.

The appearances of gummatous meningitis have already been described on p. 238.

Gliomata are infiltrating tumours. Consequently they are ill-defined, and since they may closely resemble the brain matter in tint and consistence, increase in size may be the only criterion of their presence in the pons, basal ganglia, or convolutions. In some instances their tint is reddish rather than grey. Gliomata are prone to hæmorrhage and to cystic degeneration. In all doubtful cases of hæmorrhage or cyst formation the boundary wall of the lesion should be submitted to microscopical examination.

Sarcomatous Tumours are more common without than within the brain. They may spring from the membranes, bones, or brain substance, and are, as a rule, firm, sharply defined, and easily enucleated. They may be multiple. The colour of a sarcomatous tumour varies according to the amount of blood which it contains. There is a soft, diffuse, infiltrating variety.

Endotheliomata are soft tumours which are sometimes almost gelatinous. They are of a pale or reddish colour, and may arise from the choroid plexuses or ependymal lining of the ventricles.

Carcinomatous Tumours may be secondary to carcinomata elsewhere in the body, and then their nature is evident from collateral evidence. But they may also arise in connexion with the choroid plexuses of the ventricles, to which cavities they then tend to be confined. They may give rise to hydrocephalus or compress the adjacent brain substance.

Carcinomatous tumours are generally soft, rapidly growing, decidedly vascular and not sharply defined. They tend to destroy the brain substance as they advance.

Abscesses in the Cerebral Hemispheres may be solitary or multiple.

Multiple Abscesses are due to infective embolisms and are secondary to lesions in some other part of the body such as bronchiectasis, and empyema.

Solitary abscesses are, as a rule, secondary to infective processes in their neighbourhood consequent on disease or injury.

An abscess in the temporo-sphenoidal lobe is usually due

to ear disease. The track of infection through the roof of the tympanum or antrum is often shown by adhesion and discoloration of the brain membranes in this situation.

An abscess in the frontal lobes may sometimes be traced to infection from the nasal cavities, the frontal sinuses or ethmoidal air cells.

Abscesses may also be subjacent to diseased or injured bone or occur in bruised brain tissue.

The presence of a tough inflammatory capsule around an abscess indicates that the lesion is of some standing and not recent. Old abscesses may sometimes be shelled out without rupture.

The white matter around a recent abscess may show a diffuse red or yellow softening.

Cerebral abscesses may rupture on the surface of the brain or into the ventricles.

The descending cornu of the lateral ventricle is in proximity to abscesses of the temporo - sphenoidal lobe.

Sclerosis of the Brain.—Diffuse Sclerosis of the Brain is characterised by induration of the cerebral substance, which may feel as hard as plaster of Paris.

The whole or part of a hemisphere may present this abnormal hardness, and usually is also wasted. Ascertain whether the sclerotic atrophy corresponds to the area of distribution of one of the large cerebral arteries. A microscopical examination is necessary, since the sclerosis is sometimes due to a neoplastic condition.

The clinical accompaniments of diffuse sclerosis are idiocy and cerebral birth palsy. The brains of drunkards often show an unnatural degree of firmness, as also may those of patients dying from old-standing heart disease with back-pressure, but the brain substance in such cases is not nearly so hard as in actual sclerosis.

Disseminated sclerosis is characterised by the presence of small patches of a pinkish-grey colour and rather translucent appearance. These patches are sharply defined. If recent they are soft, but if old they may be very hard.

They may be found in the centrum ovule, the corpus callosum, the basal ganglia, the pons and other parts.

Certain of the cranial nerves may present grey atrophy and sclerotic patches may also be found in the spinal cord.

Tuberose Sclerosis is a form of patchy sclerosis met with in the weak-minded and often associated with a skin affection known as adenoma sebaceum.

EXAMINATION OF THE CEREBELLUM, PONS VAROLII, AND MEDULLA OBLONGATA

Again inspect the superficial aspects of the cerebellum, pons and medulla, also the cut surfaces of the crura cerebri.

The fourth ventricle has been exposed by longitudinal division of the superior vermis ; re-examine its floor.

Split each hemisphere of the cerebellum by an incision carried horizontally inwards from its convex border towards the superior peduncle where the latter appears between the lips of the great horizontal fissure. The incisions must fully expose the interior of each lateral lobe with its white core, dentate nucleus, and the origin of the superior peduncle. If necessary other incisions may be made parallel to the first. The incisions should allow full inspection of the interior of each hemisphere without detachment of the slices from each other.

The crura, pons and medulla are examined by a series of transverse incisions carried across their ventral aspects. If necessary the trunks of the basilar and vertebral arteries are first dissected away. As a rule three incisions through the pons and three or four through the medulla are sufficient.

The knife should be sharp, as clean sections are requisite. The cut surfaces should be irrigated under the tap and carefully examined for the presence of tracts of degeneration or other disease.

The common lesions of the Cerebellum are abscesses, tumours and cysts.

Abscesses occur in the lateral lobes. They are often solitary and are practically always due to ear disease.

The track of infection from the ear should be traced if possible, it is sometimes indicated by a stalk of adhesion of the membranes over the lateral sinus, the petrous bone, or the internal auditory meatus.

Old abscesses have tough capsules, recent abscesses may be surrounded by a zone of yellow softening. In cases where the cerebellum has been explored before death a narrow track of hæmorrhage may indicate the course of the exploring trocar.

Cerebellar Tumours may be tuberculous or gliomatous. The former are sometimes multiple, the latter may be the sites of cystic degeneration or of hæmorrhage.

Care should be taken to distinguish meningeal effusions of blood or pus between the surfaces of adjacent folia; and ventricular hæmorrhage or suppuration invading the lateral recesses of the fourth ventricle, from primary hæmorrhage or suppuration into the cerebellar substance.

Cerebellar Cysts may present no evidence of their mode of origin. They sometimes arise in new growths or may be the result of softening. Hence the wall should be submitted to microscopical examination and the cerebellar arteries scrutinised for evidence of occlusion. The distribution of the posterior inferior cerebellar artery, itself a branch of the vertebral, is such that occlusion may not only give rise to softening in the posterior part of the cerebellar hemisphere, but also to an area of necrosis in the lateral part of the medulla between the inferior olive and the restiform body.

The **Crura Cerebri** may be the sites of tuberculous nodules or be invaded by tumours growing from the base of the skull.

A cerebral hæmorrhage may plough its way downwards into the upper part of the crus, but primary hæmorrhage and softening are alike rare. The vascular supply is from the posterior cerebral artery. Abscess of the crus may be found in association with pyæmic abscesses in other parts of the brain.

The Corpora Quadrigemina may be the sites of tumour; the disease, however, is rarely confined to these structures,

but implicates also the thalmus, the pons, the superior cerebellar peduncles, or even the cerebellum.

The **Pons Varolii** is more likely to be the seat of vascular lesions than of tumours. Softening, which is practically always due to thrombosis of the basilar artery, is usually syphilitic in origin. Pontine hæmorrhage owns the same causes as cerebral hæmorrhage generally. In cases of severe head injury small foci of blood are sometimes found in the substance of the pons. These minute extravasations should not be mistaken for primary pontine hæmorrhages.

Tumours which occur in the substance of the pons are usually either gliomatous or tuberculous. The former may give rise to considerable enlargement constituting the so-called 'hypertrophy' of the pons.

The pons is also a favourite position for the sclerotic foci of disseminated sclerosis.

The **Medulla Oblongata** is rarely the site of lesions. Softening is due to thrombosis or embolism of the vertebral artery and its branches.

Pressure may be exercised on the medulla by tumours or abscesses of the cerebellum or by effusions of blood which have reached the fourth ventricle. Compression of the medulla may also arise from fracture or dislocation of the atlas or axis vertebra, the result of disease of the spinal column or of violence.

In acute or chronic bulbar palsy a microscopical examination is necessary to determine the presence of the nuclear changes, but attention should be directed to the presence of hyperæmia in the acute cases and to the size of the hypoglossal and accessory nerve rootlets in the chronic ones.

Concussion of the Brain.—Cerebral bruising, with punctiform hæmorrhages and softening, or cerebral lacerations are found in most cases of fatal 'concussion.' The injury may be on the opposite side to the point struck (*contre-coup*).

In all cases, and especially where no brain injury can be detected, the safe rule is to examine the cervical region of the spine for fracture or dislocation, and the cervical cord for bruising or hæmorrhage.

CHAPTER VI

EXAMINATION OF THE VERTEBRAL COLUMN AND THE SPINAL CORD

PRELIMINARY INSPECTION

In every case where disease of the spinal cord is suspected, and where a post-mortem examination is to be made, it is well that the body should be turned face downwards immediately after death and retained in this position until the cord has been removed. Some formalin solution should also be injected into the subdural space as a precaution against post-mortem changes.

It is cleaner and more convenient to carry out the examination of the spinal canal before the thoracic and abdominal cavities are opened; first, because the entire thorax affords a better support to the spinal column, and, secondly, because fluids cannot escape from the chest and belly to soil the surroundings.

The body should lie face downwards, the head hanging over the edge of the table. A receptacle containing sawdust should be placed on the floor in a position to receive any fluids which may run from the nose and mouth.

First examine the spinal column by sight and touch for injuries, deformities or congenital defects.

Injuries, such as fractures and dislocations, may not be evident until the soft parts have been reflected. Angular curvature, due to tuberculous disease of the vertebræ, may be conspicuous, and in old-standing cases a bursa is often present over the prominence. Abscesses may complicate tuberculous disease of the bones.

Scoliosis or lateral curvature is associated with rotation of the bodies of some of the vertebræ.

Lateral curvature associated with pes cavus should suggest the presence of spinal cord disease (Friedreich's ataxia or Syringomyelia).

Localised deformities of the spinal column may be produced by the destructive action of new growths, or of aneurysms, on the bodies of the vertebræ.

EXAMINATION OF SPINA BIFIDA

Spina Bifida is indicated by the presence of a tumour in the lumbar or lumbo-sacral regions. In the absence of a tumour the presence of a dimple or of a hairy patch should suggest the presence of spina bifida occulta. Club foot, congenital dislocation of the hip or hydrocephalus may coexist.

Method of examining a spina bifida : If a sac is present look for signs of inflammation or of rupture. A dimple on the sac wall will, if seen, indicate the point of adhesion of nervous structures within. Make slight pressure on an unruptured sac and note if bulging of the anterior fontanelle is produced. Incise the sac at its lateral margin to determine its structure and the relations of the spinal cord and nerves to its cavity. Subsequent examination of the membranes of the cord will show whether spinal meningitis or hæmorrhage has occurred.

In a **Myelocele** the spinal cord has opened up posteriorly so that the central canal opens on the exposed surface in the midst of a nævoid area.

In **Syringo-Myelocele** the cord is cystic, its substance forming the inner wall of the sac. The spinal nerves pass round the convexity of the cyst. This form is rare.

In **Meningo-Myelocele** the cyst is formed by the arachnoid ; the cord is usually adherent to the cyst posteriorly. The spinal nerves pass across the interior of the sac to their foramina of exit.

In **Meningocele** the membranes alone protrude, often the dura only. The cord and the spinal nerves occupy their normal positions.

METHOD OF OPENING THE SPINAL CANAL

Next lay open the Spinal Canal.

The head should be flexed over the end of the table and firmly held by an assistant. Commencing at the external occipital protuberance, make a long median incision over the spinous processes to the base of the sacrum. Hold the knife firmly in the fist, cut with its belly not with its point, and prevent the skin from slipping laterally under its blade by placing the thumb and fingers of the left hand astride the back of the knife, thus pressing firmly on the skin at each side whilst the incision is made. Reflect the muscular masses on each side from the spines and laminæ of the vertebræ and from the lower part of the occipital bone. The laminæ should be well cleared from muscle and tendon as far outwards as the projections of the transverse processes, which are easily recognised in the dorsal region as a lateral ridge of tubercles on each side of the vertebral spines.

Examine the exposed spinous processes, the laminæ and the articular processes for signs of dislocation or fracture. Do not be misled by the marked mobility of the cervical vertebræ after the muscles have been reflected from them. A localised effusion of blood may direct attention to subjacent bony

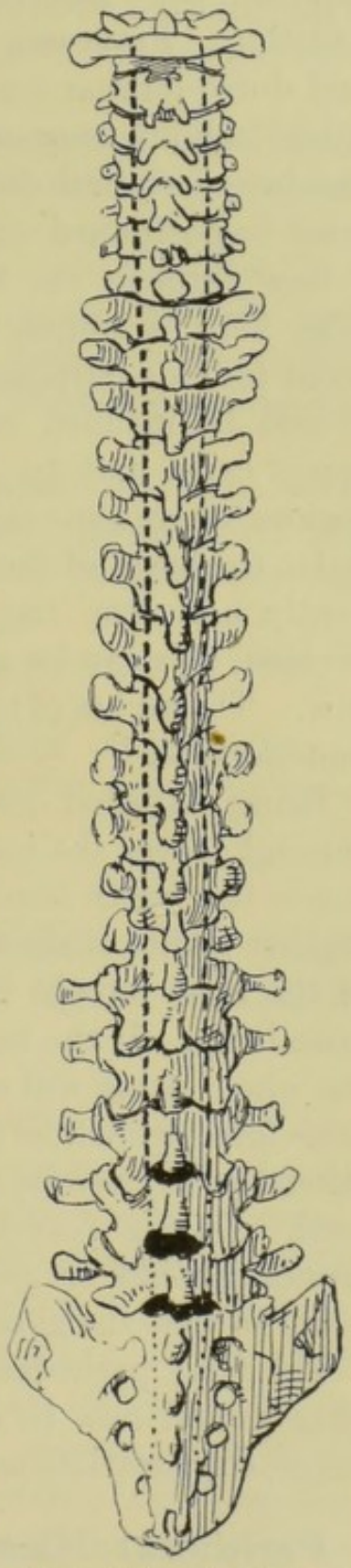

FIG. 17.—Incisions through the vertebral laminæ for removal of the Spinal Cord.

injuries, but fracture may be present without extravasation. Fractures are most common in the cervical and dorso-lumbar regions. In all cases where injury to the spinal column is suspected the bodies of the vertebræ and the intervertebral discs should also be examined, but this must be postponed until after the removal of the spinal cord.

Saw through the laminæ of the vertebræ on each side (Fig. 17). A simple back-saw is the most effective instrument for this purpose. Whilst cutting the edge of the saw should be inclined inwards as well as downwards or the spinal canal may be missed entirely. In sawing the dorsal regions where the canal is narrow apply the saw quite internal to the bases of the projecting transverse processes. Saw gently and stop immediately the laminæ are completely divided, this can be gauged by the resistance offered to the saw. The arches of the atlas and axis are best removed with bone forceps.

Being satisfied that the laminæ are completely cut through, sever the interspinous and interlaminar ligaments below the fourth lumbar vertebra (level of a line joining the highest points of the iliac crests). Insert the closed blades of the bone forceps beneath the arch of this vertebra and prise it up. If the bones have been sawn through properly the whole string will come away easily. If they do not, the bone forceps must be used to free them, particular care being taken not to exercise the slightest pressure on the subjacent cord. During removal the lower part of the chain of laminæ should be grasped with a lion forceps or a folded towel so that the glove is not scratched.

The fatty perithecal tissue and the veins external to the dura should now be inspected for the presence of hæmorrhage, suppuration, new growths or external dural adhesions.

Perithecal Hæmorrhage is usually the result of injuries. In some instances the effusion is derived from an intrathoracic or abdominal aneurysm which has eroded the spinal column. Extradural hæmorrhage may also occur

during the course of diseases characterised by a hæmorrhagic tendency, or in cases where repeated and widespread convulsions have preceded death.

Be careful not to mistake the escape of blood from spinal veins, opened during the removal of the spinal laminæ, for a pathological condition.

Perithecal Suppuration is rare. It has been met with as a primary condition. It may also extend from suppurative foci connected with the bones of the spinal column, deep sacral bedsores, or even from the abdominal or thoracic cavities.

New Growths sometimes originate in the perithecal tissues. More frequently they spread from the neighbouring bony structures or invade the spinal canal from the thorax or abdomen.

Spinal Hydatids, when present, are apt to find a nidus in the perithecal space.

Chronic Adhesive Inflammation of the spinal theca to the bones around is usually the result of bone disease or of an old fracture.

EXPOSURE OF THE GANGLIA ON THE POSTERIOR ROOTS OF THE SPINAL NERVES

If the ganglia of the posterior roots are to be examined the investigation should commence at this stage. The ganglia of the sacral and coccygeal nerves lie within the spinal canal, just outside the dura mater. To examine the other ganglia it is necessary, after exposure of the cord in its membranes and removal of the sacral laminæ with the saw, to clip away the posterior boundaries of the intervertebral foramina in which the ganglia lie. This may be effected with small bone forceps. As a preliminary the anterior and posterior spinal nerves, outside the foramina, should be laid bare by dissection and severed. Then, when the bony boundary of a foramen has been clipped away, the mass of connective tissue which invests the ganglion and nerve roots which

lie in the intervertebral canal, is gently lifted from its bed. The mass can be removed with the spinal cord to which it is still attached. On no account should the ganglia be pinched with forceps or otherwise bruised during removal. Bear in mind that the lower ganglia sometimes stray up along the roots of the cauda equina and are apt to be mistaken for small growths.

REMOVAL OF THE SPINAL CORD

If the arches of the vertebræ have been thoroughly and cleanly removed, the extraction of the spinal cord is not a difficult matter. Since its substance is easily rendered pulpy by bruising or squeezing, the greatest care must be taken not to compress the cord between the fingers or stretch it across the cut edges of the bones. First notice whether the dura appears to be unduly distended with fluid, then, with the sharpest scalpel obtainable, or with good scissors, divide the membranes and the cauda equina transversely in the lower lumbar region (in the adult the cord proper terminates at the second lumbar vertebra, in the child it reaches to the third). Note the characters and amount of any fluid which escapes from the membranes.

Grasp the cut ends of the dura with forceps and raise the cord carefully from its bed, cutting across the spinal nerves, outside the dura, close to the intervertebral foramina, as they come into view. (If the spinal ganglia are to be removed, the nerves are not to be severed.) By gentle traction on the lower end of the dura the cord is kept straight and free from twists or bends, for softening is easily simulated by injuries inflicted during removal. A few touches with the scalpel are needed to separate the attachment of the dura to the posterior common ligament of the vertebræ. Firm adhesion in this situation is pathological, however, and its cause should be sought. The chief difficulty in extraction will be experienced in the neighbourhood of the foramen magnum, around the

circumference of which the dura mater of the cord is firmly attached. If the arches of the atlas and axis have been properly removed, the attachment of the spinal dura to the foramen can be seen and severed close to the bone. This procedure is rendered easier if the brain has been removed first.

During removal of the spinal cord it is important to recognise the first pair of spinal nerves. These pass out under the vertebral arteries, the cut ends of which are easily found. The proximal ends of the nerves should be marked by ligatures, so that the recognition of any particular nerve root at subsequent stages of the dissection may be effected by counting down. Advantage may also be taken of the fact that the second dorsal and succeeding nerve roots are decidedly smaller than the first dorsal roots and those above them which participate in the formation of the brachial plexuses.

In cases of meningitis or of subtentorial lesions, where it may be necessary to test the communication between the ventricular cavities of the brain and the subarachnoid space,the cord should be exposed before the brain is removed. The dura and arachnoid must be incised in the lumbar region and the body suspended vertically with the head fully flexed. If the communication is patent the ventricles can be drained in this way. (For the special dissection required to expose the foramina of communication, see p. 221.)

Having removed the cord, complete the examination of the interior of the spinal canal. First inspect the inner aspects of the chain of laminæ removed, and then examine the sides and front of canal. The posterior common ligament is exposed on the backs of the bodies of the vertebræ, its normal appearance is smooth and pale, the presence of roughening or discoloration calls for examination of the structures in its immediate neighbourhood. Inspect the posterior aspects of the bodies of the vertebræ, and also run the finger over them to detect

any irregularities, such as elevations, depressions, or fissures. Examine the articular processes for fractures and dislocations. Pay particular attention to the position of the odontoid process of the axis, which may, by backward dislocation, cause sudden death. In such cases evidence of antecedent disease of the process or of its ligaments should not be overlooked. The condition of the intervertebral discs may be investigated by driving into them the blade of a strong knife; the resistance offered to this 'sounding' will convey some idea of the consistence of the discs, but the method of examination is hardly satisfactory. A far better way of ascertaining the condition of the discs, and also of the bodies of the vertebræ, is by removal of the suspected portion of the spinal column. This may be effected after the abdominal and thoracic viscera have been taken out, by sawing through the column some distance above and below the lesion; in the dorsal region the necks of the ribs must also be divided. The loose section is then liberated with the knife, removed and examined, the bodies of the vertebræ being divided longitudinally with the saw. This method is applicable to the investigation of fractures, dislocations, and other lesions of the vertebral column.

Fractures and Fracture-Dislocations are usually situated in the lower cervical or lower dorsal regions. Dual fractures may exist in the cervical regions, involving the first or second vertebra above and the fifth or sixth below.

As the result of judicial hanging the common lesion is an injury to the axis or the third cervical vertebra. Contrary to current belief, the odontoid process is rarely broken and the transverse ligament rarely ruptured. The cord and its membranes are often torn and the vertebral arteries lacerated. The sutures between the bones at the base of the skull may be 'started.' In ordinary hanging, as opposed to judicial, death is due to asphyxia and cerebral anæmia, not to injuries to the spinal column.

In all cases of spinal injury the condition of the nerves in the intervertebral foramina should be ascertained.

A transverse bruise of the cord, with or without intramedullary hæmorrhage, may sometimes be found in cases where the spinal column appears to be uninjured.

Caries of the Bodies of the Vertebræ may cause prominences on each side of the posterior common ligament. These consist of masses of granulation tissue which may be caseous or suppurating. The intervertebral discs are eroded and destroyed. In all cases of caries the anterior aspects of the vertebral column should be examined after removal of the abdominal and thoracic viscera.

New Growths in the Bodies of the Vertebræ may cause compression of the cord, fracture, or hæmorrhage into the spinal canal. The relations of the growth to the nerve roots should be examined, and the possibility of direct extension into the spinal canal, through the intervertebral foramina, remembered.

Sarcomata of the vertebræ may be primary or secondary to growths elsewhere.

Carcinoma is secondary, and when it is found a careful examination of the breasts, thyroid gland, œsophagus, pelvic organs, and prostate, as well as of other parts is requisite.

Aneurysms of the Aorta may erode the spinal column and project into the spinal canal. They may determine the occurrence of fracture or dislocation of the spinal column. (See also p. 92.)

Spondylitis Deformans or Osteo-arthritis of the Spine.—In this disease the interarticular and costo-vertebral joints may be eroded and eburnated. The bodies of the vertebræ may be ankylosed owing to overbridging of the intervertebral discs and the formation of bony splints alongside the anterior common ligament.

Osteitis Deformans may involve the spine as well as the bones of the cranium (p. 223), pelvis, and extremities.

EXAMINATION OF THE SPINAL CORD AND ITS MEMBRANES

The cord, still invested in its membranes, should be placed flat on the table or on a dish. Carefully inspect the outer aspect, and then, lifting up the cut edges of the dura mater with the forceps, slit up that membrane in the mid-line along its whole length. Turn the cord over and incise the dura on this aspect also. Be careful to avoid damage to the subjacent cord by the points of the scissors.

Reflect the dura on each side and inspect its inner surface, also the exposed surface of the arachnoid. Examine the nerve roots as they pass through the membranes; redness and swelling indicate recent inflammation, greyness and shrinkage indicate atrophy, such as may result from constriction, pressure, or cord disease. The posterior nerve roots should be twice or thrice the size of the anterior ones.

If healthy, the apposed surfaces of the dura and arachnoid are smooth, moist, and glistening. A dull sticky appearance indicates the presence of inflammation, which may also be indicated by obvious congestion, ecchymoses, or purulent exudation. If tumours are present within the theca their positions and relations should be accurately determined.

Spinal Pachymeningitis.—Inflammation does not readily spread through the substance of the dura mater. The outer surface may be involved by extradural inflammation or tuberculosis of the spinal column. The inner may be inflamed in association with the pia-arachnoid.

Hypertrophic Internal Pachymeningitis is a chronic inflammatory thickening of the inner layers of the dura mater. It appears, usually in the cervical region, as a fusiform tumour, evident as soon as the canal is opened. The nerve roots are compressed, and pressure may also be exercised on the spinal cord giving rise to local inflammation or softening. The thickened dura may become adherent to the pia-arachnoid.

Acute Spinal Leptomeningitis.—The early signs of this are dulling, reddening and ecchymosis of the pia-arachnoid, and sometimes also of the apposed surface of the dura. In the purulent forms the exudation may be confined beneath the thin arachnoid mater so that the space between the latter and the dura escapes, but this is by no means always the case. There may be an abundance of turbid fluid beneath the arachnoid. The position of the exudation in spinal meningitis is largely influenced by gravity, hence it may be limited to the posterior aspect of the cord. When the inflammation has spread from the cerebral meninges the cervical region alone may be affected.

The purulent varieties of spinal leptomeningitis are practically the same as the cerebral forms already described on pp. 237, 238.

Tuberculous Leptomeningitis may be recognised by the presence of miliary tubercles. The amount of exudation is scanty, and, as a rule, cerebral leptomeningitis coexists.

Syphilitic Leptomeningitis presents itself in recent cases as a gummy infiltration of the pia-arachnoid. In old-standing cases, and after treatment, the exudation may become fibrous. Occasionally patches of caseous material are present.

Chronic Spinal Leptomeningitis.—Dense localised adhesions between the leptomeninges and the dura may persist as the consequence of past meningitis. In other cases the dura may be tensely distended with fluid, the arachnoid, particularly in the lower dorsal and lumbar regions, thickened and the cord itself shrunken and indurated. Sections of the cord may show areas of system degeneration which may be secondary to the meningitis or of independent origin.

Intradural Spinal Hæmorrhage. — The blood may be enclosed by the dura or by the arachnoid. The causes are similar to those enumerated for extradural bleeding (p. 256). Hæmorrhages sometimes occur in the membranes after lumbar puncture has been performed.

Intradural Spinal Tumours. — These tumours are more likely to produce compression of the cord than

are tumours outside the dura. They are usually sarcomatous, fibrous, or syphilitic. Sarcomata may be multiple and spring from the membranes or nerve roots. Syphilomata are sometimes diffuse and infiltrating.

Meningeal neoplasms usually lie in the dorsal region, posteriorly or laterally. The cord may be narrowed or indented by their pressure, and softening may have supervened. Secondary degenerations may be conspicuous in the white columns.

Having reflected the dura and examined the membranes and nerve roots, attention should be turned to the cord itself.

Localised discoloration or bruising will not escape detection. Localised wasting or compression by tumour or caries is fairly obvious if present. General wasting, such as occurs in locomotor ataxy, may be overlooked, but disproportion in size between the cord and its sheath, coupled with wasting of the posterior nerve roots, particularly in the lumbar region, should suggest the presence of this disease. As before mentioned, in estimating the wasting of nerve roots, it is important to bear in mind that the posterior roots should be twice or three times the size of the anterior.

The fresh, normal cord has a fairly firm consistence, but it readily becomes soft as the result of post-mortem changes, especially in the mid-dorsal region. Parts of the cord which have been bruised during extraction may appear semi-diffluent, and may erroneously be supposed to be the sites of myelitis.

After a general inspection of its surface the cord should be examined by a series of transverse sections. These should be made at intervals of about half an inch with a sharp scalpel. The separated segments of the cord will be maintained in their serial continuity by the attachments of the nerve roots to the dura and arachnoid.

To make the sections, the cord should be stretched by its own weight over the left forefinger, its upper end being

grasped between the forefinger and thumb, and gradually shifted to bring the successive segments under the knife (Fig. 18).

Each section as made should be rinsed and inspected, attention being directed in turn to the white columns, the grey matter, and the central canal. The examination should be made in a good light, as slight grey degeneration of the white columns (sclerosis) is easily overlooked. Indeed for its detection it is often advantageous, after making

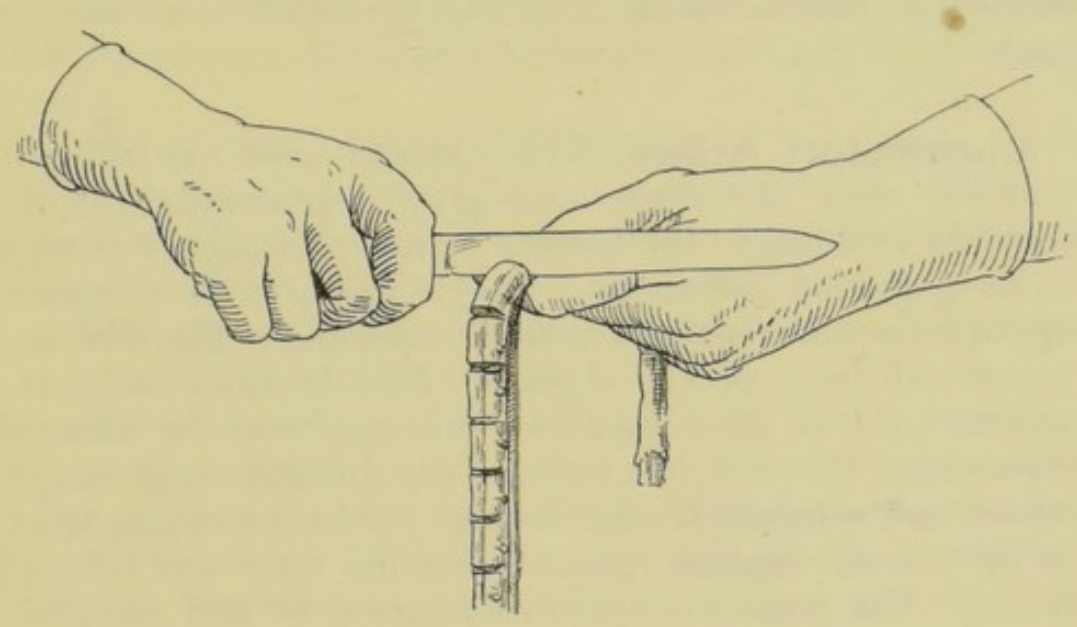

Fig. 18.—Incision of the Spinal Cord.

the incisions, to immerse the cord in Müller's fluid for a fortnight. The distribution of degeneration in the various columns should be ascertained as accurately as possible. When the posterior columns are involved the relation of the degeneration to the posterior root zone, or that part of the posterior external column which lies next to the posterior cornu and contains many fibres of the entering posterior root, is important. In the grey matter considerable changes may be present, although not obvious to the naked eye; for instance, in progressive muscular atrophy, although the motor nerve cells are wasted, the anterior grey cornua may appear normal in size; and even in acute

anterior poliomyelitis, unless congestion, hæmorrhage, or softening is present, the cells may be severely involved without much naked-eye evidence of the change. The outlines of the grey matter should be carefully observed, since gliomata and myxomata closely resemble it in colour.

When extensive disease is limited to one or more segments of the cord, tracks of degeneration in the ascending columns above the lesion and descending columns below should be looked for. Needless to say, in cases of spinal cord disease a microscopical examination should always be made.

Locomotor Ataxy.—The spinal cord is wasted. Sections show shrinkage and grey degeneration of the posterior columns in the lumbar region, particularly marked in the posterior root zones. The degeneration is continued upwards in the posterior median columns. There is also a cervical form of the disease. In advanced cases the greater part of both posterior columns may be affected throughout the cord, but tracts of endogenous origin escape. Sometimes sclerosis is also evident in the ascending tracts of the lateral columns (direct cerebellar tract and Gower's tract). The posterior nerve roots are grey and atrophic; the optic nerves may present similar degeneration.

Charcot's joints, perforating ulcers of the feet, and fragility of bones may also occur. Disease of the ascending aorta and of the aortic valves, due to syphilis, may coexist. Sometimes locomotor ataxy is associated with the cerebral changes of general paralysis. Ascending urinary infection is not uncommon.

Friedreich's Hereditary Ataxy.—In this disease sclerosis invades the posterior, lateral, and sometimes also the anterior columns of the cord. As in tabes, the posterior column degeneration involves the posterior root zones. The disease may be distinguished from ataxic paraplegia by remembering that in Friedreich's ataxy the posterior column degeneration is more intense and the posterior nerve roots are degenerated. Lateral curvature of the spine and talipes equinus or equino-varus are also present.

Lateral Sclerosis.—The essential factor in this disease is grey sclerosis of the pyramidal tracts of the lateral columns. A pure variety of the disease is exceptional; atrophy of the anterior cornual cells is usually associated, constituting the affection known as amyotrophic lateral sclerosis.

Lateral sclerosis may be secondary to lesions situated higher in the cord or in the brain. It may also be a part of a disseminated sclerosis or occur in general paralysis of the insane. In association with sclerosis of the posterior columns it may be a part of Friedreich's hereditary ataxy, of ataxic paraplegia, or of subacute combined sclerosis of the cord.

Subacute Combined Sclerosis of the Cord.—In this disease the most extensive and most recent change is often seen in the mid-dorsal region, where a marked destruction of the white matter of the periphery may be observed, whilst the grey matter and white substance immediately adjacent to it remain normal.

Sclerotic changes are traceable in the anterior and lateral pyramidal tracts and in the posterior columns, but are not necessarily limited to these parts, for the direct cerebellar tract and Gower's tract may also be degenerated. In the lumbar region the sclerosis is more restricted than elsewhere.

Other changes which may be found are anæmia; an enlarged liver, which may give an iron reaction with potassium ferrocyanide and hydrochloric acid; replacement of the yellow marrow of the bone shafts by red, or an aplastic condition of bone marrow; and evidence of secondary ascending urinary infection.

Ataxic Paraplegia.—The changes are similar to those of subacute combined sclerosis, i.e. combined degeneration of the posterior and lateral columns, but are more chronic. Anæmia is not necessarily present.

Acute Anterior Poliomyelitis.—In some recent cases of this disease the naked-eye changes are very slight, in others the changes, which are limited to the anterior cornua, vary from pinkish congestion to well-marked hæmorrhage and softening. Since the condition may be

disseminated, the whole length of the cord should be examined, particular attention being paid to the cervical and lumbar enlargements. Spinal meningitis may occur. Ascertain the condition of the anterior spinal artery; by a few authors the disease is attributed to thrombosis of the small branches of this vessel. The disease may also attack the bulb and mid-brain. When a long period has elapsed since the onset, the damaged cornua may be cystic, or indurated and shrunken. The anterior nerve roots which issue from the affected regions are small and grey, and the muscles supplied by them are wasted. Slight sclerosis of the lateral columns coexists in some cases.

Acute Myelitis.—The region of the cord involved by myelitis may be swollen and softer than normal, and the pia mater in the neighbourhood may be red and hyperæmic. On section the contrast between the white and grey matter may be very indistinct, owing to congestion. Red softening is present in cases of recent origin, but the colour alters to chocolate or yellow in those of longer duration. At a later period still the site of inflammation may present a grey, sclerotic appearance. Myelitis may be focal or disseminated. Tracts of ascending and descending degeneration of the white columns may be recognised in cases of some standing.

Pathological Softening of the Cord, without obvious signs of inflammation, is attributed to thrombosis. The condition, which is a result of syphilis, selects the lower dorsal region by preference, but may occur elsewhere. If examined soon after the onset, the softened cord has a creamy consistence and there is no overlying meningitis. The larger vessels of the surface hardly ever show any change, but the smaller arterioles which penetrate the cord are occluded in consequence of endarteritis. There is no evidence of any inflammatory reaction. If examined some time after the onset the cord may be cheesy in consistence and appear harder and firmer than normal.

Secondary changes in the form of ascending urinary infections, bed-sores, etc., may occur.

Hæmorrhage into the Spinal Cord, as a general rule, is a secondary and not a primary event. If primary, it is

the result of injury. Secondary hæmorrhage may complicate myelitis, take place into syringomyelic cavities or into the substance of a new growth. If it extends through a considerable vertical extent of the cord the presence of syringomyelia should be strongly suspected.

Progressive Muscular Atrophy.—This disease usually involves the cervical enlargement. Even in advanced cases the anterior cornua, which are affected, may not be much changed in shape to the naked eye, so a microscopical examination is requisite. As a rule widespread degeneration of the crossed and direct pyramidal tracts accompanies the disease and imparts to these tracts a grey appearance. The pyramidal degeneration may extend to the medulla, pons, or higher.

Primary atrophy of the bulbar nuclei constitutes the condition known as progressive labio-glosso-laryngeal palsy.

The anterior roots arising from the diseased cornua are small and grey. The posterior roots are normal. The affected muscles are pale and wasted; in some instances all their fibres may have disappeared.

Syringomyelia.—The cavities which characterise this disease chiefly occur in the upper dorsal and the cervical regions. They are confined to the posterior half of the cord, usually to the posterior columns. Associated with them is a quantity of grey and translucent embryonic tissue. The cavities may appear distinct from the central canal, although originally developed in connexion with this or the embryonic tissue around it; they sometimes extend into the medulla or pons. The gliomatous tissue may assume the dimensions of a tumour in one or more regions. Hæmorrhage sometimes occurs into the syringomyelic cavities. Ascending and descending tract degenerations may also be present. Localised muscular atrophy, chiefly in the upper limbs; trophic affections of the extremities in the form of whitlows, ulcerations, or gangrene; or scars due to painless burns may be found in patients suffering from this affection.

Tumours within the Cord.—The most common intramedullary tumours are syphilitic or gliomatous, but myxomatous, tuberculous, and other varieties may occur. Tumours

more often spring from the pia mater or the tissue around the central canal than from other parts. As a rule the growths are easily recognised, but myxomata and gliomata resemble the grey matter in tint. Cystic or hæmorrhagic changes may occur. Secondary degenerations of the white columns up and down the cord may often be traced from the region occupied by the tumour.

Compression of the Cord.—Compression may be due to injury; to disease of the bones, such as caries; to new growths in the vertebræ or meninges; to pachymeningitis; gummata; aneurysm; or hydatids. The compressed region is narrowed or indented. In cases where the compression is of recent origin the interior of the compressed portion may be red and soft, but in chronic cases sclerosis and system degenerations are often present. The nerve roots which issue from the compressed region may be wasted.

CHAPTER VII

EXAMINATION OF THE BONES AND JOINTS

On account of the mutilations involved it is inadvisable to subject the bones and joints to a complete examination in every case.

Investigation, however, should not be neglected when disease or injury of these structures is suspected. In all instances of fracture or dislocation a dissection should be carried out with the object of determining the exact position and extent of the injuries. In cases where dislocation or fracture has resulted from comparatively trivial injuries the presence of antecedent joint disease, undue fragility of the bones, new growths, or inflammatory conditions of the shafts and epiphyses should not be overlooked.

The amount of repair or the presence of suppuration will also need investigation. Examination of the joints and epiphyses is very necessary in obscure cases of a pyæmic character ; in certain diseases of the spinal cord, such as locomotor ataxy and syringomyelia, in connexion with which trophic lesions may occur ; and also in bodies where the visceral lesions are such as to suggest the presence of gout.

Examination of the bone marrow is especially indicated in certain blood diseases, such as pernicious anæmia and leukæmia, as well as in local bone disease.

EXAMINATION OF THE BONES

Long bones, if extensively diseased, are best examined by complete longitudinal division after removal from the body. The bones may be removed entire by suitable

incisions: for instance, the femur may be extracted through a long incision on the outer side of the thigh, the head being disarticulated first, the shaft then cleared of the soft parts, and finally the lower end separated from the tibia through the outer side of the knee-joint.

Since complete removal of a bone entails the making of incisions of considerable length, it may be preferable to expose only the diseased portion; when this happens to be the end of the bone, divide it longitudinally for a certain distance with the saw, and then by means of a transverse cut remove one half of the diseased extremity, inspecting the other half *in situ*.

Before turning attention to the condition of a bone it is often of assistance to study the condition of the overlying soft parts, since the appearance of the latter may give a clue to the nature of the underlying bone disease. Chronic localised periostitis, leading to the formation of new bone, may be found at the base of a chronic ulcer. A characteristic induration with diffuse sinus formation in the skin and tissues over the mandible may indicate the presence of actinomycosis. Sinuses, the depressed adherent orifices of which are bounded by rigid edges and sclerosis of soft tissues, are characteristic of pyogenic bone disease. Sinuses with undermined orifices, exuberant granulations, and the discharge of caseous débris or thin pus are suggestive of tuberculous infection.

To ensure complete examination of a bone attention should be directed to the following points: (1) The appearance of the periosteum; (2) The condition of the shaft; (3) The state of the epiphyses and of the lines of union with the diaphysis; (4) The appearance of the marrow in the shaft and in the cancellous tissue of the expanded ends.

The Periosteum.—The superficial layer of healthy periosteum is thin and has a firm, smooth, ligamentous appearance; the deeper layer is less dense. The periosteum of young bones is thicker and more vascular than that of old ones. Inflamed periosteum is softer, thicker, and more

vascular than in health, and also œdematous. Inflammatory exudations collect more abundantly in the deeper layers, and a collection of pus may often be found between the periosteum and the bone. Pus penetrates the outer layer of the periosteum with difficulty, hence its tendency to strip up the membrane from the bone instead.

Increased porosity and roughness of the surface of the subjacent compact bone is evidence of osteitis; if the bone is dull white and smooth, necrosis has occurred. Subacute or chronic inflammation of the periosteum may lead to the deposit of new periosteal bone on the shaft (osteoplastic periostitis); the new bone at first scales off easily, being porous and vascular, but later it becomes exceedingly dense. A sheath of periosteal bone, the involucrum, may enclose large sequestra derived from the shaft. In pulmonary osteo-arthropathy a deposit of periosteal bone is laid down along the shafts of the bones. In chloroma the periosteum becomes the site of diffuse greenish infiltrations.

The Shaft.—Irregular thickenings (periosteal nodes) or erosions (caries) of the surface of a bone are due to inflammation; the condition of the periosteum and of the soft tissues overlying the affected part should be ascertained. In all cases of acute, localised, periosteal inflammation the subjacent bone will be found roughened and the site of a certain degree of rarefying osteitis; the condition is therefore more accurately designated 'osteoperiostitis.'

The condition of the interior of the shaft is ascertained by making a longitudinal section. This will show whether the bone substance is increased in density (sclerosis) or rarefied (osteoporosis). Undue softness of bone should not escape attention. Very acute inflammation of compact bone causes necrosis; inflammation which is less acute may lead to rarefaction or 'caries'; chronic inflammation produces sclerosis. Undue softness of bone is an indication either of deficient calcification (in growing bone) or of decalcification of bone which had attained its normal

consistence. Deficient calcification is characteristic of active rickets; decalcification is met with in osteomalacia and in the pre-sclerotic stages of osteitis deformans.

The Epiphyses.—When examining an epiphysis attention should be directed to, first, the layer of cartilage which is applied to the end of the diaphysis, fitting the latter like a cap, and, secondly, the state of the ossific nucleus in the centre of the epiphysis itself. If an epiphysis is separated the presence of antecedent inflammation should not be overlooked.

In young bones a layer of proliferating cartilage should just be visible; it may be recognised by its dull white tint and is about $\frac{1}{2}\frac{1}{4}$ inch in thickness. Examination with a hand-lens reveals vertical striation in this layer. Between the proliferating layer and the diaphysis is a zone of calcified cartilage which is faintly yellow. A sharply defined band of congestion marks the end of the diaphysis. The periosteum is thickened opposite the epiphyseal cartilage and firmly attached to its edges. This attachment may be an important factor in limiting the spread of inflammation.

The Marrow.—Both red and yellow marrow, if present, should be inspected. In young individuals all bones possess a red marrow, but the change to yellow begins very early, and progresses from the centre of the bone towards the periphery, or, in long bones, towards the ends. At the age of puberty the marrow found in the shafts of the long bones is yellow and consists mainly of fatty tissue, whilst that in the articular ends of the long bones and in the flat and short bones still remains red. In old age marrow tends to become brownish and gelatinous in appearance.

Bones chosen for the investigation of marrow should be the shaft of the femur for yellow marrow, the sternum and ribs for red marrow.

To expose the marrow of the femur a longitudinal incision should be made over the shaft of the bone, the soft parts freely separated and held down by slipping a chisel between them and the shaft, whilst a piece of bone, two inches long,

is sawn out. The excised portion should be sawn longitudinally and the interior examined. Portions of ribs are easily removed with bone forceps and split open. The marrow of the sternum may be exposed by making a longitudinal saw-cut on the back of the bone and bending apart the two halves.

An increase of red marrow in the shafts of the long bones, replacing the yellow, occurs in pernicious anæmia, some other blood diseases, certain marantic conditions and osteomalacia. In the last-mentioned disease the bone at the same time becomes pliable owing to decalcification, a change which is characteristic.

In aplastic anæmia compensatory reaction of the bone marrow is absent.

In leukæmia marrow of a pale pinkish tinge may replace the yellow marrow of the shafts and occur in the ends of bones; less commonly the marrow is yellowish grey. There is a greater destruction of the true red marrow in the lymphocythæmic than in the myelæmic form of leukæmia.

Atrophy of Bone may occur as the result of old age, disuse, continuous pressure by an aneurysm or a new growth, or as a trophic disturbance induced by certain diseases of the central nervous system, such as tabes or syringomyelia.

The compact tissue of the bone is absorbed from without inwards, the Haversian canals become enlarged and confluent, and considerable deposits of fat occur in the interstices. Atrophied bones may be fractured by very slight violence. Although rarefied and wasted the bone is still hard, not decalcified and wasted at the same time, as in osteomalacia.

Rickets.—Well-marked rickety deformities are unlikely to escape detection at the post-mortem examination, but slight degrees of the disease are apt to be overlooked. Particular attention should be directed to the costo-chondral junctions and especially to their inner aspects where the swellings, if present, are very obvious. The slight enlargement which is normally present at the junction of rib

and cartilage should not be mistaken for disease. The rib and its cartilage should be split longitudinally and the line of union carefully scrutinised. The epiphyses of the wrists, ankles and knees should also be inspected and the condition of the cranial bones and fontanelles ascertained. The zone of semitransparent proliferating cartilage in a rickety epiphysis is increased both in breadth and depth; the line of ossification is very irregular in outline instead of being bounded by a fairly sharp margin as in health.

The membrane bones when affected by rickets become abnormally thick and unnaturally porous. The new bone in rickets is soft and resilient, whereas syphilitic new bone is hard. In cases of exceptional severity the periosteum of the long bones may appear thickened, soft and vascular. The periosteum when stripped may feel gritty and the subjacent bone is reddish and spongoid. In advanced cases the greater part of the thickness of the diaphysis may consist of porous bone of this character. After recovery rachitic bone may become abnormally hard.

The presence of rickets should not be overlooked in young children who have died from pulmonary disease, intestinal affections, or convulsions.

Infantile Scurvy.—In this disease the periosteum of the long bones, particularly those of the legs and thighs, is thick, vascular and more or less separated from the diaphyses by hæmorrhagic effusions. The detached periosteum may produce a thin and imperfect shell of bone in its abnormal position. Hæmorrhage may occur in the medullary cavities of the long bones and ribs, causing osteoporosis or rarefaction. Fractures through the unions of diaphyses with their epiphyses are not uncommon. Other morbid changes are swelling and sponginess of gums around erupted teeth; hæmorrhages into the skin, mucous membranes, various viscera, and maybe the joint cavities; extravasations of blood amid the muscles.

Acute Infective Bone Disease (*Acute periostitis; acute osteomyelitis; acute necrosis*). — Acute infective disease attacks the long bones, the larger more frequently than the smaller. Both periosteum and marrow may be involved or the infection be limited to the periosteum and

underlying compact tissue. In the first-mentioned form the disease commences at the end of the diaphysis and may spread over the whole length of the medullary cavity and shaft. The infected marrow is hyperæmic or infiltrated with pus and its veins thrombosed. Extensive necrosis occurs and epiphyses may become separated. Infected periosteum becomes vascular, swollen and œdematous ; pus may accumulate between the periosteum and the shaft or between the periosteal layers. Owing to the close attachment of the periosteum to the articular cartilage the joint may escape, but infection sometimes takes place ; this is specially apt to occur if the epiphyseal junction is intra-articular, as is the case with the hip-joint. Sometimes the adjacent joints contain a serous effusion.

Other pathological changes which may be met with are, abscesses in the lungs and heart-muscle, endocarditis, purulent infections of the serous sacs, abscesses or acute inflammation of the kidneys, and signs of acute systemic infection. The lesions in the bones and joints may be multiple.

In every case of acute periosteal inflammation the bone medulla should be exposed and examined.

Infective bone disease of a less fulminating character is sometimes met with and may be due to organisms similar to those which cause the acute forms, or to infection with typhoid or other bacilli. Chronic bone abscess may result.

Tuberculous Disease of Bone.—Tuberculous infection may involve the periosteum, the cancellous tissue, or both together.

Tuberculous Periostitis.—Pulpy tuberculous granulation tissue infiltrates the periosteum, and may permeate the superficial layers of the subjacent bone. Suppuration may occur between the bone and periosteum. The exposed bone is rough and friable ; the extent to which it is involved will depend on the thickness of the compact layer.

In tuberculous disease there is but little new-formed bone on the periosteal surface of the shaft, whereas in chronic periostitis of syphilitic or pyogenic origin the new bone formation may be a pronounced feature.

Tuberculous Osteitis.—The cancellous tissue of the

articular ends of the long bones or the cancellous substance of short bones is the site of this form of bone tuberculosis. Hence tuberculous disease is met with in the bodies of vertebræ, in the bones of the carpus or tarsus, in the neck or condyles of the femur, the head of the tibia, the olecranon, condyles of the humerus, and the metacarpals or metatarsals of children. It assumes the form of a rarefying osteitis, the interstices of the bone being filled with soft, greyish granulation tissue, in the peripheral parts of which tubercles are sometimes evident. The granulations are gritty from the presence in them of molecules of bone, and caseation may occur. The bone itself becomes spongy and friable; it is easily broken down by exercising pressure on it. Islets of bone may become isolated and undergo necrosis. Tuberculous sequestra are usually permeated by caseous material which gives them a yellowish colour. Suppuration is frequent, and adjacent joints may be invaded by perforation. Localised bone abscesses surrounded by thickened and sclerosed bone may be found. Diffuse tuberculous osteomyelitis is uncommon; in this variety the infective process, commencing in the cancellous tissue of the end of the bone, extends into the medullary cavity, which becomes filled with soft tuberculous granulation tissue.

Syphilitic Disease of Bone.—This may present itself in any of the following forms: Chronic periostitis, leading to the formation of nodes or of diffuse thickening; osteitis, giving rise to sclerosis and increased weight, or to rarefaction, or to both kinds of change in combination; circumscribed, or diffuse gummatous formations which may be periosteal or medullary.

Subperiosteal gummata usually cause carious erosion of the subjacent bone; this form of caries may affect wide areas of the calvarium.

Syphilitic necrosis may occur as a result of advanced sclerosis. The syphilitic sequestra are notoriously slow to separate.

Bones especially affected by syphilis are those of the skull, the nasal bones, tibia, femur, radius and ulna.

Congenital syphilis may give rise to epiphysitis (see p. 307).

Changes due to syphilis and rickets sometimes exist side by side (see pp. 223, 224).

Tumours of Bone.—These may be primary or secondary in origin. Primary tumours of bone belong to the connective tissue series, and comprise chondromata, osteomata, and various forms of sarcoma.

If on section the outline of the bone can be traced through the tumour, the latter is periosteal in origin and usually sarcomatous in nature. Fine bony trabeculæ may radiate in a characteristic manner from the surface of the shaft through the substance of the surrounding growth. Periosteal sarcomata are peculiarly malignant.

Tumours of central origin give rise to expansion and thinning of the bone which contains them. Primary central tumours may be giant-celled or small-celled sarcomata; the former variety is practically limited to its place of origin, whilst the latter diffuse themselves through the medullary cavity, giving rise also to secondary growths in the neighbouring tissues and in the viscera.

Multiple Tumours of the Bone Marrow.—In rare instances a diffuse new growth of pale or reddish colour extensively involves the bone marrow, especially that of the vertebræ, ribs and sternum. The bones become soft and friable from absorption of their compact tissue, and localised outgrowths may, in some instances, sprout through the shafts. The disease nearly always remains limited to the osseous system and a peculiar form of proteid (Bence-Jones) is found in the urine. The tumour is in some cases myelomatous, consisting of modified marrow cells; in others it consists of cells which present the characteristics of plasma cells, and hence is designated a plasmoma.

Multiple Sarcomata of Bones sometimes supervene in osteitis deformans.

Chloroma, another rare disease, is distinguished by the occurrence of characteristic greenish infiltrations in the periosteum of the vertebræ, skull bones, ribs and glands.

Secondary Tumours of Bones may be sarcomatous or carcinomatous. Bone carcinoma may be secondary to carcinoma of the breast, lung, intestinal tract, prostate, thyroid, adrenal, or other parts; hence all possible sites of

origin should, as far as is possible, be inspected at the autopsy on such cases. When involving the spine, carcinoma is especially apt to attack the lumbar vertebræ. Needless to say all cases of bone tumour should be submitted to microscopical examination in order that their exact nature may be established.

Osteitis Deformans (*Paget's Disease of the Bones*).—This disease affects the long bones of the limbs, the spine, the pelvis and the skull. It occurs in mid-life or old age. The bones of the cranium, but not of the face, become bulky, mainly from eccentric overgrowth, so that the cranial cavity is not encroached upon. All the affected bones are increased in thickness. Their compact tissue becomes finely porous and reticulated, hence the shafts have a thickened, rough appearance. The medulla also is encroached upon by a formation of porous bone. When the disease is recent the bones are quite soft, but, as a rule, by the time the patient arrives at the post-mortem table, partial sclerosis has supervened, although the bending of the shafts still indicates the presence of softening in the earlier stages of the disease.

Changes resembling those of osteitis deformans are sometimes limited to one bone. Sarcomatous disease may supervene in bones affected with Paget's disease.

Osteomalacia.—This disease affects adults, typically attacking women in connexion with pregnancy. Its pathological characteristics are decalcification and absorption of osseous tissue together with the overgrowth of a richly cellular and very vascular marrow. The bones in consequence become pliable, are liable to fracture, and on longitudinal section appear as wasted shells filled with marrow. The beaked or triradiate pelvis is very characteristic of the condition, and the osseous change is sometimes limited to this region, but the ribs and bones of the limbs may also be affected.

Pulmonary Osteo-arthropathy.—The osseous change in this disease consists in the symmetrical deposit of layers of friable new bone beneath the periosteum of the long bones, on the shafts of the metacarpals and metatarsals and sometimes also on the carpal and tarsal bones.

The joint changes, if present, are limited to cartilage erosion without either eburnation or the development of osteophytic outgrowths. The clubbing of the fingers is due to changes in the soft tissues, the phalanges being unaffected.

Osteo-arthropathy may occur in association with the following diseases: Bronchiectasis, empyema, chronic pulmonary tuberculosis, thoracic new growths, spinal caries, congenital disease of the heart, lardaceous disease of the viscera.

Acromegaly.—See p. 232.

Hydatid Disease of Bone.—Hydatid cysts may develop in the interior of bones. They cause expansion and absorption of the compact tissue and, ultimately, lead to fracture. There is no limiting cyst wall, the daughter cysts being found free in the affected bone.

Actinomycosis of Bone.—The ray fungus may attack the mandible, giving rise to periostitis and abscess formation. Sometimes the bodies of the retropharyngeal vertebræ are directly infected from lesions of the pharynx.

EXAMINATION OF THE JOINTS

When incisions have been made into a joint during life these should be utilised as far as they can be for the purpose of post-mortem examination.

Ankylosed joints should, if possible, be excised, and the examination carried out by means of sections vertical to the articular surfaces.

Attention should always be directed to the sterno-clavicular joints when removing the front of the thorax. It sometimes happens that pus is unexpectedly found in one of these articulations; its presence should lead to careful examination of the other joints of the body.

The shoulder joint can be examined without external mutilation by dissecting down inside the flap of skin and muscle which has been raised for the purpose of opening the thorax. When the neighbourhood of the joint is

reached the arm should be forcibly abducted and the joint capsule incised on its axillary aspect.

The elbow joint may be examined by a vertical posterior incision. Free access to the joint may be obtained by sawing off the coronoid process of the ulna.

The wrist joint is best examined by a transverse dorsal incision, bearing in mind that the summit of the articulation lies nearly half an inch above a line joining the tips of the styloid processes of the radius and ulna.

The hip joint may be opened by a straight incision on its outer side. The limb should be flexed to an angle of forty-five degrees, and a long incision made over the great trochanter extending from well above the latter down to the upper part of the femoral shaft.

The knee joint is most thoroughly explored by forcibly flexing the leg and then making a curved incision from the prominence of one condyle, across the ligamentum patellæ, to the prominence of the condyle of the opposite side. The synovial pouches above the knee can be opened up by prolonging the incisions up into them.

The ankle joint may be opened by a transverse anterior incision. The line of the joint lies half an inch above the tip of the inner malleolus.

The metatarso-phalangeal joint of the great toe is opened by forcibly flexing the toe, and then cutting straight across the prominence of the head of the metatarsal. As soon as the incision has passed sufficiently outwards to divide the tendon of the long flexor, the joint will open up and may be freely inspected.

The sacro-iliac joint may be examined from the front by dissecting away the psoas muscle, or from behind by sawing away the overlying portion of the ilium.

The temporo-mandibular joint should be examined from its outer aspect. The vertical incision made for reflexion of the scalp should be utilised for this purpose, the integumental structures being dissected forwards as in examination of the parotid gland (see p. 218).

Complete examination of a joint entails inspection of the synovial membrane with its fringes and recesses; careful scrutiny of the articular cartilages, attention being directed to the appearance of their free surfaces, and to any indication of encroachment or pitting by the synovial fringes at their margins; determination of the condition of the ligaments; investigation of the state of the articular ends of the bones and of the condition of their epiphyses by suitable sections; and finally an examination of the tendons, fascial structures, and bursæ around the articulation.

In many cases a bacteriological examination of the synovial fluid and synovial membrane is essential for the determination of the nature of the arthritis.

Acute Arthritis.—An acutely inflamed joint may contain turbid serum, blood-stained fluid, or pus. As a rule the amount of injection is remarkably slight, the synovial membrane being swollen and œdematous rather than discoloured. The articular cartilages undergo superficial fibrillation or necrosis, and in some cases the subjacent bone may be invaded. As the result of inflammatory softening of the ligaments, the joint may become deformed or dislocation occur. Pus which has perforated the joint capsule may spread in the connective tissue planes around. Acute arthritis may be part of a general infection, result from a perforating wound of the joint, or be due to direct extension of disease from an adjacent bone or epiphysis. In order to clear up the nature of the case a bacteriological examination is often essential; the infecting agent may be found in the synovial fluid, or, more certainly, in the synovial membrane itself.

The presence of puerperal infection, gonorrhœa, otitis media, pneumonia, or specific fevers such as typhoid or scarlet, should not be overlooked.

In **acute rheumatism** the affected joints contain an effusion which is serous, but in some cases may be blood-stained or turbid. A plastic exudation is sometimes found on the synovial membrane. Valvular disease of the heart, myocarditis, pericarditis, pleurisy, cloudy swelling

of the liver and kidneys, enlargement of the spleen, etc., may be found, but are also produced by other infections.

Tuberculous Arthritis.—Tuberculosis may occur primarily in the synovial membrane or be secondary to tuberculous bone disease.

In the primary synovial form there is a diffuse pulpy swelling of the synovial membrane which tends to fill up the joint cavity, infiltrate the ligaments, and grow over the articular cartilages. Tubercles in various stages and even caseous deposits may often be recognised. The cartilages, where encroached upon by the granulations, become pitted or perforated; separation may even occur. The subjacent bone may become carious from tuberculous invasion.

Secondary tuberculous arthritis is often set up by foci in the adjacent bone, the joint being invaded by tuberculous extension near the point of reflexion of the synovial membrane or by actual perforation of the articular cartilage.

Tuberculous joints sometimes contain an abundant serous exudate, and melon-seed bodies may also be present.

Periarticular abscesses and sinuses are not uncommon in cases of some standing.

Rheumatoid Arthritis.—This is a progressive poly-arthritis, usually symmetrical in distribution. The disease begins in the synovial and periarticular tissues giving rise to smooth, spindle-shaped swellings. The synovial membranes are thickened. There is but little effusion into the joint cavities. There is practically no proliferation of bone or cartilage, consequently osteophytic outgrowths are absent or insignificant. Eventually all the articular and periarticular structures may undergo atrophic changes, the joints becoming wasted and contracted.

Osteo-arthritis.—In osteo-arthritis inflammatory changes appear first in the articular cartilages and bones. The cartilages undergo a vertical fibrillation which causes them to assume a dull, velvety or slightly roughened appearance, and finally leads to the destruction of their central portions. The exposed bone becomes furrowed and eburnated. At the margins of the cartilages lipping and nodular excrescences occur leading to osteophytic

outgrowth. If the articular ends of the bones be sawn through their consistence may be found unduly spongy and much fat present.

The synovial membrane becomes hypertrophied, its fringes enlarged, and their villous projections may become detached to form loose bodies. Ultimately a mucoid or hyaline degeneration occurs. Synovia as a rule is deficient rather than increased.

Gouty Arthritis.—The distinguishing characteristic of a gouty joint is a deposit of urate of lime and soda in the substance of the articular cartilages. Opaque white patches are thus produced. Uratic deposits may also occur in the synovial membranes, periarticular bursæ and surrounding connective tissues. (Associated changes are granular kidneys, arterio-sclerosis, hypertrophied heart, chronic inflammations of the serous sacs, the presence of tophi in the cartilages of the ears, and sometimes also uratic deposits in the valves of the heart.)

Syphilitic Arthritis.—Either diffuse infiltration or gummata of the synovial membrane may occur as the result of syphilis. A symmetrical diffuse arthritis is sometimes seen in congenital cases.

Neuro-arthropathies : Trophic Disease of Joints.—These arthropathies occur more particularly in association with locomotor ataxy and syringomyelia. They may be characterised by rapid erosion of the articular surfaces, considerable effusion into the joint cavity, hypertrophy of the synovial fringes, and the presence of loose bodies. New bone formation sometimes occurs in the joint capsule, ligaments, and surrounding tissues. Stretching and destruction of ligaments lead to loose, flail-like joints. Spontaneous fractures of the porous and attenuated bones are not uncommon.

Loose Bodies in Joints.—These are of various kinds. They are usually free, but sometimes pedunculated. The commoner contain cartilage cells and arise in synovial fringes and mostly occur in osteo-arthritis. Other varieties of loose body are masses of blood clot, detached semi-lunar cartilages, fringes of tuberculous synovial membrane, and, very rarely, exfoliated portions of bone.

EXAMINATION OF THE MUSCLES AND TENDONS

Many of the muscles of the neck, thorax, and abdomen are exposed during the routine removal of the viscera, and are available for examination.

The muscles of the limbs may have to be exposed by special incisions, which should be planned so as to cause as little disfigurement as possible. These incisions should, as a rule, be parallel to the long axes of the limbs.

Localised Myositis may arise in the course of syphilitic or tuberculous infections. Syphilitic myositis may assume a gummatous form or occur as a diffuse infiltration of the connective tissue of muscle, leading to fibroid changes.

Gummata of muscle are usually multiple. They are circumscribed and nodular. On section they appear greyish yellow or caseous. Suppuration or necrosis may occur.

Polymyositis is a rare disease of obscure causation, it is probably infective in origin. The muscles affected show marked naked-eye changes, their normal red colour may be changed to a dirty yellowish white, mottled with red patches. The muscular tissue becomes friable and hæmorrhage may occur into it. Subcutaneous œdema and purpuric or morbilliform rashes may accompany the disease.

Suppurative Myositis may arise as a blood-borne infection in pyæmic conditions, or be due to invasion from contiguous foci of disease in the skin, subcutaneous tissue, joints, bones, etc.

Myositis Ossificans.—The formation of bony plates in the muscles is characteristic of this rare disease. It begins as a cellular infiltration of the connective tissue. The muscles affected are those of the back, neck, and the thorax.

Rupture of Muscle.—When rupture of muscle is found the presence of antecedent myositis should not be overlooked. Undue pallor, friability or œdema of the muscle may be present. A hæmatoma, which may

suppurate, usually occurs at the site of the laceration. Muscular degeneration, such as occurs in febrile diseases like typhoid, predisposes to rupture. The recti abdominis and adductors are the muscles usually affected.

Rupture without preceding disease of the muscles may occur from violence, during parturition, and in convulsive diseases or tetanus.

Muscles in a state of rigor mortis are sometimes ruptured by too forcible manipulations of the limbs after death. In such cases, although the muscles are often fatty, there is no hæmorrhage and no sign of antecedent inflammation.

Ischæmic Paralysis.—The term Volkmann's Contracture is applied to an ischæmic paralysis which involves the muscles of the forearm and is attributed to splint pressure. In the later stages of the disease the muscles are found to be wasted, firm, pale and dry. They appear mostly to be composed of fibrous tissue. The fingers, and maybe the wrists, are contracted. Scars of antecedent splint sores may be present.

Ischæmic Gangrene of Muscle.—If a muscle is completely deprived of its blood supply in consequence of embolism or thrombosis, the muscular tissue rapidly undergoes necrosis, becoming dark grey in colour and very friable.

Atrophy of Muscle will call for examination of the spinal cord, motor nerves, joints and muscle fibres.

Muscular Dystrophies (Idiopathic Muscular Atrophy, Pseudohypertrophic Muscular Paralysis).—In these diseases the muscles alone show changes, no lesions being found in the spinal cord, nerve roots, or peripheral nerves. The affected muscles are pale and translucent, being converted into masses of fat, which replaces the fibres. Some of the muscles, though wasted, may present a firm and hard appearance. In cases of some standing, various deformities and contractures occur.

Trichinosis.—Voluntary muscles infected by trichina embryos contain minute white specks, slightly elongated in the direction of the muscle fibres and distinctly visible to the

naked eye. On examination with a lens the specks are resolved into trichina cysts. The cysts are most abundant in the abdominal muscles, pillars of the diaphragm, intercostals, and muscles of the neck and larynx. They may undergo retrograde changes.

Cysticercus Cellulosæ.—It is exceptional for the cysticercus stage of a tapeworm to occur in man. The small cysts, which are quite easily visible as minute bladders, lie between the muscle fibres. The head of the worm appears as a minute white spot on the cyst wall.

Hydatid Cysts sometimes grow in the intermuscular planes of connective tissue.

Tumours of Muscle.—Tumours which arise in muscle belong to the connective tissue class and are fatty, fibrous, or sarcomatous. They originate in the connective tissue planes and not in the muscle fibres.

Carcinoma of muscle is always secondary, arising either in contiguity, as when the pectoral muscle is invaded from the breast, or by metastasis.

Teno-Synovitis may be acute or chronic. The acute suppurative form arises from direct infection or as a pyæmic manifestation. Necrosis of the tendon may result. Chronic teno-synovitis is often tuberculous and is more frequently seen in the wrist and hand than elsewhere. The infected tendon sheaths may be lined with tuberculous granulation tissue and similar tissue may ultimately infiltrate and destroy the enclosed tendons. Or the tuberculous infection may give rise to a chronic watery teno-synovitis (hydrops) with melon-shaped bodies in the sheath.

Gummata sometimes develop in tendon sheaths.

CHAPTER VIII

EXAMINATION OF THE SENSE ORGANS

EXAMINATION OF THE NASAL CAVITIES AND AIR SINUSES

As a preliminary the brain is removed.

The scalp must be thoroughly reflected from the occipital bone behind and from the frontal bone well down to the root of the nose.

The posterior flap of the scalp is divided vertically in the mid-line, the incision being carried down to the nape of the neck.

A wedge-shaped piece is next sawn out of the occipital bone, the blunt apex of the wedge being at the foramen magnum. The loose fragment of bone is turned down or detached.

A small back-saw is now applied to the base of the skull in a postero-anterior direction a little to the right of the mid-sagittal plane. The following parts should be sawn through: the vertical plate of the frontal bone, the horizontal plate of the ethmoid, the body of the sphenoid, and the basilar portion of the occipital. Care is necessary to avoid wounding the integuments of the forehead, which should be well reflected and held away from the teeth of the saw. The track of the saw traverses the frontal air sinuses, the sphenoidal sinus, and the roof of the nasal cavity. It is no disadvantage to carry the saw deeply near the occipital foramen so that the anterior arch of the atlas is severed.

The mucous membrane of the roof of the nasal fossa is divided by passing a fine-bladed scalpel along the track of the saw. The two halves of the skull are next wrenched asunder (Fig. 19). Their separation may be rendered more complete by cutting through the hard palate in an antero-posterior direction with a chisel and by dividing the symphysis menti from within outward.

Examine the frontal sinuses, exposing them further by chipping away their inner walls with bone forceps.

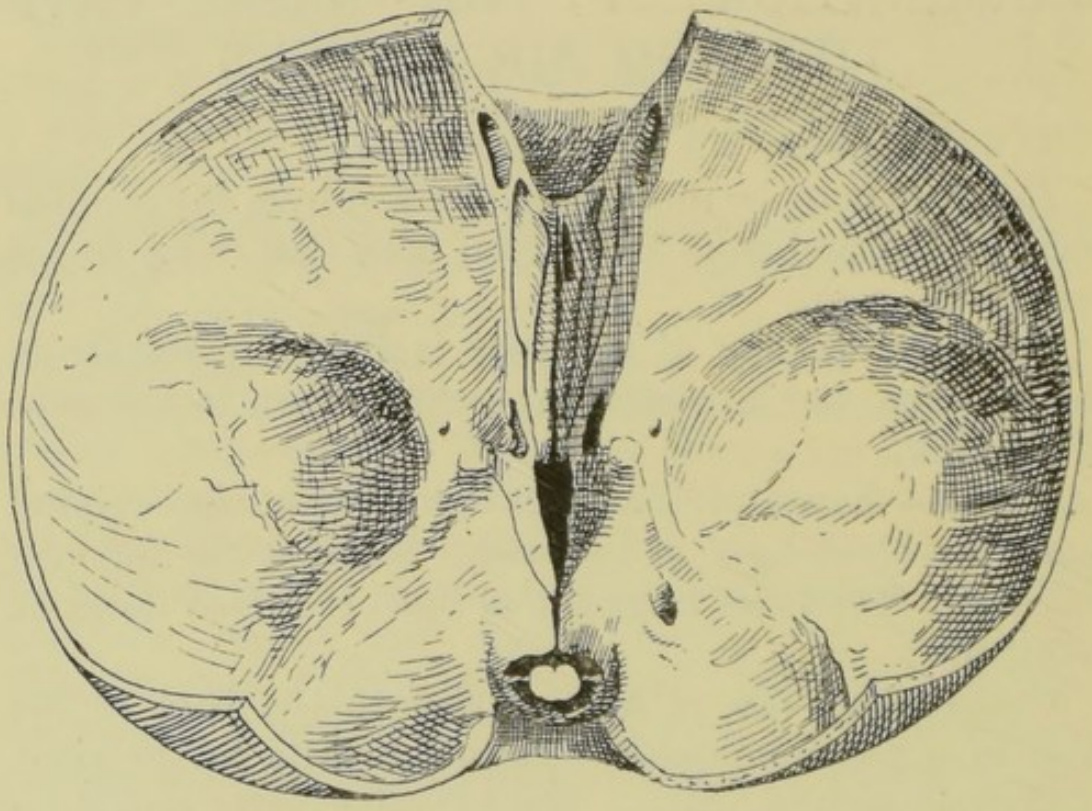

Fig. 19.—Exposure of the Nasal Fossæ and Air Sinuses of the Skull.

Examine the sphenoidal sinuses by cutting through the dorsum sellæ at its base, extracting the pituitary body, and then removing the roof of the sinuses with bone forceps.

Examine the nasal cavities. The section having been carried through to the right side of the nasal septum, the latter should carefully be removed with cutting forceps or strong scissors so that both nasal fossæ may be inspected.

Examine the Maxillary Antrum. This may be exposed

by removing the thin bony septum which shuts it off from the nasal cavity. Another method is to evert the upper lip, reflect the muco-periosteal covering of the canine fossa, and remove the outer bony wall of the sinus with chisel and forceps.

Deflections of the Nasal Septum may cause narrowing of the nasal cavity on one side, usually the left.

Rhinitis, acute, chronic, or hypertrophic is detected by examination of the mucous membrane, particularly of that covering the turbinated bones.

Membranous Rhinitis is usually, but not invariably, diphtheritic. The condition of the throat and air passages may throw light on its nature. A bacteriological investigation may be necessary.

Tuberculous Rhinitis is rarely primary, being as a rule secondary to tuberculosis of the lungs or larynx. It may present itself in the form of granulomatous swellings or grey ulcers, usually on the septum nasi.

Lupus of the Nose is usually an extension from lupus of the face. It may be primary. Pale lupus nodules or ulceration may be detected.

Syphilis of the Nose.—Primary, secondary or tertiary lesions may occur. The common form is gummatous ulceration leading to extensive destruction not only of cartilage, but also of bone. Large bony sequestra when found in the nose are usually due to syphilis. Perforation of the cartilaginous septum is often due to other causes than syphilis.

Glanders.—The nasal cavities may be the site of infection in glanders or be involved secondarily. In the latter case, the nasal mucosa may be studded with small ulcerating lesions, obviously recent, whilst older lesions occur in the lungs (*q. v.*) or elsewhere.

Leprosy may involve the nose.

Nasal Polypi.—These are the products of inflammation of the nasal mucous membrane. They may be œdematous and soft or, if older, of a fibrous consistence. The subjacent bone (usually the ethmoid) should be examined for evidence of necrosis. The mucous membrane of the turbinals, if hypertrophied, may assume a polypoid appearance.

Malignant Disease in the Nasal Cavity is more likely to be sarcomatous than carcinomatous.

Suppuration of the Accessory Nasal Sinuses may involve one or more of these cavities. The infection may have spread from the nose, or, in the case of the maxillary antrum, sometimes from the fang of a tooth. Perforation of the posterior wall of the frontal sinus or of the roof of the sphenoidal or ethmoidal sinuses may lead to intracranial infection in the form of meningitis, venous thrombosis, or cerebral abscess.

EXAMINATION OF THE EAR

Complete examination of the interior of the petrous bone is necessary in all cases of intracranial suppuration, sinus thrombosis, temporo-sphenoidal or cerebellar abscess, as well as in cases where ear disease or deaf-mutism may have existed.

After removal of the brain the surfaces of the petrous bone should be inspected, particular attention being paid to the tegmen tympani, the tegmen antri, the internal auditory meatus, and the aqueduct of the vestibule. Discolorations of the dura covering of the bone, and fistulous tracts or stalks of adhesion to the under surface of the temporal lobe or the fore part of the cerebellar hemisphere, should be investigated. The presence of temporo-sphenoidal or cerebellar abscess or of inflammation of the meninges of the brain and of the intracranial sinuses should not be overlooked.

The dura must be stripped off and the adjacent bone further examined.

As a general procedure the ear is opened *in situ* by the following method: A vertical sagittal incision is made in the upper surface of the petrous bone, a chisel being applied for this purpose just external to the eminence usually, but erroneously, stated to correspond to the superior semicircular canal. The chisel is again applied, this time horizontally at right angles to the anterior surface of the bone, to make a second incision which joins the former

at an obtuse angle. A wedge-shaped piece of bone is thus loosened, and should be prised back, exposing the tympanum and ossicles; the facial nerve also may in part be drawn out of its canal.

It is well to bear in mind that the tegmen tympani lies quite half-way along the anterior surface of the bone as measured from base to apex, and nearer to the anterior than the superior border, whilst the tegmen antri lies just external to the eminence already mentioned.

The mastoid process should be examined by reflecting the scalp and removing the outer part of the bone with chisel or saw.

If a more detailed examination of the ear is desired the whole petrous bone should be removed and then examined by sections, or microscopically, after decalcification.

The removal entire is effected as follows:

The bi-mastoid incision made for reflexion of the scalp should be prolonged along the anterior edge of the trapezius muscle half-way down the neck. The anterior scalp flap and the auricle should be dissected well forwards, the cartilaginous meatus being severed from the bone. The posterior flap is dissected back in a similar manner. The petrous bone is then loosened from above by two saw-cuts which converge towards the apex of its pyramidal portion or to the foramen magnum. The anterior cut should pass through the side of the skull near the root of the zygoma and the posterior pass down behind the mastoid process. A wedge is thus cut completely out. This is liberated from the soft parts beneath and loosened by a blow with the mallet.

After removal, the bone may be examined as follows: Carefully chip away the tegmen tympani and examine the middle ear. Remove the anterior and lower walls of the external bony meatus to expose and inspect the outer surface of the membrana tympani. Then, with as fine a saw as possible (fret-saw), divide the petrous bone from below, following a line between the posterior edge of the styloid process and the anterior edge of the orifice of the carotid

canal. The section should be made parallel to the upper anterior border of the bone and should expose the antrum, tympanum and Eustachian tube. Other sections may be made to expose the labyrinth.

The mucous membrane of the middle ear should be pale, smooth and moist. The nature of the tympanic contents (mucus, pus, blood, granulation tissue) should be noted.

The middle ear of the fœtus is filled by an embryonic gelatinous mass. This is replaced by air when respiration has been satisfactorily established, so that the presence of a distinct cavity in the middle ear is held to be proof that an infant survived its birth.

The ossicles should always be sought for and recognised. The stapes may be adherent to the piece of bone removed in opening the tympanum by the first method described. The presence of pus in the labyrinth should not be overlooked.

It may be desirable to make a bacteriological examination of the contents of the middle ear or labyrinth.

Acute Otitis Media.—The mucous membrane of the tympanum, Eustachian tube and mastoid antrum is greatly swollen, reddened and vascular. A mucous, muco-purulent or hæmorrhagic exudate fills up the cavities. The membrana tympani may be perforated.

Chronic Suppurative Otitis Media.—The mucous lining is infiltrated and thickened, or replaced by spreading granulation tissue which may partly fill the tympanic cavity. The secretion in the tympanum varies in its characters. The membrana tympani becomes opaque. Perforations or scars may be present in it. The lumen of the Eustachian tube may be obliterated. Polypi may be present. Mastoid disease, sclerosis of bone, caries, necrosis, etc., may occur as complications; also extension of infection to the meninges, sinuses, and brain.

Chronic Non-suppurative Otitis Media.—The mucous lining becomes smooth, dry, white, and thick. Fibrous bands may bind down the ossicles in various positions. The membrana tympani is indrawn and thickened.

Mastoiditis.—Mastoid inflammation is usually due to extension from the tympanum, but primary tuberculosis and osteomyelitis are not unknown.

An inflamed mastoid may contain circumscribed collections of pus in its interstices, or a large cavity filled with granulation tissue, pus and carious bone may be found. Infection of the dura or of the lateral sinus may occur.

Dense sclerosis of the mastoid results from chronic inflammation.

Labyrinthitis.—Inflammations of the labyrinth may be primary or secondary in origin. Secondary inflammations are the result of suppurative otitis media. In such cases pus may be found in the labyrinthine cavities, and may find its way into the internal auditory meatus, the aqueduct of the vestibule, etc.

The internal ear may also be involved in leukæmia (leukæmic or hæmorrhagic exudation), and in syphilis, either congenital or acquired.

Caries and Necrosis.—These may result when inflammatory disease of the aural cavities spreads to bone. Common sites are the mastoid process, the posterior wall of the auditory canal, the tegmen tympani, and the spongy bone surrounding the dense capsule of the labyrinth. Sequestra may form, comprising the core of the mastoid or portions of the internal ear. The tympanic ossicles may also be carious. Carious processes involving the carotid canal may lead to perforation or to thrombosis of the carotid artery.

Fractures of the Temporal Bone.—Fractures of the base of the skull may involve the petrous bone, which usually breaks at its weakest part, i.e. in a line through the jugular foramen and tegmen tympani or tegmen antri. The capsule of the labyrinth usually escapes. The membrana tympani may be lacerated. Hæmorrhage may occur into the middle or the internal ear in such cases.

Deaf-mutism.—Malformations of the organ of hearing should be looked for, such as absence of the labyrinth, occlusion of the fenestræ, malformations of the ossicles, etc. The auditory nerve or temporal lobes of the brain may be defective.

Acquired deaf-mutism is due to auditory disease occurring during meningitis, or the specific fevers. In such cases evidence of past otitis may be present.

Cholesteatomata (*Pearl Tumours*).—These tumours are epithelial masses mixed with cholesterin, pus, and micro-organisms.

They may occur in cases of old-standing suppurative middle ear disease, rarely as new formations without antecedent infection.

The cholesteatomatous masses have a glistening appearance and are arranged in concentric layers. They may give rise to considerable enlargement of the cavities of the middle ear.

Aural Polypi.—Polypoid granulations arising in the tympanic cavity often depend on disease of the bony boundaries. True fibromata or myxomata may also occur.

EXAMINATION OF THE ORBITS AND EYES

Removal of the entire eye is undesirable for æsthetic reasons. As a general rule removal of the posterior half of the globe is sufficient. Should complete enucleation be unavoidable an artificial eye should be provided to replace the one removed.

Before opening the orbits make an inspection of the eyelids, conjunctivæ, corneæ, and pupils, also of the integuments which cover the lacrymal sacs.

Notice any undue prominence, recession or asymmetry of the globes. Examine the eyelids for bruises, swellings or inflammation.

Inspect the ocular and palpebral conjunctivæ for pigmentations, hæmorrhages, signs of inflammation, ulceration, abnormal leashes of vessels, etc.

Pay attention to the degree of transparency of the corneæ and to the presence of arcus senilis, ulceration or opacities. Do not mistake the filminess and opacity of the corneæ, which comes on after death, for disease. Observe

the size and shape of the pupils. Discolorations, defects, adhesions or irregularities of the irides may be present. Note the colour of the eyes.

The nature of the contents of the anterior chamber should be determined. Pus or even blood may sometimes be seen.

Opacities of the lens (cataract) may, if present, be obvious on examination through the pupil.

The interior of each orbit should be exposed by removing the orbital plate of the frontal bone and the lesser wing of the sphenoid. This removal may be effected by clipping away the bone from behind forwards with bone forceps or by gentle use of the chisel and mallet. In either case the incisions made should lay open the optic foramen and diverge from this, one passing forwards and outwards in the direction of the external angular process, the other forwards parallel to the cribriform plate of the ethmoid. The anterior ends of the incisions should be united by a cut parallel to the vertical plate of the frontal bone.

Having ascertained the condition of the orbital fat and veins, and recognised the lacrymal gland, proceed to remove the posterior half of the globe with the optic nerve attached. Open the capsule of Tenon and expose the globe by dissection, tracing the optic nerve backwards, observing at the same time any distension of its sheath. When the nerve is free, make steady traction on it to draw back and steady the eyeball. Incise the latter transversely through all its tunics, a little behind its equator, with a sharp scalpel. Continue the incision round the globe with scissors, thus detaching the posterior half and optic nerve from the anterior portion, which should be left *in situ*.

Whilst making the incision the vitreous will escape, often carrying with it the lens. Both should be carefully inspected for discolorations or opacities.

During the removal of the eye the retina often becomes detached and wrinkled. It should be floated back in place by grasping the optic nerve between finger and thumb and submerging the posterior half of the globe, nerve undermost,

in a basin of water. Then by gently everting the edges of the sclerotic, so that this structure is practically turned inside out, the retina may be induced to fall smoothly over it and removed from the water in a position for examination. It should be inspected with a hand-lens.

The choroidal pigment is easily dislodged, so it should be touched as little as possible, but it is an advantage, when searching for miliary tubercles in this tunic, to brush off the choroidal pigment after first removing the retina.

Examination of the interior of the eyeball during the progress of an autopsy is necessarily very imperfect. In every case where a minute investigation is necessary the eyeball should, if possible, be removed entire and preserved in strong formalin solution, so that it may be examined properly.

DISEASES OF THE ORBITS AND EYES

Osteoperiostitis of the Orbit.—This may occur in an acute or a chronic form. In the former suppuration and bone necrosis are common. The eyeball may be protruded and the lids swollen. A diffuse abscess may form between the periosteum and the bone. In a case of orbital periostitis, the condition of the nose and accessory air sinuses should be investigated. The occurrence of punctured wounds of the orbit should not be overlooked.

Cellulitis in the Orbit usually terminates in suppuration. Proptosis and lid swelling may be present. Infection arises in various ways. The condition of the nose, accessory air sinuses, maxillary antrum, and teeth should be ascertained. Fractures of the orbital walls or punctured wounds should not be missed. The condition of the eyeball, orbital veins, and cavernous sinuses should be investigated.

Tumours of the Orbit.—Solid tumours may originate in the orbit or invade it from neighbouring structures, such as the ethmoidal air sinuses, the maxilla, or the eyelids. The new growths may be sarcomatous or carcinomatous. Chloromatous growths may infiltrate the orbital periosteum

and cause proptosis. The lacrymal gland is sometimes the seat of tumours. A microscopical examination of tumours is always requisite.

Exostoses, either spongy or compact, favour the roof and nasal boundary of the orbit, possibly because they tend to originate in the frontal and ethmoidal air sinuses.

Other orbital tumours are Meningoceles, which present at the suture between the ethmoid and frontal bones; Dermoid Cysts; Parasitic Cysts (Hydatid); and Cavernous Angiomata.

Proptosis or Exophthalmos may be due to the presence in the orbit of new growth, abscess, or aneurysm. It may also be caused by thrombosis of the cavernous sinus, or by arterio-venous communication between the cavernous sinus and the carotid artery. Displacement of the eyeball is sometimes produced by bony deformity of the orbit. In exophthalmic goitre the exophthalmos is usually bilateral.

Enophthalmos.—Retraction of the globe may be caused by a lesion of the cervical sympathetic, or be due to wasting of the eye from destructive inflammation.

Congenital Defects of the Eyelids, such as cleavage (coloboma), epicanthus, congenital ptosis, dermoid cysts, etc., if present, will be obvious.

Emphysema of the Lids may be caused by fractures which involve the nose or accessory air sinuses.

Hæmorrhage into the Lids may be local and due to direct violence, or may have extended forwards in injury to the base of the skull; in the latter case subconjunctival hæmorrhage is usually present in addition. Discoloration of the lids may also be due to scurvy or other blood diseases.

Conjunctivitis.—Acute conjunctivitis is indicated by injection or purulent secretion. Membranous conjunctivitis is not always diphtheritic; a culture should be made.

Corneal Inflammation may occur in the form of ulceration, localised white patches, or more diffuse opacities. Where diffuse keratitis is present the teeth and the shins should be examined for evidence of congenital syphilis.

Examination of the interior of the eyeball is practically limited to a search for evidence of retinitis, optic neuritis, tubercles of the choroid, opacities of the lens, or new growths.

Retinitis.—The white spots of exudation and the hæmorrhages may be recognised with a hand-lens. Particular attention should be paid to the region of the macula lutea. The condition may be renal or diabetic.

Papillitis.—When optic neuritis is present it may be possible to detect a considerable blurring of the optic disc. Retinitis may coexist (neuro-retinitis). In obscure cases the condition of the sphenoidal air sinuses which lie adjacent to the optic nerves should be investigated.

Glaucoma.—In this condition, the intraocular tension being raised, the disc may be distinctly cupped.

Tuberculosis of the Choroid.—Miliary tubercles may occur in the choroid. They are best seen by brushing away the choroidal pigment after removal of the retina.

Malignant Intraocular Growths.—These may be gliomatous or sarcomatous. Gliomata are of retinal origin. Sarcomata arise in the choroid and are melanotic. Carcinomatous growths arising from glands in the ciliary body are great rarities.

CHAPTER IX

POST-MORTEM EXAMINATION OF THE NEWLY BORN

It is necessary to conduct the examination of the body of the newly born infant in such a way as to determine the following points :

1. The degree of maturity of the child.
2. The establishment of respiration.
3. The occurrence of live birth.
4. The cause of death.
5. The period elapsed since death.

The examination should be systematically carried out in the manner indicated in this chapter, but for the bearings and value of the evidence obtained a standard text-book on Medical Jurisprudence should be consulted.

EXTERNAL EXAMINATION

Ascertain the sex and weight of the body.

Measure the length from vertex to heels and also the transverse diameters of the shoulders and hips.

Determine approximately the age.

Make a note of any peculiar marks, such as nævi, malformations, or deformities, which may aid in establishing identity.

Examine for the presence of body warmth and rigor mortis (the presence of the latter is not in itself a proof of live birth since, under certain conditions, it may come on *in utero*).

Note whether there are present any marks of putrefaction,

such as offensive odour, discoloration of the skin, or peeling of the cuticle.

Then proceed to make a more particular inspection from head to foot.

Examine the head for the presence of marked moulding or caput succedaneum. Note the size of the fontanelles and the condition of the hair.

Determine if the eyelids are adherent and whether a pupillary membrane is present. Inspect the mouth and nostrils, also the ears for the presence of foreign matter. Ascertain whether the vernix caseosa is still present, particularly about the groins, armpits and neck, or has been removed.

Pay attention to the position of the umbilicus with regard to the body and the condition of the umbilical cord.

(In the full term child the cord is inserted somewhere below the centre of the abdomen. It falls off about the fifth or sixth day after birth.)

The presence of suppuration in the navel should not be overlooked. The free end of the cord should be examined to ascertain whether it has been cleanly cut or torn; ragged edges and irregular tapering on one side afford evidence of rupture. If the cord has become desiccated, examination is facilitated by softening the end in water.

Ascertain the length of the finger and toe nails with regard to the finger and toe tips.

In boys determine the position of the testicles and condition of the scrotum.

In girls note any peculiarities of the external genitals.

Apart from decomposition, look for any exfoliation of the cuticle in scales or powder such as normally occurs on the first or second day after birth.

In every case the position and appearance of any scratches, abrasions, bruises, wounds or burns should be ascertained. A caput succedaneum should not be mistaken for evidence of injury to the head inflicted after birth.

Cord or finger marks on the neck, which may indicate strangulation, call for careful examination of the deep as

well as of the superficial structures in their neighbourhood. If the neck has been constricted by the umbilical cord discoloured impressions may be left but are free from abrasions, the cord being smooth. In such cases signs of respiration are absent as a rule.

INTERNAL EXAMINATION

Examination of all the viscera, including the central nervous system, should be carried out systematically in every case.

The primary incision is carried from the point of the chin to just above the umbilicus. At the latter point it should bifurcate and run towards the anterior superior iliac spines. The left limb of the lower part of the incision is made first in order that the umbilical vein may be inspected before it is divided.

Make the vein prominent by traction and trace it towards the portal fissure. Open it up as far as possible to determine the nature of its contents. Do not overlook suppuration around it.

When the vein has been examined complete the right limb of the incision and turn down the triangular flap of abdominal wall.

Examine the urachus, which lies on the deep surface of the flap in the mid-line, and the umbilical arteries, which converge towards the navel from each side.

Make the usual preliminary inspection of the abdominal viscera.

Before opening the thorax determine, by bimanual palpation, the height of the diaphragm with regard to the overlying ribs. To effect this introduce the index finger into the dome of the muscle and ascertain its level externally by palpation with the other hand. If the lungs do not contain air, or are only partially inflated, the diaphragm of the infant reaches up to the fourth rib, but when the lungs are fully inflated it is depressed to the fifth or sixth rib on

the right and to the sixth rib or intercostal space on the left.

Reflect the integuments from the front of the thorax and continue the chin incision upwards in the mid-line through the integuments and lower lip. Divide the symphysis menti with scissors and retract the halves of the mandible. An unobstructed view of the mouth and pharynx is thus obtained without displacing any foreign body which may be present. Note whether the tongue is rolled back into the fauces, as suffocation sometimes occurs from this cause.

The tongue, fauces, palate, and cervical viscera are liberated and drawn forwards in the ordinary way. A ligature should be placed around the trachea, above the manubrium. The thorax is next opened by cutting through the costal cartilages, near the ends of the ribs, with scissors.

Remove the manubrium and ascertain the general appearance of the thoracic contents. The degree of inflation of the lungs is gauged by noticing their positions, the extent to which they cover the pericardium, their colour and consistency.

It may at once be evident from their expanded state and 'marbled' appearance that inspiration has occurred. If the lungs appear as dark bluish or violet organs, firm, airless, and lying far back in the pleural cavities, so that the thymus gland and pericardium remain in full view, then respiration has not taken place. The absence of pulmonary inflation does not necessarily prove that the child was born dead.

The pericardium should be incised and the external appearance of the heart ascertained.

The mass of thoracic viscera is now removed from the thorax.

Examine the palate, fauces, pharynx, and œsophagus by opening them up in the mid-line posteriorly. Inspect the laryngeal aperture, but do not open the larynx or trachea at this stage.

Dissect out the thymus gland, remove and weigh it.

Before separating the heart incise the right ventricle near its apex and slit up the anterior ventricular wall and pulmonary artery from below. The orifice of the ductus arteriosus will be seen between and beyond the openings of the right and left pulmonary branches. Its patency can be investigated with a probe.

The detailed examination of the heart should now be carried out in the same way as in the adult.

Next apply the hydrostatic test to the lungs. Divide the trachea above the ligature and immerse the lungs with the portion of the air passages which still remains attached in a vessel of clean, cold water. Notice whether they float, and if so whether flotation is easy, with the lungs high above the surface, or indifferent. If they sink the lungs should be tried separately.

Lay open the trachea and bronchi from behind and examine their contents (blood, meconium, etc.). Incise each lung and notice the presence or absence of crepitation, the amount and appearance of any blood which may exude on slight pressure, and any indications of pathological consolidation. The lungs may also be incised under water; any bubbles which escape from the cut surface will then be noticeable.

Complete the hydrostatic test by subdividing each lobe and examining separate portions as to their buoyancy.

In cases where it is suspected that the smaller bronchi or alveoli may have become filled with inflammatory exudation or with foreign matter, such as vernix caseosa or meconium, which has prevented the entry of air, a microscopical examination should be carried out.

Decomposition, by liberating gases of putrefaction, may cause lungs which have not respired to float, hence the importance of noticing the signs of decomposition in other parts of the body.

The abdominal viscera should next be examined systematically and thoroughly as in the adult. The following points are of special importance:

Breslau's test for swallowed air should be applied to the

stomach and duodenum. Place double ligatures on the cardiac and pyloric ends of the stomach and at the duodeno-jejunal flexure. Detach the stomach and duodenum in one piece from the other viscera, place the mass in water and observe if it floats. Cut between the ligatures at the pylorus and observe the separate buoyancy of stomach and duodenum.

Ascertain the nature of the contents of the stomach and intestines. Especially should the presence of frothy (air-containing) mucus in the stomach and of milk or starchy or saccharine matters receive attention. Microscopical examination and the application of appropriate chemical tests is necessary.

If poison is suspected the contents of the stomach and intestines and liberal portions of the solid abdominal viscera should be reserved under seal for analysis. Examine the pelvic viscera and genital organs.

Then examine the head. Inspect the scalp again, especially in the region of the fontanelles, and also the nape of the neck for punctures or other injuries. Reflect the scalp and make further examination of its tissues.

Fractures of the skull, usually of the parietal bone, may occur during childbirth, but will, if occurring under these circumstances, be associated with other signs due to difficult delivery. Defects and fissures of the skull, due to imperfect ossification, must not be taken for signs of violence. These defects, if present, usually involve the parietal bone. Their edges are thin and smooth and there are no signs of injury.

Open the skull along the sutures, as described on p. 220; examine the dura and remove the brain. Examine the brain in the ordinary way. Bruises or hæmorrhage may be present. The spinal canal can be opened by exposing the vertebral arches and dividing them carefully with scissors.

In infants the spinal cord is unduly hard, whereas the brain is very soft.

Open the middle ear carefully, to ascertain whether a

distinct cavity is present. The embryonic gelatinous mass which fills the tympanum disappears after respiration is fully established; it is but rarely wanting in the unborn fœtus.

Lastly, examine the Ossific Centres of the Bones.

Flex the knee and lay open the knee joint by an incision below the patella. Shave off the epiphyseal cartilage of the femur by a series of transverse incisions, or split it vertically. The ossific centre lies in its thickest part and may be recognised as a red gritty patch. It appears in the ninth month of fœtal life and at full term measures from $\frac{1}{10}$–$\frac{1}{5}$ inch across ($\frac{1}{2}$–5 mm.).

The ossific centre in the head of the tibia may be exposed in a similar way. It appears simultaneously with, or shortly after, that in the lower end of the femur.

The ossific centre in the cuboid appears shortly before or shortly after birth.

The presence of an ossific centre in the os calcis is regarded as a *sign of viability*. It appears as early as the sixth month of fœtal life (between the 169th and 210th days).

The ossific centre of the astragalus also appears before birth, but is later than that of the os calcis.

The detection of syphilitic epiphysitis in an infant may have important bearings. Examine the femur by exposing the hip joint by a vertical posterior incision. Divide the ligaments and dislocate the head of the bone. Split the upper end downwards through head and shaft. A narrow gummatous line of a decided yellow colour in the epiphyseal cartilage at its junction with the diaphysis constitutes the specific lesion. The epiphysis may become separated at this line.

The head of the humerus may also be examined, the bone being dislocated forwards from the inside of the reflected thoracic flap to avoid unnecessary mutilation.

It may be necessary to examine also the epiphyses of the wrist and ankle joints. Although the presence of the characteristic epiphyseal lesion may be taken as sure proof of congenital syphilis, its absence does not necessarily indicate that the infant is not syphilitic.

Table showing the Principal Developmental Changes in the Fœtus (after Luff—modified)

Intra-uterine age in Months	Length in Inches	Weight in Pounds	Eyelids	Pupillary Membrane	Nails	Testes	Vernix caseosa	Hair	Ossific Centres
6	9–12	1–2	Adherent	Present	Just forming	In abdomen below kidneys			For manubrium and first piece of sternum. For os calcis. For bodies and laminæ of sacral vertebræ
7	12–16	2–4	No longer adherent	Beginning to disappear	Finger naïls have not yet reached extremities of the fingers	Near the abdominal rings	Vernix caseosa and lanugo present on skin	Appearing on scalp	For second and third pieces of sternum. For astragalus

8	15–18	4-5		Disappeared	Finger nails reach extremities of fingers Toe nails nearly reach extremities of toes	In inguinal canals or upper part of scrotum Left testicle lower than right			
9	18–20	5–8			Nails project beyond tips of fingers and reach extremities of toes	In scrotum		Hair thick on scalp and about an inch long	For lower epiphysis of femur. For cuboid. For first coccygeal vertebra. The fourth piece of the mesosternum and the xiphoid are usually still cartilaginous

The average weight of a new-born full-term child is, male 7½ lbs. (3,310 grms.), female 7¼ lbs. (3,230 grms.).
The lungs, before respiration, weigh about 1½ oz. (42 grms.), and after respiration as much as 2½ oz. (71 grms.) Quain.
The weight of the thymus in the newly born varies from ¼ to ½ oz. (7–14 grms.).
The brain weighs about 13½ oz. (380 grms.).
(The following weights are according to Toma :
Heart, 20·60 grms. ; Spleen, 11·10 grms. ; Kidneys, 23·60 grms. ; Liver, 118 grms.)

CHAPTER X

FINAL CONSIDERATIONS

EMBALMING

The best method of preserving a body for either short or long periods is by arterial injection.

The process must be carried out before the post-mortem examination is made, but chemicals should never be introduced into a body which is likely to become the subject of medico-legal proceedings.

The main essentials for successful injection are two: the vascular system must be intact and rigor mortis should have just passed off. The best time to choose is twenty-four hours after death. The limbs should be tested by flexion and extension to be sure of their suppleness.

The injection may be effected through the femoral artery in Scarpa's triangle, or the abdominal aorta may be chosen. In the former case the nozzle of the injector should be directed towards the heart, in the latter a T-shaped cannula is used so that the fluid may flow in both directions.*

The advantages claimed for the abdominal method are the following: The peritoneal cavity can be emptied and cleansed; fluid collections can be aspirated from the chest through the diaphragm without external puncture; the return flow of the embalming fluid in the inferior vena cava being visible, the vein can be incised and emptied if venous

* In an article published in the *Lancet*, 1902, Dr. J. G. Garson advocated injection through the abdominal aorta, and to this paper those who seek further information on the subject of embalming are referred.

engorgement obstructs the injection. In addition the abdominal incision permits the removal of the stomach and intestines, if this should be deemed necessary for the better preservation of the corpse; the roots of the severed mesenteries should of course be ligatured to prevent the escape of the embalming fluid.

If death has resulted from the rupture of a large artery in the thorax or abdomen, the ruptured vessel should be exposed and the injection performed at the site of the tear.

Rupture of a blood-vessel within the skull does not interfere with the ordinary methods of injection.

When death has occurred from hæmorrhage into the lung, stomach, or other viscus, it may be necessary to remove the diseased organ and ligature its feeding vessels before the injection can be completed.

The presence of dropsy is a great obstruction to satisfactory injection; the dropsical injections should be removed from the serous cavities by siphonage, aspiration or some other means, and the œdema of the limbs reduced by elevation and elastic bandaging or by multiple needle punctures. In such cases the use of formalin as a preservative is preferable to any antiseptics which coagulate albuminous fluids.

If a post-mortem examination as well as embalming is necessary, the preservative injection should be made at least twenty-four hours before the examination.

After a complete post-mortem examination preservation by arterial injection is practically impossible. In such cases other methods must be adopted.

About two gallons of fluid are required for the injection of a full-grown male, but for temporary preservation half this quantity will suffice.

The femoral artery having been laid bare and the nozzle tied in, or the abdominal aorta exposed through a longitudinal incision in the neighbourhood of the umbilicus and the T-cannula inserted, the fluid should be run in by hydrostatic pressure. Or a pressure bottle may be

extemporised from a large jar through the cork of which are inserted a long and a short tube. The long tube should pass to the bottom of the jar and be connected at its upper end with the injection nozzle by pressure tubing. Compression may be effected by a bicycle pump connected to the short tube.

The pressure should be just sufficient to drive the fluid slowly into the vessels. Undue pressure is apt to rupture diseased vessels, particularly in the old. Sudden acceleration of the inflow indicates the occurrence of this accident, and then the success of the injection is greatly interfered with.

It is well to continue the injection until the fluid ceases to flow, then to allow six hours or more for the fluid to diffuse, after which an additional quantity may be introduced.

In addition to intravascular injection some of the fluid should be introduced into the pleuræ, stomach, intestines, urinary bladder and nasopharyngeal cavity.

The first indication that the injected fluid is reaching the vessels of the skin is the appearance of a white mottling. The white spots become confluent, the features fill out, and finally the whole body becomes firm and hard.

After injection is complete the surface of the body and the scalp should be treated with a preservative to prevent peeling of the skin.

For brushing over the surface of the body use a saturated solution of corrosive sublimate in methylated spirit. Dilute this when prepared with 6 or 8 parts of glycerine.

Formulæ for Embalming Fluids

1. Corrosive Sublimate, 2 ozs.; Glycerine, 2 pints; Methylated Spirit, 2 gallons.
2. Liquor Zinci Chloridi (B.P.). The strength of this is 3 grains in 4 minims.

The quantity of chloride of zinc needed is 15 lbs. It may be dissolved in water as in the liquor, or better, in glycerine and spirit in the proportions given for the corrosive sublimate injection.

3. Formalin, 1 pint; Carbolic Acid Crystals, $\frac{1}{4}$ lb.; Water, $\frac{1}{2}$ gallon.

The crystals should be dissolved in the water and formalin added. If formalin alone is used the growth of certain moulds is not inhibited.

RESTITUTION AND TOILET OF THE BODY

It is extremely important that the body should be left in as natural a condition as possible at the conclusion of a post-mortem examination.

No incisions should be made upon the face and no unnecessary mutilation of the extremities practised. The long anterior incision should always stop short of the front of the chin, and in some cases may, with advantage, commence at the top of the sternum. The breasts should never be examined by external incisions, but be explored from the inside of the thoracic flaps; as already indicated, the shoulder joints may also be explored by a similar route.

The eyeballs should be left intact if possible. In cases where the eye must be examined it is, as a rule, sufficient to remove the posterior half of the globe only. The anterior half of the eyeball should be wedged in place by means of a wad of cotton wool impregnated with ink or strong permanganate solution, either of which may be handy. If complete removal is necessary an artificial eye may have to be provided.

The contour of the head should be restored as accurately as possible if the skull has been opened during the necropsy. The brain, being soft, should be placed in the thoracic or abdominal cavity and the cranial cavity filled with closely packed damp tow or a small sand-bag. The latter is

preferable, being weighty, and is easily improvised by pouring a quantity of common sand on a cloth eighteen inches square and then taking up the corners and tying them together with string. When for any reason it is necessary to keep the skull cap, a new calvarium may be fashioned out of plaster of Paris or from stout pasteboard which has been softened in water, moulded on the skull cap, and fitted to the base of the cranium.

An ugly ridge will appear on the forehead, unless particular care is taken to prevent slipping back of the skull cap. The deformity may be prevented by the use of small metal pegs which are driven into the diplöe, or by boring holes near the edges of the bones and wiring or tying the parts together. Another method of maintaining accurate apposition is to cut two transverse notches across the vertex with a saw and secure the bones with twine, which lies in the notches above and is fixed below to the remains of the temporal muscles through which it is carried on a needle.

If the skull cap has been removed by saw cuts which slope from before and behind towards the auricular regions, and the posterior incisions are continued forwards below the points where the anterior cuts meet them, the ends of the incisions thus carried forward will serve as bony slots in which a bandage passing over the vertex may be secured.

The scalp is often stretched during its reflexion, and when replaced has a baggy appearance. The redundant tissue should be removed from its cut edges before stitching is attempted. The stitches should be inserted as described for the abdomen (see next page). Another method of taking up the redundant scalp is to pass the stitches near the edge of the anterior flap but some distance behind the edge of the posterior. Then when they are tightened the scalp will be drawn taut and the skull cap firmly held in place, but a rather unsightly transverse ridge will be formed across the vertex.

All blood and other fluids should be removed from the

thoracic cavity by careful sponging. The thoracic viscera should be replaced as nearly as possible in their natural positions. The residual space is best filled up with fine sawdust (bran or even wood shavings will serve) and a bed thus formed to support the breastbone in good position. The latter may be fixed to the ribs if necessary by wire or sutures.

If the neck has been opened its contour should also be restored by packing with sawdust.

Cleanse and dry the abdominal cavity. If the stomach and intestines have not been opened they should be snipped in various places to avoid gaseous distension. Replace the viscera as naturally as possible and restore the shape of the abdomen by the use of sawdust or other absorbent material.

If the pelvic organs have been removed the pelvis should be stuffed tightly with tow to prevent leakage.

The perineum should be sewn up if it has been divided, or the natural outlets well plugged.

The long anterior incision in the body is closed by the 'glover's stitch.' A packing or large surgical needle armed with twine is used. The twine should be more than one and a half times the length of the incision. Begin the closure at the upper end. Pass all the stitches from the inner surface of the flaps to the outer. They should be about $\frac{3}{8}$ inch away from the cut edges and $\frac{1}{2}$ inch apart; they must alternate in position on the two sides so that the needle-holes on one flap are not opposite those on the other, but midway between them. The cut edges should be pushed in as the stitches are tightened up, so that no fascia or fat shows between the edges. Finish off the stitching with a firmly tied knot. If sawdust is not used in packing the abdomen, some tow, cotton wool or even paper should be placed under the incision to prevent subsequent leakage.

If it has been necessary to remove parts of the spinal column a rod of wood or metal must be inserted in the spinal canal to restore the rigidity, and the cavity further

filled in with plaster of Paris. The posterior incision is closed in the same way as the anterior.

When portions of the bones of the limbs have been removed the deficiency must be supplied. Parts of the shaft of the femur, for instance, may be replaced by insertion of a rod of wood shaved down at its ends, which are inserted into the medullary cavity. All incisions in the limbs must be carefully sewn up with the 'glover's stitch.'

If the testicles have been removed it may be advisable to replace them with dummies.

After completing the closure, the body must be thoroughly washed, particular attention being paid to the hair, nasal orifices, ears and back. No trace of blood or discharge should be left on the corpse.

The clothes should carefully be replaced.

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