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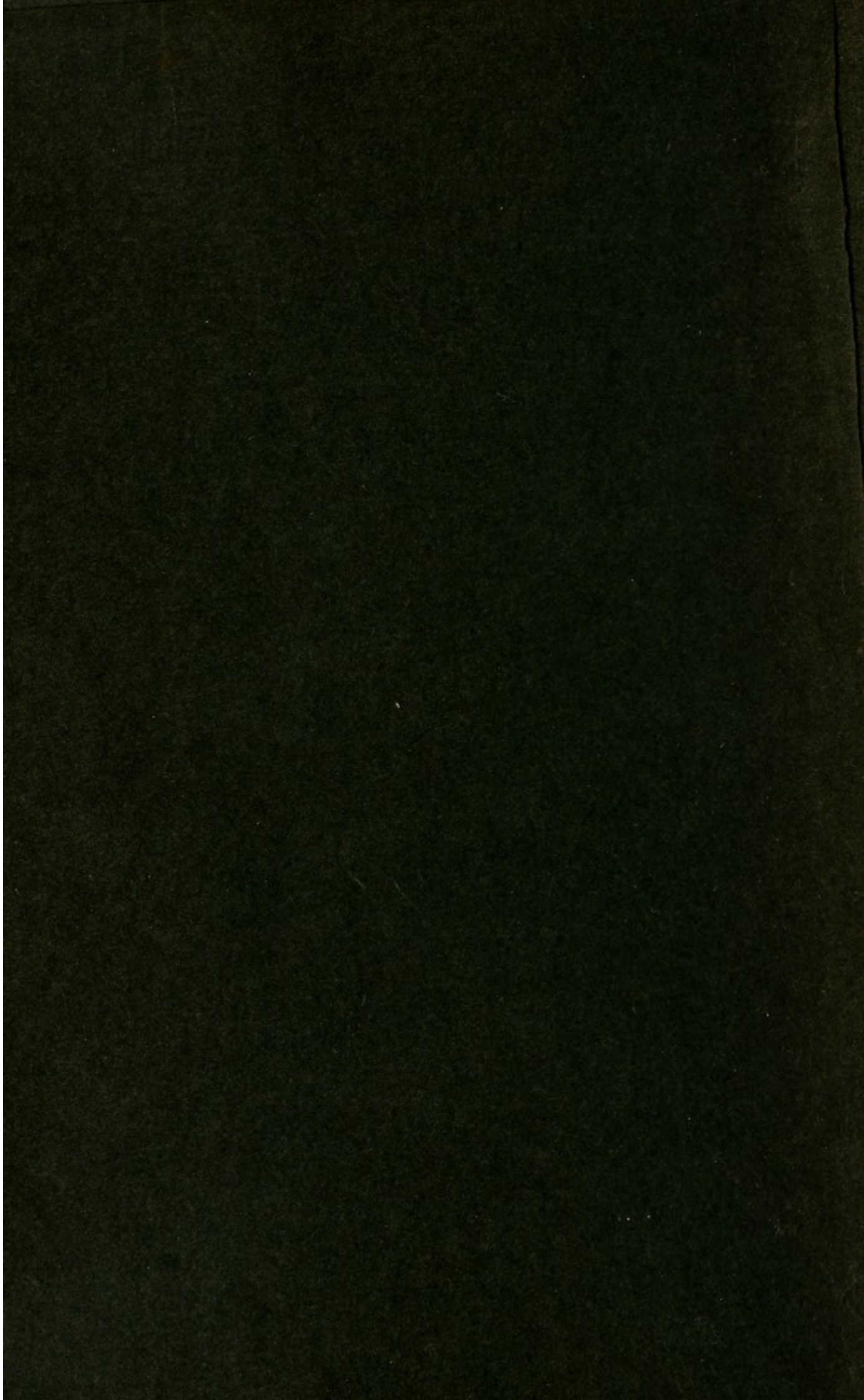
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SURGICAL DIAGNOSIS

A MANUAL

FOR

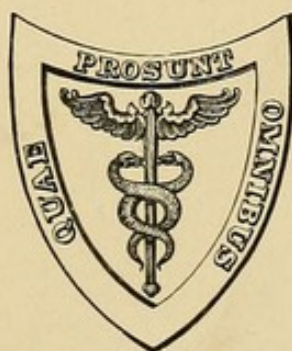
STUDENTS AND PRACTITIONERS

BY

ALBERT A. BERG, M.D.

ADJUNCT ATTENDING SURGEON TO THE MOUNT SINAI HOSPITAL, NEW YORK

ILLUSTRATED WITH 215 ENGRAVINGS AND 21 PLATES



LEA BROTHERS & CO.

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DORNAN, PRINTER

TO THE
MEMORY OF MY PARENTS
AND
TO MY TEACHER AND FRIEND

DR. A. G. GERSTER

THIS BOOK IS
AFFECTIONATELY
DEDICATED



PREFACE.

UNTIL the last few decades the surgeon's efforts were limited to organs lying on the surface and directly accessible. Aseptic methods and improvements in operative technique have meantime brought the internal organs within the range of successful treatment, and this enlargement of the surgical field has necessitated the introduction of new methods of diagnosis and improvements upon the old. In the present volume the author has endeavored to cover the whole subject concisely and in its modern development.

The light that has been thrown upon the early stages of disease-processes by laparotomy and exploratory incision, the close analysis and classification of the clinical manifestations of individual diseases that have been made by those who have the opportunity for observing large numbers of patients, and the aid that the pathological, bacteriological, and chemical laboratories afford for the interpretation of the phenomena of disease have made it possible to recognize most of the maladies that are surgical in character at their incipency. There still remain, however, some diseases which we cannot even to-day diagnosticate early, notably cancer of the internal organs. It is to be hoped that continued exploratory incision and further clinical observa-

tion will soon give the data necessary for the early recognition of these maladies.

The author has endeavored to present surgical diagnosis in a clear and definite way to meet the needs of students and general practitioners. He has also presented the methods of diagnosis of kidney function, the diagnosis of diseased conditions of the kidney from the appearance of the ureteral orifice, the early diagnosis of tuberculous disease of the articular ends of bones, etc., which he hopes will be of especial interest to his colleagues in surgery. Consideration of the best method of developing the subject for his readers has led him first to give a concise clinical picture of each disease, including its causes, onset, and course, and in certain cases the accompanying pathological changes. In each instance he has indicated the points of difference between the disease under discussion and diseases of other organs which might be mistaken for it.

The author takes this opportunity of thanking Drs. Gerster, Lilienthal, and Ware for the privilege of reproducing photographs of their cases; also Drs. Brickner, Foord, and Sternberg, of the *x*-ray department of Mt. Sinai Hospital, for the skiagraphs that are here presented, and Dr. Brenner, of the house staff of Mount Sinai Hospital, for taking most of the photographs. His thanks are likewise due to Drs. Leo Meyer and Milton Gershel for aid in revising the manuscript, and to his publishers for their unfailing courtesy during the preparation of this book.

A. A. B.

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PART I.

GENERAL CONSIDERATIONS ON DIAGNOSIS.

CHAPTER I.

THE EXAMINER—THE TAKING OF THE CLINICAL HISTORY—THE EXAMINATION OF THE PATIENT.

SUCCESS in diagnosis depends upon a careful elicitation of the patient's history and symptoms and upon their logical interpretation. The former demands from the examiner painstaking effort and the sharp use of all his senses; the latter his keenest judgment. Experience trains his discriminating faculties; it teaches him the relative clinical importance of the facts that have been elicited in the anamnesis and examination; it helps him to draw conclusions quickly and accurately. The sharper his senses, the better his judgment, and the wider his experience, so much the quicker and so much the more accurate will his diagnosis be. No diagnosis should rest on intuition; it should be the result of a rapid examination, a keen appreciation of the important symptoms, and their significance.

For the beginner in diagnostics it is essential that the investigation into the history and the examination of the patient be made in a systematic manner. While questioning the patient, the examiner should take note of his general characteristics: the demeanor, the psychical condition (whether hysterical, hypochondriacal, etc.), the accuracy with which statements are made; he should endeavor to dispel fear, or shyness, and to encourage confidence. In eliciting the clinical history, attention should be given to what are apparently

minor facts. No point is so small, no symptom so slight, but that it may have quite an important bearing on the diagnosis.

The following scheme is adhered to in taking clinical histories at Mount Sinai Hospital:

Name, age, and date of admission.

Birthplace and occupation.

The family history, especially in reference to tuberculosis, malignant disease, syphilis.

The previous personal history, habits, and in women the menstrual cycle, pregnancies, and nursing.

The present illness: its duration, and to what it is attributed; its prodromata; its onset, and its course. (In most cases it is necessary to enquire into individual symptoms and bodily functions—e. g., the gastrointestinal, urinary, genital, respiratory, and cardiac functions.)

The chief complaints, and the general appearance, nutrition, and strength.

In the examination of the patient it is likewise necessary to follow a definite systematic plan. The patient should be undressed sufficiently to permit us to make a thorough physical examination. Very sick or feeble patients will be in bed, and in our office the individual may be examined in the erect or recumbent position. The routine examination should determine:

1. *The general condition of the patient; the color of the mucous membranes and conjunctivæ; the facial expression; eruptions on the skin; scars; decubitus; the position of the patient, and the presence of œdema.*

2. *The rate and rhythm of the pulse; the character of the arteries and the arterial tension.*

3. *The rate, type, and character of the respirations.*

4. *The temperature.*

5. *The physical condition of the lungs, heart, digestive organs; of the abdomen, of the urinary organs, of the genitals, of the nervous system, of the extremities, and of the special organs.*

6. *The chemical composition and microscopic elements of the urine, and, whenever necessary, the chemical composition of the gastric juice, the appearance of the feces, etc.*

Evidences of disease are detected by the senses of sight, smell, touch, and hearing. As the former two disturb the patient least, we should gain all the information we can through these channels before proceeding to palpate, percuss, or auscultate. It is surprising how much knowledge can be gained by sight and smell. The short, rapid, forced respirations, with a flushed face, are strongly indicative of pulmonary disorder; the puffed eyelids, distended abdomen, protruding umbilicus, and œdema of the feet are pathognomonic signs of impaired general circulation; the facial expression shows actual pain, anxiety, etc.; thoracic respiration with immobility of the abdominal muscles are indications of peritoneal inflammation; shortening of limbs, axial deviation, malposition or abnormal position thereof are evidences of fracture or dislocation, etc. The diagnosis can often be made by sight alone.

The sense of smell likewise gives valuable information; thus the sweetish odor of the breath and perspiration which accompanies the septic state, and the odor of violets (acetone) in the urine and breath, that is sometimes present in diabetes, are important aids in making a diagnosis of these diseases.¹

Palpation, percussion, and auscultation are proceeded with after a thorough inspection and smell of the patient. Here there is need of a light touch and a sharp ear. It is always a good rule to compare any abnormalities with the supposedly healthy side.

The information obtained by these special sensory organs is supplemented by that which is elicited by the use of special apparatus—*e. g.*, cystoscope, proctoscope, hæmocytometer, microscope, etc.—and finally by that which is obtained by bacteriological, chemical, and histological examination. It is to be remembered that the clinical evidences of disease are the most important; the laboratory results aid, elaborate, and substantiate the bedside findings; they never replace them.

¹ The author knows of a patient admitted to the hospital in comatose condition. No history could be obtained. The bladder was empty. A painstaking physical examination did not enable the attending physician to make an accurate diagnosis. The lay superintendent who had admitted the patient ventured the diagnosis of diabetic coma from the smell of the patient's breath and perspiration. Subsequent examination of the urine proved him to be correct.

The advanced diagnostician as well as the beginner will find it essential to make the thorough examination outlined above. A complete physical examination is essential not only for making a diagnosis, but the therapeutic measures that are to be employed frequently depend upon the condition of the internal viscera. The symptoms for which the patient seeks relief may be secondary to a primary lesion that is forgotten or unnoticed until the attention is directed to it by a thorough examination; or, again, the existence of severe respiratory, cardiac, or nephritic, or other constitutional disease may contraindicate a surgical procedure that would otherwise have been employed.

THE USE OF INSTRUMENTS FOR DIAGNOSTIC PURPOSES.

Special instruments for diagnostic purposes are meant to serve one or other of the following purposes:

1. To aid and fortify the senses of sight and hearing—*e. g.*, pocket microscope, stethoscope, etc.

2. To view the interior of organs—*e. g.*, ophthalmoscope, oral and nasal specula, œsophagoscope, laryngoscope, cystoscope, proctoscope, endoscope.

3. To view the shadow cast by foreign bodies and solid organs—*e. g.*, the *x*-ray machine. To determine the outlines of a hollow viscus by transillumination—*e. g.*, gastrodiaphane.

4. To obtain the secretions and contents of the hollow viscera—*e. g.*, stomach tube, catheter, etc.

5. To determine the chemical and physical nature of the secretions and body fluids—*e. g.*, hæmoglobinometer, hæmocytometer, cryoscope, etc.

6. To determine the calibre, length, and position of hollow organs—*e. g.*, sounds and bougies.

7. To determine the presence of foreign bodies in the hollow organs—*e. g.*, the vesical sound, œsophageal sound etc.

General Considerations on Instruments.—All instruments that are introduced into body cavities should be made

clean by boiling, or, if this is not possible, by immersion in 5 per cent. carbolic acid solution for fifteen minutes. In the latter case they should be washed in distilled or sterile water just before they are used. Instruments should always be in perfect order, and should be tested and examined before each using. Those that are meant to be passed into cavities should be perfectly smooth and highly polished, lest they abrade or tear the channels through which they are to pass.

The modern surgeon must cultivate expertness in the use of all instruments and in the interpretation of the findings obtained by them. It is as necessary for the diagnosis of brain tumor, brain abscess, cerebral compression, or sinus thrombosis, to recognize a choked disk, as it is to make out axial deviation and shortening in fracture of the long bones. A specialist in the diseases of the eye, ear, etc., is not always at hand. That surgeon will make the best diagnosis whose skill in the handling of instruments and whose training in the interpretation of their findings are the most perfect.

Most of the instruments detailed above are so well known that little need be said in description or explanation of them.

Cystoscope.—Of cystoscopes there are several models. The author personally feels that the expert in cystoscopy should be equally familiar in the use of one example of each type of the instrument, viz., in the direct-vision cystoscopes—*e. g.*, Kelly's tubes; the direct telescopic cystoscopes, and the indirect (prism) telescopic cystoscopes.

The field of vision of the direct cystoscopes is larger and clearer than that of the indirect, but the former are of little value in those patients who have prostatic enlargement and a retroprostatic basin in the bladder. For general use the author would recommend the indirect catheterizing cystoscope of Nitze.

Roentgen Machine.—The Roentgen machine is daily becoming more essential to the diagnostician; not only for the information it affords of the position of fractured or dislocated bones, but also for the detection of foreign bodies, neoplasms, rarefying and sclerosing conditions of the bones, etc. For purposes of diagnosis a medium hard tube with self-regulating tension is the best.

Œsophagoscope.—The œsophagoscope has only recently found a general use. It is of decided value in the examination of the œsophagus and cardiac end of the stomach. The diagnosis of diverticula, ulcerations (benign and malignant), spasms, and stenosis of the œsophagus will be rendered more certain when sufficient expertness in the use of this instrument shall have been gained. The best type of instrument is the straight tube with a light carrier introduced down to the bottom of the tube.

Beckman's Cryoscope.—The modern surgeon desires to know not only the existence and nature of diseased processes in the kidneys, but also the separate and combined functioning power of these organs. One of the best methods of determining the latter is by ascertaining the freezing point of the urine and blood. The freezing point of a liquid depends upon the number of molecules it holds in solution; the greater the molecular concentration of a fluid as compared with that of distilled water, the lower will be its freezing point. With insufficient kidney action the concentration of the blood rises, while that of the urine falls; under these conditions the freezing point of the blood will fall below its normal point, while that of the urine will rise. Normal blood freezes at 0.56° to 0.59° below distilled water; with insufficient kidney action it freezes at 0.60° or more below distilled water. Similarly normal urine freezes at 1° to 2° below distilled water; with insufficient kidney action the freezing point rises to 0.8° or 0.9° or more below distilled water. The freezing point of the blood and urine is ascertained by the Beckman cryoscope which has recently been introduced from the physical into the medical laboratory by Koranyi of Budapest. (For further details of this instrument and the technique of its use, see *Diseases of Kidneys*, p. 377.)

Hæmoglobinometer of Dare.—The hæmoglobinometer of Dare is used by us in hospital and private practice. It is simple in construction, small in bulk, and gives sufficiently accurate results.

For counting the number of red and white blood cells we use the Thoma-Zeiss hæmocytometer. (For technique, see *Anæmia and Leukocytosis*, p. 35.)

CHAPTER II.

THE CLINICAL SIGNIFICANCE OF GENERAL SYMPTOMS IN SURGICAL DISEASES.

IN this chapter will be considered the clinical significance of the more important constitutional and physical manifestations of acute and chronic surgical diseases. Clinical evidences of disease afforded by the urine, gastric juice, and excreta will be considered in the chapters dealing with diseases of the kidneys, stomach, and intestines.

The symptoms that especially demand attention here are *cachexia, emaciation, jaundice, pain, fever, pulse rate, respiration rate, general gastrointestinal disturbances, anæmia, leukocytosis.*

Cachexia.—Cachexia evidenced by a dirty (yellowish) pallor of the skin is associated with the later stages of secondary anæmia and accompanies those diseases—*e. g., carcinoma, phthisis, syphilis, chronic septicæmia*, etc.—that give rise to this condition. It is not an exclusive nor an invariable accompaniment of malignant disease.

Emaciation.—Emaciation testifies to disturbances in metabolism, either in a faulty assimilation of food or in increased tissue waste. Excessive tissue destruction can be determined by comparing the total nitrogen ingested with the total nitrogen eliminated in the urine and feces (the latter can best be determined by the Kjeldahl apparatus).

Jaundice.—Jaundice, a yellowish discoloration of the skin and mucous membrane, is due to absorption into the blood of the coloring matters of the bile or derivatives of it. (The urine becomes very high colored; the stools may or may not be whitish in color.)

Two forms of icterus are recognized: the obstructive and the hæmohepatogenous.

The former is due to an interference with the passage of the bile into the duodenum, and the latter to an excessive

destruction of red blood cells in the liver. The hæmoglobin which is set free by the disorganization of the red cells is converted into bilirubin, and, being secreted into the finer hepatic ducts in larger amounts than can be readily carried away, part of it is resorbed into the circulation and carried to the tissues, which it stains. The excessive destruction of red blood cells in the liver, giving rise to hæmohepatogenous jaundice, occurs after the administration of certain poisons—*e. g.*, ether, chloroform, phosphorus, etc.; in some of the infectious diseases, septicæmia, pneumonia, acute yellow atrophy of the liver, etc. The intensity and constancy of the jaundice depends upon the extent and duration of the hæmolysis.

The impediment to the free passage of bile into the duodenum (obstructive jaundice) may be located anywhere, from the finest bile-capillaries to the orifice of the common bile-duct at the papilla of Vater. The causes may, therefore, be divided into the intrahepatic and extrahepatic. In the former the obstruction may involve all the bile-ducts or be limited to the ducts of larger or smaller areas of the liver, and the intensity of the jaundice will very naturally vary with the extent of the involvement. Thus the swelling and occlusion of the biliary capillaries in the liver resulting from a general or diffuse cholangitis will result in deep jaundice; whereas a cyst or carcinoma or abscess giving rise to local inflammation of the bile-ducts (circumscribed cholangitis) will be accompanied by little or no jaundice. In the extrahepatic forms of obstructive jaundice the impediment may lie within the ducts, as from a calculus, or stricture, or neoplasm; or it may be due to the pressure or traction upon the ducts by a neighboring organ—*e. g.*, floating kidney or enlarged head of the pancreas (from neoplasm or chronic inflammation), or enlarged gall-bladder or tumor of pylorus, or tumor or abscess of the liver, etc.; or it may be due to kinking of the ducts by the contraction of surrounding adhesions. The grade of jaundice varies with the extent of compression, stenosis, or obliteration of the ducts; its constancy varies according to the permanency of the compressing, stenosing, or obliterating factors. With obstruction of the common bile-ducts by calculus the gall-bladder

is usually contracted, the icterus varies in intensity, the stools being at times brown, again white; the spleen is somewhat enlarged, and there is a history of colicky pain and intermittent fever. With obturation or compression of the common bile-ducts by tumor, the gall-bladder is enlarged and distended, the jaundice grows constantly deeper, the stools are continuously white, there is no history of colicky pain or fever, and the spleen is not enlarged. These points of difference are designated as Courvoisier's law, and their importance in differential diagnosis will be discussed under Cholelithiasis. (See p. 315.)

Pain.—This is a subjective symptom, and in forming an estimate of its intensity due allowance must be made for the character of the individual. People of stoical character bear pain, even of severe degree, without much complaint; whereas, others, of sensitive nature, magnify its severity. Each individual is a law unto himself, as to how intensely he reacts to painful sensations. The physician must by observation and experience learn to estimate how much pain a patient really suffers.

Pain is either *neuralgic* or *parenchymatous* in its origin; in the former the exciting cause acts upon the trunk of a sensory or mixed nerve or upon the sensory centres. In the latter the irritating factor acts upon the peripheral sensory end organs.

Neuralgic pain is *absolutely limited* to the *area* supplied by the nerve or nerves which are the seat of irritation—*e. g.*, in trigeminal neuralgia or in the case of callus or scars pressing upon sensory nerves. In parenchymatous pain the *affected sensory end organs* may belong to *several different spinal nerves*; consequently the *painful area* is not limited to the region of distribution of one spinal nerve. Neuralgic pain is limited to a definite anatomical area; parenchymatous pain is diffuse, invading the region of distribution of several nerves. Neuralgic pains are more intense and more transient than parenchymatous pains. The latter are associated with tenderness over the affected part; whereas, with neuralgic pain there is tenderness directly over the irritated nerve, and then only in case it is superficial and can be compressed against a solid bony structure. Neuralgic

pain is due to inflammation of nerve trunks, new-growths of nerve trunks (neuroma or sarcoma), or pressure on nerve trunks by neighboring neoplasm (*e. g.*, scar, callus or exudate). A frequent error in diagnosis arises from confounding the irritation of the sciatic nerve by chronic inflammation (the common form of sciatica) with the sciatic pain which is due to compression and irritation of the sciatic nerve by small pelvic tumors. Parenchymatous pain is due to disease or injury of any part.

Character of Pain.—*Neuralgic pains* are usually very intense, transient, sharp, burning, or cutting; there are local points of tenderness over the superficial nerves.

Parenchymatous pains are apt to be less intense and constant, and are either sharp and cutting or dull and aching; there is tenderness over the entire painful area.

Colicky pain in tubular viscera is a composite one, composed of the pain produced by a foreign body, kink, or constriction which obstructs the lumen of the tube, and secondly of the pain which results from the forcible contractions of the tube in its endeavors to overcome the obstruction. The first element may be continuous, sharp, and cutting—*e. g.*, when a stone is impacted in the bile-ducts or ureters; the second element is sudden, wave-like, and sharp, and subsides suddenly. The appendix, gall-bladder, and kidneys are frequently the seat of such colicky pains.

Location of Pain.—The pain may be referred to the area of distribution of the irritated nerve or to the diseased part. In such cases the site of the pain is a valuable aid in diagnosis. Often, however, the pain is referred to some distant region (radiate pain)—*e. g.*, the pain in the knee-joint from hip disease, the pain over the appendix from right basal pleurisy. Quincke¹ has formulated these radiate pains as far as they are known and established. The most important are: trigeminal pain in frontal-sinus disease; parietal pain in diseases of the middle ear and mastoid; laryngeal pain in probing pulmonary abscesses; pain in the left shoulder, at times in the right, with angina pectoris; pain in the lower dorsal region with stomach affections; pain in the right shoulder and back

¹ Zeitschr. für klin. Med., 1890, Bd. 17.

with liver and gall-bladder diseases; pain in the back, bladder, and genitals in kidney diseases; epigastric pain in endometritis; knee pain in coxalgia; right iliac pain in right basal pleurisy; left iliac pain in left basal pleurisy; left shoulder pain in diseases of spleen. The diagnostician must constantly be on his guard in the correct interpretation of these radiate pains. He must be conversant with them, and never neglect to examine the organ from which the pain may radiate, as well as the painful region itself. Many an appendix is removed for a right basal pleurisy; many a knee-joint accused of tuberculosis when the hip is really at fault.

In addition to the radiate pain, it has been shown by the English neurologist, Head, that diseases of the internal organs are usually attended by a hyperæsthesia of definite areas of the skin. The areas of cutaneous hyperæsthesia symptomatic of the diseases of the various internal organs have been accurately mapped out by Head, and he considers them valuable aids in the diagnosis of diseases and injuries of these parts. They often correspond to the regions to which the pain radiates, as described above. In order to determine the zones of cutaneous hyperæsthesia, a blunt-pointed instrument should be passed along the skin from above downward, the patient being instructed to say when he feels the pressure of the instrument most.

The hyperalgesic skin areas in diseases of the internal viscera are, according to Head, as follows:

DESCRIPTION OF FIGS. 1 AND 2.

Zones of cutaneous hyperæsthesia symptomatic of diseases of the internal organs. They are indicated by letters and subjacent numerals which correspond to the spinal-cord segments from which the nerves supplying the affected portions of skin are derived.

Heart diseases : Pain and hyperæsthesia in zones C_3 , D_1 , D_4 .

Tuberculous lung diseases : Pain and hyperæsthesia in zones D_1 — D_7 , especially D_2 , D_4 , D_5 .

Diseases of œsophagus : Pain and hyperæsthesia, especially in D_5 , D_6 , D_8 .

Diseases of mamma : Pain and hyperæsthesia in D_4 , D_5 .

Diseases of stomach : Pain and hyperæsthesia in D_6 , D_8 , D_9 .

Diseases of intestines, pylorus, and colon : Pain and hyperæsthesia in D_{10} , D_{11} , D_{12} .

Diseases of liver : Pain and hyperæsthesia in D_7 , D_8 , D_9 , D_{10} .

Diseases of kidney and ureter : Pain and hyperæsthesia in D_{10} , D_{11} , L_1 .

Diseases of bladder : Pain and hyperæsthesia in S_2 , S_3 , S_4 .

Diseases of testicle and ovary : Pain and hyperæsthesia in D_{10} .

Diseases of uterus : Pain and hyperæsthesia in D_{10} , D_{11} , D_{12} , L_1 .

Diseases of cervix : Pain and hyperæsthesia in S_1 , S_2 , S_3 , S_4 .

FIG. 1

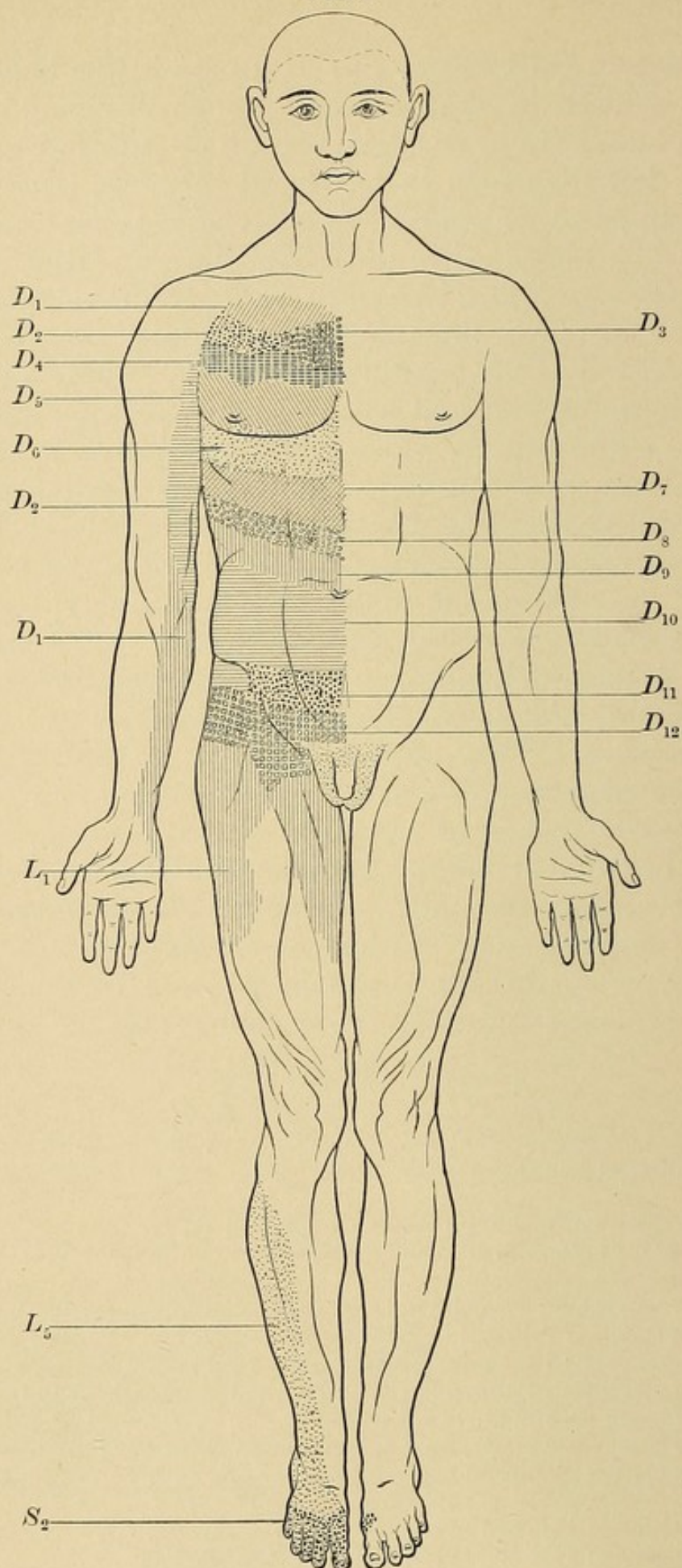
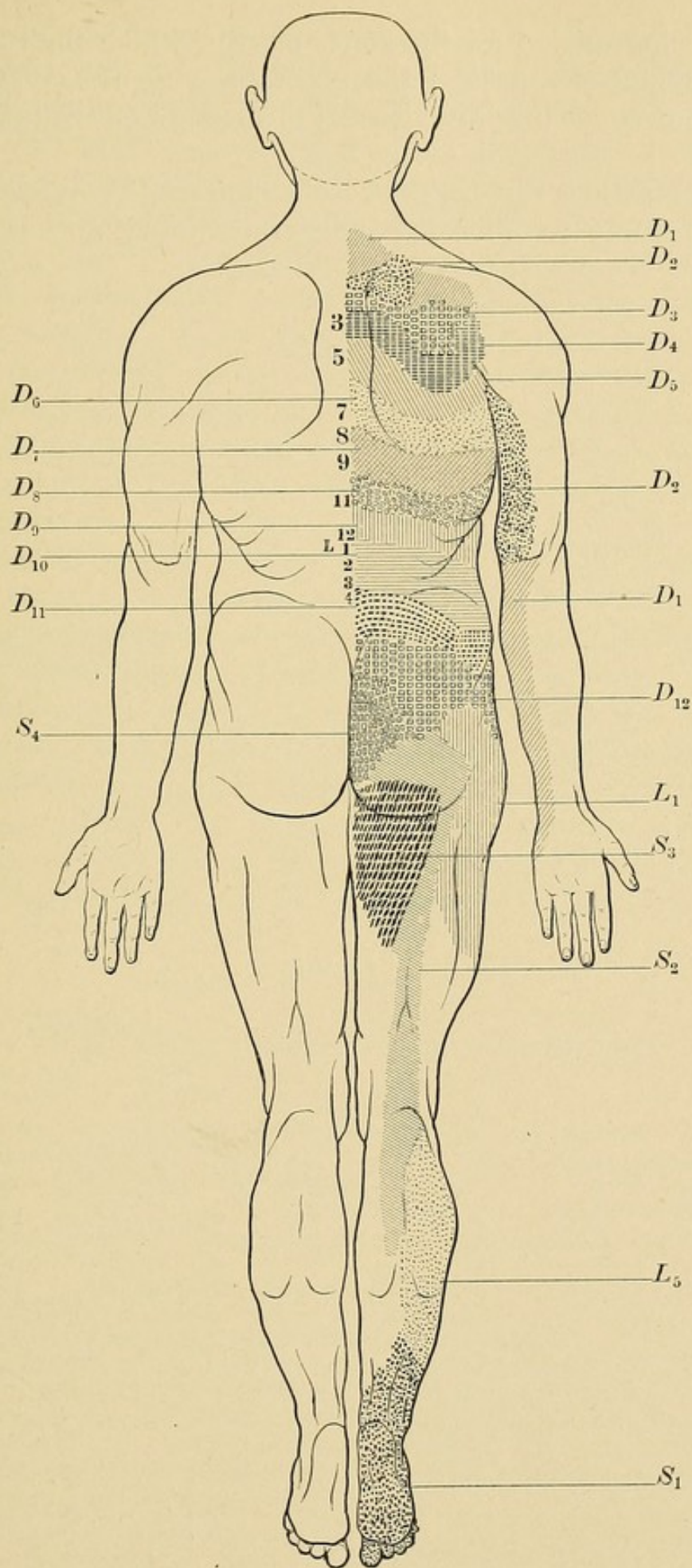


FIG. 2



The diagrams show the zones of skin which the indicated spinal segment supplies; the accompanying tables state the particular zones that are affected in diseases of the individual viscera.

Pain, Muscular Rigidity, and Reflex Spasm.—Wherever there is pain, there we find muscular immobility and muscular

FIG. 3

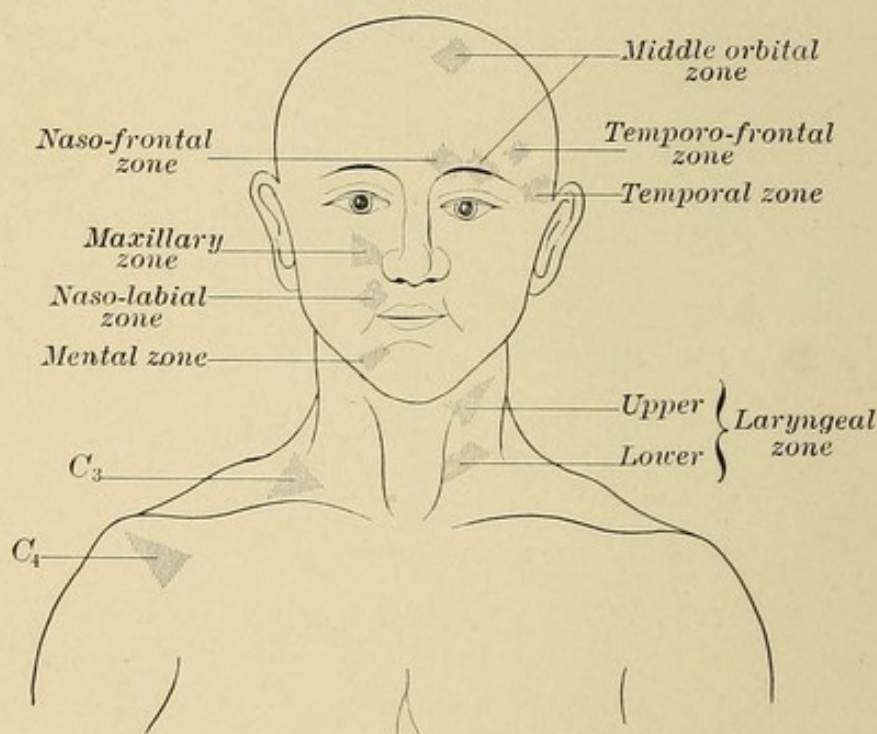
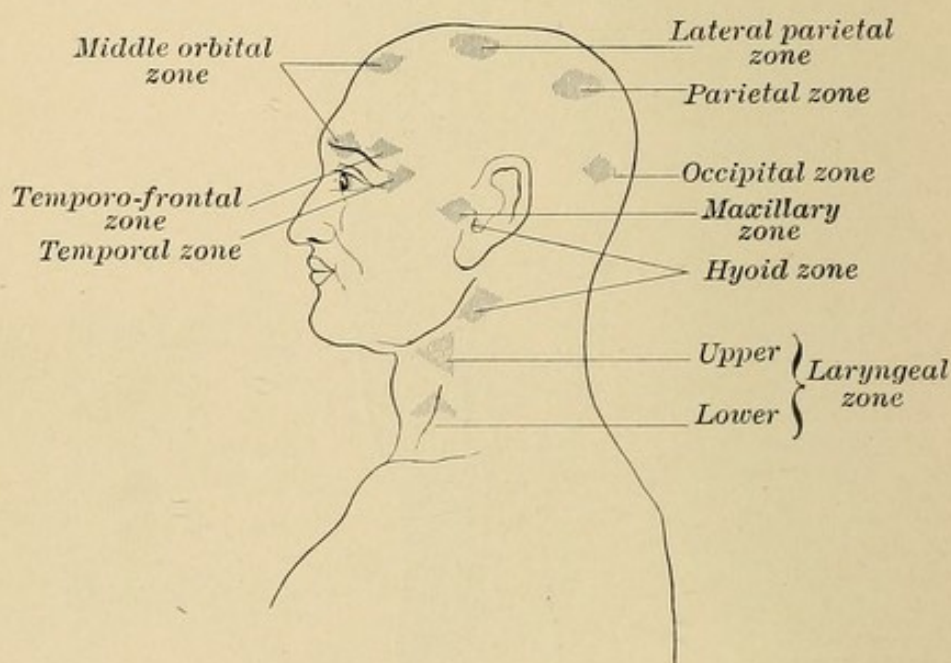


FIG. 4



rigidity. It is the attempt on the part of nature to afford protection to the painful part. Such muscular rigidity and reflex muscular spasm are often the earliest signs of inflammation. Thus the rigidity of the abdominal wall in inflammations of the peritoneum. (Rigidity of the abdominal wall is best elicited by watching whether it moves with respiration, and by passing the finger-tips *gently* over its surface. Deep palpation excites reflex contraction and thus obscures rigidity. Note that the rigidity may be local or general, depending upon the extent of involvement of the peritoneum.) The immobility of the chest in inflammatory diseases of the pleura and the reflex muscular spasm in joint disease are other instances of muscular rigidity attending painful diseases.

When a history of pain is elicited, it is well to enquire whether there have been any previous attacks. Such a history is of importance in establishing a diagnosis in doubtful cases of cholelithiasis, renal calculus, appendicitis, etc.

It is further important to ascertain whether the present attack of pain is or is not similar to that in previous attacks. Thus the patient may have had a number of attacks of sharp pain in the gall-bladder or appendix or in the stomach, etc., but in the present attack the pain is of a tearing character, is much more intense and accompanied by prostration. We

DESCRIPTION OF FIGS 3 AND 4.

Head and neck zones :

Nasofrontal zone is affected by diseased conditions of the eyes, nose, and upper incisor teeth.

Middle orbital zone ; Affected by hypermetropia.

Temporofrontal zone : Affected by diseased conditions of ear and heart.

Temporal zone : Affected by glaucoma.

Lateral parietal zone : Affected by middle-ear disease.

Parietal zone : Affected by ear and stomach diseases.

Occipital zone : Affected by diseased conditions of posterior one-half of larynx. and some of the intestines.

Maxillary zone : Affected by diseases of iris and cornea.

Mandibular zone : Affected by diseases of upper molars.

Nasolabial zone : Affected by diseases of nose and tooth pulp.

Mental zone : Affected by diseases of incisors and bicuspid teeth.

Hyoid zone : Affected by diseases of tonsils, tongue, and lower molars.

Upper laryngeal zone : Affected by diseases of dorsum of tongue and wisdom teeth.

Lower laryngeal zone : Affected by diseases of larynx.

According to Head, diseased conditions of the serous membranes are not attended with hyperæsthetic skin zones.

would infer from such a history that a rupture of the viscus had taken place during the present seizure.

The relation of pain to other bodily functions, to the ingestion of food, is also very important from a diagnostic standpoint. Pain over the pylorus immediately after the ingestion of food suggests gastric ulcer; pain in the pyloric region one or two hours after a meal, a duodenal ulcer; pain on defecation, a fissure in ano, or an ulcer of the rectum; pain before and during urination, cystitis; pain after urination, disease of the neck of the bladder. Tuberculosis and calculous diseases of the kidney frequently give as their first manifestation increased frequency and painful urination. Cholecystitic pain is apt to become worse two to three hours after a hearty meal.

The influence of bodily movement on pain is often a clue to diagnosis. Thus the pain of stone is made worse by walking, jumping, going up and down stairs. Pleuritic pain becomes worse on deep inspiration; peritonitic pain by peristalsis.

As said at the outset, pain is a purely subjective symptom. Hysterical patients may complain of intense pain and yet there is no cause therefor. It must however be strongly advised never to consider pain as hysterical or as trivial until repeated careful physical examinations have revealed no underlying cause therefor.

Fever; Pulse Rate; Respiration Rate.—The normal bodily temperature of an adult individual is 98.6° , the pulse rate 70 to 80 to the minute, and the respiration rate 18 to 20 to the minute. In all cases in which variations from the normal are suspected the temperatures should be taken per rectum every three or four hours and recorded on a chart, the pulse rate and respiration rate being coincidently noted and recorded.

An increased bodily temperature may follow (1) upon disturbances in the central nervous system—*e. g.*, after great fright or excitement, after epileptic seizures, injuries of the spinal cord, etc.; and (2) upon the resorption into the general circulation of deleterious bodies such as fibrin ferment and the toxins of bacterial life. Increased temperatures from the latter cause may be of a continuously high type with fluctuations of half a degree, in which case it indicates a continuous

resorption of deleterious bodies. The height will depend upon the virulency of the toxins and upon the amount and rapidity of their absorption, which latter in turn depends upon the tension under which the toxic products are confined in the tissues. Such continuous high temperatures accompany simple or suppurative inflammation. With the onset of the disease there is usually a distinct chill or chilly sensation.

The elevated temperature may be of an intermittent or remittent type, with marked fluctuations. With each rise there may be another chill or chilliness. Such intermittent or markedly remittent temperatures indicate an extension of the inflammation with new absorption of toxins. They occur with advancing inflammations like erysipelas, and in extending suppurative inflammations, especially those of the vascular system, for in these latter cases infected emboli from the primary seat of disease are being constantly dislodged into the blood stream and carried to distant organs, where they set up fresh suppurations (metastatic). The fluctuations of temperature have no regularity. They may occur daily, or several times daily, or at intervals of several days.

The height of the temperature is usually proportionate to the amount of resorption of toxins. This is in turn dependent on the tension under which the toxins are confined in the tissues. A small abscess under high tension—*e. g.*, in the neck under the deep fascia (angina Ludovici)—may give rise to high temperatures, whereas a large abscess in loose cellular tissue may occasion only moderate febrile elevations.

Temperature and Pulse Rate in Inflammations of Tubular Abdominal Viscera.—High temperatures accompanying suppurations in tubular viscera whose orifice of exit has become closed—*e. g.*, appendix, gall-bladder, Fallopian tube—indicate that the inflammatory products within these organs are under considerable tension. Low temperatures with rapid pulse rate in inflammatory conditions of such tubular viscera point to gangrene of the affected organ. The temperature is low because, on account of the paralysis of the inflamed part which results from the gangrene, the tension of the inflammatory products within it cannot rise very high. The pulse rate, however, is rapid and out of all proportion to the temperature. In both these conditions operation is

imperatively indicated, lest perforation with extravasation of septic material into the peritoneal cavity occur. Again the onset may have been very acute, with chill and high temperature and proportionally rapid pulse. Suddenly the temperature falls to normal or near the normal, but the general condition of the patient is not improved or it even becomes worse. This indicates a sudden lowering of tension from gangrene or perforation, and operation becomes imperative.

A rise in bodily temperature is usually accompanied by a coincident proportionate rise in pulse and respiration rate. In suppurative or gangrenous inflammations of the peritoneum, and in peritoneal septicæmia, the pulse rate rises out of all proportion to the temperature. Even with severe suppurative inflammations of the peritoneum, the temperature may be low but the pulse rate is high. The high pulse rate and low temperatures are especially characteristic of peritoneal septicæmia. By Friedländer this has been attributed to vagus disturbance.

The respiration rate is increased out of proportion to the temperature and pulse rate in all diseases of the respiratory tract.

To sum up the foregoing remarks:

Continuous high temperature, high pulse rate, and high respiration rate are indicative of continuous toxin resorption, from simple or suppurative inflammation.

Fluctuating, intermittent, or remittent temperatures indicate an extension of the original inflammation or a fresh injection of toxins or bacteria into the systemic circulation. Each extension or fresh injection may be accompanied by a chill.

The height of the temperature depends upon the rapidity and amount of resorption, which in turn is dependent upon the tension under which the inflammatory products are confined. Paralysis of a viscus or gangrene of its walls results in a diminution of the inflammatory tension, the rapidity of resorption is consequently lessened, and the temperatures are apt to decline. The pulse rate, however, remains rapid because the toxæmia continues, and is even increased by the gangrene or paralysis of the affected part.

High temperatures indicating a high tension, followed by a sudden decline of temperature with no coincident improvement in the general condition, point to gangrene or paralysis of the viscus.

Temperatures in themselves are no guide to the nature, extent, or severity of the inflammation. The pulse rate, the respiration rate, and the general condition of the patient, together with the clinical history of the present illness must always be considered in conjunction with the temperature. Peritoneal septicæmia is regularly attended with low temperatures, but high pulse and respiration rate.

A subnormal bodily temperature occurs chiefly after exposure to intense cold, at the critical period of some acute febrile diseases, *e. g.*, pneumonia, in conditions of collapse, after profuse hemorrhage, and in chronic exhaustive diseases, as the final stages of tuberculosis and carcinoma.

The fall of temperature that accompanies collapse is attended with a coincident increase in the rapidity and feebleness of the pulse, which latter signs distinguish this condition from the critical period of febrile diseases, for in this latter condition the pulse becomes slower and its quality remains good. It is to be noted that collapse may be excited reflexly by severe injuries or by mental emotions, or it may be occasioned by poisons, of which those that are elaborated by bacterial life and development are especially important. This accounts for the collapse that sometimes attends the acute bacterial diseases.

Anæmia, Leukocytosis. Method of Examination of the Blood.—The determination of the hæmoglobin percentage of the blood and the ascertainment of the number and character of the red and white blood cells are necessary for the diagnosis of essential blood diseases. They also aid in the detection of those primary organic diseases which occasion early changes in the blood, and they shed light on the nature of certain inflammatory processes.

The hæmoglobin percentage is estimated by the Dare hæmoglobinometer. The red and white blood cells are counted with the Thoma-Zeiss hæmocytometer. This latter apparatus consists of two parts: a glass graduated capillary tube with a bulbous enlargement near one end that serves for

drawing up and diluting the blood, and a counting chamber which is mounted on a glass slide. The ear or finger-tip is pricked and the blood is drawn up in the capillary tube to the mark 0.5 c.c. or 1 c.c.; the tip of the tube is wiped off and normal saline solution is then drawn up into the tube and bulb to the mark 101, thus diluting the blood two hundred or one hundred times, depending on whether we have drawn up 0.5 or 1 c.c. of blood in the capillary tube. The combined saline solution and blood in the bulb are well mixed by shaking. The fluid in the capillary tube is then expelled and a drop from the bulbous mixing chamber is put into the counting chamber. The counting chamber is 0.1 mm. deep; its floor is divided into microscopic quadrates of $\frac{1}{4000}$ c.mm. capacity, which are grouped in squares of sixteens by double dark dividing lines. The counting chamber is now covered with a cover-glass, care being taken to expel the air bubbles between them and to prevent overflowing of the mixture into the trough around the chamber. A number of the larger squares are counted and if the total number of red cells they contain is divided by the number of quadrates counted, the average contents of a quadrate will be obtained. This is then multiplied by 400,000 or 800,000, depending upon whether the blood has been diluted 100 or 200 times, and the result indicates the number of red blood cells in a c.mm. of blood. The counting of the white blood cells is done in exactly the same way; the blood is, however, only diluted ten times. The dilution is made with a 1 per cent. acetic acid solution; this takes away the color of the red blood cells by dissolving out their hæmoglobin, and so renders counting of the leukocytes more easy. The average number of leukocytes in each small square is of course only multiplied by 40,000 or 80,000, for the dilution is only one-tenth that used in counting the red cells.

In a healthy individual the red cells number 5,000,000. In chlorosis the number is not materially diminished; in anæmia it is. The number of white blood cells in a healthy individual is 5000 to 8000; an increased number (hyperleukocytosis) occurs normally during digestion of proteids; in some of the infectious diseases and with cachexia there

is likewise an increased number of leukocytes (*e. g.*, of carcinoma, see below). An increase over 50,000 warrants the diagnosis of leukæmia.

For staining the blood corpuscles, a drop of blood is placed on a perfectly clean, dry cover-slip; this is placed upon a second cover-slip, and after a second or two the cover-slips are drawn apart by a rapid gliding action, and the thin films on their surface allowed to dry in the air. The Jenner stain, consisting of methylene blue and eosin dissolved in pure methyl alcohol, is employed for staining purposes. The cover-slip is floated, film downward, in the stain for one or two minutes; it is then thoroughly washed in distilled water, dried, and mounted in Canada balsam. Under the microscope, the following forms of red cells may be distinguished:

1. Normal red cells.
2. Very large (macrocytes) or very small (microcytes) red cells.

In the severe anæmias are found:

3. Irregular form red cells (poikilocytes), or
4. Degenerated red cells with nucleus and granulated protoplasm (normoblasts, microblasts, and megaloblasts), and the following forms of white blood cells:

1. Lymphocytes, with round nucleus and small body. They originate in the lymph glands, and represent the leukocytes, which are chiefly increased in number in lymphatic leukæmia.

2. Mononuclear leukocytes, considerably larger than preceding, with ovoid nucleus and large cell body. From these develop the

3. Polynuclear leukocytes, which are large, irregular in form, finely granular and possess a lobulated nucleus or as many as four nuclei. This variety forms 70 to 75 per cent. of the leukocytes in normal blood, and it is these cells that are mainly increased in hyperleukocytosis, their number rising to 80 or 85 per cent. of the total number of leukocytes. The last two varieties are the ones which are chiefly increased in splenic and myelogenous leukæmia.

Ehrlich divides the leukocytes into three groups, according to the size and the staining qualities of the granules found in the cytoplasm:

1. The eosinophilic cells: large, round, granular bodies with granules staining deeply with eosin and other acid dyes. They come from the medulla of the bones, and normally form 1 to 3 per cent. of the leukocytes of the blood. Their presence in considerable number points to medullary involvement.

2. The basophilic cells: granules staining palely with basic dyes, and forming $\frac{1}{2}$ to 1 per cent. of leukocytes in normal blood.

3. The neutrophilic cells: granules staining only with neutral dyes.

Anæmia.—By this is designated a pallor of the skin and mucous membranes due to diminished coloring matter (hæmoglobin) in the blood (oligochromia). As a pallid color of the skin and mucous membranes may occur in completely healthy individuals, it is essential in every case to count the number of the red blood cells and estimate the hæmoglobin percentage before one assumes the existence of an anæmic state. A pallid color with normal number of red cells and hæmoglobin percentage is found in the early stages of stomach diseases, heart diseases, phthisis, and cachexia. In their advanced stages these diseases give rise to a secondary anæmia in which the destruction of hæmoglobin exceeds the loss of red blood cells.

In the early stages of cancer of the internal viscera, before a tumor is palpable, a marked anæmia in conjunction with other data, which in themselves are insufficient to warrant a diagnosis of malignant disease, furnishes strong presumptive evidence of this malady. Thus in chronic gastric disease a marked anæmia, together with an absence of free hydrochloric acid and the presence of lactic acid in the gastric contents, is strongly suggestive of a carcinoma of this organ. But *only* in the light of "additional confirmation" is an anæmia to be considered important in the early diagnosis of malignant new-growth.

In conditions of severe anæmia the administration of a general anæsthetic for surgical purposes may be followed by the most serious consequences. Mikulicz, Da Costa, Kalltayer, Cabot, and others have found that the administration of a general anæsthetic results in a marked destruction of

red blood cells and a corresponding reduction in the percentage of hæmoglobin. In patients who have a low hæmoglobin percentage the administration of a general anæsthetic may so much further reduce the hæmoglobin that the remaining amount will be absolutely insufficient to carry on the oxygen function of the blood, and death will necessarily follow. The importance of ascertaining the hæmoglobin percentage prior to the administration of a general anæsthetic is thus very evident; it should be a part of our routine examination just as is the analysis of the urine. Mikulicz advises against the administration of a general anæsthetic when the hæmoglobin is below 30 per cent.; other investigators place the limit of safety at 40 per cent.

Leukocytosis.—The number of leukocytes in the blood is often of considerable diagnostic importance. For this purpose the count must be made repeatedly and systematically, for only in this way can a rise or fall or stationary condition of the number of white cells be determined. A single count is of very little clinical value; only the course of the leukocyte count enables us to draw any valuable conclusions. It is to be noted that in young subjects the number of leukocytes, even in health, fluctuates considerably, and furthermore that a physiological hyperleukocytosis (to 10,000 or 20,000) occurs during proteid digestion.

From the results of the investigations of a number of observers (Cabot, Reich, Mikulicz, etc.) the following conclusions may be drawn:

1. **Leukocyte Count in Inflammation of Soft Parts.**—With inflammations in the soft parts (cellular tissues, etc.) a leukocyte count of 20,000 or over remaining constant or increasing for three or four days speaks strongly in favor of acute progressive abscess formation. With latent abscess or chronic abscess there is no hyperleukocytosis.

The hyperleukocytosis which goes with acute abscess frequently aids in the diagnosis of a purulent collection when other clinical evidences are wanting or obscure. It helps further to differentiate between an acute, inflammatory, non-purulent exudate and an abscess; its presence warrants an incision, even though other clinical evidences of pus are

wanting. A high leukocyte count (20,000 to 30,000) three days after the incision of an acute abscess speaks in favor of pus retention or of another abscess, even though the temperature is low; whereas a low count, even with high temperature, speaks against such a possibility. Further, if with an acute inflammatory exudate with high temperature the leukocyte count falls suddenly from high numbers, there is every probability of resolution without pus formation.

2. Leukocyte Count in Inflammation of Bones.—*Acute suppuration of the bones* is attended with high leukocytosis (over 20,000), even higher than in suppurations of the soft parts. In the very early stages of acute infectious osteomyelitis the hyperleukocytosis is not marked enough to distinguish this malady from acute rheumatism, neuralgia, or growing pains. This is especially the case in children, who are the most frequent sufferers from this affection, and who normally have a widely fluctuating number of leukocytes.

Because of the slightly increased leukocytosis attending acute infectious osteomyelitis, we cannot rely upon the number of the leukocytes for the differentiation of this malady from typhoid fever, which it often closely resembles, and which is always accompanied with a low leukocyte count.

Latent bone abscess, chronic bone suppuration, bone necrosis do not alter the number of white blood cells sufficiently to aid us in their diagnosis.

After operation a falling leukocyte count, even in the face of a high temperature, points to a recrudescence of the inflammation, and *vice versa*.

3. Leukocyte Count in Inflammations of Serous Membranes.—In *meningitis* Cabot thinks hyperleukocytosis is the rule. The leukocyte count does not aid us in the differential diagnosis between meningitis, brain abscess, cerebral hemorrhage, epidemic cerebrospinal meningitis, and pneumonia. A low white blood count in this disease is of bad prognostic import.

In *pleurisy* a high leukocyte count in acute cases speaks in favor of pus. Serous exudates ordinarily do not cause hyperleukocytosis. In chronic empyemata there is no hyperleukocytosis.

It is to be noted that the exploratory needle is a much more reliable guide to differentiate between serous and purulent effusions.

4. **In Intra-abdominal Conditions.** (a) *Intestinal Obstruction.*—The majority of observers report no hyperleukocytosis. Bloodgood would draw conclusions as to the condition of the bowel from the number of leukocytes present. Normal or very moderate leukocytosis in the first forty-eight hours of an obstruction speaks against gangrene of the bowel. The higher the leukocyte count and the shorter the duration of the obstruction, the greater the danger of gangrene.

(b) *Appendicitis or Cholecystitis.*—A rapidly rising count to 20,000 or 30,000 or over in the first two to four days of an acute attack, after which period the number of leukocytes remains constantly high, speaks strongly for abscess, even though the pulse and temperature do not coincide therewith. A moderate hyperleukocytosis or a normal number of leukocytes may go with a mild attack that does not result in suppuration, with a latent abscess, with gangrene of the viscus, or with diffuse peritonitis. The other clinical evidences, pulse, temperature, and the general and the local conditions, must guide us in determining the nature of the inflammatory process. Sondern maintains that when the polynuclear leukocytes constitute more than 70 per cent. of the total number of white blood cells, it indicates that pus or gangrene is present, and this irrespective whether there is a high total leukocyte count or not.

If with an intra-abdominal abscess that is associated with a high leukocyte count there is a sudden fall in the number of leukocytes and a coincident deterioration in the general condition of the patient, the presumption is strong that a rupture of the abscess into the peritoneal cavity has occurred.

(c) *Typhoid Perforation.*—About half the cases have a slowly rising leukocyte count (up to 18,000 or 20,000) during the twenty-four hours succeeding perforation. In the other half of the cases the blood count is low until diffuse peritonitis sets in. In some the leukocyte count remains low, even with a diffuse peritonitis. Cushing maintains that even in the preperforative stage there is a moderately increased

hyperleukocytosis. In some of the author's cases this has not been so.

A high leukocyte count in diffuse peritonitis is of good diagnostic import.

5. **In Tuberculosis and Actinomycosis.**—No increased leukocytosis accompanies a pure tuberculosis or actinomycosis, no matter what the locality or the activity of the infecting bacillus may be. An increased leukocytosis in these affections speaks in favor of a mixed infection, and the degree of leukocytosis is usually proportionate to the severity of the latter. High temperatures and normal number of leukocytes usually speak against inflammation and suppuration. High temperatures and hyperleukocytosis speak for suppuration.

6. **In Neoplasms.**—Benign tumors cause no change in the number of leukocytes. In malignant growths (carcinoma and sarcoma) the leukocyte count is inconstant.

7. **In Injuries.**—Subcutaneous injuries, fractures, etc., are associated with very moderate leukocytosis for about three days. Aseptic wounds cause a moderate leukocytosis for two to three days. If this does not subside after three days, suspicion of wound infection must be entertained. (Occasionally the leukocytosis lasts longer than three days; if the temperature and other signs point against suppuration, we may delay before opening the wound.) Suppurating wounds are attended with increased leukocytosis. (See abscess in cellular tissues, p. 39.)

It is to be noted that our present knowledge of the degrees and constancy of leukocytosis is by no means so well formulated as to warrant our neglect of other clinical evidences in forming an opinion as to the presence or absence of suppuration, perforation of a viscus into the peritoneal cavity, etc. The needle in doubtful cases of pleural effusion or exudates in the soft parts is much more reliable than the blood count for making a diagnosis of pus. He will be a sorry surgeon who relies upon the blood count to decide whether or when to operate in intestinal obstruction. The leukocyte count must only be looked upon as an aid in diagnosis, and not as a deciding factor. It is to be remembered that only positive findings (hyperleukocytosis) have a value; normal counts have little diagnostic significance.

CHAPTER III.

SURGICAL INFECTIONS.

IN connection with surgical infections it is necessary to note:

1. That they are all due to pathogenic bacteria.
2. That at the point of entry of the organisms there is usually a local lesion; and
3. That the constitutional disturbances attending the infection are due to the absorption into the blood of the products of bacterial life and development—*i. e.*, the toxins.

The knowledge given to us by Pasteur and Koch, that wound infections are invariably due to pathogenic bacteria, should lead us in each case to ascertain the identity of the infecting organism, for the prognosis and, in some instances, the therapeutic measures that are to be employed will depend upon the character of the invading bacteria.

The local lesion bears no relation to the severity of the constitutional symptoms; thus with the slightest and mildest kind of a local disturbance, and sometimes without any discernible local lesion whatever (cryptogenic infection), there may be the gravest forms of constitutional intoxication, and *vice versa*, with very severe local manifestations there may be comparatively little constitutional poisoning. The local lesion may be in the nature of suppuration, as is the case when the pyogenic bacteria are the invading organisms, or it may be a specific lesion, as happens when specific organisms—*e. g.*, the streptococcus of erysipelas, or the anthrax bacillus, or the tubercle bacillus, or actinomyces, etc.—are the infecting agents.

The constitutional symptoms of bacterial infection are due to the absorption of the toxic products elaborated by the invading bacteria; hence the technical name given to the condition of toxæmia. This is true whether the bacteria remain confined to the tissues they first infected (local

infection), or whether they invade and infect the blood (septicæmia), or whether they are carried by the blood and lodged as emboli in other organs, in each of which they inaugurate lesions similar to the primary one (pyæmia).

In this chapter our aim will be to describe and differentiate the local lesions and constitutional disturbances resulting from an invasion of the tissues with pathogenic bacteria exclusive of those due to the tubercle bacillus, the leprosy bacillus, the as yet undiscovered organism of syphilis, and the actinomyces. These will be found minutely detailed in the chapters devoted to diseases of the special organs

THE CONSTITUTIONAL SYMPTOMS OF TOXIN ABSORPTION.

The toxins of the pathogenic bacteria, excepting those produced by the tetanus bacillus and by the as yet undiscovered organism of hydrophobia, when they are absorbed into the system, produce almost similar constitutional disturbances. The symptoms are often ushered in by a chill; the temperature rises to 102° to 104°, and usually remains high with slight remissions; the pulse is rapid, the skin and conjunctivæ may be icteric, there is nausea, sometimes vomiting and diarrhœa, headache and mild delirium. The urine is albuminous and often contains casts.

The intensity of the symptoms depends entirely upon the virulence of the infecting bacteria, and upon the amount of the toxins that are absorbed into the general circulation. In some instances the toxins are so weak and so small in amount that few if any constitutional symptoms are present. In the cases of intensely virulent infections, in which a paralysis of the medullary centres is occasioned by the toxins, the temperature is subnormal, the pulse is very rapid, feeble and thready, the skin is cold and cyanosed and bathed in a cold perspiration, there is frequent vomiting of a brownish material which contains red blood cells, and the urine contains considerable albumin and casts.

In some cases the pulse rate is slow, even though the temperature is high, probably owing to an irritation of the

cardioinhibitory apparatus. When pus forms the temperature becomes more markedly remittent in character. The remissions usually occur in the morning and are apt to be attended with profuse perspiration.

In *pyæmia* the temperature curve is characterized by the occurrence of frequent sharp rises and falls. Each rise is usually preceded by a rigor and the fall is attended with profuse perspiration. Each chill and rise of temperature indicates a further extension of the infection or the lodgement of a new infected embolus in one of the other organs.

The *toxins of the tetanus bacillus* have a selective action upon the motor nerves and motor cells of the brain and spinal cord; these they at first irritate and finally paralyze. In the most virulent cases the symptoms develop in a few hours or days, after the infection has occurred; while in the milder cases the first manifestations appear after two or three weeks. The earliest symptoms are painful tonic spasms of the muscles concerned in opening the mouth (causing lock-jaw), of the muscles of the back of the neck, of the face (causing the peculiar grin-like appearance, the risus sardonicus), and of the pharyngeal muscles (causing dysphagia). These spasms soon extend to the muscles of the trunk and extremities. They become more and more frequent, and the remissions between them more partial. The respiratory muscles are usually involved late in the disease. The spasms can be elicited by the slightest stimulus; they contort the body in various directions; thus, in an arched backward position (opisthotonos), in a doubled forward position (emprostotonos), etc. A considerable degree of fever is sometimes present, though in some cases there is no pyrexia until near death, when the continuous muscular contractions may cause it to rise to 108° to 109°.

The *tetanic symptoms* may in the milder cases run a more chronic course and the spasms may be limited to the part of the body which has been the site of the infection. Under this heading may be grouped the *head tetanus* which follows injuries within the area of distribution of the cranial nerves. It is characterized by an association of trismus with facial paralysis, although there may be both tonic and clonic spasms of other muscles.

The toxæmia resulting from hydrophobia may first show itself months or even years after the infection has occurred; the usual period, however, does not exceed six weeks. The infected wound usually heals, though the scar may be tender and the seat of neuralgic pain. The earliest manifestations of the malady are restlessness, sleeplessness, loss of appetite, some slight fever, *clonic* spasms of the muscles of the tongue, of the neck, of deglutition, and of respiration. The spasms become more and more generalized; swallowing is impossible; the mouth is filled with ropy mucus; respiration is catchy, and the noise produced by spasm of the diaphragm is sometimes thought to resemble the bark of a dog. The disease usually kills in a week.

With most of the acute infections there is a *hyperleukocytosis*. Sondern believes that when the polynuclear white blood cells number more than 70 per cent. of the total number of the leukocytes pus is present.

It is always easy to correctly interpret the constitutional symptoms above detailed when there is a local lesion present. It is not so easy, nor by any means always possible, to determine whether the local infection alone accounts for the toxic symptoms, or whether there is also an infection of the blood with the pathogenic organisms—*i. e.*, a septicæmia. The severity of the symptoms is no guide, for with severe purely local infections there may be an intense grade of constitutional symptoms, and with milder blood infections the symptoms may be of only moderate severity. It is important to remember that a blood infection frequently occurs very shortly after the local infection has taken place and also that the blood infection may precede the manifestation of the local lesion. (See Osteomyelitis.)

Very frequently we shall be able to decide the question of blood infection by observing the course of the symptoms after the local lesion has been properly treated. If the toxæmia is dependent entirely upon the local trouble, then it should subside within forty-eight to seventy-two hours after this has been relieved. If, however, it is dependent upon a blood infection, its manifestations will continue unabated in spite of a subsidence of the local lesion.

In all such latter cases, and also in those where from the

severity of the constitutional disturbances we suspect the existence of a blood infection, we should have a blood culture taken. A positive culture is conclusive evidence of a septicæmia, but a negative one is not to be construed as similarly conclusive that a blood infection is not present.

The cases of cryptogenic septicæmia—*i. e.*, cases of blood infection for which no local lesion can be discovered—offer the greatest difficulty in diagnosis. Only by continued observation and by a gradual exclusion of all other maladies can we hope to arrive at a diagnosis in these cases; this is always to be confirmed by a blood culture. It is well in these cases to remember that scratches, abrasions, hang nails, urethritis, pin pricks, etc., may be the portal of entry for the bacteria into the blood stream.

The constitutional symptoms arising from a local infection or from a septicæmia are readily distinguished from those due to a pyæmia, by the absence of the repeated rigors and secondary abscesses which characterize the latter condition.

The cases of cryptogenic pyæmia may be mistaken for cases of malaria. The absence of plasmodia in the blood, and an irregularity in the occurrence of the rigors, distinguish the pyæmic condition. It is not wise, in doubtful cases, to at once administer quinine, for the antipyretic action of this drug disturbs the temperature curve and so deprives us of one of the best differential diagnostic signs.

The tetanic spasms induced by the toxins of tetanus may in rare instances be confused with those due to *strychnine poisoning*. In the latter condition, however, the spasms are less intense and between them there is complete relaxation, whereas in tetanus there is no complete relaxation between the spasms.

The clonic spasms attending hydrophobia at once distinguish this malady from tetanus, in which affection the spasms are of a tonic character.

THE LOCAL LESIONS RESULTING FROM WOUND INFECTIONS.

Here, as in dealing with the constitutional manifestations of wound infections, we will not consider the local evidences produced by the tubercle bacillus, the syphilitic virus, the leprosy bacillus, or the actinomyces. These lesions are minutely described in the succeeding chapters.

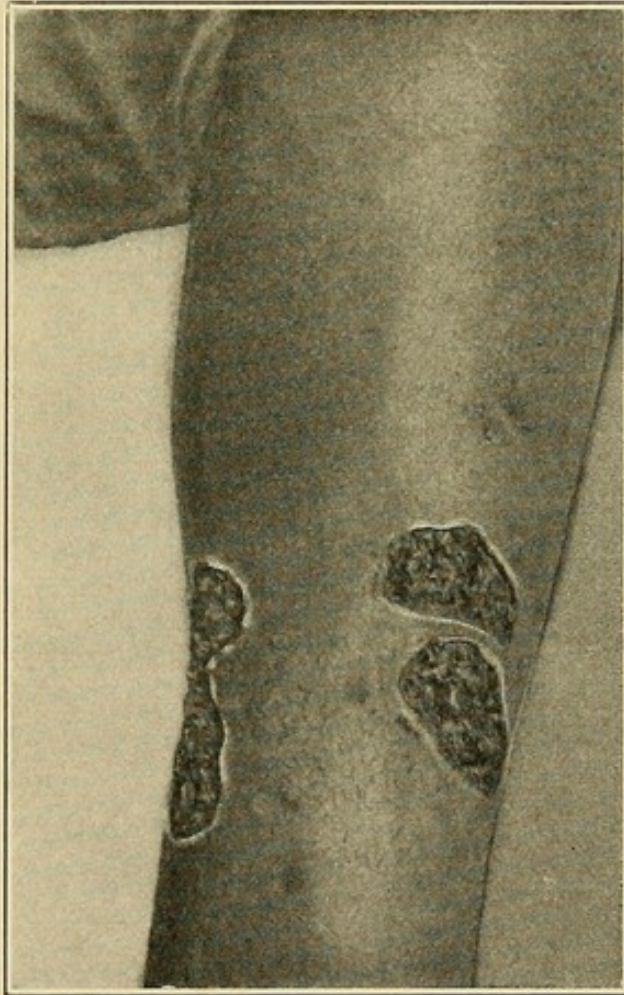
An infection of the tissues with the *pyogenic bacteria*, of which the *staphylococcus pyogenes aureus, albus and citreus*, the streptococcus pyogenes, the bacillus coli communis, the bacillus pyocyaneus, the pneumococcus, the bacillus typhosus and the gonococcus are the most important, leads to suppuration. If the pus is localized, the condition is spoken of as an abscess; if it is widely distributed in the cellular tissue the process is termed a cellulitis. An *acute abscess* forms a hot, painful swelling, hard and brawny at the outset, and later on becoming soft, elastic, and fluctuant. If it is superficially located, the overlying skin is hot and reddened. With *acute* pus formation there is considerable hyperleukocytosis, the polynuclear cells, according to Sondern, constituting over 70 per cent. of the total number of white cells. There is never any difficulty in recognizing a superficial acute abscess; the deeper ones are sometimes very hard to make out, and inasmuch as fluctuation is not always to be obtained, it is well to aspirate in every case where we suspect the presence of deep-seated pus. Abscesses lying over and accompanying the larger arteries share the pulsation of the vessels and over them can be heard a systolic murmur; they may consequently be mistaken for aneurysms. The error is especially apt to arise in the case of chronic abscesses which are not attended by local heat and redness; it will be avoided if we remember that aneurysms are usually compressible and have an *expansile pulsation* which abscesses never possess.

When an abscess has been opened and does not completely heal, a communication will often persist between the original seat of the disease and the surface of the body; such a communication is known as a *sinus* or *fistula*. A *sinus* is a granulating tract which penetrates into the tissues, and

is open at one end and closed at the other. A *fistula* is an abnormal communication between two cavities or between a cavity and the external surface.

Infection of the superficial tissues sometimes results in their molecular death, which gives rise to *ulceration*. The ulcers which follow *infection with the pyogenic bacteria* have an ash-gray, or dirty yellowish color; their edges are sharply

FIG. 5



Granulating ulcers of the leg, resulting from infection with pyogenic bacteria.
Note the sharp edges and the thickened margins.

cut; their base is fixed to the underlying structures; their margins are thickened, inflamed, and œdematous; their surface is non-granulating, and their discharge is considerable in amount, thin, sanious, often irritating and offensive, and rarely purulent.

Gangrene—*i. e.*, the simultaneous loss of vitality of a considerable area of tissue—may likewise result from infec-

tions when the bacteria, either by embolism or thrombosis, cause an obstruction of the bloodvessels and so interfere with the circulation of the affected part. This form of gangrene is always a septic one; the affected tissues rapidly disintegrate, become black, green, and yellow, the overlying cuticle is raised from the true cutis by blebs which contain a fetid serum. In the cases in which the infecting organisms are of the gas-producing types (especially the bacteria *aërogenes capsulatus*) the tissues crackle on palpation.

Carbuncles and *boils* are described in detail on page 126.

Infection of the smaller lymphatics of the skin, or of the mucous membranes, with the streptococcus *erysipelatis*¹ (Fehleisen) gives rise to *erysipelas*. This infection manifests itself by a uniform, bright, rosy-red rash with sharp, slightly elevated borders; it is not attended with much swelling except when the affected part is rich in loose areolar tissue—*e. g.*, scrotum or eyelids; it disappears on pressure and tends to spread from its initial site more or less rapidly, either with a continuous margin or in jumps, leaving a healthy interval between the areas involved. The infected part feels stiff and burns, but it is not usually painful; as the rash fades away, it leaves the skin stained slightly brown, and covered with a fine branny desquamation. Sometimes the epidermis is raised into vesicles and bullæ, and in some instances the infection invades the deeper lymphatics and occasions suppuration.

The uniformity of the redness which goes with erysipelas distinguishes this affection from a lymphangitis, in which the redness is in streaks or lines.

The redness of the skin attending a superficial abscess or suppuration in the subcutaneous cellular tissue very strongly simulates an erysipelatous redness, but it is not so sharply defined, nor are its edges elevated.

The dermatitis due to iodoform and its allied preparations may strongly simulate erysipelas. The lesion, however, is strictly limited to the region which is covered by the toxic agent, the constitutional symptoms are not so severe, and the redness at once subsides when the irritating material is removed.

¹ Some bacteriologists consider that the streptococcus of erysipelas is identical with the ordinary streptococcus pyogenes.

Erythema nodosum is attended with a slighter grade of constitutional disturbances than is erysipelas; the lesion occurs most often on the legs, in young women of a rheumatic tendency, and the redness is more sharply defined.

The local lesion resulting from *anthrax infection* (malignant pustule) is fully described on page 126. A general infection with anthrax bacilli without external lesion is sometimes found in those who handle hides, for which reason the malady is termed wool-sorters' disease. The organisms gain entrance either through the intestinal or respiratory tracts. If through the latter, a septic pleuropneumonia develops; if through the former, a septic gastroenteritis.

The local lesion of the mucous membranes of the genito-urinary system, eyes, synovial membrane, etc., following a gonococcus infection consists of a catarrhal or suppurative or ulcerative inflammation of these parts. They are minutely dealt with in the chapter devoted to diseases of the genito-urinary system, joints, etc., and are to be differentiated from the infectious processes due to other types of bacteria only by bacteriological examination.



PART II.

INJURIES AND DISEASES OF HEAD AND NECK.

CHAPTER IV.

TUMORS AND INFLAMMATORY DISEASES OF THE HEAD.

CONGENITAL TUMORS.

BABIES are sometimes born with swellings on their heads. Of these the most frequent though the least important is the *caput succedaneum*, an œdematous condition of the scalp, always lying on one side of the median line and disappearing spontaneously a few days after birth. More significant is the *cephalhæmatoma*—*i. e.*, a collection of blood beneath the periosteum, which, at its first appearance, is a soft, elastic, semifluctuating or doughy, irreducible tumor limited to one bone and never overstepping the boundaries of the sutures, but later on becoming harder and firmer and very often surrounded by a raised wall of bony tissue that simulates the margins surrounding a depressed fracture. Most significant is the *congenital protrusion* of the cranial contents, brain and membranes or membranes alone or vascular sinuses, through an opening in the skull *situated along the meeting place of one or more of the bones*.

These abnormal openings in the skull are situated in the order of their frequency of occurrence:

1. On the back of the head, above or below the occipital protuberance.

2. In the region of the root of the nose, either nasofrontal between the nasal and frontal bones, or naso-orbital.

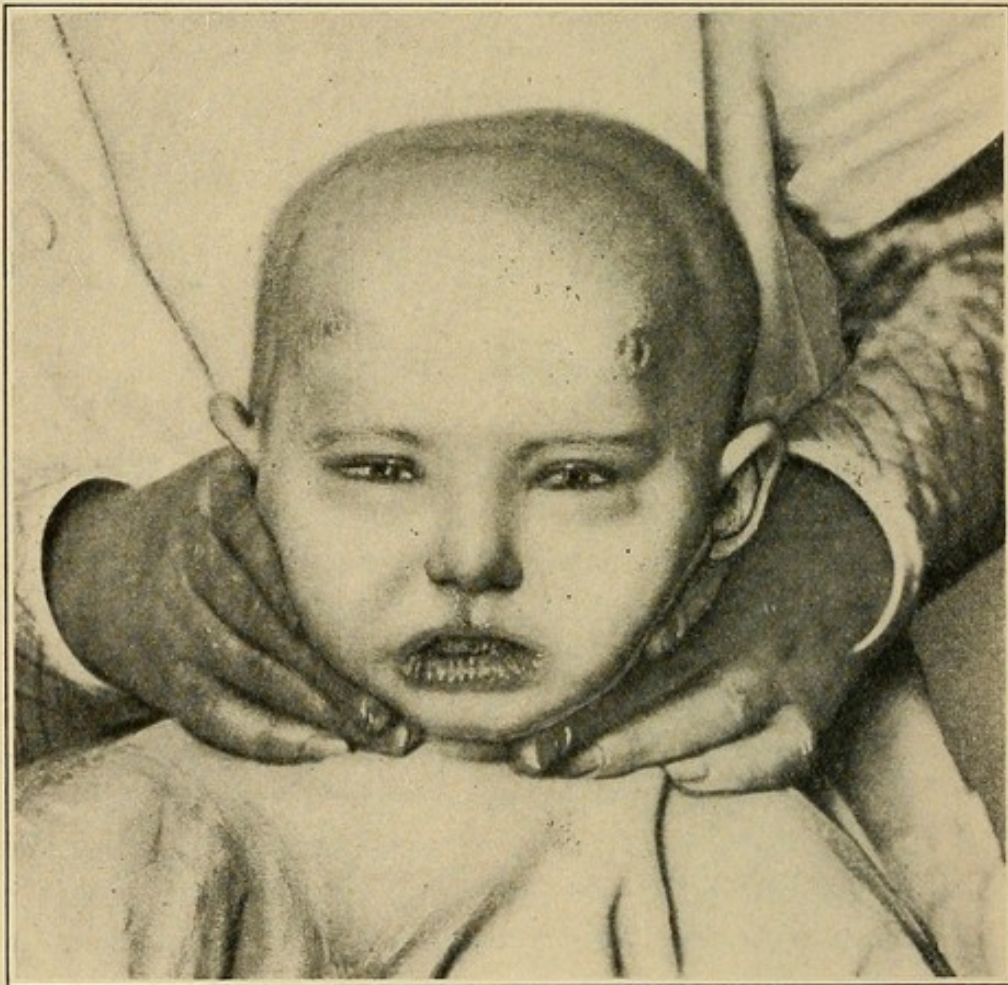
3. In the region of the sagittal suture.

4. In the region of the mastoid or the condylosquamosal suture.

5. At the base of the skull between the ethmoid and sphenoid bones, or between the ethmoid and nasal bones.

The last two sites are infrequent.

FIG. 6



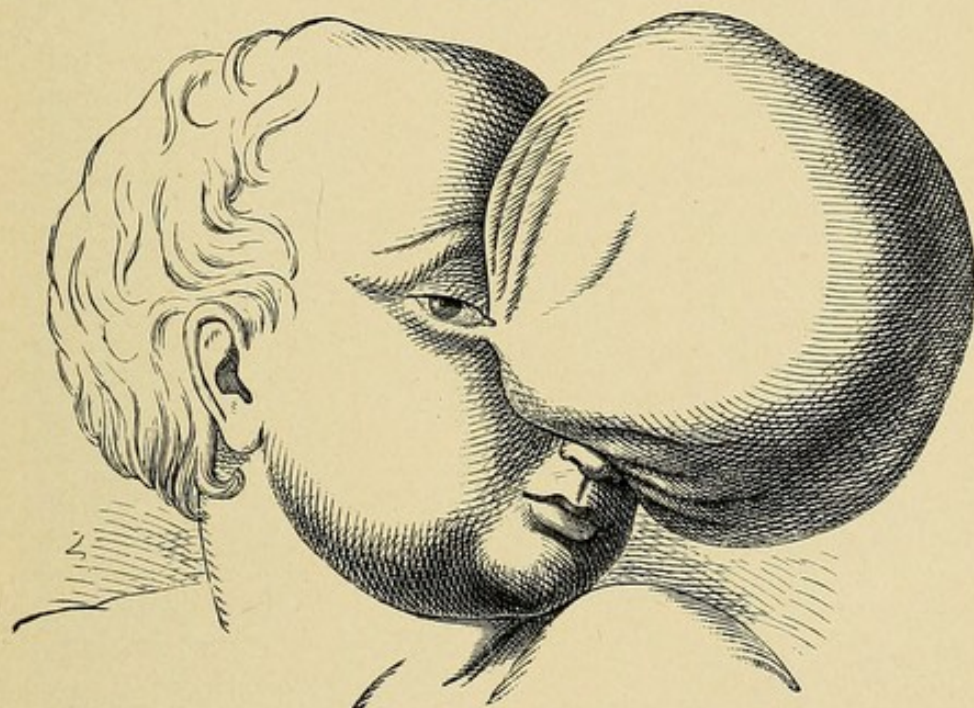
Cephalhæmatoma over the left parietal bone. Note its sharp limitations by the frontoparietal suture in front, by the sagittal suture internally, and by temporoparietal suture below.

These hernial tumors are rounded or pear-shaped, with broad base and narrow pedicle, and pulsate. The overlying scalp is smooth or wrinkled or stretched or ulcerated, or it is the seat of telangiectasis.

If the tumor is cystic, fluctuating, and translucent, it is a *meningocele*.¹

If it is soft, elastic, and more or less opaque, depending upon the proportion of brain substance it contains, it is an *encephalocele*,² and if it is bluish in color, easily replaced into the cranial cavity, and has marked respiratory pulsation, it is a *cephalhæmatocele*—i. e., a prolapsed vascular sinus.

FIG. 7



Nasofrontal cephalocele. (Von Bergmann.)

An encephalocele or false meningocele will become tense on straining or crying only when its cavity communicates with the ventricle proper; in some of such cases pressure upon the tumor produces symptoms of cerebral compression.

Before a diagnosis of a cranial hernia is made the margins

¹ *True meningoceles*, which are of rare occurrence and consist of a sac-like protrusion of the arachnoid filled with cerebrospinal fluid derived from the subarachnoid space, are not to be confounded histologically with the encephalomeningoceles, which by reason of a thinning out of the brain cortex overlying the cavity of the protruded part while the channel of communication between this cavity and the ventricle proper becomes very narrow or entirely closed, form a cystic tumor covered apparently only by arachnoid (false meningocele).

² If only the brain cortex with its overlying membranes protrudes from the skull, the hernia is spoken of as a *kenencephalocele*; if the arachnoid covering the protruding brain and ventricle becomes the seat of a cystic degeneration with the formation of one or more cysts, the tumor is spoken of as an *encephalocystomeningocele*.

of the opening in the skull through which it emerges should be palpated or viewed with the Roentgen rays. The presence of an opening in the skull at once differentiates such tumors from the caput succedaneum and the cephalhæmatoma. These latter are, further, irreducible, and always to one side of the median line, and possess none of the characteristics of cranial herniæ.

At puberty these cranial herniæ may be confounded with pulsating dermoid cysts, but the latter do not occupy the same regions in the skull as do encephaloceles, they cannot

FIG. 8



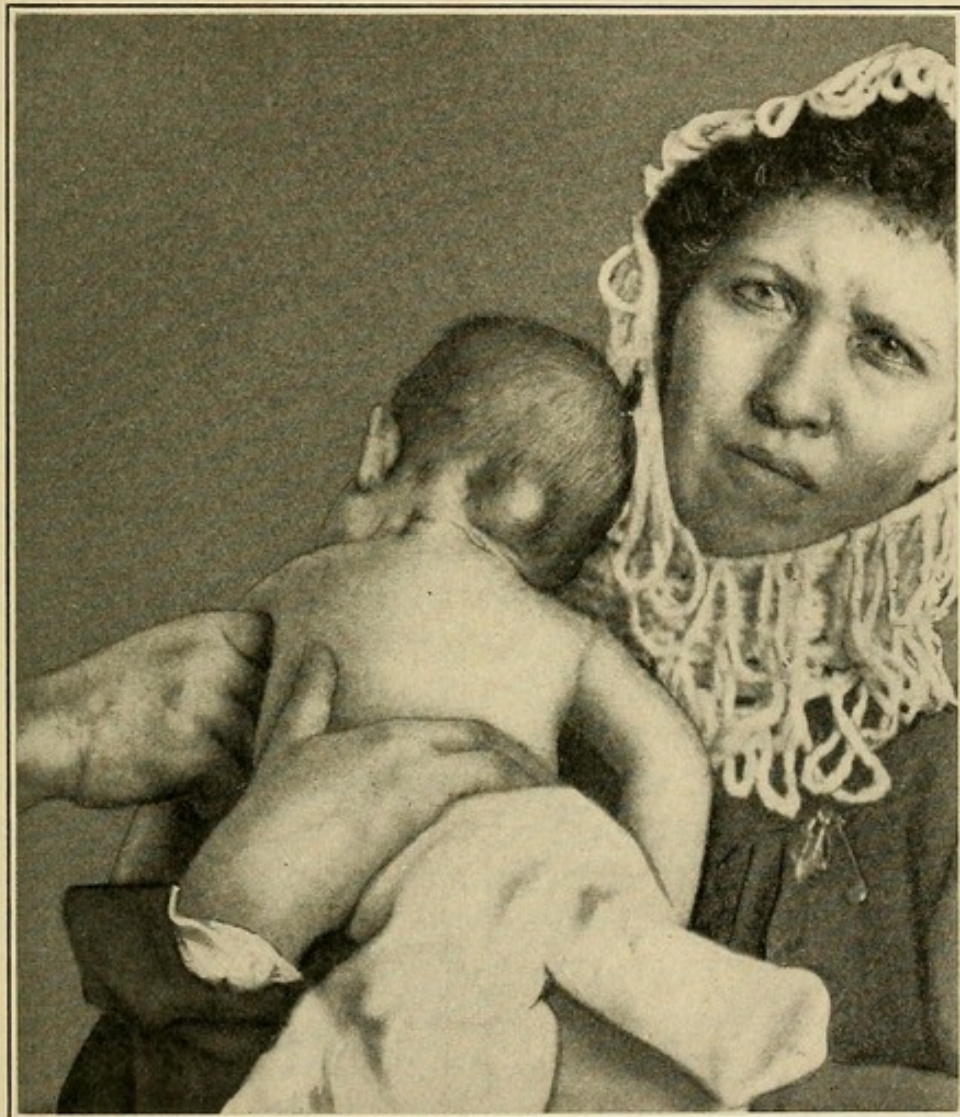
Inferior occipital cephalocele. (Von Bergmann.)

be reduced into the cranium, and on aseptic puncture yield an oily, cheesy material; whereas, encephaloceles and meningoceles yield a clear, yellowish fluid that contains grape-sugar.

Varicose veins of the scalp are to be differentiated from cephalhæmatoceles by their not reappearing after reduction, while the scalp surrounding the varix is firmly compressed. Cephalhæmatoceles deriving their blood from within the cranial cavity reappear in spite of such circular compression of the scalp.

Cephaloceles protruding through the nose have been *mistaken* for *polypi*. Fenger claims that a wide separation of the eyes and a very high attachment of the pedicle of the

FIG. 9



Cephalocele protruding through the occipital bone below the external occipital protuberance, and through the atlas.

supposed polypus are suggestive of encephalocele. Aspiration alone can decide the diagnosis in these cases. The aspirated fluid should contain grape-sugar if the tumor is a cephalocele.

NEOPLASMS OF SCALP.

Aneurysmal tumors, lipomata, and papillomata offer little difficulty in their diagnosis.

Sebaceous Cysts.—These are situated in or underneath the skin, and form tense, soft tumors of hazel-nut to walnut size. They are usually multiple. They are to be differentiated from

Dermoid Cysts.—These are more deeply situated, usually under the deep fascia and in a shallow trough of bone; they are adherent to the periosteum and bone and are consequently less movable than sebaceous cysts. Their favorite sites are on the outer and upper wall of the orbital region, the glabella, the temporoparietal region, the mastoid region, and the large fontanelle. They are congenital, grow very slowly, and, as a rule, are not noticed until puberty. The trough of bone in which the cyst lies may be very deep; occasionally its floor is entirely absorbed and the cyst thus comes to lie directly upon the dura, whose pulsation it shares (pulsating dermoid cyst). Such *pulsating cysts* are to be distinguished from encephaloceles by their location (situated in the neighborhood of the eye, about the margin of the orbit), by their irreducibility within the cranium, and by aseptic aspiration. (Dermoids contain an oily fluid, encephaloceles a clear, yellowish fluid which has a small amount of grape-sugar.)

Carcinoma of the Scalp.—This is most frequently found in the frontal or parietal regions. It is usually of the rodent ulcer type, but sometimes occurs as deeper infiltrations which originate in the sebaceous glands and hair follicles.

The tumor has the usual characteristics of carcinoma; it is hard, fixed upon the skull, with indefinite outlines, and is somewhat painful and tender. When it breaks down the ulcer that is formed has hard, everted edges, and the base is covered with unhealthy, easily bleeding granulations and crusts.

One of the superficial forms of carcinoma of the scalp resembles lupus. It commences with multiple, flattened papules, the epithelial covering of which is excoriated; as

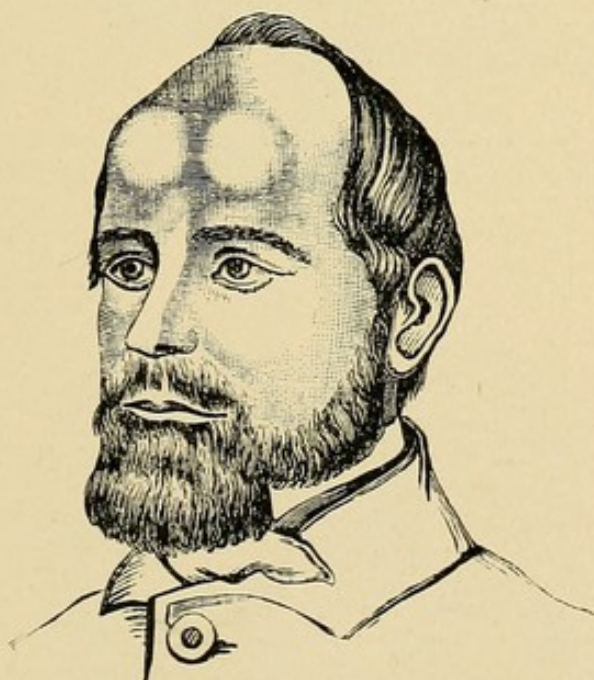
new papules form in an advancing convex border the old ones cicatrize. The diagnosis from lupus is made by the absence of all reaction after tuberculin injection.

Sarcoma of Scalp.—Sarcoma of the scalp is rare; it is usually secondary to sarcoma of the periosteum or diploë or dura.

DISEASES OF THE SKULL.

Tuberculosis of the Bones of the Skull.—This involves most frequently the frontal and parietal bones, and, as a rule, the affection is secondary to tuberculous lesions in other organs. The primary form of the disease first manifests itself by a circumscribed, œdematous, tender swelling of the overlying soft parts, which goes on to cheesy degen-

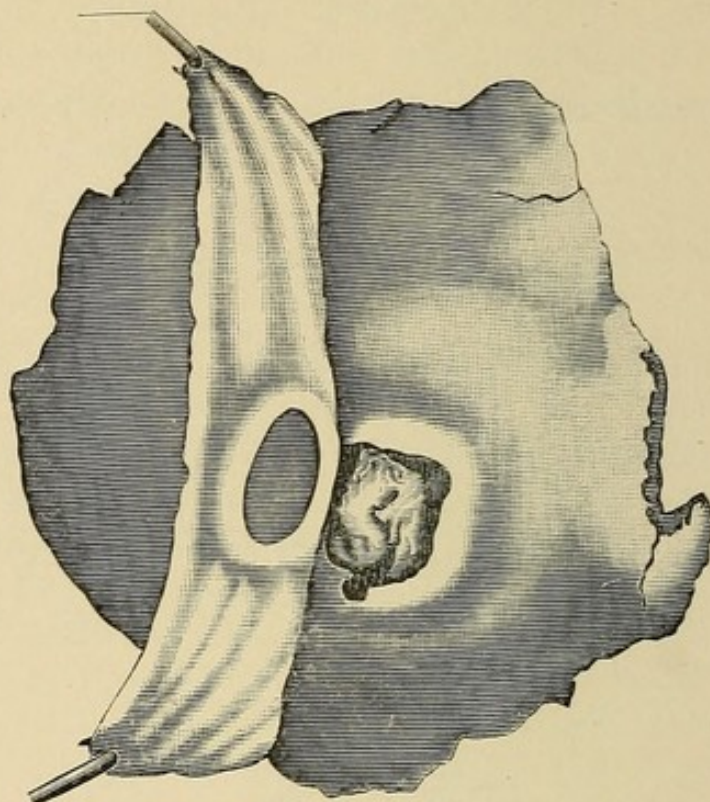
FIG. 10



Pneumatocele resulting from necrosis of frontal bone. Note the location over the frontal sinuses. Similar subperiosteal, air-containing, elastic and painless tumors are frequently found over the mastoid and over the superior maxilla.

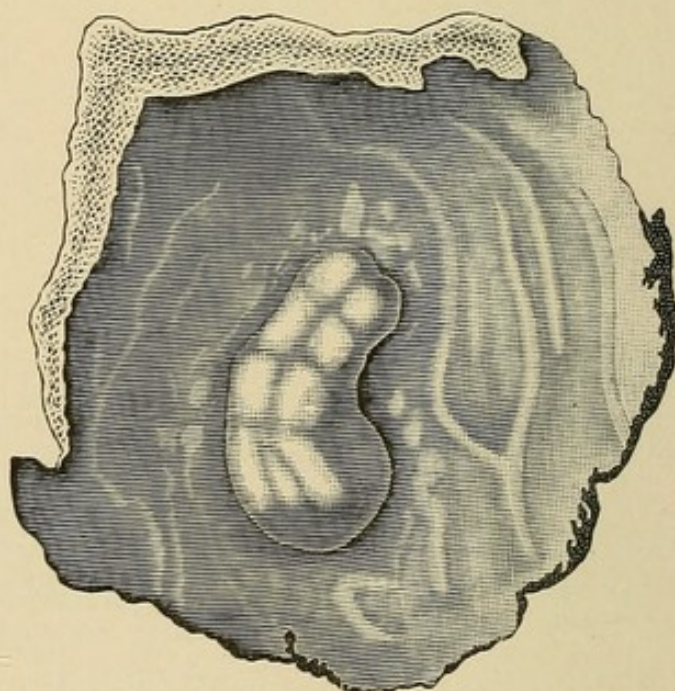
eration, abscess formation, and ulceration. The tuberculous disease eats its way inward toward the membranes and sinuses of the brain, as well as outward toward the scalp. Perforation outward occurs late on account of the firmness of the facial layers. Perforation inward is followed by meningitis or general tuberculosis.

FIG. 11



Tuberculosis of skull, the pericranium having been reflected back. Note the worm-eaten character of the sequestrum. (Duplay and Reclus.)

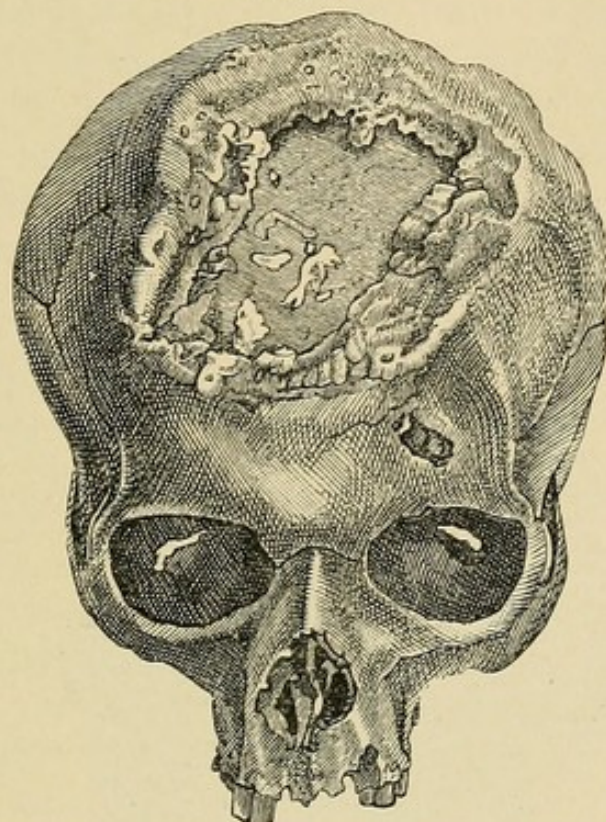
FIG. 12



Tuberculosis of skull seen from the internal aspect of the bone. Note the large tuberculous fungosities. (Duplay and Reclus.)

The diagnosis is made from the chronic course of the disease, the presence of the tubercle bacilli in the cheesy

FIG. 13



Syphilitic necrosis of the skull. (Von Bergmann.)

FIG. 14



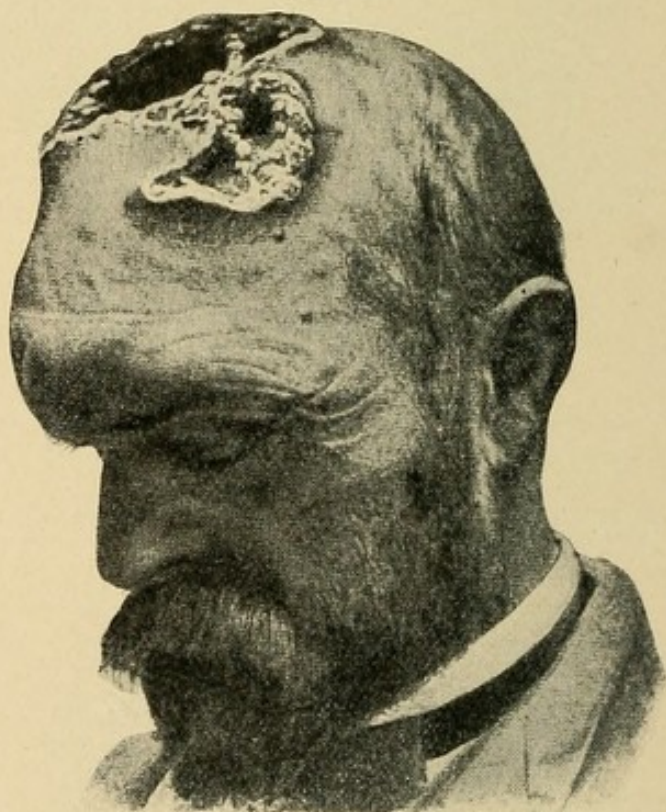
Hereditary syphilis: gummata of the cranial bones. Child, aged eighteen months. (Koplik.)

material, and from the presence of *tuberculous lesions in other organs*.

Syphilis of the Skull Bones.—This is likewise found most frequently in the frontal and parietal bones. It may be a primary process in the pericranium or diploë or it may be secondary to syphilis of the *scalp*.

Single and circumscribed or more diffuse and multiple, soft, elastic, flat tumors, having no inflammatory evidences, and with no tenderness, are indicative of gummata. They

FIG. 15



Large ulcerations resulting from extensive periosteal gummata. These are frequent sites for this malady. Note the undermined edges of the ulcers, etc.

rarely undergo ulceration. They cause bone caries and necrosis, with possible exposure of the meninges and sinuses, and also give rise to considerable and irregular thickening of the periosteum and bone.

Gummata are to be differentiated from tuberculous swellings of the bones by their lack of tenderness and by the absence of all evidences of inflammation. There is frequently, furthermore, a history of syphilis, and there are likely to be other syphilitic lesions. The administration of iodide of potassium is followed by a disappearance of the tumor.

Cephalhæmatoma.—The cephalhæmatoma of the newborn, and the subperiosteal or subfascial hæmatoma of later life, form soft, elastic, semifluctuating or doughy tumors which are limited to one bone, and never overstep the boundaries of the sutures. The swelling is surrounded by a ridge. In the newborn a cephalhæmatoma that is surrounded by a considerable ridge might give one the impression of an encephalocele. But these cephalhæmatomata are located over one or the other of the parietal bones, where an encephalocele never occurs.

Osteomata.—Osteomata of the skull are hard, sessile, smooth, painless tumors of very slow growth. They may occasionally be confounded with myelogenous sarcoma, from which they are to be differentiated by their slower growth and their painlessness.

Sarcomata.—Sarcomata originate in the periosteum or diploë; they grow very rapidly, involve the scalp early, and undergo ulceration, forming large fungous, bleeding masses. They may be very vascular and have distinct cardiac pulsation. A sarcoma of the diploë is in its early stages covered by a thin lamella of bone (the outer table), which crackles on palpation (the characteristic egg-shell crackle of myelogenous sarcoma). This feature enables us to differentiate a diploic sarcoma from a sarcoma of the dura, which has involved and then absorbed the overlying bone, and which does not manifest this crackling sensation. In the later stages of diploic sarcoma there is no characteristic egg-shell crackle, as the outer table has been gradually absorbed by the neoplasm. Fragments of it, however, especially at the margins of the tumor, remain, and these fragments are valuable diagnostic evidence of diploic as against dural sarcoma. Excessive new formation of bone at the base of the tumor is further evidence of a bony as against a dural growth. Dural sarcoma in its early stages is further likely to be reducible within the cranial cavity, and is more apt to give rise to subjective symptoms of cerebral compression, such as headache, convulsions, etc.

Akeidopeirastik, *i. e.*, puncture of the tumor with a needle, is another valuable method for differentiating dural from diploic sarcoma. If the exploring needle when it is

introduced into the tumor impinges upon bones it indicates a diploic sarcoma that has not penetrated the vitreous lamina of the bone.

Aneurysm of Bones.—Aneurysm of the bones resembles very much a diploic sarcoma. It forms a soft, non-tender

FIG. 16



FIG. 17

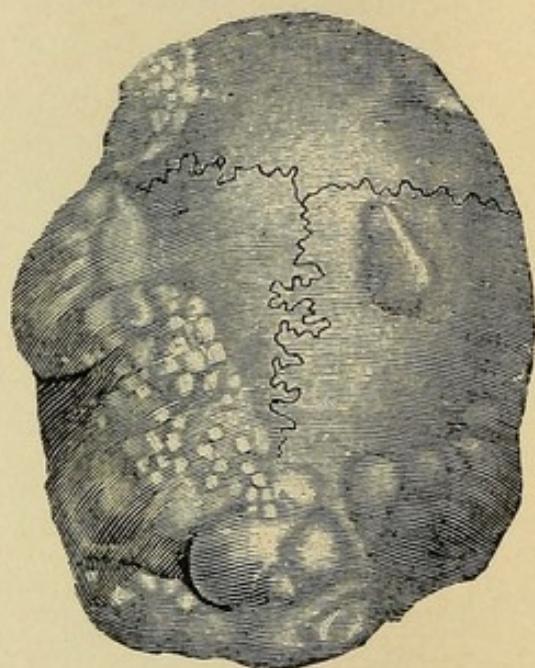


FIG. 16.—Diploic sarcoma of parietal bone. (Albert.)

FIG. 17.—Aneurysms of the skull bones. (W. Busch.)

tumor with expansile pulsation, and over it can be heard a systolic murmur and a bruit. It is easily reducible (*i. e.*, made to disappear) and stays reduced when the carotid artery is compressed. The opening in the bone through which the tumor appears can be palpated when the latter is reduced.

CHAPTER V.

INJURIES OF THE HEAD.

INJURIES of the head derive their chief clinical significance from the character and extent of the lesions the trauma produces in the brain and its enveloping membranes. In dealing with cranial injuries the diagnostician's main concern is to determine whether a cerebral lesion is present, and, if so, what its nature and localization are. Wounds of the soft parts are important only in so much as they open up an avenue for primary or subsequent infection of the brain and meninges; while the wounds and fractures of the bony capsule are of interest partly because the fragments of the bone may be depressed, and so cause compression of the brain, partly because the application of a force sufficient to fracture a bone will probably also result in cerebral concussion, contusion, or laceration, or in rupture of bloodvessels, and also because a secondary osteomyelitis may follow the injury and at a future time give rise to cerebral or meningeal lesions.

The presence of *cerebral complications* is always to be determined from the symptoms. If there are or have been no cerebral symptoms, such as headache, vomiting, stupor, unconsciousness, paralyses, convulsions, slow pulse, and slow respiration, there is, as a rule, no complicating cerebral condition. It is to be remembered, however, that the injury may have occasioned very slight primary cerebral symptoms and yet have produced a rupture of the middle meningeal artery or of one of the vascular sinuses, in virtue of which there will be manifested within a few hours the evidences of cerebral compression. It is always wise, therefore, to be guarded in estimating the severity of any cranial injury immediately after its infliction, and not to express an opinion as to the existence of cerebral complications until twenty-four hours have elapsed.

The nature of the cerebral injury is determined from the character of the symptoms.

CONCUSSION.

Concussion is always transient in its manifestations, the chief of which are unconsciousness and slow pulse.

Mild Form.—In the mildest cases the individual is stunned or dazed, possibly unconscious, but presents no marked changes in pulse, respiration, or temperature.

Moderately Severe Forms.—In the moderately severe forms the patient becomes dizzy, sees spots before his eyes, has tinnitus aurium, and falls to the ground unconscious, the muscular system being weak and relaxed. The face is pale, the expression blank, the eyelids closed, the pulse small, feeble, and slow, and the respirations very superficial. The temperature is subnormal. Recovery is the rule. After a little while the patient takes a few deep breaths, opens his eyes, stares around, and gradually returns to consciousness, the pulse becoming stronger and more rapid and the respirations deeper. Memory of the accident is usually lost; at times there is amnesia of the facts preceding the injury.

Severest Forms.—In the severest forms the coma is deeper, the pulse slower and irregular, the respirations shallow and irregular, and the cornea insensitive. The pupils may be contracted or moderately dilated and react to strong light. Urine and stools may be retained or they may be passed involuntarily. Repeated vomiting occurs, especially directly after the injury. This condition lasts for hours or even days, but reaction usually follows, the pulse becoming stronger and more rapid, the respirations deeper, the skin warm, and consciousness returning. In the fatal cases the coma becomes deeper, the pulse slower, until just before death, when it is rapid and feeble, and there are convulsions and paralyses.

Reaction from concussion is, as a rule, followed by a period of exaltation, during which the patients suffer with headache and have a hard, rapid pulse.

Simple concussion is only to be diagnosticated when the reaction is prompt. The longer the coma lasts, the more likely are there to be other circumscribed or diffuse cere-

bral lesions. In the fatal cases there are usually other cerebral lesions. It is further to be remembered that the reaction may pass into or be followed by the evidences of acute cerebral compression. *Transient loss of consciousness and slow pulse are the striking evidences of concussion.* Continued unconsciousness points to some other cerebral lesion.

COMPRESSION OF THE BRAIN.

Compression of the brain is not transient. Unless it is relieved by operation it will last until the compressing agent is removed by absorption or until the patient dies. It follows intracranial blood extravasation, depressed fractures of the skull, penetrating foreign bodies, and intracranial inflammatory exudates, and is usually complicated by cerebral concussion or other cerebral lesion.

Its chief manifestations are slow pulse and respiration, deep coma, and choked disk, besides the local symptoms from the area which is directly compressed. (See p. 70.)

Almost characteristic of compression resulting from *rupture of the middle meningeal artery* is a lucid period between the reaction from the primary concussion and the first evidences of compression.

With *rupture of the vascular sinuses or of the vessels of the pia mater* this lucid period is of considerably longer duration. If with such injuries there happens to be an external wound, so that the blood can flow out of the skull, its stream is a continuous one and is to be readily checked by compression or packing of the outer wound. If the internal hemorrhage is small in amount, and localized to one area, there will be evidence of local compression from this area (see p. 70), but no general signs of compression.

If the internal hemorrhage accumulates rapidly and is extensive, the lucid interval may be altogether wanting. Small tears of the sinuses may not occasion any symptoms of compression, the bleeding being spontaneously checked by the compression of the walls of the sinus against the bony groove in which it lies.

Rupture of the cerebral carotid is rare and, as a rule, results in immediate death. In some instances a communication between the artery and one of the neighboring venous sinuses results, with the formation of an arteriovenous aneurysm, the prominent symptom of which is a pulsating exophthalmos.

CEREBRAL LACERATION.

With cerebral laceration the primary concussion is more severe and is followed by a disturbed function of the affected part. If this happens to be one of the silent areas, there will be no localizing symptoms; but if a region is involved whose function is definitely known, there will be the evidences of suspended function of the part. Unless the laceration is extensive, with considerable extravasation of blood, there will be no evidences of compression.

INJURIES OF CRANIAL NERVES.

Injuries of the nerves are followed by a temporary or permanent paralysis of the muscles which the affected nerves supply; the paralysis is located on the same side of the body as that on which the lesion exists, and thereby differs from a paralysis due to injuries of the motor and sensory nerve centres which occasion symptoms on the opposite side. (An exception to the latter is found in patients whose medullary motor decussation does not take place.)

Injuries of the first pair of cranial nerves result in loss of smell. Such injuries are often associated with lesions of the third inferior frontal convolution (Broca's centre), which occasion motor aphasia.

Injuries of one of the second pair of cranial nerves result in complete blindness on the affected side.

Injuries of the third nerve result in ptosis and slight exophthalmos; the eyeball cannot be moved upward, downward, or inward. The eye deviates outward and somewhat downward. The pupil is dilated and immobile, and accommodation is paralyzed.

Injuries of the fourth nerve occasion limitation of movement of eyeball downward and toward the paralyzed side, and diplopia on attempting to look down. The eye is deviated upward and slightly inward.

Injuries of the fifth nerve rarely occur alone. They result in anæsthesia of the face and cornea, paralysis of the masseter, pterygoids, and temporal muscles.

Injuries of the sixth nerve occasion limitation of movement of eyeball outward and diplopia.

Injuries of the seventh nerve occasion complete paralysis of the muscles of the face, soft palate, and orbicular muscles of mouth and eye. The face is flat on the affected side, the angle of the mouth droops, and the eyelids cannot be closed; if the patient is asked to smile or show his teeth the face is drawn to the sound side; if he tries to swallow fluids, these run out of the angle of the mouth on the paralyzed side. If the soft palate is not paralyzed, the lesion is below the geniculate ganglion of this nerve.

Injuries of the eighth nerve are usually associated with basilar lesions of the facial, and result in deafness on one side.

Injuries of the ninth, tenth, eleventh, and twelfth nerves are rarely found alone. Injuries of the twelfth would result in paralysis of one-half of the tongue, which would cause it to deviate toward the paralyzed side when it is protruded from the mouth.

The coma resulting from cerebral injuries must be differentiated from that due to opium poisoning, diabetes, nephritis, alcoholism, apoplexy, epilepsy, hysteria, and asphyxia.

The coma of opium poisoning is attended by very slow respiration, small rapid pulse, and extreme contraction of the pupils. The coma is not as deep as that from cerebral compression, and there are never unilateral symptoms of paralysis or loss of corneal or tendon reflexes.

The coma of diabetes is characterized by a smell of acetone in the breath and perspiration, and by the presence of considerable sugar in the urine.

The coma of nephritis is usually preceded by convulsions, and is characterized by a urinary odor of the breath, œdema of the legs, ascites, and a diminution in the quan-

tity of urine excreted by the kidneys. The urine may be bloody, is of high specific gravity, and is loaded with albumin and casts. In neither diabetic nor uræmic coma are there any unilateral paralyses.

Alcoholic coma is not as deep as that after severe cerebral injuries. The individual can usually be aroused, and there is an alcoholic odor to the breath. The dirty appearance, the maudlin resistance, delirium, and restlessness of the patient strongly suggest alcoholism. There are no unilateral symptoms.

It is to be remembered that a cranial injury may have been sustained by an alcoholic patient or by one who is a diabetic or nephritic; in all cases of doubt the individual should be carefully watched and repeatedly examined.

In epileptic coma the onset is preceded by a cry, biting of the tongue, and general convulsion. An epileptic's tongue is scarred, and there are usually scars upon the head and extremities as evidences of prior attacks. The coma is not very deep, the individual can be aroused, and then shows no signs of paralysis. The pupils are equally dilated.

In apoplectic coma the arterial pressure is very high and the arteries are rigid; the respirations are stertorous. Apoplexy is frequent in those of thick-set stature and in syphilitics. The unilateral paralyses which go with this condition make the differential diagnosis very difficult.

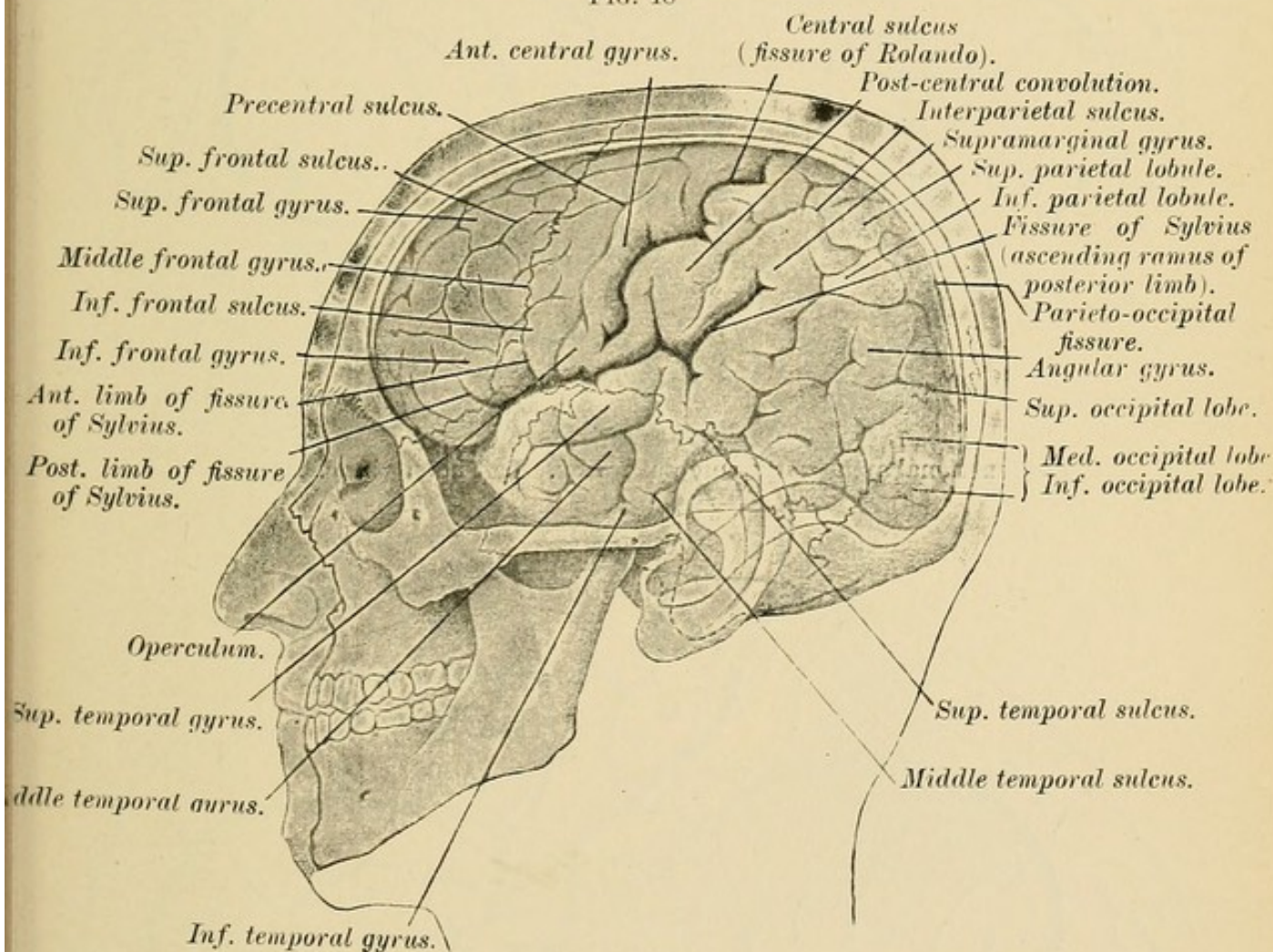
In all doubtful cases it should be the rule to observe the patient for twenty-four hours or more.

LOCALIZATION OF CEREBRAL LESIONS.

Every patient who has met with a cranial injury should be carefully examined for focal symptoms. Thus the functional power and activity of the muscles of the face and eye, the tongue, and of the extremities should be individually determined; the sensibility of the skin to touch, to heat, and cold should be tested; the condition of the reflexes should be ascertained. Tests should be made of the visual fields of both eyes, and for the various forms of aphasia; and cranial-nerve lesions should be examined for.

Hemiplegia.—Hemiplegia (paralysis of one side of the body) or convulsions of the muscles of one side of the body, point respectively to destructive and irritative lesions of the motor area of the opposite side of the brain—*i. e.*, the ascending parietal and frontal convolutions of the cortex. In this area the face is represented in the lowest part, the upper extremity and trunk in the middle section, and the lower extremity in the uppermost portion.

FIG. 18



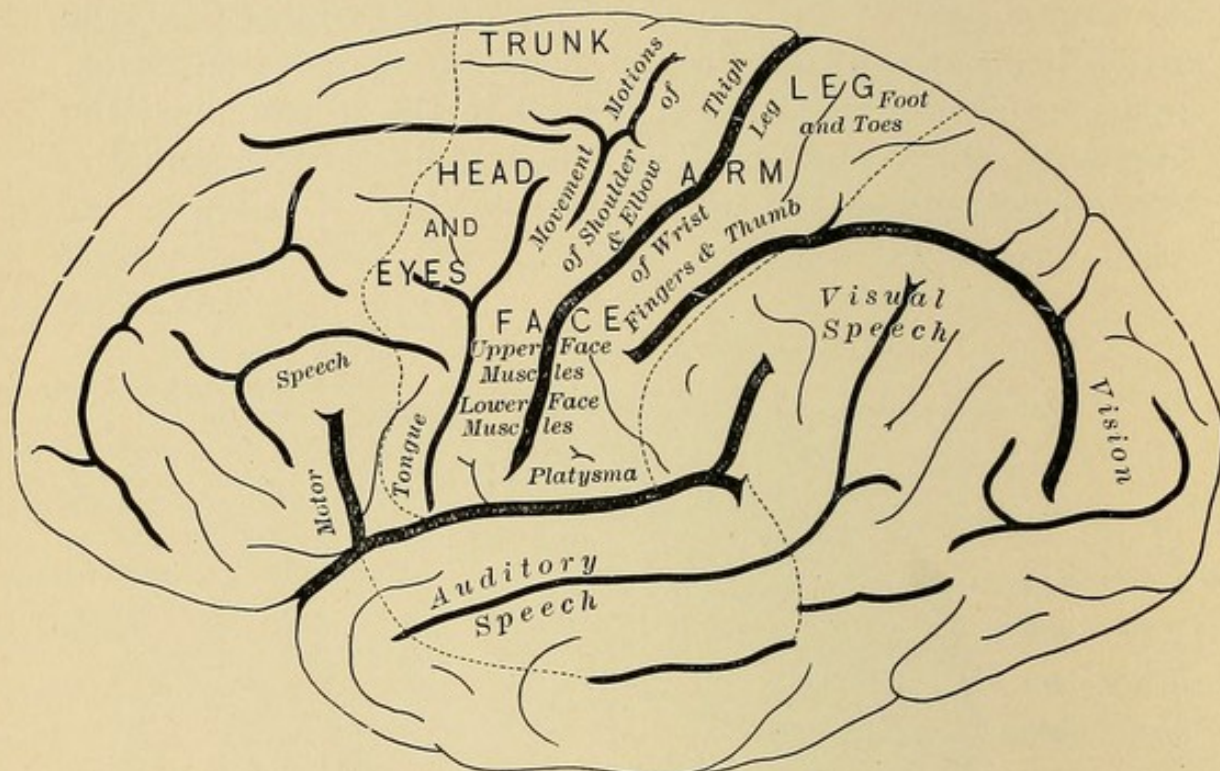
Lateral view of brain, showing fissures and convolutions. (Von Bergmann.)

Hemiplegia may also follow lesions in the internal capsule of the opposite side, lesions in the crus cerebri of the opposite side, lesions in the pons of the opposite side, and lesions in the medulla oblongata of the opposite side.

Lesions of the cortex necessary to produce complete hemiplegia must be more extensive than similar lesions of the

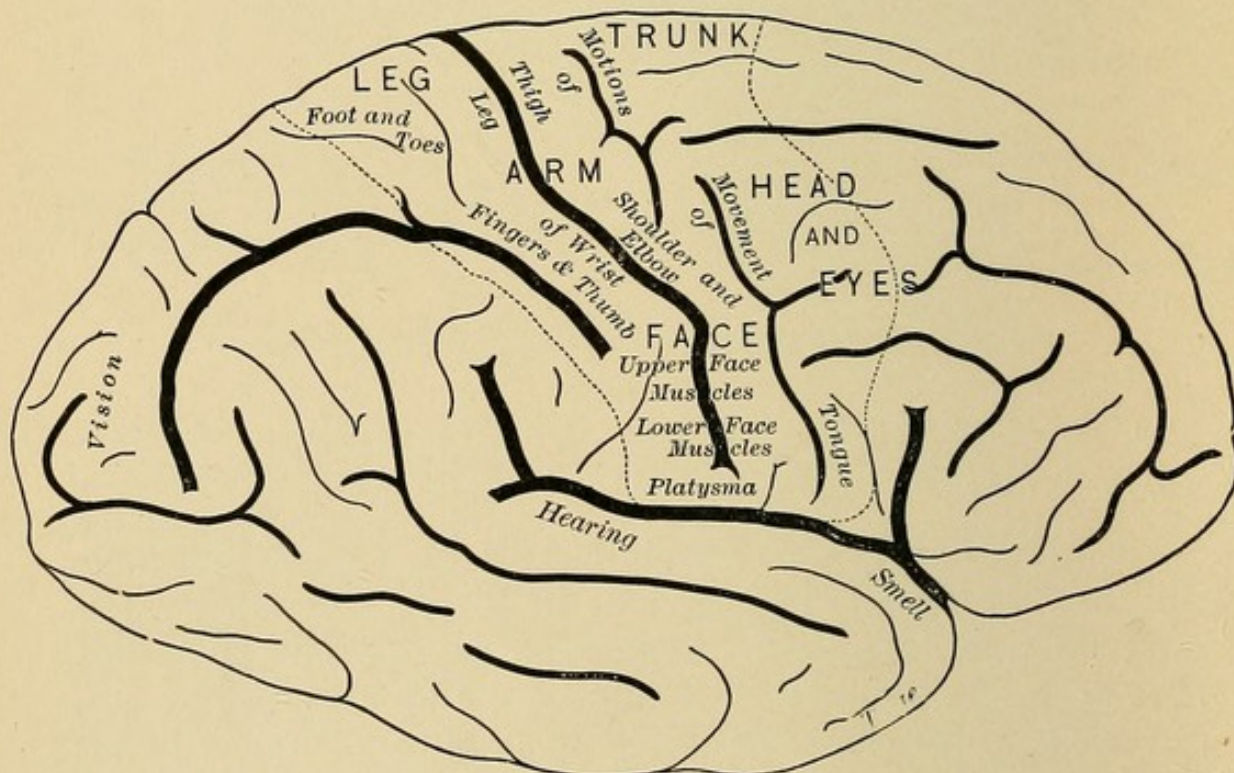
capsule, pons, or medulla. From the extent of the cerebral motor area it follows that hemiplegia due to cortical

FIG. 19



The functional areas of the cerebral cortex. Left hemisphere. (Starr.)

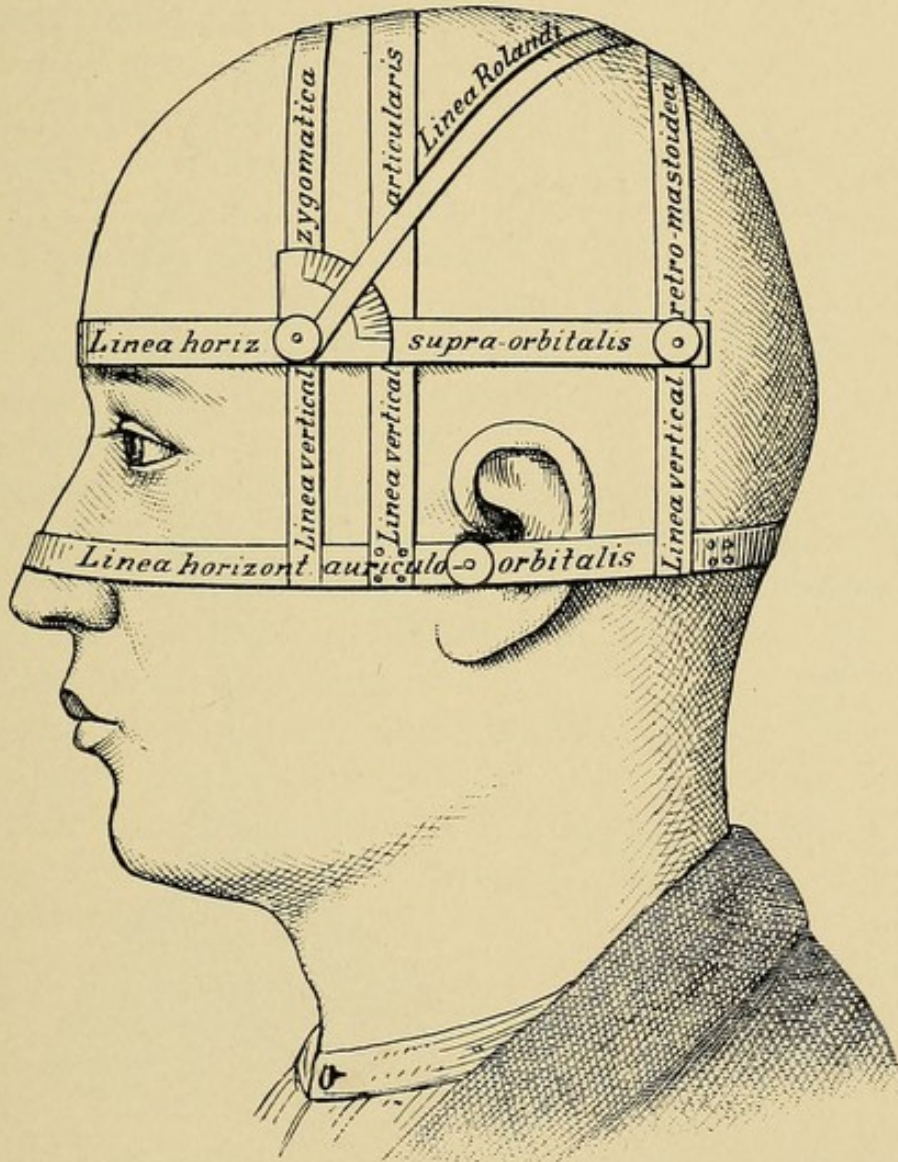
FIG. 20



The functional areas of the cerebral cortex. Right hemisphere. (Starr.)

lesions frequently commences in the arm or leg or face, and spreads with more or less rapidity to the entire half of the body. With lesions of the internal capsule, crus cerebri, pons or medulla, the paralysis usually involves the entire half of the body from the very onset.

FIG. 21



Craniometer. (Von Bergmann.)

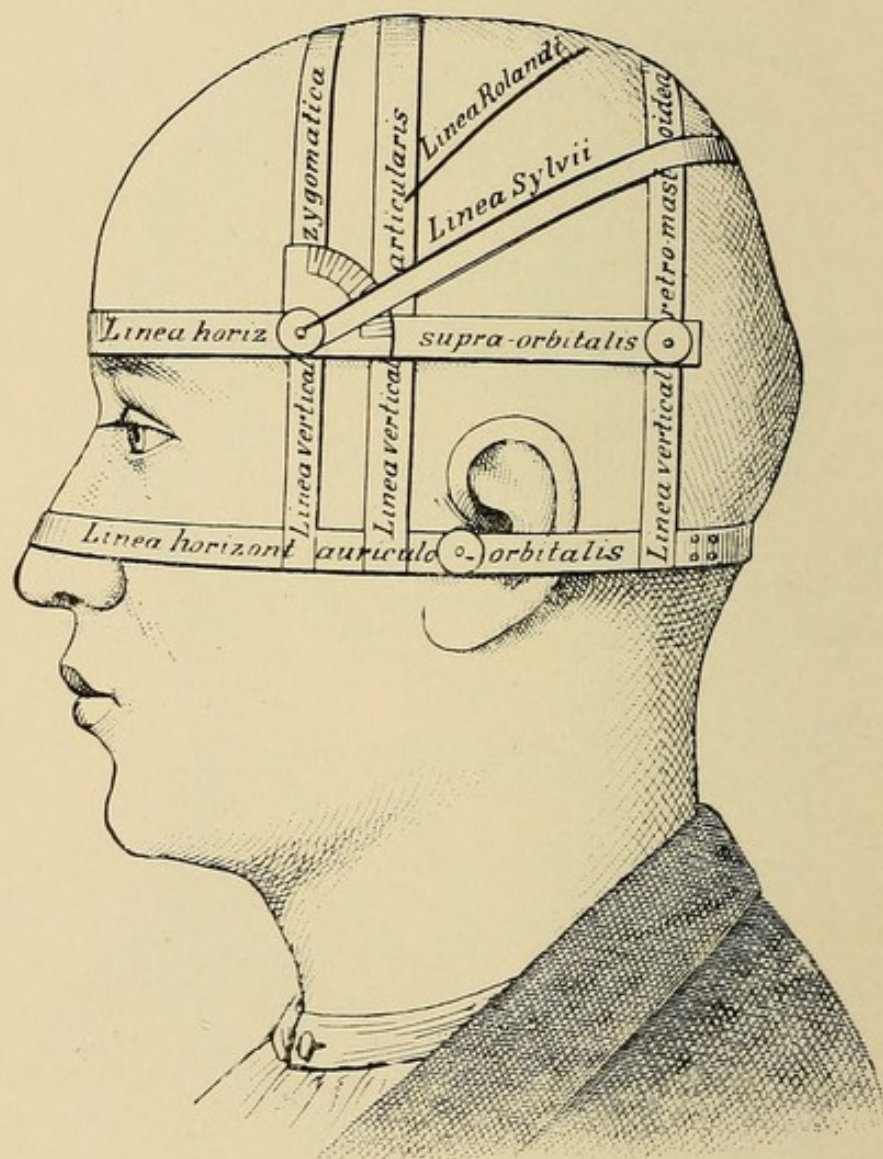
In hemiplegia due to *lesions* in the *internal capsule*, the muscles supplied by the upper branches of the facial nerve and the back muscles are usually not involved.

If in addition to the hemiplegia there is also hemianæsthesia and vasomotor disturbances, the lesion is usually located in the posterior portion of the internal capsule.

Hemiplegia due to *lesions* of the *crus cerebri* is always attended by paralysis of the muscles supplied by the third (oculomotor) nerve (see p. 77) of the side opposite to the hemiplegia.

Hemiplegia due to *lesions* of the *pons* is usually associated

FIG. 22



Craniometer. (Von Bergmann.)

with paralysis of the fifth, sixth, seventh, and twelfth nerves of the side opposite to the hemiplegia.

Hemiplegia due to *lesions* of the *bulb* of the *medulla oblongata* is associated with hemianæsthesia of the other side of the body. Hemiplegia of one side and hemianæsthesia of the other is termed "alternating hemiplegia."

It is to be remembered that although the paralysis from these several lesions usually involves the side of the body opposite to that on which the injury has been sustained, yet it may affect the muscles of the same side, either because the paralyzing factor is a *contre-coup* injury—*e. g.*, *contre-coup* rupture of the middle meningeal artery—or because the motor tracts do not decussate in the medulla.

Hemianæsthesia.—Hemianæsthesia and loss of muscle sense, with some ataxia of one-half of the body, are due to lesions of the cortical sensory centre of the opposite side of the brain, or to lesions of the internal capsule (posterior portion), crura cerebri, pons, or medulla oblongata. The cortical sensory centre overlaps the posterior portion of the motor area and extends a little farther backward. The points mentioned above for locating the site of the lesion in hemiplegia apply for the localization of the lesion producing hemianæsthesia.

Hemianopsia.—Hemianopsia, blindness of one-half of the visual field, is due (1) to lesions of the cuneus and upper part of the occipital lobe, or (2) to lesions of the optic tract behind the decussation (chiasm) of the same side as the blind halves of the retina.

Cortical lesions producing hemianopsia are usually associated with psychical or central mind blindness, in which condition visual sensations fail to summon forth any recollection of objects or circumstances acquired through previous education.

Lesions of the optic tract producing hemianopsia are usually associated with paralyzing symptoms of the oculomotor, trochlear, and trigeminal nerves of the same side as the blind halves of the retina.

With hemianopsia due to lesions *behind* the *geniculate bodies*, the pupillary light reflex is present; whereas with lesions in *front* of the *geniculate bodies* the pupillary light reflex is diminished or lost.

Complete blindness in one eye is always due to lesions of the optic nerve in front of the chiasm.

With *absolute hemianopsia*—*i. e.*, loss of light, form, and color sense—the lesion is more extensive than in *relative hemianopsia* (hemichromatopsia), in which condition the

light sense is present, but the form and color senses are lost.

Nystagmus.—Nystagmus and paralysis of some of the ocular muscles in both eyes, resulting in loss of co-ordinated movement of eyeballs, together with a staggering gait similar to that seen in cerebellar disease point to lesions in the *corpora quadrigemina*.

Aphasia.—*Motor aphasia*—*i. e.*, the inability to speak voluntarily, or to repeat words one after the other—is due to lesions of the third inferior frontal convolution and its association tracts of the left side in right-handed individuals, and of the right side of left-handed ones.

Word deafness, or *auditory aphasia*—*i. e.*, the inability to recall the spoken name of objects seen or heard or felt or tasted, or to understand speech and musical tunes, or to call to mind the objects named—is due to lesions of the posterior two-thirds of the upper temporal convolution and its association tracts.

Word blindness, or *visual aphasia*—*i. e.*, the inability to understand printed or written words or to recall objects the names of which are seen, or to write spontaneously, or to write the names of objects seen, heard, etc., or to copy and to write at dictation, or to read understandingly what has been written—is due to lesions of the angular gyrus and its association tracts.

Ataxia.—*Cerebellar ataxia*, characterized by a staggering gait, dizziness, and buzzing in the ears, is especially found after lesions of the cerebellar worm. These symptoms are also present after injuries to the labyrinth, and in fractures of the petrous portion of the temporal bone. With such fractures there is very likely to be paralysis of the facial and auditory nerves.

Rolling and rotation of the body around its long axis, with deviation of one eye upward and inward, and of the other downward and outward, are due to lesions of the *crura cerebelli*.

Cheyne-Stokes Breathing.—This is due to disturbed function of the respiratory and circulatory centres in the medulla.

Traumatic Diabetes.—Traumatic diabetes, polyuria, and albuminuria are due to lesions with disturbed function of the medulla.

Implications of the first and second cranial nerves point to lesions in the anterior cranial fossa; of the third, fourth, fifth, and sixth nerves, to the middle fossa; of the seventh and eighth nerves to the petrous bone, and of the twelfth to the posterior fossa.

THE DIAGNOSIS OF THE NATURE OF TRAUMATIC LESIONS OF THE SOFT AND BONY CAPSULES.

The scalp alone or the bony structure as well may be affected by the traumatism. Subcutaneous linear or fissure fractures of the vault of the skull without depression of the fragments cannot be made out by palpation. Symptoms of compression from ruptured vessels or from laceration of the brain cortex point rather to the presence of such fractures.

Subcutaneous fractures with depression of the fragments, but without the evidences of cerebral compression, must be differentiated from deep hæmatomata of the scalp. The margins of the ridge surrounding a hæmatoma of the scalp are soft and can be indented, whereas the margins of the ridge surrounding a depressed fracture have the very opposite characteristics.

Open wounds of the scalp should always be aseptically explored with the finger to determine the presence or absence of a fracture.

A fracture of the base is present if cerebrospinal fluid or brain substance is discharged from the nose or ear. Cerebrospinal fluid is distinguished from serum by its high albumin percentage, by its high percentage of sodium chloride, and in contradistinction to serum its flow continues in large quantities for days.

Fractures of the base usually cause paralysis of one or more of the cranial nerves. The discharge of blood from the nose, ear, or mouth, or an extravasation of blood beneath the conjunctiva or beneath the scalp in the mastoid region, with paralysis of one or more of the cranial nerves, speaks strongly for fracture of the base of the skull.

Middle meningeal hemorrhage, with evidences of compression, is usually a complication of fracture of the base.

CHAPTER VI.

INFLAMMATIONS AND NEOPLASMS OF THE BRAIN AND ITS MEMBRANES.

MENINGITIS.

IF a patient with an infected scalp wound or compound fracture of the skull develops gradually, three or four days after an injury has been sustained, a rise of temperature to 101° to 104° , a slow pulse, headache, restlessness; hyperæsthesia to light, sound, and touch; grinding of the teeth, and muscular twitchings, and then a paralysis of the arm or leg or entire half of the body, depending upon the area which is first affected, with increasing stupor, irregular respirations, and increasing pulse rate, we are safe in assuming that he has a cortical meningitis. If, instead of the paralysis of the arm or leg or entire half of the body, the patient shows paralysis of the muscles supplied by the cranial nerves, especially of the second, third, fourth, sixth, seventh, and eighth, with marked rigidity of the neck, we are equally safe in assuming that he has a basilar meningitis.

A meningitis, however, may follow other conditions than infected wounds of the scalp or skull—*e. g.*, sinus thrombosis, abscess of the brain, suppuration of the mastoid cells, thoracic suppurations, etc.; and we should be prepared to interpret the clinical manifestations above mentioned when they arise in the course of any of these maladies.

Should we be in doubt about the diagnosis we have in lumbar puncture and McEwen's sign¹—*i. e.*, a changed percussion note over the parietal regions—valuable aids in dispelling our uncertainty.

Lumbar puncture in meningitis yields a cloudy or purulent fluid containing flocculi of fibrin, polynuclear leukocytes,

¹ See p. 80.

pus, and micro-organisms. Cultures of the fluid will determine the nature of the infecting organism, whether streptococcus, staphylococcus, etc.

ACUTE SUPPURATIVE ENCEPHALITIS.

Should an acute suppurative encephalitis develop secondarily to an infected wound of the scalp or a depressed fracture of the skull with laceration of the brain, without a diffuse purulent leptomeningitis, its evidences would not be manifested until ten to fourteen days after the injury; then there would be a profuse discharge from the wound, the scalp would be swollen and œdematous, and the granulations would have a yellowish color. Retention of pus within the brain would be followed by fever, headaches, and possibly vomiting. If the abscess occupies an area whose function is known, there will be focal symptoms from this part—*e. g.*, hemiplegia, hemianæsthesia, hemianopsia, etc. (See pp. 70–77.) The later period at which symptoms develop, and the lesser intensity of these latter, differentiate acute suppurating encephalitis from purulent meningitis. It should be noted, however, that purulent meningitis, as a rule, coexists with an acute suppurative encephalitis and masks the symptoms which the latter occasions.

CHRONIC SUPPURATIVE ENCEPHALITIS.

A chronic suppurative encephalitis may likewise follow injuries to the head and also a number of other conditions—*e. g.*, chronic suppuration of the middle ear, mastoid, nasal and orbital cavities, suppuration and gangrene of the lung, malignant endocarditis, and some of the acute infectious diseases such as typhoid fever, cerebrospinal meningitis, etc. Its presence is, as a rule, readily determined, but its localization is attended with much greater difficulty, as its most frequent sites are in the temporal lobe and cerebellum, the function of which is not as yet well understood.

Very suggestive of the formation of a cerebral abscess after suppurative otitis media is the cessation of the ear discharge, with a sudden fall of temperature, the patient continuing to feel ill and having indefinite cerebral symptoms.

The cerebral symptoms, which are the same in all cases of chronic abscess, no matter what their causation is, consist of an altered mental character of the individual; he is irritable, dull, or stupid, and suffers from malaise. His temperature varies; it may be persistently low with little variation or there may be occasional chilliness or even a distinct chill with rise of temperature. The appetite is poor and the bowels are apt to be constipated. After a shorter or longer duration of these indefinite cerebral symptoms¹ the mental stupor increases and alternates with irritability and restlessness. Headaches, irregular temperatures, vomiting, and general septic appearances, together with the evidences of increased cerebral tension, develop. These last are McEwen's sign,² an inconstantly occurring optic neuritis, a slow pulse, headache which is sometimes located over the seat of the abscess, and tenderness of the head to percussion. This period is termed the active period of the abscess and at this stage it must be differentiated from acute meningitis and thrombosis of the lateral sinus.

In acute meningitis the onset and course are more rapid, there is marked hyperæsthesia to sound, light, and touch; the temperature is higher, from 101° to 104°; the pulse, though slow at the onset, becomes rapid and irregular; there are muscular twitchings, spasms, convulsions, strabismus, and pain and rigidity of neck. In doubtful cases, lumbar puncture will aid in making the diagnosis; for in meningitis the spinal fluid is turbid and contains pus cells and bacteria, whereas in cerebral abscess it is usually clear. In meningitis there is no choked disk.

In thrombosis of lateral sinus the temperature is high, and with the breaking up of the infected thrombus in the

¹ This period is known as the latent period of cerebral abscess.

² Normally the percussion note which is elicited with a rubber-tipped percussion hammer over the temporo-parietal-frontal region, while the patient is sitting up is dull, but with increased cerebral tension it changes to a higher-pitched and more resonant, almost tympanitic note, the resonance increasing as the tension rises.

sinus and metastatic lodgement of the infected fragments in the internal organs there are frequent chills and fluctuating temperatures between $97^{\circ}+$ and $109^{\circ}-$. There are apt to be mastoid tenderness, tenderness over the jugular, venous congestion of the scalp, and possibly exophthalmos. Choked disk is an early symptom.

Localization of the Abscess.—As is said above, it is not always possible to locate the site of the abscess. It is well to remember that after an injury the abscess may form superficially in the cortex at the site of the injury or it may develop in the deeper parts; and that after otitis media the abscess, as a rule, is in the temporal lobe if the primary site of suppuration is at the tegmen tympani or in the anterior mastoid cells; while if the primary suppuration is in the posterior mastoid cells or on the posterior wall of the middle ear, the abscess is likely to be in the cerebellum.

Abscesses in the *temporal lobe*, according to Freund and Pick and Starr, frequently give rise to "optical aphasia," a condition in which the patient cannot name the object which he sees. He can understand and recognize the name, can talk and describe the object, can repeat the name, and may even be able to name the object if he is permitted to touch or taste or smell it.

Abscesses in the *internal capsule* cause hemiplegia, hemianæsthesia, hemianopsia of the opposite sides.

Cerebellar abscesses cause vertigo, staggering gait (usually to the side on which the abscess lies), vomiting, diplopia, nystagmus. If the abscess presses upon one side of the pons and medulla, paralysis of the sixth and seventh cranial nerves results.

ASEPTIC SINUS THROMBOSIS.

The diagnosis of aseptic cranial sinus thrombosis is reservedly made when, after a long-continued diarrhœa or exhausting illness, such as carcinoma, pulmonary phthisis, etc., an individual suddenly commences to suffer with a unilateral headache, delirium, and somnolence. As the superior longitudinal sinus is the one that is usually thrombosed in this way, the diagnosis is materially strengthened

if there develops venous stasis and œdema of the frontal and parietal veins, distention of the veins of the orbit and eye, and nose-bleed.

INFECTIVE SINUS THROMBOSIS.

The recognition of aseptic sinus thrombosis is chiefly important to us as regards prognosis. It does not influence our therapy, for thus far no one has deemed it wise to interfere surgically to relieve the thrombosed sinus. Far different is it in infective sinus thrombosis, for here we can do a great deal by surgical measures; and if we would afford the patient the life-saving benefits of operation, we must make the diagnosis before the manifestations of pyæmia or deep septic intoxication develop; and it is not, in the majority of cases, difficult to make such an early diagnosis. Given a cause for sinus involvement, such as mastoid disease, erysipelas of the scalp, infected wounds of the scalp, or supuration in the nose, middle ear or orbit, a high rise of temperature to 104° or over, with or without a chill and headache and vomiting, should be viewed with great suspicion and the possibilities of sinus thrombosis should be borne in mind; and especially are such symptoms significant if a carefully made physical examination of all the other organs or a thorough inspection of the wound fails to reveal a cause for them.

The addition of increasing delirium, stupor, and vertigo, of focal symptoms, such as hemispasms, paralysis, aphasia, or some affection of the cranial nerves from the irritation and compression exerted by the inflamed thrombosed sinus upon the immediately surrounding nerves and brain centres, and of local evidences in the superficial or jugular veins into which the thrombosis has extended, such as painful œdema, local tenderness and abscess formation of the superficial soft parts, emptiness of the veins proximally to the thrombosis and distention distally to it, naturally make the diagnosis much more certain. Absolute certainty of diagnosis, as far as such a thing is possible in dealing with diseased conditions, is attained when the softening and disorganization of the thrombus permits

infected portions thereof to be carried into the general circulation and lodged in other viscera, with the resulting constitutional and local manifestations of pyæmia. With each lodgement there is a chill and rise of temperature to 105° – 108° , followed by a fall of temperature below the normal, and a profuse sweat. Such chills and fluctuations of temperature have no regularity; they may occur once or several times a day, or every other day or once in several days. In the organs in which the infected emboli have lodged there are the signs of metastatic abscesses. But not in all cases are these pyæmic symptoms manifested; instead the temperature in some cases remains continuously high, there is a profuse septic diarrhœa, septic roseola and rash, and an enlarged spleen. It is in these latter cases especially that the differential diagnosis from typhoid fever is very difficult.

Localization of Affected Sinus.—The location of the thrombosed sinus is determined from the site of the exciting cause thereof, from focal brain symptoms, and from local evidences in the superficial soft parts.

With *sigmoid sinus thrombosis* there is a circumscribed painful œdema along the posterior border and apex of the mastoid process, occasionally a subperiosteal abscess at the site of the mastoid vein, or a deep abscess in the suboccipital fossa corresponding to the condyloid vein; swelling and thrombosis of the internal jugular vein, which can be felt as a hard tender cord, and which occasions pain and torticollis (the patient holding the head toward the affected side to avoid the pain). Unusual emptiness of this vein in virtue of its thrombosis (especially to be detected in deep inspiration), swelling of the cervical glands, and hoarseness, dyspnœa, slow pulse, difficulty in swallowing, and spasm of the sternocleidomastoid and trapezius muscles, from compression of the ninth, tenth, and eleventh cranial nerves in the jugular foramen, are sometimes present. Nystagmus is sometimes present, and choked disk occurs in 50 per cent. of the cases.

With *cavernous sinus thrombosis* there is œdema and swelling with venous congestion in the face and about the eye, nasal hemorrhage, exophthalmos, and distention of the retinal veins, and from compression of the oculomotor,

trochlear, abducens, and first division of the fifth nerves there will be pain in the frontal and supraorbital regions, ptosis of the upper lid, various forms of strabismus, and cloudy and sometimes softened cornea. If these symptoms also appear on the opposite side it indicates that the process has extended from the sinus of the one side to that of the other.

With *superior petrosal and superior longitudinal sinus thrombosis* there are no especial local symptoms. With the latter there is at times venous congestion of the entire cranium, including the orbit and the eye, and nose-bleeds.

With *transverse sinus thrombosis* there are no local symptoms unless the clot extends into the bulb of the jugular, and then there may be evidences of compression of the ninth, tenth, and eleventh nerves.

At its beginning infective sinus thrombosis may give one the impression of a chronic brain abscess in its active period; but a brain abscess rarely occasions such high temperature, and never is attended with pyæmic temperature. In some cases sinus thrombosis and brain abscess coexist; in such instances a diagnosis of brain abscess can only be made if there are focal symptoms. If in a case of sinus thrombosis the symptoms are not relieved after incision and drainage of the infected vein, a brain abscess should be suspected and a search therefor with the aspirating needle should be made.

The patients who manifest with their sinus thrombosis a continuously high temperature, diarrhœa, roseola, and enlarged spleen, may, as stated above, be suspected of having typhoid fever; but a slow pulse, a low leukocyte count, and a Widal reaction in the serum point to typhoid, while the presence of a cause for sinus thrombosis—*e. g.*, chronic otorrhœa—favors the existence of this latter condition. The presence of tubercles on the choroid, the rapid respiration without manifest pulmonary lesion, and the existence of a primary tuberculous focus in the bones, joints, lungs, etc., similarly distinguish acute miliary tuberculosis from these forms of sinus thrombosis.

Malignant endocarditis and malaria bear some resemblance in their temperature curve and frequently occurring

chills to the pyæmic cases of sinus thrombosis. The presence of minute petechiæ in the conjunctivæ and skin, the history of an old heart lesion, and the absence of a cause for sinus thrombosis readily distinguish the endocardial cases; while the regular cycle of the chills and fever and the presence of plasmodia in the blood at once differentiate malaria from sinus infection and thrombosis.

It is a frequent but unwise practice to administer quinine in cases in which a malarial infection is suspected, for in this way we cloud the clinical picture by changing the temperature curve. It is better to wait for twenty-four hours and observe the course of the temperatures and make repeated examinations of the blood for the plasmodia of malaria.

INTRACRANIAL NEOPLASMS.

The positive evidence of the presence of a intracranial neoplasm which is afforded by a palpable tumor is only obtainable in rare instances, and that when a growth of the meninges has eroded and protruded through the cranial bones. Our diagnosis of an intracranial neoplasm does not, however, depend upon the palpation of a tumor; it can be made from characteristic general symptoms to which such a condition gives rise. These general disturbances are characterized by their gradual development and their continuous progression, and only in rare cases have intracranial tumors grown to any size without having given rise to them.

Symptoms.—The general symptoms are:

Headache; this is either frontal or occipital; it is usually very intense, and the cause of much suffering.

It is most constant and severe with neoplasms in the posterior fossa of the skull, below the tentorium cerebelli. Its site rarely indicates the position of the tumor.

Tenderness of the bone to percussion at the site of the headache may aid in localizing the tumor, unless such tenderness is due to the irritability of a single superficial nerve.

Convulsions; these may be of the nature of petit mal or general epileptic seizures. They may be an early symptom, occur once and never be repeated, or they may occur at

irregular intervals during the course of the disease, at times overshadowing all the other symptoms so that the disease is considered to be epilepsy.

Vomiting and vertigo.

Choked disk, or optic neuritis.

Change of disposition and mental power.

Slow pulse.

Attacks of syncope, polyuria, and progressive malnutrition.

These general symptoms may at times be strongly simulated by those which are occasioned by a *chronic brain abscess*; but the latter can usually be traced to a cranial injury or to suppuration of the middle ear, nasal or orbital cavities. With abscess the symptoms appear in more rapid succession, and with greater severity; and they are more apt to be attended with an irregular fever. With intracranial tumor the onset of symptoms is very gradual, but they increase steadily in intensity, and the whole course of the malady is longer. Focal symptoms are more apt to be present with cerebral neoplasms and likewise optic neuritis.

The symptoms of *tuberculous meningitis* bear some resemblance to those of tuberculous tumor, but with the former the headache is more severe and continuous; there is more likely to be hyperæsthesia to light, sound, and touch; optic neuritis is less likely to be present, and, when it is, it is less intense and later in its appearance; and the ophthalmoscope may show the presence of tubercles upon the choroid. Should there remain a doubt as to the nature of the malady, a lumbar puncture will aid in clearing it up, for in meningitis such puncture frequently yields a cloudy fluid which contains tubercle bacilli.¹

A localized tuberculous meningitis can seldom be differentiated from a tuberculous tumor.

Chronic hydrocephalus occasions general symptoms that may be mistaken for those due to neoplasm; but in this malady there is a spastic paralysis without any focal spasm, and the paralysis is always bilateral, the lower limbs being more affected than the upper. Cranial-nerve paralyses are late manifestations. Early evidences of bilateral spastic

¹ The presence of the bacilli may be demonstrated in spreads, or by culture.

paralysis and late cranial-nerve paralysis point to chronic hydrocephalus.

The presence of a tumor having been determined, the next query is, What is its site? This is ascertained from the focal symptoms. These are, as a rule, unilateral; they commence gradually and spread slowly. When they are due to cortical tumors they are at first irritative in character and later on paralytic; but when they are occasioned by a sub-cortical tumor they are usually paralytic from the beginning. Depending upon the site of the neoplasms these focal symptoms will be unilateral spasms, monoplegia, hemiplegia, paræsthesia or anæsthesia in one or more limbs; hemianopsia and various forms of aphasia, and affections of the cranial nerves and basal ganglia. (For localization of cerebral function, see p. 70.) Jackson was the first to describe the tonic and clonic convulsions which occur at intervals, limited to one part of the body—*e. g.*, the face, or hand, or foot—and sometimes extending from the part first invaded to other parts of the body in a definite order of succession—*e. g.*, from the face to the arm, to the trunk, and then to the leg. Such a localized convulsion is known as a Jacksonian epilepsy. The spasm always begins in the muscles which are represented in the centre first irritated, and naturally the determination of these muscles is of immense aid in the localization of the tumor.

An absence of focal symptoms points to the frontal or temporal lobe as the seat of the neoplasm.

The nature of the neoplasm can only be indefinitely determined. A previous history of syphilis or tuberculosis and the evidences of other syphilitic or tuberculous lesions are naturally very suggestive of a similar lesion in the brain and its membranes. A marked or complete recovery from antisiphilitic treatment is indicative of a syphilitic tumor, but partial improvement is not to be so considered, for a great many other neoplasms are temporarily and partially improved by this treatment. A variable intensity of the symptoms speaks for considerable vascularity of the tumor; hence for glioma, gliosarcoma, and aneurysms. A primary carcinoma in some other organ suggests the probability of the brain neoplasm being a metastatic deposit.

CHAPTER VII.

INJURIES, INFLAMMATIONS, AND NEOPLASMS OF THE FACE.

INFLAMMATIONS AND NEOPLASMS OF THE SKIN.

OF neoplastic and chronic inflammatory affections of the skin, it is necessary to consider especially only the lupoid, the syphilitic, and the epitheliomatous. In the early stages of these diseases the differential diagnosis can be readily made, but in the advanced stages, when ulceration has taken place, their differential diagnosis is often attended with some difficulty. Considerable aid is afforded by the previous personal and family history of the patient (thus, syphilis and tuberculosis), by the nature of the early manifestations of the malady, and by a careful physical examination for other evidences of tuberculous, syphilitic, or malignant disease. In doubtful cases a section should be given to the pathologist for microscopic examination.

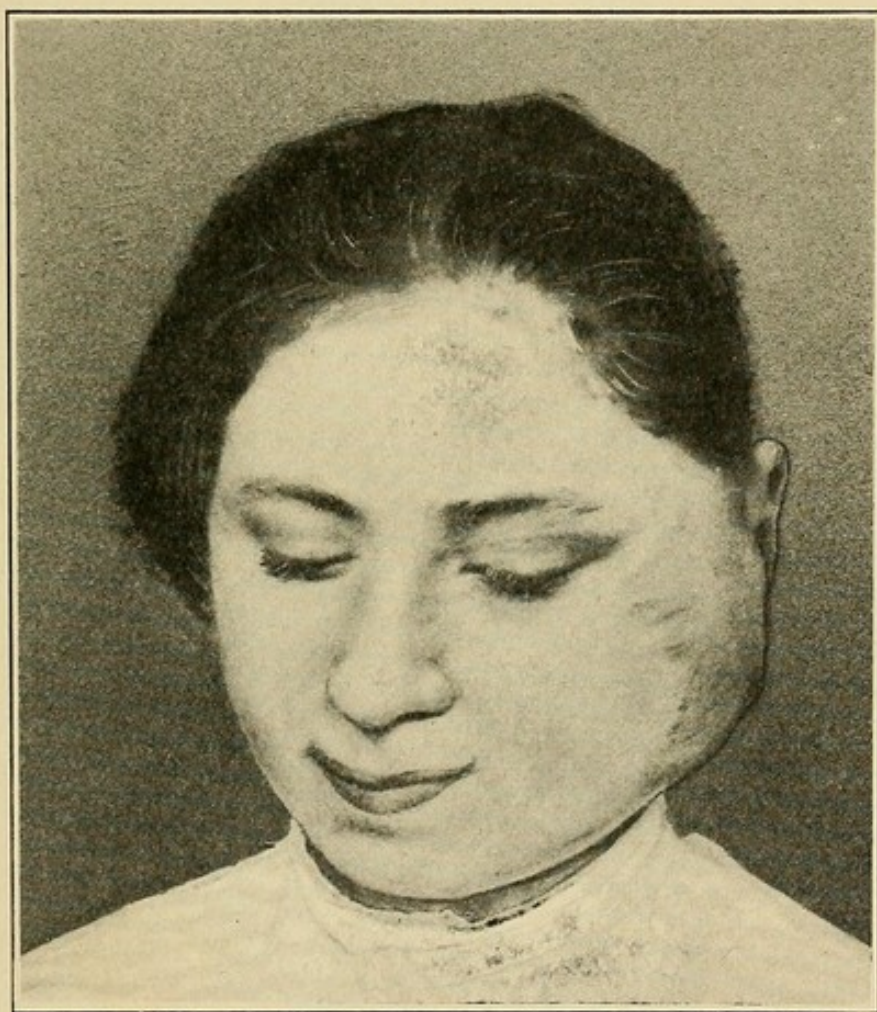
Lupus Vulgaris.—Lupus vulgaris occurs chiefly in young subjects. It first appears as a group of slightly raised, pin's-head size nodules in the skin or mucous membranes. Very gradually new nodules develop around the first group, thus forming an elevated mass that is covered with thick scales of epidermis and that soon undergoes ulceration. The ulcer may eat away the skin, muscles, and mucous membrane, but never involves the bone. At its periphery new discrete nodules are always to be seen. As the ulcer advances it tends to cicatrize in the part first attacked. This tendency toward cicatrization in one part and advancement in another part of the ulcer, with the presence of discrete nodules at the margins, is characteristic of lupus.

The Primary Chancre of Syphilis.—This is most frequently met with on the lips, and is to be distinguished from epithelioma by the following characteristics: Its first appearance is as an ulcer; its increase in size is rapid; it

occurs in younger individuals, and it is followed by the evidences of constitutional syphilis in three to four weeks. In doubtful cases a section should be given to the pathologist for microscopic diagnosis.

Ulcerating Gumma.—The ulcerating gumma appears first as a discrete, soft, elastic tumor; the ulcer usually has

FIG. 23



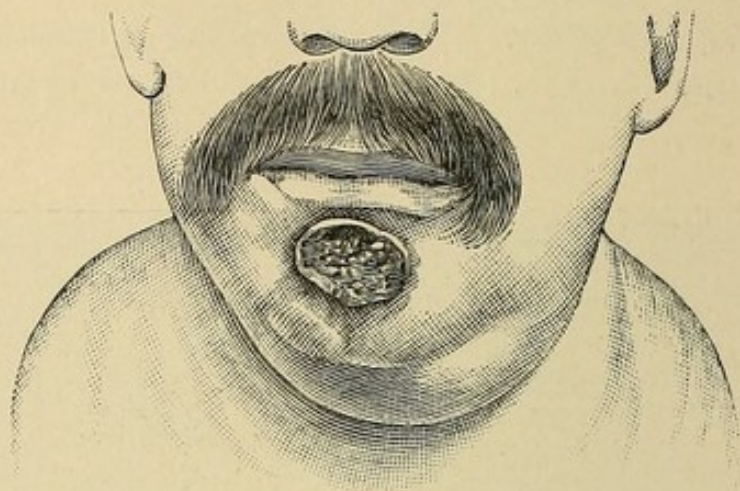
Cavernous angioliipoma of side of face and ear. Note the telangiectatic condition of the skin overlying the tumor, which was soft and doughy in character (lipoma) and pulsated markedly (cavernous angioma).

undermined, non-indurated, serpiginous edges. There are usually other evidences of syphilis that aid us in making the diagnosis.

Epithelioma.—Epithelioma of the face is met with in two forms: the *superficial* or *rodent ulcer* type, occurring chiefly above a line drawn through the angles of the mouth to the lobules of the ear, and the *deep, infiltrating carcinoma*,

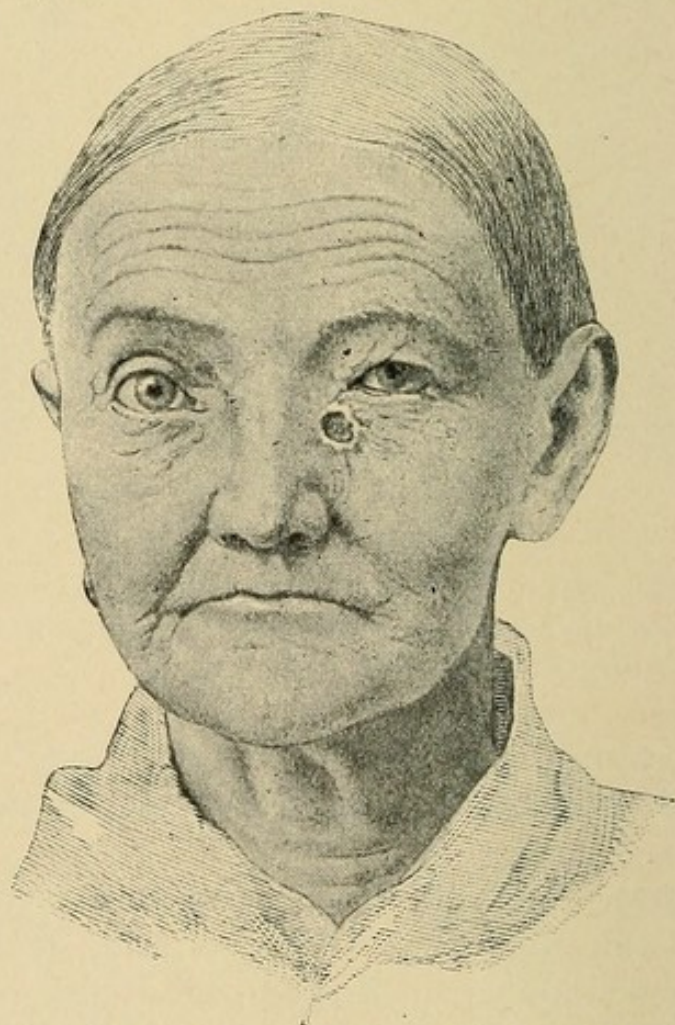
which originates in the glands of the cutis. The former commences as a small superficial ulcer, which very gradually

FIG. 24



Chancre of the chin ten days after infection by a cut with a razor. (Von Bergmann.)

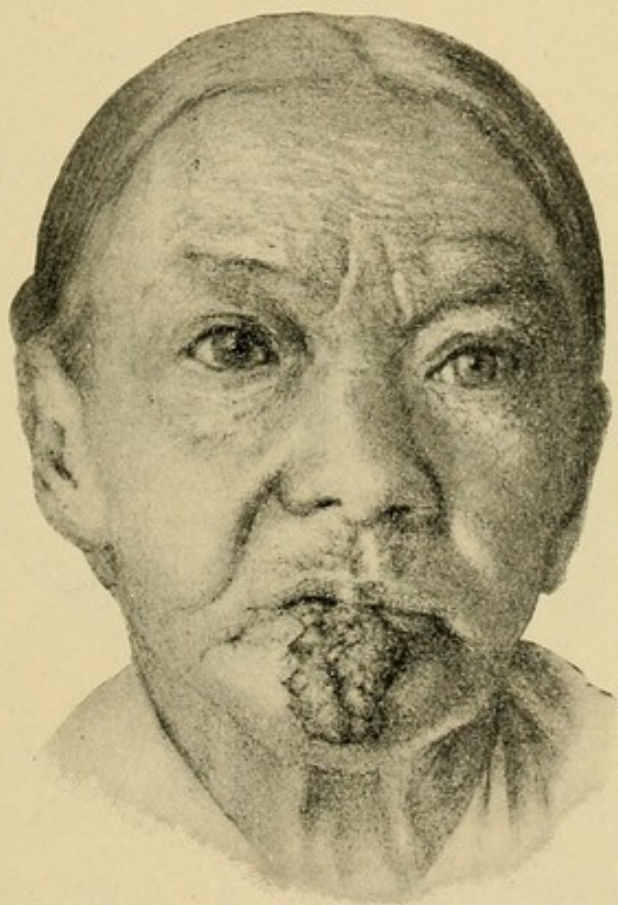
FIG. 25



Superficial epithelioma or rodent ulcer. (Von Bergmann's clinic.)

extends and tends to cicatrize in some of its parts. The latter appears as an aggregation of deep-seated, hard nodules which extend rapidly, invade the deep structures, even the bones, and soon break down, leaving an ulcer with everted, indurated edges, and unhealthy, easily bleeding, crusty base. In the lower lip it commences as a crack or fissure or hard nodule that does not heal, becomes indurated, increases in size, and undergoes ulceration.

FIG. 26



Deep, infiltrating epithelioma of the lower lip. (Von Bergmann.)

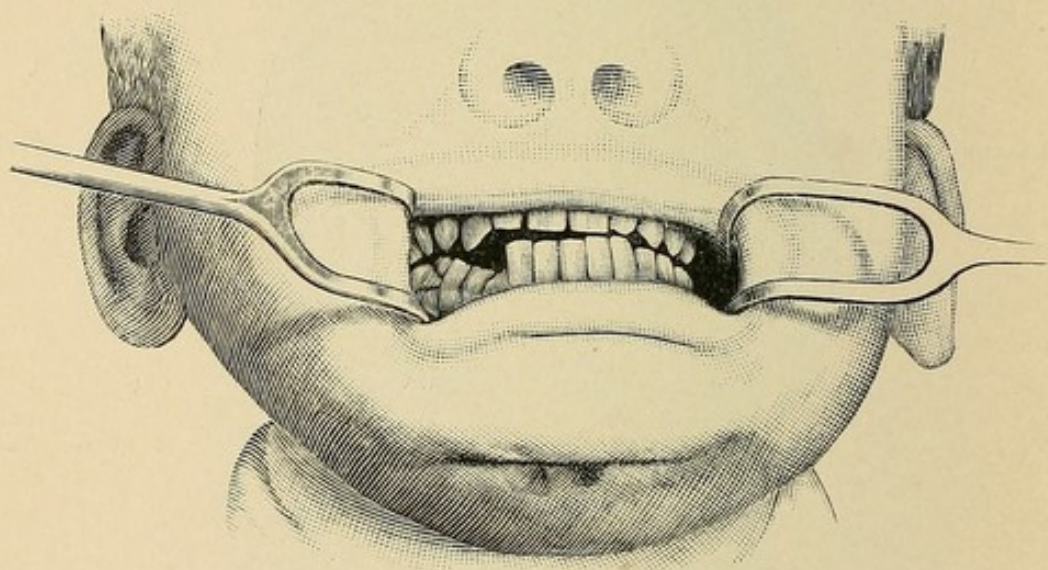
INJURIES, INFLAMMATIONS, AND NEOPLASMS OF BONES OF FACE AND JAW.

Fractures.—Fractures of the facial bones give the usual evidences of such injuries and require no especial diagnostic consideration. Fractures of the lower jaw are usually compound and are easily detected by the step-like irregularity they occasion in the alignment of the lower teeth. Fractures

of the alveolar margin of the upper jaw cause a similar irregularity in the alignment of the upper teeth.

Dislocations.—Dislocation of the lower jaw results from opening the mouth too widely and is pathognomically indicated by an inability to close the mouth. If both temporomaxillary joints are dislocated, the jaw projects directly forward and downward, the chin being very prominent. If only one of these joints is dislocated the lower jaw projects forward, and inclines downward on the affected side. Over the dislocated joint there is a decided hollow.

FIG. 27



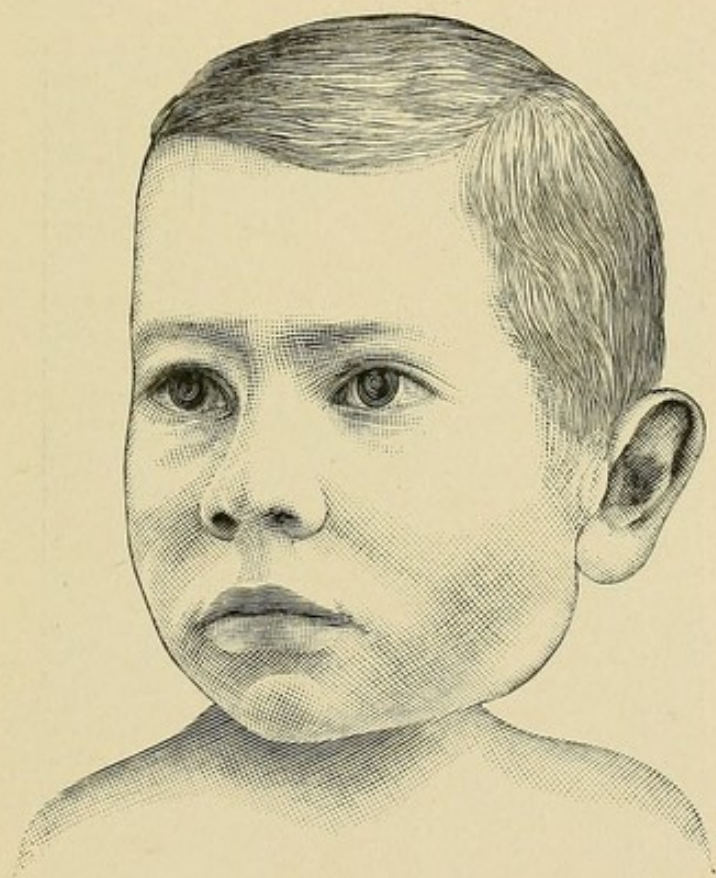
Fracture of the jaw. Note the step-like irregularity in the alignment of the lower teeth. (Von Bergmann.)

Suppurative Inflammations.—An acute onset of severe constitutional symptoms—*i. e.*, high fever, rapid pulse, stupor, and delirium—combined with swelling, pain, and exquisite tenderness of either jaw-bone, coming on after the extraction of a tooth or a compound fracture of the jaw, are almost pathognomonic of acute infectious osteomyelitis of the maxillary bones. The extremely septic character of these inflammations is to be especially noted, and the urgency for immediate operation in their presence is to be borne in mind.

A change in the normal outline of one or more of the facial bones due to irregular thickening of their periosteal covering,

and the presence of profusely discharging sinuses leading down to bare bone, are evidences of a chronic osteomyelitis. Such a chronic osteomyelitis may result from caries of the teeth, from syphilis, tuberculosis, actinomycosis, phosphorus poisoning, mercurial stomatitis, etc., and the particular cause in each case is to be determined from the personal and family history, from the site of the disease and from the especial character of the local lesions. Thus a history

FIG. 28



Osteomyelitis of the lower jaw. (Von Bergmann.)

of family or personal tuberculosis or of acquired syphilis suggests these diseases as a cause for the osteomyelitis; syphilis, furthermore, most frequently attacks the nasal and palatal processes of the superior maxillary bones; tuberculosis, the orbital margin of the superior maxilla, and actinomycosis the ramus of the lower jaw.

Phosphorus necrosis is marked by the presence of large, isolated, periosteal osteophytes, by considerable retraction of the gums with exposure of the necrotic bone, and by dropping

out of the teeth. Actinomycosis is evidenced by the marked involvement of the soft parts of the cheek and submaxillary region, the lumpy character of the lesions of the soft parts, by the discharge of the characteristic pinhead, yellowish granules which on microscopic examination are found to contain the ray fungus, and by the absence of glandular involvement during the early stages.

Chronic Hydrops.—A uniform distention of the cavities of the frontal and maxillary sinuses with such thinning of

FIG. 29



Periostitis of the lower jaw. (Von Bergmann.)

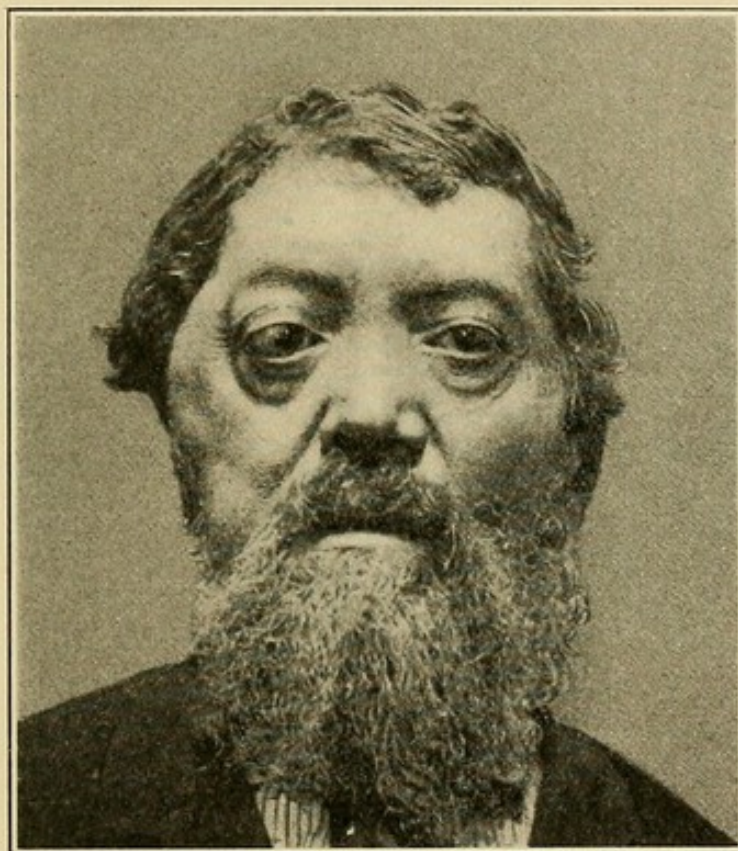
their walls as to cause these to crackle under the palpating finger, together with a feeling of weight and oppression in these parts, are evidences of chronic hydrops of these sinuses.

The distention is, in the large majority of cases, not due to an accumulation of mucoid or serous fluid in the sinus, but results from the presence of mucous polypi therein, or, in the case of the antrum of Highmore, to the presence within it of a dentigerous cyst.

In the frontal sinus the lower or orbital wall is the first

to protrude, the eyeball being displaced outward and downward; the anterior and inner walls subsequently bulge outward. In the maxillary sinus—*i. e.*, the antrum of Highmore—the anterior wall, especially at the canine fossa and the lower and inner walls, become very prominent and very thin, the gum is crowded downward, and the alveolar process is thickened. The thinning of the walls permits a feeling of fluctuation to be obtained and also allows of transillumina-

FIG. 30



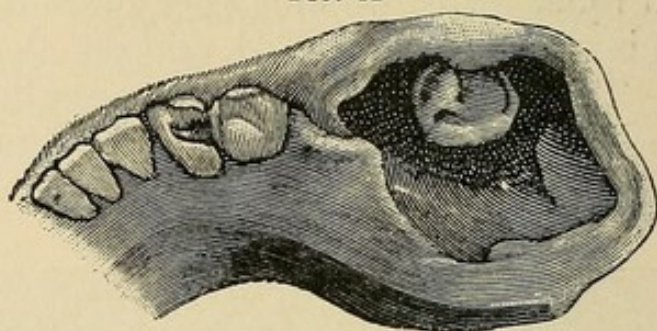
Chronic hydrops of the right antrum of Highmore. Note the bulging of the cheek and the exophthalmos without displacement of the angle of the mouth.

tion of the cavity of the sinus. The *uniform bulging* and *thinning* out of the walls of the cavities, together *with the crackling* and *sense of fluctuation* which are to be elicited upon palpation, are sufficient to establish the differentiation of a chronic hydrops from tumors of these bones.

Thus *cysts of the maxillary bones* grow only in one direction, either upward toward the antrum or nose, or downward toward the gums, or backward; they do not cause the uniform bulging of the walls of the antrum, which is characteristic

of hydrops; and tumors of the maxillary bones likewise grow only in the direction in which they encounter the least resistance. They do not cause the uniform bulging of the

FIG. 31



Dental cyst of the lower jaw in a boy aged fourteen years, causing uniform distention of the walls of the antrum. (Von Bergmann.)

antral walls and do not give the feeling of fluctuation. When they perforate externally or into the nose, mouth, or orbit, their character is easily determined.

FIG. 32



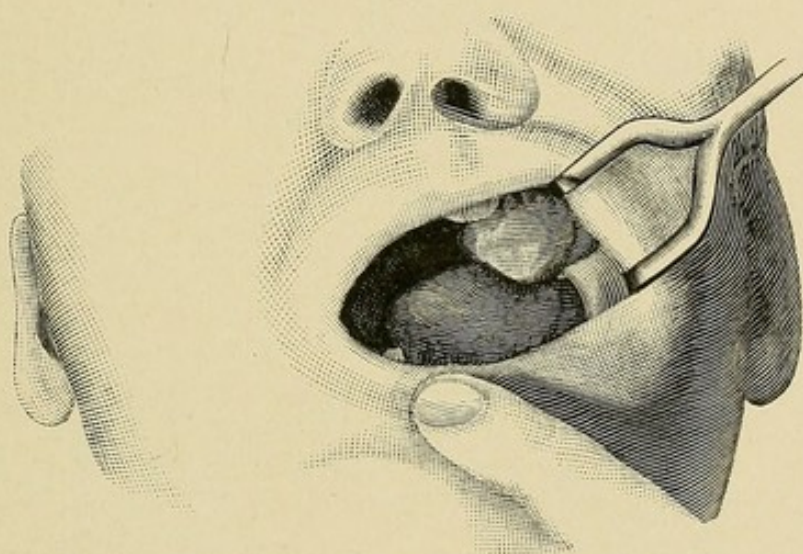
Pneumatocele of frontal sinuses. Note the distention of the anterior walls of the frontal sinuses.

It is well in the case of the antrum of Highmore to attempt to ascertain the cause of the distention. Thus one or more unerupted teeth suggest the presence of a dentigerous cyst; a carious tooth with an old history of subperiosteal abscess suggests a subperiosteal abscess cyst, which strongly simu-

lates a chronic hydrops of the cavity; and in the absence of either of these, a mucous polypus degeneration is probably present.

Empyema.—An acute or chronic empyema of these cavities usually follows an infection from the nose; the local evidences of distention resemble those just described, but there is fever and more pain with the acute purulent infection of the sinuses. The history of a preceding nasal infection suggests the character of the malady, and a periodic evacuation of considerable quantities of purulent fluid through the nose is further evidence of it. Retention of the pus within

FIG. 33



Fibroma epulis of the upper gum from a woman aged twenty-eight years. Note the smooth contour, in comparison with the irregular surface of tumor in Fig. 34. (Von Bergmann.)

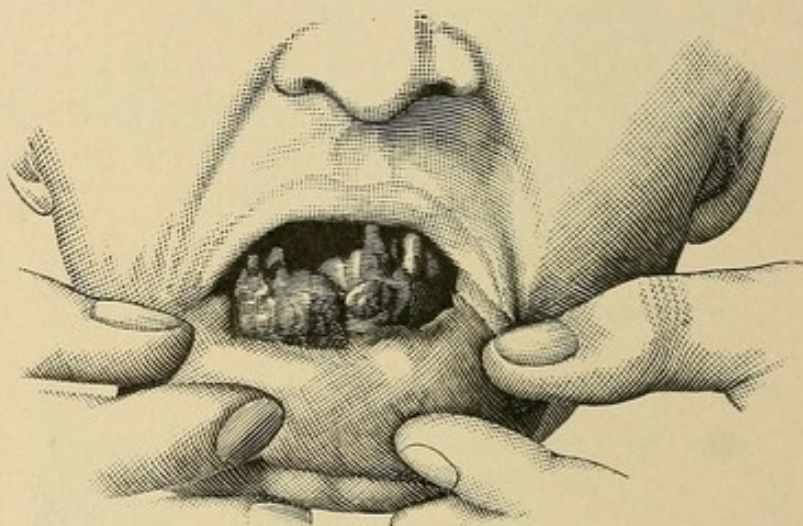
the cavities is evidenced by chills, fever, and sweating; by an increased tenderness, and by a feeling of distention of the walls of the cavity.

A fistula discharging pus and leading into the sinus cavity points to an empyema, but affords no information as to the cause thereof.

Periosteal Neoplasms.—Periosteal neoplasms of the maxillary bones, of which those that spring from the alveolar processes, *i. e.*, the *epules*, are the most common, are readily detected. They lie upon the bone. The benign tumors and the giant-celled sarcomata form hard, sessile, slowly growing tumors; the malignant neoplasms grow rapidly, undergo

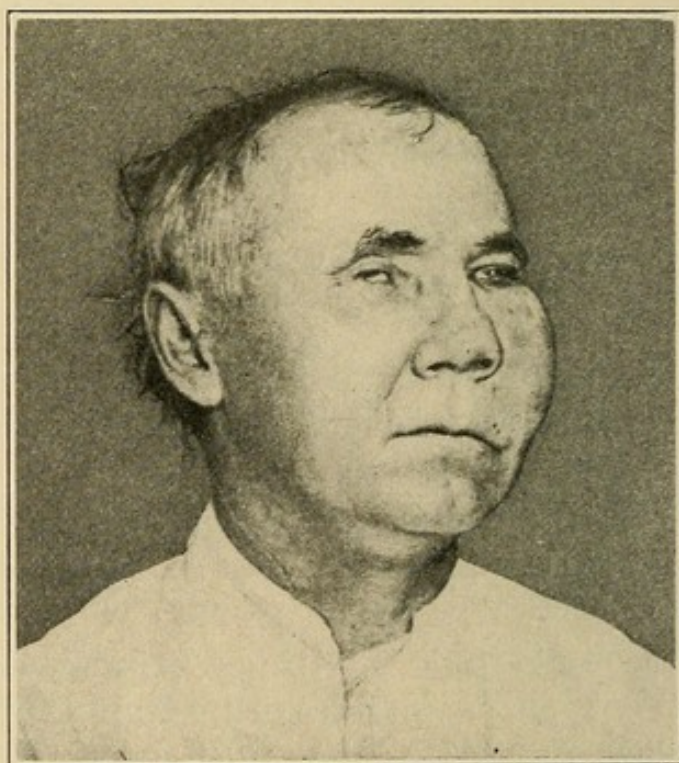
ulceration early, and cause rapid bone destruction. The nature of the periosteal growth is sometimes very difficult to determine. Malignancy is often to be decided only by the microscope and from the rapidity of growth.

FIG. 34



Sarcomatous epulis in a woman aged fifty-six years. (Von Bergmann.)

FIG. 35

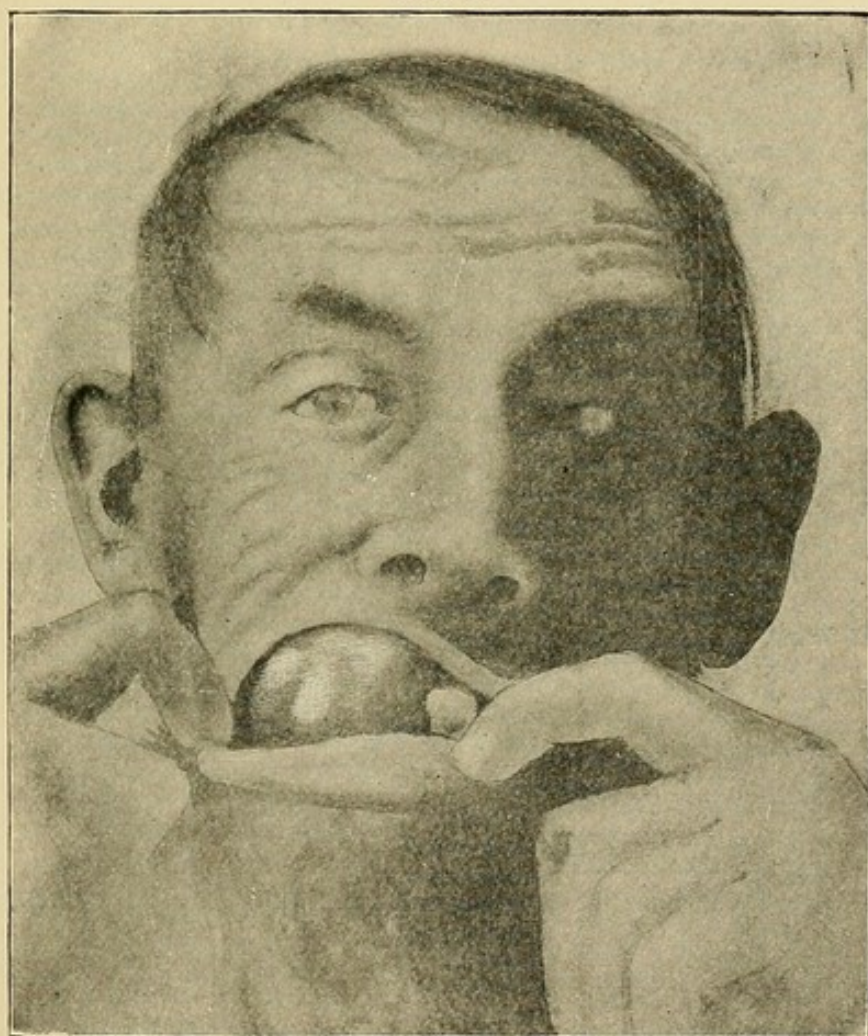


Medullary sarcoma of the left superior maxillary bone growing downward toward the mouth, and outward. Note the displacement of the angle of the mouth, the absence of exophthalmos. Compare with Fig. 30, of chronic hydrops of antrum of Highmore, in which there is uniform bulging of all the walls of the antrum.

A fibroma is not so hard as an osteoma, but periosteal and myeloid sarcomata are at times of bony hardness and thereby resemble the osteoma; with the former, however, there is marked bone destruction.

The periosteal swelling which is due to an acute inflammation is distinguished from that due to neoplasm by its sudden appearance and by the presence of acute pain, fever, and increased leukocytosis.

FIG. 36



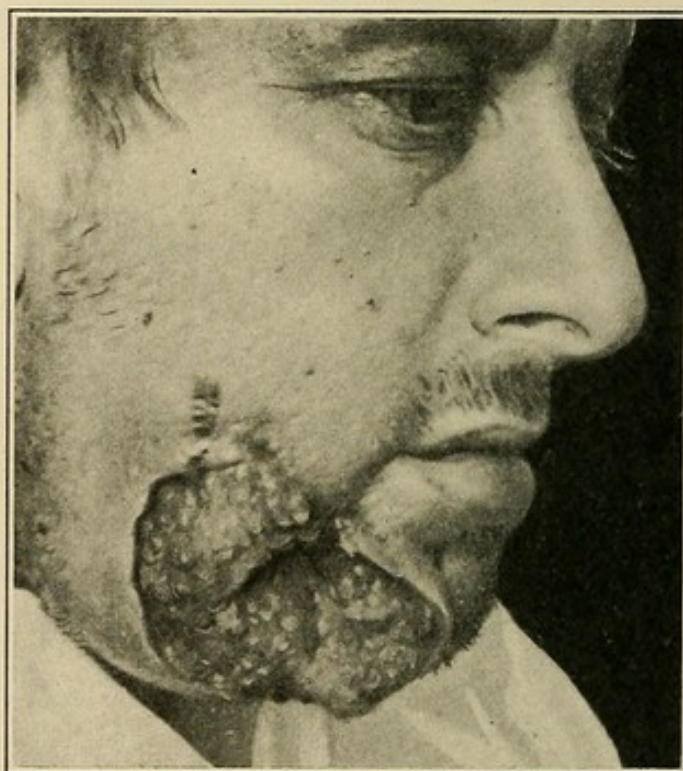
Periosteal sarcoma. (Von Bergmann.)

Medullary Tumors.—Medullary tumors can only be detected when they become large enough to fill the cavity of the bone and expand its cortical layer. The latter forms a shell around the growing tumor, and as it becomes thinned out by the increasing size of the neoplasm it crackles on palpation, and thus affords a valuable clinical sign, viz., the “egg-shell

crackle." Benign medullary tumors grow slowly and gradually cause absorption of the cortical crackling layer surrounding them, but the latter is replaced by a new one formed by the periosteum which is irritated by the growing tumor. Benign tumors are, as a rule, therefore surrounded in all stages by a crackling shell.

Malignant medullary tumors grow rapidly, and very early perforate and destroy the cortical shell; a new periosteal shell is not formed, however, for the rapidly growing neoplasm invades and destroys this membrane.

FIG. 37



Ulcerating medullary sarcoma of inferior maxilla. Note fungous character of the ulcer.

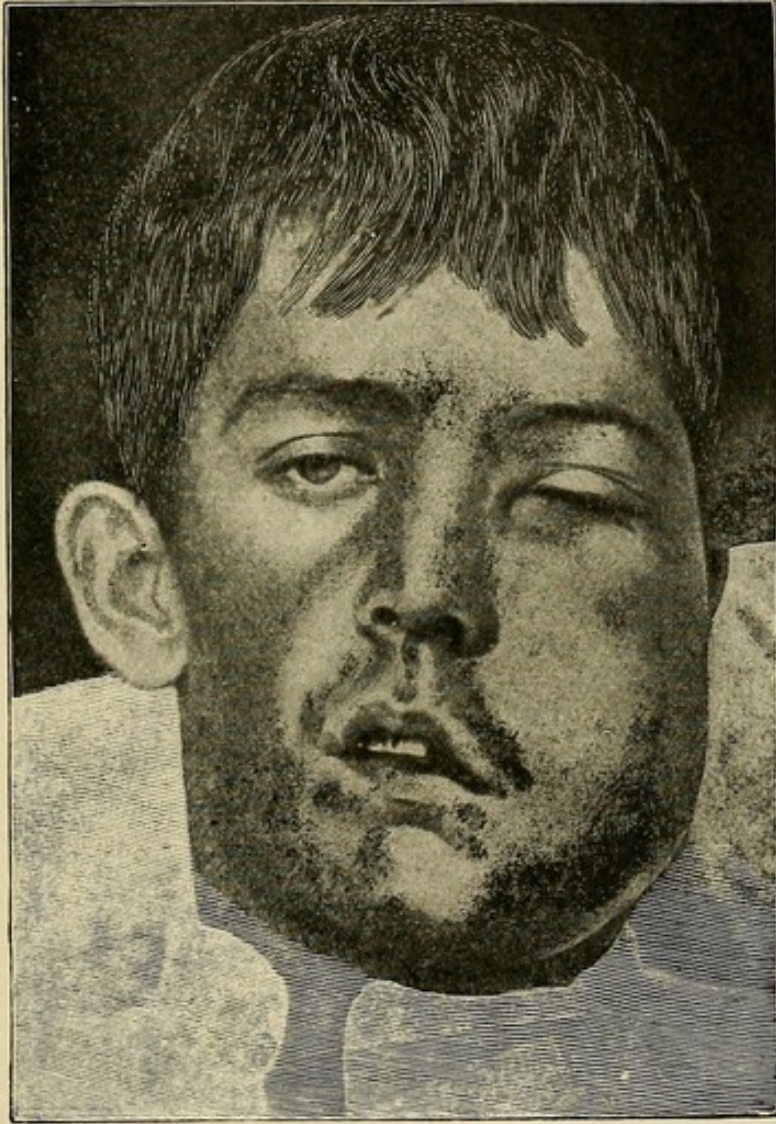
An intact shell over a slowly growing neoplasm is evidence of its benign character, while a perforated or partially destroyed shell over a rapidly growing tumor is evidence of its malignancy.

Medullary tumors grow in the direction of least resistance: upward toward the antrum, outward at the canine fossa, inward toward the nose, etc. The cavities of the bones are filled by the neoplasm, and their walls are bulged outward, thus displacing the eyeball, obstructing the nose, interfering

with the movements of the tongue and projecting into the floor of the mouth. The malignant tumors, especially the carcinomata, ulcerate rapidly.

The swelling of the bone, which is due to a chronic osteomyelitis, comprises the entire outline of the bone; it is thereby

FIG. 38



Retromaxillary fibroma, pushing the superior maxilla forward. (Von Bergmann.)

distinguished from that due to a neoplasm, which is confined chiefly to one side or border of the bone. The irregularity of the swelling, the presence of sinuses leading down to exposed bone, and the discharge of a profuse amount of pus are further data in favor of the inflammatory character of the swelling.

The differentiation of medullary neoplasms of the superior maxilla from hydrops and chronic empyema of its cavity has already been discussed on page 95.

FIG. 39



Myelogenous sarcoma. (Von Bergmann.)

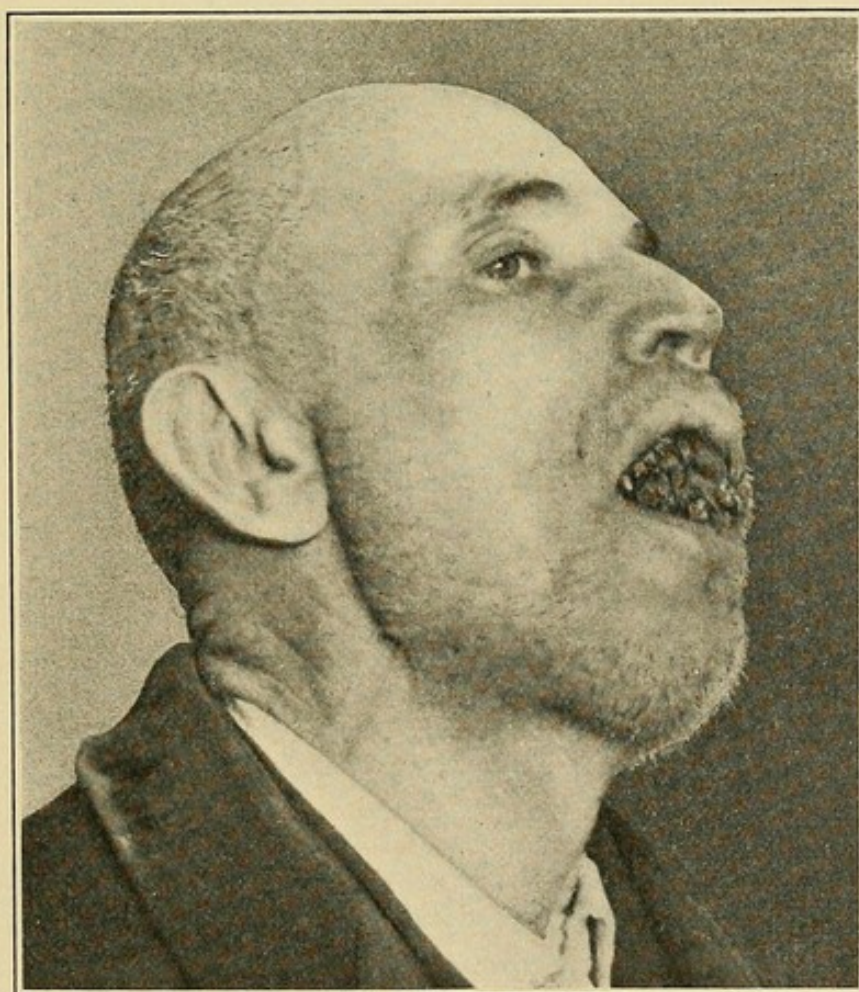
CHAPTER VIII.

INFLAMMATION AND NEOPLASMS OF THE MOUTH, TONGUE, TONSILS, PHARYNX, AND SALIVARY GLANDS.

INFLAMMATION AND ULCERATION OF THE MOUTH.

LITTLE difficulty is experienced, as a rule, in determining the nature of inflammatory and ulcerative diseases of the mucous membrane of the mouth. The gross appearance

FIG. 40



Epithelial carcinoma of the cheek. A diffuse, hard, infiltrating tumor, ulcerating at its posterior part. The ulcer has everted edges.

and site of the lesion, taken in conjunction with the antecedent personal history, will usually enable the observer to make a correct diagnosis. Thus, the extremely sensitive, grayish or yellowish spots from pin's head to lentil-seed size, occurring on the gums and buccal mucous membrane, especially in teething children, speak for *aphthæ*. The punctate and linear spots of a dirty whitish color which occur on the dried and sensitive mucous membrane of the gums, cheeks,

FIG. 41



Noma. Early stage, showing swollen, tense cheek, with gangrenous patch in one part. (Lexer.)

and tongue of weakened and marasmic individuals are characteristic of *soor*.

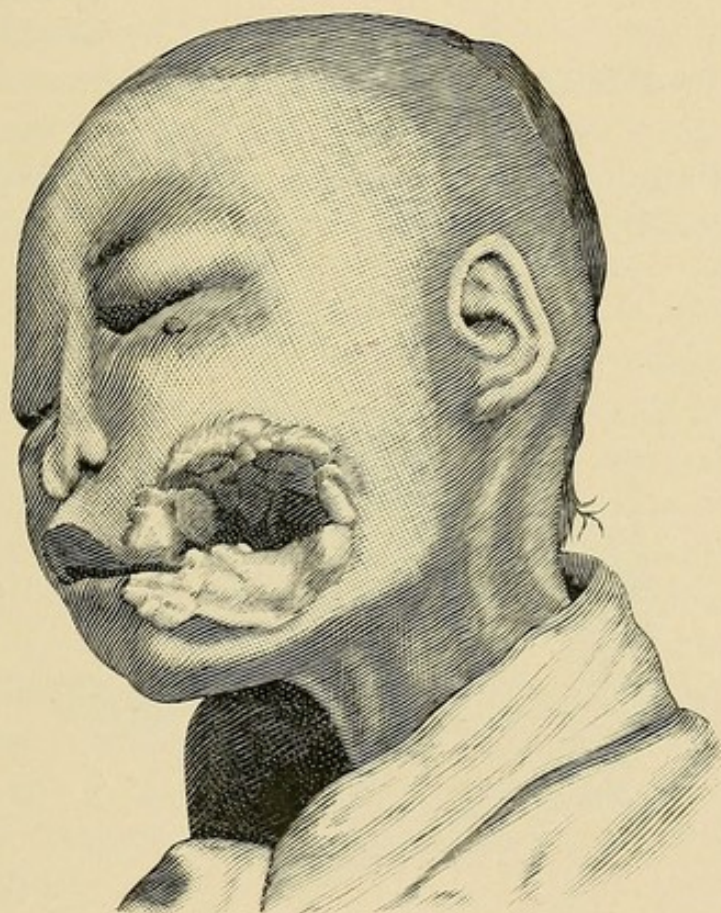
The swelling and slight bleeding from the gums of those who are taking mercury without observing the usual precautions are indicative of *mercurial stomatitis* and are a warning to the patient that, unless more care of the mouth

is observed, the teeth will become loose and ulceration and necrosis of the alveolar process of the maxilla will occur.

Swelling, congestion, softening, and bleeding from the gums in individuals who have in the bones and soft parts other evidences of scurvy or who give a history of rickets and hæmaturia, are an evidence of this affection.

The indurated sore, which within a few weeks attains a considerable size and is accompanied by submaxillary gland-

FIG. 42



Noma. Later stage, showing extensive gangrene and sloughing of the cheek.
(Lexer.)

ular swelling, followed by the other evidences of syphilis, is the initial lesion of this disease. Superficial erosions and fissures, or flat, red, circumscribed papules which sometimes ulcerate, and broad condylomata, are evidences of the secondary period of syphilis; and circumscribed soft, elastic nodules of varying size, sometimes ulcerating and tending to perforate the part which is affected, are indicative of the final stage of this disease.

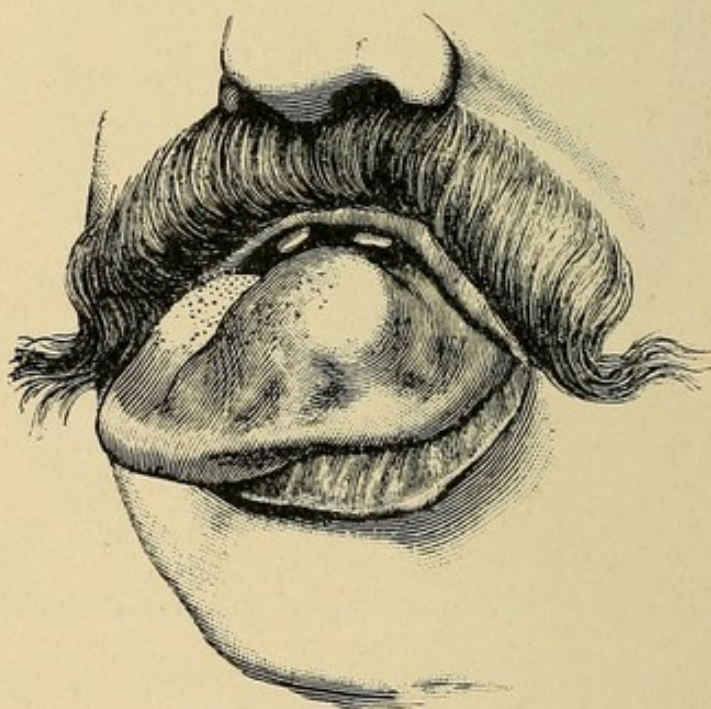
A gangrenous patch on the mucous membrane of a tense, shiny, and swollen cheek in marasmic and much-weakened individuals, especially children, the gangrene having a tendency to spread through the entire thickness of the cheek and even to the bones of the face, together with severe constitutional symptoms of sepsis, is pathognomonic of *noma*.

Tuberculosis, actinomycosis, and cancerous disease of this region have the same characteristics as similar affections of the tongue and pharynx.

DISEASES OF THE TONGUE.

Acute inflammatory conditions of this organ manifest their presence by an acute onset with high temperatures,

FIG. 43



Chronic abscess of the tongue ; a painful, fluctuating swelling.

together with a rapid, painful swelling of the tongue which either resolves in a few days or goes on to form an abscess. Simple as is the diagnosis in these cases, so difficult is it at times in the ulcerative lesions that result from chronic inflammations such as syphilis, tuberculosis, and actinomycosis. These bear so much resemblance to one another and

to the ulcerations which result from malignant disease that their differentiation is often attended with considerable difficulty. A preceding history of syphilis, or the presence of other syphilitic lesions in the bones or the skin or mucous membranes, or the evidences of tuberculosis in the lungs, bones, joints, etc., will afford valuable data for establishing a diagnosis; but in all doubtful cases we should remember, first, to try the effect of antisyphilitic treatment, and, secondly, to submit an excised specimen of the lesion to expert pathological examination.

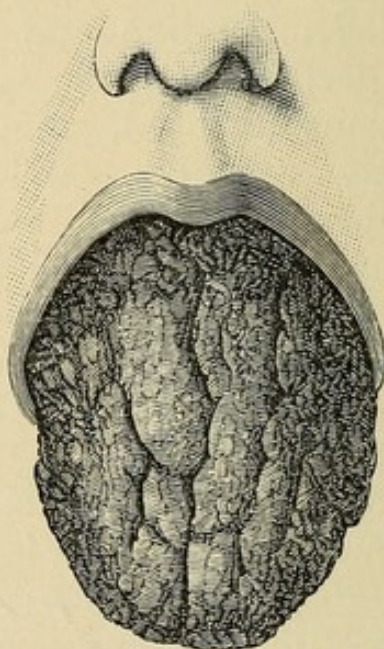
Tuberculosis.—Tuberculosis of the tongue may resemble both carcinoma and syphilis. It is very rarely a primary lesion; most frequently it is secondary to pulmonary tuberculosis, the bacilli-laden sputum and the injury of the tongue from sharp teeth being the causes of infection. Its initial manifestations are hard, painless, slowly growing nodules that strongly resemble carcinomatous nodules. When these break down they leave an indolent, fungous, indurated ulcer, which is distinguished from carcinoma by its worm-eaten, cheesy base, often surrounded by miliary tubercles, and by the pale, flabby granulations with which it is covered; the presence of tuberculous lesions elsewhere in the body assist in making the diagnosis. From ulcerating gummata the softened, broken-down, and ulcerated tuberculous nodes are distinguished by their initial hardness, their cheesy base and indurated edges; the presence of tuberculous lesions in the lungs and the absence of a history of syphilis or other evidences of this disease are further aids in their differentiation.

Syphilis—A rapid development of an indurated sore on the tongue, with an early, painless, submaxillary glandular involvement, together with a rapid improvement from the administration of mercury, speak strongly enough for the initial lesion of syphilis; while sharp margins, rounded form, multiplicity, and lack of all inflammatory reaction distinguish the superficial ulcerations which attend the secondary period of this disease. The ulcers which arise from the softening and breaking down of gummata are distinguished from tuberculous lesions by their initial appearance as soft, elastic nodules usually multiple in number, and by the character

istics of the ulcer, viz., soft, sharp borders with lardaceous base that clear up rapidly on taking iodides and mercury. To these data a previous history of syphilis and the presence of syphilitic lesions in the other organs lend confirmatory evidence. The sclerosing form of syphilitic glossitis is not very common. It is well pictured in Fig. 44.

Actinomycosis.—The first manifestations of this malady are pea to hazelnut-sized, sharply circumscribed, hard, usually painless nodules at the sides and tip of the tongue, covered by congested mucous membrane. These grow slowly larger, then softer, and later on fluctuant, and eventually break

FIG. 44



Sclerosing syphilitic glossitis. The tongue is hard ; the mucous membrane is red in places and white in others ; the organ is atrophied, and its surface is covered by a series of projections and fissures. (Reclus.)

down and leave an ulcer. The diagnosis of actinomycosis is confirmed by a discharge from the ulcerating area of minute yellowish granules that on microscopic examination are found to contain the ray fungus. The nodules are differentiated from those due to tuberculosis or syphilis by the course of their development and by the absence of other tuberculous and syphilitic lesions ; and the ulcers which result from these nodules are distinguished from those due to carcinoma by their multiplicity, their sharp margins and their softer consistency.

Carcinoma.—This affection usually occurs in middle-aged or elderly subjects; it commences as an indurated crack or fissure or nodule on the side or dorsum of the tongue which persistently refuses to heal; it advances rather rapidly, and is later on followed by enlargement and induration of the submaxillary glands, with ulceration at the site of the lesion on the tongue, the ulcer having hard, everted edges and an unhealthy, easily bleeding base. The early nodules are to be distinguished from those due to tuberculosis, syphilis,

FIG. 45

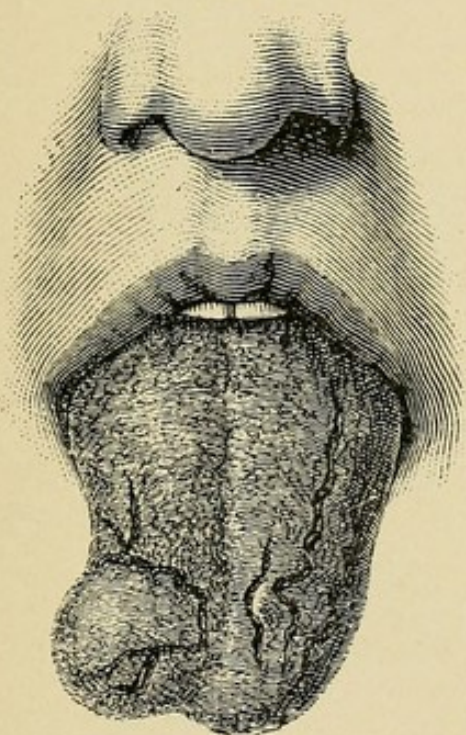


FIG. 45.—Gumma and syphilitic fissures of tongue. Note soft, elastic, circumscribed tumor and the other evidences of syphilis in the fissures of the tongue. (Reclus.)

FIG. 46

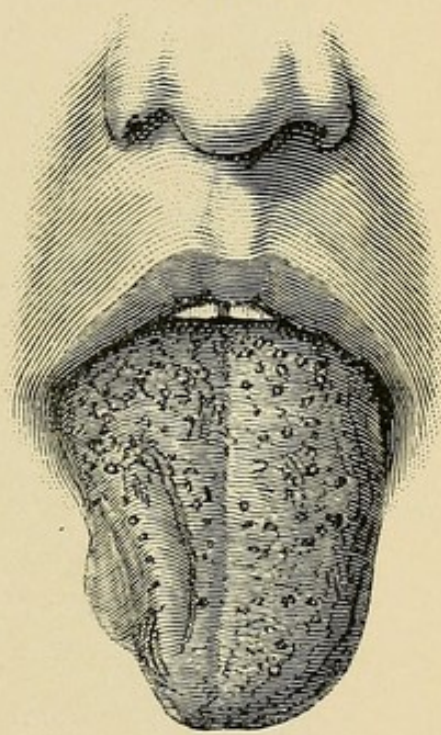


FIG. 46.—Syphilitic tongue after resorption of the gumma by iodides. Note the fissures and the retraction of the tongue at the site of the gumma. (Reclus.)

and actinomycosis by their hardness and their lack of sharply defined margins, by the course of their development, and by the absence of other tuberculous or syphilitic lesions. Carcinomatous ulcers differ from tuberculous and syphilitic ulcers in their indurated, everted edges, and their unhealthy, easily bleeding, rather extensive base. A marked interference with the movements of the tongue, a considerable enlargement of the submaxillary glands and early cachexia, are further evidences of malignant disease.

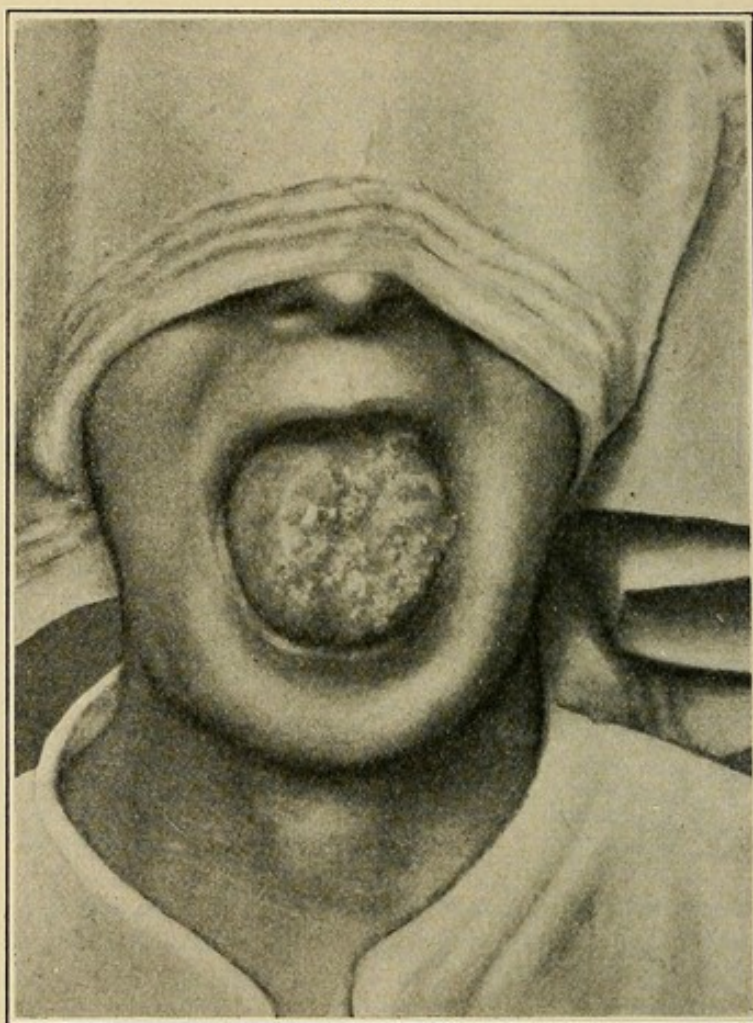
To recapitulate:

Nodules.—*Nodules in the tongue may be:*

Tuberculous. Occurring in young subjects, with other evidences of tuberculosis, hard at onset, painless, of slow growth.

Syphilitic Gummata. These occur at any age, with other syphilitic lesions in the bones, skin, etc.; they are usually multiple and have a soft and elastic consistency.

FIG. 47



Carcinoma of the tongue. Note its site, its diffuse character, its irregular, ulcerated surface, and the everted edges of the ulcer.

Actinomycotic. These occur in young adult life; the nodules are multiple, sharply circumscribed, hard at the beginning, usually painless, located at tip or sides of tongue, and develop in two to eight weeks.

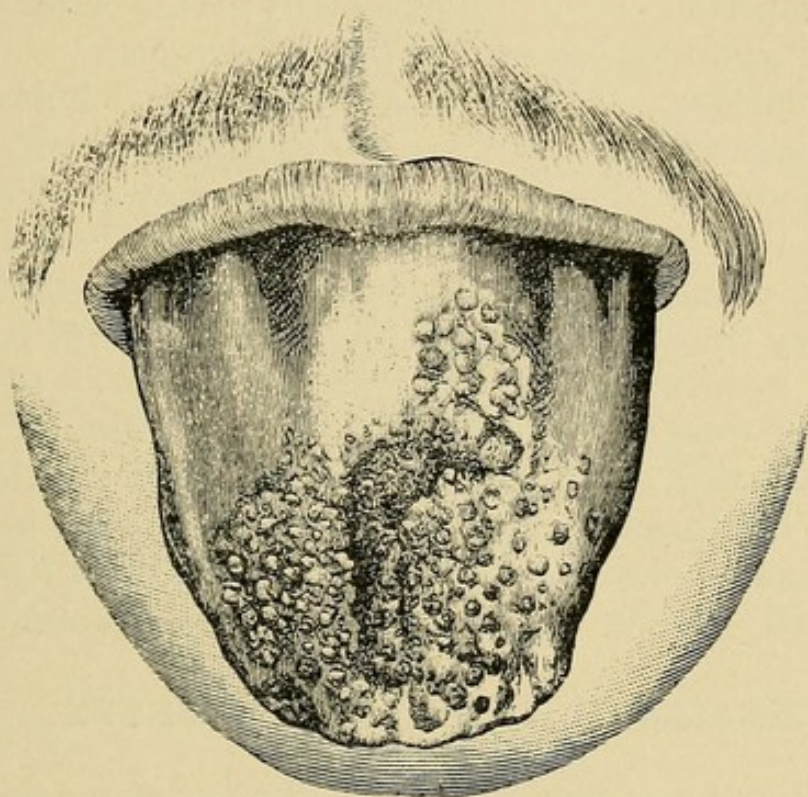
Carcinomatous. These occur in middle or advanced age; they are very hard, with diffuse margins, somewhat painful, and are attended with marked glandular involvement.

Chronic Abscess. This is very painful and fluctuates.

Ulcers.—*Ulcers of the tongue may be:*

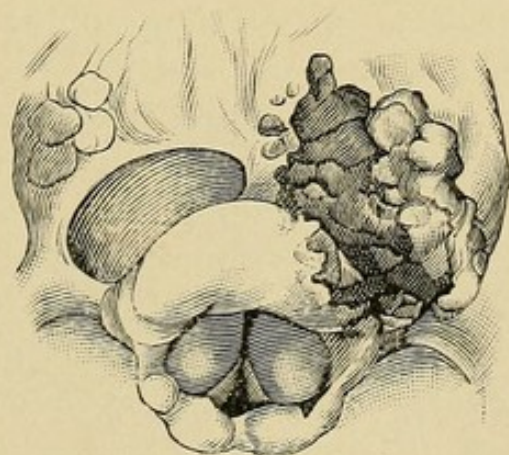
Tuberculous. The edges are soft, the base worm-eaten, cheesy, covered by pale, flabby granulations, and surrounded,

FIG. 48



Papillomata of tongue. Note their multiplicity, their sharply circumscribed character, and the absence of ulceration.

FIG. 49



Tuberculosis of tongue, secondary to laryngeal tuberculosis. Note worm-eaten character of ulcer and the smaller tubercles and ulcers which surround it.

as a rule, by miliary tubercles or smaller ulcerations. There are usually evidences of tuberculosis in the lungs, bones, or joints.

Syphilitic. The edges are soft and sharp, the base waxy, and there are, as a rule, other evidences of syphilis in the bones, skin, etc. On using iodides and mercury the ulcer rapidly heals.

Actinomycotic. Are distinguished by the discharge of minute yellowish granules *containing the ray fungus*.

Carcinomatous. The edges are everted and indurated, the base is extensive and covered by unhealthy, easily bleeding granulations, and there is marked impairment of the movements of the tongue. The course is rapid; there is marked glandular involvement, early cachexia, and metastases.

In every case it is essential to obtain a careful family and previous history, and to elicit as accurately as possible an account of the earliest evidences of the disease, its duration, and its course. The physical examination should include the skin, bones, and internal organs; and, finally, in doubtful cases a section should be given to the pathologist for examination.

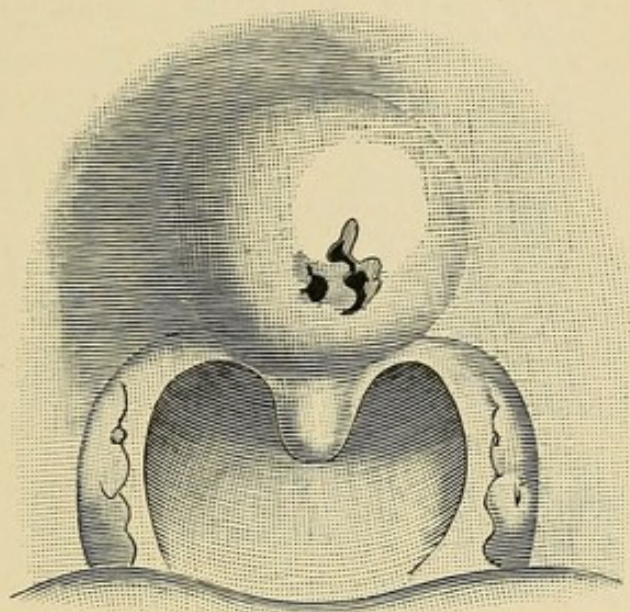
DISEASES OF TONSIL AND PHARYNX.

What has been said of tuberculosis, syphilis, and actinomycosis of the tongue applies equally well of these affections when they are located in the tonsils and pharynx. Enlargement of the tonsils is due to abscess, chronic hyperplasia, lues, or malignant disease. An acute onset of constitutional symptoms, together with pain, swelling, and fluctuation of the organ, characterize the acute abscesses; while a soft, boggy, fluctuating enlargement coming on after one or more previous attacks of acute inflammation, and without constitutional disturbances, speaks for a chronic abscess.

Gradually developing nasal voice and mouth breathing, with repeated attacks of acute inflammation of the throat, cough, or ear discharge, especially in young children, suggest chronic hyperplasia of the tonsils and adenoid vegetations in the nasopharynx. The tonsils in these cases are enlarged and soft, sometimes their crypts are distended into little cysts, and again the orifices of the crypts are plugged with a pearly white material. The cysts may attain the size of a large walnut; their rather rapid development, tense and

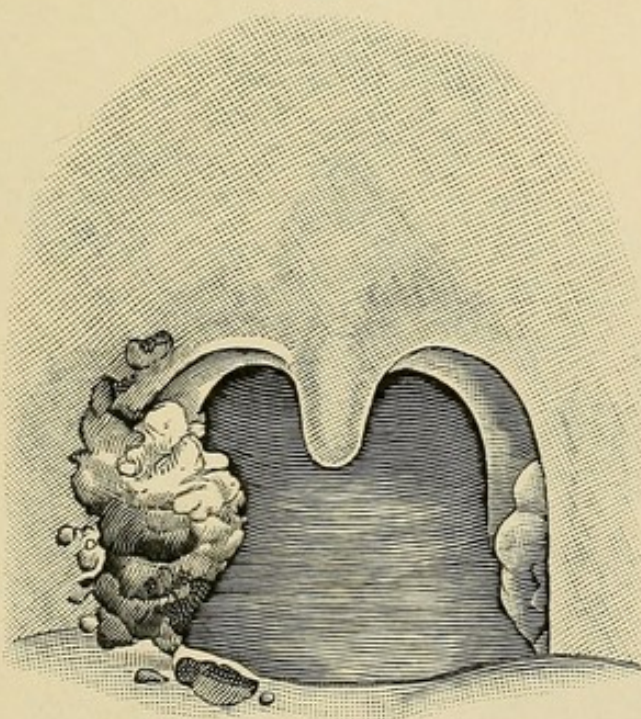
fluctuating character, and mobility upon the deeper structures, readily proclaim their benign cystic nature.

FIG. 50



Ulcerating gumma of soft palate; smooth, soft, elastic tumor in a syphilitic subject.

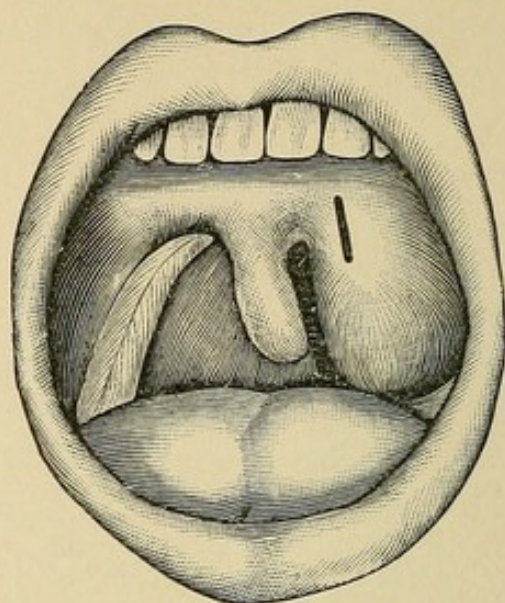
FIG. 51



Tuberculosis of the right tonsil. Note worm-eaten character of edges of ulcer and the surrounding tubercles and smaller ulcerations.

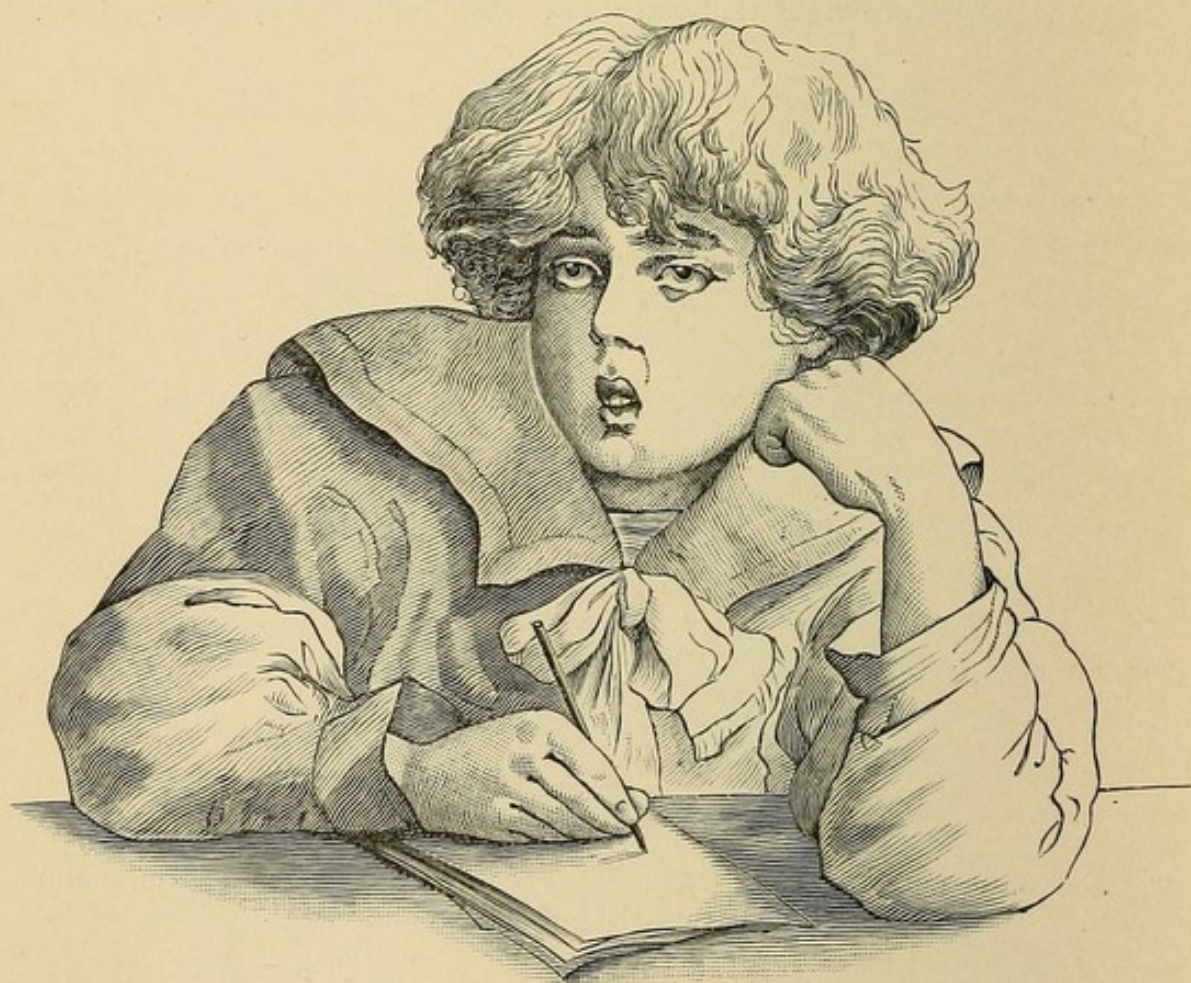
The swelling of the tonsil due to sarcoma is hard, increases rapidly in size, soon becomes fixed to the surrounding tissues

FIG. 52



Peritonsillar abscess on the left side. (Coakley.)

FIG 53



Adenoid facies. Note the half-open mouth, the atrophic and immobile nostrils, the obliteration of the naso-labial folds, and the lack of facial expression.

and ulcerates, leaving an easily bleeding, fungous, indurated mass. It occasions considerable pain, which radiates to the ears, and when the tumor attains sufficient size it interferes with breathing and swallowing. In its early stages it may be mistaken for chronic hyperplasia, but this latter is, as a rule, bilateral and with it the tonsil is much softer in consistency. A gumma, especially when ulcerated, may be mistaken for sarcoma; but its appearance and feel before ulceration, viz., that of an elastic, circumscribed, soft nodule which is not fixed, and the entire dissimilarity of the ulcers, together with a previous history of syphilis and the presence of other syphilitic lesions, readily enable us to differentiate the two conditions.

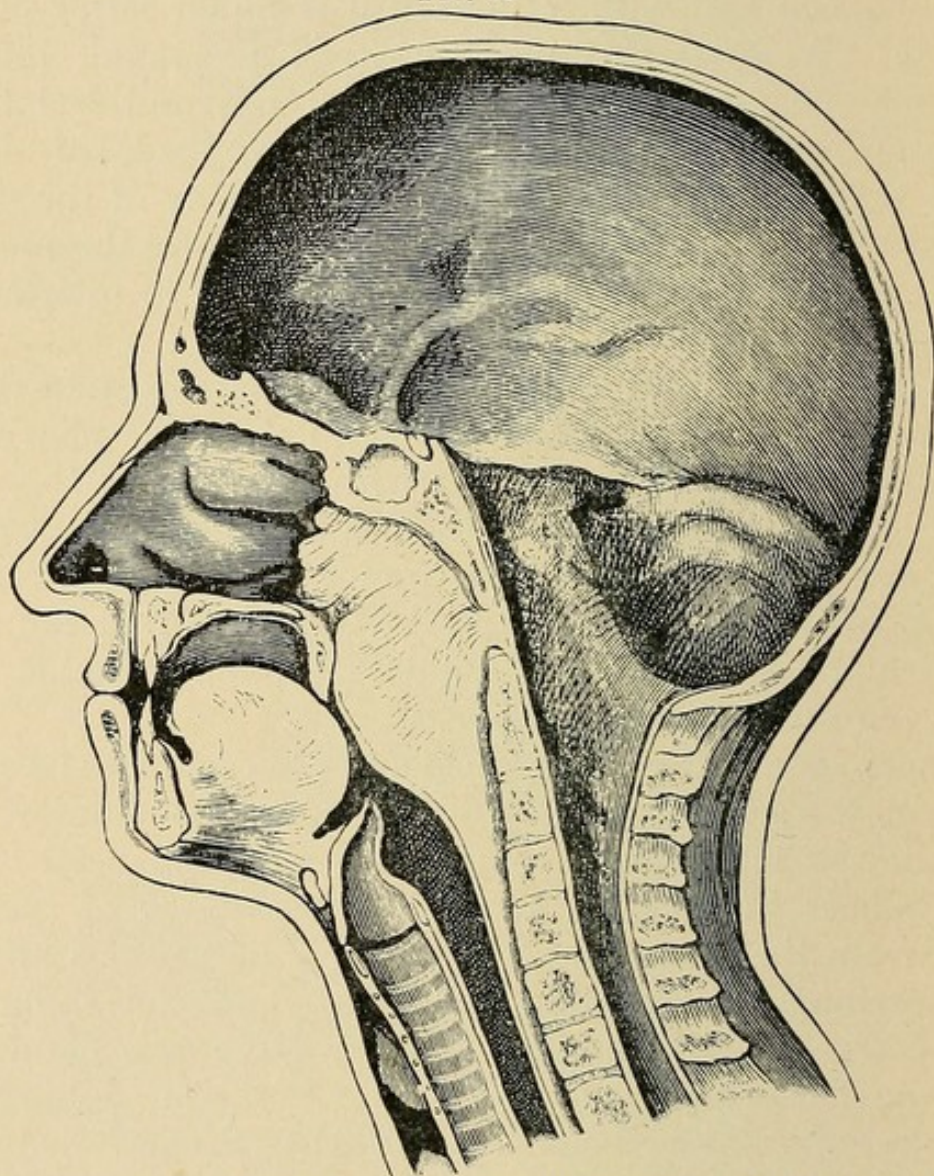
We must be careful not to confound a sarcomatous degeneration of the lymphatic gland which lies on the pharyngeal wall directly behind the tonsils with sarcoma of the tonsil itself. This is a likely error, because the enlargement of the lymphatic gland gives rise to a tumor in the oropharynx on the top of which rides the tonsil. The fact that the tonsil is soft and movable and not enlarged should enable us to make the differential diagnosis.

Cancerous disease of the tonsil and pharynx is to be recognized by the same characteristics as pertain to similar affections of the mouth and tongue. Pain in the head or ear, disturbances in hearing, and a foul discharge of pus and blood from the nose are often the first indications afforded by a carcinoma in the vault of the pharynx, just as induration and enlargement of the submaxillary lymphatic glands are often the first evidences of a carcinoma in the mesopharynx or hypopharynx. These facts should teach us to carefully inspect and palpate these parts when an elderly subject complains of pain in the ear, or foul nasal discharge, or notices a swelling in the submaxillary region. In the later stages of cancerous disease of these parts the characteristic ulcer and the marked disturbances in breathing and swallowing leave little doubt as to the nature of the malady.

In children and in young adults we sometimes find, as a cause for nasal voice, mucopurulent nasal discharge, and mouth breathing, a tumor in the vault of the nasopharynx which projects from the base of the skull. The tumor is

hard and nodular; as it grows it fills the nasopharynx, pushes the superior maxillary bones forward, the roof of the mouth downward, causes the eyeballs to protrude from their sockets, and completely obstructs the posterior nares. It thus inter-

FIG. 54



Retropharyngeal tumor, causing interference with breathing and swallowing. The diagnosis is readily made by inspection and palpation of the oropharynx. (Albert.)

feres with breathing and swallowing. Such tumors are usually fibrosarcomata.

DISEASES OF THE SALIVARY GLANDS.

Tumors of the Floor of the Mouth.—Of swellings which bulge into or occupy the floor of the mouth, we must distinguish between those which develop acutely and those

which form slowly; the former are due to acute inflammation, the latter to chronic inflammation, syphilis, tuberculosis, neoplasms, or cysts of the submaxillary or sublingual salivary glands, the submaxillary lymphatic glands, the Blandin-Nunin glands, to cell inclusions that have taken place during fetal development, or to affections of a persistent thyroglossal duct.

Acute Inflammatory Swellings of Salivary Glands.—An acute onset of pain, tenderness, and moderate swelling of the submaxillary or sublingual glands, with slight constitutional disturbances, characterize simple inflammation of these organs;

FIG. 55

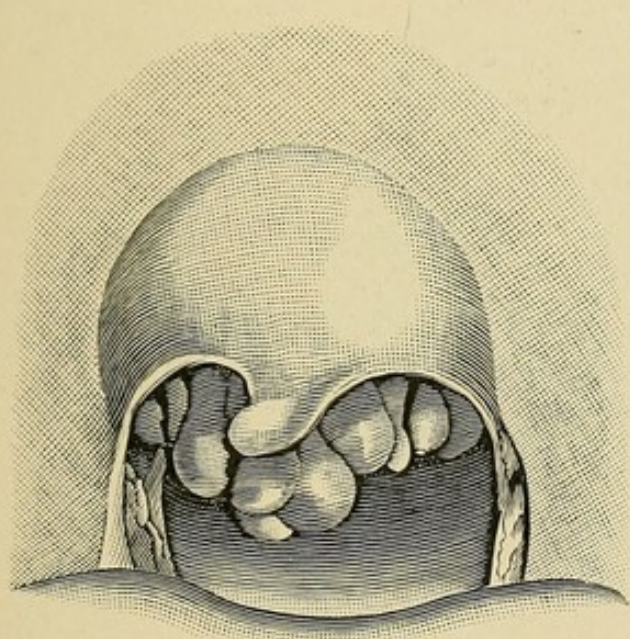


FIG. 56

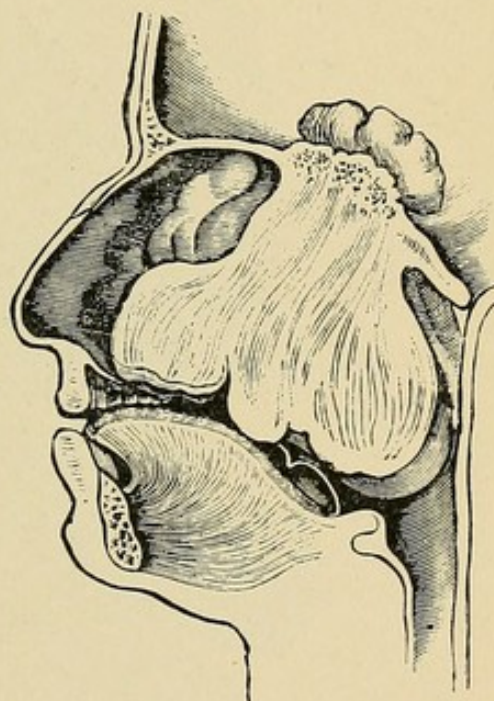


FIG. 55.—Vegetant myosarcoma of nasopharynx.

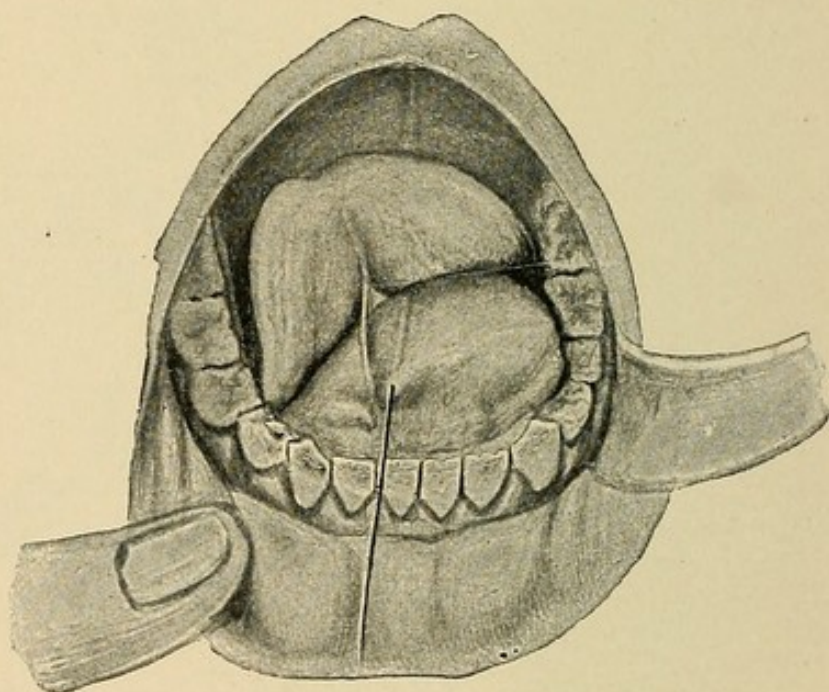
FIG. 56.—Fibrosarcoma of the nasopharynx. (Albert.)

whereas exquisite pain and tenderness with severe constitutional symptoms and extensive brawny induration of the floor of the mouth and submaxillary region of the neck indicate a phlegmonous inflammation of the submaxillary gland—*i. e.*, an angina Ludovici or cynanche. The more gradually appearing swellings most often seen are ranulæ, dermoid and thyroglossal cysts, chronic inflammatory tumors of the submaxillary and sublingual salivary glands, neoplasms of these latter organs, and inflammatory or neoplastic tumors of the submaxillary lymphatic glands.

Ranula.—A rounded or egg-shaped cystic tumor which is neither tender nor painful, situated under the tongue, is characteristic of ranula; such a tumor is due to a cystic dilatation of the chief ducts of the Blandin-Nunin glands, which are situated at the tip of the tongue.

Salivary Calculus.—A smooth, somewhat tender, fluctuating swelling of the submaxillary or sublingual salivary glands appearing somewhat rapidly, but without constitutional symptoms, should always suggest the possibility of its being

FIG. 57

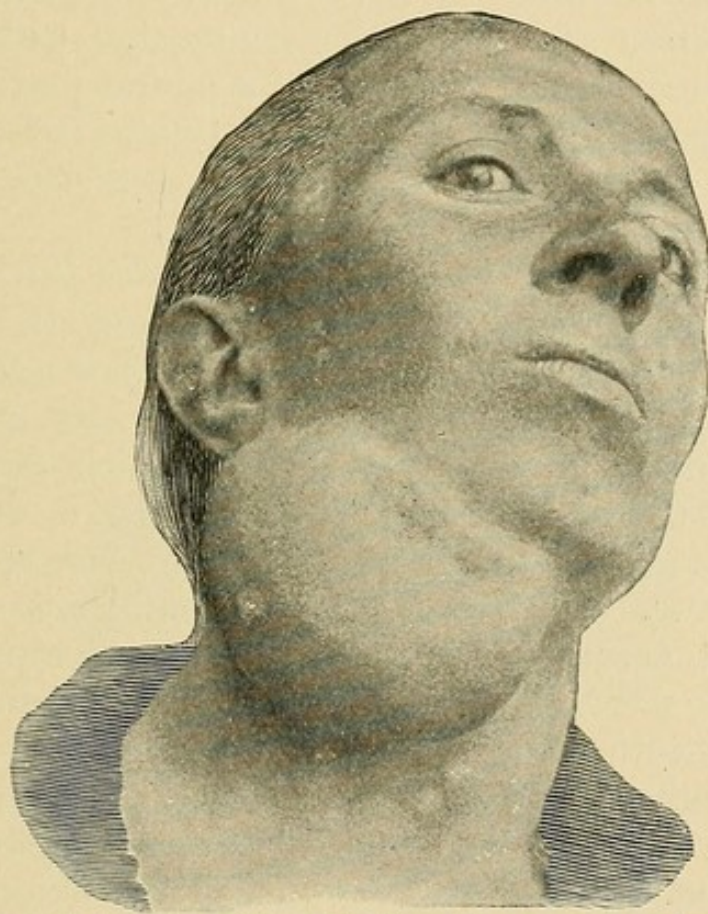


Ranula. (Von Bergmann.)

due to occlusion of the ducts of these glands by a calculus. If a hard nodule is felt in the duct, or if a bristle passed into the duct meets with a hard, gritty obstruction, the diagnosis of a calculus in the duct may safely be made; only rarely does the calculus protrude from the duct so as to be visible. It should also be remembered that an acute inflammation may develop in such an occluded gland at any time; if a calculus protrudes from the orifice of the duct, or if the patient gives a previous history of pain or of irritation in the floor of the mouth with or without a swelling of the gland, the cause of the inflammation will become clear.

Chronic Inflammatory Tumors of the Salivary Glands.—The swellings due to chronic inflammation, syphilis, tuberculosis, and to the early stages of the neoplasms of the submaxillary and sublingual salivary glands are very similar in their characteristics and can in most instances only be told apart by histological examination of an excised specimen. A previous history of syphilis, or the presence of other syphilitic lesions, or an *entire* disappearance of the swelling from

FIG. 58



Mixed tumor of the submaxillary gland. (After a case in v. Bruns' clinic.)

antisyphilitic treatment, are strong evidences of its syphilitic character. The safest plan of procedure in the differentiation of these swellings is the following: First administer anti-specific treatment for ten to fourteen days, and if *no marked* improvement follows therefrom, immediately excise a specimen and submit it to histological examination.

Not only are these various swellings of the salivary glands difficult to differentiate from one another, but they are equally hard to distinguish from chronic hyperplasia or other

affections of the submaxillary lymph glands. It happens now and then that an isolated lymph gland in this region becomes enlarged and in every way simulates a swelling of the salivary glands. As the indication for treatment is the same in either case, viz., to excise a specimen for examination, if the swelling is increasing and does not disappear after antispasmodic treatment, the necessity for differential diagnosis is not so pressing.

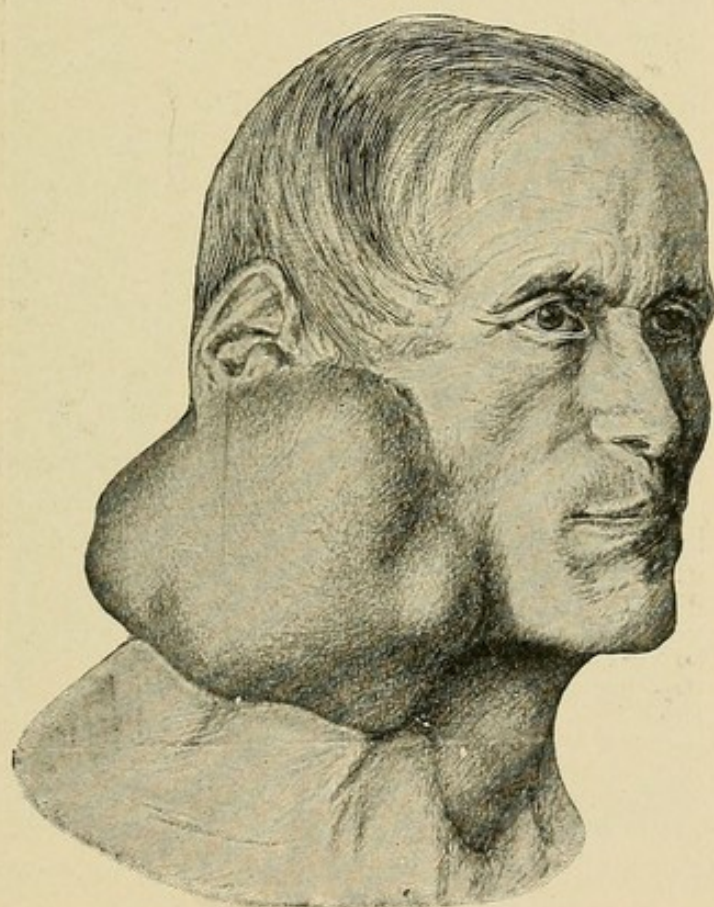
Dermoid cysts and thyroglossal cysts are readily distinguished from swellings of the submaxillary glands by their median position and their close connection with the hyoid bone. Branchiogenetic cysts have a lateral position and so may be erroneously considered as tumors of the salivary glands, but they are fluctuating and have a close connection with the hyoid bone or inferior maxilla.

Tumors of Parotid.—In connection with the swellings of the submaxillary and sublingual salivary glands, those of the *Parotid* are of interest. Pain behind the jaw and below the ear, ushered in by a chill or sudden fever and accompanied by a painful swelling in this region which raises up the lobule of the ear and fills up the hollow in front of the sternocleidomastoid muscle, is due most frequently to an acute parotitis. The existence of an epidemic of parotitis and a slight severity of the constitutional symptoms speak for epidemic parotitis or mumps, whereas a greater severity of the constitutional symptoms in a patient who is suffering from some other acute infectious disease, such as pneumonia, are in favor of the metastatic or suppurative character of the inflammation. The possibility of such an inflammation following operations should not be forgotten.

Circumscribed or diffuse enlargements of the parotid are sometimes due to benign neoplasms—*e. g.*, angioma, fibroma, lipoma, or myxoma; they all grow slowly and are encapsulated and movable. It is to be noted that no tumor is to be considered of parotid origin unless it is connected with the gland and lies beneath the parotid masseteric fascia. Probably the most interesting tumors of the salivary glands are those of mixed character. These are benign, but they may undergo malignant degeneration. They are usually sharply circumscribed and defined from the surrounding

tissues; they are rounded or of elongated oval form, have a smooth or lobulated surface, or, when cartilaginous nodules are present, a nodular surface. Their consistency varies from pseudofluctuating to hard, depending upon the character of the constituent parts; they grow slowly, are covered by normal skin, and are perfectly movable. They may attain considerable size; the presence of hard, nodular masses in a tumor of these organs may be taken as a sign of its mixed character. Such tumors, when small, may be mistaken for

FIG. 59



Mixed tumor of the parotid. (After a case in v. Bruns' clinic.)

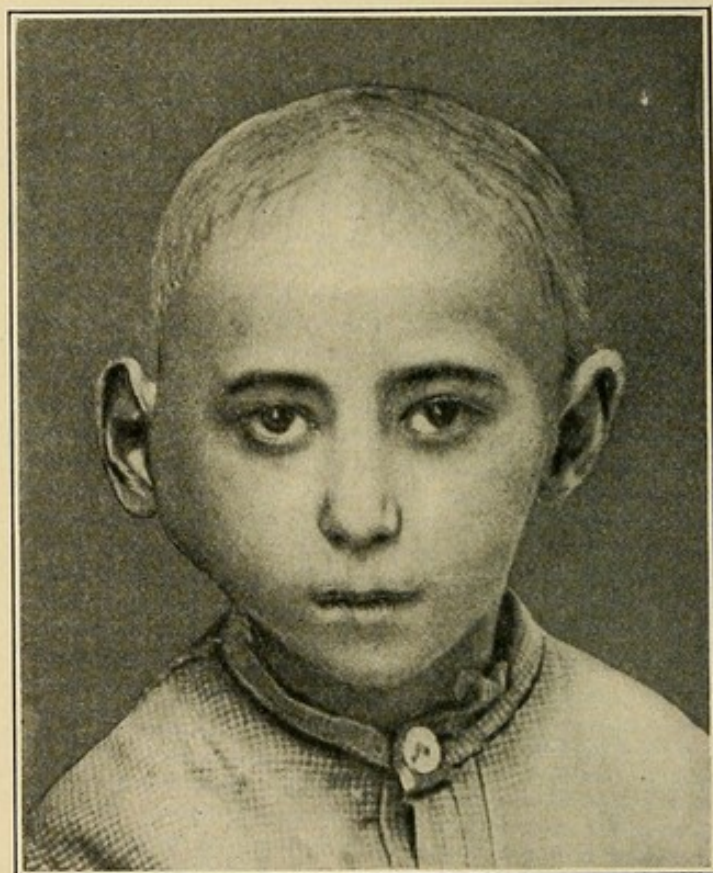
enlargements of the submaxillary or parotid lymphatic glands, from which they can be differentiated only by continued observation and by microscopic examination.

Malignant tumors of the parotid gland in their early stages very strongly resemble the chronic inflammatory, syphilitic, and tuberculous enlargements of this organ. No positive means exist to accurately differentiate these various maladies, and the same rule applies here as was given for the differ-

entiation of the swellings of the submaxillary salivary gland, viz., first try antisyphilitic treatment, and if it does not cause material improvement within ten to fourteen days, cut down upon the tumor, excise a specimen, and examine it under the microscope.

The late stages of malignant disease of these glands are readily recognized, but at this time our diagnosis avails us but little, as no radical cure can be effected. A hard, retracted

FIG. 60



Angiosarcoma of parotid. A diffuse hard tumor situated below the parotid fascia and raising up the lobule of the ear.

tumor with possibly a brawny induration of the skin, as occurs with cancer *en cuirasse*, and an early development of facial palsy with late induration of the submaxillary lymph glands are in favor of the tumor being a scirrhus cancer; whereas a large tumor that early perforates through the skin and forms a large, fungous, ulcerating mass, and that is attended with late facial paralysis and early lymphatic enlargement, speaks for a medullary cancer.

CHAPTER IX.

INFLAMMATORY DISEASES OF THE NECK.

THE complexity of the anatomical structure of the neck and the varied character of the diseases which originate in them make it very difficult at times to determine the exact nature and starting point of a new growth or inflammatory process in this region. Considerable aid in both these particulars is afforded by a complete anamnesis and a thorough physical examination, but a decision will often be best arrived at by a process of exclusion.

ACUTE SUPPURATIVE INFLAMMATIONS.

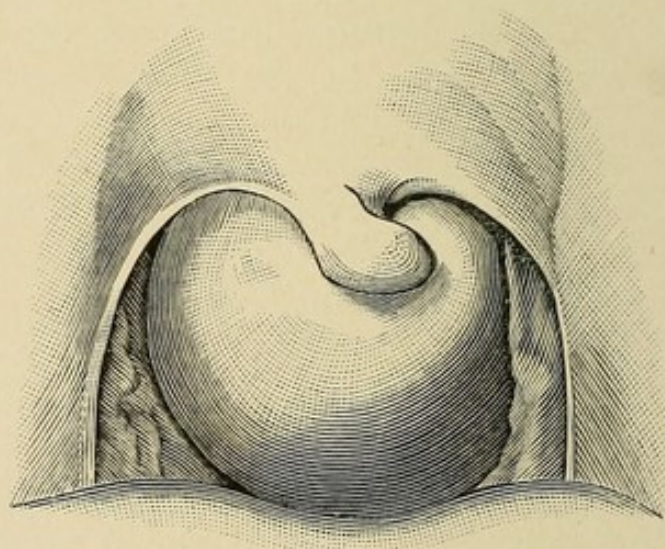
The severity of the constitutional symptoms that attend the acute inflammatory affections of the neck causes these maladies to stand out in strong contrast to the chronic inflammatory and neoplastic diseases of these parts.

Frequently, and especially in the case of phlegmonous inflammations arising in structures which are encapsulated in or covered by the dense fibrous cervical fascia, these constitutional symptoms are very severe; the temperature rising to 104° or 105° , the pulse to 120 or over, and that with great prostration. An acute onset of such severe constitutional symptoms, together with the rapid formation of a brawny, tender, exquisitely painful swelling in the neighborhood of the submaxillary gland, in the thyroid gland, in the deep cervical glands along the anterior border of the sternocleidomastoid muscle, or in the deep cervical cellular tissue, are pathognomonic of a deep phlegmonous inflammation. The most frequent site of such an inflammation is in the submaxillary gland or its immediate surroundings, where it goes under the name of *Angina Ludovici* or *Cynanche*. In this location it interferes markedly with opening the mouth, mastication,

and swallowing. The breath has a fetid odor, the saliva dribbles constantly, and at any time the swelling may extend to the glottis, in which case suffocation results unless prompt relief by tracheotomy is afforded.

Acute infection of the retropharyngeal glands from the nasopharynx during scarlet fever, influenza, diphtheria, typhoid fever, etc., or perforation of the retropharyngeal wall by neoplasms or foreign bodies, frequently gives rise to *acute retropharyngeal abscess*. The pus in these abscesses may burrow forward toward the pharynx, or laterally to the inner or outer side of the sternocleidomastoid muscle, or downward to the mediastinum or upward to the submaxillary

FIG. 61



Retropharyngeal abscess.

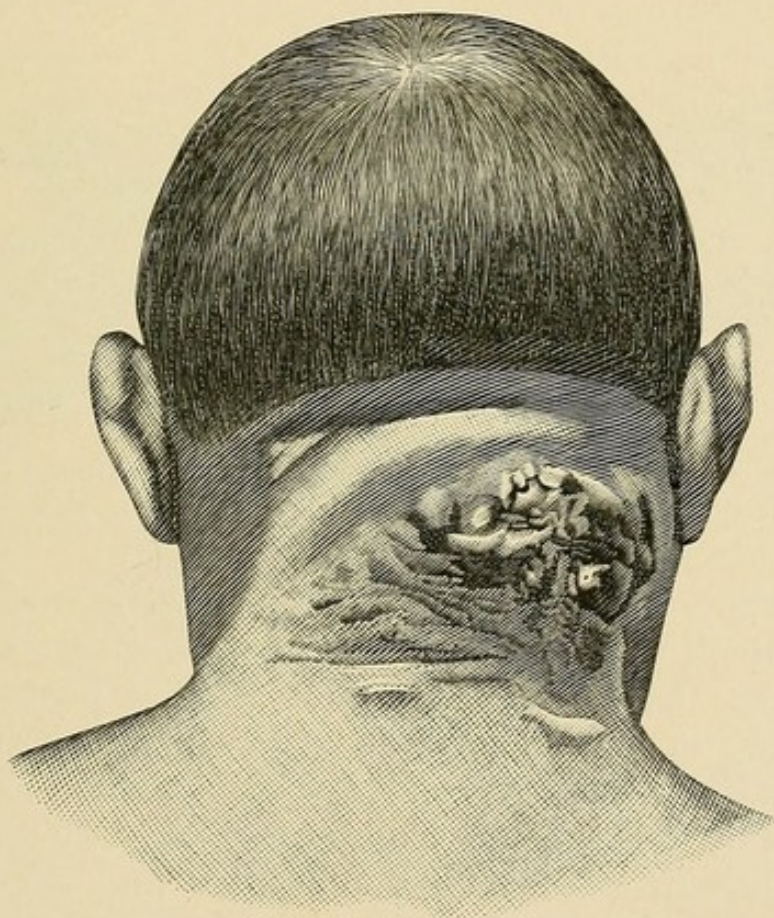
region, forming at these sites a fluctuating, tender, painful mass. When an abscess forms in the retropharyngeal tissues, the neck is held rigid and dorsally flexed, and swallowing and respiration are impeded in proportion to the extent of compression or encroachment of the swelling upon the larynx and œsophagus.

A sudden onset of fever, with nasal voice, impaired respiration and difficulty in swallowing, in a child who has suffered with nasopharyngeal disease, are very suggestive of a retropharyngeal abscess, and they should always prompt us to examine the retropharyngeal wall with the finger, and to search for a fluctuating, tender swelling in the neck, behind

the carotid vessels to the inner or outer side of the sternocleidomastoid muscles.

The impaired respiration and difficulty in swallowing may lead to the supposition of laryngeal or œsophageal stenosis, a supposition that can at once be set aside by visual examination and palpation of the oropharynx. Laryngeal stenosis in children is furthermore usually diphtheritic and is evidenced by patches of membrane on the uvula, tonsils, or in

FIG. 62



Carbuncle of neck. (Lexer.)

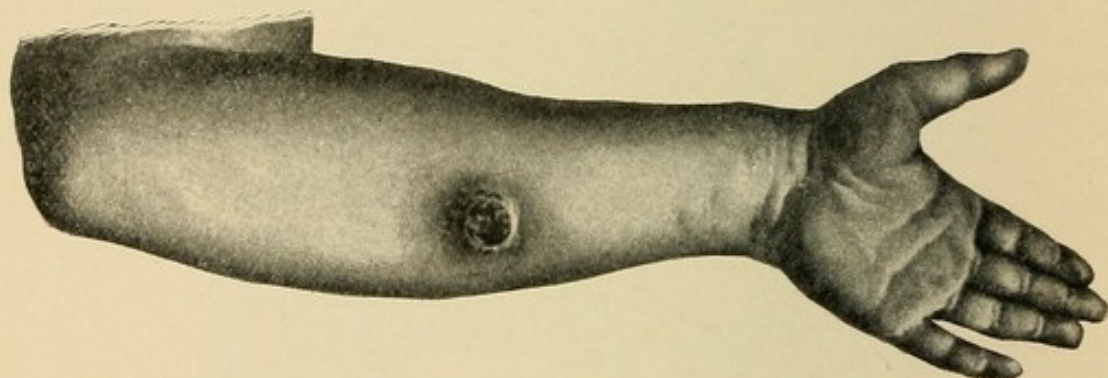
the nasopharynx, by a characteristic metallic barking cough, by respiratory stridor and hoarseness.

It is to be noted, however, that retropharyngeal abscess may complicate diphtheria of the nasopharynx. The exact condition of affairs is at once recognized when the bulging, fluctuating swelling behind the pharynx and overhanging the superior laryngeal and œsophageal orifices is seen and palpated.

Of the superficial acute suppurative inflammations it is necessary to differentiate the carbuncles, the furuncles, and the anthrax pustules.

Carbuncles.—These are frequently found in diabetics, hence the importance of always examining the urine of such patients. They occur most frequently on the back of the neck, as single, livid, red, painful, tender, ill-defined swellings, the surface of which is studded with small vesicles, which become pustular, break down and lead down to a soft, grayish slough.

FIG. 63



Anthrax carbuncle. Note the area of infiltration on which there are several small vesicles surrounding the central, gangrenous, crust-covered patch. (Lexer.)

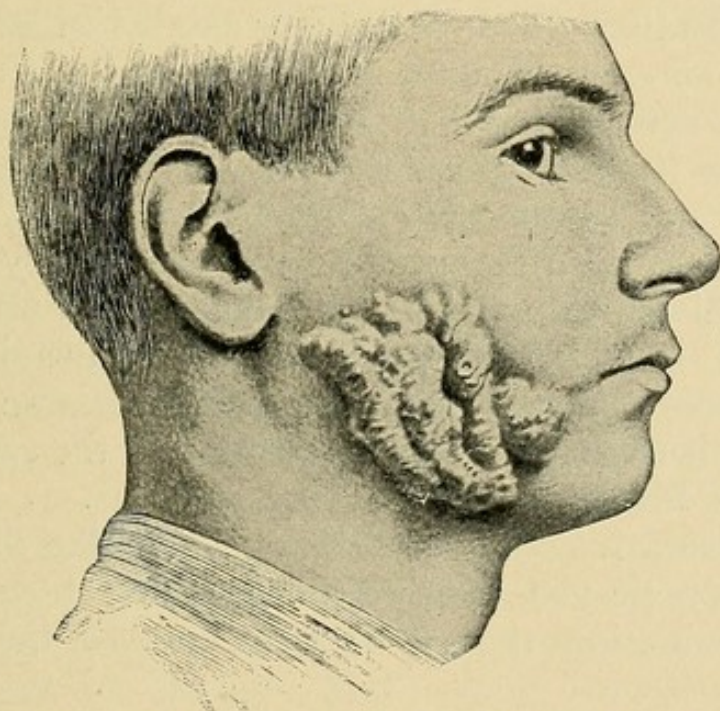
Furuncles.—Furuncles or boils are usually multiple and occur as small, well-defined, painful and tender, red, conical swellings, on the summit of which a small pustule forms, which breaks open and leads down to a greenish slough.

Anthrax Pustules.—Anthrax pustules start as dark vesicles which rapidly become pustular, then burst and dry into a crust. The surrounding skin is swollen and covered by a crop of fresh vesicles. After forty-eight hours constitutional symptoms appear: fever, delirium, diarrhœa. In some instances the pustules resemble a diffuse carbuncle, the overlying skin being of erysipelatous redness. The diagnosis is always to be confirmed by an examination of the vesicular contents for the bacillus anthracis.

CHRONIC SUPPURATIVE INFLAMMATIONS.

The chronic suppurative inflammations which originate in the glandular structures are tuberculous, pyogenic, or syphilitic in character, while those that occupy the cellular tissue are due to actinomycosis, or to tuberculous or syphilitic caries of the cervical vertebræ, mastoid process, sternum, etc. Caries of the vertebræ occasion the chronic retropharyngeal abscess.

FIG. 64



Actinomycosis of cheek originating in lower jaw. (Illich.)

Tuberculous Glandular Abscesses.—The tuberculous glandular abscesses form characteristic, slowly growing, circumscribed, fluctuating, painless swellings. As a rule, the lymphatic glands of an entire group become fused together, and break down into one or more abscesses. The presence of scars and fistulæ from previous suppurations and the existence of tuberculous foci in the lungs, bones, etc., make the diagnosis positive.

Actinomycosis of Cellular Tissue.—Actinomycosis of the cellular tissue manifests itself as a diffuse, slowly growing, painless swelling which gradually softens in the centre while its periphery is hard and brawny. The skin becomes livid

and breaks down, and from the sinus is discharged a slimy, purulent material containing yellowish granules that on microscopic examination show the ray fungus. Acute phlegmonous actinomycosis due to a mixed bacterial infection is a rare manner in which the disease manifests itself. The actinomycotic infection can usually be traced to the socket of a carious tooth where the fungus has gained entrance; the disease spreads by direct continuity through the cellular tissues; it is never carried by the lymphatics to the glands, which latter are consequently not enlarged.

Actinomycosis is distinguished from *tuberculosis* by the diffuse induration which surrounds small foci of softening, and by the absence of glandular enlargement. From *malignant disease* actinomycosis is to be differentiated by the areas of softening in the indurated mass, by the extent of the induration, and by the absence of glandular involvement.

From *gumma* the differentiation is to be made by the lack of a previous history of syphilis, by the absence of other tertiary lesions, and by the finding of the ray fungus in material aspirated from the swelling. The administration of iodides will not avail much in the differentiation of the two diseases, for this acts favorably upon both conditions.

In the ulcerative stage of actinomycosis, the discharge of the characteristic yellowish granules, which show on microscopic examination the ray fungus, makes the diagnosis clear.

Tuberculosis or Syphilis of the Cellular Tissue.—Tuberculosis or syphilis of the cervical vertebræ may occasion a *chronic retropharyngeal abscess* which points either in the retropharyngeal wall, causing a nasal character to the voice, impaired respiration and dysphagia, or appears in the lateral region of the neck, to the inner or outer side of the sternocleidomastoid muscle, but always behind the carotid vessels.

The usual evidences of spondylitis—viz., rigidity of the neck, deformity of the cervical spine, and local pain over the diseased vertebræ on pressure and motion—will throw light upon the character and origin of a fluctuating swelling in the retropharynx, or in the lateral region of the neck to the inner or outer side of the sternocleidomastoid muscle with the carotid vessels lying upon it. The nasal voice and impaired

respiration and dysphagia may first attract attention to the retropharyngeal tumor. The previous history of syphilis will lead us to suspect this as a cause of the vertebral caries, especially if the patient is otherwise healthy and has no other tuberculous foci.

Tuberculosis or syphilis of the mastoid process, sternum, etc., may give rise to a cold abscess in the cellular planes, which can be readily recognized and traced to the primary focus of disease.

CHRONIC FLUCTUATING SWELLINGS.

A chronic fluctuating swelling of the neck unattended by constitutional symptoms, and with no especial pain or tenderness, may be a *cystic neoplasm*, an *aneurysm*, or a *chronic abscess*.

For their differentiation it is important to remember that chronic abscesses are in the vast majority of cases tuberculous, actinomycotic, or syphilitic, and are secondary to diseases of the glands, cervical vertebræ, sternum, mastoid process, or lower jaw, etc. If the glands do not show the characteristic fusing into masses; if there are no irregular scars or fistulæ on the neck, pointing to previous suppuration of these structures; if the cervical vertebræ, mastoid, sternum, lower jaw, etc., are not diseased, and if there is no primary actinomycotic focus in the lower jaw, the swelling is not an abscess, but a neoplasm or an aneurysm, for the differentiation of which the reader is referred to page 58.

CHAPTER X.

TUMORS OF THE NECK.

LYMPHATIC GLANDULAR TUMORS.

THE lymphatic glandular apparatus may be affected by a variety of inflammatory and neoplastic diseases and from macroscopic appearance the differentiation between them is often extremely difficult and at times even impossible. The difficulty will be appreciated when it is remembered that the pathologist is often unable to positively determine by microscopic examination the exact nature of the diseased process. The trouble lies partly in our imperfect classification of the diseases of the glandular apparatus, which permits clinicians to group under one nomenclature entirely different types of pathological processes, and partly in the fact that the causation of some of the glandular diseases is still very little understood. It will frequently be necessary to use all the means at our disposal in order to arrive at a correct diagnosis of the abnormal conditions of the lymphatic glandular apparatus; this includes a careful antecedent history of the patient, a thorough physical examination of the internal organs, and of the region which is drained by the lymphatics emptying into the enlarged glands, a careful blood count and hæmoglobin estimation, and, finally, in cases of doubt, an excision of one of the glands for pathological examination and animal inoculation.

While this chapter is to be especially devoted to a study of the diagnosis of and differentiation between the various diseased conditions of the cervical lymph glands, it is to be noted that what is true of these glands is equally true of the glands in other regions. It is more difficult, however, to correctly determine the nature of an enlargement of the cervical nodes than it is to decide upon the character of diseased conditions of the glands in other parts, because

several of the glandular diseases give their first physical manifestations in an enlargement of the cervical nodes, and by the time the axillary, inguinal, mediastinal, or retroperitoneal glands become appreciably affected, such other constitutional signs of the malady have become evidenced as to make the entire clinical picture very clear.

The glands of the neck may be roughly divided into the superficial and deep according as they lie above or behind the deep cervical fascia. The deep glands are again arranged in three main groups, viz., those of the submaxillary region, those between the anterior border of the sternocleidomastoid muscle and the trachea, and those in the occipital triangle, the latter being especially numerous in the supraclavicular fossa.

Single glands of these groups may become swollen and diseased, forming smooth, round or ovoid tumors; or a number of glands of one group or an entire group may be involved, forming nodular tumors, the size and conformity of which vary with the number of glands affected; or all the groups of one or both sides may be involved, forming single or multiple nodular tumors of different size and shape. The *consistency* of glandular tumors varies considerably, depending chiefly on the nature of the disease, and the presence or absence of periadenitis. In the same group may be found soft and hard and fluctuating nodules. Pseudofluctuation in very soft swellings of the hyperplastic type is often to be elicited.

As long as the disease is confined within the capsule of the gland the latter remains movable under the skin and upon the deeper structures. *Periadenitis* results in its becoming adherent to its fellows, to the deeper structures, and to the skin.

The presence or absence of a periadenitis may be taken as a basis for a clinical classification of diseased glands. Thus hyperplasia without periadenitis may be due to (1) a local irritative focus—*e. g.*, eczema, local infection, etc.; (2) tuberculosis; (3) pseudoleukæmia; (4) leukæmia; (5) sarcoma; (6) secondary or tertiary stages of syphilis.

Hyperplasia with periadenitis may be due to (1) tuberculosis; (2) carcinoma; (3) sarcoma; (4) initial and tertiary forms of syphilis.

Glandular Enlargement without Peradenitis. Simple Hyperplasia.—The simple hyperplasia of glands occurs most frequently in young children in whom diseases of the mouth, tonsils, and gums are very frequent, but it may occur at all ages and in all regions of the body from a local focus of irritation and infection. Such glandular enlargement is found in otherwise healthy subjects and subsides when the primary focus of infection or irritation has been done away with.

The differentiation of this condition from the initial stages of tuberculous hyperplasia is very difficult and often impossible; for such swollen glands are more susceptible to infection with tubercle bacilli than healthy ones, and there is no way in which we can determine when the simple hyperplasia becomes tuberculous. A persistent and increasing glandular enlargement with a progressive involvement of new nodes after the subsidence of a local focus of infection or irritation, and especially if peradenitis develops points very strongly to tuberculosis, and even more so when there are other tuberculous lesions in the lungs, bones, etc. From pseudoleukæmic nodes the simple hyperplastic glands are more easily differentiated. In the former there is no local focus of infection, the enlargement is progressive, new nodes being constantly involved, the general health is gradually impaired, and secondary anæmia develops.

Tuberculous Hyperplasia.—Tuberculous hyperplasia without peradenitis must be differentiated from simple hyperplasia occurring in children and from pseudoleukæmia and the secondary and tertiary stages of syphilitic glands.

Clinical evidences in favor of tuberculosis are the presence of other foci of this disease, the tendency to peradenitis and softening of the glands, and a positive response to a tuberculin injection. In all cases of doubt a single node should be excised and submitted to microscopic examination and animal inoculation.

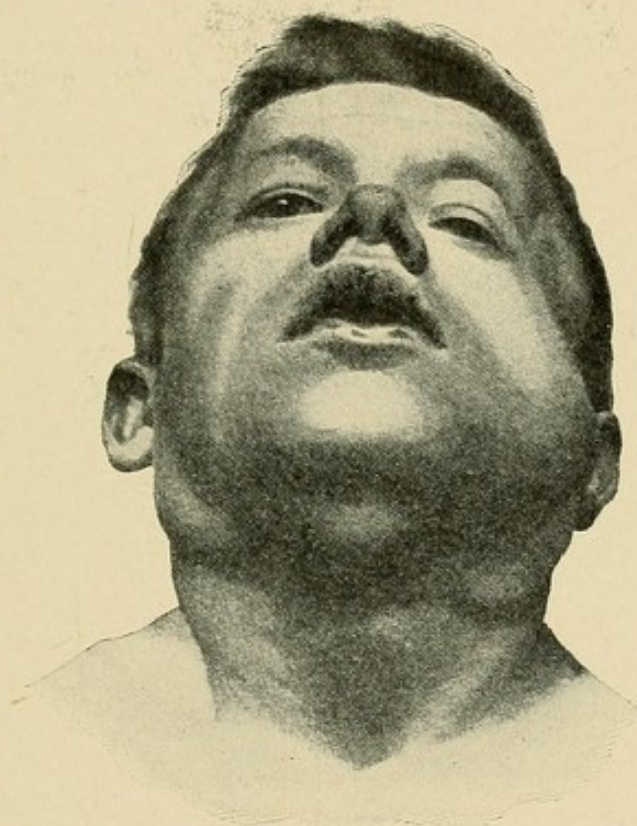
The differentiation of tuberculous from simple hyperplasia has been considered above.

From pseudoleukæmia the differential diagnosis is often impossible, and always very difficult. In favor of the latter are the absence of tuberculous lesions in other organs; the

progressive involvement of new glands in the neck, axillæ, groin, etc.; the uniform consistency of all the glands, deterioration of the general health, the presence of a *secondary anæmia*, and the improvement which may follow from internal administration of arsenic.

Leukæmic glands are easily differentiated from tuberculous ones by the characteristic blood changes that attend this malady. (See p. 42.)

FIG. 65



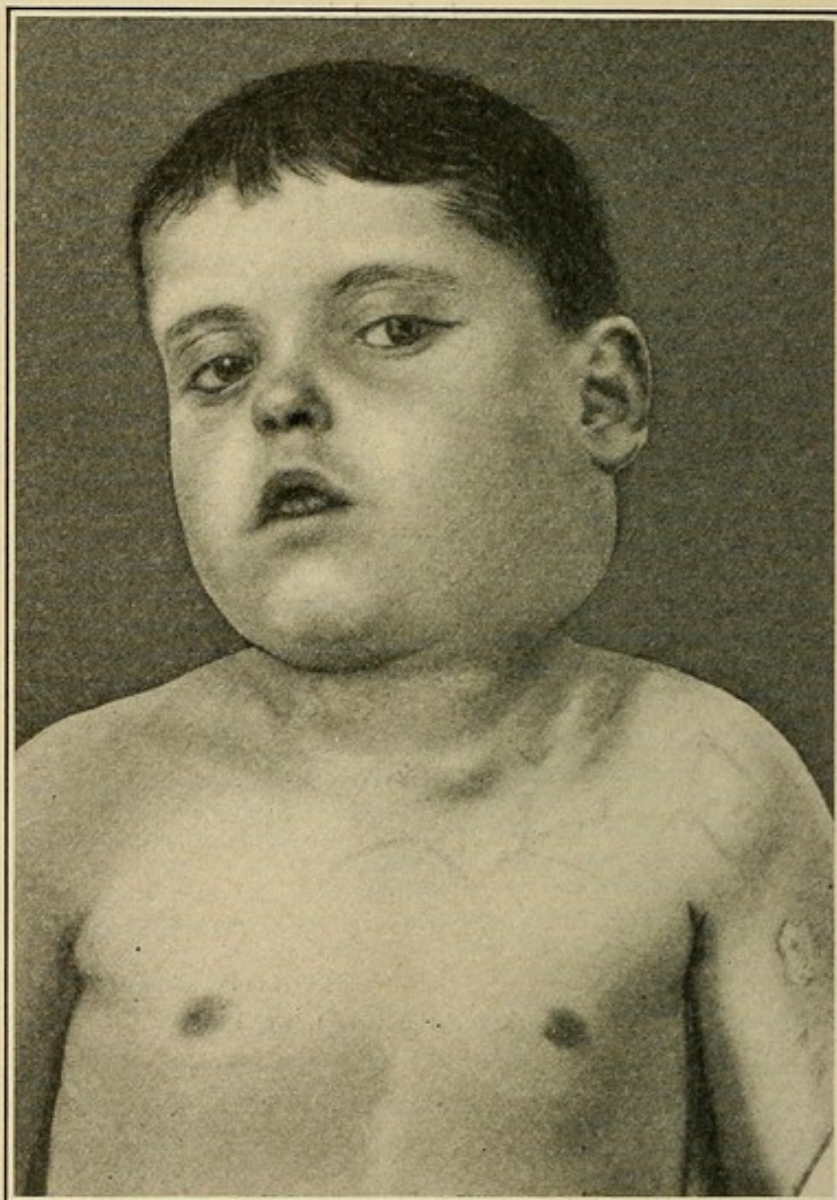
Scrofula. Note the tuberculous enlargement of the cervical lymph nodes, the rhagades of the upper lip, the chronic nasal catarrh, and the conjunctivitis. (Lexer.)

Glandular hyperplasiæ occurring in the secondary and tertiary periods of syphilis are distinguished from tuberculous glands by a history of syphilis, by the existence of other syphilitic lesions on the skin, mucous membranes, etc., and by their disappearance under antisyphilitic treatment.

Pseudoleukæmic Hyperplasia.—Glandular hyperplasia due to pseudoleukæmia (malignant lymphoma) commences as nodular, uniformly soft enlargements of the glands on one or both sides of the neck. The glands of the axillæ, groin, mediastinum, retroperitoneum, become successively enlarged; the ones first affected gradually increase in size, and metas-

tases form in the internal organs. In the latter stages of the disease a marked anæmia and general physical deterioration develop. The glands are never fixed and there is no difference in the consistency of different parts of the tumors.

FIG. 66



Pseudoleukæmic glands of the neck. Uniformly soft, movable tumors.

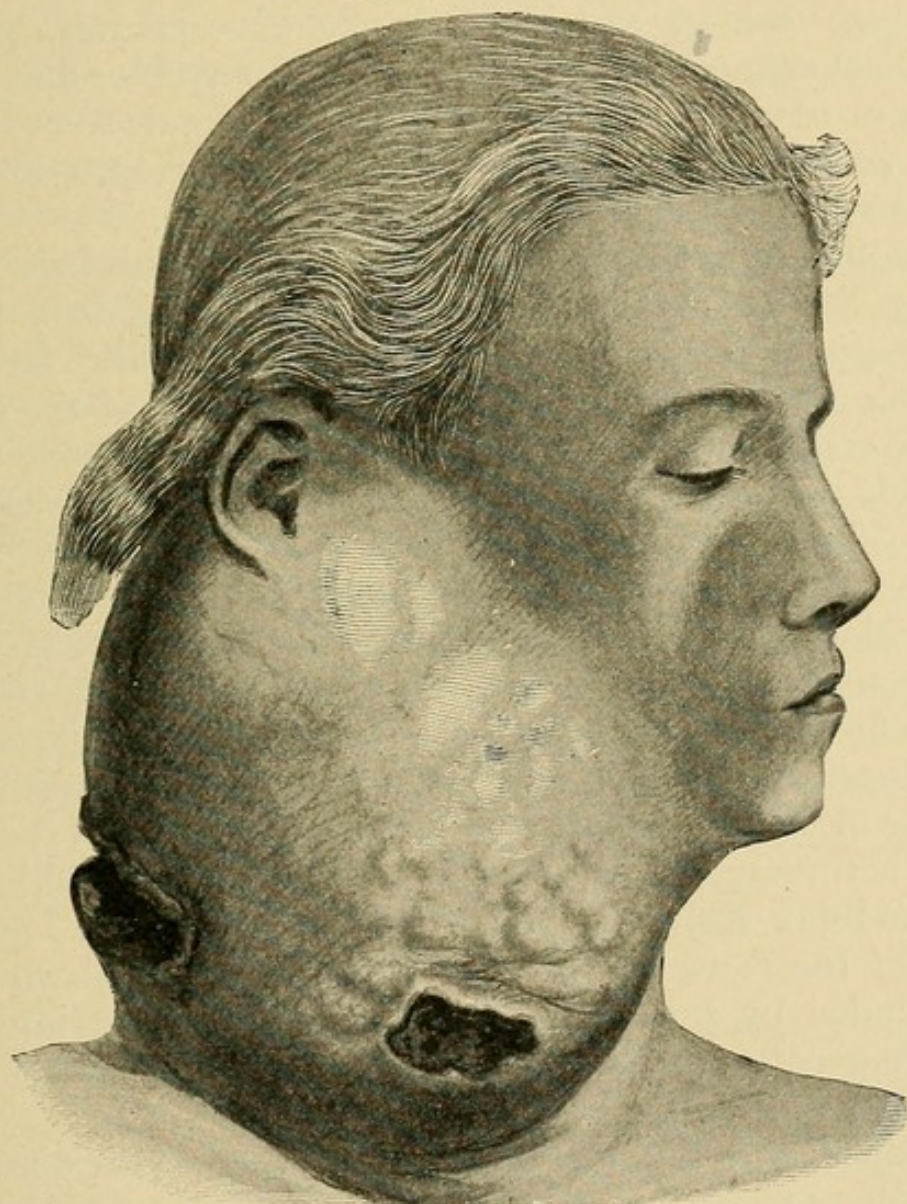
The glandular enlargement due to leukæmia resembles that due to pseudoleukæmia. But in the former the blood shows decided changes (see p. 35), the spleen is enlarged and the anæmia is intense.

The differentiation from simple and tuberculous hyperplasia has already been considered. It is to be noted that

tuberculous and pseudoleukæmic hyperplasia may exist side by side in the same individual.

From leukæmic hyperplasia these glandular enlargements are distinguished clinically by a late appearance of anæmia and an early manifestation of the glandular tumors, whereas

FIG. 67



Lymphosarcoma in a woman twenty-five years of age. Note the regular margins of the ulcer and fungous granulations of the base. (Von Bergmann.)

in leukæmia the anæmia appears first and the glandular enlargement later. An examination of the blood at once decides the diagnosis. (See p. 39.)

Sarcomatous Hyperplasia.—Sarcoma of the cervical glands starts either in the nodes along the carotid vessels or in

the nodes below and behind the angle of the lower jaw. The tumor is smooth, of uniform consistency, and movable in its early stages; later it becomes softer in some of its parts, fixed to the skin and deeper structures, and eventually it ulcerates through its cutaneous covering. In its early stages sarcoma cannot be macroscopically differentiated from pseudoleukæmia. Sarcomatous glands usually grow rapidly, but those behind the jaw may be of very slow growth. Pseudoleukæmic glands are always movable and never soften or ulcerate, whereas sarcomatous glands soon become fixed, and finally soften and ulcerate. Microscopic examination will aid in making an early diagnosis.

Syphilitic Hyperplasia.—Syphilitic hyperplasia of the glands during the secondary stages is readily recognized by the anamnesis and the presence of other syphilitic lesions of the skin and mucous membranes. Gummata of the glands, a rare condition, form smooth elastic tumors which remain movable unless degeneration occurs, in which case they soften, break down, become fixed, and ulcerate. Before degeneration, the history, the otherwise good health (no tuberculosis) of the individual, the absence of other glandular enlargement, and the absence of progression to other glands are sufficient to distinguish these glandular enlargements from those due to tuberculosis, sarcoma, and pseudoleukæmia. In the ulcerating stages the differentiation must be made from actinomycosis and tuberculosis. (See p. 42.)

Glandular Enlargement with Peradenitis.—The diagnosis of the cause of the enlargement of glands which are surrounded by a peradenitis and fused together into irregular masses more or less fixed upon the deeper structures and skin is usually easy.

Tuberculosis.—In tuberculosis at this stage of the disease the glandular masses are soft in some spots and fluctuating in others, there are usually other evidences of tuberculosis in the lungs, bones, or internal viscera, and there may be scars of healed tuberculous abscesses in the neck or in other parts of the body. Such scars are irregular in form, broad, show depressions and elevations, and are adherent to the underlying structures. If the glands are ulcerated the ulcers are irregular and worm-eaten and covered by fungous granulations.

Lymphosarcoma.—In lymphosarcoma there is a single tumor made up of many irregular nodules, the soft, pseudofluctuating consistency of which is uniform. The tumor grows rapidly, attains considerable size, compresses the neighboring structures, and causes rapid deterioration of the general health. Metastases in distant organs are frequently found. After ulceration the defect is surrounded by more regular margins and the base is covered by easily bleeding, sloughing granulations. Microscopic examination at once reveals the nature of the malady.

Carcinoma.—Carcinomatous glands are always secondary to a primary focus of the disease in the face or oropharynx. The glands are uniformly hard; after ulceration the defect is surrounded by everted, hard edges, and the base is covered by reddish, easily bleeding, crusting granulations. In old age a tuberculous gland with hard, calcareous capsule may simulate a carcinomatous nodule, but careful search shows no primary growth, and carcinomatous glands are always fixed.

Glandular Enlargement Accompanying Syphilis.—The glandular enlargement which accompanies the primary stage of syphilis is always attendant upon an initial sore. The swelling attains its maximum size rapidly, and disappears after the use of mercury. Breaking down and ulcerating gummata of the neck have been considered on page 126.

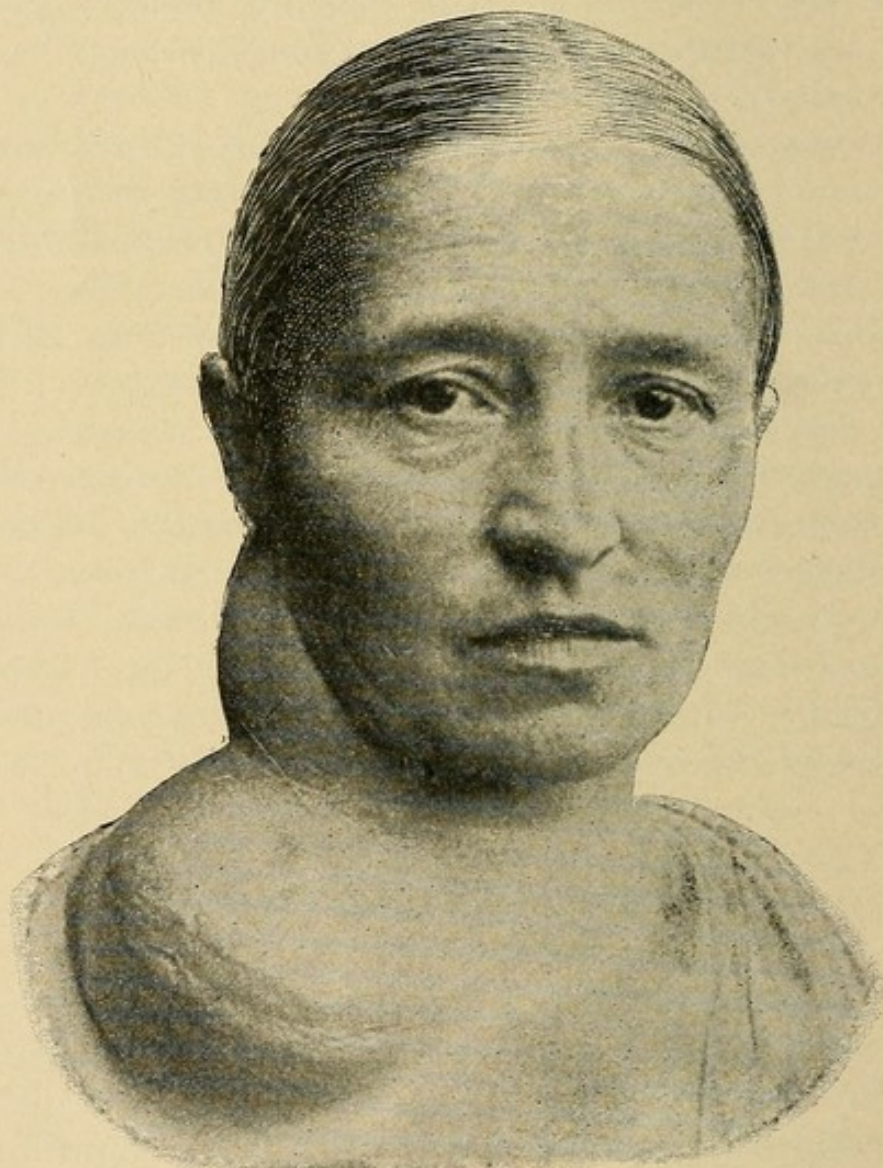
MISCELLANEOUS TUMORS.

For the purposes of diagnostic study we may group these tumors into those of solid consistency and those with fluid contents. The former are similar histologically to the benign and malign solid tumors which arise from connective and epithelial tissues in other parts of the body, but the latter are, for the most part, unique tumors to this region.

Fluid Tumors.—These are wholly or in part fluctuating and elastic. In this class belong the branchiogenetic cysts, the congenital cystic hygroma or lymphangioma, the acquired cavernous lymphangioma, the enlarged bursæ, the echinococcus cysts, the blood cysts, the hæmangioma, the cavernous angioma, and the aneurysms.

These may be again grouped according as the contents are cystic material or blood. If the tumor is compressible, it is a blood-containing one, and is therefore a blood cyst, an angioma, or an aneurysm. It is to be noted, however, that not every blood-containing tumor is compressible—*e. g.*, a blood cyst whose communication with the vein from which

FIG. 68



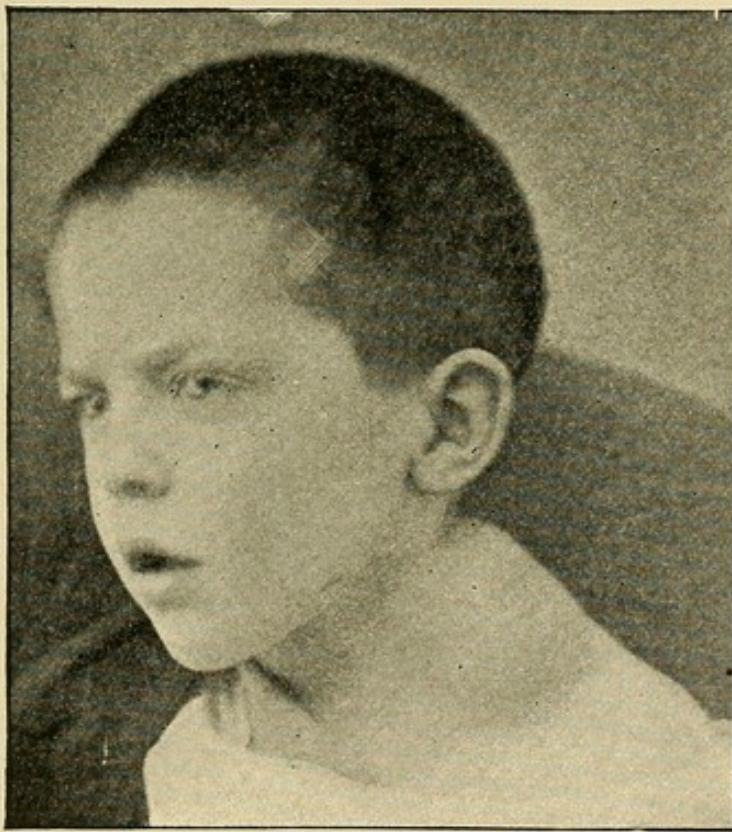
Multilocular branchiogenic cyst. (Von Bruns.)

it arises has become closed. In the absence of compressibility other clinical evidences often aid us in the recognition of blood tumors. Thus, an expansile pulsation in a tumor points to its aneurysmal character; likewise systolic bruit, and increase or decrease in its size when the efferent or afferent vessels are respectively compressed. Should all the

data we can collect be insufficient to enable us to make a diagnosis, then aseptic puncture with a fine needle will afford conclusive evidence as to the contents of a fluid tumor.

Cystic Tumors. 1. **Branchiogenetic Cysts.**—The branchiogenetic cysts are rarely noticed at birth; they develop chiefly in the first three decennials of life, and especially at puberty. They either have a lateral position between the sternocleidomastoid muscle and the median line anywhere from the mastoid process down to the jugulum, sometimes attached to

FIG. 69



Lateral branchial cyst. (Dennis.)

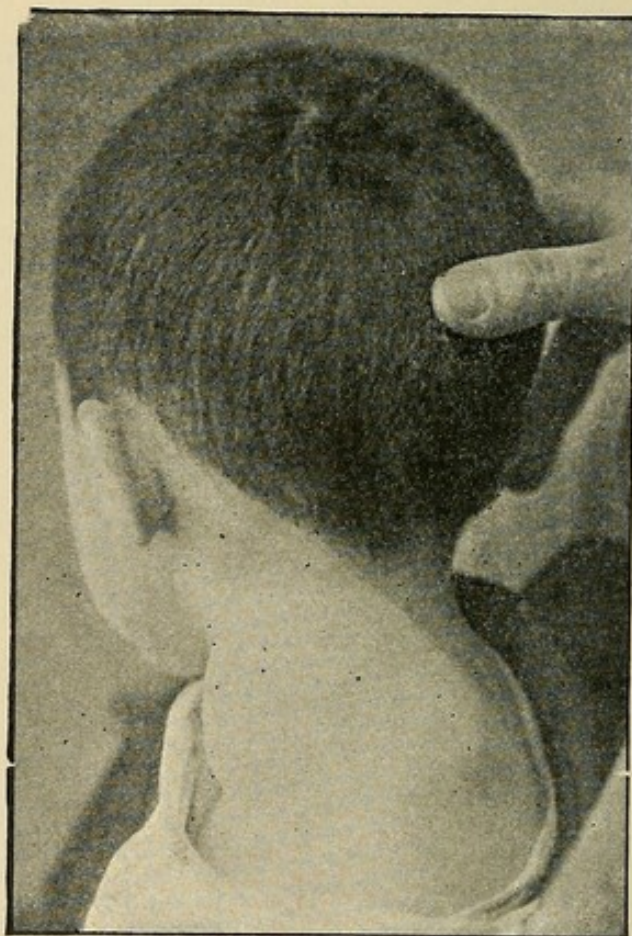
the lower jaw, to the hyoid bone or styloid process, or they have a median position above or below the hyoid bone.

The lateral cysts arise from the second branchial cleft, the median from the thyroglossal duct and sinus cervicalis. They are due to closure of the orifices of a complete or incomplete branchial fistula or to the persistence of the branchial canal or to cell inclusions during the closure of the sinus cervicalis (the latter forming median dermoid cysts).

The contents of these cysts are either a serous fluid, in which case they are known as *hygromata*, or a mucoid material, or an oily, fatty material with hair, teeth, bones, etc., in which case they are known as *dermoid cysts*.

They form ovoid, hen's-egg-sized, usually fluctuating and elastic, unilocular or multilocular tumors, which are movable under the skin, but less movable on the deeper parts, and are not tender. The overlying skin is of normal appearance. They grow slowly and compress the neighboring structures.

FIG. 70

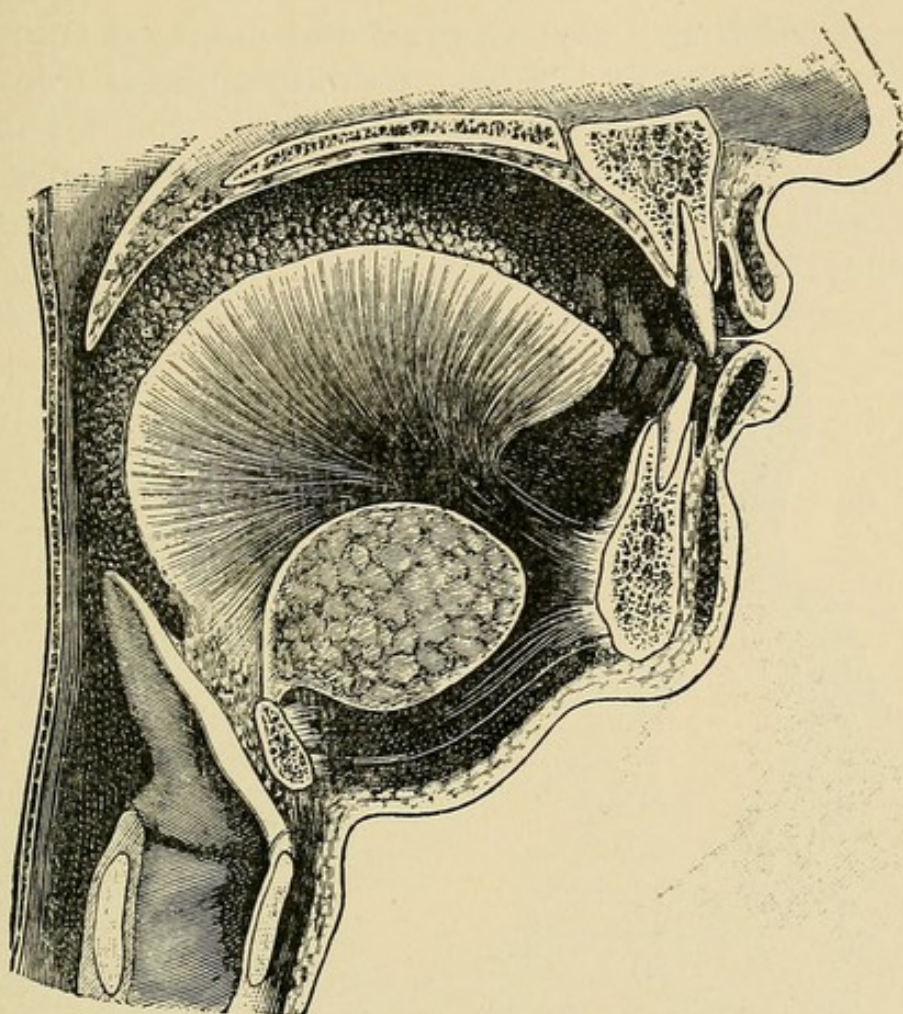


Lateral branchial cyst. (Dennis.) (Side view of Fig. 69.)

The lateral cysts must be differentiated from cystic lymph angioma and aberrant thyroid cysts, and the median ones from enlarged bursæ that lie above or below the hyoid bone, and from chronic glandular abscess.

The cystic lymph angioma are multilocular, lobulated, flaccid, congenital tumors of larger size and more rapid growth; they are more superficial and on aspiration yield a

FIG. 71



Dermoid cyst at base of tongue. (Marehant.)

FIG. 72

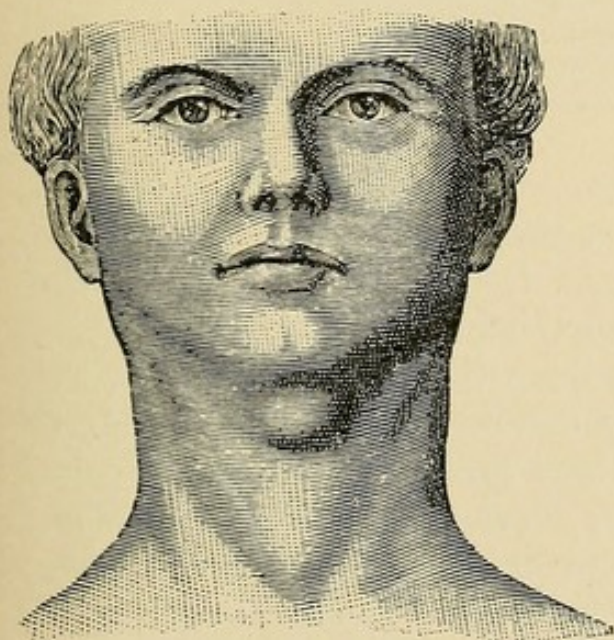
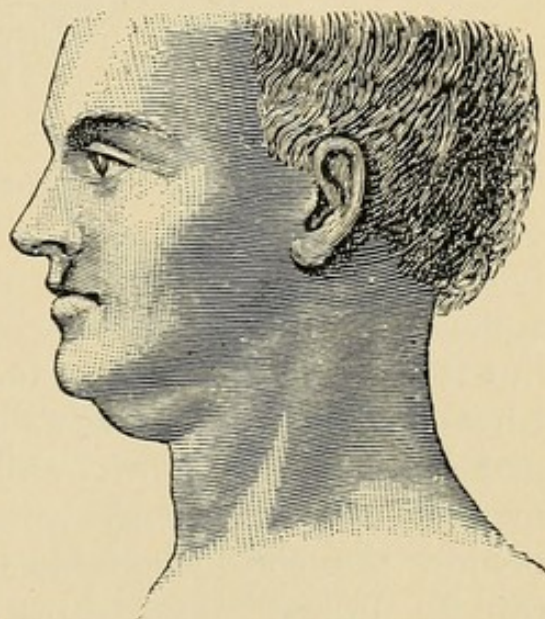


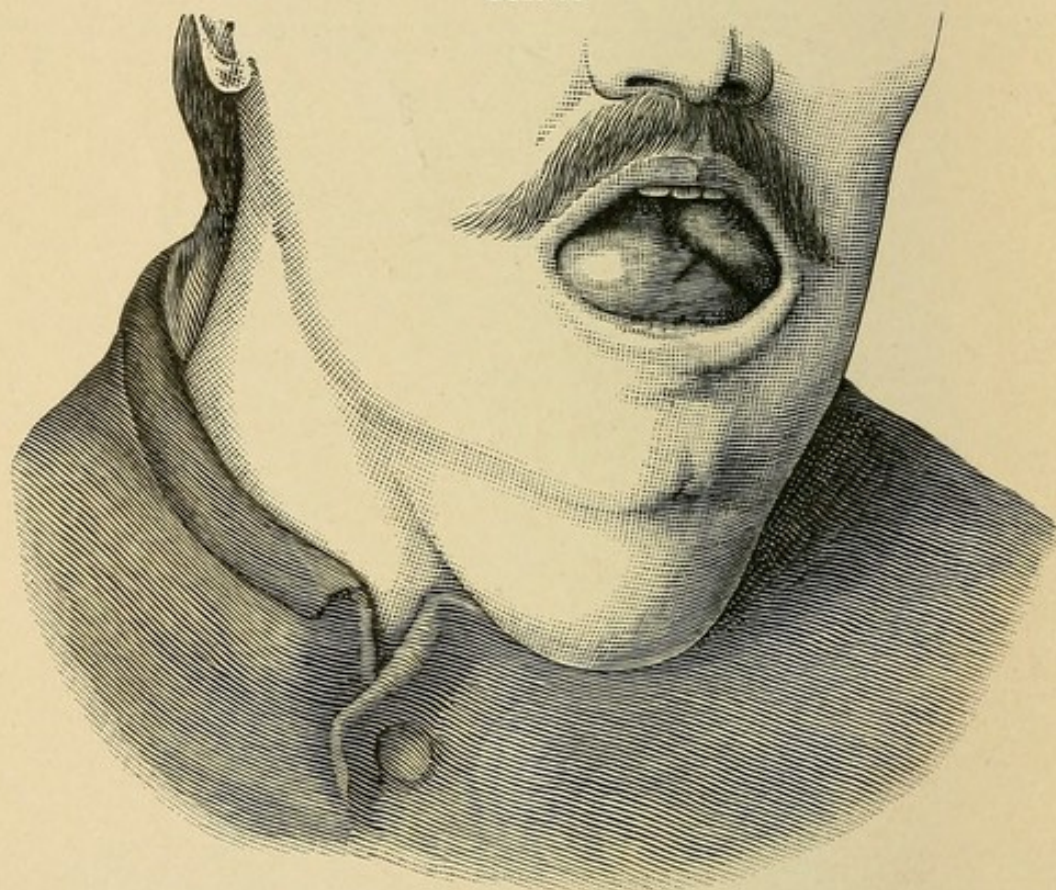
FIG. 73



Median suprahyoid dermoid cysts.

clear, serous, milky, or brownish fluid which coagulates on standing. Aberrant thyroid cysts and enlarged suprahyoid and infrahyoid bursæ can only be differentiated from branchial and dermoid cysts by a microscopic examination. A chronic glandular abscess is slower in its development than is a dermoid cyst; aspiration yields pus, and there are likely to be other glandular nodes.

FIG. 74



Dermoid of the floor of the mouth. (Von Bergmann.)

2. Congenital Cystic Hygroma.—Congenital cystic hygroma or lymphangioma is due to a progressive dilatation of the lymphatic vessels. It is most frequently located in the subcutaneous fatty tissue, whence it invades the deeper parts, but it may be primary in the deep tissues, especially in the carotid sheath, and gradually extend to the surface. It is met with in very young children and in infants and commences, as a rule, in the upper cervical region in front of or behind the sternocleidomastoid muscle, and grows fairly rapidly upward and downward, penetrating everywhere between the various structures and compressing them.

It forms a fluctuating, usually flaccid, lobulated tumor. The overlying skin is movable, at times so thinned as to be translucent, but again thickened and oedematous (elephantiasis). The superficial veins are enlarged.

Aspiration yields a clear, serous, milky, or brownish fluid which coagulates on standing.

Their differentiation from branchial cysts has already been discussed. Accessory thyroid cysts are not lobulated and do

FIG. 75



Congenital cystic hygroma.

not yield a coagulating fluid on aspiration. Deep branching lipomata are not truly fluctuating, do not yield the characteristic fluid on aspiration, and are much more deeply situated.

3. **Cavernous Lymphangioma.**—Cavernous lymphangioma is a rare tumor and develops during adult life. As the preceding growth, it is due to a progressive dilatation of the lymphatic vessels and usually commences in the lower carotid triangle. It forms a lobulated, fluctuating, flaccid

tumor in front of or behind the sternocleidomastoid muscle; aspiration yields a clear or milky fluid which coagulates on standing. These characteristics are usually sufficient to differentiate it from a branchial cyst, or deep lipoma, or cavernous angioma.

4. **Enlarged Bursæ.**—Enlarged bursæ (hygroma), either suprahyoid or infrahyoid, are only occasionally met with. The tumor develops slowly and painlessly; rarely becomes larger than a walnut; is rounded and fluctuating, and is fixed to the hyoid bone, but movable under a normal-appearing skin. Only by microscopic examination can it be differentiated from a median branchial cyst, or an accessory thyroid cyst. When these bursæ become acutely inflamed and suppurate they are to be distinguished from suppurating lymphatic glands by their more acute course and greater tenderness.

5. **Echinococcus Cysts.**—Echinococcus cysts of the neck are rare. They are, for the most part, situated under the sternocleidomastoid muscle, and are characterized by an intermittent increase in size. The cyst is smooth and fluctuating; aspiration thereof yields a clear or turbid, serous fluid which does not coagulate, and which, under the microscope, may show the hooklets of the echinococcus.

The diagnosis from other cysts of the neck is exceedingly difficult, and will usually be made only after incision. Lymphatic cysts are to be differentiated by their coagulating contents, subfacial lipoma by their solid consistency.

Blood Tumors. 1. **Blood Cysts.**—Blood cysts are of uncertain pathogenesis. They are either congenital or first appear in adult life, especially on left side of the neck, and vary in size from a walnut to a child's head. They form smooth or slightly lobulated, fluctuating, elastic tumors, which are movable under a normally appearing skin, and upon the deeper parts. The tumor is compressible only when it communicates with a large vein, and in such cases its tension increases on coughing and straining.

These cysts which are compressible and fluctuating are easily recognized. Non-compressible fluctuating tumors are to be distinguished from subfacial lipoma, cold abscesses, and soft malignant tumors by aspiration. From aneurysm they differ in their lack of expansile pulsation.

2. **Angiomata.**—Angiomata of the simple type occur in the form of flat vascular *nævi*, and also as subcutaneous tumors in any part of the neck; their diagnosis is self-evident. The cavernous angioma is usually connected only with the veins, but may be in communication with the arteries, in which case the tumor has expansile pulsation. The tumors are flat and irregular, not truly fluctuating, but are compressible, and their tension is increased by straining and coughing.

Their compressibility and pseudofluctuation are sufficient to distinguish them from blood cysts and other forms of cystic growths. Deep-seated cavernoma are difficult to diagnose, because their physical characteristics cannot be elicited.

3. **Aneurysms.**—True and false and arteriovenous aneurysms of the neck are of frequent occurrence and give sufficiently clear signs to render their diagnosis easy in the great majority of cases.

They form expansile, pulsating tumors that are usually compressible, that increase in size when the efferent vessel is compressed, and that afford, on auscultation and palpation respectively, a systolic murmur and a thrill.

As the tumor grows it compresses the neighboring structures; compression of the recurrent laryngeal nerve gives rise to aphonia or hoarseness from paralysis of the abductor muscle of the vocal cord, the cord resting in the cadaveric position; compression of the sympathetic results in dilatation of the pupil on the same side. Pressure upon the trachea and œsophagus occasions tracheal stridor, dyspnœa, and dysphagia. Depending upon the position of the aneurysm there will be manifest inequality and retardation in the temporal or radial pulses.

An abscess, neoplasm or tumor, lying upon a large artery, may pulsate upward and downward, but, with one exception, they never have expansile pulsation. The exception is a very vascular neoplasm, but this grows more rapidly than an aneurysm, does not retard the pulses, and is not compressible to the extent of aneurysms. A thyroid tumor lying upon the carotid may pulsate, but not in expansile fashion (except in case of aneurysmal goitre, and this is truly a variety of aneurysm), and rises and falls with deglutition, which aneurysms never do.

Cavernous angiomata connected with an artery, if they are deeply seated, cannot be differentiated from aneurysms, for they have all the physical characteristics of the latter and are in fact aneurysmal tumors.

It is a matter of surgical importance to determine the point of origin of the aneurysm. Those that spring from the carotid in the neck, or from the subclavian in the posterior triangle, are easily located; those that arise from the innominate, or from the first portion of the subclavian and carotid, must be placed by a study of the radial and temporal pulses. If the temporal pulses are the same, the aneurysm is neither innominate nor carotid; if the radial pulses are the same, the aneurysm is neither innominate nor subclavian. Thus by exclusion the starting point or origin of the aneurysm can be determined.

SOLID TUMORS OF THE NECK.

Some of these have already been considered under glandular tumors, for which the reader is referred to page 130.

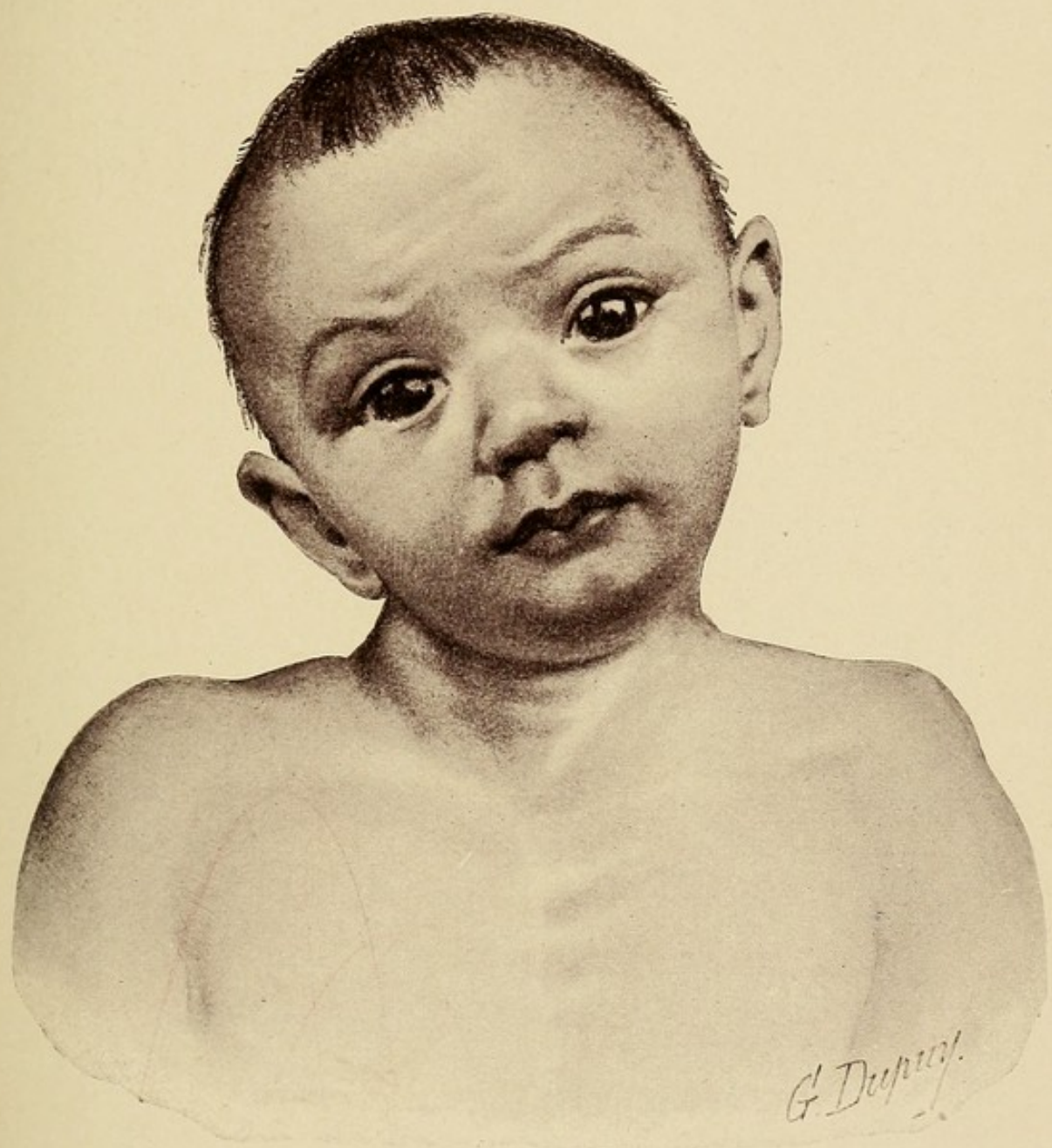
In the newborn, a *hæmatoma of the sternocleidomastoid muscle*, or a *rupture of this muscle*, manifests itself by a soft, doughy tumor, somewhat tender and causing the infant to hold the head laterally flexed and rotated to the opposite side. Such tumors can be confused with nothing else.

Fibromata.—Fibromata may be superficially or deeply situated in any part of the neck. Their hard consistency differentiates them from lipomata, while their slow growth distinguishes them from sarcomata.

Lipomata.—Lipomata are likewise either superficial or deep, the former being especially common on the back of the neck, where they form comparatively large, lobulated, soft, slowly growing tumors.

The deep lipomata of the neck are much less frequently met with. They originate in the subfascial layers, and penetrate in a dendritic fashion between all the structures and into the mediastinum. In its diffuse character it resembles a malignant growth, but is not adherent, and if thoroughly removed in all its parts it does not recur.

PLATE I.

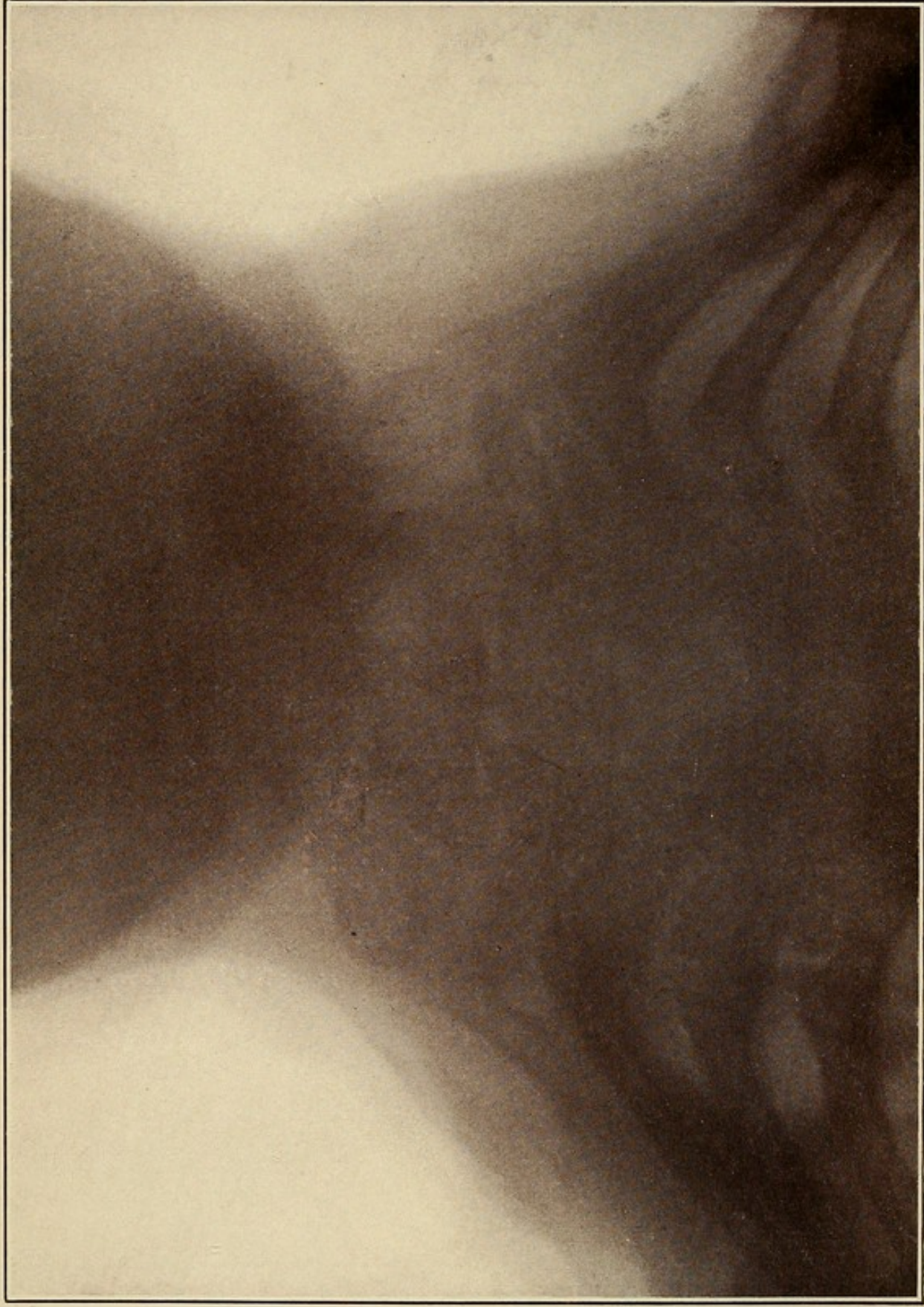


Hæmatoma of the Sternomastoid Muscle of the Right Side in a Newborn Infant. Swelling at the centre of the anterior border of the muscle; contraction of the muscle with torticollis. (Koplik.)





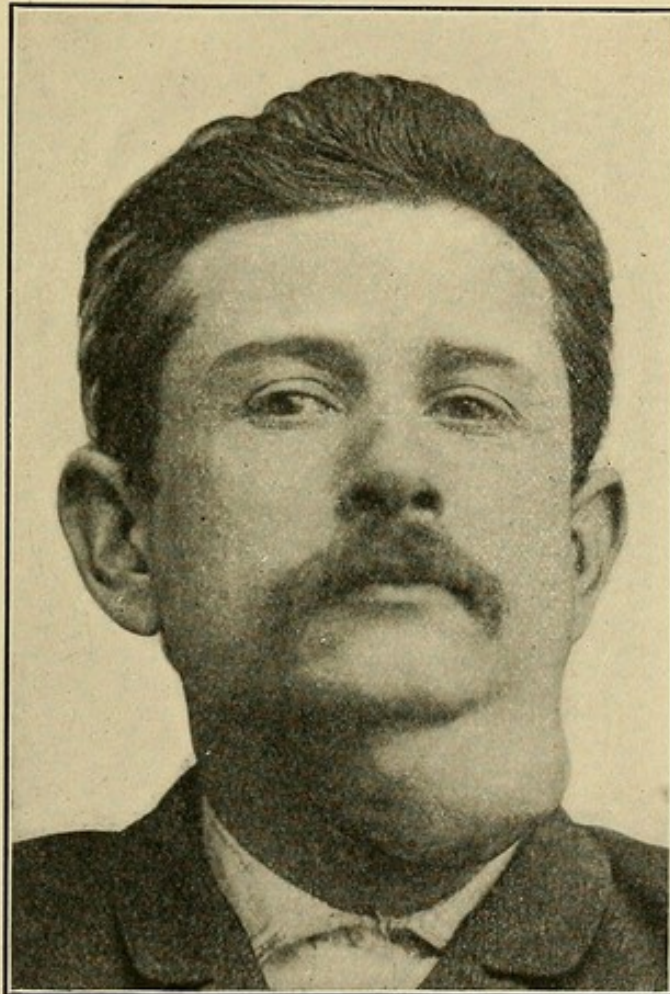
PLATE II.



Bilateral Cervical Rib. The patient, a young girl, complained only of intense pain in the left arm and inability to move the left shoulder-joint.

The diagnosis of these deep lipomata is always difficult; their soft, pseudofluctuating consistency may lead to their being mistaken for cystic or blood tumors, from which they can only be differentiated by aseptic aspiration. From soft, malignant neoplasms they can only be differentiated when they are exposed on the operating table.

FIG. 76

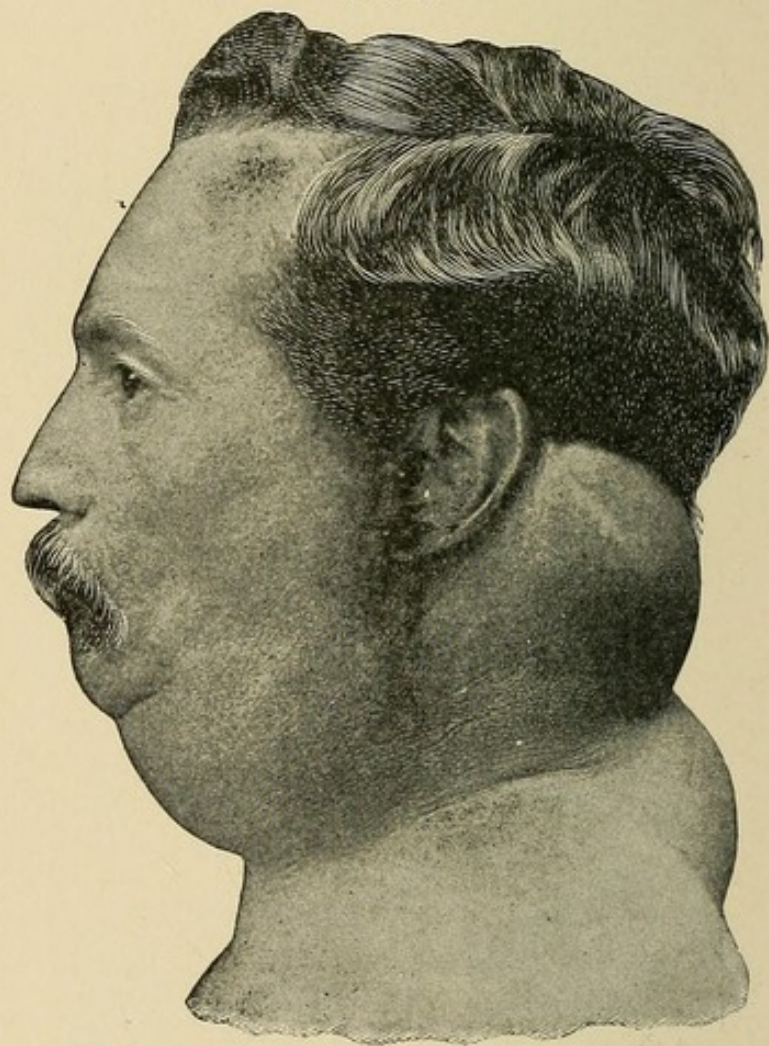


Lipoma of the neck. (Von Bergmann.)

Cervical Rib.—Occasionally a patient will complain of neuralgic pains in the shoulder, arm, and forearm—*i. e.*, in the region of distribution of the brachial plexus—and on examination a hard, fixed tumor is felt in the supraclavicular region, usually on one side, but at times bilaterally. The general health is not impaired; the tumor does not grow, and it is somewhat tender. A cervical rib should be suspected and an x -ray examination ordered.

Malignant Growths.—Of malignant growths, sarcoma of the glands has been considered. Primary carcinoma may originate in the remains of the branchial canal and manifest itself as a hard nodule in the carotid triangle, circumscribed at first, but growing rapidly and infiltrating the neighboring

FIG. 77



Diffuse lipoma of the neck. (Von Bruns.)

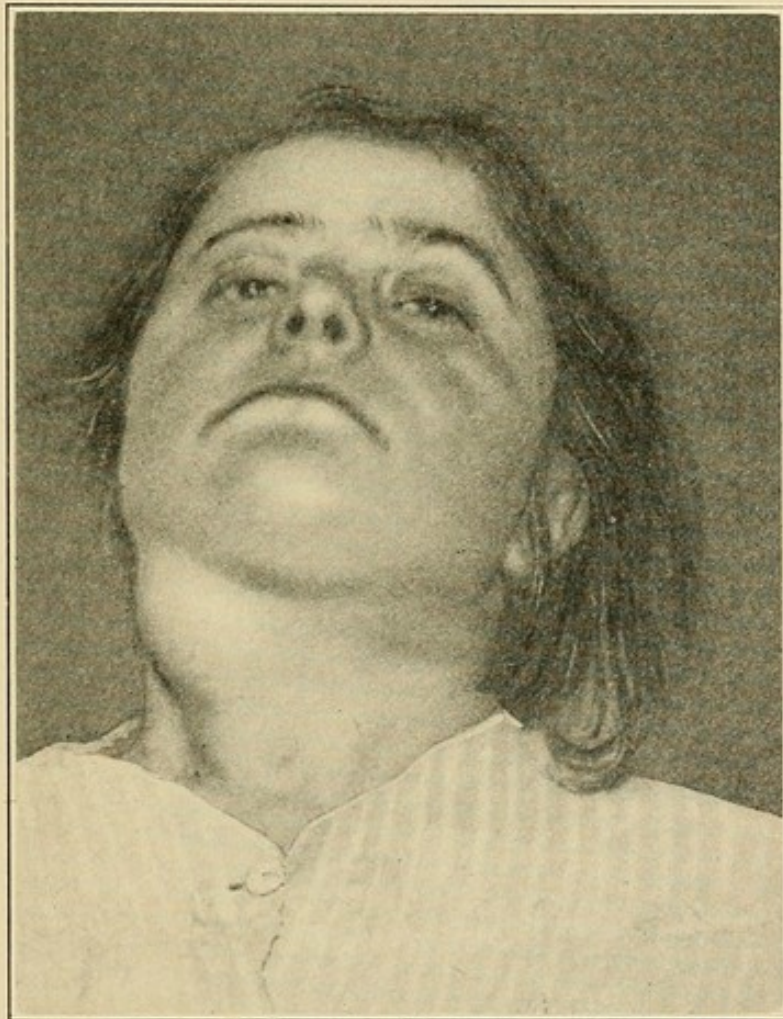
structures, and causing pain which radiates into the ears and occiput. Glandular enlargement and ulceration follow early.

The diagnosis is made from the evident character of malignancy in the neoplasm, and by the absence of a primary carcinoma in the mouth, oropharynx, œsophagus or larynx, to account for the glandular involvement.

THYROID TUMORS.

Tumors of the neck are of thyroid origin if they occupy the region of the thyroid gland, lie behind the sternohyoid and sternothyroid muscles, and move up and down with deglutition. It is to be especially noted, however, that aberrant (accessory) thyroid lobes are sometimes present on

FIG. 78



Thyroid tumor springing from the tip of the upper pole of the thyroid gland. It occupies the same position as would an aberrant thyroid tumor.

the side of the thyroid cartilage, or beneath the outer edge of the sternocleidomastoid muscle, or behind the sternum, and that tumors developing in these aberrant lobes do not have these physical characteristics. The character of such tumors can only be determined by microscopic examination; their

FIG. 79

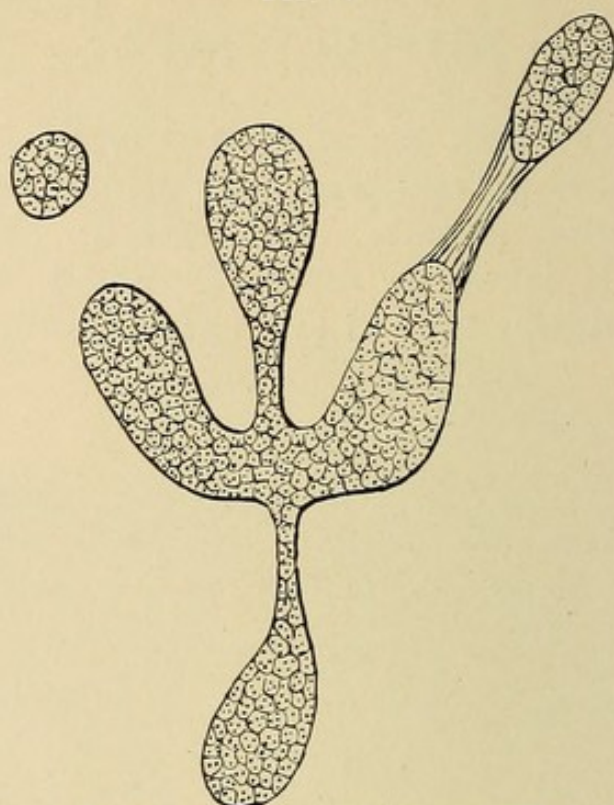
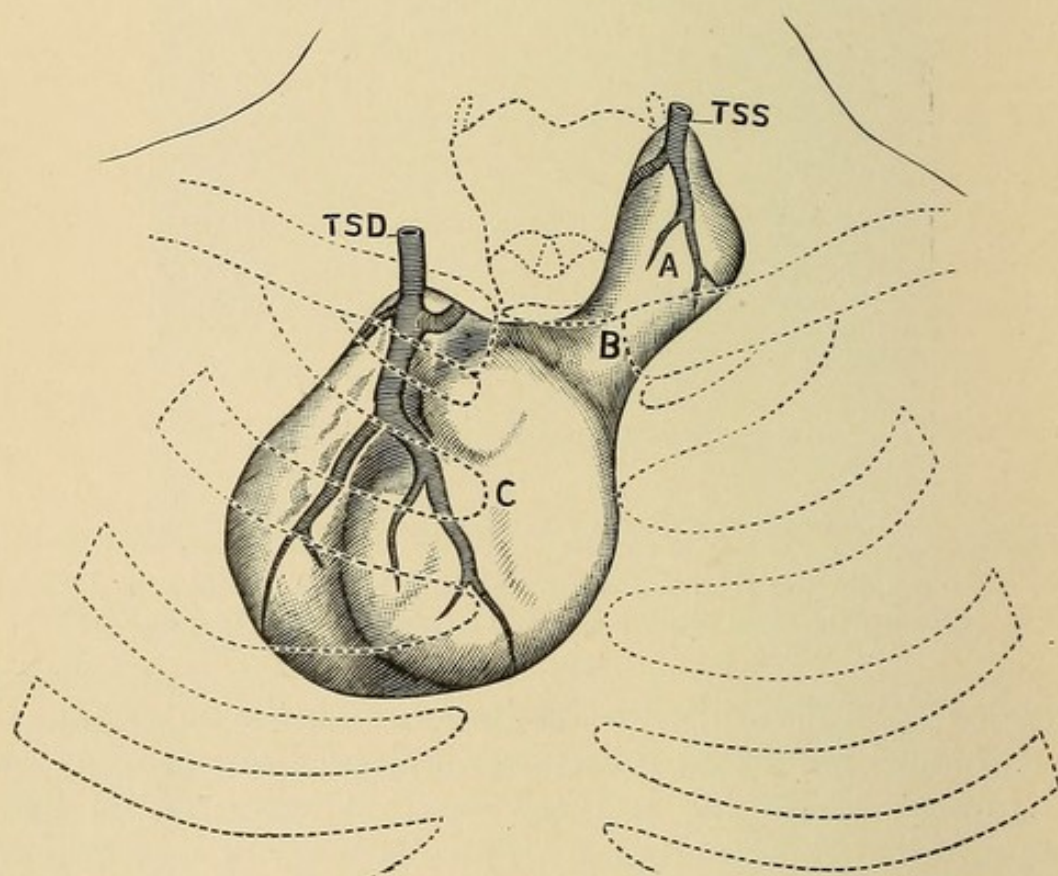


Diagram of accessory thyroids. (Von Bergmann.)

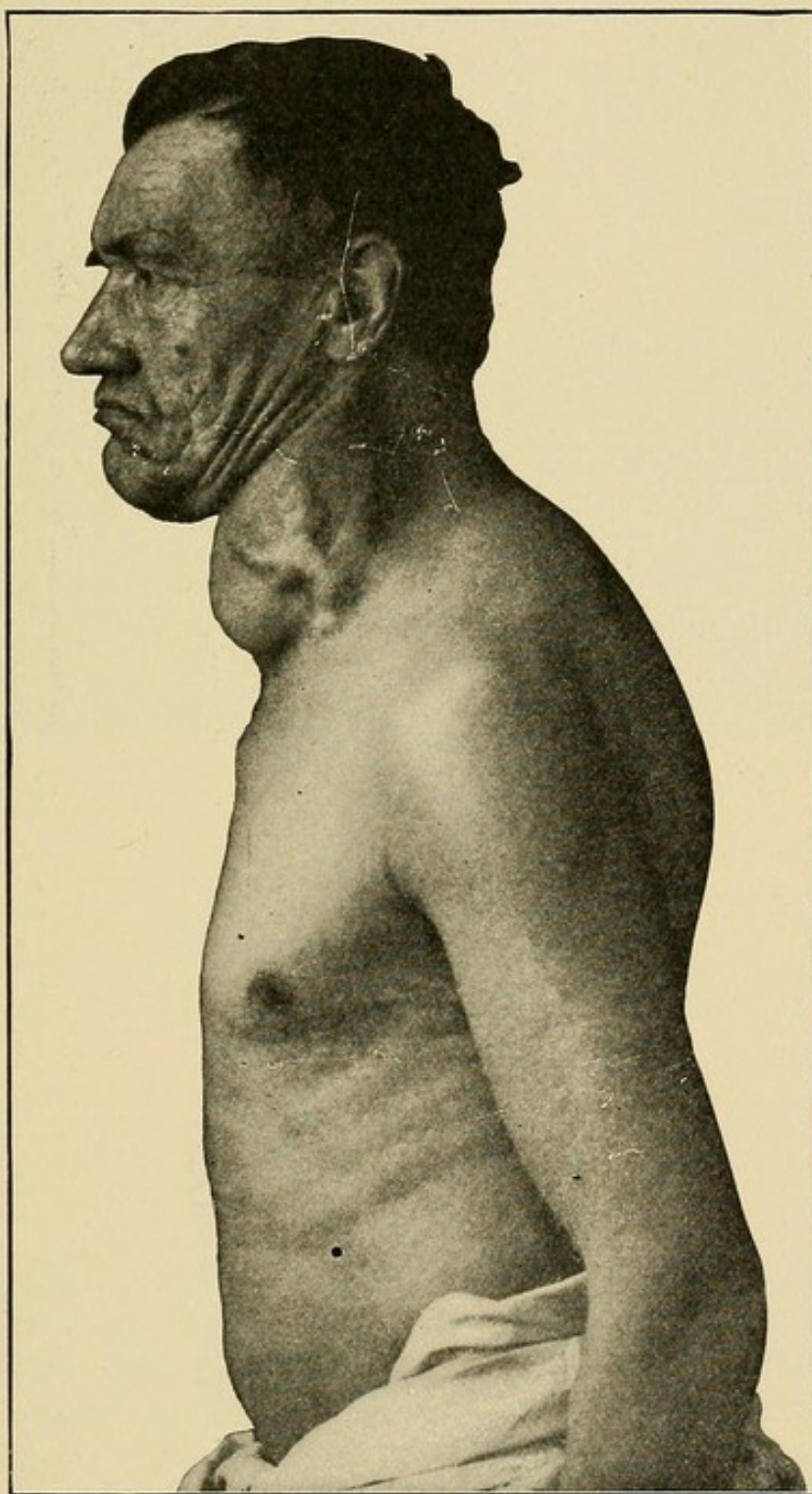
FIG. 80



Intrathoracic thyroid : TSD, right superior thyroid artery ; TSS, left superior thyroid artery. (Von Bergmann.)

thyroid origin can only be suspected, but never positively determined, before their removal.

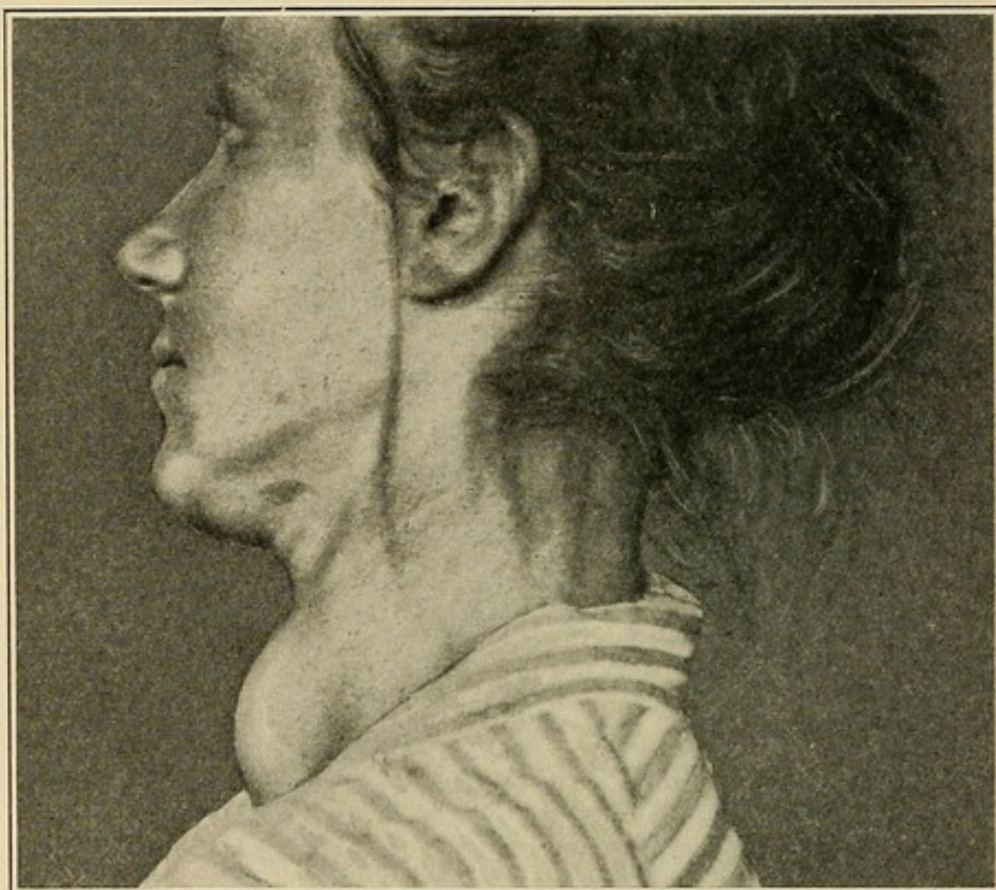
FIG. 81



Parenchymatous stroma. (Von Bergmann.)

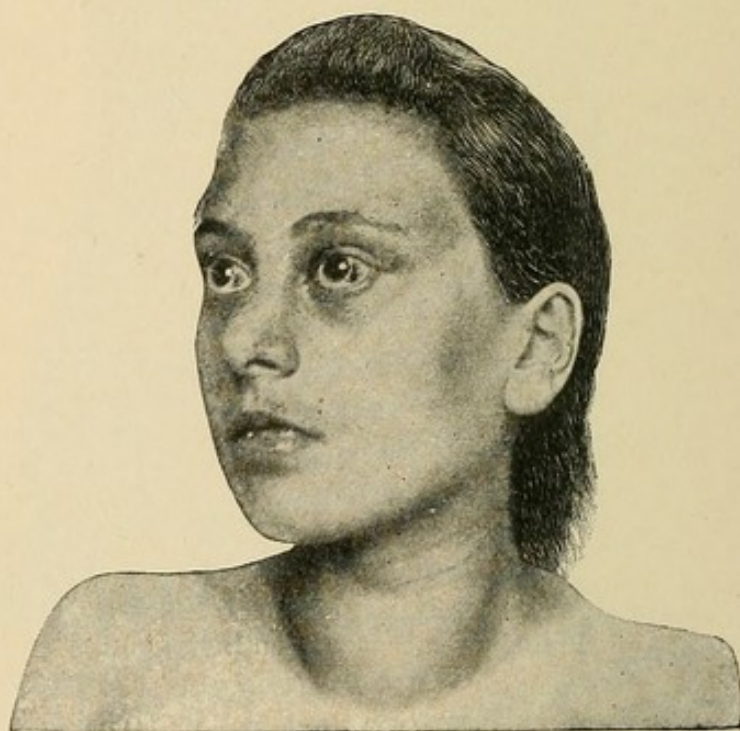
Nature of Thyroid Tumors.—The nature of a thyroid tumor is determined from its rapidity of growth, its outline,

FIG. 82



Cystic goitre. A single, rounded, circumscribed nodule, having distinct fluctuation.

FIG. 83



Struma in Graves' disease. (Von Bergmann.)

whether it involves the whole or part of the gland, its consistency, its mobility, its pulsation, and its encapsulation.

If the whole gland is uniformly and symmetrically enlarged and soft, or contains numerous small nodules, the tumor is a parenchymatous goitre.

If the whole gland is enlarged and soft and vascular, or contains several small nodules, and there are tachycardia, exophthalmos, inco-ordination of associated movements of the eyeball, eyelid and brows, together with nervousness, etc., the tumor is a manifestation of Graves' disease.

FIG. 84



Follicular goitre. (Von Bruns.)

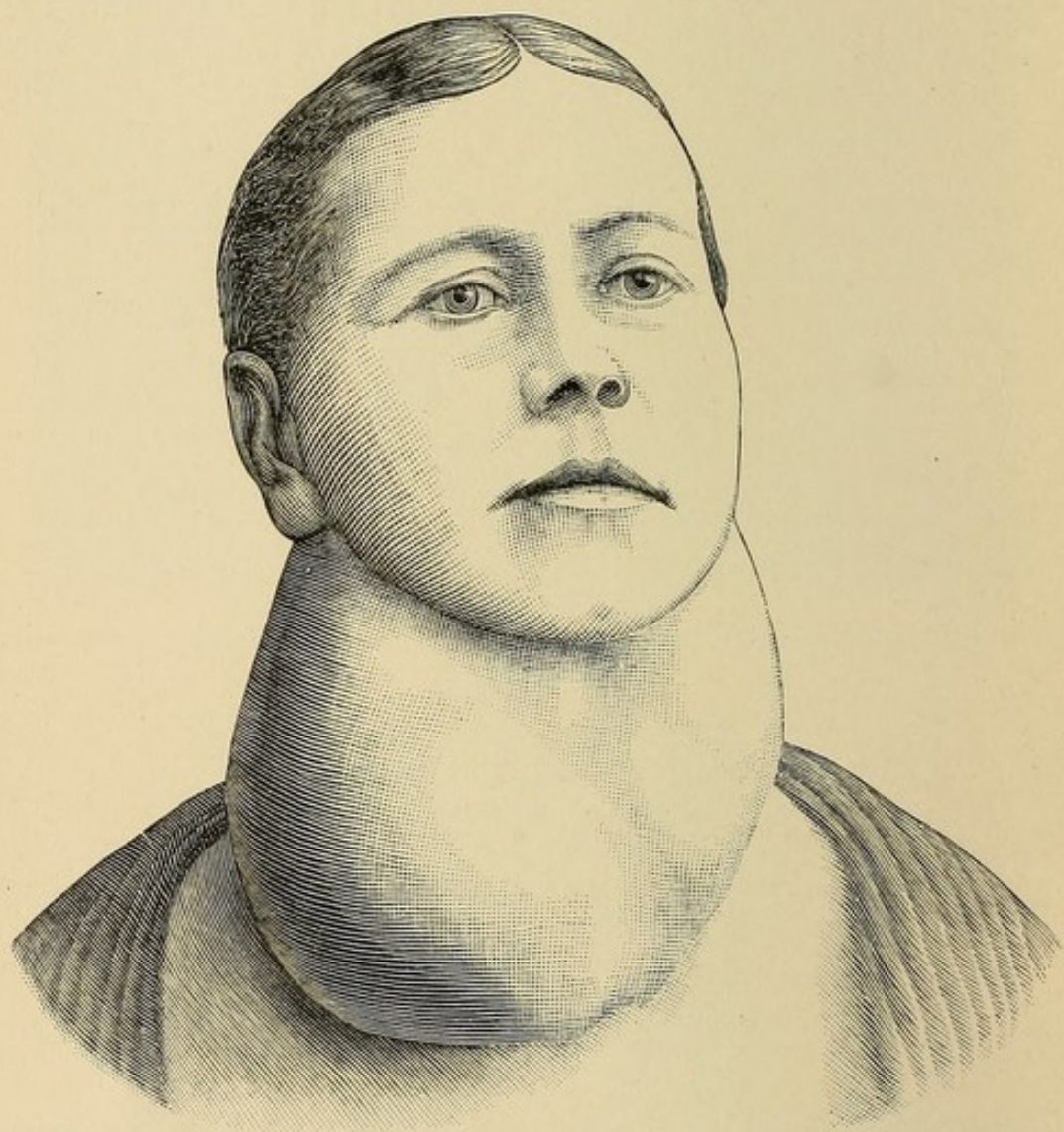
If the gland contains one or more rounded, sharply circumscribed nodules, it is an *adenomatous goitre*. Such adenomatous nodules may undergo degeneration and become *cystic* (fluctuating) or *hyaline* (soft and doughy) or *calcareous* (stony, hard). In the same gland, one type or several types of degenerated nodules may be present.

If the tumor is made up chiefly of enlarged and dilated veins and arteries (it being compressible and having expansile pulsation and affording a systolic bruit) it is a *vascular goitre*

Expansile pulsation is present only when numerous arteries go to make up the tumor.

If the tumor begins in one lobe, is hard and increases in size rapidly and infiltrates the rest of the gland, it is *malignant*, either sarcoma or carcinoma. Pain shooting up to the

FIG. 85



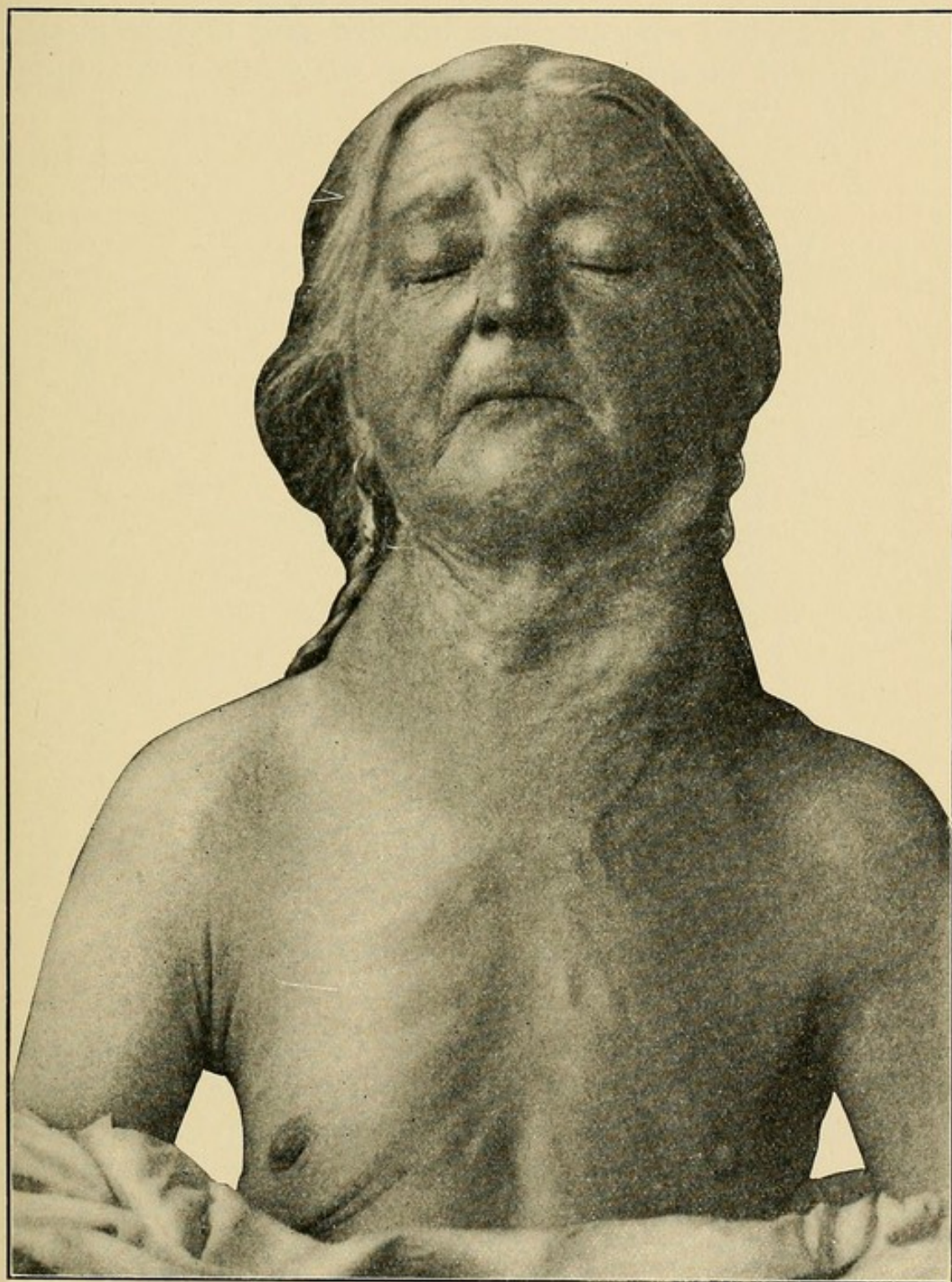
Colloid goitre. (Von Bruns.)

ears and secondary growths in the bones and glands are confirmatory evidences of malignancy.

One should never end his examination of a thyroid tumor until he has looked for evidences of thyroidism, such as tachycardia, tremors, nervousness, etc.; inspected the vocal cords and observed their movements, and palpated the

trachea and large vessels in the neck. For the presence or absence of thyroidism, paralysis of the cords and compression

FIG. 86



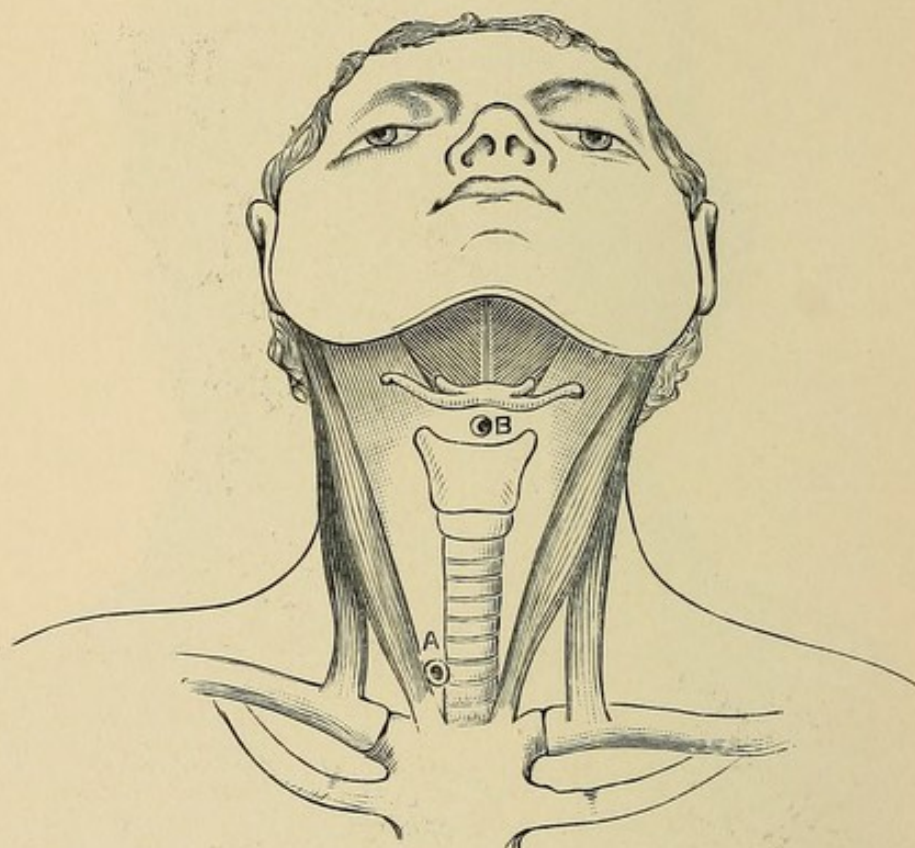
Carcinoma of thyroid gland. (Curschmann.)

of the trachea are most important data for the surgeon to know prior to operative interference.

FISTULÆ AND SINUSES OF THE NECK.

If a branchial cyst becomes infected and perforates externally, or if on account of a wrong diagnosis it is simply

FIG. 87

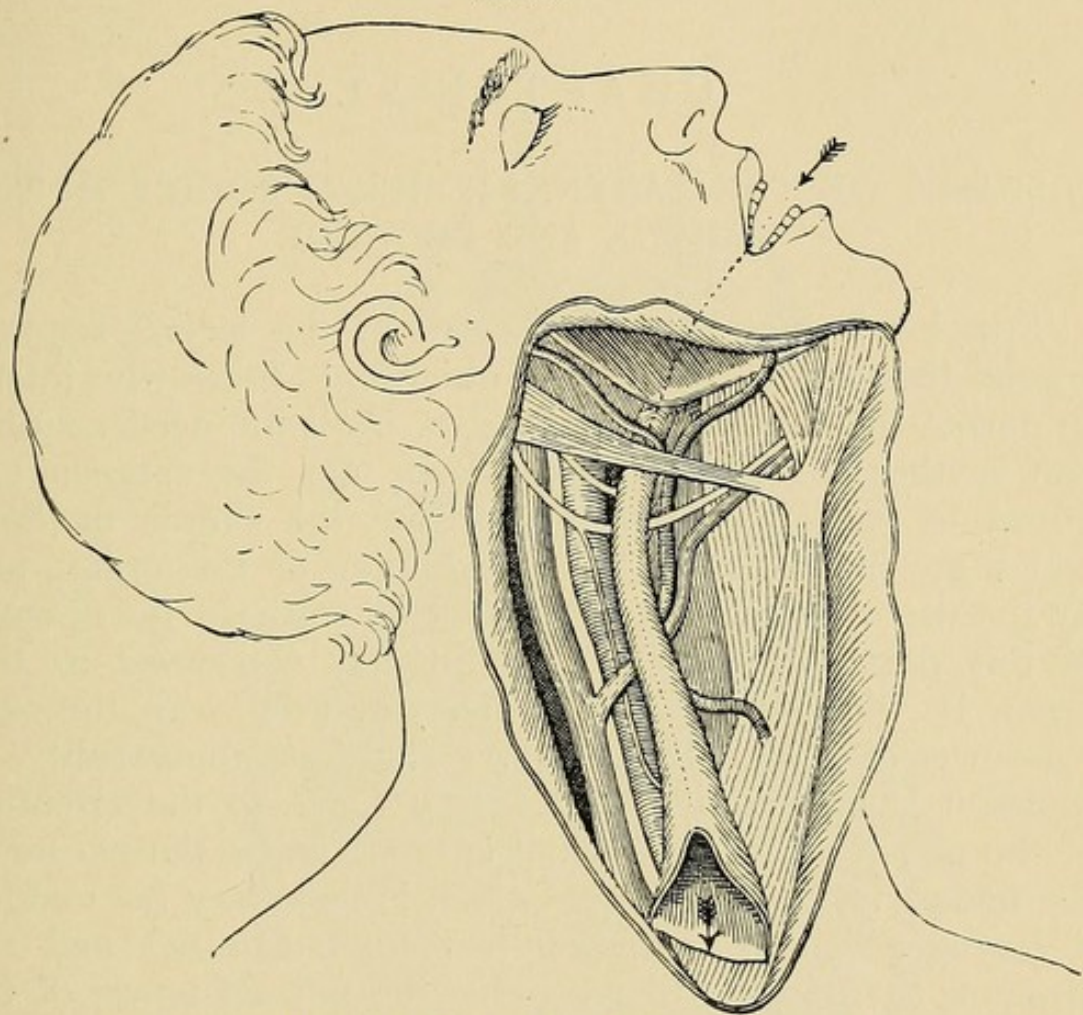


A, lateral sinus ; B, median sinus. (Von Bergmann.)

incised without complete removal of its walls, a fistula will result. If the opening of the fistula is in the median line of the neck, and if the tract thereof extends up to the hyoid bone and root of the tongue, we have to do with a median branchial cyst that originated in a patent thyroglossal duct. If, on the other hand, the sinus opens directly above the sternoclavicular joint and runs upward and outward to open at the pillar of the fauces, we have to do with a lateral branchial cyst that developed from the branchial canal. The external opening of these latter fistulæ is sometimes

situated beneath a little pendulous fold of skin enclosing a bit of cartilage; it may be located in the tragus or helix of the ear.

FIG. 88



Incomplete lateral fistula. (Watson.)

CHAPTER XI.

DISEASES OF THE LARYNX: FOREIGN BODIES IN THE LARYNX AND BRONCHI.

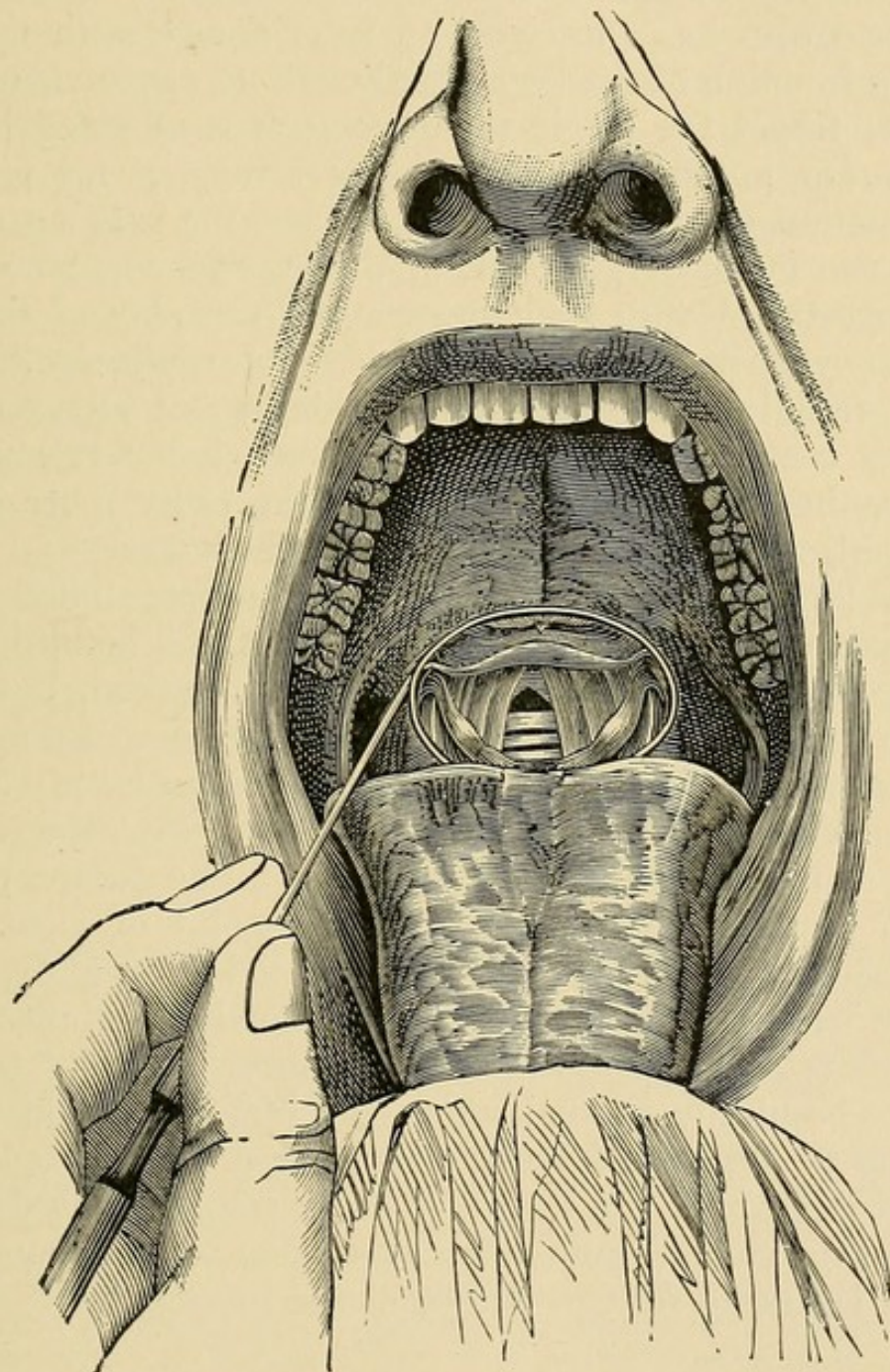
THE local treatment of diseases of the larynx requires special training and technique which only those who intend to devote themselves to this field of medicine need acquire. But neither the general practitioner nor the surgeon can afford to neglect or fail to cultivate the simple methods which are employed in the examination of this organ, and by the use of which alone a correct diagnosis of the diseases of this part can be made. Intimately concerned as this organ is with the every-day life, yes, even with the very existence of the individual, its slightest abnormality of function is at once detected and brought to the attention of the physician, whose duty it is to recognize the nature of the malady. The technique which is necessary for making a laryngoscopic examination is readily acquired and the observer soon learns to recognize the normal aspect of the parts and the significance of the typical abnormalities thereof. Only in the atypical manifestations of diseased processes is difficulty in diagnosis encountered, and in these instances the specially trained eye and the pathologist's examination of sections of the diseased tissue should be early called upon to aid in the interpretation.

Method of Laryngoscopic Examination.—The essentials for a laryngoscopic examination are a good light, a forehead mirror, and a laryngoscopic mirror. The patient should sit upright in front of the examiner, grasp his own tongue with a small napkin and draw it well forward. With the forehead mirror the light which comes from behind the shoulder of the patient is focused upon his uvula and soft palate. The laryngoscopic mirror, which has been just previously gently warmed over the light, is then introduced up to the uvula

without touching the tongue, and while the patient phonates *e* the picture of the larynx is observed.

Visual inspection of the exterior of the organ, together with external and pharyngeal palpation, and in some

FIG. 89



Laryngeal mirror in position, displaying the laryngeal image. (Cohen.)

instances sounding of the laryngeal canal, also afford valuable clinical data upon which to base a diagnosis of injury or disease of this part.

Manifestations of Diseases of the Larynx.—Diseases of the larynx manifest their presence by signs which indicate an interference with its functions, viz., that of a tube carrying air to the lungs, and that of an organ of phonation; alterations in the normal external and internal appearances and feel of the organs are further evidences of its disease.

No one who has witnessed an interference with the air-carrying function of the larynx sufficient to cause air hunger can ever forget the tragic picture which is afforded by the unfortunate individual. The early struggles for air, the forced action of the respiratory muscles, the tugging at the throat, the marked retraction of the supraclavicular spaces and epigastrium with each inspiration, the play of the alæ nasi, the cyanosis, and the loud, stridulous respiration. More or less rapidly, depending on the degree of stenosis, the struggles cease, the respiration becomes shallower and less audible, the cyanosis more marked, the pulse more feeble and rapid, until death puts an end to the scene.

Such laryngeal dyspnœa is to be differentiated from bronchial, pulmonary, or cardiac dyspnœa. The air hunger in these latter conditions may be just as marked, but there is no stridulous respiration, there is no huskiness of the voice, no laryngeal cough, and physical examination reveals in the bronchi or air cells of the lung or in the heart evidences of disease which account for the dyspnœa. The differentiation is most important from a therapeutic point of view. No patient should be permitted to succumb to laryngeal dyspnœa if a physician is in attendance, no matter how primitive the instruments at his disposal for the performance of tracheotomy. Naturally in such cases the high operation is the only one to perform, for this can be done rapidly even by inexpert surgeons. On the other hand, tracheotomy does no good for bronchial, pulmonic, or cardiac dyspnœa, except when it is due to foreign bodies in the bronchi.¹

¹ I was recently urgently summoned to the hospital to perform tracheotomy upon a patient who was suffering with severe dyspnœa, owing, I was told, to compression of the trachea by a cervical neoplasm. The dyspnœa being extreme, the house surgeon proceeded to operate before my arrival. He intended to make a *low tracheotomy*. I found him considerably bothered by a profuse venous hemorrhage from the soft parts overlying the trachea, and as he had difficulty in checking this, I rapidly prepared my hands and assisted him. After stopping the

ACUTE LARYNGEAL DISEASES.

In the acute laryngeal diseases the dyspnœa and stridor are the most striking symptoms, but no less important for diagnosis are the huskiness of the voice, aphonia, and characteristic metallic laryngeal cough. In the urgent cases of dyspnœa no time is afforded for laryngoscopic or even visual inspection of the parts in these cases. The diagnosis will have to be made from a rapidly taken anamnesis and a hurried physical examination. In the less urgent acute cases, and in the chronic laryngeal maladies, which, by the way, give the first evidences of their presence by changes in the character of the voice, and by pain, and only later on by dyspnœa, there is ample time for laryngoscopic and general physical examination.

Causes of Acute Laryngeal Stenosis.—The causes of acute laryngeal stenosis are: *Acute laryngitis*, *diphtheritic laryngitis* (*croup*), *laryngismus stridulus* (*spasm of the larynx*), *œdema of the structures around the glottis*, *foreign bodies in the larynx*, *wounds and fractures of the larynx*.

Acute Laryngitis.—Laryngeal stenosis due to simple inflammation is rarely met with and then it is chiefly dependent upon an associated œdema of the structures around the glottis.

bleeding, which came from the median jugular vein, this having been incised longitudinally, I palpated the cervical tumor and the trachea, and to my surprise found that the latter was neither displaced nor compressed by the neoplasm. I rapidly asked for the previous history of the patient and was told that she had for some time been treated by her family physician for a mediastinal tumor; upon percussion I found extensive dulness over the entire anterior mediastinum and over the upper lobe of the left lung. The cervical tumor represented a bunch of enlarged glands; it played no part in the causation of the dyspnœa. This latter was due to compression of the bronchi at the root of the lungs by the mediastinal tumor. I at once informed the house surgeon that tracheotomy would not relieve the patient unless it was possible to pass a stiff drainage tube through the tracheal opening down past the site of obstruction. Though this was successfully accomplished, the patient nevertheless succumbed within a few hours. I quote this history for three reasons: first, to show the utter impossibility of relieving dyspnœa from bronchial obstruction by a tracheotomy, unless such obstruction be due to a foreign body; second, to illustrate the great importance of a careful examination and a good history in establishing a diagnosis; and third, to demonstrate that in great urgency one should never try to do a low tracheotomy, for its performance requires careful and accurate dissection, which is necessarily time-consuming, whereas a high tracheotomy can be rapidly and safely done with a few strokes of the knife.

The mild severity of the constitutional symptoms, the absence of patches of false membrane in the nasopharynx or on the tonsils, and of enlarged cervical glands, will serve to differentiate this form of laryngeal obstruction from that due to membranous croup.

Diphtheritic Croup.—Laryngeal stenosis due to membranous croup is met with chiefly in young children, whose laryngeal aperture is small and consequently more easily occluded than that of adults. Except in the cases of primary laryngeal diphtheria, patches of false membrane are also present in the nasopharynx and on the tonsils; the cervical nodes are enlarged; the temperature is high and the patient is usually sick a number of days before the membrane accumulates sufficiently to occlude the glottis. It is to be remembered that in some instances severe laryngeal dyspnoea from laryngeal croup may be met with in children who have been well up to the onset of the attack. In these cases it is not the membrane that occludes the larynx, but the catarrhal swelling. Such dyspnoea is rarely severe enough to urgently require tracheotomy or intubation. It is relieved by an emetic, but as the false membrane accumulates it again becomes severe. In these cases the initial attack may be taken for a seizure of spasmodic croup.

Laryngismus Stridulus.—Laryngismus stridulus occurs in rickety children; the dyspnoea it occasions is marked, but there is neither laryngeal cough nor hoarseness, both of which are so characteristic of laryngeal croup.

Spasmodic Croup.—Spasmodic croup of young children comes on, as a rule, in the middle of the night. The previously healthy child wakes up out of a sound sleep with a croupy cough, some hoarseness and dyspnoea, which for a time seems serious. The attack passes off abruptly, the child falling asleep and the next morning on awakening the patient feels perfectly well. Such a condition is differentiated from diphtheritic croup by the previous healthy condition of the patient, the absence of membrane or swelling in the nasopharynx or tonsils, the absence of temperature elevations and the history of previous attacks of a similar spasm.

Œdema of Structures of Glottis.—Œdema of the structures which form the glottis occasions a rapidly increasing dyspnoea

usually without *stridor*, but the voice becomes husky and disappears. Laryngoscopic examination shows the ary-epiglottic folds and epiglottis enormously swollen and distended into fluid sacs. Such an œdema is met with as a sequence to acute laryngitis, diphtheritic croup, severe paralaryngeal inflammations, as angina ludovici, chronic laryngeal ulcerations from tuberculosis or syphilis; the acute infectious diseases, as scarlet fever, typhus and typhoid, and to Bright's disease. A carefully taken previous history and a careful physical examination of the patient, his urine, etc., will usually explain a sudden rapidly increasing dyspnœa and indicate the proper therapeutic procedure that is to be employed.

Foreign Bodies in the Larynx and Bronchi.—Foreign bodies in the air passages may lodge in the larynx above the vocal cords, or in the rima glottidis; or they may bob up and down in the trachea, closing the air tubes and glottis like a ball valve; or they may become impacted in the bronchi. Foreign bodies may further lodge in the pharynx just opposite to the superior aperture of the larynx, and either compress or entirely occlude the latter.

Foreign bodies lodging in the larynx occasion inspiratory or expiratory dyspnœa or both, rarely stridor or laryngeal cough, and hoarseness only if the body lies upon or between the vocal cords. The degree of dyspnœa depends entirely on the size and the site of lodgement of the foreign body. The diagnosis is usually made from the history. Immediately on being summoned to such a case, the physician should carefully palpate and search in the pharynx or superior laryngeal aperture to ascertain whether a foreign body is lodged therein. If none is found and the dyspnœa is great, immediate tracheotomy should be made.

Foreign bodies in the trachea occasion attacks of severe spasmodic dyspnœa which recur from time to time as the result of a cough; there is also a feeling of something moving up and down in the trachea. By auscultation over the trachea the foreign body may be heard moving up and down the trachea with respiration. The spasmodic attacks of dyspnœa are excited by the impact of the foreign body against the vocal cords. Where the case permits of delay, an x-ray picture will positively reveal the presence of the foreign

body; in urgent cases immediate tracheotomy relieves the dyspnoea and movable bodies will very likely be expelled through the tracheal opening on forced expiration.

Foreign bodies in the bronchi are usually impacted in one or the other of the main or primary bronchial tubes.

FIG. 90



Bean in right bronchus. (Fürbinger.)

When this is the case the corresponding side of the chest does not move with respiration, and there is an absence of vesicular murmur over the corresponding lung, or a loud sibilant rale is to be heard on this side of the thorax, with greatest intensity at the root of the lungs. There may be

no dyspnoea while the patient is quiet. An absence of normal breathing sounds, or a loud sibilant rale over a part of the lung, points to the lodgement of the foreign body in one of the smaller bronchi.

It is to be noted that small foreign bodies lodging in the smaller bronchi may occasion few if any primary symptoms, and only after the formation of a pulmonary abscess or bronchiectasis or a localized consolidation is attention drawn to the affected part. The anamnesis may at once reveal the cause, for the pathological changes and the presence and site of the foreign body may sometimes be accurately determined by the Roentgen ray.

Wounds and Fractures of the Larynx.—In these days of intubation, wounds and fractures of the larynx are comparatively frequent from inexpert use of the O'Dwyer apparatus. The diagnosis is readily made. The injury may be inflicted during intubation or extubation, more frequently the latter, and is at once followed by an increased or renewed dyspnoea, hemorrhage, and sometimes by emphysema of the neck.

CHRONIC LARYNGEAL DISEASES.

Changes of the voice and pain are the first indications of the chronic diseases of the larynx; the pain is referred to the diseased part, radiates to the ears, and is increased on swallowing. Impairment in breathing is a later symptom, though it is to be remembered that sudden dyspnoea from oedema of the structures around the glottis is a possible and ever-threatening complication of these chronic laryngeal affections.

Changes in the voice, with or without pain, or impaired respiration should lead the attendant to make a laryngoscopic examination.

The pictures to be seen in the most common of the chronic diseases of this organ are as follows:

Chronic Laryngitis.—In chronic laryngitis the mucous membrane, especially the posterior part, is red, swollen and thickened; sometimes there are superficial ulceration and desquamation; there is also an impaired motility of the vocal

cords from swelling and thickening of the mucous membrane.

Tuberculous Laryngitis.—In the preulcerative stage, the mucous membrane is swollen and rather nodular, but not discolored; if the membrane is reddened it is unevenly so. The ulcerations usually commence posteriorly and extend thence around the larynx; they are ragged, worm-eaten and sloughy, are surrounded by pale, swollen mucous membrane, and show no tendency to heal; hence there are no scars. In the ulcerative stage the pain is marked, especially in swallowing.

The general condition and history of the patient are of great importance in the diagnosis. The existence of other tuberculous lesions, especially in the lungs, should be carefully sought for, and the sputum or scrapings from the ulcer should be examined for tubercle bacilli.

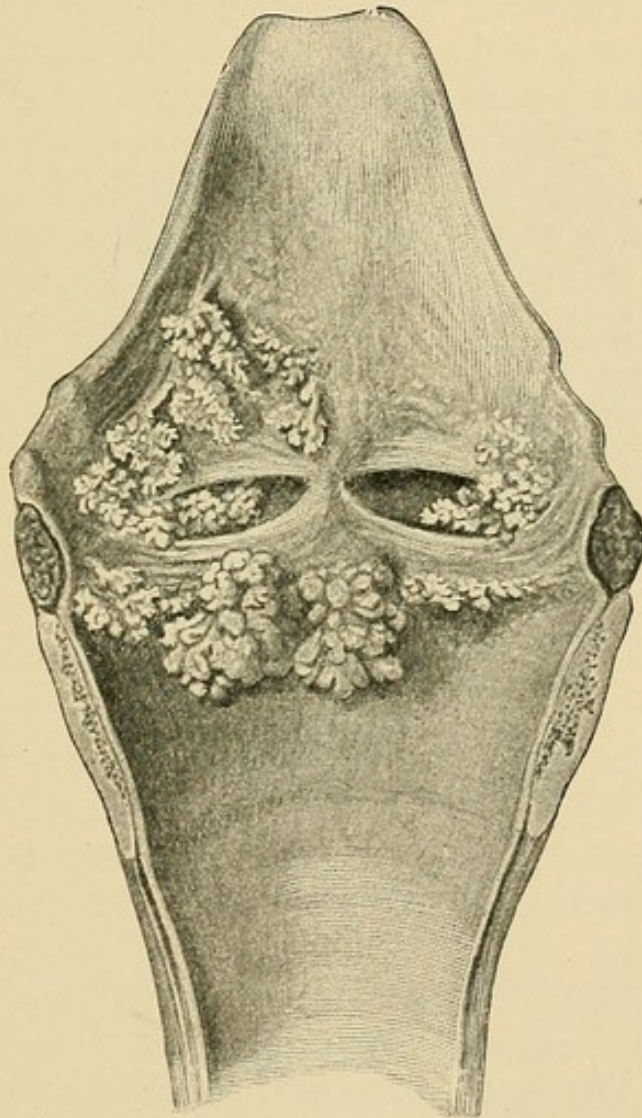
Syphilitic Laryngitis.—In syphilitic laryngitis the picture as seen with the mirror varies. In the secondary stages of the disease, the whole or only parts of the mucous membrane may show a uniform or mottled congestion, or superficial, shallow, irregularly rounded ulcers surrounded by an inflammatory areola, the floor of which is covered by a yellowish material. These ulcers heal and new ones form, the healed ulcers being represented by stellate scars. In rare instances mucous patches are seen on the epiglottis, arytenoids, and vocal cords. They have a regular rounded outline, slightly elevated margins, and are surrounded by an inflammatory areola, their base being covered by whitish exudate.

In the tertiary stages gummata are met with. They are located usually upon the epiglottis or arytenoids, are single or multiple, and form smooth, rounded nodules, varying in size from a pin's head to a small marble. They appear suddenly, grow slowly, and undergo softening and ulceration. The defect thus created is deep, has ragged, sharply defined edges, surrounded by a deeply inflamed, elevated areola, and the base is covered by a foul greenish or yellowish necrotic tissue. If healing occurs, stenosis and deformities result from the contraction of the irregular scars. In establishing the diagnosis, it is of considerable aid to elicit a history of syphilis, and to find other evidences of syphilis in the bones, on the skin and mucous membrane, etc.

Neoplasms.—With neoplasms the local manifestations depend upon the character and stage of growth of the tumor.

Papillomata are usually multiple, prominent, grayish-white growths, with irregular surface, but not ulcerated, and have little tendency to bleed. They may be located above, upon, or below the vocal cords. If the growth is above the cords

FIG. 91



Multiple papillomata of larynx. (Von Bergmann.)

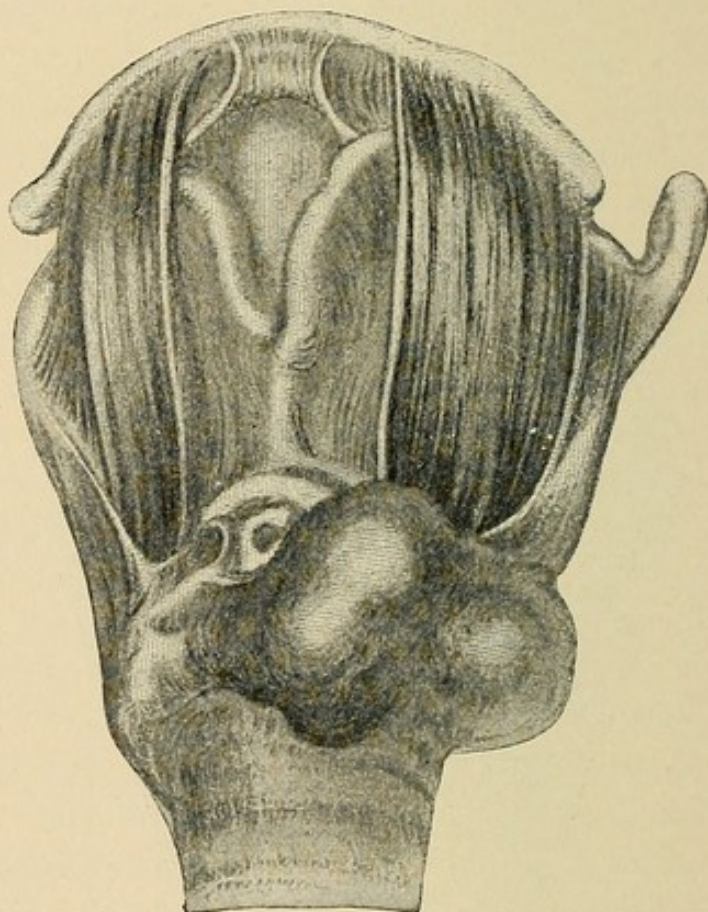
the dyspnœa will be chiefly on inspiration; if below, chiefly on expiration.

Fibromata are usually smooth and vascular single tumors; they sometimes ulcerate and grow very slowly.

Sarcoma and *carcinoma* form rapidly growing tumors which infiltrate the neighboring parts, undergo early ulcer-

ation, and markedly interfere with respiration and deglutition. The pain in carcinoma is very marked and radiates to the ears; it is less severe in sarcoma. In carcinoma more frequently than in sarcoma the glands along the carotid are enlarged and hard and fixed. In all doubtful tumors, a specimen should be excised and submitted to a competent pathologist for examination.

FIG. 92



Perichondritis cricoideus with external abscesses. (Türk.)

The other laryngeal neoplasms, such as lipoma, polypi, chondroma, dermoid and retention cysts, are more rarely encountered, and their diagnosis is readily made.

Chronic Pachydermatous Laryngitis.—A form of chronic pachydermatous laryngitis is quite frequently met with. The laryngeal mucous membrane is thickened, roughened, and whitish in color; ulceration is rare. This condition lasts a long time, and in many instances finally degenerates into epithelioma.

Chondritis. — Syphilis, tuberculosis, malignant disease, typhoid fever and rheumatism frequently invade the *cartilages* of the larynx, either primarily or secondarily to an intralaryngeal lesion, and give rise to chondritis and perichondritis, the latter being sometimes of a purulent character. The diagnosis is made from the previous history and the evidences afforded by other foci of the disease. In syphilis there is more likely to be an external swelling than in tuberculosis, and the syphilitic affections show a tendency to heal. The ulcerations that result from tuberculosis are ragged and worm-eaten and are surrounded by protuberant granulations. In typhoid fever the mucous membrane of the larynx is red, boggy, and œdematous, and if the perichondrium is invaded a smooth, tender swelling becomes visible either externally or internally. There may be associated with these lesions considerable pain and dysphagia, and the patients may suffer from attacks of severe dyspnœa. The presence of *gouty nodes* in the cartilaginous structures of other parts, especially of the ear and of the finger-joints, indicates the character of firm nodes in the laryngeal cartilages.

CICATRICIAL STRICTURES.

Cicatricial strictures of the larynx and trachea occasion permanent hoarseness and dyspnœa, the severity of which depends on the degree of stenosis which is present. Such cicatrices result from injuries and wounds of the larynx or trachea inflicted with suicidal intent, or from rough and inexpert intralaryngeal or intratracheal manipulations or instrumentation, from pressure ulcers caused by the O'Dwyer tubes, and especially by the vicious modifications of this instrument, from syphilitic ulcerations, and from cicatrization of ulcers due to the inhalation or application of irritating chemicals. With the mirror the adventitious tissue is readily seen and the diagnosis is easily established. The causation of the stricture, which has important bearing upon its treatment, must be ascertained from the history and from the physical examination of the rest of the body.

IMPAIRED RESPIRATION.

Impaired respiration and even severe dyspnœa are not infrequently due to compression of the larynx, trachea, and bronchi by neighboring tumors or to encroachment upon the superior laryngeal aperture by tumors, foreign bodies or abscesses in the pharynx. The diagnosis of such conditions is, as a rule, readily made by palpation, oropharyngeal inspection, and laryngoscopic examination. Especially significant and of serious import, on account of the danger of urgent dyspnœa, are those cases in which the neoplasm has eroded and thus caused a pathological fracture of the laryngeal cartilages or tracheal rings. The sudden collapse of the laryngeal or tracheal walls at the points of fracture occasions such sudden severe dyspnœa that death results unless relief by tracheotomy is immediately afforded.

PART III.

INJURIES AND DISEASES OF THE THORAX.

CHAPTER XII.

INJURIES OF THE THORAX.

THE chief significance of injuries of the thorax lies in the lesions the traumatism inflicts upon the contained viscera. Open penetrating wounds differ from subcutaneous ones in their greater likelihood to visceral complications, in their almost invariably resulting in pneumothorax, and in their liability to infection. However, it must not be forgotten that pneumothorax may likewise follow subcutaneous rupture of the lung, and, further, that subcutaneous wounds may become infected either through their communication with the air tubes or through the medium of the blood. It is natural to expect a visceral complication to an open penetrating wound, but it must also be remembered that serious visceral lesions may attend subcutaneous injuries, even when the external manifestations are very slight. The main point to keep in view is that every patient who has sustained a thoracic injury should be carefully examined for signs of complicating visceral lesions, as well as for the more striking and manifest external ones.

Commotio Thoracis.—Thoracic injuries usually give rise to shock, which is proportionate in degree to the severity of the injury. In certain rare instances, however, comparatively slight injuries have caused alarming and even fatal collapse. In such cases the trauma has usually been inflicted over the

anterior sternal region. The individual at once falls over, becomes pale and cold, with small, thready, slow, irregular pulse and superficial, rapid, and irregular respirations. Most of the patients recover rapidly, some more slowly, and a few succumb. The symptoms have been ascribed to an irritation of the vagus and cardiodepressor nerves, and the condition has been termed *commotio thoracis*. Such a diagnosis is only to be made after a careful examination has excluded an injury of the visceral organs. Severe or continued hemorrhage from wounds of the heart or vessels or lungs naturally increases and prolongs the shock resulting from the immediate injury, and where reaction therefrom is delayed the suspicion of a visceral complication should always be entertained.

INJURIES OF THE THORACIC WALL.

Injuries inflicted upon the thoracic wall may occasion thereon an open wound, or a subcutaneous one, with fracture of the ribs or sternum, severing or laceration of an intercostal or the internal mammary artery, or a hæmatoma.

Hæmatomata form soft, doughy, tender swellings that gradually shrink and become harder and finally disappear. They are differentiated from abscesses by the absence of local heat, temperature elevations, and true fluctuation. Abscesses grow larger, whereas hæmatomata become smaller and harder. Exploratory aspiration will positively decide the diagnosis.

Fracture of the ribs is evidenced by distinct local pain on pressure, pain at the injured spot on deep inspiration or when pressure is made over the anterior or posterior ends of the rib, and pleuritic friction sounds. Abnormal mobility, displacement, and crepitus are sometimes present.

Fractures of the sternum are to be recognized by abnormal mobility, crepitus, and displacement of the fragments, and by the pain which is provoked over the site of the injury when one end of the sternum is pressed upon or when the head is forcibly extended.

Rupture of an intercostal artery or the internal mammary artery is followed by the formation of a false aneurysm, which gives the usual signs of this condition, viz., a compressible tumor, over which there is a systolic murmur and a thrill.

Subcutaneous emphysema may form around non-penetrating wounds; it is always circumscribed, does not spread, and tends to disappear rapidly.

INJURIES OF THE LUNGS AND PLEURA.

Wounds of the lungs and pleuræ are recognized by the following conditions and symptoms:

1. *Pneumothorax*. This causes respiratory immobility of the affected side of the chest; rapid, irregular, labored respirations; slow, irregular, small and thready pulse; and tympanitic resonance over the entire affected side of the thorax. Such pneumothorax is less marked in penetrating wounds which involve the pleura alone than in those which are complicated by a wound of the lung.

2. *Hæmopneumothorax*. This occasions the same disturbances of the pulse and respiration as does simple pneumothorax, but gives different physical signs, viz., tympanitic resonance over the apex of the chest, with flatness at the base, and succussion.

3. *Subcutaneous emphysema*. This is usually limited to the cellular tissues at the site at which the injury was inflicted, but sometimes extends to the cellular tissues of a large part of the body.

4. *Hæmoptysis*, of varying profuseness and duration.

5. *Irritable cough*, and

6. *Dyspnæa*. This is usually dependent upon the hæmopneumothorax, in which case it appears immediately upon reception of the injury; sometimes it is due to an accumulating exudate within the thorax with resultant compression of the lung, and in this instance it is first manifested several days after the injury.

Pneumothorax or hæmopneumothorax may result from a penetrating wound involving the pleura alone, or from a

penetrating wound involving lung and pleura, or from a rupture of the lung.

Subcutaneous emphysema may be present in simple non-penetrating wounds of the chest, but it is usually due to a complicating wound or to a rupture of the pleura and lung. With non-penetrating wounds it is always of very limited extent, is confined chiefly to the margins of the wound, and therefore cannot be confounded with the more extensive emphysema that goes with pleural or lung injuries.

If, with a penetrating wound, there is present only pneumothorax, or only a very slight hæmopneumothorax, and no hæmoptysis, and if the pneumothorax rapidly disappears upon closure of the outer wound by an abundant gauze dressing, the lung has in all probability not been injured. But if the pneumothorax constantly increases, as evidenced by increasing dyspnœa and higher-pitched tympanitic note over the chest, or if there is much blood in the pleural cavity, and if there is hæmoptysis, the likelihood of pulmonary injury being present is very strong. It is to be noted that very considerable hæmopneumothorax may result from wounds of the pleura alone, the blood coming from an injured intercostal or internal mammary vessel; and, further, that although hæmoptysis usually follows a pulmonary injury, yet small wounds of the lung which are not in communication with a bronchus, may not be attended by this symptom; and again, that should the pleural layers have been adherent at the site over which the injury was inflicted, neither pneumothorax nor hæmopneumothorax would result.

INJURIES OF HEART AND PERICARDIUM.

Wounds of the heart and pericardium are in the vast majority of instances due to penetrating injuries, the site of which over the cardiac area offers a hint to the possibility of involvement of these viscera. Though wounds of the pericardium alone are of infrequent occurrence, clinical evidences have nevertheless shown that this structure may be injured independently of the heart. As the left pleura and lung almost cover that portion of the heart and pericardium which

is not protected by the sternum, penetrating wounds of these latter viscera are usually complicated by wounds of the former.

Wounds of the heart and pericardium are attended by one invariable symptom, viz., an effusion of blood into the pericardial sac. With wounds of the pericardium alone, and with non-penetrating wounds of the cardiac muscle, the hæmopericardium may be very slight, but with penetrating wounds of the latter, and sometimes with wounds of the pericardium alone, it is very great. Small amounts of blood in the pericardial sac occasion few, if any, disturbances; its presence may never be noticed or it is only detected from the pericardial friction sounds which it gives rise to. Large amounts of blood in the pericardial sac occasion the most severe and urgent manifestations, viz., marked dyspnoea, cyanosis, and rapid, irregular, intermittent pulse; on physical examination the apex beat cannot be felt, there is an increased area of cardiac dulness, and faintness or entire absence of heart sounds.

Hæmopneumopericardium from associated injury of the lung and pleura is rare; it occasions a tympanitic resonance over the pericardial sac with succussion, in addition to the other physical signs.

Should wounds of the lung and pleura complicate the injury to the heart and pericardium, they would be indicated by the symptoms which have been detailed above (p. 173). Pericardial and cardiac injuries are likewise attended by grave shock.

It is, of course, difficult in a given case, to state definitely at first sight whether a penetrating wound in the cardiac area involves the pericardium alone or also the heart. Deep shock, great anxiety, and large degrees of hæmopericardium point to coincident lesions of the heart, an indication that is made more certain by a pulsating hemorrhage from the external wound, and by the presence of blowing, metallic, or purring murmurs over the heart. It is not to be forgotten, however, that even severe injuries of the heart may be attended by no shock and little hæmopericardium, the wound in the viscus being temporarily closed or being not completely penetrating. On account of the danger of carrying in infec-

tion, probing of the wound is not advisable. In cases of doubt it is better to freely expose the pericardial sac and determine by direct sight and touch the extent and character of the injury.

INJURIES OF LARGE BLOODVESSELS.

Injuries of the large bloodvessels result in such speedy death from hemorrhage that no opportunity for their diagnosis is afforded.

INJURIES OF ŒSOPHAGUS.

Injuries of the œsophagus are readily recognized by the escape of ingesta and mucus through the external wound.

INJURIES OF DIAPHRAGM.

Injuries of the diaphragm result from penetrating wounds and from severe crushing violence. They are usually complicated by lesions of the thoracic and abdominal viscera and are masked by the symptoms which the latter occasion. The chief significance of tears and wounds of the diaphragm lies in the possibility such injuries offer for the prolapse of one or more of the abdominal viscera into the thorax. If a considerable part of the stomach and intestines are so prolapsed the heart is displaced, the lungs are compressed, there is marked dyspnœa and tympanitic resonance over the chest, and there is danger that the prolapsed bowel will be constricted and strangulated by the opening in the wounded midriff with resulting intestinal obstruction and gangrene.

Uncomplicated wounds of the diaphragm usually pass unnoticed. They occasion pain at the site of the lesion, which sometimes radiates to the shoulder and is especially marked on forced respiration or cough. Visceral prolapse should be strongly suspected when there is dyspnœa, tympanitic resonance over the lower thoracic area, and displacement of

the heart, and especially if there is intestinal obstruction and vomiting. It should be remembered, however, that a thoracic injury, followed by a diaphragmatic pleurisy, sometimes occasions intense abdominal pain, constipation and vomiting, and so resembles the cases of diaphragmatic injury in which visceral complications are present.

The difficulty of differentiating the two conditions is especially great when only a single knuckle of intestine or small section of the stomach is prolapsed into the thorax and strangulated, and when there is consequently an absence of dyspnœa, tympanitic resonance, and displaced heart. With pleurisy, however, the constipation is never absolute, nor the vomiting persistent, as is the case with strangulation and obstruction of the bowel.

CHAPTER XIII.

INFLAMMATIONS AND NEOPLASMS OF THE CHEST WALL.

ABSCESSSES OF THE THORACIC WALL.

Acute Suppuration.—Acute suppuration of the thoracic wall affords the signs that attend such a condition in other parts—viz., local pain, heat, temperature elevation, and increased leukocytosis. If the abscess is superficially located, fluctuation appears early; otherwise this is not palpable at all or only after the abscess has come nearer to the surface. Superficial abscesses become more prominent when the underlying muscles are contracted, whereas deeply seated ones become flatter and less prominent when the muscles are put on the stretch. In doubtful cases aspiration will decide whether pus is present.

Mural abscesses secondary to pulmonary abscesses usually contain gas. When they do, they are tympanitic to percussion and therein resemble herniæ of the lung. The differentiation is, however, readily made; for herniæ of the lung can be easily replaced into the chest, which abscesses cannot be, and they do not present the usual signs of inflammation.

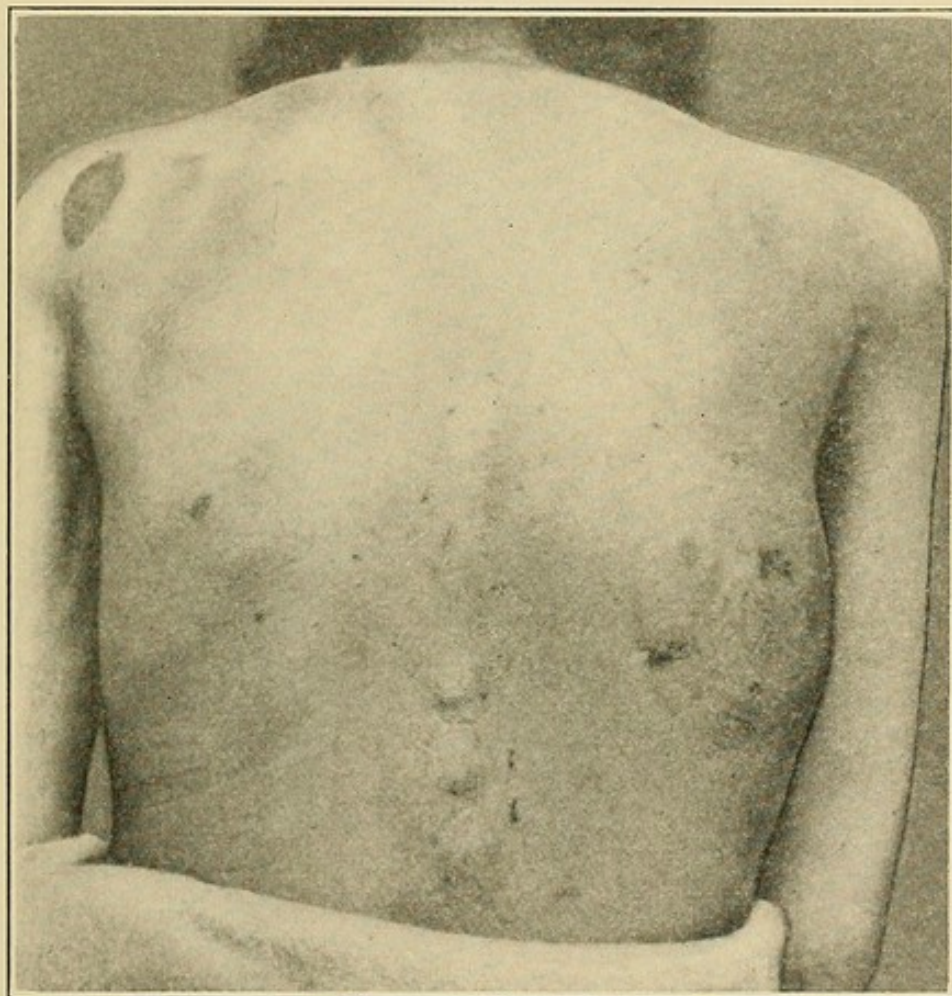
With mural abscesses secondary to empyema there will be, in addition to the above signs, those characteristic of empyema and aspiration of the pleural cavity will yield pus.

Deeply seated abscesses and phlegmons of the chest wall may at their incipiency be confounded with intrathoracic lesions, pleurisy, and pneumonia, but the subsequent development of the physical signs which attend the latter maladies will serve to differentiate them.

Chronic Suppurations.—Chronic suppurations of the thoracic wall are usually secondary to tuberculous disease of its bony structures; more rarely they are actinomycotic or syphilitic, and sometimes they are due to an extension

of a suppuration from glandular abscesses in the axilla. They form soft, fluctuating, slowly growing, painless swellings that are made more prominent by muscular contraction if they are superficial, and less so if they are deeply placed. Their tendency is to burrow along the muscular planes, perforate the latter, and eventually break through the skin in one or more places, thus creating profusely secreting

FIG. 93



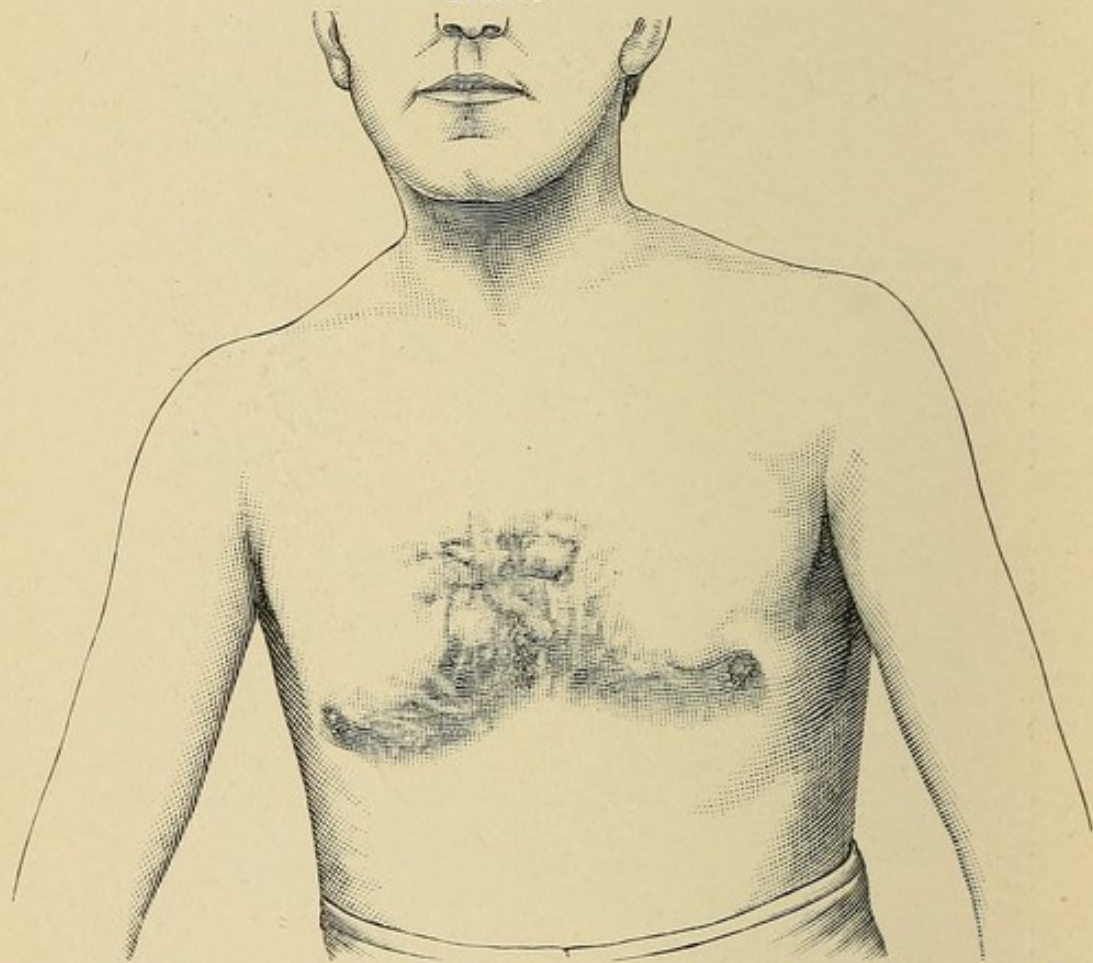
Mural thoracic abscess from perforation of empyema. Note enlargement of the right side of chest, suggesting large pleural exudate.

sinuses, whose external opening bears testimony, both by its appearance and by the character of its discharge as to the nature of the underlying malady. Thus, retracted, irregular scars and fistulous openings, from which a cheesy, flocculent material is discharged, are very suggestive of tuberculosis; a great deal of brawny induration surrounding small areas of softening and a discharge of minute yellowish granules

containing the ray fungus are indicative of actinomycosis; and a discharge of a homogeneous stringy material suggests syphilis. Other evidences of syphilis or tuberculosis will help to substantiate the diagnosis.

The tortuosity of the channels along which the pus has burrowed often makes it difficult to locate the site of the primary affection. This is best done by withdrawing the contents of the abscess cavity with an aspirating needle and

FIG. 94



Tuberculosis of the ribs and sternum. Note the retracted, irregular scars and the multiple fistulæ. (Von Bergmann.)

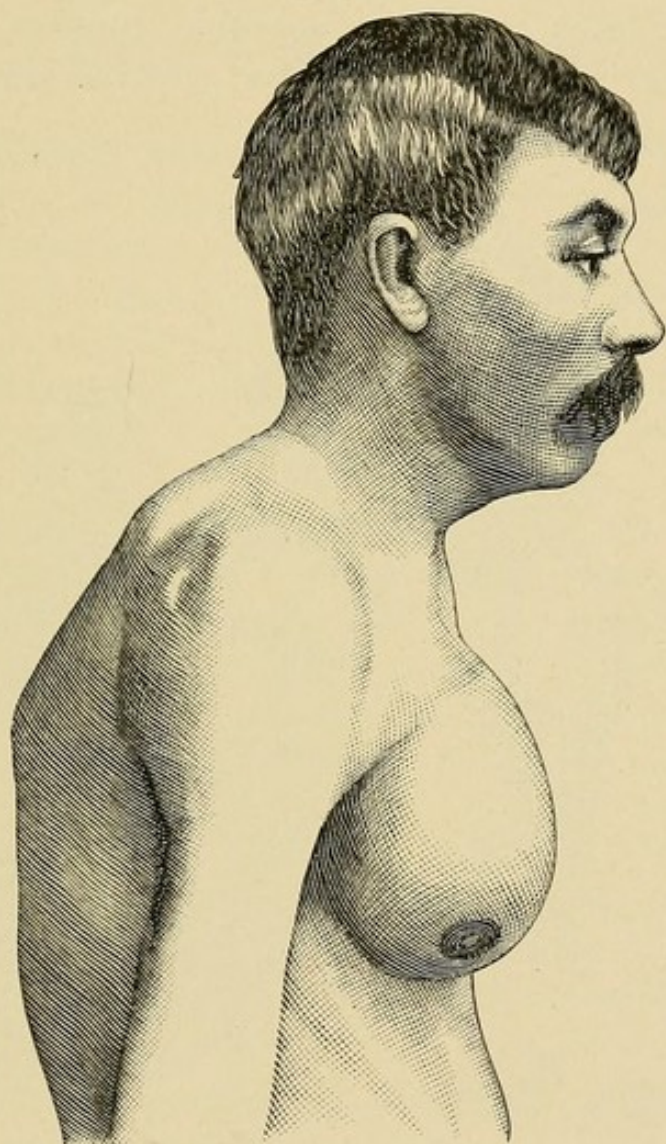
then injecting into it, under considerable pressure, some 10 per cent. solution of iodoform glycerin. The latter makes its way along the tortuous channels, and on exposing the patient to the *x*-ray, the entire course of the abscess down to its primary site is easily followed.

Aneurysms of the large intrathoracic vessels or of the intercostal or mammary arteries are differentiated from chronic abscesses by their compressibility, their expansile

pulsation, the systolic murmur and bruit to be heard over them, and the intense boring pain to which they give rise.

Herniæ of the lung, in contradistinction to chronic abscesses, are *tympanitic to percussion* and can be easily replaced within the chest.

FIG. 95



Large cold abscess of the thorax, from syphilitic disease of the ribs. Note the absence of tuberculous lesions in other organs. (Von Bergmann.)

NEOPLASMS OF THE CHEST WALL.

Benign neoplasms offer little difficulty in their diagnosis. *Atheromata*, *dermoids*, and *lipomata* occur most frequently on the back as soft elastic tumors, having pseudofluctuation. They may be confounded with chronic abscesses; the latter,

however, are of more rapid growth, are distinctly fluctuating, and are not lobulated nor attended with dimpling of the skin as are lipomata. In doubtful cases aspiration will at once clear up the diagnosis. *Retromammary lipoma* push forward the breast and make the impression of an hypertrophied gland. *Fibromata* form hard, very slowly growing, usually submuscular, and not very movable tumors, and *neurofibromata* form hard, very sensitive growths along the inter-

FIG. 96



Retromammary lipoma. (Albert.)

costal nerve trunks and provoke severe neuralgic pain. Of *vascular tumors*, the *nævi* and *telangiectatic growths* are readily recognized by their surface appearance and their compressibility. *Enchondromata* occasionally form on the ribs and sternum, usually at their junction with the cartilages, constituting hard, very slowly growing tumors, that may, however, attain considerable size and give rise to metastases in other organs.

CHAPTER XIV.

DISEASES OF THE BREAST.

Clinical History and Method of Examination.—In the clinical history of those suffering with affections of the breasts it is important to ascertain whether there has ever been any injury to the organ, whether the patient has ever nursed and the time of the last nursing, whether there have been previous inflammatory conditions in the gland, and whether there is any evidence pointing to tuberculosis or syphilis.

In examining the breast, the physician should sit in front of the patient. He should compare the size of the two glands and should take note of their surface appearance, the condition of the nipples and their areola, and when there is a discharge from the nipple he should mark its amount and its character. In palpating the organ, the pectoral muscles should be relaxed by raising and supporting the corresponding arm at a level with the shoulder. The whole gland should then be rolled beneath the flat of the hand, and any thickenings, irregularities or neoplastic formations within it noted. The mobility of the gland upon the thorax and the mobility of its cutaneous covering should also be determined. No examination is complete until the axilla has been carefully palpated. This is best done by crowding the axillary contents against the chest wall, or against the humerus, and then palpating them with the finger tips. The supra-clavicular spaces should also be palpated for enlarged glands.

INFLAMMATORY DISEASES OF THE BREAST.

Acute Inflammation.—Acute inflammation of the breast, characterized by a painful swelling of the gland and at times by a reddening of the skin over it, occurs with especial

frequency in the newborn (in males more frequently than in females) and at puberty.

Acute Suppurative Mastitis.—Acute suppurative mastitis is met with most often in women during the lactation period. It is characterized by the usual signs of acute suppuration—viz., swelling, redness, heat, rise of temperature, and increased leukocyte count. The abscesses may be superficial, beneath the skin, or within the substance of the gland, or retro-mammary. In the first two instances fluctuation appears early, but in the latter it is to be obtained only very late or not at all. Some axillary glandular swelling is always present.

In a patient that recently came under the care of the author, the tenderness and redness of the skin overlying a tumor in the breast and the axillary enlargement suggested to her family physician an abscess formation, which he proceeded to incise. The advanced age of the patient, the hardness and immobility of the tumor and of the axillary glands, with the absence of constitutional symptoms, should have easily enabled him to exclude abscess and diagnosticate a malignant neoplasm, which had become adherent to the skin and was about to ulcerate through it.

Chronic Mastitis.—The chronic forms of mastitis are difficult of differentiation from neoplastic formation. As a result of chronic inflammatory processes, multiple smaller and larger cysts form in the breast, or isolated lobules of the gland, become the seat of a nodular infiltration, occasionally accompanied by axillary glandular enlargement and atrophy and retraction of the breast. Such chronic mastitis is met with after puberty, especially in women who have borne children, but not nursed them. It may involve one or both breasts. The nodules and cysts are best appreciated by squeezing the breast between the thumb and forefinger. The cysts are rarely larger than a walnut, are tense and at times fluctuating, while the nodules are firm and leathery. Compression of the breast may cause a few drops of milky or brownish fluid to exude from the nipple.

The multiplicity of the tumors, their smooth surface, the fluctuation of the cysts, the variations in size and consistency of the nodules (now hard, now soft, now large, now small), the absence of lancinating pain in the breast (though radiat-

ing pain to the axilla may be complained of), the mobility of the breast upon the thorax and muscles, and the long duration and slow progress of the malady, usually suffice to differentiate these cases from neoplasms. The internal administration of the iodides acts favorably upon the size and consistency of the nodules, and they should always be given to aid us in the diagnosis. In doubtful cases an exploratory incision, with immediate examination of a frozen section under the microscope, is to be made. Under no condition is a patient with chronic mastitis to be dismissed from our observation, for malignant degeneration may occur at any time, and the patient should have the benefit of an early detection of such a change in the breast.

Benign Hypertrophy.—Benign hypertrophy of the breasts may occur at the time of puberty. It usually affects both breasts. Unilateral hypertrophy is suggestive of neoplasm. The breasts rapidly become very large, but remain soft, elastic, and movable, and do not cause pain.

Tuberculosis.—Tuberculosis is rarely primary in the breasts, though secondary infection from tuberculosis of the lungs, ribs, sternum, etc., is fairly frequent. It appears either as a circumscribed cold abscess or more usually as disseminated indurated nodules, having an irregular surface. The nodules tend to enlarge slowly, become soft in their centre and eventually perforate, leaving fistulæ which discharge cheesy, flocculent material. The axillary glands sooner or later become enlarged.

In the early stages the nodules are difficult to differentiate from cancerous nodes. Other evidences of tuberculosis may suggest the diagnosis, but no positive conclusion can be arrived at until a microscopic examination of a section of the nodule has been made. When caseous degeneration and fistulæ have formed the diagnosis is easy.

NEOPLASMS OF THE BREAST.

Clinically we are chiefly concerned as to whether a neoplasm in the breast is of a benign or malign character. Histologically it is essential to differentiate between the

various connective tissue and epithelial tumors and their combinations (the fibroma, myxoma, lipoma, angioma, chondroma, atheroma and sarcoma among the former, and the adenoma and carcinoma among the latter), but clinically this differentiation, at least in the initial stages, is often impossible. In this organ especially, where early positive diagnosis of benignancy or malignancy is not always possible, there is need of frequent resort to exploratory incision and histological examination of an excised specimen of the growth. Only in this way can we hope to arrive at an early positive diagnosis in the obscure cases.

Benign Tumors.—Benign tumors grow slowly, and are and remain circumscribed, encapsulated and movable. Of tumors with these characteristics the most common are the *fibroadenoma*, which are usually rounded, smooth (rarely irregularly lobulated), firm tumors while small, with areas of softer consistency when they attain larger size, and with fluctuating areas when cysts develop in them. *Lipoma*, *angioma*, and *chondroma* are rare, and are recognized by the special characteristics of fatty, vascular, and cartilaginous tumors.

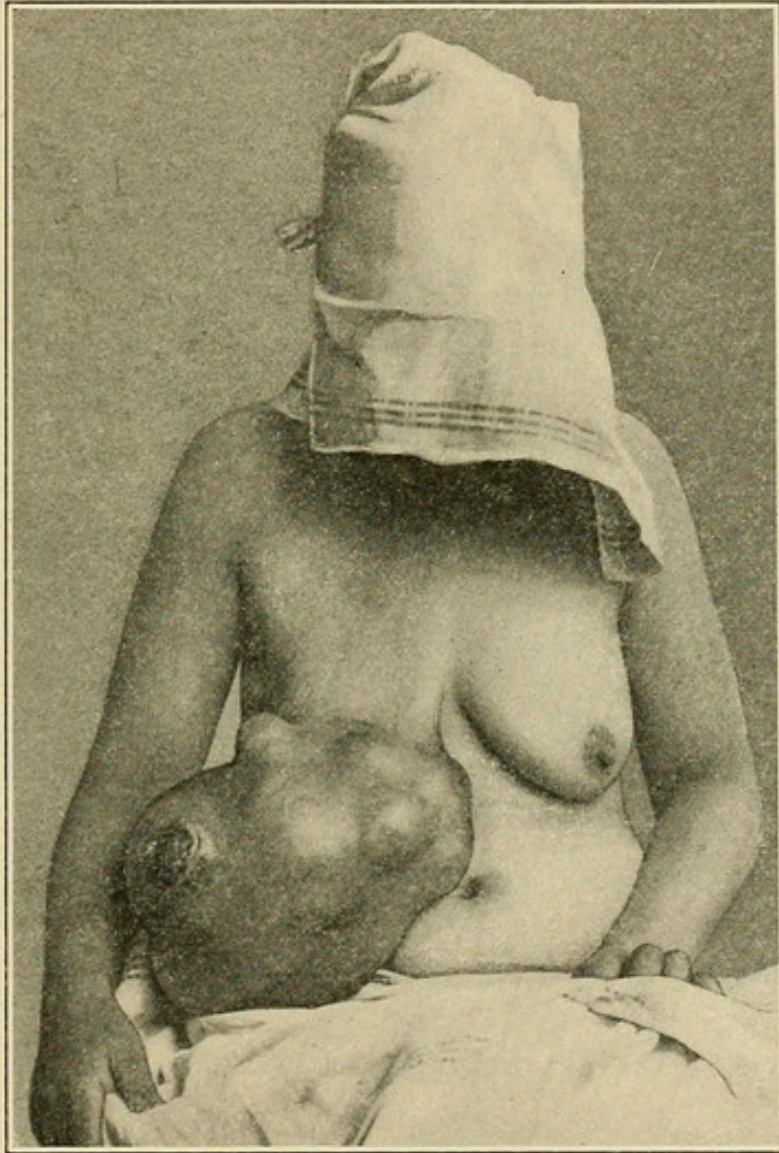
Simple cystic tumors, not as parts of fibroadenoma, sarcoma, or carcinoma, also occur; they are usually retention cysts, their contents being serous or mucoid or oily or creamy material. In the latter instance the cyst is termed "galactoceles."

Malignant Tumors.—Malignant tumors are most frequent after the fortieth year of life; they are hard (in many cases stony hard), grow rapidly, either are not or speedily lose their encapsulation, and very early become adherent and fixed in the breast, to the thorax, and to the skin. *Carcinoma* are very early attended with enlargement and induration of the axillary glands; in *sarcoma* the glandular enlargement appears late or not at all. These tumors cause marked interference with general health, cachexia, and are followed sooner or later by secondary growths in other organs, most especially the lungs, pleura, and vertebræ. As the growth enlarges it becomes adherent to the skin, breaks through it and leaves an ulcer which in carcinoma has everted, indurated edges, and a granulating, easily bleeding, crusty

base, and in sarcoma undermined, indurated edges and a fungous base.

The rapidity of growth depends upon the vascularity of the breast, and upon the amount of fibrous tissue in the neoplasm. Thus, in nursing breasts the growth is very

FIG. 97



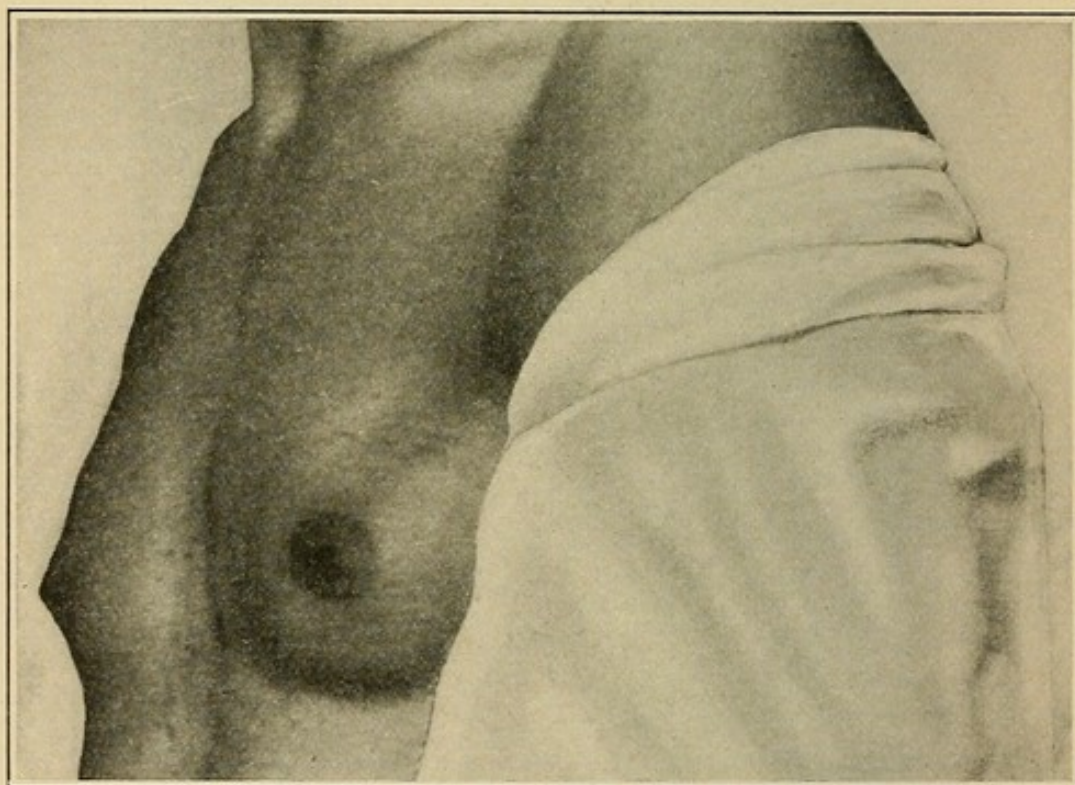
Large ulcerating sarcoma of the breast. Note fungous character of the ulcer which suggests the nature of the tumor.

rapid, while in an old subject the progress is considerably slower; similarly the richly cellular tumors increase in size very rapidly, while the fibrous ones grow very slowly. The presence of the fibrous tissue leads to retraction of the nipple and the skin overlying the tumor, but the absence of this retraction is not an evidence of non-malignancy any more

than its presence is a proof of it. (With the contraction of fibrous tissue, no matter whether benign or malign in character, retraction of the skin and nipple always occur.)

Of the malignant tumors, we are wont to differentiate the sarcoma and carcinoma. The former are more frequent between the twentieth and fortieth year, the latter after forty; and with the latter the axillary glandular involvement appears early, while in sarcoma the glands enlarge late or not at all. Clinically, however, it is not always possible to distinguish between them.

FIG. 98



Carcinoma of left breast in a male. Note diffuse enlargement of the gland, which was hard and fixed to the chest; axillary lymph nodes enlarged.

Sarcoma.—Of the sarcoma we may distinguish clinically:

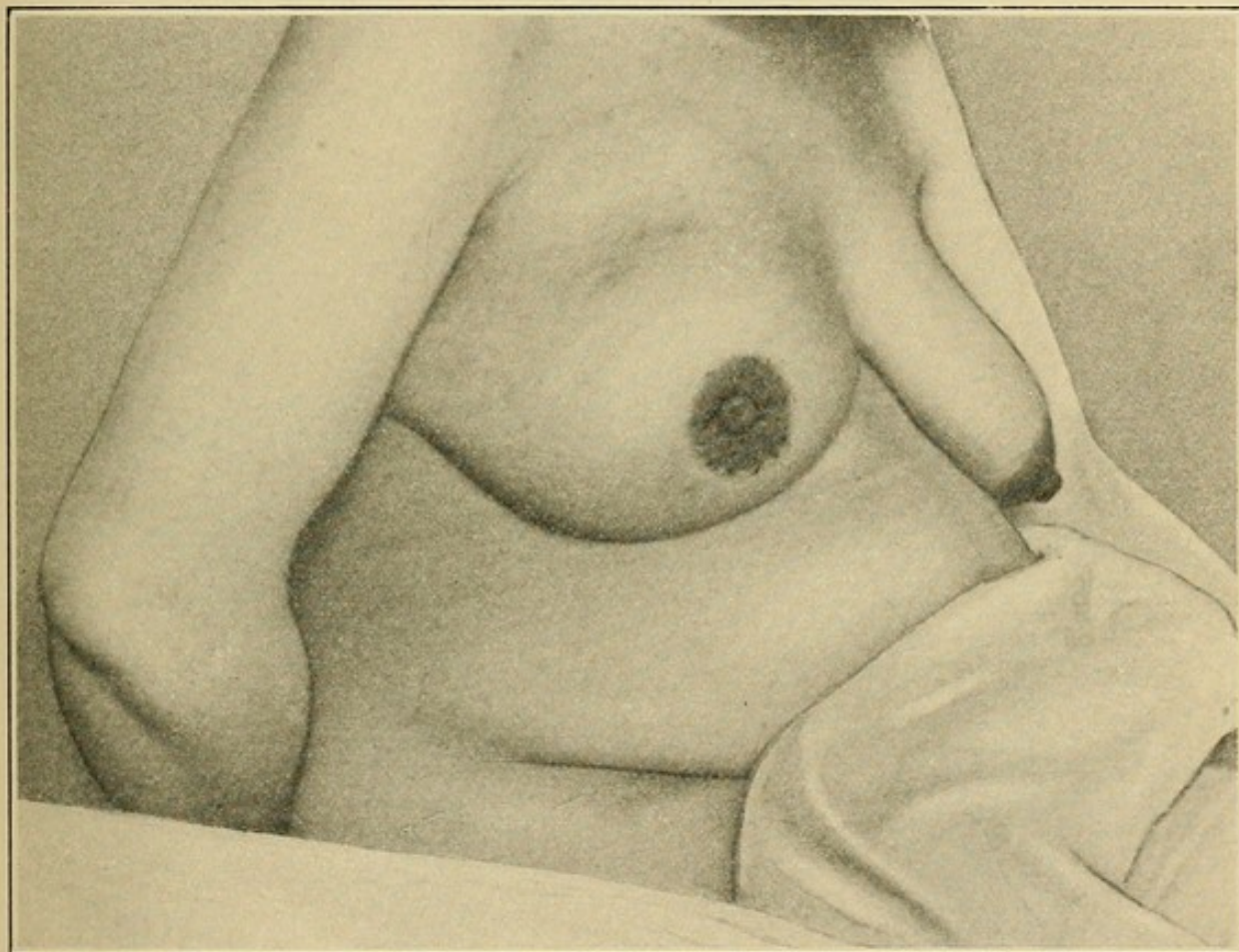
1. *The cystic sarcoma*, forming encapsulated movable tumors with irregular surface and of unequal consistency, with varying rapidity of growth, and attaining at times very large size.
2. *The soft sarcoma* (medullary and melanosarcoma), which grow very rapidly and infiltrate the breast.
3. *The hard sarcoma* (fibro- and spindle-celled sarcoma), which grow slowly and infiltrate the breast.

Carcinoma.—Of the carcinoma we may distinguish:

1. *The acinous* (medullary carcinoma), forming soft tumors which tend to early degeneration and ulceration, and from the base of which fungoid granulations appear.

2. *The tubular or simple carcinoma*, forming flattened tumors that infiltrate the skin, muscles, ribs and pleura, often in a disconnected chain (at times there are multiple

FIG. 99



Carcinoma of the right breast in pregnant woman. Its character could only be determined by histological examination of a frozen section.

hard nodules in the skin of the breast, back, and arm (*cancer en cuirasse*), while the remaining skin is of board-like hardness and inflamed).

3. *The scirrhus carcinoma*, of slow growth with contraction and atrophy of parts of the neoplasm; it is met with commonly in old subjects.

4. *The colloid carcinoma*, of slow growth and affording the best prognosis after removal.

It is to be especially noted that differentiation between small sarcoma, carcinoma, fibroma, adenoma, and chronic mastitis is not always possible. Stony hardness, non-encapsulation, and lancinating pain, steady progression and unchanging character should always excite suspicions of

FIG. 100



Atrophic scirrhus carcinoma of the breast. (Von Bergmann.)

malignancy and prompt an early exploratory incision with microscopic examination of an excised specimen of the growth. When the axillary glands have become intumescent and indurated the diagnosis is easy, but the radical cure of the disease is usually out of the question.

Diseases of the Nipple.—The nipple and its areola may be the seat of *erosions*, *syphilitic condylomata*, *atheroma*, and *epithelial carcinoma*. *Paget's disease*, falsely styled chronic eczema of the nipple, is an epithelioma, which originates in the epithelia surrounding the orifices of the ducts. In the earlier stages of this affection the nipple is reddened and painful, has a granulating appearance, and discharges a clear, yellowish fluid; or a psoriatic condition develops around the nipple. The differentiation from chronic eczema or psoriasis is difficult, but our suspicion should be aroused if the malady proves rebellious to local treatment.

CHAPTER XV.

DISEASES OF THE PLEURA AND LUNGS.

THIS chapter will be devoted to the diagnosis of empyema of the pleura, and abscess, gangrene, and neoplasms of the lung; but for the detailed physical evidences of these diseases the reader is referred to books on internal medicine.

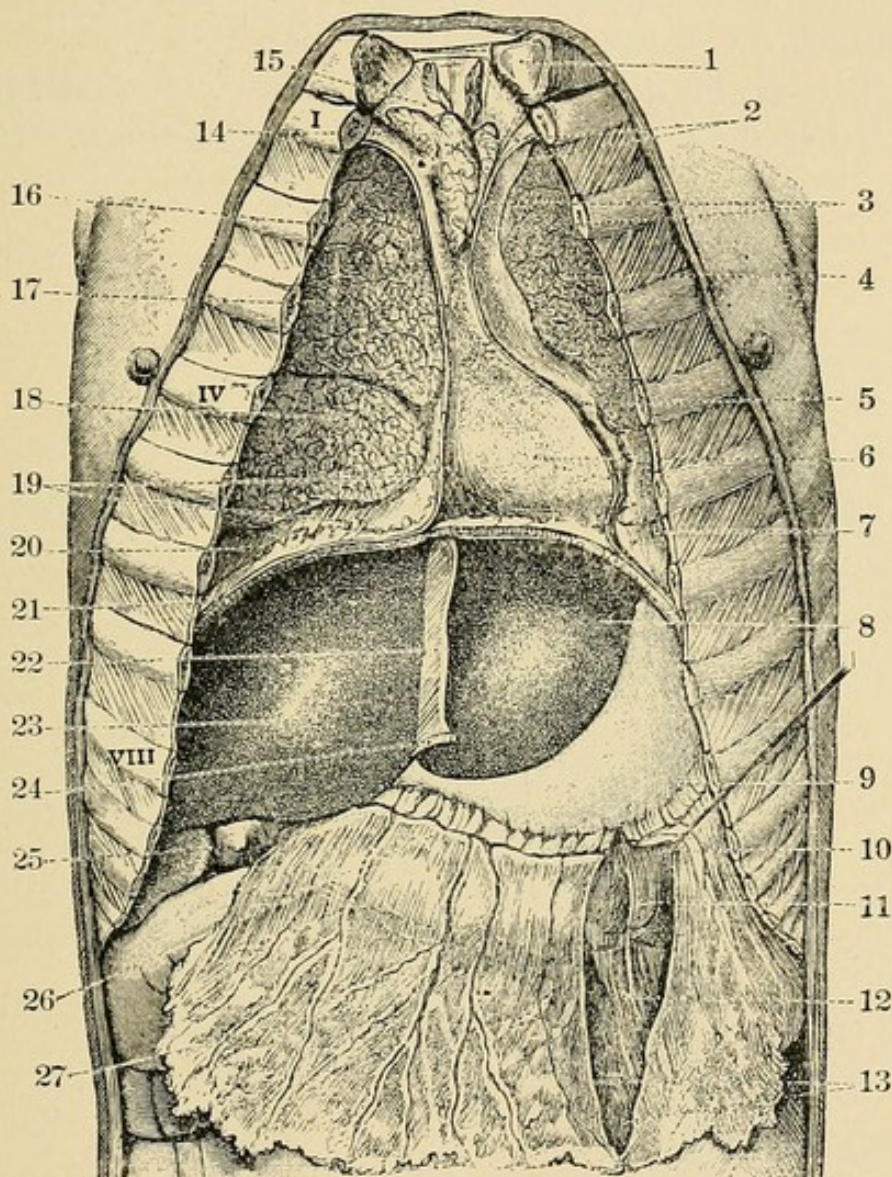
EMPYEMA.

Empyema of the pleura is usually secondary to pulmonary inflammation, less frequently to infradiaphragmatic suppuration, or suppuration of the chest wall, rarely primary without pulmonary lesion, exceptionally a manifestation of pyæmia, and sometimes due to an infected penetrating wound of the chest. The pus may be free in the pleural cavity, or it may be encapsulated; if the encapsulation is between the lobes of the lung, an *interlobar* abscess is formed. The free exudates are easier of diagnosis and differential diagnosis than are the encapsulated ones.

It is to be distinctly noted that although the presence of fluid within the pleural cavity can usually be diagnosticated from the physical signs, and its character be probably determined from the temperature curve and blood count (high leukocytosis, above 20,000, pointing to pus), yet the only positive way of ascertaining the presence and nature of a pleural exudate is by aseptic puncture. If this is done properly, no harm can result, and only in rare instances of encapsulated exudates are negative results obtained when pus or fluid is present in the pleural cavity. If the first puncture is negative, we should not hesitate to repeat the procedure a number of times if the physical signs and constitutional symptoms point to the presence of pus.

Physical Signs.—The classical physical signs afforded by a pleural exudate—viz., *enlargement of the affected side of the*

FIG. 101

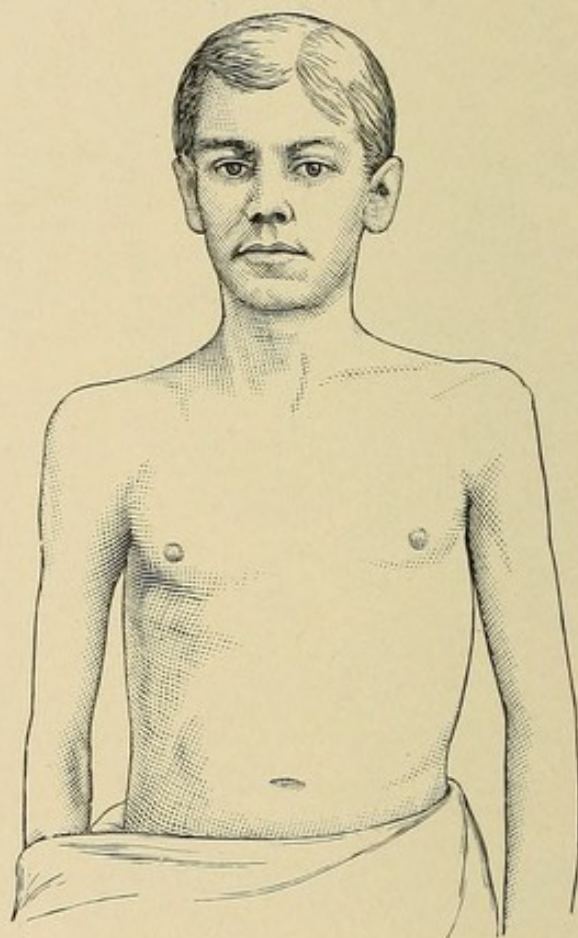


Topography of the thoracic and upper abdominal organs. Note limits of the pleuræ and lungs in reference to the bony chest wall. 1. Sternal end of the clavicle. 2. Thymus gland. 3. Mediastinal layers of the pleura. 4. Upper lobe of left lung. 5. Pericardial layer of pleura. 6. Anterior mediastinum. 7. Costophrenic sinus. 8. Left lobe of liver. 9. Greater curvature of stomach. 10. Bursa omentalis. 11. Transverse colon. 12. Great omentum. 13. Inferior omental recess. 14. Dome of the pleura. 15. Upper mediastinum. 16. Right lung. 17. Mediastinal layers of the pleura. 18. Right lung. 19. Fatty folds of the pleura. 20. Diaphragmatic pleura. 21. Diaphragm. 22. Falciform ligament of the liver. 23. Right lobe of liver. 24. Round ligament of liver. 25. Fundus of gall-bladder. 26. Gastrocolic ligament. 27. Great omentum.

chest, diminution of its respiratory movement, absence of vocal fremitus and voice, dulness on percussion, absence of or

greatly diminished respiratory murmur, and displacement of the heart, diaphragm, and lung—may be somewhat irregular and resemble those which are given by a pneumonic consolidation. If the exudate forms during the existence of a pneumonia or immediately after its defervescence, the physical signs, high fever and high leukocyte count, may be attributed to delayed resolution of the pneumonia. In these

FIG. 102



Enlargement of the left chest from large pleuritic exudate. (Weintraub.)

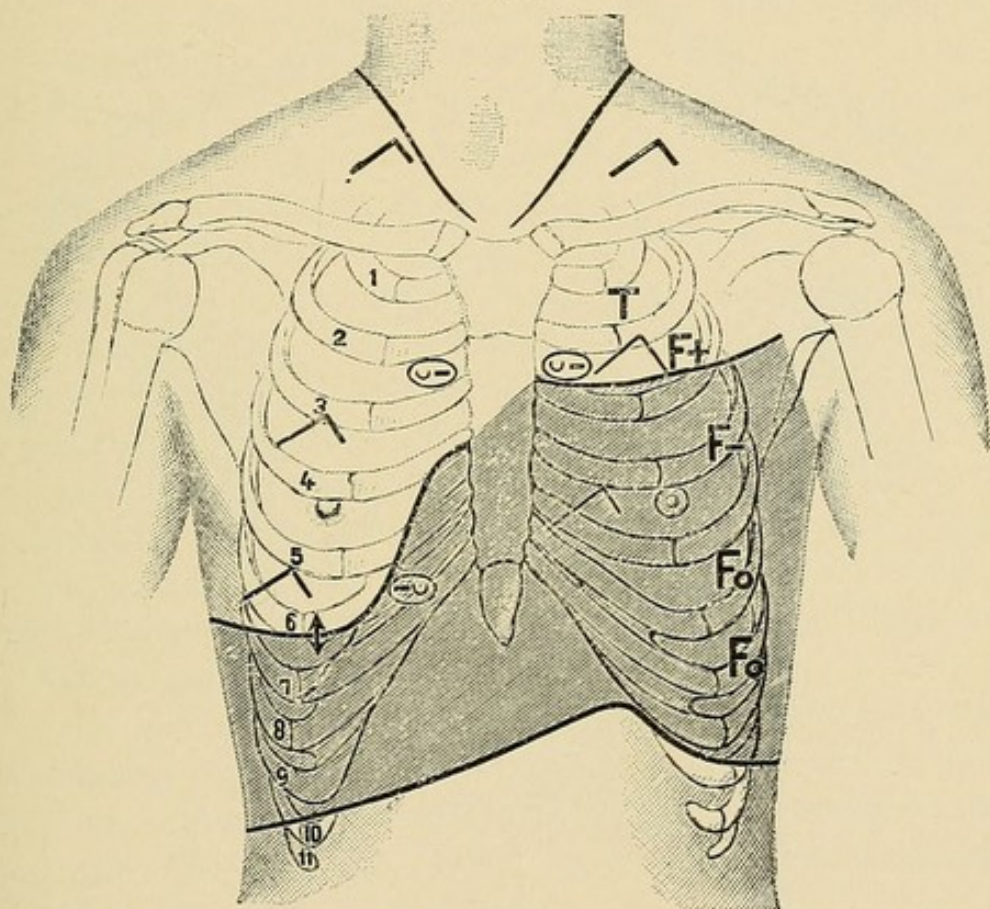
cases especially is exploratory aspiration to be practised, in order to make an accurate diagnosis.

Purulent collections below the diaphragm (subphrenic abscess) give physical signs that are somewhat similar to those afforded by empyemata. But with subphrenic abscess there is an elevated, dome-shaped, basal dulness, with normal pulmonary resonance above it, and normal vesicular breathing can be heard up to the level of dulness; whereas in empyema the breathing is altered up to the top of the chest,

and the basal dulness is concave upward. The history of a preceding intra-abdominal suppuration or of visceral perforation is strongly suggestive of subphrenic abscess. In some instances a purulent or serous pleural effusion develops secondarily to the subphrenic abscess, and its presence makes the diagnosis of the subphrenic abscess more difficult.

A coincident pleural and subphrenic exudate is to be strongly suspected when on transpleural aspiration we obtain

FIG. 103



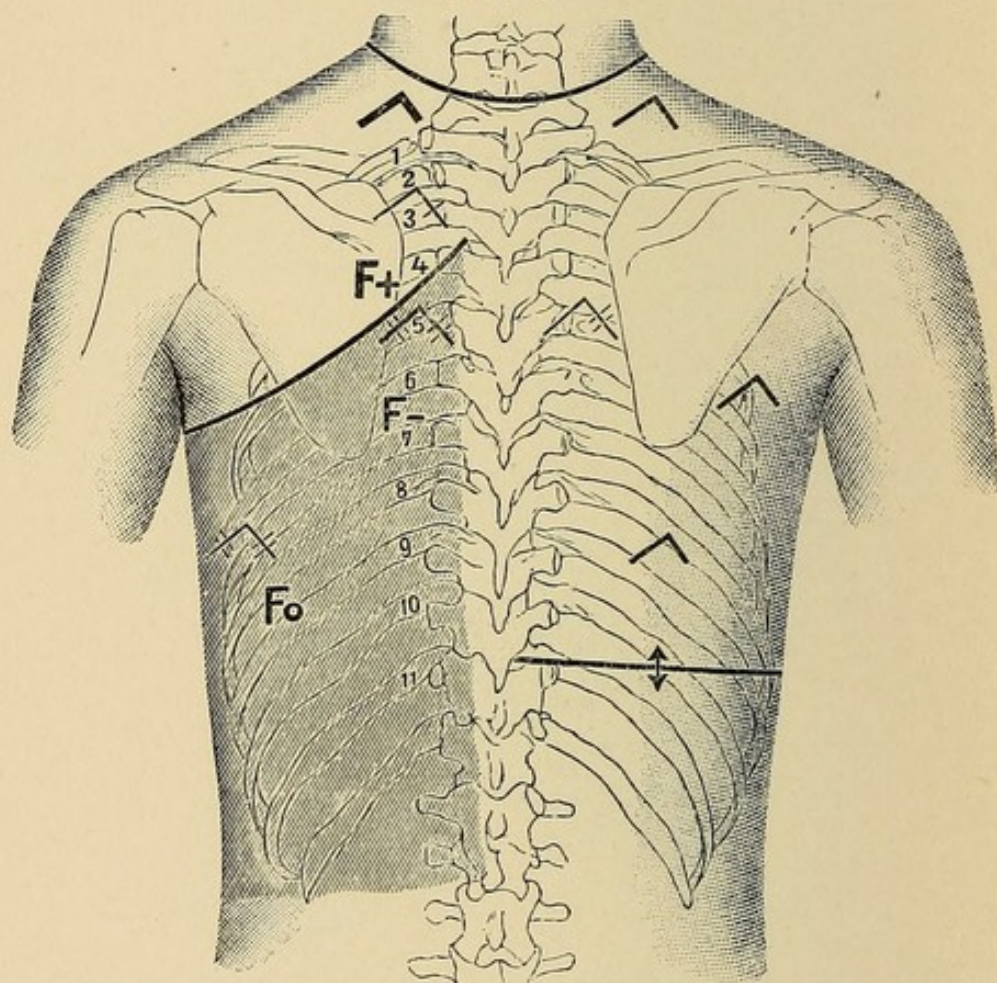
Anterior view of shape of the upper line of flatness in left pleural exudate.
(Weintraub.)

one kind of pus from the subphrenic space and another kind of pus or a serous fluid from the pleural cavity.

The *sacculated pleural exudates* are more difficult of diagnosis because the classical physical signs are apt to be somewhat irregular, and especially difficult are the cases in which the pus is encapsulated between the lobes of the lung (the interlobar abscesses). It is in this class of cases that we have the greatest use for aseptic exploratory aspiration, and if the

first puncture is negative the procedure should be repeated until the pus is found. The interlobar abscess usually points between the anterior and posterior axillary lines, over the course of the incisures of the lung. An interlobar exudate is distinguished from a pulmonary abscess which communicates with a bronchus by the absence of elastic fibres in the sputum, the absence of a profuse three-layer sputum, and by

FIG. 104



Posterior view of same as Fig. 103. (Weintraub.)

the constancy of the physical signs. (With pulmonary abscess the physical signs vary before and after expectoration.) From pulmonary abscess not in communication with a bronchus the differentiation is impossible, and may even remain so on the operating table.

Chronic Sacculated Empyema.—A chronic sacculated empyema may be simulated by a *chronic pneumonia with bronchiectasis*, in which the large bronchi and the cavities

are filled with exudate and sputum. Aspiration may even yield a purulent material which is derived from the cavities. An abrupt outline of the area of dulness and the expectoration of large masses of sputum are characteristic of the latter malady.

A chronic sacculated empyema may be simulated by a *mediastinal* or *pleural tumor*. An irregular area of dulness which gradually extends in several directions, the presence of some vocal fremitus over this dull area, and negative results from repeated aspirations are characteristic of the latter conditions.

A chronic sacculated empyema which transmits the cardiac movements to the chest wall may pulsate *visibly* and *sensibly*, more frequently only the latter. When such pulsating empyemata are located in the anterior part of the chest over the aortic region, they may be confounded with aortic aneurysms. The similarity of the radial pulses, the absence of systolic murmur over the pulsating swelling, and the absence of signs of compression of the nerves, œsophagus and trachea, and of severe boring pain at the site of the bulging will enable us to exclude aneurysm. Exploratory aspiration is not to be made in these cases until every other diagnostic aid, including fluoroscopic examination, has been employed.

Large peripleuritic exudates may simulate *sacculated empyemata*; but with such collections of pus there is no evidence of pulmonary compression, no displacement of viscera, and the bulging of the intercostal spaces is strictly limited to the area of dulness.

The presence of air and pus in the pleural cavity (*pyopneumothorax*), the result of an infected pneumothorax or of a perforation into the pleura of a pulmonary abscess, is recognized from the signs of fluid, with the addition of tympanitic resonance at the top of the chest, and succussion. It is distinguished from *pyopneumothorax subphrenicus* by the fact that in the latter the tympanitic resonance is at the base of the chest, while in the former it is at the top. The anamnesis is also of value in making the differentiation. (See p. 369.) Thus a history of intraperitoneal suppuration or of ulceration of an intra-abdominal organ speaks

strongly for a subphrenic suppuration, while a preceding thoracic disease is strong evidence in favor of *supraphrenic* pus formation.

GANGRENE AND ABSCESS OF THE LUNG.

Gangrene of the lung occurs when necrotic areas thereof undergo putrefaction. In the early stages of the process—*i. e.*, before the necrotic tissue breaks down—the physical signs over the affected portion of the lung are similar to those which are afforded by a pneumonic consolidation. Later on—*i. e.*, when softening and liquefaction of the gangrenous area has taken place—the physical signs are those of an abscess cavity, together with a fetid odor of the breath and a fetid expectoration. It must not be considered, however, that every case of pulmonary abscess attended with fetid breath and fetid expectoration is one of pulmonary gangrene, for the organisms which cause putrefaction are always present in the air passages and very often they occasion decomposition of the purulent contents of stagnant abscess cavities that result from causes other than gangrene, and so give rise to fetid breath and fetid expectoration. It would be desirable to distinguish between the cases in which the pulmonary gangrene is the primary lesion and the abscess formation the secondary one, and those in which the abscess is the primary affection and the decomposition of the contents the complicating condition, for in the latter there is really no pulmonary gangrene, as the putrefactive organisms which are resident in a stagnant abscess cavity rarely invade its walls. Clinically it is not possible to separate these groups of cases, unless the history points to the presence of a pulmonary abscess prior to the time when fetid breath and expectoration were noticed, or unless there is in the history a distinct cause for the gangrene of the lung—*e. g.*, cardiac or vascular disease, pneumonia, etc.

The presence of an abscess in the lungs is determined from the anamnesis, from the constitutional manifestations of suppuration, by physical examination of the chest and sputum, and from the leukocyte count.

A previous pneumonia, especially of the aspiration type, or an old bronchiectasis or tuberculosis are the most frequent conditions that lead to the formation of pulmonary abscess. Less commonly the latter are the result of the lodgement of infected emboli in the lungs; occasionally they are due to a rupture of an empyema, liver abscess, a subphrenic abscess, suppurating echinococcus cyst, suppurating bronchial gland, malignant growth of the œsophagus, or purulent mediastinitis into the lung.

Percussory and Auscultatory Phenomena.—The percussory and auscultatory phenomena pointing to a pulmonary abscess are:

1. *A dull tympanitic resonance* over a circumscribed area (the tympanicity being the predominant sign), which disappears on change of posture and reappears on assuming the former position, or a tympanitic resonance over a circumscribed area while the patient is in the *lying position*, changing to *dulness* in its lower part and deeper tympanitic resonance in its upper zone when the *upright* position is assumed.

2. *Metamorphic breathing*—*i. e.*, a respiratory murmur whose first part is vesicular and whose final part is amphoric or bronchial. This is an almost certain sign of cavity.

3. *Metallic tinkle.*

4. *Amphoric breathing*, a very doubtful sign.

It is important to compare the physical signs which are obtained *before* and *after* expectoration. Thus, a dulness and absence of breathing over a circumscribed area before expectoration may change after it to a dull tympany with bronchial breathing or one of its varieties or amphoric breathing with metallic tinkle.

Expectoration.—The expectoration in cases of pulmonary abscess is usually of a fetid character due either to gangrene of the walls of the cavity or to decomposition of the contents. In the latter instance the fetid character may not be constantly in evidence. The patient is often able to tell by taste and smell the region of the lung from which the putrid material is derived. *On standing* the sputum separates into *three layers*, the lowest being a purulent fluid material of

varying consistency, the middle a turbid fluid, and the upper a ropy, frothy material, to which frothy, purulent, rounded balls and thready matter adhere. Where there is little secretion from the bronchial tubes in the expectorated material, the pus is more confluent in the lowest layer, and there are but few purulent balls in the upper layer. The latter are suggestive of a tuberculous bronchiectasis.

In fresh cavities and in advancing cavities elastic fibres are present in the expectorated material, and can be demonstrated by boiling the latter in caustic potash.

Various forms and varieties of bacteria are present in the sputum when the bronchial tubes share in the suppurative process. A preponderance of a single variety of bacteria, either streptococcus, or pneumococcus, or staphylococcus, points to a *perforation of an extrapulmonary abscess or empyema* into the lung.

When the abscess cavity occupies the lower lobes, the expectoration of pus usually takes place at periodic intervals, at which time large quantities will be coughed up. The discharge is often influenced by posture and local pressure; this affords the observer an idea of the site of the abscess.

Roentgen Ray.—The Roentgen rays show air-containing cavities as lighter areas surrounded by darker ones.

Diagnosis of the Site of the Abscess.—It seems reasonable to suppose that by percussion and auscultation it would be comparatively simple to locate an abscess cavity. It is to be noted, however, that it is only the abscesses in the upper lobes and those in the lower lobes which are surrounded by rigid, non-collapsible walls, as the metapneumonic abscesses and the bronchiectatic abscess following empyema that are likely to give percussory and auscultatory evidences of their presence. Where the anamnesis, profuse expectoration, fever, and leukocyte count point to an abscess formation the site of which cannot be determined by percussion and auscultation, it is well to remember that periodic profuse expectoration is more likely to go with lower lobe abscesses, and that long-standing abscesses are usually in the lower lobes behind, and that the patient by the taste and smell of the sputum may be able to state from what

region the fetid expectoration is derived, and finally that the *x*-ray may indicate its position.

The depth of the cavity from the surface of the lung cannot be foretold, even after exposure of the lung on the operating table. Numerous pleuritic friction rales would seem to indicate that the suppuration is near the surface of the lung.

Diagnosis of the Number of Abscesses.—Single abscesses are only to be assumed in cases in which, within a short time, there is a considerable amount of lung tissue expectorated and when the signs of cavity formation persist over one and the same region.

Single abscesses following croupous pneumonia may be confounded with interlobar or other encapsulated empyemata. They may all occasion dulness and diminished breathing, but if after expectoration the dulness changes to dull tympanicity, and the breathing becomes metamorphic and there are elastic fibres in the sputum, an abscess is to be inferred. Multiple abscesses may be assumed when cavity symptoms are manifested over different and separate regions, and when different metamorphic breathing sounds are heard over neighboring areas. Bronchiectatic cavities are multiple.

Character of Abscesses.—The character of the abscess, whether tuberculous or not, is to be determined from the anamnesis, the duration of the malady, and from the presence or absence of tubercle bacilli in the sputum. Breaking down gummata are rare; their presence is to be determined from the previous history of the patient and their yielding to antispecific treatment; other evidences of syphilis speak for this condition.

NEOPLASMS OF THE LUNG.

Primary Neoplasms.—Primary neoplasms of the lung can only be diagnosed when they attain sufficient size to compress the mediastinal structures and to give dulness on percussion. The benign growths are of very infrequent occurrence. The malignant tumors, sarcoma and carci-

noma, are more frequently metastatic than primary growths; the metastatic sarcoma to primary sarcoma of the femur, and the metastatic carcinoma to primary carcinoma of the breast being especially common.

The initial stage of malignant disease is attended with cough, pain at the site of the neoplasm, expectoration, and a hemorrhagic exudate into the pleural cavity. As the tumor enlarges, it compresses the bronchi and causes dyspnoea and profuse hemorrhagic, at times fetid, expectoration. In cases of pulmonary sarcoma Lenhardt lays stress on the presence of rounded, fatty kernels in the sputum, and others dwell on the significance of giant vacuole cells (ten to twenty times as large as leukocytes) in the pleuritic exudate. Physical examination reveals relative or absolute dulness over an irregular area corresponding in its outlines to the limits of the tumor, with diminished or absent breathing over this area of dulness. X-ray examination shows a deeper shadow corresponding to the site of the tumor. A radiating deep shadow is considered pathognomonic of a tumor of the bronchi.

Echinococcus Cysts.—A small and centrally located echinococcus cyst of the lung gives no evidences of its presence, and hence its detection is not possible. But as the cyst grows and approaches the surface of the lung it occasions a superficial dulness, pleural friction rales, and diminished breathing over a circumscribed area. The larger cysts compress the lung and sometimes the bronchi, and thus occasion dyspnoea, cough, and expectoration of a hemorrhagic or wine-colored sputum. In the absence of physical signs which would point to the presence of a neoplasm this expectorated material would suggest tuberculosis; this can, however, be excluded by the absence of tubercle bacilli in the sputum, and by the lack of a reaction after a tuberculin injection.

Perforation of the cyst into a bronchus is indicated by an expectoration of clear echinococcus fluid in which hooklets are to be found, and sometimes daughter cysts. As the walls of the parent cyst collapse after its perforation, the physical signs of a cavity are rarely to be obtained. Perforation into the pleural cavity is evidenced by severe pleuritic pain and

by the immediate presence of an exudate in the pleural cavity; after a few days an urticarial eruption may appear.

The presence of bile in the expectorated echinococcus material is evidence of a primary liver cyst which has ruptured into the lung and then into a bronchus.

CHAPTER XVI.

DISEASES OF THE MEDIASTINUM.

IN the mediastinal space there are contained within narrow limits a number of important organs—viz., the heart and the large bloodvessels, the trachea, œsophagus, nerves, lymphatic and thymus glands, and occasionally accessory lobes of the thyroid. The enlargement of any of these organs, whether from inflammation or neoplasm, produces pressure upon the neighboring structures, and it is the evidences of such pressure that first attract attention to the mediastinal disease.

Thus the patients complain of vague pain in the back, from the second to the sixth dorsal vertebræ or in the sub-sternal region; of dyspnœa, paroxysmal cough, and dysphagia; and on physical examination there is found a prominence of the veins of the face, neck, and thorax, occasionally œdema of these parts, and a rough, blowing, respiratory sound, with much prolonged expiration over the interscapular region.

The severity of the pressure symptoms is of course in proportion to the size of the swelling. When the latter attains considerable dimensions there is a dulness in the interscapular region from the second to the sixth dorsal vertebræ, or in front to one or both sides of the sternum.

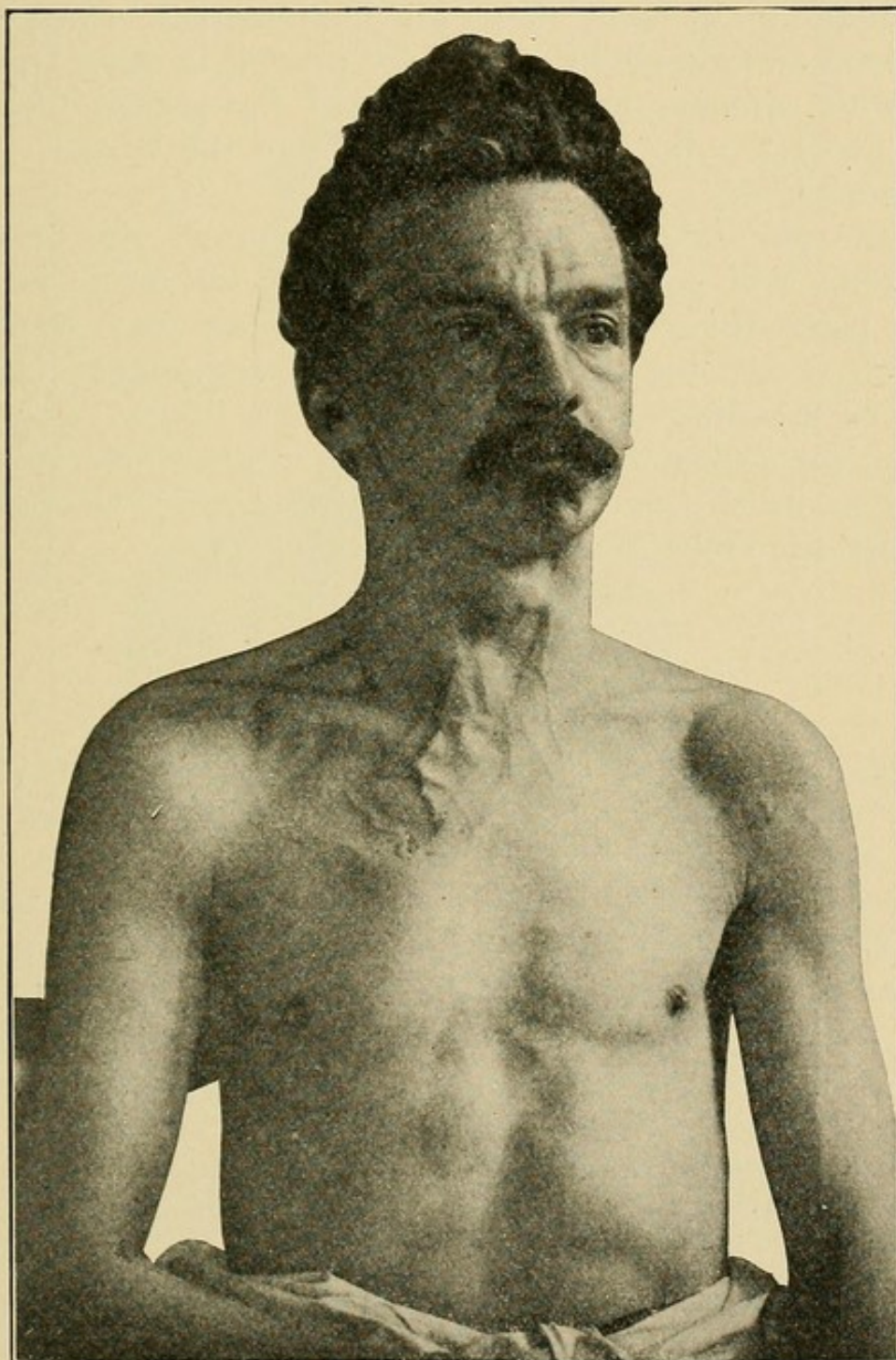
These symptoms and physical signs point only to the presence of mediastinal disease; they tell us nothing of the nature of the malady.

In determining this we are very materially aided by a complete previous history and by a general physical examination, but quite often we will be able to arrive at a decision only by a process of exclusion.

GLANDULAR ENLARGEMENT.

Glandular enlargement will be suggested if there is swelling of the glands in other regions—*e. g.*, the neck, groin,

FIG. 105



Carcinoma of the mediastinum. Note the prominence of the veins of the neck and thorax. (Curschmann.)

abdomen, and axillæ. With tuberculosis of the glands the swelling is rarely so great as to cause pressure symptoms, and there are very apt to be other evidences of tuberculosis in the lungs, cervical glands, etc. With syphilis of the glands there is a history of an initial chancre, and there are apt to be other lesions of this disease in the bones, skin, and mucous membranes. With leukæmic and pseudoleukæmic glandular enlargement there are characteristic changes in the blood and glandular masses are present in other regions; and with sarcoma and carcinoma of the glands there is, as a general rule, a primary growth in the breast, lower extremities, etc.

If the glandular swelling is inflammatory the pressure symptoms will not be very pronounced, but when healing takes place the contraction which follows may result in traction diverticula of the bronchi or œsophagus (see p. 210), or in adhesions to the pleura and lungs. The neoplastic, pseudoleukæmic, and leukæmic glandular enlargement is usually very considerable and the evidences of pressure are consequently very marked.

NEOPLASMS.

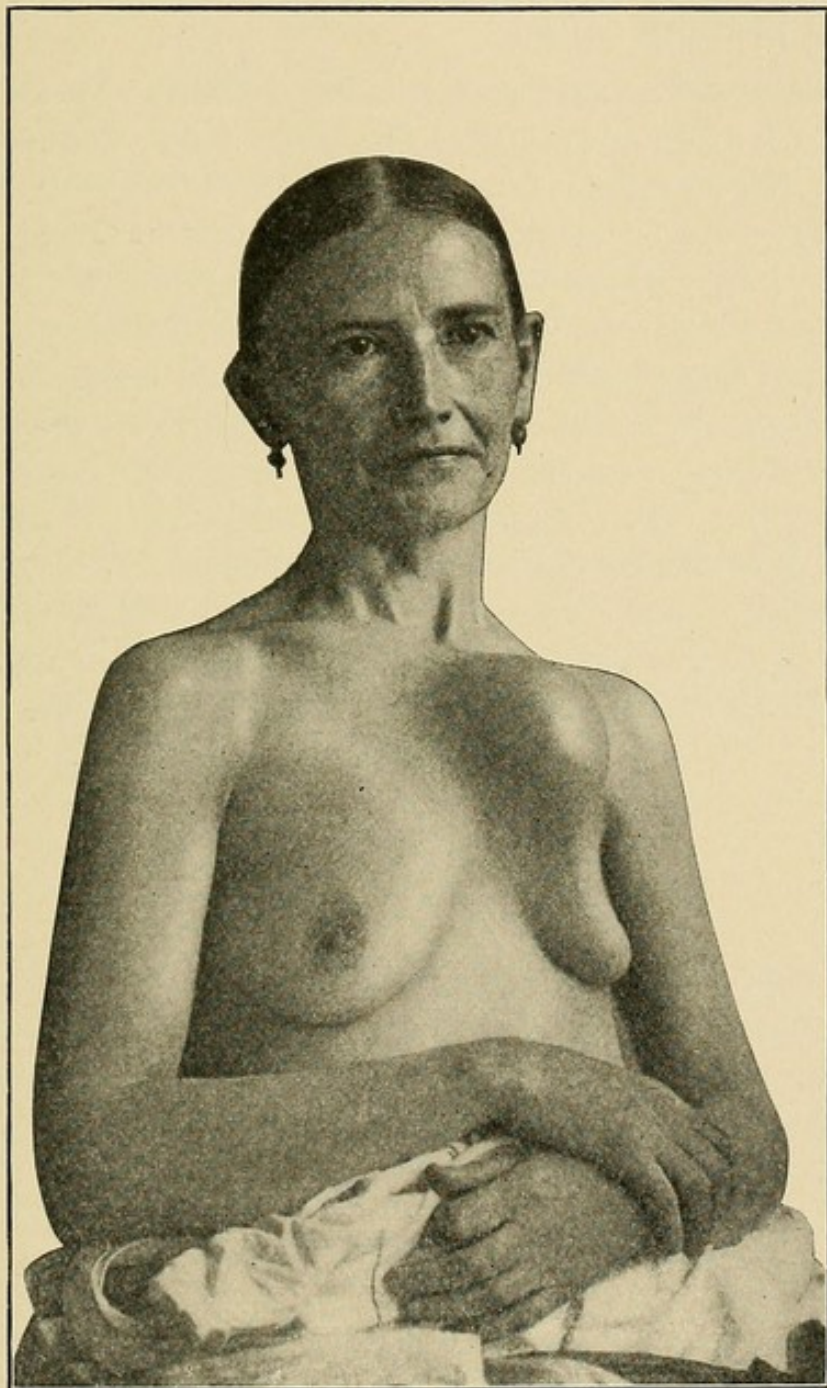
Benign neoplasms of the mediastinum are infrequent, the more common being the branching lipoma and dermoid cysts, and the more rare the echinococcus and simple cysts. Their slow growth and the absence of cachexia and metastases differentiate them from malignant growths, and the absence of the classical symptoms of aneurysm distinguishes them from vascular tumors. The possibility of the tumor being a diseased accessory thyroid lobe should always be borne in mind.

Tumors which originate in the lungs, pleuræ, œsophagus, or bloodvessels, and secondarily invade the mediastinum, usually give some evidences from the primarily affected viscus which leads to the correct diagnosis. In the absence of such signs their differentiation from primary mediastinal tumors and diseases will be very difficult, if not impossible.

SUPPURATIVE MEDIASTITIS.

The presence of a suppurative mediastinitis may be assumed if to the evidences of pressure upon the mediastinal

FIG. 106



Aneurysm of the arch of the aorta, giving the classical signs of this disease and compressing the structures in the upper mediastinal region. (Curschmann.)

organs there are added the symptoms of pus formation—viz., fever, chills, sweating, rapid pulse, throbbing pain in the

chest and back, and a high leukocyte count. As such sup-
puration is never a primary process, but always follows
perforation or ulceration of the organs in the mediastinum
or neck, or suppuration of the vertebræ, or some acute
infectious disease like typhoid fever, pneumonia, smallpox,
etc., we should always try to ascertain the nature and the
site of its primary cause.

CHAPTER XVII.

DISEASES OF THE ŒSOPHAGUS.

THE Œsophagus is a muscular tube, the function of which is to transport food from the oropharynx into the stomach, and the first result of its disease is consequently a disturbance of this function—*i. e.*, dysphagia. Those surgical diseases as retropharyngeal abscess, tumors of the oropharynx, tonsils, and cervical glands, laryngeal ulcerations, etc., which prevent ingesta from reaching the Œsophagus and so interfere with swallowing, usually afford other clinical evidences besides the dysphagia—*viz.*, nasal voice, laryngeal cough and breathing, disturbances of the spine, etc.—that should prompt the examiner to look carefully into the mouth and larynx for the cause of the difficulty. Once the food enters into the Œsophagus its further passage downward into the stomach depends upon the integrity of the muscular wall of the tube and upon the calibre of its canal.

DISTURBANCES IN ACTION.

Disturbances in the action of the muscular fibres may be in the nature of *overaction* or *paralysis*, the former affecting most frequently the fibres around the cardiac orifice and resulting in what Mikulicz has described as cardiospasm.¹

DISTURBANCES IN LUMEN.

Disturbances in the lumen of the Œsophagus may be in the nature of a *total* or *partial occlusion*, as by foreign bodies,

¹ Under normal conditions the cardiac orifice opens automatically during the act of swallowing, but in cardiospasm it remains closed and must be overcome by the forcible contraction of the muscular wall of the Œsophagus, with consequent hypertrophy and dilatation of it.

or of a *contraction* (stenosis) or *dilatation*. The stenosis may be due to simple or malignant ulceration or stricture of the œsophageal wall or to compression of the organ by neighboring neoplasms, aneurysms, etc. Dilatation of the lumen may be present throughout the entire extent of the organ—*e. g.*, after paralysis of the muscular coat (as occurs in bulbar paralysis); or it may be limited to a portion of the tube—*e. g.*, above a stricture or spasm; or it may be confined to a part of the circumference of the tube—*e. g.*, a circumscribed pouch-like protrusion or bulging of the wall (a diverticulum).¹

Clinical Evidences.—The clinical evidences resulting from partial or complete occlusion by foreign bodies, from stenosis, from dilatation, and diverticula, and from spasm are about the same. The patients complain of *dysphagia*, *regurgitation* of ingested material immediately or some time after their taking, and *inanition*. The dysphagia in the early stages of the disease is noticed only when solid food is taken, but later on it is also present when liquids are ingested. The diagnosis of the nature of the malady must be made by a careful examination.

The examination should always be preceded by the elicitation of a complete previous history, in which it is especially essential to ascertain the age of the patient, the duration and character of the initial symptoms; whether the individual was ever afflicted with syphilis or severe typhoid fever, in the course of which œsophageal ulcerations and dysphagia were in evidence; whether caustic acids or alkalies or foreign bodies have been swallowed, and whether any other symptoms have attended the dysphagia—*e. g.*, aphonia, boring pain in the sternum or vertebræ, dyspnœa, profuse fetid expectoration of purulent material and food. All these data are essential for differential diagnosis. A spontaneous onset of the symptoms suggests a carcinomatous or syphilitic stricture or a diverticulum.

The physical examination is made with the œsophageal

¹ *Diverticula* are divided into the pulsion and traction varieties; the former result from increased pressure within the œsophagus upon a weakened area of its wall, and are located mostly in the cervical portion of the tube; the latter are due to traction upon the wall of the œsophagus by adhesions, bands, etc., and are located in any portion of the organ.

bougie, the œsophagoscope, the *x*-ray, and by percussion and auscultation over the posterior mediastinum, including auscultation of the œsophagus during deglutition.

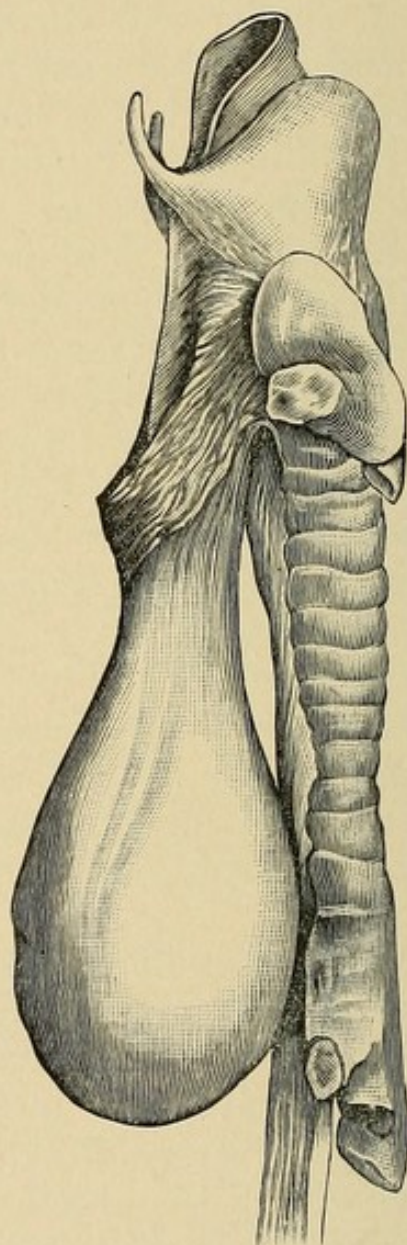
The use of the œsophageal bougie will usually determine the patency or non-patency of the œsophageal lumen. The larger-sized bougies are first to be employed, and if their passage is obstructed smaller and smaller ones are to be used until one is passed or the non-patency of the canal for bougies is established. Arrest of the instrument is usually due to stenosis, but it is not to be forgotten that diverticula may likewise interfere with its passage. In this case, however, a change in the patient's position, or throwing his head far back or to one side, may enable the obstruction to be overcome and the sound to be passed into the stomach. It is to be further noted that diverticula may arrest the passage of small bougies, whereas larger ones easily pass into the stomach. One trial with the bougie is therefore not sufficient. Repeated attempts with large and small-sized instruments should be made, always gently, and with the patient's head in various positions.

If an obstruction of the lumen and its site has been established, the next concern is to determine its *nature*. For this the anamnesis and the age of the patient furnish valuable data, and they are to be supplemented by those which are afforded by œsophagoscopy, *x*-ray, and general physical examination.

Thus a history of having swallowed a foreign body will point to partial or complete obturation by such foreign material; a history of having swallowed carbolic acid, or caustic lye, or other corrosive, will indicate benign cicatricial stenosis; a previous history of syphilis or other evidences of this disease will, in the absence of another cause for the symptoms, suggest a syphilitic ulceration with cicatricial stenosis; whereas advanced years (above forty) with rapidly increasing stenosis, vomiting of blood, and cachexia and emaciation out of proportion to the degree of stenosis, will point strongly to malignant neoplasm of the organ. It is infrequent to find fragments of the neoplasm in the vomitus, but when they are present they substantiate the diagnosis. A very gradual onset of symptoms, with a profuse expecto-

ration of mucus, a feeling of fulness, with colicky pains in the neck relieved by vomiting, a difficulty in swallowing the first parts of a meal, while the rest of it passes down easily, and finally a pronounced dysphagia and regurgitation

FIG. 107



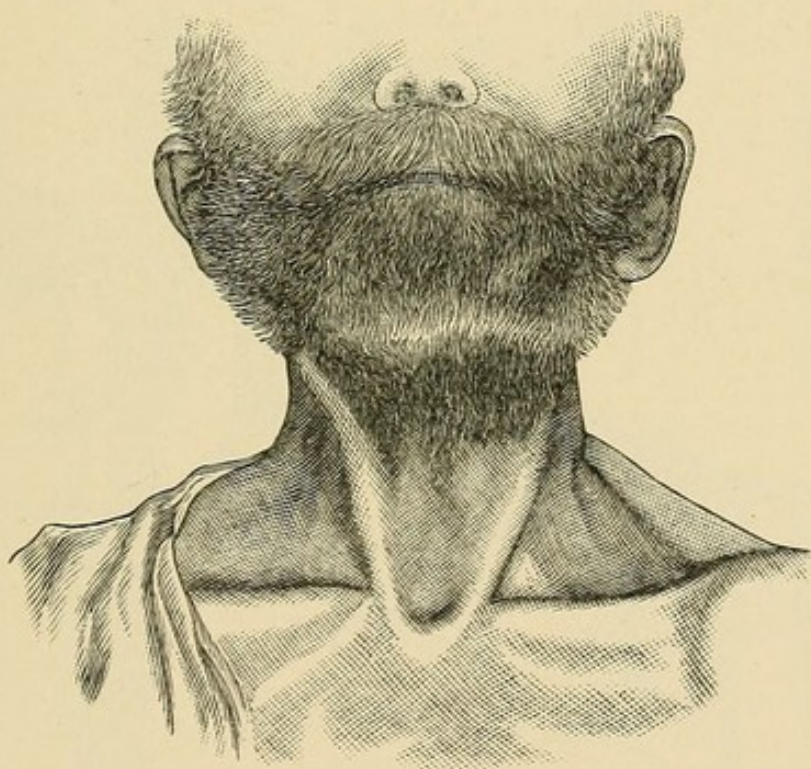
Pulsion diverticula of the œsophagus. (König.)

of large amounts of unmodified ingesta, some of which may have been taken several days before, suggest a pulsion diverticulum; the diagnosis is confirmed by the appearance of a tumor in the neck after eating, and its disappearance after regurgitation or expression,

The absence of data pointing to any of the above causes for the stenotic symptoms, and especially if the patient is young and the obstruction is at the cardiac orifice, will permit of the assumption of a cardiospasm. Should the individual show evidences of hysteria and the site of the stenosis change from time to time, an hysterical spasm may be thought of, but the diagnosis in either case must be verified by the œsophagoscope.

If the physical examination reveals a solid or cystic tumor in the neck, and the stenosis is in the cervical portion of the

FIG. 108



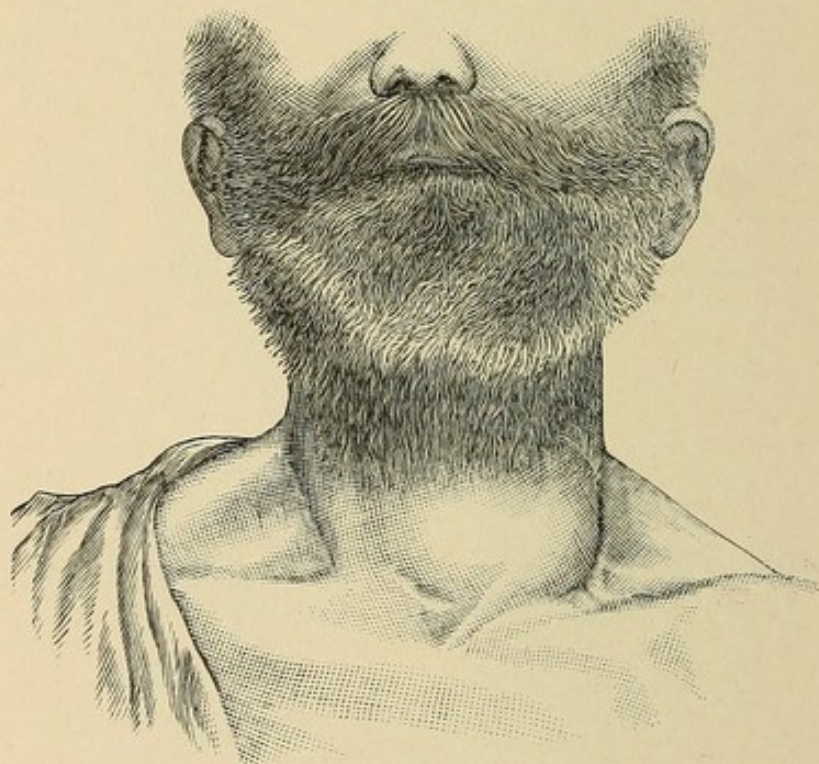
Pulsion diverticulum, empty. (Observed in Billroth's clinic.) (Von Bergmann.)

tube, a compression of the œsophagus by the neoplasm may be inferred; similarly, intense pain in the vertebræ, with dullness in the mediastinum, systolic murmur, thrill and retardation of the pulse at the wrist, suggest an aortic aneurysm with œsophageal compression. Again, dyspnœa, loud harsh breathing over the root of the lung, dullness in the posterior mediastinum, and absent breathing over one or more pulmonary lobes point to a mediastinal neoplasm compressing the œsophagus.

A careful physical examination, with a close study of the anamnesis, will thus throw much light on the nature of œsophageal disease. Positive conclusions can of course only be formed from seeing and feeling the diseased parts. The latter is only possible in case the lesion is located in the cervical portion of the organ, but the former is at our disposal through use of the œsophagoscope and *x-ray*.

Employment of Œsophagoscope.—The systematic employment of the *œsophagoscope* and *x-ray* has but recently come into practice, and with perfection in the technique

FIG. 109

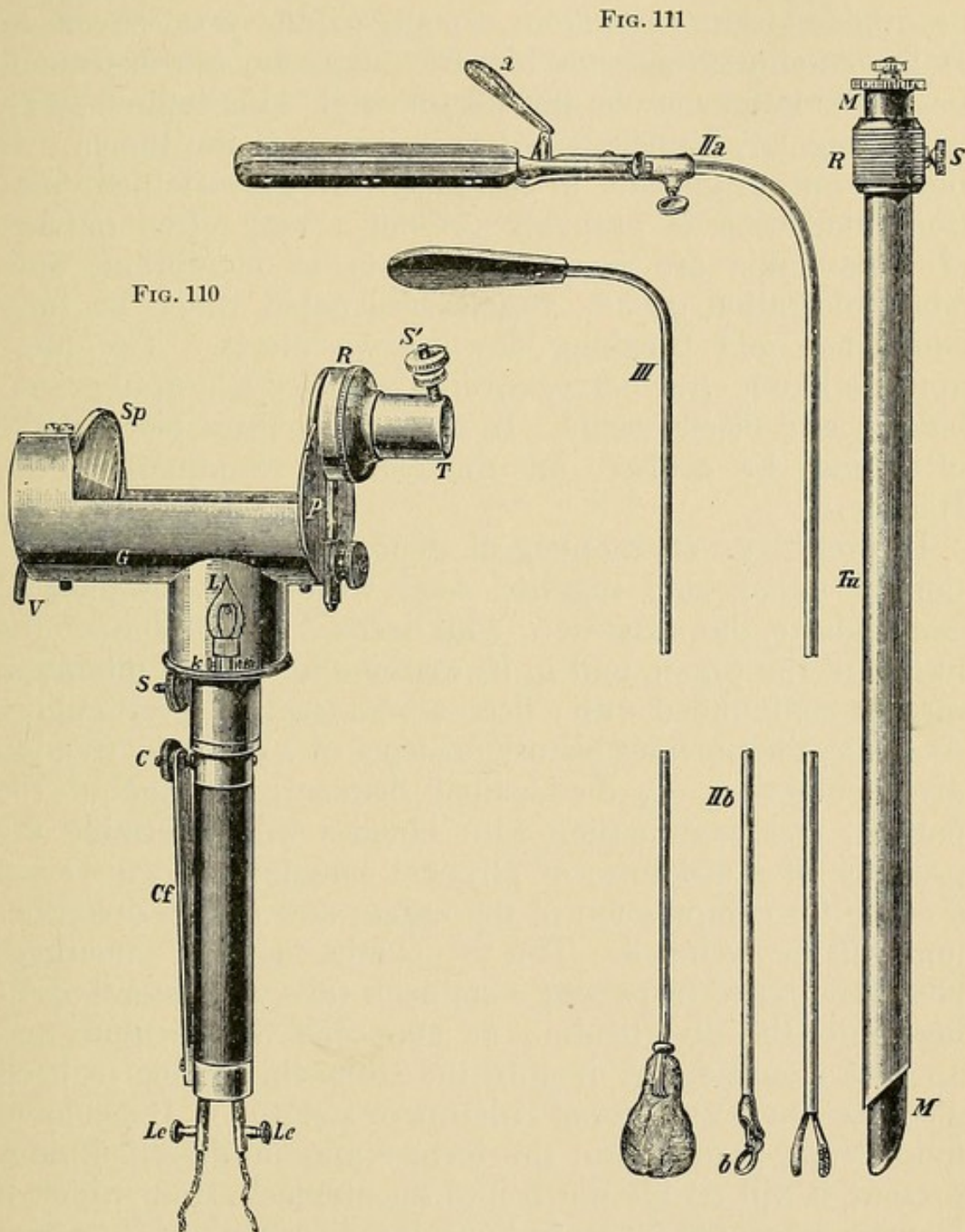


Pulsion diverticulum, filled. (Observed in Billroth's clinic.) (Von Bergmann.)

of their use, and greater skill in the interpretation of the findings afforded by them, earlier and better diagnoses will result. The nature of a stenosis, the orifice of a diverticulum, and the presence of a cardiospasm are easily studied and observed through the œsophagoscope, while the calibre and course of the organ are just as easily to be seen with the fluoroscope after its canal has been coated with bismuth.

The picture afforded by the œsophagoscope in cases of *cicatricial strictures* due to swallowing a corrosive material is as follows: In the cervical and upper thoracic portions

of the canal numerous ribbon-like, longitudinal, speckled white scars are seen. Nearest to the stricture these scars are the most numerous. The entrance to the stricture is



Œsophagoscopes.

shaped like a funnel, and the strictured portion rarely has respiratory mobility (von Haeker).

During the early stages of carcinoma the œsophagoscope

shows an infiltration of the mucosa, which is recognized by its rigidity and respiratory immobility or by a circumscribed protrusion of the affected area, the mucous membrane over this being paler or more cyanotic than that in other parts, or studded with numerous, small, papillary excrescences. If the carcinoma extends more beneath the mucous membrane the latter may be thrown into rigid longitudinal folds, with irregular, funnel-shaped appearance of the lumen. If bloody mucus appears in the lumen of the strictured portion, ulceration is probably present. Very often patches of leukoplakia are seen on the mucous membrane, and, when ulceration occurs, vesicles are noted which by their confluence and breaking down leave ulcers. The ulcer appears like a strawberry, covered with reddish-gray granulations, and bleeds easily. In cases of doubt a piece of the ulcer may be evulsed for microscopic examination (von Haeker).

In every case of œsophageal stenosis there is some retention of mucus and ingested food within the œsophageal canal above the stricture. This leads to dilatation of the lumen of the organ, and in its extreme form such dilatation may be confounded with a deep-seated traction diverticulum. As a rule, the previous history (absence of a cause for traction diverticulum—*e. g.*, mediastinal disease), the age of the patient, and examination with bougies will determine the presence of a stricture, or physical examination will reveal a cause for compression of the organ, and thus a diverticulum will be excluded. The two conditions may sometimes be differentiated by passing a stomach tube with lateral openings into the diverticulum or supposed diverticulum and another alongside of it into the stomach. (This in itself dismisses the possibility of stricture or stenosis.) If a colored fluid is now poured into the former and no diverticulum is present, it will trickle down into the stomach, from which it may be recovered by aspiration; if a diverticulum is present the fluid finds its way into it, and nothing is recovered from the stomach, or only the overflow from the diverticulum. The skiagraph further helps to differentiate them, while the œsophagoscope makes the diagnosis positive.

A perforation of the œsophagus into the lung or posterior

mediastinum is followed by evidences of abscess in these parts, which gives the signs already described as due to these lesions.

Benign tumors of the œsophagus, especially polypi, cause no disturbances while they are small; when large they produce obturation and give rise to the usual symptoms thereof. Their diagnosis is to be made with the œsophagoscope.



PART IV.

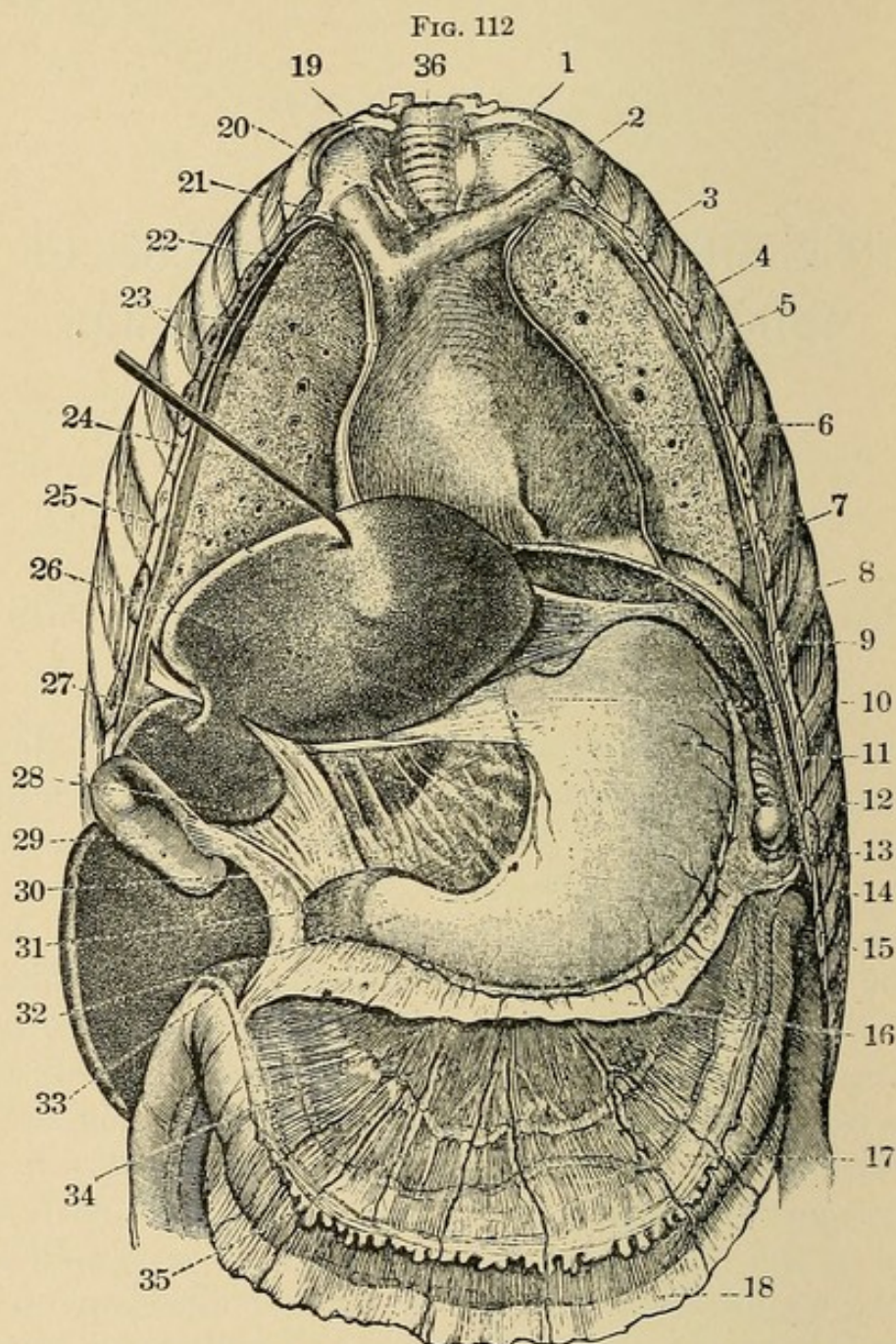
DIAGNOSIS OF INJURIES AND DISEASES OF THE ABDOMINAL WALL AND VISCERA.

CHAPTER XVIII.

GENERAL REMARKS ON ABDOMINAL DIAGNOSIS AND EXAMINATION.

THE introduction of aseptic and antiseptic methods of operating opened up new fields of usefulness to the surgeon, and in none of them has he gained greater glory than in the domain of abdominal diseases. By incessant study, labor, and research he has brought his technical skill in dealing with the abdominal viscera to a high plane of efficiency, but his diagnostic ability has not been similarly advanced, he having need of much improvement in this direction before he achieves the perfection that he has acquired in his technical accomplishments. At the present time he must still resort to laparotomy to aid him in diagnosis during the early stages of some of the abdominal diseases, though he is well aware that such exploratory laparotomy is a confession of diagnostic weakness on his part. It is a weakness, however, that it is neither shameful nor sinful to acknowledge. If one takes into account the close anatomical proximity and similar physiological function of many of the important organs within the abdomen, it is not to be wondered at that the surgeon often finds himself in a quandary as to the exact character of, nay, even as to the organ which is involved in the diseased process. In far-advanced cases no well-trained surgeon will need an exploratory laparotomy

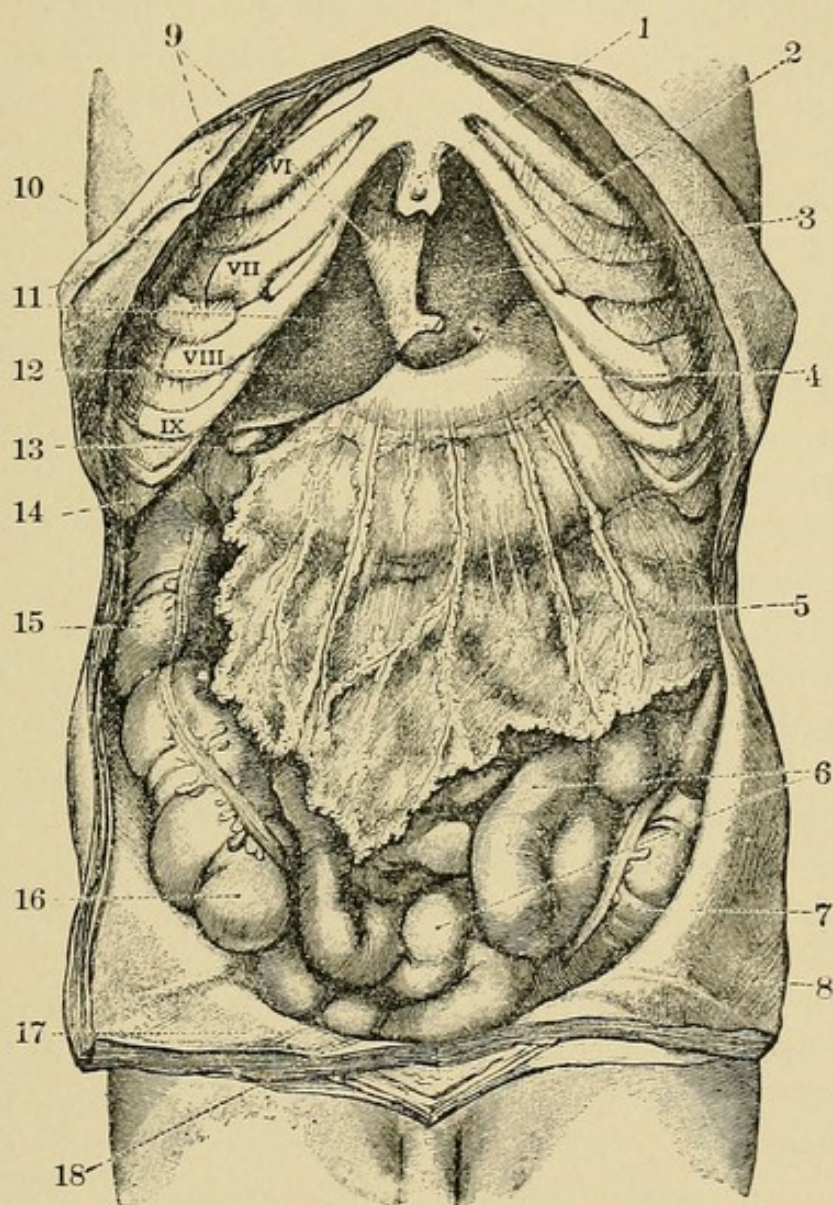
to aid him in establishing a diagnosis. Little benefit, however, is to be derived by the patient from surgical interfer-



Topography of thoracic and upper abdominal organs. (Toldt.) 1. Left common carotid artery. 2. Left innominate vein. 3 and 4. Pulmonary and costal pleura. 5 and 23. Pericardial layer of the pleura. 6. Pericardium. 7. Left lateral ligament of the liver. 8. Diaphragmatic pleura. 9. Fundus of stomach. 10. Cardiac end of stomach. 11. Anterior margin of spleen. 12. Gastrolial ligament. 13. Bed of spleen. 14. Colicophrenic ligament. 15. Splenic flexure of the colon. 16. Great omentum. 17. Transverse colon. 18. Posterior layer of great omentum. 19. Dome of pleura. 20. Innominate artery. 21. Right innominate vein. 22. Superior vena cava. 24. Right lung. 25. Left hepatic lobe. 26 and 27. Gastrohepatic omentum. 28. Hepatoduodenal ligament. 29. Gall-bladder. 30. Hepatocolic ligament. 31. First portion of duodenum. 32. Pylorus. 33. Hepatic flexure of colon. 34. Bursa omentalis. 35. Transverse mesocolon. 36. Trachea.

ence in such late stages of disease; if any good is to be conferred by operative efforts the diagnosis must be made in

FIG. 113



Topography of the abdominal viscera. Note relation of the organs to the costal arch and the abdominal parietes. 1. Xiphoid process. 2. Costal arch. 3. Left lobe of liver. 4. Stomach. 5. Great omentum. 6. Small intestines. 7. Sigmoid flexure of colon. 8 and 10. Parietal peritoneum. 9. Falciform ligament of the liver. 11. Round ligament of the liver. 12. Right lobe of the liver. 13. Fundus of the gall-bladder. 14. Hepatic flexure of the colon. 15. Ascending colon. 16. Cæcum. 17. Lateral umbilical fold. 18. Median umbilical fold.

the early stages of the malady, and at the present day such early diagnosis frequently needs the help of an exploratory incision.

The immunity against infection conferred upon our patients by a strict observance of the aseptic and antiseptic methods of operating has reduced the dangers of such exploratory incisions to a minimum; why then should we deny our patients the great benefits which are to be derived from exploratory incision in making an early correct diagnosis? We do not hesitate to expose a tumor of doubtful character when it is located in the soft parts, why should we hesitate to do likewise in doubtful diseases of the abdomen when the risk is in nowise greater? It is to be hoped that continued exploration will throw the much-needed light upon the early stages of the diseased conditions of the abdominal organs that it has thrown upon the early stages of bone and joint and appendicular disease, and that in the not far distant future we may be able to make early diagnoses without the aid of an abdominal incision. But at the present time the surgeon must, in the early stages of some of the abdominal diseases, resort to diagnostic laparotomy; and the author strongly seconds the opinion expressed by Dr. Wm. H. Mayo in regard to the diagnosis of the diseased conditions occurring in the right hypochondriac region—viz., “that the essential point to determine in a given case is whether it should be treated medically or surgically, and not so much the exact character or starting point of the diseased process.”

Though I have thus pleaded for early exploration in doubtful diseases of the abdominal viscera, I do not by any means wish it to be inferred that a diagnosis cannot be made in any other way. On the contrary, the rule is that a careful history and a painstaking physical examination will lead to a correct diagnosis.

The method of taking the patient's history has already been dwelt upon, and in the following pages the method of making a physical examination of the abdomen will be briefly described.

Examination of the abdomen is made by inspection, palpation, percussion, and auscultation, and in some instances by exploratory puncture.

Inspection.—For inspection the patient should be in the upright, dorsal recumbent, lateral prone or knee-elbow

position, and note should be taken of the following points:

1. The presence of *scars* or *striæ* in the skin due to long-continued distention—*e. g.*, by pregnancy, ascites, etc.

2. The *movement of the abdominal wall* with respiration; respiratory immobility points to a local or to a general painful peritoneal inflammation.

3. The presence of *fat accumulation*; when this is excessive it is especially abundant on the summit of the abdomen, but the umbilicus is not thereby elevated.

4. The presence of *œdema of the abdominal walls*. When present it is especially apparent in the most dependent portions—*viz.*, in the flanks and groins when the patient is recumbent; it pits on pressure and it shifts with a change of the patient's position. It is to be noted, however, that the umbilicus remains retracted. If there is a coincident œdema of other regions of the skin, a general circulatory disturbance is to be suspected. If it is confined to one area of the abdominal wall, a local inflammation of the latter or of an underlying intraperitoneal viscus is likely to be present.

5. The presence of *intestinal distention*. This may be local or general, and if there is also an increased peristalsis it speaks for intestinal obstruction. A painful distention of one coil of intestine alternating with its collapse (intestinal erection) is a pathognomonic sign of a chronic intestinal stenosis just below the erected coil.

6. The presence of *ascites*. When there is an accumulation of considerable amounts of fluid in the peritoneal cavity the flanks are ballooned out, the abdomen is barrel-shaped, and the *umbilicus protrudes*. (See Fig. 124.)

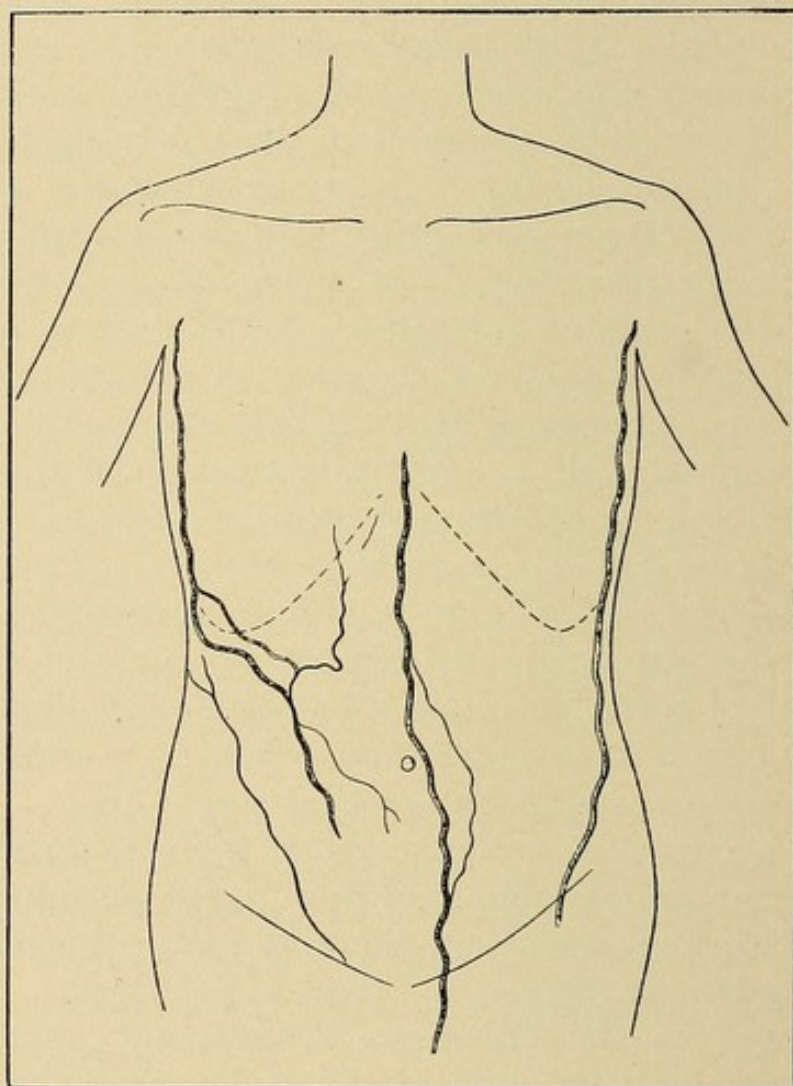
7. Changes in the *cutaneous circulation*. A distention of the veins along the side of the abdomen indicates compression or thrombosis of the vena cava inferior. A distention of the veins around the umbilicus (*caput medusæ*) speaks for obstruction by compression or thrombosis of the portal circulation. (See Figs. 114, 115, and 116.)

Visual inspection is aided by (1) *fluoroscopy*, by which the contour and consistency and outlines of tumors and organs may be determined; (2) by *gastrodiaphany*, by which the outlines of the stomach and the presence and limits of the

neoplasm or exudate around the organ are fixed; (3) by *œsophagoscopy*, and (4) by *cystoscopy*.

Palpation.—For palpation the patient is usually placed in the dorsal recumbent position with the limbs slightly drawn up. The knee-elbow position and Trendelenburg's position afford considerable aid in palpating the pelvic organs and pelvic neoplasms. Strong flexion of the thorax

FIG. 114

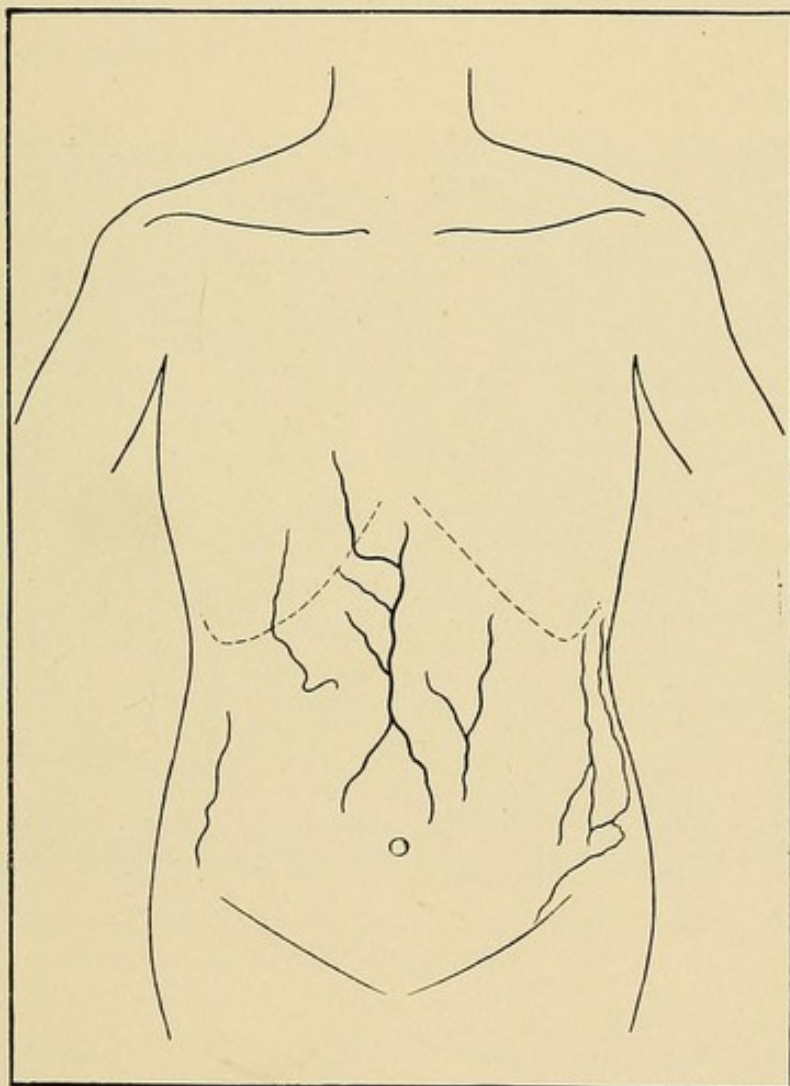


Enlargement of the superficial veins along the side of the abdomen from obstruction of the inferior vena cava.

and lower limbs upon the abdomen (Gerster) often facilitates palpation of the organs and neoplasms in the upper abdominal region. The dorsal recumbent position with moderate elevation of one extended limb (Meltzer) is of service in palpation of the corresponding iliac fossa. The lateral prone position sometimes renders palpation more easy.

Before resorting to palpation the bladder and rectum should be emptied. The greatest barriers to satisfactory abdominal palpation are rigidity and excessive adiposity of the abdominal walls. The former, except when due to peritonitis, can often be overcome by directing the patient to take deep respirations and keep his mouth open. Mus-

FIG. 115



Eulargement of the superficial veins around umbilicus from obstruction of the portal vein.

cular relaxation can sometimes be brought about by placing the patient in a warm bath, and, where everything else fails, by the administration of an anæsthetic.

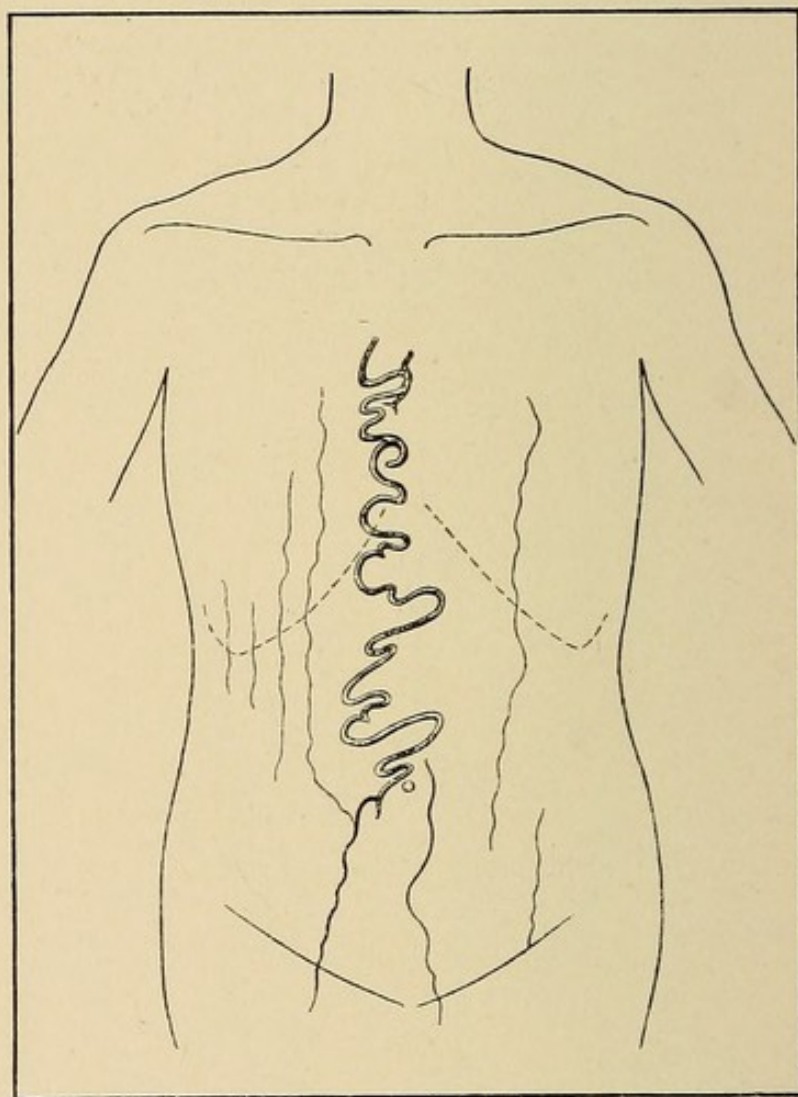
By palpation note is to be taken of:

1. *Irregularities* in the abdominal wall. If these are due to fat, the masses are elastic, soft, and lobulated, and

generally distributed. If they are due to new-growths, they are local, and become more prominent on contraction of the abdominal muscles (*e. g.*, by straining).

2. *Rigidity* of the abdominal walls. This may be local or general, and indicates a circumscribed or diffuse peritoneal inflammation, or reflex muscular contraction without any organic basis.

FIG. 116



Enlargement of superficial abdominal veins from portal obstruction.

3. The presence of *peritoneal crepitation*. This results from the rubbing upon one another of adjacent fibrin-covered surfaces of peritoneum.

4. The presence of the *fremitus of hydatid disease*. This is rarely obtained; it is due to the impact of the hydatid cyst contents against the cyst wall and is most frequently found in superficial hydatid cysts of the liver.

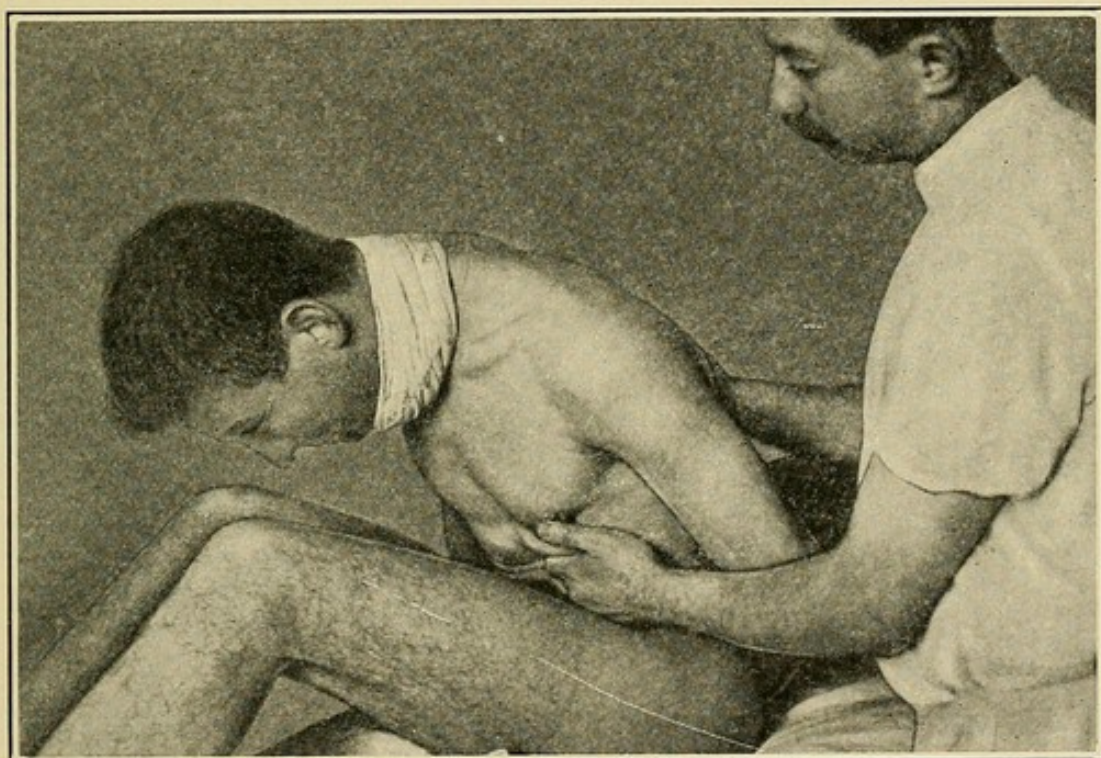
5. The presence of *increased peristaltic waves*. These speak for intestinal obstruction. Of special import is intestinal erection (*i. e.*, a sudden painful distention of one loop of bowel, which is pathognomonic of chronic intestinal stenosis.

6. The presence and consistency of *abdominal tumors*.

7. The *mobility of abdominal tumors or dislocated viscera*.

Mobility may be active, passive, or respiratory in character; it is materially hindered or even entirely suspended by peritoneal adhesions.

FIG. 117



Gerster's position (thorax and lower limbs strongly flexed upon the abdomen) for palpation of epigastric region.

All *intraperitoneal tumors* have some *respiratory mobility*; this is interfered with by paralysis of the diaphragm and by large amounts of fluid in the pleural cavities.

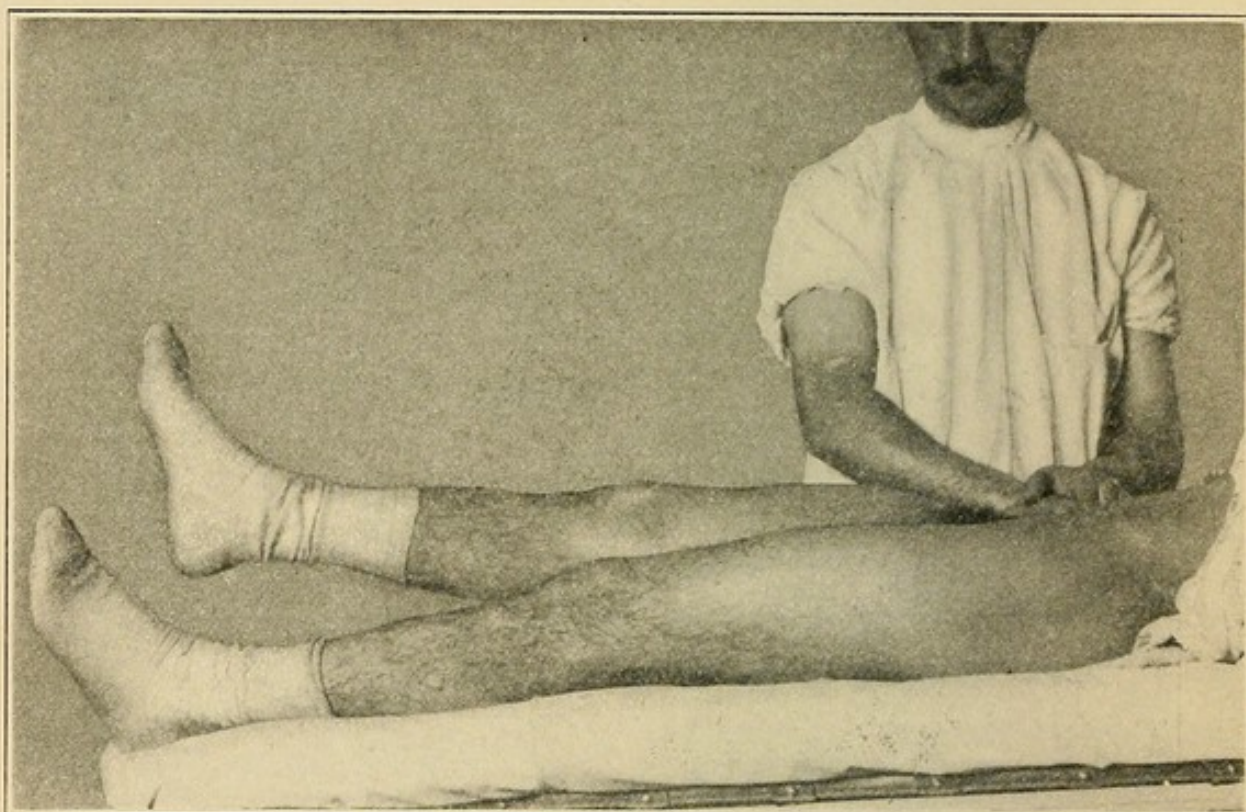
Tumors of the stomach, of the small and large intestines and their mesenteries (except when located at the root of the latter), of the ovary, and uterus, and a wandering spleen have active mobility, the range of which depends on the length of their pedicle and on the mobility of the organs from which such tumors spring. Some of them move in arcs of circles or in ellipses, the centre of which lies in the affected organ. The gall-bladder is very freely movable

when it is connected to the liver by a long mesentery, but when it is closely attached to the liver it has no independent mobility.

Retroperitoneal tumors have no *respiratory mobility*. They may have active mobility—*e. g.*, a floating kidney. Pancreatic swellings and tumors have no mobility.

Intraperitoneal or extraperitoneal adherent exudates have no mobility.

FIG. 118



Meltzer's position (moderate elevation of one extended limb) for palpation of the iliac fossa.

Passive mobility is enjoyed by pedunculated tumors, or dislocated viscera with long mesenteries—*e. g.*, ovarian tumors, wandering spleen, etc.

8. The *relation* of the distended stomach and colon to dislocated viscera or neoplasms. The stomach is best distended by air injected through a stomach tube with a Davidson syringe. The large intestine is best distended through a rectal tube, which is passed about six inches into the bowel and whose outer end is connected with the nozzle of an inverted bottle filled with charged CO_2 water.

Minkowski has shown that after the stomach and colon

are distended, abdominal tumors tend to recede to those parts of the cavity in which are situated the organs from which they arise. A distended stomach pushes the liver and gall-bladder upward under the ribs; the spleen downward and to the left; the transverse colon and omentum downward. The lesser curvature moves upward under the ribs. The kidneys remain unaffected by gastric distention, but are pushed upward and backward by a distended colon.

A distended stomach and large intestine cover over pancreatic tumors with the exception of those which push forward between the stomach and bowel.

Percussion.—By *percussion* we should determine (a) the limits of dulness and flatness of the solid organs, neoplasms, or exudates, and (b) the limits of gastric, intestinal and colonic tympany.

Diminution of liver or splenic dulness from *below* is usually due to a distended stomach or intestine.

Diminution of liver or splenic dulness *above and below* (concentric diminution, H. W. Berg) speaks for free gas in the peritoneal cavity.

Dulness or flatness in the loins, which shifts with a change of the patient's position, speaks for free exudate in the peritoneal cavity. (In percussing the loins for the presence of a free peritoneal exudate we should see to it that the large intestine is empty, lest fluid therein be mistaken for free fluid in the peritoneal cavity.) Irregular areas of dulness or flatness speak for a chronic adhesive peritonitis with or without a sacculated fluid exudate, for new-growths, or for dislocated viscera. It is to be remembered that over collapsed intestine the percussion note is dull.

Auscultation.—By *auscultation* we are enabled to elicit:

(a) *Bruits over the large vessels*; these point to the presence of aneurysms or to a compression of the vessels by new-growths, exudates, or viscera.

(b) *Intestinal bruits* from abnormal peristalsis, and loud noises made by gases and fluids passing through a stenosed intestinal canal.

(c) *Succussion*; this results from an admixture of gas and fluid in a cavity. It is to be heard over the stomach and intestine and over gaseous abscesses.

Probatory Puncture.—Probatory puncture with a long, fine needle is resorted to in the differential diagnosis of liver and splenic abscesses, retroperitoneal exudates, or neoplasms (*e. g.*, of the kidney) or pelvic exudates. In the case of the liver and spleen the puncture should be made through the back or axillary region, or over the convexity of the liver from the anterior aspect. The point of maximum tenderness should be selected as the site at which to introduce the needle. The kidney is aspirated through the loin. The material which is obtained by aspiration should always be examined microscopically and histologically, and cultures should be made therefrom.

CHAPTER XIX.

DISEASES OF THE ABDOMINAL WALL AND INJURIES OF THE ABDOMEN.

TUMORS, SWELLINGS, EXUDATES.

TUMORS, swellings, and exudates in the abdominal wall share its movement in respiration, which in the dorsal recumbent position is forward and backward. Subcutaneous tumors are movable on the aponeurotic layers; intramural tumors become more prominent on forcible contraction of the abdominal muscles—*e. g.*, during straining; and peritoneal tumors become less prominent on such forcible muscular contraction. These general characteristics of abdominal-wall tumors and swellings should be carefully noted and remembered, for they will frequently stand us in good stead in differentiating mural swellings and neoplasms from intraperitoneal ones.

The failure of the recti muscles to unite in the median line is recognized by a hernial protrusion of the abdominal viscera at the site of the deficiency. The hernia is always in the median line, at the umbilicus, in the epigastrium, or above the symphysis pubis. In the latter instance there may be an associated lack of closure of the bladder and symphysis pubis, giving rise to the malformation of ectopia vesicæ. (See also Ventral Hernia, p. 241.)

Painful, discolored, soft, doughy, non-fluctuating swellings, giving no impulse on coughing, unattended with fever, or increased leukocyte count, and coming on after slight or severe trauma, or overexertion of the abdominal muscles, are characteristic of hæmatomata of the abdominal wall. Their occurrence in the course of exhausting diseases or in marasmic states after very slight injuries to or exertions of the abdominal muscles are no uncommon event.

The absence of fever and leukocytosis, their moderate tenderness, and their tendency to become smaller distinguish them from abscesses; while from neoplasms they are to be differentiated by the facts that they appear suddenly and do not steadily increase in size.

The location of herniæ in the median line of the abdomen or at the inguinal or femoral rings; their soft, elastic, or doughy character; their usual reducibility with impulse on coughing, and their lack of pain or tenderness unless inflamed readily distinguish the swellings to which they give rise from hæmatomata of the abdominal wall.

TEARS AND RUPTURES OF MUSCLES.

Tears and ruptures of the abdominal muscles give rise to hæmatomata, which in conjunction with the palpable hiatus between the torn fibres render it easy to make the diagnosis of this condition.

ABSCESSSES OF ABDOMINAL WALL.

Acute.—Acute abscesses form painful, œdematous, hot, tender, fluctuating, circumscribed or diffuse swellings. They are accompanied by a rise of temperature to 103° or 104°, and a leukocytosis after three days of from 20,000 to 30,000. They follow traumata or ulceration and perforation of the intestine, appendix, cæcum, sigmoid flexure, gall-bladder, tubes, ovaries, or urinary bladder, the diseased viscus having become adherent to the abdominal parietes before the actual perforation occurred; or they are secondary to disease of the costal cartilages, symphysis pubis, or iliac bones. Sometimes these mural abscesses are metastatic to suppuration or infection in distant organs, and if no local cause for their presence can be ascertained the previous history should be carefully sifted for such a distant infection or suppuration.

The abscesses may be located superficially beneath the skin or within or behind the muscular layer (mural), or

properitoneally. The most important of the properitoneal abscesses are the prevesical. Where the abscess is deep the pelvic organs, kidneys, abdominal viscera, and bony walls of the abdomen should be carefully examined to ascertain the primary cause of the affection.

Chronic or Latent.—Chronic or latent abscess and tuberculous and actinomycotic abscesses occasion little or no fever, and no leukocytosis. Their differentiation from hæmatomata or neoplasms is not easy, and must be made from the clinical history, the evidence of suppuration or of tuberculosis or actinomycosis in other parts of the abdominal cavity or its walls, and by exploratory puncture. Small yellowish granules, the size of millet-seed, which under the microscope are found to be made up of the ray fungus, are characteristic of actinomycosis. The pus obtained by aspiration should always be examined in smears and cultures.

NEOPLASMS.

Benign tumors are circumscribed, encapsulated, of slow growth, painless, and cause no interference with the general health. Malignant growths are more diffuse, infiltrating, of rapid growth, somewhat painful, and cause deterioration of general health with cachexia.

Neoplasms may be subcutaneous, intramural, or properitoneal in their situation (for differentiation, see remarks at beginning of this chapter).

Benign Tumors.—*Fibromata molluscum* are situated in the cutaneous and subcutaneous tissues; they are soft, small, tender, movable, at times pedunculated, slowly growing tumors.

The *lipomata* are situated chiefly in the flanks and in the median line of the abdomen above the umbilicus. In the latter site they are usually properitoneal in origin, and push forward between the interstices of the linea alba, resembling in many respects epigastric herniæ, from which, however, they are to be distinguished by the absence of gastric pain and digestive disturbances.

The *fibromata* occur most frequently in women, developing in the striæ gravidarum, but they also form in the cicatricial

tissue left in the abdominal wall by hæmatomata and muscular tears. They are located, as a rule, in the median part of the abdominal wall, closely attached to the aponeurotic structures, and form smooth or knotty, very hard, circumscribed, non-tender tumors which have a very slow growth, and cause no disturbance in general health.

The absence of acute pain, temperature elevations and increased leukocytosis, and of any primary inflammation or disease of the abdominal or pelvic viscera, or of the bony margins of the abdomen, distinguish the fibromata from abscesses. *Properitoneal fibromata* may bear a strong resemblance to intraperitoneal tumors of subjacent viscera, omentum, etc. Such fibromata are to be differentiated from hepatic growths by their lack of respiratory mobility, and from splenic tumors by the absence of the characteristic shape and notched border of this organ. Omental growths and intraperitoneal encapsulated exudates which are adherent to the abdominal wall are more difficult of differentiation. Omental tumors of an inflammatory nature are tender, painful, of more rapid growth, and are possibly attended by fever and leukocytosis, while those of a malignant character are of rapid growth, are frequently attended with ascites, and with rapid deterioration of the general health. Inflammatory exudates are of less density than fibromata, and become flattened when the patient assumes the recumbent position.

Mural fibromata are distinguished from retroperitoneal tumors by the fact that they become more prominent when the stomach and colon are distended, whereas the retroperitoneal tumors disappear under these conditions.

Malignant Tumors.—Malignant neoplasms of the abdominal walls may be located in the skin, in which case they are the evidences of a general sarcomatosis or carcinosis or of a melanosarcomatous degeneration of an old nævus. More rarely they occur in the deeper layers as primary sarcoma or carcinoma. At the navel they may be primary, but in the other regions they are usually metastatic or extensions from growths in the subjacent viscera.

Dermoid cysts and echinococcus cysts are occasionally found on the abdominal wall. The former are situated at

the umbilicus. Should they become inflamed and perforate into the umbilical area, there would be a discharge of an oily, cheesy substance, and occasionally hair. Echinococcus cyst follows echinococcus disease of the subjacent viscera.

Growths involving the umbilicus may be:

1. *Navel granulomata*; these are seen in the first few weeks of infancy. They lack an orifice, and hence cannot be mistaken for Dotter's tract.

2. *Papillary fibromata*.

3. *Primary sarcomata or carcinomata*; these form hard, painful, diffuse tumors which undergo rapid ulceration.

CONTUSIONS OF THE ABDOMEN.

The abdominal wall alone, or an underlying viscus alone, or both together, may be ruptured by a traumatism. If the injury is limited to the abdominal wall, rupture of one or more of its constituent layers may result. If an underlying viscus is ruptured, the immediate symptoms are due to shock, hemorrhage from the ruptured organ, and peritoneal irritation from the extravasation of blood or septic intestinal contents; the later symptoms are those of acute diffuse peritonitis, the severity of which depends on the character of the extravasated material. (See Perforations into the Peritoneal Cavity, p. 274.) Immediately after the injury the patient is in a shocked condition, with pallid, cold skin; rapid, feeble pulse; slow, shallow respiration, and subnormal temperature. If there is a profuse hemorrhage into the peritoneal cavity—*e. g.*, from rupture of liver, spleen, or bloodvessels of large size, or extensive extravasation of septic matter—stimulation and the application of heat do not succeed in reviving the patient from this shocked condition; the pulse becomes progressively more rapid and feeble, and death ensues. With slow hemorrhage or gradual extravasation of septic material, the patient usually reacts from shock and within the next few hours manifests the following symptoms: The pulse rate gradually but continuously rises in rapidity (to 90, to 100, to 110, to 120); the temperature rises to normal;

vomiting may or may not occur. The abdominal wall becomes rigid, and does not move with respiration. There is pain and tenderness at the site of the contusion; possibly a local area of dulness corresponding to extravasated blood or intestinal contents. If the condition is unrelieved, death from continued hemorrhage may occur, or diffuse or circumscribed peritonitis develop. If no infected material is extravasated into the peritoneal cavity, or if the hemorrhage ceases and no secondary infection of the blood clot takes place, an aseptic peritonitis develops from which recovery usually occurs. (For symptoms of peritonitis, see p. 248.)

Visceral involvement with an abdominal contusion is recognized during the first few hours after the injury from abdominal rigidity and increasing pulse rate. The presence of these two signs should always prompt immediate operative exploration. If a hollow viscus has been ruptured, the concentric obscuration of liver and splenic dulness is important evidence. When a diffuse or circumscribed peritonitis has developed the diagnosis becomes very simple. In connection with abdominal injuries we should remember that even without visceral lesions death from reflex shock may result.

OPEN WOUNDS OF THE ABDOMEN.

Non-penetrating wounds of the abdominal walls have no especial significance. Penetrating wounds may or may not involve the abdominal viscera. In all cases, just as with contusions, there is shock and hemorrhage of varying degree. Penetrating wounds without visceral involvement may give rise to local or diffuse peritonitis. With visceral involvement the early and late symptoms resemble those of contusions with visceral rupture. The diagnosis of visceral complication in the early stages is made from the abdominal rigidity and the increasing pulse rate, with or without concentric obliteration of liver and splenic dulness. In the late stages the presence of a diffuse peritonitis makes the diagnosis easy.

CHAPTER XX.

HERNIA.

GENERALLY speaking, a hernia may be defined as a swelling which appears on assuming the erect position, grows larger on straining, gives an impulse on coughing,

FIG. 119



Oblique inguinal hernia.

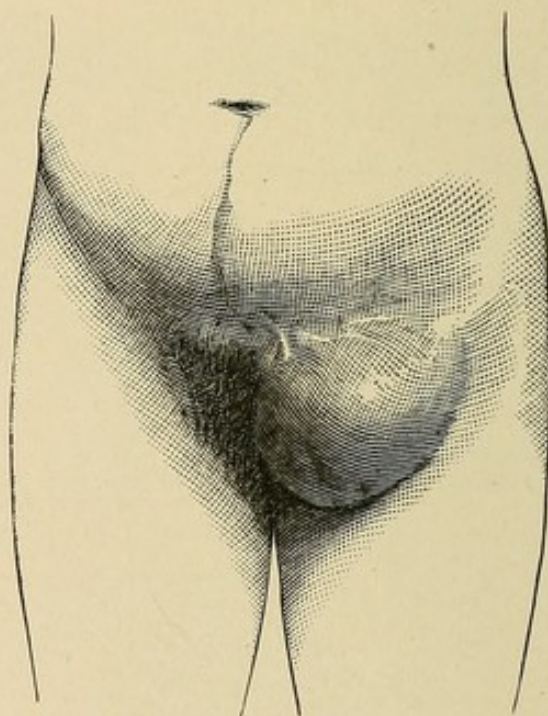
and is reducible. These general characteristics enable one to recognize an uncomplicated hernial protrusion, but one should never rest satisfied with this; he should endeavor to

ascertain the definite variety of the hernia, the nature of its contents, and the presence of any complications.

VARIETIES OF HERNIÆ.

In *inguinal hernia* the hernial orifice is *above* Poupart's ligament, the swelling is internal to and covers the pubic spine, the canal is oblique or straight, and the neck in the oblique variety is just external to the deep epigastric vessels and internal to them in the direct variety. The latter herniæ

FIG. 120



Femoral hernia. (Von Bergmann.)

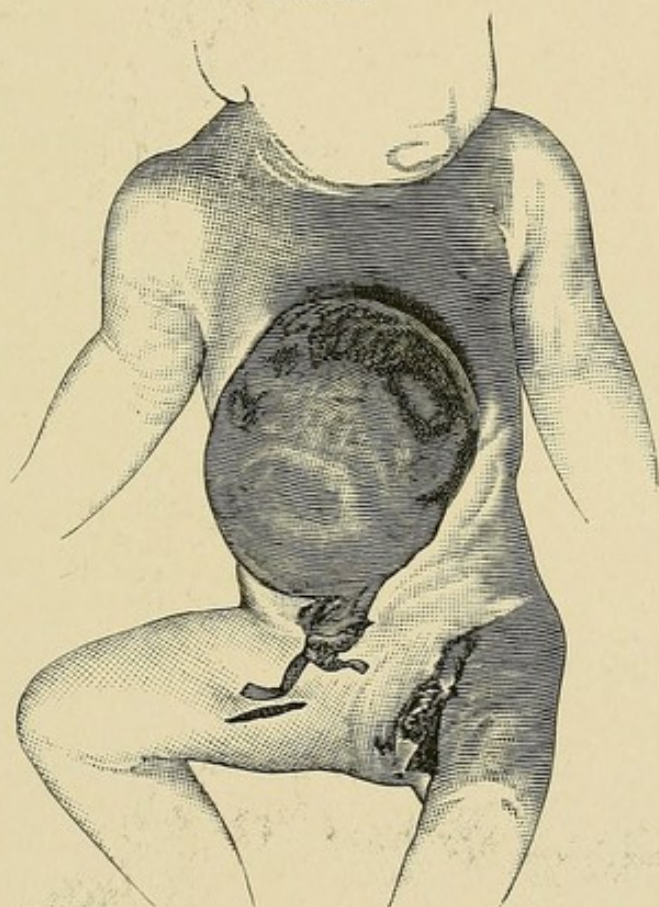
lie internal to the spermatic cord, are usually small, of rounded shape, and rarely become scrotal.

Properitoneal and *superficial inguinal herniæ* are usually associated with an undescended testicle of the corresponding side. In the former the testicle is just outside the internal inguinal ring, and in the latter just outside of the external ring. In either case the corresponding testicle is absent from the scrotum. A properitoneal hernia forms an elongated, rounded tumor, lying just above Poupart's ligament, external to the pubic spine. It does not occupy the inguinal canal

except when the sac is bilocular, in which case one division thereof extends into it. In the bilocular variety the hernial contents often reduce from the lower into the upper division of the sac. The bilocular condition should always be suspected in cases of strangulated properitoneal hernia when the symptoms of strangulation persist after apparently successful reduction by taxis.

A superficial inguinal hernia lies just beneath the skin of the inguinal region. When it becomes strangulated it

FIG. 121



Hernia of cord. (Von Bergmann.)

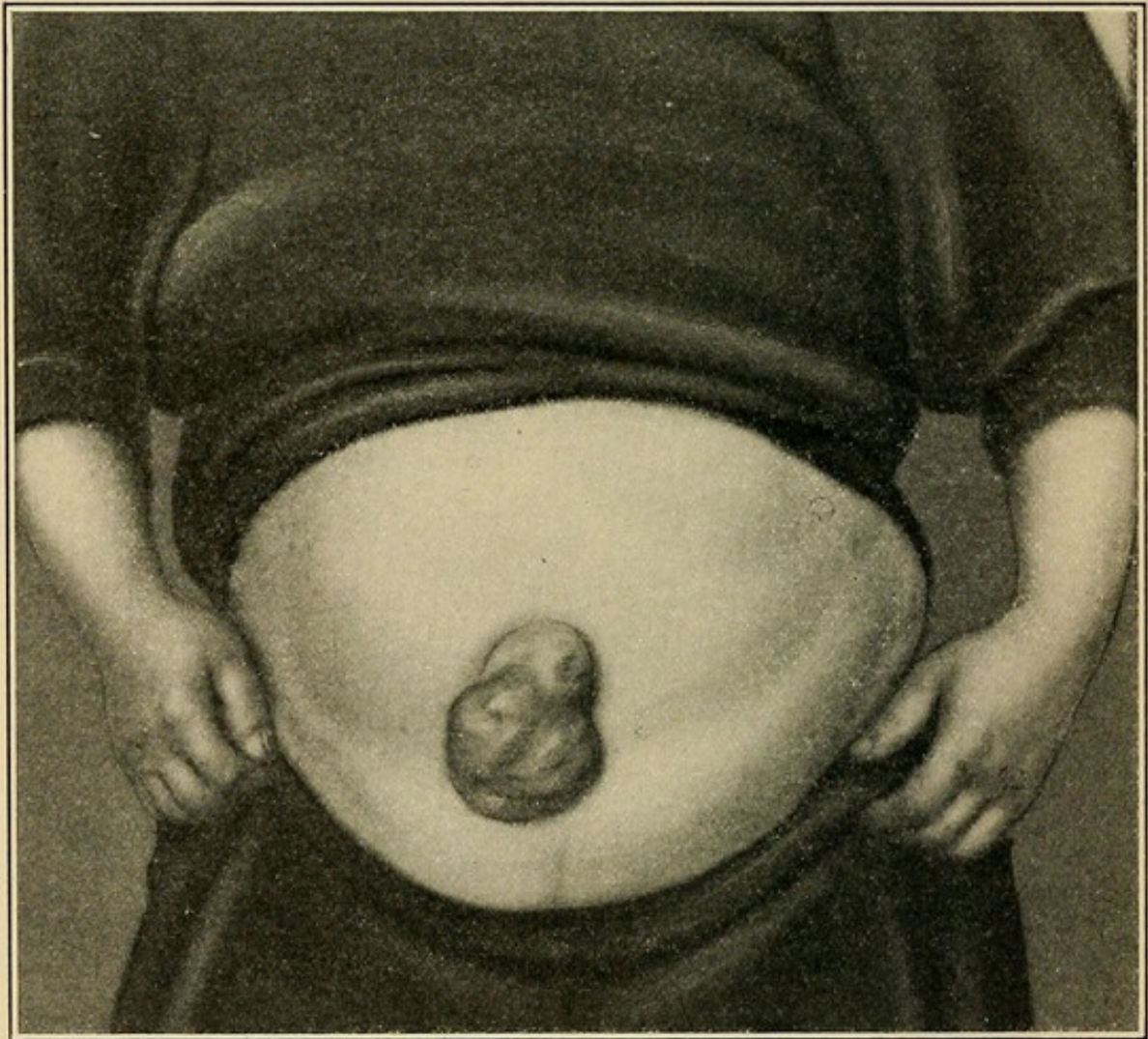
must be carefully differentiated from inflamed ectopic testicle. (See p. 243.)

In *femoral herniæ* the orifice lies below Poupart's ligament, external to the pubic spine, and the neck lies internal to the femoral vessels.

Obturator herniæ appear beneath the horizontal ramus of the pubis, internal to the femoral vessels, and are covered by the pectineus and adductor longus muscles. They form

small tumors which press upon the obturator nerves, causing thereby pain along the inner side of the thigh, in the hip and knee-joints, and paresis of the adductor muscles. When their existence is suspected an attempt should be made to palpate through the rectum or vagina, the internal hernial orifice at the obturator foramen. By flexing and adducting

FIG. 122



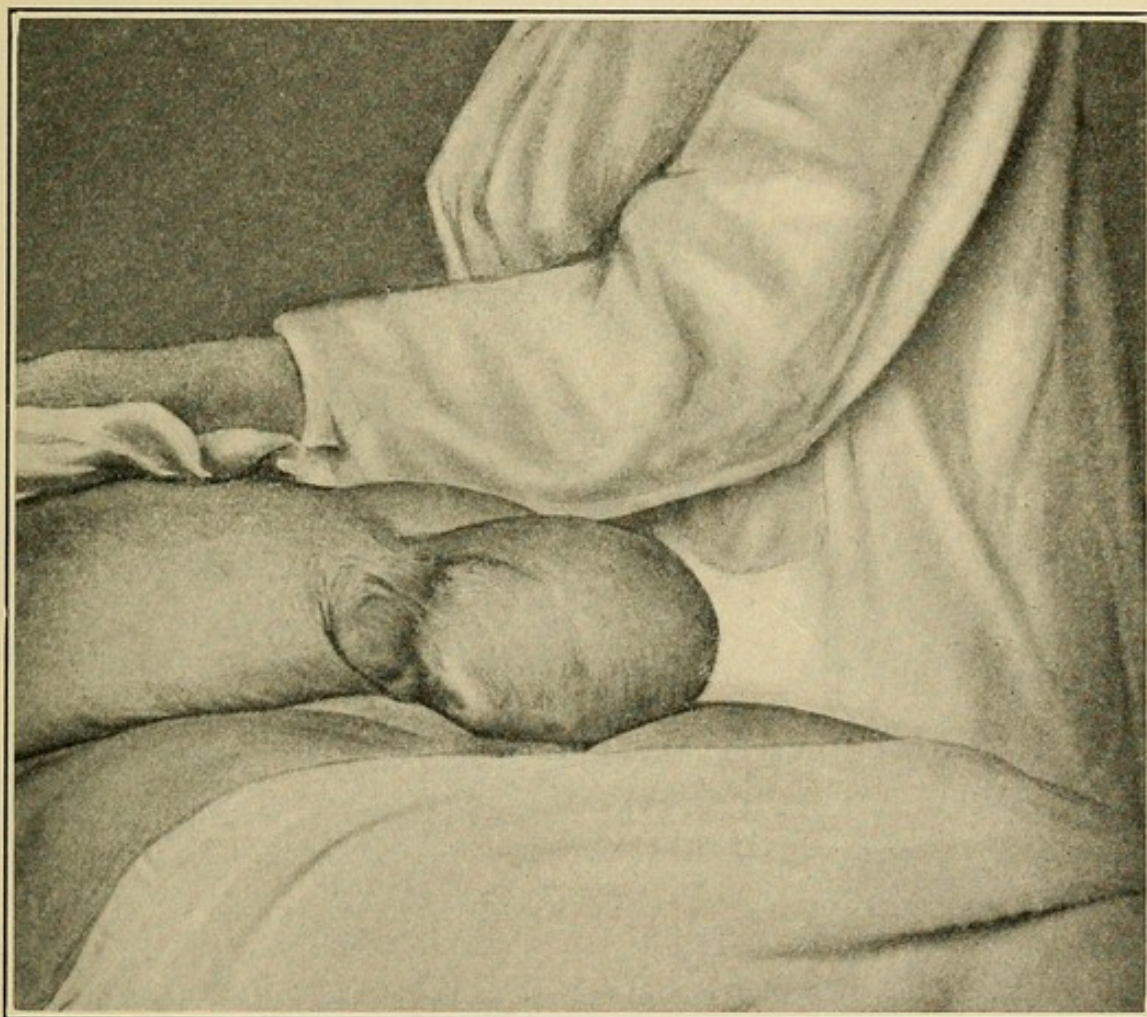
Large irreducible umbilical hernia.

the limb, one is sometimes enabled to palpate the hernia beneath the adductor longus muscle, or appreciate the bulging of the hernial tumor with the naked eye. The diagnosis is especially important when the hernia becomes strangulated, and one should never forget to look for an obturator hernia when there are symptoms of acute intestinal obstruction that are not to be accounted for by some other

cause. Pelvic exudates and neuritis of the obturator nerve may simulate this form of hernia. Vaginal examination in the former and the improvement of the symptoms by treatment in the latter should establish the correct diagnosis.

Herniæ of the umbilical cord are noticed at the birth of the child. These infants usually die shortly after birth, from sepsis due to the sloughing of the hernial sac and its

FIG. 123



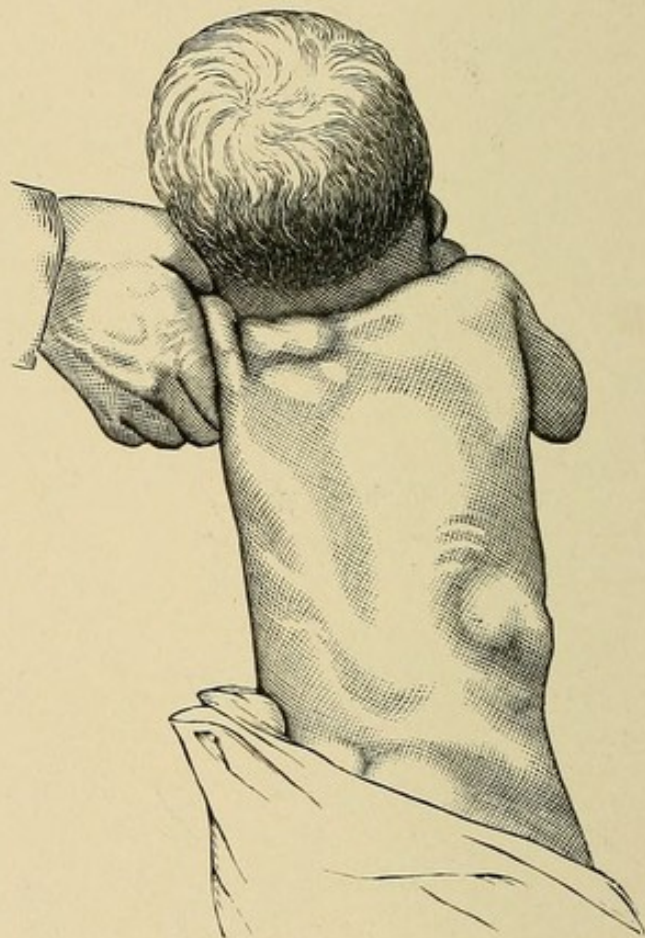
Large irreducible ventral hernia.

contents. Occasionally when the hernia is small and contains only a single loop of intestine, the child survives the sloughing and retains an intestinal fistula at the umbilicus, which must not be confounded with a patent omphalomesenteric duct (Dotter's fistula). These herniæ are sometimes very large, as when the liver occupies the hernial sac; their covering is derived from the amnion, over which are spread out the umbilical vessels.

Herniæ at the umbilicus occur in children and in adults. In the former they form small tumors with the navel on their under side. In the latter they usually form large tumors with diverticula going off from the main sac; they frequently become irreducible.

Ventral herniæ develop in anatomically weak places of the abdominal wall—*e. g.*, in the linea alba (owing to a

FIG. 124



Congenital lateral ventral hernia. (Wyss.)

diastasis of the recti muscles), in the triangle of Petit, and in the linea semilunaris.

Diastasis of the recti muscles permits of a protrusion of the abdominal contents during straining. There is no distinct sac and no hernial orifice.

Epigastric herniæ develop in the linea alba of the epigastrium. They are usually small, and associated with preperitoneal lipomata. They occasion continuous or colicky gastric pains and disturbances of gastric digestion, simulat-

ing thereby chronic diseases of the stomach; the differentiation from the latter is to be made by an examination of the gastric contents, by careful observation, and by the presence of the hernial orifice.

Herniæ at the linea semilunaris are usually found in elderly or feeble subjects, and are usually properitoneal.

Herniæ through the triangle of Petit (which is located between the twelfth rib and iliac crest, the external oblique origin and the outer border of the latissimus dorsi) follow trauma or muscular weakness or operations in this region. They should be distinguished from the outward bulging which is due to a paretic condition of the abdominal muscles. These latter are not true herniæ; they have neither hernial orifice, nor sac, nor contents.

Herniæ after laparotomy and trauma are large or small; they are apt to be irreducible and do not often become strangulated.

CONTENTS OF HERNIA.

The contents of the hernial sac may be intestine, omentum, testicle, ovary, appendix, bladder, or other intraperitoneal viscus, and occasionally foreign bodies derived from these viscera—*e. g.*, vesical stone, round worms, etc.

Intestines are recognized by their elastic feel, their tympanitic percussion note, and by the gurgling sound which is heard on their reduction into the abdominal cavity.

Omentum is recognized by its doughy feel, dull percussion note, and the absence of a gurgling sound on reduction.

The ovary and tube are recognized by their swelling at the menstrual periods and by the peculiar ovarian sensation which is felt by the patient when the organ is squeezed. By vaginal examination the broad ligament and appendages of the corresponding side can be traced into the hernial ring.

The testicle is recognized by its shape, and the peculiar testicular sensation which is experienced when the organ is squeezed. The absence of this organ from the scrotum is additional evidence.

The bladder is suspected when there is vesical tenesmus and irritation; the suspicion can be verified by cystoscopic

examination, by distention of the bladder (the hernia swelling as the fluid is injected), and by passing a sound into the bladder (if the bladder is in the hernial sac the sound can sometimes be made to enter the hernial canal).

The appendix can be palpated as a long, narrow, cylindrical body.

The liver and spleen are recognized by their shape and consistency.

The stomach is suspected if there is marked gastric pain and vomiting.

These viscera while in the hernial sac may become diseased; thus in the Fallopian tube a pyosalpinx or hæmato-salpinx or extrauterine pregnancy may form; in the ovary, an ovarian abscess or cyst; in the appendix, an acute inflammation. From the physical examination as outlined above the organ which occupies the hernial sac is to be recognized, and from the clinical symptoms the pathological process which is going on in the affected organ is to be diagnosed.

DIAGNOSIS OF COMPLICATIONS OF HERNIA.

Irreducibility is characterized by an inability to replace the hernial contents within the abdomen. If there is no coexisting incarceration or strangulation of the hernial contents there will be no other changes in the hernia. Its impulse on coughing persists.

Inflammation is evidenced by severe, continuous, or colicky pain, by vomiting, and by constipation; the hernia becoming larger in size, usually irreducible, and affording no impulse on coughing; the overlying skin is red or bluish or œdematous; the sac is tense, and there may be peritoneal crepitation over it. The vomiting is especially marked if there is peritonitis; the constipation is never absolute, for even with peritoneal involvement foul-smelling gases may be passed per rectum.

Inflammation results from trauma to or strangulation or disease of the hernial contents, and as a result thereof perforation of the prolapsed viscus may result. The latter is attended by sudden severe pain in the hernia and possibly

by collapse, by gaseous crackling, and in some instances by abscess formation in the sac. When the latter occurs the hernia becomes hard and very tender.

Obstruction results from stasis of the material contained within the prolapsed intestine. It is especially apt to occur in large irreducible herniæ and in old subjects. There is in simple obstruction no compression or strangulation of the bowel. It is evidenced by constipation, nausea, sometimes by vomiting and by severe colicky pain. The constipation is never absolute, as foul-smelling gases are passed by rectum. This condition is distinguished from *strangulation* by its gradual onset, by the absence of tension within the sac, by the presence of soft, doughy, indentable (fecal) masses in the prolapsed bowel, and by the persistence of the impulse on coughing or straining. But such cases should always be carefully observed, and until the bowels move satisfactorily the possibility of a strangulation being present should never be dismissed. One successful enema is no proof of the patency of the lumen of the bowel; repeated successful enemata point to the absence of strangulation.

Torsion of the contents gives the same signs as inflammation or strangulation, though usually of a less intense grade. It should be suspected in cases of long-standing omental hernia in which there is a mass in the abdomen that reaches down into the hernial sac.

Strangulation occurring in a previously reducible hernia results in its becoming at once irreducible. The hernia, whether previously reducible or irreducible, increases in size, becomes tender and tense, and loses its impulse. The patient vomits, at first the contents of the stomach, and later on the contents of the upper and lower bowel (biliary and fecal material), the abdomen becomes distended, the urine diminished in amount, and the bowels absolutely constipated. One or two evacuations from the bowel below the site of strangulation may take place after the occurrence of this complication, and in those cases in which only a portion of the wall of the intestine is strangulated, gases and bloody, diarrhœal mucus with fecal particles may be passed by the rectum. There is severe pain in the hernia and the patient is more or less prostrated. If the

affected loop of intestine perforates, there is a sudden tearing pain in the hernia with diminution of its tension, and temporary relief of the previous pain, but the patient becomes more or less deeply collapsed. Gangrene of the strangulated hernial contents is attended by temporary relief of pain and tension, but is followed by peritonitis.

Obstructed, inflamed, and strangulated herniæ may be confounded with an inflamed, undescended testicle, or with inflamed inguinal and femoral glands. With an inflamed, undescended testicle this organ is not in the scrotum; the constipation is not absolute, and the vomiting is not incessant nor fecal. With inflamed glands there is no previous history of hernia, there is a primary focus of infection on the leg or thigh or genitals, the constipation is not absolute, nor is the vomiting so marked. In all of these cases careful observation and repeated examinations are necessary, and should doubt exist it is better to operate than risk the dangers of an unrelieved strangulation.

DIFFERENTIAL DIAGNOSIS OF THE SPECIAL HERNIÆ.

Inguinal Herniæ. Congenital.—Congenital inguinal herniæ date from early infancy. They are scrotal from the very beginning, have an elongated, cylindrical form, are closely adherent to the spermatic cord, and are frequently associated with an undescended testicle and hydrocele of the cord and tunica vaginalis. If the testicle is in the scrotum the hernia lies in front of it. In the funicular variety the testis lies below the hernia.

Acquired.—Acquired inguinal hernia may be entirely within the inguinal canal (bubonocoele), or they may descend into the scrotum, in which case the testis is always below the hernia.

A bubonocoele or incomplete hernia is distinguished from an undescended testicle by the presence of the testicle in the scrotum, and by the absence of the peculiar testicular sensation when the swelling is compressed. An inflamed, undescended testicle is to be especially differentiated from a strangulated hernia.

Hydrocele of the cord and of the tunica vaginalis are distinguished from this variety of hernia by their translucency, dulness to percussion, fluctuation, and slow or entire absence of reducibility. If they are reducible there is no gurgle. A congenital hydrocele reduces into the abdomen and is, as a rule, associated with a hernia. A bilocular hydrocele reduces into the iliac fossa, where it forms a distinct tumor, the tension of which rises when pressure is made upon the scrotal portion.

A hydrocele of the tunica vaginalis always lies in front of the testicle.

A varicocele feels like a mass of earthworms; it gives no impulse on coughing; it does not reduce with a gurgle and when it is reduced it returns even though pressure is made over the external inguinal ring. The swelling enlarges from below upward.

A *lipoma* of the cord is soft and lobulated; it does not reduce with a gurgle, it gives no impulse on coughing, and unless it is very large it causes no pain or dragging sensation.

Femoral herniæ are distinguished from enlarged femoral glands by their reducibility with a gurgle and their impulse on coughing. A varicose internal saphenous vein is differentiated from a femoral hernia by the absence of all the physical signs of a hernia and by its reappearing from below upward, even though pressure is made over the saphenous opening.

Before closing this chapter let me advise all beginners to carefully examine their patients who complain of intestinal colic or of dragging pains in the abdomen, or of gastric pain and vomiting for a hernia. A radical operation for hernia or a well-fitting truss will often put an end to the obscure intestinal or gastric pains, that may have been ascribed to nervous or functional or more serious organic lesions.

CHAPTER XXI.

DISEASES OF THE PERITONEUM. ASCITES. SUB-PHRENIC ABSCESS.

ACUTE INFECTIOUS PERITONITIS.

THE clinical manifestations of acute circumscribed and diffuse infectious peritonitis are so characteristic that even beginners in diagnosis will have no difficulty in recognizing these conditions.

With acute diffuse peritonitis the patient looks sick, anxious, and restless; he lies on his back with his thighs and legs flexed, and in the later stages of the disease he is cyanosed, the features having a pinched expression (the *facies hippocratica*). The sensorium is clear or somewhat excited and elated, the skin is cool and sometimes clammy, the breathing is thoracic and rapid, the pulse rate is rapid and increases in rapidity, the temperature is usually elevated, the bowels are constipated, there is incessant vomiting of gastric and intestinal contents, and a markedly diminished excretion of urine, which contains indican. The leukocytes may be normal or increased to 15,000 or 20,000 or over. The number of leukocytes is, however, of no diagnostic value; high counts seem to be present in the less severely septic cases.

The abdomen is distended and painful; its wall does not move with respiration, and is rigid, hard, and tender to the slightest touch; there is a gradually increasing movable dulness in the flanks. (In percussion of the abdomen for detecting an exudate it is necessary to first of all empty the large bowel, for fluid in a distended colon will often give a shifting dulness in the flanks.)

PERITONEAL SEPTICÆMIA.

With peritoneal septicæmia the general symptoms of toxæmia and vagus paralysis are much more marked than the abdominal ones. The patients are collapsed, the pulse is very rapid and feeble, the respirations rapid, and the temperature not much elevated. The sensorium is clear, and there is a feeling of elation. The leukocyte count is not much higher than the normal.

(For circumscribed and diffuse peritonitis following perforation of a hollow viscus or abscess or infected neoplasm, see p. 274.)

CIRCUMSCRIBED PERITONITIS.

With circumscribed peritonitis the general manifestations are much less severe than in the preceding. The facial expression, color, and type of respiration are not much altered. The pulse rate is either normal or only slightly elevated; the temperature varies from moderate to considerable elevations. With an encapsulated purulent exudate under considerable tension, the temperature will be quite high and the leukocytes increased to 20,000 or more; but when the tension is low even purulent exudates do not occasion a very high temperature.

At the outset there may be general abdominal pain, vomiting, intestinal distention, and constipation. These disappear after twenty-four or forty-eight hours, and the pain becomes localized to the region occupied by the primary focus of the disease. This area becomes tender, the abdominal wall over it is rigid, and the intestine at this site may be distended.

Such clinical pictures will very rarely leave any doubt as to the nature of the disease. But our efforts do not and should not end with having established the presence of a peritonitis. This malady is *always secondary* to injury or disease of some one or other of the intra-abdominal organs or of the abdominal wall, or of a constitutional disease like

pneumonia, etc., and if we would be successful in our treatment of the peritonitis we must first of all ascertain and remove its cause. The primary focus of disease and its character will often be ascertained from the previous history, for the diseased organ has usually manifested its own train of symptoms for some time prior to the onset of the peritonitis—*e. g.*, a diseased appendix, or a gastric or duodenal ulcer, or cholelithiasis, etc. The point of origin of the pain and the site of its greatest intensity will frequently guide us to the diseased organ that has provoked the peritonitis.

At the outset of the peritonitis it is impossible to differentiate the diffuse from the circumscribed forms, or to predict that in a certain case the peritonitis will remain circumscribed. A rising pulse rate, anxious expression, and increasing abdominal rigidity speak for advancing peritonitis and urgently indicate operative interference.

Stone colic may simulate peritonitis. In both there is marked abdominal pain, vomiting, constipation, and distention. In stone colic the pain is most intense at the site of impaction of the stone, and radiates from this point in a direction that is characteristic to the organ in which the calculus is contained. There is no general abdominal rigidity and no pain or tenderness.

Acute enteritis is to be differentiated from peritonitis by the absence of abdominal rigidity, thoracic breathing, and increasing rapidity of the pulse.

Intestinal obstruction differs from peritonitis in that it is not attended with constitutional symptoms during the early stages. Furthermore, in obstruction the pain comes on in attacks; there is at the outset increased peristalsis, and on palpation of the abdomen a fixed loop of bowel can often be detected; the abdominal wall is not rigid and tender, the distention is at first limited to the site of obstruction, and the breathing is not thoracic. In obstruction the constipation is absolute, whereas in peritonitis gas and some fecal matter may be passed. When peritonitis supervenes upon the intestinal obstruction these differences gradually disappear.

CHRONIC ADHESIVE PERITONITIS.

The intraperitoneal adhesions or bands, which result from a chronic adhesive peritonitis, may occasion compression or kinking or strangulation or contraction of the mesentery of the hollow intraperitoneal viscera, with consequent stenosis or complete obstruction of their lumina or strangulation of their nutrient vessels.

The stenotic symptoms (see p. 250) are of gradual onset and development, and are distinguished from those due to a malignant neoplasm by the absence of a tumor and cachexia. In the case of the pylorus, the presence of free hydrochloric acid and ferments in the gastric juice and the absence of lactic acid therefrom are additional data to distinguish a pyloric stenosis produced by adhesions or bands from that due to a cancer.

The onset and course of acute intestinal obstruction or of volvulus due to such adhesions are similar to those which attend these conditions when arising from other causes.

The dependence of these conditions upon peritoneal adhesions can be established prior to operation only if the patient gives a history of previous inflammation of an intraperitoneal organ—*e. g.*, of the appendix, gall-bladder, genital organs in the female, etc.—or a history of an abdominal injury or operation, of coprostasis, or of syphilis.

TUBERCULOUS PERITONITIS.

Tuberculosis of the peritoneum may manifest itself by the symptoms of chronic intestinal stenosis, or by the presence of nodular masses in the abdomen, or by the presence of a free or encapsulated exudate in the peritoneal cavity, or by combinations of these. Patients suffering with this malady usually have some other focus of tuberculous disease—*e. g.*, in the genital organs, lungs, intestines, lymphatic glands, bones, or joints. They become emaciated and weak, have an irregular, moderate fever, and suffer from gastrointestinal disturbances.

The nodular masses are few or many, soft or hard, large or small. Free exudates give rise to shifting dulness in the flanks, distention of the abdomen, protrusion of the umbilicus, and bulging of the flanks. Encapsulated exudates occasion irregular areas of dulness in the abdomen, which change their size and position from day to day; they may resemble intra-abdominal cystic tumors, but they are distinguished from them by their irregularity, their change in size and position from time to time, their multiplicity, their intimate adherence to the abdominal wall, intestines, and omentum, their flattened shape when the patient assumes the recumbent position, and by the presence of tuberculous foci in other organs.

The nodular masses may be mistaken for malignant disease of the peritoneum; but the latter is always secondary to carcinoma of other abdominal organs, occurs in much older subjects, and causes marked cachexia. The free peritoneal exudate may be thought to be due to cirrhosis of the liver, but this condition is found chiefly in alcoholics and in syphilitics; with it the liver is knobby, irregular, and usually small, and there are other evidences of portal obstruction—*e. g.*, hæmatemesis, melæna, hemorrhoids, enlarged spleen, etc.

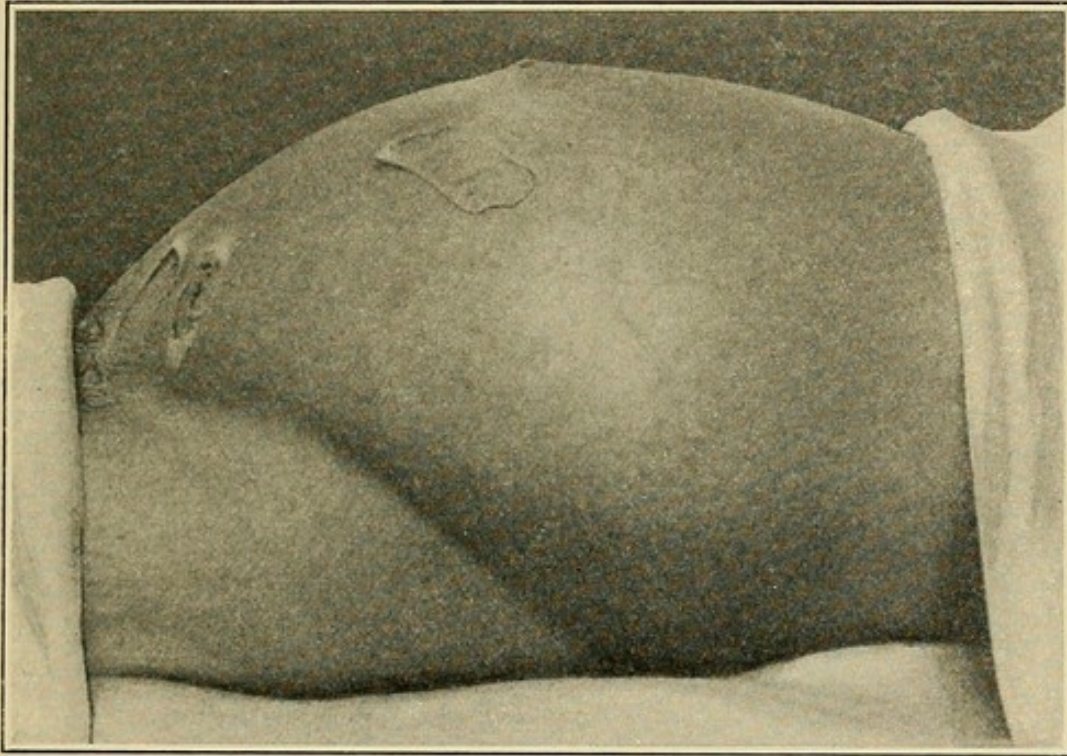
ASCITES.

An accumulation of fluid in the peritoneal cavity is manifested by a protruding umbilicus, a distended condition of the abdomen, which becomes flattened in the middle and ballooned out on the sides when the patient assumes the dorsal recumbent position, which is tympanitic to percussion in the centre and above and flat in the flanks, the latter being replaced by tympanitic resonance when the patient's position is changed to one or the other side (shifting flatness), and which gives the sensation of a fluid wave when it is lightly tapped. The shifting dulness in the flanks is naturally one of the first indications of such a fluid collection, and as this is very strongly simulated by fluid accumulations in the large intestines, we must see to it that the bowel is emptied before we interpret this physical sign as due to fluid within the peritoneal cavity.

Purulent and hemorrhagic fluid exudates are rarely sufficient in amount to distend the abdomen; their presence is usually determined from the shifting dulness in the flanks.

Serous or ascitic exudates due to disturbances in the general circulation from pulmonary, cardiac, or nephritic disease, or to disturbances in the portal circulation from portal thrombosis, liver cirrhosis, or diseases of the spleen,

FIG. 125



Shape of the abdomen in ascites. Note the protruding umbilicus, the bulging flanks, and the barrel shape.

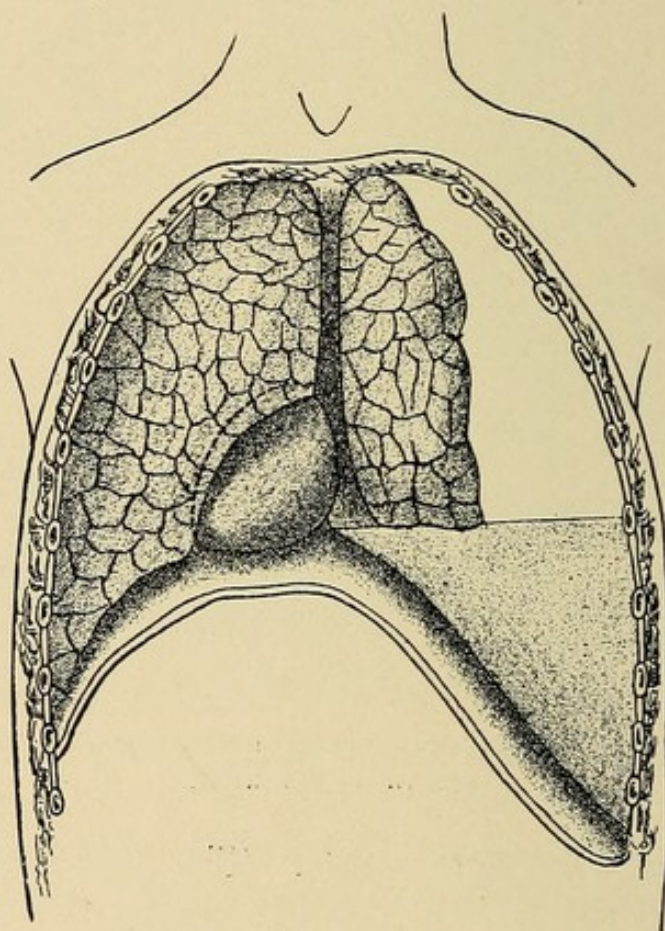
frequently reach large amounts. The fluid is usually clear, of a yellowish color, 1012 specific gravity, alkaline in reaction, coagulates slightly on standing, and is rich in proteids. Microscopically it contains but few formed elements.¹ The admixture of blood points to carcinoma, tuberculosis, or traumatism; a milky admixture, chylous ascites, points to obstruction of the chyle-ducts or to obstruction of the thoracic ducts by tumors, entozoa, or thrombosis.

¹ These characteristics distinguish it from inflammatory exudates and cystic contents. Peritoneal exudates have a specific gravity of 1028, coagulate on standing, and contain many formed elements. *Ovarian cystic fluid* does not coagulate on standing and *echinococcus cystic fluid* is poor in proteids.

SUBPHRENIC ABSCESS.

The presence of pus, or of gas¹ and pus, in the subphrenic spaces, the latter condition constituting the pyopneumothorax subphrenicus of Leyden, is readily determined from the constitutional evidences that always attend pus formation, and from the physical signs which an abscess in these

FIG. 126



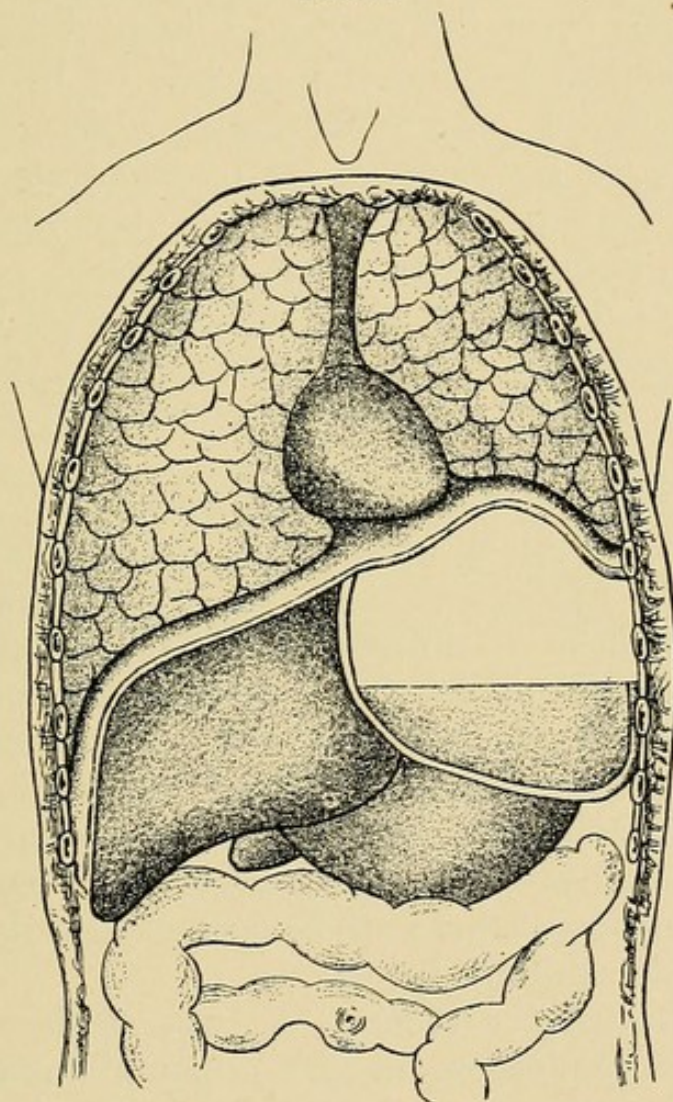
Left pyopneumothorax. Note the displacement of the heart to the right, the compression of the left lung, and the depression of the diaphragm. (Maydl.)

regions affords. Our diagnostic efforts should not, however, end with this determination. These abscesses are almost invariably secondary to suppuration or ulceration within the abdominal or thoracic cavities, and our efforts should be directed to ascertaining the location and character of the primary disease. This latter is not so easy, but if we

¹ The gas is derived from a ruptured hollow viscus or from gas-forming bacteria.

probe carefully into the patient's history prior to the advent of the subphrenic suppuration, study the character of its onset, and examine the pus, both in smears and by culture, we shall in most instances be able to arrive at a correct diagnosis of the primary malady.

FIG. 127



Left pyopneumothorax subphrenicus. Note displacement of heart upward, the upward compression of the lung, and upward displacement of the diaphragm. (Maydl.)

Abscesses in the right subphrenic space are mostly occasioned by diseases of the appendix, liver, gall-bladder, duodenum, and right kidney, and by diffuse purulent peritonitis. The accumulation of pus in this region depresses the liver, raises the diaphragm, and compresses the lower lobe of the right lung. It gives rise to an area of dulness,

with convexity upward at the base of the right chest; above this area there is normal pulmonary resonance and normal vesicular breathing. If there is gas in the abscess cavity its presence is indicated by a characteristic succussion sound on shaking the patient and by a zone of three-layer percussion sound at the base of the right chest—*i. e.*, from above downward there is first normal pulmonary resonance, next tympanitic (over the gas), and lastly dulness over the liver. The elevated, dome-shaped line of liver dulness in front is characteristic of a subphrenic exudate or tumor, and between these we can decide only by exploratory puncture. When the abscess is far back in the subphrenic space, the dome-shaped, basal dulness is not so marked. This dome-shaped area of dulness in a patient who gives a preceding history of intra-abdominal suppuration, or of visceral perforation, should strongly suggest the presence of a subphrenic abscess.

Abscesses in the left subphrenic region are usually due to perforations of the stomach, or to diseases of the spleen, left kidney, and left lobe of the liver, or to sacculation of pus which results from a diffuse purulent peritonitis. They depress the left lobe of the liver, displace the heart upward, and give rise to the characteristic dome-shaped area of basal dulness. If they are gas-containing abscesses, there will be the characteristic succussion on shaking the patient and the three-layer, dome-shaped, basal percussion area. Unless the lung is compressed there will be normal vesicular breathing down to the dome-shaped area.

The constitutional evidences of subphrenic suppuration are: pain and tenderness in the upper abdominal region; fever, either continuous or remittent; disturbed nutrition and weakness, and increased leukocytosis.

A coexisting pleural exudate, a by no means infrequent complication to a subphrenic exudate, and even in some instances the primary cause of the subphrenic abscess, makes the diagnosis of the subphrenic condition much more difficult. The previous history will often aid us in that it affords a cause for the subphrenic suppuration, and in some instances exploratory puncture will throw light upon the diagnosis. Thus if we introduce the needle deeply and trans-

pleurally and aspirate one kind of pus from the subphrenic space, and then on withdrawing the needle aspirate a different kind of pus or serum from the pleural cavity, we may assume that a subphrenic abscess and a pleural effusion are both present. In the remaining doubtful cases the diagnosis will only be made on the operating table.

From a *pleural exudate* a subphrenic abscess is readily differentiated, for with the former there is, as a rule, a history of preceding pulmonary or thoracic disease, the basal dulness is not dome-shaped but is concave upward, and the breathing is altered up to the top of the chest.

True pyopneumothorax is distinguished by a history of preceding pulmonary disease, to which the numerous rales over the affected lung bear testimony. The percussion note over the affected side of the chest is tympanitic above and dull below, and not, as in the gas-containing subphrenic abscess, normal above with a three-layer zone of basal percussion sound. The normal vesicular breathing at the top of the chest in the latter condition is replaced in pyopneumothorax by compressed or distant breathing.

CHAPTER XXII.

DISEASES OF THE STOMACH.

METHODS OF EXAMINATION OF THE STOMACH AND ITS SECRETION.

UNTIL comparatively recent years our diagnosis of gastric affections was made chiefly from the clinical symptoms, with such aid as was to be had from the incomplete methods of physical examination then in vogue. Resting upon the data obtained in this way, it is not to be wondered at that the diagnosis was often entirely wrong, and that little benefit was conferred upon the patient by prolonged methods of treatment. To-day we are considerably better off, inasmuch as the methods for examination of the stomach and its secretion are more exact and accurate; thus we have learned to ascertain the position, size, and motility of the stomach, the composition of its secretion, and with the fluoroscope and gastrodiaaphane we can see the outlines of the organ and in some instances the shadow of a tumor or exudate that lies upon or in its walls. Further, our understanding and appreciation of the clinical evidences of its disease have, thanks to the combined efforts of the internist, surgeon, and pathologist, been considerably advanced.

But though we have made great strides toward the more accurate diagnosis of stomach disorders, considerable further progress is necessary, before we will be able to diagnosticate in their early stages those diseases of the stomach which at their beginning afford very obscure and indefinite symptoms. Especially is this true of ulcer and cancer of the stomach, both of which diseases very frequently pass by unnoticed until some serious life-threatening complication arises—*e. g.*, hemorrhage or perforation, or until it is too late to effect a radical cure. Such early diagnosis is not, however, always

possible from the data which are to be obtained from the clinical history, the physical examination, and the chemical analysis of the gastric secretion; and in the doubtful cases we must have the aid which is afforded by diagnostic laparotomy. The risks attending this procedure are slight and are not to be compared with the immense benefit that may accrue to the patient from an early accurate diagnosis of his malady. A good working rule for its employment is the following: "If the symptoms are progressive, and no adequate constitutional or local cause for them can be found, and no benefit is derived from a carefully instituted internal and local treatment, diagnostic laparotomy is to be considered not only admissible but obligatory."

Turning now to the examination of the stomach, it is important to ascertain its position, its size, its muscular function, the presence of abnormal tumor formation, the chemical composition of its secretion, and the shadow pictures which are afforded by the fluoroscope and gastrodiaephane.

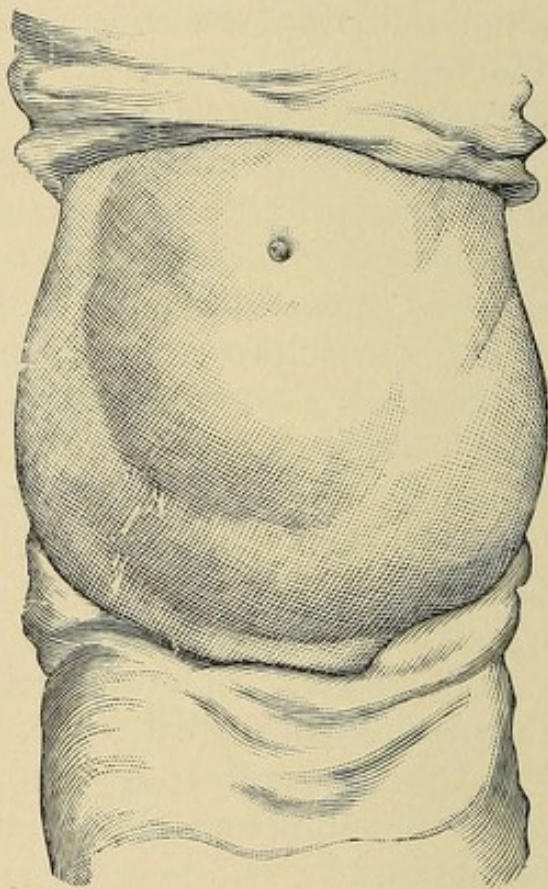
Position.—The position of the organ is determined by percussion and palpation in its empty and distended state, and also by fluoroscopic and gastrodiaephantic examination. The viscus is best distended with a Davidson syringe through a stomach tube passed just beyond the cardiac orifice. Seidlitz powders are less satisfactory for this purpose. A recent hemorrhage, or the suspicion of an ulcer, or perforation, contraindicates distention, which in any instance should not be carried to excessive degrees. A large dose of bismuth subnitrate should be given before fluoroscopic examination.

The normal position of the stomach is a vertical one; its cardiac end lies behind the costal cartilages of the sixth and seventh rib; its pyloric end just to the right of the sternum at the level of the tip of the ensiform cartilage. Its highest and lowest points vary according to its state of distention. In the fully distended state, the highest point corresponds to the top of the fundus and lies in the mammary line behind the fifth rib; and the lowest point several inches above the umbilicus. (See Fig. 112, p. 220.) In an empty condition the stomach is like a hollow tube, the greater curvature being almost parallel with the lesser.

Changes in position are due to gastropptosis, or neoplasms, or adhesions.

Size.—The size of the stomach is variable; normally we expect the lower border in the fully distended state of the viscus to lie several fingers' breadth above the umbilicus, and its upper border to reach to the fifth rib in the mammary line. If the former descends below the umbilicus, with constancy of the upward extension, dilatation may be

FIG. 128



Stomach inflated, showing gastropptosis. (Von Bergmann.)

assumed. Such dilatation is due to pyloric stenosis and to relaxation or acute paralysis of the muscular coat of the organ.

The anatomical relation of growths and exudates around the stomach can be best determined when the stomach and colon are distended. Only the pyloric and lower portions of the body of the stomach are accessible to palpation, and that in the space between the left hepatic lobe and the costal margin. When the stomach is distended the upper

border may, upon deep expiration, be felt to slip upward under the ribs and costal cartilages.

Motor Function.—*The motor function* of the stomach is determined by giving the patient a test meal in the evening and siphoning out the organ on the following morning. If food particles are obtained it points to muscular insufficiency. This is present in acute dilatation of the organ and in the dilatation that accompanies long-standing pyloric stenosis. If large amounts of clear gastric juice (above 100 c.c.) are obtained, it points to hypersecretion, a condition that accompanies gastric ulcer or that results from a nervous disturbance.

Chemical Composition of Gastric Juice.—*The chemical composition of the gastric juice* is determined by an examination of the gastric contents, which are obtained by siphoning out the stomach one hour after a test meal has been taken. The test meal should consist of a cup of black, unsweetened coffee or tea and one piece of toast, or, better, one shredded-wheat biscuit.

The examination is made for:

1. **Acidity**, with blue litmus paper.
2. **Free hydrochloric acid.** A few drops of a weak watery solution of methyl violet are added to 1 c.c. of the gastric juice, and to a similar quantity of distilled water. In the presence of free hydrochloric acid a deep-blue color appears.

Or a few drops of Ginzberg's solution (composed of two parts of phloroglucin, one part of vanillin, in three parts of absolute alcohol) are dried on a porcelain dish over the Bunsen flame. A few drops of filtered gastric juice are added and the dish warmed. In the presence of free hydrochloric acid, a brilliant red color results. In chronic atrophic gastritis and carcinoma there may be an absence of free hydrochloric acid.

3. **Lactic acid.** A few drops of gastric juice are added to a test-tube full of Uffelmann's reagent, which is made as follows: To 10 c.c. of a 1 per cent. solution of carbolic acid are added 1 to 2 drops of liquor ferri sesquichlorati. In the presence of large amounts of lactic acid a canary yellow color appears.

The presence of large amounts of lactic acid points to

gastric stagnation, absence of hydrochloric acid, and diminution of ferments. These conditions are usually found in pyloric carcinoma with dilatation of the stomach.

4. **Peptones.** To a cold, strongly alkalinized solution of gastric juice is slowly added a 1 per cent. solution of copper sulphate. A deep-red color points to presence of peptones (Biuret reaction). Its absence indicates absent free hydrochloric acid and ferments, conditions found in chronic atrophic gastritis and carcinoma.

5. **The total acidity.** Ten c.c. of filtered gastric contents are diluted with two or three times the quantity of water, and a few drops of alcoholic phenolphthalein added. Alkalies turn the latter red; in acid or neutral solutions it remains colorless. A decinormal sodium hydrate solution is added from a graduated burette to the diluted gastric juice with phenolphthalein until a weak red color permanently appears. From the number of cubic centimetres of sodium hydrate used, the amount of total acids is computed. Thus, if for the neutralization 3 to 4 c.c. of decinormal sodium hydrate solution were used, for 100 c.c. of gastric juice, 30 to 40 c.c. would be used, which is put down as the total acidity. This is about the normal.

ULCER OF THE STOMACH.

A diagnosis of ulceration of the stomach can, as a rule, be made from the symptoms to which such a condition gives rise—viz., sharp cutting pain in the epigastrium which shoots into the back and is possibly made worse by eating, eructations and vomiting of intensely acid material soon after taking food, heartburn, anæmia, impoverished nutrition, hyperacidity of the gastric juice, local tenderness in the stomach region, a Head zone, and when the edges of the ulcer are very much thickened, a tumor in the stomach region. The hyperacidity of the gastric juice, the Head zone, and the tumor are very inconstant manifestations and are consequently of little aid in making the diagnosis. In the majority of cases the subjective symptoms alone are present, and as these very closely resemble those which are occasioned

by calculi in the gall-bladder, duodenal ulcer, and epigastric hernia, we often find it very difficult to make the differential diagnosis between these last-named conditions and gastric ulcer.

With *gallstone disease*, however, there is apt to be a history of repeated attacks of biliary colic, some of which have been followed by jaundice, and after some of the attacks of colic stones may have been found in the stools. Furthermore, the pain in gastric ulcer is likely to be worse right after eating, whereas in gallstone disease the pain commences two or three hours after a meal. A history of preceding attacks of inflammation of the gall-bladder or the occurrence at any time of a cholecystitis with fever and distention of the viscus are important data for making the differentiation. We must not forget that gallstone disease and gastric ulcer may coexist, and that the contraction of perigastric adhesions which have originated from a calculous cholecystitis may occasion a benign pyloric stenosis that in every detail clinically resembles the stenosis which follows the cicatrization of a pyloric ulcer. In such cases a history of preceding attacks of cholecystitis gives us a clue to the diagnosis.

Duodenal ulcer is distinguished from pyloric ulcer by the fact that the pain is located more to the right and comes on two or three hours after a meal. The occasional occurrence of jaundice and the radiation of the pain to the right shoulder which occur in some of the cases of duodenal ulcer may lead to an erroneous diagnosis of biliary lithiasis; the differentiation is, however, readily to be made from the absence of attacks of biliary colic and cholecystitis.

An epigastric hernia is readily detected by physical examination, and if in every case presenting gastric symptoms we remember to examine for it, many mistakes in diagnosis will be avoided.

It seems almost impossible that any difficulty should be experienced in differentiating two such radically different conditions as gastric ulcer and gastric cancer; and yet an ulcer with thickened edges that gives rise to a palpable tumor in the epigastrium may readily be mistaken for a carcinoma of the stomach. If we remember the similarity of the gas-

tric symptoms in both conditions, the inconstancy of the changes in the chemical composition of the gastric juice that attend them, and the unreliability of the changes in the blood that are associated with carcinoma and ulcer, we will appreciate the difficulty that arises in their differentiation. This difficulty becomes all the greater if we also remember that carcinoma only too often develops on the base of an old ulcer. The only safe plan of procedure in such cases is to explore by laparotomy every palpable tumor of the stomach, and that without too much delay. Exploratory laparotomy in these cases is not *only admissible*, but *obligatory*.

As was stated above, carcinoma frequently develops on the base of an old ulcer, and there are no signs by which the change from a benign to a malign affection can be detected.

Our experience with such cases has taught us that when a patient who has or had a gastric ulcer suffers with an aggravation or recurrence of the symptoms with coincident loss of weight and strength, and does not improve under appropriate medical treatment, exploratory laparotomy is the safest procedure.

The later stages of cancer, especially when located at the pylorus, are readily differentiated from ulcer. The marked wasting and loss of strength, the cachexia, the anacidity of the gastric juice, the absence of ferments, and the presence of lactic acid therein when the growth is at the pylorus, all bear strong testimony to the existence of a carcinoma; testimony that is considerably strengthened if a hard, nodular tumor and supraclavicular glandular enlargement are to be felt.

In connection with gastric ulcer we must not forget the complications to which it may give rise—viz., hæmatemesis and melæna, pyloric stenosis with secondary gastric dilatation, acute perforation, chronic perforation with perigastritis, and subphrenic abscess. The diagnosis of benign pyloric stenosis, acute perforation, and subphrenic abscess will be considered on pages 270 and 274.

Chronic or gradual perforation often gives rise to perigastritis, with the formation of an exudate around the perforation. The exudate may become adherent to the anterior

abdominal wall or to neighboring organs, and when it is originally situated in the bursa omentalis it may burrow upward into the subphrenic regions. Such an exudate is hard, very sensitive, has indefinite borders and a smooth surface. Should it break down into pus the temperature would rise and the leukocytes increase in numbers. The tumor formed by a perigastric exudate may simulate cancer of the stomach, from which the clinical history and the hyperacidity of the gastric juice differentiate it. From a neoplasm of the colon a perigastric exudate is distinguished by its clinical course and the symptoms which attend it and by colonic distention.

Perigastric adhesions from ulcer, gall-bladder, or pancreatic disease may cause a benign pyloric stenosis. A clinical history pointing to ulcer and the examination of the stomach contents will help to establish the cause of the perigastritis.

NEOPLASMS OF THE STOMACH.

Benign as well as malign neoplasms develop in the walls of the stomach. The former grow slowly, attain considerable size, have a smooth, hard surface, and cause no disturbances in gastric function or general nutrition. The latter,¹ on the other hand, grow rapidly, give rise to chemical changes in the gastric juice and disturb gastric motility, and very early in their course occasion marked emaciation and cachexia.

It is unfortunate for our patients that too many physicians still rely upon the presence of a *palpable tumor* for the diagnosis of gastric cancer, because when a tumor becomes palpable it is too late to effect a cure of the malady. If we are to hold out to patients afflicted with this disease any hope for a radical cure, we must make the diagnosis before a tumor becomes palpable. Czerny has stated "that no cancer of the stomach should be operated upon radically at a time when its

¹ The carcinomata are more frequent than the sarcomata. The latter, according to Fenwick, constitute from 5 to 7 per cent. of stomach tumors. They are often associated with sarcomata of the ovaries.

recognition as a tumor has become certain," and Kraske is of the opinion "that operation for pyloric cancer is desirable only when a tumor cannot be felt, and when on this account no positive diagnosis is possible." A palpable tumor is usually one of the late manifestations of the malady, and while its presence in conjunction with other clinical symptoms affords almost conclusive evidence of the existence of a cancer, it comes too late to permit us to wait for it before establishing a diagnosis. In the early stages of the malady we must rely upon other clinical evidences than the presence of a tumor for making the diagnosis; these evidences, as Kraske states, are not positive, but they are strongly suspicious and warrant, nay, even oblige, us to resort to exploratory laparotomy in order to ascertain their cause.

The most important of the early signs of cancer of the stomach are (1) the occurrence of dyspeptic symptoms in a patient who is beyond the fortieth year of life; (2) changes in the chemical composition of the gastric juice; (3) disturbances in swallowing or in gastric motility; (4) a secondary anæmia.

The earliest manifestations of the disease are usually of a dyspeptic character. These may remain the only evidences during the early stages—*e. g.*, when the neoplasm is limited to the body of the organ—or to them may be added the evidences of pyloric or cardiac stenosis when the new-growth is located at one or the other of the orifices of the organ.

It would be better for our patients if we were to suspect every one beyond forty who suddenly presents gastric symptoms as being affected with carcinoma and devoted our efforts to disprove its presence, than if we look upon these manifestations with a favorable eye until the development of a tumor dispels our false hopes of their benign character.

The gastric juice in the early stages of carcinoma of the stomach usually contains *no* free hydrochloric acid; in those instances in which the cancer develops on the base of an ulcer there may be a hyperacidity. The absence of digestive ferments (pepsin and rennet) is also an inconstant phenomenon. Several other conditions besides cancer—*e. g.*, chronic catarrh of the stomach and atrophy of the gastric mucosa—

are sometimes accompanied by a marked diminution or absence of free hydrochloric acid from the gastric juice. A chronic catarrh, however, runs a lengthy course with improvements and regressions, and with no grave impairment of the general condition. Atrophy of the gastric mucosa, while it may determine an alarming state of cachexia, has likewise a protracted course and will not be ordinarily complicated by disorders of motility. The absence of motor insufficiency will likewise differentiate the cases of gastric atrophy due to cancer in other parts of the body from pyloric cancer. The greatest difficulties will be experienced in the differentiation between cancer and the atrophy which is due to gastric distention from benign stenosis of the pylorus. Here again the longer duration of the malady is important for differentiation.

Should the cancer be located at the pyloric orifice, there will be quite early manifested an *insufficient motor function*, with vomiting, stagnation of contents within the viscus, and the presence of lactic acid in the gastric juice. These additional symptoms make the early diagnosis much easier. All these symptoms may again attend a benign pyloric stenosis with atrophy of the mucous membrane, but the progress of the symptoms in this latter condition is very slow, and is usually preceded by the symptoms of gastric ulcer.

Should the cancer be located at the cardiac orifice, an increasing difficulty in swallowing with absence of or delay in the second swallowing sound will materially aid in establishing an early diagnosis.

During these early stages the blood frequently shows a *secondary anæmia*, the hæmoglobin falling to 30 or 40 per cent.

While the symptoms just detailed are not positive proof of gastric cancer, they are nevertheless strong enough evidence to justify us in advising an exploratory laparotomy in order to ascertain their cause. The question may be summed up as follows: When a patient beyond forty presents the symptoms of an intractable chronic dyspepsia, with absence of free hydrochloric acid in the gastric juice and with evidences of motor insufficiency of the stomach

(such as vomiting, stagnation in the stomach, and lactic acid in the gastric juice), and continuously loses weight, he should be urgently advised to have an exploratory laparotomy done in order to ascertain the cause of the symptoms.

Difficult as is the early diagnosis of gastric cancer, so easy does it become after the development of a tumor, which, in conjunction with the symptoms above detailed, is almost conclusive evidence of this disease.

The *tumor* is hard and nodular; when not adherent it enjoys respiratory, active, and passive motility, and should there be gastropexia it may have a very low site, even sagging into the right kidney region. If it is located upon the posterior wall or lesser curvature, it is obscured by distention of the organ. Colonic distention causes the tumor to ascend.

The tumor of a gastric cancer is to be distinguished from tumors of the colon, omentum, gall-bladder, liver, pancreas, and abdominal wall, and from a floating kidney or spleen.

Colonic Tumors.—Colonic tumors are differentiated by gastric and colonic distention, the absence of chemical changes in the gastric juice, and the presence of stenotic symptoms of the colon with colonic erections. (See Chronic Intestinal Obstruction, p. 283.)

Omental Tumors.—Omental tumors are elongated in form and are pushed downward by gastric and colonic distention. If they are adherent in front of the stomach, distention of the latter makes them more prominent. They are not attended with chemical changes in the gastric juice.

Tumors of Gall-bladder.—With tumors of the gall-bladder there is a history of previous attacks of biliary colic and cholecystitis, possibly of jaundice, and of calculi being found in the stools. The gastric secretion is normal. The tumor has the shape of a gall-bladder.

Tumors of Liver.—Tumors of the left lobe of the liver are not affected by gastric distention.

Tumors of Pancreas.—Tumors of the pancreas are obscured by gastric and colonic distention; the gastric secretion is normal; there may be jaundice; cachexia occurs

early and is marked. There are evidences of pyloric stenosis.

Wandering Spleen.—The wandering spleen is easily recognized by its notched border; the *kidney* by its shape. Both organs can be replaced into their respective positions.

CHAPTER XXIII.

DISEASES OF THE STOMACH (*Continued*).

PYLORIC STENOSIS.

MODERATE or severe forms of congenital pyloric stenosis, which are presumably due to a spasm of the normal or hypertrophied pyloric muscle, are readily recognized from the symptoms which they occasion. Either right after birth or in the course of the first year of its life, the infant persistently vomits all ingesta, and that without any cause. In spite of change in and regulation of the diet and of all local treatment, the vomiting continues and soon causes the death of the patient from inanition. The stomach is rarely much dilated; occasionally the hypertrophied pylorus is to be felt as a distinct tumor.

The acquired forms of pyloric stenosis are either benign or malign in character; the former are due to the contraction of a healed pyloric ulcer, to kinking of the pylorus by perigastric adhesions or to pressure on the pylorus—*e. g.*, by an exudate or an enlarged gall-bladder or a floating kidney; the latter are due to malignant disease of the pylorus or adjacent viscera.

The existence of a *pyloric stenosis* is not hard to diagnose, for the *disturbed gastric motility* which it occasions bears strong testimony thereof. The evidences of such disturbed motility are *vomiting* (often of large quantities of food, some of which may have been ingested several days before), more or less *prolongation* of the time in which the stomach should empty itself after a meal, and in the more advanced cases *stagnation of food* within the stomach, *gastric eructations* and more or less *gastric dilatation*, the lower border of the viscus in its distended state descending below the umbilicus.

The character of the stenosis is not always so easy to

determine. In most cases a preceding history of gastric ulcer, of cholelithiasis with cholecystitis, of chronic pancreatitis, or the physical evidences of a floating kidney will suggest the possibility of its benign character, a possibility that is rendered more certain if the gastric juice contains free hydrochloric acid and ferments and no lactic acid, and if the hæmoglobin percentage remains fairly high. In some cases, however, the distention of the stomach which follows a benign stenosis occasions an extreme grade of atrophy of the gastric mucosa, in consequence of which the gastric secretion will contain no free hydrochloric acid and no ferments, but will contain lactic acid;¹ the emaciation and cachexia may, furthermore, be just as marked as is present in malignant disease of the organ. In these cases the differential diagnosis is attended with a great deal of difficulty, and even the presence of a tumor at the pylorus may not be sufficient to distinguish the two conditions, for the tumor may represent an ulcer with thickened edges or a perigastric exudate or a contracted adherent gall-bladder, as well as a malignant neoplasm. The previous history may help us in the differentiation, but as surgical interference is indicated to relieve the stenosis whatever its cause, no time should be lost in proceeding to exploratory laparotomy.

ACUTE DILATATION OF THE STOMACH.

This follows a variety of local and constitutional diseases and also overfeeding. The condition manifests itself with a sudden onset of persistent and uncontrollable vomiting of large amounts of acid material, with marked prostration and collapse (weak pulse, subnormal temperature, and diminished urine elimination). The stomach is much dilated, the greater curvature descending considerably below the umbilicus.

¹ The presence of lactic acid in the gastric juice depends upon the absence therefrom of hydrochloric acid and of ferments and upon the presence of motor insufficiency; it is consequently found in the benign as well as the malign forms of pyloric stenosis attended with atrophy of gastric mucosa.

HOURLASS STOMACH.

The evidences of disturbed gastric motility—viz., repeated vomiting, stagnation of food within the stomach, dilatation of the organ, and disturbed nutrition and emaciation—may also be due to an hour-glass condition of the viscus. This is always acquired and is occasioned by a constriction of the stomach anywhere between its two orifices. The constriction may be due to perigastric adhesions,¹ or to contraction and spasm of the stomach wall at the site of a chronic ulcer, or to an annular neoplasm of the stomach.

The condition is to be recognized by physical examination, and its underlying cause determined from the anamnesis and from the examination of the gastric secretion. Thus a preliminary gallstone history, or a history of gastric ulcer with hyperacid gastric juice would indicate a benign type of hour-glass contraction, whereas anacidity of the gastric juice, with the presence of lactic acid therein, a low hæmoglobin percentage, and a hard, nodular, palpable tumor of the stomach, would point to malignant disease as the cause therefor.

According to Moynihan the following signs point clearly to hour-glass stomach:

1. If a stomach tube be passed and the viscus washed out with a known quantity of fluid, the loss of a certain quantity will be observed when the return fluid is measured (Woelfler's first sign).

2. If the stomach is washed out until it is clean, a sudden rush of foul, ill-smelling fluid may occur; or if the stomach is washed clean and a tube is reinserted after a few minutes, several ounces of offensive fluid may escape (Woelfler's secondary sign).

3. Paradoxical dilatation of the stomach (Jaworski). The stomach is first dilated and a succussion splash obtained; the stomach tube is passed and the viscus apparently emptied;

¹ Perigastric adhesions have as their most frequent cause an ulceration of the stomach or duodenum and a calculous cholecystitis.

on again palpating, the splashing succussion sound will be again elicited. Jaboulay has added to this the following: If the cardiac pouch be filled with water, the splashing succussion sound may still be obtained in the pyloric portion.

4. Von Eiselsberg observed that on distention of the stomach with a Seidlitz powder, a bulging of the left side of the epigastrium was produced. After a few moments this subsided and concomitantly there was a bulging of the right side.

5. Von Eiselsberg also heard a rumbling sound at the site of the constriction as the gas passed through the narrowed orifice.

6. Moynihan has found the following of service: The stomach resonance is percussed; the viscus is then distended with a Seidlitz powder. After a few minutes there is an enormous increase in the resonance of the upper part of the stomach, while the lower part remains unaltered. If the pyloric pouch can be felt or seen, the diagnosis is clear, for the upper, distended pouch is the cardiac segment.

7. Schmidt-Monard and Eichhorst have noticed a distinct sulcus between the two distended pouches, and one pouch can be emptied into the other.

8. Ewald distends the stomach and examines with the gastroduaphane; the transillumination is seen only in the cardiac pouch; the pyloric segment remains *dark*.

CHAPTER XXIV.

PERFORATIONS INTO THE PERITONEAL CAVITY.

PERFORATION of a hollow viscus, infected cyst, or abscess into the peritoneal cavity is usually attended with local or diffuse extravasation of infected material therein, and it is this latter circumstance that gives to this accident its sinister significance. Two problems stare us in the face in every case of suspected perforation: the first is to decide whether a perforation has really taken place, and if it has, the next thing to determine is whether the attending extravasation of intestinal contents or other infected material is confined by limiting adhesions to one part of the peritoneal cavity or whether it is free in the general peritoneal cavity.

The study that has been recently expended upon this subject has enabled us to diagnosticate with a fair amount of certainty the acute and sudden and the slow and gradual perforations that are attended with local or diffuse extravasation of septic material; but the slow and gradual perforations which are not attended with extravasation because of a previous adhesion of the diseased focus to a neighboring viscus or part of the abdominal wall are very difficult of recognition, and are sometimes only detected on the post-mortem table.

But though we are in a position to detect a perforation that is attended with extravasation of septic material, we are absolutely unable, from our present knowledge and experience, to positively state at an early stage after such a perforation has occurred whether or not the resulting extravasation will be confined by adhesions to one portion of the peritoneal cavity; and yet this is a most important question. For if the extravasation of infected material is into the free peritoneal cavity, an immediate laparotomy with repair of the perforation must be undertaken if we are to prevent the

diffusion of the septic matter throughout the peritoneal cavity with the consequent development of a diffuse septic peritonitis; and yet such immediate laparotomy may be most undesirable on account of the patient's otherwise poor general condition. If, however, we could foretell that the extravasation would be confined to a local area of the peritoneum, this immediate interference could be delayed, and if an abscess formed at the site of the extravasation it could be evacuated when it had become well walled off from the free peritoneal cavity. At such a time the operation would be comparatively simple, amounting only to incision and drainage of an extra-peritoneal abscess, and the patient would be in many instances far better able to withstand it.

We have, however, not yet learned to recognize in which cases the extravasation will remain localized, and until we can do so we shall have to operate all cases immediately upon making the diagnosis of a perforation, in order to prevent in some of them the possible complication of diffuse peritonitis.

The earlier the diagnosis is made and the quicker the proper surgical treatment is applied in the cases that are attended with extravasation into the free peritoneal cavity, the less will be the area of peritoneum which is soiled with the septic material and the less will be the likelihood of a diffuse septic peritonitis. The aim should be to establish a diagnosis before a septic peritonitis develops, and this is possible only by a careful study of each individual case. When a peritonitis has developed the diagnosis is very easy, but the patient's chances for recovery with or without operative interference is proportionally bad.

Turning now to the clinical evidences of perforation, we find that *the perforation, per se*, gives rise only to local pain; this is of varying severity, being very intense and tearing in the acute cases, and so slight as to pass unnoticed in the gradual ones. The pain may occasion some slight reflex shock and reflex vomiting. The other symptoms attending the perforation are due entirely to the extravasation of septic material. Their intensity depends upon the rapidity with which the septic material escapes into the peritoneal cavity, and upon the area of the peritoneum which is soiled.

With sudden acute perforations of large size that are not

surrounded by limiting adhesions, there is a rapid extensive soiling of the peritoneum. The earliest symptoms are those of deep shock—viz., cold, clammy skin; feeble, rapid pulse, and subnormal temperature—and to this the patient may succumb. If he does not, there rapidly develops a diffuse septic peritonitis, with free gas in the peritoneal cavity, the evidences of which are a rigidity, hardness, and respiratory immobility of the abdominal wall; an increasing dulness in the flanks, which shifts with a change in the patient's position; an increasing abdominal distention, a concentric obliteration of liver and splenic flatness, general abdominal pain and tenderness, vomiting, constipation, and a rise in the pulse rate out of proportion to the rise in temperature. The leukocyte count is not constant; it may be normal or considerably increased. It is a good prognostic sign if it is high, but is of no diagnostic value.

With a small pin-hole perforation into the general peritoneal cavity the leakage is very gradual. The initial shock is much less severe, and the development of the symptoms of diffuse peritonitis is much slower.

With perforations that have been completely closed by adhesions before actual rupture occurred, the latter event is attended only with pain, and there are apt to be no subsequent evidences.

With perforations in which the extravasation of infected matter is confined by limiting adhesions to a local area of the peritoneal cavity, or with perforations into the retroperitoneal cellular tissue, the initial shock is of moderate severity, and it is followed by the formation of a localized intraperitoneal or retroperitoneal or subphrenic exudate. This occasions local pain and a rise of temperature and pulse rate, local abdominal rigidity and immobility, local distention, possibly vomiting and constipation. The leukocyte count rises to 15,000 or 30,000. The physical signs are those of an intraperitoneal or retroperitoneal or subphrenic abscess, which sometimes contains gas.

To recapitulate:

☞ *Large perforations into the free peritoneal cavity*, which are accompanied by extensive extravasation of septic material, occasion deep shock; whereas, *small perforations*, which

are attended *with gradual extravasation* of infected matter, and *those that are completely shut off from the peritoneal cavity*, and therefore not attended with any extravasation, and those in which the extravasation is confined to a local area, occasion little or no shock.

In all there are evidences of peritonitis. With the cases of perforation and extravasation into the free peritoneal cavity, the peritonitis advances rapidly; with those in which the extravasation is absent or confined to a local portion of the peritoneal cavity, the peritonitis usually remains local, though progression is not impossible. The earliest symptoms of peritonitis are abdominal rigidity and immobility and increased pulse rate. Temperature elevations are usually present, but they are not in proportion to the increased pulse rate. The leukocyte count in diffuse peritonitis may be high, but this is not constant. Its presence is of good prognostic import. With a sacculated abscess the leukocytes rise to 20,000 or 30,000. Vomiting and distention appear late.

PERFORATIONS OF THE SPECIAL VISCERA.

Stomach.—Perforations of the anterior wall of this viscus are apt to be into the free peritoneal cavity. The extravasated material and the subsequent peritonitis remain confined to the upper quadrant of the abdomen for twelve to twenty-four hours, their downward spread being retarded by the transverse colon and mesocolon. The peritonitis is likely to be less severe on account of the diminished virulence of the organisms residing in the stomach. Perforations of the posterior wall of the stomach are into the bursa omentalis, in which the extravasated material is very apt to be confined; though it may subsequently burrow upward into the subphrenic spaces, causing a subphrenic abscess.

Duodenal Perforations.—If they are on the anterior wall of the viscus the rupture is usually into the free peritoneal cavity. The extravasated material gravitates along the outer side of the ascending colon into the right iliac fossa, and gives rise to a peritonitis that appears to originate from a diseased appendix. If they are on the posterior wall the extrav-

asation takes place into the retroduodenal cellular tissue; here it gives rise to an abscess, which may burrow upward to the right subphrenic region or downward into the iliac fossa; and in the latter instance it may be confounded with a retrocolic appendicular abscess.

Typhoid Perforations.—Typhoid perforations are, as a rule, attended with extravasation into the free peritoneal cavity, and are rapidly followed by the development of a diffuse septic peritonitis; only occasionally are they surrounded by adhesive barriers which confine the extravasated material, and so lead to the formation of a local intraperitoneal abscess. The perforated loop of intestine is usually at or near the right iliac fossa, and for this reason the symptoms resemble those of perforative appendicitis. Diagnostic errors are especially likely to arise in the ambulatory cases of typhoid.

Appendicular Perforations.—The organ is usually surrounded by omental and intestinal adhesions before the actual perforation occurs. These adhesive barriers usually confine the extravasation to the right iliac fossa, where an abscess forms. Sometimes, however, the perforation is into the free peritoneal cavity or into the retrocolic cellular tissues; in the latter instance the pus may burrow upward to the liver and right subphrenic space.

Other parts of the intestinal tract, the gall-bladder and bile-ducts, or the urinary bladder may perforate into the free peritoneal cavity or into a space that is surrounded by limiting adhesions. The accident occasions either a diffuse peritonitis or a local peritonitis with abscess formation at the site of the perforation.

It is to be noted that a strictly limited abscess may at any time rupture into the general peritoneal cavity and occasion a diffuse purulent peritonitis.

The diagnosis of a perforation is made by a careful consideration of the patient's previous and present history (an old ulcer or neoplasm in the gastrointestinal tract, or the existence of typhoid fever, or of recurring attacks of appendicitis, or cholecystitis being especially significant), and from a study of the symptoms. The occurrence of shock, tearing pain in the abdomen, rigidity of the abdominal wall, concentric

obliteration of liver and splenic dulness, with a gradually rising pulse rate, are early and fairly conclusive signs. The development of a diffuse septic peritonitis merely confirms our early diagnosis.

The viscus which is perforated is determined from a consideration of the previous history; thus a history of gastric or duodenal ulcer, of typhoid fever, of previous attacks of appendicitis, cholecystitis, etc., is most valuable as a guide to the site of the disease. A further guide is afforded by the fact that the peritonitis is always most intense at its point of origin.

Perforations of duodenal ulcers and of typhoid ulcers, especially in the ambulatory type of the latter disease, closely resemble in their clinical manifestations perforations of the appendix vermiformis, and they are frequently diagnosed as perforative appendicitis. The error is all the more pardonable in cases of perforating duodenal ulcer because this sometimes gives no evidence of its presence until perforation occurs. The cases of this latter malady that do give symptoms before perforation can be distinguished from cases of appendicitis by the previous history of pain in the right hypochondrium several hours after eating and by the absence of previous attacks of appendicitis. The Widal reaction of the blood and other evidences of typhoid fever will distinguish typhoidal from appendicular perforations.

Rupture of a hollow viscus, infected cyst, or abscess into the peritoneal cavity is distinguished from intestinal obstruction by the lack of the absolute constipation which attends this latter disease, and by the presence of concentric obliteration of liver and splenic dulness, and by the history of a cause for the perforation.

From *mesenteric thrombosis* and *embolism* this condition differs, in that the former frequently complicates cardiac and kidney disease and has an acute onset, with little or no shock, but with one or more bloody evacuations from the lower bowel.

CHAPTER XXV.

INTESTINAL OBSTRUCTION.

ACUTE OBSTRUCTION.

THE clinical picture of acute intestinal obstruction is so typical that its ready recognition is comparatively easy. The onset is acute, with intense colicky pain at the site of obstruction, varying degree of shock, vomiting, absolute constipation, and gradually increasing intestinal distention. The first vomitus consists of the contents of the stomach; later on it contains bile and fecal-smelling material. It is to be noted that one or two evacuations from the bowel frequently occur after the onset of the symptoms, but these are not to be looked upon as evidence of the patency of the intestinal lumen; they are derived from the bowel below the site of obstruction, and after this has emptied itself, absolute constipation is present.

When an acute obstruction is suspected, a high enema, consisting of a quart of soapsuds with half an ounce of ox-gall and a drachm of the spirits of turpentine should be administered, the patient being placed in the dorsal recumbent position, with the hips elevated on several hard pillows. The enema to be successful must be followed by the passing of *foul-smelling gases* and *fecal particles*. It is to be remembered that the first enema may be attended with a good result, but that in complete obstructions subsequent enemata will prove absolutely ineffectual.

In incomplete obstructions foul gas and blood-stained mucus and fecal particles may continue to be evacuated per rectum; in these cases the diagnosis must be made from the pain, the persistent vomiting, and gradually increasing abdominal distention.

The administration of a cathartic in suspected cases of acute obstruction is rarely advisable, for should there be

obstruction of the bowels, the symptoms will be materially aggravated thereby.

The character of the obstruction, whether dynamic or mechanical, and the cause to which it is due, are to be determined from the anamnesis and by physical examination. *The dynamic varieties* result from a weakness or paralysis of the muscular coat of the bowel; they occur with peritonitis, thrombosis, or embolism of the mesenteric vessels and as a sequence to overdistention of the bowel. A temporary parietic condition occurs in conjunction with strangulation of the testicles, diseases of the pancreas, after laparotomy, especially when the omentum has been deligated, and after operations upon the rectum, etc.

The mechanical varieties result from strangulation or obturation of the bowel, an interference with the blood supply of the affected loop of intestine attending the strangulation types. Examples of the latter are volvulus, knots, strangulation by bands, and by normal or abnormal orifices, etc.; of the obturator variety, examples are furnished by the obstruction from foreign bodies—*e. g.*, gallstones, by constricting or compressing neoplasms, feces, etc. The invagination of the bowel represents a combination of the strangulation and obturation types.

The cause of the obstruction can usually be determined from the previous history and from a careful physical examination. Thus a history of an old peritonitis or of an inflammation of an intraperitoneal organ suggests an obstruction by a band. A previous hernia points to this condition as a cause of the strangulation. A sudden onset with bloody evacuations per rectum, and a previous history of chronic cardiac or Bright's disease, or diabetes, suggests the possibility of mesenteric thrombosis or embolism. A history of cholelithiasis, with a recent severe attack of biliary colic, suggests the possibility that a gallstone has ulcerated into the bowel and is acting as an obturator. A history of blood and mucous evacuations in a child points to intussusception.

A hernia should always be looked for when there are evidences of obstruction. A tense, suddenly enlarged, tender swelling in the inguinal, femoral, or umbilical regions or other weak part of the abdominal wall points to a strangulated

hernia. The occurrence of painful spasmodic erections of the bowel suggests a long-standing chronic stenosis of the bowel with a sudden complete obstruction. *A doughy, tender, movable tumor* in the abdomen, with a history of bloody, mucous evacuations, points to acute intussusception. Invagination may further be recognized if the invaginated gut protrudes at the anus, or if it can be palpated per rectum. A much distended, dull, tympanitic tumefaction, surrounded by coils of distended intestine, suggests a volvulus of the bowel. In peritonitis the onset of the obstructive symptoms is slower: there is a preceding history of local intraperitoneal inflammation—*e. g.*, appendicitis, cholecystitis, pyosalpinx, etc.—the abdomen is generally tender; there is no peristalsis; the facies is drawn and anxious, the pulse is very rapid, and the temperature is slightly or considerably elevated. Such a picture following upon an acute obstruction of the bowels shows that a peritonitis has supervened.

The Site of Obstruction.—Very early vomiting and one or two good fecal movements, with escape of flatus after the onset, indicate a high site of the obstruction; whereas, early absolute constipation with late vomiting points to a low site of the obstructed bowel. Meteorism of the central part of the abdomen, with flattened flanks, indicates an obstruction above the ileocæcal valve, and general abdominal distention points to obstruction in the lower bowel. The cæcum is always *very much distended* in obstruction of the colon distally to it, and forms a bulging, tympanitic mass. Very slight abdominal distention points to obstruction high up in the bowel.

The obstructed coil can sometimes be palpated; it has a greater resistance and less motion than the other coils, and is always felt at the same place at repeated examinations. This fixed position has given it the name of the “fixed loop.” The presence of a fixed loop helps to differentiate between obstruction and peritonitis. In obstruction of the small intestine indican appears early in the urine; in obstruction of the large bowel indican is absent from the urine or appears late.

The mechanical and true paralytic types of acute intestinal obstruction are distinguished from the paretic types by the continuously progressive character of the symptoms that

attended them, the presence of a fixed loop of bowel, the rapid deterioration of the general condition, and the increasing rapidity of the pulse.

In *peritonitis* there is a preceding history of local intra-peritoneal inflammation, and there is general abdominal tenderness, rigidity of the abdominal wall, a drawn, anxious appearance, and very rapid pulse.

In the reflex paresis of the bowels accompanying acute and subacute pancreatic disease, the general symptoms are very much like those of acute obstruction. In pancreatic disease, however, the distention does not become so marked, there are pain and tenderness in the epigastrium, possibly sugar in the urine, and free fat in the stools, and there may be a preceding history of cholelithiasis.

CHRONIC OBSTRUCTION.

With the lesser grades of chronic obstruction of the bowels the symptoms are but slightly pronounced and the diagnosis is consequently difficult to make. Attacks of colicky pain, with pronounced tendency to constipation and distention of the bowel, are very suggestive of chronic obstruction and demand that we keep the patient under careful observation until the character of the malady becomes clear.

As the obstruction becomes more complete the attacks of colicky pain become more severe and more frequent, and in conjunction with them occur visible or palpable erections of the bowel above the seat of obstruction. These painful intestinal erections are pathognomonic of the condition. Coincidentally the constipation becomes more marked, but sometimes it alternates with a watery mucous diarrhoea, the latter being due to a catarrhal enteritis or colitis set up by the irritating fecal matter which is retained above the site of the obstruction. If at any time the stenosed orifice becomes totally occluded by swelling or by the lodgement of a foreign body within it, the signs of an acute obstruction would be manifest.

Physical examination and the previous history of the patient frequently help to determine the cause and site of the obstruction.

Cachexia, diminution of hæmoglobin to 30 per cent. or 40 per cent., marked loss of weight, and the presence of tumor particles in the stools point to cancer of the wall of the bowel, which the presence of a hard, nodular, fixed tumor confirms.

Persistent constipation and hard feces, with no deterioration in general health, or diminution of hæmoglobin percentage, and no cachexia, point rather to impacted feces; a diagnosis that is confirmed by the presence of hardened feces in the rectum or a soft, indentable tumor of the intestine.

A previous peritonitis suggests compression of the intestine by a band.

An elongated, sausage-shaped tumor, which is not very tender, but is soft and elastic, in conjunction with mucous stools, suggests a chronic intussusception.

Moderately marked symptoms of stenosis, with a hard, irregular, sausage-shaped, tender, adherent tumor in the ileocæcal regions, suggests tuberculosis; the diagnosis is verified by the presence of other foci of tuberculosis and by a positive tuberculin test.

Very chronic stenotic symptoms, with an ileocæcal tumor that is adherent to the iliac wall, fixed, not tender, hard, with spots of softening, point to actinomycosis of the bowels.

Site of Obstruction.—The site of obstruction is determined in the same way as in the acute obstruction. (See p. 282.) It is to be noted that obstruction of the large intestine below the cæcum is accompanied by considerable distention of the cæcum.

CHAPTER XXVI.

DISEASES OF THE APPENDIX VERMIFORMIS.

THE appendix usually lies in the right iliac fossa, its base corresponding to the mid-point of a line drawn from the superior iliac spine to the umbilicus (McBurney's point). Occasionally it lies in the pelvis, and sometimes, either because of a long mesenterium or a very freely movable caput coli, it lies in the left iliac fossa.

The appendix can be best palpated when the patient is in the dorsal recumbent position. By having the patient slightly raise the extended right lower limb from the plane of the examining table or bed (see Fig. 118), thereby contracting the psoas muscle, the appendix and caput coli are brought nearer to the anterior abdominal wall and their palpation is thereby rendered more accurate and easy. If the appendix lies in the pelvis, it is best palpated through the rectum or vagina.

The diagnosis of diseased conditions of the appendix vermiformis is in most instances readily and easily made, and yet it is no exaggeration to say that more diagnostic errors are committed in the name of this organ than in that of any of the other abdominal viscera. This is partly due to the close anatomical proximity of the appendix to the uterine appendages, the right ureter and kidney, and in some cases the gall-bladder, and partly to the similarity of the clinical manifestations of its diseased conditions to those which are due to affections of the pancreas, duodenum, diaphragmatic pleura, etc.

The most important symptoms of an acute attack of appendicitis are crampy, abdominal pain which becomes localized in the right iliac fossa (rarely in the pelvis or left iliac region), one or more attacks of vomiting, constipation (sometimes diarrhoea), fever, increased frequency of the pulse,

tenderness at the site of the diseased appendix (most frequently in the right iliac fossa, but sometimes in the pelvis, or left iliac fossa or right hypochondrium), rigidity of the abdominal wall overlying the inflamed organ, and often a tumefaction or swelling at the appendicular site. From these manifestations the diagnosis of an attack of appendicitis can usually be made, and by a carefully taken anamnesis, with a thorough and complete abdominal and thoracic examination, in which we should always include a rectal and vaginal palpation, we shall be enabled to exclude or confirm the presence of diseased conditions of other abdominal or thoracic organs that may simulate appendicitis. There is, however, a small proportion of cases in which it is impossible to differentiate between disease of the appendix and that of other right-sided abdominal organs; in such cases the important point to decide during an acute stage of inflammation is whether or not operative interference is urgently demanded.¹ If so, the abdominal incision will clear up the diagnosis. If, however, delay can be safely practised, further and continued observation will, as a rule, throw light upon the nature and site of the disease.

If it is impossible to determine in all acute cases whether or not the appendix is at the root of or is concerned in an inflammatory process on the right side of the abdomen, much less is it always possible to determine from the clinical evidences the exact character of the lesions that are present in the diseased organ. Those who insist upon immediate operation in *all acute cases* of appendicitis advance this inability to determine the nature of the lesions within an inflamed appendix as a cause for their practice. But while it must be confessed that it is not always possible to accurately state what are the pathological changes in the inflamed appendix, we are concerned in forming our decision for or against immediate operation, not so much in the *pathological alterations* which the diseased appendix has undergone, as we are in the

¹ The urgency of operative interference is determined from the rapidity of the pulse, the general appearance of the patient, the abdominal rigidity, and from the history of the onset and course of the attack. A rising pulse rate to 110, then to 120 and over, an anxious expression and increasing abdominal rigidity, irrespective of the temperature elevations, speak for immediate operation. (See p. 287.)

severity of the inflammation; and while we cannot predict from the clinical signs what *will be the severity*, we can, in the vast majority of cases, tell *what is the severity* at a particular time. If, therefore, we keep a sharp and close watch of the patient we will be able to determine when complications arise and whether the inflammation is progressive or regressive in character, and upon this determination we can base our decision for or against immediate operation.

The severity of the inflammation is *judged* from the *character of the onset of the attack, its subsequent course*, and from the *rapidity of the pulse rate, the rigidity of the abdominal wall, and general appearance of the patient*. Its progression is determined by *observing the pulse rate, the abdominal rigidity, and the general condition of the patient*.

An anxious, drawn expression is always a sign of serious disease of the appendix. An hourly increasing pulse rate, even though the temperatures are normal or slightly elevated, is a most important indication of a rapidly progressive severe inflammation of this organ. Marked abdominal rigidity is likewise a valuable indication of a severe inflammatory condition. *A sudden onset with a chill*, high temperature, and rapid pulse rate, with sudden abatement, points to a sudden diminution of high tension within the appendix. This may be due to a re-establishment of the patency of the orifice of exit of the appendicular canal—*e. g.*, by the expulsion of a foreign body, or it may be due to perforation or gangrene of the appendix, the latter being much the more frequent.

An onset with severe chill, and one or more subsequent chills, irrespective of the local conditions, but especially if no abscess is present, strongly suggests the possibility of an infected mesenteric thrombosis and septicopyæmia.

A very rapid pulse rate, with slightly elevated temperature, persistent vomiting of brownish material, with marked prostration and feeling of well-being, points to a septicæmia.

A severe chill, increasing pulse rate, and marked abdominal rigidity, with or without temperature elevation, or the presence of a mass in the appendicular region, speak strongly for immediate operation.

But while the exact character of the lesions in the diseased

appendix cannot be accurately determined in each case, there is a clinical picture that usually goes with definite lesions of the diseased organ, and from such clinical picture we can form a working estimate of the character of the inflammatory process.

ACUTE AND CHRONIC FOLLICULAR APPENDICITIS.

With acute follicular (catarrhal) appendicitis the symptoms are not very severe. The onset is sudden, with or without a chill, the temperature rising to between 99° and 103° per rectum, the pulse rate rarely rising above 100 to the minute. At first there are crampy pains around the umbilicus, but after a few hours these localize in the right iliac fossa. The patient may vomit or be constipated or have diarrhoea. The abdominal wall in the right iliac region is somewhat rigid, and there is distinct tenderness over the appendix.¹

The leukocytes are not materially increased in number. The attack subsides within twenty-four hours to several days or a week, and during it the patient is usually confined to bed. With the severe grades of this type of appendicitis, a slight local peritonitis, with formation of adhesions, develops. The first attack does not, as a rule, produce organic changes in the walls of the appendix, but succeeding attacks usually leave them thickened and infiltrated, thus resulting in a chronic follicular (catarrhal) appendicitis. This manifests itself by recurring acute seizures in the intervals between which the appendix is quite tender and thickened. If adhesions have formed, there is a tumefaction around the appendix.

¹ If the appendix is situated in the right iliac fossa the tenderness will very likely be at McBurney's point, but if it is in the pelvis or on the left side of the abdomen, or behind the cæcum the tenderness will be at the corresponding site.

ACUTE ULCERATIVE AND GANGRENOUS APPENDICITIS.

With acute ulcerative or gangrenous appendicitis¹ *without perforation* the onset is usually sudden; there may or may not have been previous attacks of follicular appendicitis. There is an initial chill or chilly sensations; the temperature rises to 102° or 104°; the pulse rate varies between 90 and 120 or over. There is vomiting, constipation, or diarrhoea. Locally there is considerable pain and tenderness over the appendix, and marked localized abdominal rigidity. The leukocytes are not regularly or persistently increased. The attack may subside within several days to a week, and during it the patient is quite sick; or the inflammation goes on to perforation and to the development of other complications.

With acute ulcerative or gangrenous appendicitis *with perforation* the attack commences just as the preceding one. After one to several days perforation occurs, at which time there is a sudden abatement in the local and general symptoms. This relief, however, is very shortly followed by another train of symptoms, which vary according to whether the extravasation of the appendicular contents into the peritoneal cavity which follows upon the perforation is confined or not by limiting adhesions.

If the extravasation remains confined, a local abscess forms around the perforated appendix.² Such an abscess may be located in the right iliac fossa, or behind the colon, even extending upward to the liver and subphrenic space, or in the pelvis or in the left iliac fossa. It forms a smooth, rounded swelling that usually has no mobility, and the soft parts overlying it are rigid, tender, and painful. If it lies in contact with the abdominal wall, the percussion note over it is dull; but if

¹ Gangrene occurring early in the course of an attack is usually due to an interference with the vascular supply of the organ—*e. g.*, by volvulus or kink of the mesentery, or by embolism or thrombosis of its nutrient vessels; occurring late, it is due to the intensity of the inflammatory process. It may be confined to one or more local spots of the mucous membrane, or it may involve the entire wall of the organ.

² It is to be especially noted that a local abscess sometimes forms without perforation or without gangrene of the appendix, and that there is no way in which we can tell prior to operation whether the abscess is or is not due to gangrene or perforation of the organ.

it lies behind or between the coils of intestines, the percussion note over it is tympanitic. If it lies in the pelvis a bulging, tender, painful swelling, more or less distinctly fluctuating, can be felt through the vagina or rectum. After the formation of the abscess the temperature varies between 101° and 104° , the pulse is usually below 120, and the leukocyte count is high, 20,000 to 30,000; occasionally there is vomiting, and the bowels are usually constipated. If the abscess is not incised and drained, spontaneous rupture into a hollow viscus or into the free peritoneal cavity usually takes place. If the rupture has occurred into a hollow viscus, the pus is discharged through this channel; if into the peritoneal cavity, a diffuse peritonitis results.

If the extravasation of appendicular contents following upon the perforation takes place into the free peritoneal cavity, its immediate manifestation is shock of varying severity. A diffuse purulent peritonitis soon develops, which gives the following symptoms: The pulse rate rises rapidly (to above 120), the temperature becomes slightly or considerably elevated (to 104° or 105°); the expression is drawn and anxious; there is vomiting of gastric, or bilious, or fecal material, and obstinate constipation. The leukocyte count is either low or high. (High temperatures and high leukocyte count are favorable prognostic signs, but are of no help in diagnosis.) Locally the abdominal wall is tender and rigid; there is gradually increasing distention, absence of peristalsis, and a free, shifting exudate in the loins. The exudate may become encapsulated in various and multiple regions of the peritoneal cavity, thus giving rise to irregular areas of dulness in the abdomen.

SEPTICÆMIA AND SEPTICOPYÆMIA FOLLOWING APPENDICITIS.

Acute follicular, or ulcerative, or gangrenous appendicitis may be followed early or late in the attack by acute septicæmia or by septic thrombosis of the venous radicles of the appendicular mesentery. The thrombosis may remain localized in the appendicular mesenteric veins or it may extend

upward into the superior mesenteric vein, portal vein, and liver. In the liver multiple abscesses may develop from the lodgement of minute septic emboli derived from the disorganization of septic blood clots in the mesenteric and portal veins (septicopyæmia).

Such involvement of the vascular system is especially likely to occur when the tension of the confined septic contents in the appendix becomes excessively high. Under these conditions the slightest variation, especially a further increase of pressure, either by vomiting or by the manipulations of the surgeon, may precipitate an injection of infectious and toxic material into the blood stream, causing toxæmia and bacteriæmia. Such a septicæmia may result fatally before the local changes in the appendix have had time to become manifest. The slower the rise in tension the more apt are the bloodvessels to be shut off by the inflammatory processes, and consequently the less are the dangers of acute septicæmia. In making the prognosis in a case of acute appendicitis the surgeon should bear in mind that in a foudroyant case a general infection may have taken place before his arrival, or be precipitated by his manipulations. With such a bacteriæmia the patient is much sicker; he usually has had a severe chill; the pulse is especially rapid, 120 or over; the temperature is not much elevated. The leukocyte count is apt to be low. Locally there are pain and tenderness and abdominal rigidity at the appendicular site. No mass or only a thickened appendix is to be felt.

Besides this immediate blood infection there is the thrombosis of the venous radicles of the appendicular mesenterium. Either from an extension of the inflammation within the appendix to the vascular structures, or from a volvulus of the appendix and its mesenterium, a thrombosis of the venous radicles results, which necessarily implies, especially if the thrombus is an infected one, the possibility of metastatic lodgement of portions of this thrombus in the portal vein, liver, and other organs.¹ Such a thrombosis of the mesenterium may be markedly in evidence, even

¹ The lodgement in other organs than the liver is possible only when there is a communication between the portal and hepatic veins.

though the appendix is neither gangrenous nor perforated. If the thrombosis remains localized in the appendicular mesenteric veins, no additional symptoms from this are manifest; but if it spreads and gives origin to a metastatic lodgement of infected emboli in the superior mesenteric or portal veins, or liver, there develop the symptoms of septicopyæmia—*i. e.*, repeated chills, temperatures fluctuating between subnormal and 107° or 108° , gradually increasing pulse rate, rapid deterioration in general condition, and increasing icterus. The abdomen is flat, not tender or painful or rigid. The liver is enlarged and somewhat tender. The appetite may continue good, and the bowels may move regularly. Death from exhaustion and sepsis occurs in from several days to several months. The removal of the appendix after infective thrombosis has taken place has no influence upon the subsequent course of the disease.

An *appendicitis* may be simulated by *intestinal colic*, *enteritis*, *cholecystitis*, *acute pancreatitis*, *displaced and diseased conditions of the kidneys*, *pedunculated ovarian and uterine tumors*, *tumors of the small intestine and caput coli*, and *diseases of the ureter*; also by *perforations of the stomach*, *duodenum*, and *typhoid ulcers of the intestine* and by *diaphragmatic pleurisy*.

In *intestinal colic* the pulse rate is not materially increased; there is no abdominal rigidity, and the appendicular region is not sensitive; high enemata relieve the pain.

In *enteritis* there is no appendicular pain, no material elevation of the pulse rate, and the stools usually contain mucus and blood.

Cholecystitis is usually distinguished by a history of previous attacks of biliary colic, the pain in which radiates into the back and right shoulder; some of these attacks may have been followed by jaundice and by the passage of calculi in the stools. A distended gall-bladder that has a long mesentery may occupy the right iliac fossa and so simulate an appendicular abscess or exudate, but the tumor caused by such a distended gall-bladder is smooth and rounded, and is apt to be quite movable from side to side and with respiration. If the examiner stands on the right side of the patient, facing his feet, and palpates the under surface of the liver

and neck of the gall-bladder, the smooth, rounded tumor formed by diseased conditions of the latter organ can be traced downward from the liver. Distention of the colon causes a tumor which represents the gall-bladder to move upward.

Acute pancreatitis is marked by a greater initial prostration, and possibly by a preceding history of gallstone disease. With this malady the pain and tenderness, and possibly swelling, are especially marked above the umbilicus; the urine sometimes contains sugar and the stools free fat. It is often impossible to make a differential diagnosis, and if on opening the abdomen the appendix is found to be healthy, the pancreas should always be palpated and fat necrosis looked for.

In *displaced kidney* the kidney-shaped tumor can always be replaced into the loin; there may be a history of periodical attacks of hydronephrosis.

With *pyelitis*, *pyonephrosis*, *hydronephrosis*, *stone* or *kink in the ureter* there is a preceding history of cystitis or kidney colic;¹ there are pathological changes in the urine, and the cystoscope reveals changes in the ureteral mouth. With *diseased conditions of the ureter* there is always a preceding cause for the inflammation—*e. g.*, cystitis, diseased kidney, stone, etc.—and the ureter can be palpated through the abdomen, vagina, or rectum as a thickened cord passing from the kidney to the bladder; the cystoscope also reveals changes in the ureteral orifice.

A kidney colic may very strongly resemble an appendicular colic, and many an appendix has been removed when the kidney was really the cause of the trouble. In all cases, therefore, in which there are no physical evidences that conclusively point to disease of the appendix the possibility of a diseased kidney or of a stone in the kidney or ureter being the cause of the pain and other symptoms should be remembered. The aid of the *x-ray*, cystoscope, and ureteral catheter, with separation and careful examination of the individual urines, should be invoked in all such cases in order to ascertain the location and character of the malady.

¹ In kidney colic the pain radiates to the groin, testicle, or ovary, thereby differing from the site of the pain in appendicular disease.

Pedunculated ovarian and uterine tumors are, as a rule, to be differentiated from appendicular exudates and abscesses by vaginal and rectal examination. The cysts with flabby walls are the most difficult to palpate. A history of menstrual irregularities should excite our suspicion of ovarian or tubal or uterine disease or neoplasm being present.

Diseased conditions of the right Fallopian tube sometimes resemble very strongly diseased conditions of the appendix. A history of genital infection, with the presence of an enlarged and tender left tube, and the close proximity of the inflammatory mass to the uterus are in favor of the trouble being a pyosalpinx. A history of menstrual irregularity (see p. 351), together with colicky pelvic pain, which is possibly accompanied with fainting spells, the early signs of pregnancy, and the presence of a tender, soft, doughy tumor that is closely related to the uterus, speak for extra-uterine pregnancy.

Perforating ulcers of the stomach and duodenum are usually preceded by a long history of epigastric pain, heart-burn, eructations, and vomiting of acid material, and sometimes by hæmatemesis and melæna. At the time of perforation the pain is of a tearing character and is located in the epigastrium, and only later does it become located in the right iliac fossa. Duodenal ulcers sometimes give no evidences of their presence until perforation occurs. In such cases the location of the tearing pain in the epigastric or hypochondriac regions at the beginning of the trouble should excite our suspicions of perforating duodenal ulceration.

Diaphragmatic pleurisy is readily detected from the friction sounds which are to be heard over the base of the chest. It should be an invariable practice to carefully examine the base of the right chest in every case in which there is pain in the right iliac fossa. If this is religiously done the diagnosis of appendicitis will not be made when the patient really has a right basal pneumonia.

One should further remember to exclude *neuralgia* of the *ilioinguinal nerve* in cases where the diagnosis of appendicitis is not absolutely positive. Such neuralgia provokes pain in the area supplied by this nerve which roughly corresponds to the iliac region, and may in this respect resemble

appendicular disease. This ailment is especially likely to be occasioned by a lateral curvature of the spine, and it is in patients with this deformity that we should especially be on our guard. Continued observation and relief from pain which is experienced by the proper orthopædic apparatus for the spinal deformity will enable us to make the correct diagnosis.

Sarcoma, tuberculosis, actinomycosis, and carcinoma of the intestines are often very difficult to differentiate from a chronic diseased appendix that is surrounded by an exudate and adhesions. The presence of multiple tumors, the long duration of the disease, and the absence of a high leukocyte count speak in favor of the former. If an abscess has formed secondarily to these affections, the incision of the abdomen alone can clear up the diagnosis.

Perforating typhoid ulcers, especially in the ambulatory type of the disease, bear a close similarity to appendicular perforation. A preceding history of fever with an enlarged spleen, roseola, and Widal reaction makes a differential diagnosis very easy, but these symptoms and signs are not always present. During the typhoid season it is always well to have in mind the possibility that a perforating ulcer may be the cause for symptoms that simulate appendicitis, and in every case of this latter malady we should look for evidences of typhoid fever. The possibility of the perforating ulcer in typhoid being located in the appendix should not be forgotten.

CHAPTER XXVII.

NEOPLASMS OF THE INTESTINE, MESENTERY, AND OMENTUM.

NEOPLASMS OF THE INTESTINE.

Benign Neoplasms.—A *benign* neoplasm of the bowel gives rise to no symptoms beyond the presence of a palpable tumor unless it occasions obturation or invagination of the affected coil of intestine with resulting intestinal obstruction. The tumor varies in size from a walnut to an orange, grows very slowly, and causes no disturbances in the general health of the patient. Histologically these benign tumors belong to the lipoma, myoma, or adenoma groups, the myoma being at times mistaken for sarcoma, from which, however, the slow growth and continued well-being of the patient differentiate it.

Malignant Neoplasms.—Rapid growth and rapid loss of flesh and strength distinguish the malignant neoplasms of the intestine. *The sarcomata* occur frequently during young adult life as multiple tumors, and spread rapidly to the mesentery and omentum. They differ from the carcinomata in one very essential particular in that they do not frequently occlude the lumen of the intestine, and hence do not occasion a chronic intestinal stenosis. Their disintegration gives rise to moderate temperature elevations between 101° and 102°. The presence of one of these tender, somewhat painful tumors in the right iliac fossa, together with temperature elevations and a free ascitic exudate in the peritoneal cavity, may lead one to the impression that he is dealing with a subacute appendicular exudate. The presence of multiple tumors within the abdomen which are often to be best palpated through the rectum, the wretched general condition of the patient, the history of the course of the disease

(appendicitis having an acute onset with possible previous attacks), and a low leukocyte count point very strongly to malignant disease of the bowel.

The carcinomata occur usually in late adult life and are most frequently located in the large intestine, especially in the sigmoid flexure and rectum. They tend to spread in an annular fashion around the intestine and thus occasion a chronic intestinal stenosis, the clinical signs of which have already been described (p. 283). A palpable tumor is usually a late manifestation of the disease, and in some instances—*e. g.*, when the neoplasm is located at the hepatic or splenic flexure—it is never to be felt. It is hard, irregular, nodular, and tender, and enjoys some mobility until it becomes adherent, which often occurs very early. In most instances a gradually increasing intestinal stenosis with intestinal erections, and fetid, bloody, purulent, mucous stools are the clinical evidences to which this malady gives rise. In some cases, however, especially in scirrhus carcinoma of the sigmoid flexure, the first indication which the neoplasm affords is an acute intestinal obstruction, and in others an increasing otherwise unexplainable cachexia first suggests the possibility of an intestinal carcinoma.

Tuberculosis and *actinomycosis* of the ileocaecal region are distinguished from malignant disease by their more chronic course. The tumor which they occasion is not so hard, and is very apt to have soft areas. The presence of other foci of tuberculous or actinomycotic disease are valuable aids in making a diagnosis, but the occasional simultaneous occurrence of tuberculous and malignant disease is not to be forgotten.

The evidences of *chronic intestinal stenosis* and a *palpable tumor* which go with chronic intussusception may lead to the diagnosis of malignant disease; but the absence of emaciation and cachexia, and the smooth, doughy, non-adherent characteristics of the tumor should in most instances enable us to differentiate the two.

A *chronic appendicitis* with *adhesions* and *exudate* is distinguished from malignant disease by the clinical history of the case, by the absence of cachexia, and by the usual absence of intestinal stenosis; the tumor which is due to

appendicular affections is not so hard or nodular as that due to a malignant neoplasm.

Fecal masses in the bowel are distinguished from malignant tumors by their soft, indentable character and by their disappearance after free catharsis. Our suspicion of malignant disease should be aroused if a patient has repeated attacks of obstinate constipation, and gradually loses flesh and strength.

TUMORS OF THE MESENTERY AND OMENTUM.

Mesenteric Tumors.—Tumors of the mesentery are to be recognized only when they attain sufficient size to be palpated. If the tumor is located near the intestinal end of the mesentery it enjoys considerable mobility unless it has become adherent to the neighboring viscera. Its adhesion to the abdominal wall is infrequent.

The nature of the tumor is difficult to determine.

Fluid Tumors.—The fluid tumors, which comprise the serous, hemorrhagic, chylous, echinococcic, dermoid, teratoid, and fetal inclusion cysts, are more or less fluctuating and of slow growth. They are usually unilocular and single; the lymphatic (chylous cysts) may be multiple.

Mesenteric tumors of this class are distinguished from *omental cysts* by the fact that the latter lie nearer to the abdominal wall and very early become adherent to it. From *pancreatic cysts* they are to be differentiated by the history of digestive disturbances or cholelithiasis, which usually precede the former, and by the fact that the outlines of pancreatic tumors are obliterated when the stomach and colon are distended. The presence of sugar in the urine and of free fat in the stools are further confirmatory evidences of the pancreatic origin of the cyst.

Mesenteric cysts are to be distinguished from *ovarian cysts* by the relations which the latter have to the uterus and broad ligament (p. 347). The extension of ovarian cysts from below upward and the palpation of their pedicle through the vagina or rectum are further proofs of their ovarian origin.

Sacculated intraperitoneal exudates, usually of tuberculous origin, may very much resemble a mesenteric cyst; but

these become flattened out by the pressure of the abdominal wall and overlying intestines when the patient assumes the recumbent position, whereas mesenteric cystic tumors retain their globular form.

Solid Tumors.—The *solid* mesenteric tumors belong histologically to the lipoma, lymphadenoma, tuberculoma, sarcoma or carcinoma.

The *lipomata* resemble the cystic tumors, but are said to grow much more rapidly. The malignant neoplasms are usually secondary deposits in the glands of the mesentery and cause a rapid deterioration of the general physical condition.

Omental Tumors.—Omental tumors form rounded or flattened masses which always lie close to the abdominal wall, to which, as a rule, they are closely connected. If the tumor develops after a laparotomy or herniotomy or inflammation of the abdominal viscera, it is of an inflammatory character. Such inflammatory tumors of the omentum are apt to occasion considerable vomiting of a hemorrhagic material and obstinate constipation; should pus form it would be indicated by a rise in temperature and an increase in the leukocytes up to 20,000 or 30,000.

Cystic omental tumors are usually due to the echinococcus.

Carcinoma and sarcoma are usually secondary tumors.

Torsion of Omentum.—Torsion of a part or the whole of the omentum results in the formation of a lumpy tumor with narrow or thick pedicle. It occasions considerable peritoneal irritation and serohemorrhagic exudate in the peritoneal cavity. It simulates twisted ovarian cyst or acute appendicitis. The diagnosis is usually made only after abdominal section.

CHAPTER XXVIII.

DISEASES OF THE LIVER.

THE normal position of the liver is in the right hypochondrium and epigastrium, behind the lower chest wall. Its upper border in the sternal line is at the base of the ensiform cartilage; in the parasternal line at the lower border of the fifth rib; in the mammary line $2\frac{1}{2}$ cm. above the upper border of the sixth rib; in the axillary line at the upper border of the seventh rib. The lower border in the axillary line is between the tenth and eleventh ribs; in the mammary line at the costal margin; in the median line about midway between the ensiform cartilage and the umbilicus, and then it rises in a curved direction upward, reaching the diaphragm between the left parasternal and mammary lines. In those who have lax ligaments and flaccid abdominal walls, and in those who lace tightly, the organ always lies somewhat lower.

Only the lower border of the organ can under normal conditions be palpated. It should feel sharp and smooth and soft; an increased resistance or an undue roundness, or unevenness or tenderness thereof point to diseased conditions of the organ.

ABNORMAL LOBES.

Abnormal lobes, the most common of which is a tongue-shaped prolongation of the right lobe, are not infrequently encountered, and are especially common in women with lax abdominal walls. They result, as a rule, from tight lacing or constriction of the lower costal margin during adolescence, when the thorax is soft and flexible. The lobe itself represents a movable prolongation of the liver and is connected therewith by a broad or narrow pedicle. The pres-

ence of such an abnormal lobe is made manifest by pain or by attacks of twisting or inflammation of its pedicle on account of which the lobe becomes painful, tender, and of increased size. The symptoms occasioned by biliary calculi, to the formation of which the abnormal lobe has predisposed by compression or kinking of the cystic duct, or the presence of a tumor in the right side of the abdomen, are additional evidences of such abnormal hepatic lobes.

Such tumors are directly connected with the liver, are demarcated from it by a transverse groove, and unless adhesions have formed around them are freely movable. They lie *above* the *transverse colon*. Sometimes they appear to occupy the loin and so may be mistaken for displaced kidneys and tumors of the kidney and adrenals. They cannot, however, be fully replaced into the loin as kidney tumors can be, and when pressure upon them is relaxed they immediately assume their former position. Kidney tumors furthermore *lie behind the colon*, or, when very large, protrude forward with the colon along their inner side, and if the kidney is diseased the cystoscopic examination of the bladder usually reveals changes in the ureteric mouths, and the ureteric catheter withdraws abnormal urine from the corresponding side.

A distended gall-bladder is to be distinguished from an abnormal lobe by its greater lateral mobility and by the absence of a groove between it and the liver. It is well to remember, however, that the two conditions are very frequently associated, the lobe lying external to the gall-bladder or spread out over its surface; they may both have long pedicles and reach down to the umbilicus.

Tumors around the appendix can be differentiated from abnormal hepatic lobes by their clinical history, their relation to the distended colon, and the absence of the groove that always separates an abnormal lobe from the liver. Appendicular tumors are rarely as smooth or tongue-shaped, and are apt to be more tender and less movable.

Tumors of the hepatic flexure of the colon are distinguished from abnormal hepatic lobes by the chronic intestinal stenosis which attend them.

The characteristic flexion of the thigh and deformity of

the spine that attend psoas abscess readily differentiate this condition from the one under discussion.

FLOATING LIVER.

Floating liver is a rare occurrence. It is found in women with very lax abdominal walls, and is indicated by a tympanitic resonance where the liver dulness should be. When the patient assumes the recumbent position the normal area of liver dulness returns. Such patients suffer from pain, dragging sensations, gastric disturbances, portal congestion, and hemorrhoids.

CONGENITAL MALPOSITION OF LIVER.

Congenital malposition of the liver in a hernia into the umbilical cord has already been referred to (p. 240). Such children usually succumb a few days after birth.

LIVER ABSCESS.

It is essential first of all to disabuse our minds of the impression that hepatic suppurations are infrequent in these temperate zones; for while the tropical abscesses of the liver which are secondary to simple or amœbic dysentery are relatively infrequent in our climates, those which are secondary to purulent affections of the gall-bladder and bile-ducts—to suppurative and ulcerative diseases within the area drained by the branches of the portal vein and to severe traumatisms of the liver—are quite frequently encountered.

Tropical Abscess.—The clinical story of a patient who has a tropical abscess of the liver is about as follows: He has or had a diarrhœa;¹ becomes gradually more and more sallow, almost cachectic in appearance, emaciated, is subject to psychical depression, gastric disturbances, and profuse sweating, and runs a continuously or remittently high tem-

¹ It is strange that even a severe diarrhœa often ceases with the advent of the liver suppuration.

perature. It is a mistake to think that all patients with liver abscess are jaundiced; they become so only when there is an associated cholangitis, or when the abscess presses upon and occludes the hepatic or common bile-ducts. Nor is the high temperature an invariable accompaniment of this variety of liver suppuration, for in those cases in which the abscess is surrounded by a dense fibrous wall the temperature may be normal or but slightly elevated.

Such a clinical picture should always arouse our suspicion of a liver abscess, and should prompt us to carefully examine this organ. A small, deeply seated abscess may afford no local evidences of its presence; if, however, we are reasonably sure of the diagnosis we may explore the liver for pus with an aspirating needle. It is to be remembered, however, that while a positive result of such aspiration is conclusive evidence of the presence of an abscess, a negative result is not similarly conclusive against it, for the focus may be so small and so deep as to escape our aspirating needle.

Larger and more superficially seated abscesses provoke *pain*, which may radiate to the shoulder and groin, and which is increased by deep respiration and compression of the thorax. *Tenderness* over the liver is likewise present, and is most acute directly over the seat of the abscess. *Enlargement* of the liver goes with all abscesses of any size, and the direction of the enlargement is a clue to their seat. Thus, if the abscess is in the upper posterior portion of the liver the enlargement of the organ is upward and backward, the right lower lobe of the lung being compressed and the lower limits of pulmonary dulness being convex upward; if it is in the lateral portion of the convexity, the lower ribs and intercostal spaces are bulged outward; and if it is in the anterior portion of the organ, either near the convexity or lower surface, the abdominal wall below the costal margin is bulged forward, and the anterior border of the liver is thickened and rounded. If the abscess is in the left lobe the area of liver dulness to the left is increased.

With superficial abscesses the cutaneous veins are engorged, and the soft parts are œdematous and fluctuating.

Multiple Abscesses.—With *multiple abscesses* of the liver there is an entirely different preceding history and clinical

course. Thus, as a rule, some intraperitoneal suppuration or an ulceration of one of the intra-abdominal viscera, or gallstone disease, cholangitis, appendicitis, etc., precedes the development of the liver suppuration. The course of the malady is marked by repeated chills with intermittently high temperatures, profuse sweats, rapid emaciation, and increasing jaundice. In some instances the course is less acute; the chills are infrequent, sometimes altogether wanting; the temperatures are very irregular, sometimes quite high and again near to the normal; and gradually increasing pallor, slight or marked jaundice, emaciation, and weakness develop.

The physical signs in the liver are the same as with the single abscesses, and the spleen is especially likely to be enlarged.

Though the diagnosis of liver abscess can, as a rule, be made from the above-described clinical manifestations, it should always be confirmed by exploratory puncture. This should be made with a fine-calibred needle, which is introduced at the point of greatest tenderness. Aspiration is perfectly safe if it is made through the back or side of the chest, but if the abscess is located in the epigastrium or in the left lobe of the liver, exploratory incision is to be preferred to aspiration. The pus which is most frequently obtained from liver abscesses is thick, of a dirty brown color (like soft chocolate ice cream), and contains liver cells, pus cells, and in most instances some variety of bacteria or the *amœba coli* or *actinomyces*. A culture should always be made from it at once, and cover-glass spreads therefrom should be examined under the microscope.

The leukocyte count is not of much aid in the diagnosis of liver suppuration. With the acute varieties it is usually somewhat increased, but with the chronic types it is not materially altered.

The possibility that a hydrothorax, pyothorax, or ascites may complicate the liver abscess should be kept in mind; so that the physical signs which result from these conditions should be properly interpreted. Perforation of the abscess into the pleural cavity is indicated by shock and intense dyspnœa, and is followed by a pyothorax. Perforation into the peritoneal cavity has already been considered (p. 274).

If the abscess ruptures into the intestine or through the diaphragm into the lung and then into a bronchus, there will be an evacuation of the pus by the rectum in the first instance and by the mouth in the latter.

A liver abscess is to be distinguished from an uninfected echinococcus cyst of the liver by the history of one of the usual etiological factors for this condition, by the presence of fever, a painful and tender liver, and by the emaciation and sallowness of the patient. In hydatid disease the liver is usually rounded, smooth, and tense, and in some instances hydatid fremitus may be obtained. Infected hydatid cysts differ from abscesses of the liver in that the acute stage of infection is preceded by a long period of digestive disturbances with enlarged liver.

The swelling in the right hypochondrium occasioned by a hydrops or empyema of the gall-bladder might possibly be mistaken for a liver abscess. With hydrops, however, there are no constitutional disturbances and the gall-bladder is tense, only slightly tender, smooth, and movable; with *empyema* the gall-bladder is tender, usually enlarged, and somewhat movable. The liver is not enlarged or painful or tender in either case. If a suppurative cholangitis develops secondary to these inflammations of the gall-bladder the symptoms of hepatic abscesses would also become manifested.

A kidney shape, a hilus, and a rounded lower border distinguish a tumor due to an *inflamed right kidney, hydronephrosis, or pyonephrosis* from that which is occasioned by a liver abscess. Furthermore, a kidney can be replaced into the loin; it has no respiratory mobility; it lies behind the distended colon, and if it is very much enlarged it approaches the anterior abdominal wall to the inner side of the distended colon. With disease of the kidney the urine often contains pus or blood. With periodical hydronephrosis the tumor appears and disappears; with its appearance there is pain in the loin, vesical tenesmus, and diminution in the amount of urine passed per urethram; with its disappearance the pain is relieved and there is an increased amount of urine voided. With disease of the kidney the cystoscope will reveal changes in the mouth of the right ureter, and catheterization of the right ureter will withdraw

altered urine. There may be a preceding history of tuberculosis or renal calculi or neoplasm. With hydronephrosis there are no constitutional symptoms.

A history pointing to preceding kidney suppuration would help to distinguish a *paranephritic abscess* from liver abscess, for most all of the former follow upon abscesses of the kidney. In those instances in which the paranephritic suppuration is metastatic to suppuration in other parts of the body, and especially if in these cases the pus burrows upward behind the liver, the differentiation from liver abscess may be very difficult.

The relation which a *pancreatic swelling* bears to the stomach and colon will usually suffice to distinguish it from that which is due to a liver abscess. A preceding history of gallstone disease, the presence of sugar in the urine, and fat in the stools are additional evidences in favor of pancreatic disease. Furthermore, cysts and tumors of the pancreas are not attended with any febrile constitutional disturbances, and in the acute forms of pancreatitis these evidences are much more intense than they are with liver abscess.

The lack of the characteristic splenic shape and notched anterior border help to differentiate swellings due to abscesses of the left lobe of the liver from *swellings of the spleen*. The absence of marked gastric symptoms and disturbance of gastric motility, and of chemical changes in the gastric juice help to distinguish gastric tumors from those due to chronic liver suppuration.

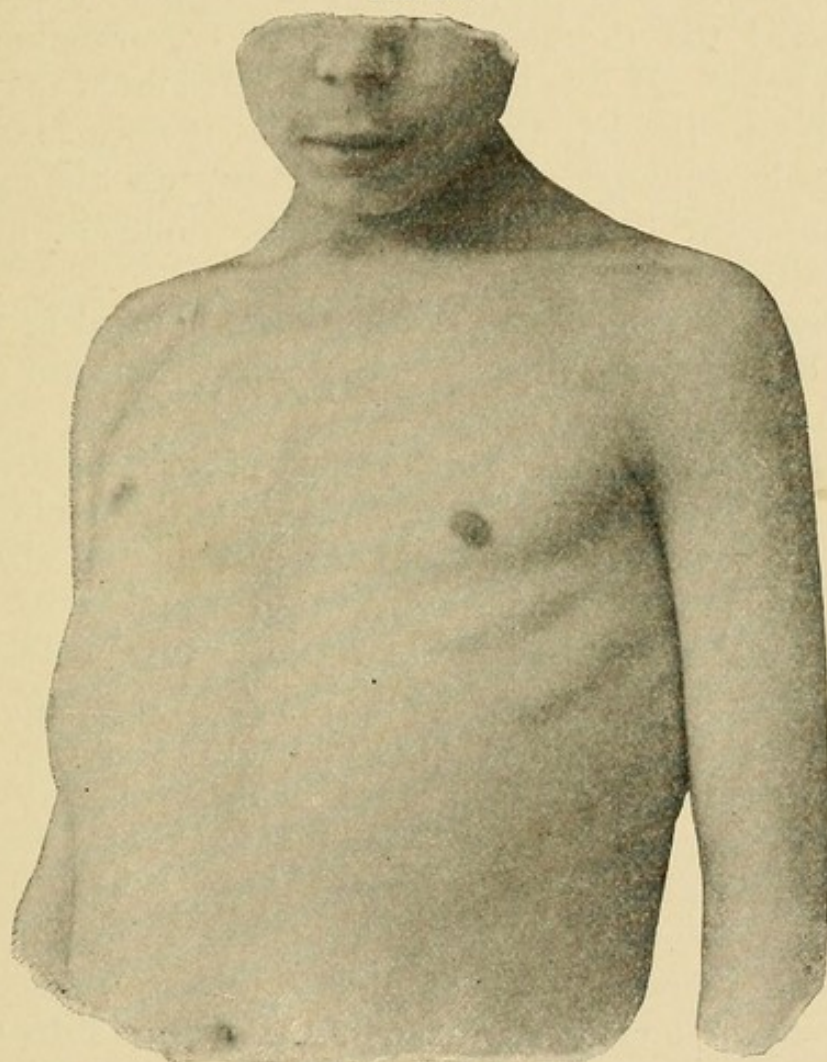
The chills and fever attending multiple liver abscesses may suggest malaria, but there is here no regular cycle to the paroxysms, and no plasmodia are to be found in the blood. Furthermore, there is in the history of the patient's illness a distinct cause—*e. g.*, dysentery or intraperitoneal suppuration or ulceration for liver abscesses.

NEOPLASMS OF THE LIVER.

Hydatid (*Echinococcus*) Cysts.—These constitute the large majority of the fluid tumors. These cysts are usually unilocular and single, but they may be multilocular and

multiple; they occur most frequently in women, and rarely in children; and their favorite site is in the right lobe. When small and located in the centre of the liver they give no indication of their presence; larger cysts form elastic, non-tender, fluctuating tumors that share with the liver its

FIG. 129



Hydatid cyst of the liver. (Francis H. Markoe.)

respiratory mobility, are dull to percussion, and occasionally afford an hydatid fremitus.¹ They may occasion pain which radiates to the right shoulder, shoulder-blade, and loin, some fever from resorption of the cystic contents, emaciation, and weakness. Icterus is present only when the chole-dochus is compressed by the tumor or when there is an

¹ Hydatid fremitus is a peculiar vibratory sensation that is experienced when the cyst is palpated.

associated cholangitis. The cyst usually projects from the convexity of the right lobe as a semiglobular tumor, which causes the lower thoracic wall to bulge outward; it may project from the under surface of the liver in the form of a pedunculated tumor, and if the pedicle is long enough the cyst may reach down into the lower part of the abdominal cavity; or it may project from the upper surface of the liver toward the thoracic cavity with compression of the lung and heart. If it is located in the left lobe it may extend over so far to the left that the dulness it occasions merges with splenic dulness. Large cysts compress the neighboring organs—the lungs, heart, kidneys, stomach, intestines, and gall-ducts.

Cyst Contents.—The cyst contents are neutral in reaction, 1000 or 1015 specific gravity, colorless, contain no albumin, but do have a trace of sugar.

Infection of Cyst.—The development of fever, of continuous, remittent or intermittent type, with or without chills, rapid emaciation, sweating, and rapid increase in the size of and tenderness over the tumor indicate an *infection of the cyst*.

Rupture of Cyst.—Rupture of the cyst into a neighboring hollow viscus is recognized by the escape of daughter cysts in the stools or in the urine, or in vomited or expectorated material. Rupture into the peritoneal or pleural cavity is followed by a varying degree of shock, rise of temperature, and an urticarial eruption over the body. The extravasated cyst contents excite a peritonitis or pleuritis, and if recovery takes place a new growth of cysts will take place in these cavities within one or more years. Rupture into the pericardium is usually fatal. Rupture into the bile-ducts is followed by cholangitis and increasing icterus, and as the daughter cysts pass through the ducts they occasion attacks of biliary colic.

In another chapter we have mentioned that the changes in the size and shape of the liver due to abscess are very similar to those which result from echinococcus disease. Of course the severe grade of constitutional symptoms readily distinguishes the acute liver abscesses from hydatid cysts, but the chronic variety of abscesses bear them some resemblance. It is to be noted, however, that patients

suffering with this variety of abscess usually give a preceding history of diarrhœa, or of trauma over the liver, or of cholelithiasis; they are much more sallow and emaciated, and the liver or tumor is more tender and less tense. If the cyst has ruptured and daughter cysts have escaped in the stools or in the urine or have been expectorated or vomited, the differential diagnosis is very easily made.

A preceding history of ulceration or suppuration of an intraperitoneal organ and an acute onset and course distinguish the acute liver abscesses from the infected hydatid cyst; furthermore the echinococcus cysts, as a rule, exist for a long time prior to their infection, and during this uninfected period the patient suffers from chronic gastrointestinal disturbance with some emaciation.

The multiplicity of the nodules, their hardness, tenderness, and rapid growth distinguish carcinoma of the liver from hydatid disease. The presence of a primary growth in some one of the other abdominal viscera, to which the liver manifestation is secondary, and the cachectic and somewhat icteric appearance of the patient help us materially in making the differentiation between the two conditions.

Wasting diseases and prolonged suppuration are attended with an enlargement of the liver due to its amyloid or fatty degeneration. But in these cases the organ is uniformly and generally enlarged and smooth and not painful, and the preceding history of a wasting malady furnishes an explanation for the liver and splenic changes.

Non-parasitic liver cysts can only be differentiated from the hydatid variety by examination of the cyst wall and the cyst contents. The cyst contents are to be obtained by exploratory puncture; this is never to be practised, however, unless all preparations for immediate operation have been previously made, for extravasation of the cyst contents into the peritoneal cavity along the track of the needle may follow such puncture and fever, urticaria, and peritonitis result therefrom.

A *pedunculated hydatid cyst* projecting from the under surface of the liver may be mistaken for a distended gall-bladder. But with this latter condition there is a preceding history of biliary colic, possibly of jaundice, and the passage

of stones in the stools. The tumor formed by a distended gall-bladder is oval-shaped, smooth, and is more frequently movable than a hydatid cyst.

Hydronephrotic and *pyonephrotic tumors* bear a superficial physical resemblance to pedunculated hydatid cysts, but they can be readily differentiated from them, for they lie behind the colon, have no respiratory mobility, and can be replaced into the loin, which position they maintain as long as the patient occupies the recumbent position. Furthermore, with these kidney lesions there are usually changes in the urine. With periodical hydronephrosis there are variations in the size of the growth; and as the kidney tumor diminishes in size there is an increase in the amount of urine which is voided. The altered appearances of the ureteral mouths in diseased conditions of the kidney, together with the abnormal constituents in the urine drawn from such kidneys by the ureteral catheter, are additional data for the recognition of kidney abnormalities and disease.

The position of pancreatic cysts behind the stomach and colon, or below the latter or above the former, and their lack of respiratory mobility, distinguishes them from hydatid disease of the liver. A preceding history of gallstone disease and the presence of sugar in the urine and fat in the stools are additional diagnostic evidences in favor of the pancreatic origin of the cyst.

Mesenteric cysts. (See p. 336.) *Splenic tumors* differ from cysts of the left lobe of the liver by their characteristic shape and their notched border.

Subphrenic echinococcus liver cysts are to be differentiated from pleural effusions and subphrenic abscesses.

Pleurisy with effusion has an acute onset with an acute course. It commences with a chill, high fever, pain in the side, and dyspnoea; as the effusion accumulates the dyspnoea becomes worse, the thorax (ribs and intercostal spaces) distended and barrel-shaped, the heart and mediastinum displaced, and the entire liver depressed; if the effusion is free it changes its level with changes of the patient's position. With pleurisy the area of compressed breathing extends up to the upper lobe, while with a subphrenic hydatid cyst vesicular breathing extends down to the level of the cyst. In

the latter affection the onset and progress of growth are slow, and the dyspnœa becomes very gradually worse; as the cyst increases in size it distends the thorax in a single area and the thorax has a bell-shape. The liver is depressed by it only in part—*e. g.*, in front or in back. Pain radiating to the back or shoulder is present, and if the liver can be palpated the circumscribed bulging of the cyst can be detected. With *empyema* or *chronic serous pleurisy* there is deterioration of the general health. Exploratory puncture determines the presence of serous or purulent exudates.

Subphrenic abscess is secondary to a ruptured hollow viscus, or to suppuration within the abdomen, and the signs of these primary maladies are, as a rule, quite clear. There is no previous history of liver pain or of an enlarged liver or of the passage of daughter cysts.

Pyopneumothorax has an acute onset, with cough, dyspnœa, expectoration, and some collapse. The heart and mediastinal structures are displaced. The entire lung is compressed; the breathing is labored and dyspnœic; there is a succussion splashing sound on shaking the patient.

Subphrenic cysts are distinguished from *supraphrenic cysts* by the following signs: In subphrenic cyst the thorax bulges in one place like a bell; in supraphrenic cyst it is uniformly bulging like a barrel. Subphrenic cysts push up the diaphragm, and soon paralyze it; they consequently cease to move with respiration. Supraphrenic cysts push down the diaphragm and the entire liver, but as they do not paralyze the former, they retain respiratory mobility. The supraphrenic cysts very early give evidences of pleural involvement.

Nonparasitic Cysts.—*Nonparasitic cysts* of the liver are either *biliary duct retention cysts*, or *dermoids* or *lymph cysts*, or *cystic adenoma*. They form solitary cysts, and are located near the liver surface. The symptoms are similar to those given by the hydatid cysts.

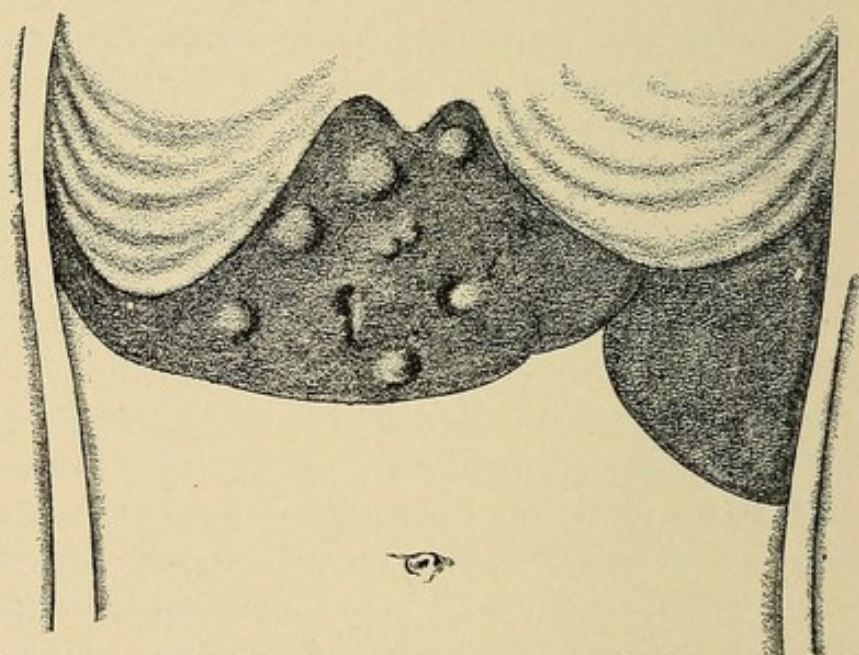
Cystic Degeneration of Liver.—Cystic degeneration of liver occurs frequently in conjunction with cystic degeneration of the kidneys. The liver feels lumpy. The diagnosis is to be made only by exploratory incision.

Solid Tumors.—Of the solid tumors of the liver the *syphilomata*, the *fibromata*, the *angiomata* and the *adenomata*

are the most frequently encountered benign growths, while the *carcinomata* and *sarcomata* are the usual varieties of the malignant neoplasms.

The syphilitic lesions of the liver that concern us most from a diagnostic standpoint are the nodular and lobulated conditions of the organ. The nodules vary in size from a split pea to a hen's egg, and are most frequently located along the suspensory ligament, in Glisson's capsule, at the portal fissure and along the main branches of the portal vein. The diagnosis of this condition depends upon an antecedent

FIG. 130



Syphilitic enlargement of the liver and spleen; multiple gummata of the liver; laparotomy and removal of one tumor for examination; cure by subsequent treatment (drawn from life). (Von Bergmann.)

history of syphilis and upon the improvement that follows from the administration of iodide of potassium. Sometimes these nodules become quite painful and sensitive, the pain being very similar to that of gallstone disease, and should there be a nodule at the gall-bladder site, it may be mistaken for a hard, contracted gall-bladder containing calculi. In such cases a previous history of syphilis, the presence of syphilitic lesions in the other organs (bones, skin, mucous membranes), the presence of other nodular masses in the liver, and the improvement which is obtained from anti-syphilitic treatment speak strongly in favor of the swelling

being a syphiloma. Should these nodules undergo softening or suppuration, the symptoms to which they would then give rise, viz., irregular fever, disturbances in the general health, and the presence of a painful, tender mass in the liver, would resemble very much those which accompany a chronic liver abscess; the differentiation is in most instances impossible without the aid of a definite antecedent history of syphilis or of an etiological cause for the liver abscess. If these cannot be elicited, an exploratory incision is the only way in which we can make a diagnosis.

An isolated lobule that is sometimes formed in a syphilitic liver as the result of the contraction of newly formed connective-tissue bands may resemble a floating kidney, for the differentiation of which the reader is referred to page 384.

Fibromata and *angiomata* give rise to symptoms only when they are large enough to cause pressure upon the neighboring structures. Examination then shows the presence of a rounded, firm, non-tender tumor.

Adenomata occur in the form of multiple nodes or as single tumors, which are well encapsulated. They may occasion toxic marasmus from pressure upon the bile-ducts. Attention is drawn to their presence when they produce pressure upon neighboring structures and interfere with general well-being.

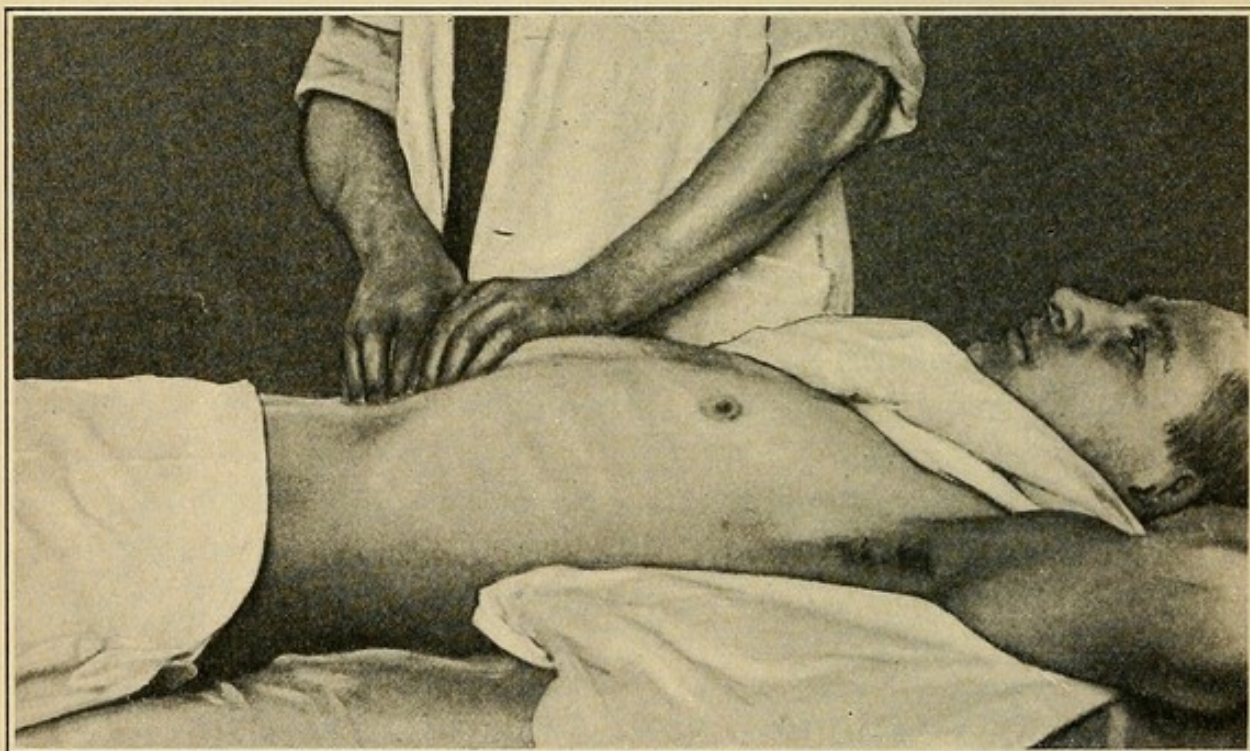
Carcinomata and *sarcomata* are usually metastatic tumors. Their presence in the liver adds to the cachexia resulting from the primary growth and causes icterus (usually from pressure on the bile-ducts), loss of flesh and strength, disturbed digestion, and ascites. The liver is enlarged and nodular, or it presents a single tumor which is painful and tender, and its anterior margin is much thickened.

CHAPTER XXIX.

DISEASES OF THE GALL-BLADDER.

THE gall-bladder normally occupies a position on the under surface of the right lobe of the liver, its fundus touching the anterior abdominal wall below the ninth costal

FIG. 131



Palpating gall-bladder : Examiner stands on right side of the patient, facing his feet, and palpates the under surface of the liver and gall-bladder region.

cartilage, just outside of the external edge of the right rectus muscle. Changes in the position of the liver or adhesions between the gall-bladder and adjacent viscera alter its position. This viscus can only be palpated when it is distended. To palpate, the examiner should stand on the right side of the patient, facing his legs, and press his fingers underneath the liver.

CHOLELITHIASIS.

Mainly through the efforts of Riedel, Courvoisier, and Kehr, and, for the most part, from the accurate data obtained by them from exploratory laparotomy, has the diagnosis of gallstone disease been placed upon a sound basis. We are no longer satisfied when we have established the fact that our patient suffers with cholelithiasis; we desire also to know the site of the stones, and whether any complicating diseases or conditions of the biliary apparatus are present.

While biliary calculi do occasionally form in the bile-ducts, their most frequent site of formation is in the gall-bladder, from which place they, as a rule, sooner or later wander into the ducts, there to be impacted or, as more frequently happens, to be passed on into the intestine. Their mere presence in the uninfected gall-bladder usually gives rise to no symptoms, but in some cases they occasion more or less severe pain, which is located in the epigastrium and radiates to the back and right shoulder, or is confined only to the back opposite the eleventh and twelfth dorsal vertebræ. As there are no physical findings to explain this pain, and because it is frequently aggravated by a heavy meal and is attended with gas and acid eructations, the diagnosis of gastric neuralgia, hyperacidity of the gastric juice, etc., is frequently erroneously made. But these are not diseases; they are merely symptoms; and if at first we are not able to positively decide upon their underlying cause, we should continue to carefully observe the patient until we have additional data upon which to base a diagnosis. In most cases of gallstone disease such data will sooner or later be forthcoming, either from the wanderings of the calculi into the ducts or from the addition of a secondary infection of the biliary apparatus.

Passage of Stones into Ducts.—The passage of a stone into or through the ducts into the intestine usually excites an *attack of biliary colic*, which is marked by severe cutting, colicky pain referred to the right hypochondrium and radiating to the back and right shoulder, by prostration, and by vomiting of gastric contents and bile. With it the temperature may be moderately elevated, and the liver slightly

enlarged; directly after it the patient becomes icteric, the stools clayey in color, and if they are carefully washed and sifted the calculi may be found.¹

Impaction of Stones in Ducts.—If the stone becomes impacted in the *cystic duct* and occludes it, or if the latter becomes obstructed in any other way—*e. g.*, by a kink or by contraction of surrounding adhesions, the gall-bladder usually, but by no means always, becomes distended; its distention is impossible if from previous inflammation and ulceration it has become shrunken and contracted and its walls thick and inelastic. A distended gall-bladder lies along a line drawn from the tip of the ninth costal cartilage downward parallel to the outer border of the rectus muscle. It forms a smooth, rounded, tender tumor that has respiratory and lateral mobility, and the dulness it affords on percussion is usually continuous with that of the liver. Jaundice is not present in such cases unless the distended gall-bladder compresses the common bile or hepatic ducts, or unless there is an infection of the ducts (cholangitis).

A distention of the gall-bladder without *icterus* points to cystic duct obstruction, the obstructing cause being usually a calculus, less frequently a kink or cicatricial stenosis. A distention of the gall-bladder with *icterus* points to compression or occlusion of the common bile-duct by a tumor. The reason for this latter will be seen later.

If the stone becomes impacted in the *hepatic or common bile-ducts* and causes their *sudden and complete occlusion*, there will be a gradually increasing *icterus*, emaciation, acholic stools, slight fever, and a tendency to hemorrhages from the mucous membranes. The gall-bladder is *not likely* to be *distended*, for, as a rule, the impaction of stones in the ducts goes with the later stages of gallstone disease, after there have been repeated attacks of cholecystitis, as a result of which the gall-bladder has become contracted. The liver may be enlarged and cirrhotic, but it is not tender.

If the stones in these ducts only *partially occlude* them or *act as ball valves*, sometimes occluding and sometimes not

¹ It is important to note that the calculi may not be passed in the stools for several days after the colic has subsided, hence the importance of examining the stools for at least a week after an attack.

occluding the channels, there may be no jaundice, or periodical jaundice, or jaundice of varying intensity, and the disturbances in the general health are only very slight. Jaundice may be *absent* with stones lodged in the *common bile-duct* under the following conditions: Either they are too small to entirely obstruct the channel, or the cystic duct is patent and a pathological fistula exists between the gall-bladder and intestine.

Infection of the Biliary Apparatus.—The presence of stones in the biliary apparatus renders this much more likely to infection. The wanderings of the stones are by many attributed to such a mixed or secondary infection; but however this may be, the other clinical evidences of such infection are very pronounced.

If the *infection* is *limited* to the *gall-bladder*, and the *cystic duct* is *coincidentally occluded* either by a stone that has wandered into it or by the inflammatory swelling of its mucous membrane, the symptoms will vary with the virulency of the infecting organism and with the severity of the inflammation.

Acute Cholecystitis.—A *mild inflammation* is attended with moderate fever, somewhat increased pulse rate, pain, and distention of the gall-bladder. *With the severer forms of inflammation* the fever is higher (103° to 104°), the pulse rate more rapid, there is vomiting, severe pain, and considerable gall-bladder distention. *In the severest cases*, with local or general gangrene of the gall-bladder, and local or diffuse peritonitis, the patient is very sick; the expression is drawn and anxious, the tongue is dry, the pulse rapid, the temperature 104° or over, and the abdomen distended; there is severe pain over the gall-bladder, with considerable distention of the viscus and local or general abdominal rigidity. *With perforation or complete gangrene* of the gall-bladder a septic peritonitis ensues. With the mild grade of infection of the gall-bladder the viscus is filled with mucus and bile and serum (acute hydrops); with the severer grades it is filled with bile and pus (acute empyema).

The milder grades, and even some of the severer ones, subside spontaneously if the obstruction to the cysticus is relieved by the passage of the stone into the intestine, or the

falling back of the stone into the gall-bladder. Pericholecystitic adhesions or ulcerations of the gall-bladder remain after the severer forms. A mixed infection of the gall-bladder having once taken place, recurrent attacks are sure to occur, until the gallstones are removed and the viscus drained, and thereby its infection relieved. The ulceration and subsequent cicatrization which result from recurrent attacks of acute cholecystitis finally bring about a contraction of the gall-bladder, and a thickening and inelasticity of its walls.

Chronic Cholecystitis.—The contraction of adhesions may cause a kink in the cysticus; likewise the cicatrization of ulcers near the cystic orifice of the gall-bladder may result in stenosis or permanent closure of the cystic duct. The mucus and serum and pus which is secreted by the infected mucous membrane cannot then escape, and chronic hydrops or chronic empyema of the gall-bladder results. With these chronic forms of cholecystitis there are very slight constitutional symptoms. The distended gall-bladder occasions a feeling of weight and pain.

It is to be *especially noted* that *no jaundice accompanies acute or chronic cholecystitis*, unless the *distended viscus compresses the common bile or hepatic ducts*.

Cholangitis.—If the infection is located in the ducts, a cholangitis develops.¹ This may occur together with or independently of a cholecystitis. If it is acute and septic in character, it occasions repeated chills, intermittent or remittent temperature elevations, sweating, jaundice, rapid emaciation, and a tendency to bleeding from the mucous membranes. The stools are not completely acholic. The liver becomes much enlarged, tender, and painful, and may contain small abscesses.

Ulceration of Stones Through Biliary Channels.—One other complication may result from biliary calculi. When they are confined in the gall-bladder or ducts, they may ulcerate through these structures into a neighboring hollow

¹ It is to be especially noted that the infection of the gall-bladder and bile-ducts may remain after the calculi have passed. This fact will explain those increasingly frequent cases in which a diagnosis of gallstones with cholecystitis or cholangitis was made, and yet no stones were found on opening the abdomen, but only an acute or chronic cholecystitis or cholangitis.

viscus—*e. g.*, the stomach, duodenum, intestines or kidney, or into the peritoneal cavity. There may be no evidences of such ulceration, or there may be signs of a local adhesive peritonitis. Once the stone has passed into a neighboring viscus, it may give no further trouble, or if it is very large it may obstruct the channel of its new habitat. (See Intestinal Obstruction from Gallstones.)

The diagnosis of gallstone disease is thus seen to depend on the presence of pain in the right hypochondrium or epigastrium or in the back opposite the eleventh and twelfth dorsal vertebræ, on the occurrence of attacks of biliary colic with the passage of stones in the stools, on the presence of jaundice and of fever. No aid is to be obtained from the *x*-ray examination, for biliary calculi rarely cast shadows.

It is important to remember that jaundice in gallstone disease occurs only from obstruction or compression or inflammation of the primary and subsidiary hepatic ducts or common bile-duct, and that when the calculi are confined to the gall-bladder or cystic duct, there is *no jaundice*, unless the distended gall-bladder presses upon the hepatic or common bile-ducts, or unless there is a cholangitis. Further, it is important to note that the gall-bladder is not always distended in calculus disease. Distention of the gall-bladder points to obstruction of the cystic duct; a gall-bladder with elastic walls distends so as to accommodate the stagnant contents, but a gall-bladder whose walls are infiltrated, thickened, and cicatricial from repeated attacks of inflammation is inelastic and cannot distend.

Determination of Site of Stones.—The location of the stones is to be determined from the condition of the gall-bladder and from the presence or absence of jaundice and clayey stools; the existence of complicating inflammation of the gall-bladder and ducts is to be determined from the constitutional symptoms and from the presence of a distended, painful, exquisitely tender gall-bladder, or of an enlarged, painful and tender liver.

If the stones are located in the gall-bladder and the cystic duct is open, there will be only pain and attacks of colic; the gall-bladder is not distended. *If the cystic duct is obstructed* by a calculus or by a kink or by cicatricial stenosis,

the gall-bladder is distended. There is no jaundice unless the distended viscus presses upon the hepatic or common bile-ducts, or unless there is a cholangitis present.

Firmly impacted stones in the choledochus or hepaticus cause, in addition to the pain and colic, an increasing jaundice with acholic stools; *with ball-valve stones* there may be no jaundice or intermittent jaundice or jaundice of a varying intensity; the color of the stools varies according to the presence and degree of jaundice. The gall-bladder is not likely to be distended in either case, because preceding attacks of cholecystitis have rendered its walls rigid, inelastic and contracted, and incapable of yielding to the increasing pressure of the dammed-up bile.

Those cases of cholelithiasis that only occasion *cholecystitic pain* must be differentiated from cases of *lead colic*, *gastric ulcer*, *kidney colic*, and *intestinal colic*. If the individual is a painter or worker in lead and has a blue line on the gums, and confesses to neglect in the care of his hands and clothing before eating, we are safe in diagnosing a lead colic. If the pain is aggravated by eating, is attended with a gastric ulcer "Head" zone, with hyperacidity and possibly with hæmatemesis, a gastric ulcer is probably its cause. If it is attended with frequent urination, possibly with hæmaturia, and radiates downward into the groin, testicle, and thigh, and if the cystoscope reveals changes in the ureteral orifice (for which see p. 374), a kidney stone or other abnormal kidney condition is probably present. In intestinal colic the pain is more general over the abdomen, and is relieved by a cathartic or high enema.

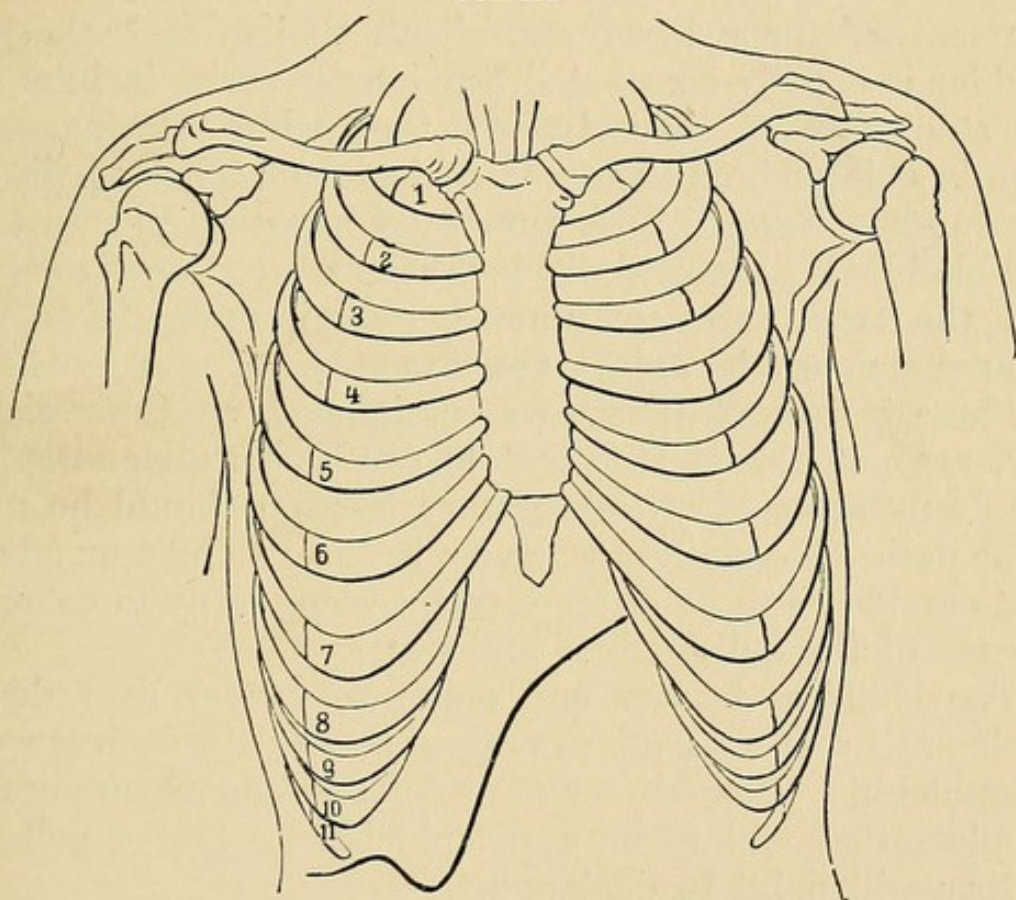
As the formation of biliary calculi is favored by biliary stasis and excited by an infection of the biliary passages with bacteria of diminished virulence, the presence of conditions favoring biliary stasis—*e. g.*, nephroptosis, or enteroptosis, and the history of a preceding systemic infection—*e. g.*, by the typhoid bacillus or other organisms whose elimination from the blood is accomplished by the liver and carried by the bile into the bile passages, are points in favor of calculous disease of the biliary organs.

Those cases that exhibit cholecystitic pain plus cholecystitis and a distended gall-bladder must be differentiated

in their acute stage from *acute appendicitis*. Gall-bladder disease is distinguished by the history of previous attacks of colic with or without fever or jaundice, and by the pear-shaped tumor, with its respiratory and lateral mobility and its close relations to the liver.

A preceding history of pain and colic distinguishes a tumor due to a *chronic hydrops* or *empyema* of the *gall-bladder*. The tumor itself differs from kidney tumors in that

FIG. 132



Reidel's lobe of liver, frequently present in gallstone disease, and often spread out over an enlarged gall-bladder.

it has not the characteristic kidney shape, it lies above the colon and not behind it, and it cannot be fully replaced into the loin. By distention of the stomach and by chemical examination of the gastric contents tumors of the stomach may be differentiated from enlargements of the gall-bladder.

Echinococcus cysts can be differentiated from enlarged gall-bladders by their history and their slow growth, and *syphilomata of the liver* by an antecedent history of syphilis,

the multiplicity of the nodules, the thickening of Glisson's capsule with the consequent rounding out of the sharp anterior border of the liver, and by the improvement from antispecific treatment. Riedel lays great stress on the presence of a tongue-shaped lobe of the liver at the site of the gall-bladder as pointing to a hydrops of the viscus.

Cholelithiasis with obstructive jaundice, the stones being impacted in or floating up and down in the common bile-duct, is to be differentiated from *obstructive jaundice due to compression of the ducts by tumor*—*e. g.*, of head of the pancreas, of the pylorus, etc. With cholelithiasis the gall-bladder is usually contracted, the icterus varies in intensity, the stools are at times brown, then white, the spleen is slightly enlarged, and there is a history of colicky pain and intermittent fever. With tumor compressing the duct the gall-bladder is distended, the icterus grows constantly deeper, and the stools are continuously white; colic, fever, and enlargement of the spleen are absent.

These points of difference, designated as Courvoisier's law, are not always sufficient to establish a diagnosis. In the doubtful cases an exploratory incision should be made if the patient's condition warrants it. It is to be remembered that carcinoma quite often develops secondarily to calculous disease of the gall-bladder and ducts.

The chills and fever and enlarged, tender liver due to cholangitis may simulate pyæmic abscesses of this organ with pylephlebitis. The history of an intraperitoneal suppuration or ulceration will favor a pylephlebitis, while a gallstone history will point to cholangitis.

CARCINOMA OF THE GALL-BLADDER.

Malignant disease of the gall-bladder frequently develops after long-standing cholelithiasis. It appears as a hard, irregular, rapidly growing tumor at the site of the gall-bladder. The patient emaciates rapidly, ascites develops early and especially if the glands at the portal fissure of the liver are enlarged and press upon and occlude the portal vein. The cystic and common bile-ducts become obstructed,

with consequent icterus, acholic stools, and more rapid emaciation and fever.

The differential diagnosis from cholelithiasis is to be made from the hardness and irregularity of the tumor, the icterus, cachexia, and ascites. Unless the carcinoma follows cholelithiasis, there are usually no colicky pains.

CARCINOMA OF THE BILE-DUCTS.

The usual sites of this affection are either at the papilla of Vater or at the cystic and hepatic junction. According to Courvoisier's law the gall-bladder becomes distended; the icterus appears slowly, but grows steadily deeper. The pain is slight, the liver is enlarged; and finally becomes the seat of biliary cirrhosis. There is rapid emaciation and ascites.

The differential diagnosis from impacted calculus in the choledochus is made according to Courvoisier's law; colicky pain and fever and enlarged spleen being absent, and the gall-bladder being distended.

CHAPTER XXX.

DISEASES OF THE PANCREAS.

THE pancreas lies transversely across the abdomen behind the peritoneum, at the level of the body of the first lumbar vertebra. If the organ is enlarged upward it pushes the retroperitoneal peritoneum and the gastrohepatic ligament forward and approaches the anterior abdominal wall between the liver and the lesser curvature of the stomach; if the enlargement is forward it pushes the stomach upward, the transverse mesocolon downward, and the gastrocolic ligament forward, and appears between the stomach and colon; and if the enlargement is downward it separates the layers of the transverse mesocolon and appears below the transverse colon, being covered by the great omentum. Should the enlargement downward be very marked, the descending colon would thereby be raised up from the posterior abdominal wall. Enlargements of the pancreas are due chiefly to cysts, neoplasms, and inflammation.

Our appreciation of the clinical manifestations of pancreatic disease and neoplasms, and our ability to recognize these conditions have been materially advanced by the experimental labors and researches of Fitz, Körte, Mikulicz, Opie, Mayo Robson and others; and while we are still far from being able to make a certain diagnosis of these maladies, we are nevertheless in a position to suspect their presence, and if we exclude the diseases which in their clinical course and symptomatology resemble the disorders of this organ, we will be able in many cases to arrive at a correct diagnosis.

There is one characteristic lesion of the acute hemorrhagic, gangrenous and some of the more chronic diseases of the pancreas, and that is spots of necrosis in the fat surrounding the organ, in the fat of the omentum, and also, though

with less frequency, in the subpleural and subpericardial fat. These spots are small, opaque, and white, and are probably due to a diffusion of the pancreatic secretion with its fat-splitting ferment into the surrounding tissue.¹ Unfortunately, we are not able to recognize this fat necrosis before opening the abdomen, but we should not neglect to look for it in all doubtful cases of abdominal disease when the abdomen has been opened, and if it is present we may be assured that a grave disease of the pancreas is present.

INFLAMMATION OF THE PANCREAS.

Acute Inflammation.—The onset and course of pancreatic hemorrhage, acute hemorrhagic pancreatitis, and necrosis of the gland are eminently acute and rapid. The presence of these maladies should always be suspected where, with a previous history of gallstone disease or gastrointestinal disorders, there is a sudden onset of severe epigastric pain, vomiting, constipation, abdominal distention and extreme prostration, with rapid pulse and normal or moderately elevated temperatures. Opie considers that these three conditions are different grades and stages of the same pathological process, and that they are due to the action of irritating and destructive agents, chief among which is the bile, upon the gland. This, he believes, gains access to the gland when the papilla of Vater is obstructed by a calculus that is large enough to occlude the orifice of the latter and so prevent the entrance of the bile into the intestine, but is not of sufficient size to completely fill the ampulla of the common bile and pancreatic ducts, and so prevent the reflux of bile along the pancreatic duct into the pancreas. The significance of a history of gallstone disease in connection with the acute forms of pancreatitis will according to this be appreciated.

The onset and initial symptoms of *acute intestinal obstruction* and of *acute appendicitis* resemble very much those which attend these forms of acute pancreatitis. But the initial prostration is much more intense in the latter affection.

¹ The extent of the diffusion depends on the duration of life after the onset of the pancreatic disease.

With intestinal obstruction there is absolute constipation and persistent vomiting, whereas in acute pancreatitis high enemata withdraw foul gases and even fecal particles from the bowel. With acute appendicitis the pain in the beginning of the attack is centred around the umbilicus or is general in the abdomen, and later becomes localized in the right iliac fossa, and the right rectus muscle is rigid; while with acute pancreatitis the pain at the outset is in the epigastrium, which region is tender and rigid. A history of gallstone disease would speak in favor of acute pancreatitis.

Should the patient survive this acute stage of the disease, he would then go on to present the evidences of a pancreatic hemorrhage and necrosis. With local necrosis and with complete sequestration of the gland the course is subacute, and is marked by moderate elevations of temperature, occasional vomiting, constipation, free fluid in the abdomen, emaciation, loss of strength and bronzing of the skin, and by the appearance of a swelling in the epigastric region corresponding to the position of the pancreas. This swelling is retrogastric and has no respiratory mobility, and is tender and smooth. If this swelling breaks down into pus, the latter may gravitate downward, especially to the left, or more rarely between the layers of the transverse mesocolon.

Such a swelling may be mistaken for a neoplasm on the posterior wall of the stomach, but the clinical history is entirely different in these two affections, and, furthermore, the lack of respiratory mobility of the tumor, and the absence of any marked change in the chemical composition of the gastric juice, together with the absence of cachexia, readily enable us to exclude a gastric new-growth.

A less intense onset of epigastric pain, nausea, vomiting and constipation, and a more subacute course which is marked by moderate fever, emaciation, occasional vomiting, abdominal distention, constipation, the development of free ascites, and by the appearance of a tender swelling in the epigastrium characterize the acute suppurative forms of pancreatitis. The swelling is retrogastric, has no respiratory mobility, and is very tender. The leukocytes are increased in number. A history of preceding gallstone disease is also suggestive of this form of pancreatitis, for the infection of

the bile-ducts that is so commonly present with biliary lithiasis is quite likely to extend up into the radicles of the pancreatic ducts.

Chronic Inflammation.—The forms of chronic inflammation of the pancreas that are attended with enlargement of the head of the organ are especially important diagnostically because of the resemblance their clinical manifestations bear to malignant disease of the pancreas, pylorus, duodenum, and gall-bladder.

In all of these affections the patients complain of gastrointestinal disturbances, such as nausea, vomiting, and constipation; they are emaciated and usually icteric, and on examination we find a hard, nodular tumor, somewhat to the right of the epigastrium, and if the patient is icteric a distention of the gall-bladder is present.

Evidences of disturbed gastric motility, such as dilatation of the stomach, prolongation of the time the organ requires for emptying itself after a meal, and stagnation of the contents within the stomach, combined with an absence of free hydrochloric acid and ferments from the gastric juice, and the presence of lactic acid therein, enable us to distinguish neoplasms of the pylorus and duodenum from pancreatic tumors. The more superficial location of the tumor and an earlier appearance of icterus help to differentiate the neoplasms of the gall-bladder. But the tumor which is due to carcinoma of the head of the pancreas cannot in most instances be differentiated from that due to a chronic inflammation of this part of the organ without the aid of exploratory laparotomy, and in quite a number of cases the surgeon is unable to decide between the two affections even when he has the parts directly accessible to inspection and palpation.

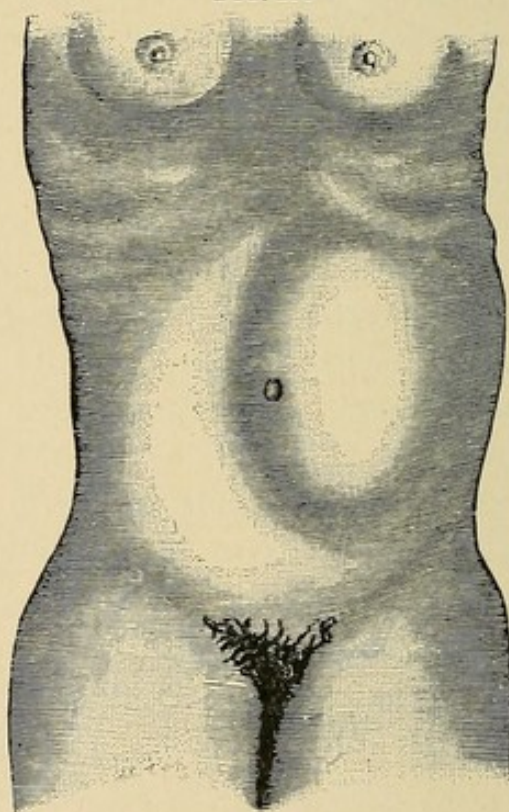
The presence of sugar in the urine and of fat in the stools is an infrequent occurrence in these forms of pancreatic disease, but when they do exist they afford additional evidences of such malady.

The differentiation of tumors, especially those of the pancreas that cause obstructive jaundice from biliary lithiasis with obstructive jaundice, has already been considered. (See pp. 310 and 324.)

TUMORS OF THE PANCREAS.

Cysts.—A pancreatic cyst manifests itself by the symptoms of gastric catarrh—viz., loss of appetite, nausea, vomiting of gastric and biliary matter or of blood, constipation or diarrhœa, together with the presence of a retroperitoneal tumor that is tense and fluctuating, but not tender, that usually has no respiratory mobility, and that has definite and special relations to the stomach and transverse colon. These tumors are of slow growth and the gastric disturbances may exist for some time prior to the development of a palpable tumor.

FIG. 133

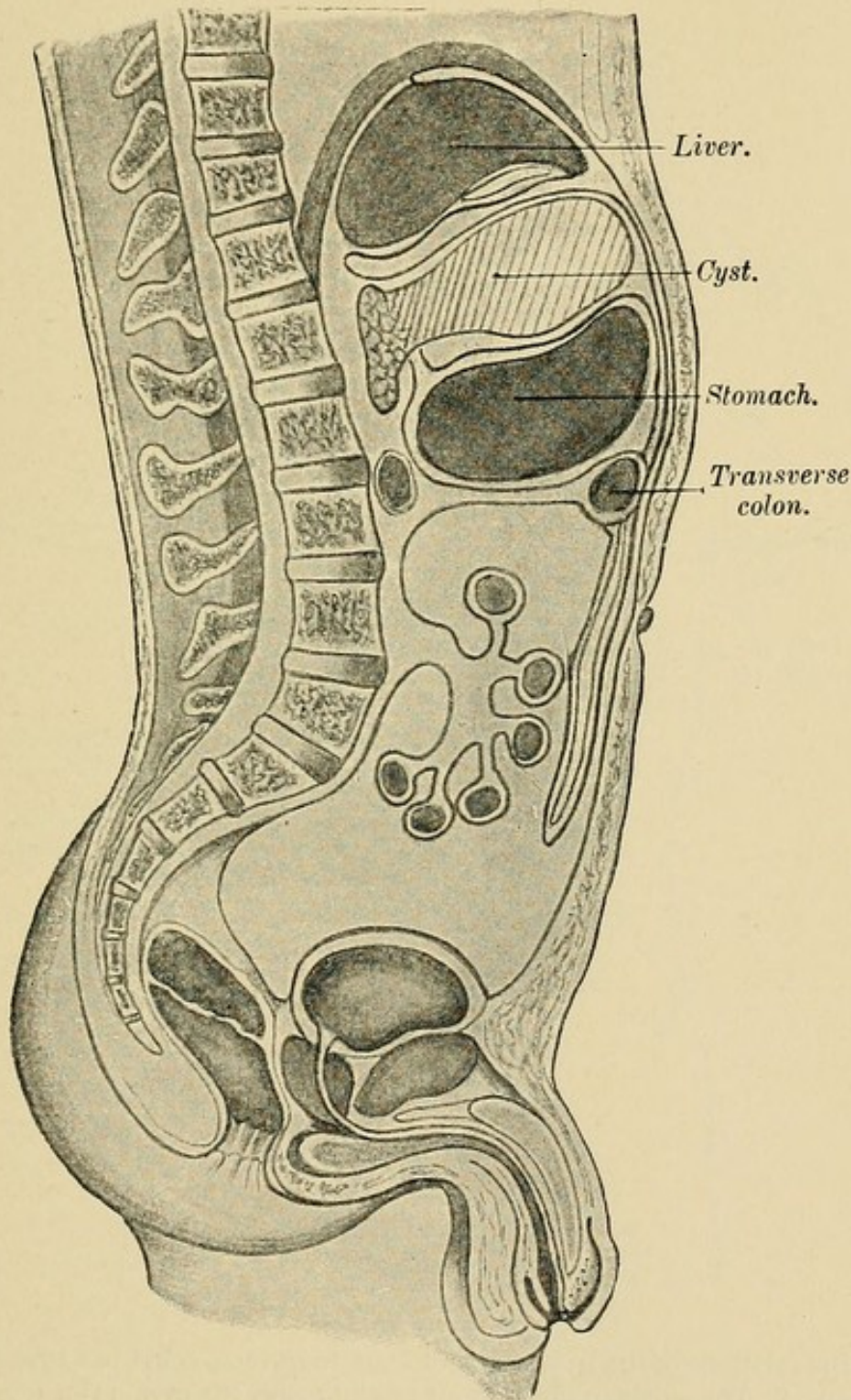


A pancreatic cyst, type C. (Flaischlen.)

The relation which the cyst bears to the stomach and transverse colon is its important and distinguishing characteristic. At first it is retroperitoneal and usually lies behind the stomach; as it grows it may project upward, push the hepatogastric ligament (lesser omentum) forward, and approach the anterior abdominal wall above the lesser curvature of the stomach (type A). Larger cysts of this class drop down in front of the stomach, and in their anterior gas-

tric portion they enjoy considerable respiratory mobility. A second class of cysts (type B) push directly forward between

FIG. 134

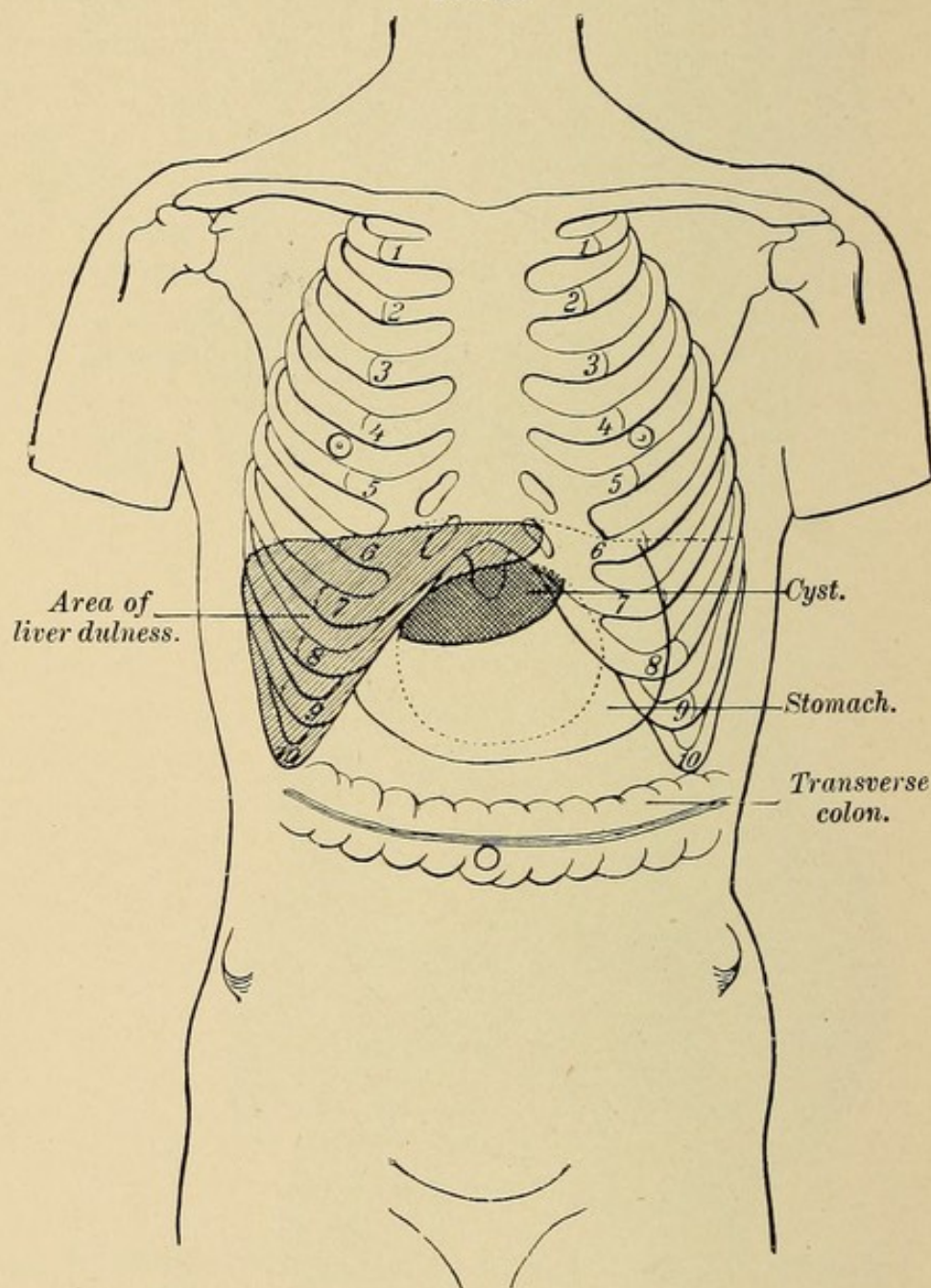


Cyst of the pancreas developing between the stomach and liver, type A. The lesser omentum, and possibly the foramen of Winslow, is crowded forward by the cyst. (Von Bergmann.)

the stomach and transverse colon, being covered by the gastrocolic ligament; and a third class (type C) bulge downward,

pushing the lower layer of the transverse mesocolon downward, and appearing below the transverse colon, being covered by the greater omentum. These relations of the

FIG. 135

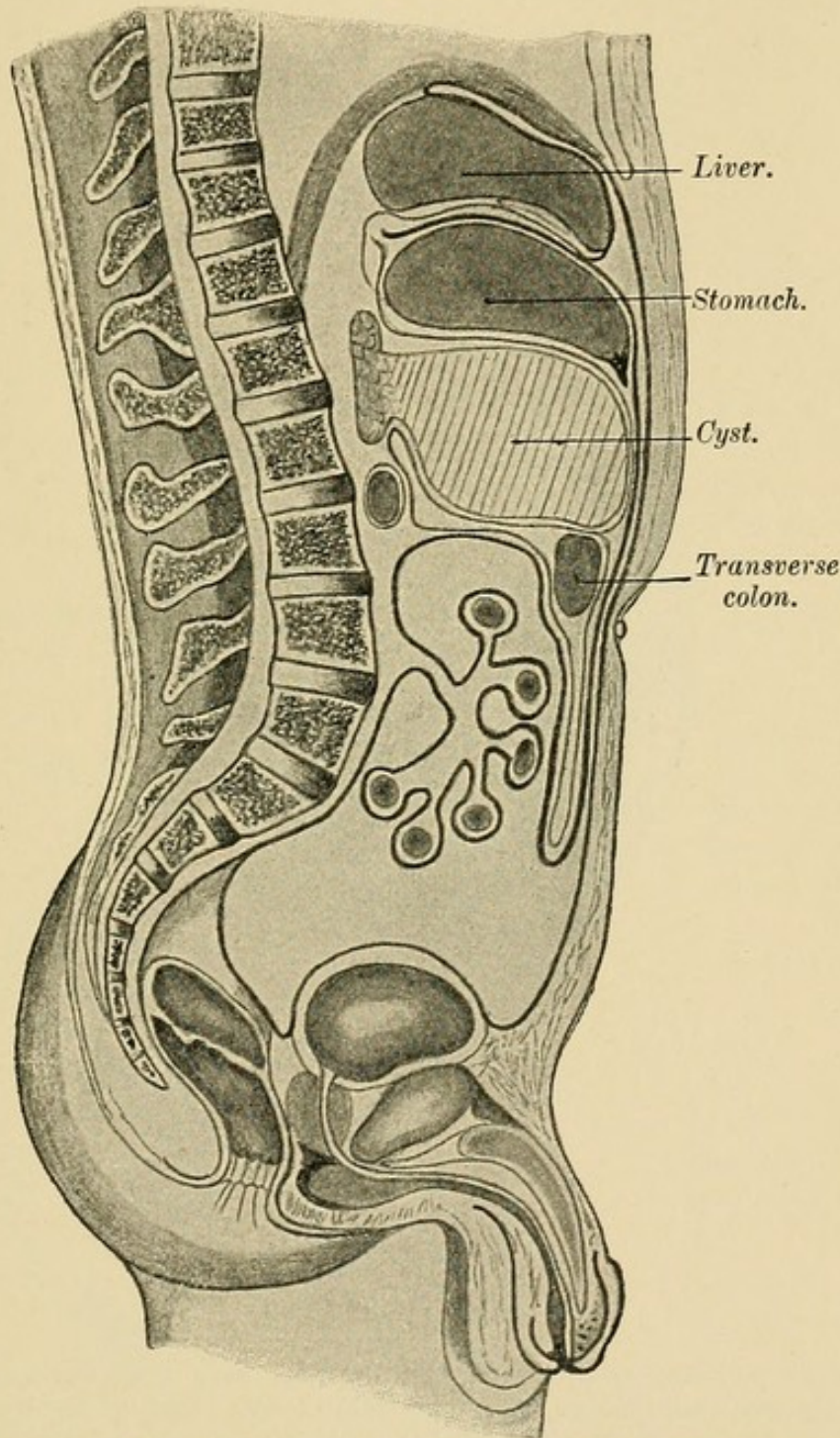


Showing the relations of the liver, stomach, and transverse colon to a cyst of the pancreas, type A. The stomach lies in front of and below the cyst. (Von Bergmann.)

tumor to the stomach and transverse colon are so important for establishing a diagnosis that we should never neglect in cases of abdominal cystic tumors to distend both these viscera and determine their position in reference to the growth.

The presence of undigested proteid matter or of fat in the stools, or the presence of sugar in the urine are to be

FIG. 136

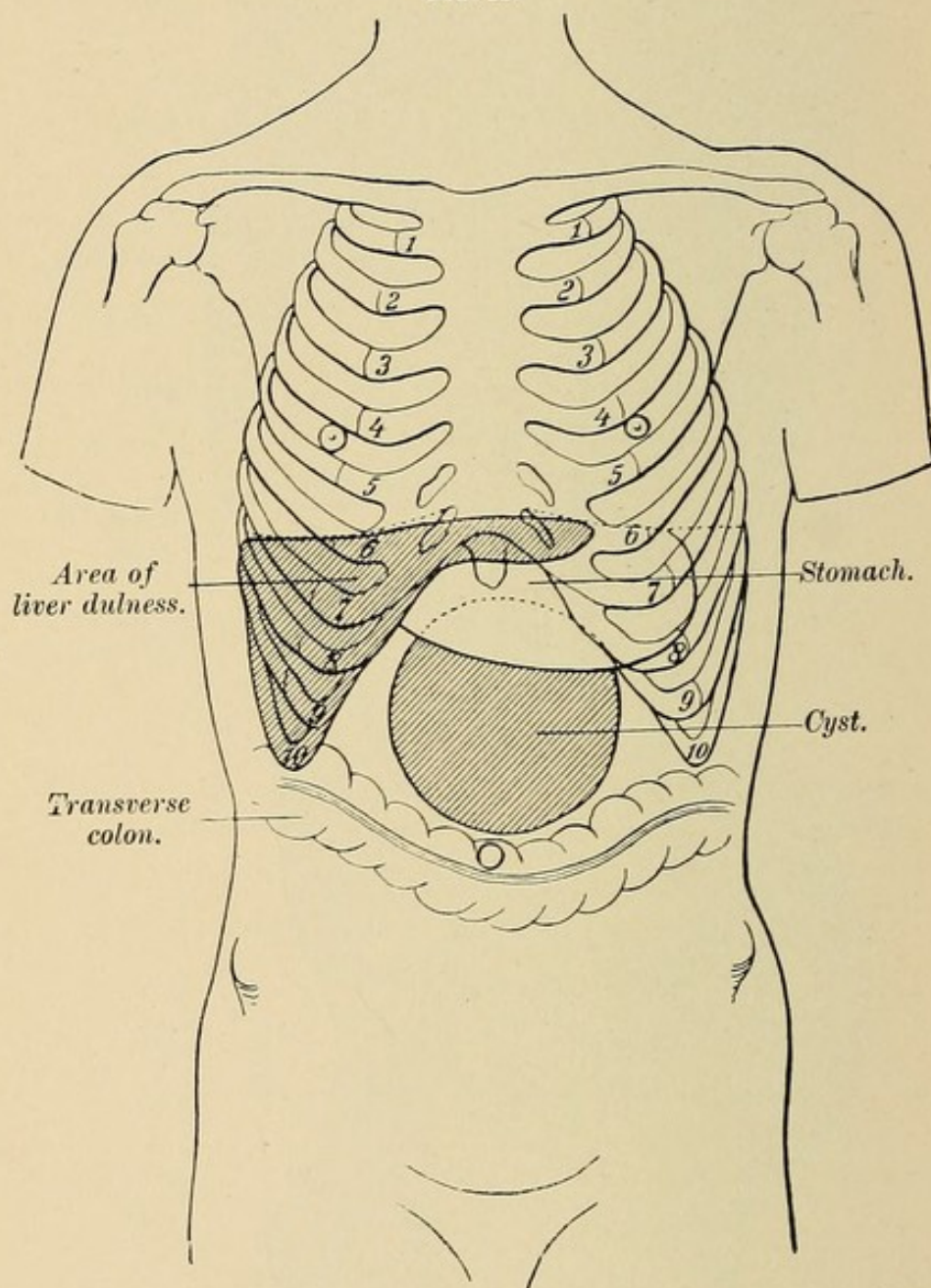


Cyst of the pancreas developing between the stomach and transverse colon, type B.
The gastrocolic omentum lies in front of the cyst. (Von Bergmann.)

looked upon as confirmatory evidences of a pancreatic cyst. Their absence does not speak against the cyst being of pancreatic origin.

Disturbances of gastric motility leading to dilatation of the organ and stagnation within it, or the evidences of obstructive jaundice, hydronephrosis or ascites, are the con-

FIG. 137

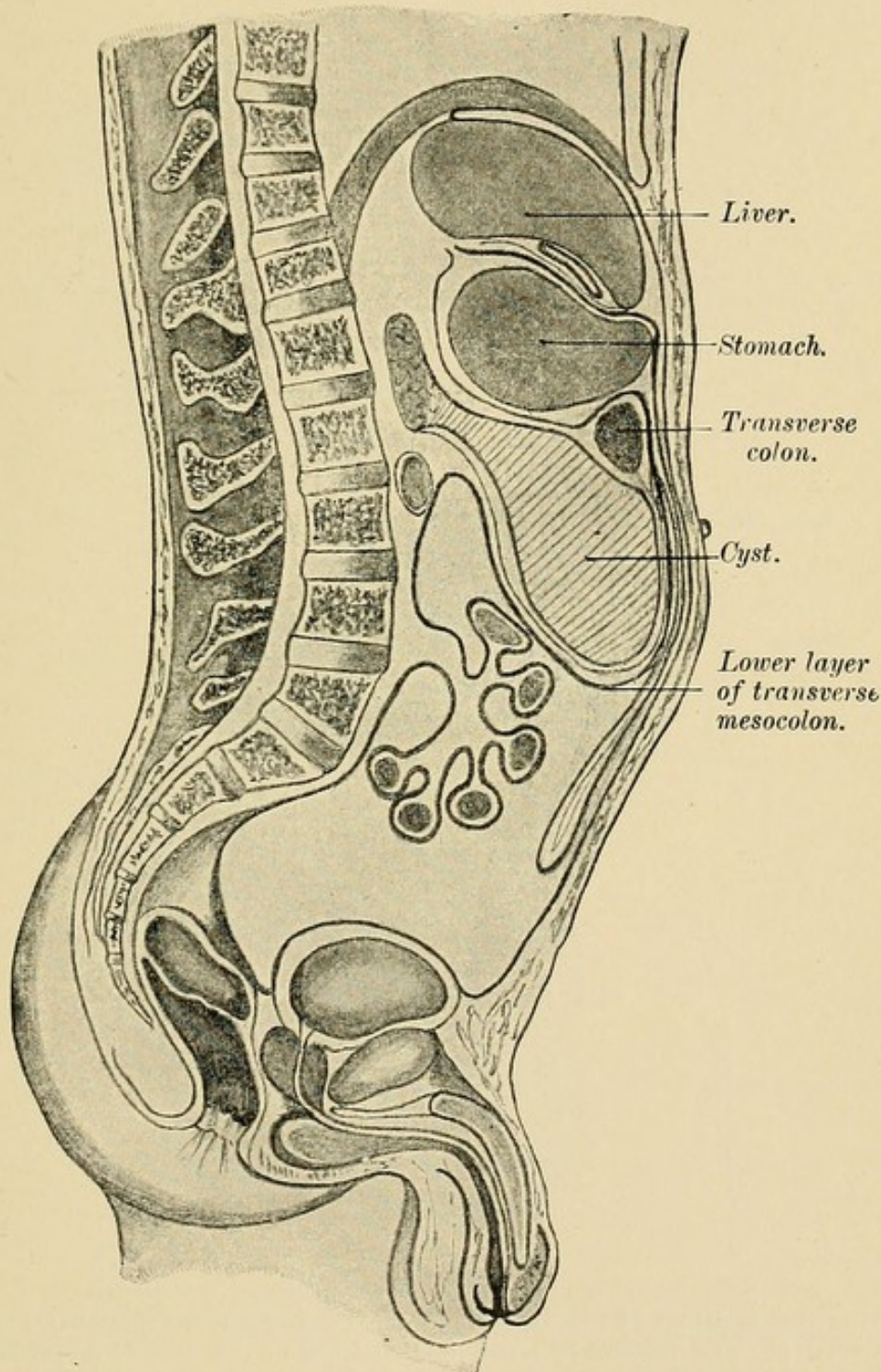


Showing the relations of the liver and transverse colon to the tumor caused by a typical pancreatic cyst, type B. The stomach is displaced upward and partially overlaps the cyst. The transverse colon follows its lower margin. (Von Bergmann.)

sequences of pressure exercised by any large abdominal tumor upon the stomach, the common bile-duct, the ureter or the portal vein; these symptoms do not indicate the pancreatic origin of a tumor.

The composition of the cyst contents will help to differentiate cystic tumors that are surrounded by such

FIG. 138

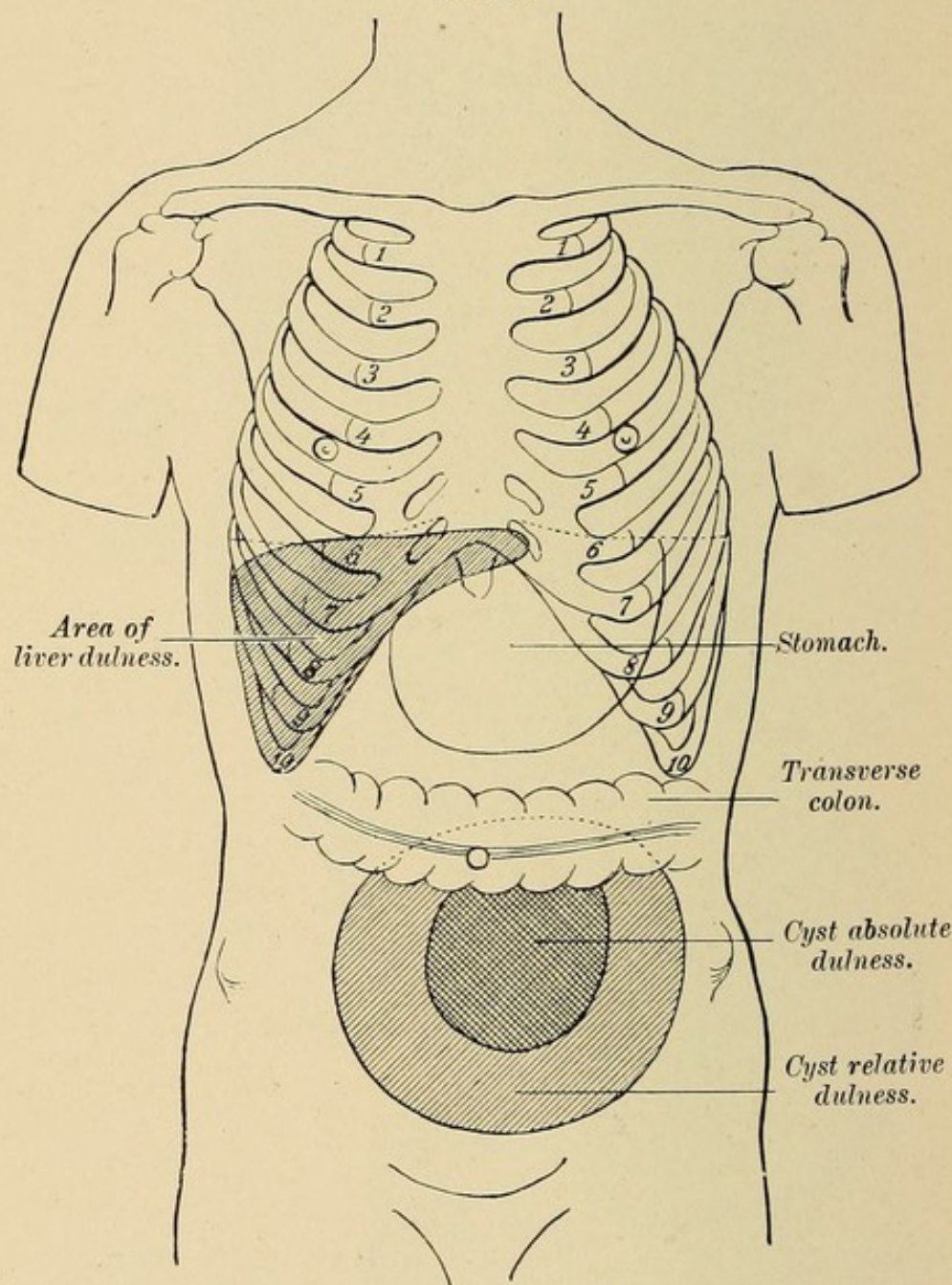


Cyst of the pancreas developing between the layers of the transverse mesocolon, type C, and stretching the lower layer more than the upper. (Von Bergmann.)

dense adhesions as to make their origin uncertain, even after the abdomen has been opened. These cyst contents are

cloudy, brownish in color, of syrupy consistency, occasionally colloid or purulent. They are usually mixed with some fluid or clotted blood; they are alkaline, of 1007 or 1028 specific

FIG. 139

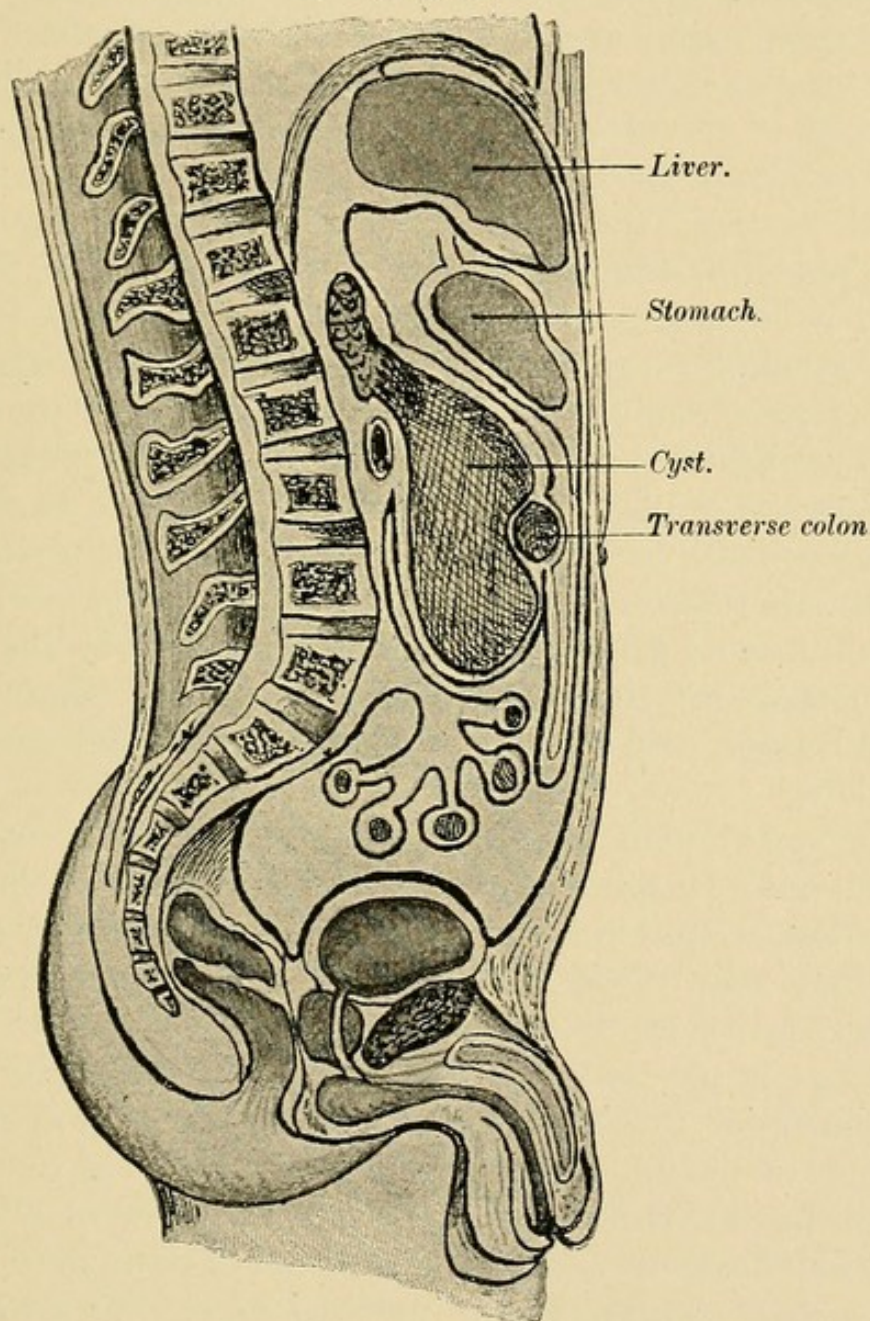


Showing the relations of the liver, stomach, and transverse colon to a pancreatic cyst of type C. The transverse colon lies in front of the cyst or along its upper border. (Von Bergmann.)

gravity, have some albumin, rarely any sugar, and sometimes cholesterin. Ferments are not always present; sometimes there is diastase, and again trypsin and steapsin.

The cause underlying the formation of a pancreatic cyst is often to be gleaned from the previous history of the patient. Thus trauma, or the acute infectious diseases, or chole-

FIG. 140



Cyst of the pancreas developing between the layers of the transverse mesocolon. The colon lies directly in front of the cyst. (Von Bergmann.)

lithiasis or hemorrhage into the pancreas are the most frequent causes, and in most instances the patient will give a history of trauma or present the evidences of the previous or present existence of these maladies. A chronic pancreatitis

or an obstruction of the duct of Wirsung by a calculus that has formed within it are less frequent causes for pancreatic cysts. The symptoms afforded by pancreatic calculi would not differ in any way from those of biliary calculi.

Pedunculated cystic tumors of the liver, and especially the echinococcus cysts, are distinguished from pancreatic cysts in that they have respiratory and independent mobility and lie in front of the stomach, whereas the pancreatic cysts lie behind or above or below this organ. Liver cysts remain attached to the liver even when the patient assumes the upright position, whereas pancreatic cysts fall away from it. When it is impossible to make the differentiation it is better to make an exploratory incision than an exploratory puncture.

A hydrops of the gall-bladder is distinguished from a pancreatic cyst by the pear-shape of the tumor, its proximity to the abdominal wall, and its free mobility. It is attached to the liver and lies to the right and in front of the stomach, whereas pancreatic cysts lie behind the stomach.

Splenic tumors are differentiated from cysts in the tail of the pancreas by their characteristic splenic shape, their notched border, and by their pushing the stomach and colon to the left.

Aortic aneurysms are distinguished by their expansile pulsation and systolic murmur. We must note, however, that a pulsation and murmur are to be heard over any abdominal tumor that compresses or lies upon the aorta; such pulsation, however, is *not* expansile.

A hydro- or pyonephrosis differs from a pancreatic cyst in that it occupies the loin, with the distended colon lying in front and to its inner side; when the tumor is very large, the colon lies to its outer side. Cysts lie below or above the colon. Furthermore, in kidney disease cystoscopic examination reveals changes in the ureteral mouth, and the urine which is drawn by ureteral catheterization from the corresponding kidney will, as a rule, show abnormalities. The patient is also apt to suffer from urinary disturbances.

Mesenteric cysts usually lie below the umbilicus; *ovarian cysts* grow from below, and by vaginal or rectal palpation we can feel the pedicle that connects them to the ovary. In either case the relations of the stomach and colon to the

tumor will usually enable us to differentiate them from pancreatic cysts.

Malignant growths of the pancreas are distinguished from cysts by their hardness, irregularity, and rapid growth; they are furthermore attended with cachexia and do not fluctuate. Chronic abscesses of the pancreas are more tender than cysts, and are accompanied by fever and emaciation. Their course is much more rapid.

MALIGNANT TUMORS OF THE PANCREAS.

The evidences afforded by a malignant growth (usually carcinoma) of the head of the pancreas are a tender, hard, smooth or irregular tumor to the right of the vertebral column and behind the stomach, together with epigastric pain, early cachexia, and ascites. Obstructive jaundice from compression of the choledochus develops in most cases, and when it does the gall-bladder is felt to be distended. Glycosuria and fat in the stools are much less frequent symptoms.

The absence of a palpable tumor and the paucity of the physical sufferings preclude the possibility of making a diagnosis during the early stages of the disease. Many of these patients first present themselves to the physician when they become jaundiced, and at such a time the differentiation must be made between obstructive jaundice due to biliary calculi, and that due to chronic inflammatory and malignant disease of the pancreas.

With *calculous disease* there is a preceding history of cholecystitic pain, biliary colic, attacks of jaundice, and, possibly, temperature elevations. The gall-bladder is tender and cannot, as a rule, be palpated, and the liver and spleen are enlarged. While the patients are much emaciated, they are not cachectic. (See Courvoisier's law, pp. 319-322.)

The chronic inflammatory forms of pancreatitis cannot with any degree of certainty be differentiated from malignant disease of the organ even when the abdomen has been opened and the parts are directly accessible to palpation. An absence of cachexia and the existence of fever speak for chronic pancreatitis; ascites is indicative of carcinoma.

Cancer of the pylorus is to be distinguished from cancer of the pancreas-head by the disturbed gastric motility and the chemical changes in the gastric secretion—viz., an absence of free hydrochloric acid and ferments, and a presence of lactic acid—that attend it.

The evidences of chronic intestinal stenosis, erections of the colon, and dilatation of the caput coli distinguish colon cancers from neoplasms of the pancreas.

Cancer of the papilla of Vater or of the choledochus can only be differentiated from pancreatic neoplasms by exploratory laparotomy.

PANCREATIC CALCULI.

The clinical evidences afforded by these stones are scarcely different from those which are occasioned by biliary calculi. Glycosuria and fat in the stools speak strongly for pancreatic as against biliary calculi. Should a history of colicky pain in the epigastrium precede the development of a cystic tumor of the pancreas, the chances would be very great that the latter was a true retention cyst from obstruction of the duct of Wirsung by a calculus.

CHAPTER XXXI.

DISEASES OF THE SPLEEN.

THE spleen normally lies in the left hypochondrium. It extends from the ninth to the eleventh ribs, its long axis being parallel to the tenth rib. Its posterior border lies near to the vertebral column and its anterior border reaches to the mid- or anterior axillary line.

MOVABLE SPLEEN.

A movable spleen manifests itself by a dragging pain in the left hypochondrium, which radiates to the thigh, and by the evidences of compression of the neighboring viscera—*e. g.*, strangury from compression of the kidney, constipation or even ileus from compression of the intestine. The organ can be recognized by its notched anterior border and hilus; it may be of normal size or it may be considerably enlarged—*e. g.*, from malaria, leukæmia, or neoplasm.

A floating kidney is easily differentiated from the spleen because it has not the splenic shape or notched anterior border.

The possibility of such a movable organ suffering a torsion of its pedicle should be remembered, so that the pain and peritonitis to which this accident gives rise should be attributed to its proper cause.

ABSCESS OF THE SPLEEN.

Abscess formation in this organ is to be suspected if it becomes swollen and tender, and if there are constitutional

evidences of pus accumulation. Fluctuation is only to be elicited in the large abscesses. The acute infectious diseases, typhoid, rheumatism, and pyæmia, are the most frequent causes of this condition. In typhoid the presence of a splenic abscess should be suspected if, after the subsidence of the fever, the organ becomes swollen and tender and the fever rises again.

RUPTURE OF SPLEEN.

Rupture of the spleen is predisposed to by the pathological conditions of the organ which are induced by malaria, typhoid fever, and other infectious diseases, and is excited by trauma and forcible contraction of the abdominal muscles.

The symptoms are pain, rigidity of the abdominal wall, and internal hemorrhage. (For the diagnosis of this accident see Contusions of the Abdomen, p. 235.)

NEOPLASMS OF THE SPLEEN.

The benign cystic growths, of which there are serous, hemorrhagic, hydatid, and dermoid varieties, occasion an enlargement of the organ, which causes pressure upon the neighboring viscera. *Benign solid enlargements* of the spleen are due to malaria and leukæmia; these two conditions are readily differentiated by examination of the blood for plasmodia and by counting the leukocytes.

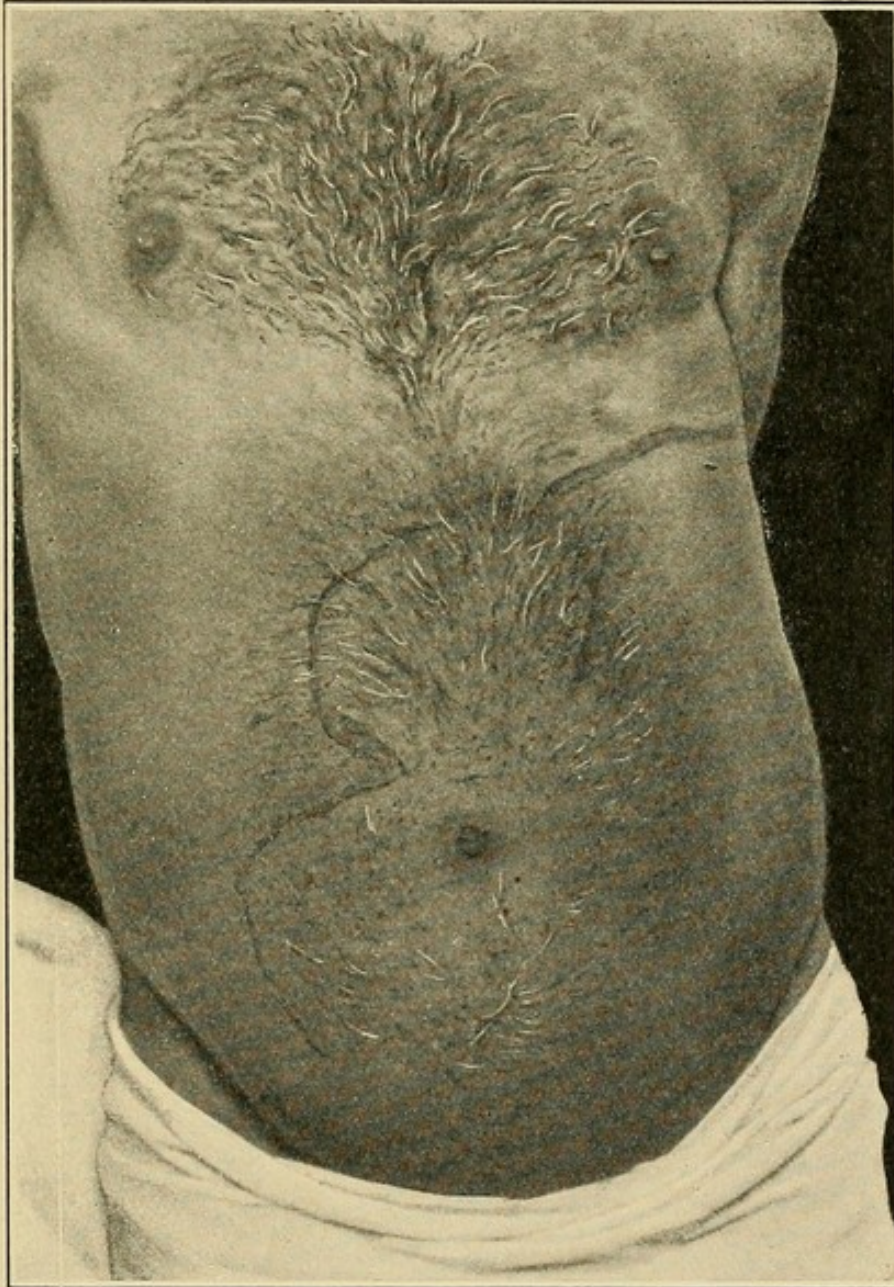
Hydronephrotic cystic tumors differ from splenic cysts in that they are attended with a history of urinary disturbances, and by alterations in the amount and character of the urine coming from the corresponding kidney. These tumors, furthermore, have no notched anterior border.

Hydatid cysts of the tail of the pancreas are to be differentiated from splenic cysts by the characteristic relation which they have to the stomach and transverse colon. *Hydatid cysts of the left lobe of the liver* are much more

difficult to differentiate from splenic tumors, unless a tympanic zone separates the liver from the spleen.

Ovarian cysts are distinguished from splenic cysts by their growth from below upward, and by their connection to the

FIG. 141

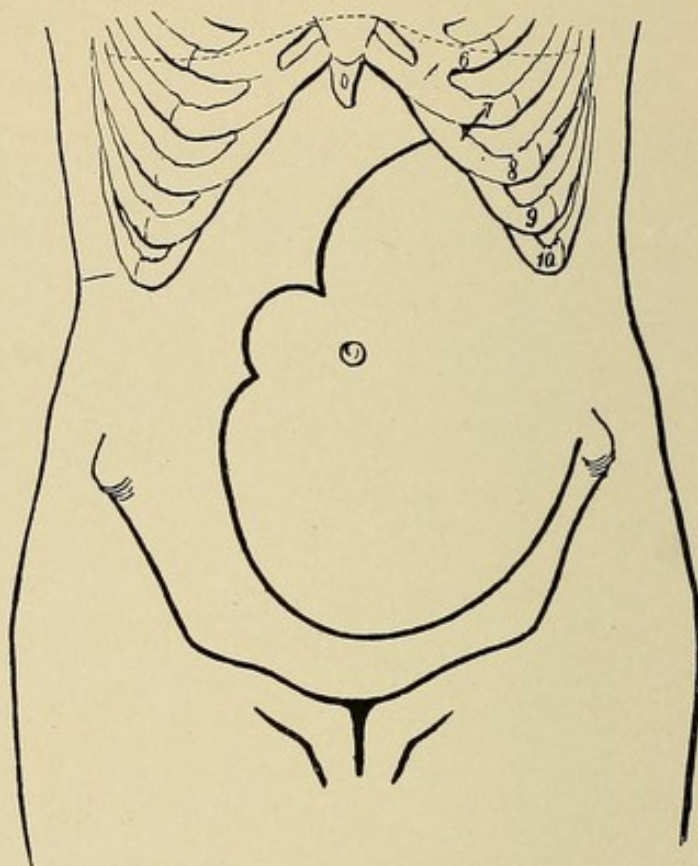


Large leukæmic splenic tumor. Note characteristic notch on anterior border, which alone is sufficient to distinguish a splenic tumor from all other abdominal swellings.

ovary, broad ligaments, and uterus with a pedicle that can be palpated through the vagina or rectum.

A rapid enlargement of the spleen attended with pain and cachexia, occurring in a young individual in whom leukæmia

FIG. 142



Large splenic tumor. Note characteristic notch and sharp anterior border.
(Winter.)

has been excluded by a blood examination, speaks for *sarcoma of the organ*.

CHAPTER XXXII.

DISEASES OF THE FEMALE PELVIC ORGANS.

METHOD OF EXAMINATION.

ABNORMALLY profuse or irregular uterine bleeding, vaginal discharge, pelvic pain, or the evidences of pressure upon the bladder, rectum or ureter, such as increased frequency of or pain on urination, and constipation, are the first indications to women that something is wrong with their pelvic organs. The physician to whom they apply should in every case take a careful history (for the details see p. 18), and then proceed to a careful abdominal and pelvic examination.

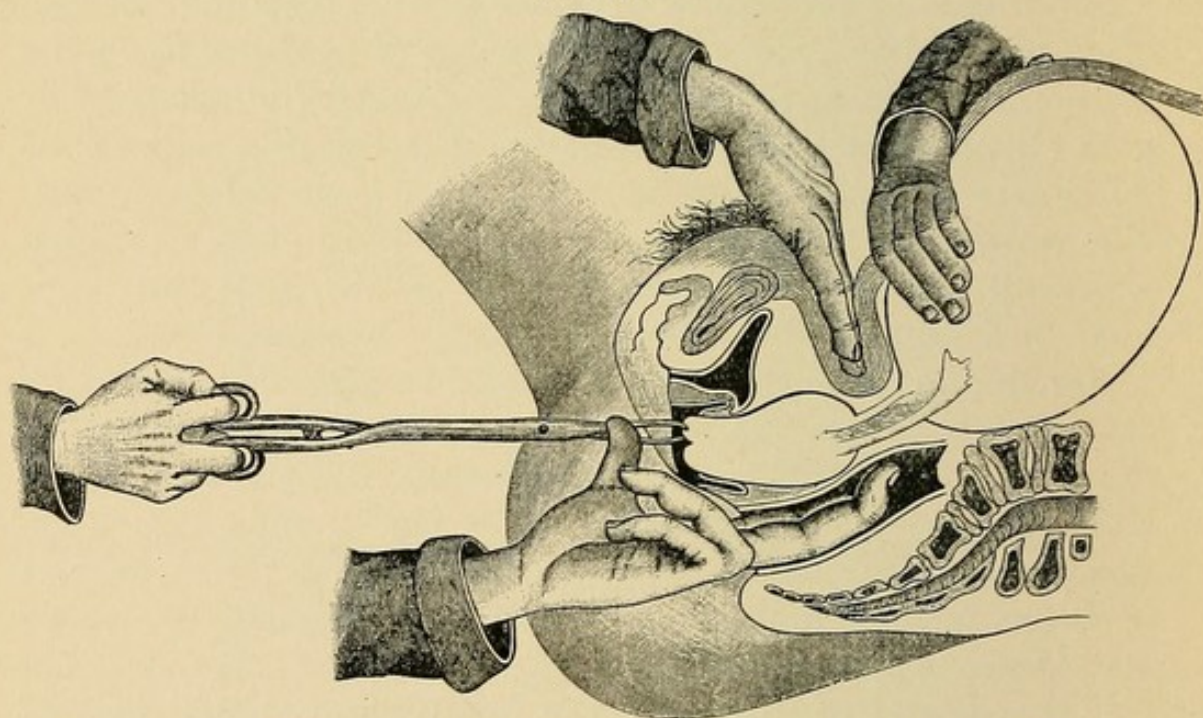
For this purpose the patient should be placed on her back transversely across the bed or on an examining couch, with the thighs semiflexed upon the abdomen and the legs flexed and supported by an assistant or by leg stirrups. In some special instances the knee-chest or the lateral prone (Sims') positions may be employed. The bladder should be evacuated prior to the examination.

The external genitals and the anal region should first of all be inspected and note taken of any abnormalities in these parts. Any bulging or prolapse of the vaginal walls or internal genital organs on coughing or straining, the appearance of the vaginal mucosa as seen through a speculum, and of the uterine cervix and os should also be noted. The examiner should then proceed to palpate bimanually through the vagina and rectum the pelvic organs, uterus, broad ligament, ovaries and tubes, parametria, bladder, and rectum. In thin subjects with lax abdominal walls the Fallopian tubes can be palpated from their inner uterine end, where they feel like firm cords of lead-pencil thickness, to their outer extremity, where they are intimately related to the ovary. The consistency of the cervix; the position, size,

flexure, and mobility of the uterus; the presence of neoplasms and their relation to the uterus, broad ligaments, and ovaries; the presence of exudates and their relation to the uterus, and last, but not least, the amount of pelvic pain and tenderness the abdominal condition provokes should all be carefully noted.

Hegar has introduced a plan which is very useful in detecting the connection or otherwise of a tumor with the uterus or broad ligament. The uterine os is seized with a vulsella forceps and drawn downward, and the forceps

FIG. 143



Hegar's method of palpation of pedicle of uterine or ovarian tumor. (Winter.)

placed in the hands of an assistant. The right hand is laid upon the abdominal wall above the pubes and the left forefinger passed into the rectum. The tumor being outlined between the two, is drawn upward; if it has any connection with the uterus, the forceps will rise. If the assistant pulls gently on the forceps while the tumor is held between the two hands of the surgeon, its pedicle can usually be felt and outlined by the rectal finger.

The abdominal examination should determine the shape of the abdomen, the presence of undue bulging in some eccentric position, the retraction or protrusion of the navel, the presence

of free fluid in the peritoneal cavity, the presence of tumors, their size and mobility, and finally the size and consistency of the liver and other abdominal organs.

The uterine sound affords us an invaluable means of determining the length of the uterine canal and the position of the uterus with reference to an exudate or neoplasm in the pelvis. It goes without saying that a uterine sound should never be introduced until we have done our utmost to ascertain that the woman is not pregnant, and, further, that all aseptic precautions should be observed in its introduction.

FIBROMYOMATA OF THE UTERUS.

The physical complaints that lead patients with fibromyomata of the uterus to apply to us for treatment are menorrhagia, metrorrhagia, or dysmenorrhœa, or attacks of local pelvic peritonitis provoking nausea, vomiting, constipation, intestinal distention, some free exudate in the peritoneal cavity and pelvic pain, or the evidences of pressure upon the bladder, rectum, ureter, pelvic vessels and nerves, such as increased frequency of urination, pain on urination, constipation, neuralgic pain in the pelvis shooting down into the lower limbs, swelling and congestion of the feet and legs, or sterility. By this it is not meant to imply that all those who have fibroid tumors of the uterus necessarily suffer from one or more or all of these symptoms, for even fairly large fibroids may be present and not give rise to any disturbances. The examination of these patients reveals conditions that vary according as the tumor is submucous, intramural or subperitoneal, and according to its location on the uterine wall.

Submucous Fibromata.—Submucous fibromata are to be detected only when they protrude as polypi through the cervix, or when they can be palpated by inserting the finger through the cervix into the uterine cavity. The presence of other fibroid masses in the wall of or beneath the peritoneal coat of the uterus usually enables us to make the correct diagnosis. These are the tumors that occasion the most profuse uterine bleedings, especially at the menstrual

periods. The introduction of the sound affords important data for the diagnosis, for the uterine canal in these cases is found distorted and usually somewhat lengthened. As a rule, such submucous tumors tend eventually to protrude through the cervical canal, and in some instances spontaneous delivery takes place. If the latter occurs the tumor which presents in the vagina, if of large size, may feel like a presenting fetal head, and may give one the impression that labor is going on. The impression is quickly dispelled if palpation of the abdomen reveals no fetal parts, if on auscultation no fetal heart sounds are to be heard, and if there are no signs of pregnancy in the breasts, etc.

Smaller tumors that present in or that have passed through the cervix appear as smooth, rounded or pear-shaped or cylindrical masses, the mucous membrane over which is either intact or ulcerated.

The bleeding which attends submucous fibroids is often very suggestive of abortion. The latter is, however, accompanied by the usual signs of early pregnancy and by the presence of placental tissue in the discharge. All doubt as to the diagnosis is at once dispelled if, when the finger is introduced into the uterus, a firm mass of varying size and shape, covered with smooth or ulcerated mucous membrane, is to be felt.

Intramural Fibromata.—*Intramural fibromata* are much easier to detect after they have reached a palpable size. They enlarge and thicken the uterus, which has an irregular, smooth, nodular shape, and elongate and distort the uterine canal. The size of the nodules vary; they are of very slow growth, and though the patients may become very weak and anæmic, they are not cachectic.

Such fibroids may give one the impression of a sarcoma, but their multiple character, their slow growth, and the absence of cachexia serve to differentiate them from this condition. The possibility of a sarcomatous degeneration of the fibroids taking place should not be forgotten. This would be indicated by a suddenly assumed, much more rapid growth of the tumor, by increased pain and bleeding, and by emaciation; these latter symptoms are much more signifi-

cant if they occur after the menopause, when fibroid tumors should under natural conditions gradually shrink.

Subperitoneal Fibroids.—The subperitoneal fibroids are readily recognized from their smooth, rounded, hard, non-tender, and multiple character. They move with the uterus; if they are pedunculated they enjoy considerable independent mobility, but otherwise they are fixed to the uterus. Those which are located at the fundus of the uterus grow upward into the abdominal cavity; those which spring from its sides push apart the layers of the broad ligament and with continued growth raise up the retroperitoneal peritoneum, and thus come to lie intraligamentously or subcæcally or sub-sigmoidally.

They must be distinguished from ovarian and tubal enlargements. *Ovarian tumors* can be traced to this organ. They are usually cystic and fluctuating, or if solid they are softer than fibroids. They rarely cause uterine bleeding, and never elongate or distort the uterine canal. Their base is external to the round ligament. *Tubal swellings* are connected by a narrow isthmus with the cornu of the uterus. They are tender, of sausage-shape, and tend to prolapse behind the uterus, where they become adherent. Their development is preceded by a history of genital infection, either septic or gonorrhœal or tuberculous. They rarely cause much bleeding, and never elongate or distort the uterine canal. *Intraperitoneal hæmatocele and pelvic hæmatoma* are distinguished from uterine fibroids by the fact that they usually follow a tubal pregnancy, a history of which can be elicited on cross-questioning. These tumors are soft at first and gradually become harder; they are adherent and tender and have ill-defined outlines.

Other intra-abdominal tumors located in the upper segment of this cavity are distinguished from pedunculated uterine myomata in that they fall away from the pelvic organs when the patient is placed in Trendelenburg's position.

Ballottement, swelling of the breasts, morning nausea and vomiting, fetal heart sounds, and *amenorrhœa* readily distinguish a pregnant uterus from one that is the seat of a large, succulent fibroid tumor.

A bicornate uterus may to palpation feel like a uterus with a fibroid at one cornu. The possibility of introducing the sound into two canals and the relation of the round ligaments to the uterus are the only data we have for a differential diagnosis.

MALIGNANT NEOPLASMS OF THE UTERUS.

When a woman at the menopause commences to flow irregularly and profusely, and has a foul, watery, or purulent vaginal discharge, she should not be considered as following the normal course at this period, but she should be strongly suspected of having a carcinoma of the uterus. Not until a careful examination has shown that no cancer is present should we dismiss this suspicion, and even then it is best to keep such a patient under close observation for some time.

An epithelioma of the cervix first appears as an indurated or infiltrated area, the rest of the cervix having a glazed or granular appearance. It soon forms a cauliflower-like growth which fills the vaginal vault and tends to ulcerate, leaving an indurated, sloughing ulcer, with everted edges.

Adenocarcinoma of the cervix first appears as a nodule, which grows rapidly, invades the vaginal surface of the cervix, and forms a cauliflower, villous mass that resembles an epithelioma.

Adenocarcinoma of the body of the uterus appears as a papillary, dendritic growth and causes an enlargement of the organ. If there is any doubt as to the diagnosis, a section of the cervix or scrapings from the uterine body should be sent to the pathologist for microscopic examination.

The clinical evidences of sarcoma are similar to those of carcinoma; they can be differentiated only by microscopic examination. The possibility of a sarcomatous degeneration of benign myomata should be kept in mind, and if a previously benign tumor takes on a very rapid growth, bleeds profusely, and causes considerable pain, this condition should be suspected.

INFLAMMATIONS OF THE FALLOPIAN TUBES.

When the Fallopian tube becomes inflamed its outer end becomes closed by the adhesions which form between it and the ovary, omentum, or intestine, or by the adhesion of its own fimbriæ to one another. The cavity of the tube then becomes distended with inflammatory products, and owing to its increased weight the organ falls down into Douglas' space, where it becomes adherent. The early formation of adhesions to the lateral or posterior pelvic floor prevents this prolapse into Douglas' cul-de-sac, and in such cases the distended tube bulges downward, separates the layers of its mesentery and those of the broad ligament, and thus come to lie in an intraligamentous position. The presence of adhesions and parametritis often make it difficult to decide whether the tube is intraperitoneal or intraligamentous.

The distal two-thirds of the tube is the usual site of disease. Inflammation of the tubes is excited by infection with the gonococcus, tubercle bacillus, bacillus actinomycosis, pyogenic organisms, etc.

The terminations hydro-, or pyo-, or hæmatosalpinx indicate a distention of the tube with serum, or pus, or blood.

The milder forms of salpingitis (catarrhal and hydrosalpinx), and even some of the cases of pyosalpinx, give rise only to distention of the tube and the feeling of pelvic weight and tension.

The severer forms, and especially the cases of pyosalpinx, manifest themselves by recurring attacks of pelvic pain and tension, fever, nausea, vomiting, and meteorism. The attacks may recur very frequently, and between them the patient suffers from pelvic weight and vesical or rectal disturbances. In the purulent forms the patient may develop chronic sepsis, with fever, emaciation, and secondary anæmia. With each recurrent attack a fresh local peritonitis is set up, which results in a matting together of the tubes, ovaries, omentum, bladder, and rectum. The distended tube forms a rounded tumor that lies behind or to the side of the uterus, to which it is connected by a cord-like strand of lead-pencil thickness, and which in the absence of adhesions is movable.

With *hydrosalpinx* the tube is tense and not very tender; it is not likely to be adherent. With *hæmatosalpinx* the tube is tense and tender and not usually adherent. With *pyosalpinx* the tube is thickened, sausage-shaped, doughy, and adherent.

The contents of the tube are determined from the patient's temperature, the leukocyte count (above 15,000 or 20,000 indicating a pyosalpinx), and from the shape and thickness of the tube walls.

A pyosalpinx may perforate through the abdominal wall, or into the uterus or vagina, or into the bladder or rectum, or into the peritoneal cavity. Perforation into the peritoneal cavity is followed by peritonitis, and perforation into the bladder, rectum, uterus, or vagina is followed by a discharge of pus with the urine or feces, or through the vagina. A periodical discharge of pus accompanied by a relief of pain and by a coincident diminution in the size of the tumor points to a valve-like orifice of the perforation, with periodical retention and discharge of the contents of the tube.

Ovarian cystic tumors are with difficulty distinguishable from tubal enlargements. Bilaterality of the tumor, sausage-shape, and a history of infection (puerperal, gonorrhœal, or tuberculous) speak for tubal disease. Ovarian tumors are more movable.

Soft uterine myomata are usually multiple; they are also harder, rounder, and more movable than tubal tumors. The history of infection is absent.

It is often impossible to differentiate *appendicular exudates or abscesses* occupying the right side of the true pelvis from tubal abscesses. The history of previous attacks of appendicitis, the more acute onset of the latter illness, the facts that with appendicitis the abscess occupies a higher site, and that part of the abscess is in the right iliac fossa, that the broad ligament is not apt to be involved, and that the uterus is freely movable, and that with tubal diseases both sides are likely to be affected, will aid us in making the differential diagnosis. It is to be remembered that with an appendicitis the tube may form one of the walls of the abscess cavity, and that with salpingitis the appendix may become adherent to

the tube. It is not unusual for both organs to be coincidentally diseased.

In pelvic peritonitis the tubes and ovaries are, as a rule, coincidentally diseased, and can with difficulty be separated from the inflamed tissues.

In pelvic cellulitis and intraligamentous exudates the vaginal vault is depressed and the exudate can be traced in front of and behind the cervix to the opposite side. Frequently the appendages are involved in the inflammation, and as the exudate disappears, the tubes can be easily recognized. In pelvic abscess it is impossible to distinguish the appendages.

Ovarian abscesses or inflammation or cysts of the ovary can rarely be accurately differentiated from tubal disease. As a rule, the tube is diseased coincidentally with the ovary.

EXTRAUTERINE PREGNANCY.

Pregnancy may occur in any part of the Fallopian tube, most frequently in the body of the tube, rarely at the fimbriated end, or in the intrauterine segment. After the impregnated ovum has become lodged in the tube, the fimbriated end becomes closed by adhesions and the ovum thus lies in a closed sac.

With the exception of amenorrhœa, the early signs of pregnancy—viz., morning nausea, vomiting, swelling of the breasts, pigmentation of the skin, etc.—are present in cases of tubal gestation. Instead of amenorrhœa there is an *irregularity of menstruation*, the woman going several days or weeks beyond her time and then commencing to bleed irregularly.

If the woman does not know or suspect that she is pregnant, this irregular bleeding may not give her much concern; but if she believes herself pregnant, the bleeding, especially if it is profuse, together with the discharge of membrane with the blood, may cause her to think that she has aborted. If she seeks medical advice at this time, there is found on examination either a solitary hard mass (a tubal mole), or a soft, doughy, usually tender, not adherent mass corresponding to

the tube. The uterus is enlarged, but not to a degree to correspond with the supposed term of pregnancy. If the membrane has been preserved it will be seen to be a decidual membrane. In many instances the patient will have experienced crampy, colicky pains in the pelvis.

If the diagnosis is not made in this stage and the tubal pregnancy continues to progress, the patient will very shortly have attacks of severe, colicky pains in the lower abdominal and pelvic regions, in some of which she may feel faint; and, finally, comes a severe attack of pain, accompanied with collapse and the evidences of internal hemorrhage, due to rupture of the tube. If the rupture is intraperitoneal and occurs during the early months of the pregnancy, there will be the physical signs of free fluid in the pelvis and flanks; sometimes the above-described soft, doughy, tender tumor of the tube is to be felt, and sometimes no mass whatever can be palpated. If the rupture is extraperitoneal, or if the intraperitoneal hemorrhage ceases and the blood becomes encapsulated, there will be a soft, boggy, tender, immovable mass with indefinite outlines either between the layers of the broad ligament or behind or to the side of the uterus, which pushes the latter to one side and depresses the vaginal vault. The temperature may be slightly elevated. The mass becomes gradually harder; should it become infected, there would be chills, fever, and septic symptoms. Large swellings may fill the entire pelvis, pushing the uterus to one side or backward.

If the fetus remains alive after the rupture of the tube, heart sounds will be heard after the seventh month, and fetal movements will be felt after the sixteenth to the eighteenth week. A continued growth of the fetus causes peritoneal irritation with abdominal pain, tenderness, vomiting, and distention; the foetus occupies an abnormal position in the abdomen and compresses the neighboring viscera.

Death of the foetus gives the same symptoms as fetal death in uterine pregnancies—viz., receding of the abdomen and breasts, chills, fever, foul discharge from the uterus, and general physical deterioration. Infection of the dead foetus leads to abscess formation and peritonitis.

Perforation of the abscess into a hollow viscus is preceded

by symptoms indicating irritation of this viscus, and is followed by a discharge of fetal parts, pus, etc., through the channels of exit of these viscera.

Unruptured.—An *unruptured tubal pregnancy* is to be distinguished from *tubal enlargements due to other causes*, from *uterine fibroids* and *small ovarian cysts*.

Irregularity in menstruation is present in both salpingitis and tubal pregnancy, but in the former there is a history and evidence of previous genital infection. A *pyosalpinx* is more tender, except in the chronic stage, and then it is considerably harder and more adherent, and is apt to be prolapsed into Douglas' space. A tubal mole is quite hard, but it is painless, not tender, and non-adherent. Bilaterality of the affection speaks for salpingitis, although it must be remembered that tubal pregnancy most frequently occurs in a tube that has been the seat of previous disease. A careful anamnesis is of considerable value in making the diagnosis.

Uterine fibroids are usually multiple, more irregular and hard. They have an entirely different history (see p. 345) and can be traced to the body of the uterus. There are no evidences of pregnancy.

Ovarian cysts have a pedicle, and are not attended with the signs of early pregnancy; they are usually fluctuating and tense, and have a smooth surface.

Ruptured.—A *ruptured tubal pregnancy* may be mistaken for *acute perforative appendicitis* or *twisted ovarian cyst*.

In *acute appendicitis* there is rarely the profound collapse or the evidences of internal hemorrhage. Appendicular exudates are wholly or in part higher up than are tubal pregnancies. The tube is felt to be normal. There are no evidences of pregnancy.

An *ovarian cyst* whose pedicle has become twisted is attended with crampy, colicky, pelvic pain that may suggest tubal rupture; but such a cyst and its pedicle are to be readily palpated, the accident is not attended with collapse, and there are no signs of early pregnancy.

Pregnancy in a bicornate uterus is distinguished from tubal pregnancy by the lopsided shape of the uterus (owing to the unimpregnated horn), and by the low, broad connection of the sac with the cervical end of the uterus.

CHAPTER XXXIII.

DISEASES OF THE FEMALE PELVIC ORGANS

(Continued).

INFLAMMATION OF THE OVARIES.

WOMEN may suffer considerable pelvic weight and pain which radiates down into the thighs, from a chronic follicular oöphoritis. The ovaries in these cases feel swollen, soft, and very tender. Hemorrhage into or suppuration of the small cysts, which go with this condition of the organ, causes increased pain and swelling, with moderate temperature elevations. Occasionally one of these cysts grows to the size of a child's head; it is tense and has thin walls, and can only be differentiated from neoplastic cysts of the organ by laparotomy.

NEOPLASMS OF OVARY.

Among the chief varieties of ovarian new-growths are:

Multiple cystic adenoma, with serous or pseudomucinous contents.

Papillary cystic adenoma, with pseudomucinous contents.

Solid papillary tumors.

| *Papillary adenocarcinoma and sarcoma*.

Carcinoma, sarcoma, fibroma, dermoids, and parovarian cysts.

Ovarian tumors are attached to the broad ligament by a pedicle, in which the meso-ovarian and the utero-ovarian ligaments, the mesosalpinx, the uterine tube, and the broad ligament enter. The relations of these structures vary greatly, according as the tumor grows upward into the abdominal cavity or downward toward the pelvic floor. Thus the meso-ovarian and the utero-ovarian ligaments may

be stretched out into a long pedicle; again, the tumor may develop in the outer part of the mesosalpinx, and the ampullar end of the tube be spread out over the surface. The whole mesosalpinx may be spread apart, and the tube stretched out on its surface. Continued development of the latter tumors opens up the lower part of the broad ligament, and raises the pelvic and abdominal peritoneum (thus forming the broad-ligament tumors). The pseudointraligamentous or, better termed, the retroligamentous tumors attain this position from having fallen backward into the pelvis behind the broad ligament, to the posterior layer of which they become adherent. The mesosalpinx covers over these tumors as a hood.

As long as ovarian tumors are small and do not distend the abdominal wall or cause pressure upon the abdominal viscera, or suffer any complications, they will not attract attention, for their mere presence does not, as a rule, cause any symptoms. Not even menstrual irregularities are provoked by them, to disturb the usual equanimity of the patient, or to warn her that something is wrong with her pelvic organs. The woman's first complaints are usually from pressure of the tumor upon the pelvic and abdominal organs, pressure being indicated by irritability of the affected viscus or by stenosis of its lumen, or by pain. The last is especially marked if the nerves are compressed. The pain is pelvic; it may radiate to the small of the back or down the thigh. Distention of the abdomen occurs only with large tumors, unless it be due to ascites, which is an early symptom of all malignant and papillary growths.

It frequently happens that the cyst causes no disturbances and consequently escapes detection until a complication arises. Such complications are twisting of its pedicle, rupture of its walls, incarceration, inflammation and suppuration, and malignant degeneration.

Twist of the pedicle is attended by crampy pelvic and abdominal pains, vomiting, shock, and peritoneal irritations. If the twist is sufficient to cause strangulation of the pedicle, gangrene of the cyst with subsequent peritonitis will result. If the presence of the cyst and its size were known before the twist of its pedicle occurred, an increase in its dimensions will be observed to follow this condition.

Rupture of the cyst may be followed by death from internal hemorrhage if a large vessel is torn. This is, however, a rare occurrence. If the cyst contents are bland, there follows diarrhœa or polyuria; if they are irritating, peritonitis with the formation of adhesions ensues. Rupture of papillary cysts has an especial significance, for dissemination of the growth over the peritoneum follows upon it. If the presence of the cyst was known prior to its rupture, this complication would be observed to cause a decrease in its size or even its entire disappearance.

Incarceration of the cyst in the pelvis is manifested by pelvic pain and the signs of peritoneal irritation. Gangrene of the cyst is followed by septic peritonitis, but this rarely occurs from this accident.

Suppuration of the cyst is a rare occurrence; it is indicated by fever, emaciation, and increase in the size of the tumor.

DIAGNOSIS OF THE PRESENCE OF A TUMOR.

It is a good rule never to diagnosticate the presence of an ovarian cyst unless it can be palpated. This is not always easy; and especially is it difficult where the woman is fat, and the cyst is small and flaccid. If palpation is rendered difficult by rigid abdominal walls, an anæsthetic should be administered.

Ovarian tumors have the following characteristics: Those that extend up into the abdomen are dull to percussion over their convexity and below toward the pelvis, and are surrounded above and at the sides by a zone of tympanitic resonance. They are attached to the broad ligament by a pedicle, which can often be palpated per rectum, and they replace the ovary of the side from which they spring.

The distention of the abdomen due to intestinal distention, to bladder distention, and to tuberculous and chronic exudative peritonitis, is readily distinguished from that due to an ovarian tumor. With intestinal distention the abdomen is everywhere tympanitic. An overdistended bladder has an elongated, ovoid shape, a symmetrical form, lies in the median line, and disappears when the bladder is emptied

by a catheter, a procedure, by the way, that should always be carried out before an examination is made. Tuberculous and other chronic forms of peritonitis with exudation are characterized by irregular areas of tympany and dullness, which alter their position and their size from time to time. The sacculations of fluid have indefinite outlines, and slight mobility.

Small ovarian tumors must be differentiated from uterine tumors, tubal enlargements, pelvic exudates, and fecal masses. Ovarian tumors lie to one side of or behind the uterus, are usually smooth and round, cystic and fluctuant, enjoy independent mobility, and are connected to the uterus by the corresponding utero-ovarian ligament. *Myomata* are usually multiple, grow more slowly, and are harder. Their attachment to the uterus is not by the utero-ovarian ligament. They distort and elongate the uterine canal. Menstruation is usually profuse and painful. *Tubal tumors* are sausage-shaped; they follow a preceding vaginal and uterine infection; they are usually adherent in Douglas' sac, and their narrow, pipe-stem pedicle, which can, as a rule, be palpated, corresponds to the uterine end of the tube. *Pelvic exudates* follow a genital infection. The swelling they occasion is diffuse, not mobile, and is attached to the uterus and iliac wall; the uterus is fixed and very painful when attempts are made to move it. They are attended with fever and constitutional disturbances.

An *extraperitoneal hæmatoma* is diffuse and non-mobile. At first it is soft and boggy; later on it becomes hard and tender. Both hemorrhage and exudates tend to become smaller; cysts larger.

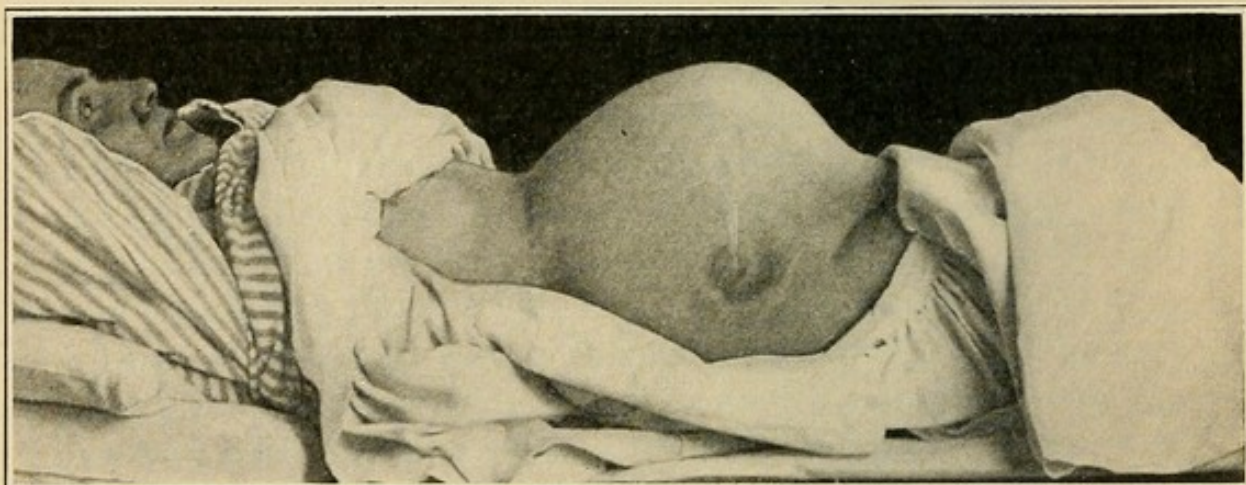
Fecal masses differ from ovarian cysts in that they disappear when the bowels are thoroughly purged.

Medium-sized ovarian tumors are oval, smooth, and have at times a knobbed surface. They are easily palpated. By grasping the cervix with a bullet-forceps and dragging it downward, and at the same time pulling the tumor upward into the abdomen, the pedicle is put on the stretch and can be then easily palpated through the rectum or vagina. The uterus can also by this procedure be more easily and definitely palpated; it is felt to be distinct from the tumor, which latter

nevertheless participates in the downward movement that is imparted to the uterus by the traction on the cervix.

Medium-sized tumors are to be differentiated from pregnancy in the fourth to the sixth months, from tumors of other abdominal organs, from tumors of the abdominal wall, from other pelvic tumors, and from ascites. In pregnancy the tumor which is formed by the body of the uterus is directly continuous with the cervix uteri. This characteristic of the tumor, together with the other signs of pregnancy, of which ballottement is especially important, readily enables us to make the differentiation. In the fifth and sixth months of pregnancy the fetal heart sounds, the palpation of fetal parts,

FIG. 144.



Shape of abdomen in ovarian or uterine tumor. Compare with Fig. 125, showing distention of abdomen with ascitic fluid. Note absence of protruding umbilicus and flatness of the flanks.

and the uterine bruits leave no room for mistake in diagnosis. With hydramnios or death of the fetus, the signs of pregnancy, and the fact that the tumor is directly continuous with the cervix will enable the examiner to rule out an *ovarian cyst*.

Tumors of other abdominal organs, stomach, intestines, kidney, spleen, omentum, etc., all fall away from the pelvis and toward the diaphragm when the patient is put into Trendelenburg's position, thus showing that they have no pelvic connection. When such tumors are adherent, the pelvic organs must be carefully mapped out per vaginam, and their relations to the tumor determined.

Tumors of and exudates in the *abdominal wall* are usually harder than ovarian cysts. They move with the abdominal wall in respiration—*i. e.*, with the patient in the dorsal recumbent position they move forward and backward. Their shape is flattened, and they have no relation to the pelvic organs.

Free ascites gives to the abdomen a barrel-shape when the patient is in the dorsal recumbent position; the flanks bulge, have a dull note on superficial percussion, and a dull tympanitic note on deep percussion, and the centre of the abdomen is tympanitic. With change in the patient's position to one side the upper flank becomes tympanitic to percussion. With tumor the percussion note over the tumor is always dull.

Echinococcus cysts of the pelvis are usually adherent; they are of slow growth, and are apt to be associated with echinococcic disease of other organs. The normal condition of the uterus and ovaries shows that these organs are not involved in the cystic degeneration.

Very large ovarian tumors are more difficult of differentiation because they occupy the entire abdominal and pelvic cavities. They have no mobility from lack of space, and their pedicles are difficult to make out. Tympanitic resonance over the abdomen is only present well back in the flanks, and high up under the ribs; in all other parts the percussion note is dull. The uterus is displaced; either it is crowded down into the pelvic floor or elevated out of the pelvis in front of the tumor, where it can often be felt above the symphysis. The entire pelvis is filled with the tumor.

The differential diagnosis must be made from free ascites. (See above and p. 252.)

Ascites complicating tumor: The tumor can at times be felt per vaginam, while the ascites gives the usual symptoms.

DIAGNOSIS OF THE NATURE OF THE TUMOR.

As a rule the cystic tumors can be differentiated from the solid ones by their markedly different resistance, their larger size, and their more frequent association with ascites. Among the cystic tumors must be distinguished the multilocular cysts, cystic papilloma, dermoids, parovarian cysts, cystic sarcoma, and carcinoma.

A *multilocular cyst* is irregular in contour, the irregularity corresponding to the depressions between the cysts. The wall feels thick, and as its contents are viscid the sensation of fluctuation is indistinct. Irregular bosses of varying tenseness may be made out. *Cystic papillomata* that have a smooth cyst wall cannot be distinguished from other cystic tumors. If the papillomata form excrescences on the surface of the cyst, these may be felt through the vagina. The presence of ascites with a small, fixed pelvic tumor and with no cachexia should arouse our suspicion of such growths. Extensive metastatic, papillary excrescences in the abdomen are easily palpable when the coexistent ascitic fluid is removed.

Dermoids are usually monocystic, single, and not much larger than an adult head. They are of soft, mushy consistency, of slow growth, float up in front of the uterus, occur chiefly in children and in young women, and are frequently tender.

Parovarian cysts are unilocular. They usually have thin walls and a broad base of origin, and are intraligamentous. They may have an irregular contour, and resemble multilocular cysts. They occur chiefly in young subjects.

Cystic sarcomata and *carcinomata* are unilocular; they have thick walls. When they reach large size they occasion ascites, cachexia, emaciation, and metastases. When they are small they have no especial characteristics.

Among the hard tumors are the *fibroma* and *solid sarcoma* and *carcinoma*. The *fibroma* is a hard tumor, not very large, is accompanied by ascites, but not with cachexia, emaciation, or metastases. Carcinoma and sarcoma are larger, and may involve both ovaries. The latter occur in young subjects, are often secondary to sarcoma of the stomach, and are attended with ascites, cachexia, emaciation, and metastases.

Malignant degeneration of a benign tumor is indicated by its rapid enlargement, by pain, ascites, cachexia and œdema of the legs and abdominal walls.

Adhesions between the cyst and neighboring viscera are indicated by a loss of its mobility, an increase of its tenderness, and by the presence of peritoneal friction sounds.

CHAPTER XXXIV.

DISEASES OF THE RECTUM.

METHOD OF EXAMINATION OF THE ANUS AND RECTUM.

THE symptoms which are occasioned by diseased conditions of the rectum are constipation or diarrhœa, tenesmus, pain, itching, discomfort or uneasiness about the anus, prolapse or protrusion about the anus, bleeding from the rectum, and a discharge of mucus or pus or blood with the stools.

It is unfortunately too common for physicians to make their diagnoses of rectal disorders from these symptoms, without the aid of the essential data that are afforded by a local examination. This explains why their diagnoses are so often wrong, and further accounts for their frequent inability to effect a cure of the malady. The first requisite for a successful issue of our therapy in diseases of this organ is an accurate diagnosis not only of the local disorder, but of the cause therefor, and this can only be made by a thorough examination of the rectum and of the other abdominal and thoracic organs.

The local examination should be made soon after the rectum has been evacuated, though where there is a discharge of blood, or pus or mucus, it may be advisable to make a re-examination several hours after defecation. In such cases it is best to make the examinations on two successive days. The patient for examination should be laid upon a couch or table in the left lateral (Sims') position, in the exaggerated lithotomy position, in the knee-chest position, or, when protrusion or prolapse is suspected, in the squatting position.

Our attention should first be directed to the external anal appearances; its shape, the condition of the surrounding skin; whether it is moist, or dry, or brittle, or red, or excoriated; whether parasites or pediculi are lodged in the external

hairs, and whether there are present protrusions, or scars, or ulcerations, or fistulous openings, or external growths, such as condylomata, fibroids, polypi or skin-tabs. The buttocks should then be pulled well apart and, while the patient strains slightly, we should inspect the anal canal, noting the mucocutaneous border; whether it is soft and moist, or dry, brittle, crackling, and bleeding, as in atrophic rectal catarrh and syphilis. The condition of the anal folds, whether they are normal or inflamed and swollen, and the presence of fissures, hemorrhoids, polypi, or new-growths, are likewise to be taken note of.

The finger, covered with a rubber finger-stall and well lubricated, should now be slowly introduced with a boring motion, at first upward and forward and then backward, feeling as it enters the tonicity and irritability of the sphincter muscle. Irritability points to acute disease; a hard, resisting sphincter indicates hypertrophy from chronic disease, and a relaxed, flaccid muscle speaks for an exhausting general disease or neoplasm of the rectum. As the finger passes into the bowel the presence of ulcers, of abnormal openings, of abscesses, of hypertrophied, inflamed or thrombosed hemorrhoids, of foreign bodies, of neoplasms or of strictures, should be noted, and likewise the position, mobility, and configuration of the generative organs, of the bladder, of the prostate, and of the coccyx. The presence of pelvic tumors should also be ascertained. As the finger is withdrawn the patient should be asked to bear down, so that if prolapse or hemorrhoids or pus or blood are present, they should follow it out through the anus.

Inspection of the rectum through the proctoscope is often necessary to differentiate the digital findings, and for the examination of the upper rectum and sigmoid flexure the proctoscope and sigmoidoscope should be employed.

When we have thus ascertained the nature of the rectal malady, we should direct our attention to the internal organs and to the anamnesis for a possible explanation of the local condition. The diseases of the liver are especially likely to be the cause of rectal disorders, and conversely diseases of the rectum frequently give rise to secondary lesions in the liver. The careful percussion and palpation of this organ

should therefore never be neglected when the rectum is diseased. The antecedent family and personal history, especially as to gonorrhœa, syphilis, and tuberculosis, should also be elicited, and their bearing upon the diseased condition given due consideration. The previous and present health and weight of the patient, and the presence of symptoms referable to the genitourinary organs are often valuable guides toward indicating the extent and character of the rectal malady.

THE SPECIAL DISEASES OF THE RECTUM.

Atresia of the Anus and Rectum.—It should be the practice of obstetricians and nurses to inspect and determine the patency of the anal and urethral orifices of the newly born babe, for among the ignorant a congenital defect or malformation that is not detected and announced at the time of birth is often ascribed to the carelessness or incompetency of the physician in charge. Such examination will at times reveal that there is no anal orifice, but while the child cries a bulging may be noticed in the perineum; the condition is one of *atresia ani*. Or the anus is absent and no bulging in the perineum is to be seen; the condition is one of *atresia ani et recti*. Sometimes the anus is present, but it leads into a blind cul-de-sac; the condition is one of *atresia recti*. In all these cases there will develop the symptoms of intestinal obstruction, unless an opening for the evacuation of the contents of the bowel is speedily made. Abnormal congenital openings between the rectum and bladder, urethra, vagina, or external genital organs are readily recognized by the discharge of fecal matter through these abnormal channels. Such conditions are rare and are usually associated with imperforate conditions of the anus.

Prolapse.—A conical *protrusion of the mucous membrane* or of the entire wall of the rectum is seen quite often in childhood, less frequently in adult and advanced life. The protrusion is attended with bleeding and some pain, and is first noticed by the mother or patient at the end of defecation. The protruded part may be a simple prolapse of the

end of the rectum, or it may be a higher portion of the rectum that has invaginated itself through the anus. In the former instance the mucous membrane covering the protruded part is directly continuous with the mucocutaneous border, and the finger cannot be introduced into the anus alongside of it, whereas in the latter the finger can be passed into the anus alongside of the protrusion, and can be swept entirely around it. A protruding invagination of the rectum through the anus is differentiated from a protruding intussusceptum of a higher portion of the bowel by the fact that in the latter condition the finger, when introduced into the rectum alongside of the protrusion, meets with no obstruction to its passage upward, while in the former an obstruction is encountered a short distance from the anal aperture.

Prolapsing hemorrhoids are distinguished from simple prolapse by their irregular, lobulated shape, the varicose condition of the vessels, and by the fact that at certain portions of the circumference of the rectum the mucous membrane remains *in situ*.

When the *prolapsed mucous membrane* becomes excoriated, hypertrophic, and nodular, it bears a strong resemblance to a neoplasm, and can only be distinguished from it by microscopic examination of an excised specimen.

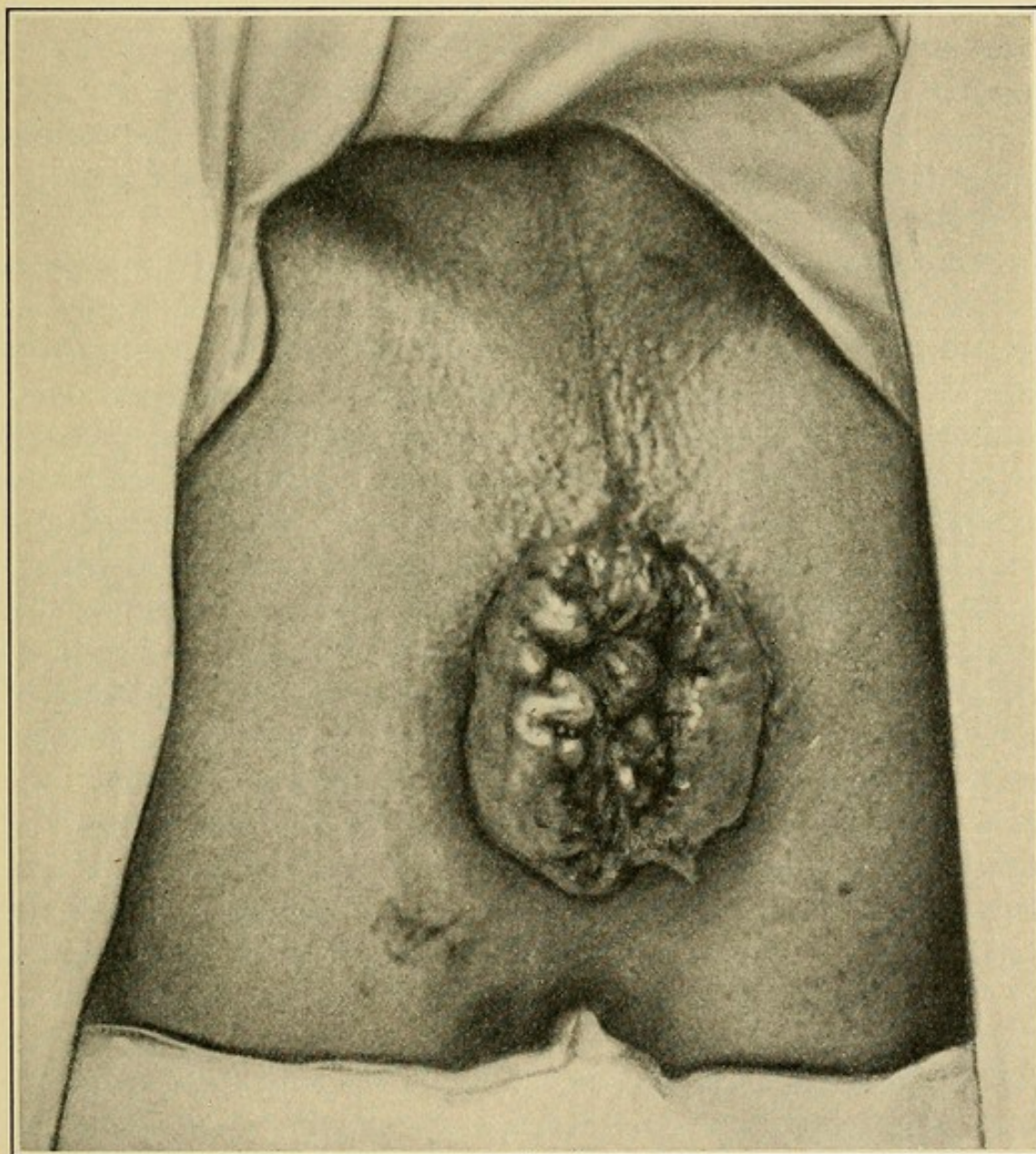
Hemorrhoids.—A *varicose condition* of the hemorrhoidal veins (*hemorrhoids*) is very readily recognized. When the vessels external to the sphincter ani are affected (*external hemorrhoids*), they appear as single or multiple, bluish, soft nodes with a broad base, that swell upon straining and that are easily emptied by pressure. When they become inflamed, they are swollen, painful, tender and hard, and can no longer be emptied by pressure; at such time they occasion marked rectal tenesmus. Acutely inflamed or thrombosed external hemorrhoids or a chronic irritation about the anus often result in the formation of skin-tabs about the anus, sometimes called *fleshy piles*. These cause annoyance only when they become inflamed.

The internal hemorrhoids appear as sessile prominences, sometimes single, and again multiple, of irregular, lobulated shape; they occasion bleeding, prolapse and pain. When

they are prolapsed or inflamed, they form globular swellings with glistening surface.

Hemorrhoids are always to be considered as symptomatic of some other disease or as due to functional derangement of the bowel, and to the ascertainment of their underlying

FIG. 145.



Ulcerating and prolapsed internal hemorrhoids. Note the lobulation of the protruding masses.

cause we should in every case devote our attention. The most frequent causes of hemorrhoids are diseases of the heart, lungs, and liver, neoplasms of the rectum, neoplasms or malposition of the internal female genital organs, stricture

of the urethra, enlarged prostate, and vesical stone, and until a thorough examination has shown an absence of these conditions we are not justified in considering that they are due to functional disturbances of the bowel.

Inflammation and Ulceration.—A *discharge of mucus*, blood, and pus from the rectum, with tenesmus and reflex urethral and vesical irritability, is indicative of inflammation and ulceration of the rectum. The previous history of the patient, the evidences of some constitutional disease in other organs, and the appearances of the ulcerations throw light upon the especial character of the inflammatory or ulcerative process.

An acute onset with some fever, malaise, rectal pain, rectal and vesical tenesmus, and the discharge of mucus or blood or pus from the rectum speak for *acute proctitis*, the cause of which must be sought in some error or over-indulgence in diet or drink, in some constitutional disease, in gonorrhœa, worms, etc. Through the speculum the mucous membrane appears of a bright-red color, at first dry and swollen, and later on covered with mucus or blood or pus.

A gradual onset marked by flatulence, tenesmus, loss of appetite, alternating constipation and diarrhœa, a discharge of mucopus sometimes tinged with blood, and a feeling of weight and discomfort in the rectum are suggestive of *chronic inflammation*. Such a chronic process may also be the remains of an acute inflammation. If there is a marked tendency to bleeding and the development of fissures around the anus, if the mucous membrane is dry and cracks easily and tends to ulcerate, the inflammation is of the *atrophic variety*, a frequent result of sodomy, improper hygienic conditions, or syphilis.

The *ulcerative lesions* are chiefly of the tuberculous, syphilitic, gonorrhœal, inflammatory, or neoplastic types. They all occasion about the same character of symptoms—viz., pain in the back, diarrhœa, tenesmus, and a discharge of blood, mucus, and pus. The discharge is very profuse in the syphilitic ulcerations, and foul and decomposing in odor in the tuberculous and neoplastic.

An acute onset of the symptoms in a patient who has recently lived in the tropics, and the presence of narrow,

linear, or stellate ulcerations having sharply defined borders, the scrapings from which contain the *amœbæ coli*, are indicative of *amœbic proctitis*.

A very slight grade of symptoms with marked chronicity occurring in a patient who suffers with hemorrhoids, and the presence of sharply defined, irregular ulcers of the mucous membrane between the hemorrhoidal masses speak for *varicose ulcers*. These must be distinguished from the superficial or teat-like ulcers on top of the hemorrhoidal masses which are due to inflammation of the latter.

A gradual, insidious onset of the symptoms in a tuberculous subject, with the presence of irregular, grayish ulcers having undetermined and worm-eaten edges, points to *tuberculosis*.

A gradual onset in a syphilitic subject who presents other evidences of syphilis in the skin, bones, mucous membranes, etc., together with crater-shaped, sharply defined ulcers that discharge profusely, speak for *syphilis*. In connection with this disease it is well to remember the characteristic papillary growths (broad condylomata) and the mucous patches that occur during the secondary period, and the soft, elastic, rounded swellings (gummata) that develop during the tertiary period, and also the initial erythema or dermatitis with shallow fissures between the anal folds followed by perianal ulcerations with seropurulent discharge that occurs in infants as a result of hereditary infection.

The presence of a hard, fixed, rapidly growing tumor in the wall of the rectum, which breaks down and leaves an ulcer that has hard, everted edges and necrotic base, is indicative of a *malignant neoplasm*.

A gradual onset of the symptoms in a patient who is in the terminal stages of chronic Bright's disease, diabetes, cirrhosis of the liver, or marasmus, points to ulceration due to these constitutional disorders. As these individuals are already near their end, the rectal ulcerations are of importance only in so far as the sufferings of the patient are thereby considerably increased.

Stricture of Rectum.—Stricture of the rectum, which is manifested by an increasing tendency to constipation alternating with diarrhœa, tape-like stools, and mucopurulent, bloody, or foul rectal discharge, is either benign or malign

in character. A preceding history of simple or syphilitic ulceration and a multiple number of strictures speaks for their benign character, while a hard, fixed, ulcerating tumor in an elderly person that occasions a foul discharge and that is attended with emaciation and cachexia is indicative of its malignant nature.

Most of the cases of stricture of the rectum, and especially those of the malignant type, are attended with hemorrhoids, and it is the disturbances which these latter provoke that very often first attract the patient's attention. This explains why so many of these cases are diagnosticated as hemorrhoids, and it should teach us never to rest satisfied with the diagnosis of hemorrhoids until a thorough and careful rectal, pelvic, abdominal, and thoracic examination has shown that they are not dependent upon some other malady.

Polypi.—Frequently recurring severe hemorrhages from the rectum, especially in children, are very suggestive of *rectal polypi*. The diagnosis is easily verified by digital examination.

Periproctitis.—Pain, tenderness, and rectal tenesmus, together with the presence of a doughy or fluctuating swelling in the perianal or perirectal cellular tissues, are evidences of a *periproctitis*, the cause for which must be sought in some inflammatory or ulcerative condition or injury of the rectum, or in some inflammatory disease within the pelvis or of its bony walls.

Fistula.—Periproctitis is frequently followed by fistulæ in ano or fistulæ in recto. If these are complete—*i. e.*, have both an internal and external opening—there is usually an escape of gas and fecal matter from the external orifice. The internal opening can sometimes be palpated by the finger in the rectum and sometimes be seen with the proctoscope, but the most certain way to locate it is to pass a probe through the fistula into the anus or rectum. Fistulæ may be blind internally or externally. In such cases there is no escape of gas or fecal matter, but as long as the orifice of the fistulous tract remains open, there will be a discharge of pus either into the bowel or onto the surface of the buttock. When the orifice becomes closed a periproctitic abscess develops.

PART V.

DISEASES OF THE GENITOURINARY ORGANS.

CHAPTER XXXV.

DISEASES OF THE KIDNEY.

GENERAL CONSIDERATIONS ON DIAGNOSIS.

THE accurate methods at our disposal for the examination of the kidneys and their secretion enable us to ascertain not only whether these organs are healthy or diseased, but also in most cases the exact nature of the malady which affects them. We are further in a position to ascertain whether one or both organs are diseased, and if the latter is the case, which kidney is the most affected, and finally we are able to determine the combined and individual functioning power of the two organs.

The health or disease of the kidneys is determined from a study of the anamnesis, from physical examination, from careful urinary analysis, from the appearances of the ureteral mouths as seen with the cystoscope, and from *x*-ray examination. Unilateral or bilateral disease is positively determined by ureteral catheterization with collection and examination of the individual urines; and the combined and individual functioning power is ascertained from the total urea eliminated in twenty-four hours, from the urea percentage in the individual urines, from cryoscopic examination of the blood, cryoscopic examination of the individual urines, and by the phloridzin and methylene-blue tests.

The Anamnesis.—It is well to remember that while most individuals who suffer with surgical diseases of the kidney

afford at some time clinical evidences of their malady, yet it is not infrequent to see patients who have been afflicted with such ailments for a long time without having noticed anything abnormal in the urinary tract or urine. As only the intelligent are accustomed to watch the gross appearances of the urine, it is easy to understand how it is that even considerable grades of pyuria or hæmaturia pass unnoticed, unless pain or frequent urination direct attention to the urinary organs and their excretion. Such patients often seek the advice of the physician on account of disturbances of their general health, such as anæmia, loss of weight, or gastric and intestinal disorders; their thorough physical examination then reveals the existence of advanced disease of one or both kidneys.

As a rule, however, the patients complain of urinary disturbances, and in every case it is necessary to make enquiries as to the following facts: *The frequency of urination* (during the day and night); *whether it is painful or not*, and at what stage of the urinary act the pain occurs; *the gross appearances of the urine*, whether purulent, or cloudy, or bloody (and in the latter instance whether the urine is bright red or smoky in color); whether the pus or blood is uniformly distributed in the urine, or whether it is present only in the first or last urines; the *total quantity* of urine which is passed in twenty-four hours, and the *readiness or ease* with which the stream is started.

All these data are most important; thus the only evidences of early renal tuberculosis may be painful, frequent urination. Pus or blood appearing only in the first urines never comes from the kidney; from the latter source it is always uniformly mixed with the urine. Smoky urine is believed by Gomprecht to indicate the kidney as the source of the blood. Pain which is experienced only at the end of urination is not due to kidney disease; from this cause there is pain throughout the whole act of urination, though it may be most severe at the beginning or end of the act. Thus from a few questions and answers the examiner is able to gain some insight into the location of the disease in the urinary organs.

In the anamnesis it is further important to elicit such general data as may aid in making the diagnosis. Thus the

age of the patient, the duration of the symptoms and the rapidity with which they have developed and progressed, the existence of family or personal tuberculosis, previous gonorrhœa or syphilis, the history of a trauma or operation upon the urinary organs in women, pelvic inflammation, pregnancy and child-bearing, and the past or present affliction with other acute or chronic diseases, such as infections, pyæmia, hæmophilia, etc.

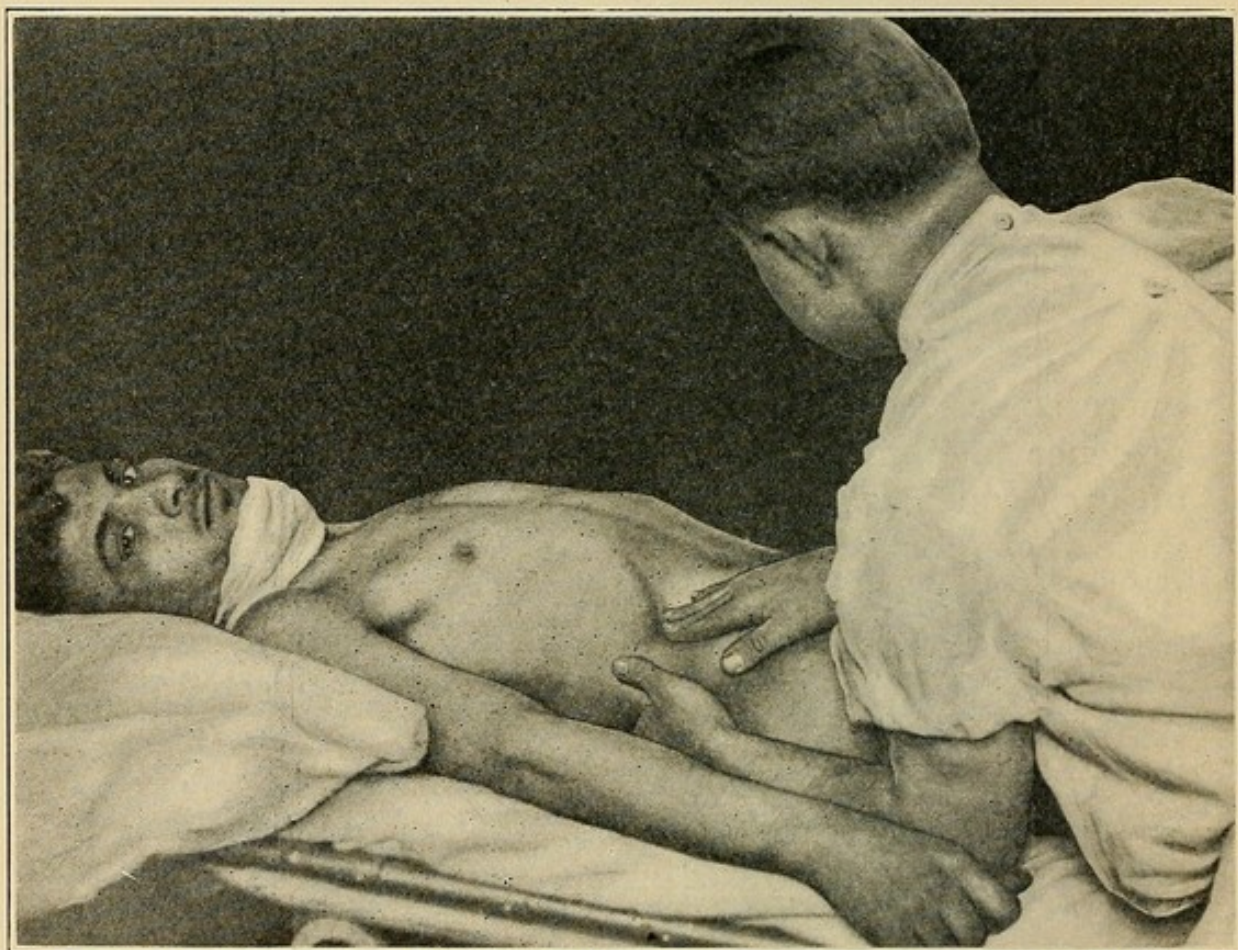
The diagnosis, however, cannot be made from the information which is obtained from the anamnesis; only a clue can be had from it as to the site and possible nature of the urinary malady. The diagnosis always rests upon the knowledge which is obtained from the examination of the patient and his urine.

By physical examination we can determine the presence of any abnormal position, form, or swelling of the kidneys and other parts of the urinary tract; by chemical analysis and microscopic examination of the urine we can determine the presence of abnormal constituents therein and the amount of urea which is excreted. With the *x*-ray we can determine the presence of foreign bodies in the urinary organs; with the cystoscope we can see the appearance of the ureteral orifices and of the vesical mucosa; by ureteral catheterization and analysis of the separately collected urines we can ascertain whether one or both kidneys are diseased; by cryoscopy of the blood we can ascertain the sufficiency or insufficiency of the combined kidney function, and by cryoscopy of the separated urines we can find out the individual kidney functioning power.

Physical Examination.—The normal position of the kidneys is retroperitoneal, in the upper one-third of the lumbar fossa, reaching from the upper border of the twelfth dorsal vertebra to the lower border of the second or middle of the third lumbar vertebra, the right organ being about one finger's breadth lower than its fellow. The hepatic flexure of the colon covers the lower pole of the right kidney, the descending duodenum its pelvis; the splenic flexure and tail of the pancreas cover the lower one-half of the left kidney, and the spleen its external lateral border. Abnormalities in size, form, or consistency may be detected by inspection,

bimanual palpation and percussion, with or without preliminary distention of the stomach and colon. In palpating the kidney, the patient should lie on his back, with the lower limbs flexed and the shoulders slightly elevated, or on the healthy side with limbs drawn up, or in the knee-chest position. The one hand of the examiner is placed in the loin and the other over the anterior abdominal wall. Where

FIG. 146



One method of palpating kidney. Patient in dorsorecumbent position, with lower limbs flexed and shoulders slightly elevated.

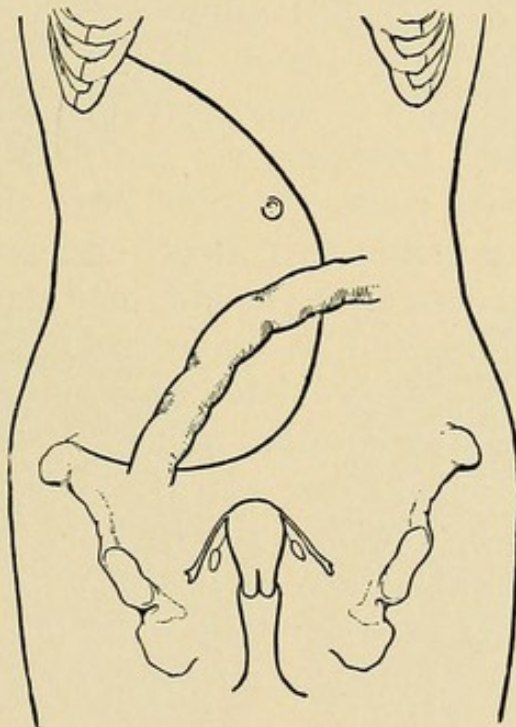
the latter is very rigid, or the individual very fat, a general anæsthetic may be required in order to make satisfactory palpation. In thin subjects of both sexes who have lax abdominal walls the lower pole of the kidney may normally be palpated.

It is always important to ascertain the relations which enlargements and tumors of the kidney bear to the colon. For this purpose the colon should be distended with air, and

then outlined by percussion. On either side the colon lies in front and internal to the tumor, the ascending colon passing across it from below and to the right upward to the left, and the descending colon from above and to the left, downward to the right.

Exploratory Puncture.—Exploratory puncture is often of value to ascertain the nature of the kidney swelling. It should always be done through the loin, and under strict aseptic precautions.

FIG. 147



Relation of ascending colon to tumor of the right kidney. (Winter.)

Urinary Examination.—The importance of careful and thorough urinary examination in establishing a diagnosis of kidney disease cannot be overestimated. The total quantity of urine which is passed in twenty-four hours should be collected and a specimen thereof examined chemically and microscopically. Its reaction and color and the presence of sediment should be noted. If there is a sediment, the urine should be repeatedly filtered until it is clear, the filtered urine being examined for albumin and sugar, and urea percentage, and the sediment being examined under the microscope for crystals of uric acid, oxalates, triple phosphates, leucin, and

tyrosin, for pus and red blood cells,¹ casts, epithelia, and micro-organisms, especially tubercle bacilli, staphylococci, streptococci, or colon bacilli. (Examination of the urine for the presence of bacteria always demands that it be drawn by aseptic catheter.) The methods of performing these tests and the appearance of the microscopic elements are to be obtained from books on urinary examination.

Cystoscopic Examination.—The appearance of the ureteral openings as seen with the cystoscope and the character of the urinary efflux are valuable aids in making a diagnosis of the presence and nature of a kidney affection. Naturally a trained eye is essential to appreciate the changes in the shape, position, and appearance of the ureteral orifice, and considerable experience is necessary to interpret the cystoscopic findings. If, however, we are to establish an early diagnosis in obscure cases we must have the aid of all the data which it is possible to collect, and some of the most important of these data are to be obtained only by cystoscopic examination. Though it is impossible from our present experience to state exactly what kidney changes and lesions correspond to the appearances of the ureteral orifices, still there are certain cystoscopic pictures which are known to be due to definite kidney affections. The following descriptions of the ureteral orifice in health and disease and the clinical significance of the changes in its appearance and position are for the most part taken from Hurry Fenwick (*Obscure Diseases of the Kidneys*), a pioneer observer in this new field of diagnosis.

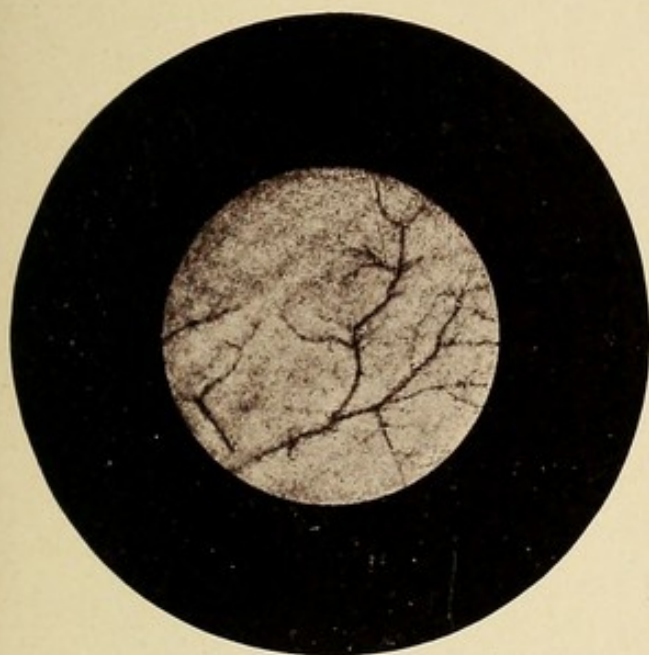
In practising ureteral meatoscopy the observer should note the position of the ureteral orifice, its shape and size, and the appearance of the surrounding and adjacent vesical mucosa. *The normal ureteral orifice* lies in the lateral angle of the trigonum, on a slightly elevated hillock, and appears as a faint, flesh-colored slit, or rounded hole.

The ureteral orifice may become elongated and furrowed, arched, dilated like a golf-hole, puckered, distorted, and warped; its *mucous membrane* may be congested, œdematous,

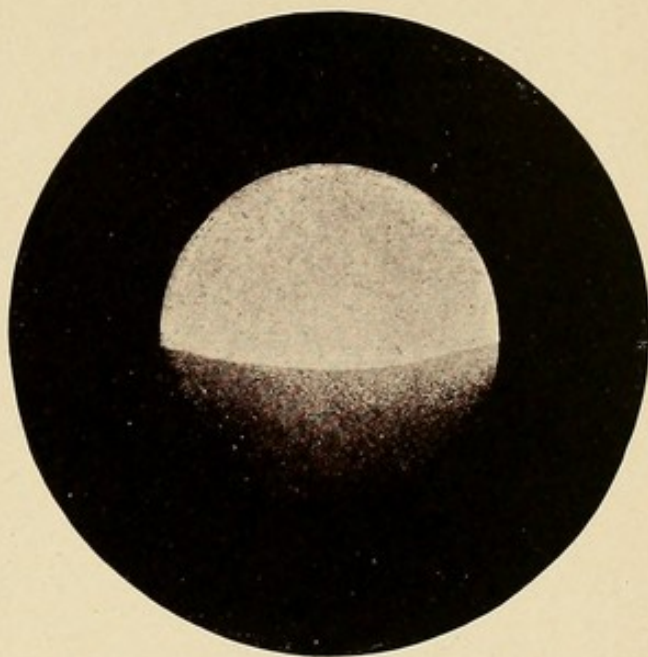
¹ The shape of the red blood cells is important as affording a hint to their source; thus crenated cells come from the higher urinary organs.

PLATE III.

A.



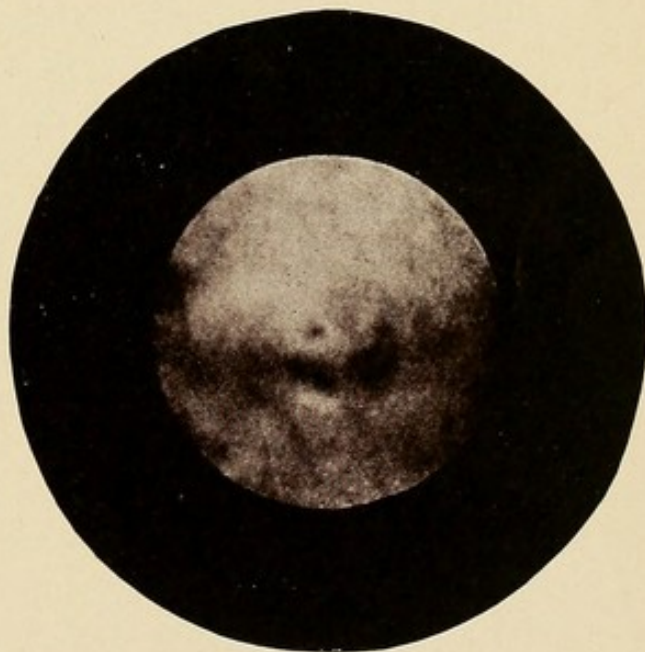
B.



C.



D.



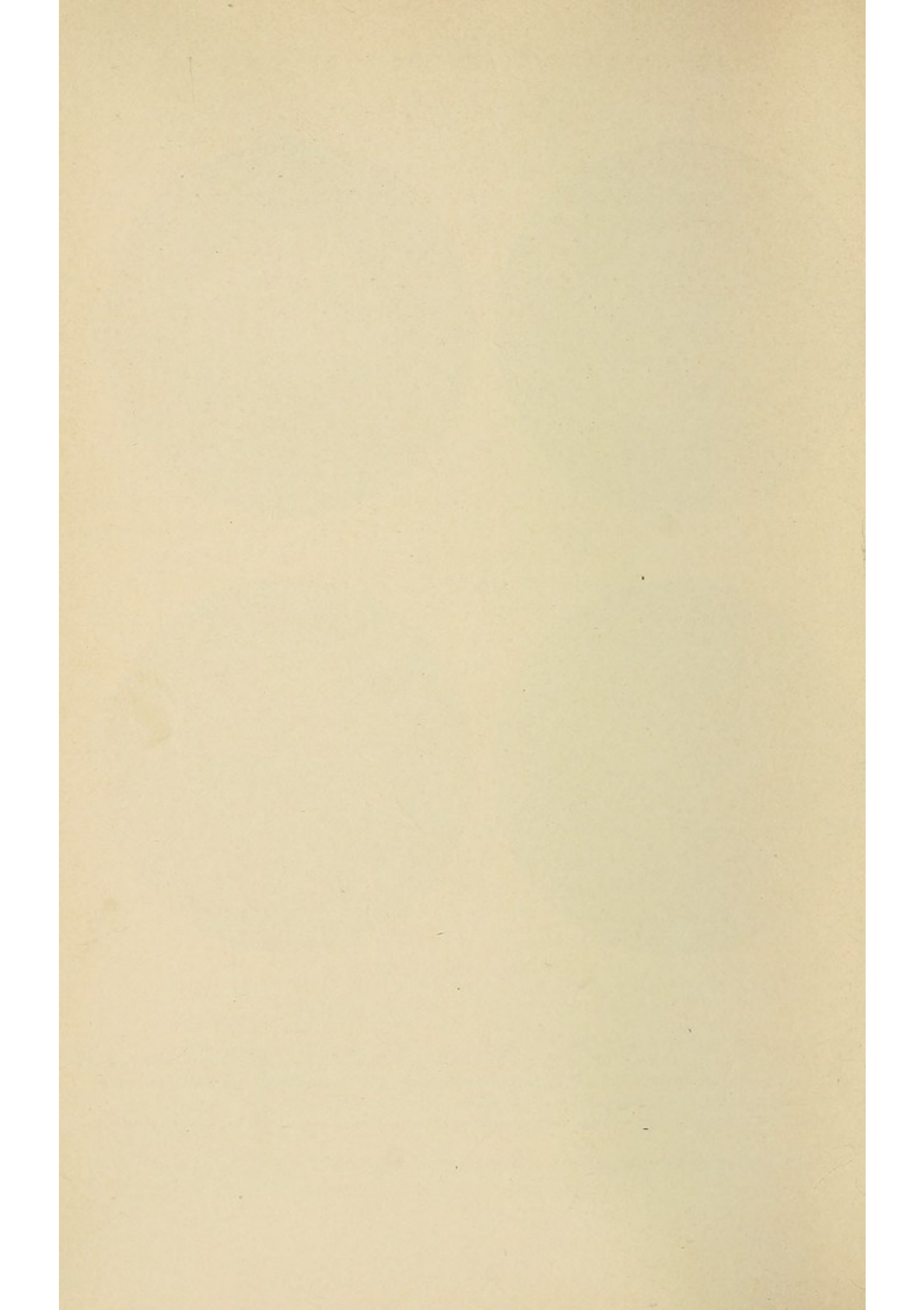
Cystoscopic Pictures of the Normal Bladder and Ureteral Orifices. (From Nitze.)

A. Appearance of the normal vesical mucosa.

B. Trigonum and adjoining portion of the base of the bladder. The darker lower half represents the trigonum. Note the unbroken slightly concave line of the normal prostate which separates the trigonum from the remaining portion of the vesical floor.

C. and D. Different varieties of the *normal ureteric orifice*. In C. the ureteric bar is very strongly marked.

Note the position of the orifice in the lateral angle of the trigonum.



eroded, ulcerated, or the seat of pinhead to pea-sized papillomata, or everted or prolapsed into the bladder.

Elongation of the orifice indicates a dilatation of the kidney pelvis, while *an arched shape* points to dilatation of the ureter from below upward, as occurs whenever there is an obstruction to the free passage of urine through the urethra.

The golf-holed orifice corresponds to a dilated, atonic kidney pelvis and ureter, and conical protrusion usually points to a kidney-stone which provokes colics.

With the *painless hæmaturia* which results from chronic granular nephritis, malignant disease, aseptic calculus, and some forms of tuberculosis, the ureteral orifice may be elongated and furrowed from distention of the kidney pelvis with blood, and somewhat congested as though streaked with fresh blood, or it may be unaltered.

With *renal calculus* the appearance of the ureteral orifice varies. In some cases there is no change; if the pelvis is *dilated*, the orifice is elongated and furrowed, possibly congested; and if there is *pyelitis* with alkaline urine, there are scald-like erosions and congestion of the immediate area around the orifice, its lips being everted and the trigonum congested and swollen; ultimately the orifice becomes warped and contracted. With *pyelitis* the efflux is muddy, the jets of urine being forcible and rapidly repeated or slow and sluggish. If there is entire *cessation* of the function of the kidney from coincident suppuration, the ureteral orifice is golf-holed and the efflux is slow and consists of solid or semisolid pus. If a stone is *passing* through the ureter and is near its *vesical* end, there may be punctate extravasations in the immediate neighborhood of the orifice, and the corresponding side of the interureteric bar becomes thick and swollen; as the stone approaches nearer the bladder the extravasations become more numerous, until finally the orifice and its immediate surroundings become uniformly red and appear to be bruised. Sometimes there is considerable œdema. *After* the transit of the stone the orifice may be œdematous; it is large and patulous if the stone was smooth, eroded and irregular if rough, and the surrounding area is red. If the stone is *arrested* in the lower third of the ureter, the mucous membrane of the orifice is protruded and everted, sometimes

roughened, again œdematous; the efflux may be perfectly normal.

With *renal tuberculosis* there may be no changes in the ureteral orifice. In the early stages of the disease the opening is usually golf-holed; all around it the mucous membrane is reddened, and on it are to be seen a few sparsely scattered tubercles, and not infrequently there are one or more sharply cut, sloughing ulcers behind the affected orifice; on the rest of the vesical mucosa further evidences of tuberculosis may be present, in the shape of red, extravasated areas with white, necrotic flakes on them or superficial erosions or worm-eaten ulcerations. In more *advanced* tuberculosis the ureter and corresponding part of the bladder wall are dragged out of their normal position, and ulcerations with red patches of extravasation are visible on other parts of the bladder wall; the orifice is patulous and irregular in shape, and its wall is thick and caked; or the orifice is thickened, irregular, and choked, or the seat of massive œdema, the latter being probably the result of an acute tuberculous pyelitis.

With *neoplasm* the orifice may be elongated and furrowed from distention of the kidney pelvis.

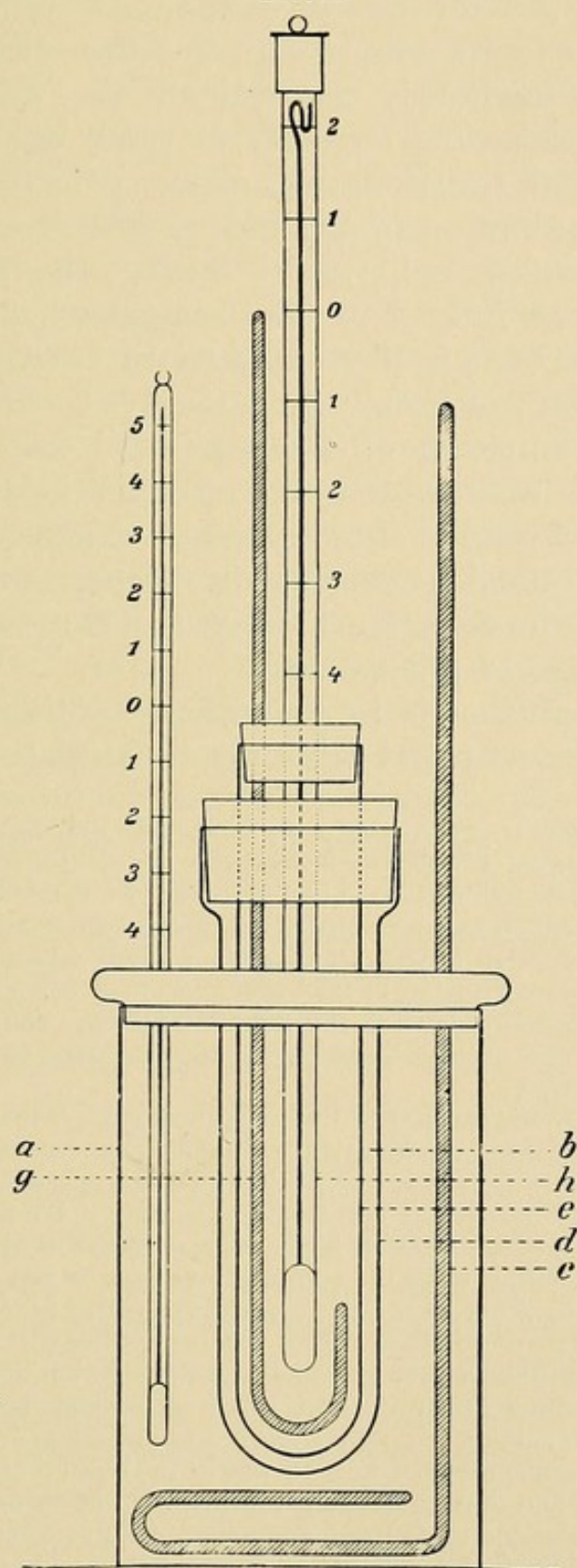
If the *peristaltic wave* at the ureteral orifice is strongly marked, the lips of the opening being drawn in, and the urine forcibly ejected, it indicates an hypertrophy and some dilatation of the ureter from increased work of the corresponding kidney, or from irritation of the ureter by a foreign body—*e. g.*, a calculus or blood clot or plug of mucus and pus.

An *efflux of clear urine* appears like a wave of glycerin passing inward across the trigonum. The efflux may be clear or opaque (bloody or purulent), rapidly repeated and forcible or slow and trickling. A *puriform* trickle indicates a crippled, suppurating kidney; a *blood* trickle points to a crippled kidney or to a moderate hemorrhage with diminished secretion of urine by the affected organ. A *solid pus* efflux indicates secretory death of the corresponding suppurating kidney; a *solid bloody efflux* points to a profuse renal hemorrhage.

X-ray Examination.—X-ray examination is especially important for detecting and locating calculi within the urinary

passages. Neoplasms may be recognized as deeper shadows surrounded by lighter areas.

FIG. 148



Beckmann's freezing apparatus. (Von Bergmann.)

Determination of Kidney Function.—The determination of kidney function is especially important when operative attack upon these organs is contemplated. No surgeon is justified at the present time in planning to remove one kidney or even to incise one except for the relief of anuria, unless he has previously ascertained the combined and individual functioning capacity of these organs.

The combined functioning capacity is determined by cryoscopic examination of the blood, and of a specimen of the total twenty-four-hour urine, during which period moderate amounts of fluid have been ingested, and from the amount of urea which is eliminated in twenty-four hours. The individual functioning capacity is determined by cryoscopic examination of the separated urines and from the amount of urea which is contained in the separated urines, and the relative functioning capacity of the two organs is determined by comparing the percentages of sugar which are contained in the individual urines after a subcutaneous injection of phloridzin.¹

The cryoscope that is in general use is that designed by Beckmann. Its essential parts are a vessel (*a*) to hold the

¹ There is at present no unanimity of opinion as to the relative value of these methods for determining the functional capacity of the kidneys. Some (Israel, Roosing, and others) maintain that the best indicator of a combined and individual sufficient kidney function is the percentage of urea in the total twenty-four-hour urine, and in the separated urines respectively; whereas others (Casper, Kummel, and others) believe that cryoscopy of the blood and of the combined and separated urines furnishes the best data as to the kidney function. The author uses both methods, and with one exception has never been deceived as to the functional capacity of the kidneys.

After all, it is functional capacity of the individual kidneys that concerns us most when operative attack upon these organs is contemplated. It would be manifestly wrong to remove one kidney or severely injure it if the opposite kidney were seriously diseased and of insufficient functioning capacity. We must not, however, accept blindly nor rest our decision for or against operation upon the knowledge of kidney function afforded us by the percentage of urea, or by the freezing point of the urine and blood. Thus, very often a perfectly healthy kidney works insufficiently because of disease of its fellow-organ. Such a kidney will resume its normal sufficient action when the diseased organ is removed. When, therefore, in a case of contemplated nephrectomy the urine drawn from the opposite kidney shows it to be the seat of no serious organic disease, we proceed to operation, even though its urea output and the cryoscopic index of its urine and of the blood indicate an insufficient function on its part, and thus speak against removal of the one organ. Each case of kidney disease must be studied by itself, and not only must we endeavor to ascertain the combined and individual functioning power of the two organs, but the causation of abnormalities in this latter respect must likewise be sought.

freezing mixture, a glass air-chamber (*b*), closed at the top with a perforated cork through which passes a test-tube (*e*) to contain the fluid, whose freezing point is to be determined. This last test-tube (*e*) is also closed at the top with a double perforated cork, one of the openings being for the Beckmann thermometer (*h*) and the other for a platinum glass stirrer (*g*). (The function of the latter is to enable us to keep the entire solution that is to be frozen at a uniform temperature.) The thermometer is peculiarly constructed. It has at the top a mercury reservoir, which is connected by a U-tube with the tube of the thermometer. From the reservoir sufficient mercury is shaken up into the U-tube so that the thermometer will register the freezing point of distilled water somewhere near the middle of the graduated portion. The graduations are in degrees, which are again subdivided into 100 parts. It is very important to bear in mind that this thermometer is not meant to register the temperature of the solution which is to be frozen. It is designed to register the point at which various fluids freeze, so that their freezing point can be compared with that of distilled water. This latter point on the thermometer scale is not a fixed one, but depends entirely upon the amount of mercury which we shake up into the U-tube at the top of the thermometer. We try to have sufficient mercury in this tube so that the thermometer will register the freezing point of distilled water somewhere near the middle of its graduated section.

The freezing point of water on the thermometer scale is to be determined first of all, and then the freezing point of the other solution; their difference represents the cryoscopic coefficient of the latter. We should use sufficient solution to completely cover the bulb of the thermometer. As a freezing mixture, ice and salt or a solution of ammonium nitrite in water will answer every purpose. During the freezing process the platinum glass stirrer is worked continually up and down, so as to maintain a uniform temperature of the solution that is to be frozen. As the freezing proceeds the mercury will be noticed to fall in the tube, and after reaching a certain level it suddenly rises and remains at a fixed point. The latter point is the freezing point, the fall below this being due to the fact that

the solution undercools before it really solidifies. If the freezing is continued with, the mercury will, of course, continue to fall as we lower the temperature of the ice. The freezing point of a solution depends on the amount of its dissolved molecules. It is evident that with insufficient kidney function the dissolved molecules in the blood which normally should be eliminated by the kidneys remain to a greater or less extent in solution in the blood, and consequently the molecular concentration of the latter rises, with corresponding lowering of its freezing point; and conversely the molecular concentration of the urine diminishes with consequent elevation of its freezing point as compared with that of distilled water, which is taken as a standard.

The blood with normally functioning kidneys freezes at 0.56° C. below distilled water; variations to 0.60° C. are within the limits of normality. If the freezing point falls below 0.60° C. renal insufficiency may be inferred. It is to be noted, however, that the freezing point may be below 0.60° C. as a result of heart disease, or from increased intra-abdominal pressure occasioned by large tumors, or from insufficient oxidation of the blood on account of respiratory disease, or during severe one-sided renal pain, even though one or both kidneys are healthy; due allowance must be made in such conditions for the lowering of the freezing point.

The urine normally freezes at 1.2° C. to 2.2° C. below distilled water. With renal insufficiency the freezing point of the urine approaches or equals that of the blood. With ingestion of large amounts of water the freezing point of the urine rises even to 0.1° C., and with ingestion of little water the freezing point falls to 3° C. The freezing point of urine should, therefore, be determined only after a moderate ingestion of fluids. If, under such circumstances, the freezing point is less than 1° C. and no severe anæmia is present, renal insufficiency must be assumed.

By catheterizing the ureters and collecting the secretion of each kidney separately, and then ascertaining their individual freezing points, we may by comparing the two results determine the functional activity of each organ; and as the functioning activity of an organ depends on the amount of

secreting structure in it, we may, also from a comparison of the individual freezing points of the urines of two diseased kidneys, ascertain which organ is most diseased.

The individual functional activity may also be determined by the methylene-blue and phloridzin tests, combined with the separate collection of the urines with the ureteral catheter. Of the two tests the phloridzin is the more rapid and reliable. To carry out this test both ureters are catheterized and then 0.005 gm. of phloridzin is injected subcutaneously into the buttock; the amount of *sugar* eliminated during the *same* time by each kidney is then to be determined.

Healthy organs eliminate from phloridzin the *same* amount of *sugar* at the *same* time. By comparing, therefore, the percentage of sugar eliminated by each kidney during the *same* period it is possible to determine the amount of working parenchyma in each organ.

(NOTE.—In the methylene-blue test—which is made by injecting 0.05 gm. of methylene blue into the buttock after preliminary catheterization of the ureters—it is necessary to watch for the beginning of the elimination, its cessation, its course, and its intensity. As the elimination may extend over three days, during which time the ureteral catheters would have to remain in place, this method is not as good a one as the phloridzin.)

CHAPTER XXXVI.

MALFORMATIONS AND DISPLACEMENTS OF THE KIDNEY.

CONGENITAL MALFORMATIONS OF THE KIDNEY.

Absence of One Kidney.—Absence of one kidney can only be diagnosed by the impermeability of the corresponding ureter in the whole or part of its extent, and by the absence of urinary secretion from this ureter.

Fusion.—*Fusion of the organs* into one horseshoe kidney or into an elongated kidney can be recognized in thin individuals by palpation, especially if the organ is movable. An increased number of ureteral openings in the bladder or other deformities of the urogenital system should excite our suspicions of such congenital malformations of the kidney. If, on exposure of one organ, it is found to be abnormally large, the suspicion of an abnormality in the number of kidneys should be entertained.

Congenital Sacral Kidneys.—*Congenital sacral or pelvic kidneys*, which are distinguished by their fixed position from floating kidneys that may lie in these regions, should be recognized by their pyramidal form and their lobulation. They attract attention only when they become inflamed or swollen, or when they exert pressure upon the pelvic organs.

Two or More Ureters.—Two or more ureters on one side can be made out with the cystoscope, provided the accessory ureters have a vesical opening into which a catheter can be passed. If the accessory ureter opens into its fellow the diagnosis cannot be made. If the accessory ureter opens into the urethra or seminal passages, the diagnosis may be made from the history of constant dripping, in spite of which the patient is still able to urinate voluntarily in a stream.

A pouch-like, mucous-membrane-covered tumor which lies in the bladder or urethra and which, in the latter instance, can be replaced into the bladder, and which on cystoscopic examination is seen to spring from the region of the ureteral orifice, suggests a cystic intraparietal dilatation of the vesical end of the ureter.

NEPHROPTOSIS AND REN MOBILIS.

A movable kidney is readily recognized by its kidney shape, its hilus, and its easy reposition into the loin, a position that it maintains as long as the patient remains recumbent and does not strain. Such a movable kidney may provoke no symptoms, or it may occasion a dragging or colicky pain in the side, nausea, vomiting, constipation, and occasionally jaundice. In some cases the pain is referred to the appendicular region, and this may occasion the belief that the patient has appendicitis. As chronic affections of the colon and appendix are frequently combined with nephroptosis, the only way we can decide whether the symptoms are due to the floating kidney alone is to support the kidney by a belt and see whether they disappear.

The kidney may be the only abdominal organ that is loose and prolapsed, or there may be a general visceral ptosis, the latter constituting *Glenard's disease*. Patients who suffer from this latter malady are, as a rule, thin, anæmic individuals who complain of gastric dyspepsia, constipation, general bodily aches and pains, general lassitude and other neurasthenic symptoms. As the prolapse of the kidney in these cases is but one of the disturbances from which the patient suffers, it is evident that nephropexy will do no material good, and therefore before proceeding to anchor a kidney we should always ascertain whether this organ alone or whether all the abdominal organs are prolapsed. This can readily be done by outlining the limits of these organs by percussion and palpation, a preliminary distention of the stomach and colon affording considerable aid in this examination.

It sometimes happens that the ureter of a floating kidney

becomes kinked, or that its pedicle becomes twisted. In the former instance the kidney becomes enlarged and tender (acute hydronephrosis) and the patients have colicky pains in the loin and sometimes hæmaturia; all of these symptoms disappear when the ureter becomes straightened out. Such attacks may be repeated from time to time, thus causing an intermittent hydronephrosis.

Strangulation of the kidney from twist in the pedicle occasions sudden intense pain with nausea, vomiting, faintness; even collapse, chills, and fever. The kidney is enlarged, tender, and painful, and the amount of urine which is voided is considerably diminished.

A movable kidney may be confounded with an *accessory hepatic lobe*, with *cysts of the liver*, and with *gall-bladder enlargements*. All tumors of the liver and gall-bladder share the respiratory mobility of these organs, whereas floating kidneys, except they have a long pedicle and a mesentery, have no respiratory mobility. The kidney is usually covered by the colon, which gives tympanitic resonance, whereas the gall-bladder and liver lie close to the anterior abdominal wall and the percussion note over them is dull. The most conclusive sign for differentiation lies in the ease with which a floating kidney can be replaced into the loin, which position it furthermore maintains when pressure upon it is relaxed, whereas all other tumors and swellings cannot be fully pushed into the loin and on their relaxation at once come out again.

A floating spleen or *enlarged spleen* is differentiated from a floating kidney by its characteristic form and its notched anterior border.

Tumors of the stomach and colon cannot be fully replaced into the loin, and they furthermore afford other clinical evidences that enable us to readily differentiate them from floating kidneys; thus with *pyloric tumors* of a malignant character there are the manifestations of pyloric stenosis and the gastric juice contains no free hydrochloric acid, but does contain lactic acid, and with benign pyloric tumors the evidences of stagnation within the stomach, with or without changes in the chemical composition of the gastric juice, are present. (See p. 270.)

With *intestinal tumors* that cause stenosis of the affected loop of bowel there are the evidences of chronic intestinal obstruction. (See p. 283.) In doubtful cases distention of the stomach and colon will throw light on the site and origin of the tumor.

Long-pedicle tumors of the ovaries may have the same shape and consistency as the kidneys, but they cannot be fully replaced into the loin, and on vaginal examination the pedicle of the tumor can be easily appreciated.

CHAPTER XXXVII.

INFLAMMATIONS OF THE KIDNEY.

SUPPURATIVE NEPHRITIS.

Acute Abscesses.—Of the acute abscesses of the kidney the large single or multiple ones are readily recognized from the following signs: A palpable enlargement and tenderness of the organ, associated with colicky pain in the side, pyuria and moderately severe constitutional manifestations of sepsis, such as repeated chills, continuous or intermittent fever, rapid pulse, profuse sweating, rapid emaciation, and high leukocyte count. Such abscesses are most frequently traceable to suppurations in other parts of the body, and especially to furuncles and acute osteomyelitis of the fingers and toes. The recognized frequency of kidney infection in such diseases should be ever kept in mind, and it should prompt us to carefully examine these organs when during their course the general constitutional symptoms point to other foci of suppuration.

The acute miliary abscesses of the kidney, a pathological condition of the organ that is better known to us as "surgical kidney," are especially apt to follow operations upon the lower urinary organs and the acute infectious diseases. Their presence is to be strongly suspected when, in these conditions, there is colicky pain in, some tenderness over, and possibly some enlargement of the kidneys, together with severe constitutional symptoms.

Chronic Abscesses.—It is but too commonly considered that suppuration within the kidney is always attended with pyuria, swelling of the organ, and more or less severe constitutional symptoms. This is by no means the case, for it not infrequently happens that one or several abscesses are present in the cortex of the kidney without there being any constitu-

tional or local symptoms or pus in the urine to indicate their presence. In such instances the abscesses are small, and not in communication with the kidney pelvis, and the organisms which have excited the suppuration have little or no virulence. Such cases of latent kidney abscesses have no interest to us, for the lack of all constitutional or local disturbances precludes the possibility of their coming under treatment. In most instances, however, the abscesses sooner or later rupture into the pelvis of the kidney, and from the clinical picture that is then afforded the diagnosis is readily made. The patients are thin, anæmic, and weak, and have a slight evening rise of temperature; the kidneys are enlarged, somewhat tender, and excite colicky pain in the loin; urination is frequent and the urine contains considerable quantities of pus. The cystoscopic appearances of the ureteral orifices help to confirm the diagnosis. Thus, soon after the rupture of the abscess into the kidney pelvis has occurred the ureteral orifices appear congested, scalded, and eroded, the efflux being muddy and forcible; later on the orifice is contracted, irregular and warped, and the efflux is slower and thicker—indications that the function of the kidney is being destroyed; and finally the efflux becomes a solid, puriform trickle—an indication of the secretory death of the kidney. The remaining vesical mucosa shows the evidences of chronic cystitis.

While it is easy to make the diagnosis of suppurating kidney at this stage, it is not always as easy to determine whether the suppuration was the primary malady or whether it was engrafted upon a preceding tuberculous or calculous condition. The previous history may afford us a clue as to this point. Thus a chronic suppuration in some other part of the body, especially the lower urinary organs, will suggest a primary suppurative process, whereas the history of previous attacks of kidney colic with hæmaturia, or a history that goes with tuberculosis of this organ (for which see p. 391) would rather indicate a secondary pyogenic infection upon a pre-existing stone or tuberculous condition. The presence of tubercle bacilli in the urine and the characteristic tuberculous lesions around the ureteric orifice and on the vesical mucosa are conclusive evidences of a mixed tuber-

culous and pyogenic infection, and similarly the shadows of stones in the affected kidney as shown by the *x*-ray affords us positive proof of a combined pyogenic and calculous condition.

The absence of a fluctuating tumor which varies in size from time to time, the fluctuations in size being coincident with a diminution or disappearance of or increase in the amount of pus in the urine, distinguishes abscesses of the kidney from pyonephrosis.

Some cases of surgical kidney do not manifest the local evidences which were described above as going with this condition; they exhibit only the constitutional symptoms, and as these simulate those which are occasioned by acute miliary tuberculosis, typhoid fever, and cerebrospinal meningitis, we must carefully exclude these maladies before deciding upon a diagnosis of surgical kidney.

HYDRONEPHROSIS AND PYONEPHROSIS.

Distention of the kidney pelvis with fluid is the characteristic sign by which we distinguish *hydronephrosis* and *pyonephrosis*. In the former clear urine is the distending material, and in the latter pus and urine. Such a distention is always due to an obstruction in the ureter, bladder, or urethra, which interferes with the free drainage of the kidney pelvis. This obstruction may be a continuously acting one—*e. g.*, a cancer of the uterus which compresses the ureter or a stricture of the ureter or urethra; or it may be present only at times—*e. g.*, a kink in the ureter or a calculus which intermittently blocks the ureter; in the former instance the distention is constant, and in the latter it is intermittent.

An acute onset of exquisite pain in the kidney, which becomes enlarged and stony hard, together with moderate fever, prostration, and diminution in the amount of urine which is voided, is indicative of an *acute hydronephrosis*. A large, gradually developing, smooth, fluctuating sac that is attended only with vague gastrointestinal symptoms, such as nausea, loss of appetite, thirst and constipation, points to a *chronic hydronephrosis*.

An acutely enlarged, painful tumor in which fluctuation may be obtained, with continuous or hectic fever with or without chills, emaciation, loss of appetite, profuse sweats, and high leukocyte count, etc., speak for acute pyonephrosis, and a more slowly forming tumor with gradual deterioration of the general health and moderate fever point to a chronic pyonephrosis.

During an attack of acute hydronephrosis the urine is diminished in amount, but it is clear and appears normal. The opposite kidney if healthy may take up the function of the obstructed one or it may itself suspend function, in which case anuria results. Between the attacks the urine is normal, both in amount and in composition.

With chronic hydronephrosis the urine may be cloudy from mucus, epithelial cells, and leukocytes. With pyonephrosis the urine contains pus, often in considerable amounts. If the opposite kidney is healthy and the ureter on the affected side becomes obstructed from time to time, the urine during such periods of obstruction is clear, but becomes purulent again when the obstruction is relieved. The pus is uniformly mixed with the urine. In hydronephrosis the urine is acid or neutral; in pyonephrosis, acid or alkaline.

The ureteral orifice in chronic cases of hydronephrosis is elongated and furrowed; with chronic pyonephrosis it is usually warped, contracted and irregular, or elongated and furrowed, or oval-shaped. By squeezing the kidney, pus may be seen to emerge from the ureteral orifice. In the acute cases of hydronephrosis the ureteral orifice is not changed; in acute pyonephrosis it may be congested or scalded or eroded.

The efflux during the period of obstruction is absent on the affected side, but on the opposite side it is likely to be more forcible. With pyonephrosis the efflux is muddy and trickling if the kidney is crippled, or a solid string of pus if its secretory function is lost. In the absence of a tumor and pain in the affected kidney the cystoscope is the only means by which we can ascertain from which kidney the pus in the urine is derived.

Pyonephrosis is readily differentiated from hydronephrosis by its attendant constitutional symptoms, by the pyuria,

and the eroded or warped or irregular ureteral orifice. Its differentiation from abscess of the kidney is not so easy, nor is it always possible. Intermittent pyuria, accompanied by variations in the size of the tumor, speaks for pyonephrosis. Pyuria, constitutional symptoms, and the appearance of the ureteral orifice distinguish pyonephrosis from all other abdominal tumors.¹

An acute, small hydronephrosis may from its stony hardness resemble a *carcinoma*, but it is distinguished from this by the exquisite pain and tenderness that accompany it.

Echinococcic cysts of the liver, enlarged gall-bladders, cysts of the pancreas, ovarian cysts, and cysts of the kidney must be differentiated from the chronic forms of hydronephrosis. In the first place the history or physical examination affords a cause for the hydronephrosis; then, again, the tumor occupies the loin and has the colon in front and to its inner side. Cysts of the liver and gall-bladder have moderate respiratory mobility, are directly connected with the liver, and lie above the colon. They can sometimes be pushed into the loin, but they do not remain there when pressure upon them is relaxed. With gall-bladder tumors there is a history of attacks of biliary colic, with or without jaundice, and stones in the stools. With echinococcic cysts there may be the characteristic fremitus.

Pancreatic cysts are retroperitoneal, but do not occupy the loin; they lie above the stomach, or between it and the colon, or below the colon; they may occasion intermittent glycosuria or lipuria. It is to be noted that large pancreatic cysts, like other large abdominal tumors, may by pressure on the ureter give rise to hydronephrosis.

Ovarian cysts with elongated pedicle may reach up into the loin, but they never fully occupy it; their pedicle can be felt per vaginam; they are freely movable; by traction upon the uterus the tumor is pulled downward; they are furthermore surrounded by tympanitic coils of intestine, thereby differing from hydronephrotic tumors, which have intestines only to the inner side.

The percentage of albuminuria due to pus can be estimated by counting the leukocytes; 50,000 to 70,000 cells correspond to 1 per cent. of albumin by the Esbach test. Rarely does albuminuria from this cause reach above 1 per cent.

It is to be especially noted that in chronic hydronephrosis the cystoscope shows the ureteral orifice elongated and furrowed, whereas in all the other conditions above mentioned the ureteral orifice is normal. By puncture of the hydronephrotic tumor little is to be learned. Its contents rarely contain much urea, in fact no more than may be found in the fluid of some ovarian cysts.

RENAL TUBERCULOSIS.

Recent clinical experience and experimental investigations would go to show that tuberculosis of the urinary organs originates in the kidneys; that ascending tuberculous infection of these organs is most improbable, if not impossible, and that a combined infection of the kidneys and genitals must explain the combination of urinary and genital tuberculosis. The infection of the urinary organs being primary in the kidneys, it is evident that the earlier the affected organ is removed, the better will be the prognosis for radical cure of the disease. The difficulty lies in making an early diagnosis of such renal infection. In the early stages there are very few symptoms, and these on account of their slightly annoying character are apt to escape the attention of the patient and his physician. After a shorter or longer period of such latency, however, during which the tuberculous process in the kidney is constantly progressing, active and distressing symptoms appear, and at this time the diagnosis is easy and should be readily made.

The earliest manifestations of the disease come from the bladder, which the tubercle-bacilli-laden urine irritates and subsequently infects. The early vesical symptoms are burning pain at the end of urination, frequency of urination, and inconstantly cloudy urine; in other words, symptoms of mild cystitis. Every case presenting such symptoms, that is not gonorrhœal in origin or due to infection by instrumentation or trauma, should be suspected of being tuberculous; in them the urine should be repeatedly and carefully examined for tubercle bacilli, and the cystoscopic appearances of the ureteral orifice and vesical mucosa should be ascertained.

(The differentiation of tubercle bacilli from smegma bacilli is made by drawing the urine with aseptic catheter and by staining methods.)

During the early stages of the disease there are not any or only very slight constitutional symptoms, and these do not point to the kidney. The patients may feel perfectly well or complain of lassitude, or of being easily exhausted; they may be pale and anæmic, but the kidneys are not enlarged, nor sensitive, nor tender. (Tuberculosis in other organs—the lungs, glands, etc.—should always be looked for, as its presence aids us in making the diagnosis.)

Cystoscopic examination in the early stages of tuberculous disease of the kidneys may reveal no changes in the bladder; these are to be looked for especially at the ureteral orifices. The first manifestations are a dilatation of the ureteral orifice, which is surrounded by reddened mucous membrane. A little later a few sparsely scattered tubercles and one or more sharply cut, sloughing ulcers become visible around or behind the ureteral orifice. The efflux is cloudy, but of normal force. Gradually the rest of the vesical mucosa shows evidences of tuberculous infection, in the shape of red, extravasated areas with white, necrotic flakes upon them, or superficial erosions, or worm-eaten ulcerations. These ureteral orificial changes are very characteristic and should prompt frequent and repeated examinations of the urine for tubercle bacilli.

As the tuberculosis of the kidney and the secondary infection of the bladder advance, the vesical and kidney symptoms become more marked. The kidney becomes enlarged, tender, and painful; tenesmus and frequency of urination are distressing, and pyuria, less frequently hæmaturia,¹ are present. Hectic fever, night-sweats, loss of weight, anæmia, and loss of appetite are the constitutional manifestations, and between the fever and the distressing tenesmus and frequent urination the individual is most wretched and miserable. At this stage the cystoscope shows the ureteral orifice and corresponding part of the bladder-wall dragged out of the normal position, ulcerated, patulous, and irregular, together with ulcerations

¹ Painless hæmaturia is sometimes the very first evidence of renal tuberculosis.

and extravasations on other parts of the vesical mucosa. At times the ureteral orifice is thickened, irregular, and choked or very œdematous, the result probably of a very acute tuberculous pyelitis. At this period the efflux is a puriform trickle; the urine contains many tubercle bacilli.

Every case of cystitis that is not gonorrhœal in origin or due to infection by instrumentation and trauma is to be viewed with suspicion, and especially so in women. Cystoscopic examination should be made at once and repeated from time to time, especial attention being directed to the appearances of the ureteral orifices and the character of the efflux. The urine should be repeatedly examined for tubercle bacilli, for which purpose several quarts of urine should be sedimented or centrifuged. If the urine is turbid the cystoscope will enable us to ascertain from which kidney the turbid urine descends, a knowledge that in the absence of all kidney symptoms is of the utmost importance for the localization of the disease. If the ureters are catheterized and the separated urines collected, the functional activity of each organ can be estimated, without which determination no extirpation of the kidney should be contemplated.

Floating and movable kidneys, and some cases of renal calculus, occasion frequent and painful urination and somewhat cloudy urine. The differentiation of the early stages of tuberculosis of the kidney from these conditions is attended with difficulty, and especially so if a tuberculous infection has been engrafted upon a movable or calculus-containing kidney, an occurrence that is by no means infrequent or rare. The cystoscopic appearance of the ureteral orifice and the presence of tubercle bacilli in the urine will have to be relied on to establish the differential diagnosis, or the combined affection.

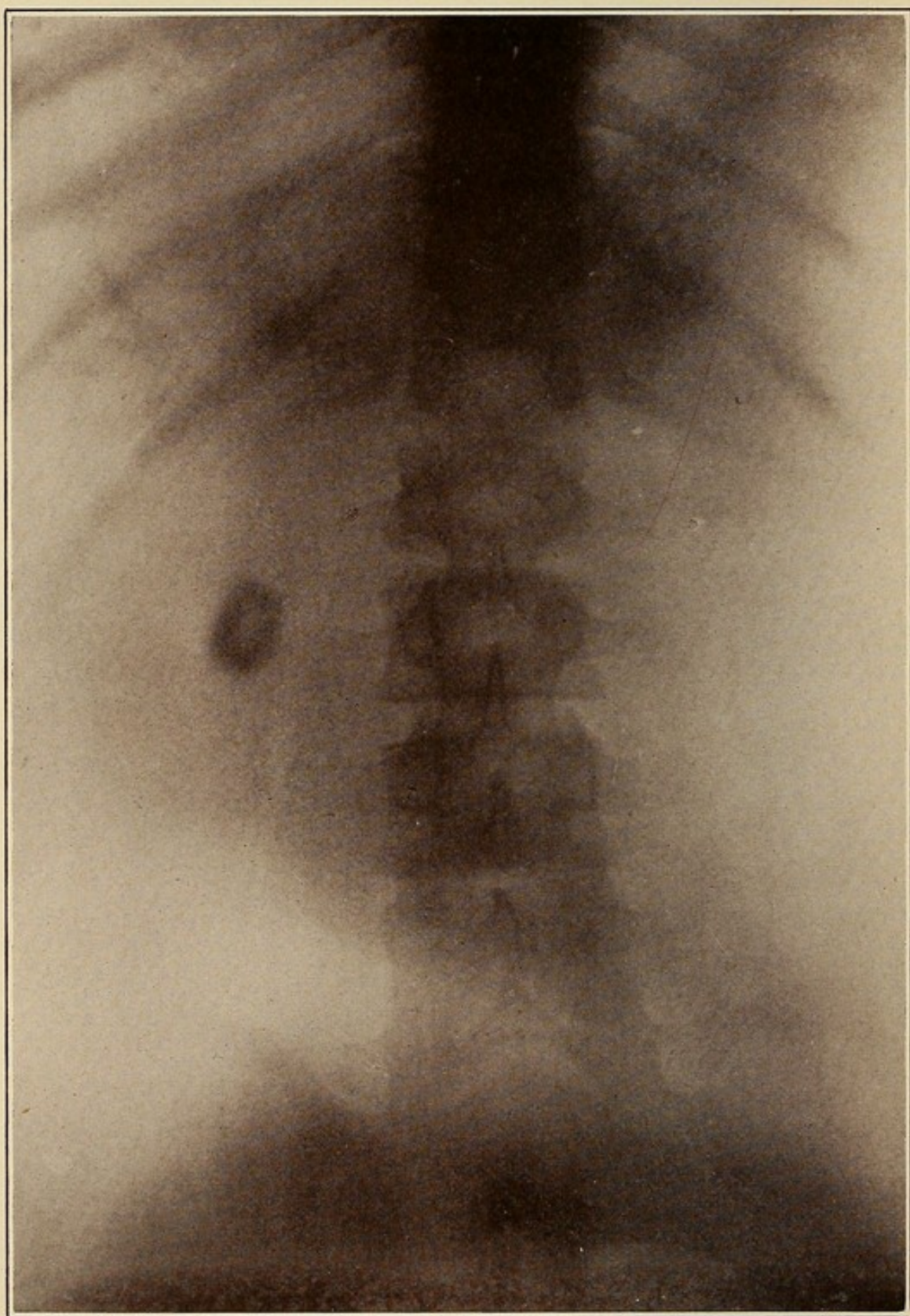
The later stages of tuberculous kidney disease may without the cystoscope be confused with suppurative nephritis, with which a tuberculosis may also be combined. The cystoscopic appearance of the ureteral orifices and of the vesical mucosa and the presence of tubercle bacilli in the urine will serve to differentiate the two affections when they are singly present, while a rapid course of the disease, with numerous pyogenic bacteria in the urine, will point to a mixed or com-

bined infection. Secretory death of the kidney is indicated by an efflux of solid pus from the ureter.

If calculus disease has been the primary affection the history would very likely afford the information of repeated kidney colics and hæmaturia, the *x*-ray would show the presence of calculi, and the cystoscopic picture of the ureteral orifices and the presence of tubercle bacilli in the urine would indicate the existence of a tuberculous infection. The secondary formation of calculi in a tuberculous kidney is only to be recognized from the passage of small calculi, and by *x*-ray examination.



PLATE IV.



Aseptic Renal Stone.

CHAPTER XXXVIII.

RENAL STONE.

UNTIL the introduction of the Roentgen ray our diagnosis of those aseptic renal stones that provoke only pain in the loin and hypochondrium was always uncertain, for there were no physical findings to direct us to the seat and cause of the suffering. In some of these cases we were able by a process of exclusion after long-continued observation of the patient, and after careful analysis of the gastric secretion, to rule out gastric and biliary affections as a cause of the symptoms; in some we were able to narrow down the possibilities to some affection of the kidney, but further than this we could not with any degree of certainty go. Nor did the occurrence of even repeated attacks of kidney colic with hæmaturia help us to a positive diagnosis of kidney stone, for in many of such cases in which we were led by these symptoms to expose and incise the kidney with the almost certain expectation of finding a stone, we were surprised to find no stone, but sometimes a chronic nephritis and again only perinephritic adhesions. A most carefully conducted urinary examination did not help us to differentiate these conditions, though the continued presence of crystals of uric acid or oxalate of lime strongly suggested a calculus, just as a preceding infection of the kidney during an acute infectious disease or otherwise suggested a chronic nephritis or perinephritic adhesions.

With the introduction of the *x*-ray, however, this difficulty has to a large extent disappeared, for it is the author's experience that an *expert radiographist* will seldom, if ever, fail to find an oxalate renal stone, and in most instances he will be able to obtain a shadow of a uric acid stone.¹ He may

¹ In very stout individuals the results of *x*-ray photography for renal calculi are not as satisfactory as they are in thin subjects. Due allowance must be made in such obese patients in forming our conclusions as to the presence or absence of a renal stone from the *x*-ray photograph.

have to take a number of exposures, but if he persists, he will be able to locate the stone if it is present.

When the stone commences to travel down the ureter the diagnosis becomes very much easier, for in addition to the kidney colic and hæmaturia, the passage of the stone through the lower portion of the ureter is attended with typical changes in the appearances of the vesical orifice of the ureter. (See p. 375.) Similarly when the stone becomes impacted in the lower third of the ureter there are characteristic changes in the vesical ureteral orifice. An acute hydro-nephrosis, indicated by exquisite pain, swelling and stony hardness of the kidney, following an attack of kidney colic with hæmaturia is strong evidence of an impacted stone in the ureter, just as is a complete anuria after a kidney colic. Complete anuria does not always signify that both ureters are obstructed by calculi, for it also occurs when only one of the ureters is obstructed, the function of the remaining kidney being reflexly suspended. As in these latter cases incision of the obstructed kidney alone usually suffices to relieve the anuria, it is a matter of the utmost importance to ascertain which is the obstructed kidney. This can be done only by the ureteral catheter; it is not safe to rely on the location of the pain for this determination, for in many instances of unilateral renal stone the patients complain of pain in the opposite healthy organ. The ureteral catheter informs us not only of the presence of an obstruction, but also of its site, a most important point when operative interference for its removal is to be undertaken.

When a kidney that is the seat of a stone becomes infected and in virtue of such infection a pyelitis or a pyonephrosis or suppurative nephritis develops, the evidences afforded by the latter conditions are usually clear enough to render their recognition easy. It is not always so simple, however, to ascertain that they are primarily due to a stone. A history of attacks of renal colic and hæmaturia is very suggestive of their connection with renal calculus.

The pain in renal colic is sharp and cutting in character, commencing in the loin and radiating down into the genitals and thigh. It resembles in its character that which is due to biliary colic, and gastric or duodenal ulcer. Renal colic,

however, is usually attended with hæmaturia; as this hæmaturia may be only microscopic, it is of the highest importance to carefully examine the urine in all cases for the presence of red blood cells. In biliary colic the pain starts in the right hypochondrium and radiates to the back and right shoulder. The pain due to gastric ulcer is usually accompanied by other evidences of this malady—viz., vomiting, hyperacidity, hæmatemesis, or melæna. The pain in duodenal ulcer is most severe two or three hours after a meal, but the differentiation of the two conditions without the *x*-ray is sometimes exceedingly difficult. The plan suggested by Kelly to demonstrate the presence of a stone in the ureter or kidney pelvis—viz., to pass a waxed-tipped catheter into the ureter and see whether the wax is scratched—is of much less diagnostic value than is the Roentgen-ray examination.

CHAPTER XXXIX.

NEOPLASMS OF THE KIDNEY.

THE clinically important kidney tumors are chiefly met with during two periods of life—before puberty (in some cases very shortly after birth) and after the fortieth year. Benign tumors, as lipoma, fibroma, adenoma, and chondroma, are of infrequent occurrence; malign tumors, as carcinoma and sarcoma, are relatively frequent; and hypernephroma, simple cysts, polycystic degeneration, and echinococcic cysts less so.

The best clinical evidence of a neoplasm is the presence of a palpable tumor. Should the tumor be located at the upper pole or on the posterior surface of the organ, its detection by palpation would be long delayed, unless the patient is thin and the kidney movable. Malignant tumors are usually hard with irregular nodular surface, whereas benign growths and cysts have a smooth surface.

Other clinical evidences of a neoplasm are renal pain and hæmaturia. These are usually present before a tumor can be palpated. The pain is due either to distention of the kidney pelvis with blood, in which case it is colicky in character, or it is due to the neoplasm itself, in which case it is a continuous dull ache. The hæmaturia varies in the severity and constancy of its occurrence. With benign growths and some malignant ones, it never occurs; again, in some cases it is very slight in amount, and unless accompanied by pain it is not apt to attract the patient's attention. In other cases it is very profuse and lasts several days, and is accompanied by severe colicky pain.

The urine at the times of bleeding is bright red or smoky in color, and at times contains ureteral and urethral blood clots. Israel considers that old, decolorized casts passed with clear urine undoubtedly come from the ureter, and that soft,

white or light yellow or red, earthworm-like clots are characteristic of kidney tumor. Under the microscope numerous red and white blood cells, fat droplets, casts, and epithelia of all kinds are to be seen.

Anæmia, cachexia, and emaciation are late evidences of kidney tumors. Guyon lays stress on the occurrence of varicocele with neoplasms of the kidney.

Benign Tumors.—Benign tumors are of little clinical importance; they rarely attain large size and do not give rise to any symptoms.

Malignant Growths.—Malignant growths are quite common. The *carcinomata* form nodular or smooth infiltrating tumors of hard or soft consistency, which, as a rule, can be palpated, and are accompanied by renal pain and hæmaturia. The *sarcomata*, *endotheliomata* and *embryonal sarcomata* of children exhibit the usual characteristics of malignant tumors, though the bleeding may not be marked. *Embryonal sarcomata* occur during the first two years of life, though they have been met with as late as the fourteenth year. They grow very fast, rarely give rise to hæmaturia, and are not very likely to occasion metastases. In almost every one of these particulars they differ from the *hypernephromata* (tumors of aberrant adrenal tissue in the kidney). These latter are almost invariably encountered after the thirtieth year of life, grow slowly, and are attended with marked and profuse hæmaturia, which is, as a rule, the first indication of their presence.

Cystic Tumors.—Of cystic tumors there may be single cysts, or echinococcus cysts, or the entire organ may be the seat of polycystic degeneration. The last mentioned may be congenital in origin or develop in later life; it is usually bilateral. Many of the infants affected with bilateral cystic degeneration of the kidneys are not viable; their kidneys are represented by large tumors with nodular surface, which it is scarcely possible to differentiate from solid growths. The acquired polycystic degeneration is often combined with cysts of the liver; it is likewise bilateral in the majority of the cases. As the hæmaturia, colicky pain, and tumor which result from this disease also accompany other neoplasms of the kidney, it is not always possible to make the diagnosis.

In favor of polycystic degeneration are the following facts: A history of other members of the family having suffered from the same affection, a slow growth of the tumor, and an absence of cachexia and of all data pointing to tuberculosis or suppuration of the kidney. Probatory puncture through the loin will differentiate a polycystic kidney from a hydro-nephrotic sac, for by puncture we empty only one of the cysts and the tumor consequently remains, whereas hydro-nephrotic sacs are entirely evacuated by puncture, with disappearance of the tumor. The bilateral character of the disease may be recognized from the symptoms of contracted kidney with cardiac hypertrophy. Bilateral disease, accompanied by cysts of the liver, and the symptoms of contracted kidney and cardiac hypertrophy make the diagnosis positive.

Single cysts of the kidney are not common. They vary in size, and may grow to the size of an adult head. They have been noticed between the eighteenth and sixty-fifth years of life.

Echinococcic cysts are first noticed when they reach a large size; they grow slowly, form smooth, rounded tumors, and do not occasion any material symptoms. If they perforate into the ureter so that daughter-cysts are passed with the urine (always with an attack of kidney colic), the diagnosis is clear, otherwise this will remain doubtful until the tumor is exposed on the operating table.

Adrenal Tumors.—Adrenal tumors, which are most frequently of a tuberculous nature, cannot be palpated unless they reach a large size, and at this time their differentiation from kidney tumors is very difficult. Clinically they are distinguished from renal neoplasms by the fact that they do not frequently occasion hæmaturia. The chief symptoms to which they give rise result from compression of the lumbar nerves and vena cava inferior—viz., pain in the area of distribution of these nerves, ascites and œdema of the legs. Bronzing of the skin and mucous membranes, which frequently accompanies adrenal disease, is likely to be absent with neoplasms of this organ.

CHAPTER XL.

DISEASES OF THE URETER.

Accidental trauma rarely results in injuries of the ureter alone; it generally occasions wounds of other viscera as well, and the evidences of these mask the symptoms which come from the injured ureter. The usual sequela of a torn or crushed ureter is the formation of a retroperitoneal tumor; this is tender and painful and yields, on aspiration, urine or urine mixed with a little blood.

Accidental injuries of the ureter during pelvic and abdominal operations are recognized from the profuse secretions from the wound immediately after operation, and from the diminished amount of urine, possibly bloody in character, which is voided from the bladder. Intraperitoneal injuries with consequent escape of urine into the peritoneal cavity are recognized only after the abdomen has been opened for treatment of the septic peritonitis to which the lesion gives rise.

The *inflammations* of the ureter are usually secondary to those of the kidney and bladder, and are recognized from the thickening of the tube and the pain and tenderness along its course. The thickening can be appreciated by abdominal, by rectal, and by vaginal palpation. Inflammation of the right ureter has been mistaken for disease of the appendix, but the presence of vesical or kidney disease and the absence of a typical history of appendicitis should enable us to readily differentiate the two. Sometimes appendicular or other abscesses perforate into the ureter, and so occasion vesical tenesmus and pyuria. This condition will be understood if we find from the history and from examination that an abscess was present prior to the onset of the vesical tenesmus and pyuria. The cystoscope will distinguish perforations of abscess into the bladder from those into the ureter; with the former we ought to be able to see the site of perforation.

Kinks, strictures, and valve formation are to be recognized from the hydronephrosis which they occasion and from the obstruction such conditions afford to the passage of the ureteral catheter.¹

Neoplasms are very rare, but papilloma, sarcoma, and carcinoma have been reported. They obstruct the ureteral channel, cause a hydronephrosis, and occasion hæmaturia, from which signs their presence may with considerable reservation be assumed.

¹ Incomplete obstruction of the ureter by stricture or kink is evidenced by dull pain along the course of the ureter and by frequent urination. The diagnosis can only be made by the ureteral catheter.

CHAPTER XLI.

DISEASES AND INJURIES OF THE URINARY BLADDER.

A SOMEWHAT similar symptom complex attends most of the diseases of the urinary bladder, and by its close study we will be able, as a rule, to differentiate the disorders of this organ from those of the rest of the urinary apparatus. But, though it is possible from the physical sufferings of the patient to locate the seat of the urinary malady in the bladder, it is not usually possible to learn the precise nature of the affection in this way. To ascertain this we must palpate,¹ percuss, cystoscope, and sound the bladder, estimate its capacity, and find out whether it empties itself at each act of urination.

The complaints that direct our attention to the urinary bladder are pain, tenesmus, and increased frequency of urination. Changes in the force, calibre, and character of the urinary stream are sometimes in evidence, pyuria only with the inflammatory and ulcerative diseases, and hæmaturia with the inflammatory, ulcerative, neoplastic, and calculous diseases of the viscus.

In vesical disease a dull ache or *pain* may be continuously present in the hypogastric or perineal regions. It is usually severe at the beginning of and throughout urination, and when the vesical orifice is involved it is especially acute at the end of the act. In the latter instance it is referred to the glans penis.

Tenesmus is especially marked in the inflammatory diseases. The patient may have to pass water every few minutes, and that with great pain and straining. The pain is most severe just before and at the end of urination.

¹ In palpating the bladder, we should not forget to avail ourselves of bimanual palpation, the finger of the one examining hand being introduced into the rectum or vagina.

The *frequency* of urination depends on the nature of the malady, its location, the size of the bladder, and upon whether the viscus empties itself at each act of urination. In inflammatory and ulcerative diseases, especially if they involve the vesical neck, there is a desire to pass water every few minutes; with neoplasm and stone, not complicated by cystitis, the frequency is not much altered; and in cases of partial retention or with a contracted bladder the frequency depends upon the amount of residual urine and the capacity of the organ.

Pyuria occurs only with the inflammatory and ulcerative diseases. *Vesical pyuria* is readily differentiated from *renal pyuria* by the following procedure: the bladder is washed until the water returns clean; if now the bladder is filled with clean water, this will become rapidly turbid if the pus descends from the kidneys, and remain clean for a much longer time if the pus is vesical in its origin. Vesical pyuria is to be differentiated from urethral pyuria by making the patient urinate into three glasses. The first couple of ounces, which wash out the urethra, are passed into one beaker, and the rest into two other beakers. If the first urine is turbid, whereas the second and third are clear, the pus comes from the anterior urethra; if all are cloudy it comes from the bladder; if the first is most turbid, the second less so, and the third still less, the pus is derived from the posterior urethra; if the first and second are cloudy and the last most so, the pus is derived from the bladder and trigonum.

Cloudiness of the urine is not to be interpreted in every case as due to pus. It may be caused by uric acid, phosphates, oxalates, fat, chyle, or bacteria. If it is due to uric acid it is dispelled by heat; if it is due to phosphates it disappears on the addition of acetic acid, and if it is caused by oxalates it is cleared up by adding hydrochloric acid. If these tests do not succeed in clearing up the urine, and potassium hydrate is now added to it, a gelatinous transparency will take the place of the turbidity if it is due to pus (Donné's test). If this test fails we may assume that the cloudiness is due to bacteria, and their presence is then to be verified by microscopic examination. Cloudiness of the urine due to fat is dispelled by the addition of alcohol ether.

With the inflammatory and ulcerative diseases of the bladder, the urine is usually alkaline in reaction. The ammonia which is set free by the alkaline decomposition of the urine reacts upon the pus and changes it into a ropy, reddish sediment, which adheres to the vessel into which the urine is passed.

Abundant epithelial cells are present in the urine of those suffering with *vesical disease*, but it is not possible to state from the shape and character of the epithelia what their source is. The presence of fragments of papillary or malignant tumors in the urine usually points to such disease of this organ.

Hæmaturia occurs in greater or less amount and at some time or other with most of the vesical diseases. The blood is uniformly distributed in the urine, and the clots are rounded. Vesical hæmaturia is to be differentiated from renal hæmaturia only by the aid of the cystoscope, and from urethral hæmaturia by cystoscopy and endoscopy.

While the symptoms just detailed point to vesical disease, the nature of the latter can only be ascertained by physical examination, cystoscopy, and sounding. A hint as to its character may sometimes be gleaned from the history. Thus with stone and pedunculated tumors the patient experiences sudden stoppage of the urinary stream, which a change in his position may prevent, and which a certain posture during the act of urination may avoid. Again, patients with stone in the bladder, and especially the children who are thus afflicted, are noticed to pull on the penis and frequently masturbate, and their straining to overcome the sudden impediment to urination leads to prolapsus ani and hernia.¹ With cystitis the tenesmus is especially marked and the urine is usually turbid and alkaline. With neoplasm the first symptom is very often "hæmaturia." Such data suggest the diagnosis, which can then be confirmed with the cystoscope and searcher. In acute diseases, however, the use of these instruments is attended with so much pain, and the possibility of aggravating the malady by their use is so great, that

¹ It is worth while to examine children, who have an acquired hernia from no accountable cause, for vesical calculus.

it is preferable to rely upon the symptoms for making a diagnosis during the early stages.

In contracted bladder due to tuberculosis cystoscopy is likewise not well borne, and if the diagnosis can be made without its aid, the patient will be spared much pain and possibly an exacerbation of the malady.

If there is much bleeding from the bladder, cystoscopy will usually be impossible. The lesser degrees can be overcome by irrigating the bladder with 1 to 10,000 adrenalin solution or by using the Nitze irrigation cystoscope.

CYSTOSCOPIC APPEARANCES IN DISEASES OF THE BLADDER.

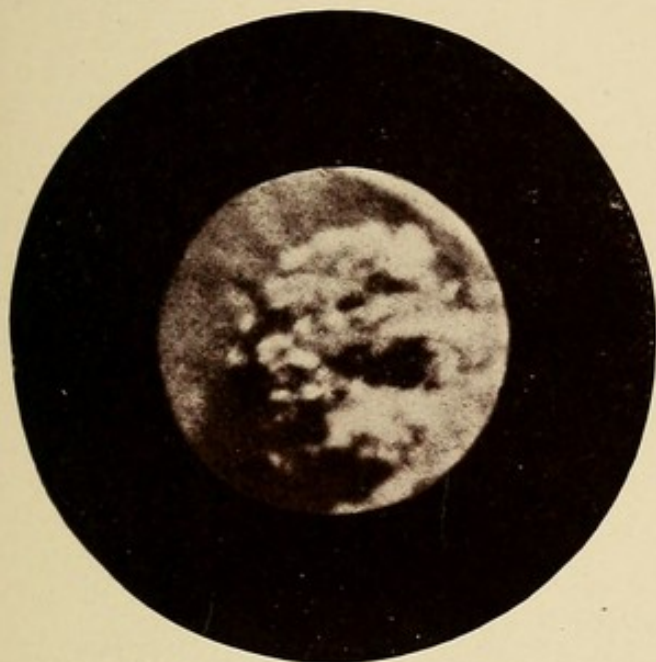
Cystitis.—In *acute* cystitis the mucous membrane, especially that of the trigonum and vesical neck, is deeply injected, swollen, of intense red color, and almost velvety in appearance. Flakes of mucus and pus are visible on its surface.

In *chronic* cystitis the mucous membrane appears swollen, somewhat thickened and congested, is covered with patches of grayish-yellow pus and mucus, and is studded with ecchymotic areas; sometimes there are to be seen ulcerations of varying size and depth, and occasionally there are pseudovesicles in the œdematous mucous membrane or polypus-like proliferations in the mucosa. These evidences are most marked at the trigonum, which is of a dark-red color, swollen, and raised into numerous thickened folds. In cases of cystitis secondary to conditions which interfere with the free passage of urine from the bladder—*e. g.*, urethral strictures, hypertrophy of the prostate, etc.—there are prominent ridges and trabeculæ on the interior of the vesical wall from thickening of the muscular coat, and between them the mucous membrane is pouched out into diverticula and sacculations which sometimes contain secondary phosphatic calculi.

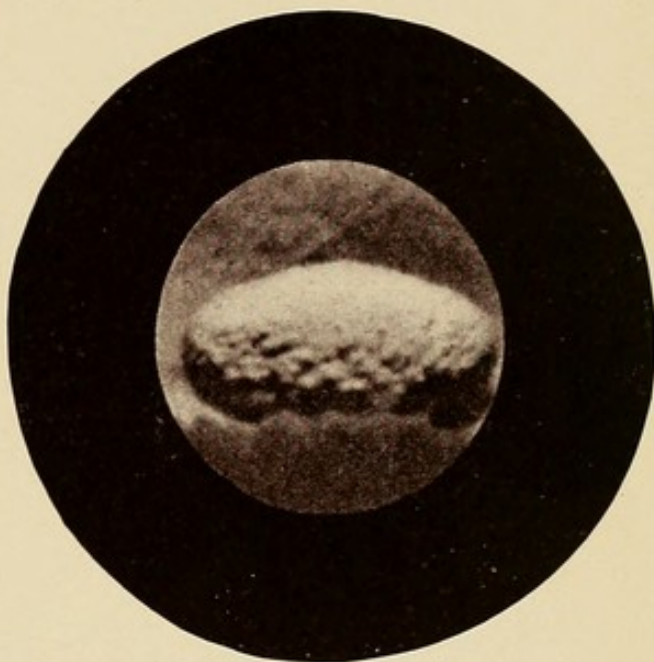
Tuberculosis.—If the vesical affection is secondary to a kidney tuberculosis, the lesions are most marked around the ureteral orifices; on the other hand, when it is second-

PLATE V.

A.



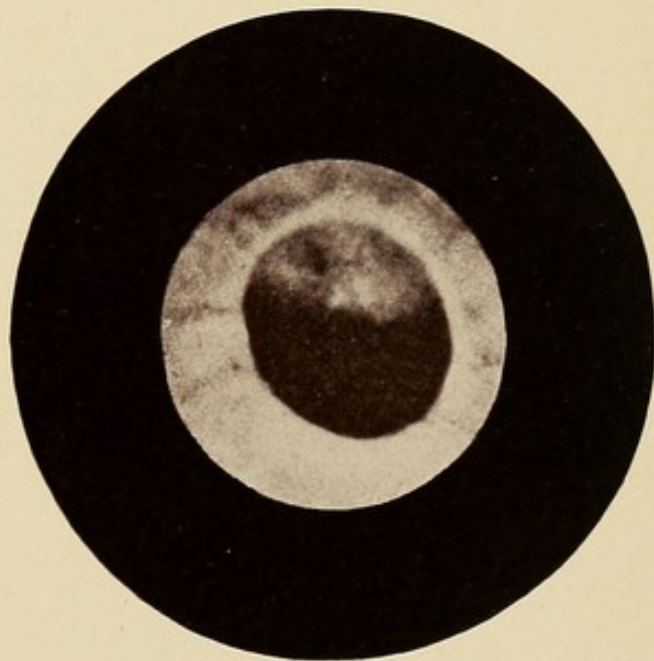
C.



B.



D.



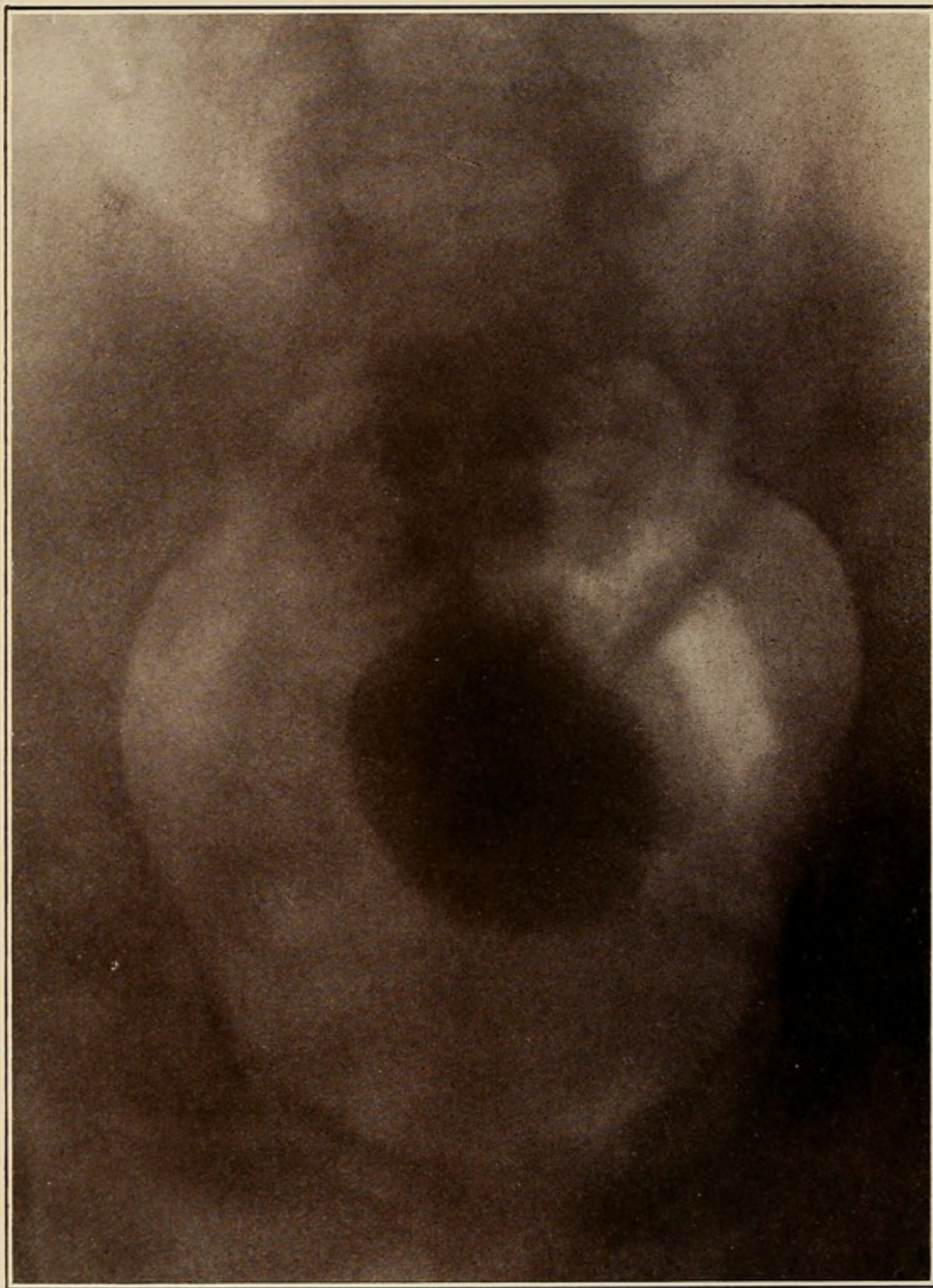
- A. Villous tumor of the bladder.
B. Cancerous tumor of the bladder.
C. Uric acid stone.
D. Congenital diverticulum.

Note absence of trabeculation of bladder wall in *D*, which differentiates a congenital diverticulum from an acquired diverticulum. (From Nitze.)





PLATE VI.



Large Vesical Stone.

ary to genital tuberculosis the lesions are diffused over the whole bladder wall. For the description of the lesions around the ureteral orifices, see p. 374. On the rest of the vesical mucosa in the early stages of the disease are red extravasated areas with white necrotic flakes on them or grayish-white tubercles, or superficial erosions or worm-eaten ulcerations. In more advanced stages of the disease the bladder is contracted and trabeculated, the mucous membrane is thickened and covered with thick pus and mucus and is studded with worm-eaten ulcerations, red areas of extravasation, and groups of grayish-white tubercles.

Papillomata.—Papillomata appear as shaggy, papillary, pedunculated, or sessile, single or multiple tumors, generally situated at the ureteral orifices or fundus of the bladder.

Carcinomata.—Carcinomata appear as shaggy, infiltrating, thickened tumors, or as flattened infiltrations or as everted, irregular, easily bleeding ulcerations.

Calculi.—Calculi are readily recognized, and their shape, size, color, and number easily determined.

The presence or absence of stone may also be determined by the searcher and *x*-ray examination. By palpation of the base of the bladder through the rectum the presence of a neoplasm and occasionally that of calculi can be determined, and by bimanual palpation through the rectum and over the hypogastric region the presence of mural neoplasms or abscesses, or of large calculi may at times be detected.

By percussion and palpation over the hypogastrium the oval, fluid-containing (fluctuating) dull tumor representing a distended bladder can be detected. Digital examination of the interior of the bladder in the female after dilating the urethra is rarely resorted to for diagnostic purposes.

The examination for tubercle bacilli should be made in all suspected cases of tuberculosis; in this connection it is to be especially noted that cases of chronic cystitis for which no etiological cause can be ascertained and which are rebellious to local treatment are very likely tuberculous in character, and careful examination of the urogenital system, especially the kidneys and testicles, is to be made for primary foci of this disease.

INJURIES OF THE BLADDER.

The history of an injury¹ over the hypogastric region, and especially if such injury has caused a fracture of the pelvis, together with difficulty in urination, tenesmus, and bloody urine, is very suggestive of an injury to the bladder or urethra. A rupture of the bladder may be assumed if catheterism is easy and if very little bloody urine is found in the bladder. The assumption can be verified by injecting a measured quantity of fluid into the bladder and then measuring the amount that returns through the catheter. With a bladder rupture the return amount does not correspond with the amount which has been injected. Bleeding from the urethra independent of urination suggests a rupture or injury of the urethra, in which case the passage of the catheter is likely to be obstructed.

It is not always possible to tell at the outset whether the site of the rupture in the bladder is on its peritoneal or extraperitoneal surface. But if the rent is not at once repaired the subsequent developments will reveal its site; for with intra-peritoneal rupture there develops sooner or later, depending on the aseptic or septic condition of the urine, a diffuse septic peritonitis, and with extraperitoneal rupture there develops a gangrenous, phlegmonous inflammation of the cellular tissues in front and at the base of the bladder.

¹ It is to be noted that the patient may have been under the influence of alcohol or other narcotic when the injury was sustained and consequently is not conscious of it, and also that in distended conditions of the bladder a very slight traumatism that ordinarily is not severe enough to make an impression upon the individual may result in a rupture of this viscus.

CHAPTER XLII.

URINARY FISTULÆ: DISTURBANCES OF MICTURITION.

Fistulæ connected with the urinary organs are readily recognized from the urinous fluid which they discharge. The internal orifice may be located in the kidney, ureter, bladder or urethra, and the external opening in the loin, on the abdominal wall, the external genitals, in the uterus or vagina, in the rectum or on the thighs.¹

In every case of such fistula it is essential to locate the internal and the external openings. If the internal orifice is situated in the kidney, in the ureter or in the bladder, there is a constant dribbling of urine from the external opening, whereas if it is situated in the urethra there is a leakage of urine only during urination, unless the patient is incontinent from other causes. To differentiate the ureteral from the vesical fistulæ, we fill the bladder with colored fluid and watch the external opening of the fistula. If the fistula is a vesical one the colored fluid will at once appear at the external orifice. Cystoscopic examination will also aid us; for in ureteral fistulæ there is no urinary efflux² from the corresponding ureter.

The location of the external opening of the fistula is easily determined except in those cases in which its site is in the cervix uteri, rectum, or vagina. In such cases considerable aid is afforded by giving the patient a dose of methylene blue and then watching the suspected region for the discharge of the blue-stained urine.

The internal orifice of urethral fistulæ may often be located by passing a metal instrument into the urethra and

¹ Fistulæ are designated according to the site of the internal and external orifices; thus, vesicovaginal, ureterovaginal, etc.

² The efflux can be more easily seen and watched if we inject subcutaneously twenty minutes before our cystoscopic examination about thirty minims of a 4 per cent. solution of indigo carmine.

a metal probe through the fistulous canal from the external opening. *Their first point* of contact indicates the site of the internal orifice. With tortuous fistulæ it may be very difficult to pass a probe; in such cases the internal orifice may be located by endoscopic examination, combined with injections of colored fluid into the fistulous canal.

Disturbances of micturition are always symptomatic of disease of the urogenital organs, and we should in every instance attempt to discover their underlying cause. Normally the urine should be passed at regular intervals of several hours, in a good-sized stream, with good force and without pain. Extreme deviations from such a normal course are a total inability to pass urine (retention) and a total inability to retain any urine in the bladder (incontinence). Lesser degrees of these conditions also occur.

Complete retention is due to vesical paralysis, as occurs in diseases or injuries of the spinal cord; to obstruction of the ureteral canal—*e. g.*, by neoplasm, foreign bodies, calculi, etc.; to stricture of the urethra, to spasm of the sphincter vesicæ, or to rupture of the urethra. With this condition the bladder becomes distended and forms a globular fluctuating tumor that gives a dull percussion note in the hypogastric region. The inability to pass urine on account of any of the above causes should not be confounded with suppression of urine, in which condition no urine is secreted and the bladder is consequently empty and collapsed.

Obstruction of the urethral canal rarely causes complete retention; a little urine, if only a few drops, can usually be passed. The nature of the obstruction is to be determined from the anamnesis, by palpation of the prostate, by sounding, and by endoscopic and cystoscopic examination.

Spasm of the vesical sphincter may be hysterical or reflex in character. Other neurotic symptoms and variations in the intensity of the spasm, together with an absence of an organic basis for the spasm, are in favor of its being hysterical in character, while the presence of vesical inflammation, ulceration, or other disease as seen with the cystoscope signifies to its reflex nature.

Incontinence of urine—*i. e.*, an inability to hold urine—may be complete or partial and either functional or organic

in nature. Of the functional incontinence the most familiar example is the nocturnal enuresis which is frequently met with in male children during their early life and which disappears spontaneously when they reach puberty.

Paralysis of and changes in the sphincter, foreign bodies which are lodged in the internal urethral orifice and thereby interfere with sphincteric closure, and large vesical fistulæ lead to complete incontinence.

Tardiness in starting the stream may be due to general muscular debility, or to disorders at the vesical neck—*i. e.*, hypertrophied prostate, deep urethral strictures, etc.

Diminution in the calibre of the stream and forking and twistings thereof as it emerges from the urinary meatus point principally to strictures of the urethra, but may also be due to obturation or compression of the urethra by foreign bodies, neoplasms, etc.

CHAPTER XLIII.

DISEASES OF THE PROSTATE GLAND, POSTERIOR URETHRA AND SEMINAL VESICLES.

THE intimate anatomical and physiological relations of the prostate gland, vesical neck, posterior urethra and seminal vesicles explain their frequent simultaneous involvement in disease, and the similar symptom-complex that attends their disorders.

We may broadly divide the maladies which affect these organs into those of an inflammatory nature, whose chief local manifestations are increased frequency of urination, painful urination, pyuria, and hæmaturia, and those which occasion a narrowing, compression, or obturation of the urethral canal, and manifest their presence by a difficulty in passing water.

[INFLAMMATORY AFFECTIONS.

☞ The symptoms attending this group are similar to those which are provoked by allied affections of the urinary bladder, but they have points of difference that enable us to tell in which region the disease is located. Thus in these inflammations the pain, while present throughout the whole act of urination, is most severe at its end; and when the patient urinates into three glasses the first one contains much more pus than do the second and third. Should there be bleeding, there will be blood only in the first glass if the hemorrhage is small; with larger amounts all three glasses will be bloody, but especially the first and third. If these data are compared with those which are obtained by examination in cases of vesical disease, no difficulty will be experienced in determining the site of the inflammatory process.

The nature of the diseased process and its further localization to one or more organs of this portion of the genito-urinary tract can be determined from the special character of the symptoms, and by physical, endoscopic and cystoscopic examination. While the last two methods afford us the most reliable and positive data for diagnosis, they cannot be employed in the acute stages of the inflammatory diseases because of the likelihood of increasing the severity of the inflammatory process and because of the exquisite pain which every manipulation with the inflamed parts during this stage provokes.

Urethritis, Prostatitis, Vesiculitis.—A moderate increase in the frequency of urination with slight pain and mild constitutional symptoms, with no tenderness or enlargement of the prostate or seminal vesicles, speaks for acute posterior urethritis. If the prostate is hot, enlarged and tender, and the frequency of and pain on urination are very marked, there is an acute prostatitis; abscess formation in this gland is indicated by the development therein of a soft or fluctuating area, by an increase in the severity of the other symptoms, and by fever. If there is doubt as to the presence of pus an exploratory aspiration should be made through the perineum, the needle being guided into the suspected area by the finger in the rectum. Acute tenderness and enlargement of the seminal vesicles so that they can be palpated as sausage-shaped masses above the prostate point to acute seminal vesiculitis.

The appearance of shreds in the urine, with or without increased frequency of urination, following upon an acute urethritis is suggestive of a chronic posterior urethritis; the diagnosis is confirmed if on endoscopic examination the mucous membrane of this portion of the urethral canal appears dull, rigid, irregularly spotted, with its folds absent or flattened out.

A urethral discharge of clear, milky, or cloudy, slimy, viscid fluid which varies in amount from a few drops to a couple of teaspoonfuls, and which is especially noticed after defecation and when the stools are hard, together with a feeling of soreness of the posterior urethra, and various neurotic symptoms, such as lassitude, depression, etc., are

indicative of a chronic prostatitis. In these cases the prostate feels enlarged with areas of softening and is prominent in some places and depressed in others; between the softened areas the gland feels firmer than is normal. The soft areas disappear after the gland is massaged through the rectum. Upon cystoscopic examination the prostatic outline around the internal urethral orifice is not smooth and rounded, but irregularly elevated and depressed.

Prostatic Tuberculosis.—Firm nodules in the prostate, together with hæmospermia, are very suggestive of *prostatic tuberculosis*. Tubercle bacilli will not be found in the urine or in the prostatic secretion until caseation of the nodules occurs; this is indicated by softening of the centre of the nodules. The tubercle bacilli can be best obtained by expressing the prostatic secretion through rectal massage. The procedure is to be carried out as follows: The urethra and bladder are first thoroughly cleansed by irrigation and the bladder filled with four to six ounces of saline solution. The gland is then expressed and after this the patient empties the contents of the bladder. This fluid is centrifuged or sedimented and the sediment is examined for tubercle bacilli.¹

The presence of enlarged, thickened, nodular masses corresponding to the site of the seminal vesicles, in connection with evidences of tuberculosis in the testicles and prostate, points to the participation of these structures in the tuberculous process.²

¹ Recent investigations (Baumgarten) and clinical experience would go to show that genital tuberculosis is primary in the testicles and secondarily extends to the seminal vesicles and prostate, just as urinary tuberculosis is primary in the kidneys and secondarily extends to the ureter and bladder. Extension to the testicle from primary prostatic tuberculosis is considered to be impossible, though it cannot be denied that a primary nidus may sometimes be located in the prostate and subsequently extend to the bladder. If prostatic tuberculosis is, as a general rule, secondary to testicular tuberculosis, the evidences afforded by the latter (see p. 434) should aid us in the diagnosis of the former.

² The frequency with which tuberculous disease of the testicle is complicated with tuberculous affection of the prostate and seminal vesicles should always prompt us to examine these latter organs before proceeding to radical operation upon the testicle, for it is but rational that if these structures are involved a radical cure is only possible if they are simultaneously removed, a procedure that many surgeons do not look upon with favor, especially if the disease is bilateral.

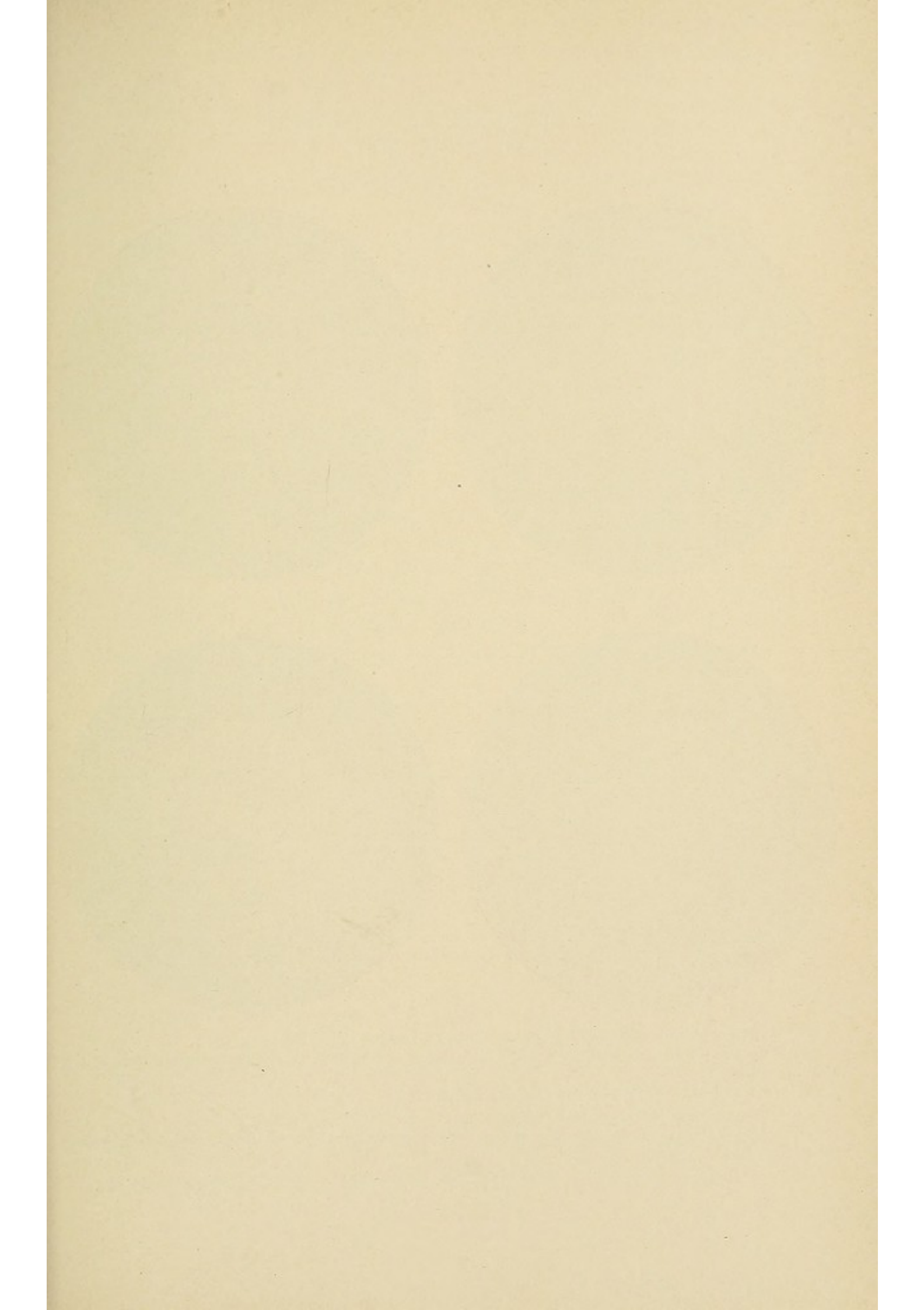


PLATE VII.

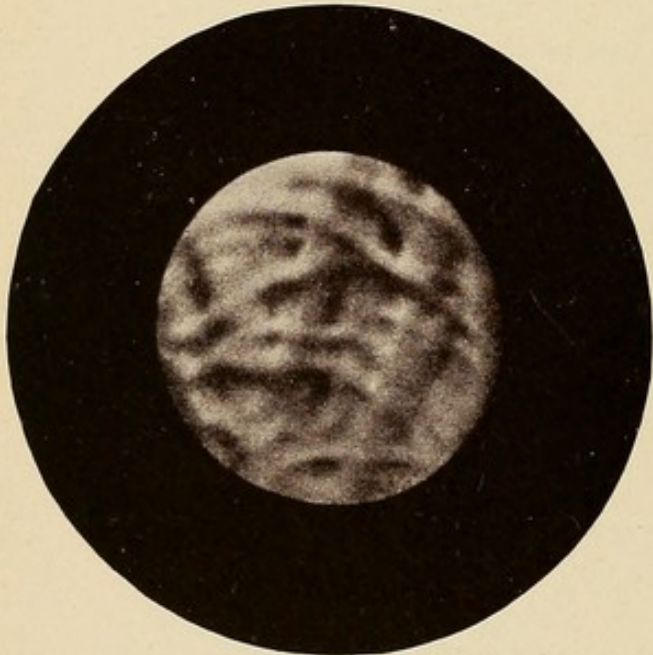
A.



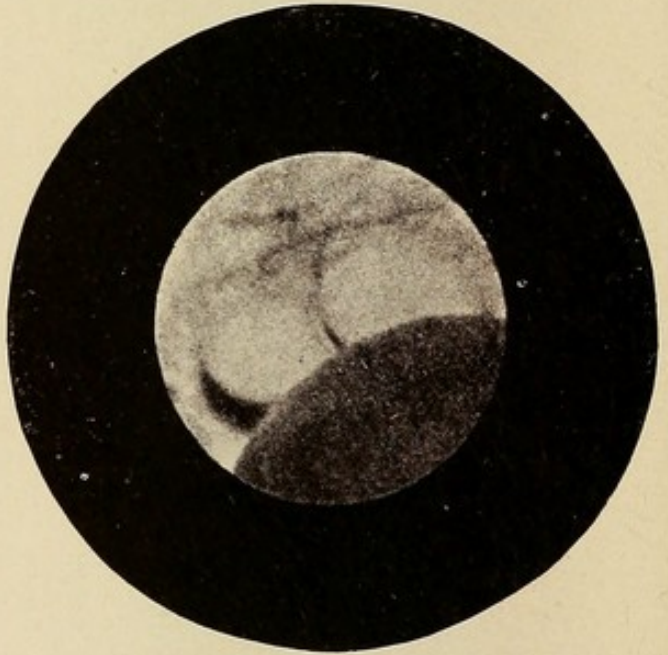
B.



C.



D.



A. Hypertrophied prostate; lateral lobes projecting into the bladder.

B. Hypertrophied prostate projecting into the bladder, with moderate trabeculation of the bladder walls.

C. Marked trabeculation of the bladder walls.

D. Hypertrophied prostate and vesical calculi.

Note in all the prostatic pictures the greater or less alteration of the normal prostatic line seen in Plate III. (From Nitze.)

OBSTRUCTIVE DISEASES.

The diseases causing obstruction of the urethral canal are enlargements and neoplasms of the prostate, strictures of the urethra, calculi and foreign bodies in the urethra, and neoplasms of the urethra. All these affections give as their first and chief clinical manifestation a difficulty in passing water, and differentiation between them is possible only by a careful investigation of the mode of onset and course of development of the urinary disturbance, and by a complete local examination.

Prostatic Hypertrophy and Atrophy.—The symptoms resulting from prostatic hypertrophy or atrophy, for by raising a bar across the neck of the bladder an atrophic prostate may offer the same obstruction to urination as is caused by a hypertrophic prostate that compresses the urethra or blocks its internal vesical orifice like a ball valve, may be grouped into three stages: the initial stage in which there is no urine retained in the bladder, the intermediate stage with partial retention, and the final stage with complete retention.

During the first stages the individual is not much disturbed; he must urinate somewhat more frequently during the night as well as during the day, experiences a burning pain in the urethra, especially at the glans penis, finds that he must wait a little longer than heretofore for the urine to commence to flow, and that the stream has lost its force, the urine dropping down between his legs. The urine is clear, and the general health remains good. These symptoms are not constantly in evidence, nor always equally severe. They vary from week to week, and are invariably made worse by any cause which produces pelvic congestion—*e. g.*, constipation, sexual over-indulgence, and exposure to cold. The symptoms may not increase materially for a number of years, but gradually the bladder becomes unable to entirely overcome the obstruction offered by the prostate and fails to empty itself at each urination. The retention of urine in the bladder increases the frequency of urination and occasions a chronic urosepsis, which gives rise to dyspeptic symptoms. The retention of

urine leads further to dilatation of the bladder, and this, together with the hypertrophy and subsequent degeneration of its muscular coat, which are occasioned by the increasing urethral obstruction, ultimately result in complete retention of urine.

Acute complete retention may set in at any time during the first two stages from acute congestion of the gland; in fact, such acute retention is often the very first sign of the hypertrophied prostate. Repeated attacks of acute complete retention may occur or the acute complete retention may be followed by a chronic partial retention. At any time the symptoms are made considerably worse by an infection of the bladder and kidneys.

The diagnosis of prostatic enlargement or atrophy is made by palpating the gland through the rectum, and by the cystoscope, while the presence of residual urine is determined by the urethral catheter.

As a rule, an enlargement of the prostate can be appreciated through the rectum. Its consistency varies; sometimes it is soft, again firm, and it may contain isolated, firm, elastic nodules. The rectal wall is movable upon the gland. It is desirable during the palpation to try and feel the upper border of the gland, so as to ascertain the upper limits of the enlarged organ. It must not be assumed, however, that every prostate that causes prostatic symptoms feels enlarged through the rectum. Very often the limits and surface configuration as far as can be made out per rectum are not materially changed, and in not a few cases the gland feels smaller and firmer than is normal. In the presence of prostatic symptoms, when the palpating finger is unable to detect sufficient enlargement of the organ to account for them, a further examination is necessary in order to arrive at a diagnosis; for stricture of the urethra and muscular weakness of the bladder from general arteriosclerosis give a clinical picture that much resembles prostatism.

In just these cases does cystoscopy find its chief diagnostic value. As a rule instrumentation is to be avoided in prostatitis, for these patients are very easily infected; but when it is necessary for diagnosis no hesitancy should be had in resorting to it, provided the very strictest aseptic and anti-

septic precautions are observed. The cystoscope shows the enlarged prostate bulging into the bladder, and with a retrograde cystoscopic instrument its encroachments upon the urethral orifice can be distinctly seen. The hypertrophied middle lobe and prostatic bar, when present, are readily made out, and the trabeculated bladder, with its pouches and diverticula and possibly calculi, are clearly seen.

Should partial retention of urine be suspected, its presence can be demonstrated by the urethral catheter. Catheterization belongs in the same category as other instrumentation, and in its employment the strictest aseptic precautions are to be observed. The patient first passes all the urine he can, then the catheter is inserted, and the presence and amount of residual urine determined.

The symptoms above described as resulting from prostatic hypertrophy and atrophy are closely simulated by those which are occasioned by stricture of the urethra, vesical tumors, prostatic neoplasms, and degeneration of the vesical wall from arteriosclerosis.

Stricture of Urethra.—Stricture of the urethra occurs, as a rule, in younger subjects, and is preceded by one or more attacks of gonorrhœa, to which the urinary disturbance can be distinctly traced. The stream with stricture is forked and twisted. Examination of the urethra by sounds demonstrates its stenosis, while with the endoscope the diaphragm or funnel-shaped contraction of the canal is seen. The prostate does not feel enlarged per rectum, and if a cystoscope can be passed into the bladder, no median lobe, no prostatic bar, nor bulging lateral lobes are to be seen. The association of prostatic hypertrophy with stricture is diagnosed from the presence of clinical symptoms and objective findings belonging to both diseases.

Vesical Tumors.—Vesical tumors do not give the same clinical history. With them hemorrhage is often the first symptom, and difficulty in passing urine is experienced only when the growth encroaches on the urethral orifice. Early cachexia, inguinal glandular enlargement, the presence of a hard, infiltrating tumor which can be appreciated per rectum, and, chief of all, the cystoscopic picture will permit a differentiation from prostatic hypertrophy to be made.

Malignant Tumors of Prostate.—Malignant tumors of the prostate are in their early stages very difficult, if not impossible, to differentiate from benign prostatic hypertrophy. The former occur in the young as well as the old, but they are harder, more irregular, more rapidly growing tumors, and are attended with cachexia and enlargement of the inguinal glands. They more frequently give rise to hæmaturia.

Retention of Urine.—Retention of urine without prostatic disease, as occurs with arteriosclerosis and muscular degeneration of the vesical wall, is to be recognized by the absence of a prostatic enlargement, by the absence of an elongated posterior urethra, and by the presence of general arteriosclerosis.

Urethral Calculi.—The difficulty in urination that is due to the impaction of a calculus in the urethra is usually attended with urethral tenesmus, pain, and sometimes with hæmaturia and pyuria. The previous history will, as a rule, suggest the presence of a stone in the kidney or bladder. The diagnosis is readily confirmed in suspected cases by passing a metal searcher into the urethra; if a calculus is present a grating sensation will be experienced. Sometimes a calculus that forms in the prostate protrudes into the urethra and occasions more or less difficulty in urination, pain, pyuria, and hæmaturia. Its presence is recognized from the hard, nodular, circumscribed mass in the prostate, and by *x*-ray examination.

Neoplasms of Urethra.—Neoplasms of the urethra only interfere with the lumen of the canal when they attain some size. *Papillomata* occur in any part of the canal, but most frequently near the external meatus. They occasion a seroturbid urethral discharge that is often attributed to a gonorrhœal infection. A small papillary growth on the external meatus with a seroturbid urethral discharge should suggest the possibility of more such growths being present deeper down in the canal, and should prompt us to make an endoscopic examination. A *malignant tumor* should be thought of if with the evidences of urethral obstruction, such as go with stricture of the urethra, there is a hard, irregular, infiltrating tumor of the urethra, to account for

which there is present neither a gonorrhœal periurethritis nor a stricture of the urethra. As the malignant tumors are rarely primary growths, the original focus, which is most frequently located in the rectum or prostate, should be sought for.

A smooth, elastic tumor in the neighborhood of Cowper's glands with or without urinary disturbance is suggestive of a retention cyst of this organ.

CHAPTER XLIV.

INJURIES OF THE URETHRA AND URINARY EXTRA- SATION: URETHRAL FEVER.

INFILTRATION with urine of the cellular tissues of the thigh, buttocks, hypogastrium, scrotum, and penis has its most frequent cause in injuries of the urethra, whether sustained by accidental trauma or by instrumentation; it likewise follows rupture of the urethra from destructive inflammations thereof and spontaneous rupture of a weakened portion of the canal—*e. g.*, behind a stricture. Not every injury of the urethra is attended with urinary extravasation. The lesser injuries—*e. g.*, a false passage, etc.—are apt to be followed only by hemorrhage into the canal, or by a submucous hæmatoma; the blood in the urethra appears at the external meatus if the site of the injury is in front of the triangular ligament, otherwise it flows back into the bladder and is voided with the urine. A large hæmatoma may occlude the urethra and cause difficulty in urination, even complete retention; it appears immediately after the reception of the injury and forms a soft, doughy, somewhat tender tumor.

Tearing, laceration, or perforation of the urethra leads, as a rule, to urinary extravasation. If the solution of continuity is in front of the triangular ligament, the urine extravasates into the scrotum and penis, down the thighs and upon the abdominal wall; if it is behind the triangular ligament, the urine infiltrates into the cellular tissue of the pelvis and space of Retzius. When the extravasated urine occupies the superficial structures, these rapidly become swollen, red, and gangrenous in patches, and severe constitutional symptoms, high temperatures, rapid pulse rate, and prostration develop. When it is located in the deeper cellular tissues a rapidly spreading, brawny, exquisitely

painful, somewhat fluctuating swelling appears, attended with very severe constitutional disturbances.

Very small tears may be followed by the formation of a small abscess at the site of the injury, which may in turn perforate into the urethra or externally, with or without the formation of a urinary fistula.

The existence of an injury or rupture of the urethra is readily ascertained from a history of accidental trauma or instrumentation, from the presence of a destructive inflammation of the urethral wall, or from an old history of stricture followed by the sudden appearance of extravasated urine. The appearance of blood at the meatus or of a large hæmatoma in the perineum after an injury or after instrumentation points to a lesion of the urethra. If a catheter can be easily passed into the bladder and clear urine is withdrawn therefrom, and if the patient is able to void urine spontaneously, the solution of continuity cannot be very large. If, on the other hand, a catheter cannot be passed into the bladder, and the patient cannot urinate, the defect must be extensive. The examiner must be cautious in interpreting the findings with the catheter. Thus the patient may urinate spontaneously into a pouch in the cellular tissue at the site of the injury, and the catheter passing into this space withdraws clear urine or bloody urine, giving the impression that no severe injury of the urethra has taken place. By measuring the length of the part of the catheter that must be passed until urine commences to flow, it will be found that this does not correspond with the length of the urethral canal, and further, with the finger in the rectum or on the perineum, it will be found that the catheter has not been passed through the entire canal. The rapid development of urinary extravasation at once points to a severe urethral injury.

URETHRAL FEVER.

We have already alluded to the fact that the passage of an instrument into the urethra is a serious and important surgical procedure, on account of the risks it entails of carry-

ing infection to the upper urinary organs. In some individuals, especially those who are susceptible to infections and those whose urine is foul and septic, instrumentation of any kind, even though done with the greatest of aseptic and antiseptic precaution, is apt to be followed by a chill and high rise of temperature. Some of the patients become seriously ill, the high fever continues, and death may follow in twenty-four hours of profound sepsis. In others the fever subsides in a few days, with complete recovery of the patient. Local evidences of infection may be altogether wanting, or the kidneys become enlarged and tender or a severe cystitis develops.

CHAPTER XLV.

DISEASES OF THE EXTERNAL GENITAL ORGANS, TESTICLE, AND CORD.

ABNORMALITIES.

ABNORMALITIES in the position of the meatus urinarius—*e. g.*, epispadias (on the dorsum of the penis), hypospadias (on the ventral surface)—and abnormalities in the tightness of the foreskin, phimosis (excessive tightness), need only to be seen to be recognized. Paraphimosis results when a very tight foreskin is drawn back over the glans and cannot be reduced again. If the glans is constricted by the tight foreskin, strangulation and ultimate necrosis thereof will take place.

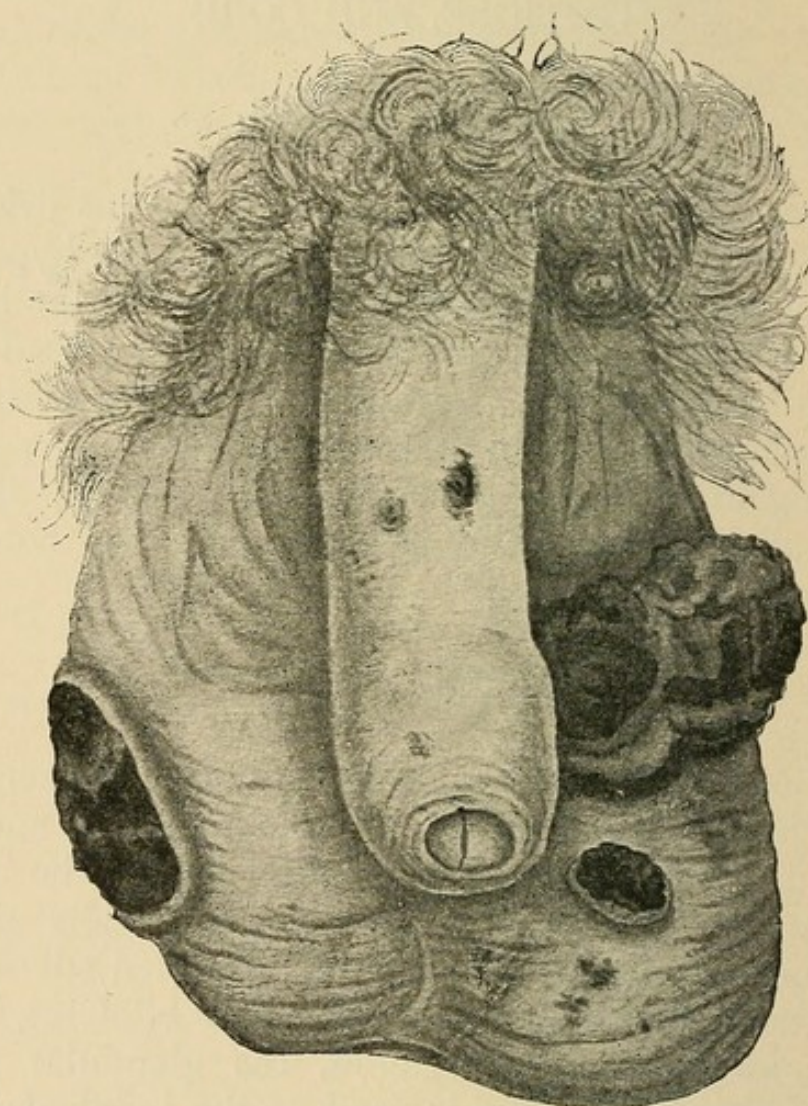
ULCERATIONS.

Ulcerations of the external genitals may be venereal (soft or hard chancres), cancerous (epitheliomatous), and rarely tuberculous. In contrast to the hard chancre the soft sore has a shorter period of incubation, is usually multiple, secretes more profusely, has not the indurated edges, and is usually attended with unilateral, instead of bilateral, inguinal glandular swelling. Furthermore, the glandular involvement is of the dolent type instead of the indolent, as goes with the hard chancre. The soft chancre may be atonic (no tendency to heal), or attended with considerable inflammatory reaction, or phagedenic, or serpiginous, or gangrenous.

Tuberculous Ulcerations.—Tuberculous ulcerations are infrequent, and more common in the female than in the male. They are always secondary to tuberculosis of other parts of the genital tract, and have an irregular, worm-eaten, cheesy base.

Cancerous Ulcerations.—Cancerous growths of this region are most frequently of the epitheliomatous type, rarely of the medullary variety. The former commence as indurated, papillary excrescences that rapidly break down, leaving an everted, indurated ulcer with a crusty, easily bleeding, unhealthy, granulating base. The latter com-

FIG. 149



Chimney-sweep's carcinomata of the penis and scrotum. (Bramann.)

mence as circumscribed, indurated nodules that rapidly fuse together and break down, leaving an everted, indurated ulcer with exuberant bleeding, granulating base. The surface of the foreskin and the surface of the vulva which oppose the seat of the neoplasm are very liable to contact infection.

Acuminate Condylomata.—Acuminate condylomata form fine papillary excrescences without induration.

Herpes Progenitalis.—Herpes progenitalis manifests itself by a group of small vesicles which rapidly burst, leaving superficial, not indurated erosions, which heal readily under a soothing ointment. Their tendency to recurrence adds another characteristic to them.

Benign Nodular Infiltrations.—Benign nodular infiltrations of the penis are not uncommonly encountered and are sometimes mistaken for cancer. Such nodules are either gummata, or gouty nodes, or distended, chronically inflamed follicles with considerable fibrous induration around them, or they are thickened lymphatics arising from an eczema of the penis. *Gummata* occur only in syphilitics and are elastic and firm. *Distended follicles* are preceded by a history of gonorrhœa; they are of slow growth and painless. *Gouty nodes* are attended with other evidences of this disease—*e. g.*, deposits on the fingers and toes, in the tendon-sheaths, and around the larger joints.

These nodes are distinguished from *malignant deposits* by their softer consistency, and by their circumscribed, non-infiltrating character.

Prolapse of the anterior and posterior vaginal walls (cystocele and rectocele), retention cysts of the vaginal walls, retention cysts of Bartholini's gland, and suppurations of the latter are readily recognized when seen.

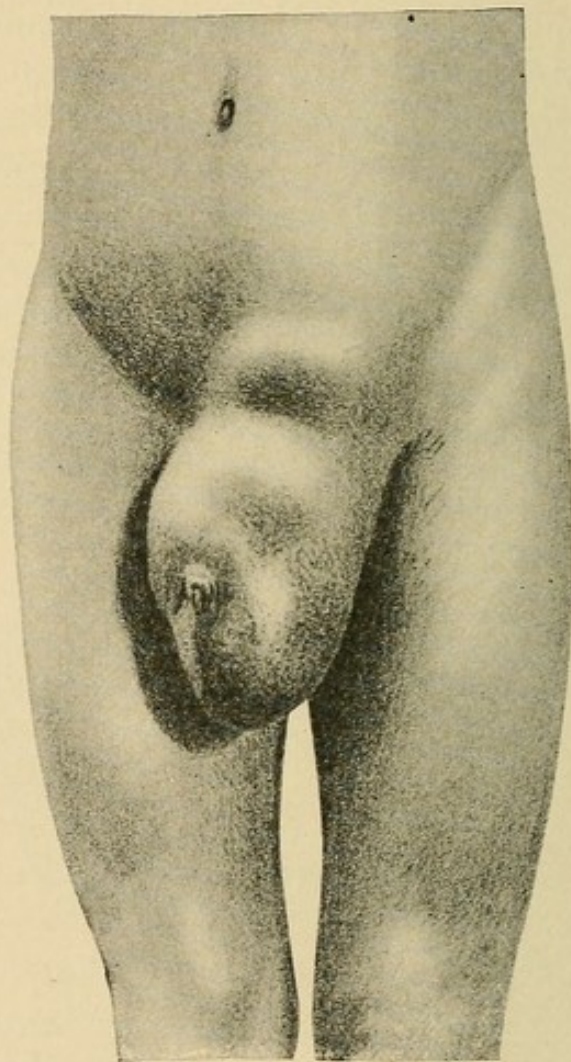
SCROTAL SWELLINGS.

It is of prime importance in the diagnosis of scrotal swellings to determine whether they originate in the structures of the cord or testicle or whether they project into the scrotum from the abdominal cavity. The latter class is characterized by entire reducibility¹ into the abdominal cavity, by an impulse on coughing, and by the fact that they extend into and fill the inguinal canal. If the swelling reduces with a gurgle and is tympanitic to percussion, it is a hernia of the intestine; if it is soft and doughy and dull to percussion and does not reduce with a gurgle, it is a

¹ The swelling may have lost its reducibility, but the other characteristics remain.

hernia of the omentum; if it reduces slowly without a gurgle and is fluctuating and translucent, it is a hydrocele of the open processus vaginalis peritonei; if it is only partially reducible, but has the other characteristics just enumerated, and if upon reduction a fluctuating swelling appears beneath the aponeurosis in the inguinal region which later becomes

FIG. 150



Bilocular hydrocele, partially intra-abdominal. Before operation. (Von Bergmann.)

tense when the scrotal portion of the swelling is compressed, it is a bilocular hydrocele.

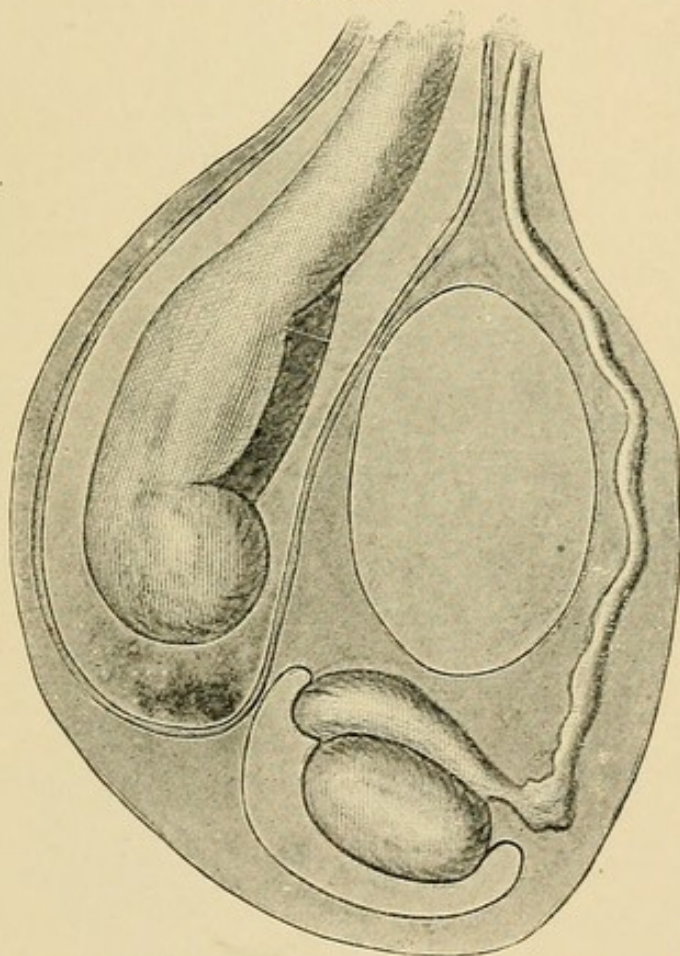
Swellings in the scrotum that have not the characteristics of reducibility and impulse on coughing originate in the cord or testicle.

If the swelling is funicular in origin, a fact that is easily recognized by the ability to palpate a normal testicle below

it, it is either a hydrocele or hæmatocele, or varicocele, or lipoma.

Hydrocele.—A hydrocele of the cord—*i. e.*, a collection of serum in the unobliterated remains of the processus vaginalis—forms a smooth, rounded, or cylindrical, usually tense, fluctuating, and translucent tumor. Translucency may be absent if the sac of the hydrocele is much thickened or calcareous, a condition that is often found in the

FIG. 151



Acquired inguinal hernia in front of the hydrocele of the cord. (Von Bergmann.)

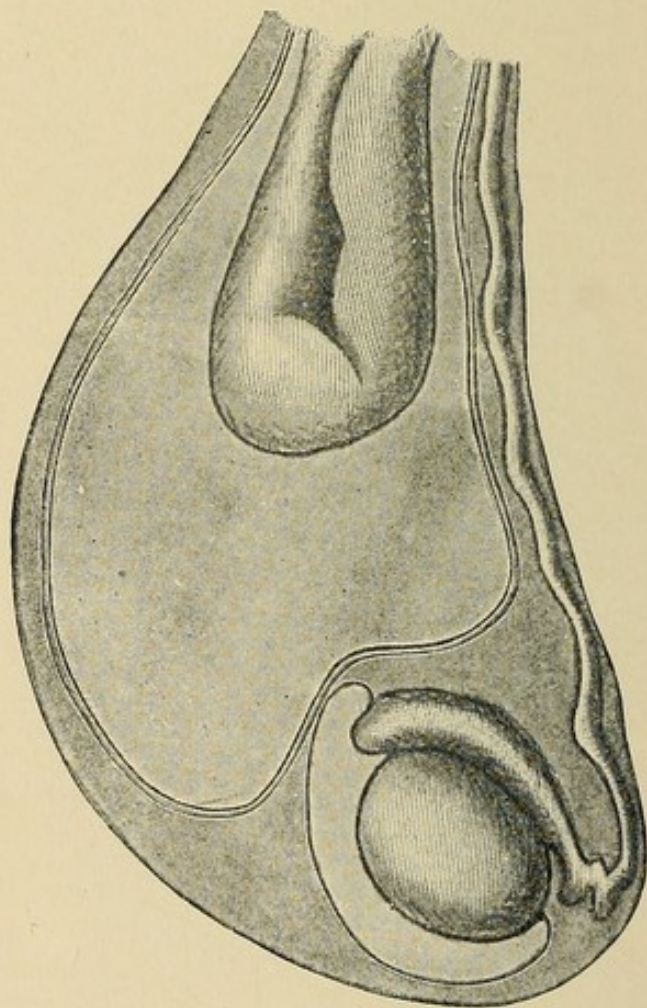
long-standing cases. Sometimes the fluid is encapsulated in several compartments, thus constituting the multilocular hydroceles.

Hæmatocele.—An hæmatocele forms a soft, doughy, semi-fluctuating, tense swelling, and usually develops after a trauma to this organ, or follows upon a puncture of a pre-existing hydrocele sac. The hemorrhagic infiltration of the subcutaneous tissues, the clinical history of the case, and the

absence of translucency without a marked thickening of the sac readily distinguish these swellings from hydroceles.

Lipomata.—Lipomata of the cord form diffuse, soft, semi-fluctuating tumors, which are distinguished from the preceding swellings by the absence of true fluctuation, translucency, and tenseness. In doubtful cases aspiration will at once enable us to determine the character of the swelling.

FIG. 152



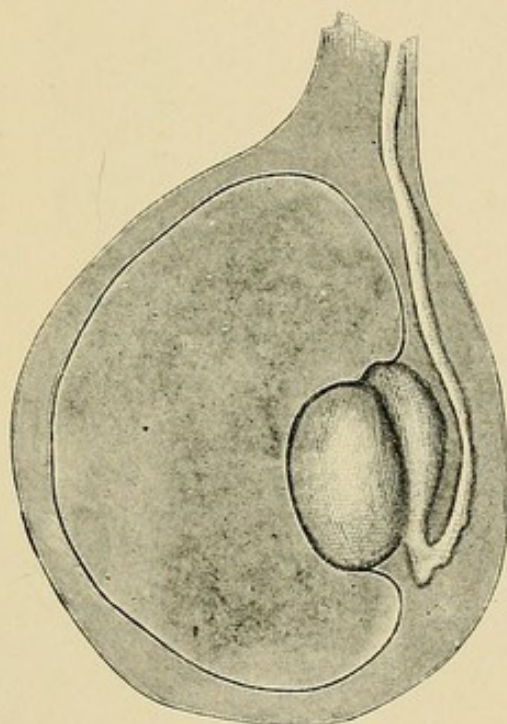
Acquired inguinal hernia with hydrocele of the sac. (Von Bergmann.)

Varicocele.—Dilatation and tortuosity of the veins of the cord (spermatic plexus) constitute a varicocele, which is readily recognized by the compressibility and earth-worm feel of the mass, and by its disappearance when the patient is recumbent. It is most often found on the left side, and is accompanied by dilatation of the superficial scrotal veins, and by atrophy of the corresponding testis. It may be con-

founded with an epiplocele (hernia), but the reducibility of the latter into the abdomen and its impulse on coughing readily distinguish it.

Hydrocele of Tunica Vaginalis Testis.—Hydrocele and hæmatocele of the tunica vaginalis testis have the same characteristics as similar conditions of the cord, from which they are to be distinguished by the fact that they encircle and hide the testicle. Herniæ and hydroceles of the testis

FIG. 153



Hydrocele of the tunica vaginalis. (Von Bergmann.)

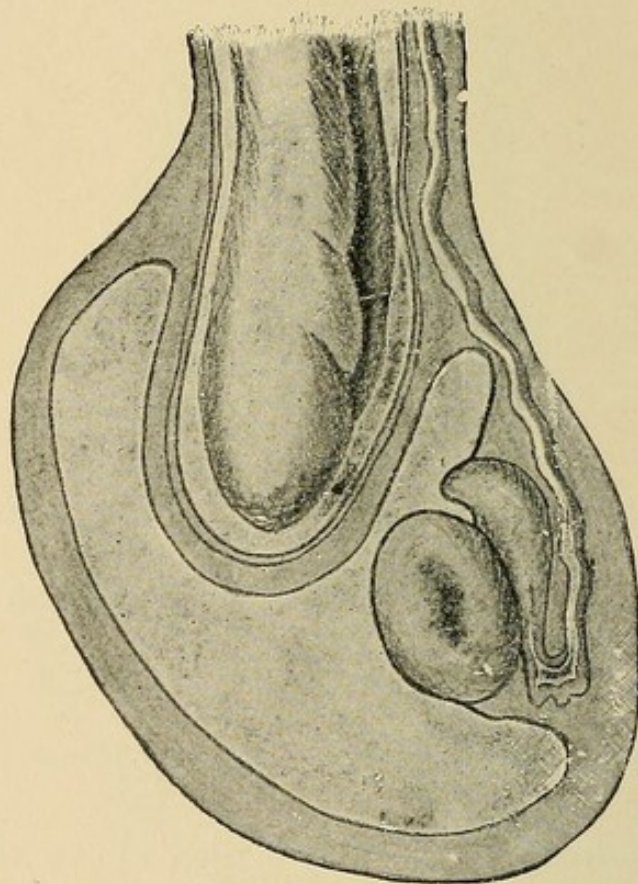
may exist together; if the hernia lies above the hydrocele, there is a reducible, non-translucent tumor above, and an irreducible, translucent, fluctuating tumor below; if the hernia invaginates itself into the hydrocele (encysted hernia), there is a tense, fluctuating, non-reducible swelling which surrounds a reducible, non-fluctuating one.

Spermatocele.—Spermatoceles (cysts of the testicle) possess some of the characteristics of hydroceles of the tunica testis, but they are distinguished from them by the fact that as they usually lie between the testicle and epididymis, they do not cover the front and sides of the testicle as do the hydroceles. Hydrocele of the cord is differentiated from spermatocele by

the fact that in this condition the testicle can be isolated from the tumor, which is not possible in the case of a spermatocele. In doubtful cases exploratory aspiration and examination of the fluid under the microscope for spermatozoa will clear up the diagnosis.

Swellings of Epididymis.—*Swellings of the epididymis* are to be differentiated from those of the testicle proper by their crescentic shape, in the hollow of which lies the globular

FIG. 154

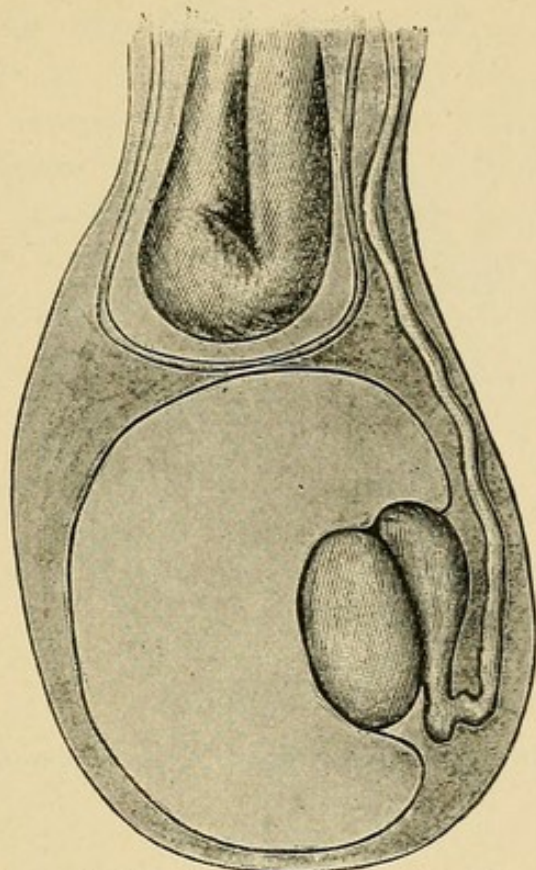


Encysted hernia with invagination of a hydrocele of the tunica vaginalis.
(Von Bergmann.)

testicle. *Tumors of the testicle* are of globular shape; their outlines may be obscured by a hydrocele which accompanies the diseased process, and we should, therefore, always be guarded in expressing an opinion as to the condition of the testicle until the hydrocele fluid has been removed.

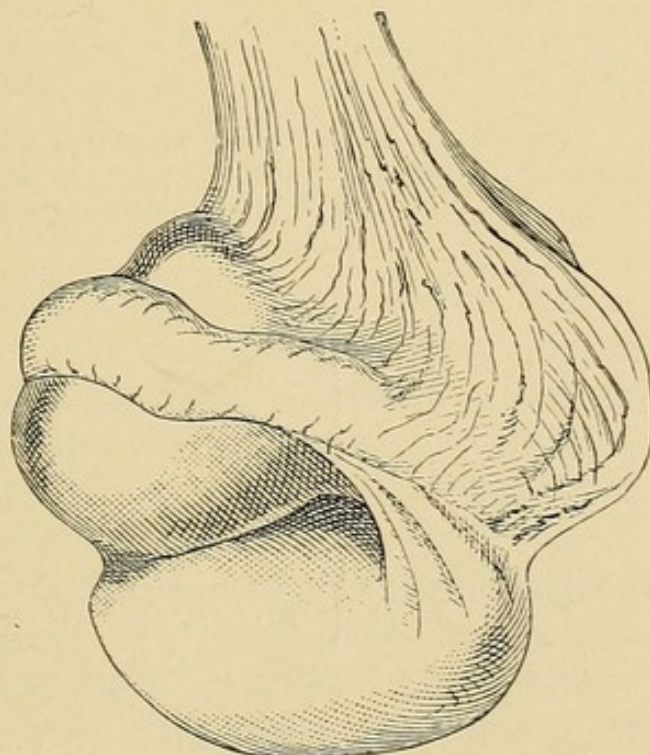
Rapid enlargement, exquisite pain, and tenderness, with temperature elevations, are evidences of acute inflammatory conditions of the testicle or epididymis, the cause for which

FIG. 155



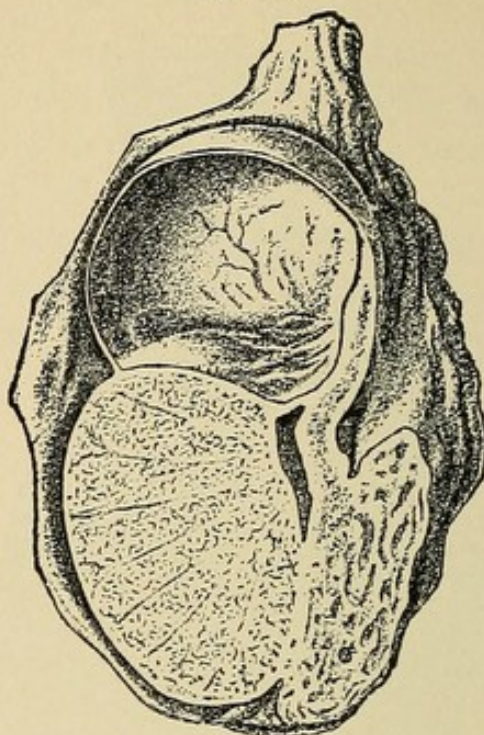
Hydrocele of the tunica vaginalis with acquired inguinal hernia. (Von Bergmann.)

FIG. 156



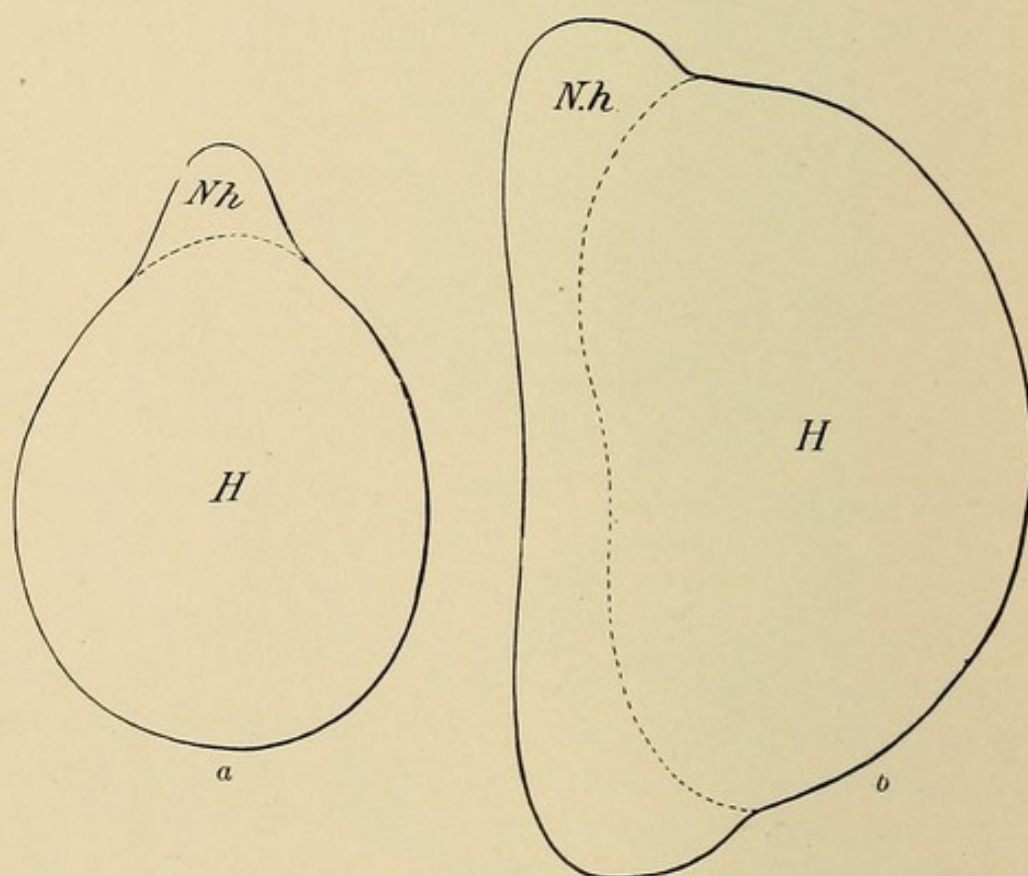
Spermatocele between the testis and epididymis, giving the testis a horizontal position. (Kocher.)

FIG. 157



Intravaginal spermatocoele. (Von Bergmann.)

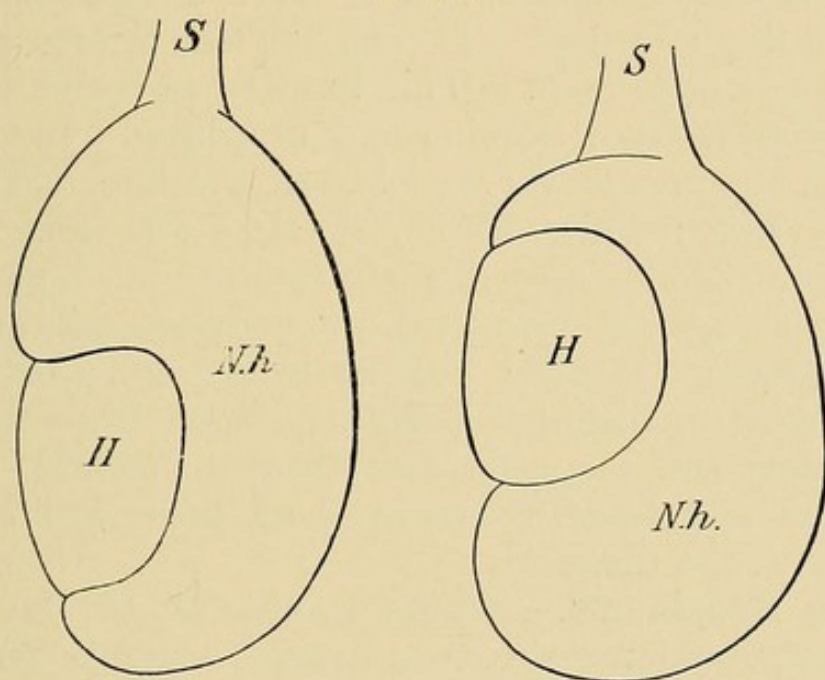
FIG. 158



Showing the relations of the testis and epididymis in acute orchitis ; *H*, testis ; *Nh*, epididymis ; *a*, sagittal section ; *b*, horizontal section. (Kocher.)

must be sought in a posterior urethritis or in an acute infectious disease, especially likely being mumps, typhoid fever, or some eruptive fever. The enlargement of the testicle which sometimes remains after an acute inflammation bears a strong resemblance to chronic orchitis from syphilis, and in the absence of a syphilitic history the diagnosis is always uncertain. If the swelling is very hard, has a smooth and regular outline, is limited to the body of the testicle, is ac-

FIG. 159



Illustrating the relations of the epididymis and testis in acute epididymitis. In the first drawing the head of the epididymis is chiefly affected, and in the second drawing, the tail; *H*, testis; *Nh*, epididymis; *S*, spermatic cord. (Von Bergmann.)

accompanied by a hydrocele, and does not impart the testicular sensation when it is compressed, it is probably syphilitic.

A chronic orchitis is to be differentiated from a chronic hydrocele with much thickened and calcareous sac by the fact that with the latter condition the tumor is in some places of very hard, almost bony consistency.

Syphilitic Gummata.—Prior to softening and perforation, *syphilitic gummata* of the testicle cannot be distinguished from a diffuse syphilitic orchitis. After perforation they may be confused with tuberculosis or neoplasm of the testicle, but the regular margins of the fistulous opening, the profuse serous discharge therefrom, and the absence of secondary

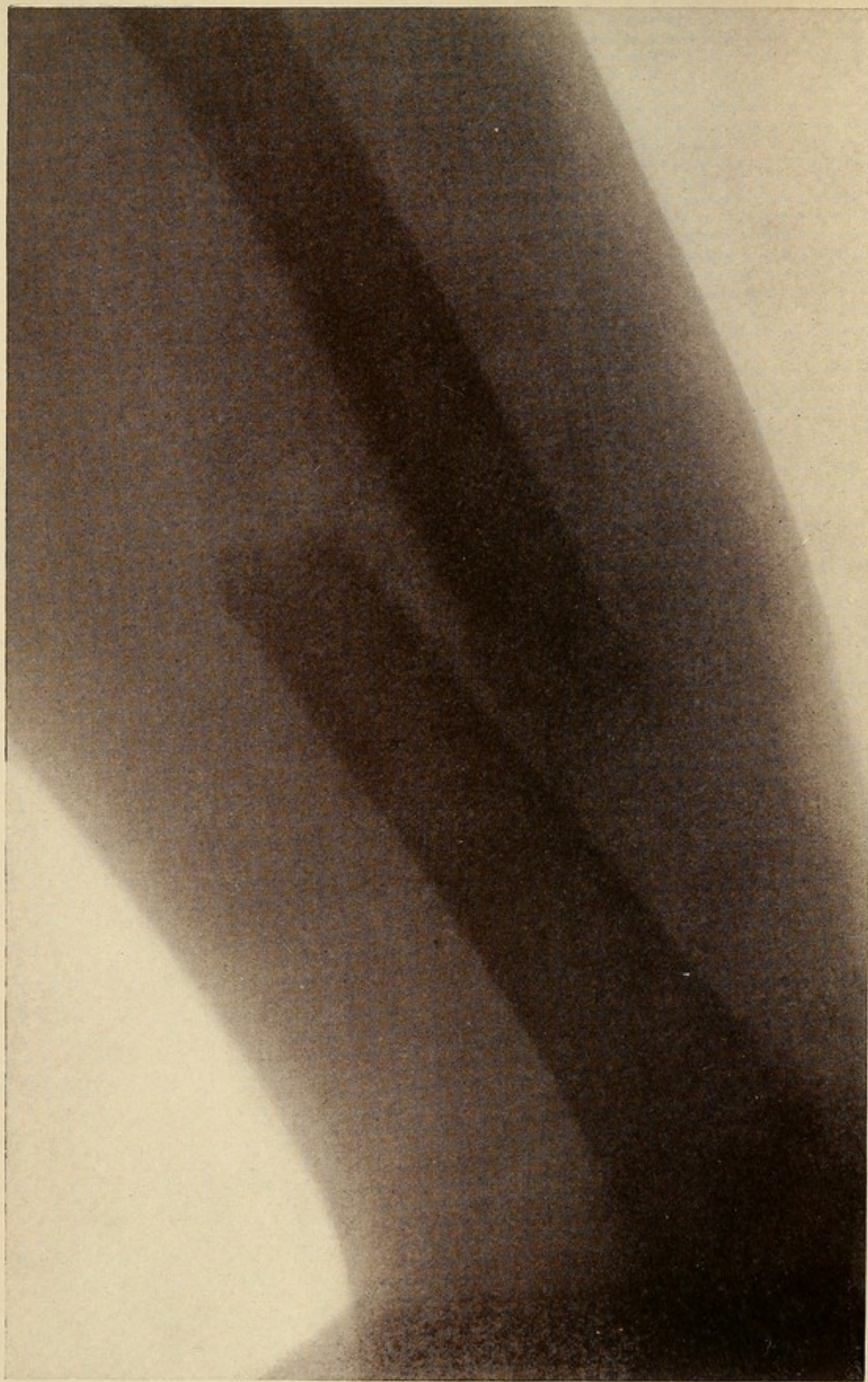
foci in the cord, prostate, and seminal vesicles, together with a syphilitic history and other evidences of this disease, serve to differentiate them. Gummatous nodules in the epididymis may be confused with the nodules in this organ that result from a preceding gonorrhœal infection, but those due to syphilis are usually located in the globus major, while those due to gonorrhœa are, as a rule, in the globus minor.

Tuberculous Disease.—Tuberculous disease of the testicle occurs most frequently in young subjects who have other foci of tuberculous disease or who have a family predisposition for this affection. The epididymis is first attacked and becomes nodulated. This location in the early stages distinguishes it from syphilis and neoplasm. Later on it spreads to and invades the testicle, vas, prostate, and seminal vesicles. The nodules soften, break down, and become adherent to the skin and ulcerate through it. The cheesy, worm-eaten edges of the ulcers, the seropurulent discharge, and the presence of other nodules in the vas, prostate, and vesicles serve to distinguish this condition from the other inflammatory and neoplastic diseases of the testicle. If both epididymes and testicles are involved there is no further doubt about the diagnosis.

Benign Neoplasms.—Benign neoplasms of the testicle grow slowly, and remain encapsulated, whereas malignant growths increase in size rapidly, and extend at an early period to the epididymis and lumbar glands. All neoplasms of the testicle impart a feeling of great weight to the organ, which at once distinguishes them from the inflammatory conditions.

Sarcomata are most frequent in young individuals; they grow rapidly and are quite soft in consistency. The *carcinomata* are hard, grow less rapidly, and are most common in advanced life. Prior to perforation a sarcomatous tumor may resemble a gumma or syphilitic orchitis, but mixed treatment has no marked influence upon it, the patient does not give a distinct syphilitic history and does not show other evidences of the specific disease. In all doubtful cases exploratory incision is to be resorted to as early as possible. After perforation the fungous character of the ulcer, the rapid enlargement of the tumor, and the presence of secondary deposits readily enable one to make the diagnosis.





Fracture of the Femur, showing considerable Vertical and Lateral Displacement of the Fragments, without change in the External Contour of the Limb.

PART VI.

INJURIES AND DISEASES OF THE EXTREMITIES.

CHAPTER XLVI.

INJURIES OF THE BONES.

THE *x*-ray¹ has simplified the recognition of fractures, and made the determination of their direction, their number, and the displacement of the fragments very much more certain. Even in the apparently simple cases this method of examination should be employed wherever possible, if only to confirm the other clinical findings.

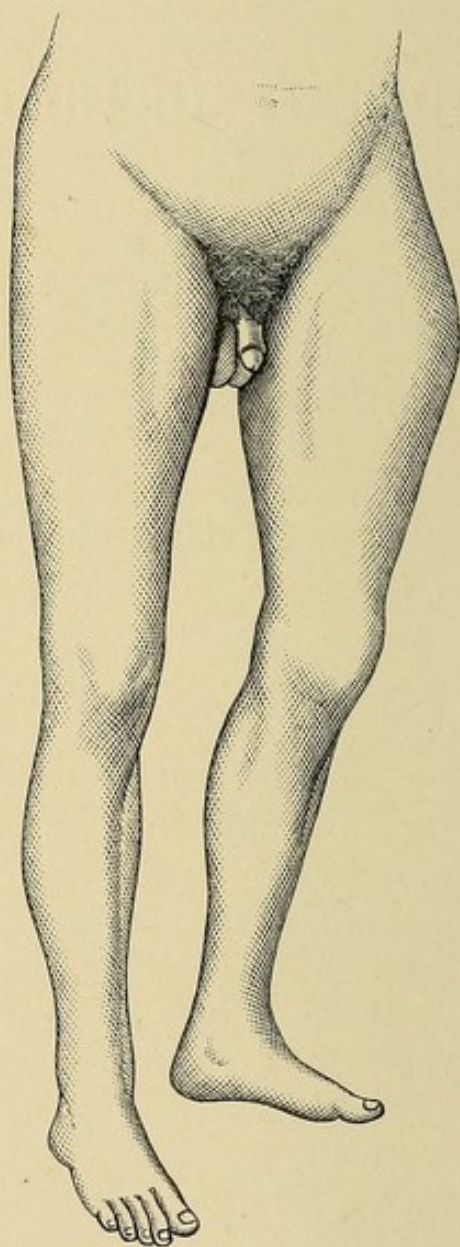
In addition to this positive and accurate method for the diagnosis of fractures and other injuries of the bones there are other clinical signs by which the presence of a fracture may be determined, though the relation of the fragments to each other and the direction of their displacement cannot be as well elicited by them as by the *x*-ray examination.

These clinical evidences are swelling and ecchymosis in the region where the injury was inflicted, and extending from here up and down the limb, change in the axis of the limb (either an angular deviation of its own axis, or displacement of its entire axis inward or outward in reference to neighboring fixed points), local tenderness over the fracture, crepitus on rubbing the apposed surfaces together, shortening of the limb, false point of motion in the limb, and usually a loss of active motility.

¹ The use of the *x*-rays in the treatment of fractures, to determine whether the fragments are in good apposition, need only to be mentioned to at once suggest its great value to those who are not accustomed to systematically employ the rays for this purpose.

Method of Examination for Fracture.—A patient who has sustained an injury to his limbs should be examined in the following manner: He should be undressed, the clothing if necessary being cut away, so as to expose the *injured* and

FIG. 160



Angular deviation of the axis of the left thigh due to fracture of the femur.
(Hoffa.)

the *uninjured* extremity. His attitude, his position; the manner in which he carries the injured part; the presence of external wounds, of projecting bones, of ecchymoses; the axis of the limb (its own integrity and its direction to neighboring fixed points), and the possibility of active motion

should all be carefully noted. In other words, all the information that can be obtained by inspection should be elicited before the injured limb is touched. The tip of a single finger should then be passed along the entire length of the bone, the patient being asked to state when he feels an acute, sharp pain (as the location of this corresponds to the seat of fracture, the examiner knows at once where to look for crepitus and false point of motion). The length of the limb should then be measured and compared with that of the opposite side, and the relation of fixed points on it to fixed points on neighboring parts should also be ascertained. Open wounds should not be probed, but after being thoroughly cleansed they should be covered by an aseptic dressing. In every detail of the examination comparison of the injured with the healthy side should be made.

With such a routine method of examination it is possible to determine the probable presence of a fracture by inspection alone. It is never right, however, to rely upon this means alone for making the diagnosis. The data it affords should always be reinforced by those that are to be obtained by palpation, measurement, and the *x*-ray examination.

SPECIAL FEATURES OF FRACTURES OF THE INDIVIDUAL BONES.

Only the fractures of the shafts of the long bones will be here considered; those of the articular ends of the bones will be included under injuries of the joints.

Fractures of the Clavicle.—The attitude of a patient who has sustained a fracture of the collar bone is characteristic. His head is inclined to the injured side, the affected shoulder is depressed, and the corresponding elbow and forearm are supported by the well hand. The space between the neck and the outer aspect of the shoulder is shortened.

Fractures of the Humerus.—It is most important in these fractures to carefully examine the sensation and power of the arm, forearm, and hand, in order to ascertain whether the fracture has involved the musculospiral nerve. The characteristic wrist drop follows paralysis of this nerve.

Many a malpractice suit will be avoided and many a patient will be prevented from being dissatisfied with the treatment he has received if he is told at the outset that the injury he has sustained has involved this nerve and may be followed by paralysis of the muscles supplied by it. Measurements of the humerus should be made from the tip of the acromion process to the external epicondyle of the humerus.

Fractures of the Radius and Ulna.—The deformity may be slight, especially in fat subjects, with little displacement of the fragments. With green-stick fractures, which occur quite frequently in these bones between the ages of two and fourteen years, crepitus is absent unless one bone is completely fractured; the deformity, however, is very evident.

Fractures of the *coronoid process* of the ulna are usually associated with backward dislocation of the ulna, and should be suspected if the dislocation recurs readily after reduction.

The fractured process should be sought about one finger's breadth above the bend of the elbow; its palpation may be difficult, but its discovery with the *x*-ray will be easy.

Fractures of the *olecranon process* result in an inability to forcibly extend the arm; a depression marks the separation between the fractured ends.

Fractures of the Femur.—Shortening is determined by measuring from the anterior superior iliac spine to the internal malleolus of the same side, the patient lying flat upon his back and both iliac spines being on the same straight plane.

Fractures of the Patella.—These are attended with partial or complete loss of power of extension of the leg, the degree of interference with this movement depending upon the extent of the associated tear of the quadriceps muscle. The patient is often unable to raise the heel from the bed when lying upon the back. The knee-joint is distended with blood.

Fractures of the Tibia and Fibula.—Shortening should be determined by measuring from the internal condyle of the femur to the tip of the internal malleolus. Fracture of both bones is attended by eversion of the foot.

CHAPTER XLVII.

INFLAMMATIONS AND NEOPLASMS OF BONES.

ACUTE OSTEOMYELITIS.

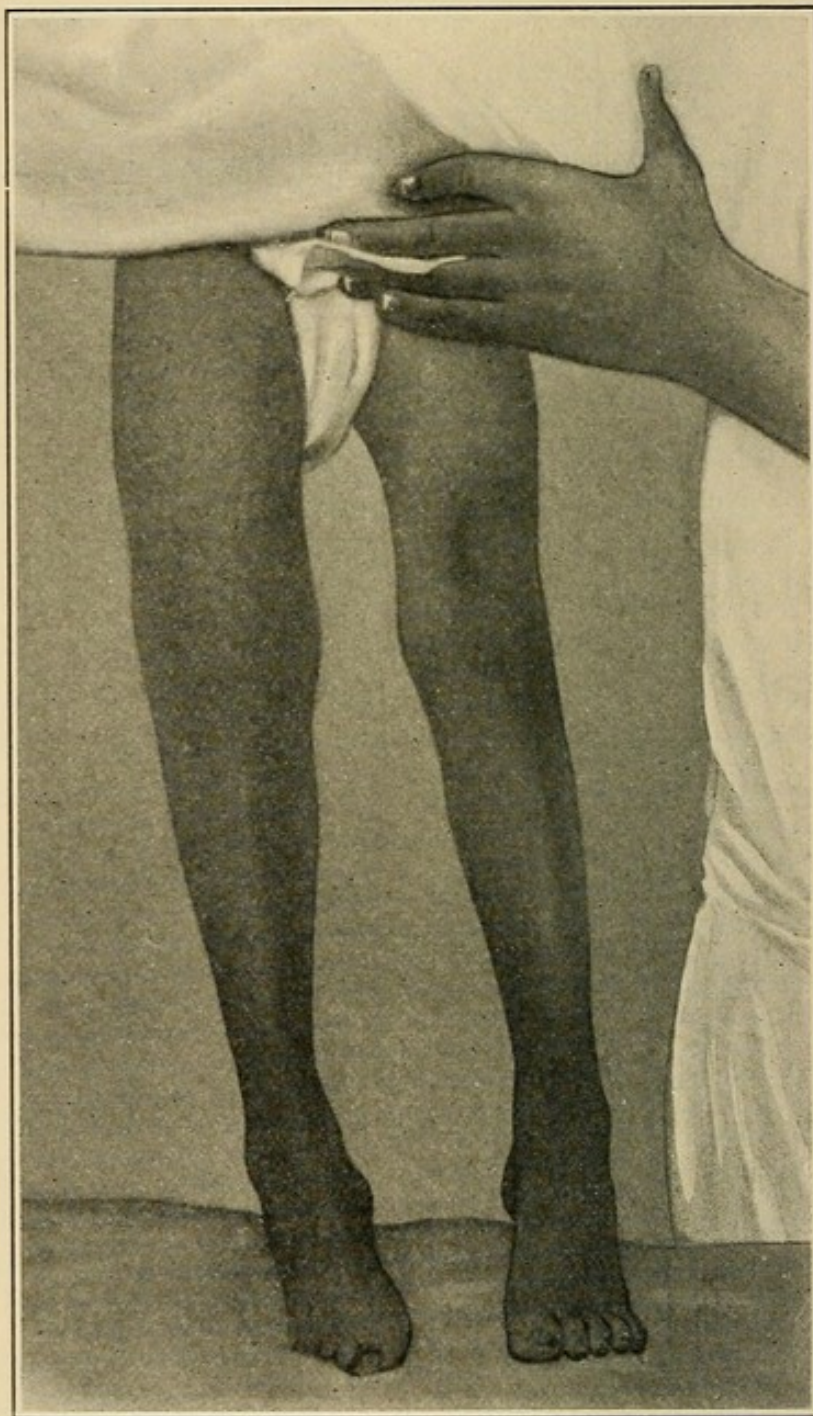
WHILE the acute infective bacterial osteomyelitis and periostitis are among the most serious and often rapidly fatal diseases which the surgeon is called upon to treat, the acute non-infectious, non-bacterial periostitis and osteomyelitis never threaten life or limb, and never provoke anything but the mildest constitutional manifestations. The former are rapidly destructive and eminently septic inflammations; they follow an infection of the medullary canal and periosteum by the pyogenic bacteria, most commonly the staphylococcus aureus, which are carried to the bone by the blood from other foci of suppuration (most frequently from furuncles, from the intestines, air tubes, etc.), or are directly introduced through open wounds. The latter is a reparative process and is seen after fractures, aseptic bone injuries, etc.

In no other surgical affection is it so essential to make an early diagnosis and rapidly institute the proper treatment as in the acute bacterial forms of osteomyelitis. In many instances the integrity of the limb and the very life of the patient depend upon an immediate opening and drainage of the infected medullary canal, and only too often does it occur that extensive bone necrosis and severe, even fatal, septicæmia ensue, even though a diagnosis has been promptly made and the proper procedures rapidly carried out.

The disease occurs at any period of life, but is especially frequent during childhood and adolescence. In the *foudroyant cases* the patients are from the outset intensely ill. The constitutional symptoms are those of a severe intoxication—viz., extreme prostration, delirium, high fever, 105°

to 106°; rapid pulse, 120 to 150, and dry tongue; the local symptoms are very slight or are not at all in evidence. After

FIG. 161



Acute osteomyelitis of the femur, due to the streptococcus pyogenes, with sero-purulent gonitis. Note the swelling of the affected limb and the periarthrititis at the knee.

twenty-four or forty-eight hours these patients die or the constitutional symptoms abate somewhat, owing to spontaneous perforation of the bone and periosteum; the local

manifestations then become more evident and palpable. In this class of cases the septicæmia commences with the osteomyelitis. The clinical phenomena are due to the blood poisoning, which overshadows, both in its importance and in its manifestations, the local bone disease which has been its cause. In some of the patients the local evidences afforded by the diseased bone—viz., pain, swelling, and a limited use of the affected limb—may not have had time to develop before death occurred.

In the *moderately severe cases* (which constitute the largest number) the onset of the disease is attended by a chill, to which follow a continuously high fever, rapid pulse rate, coated, dry tongue, drowsiness, mild delirium and constantly high leukocytosis (20,000 or over). Over the infected bone the soft parts become considerably swollen, œdematous, and exquisitely painful and tender; active motion is suspended and passive motion is attended with excruciating pain. If the infection is at the epiphyseal end of the bone the neighboring joint becomes coincidently involved; at first the periarticular structures become œdematous, then a serous or sero-purulent exudate forms within the capsule of the joint, and if infection of the joint cavity occurs, a purulent arthritis develops. If no relief is afforded by surgical means, the patient, usually, succumbs to blood poisoning; in some instances the life of the patient is saved by spontaneous perforation of the bone and periosteum. The perforation is followed by the formation of an abscess in the soft parts which occasions increased swelling and redness. An abscess, however, is not always to be construed as an evidence of a perforation of the bone; in many instances it is due to the breaking down of an infected vascular thrombus, or to suppuration of the lymphatic vessels or glands. With perforation of the bone the constitutional symptoms regularly subside in intensity because the tension of the inflammatory products within the medullary canal is thereby relieved.

The cases in which the local symptoms are present in conjunction with high fever, rapid pulse, drowsiness, and coated, dry tongue are easily recognized; but in the foudroyant cases, and in the very early stages of the less severe cases

before the local signs have fully developed, the diagnosis is not so easy, and yet it is in these septic cases that it is most necessary to make an early diagnosis and promptly institute the proper methods of treatment. A good working rule is the following: Every child and young adult in whom there is a sudden onset of high fever, rapid pulse, etc., for which no adequate cause can be found, should be suspected of being afflicted with an acute infectious osteomyelitis, and for the presence of this malady every bone, and especially its epiphyseal ends, should be carefully examined. A child may be too sick to complain of local pain, and in the absence of swelling, redness, etc., the existence of a bone disease may not be suspected. It is the author's experience that even delirious and comatose patients manifest by a shriek or a cry or by restlessness an exquisite tenderness over an infected bone, and that this tenderness is present before swelling and œdema have become evident. This tenderness, taken in conjunction with the constitutional symptoms for which on careful examination no other adequate cause can be found, forms a most valuable early indication of the presence of an acute septic osteomyelitis; in the later stages of the malady, when the entire affected limb has become much swollen, the location of the acute pain enables the examiner to accurately determine the site of the disease.

The other constitutional diseases, which in their onset resemble acute infectious osteomyelitis, are typhoid fever, cerebrospinal meningitis, and miliary tuberculosis.

In *typhoid fever* the leukocyte blood count is low, the pulse is slow; there may be a Widal reaction. The Ehrlich diazo-reaction in the urine may be present in both affections.

In *cerebrospinal meningitis* the neck is very apt to be rigid, Kernig's sign is present, the pulse is slow, the leukocyte count is not apt to be as high, and lumbar puncture yields a turbid fluid containing bacteria, usually the diplococcus or streptococcus.

In *miliary tuberculosis* careful search will reveal in the glands or in the lungs or bones a primary focus of the disease. Furthermore, the leukocyte count is not apt to be so high, the temperature curve is irregular, the respirations are frequent, and tubercles may be found on the choroid.

Subacute types of epiphysitis occur, in which the distention of the neighboring joint by a viscid catarrhal fluid forms the chief symptom, the constitutional manifestations being very mild. Such conditions of the joints have been styled "Volkmann's catarrhal synovitis." They, as well as the more acute forms of joint manifestations that attend acute infectious epiphysitis and osteomyelitis, must be differentiated from the rheumatic forms of synovitis. The latter are not as frequently met with in children; their onset is not so severe, nor their course so rapid; they are frequently preceded by tonsillitis, and are more apt to be polyarticular. Furthermore, the arthritides complicating epiphysitis and osteomyelitis are always associated with periarthrititis, whereas with the rheumatic joints there is no periarthrititis at all, and there is no local tenderness over the bone.

It is doubtful whether the metastatic joint lesions following the acute exanthemata are not due to a primary metastatic osteomyelitis, the joint symptoms being the most striking manifestation. A primary pyarthrosis occurring in these diseases is to be distinguished from the joint lesions, which are secondary to bone involvement, by the absence of bone tenderness, and by the previous history of an eruptive disease.

The joint lesions due to gonorrhœa are to be distinguished from those which are secondary to acute osteomyelitis by their greater frequency in young adult males, by the presence of a urethral discharge or shreds in the urine, by the considerable periarthrititis that attends them, by the absence of tenderness over the epiphyses, and by the less severity and less rapid development of the constitutional symptoms.

It is to be especially noted that a joint effusion, together with periarthrititis, may *precede* the development of the other local evidences of acute osteomyelitis.

Some forms of *acute tuberculous arthritis* in infancy resemble acute osteomyelitis with joint complications. The joint is hot, swollen, and sensitive, the onset is sudden, and the constitutional symptoms severe. Such cases are usually observed in children whose mothers suffer from advanced disease of the lung.

CHRONIC INFLAMMATIONS OF BONE.

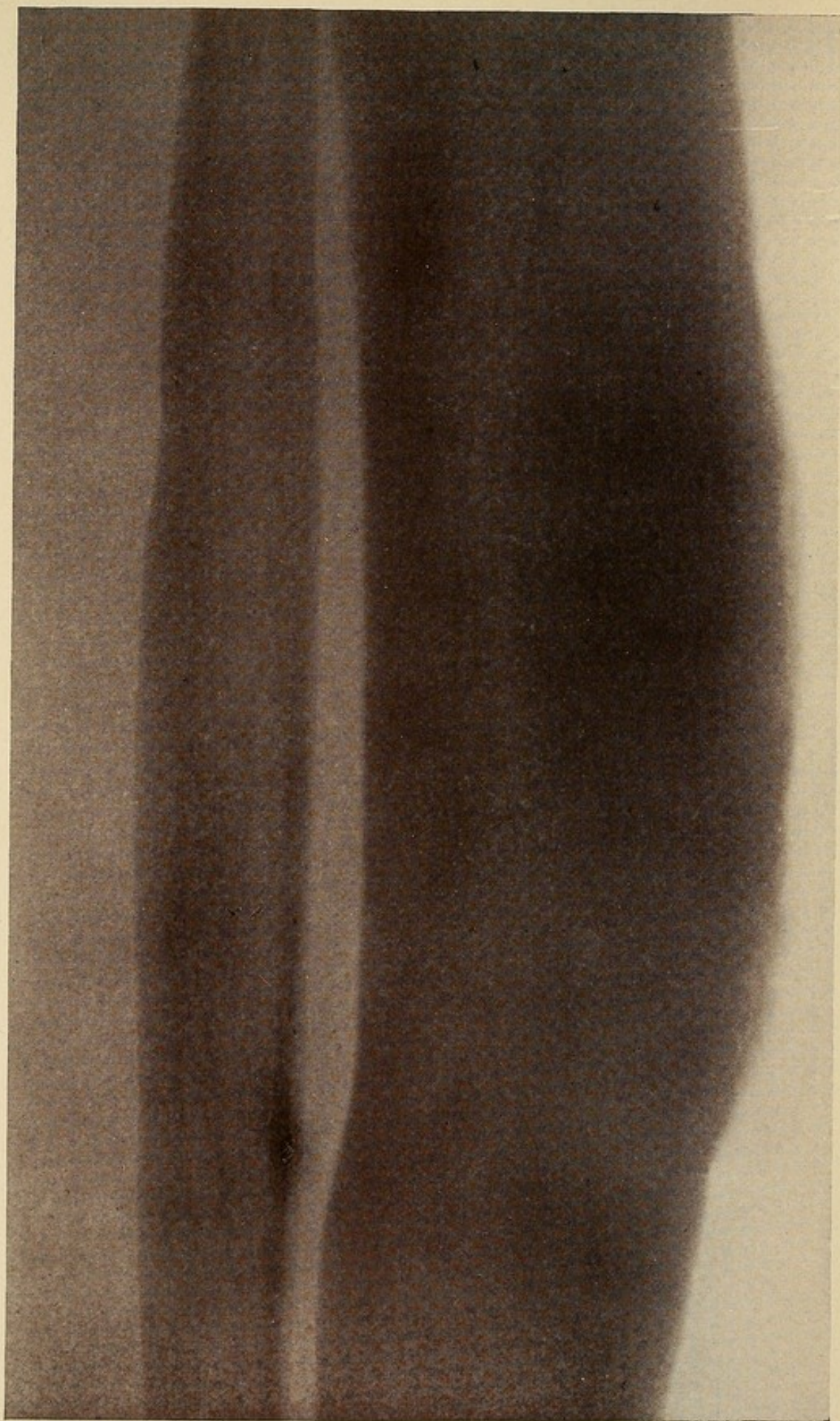
Severe, rapidly progressing, and destructive as are the acute infective forms of osteomyelitis, so chronic, slow, and function-disturbing are the chronic types of this malady. They commence insidiously or acutely and then subside into a chronic stage, progress slowly and steadily, or lie dormant for months and years, and flare up at times in acute exacerbations. They cause a gradual impairment in the general health, and when attended with prolonged suppuration they give rise to waxy degeneration of the internal viscera. They likewise result in a gradually increasing impairment of the functional activity of the diseased limb.

The most common varieties of chronic osteomyelitis are the pyogenic (the invading organisms being of diminished virulence), the typhoidal,¹ the tuberculous, the syphilitic, and the actinomycotic.

The local evidences of chronic bone disease are, as a rule, well marked and readily recognized. The patients complain of pain and tenderness in the diseased bone; the pain is deep seated, intense, boring or gnawing in character, often intermittent, and generally most severe after exposure to cold, after active exercise or when the patient retires to bed and the limb becomes warm; the tenderness is circumscribed and corresponds to the site of the disease. The physical evidences may be very slight in the early stages of the disease, but they become more marked with its development. If the malady is confined to the *medullary cavity* the bone feels uniformly thickened and enlarged; if the periosteum is also involved the bone is irregularly *thickened* and its surface is uneven and of varying consistency, harder in some places, softer in others. In some instances an exudate forms between the periosteum and the bone. The soft parts overlying the diseased bone may be normal or thickened, or adherent to the bone, or infiltrated, or the seat of abscesses. The pus in the abscesses may burrow by gravity along the planes of least resistance to distant points, and

¹ When the Eberth bacillus produces suppuration it probably has changed its specific character and become a pyogenic organism.





Chronic Osteoperiostitis of the Tibia.

Note the thickening of the periosteum, the deeper shadow of the cortical tissue of the tibia, and the irregular, shaggy projections from the periosteum of the tibia. Compare with the picture of the healthy fibula alongside.

perforate on to the skin with the formation of sinuses and fistulæ. Such sinuses always lead down to necrotic or carious bone. The appearance of the fistulæ and the character of their discharge depend on the nature of the disease in the bone. (See below.)

The functional disturbances which are occasioned by these forms of osteomyelitis depend chiefly on the location of the diseased process. Where the inflammation is limited to the shaft of the bone the interference with function is usually very slight; at times, however, the boring pain and tenderness restrict the active use of the limb, though passive motion remains normal; those movements are especially restricted which are performed by muscles whose place of insertion on the bone corresponds to the site of the disease. When the articular end of the bone is involved, as is most frequently the case in the tuberculous forms of the disease, active and passive movements of the corresponding joints are restricted. It is to be noted that such restriction may not be in evidence in all of the movements which the joint normally enjoys. (See Tuberculous Joint Disease.)

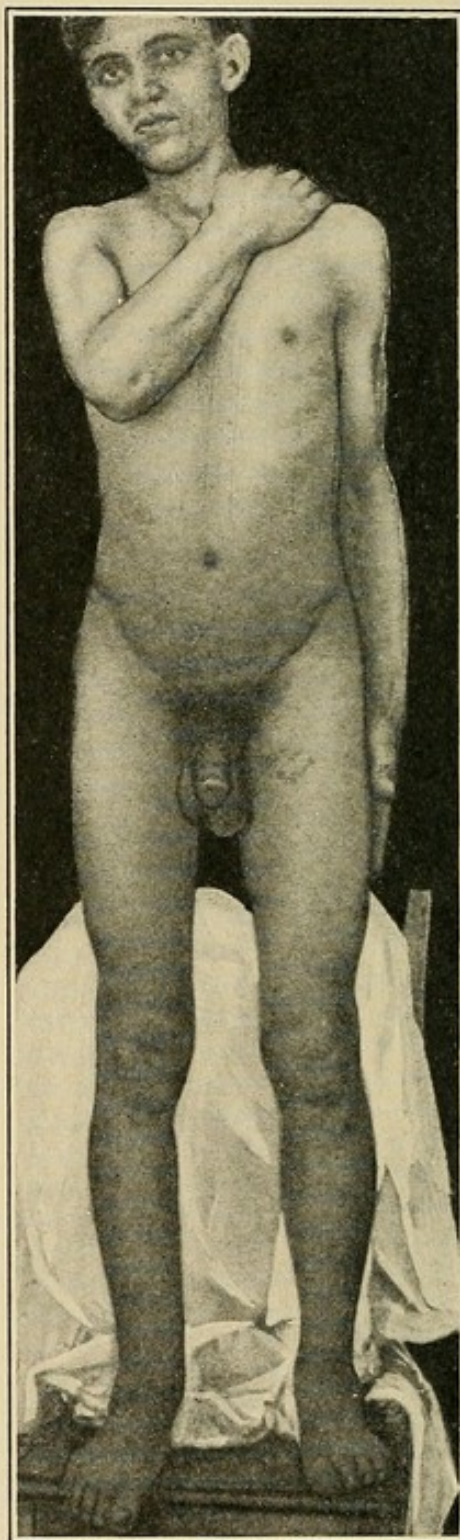
After long continuance of the disease, especially if it is attended with suppuration, the general health suffers; the individual becomes pale, anæmic, and thin, may sweat at night, has occasional elevations of temperature, loses his appetite, and in the final stages waxy degeneration of the internal viscera, of the kidneys, liver, spleen, etc., develops.¹

The *x*-ray affords valuable clinical data for the diagnosis and differentiation of chronic bone disease, and as greater experience is obtained in the interpretation of the pictures it affords more aid from its use can be expected. In the shadowgraphs note should be taken of changes in the outline and thickness of the bone and its periosteum, of the structure of the cancellous tissue, and of the amount of rays which are absorbed by the tissues. No radiograph is good unless it shows the details of the bones and of the soft parts. When the periosteum is diseased it appears thickened, its under surface being irregular, with shaggy projections here

¹ It is always most important to ascertain whether the internal viscera are healthy before a prognosis is made in cases of chronic bone disease.

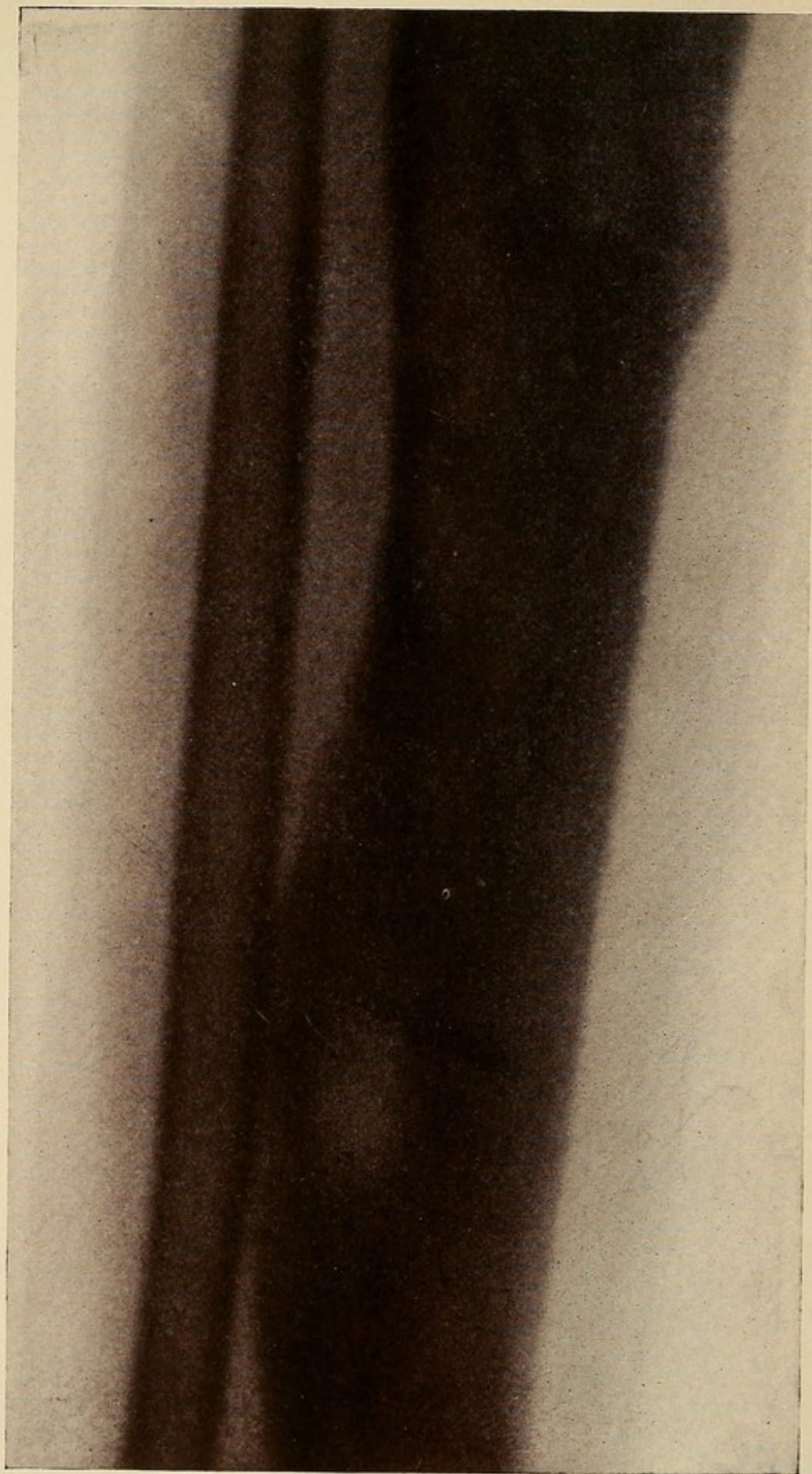
and there; the bone shadow is darker in osteosclerotic areas and lighter in the osteoporotic parts. Sequestra are recog-

FIG. 162



Chronic osteoma of the right tibia (recurrent). Note the diffuse swelling and thickening of the right leg, which, together with the history of repeated acute exacerbations of the malady, readily permitted us to make the diagnosis of chronic pyogenic osteomyelitis.





Chronic Osteomyelitis of the Tibia, with Sequestra Formation.

The lightest areas correspond to the cavities in the bone which contain the sequestra. The slightly darker shadows correspond to the osteoporotic bone, and the darkest shadows represent the osteosclerotic portions of the bone. A sinus leading down to a sequestrum is visible in the upper part of the picture.

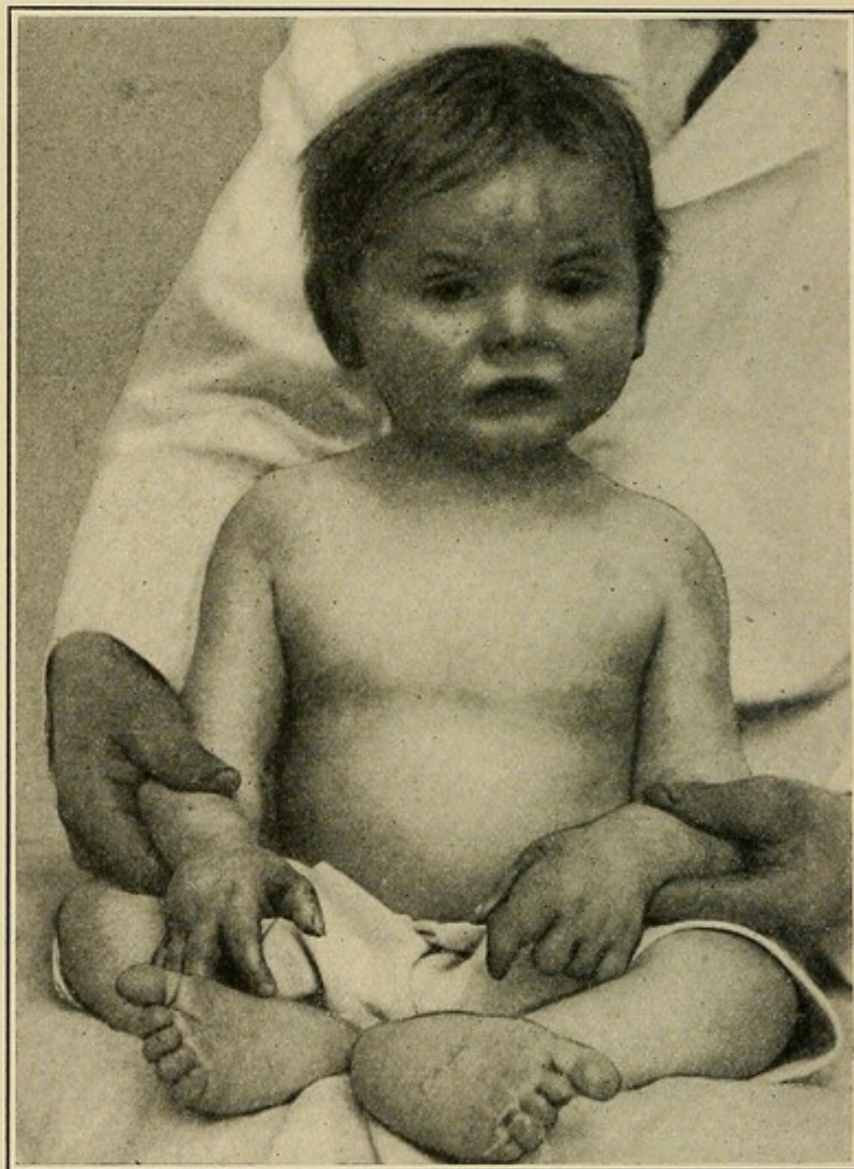
nized as light shadows lying in a still lighter area, which latter corresponds to the cavity containing the sequestrum. The reading of the radiograph correctly demands considerable experience; the picture of the healthy part should always be read in conjunction with that of the diseased side.

In a well-marked case of chronic bone disease little difficulty should be experienced in making the diagnosis. The spontaneous, intense, boring pain; the local tenderness, the changes in the contour and thickness of the bone, the abscesses in the soft parts, and the presence of sinuses and fistulæ leading down to carious or necrotic bone clearly indicate the disease which is present. But easy as it is to make the diagnosis in the fully developed cases, so difficult is it often in the initial stages. In children who are the most frequent sufferers from this form of bone disease the slight functional disturbances which are present in the early stages of the disease are often ascribed to a fall or trauma; on cross-questioning, however, we will, as a rule, ascertain that the trauma was very slight and not sufficient to account for the symptoms, and frequently we will find that the traumatism was not really noticed by the parent, but only subsequently thought of in order to account for the little patient's trouble. One sign is pathognomonic of early bone disease when it is located at the epiphyseal ends, and that is restricted movements of the adjacent joint in one or more directions, the limitation of movement being attended with a *reflex muscular spasm*. Continued use of the x-ray and careful study of the pictures which it gives may in the future help us considerably in the early diagnosis of chronic bone disease.

The nature of a chronic bone inflammation is determined from a study of the anamnesis and from the especial characteristics of the local lesion. The family and personal past history of the patient and other evidences of a constitutional disease may furnish us with valuable data for this determination. Thus a history of a previous attack of acute osteomyelitis in the same bone, or in another bone, or of recurrent exacerbations of the chronic inflammation, points very strongly to a *chronic pyogenic osteomyelitis*. If the disease

is located at the diaphyseal end of one of the long bones, and commenced acutely or subacutely with moderate fever and pain, which subsided after a few days, and if its subsequent course was characterized by a very gradual but considerable cystic swelling of the periosteum and bone from

FIG. 163



Spina ventosa. Note the swelling of the fingers, due to distention of the medullary canal of the phalangeal bones by tuberculous tissue.

an accumulation of synovial-like, mucoid material between the bone and periosteum, or in the periosteum, or external to it, it is very likely to be a *periosteo-osteomyelitis albuminosa* (Ollier)—*i. e.*, a subacute, serous osteomyelitis; such an inflammation is due to bacteria of diminished virulence.

If the disease is very chronic and is located at the epi-

physeal ends of the long bones; if there are abscesses in the soft parts which on aspiration yield a yellowish, flocculent material, and, further, if there are worm-eaten, cheesy, undermined, retracted, fistulous openings which lead down to bare carious bone, and from which a cheesy, flocculent material is discharged, it is *tuberculous* in character. Occasionally the medullary canal is much distended by the tuberculous deposit within it, and its cortex is consequently thinned and expanded. The *spina ventosa* most frequently seen in the phalanges of the fingers are examples of such lesions.

If there is a previous history of syphilis and if there are in the periosteum one or more flat, elastic swellings, which contain a fatty, cheesy, somewhat purulent material, the disease is *syphilitic*; these lesions are styled *gummata*. Under antisyphilitic treatment these swellings shrink into firm, fibrous nodes. Such periosteal swellings are especially frequent in the cranial bones and clavicle, but they are rare in the diaphyses of the long bones.

Gummata of the bone itself form nodes varying in size from a lentil-seed to a nut. They are single or multiple, and may remain unrecognized until spontaneous fracture takes place; this occurrence, together with a history of syphilis and the absence of a distinct neoplasm, should enable us to make the diagnosis.

A previous history of syphilis with hypertrophy and irregular nodulation and sclerosis of the bone is indicative of a *syphilitic ossifying osteoperiostitis*.

The picture of rachitis with a congenital luetic history suggests syphilis as a cause of the rachitical symptoms.

If the bone symptoms develop during the course of or during the convalescence from typhoid fever and are sub-acute in character, the process is in all probability due to the typhoid bacillus. It usually commences at those portions of the bone to which the muscles are attached.

Benign and malignant neoplasms of bone give rise to pain, local tenderness, thickening, and hypertrophy of the bone, and so resemble the chronic inflammatory conditions. The enlargement which is due to periosteal or cortical neoplasm, however, is circumscribed; it commences abruptly, whereas that due to chronic inflammation is diffuse and more irregular.

Central sarcomata occasion a diffuse enlargement of the bone, and are to be differentiated from inflammatory affections by their rapid growth, by the absence of previous inflammatory, syphilitic or tuberculous history, and by the egg-shell crackle of the thinned-out cortical tissue over the tumor. The *x*-ray often aids in the differentiation of these cases.

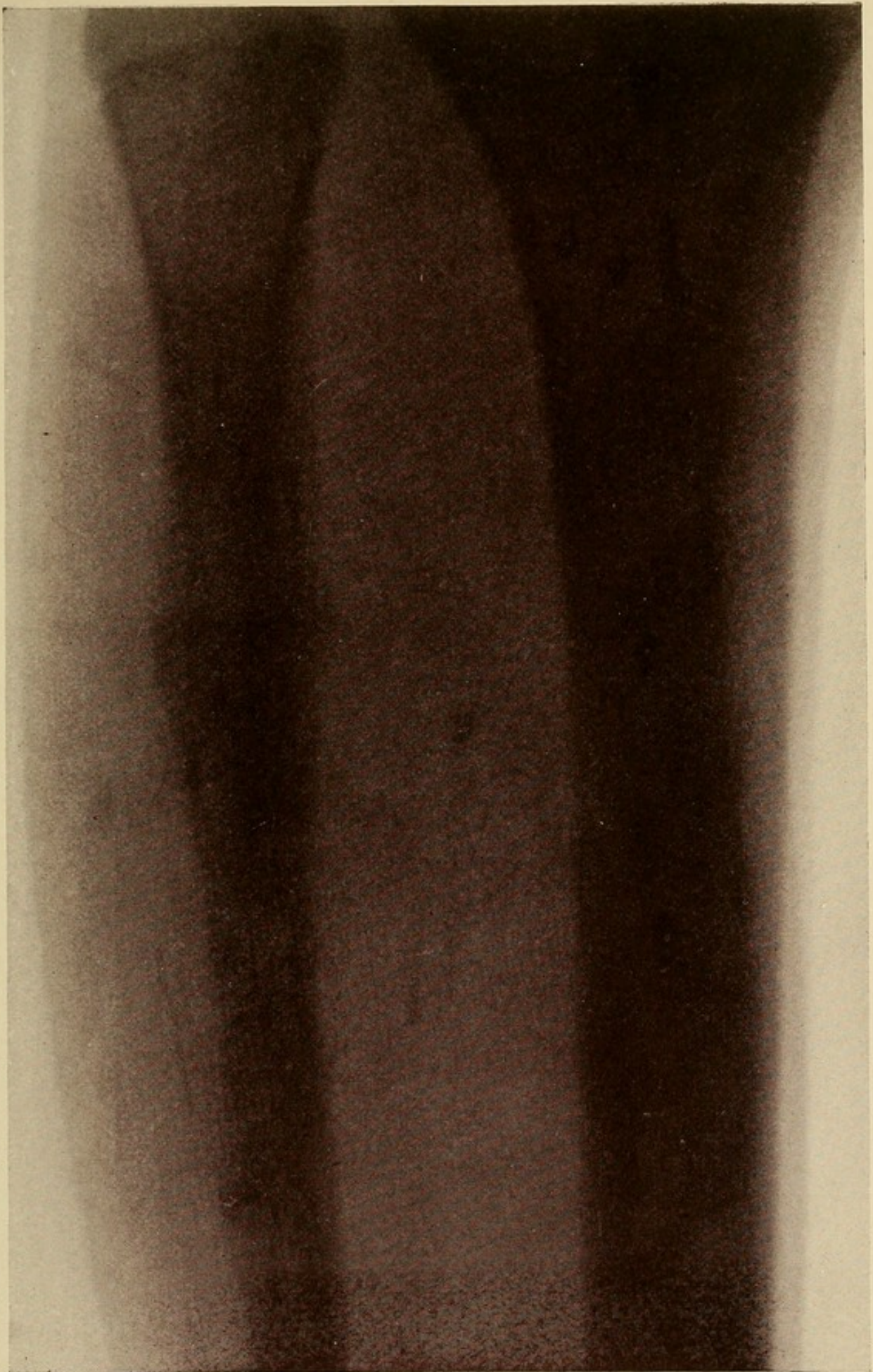
In a number of cases, however, it is impossible to decide upon the exact nature of the swelling of the bone prior to its exposure and microscopic examination. It is well to consider every doubtful neoplastic swelling as syphilitic until a lack of improvement thereof from antispecific treatment has proven it not to be so.

NUTRITIVE DISEASES OF BONES.

A local malnutrition of bone with consequent atrophy and fragility thereof accompanies disease of contiguous joints, while nutritive disturbances of a number or all of the skeletal bones are dependent upon some constitutional disease, such as rickets, osteomalacia, fragilitas ossium, and osteitis deformans.

Rachitis.—Rachitical disease occurs in insufficiently or improperly fed children and in those who suffer from gastrointestinal diseases with consequent impaired digestion, absorption, and assimilation. It is readily recognized from the changes it occasions in the osseous system; these include a softening of the cranial bones (craniotabes), a delayed closure of the fontanelles, a broadening of the anterior portion of the skull, an enlargement at the chondrocostal junction (the latter forming the rachitical rosary), a flattening of the chest, and tender, enlarged epiphyseal ends of the long bones, with relaxation of the joint ligaments, thus permitting of abnormal mobility of the joints. As the children grow older spinal curvature, bow-legs, genu valgum or varum, bear testimony to the past or present existence of rachitical disease.

Osteomalacia.—Osteomalacia occurs chiefly in pregnancy or nursing women, though it is met with in men and in non-puerperal women. In the former it starts in the pelvic bones, in the latter in the spinal column and bones of the chest. The disease is readily recognized from the softening, bending,



Periosteal Sarcoma of the Fibula.

Note the spindle-shaped enlargement of the upper end of the bone; there is no thinning out of the cortical tissue and no involvement of the medulla by the neoplasm. Note the smooth surface of the periosteum and compare with Plate IX., of inflammatory osteoperiostitis. This tumor gave rise to a metastatic sarcoma of the brain. It was unnoticed by the patient, who came to the hospital for his cerebral condition.



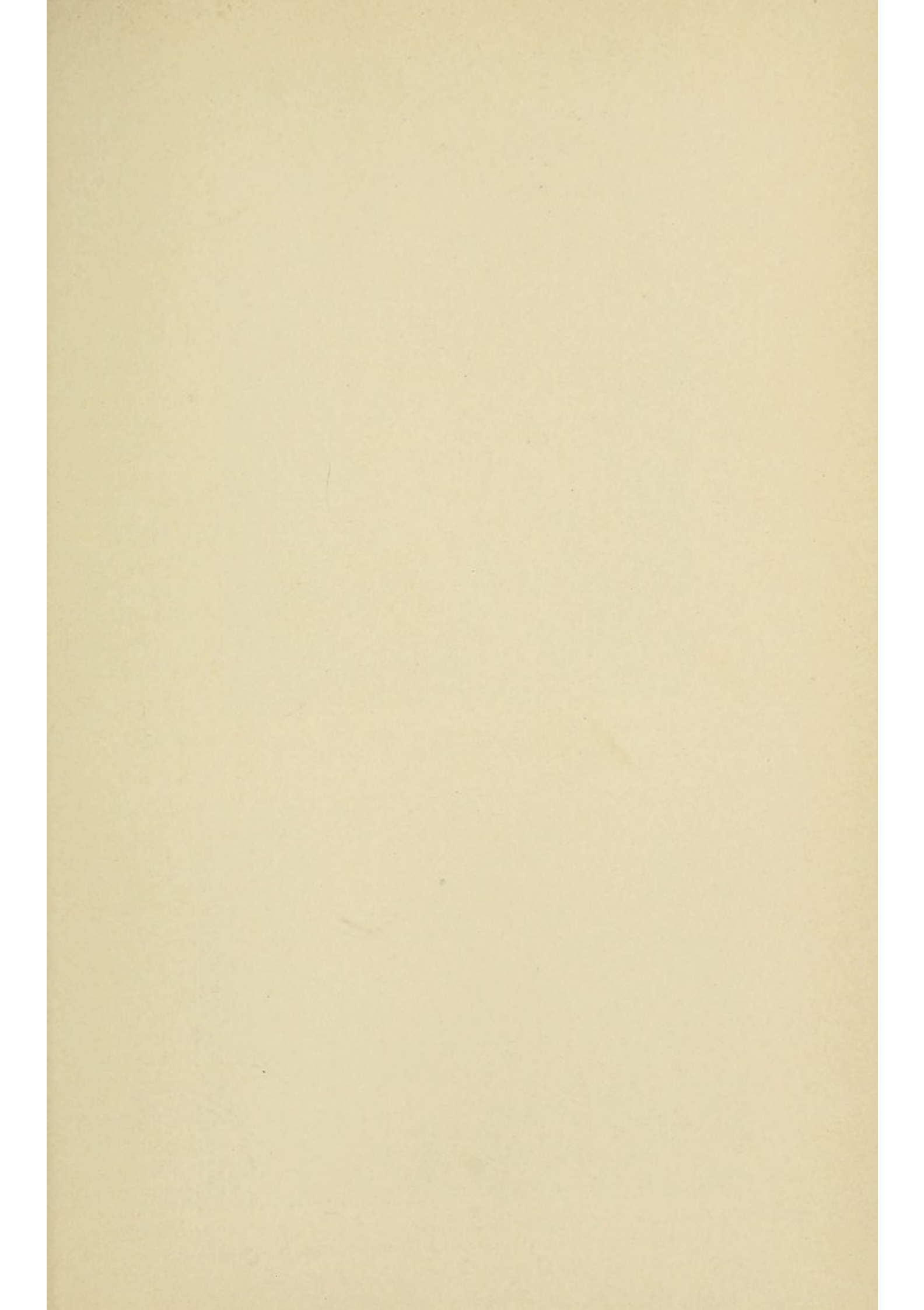
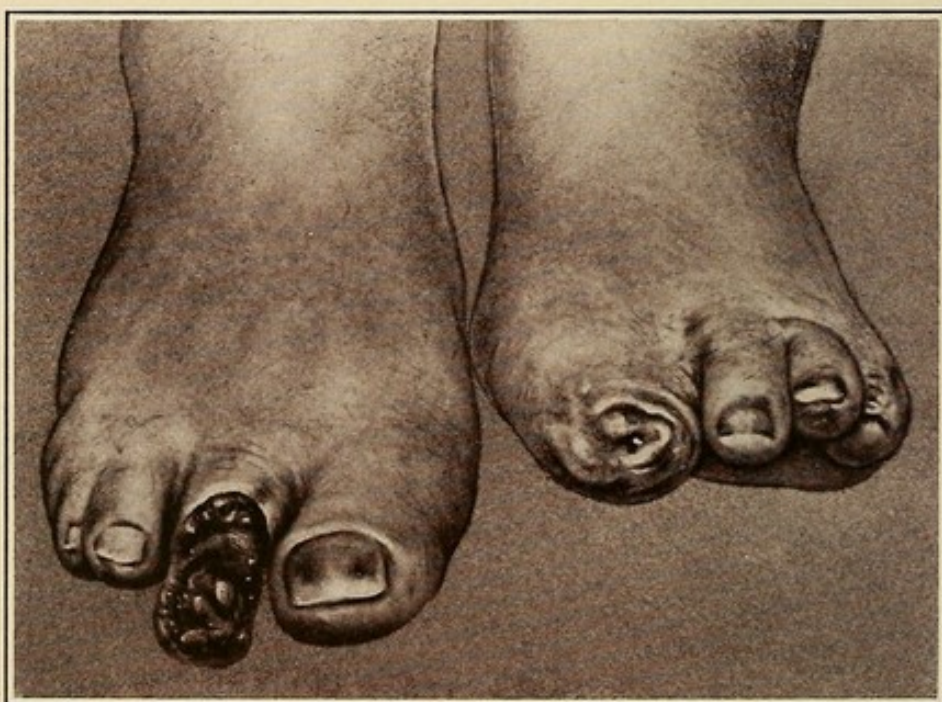


PLATE XII.



Sarcoma of the Leg, on the basis of an old varicose ulcer of the leg. Note character of the ulcer.



Raynaud's Disease, with Gangrene of Second Toe Note that the big toe on the other foot is also partly missing from gangrene.

and spontaneous fracture of the bones which it occasions. The wandering pains in the bones, which attend the early stages of the disease, may suggest rheumatism, but the latter is readily excluded by the absence of joint involvement. From rachitis it is differentiated by the infrequency with which the cranial bones are involved, while the spontaneous fractures to which it gives rise are differentiated from those due to malignant disease by their multiple character and the absence of a tumor.

Osteitis Deformans.—Osteitis deformans is characterized by its development in middle life or later, the symmetrical distribution of the lesions, its very slow progress, and its predilection for the long and cranial bones. The affected bones become enlarged and softened, in virtue of which they yield and bend.

Exostoses.—Exostoses may likewise be looked upon as disturbances of nutrition. Their favorite sites are the epiphyseal ends of the long bones, especially the inner surface of the femur just above the condyles, the head of the tibia, and the phalanges of the fingers and toes. They are often multiple and their surface is irregular.

NEOPLASMS OF BONE.

Benign tumors, of which the osteoma, chondroma, and fibroma are the chief types, develop either from the periosteum or from the epiphyseal cartilages.

The *fibromata* originate from the periosteum and form hard, strictly circumscribed, painless, very slowly growing tumors. The jaw-bones are the favorite sites of these tumors, and in this situation they go under the name of *fibrous epules*.

The *chondromata* usually start from beneath the periosteum of the long bones independent of the epiphyseal cartilage, and form firm, lobulated, encapsulated tumors, which are *painless* unless they press upon nerve trunks. They may extend into the medullary canal and cause expansion of the bone, or they may cause erosion of the bone with resulting spontaneous fracture. The chondromata growing from the smaller bones (most usually those of the hand) are frequently

multiple and commence in the interior of the bone close to the epiphyseal cartilage and cause its expansion.

Osteomata are usually met with near the articular end of the bone, forming pedunculated or sessile tumors, often of large size. They start to develop during youth, and as the bones grow their base of attachment may become separated from the epiphysis to an extent corresponding to the amount of growth which has taken place. As a rule the growth of the tumor ceases at maturity. Their most common sites are the inner condyle of the femur close to the adductor tubercle, and the inner aspect of the jaw. *Ivory exostoses* develop most frequently on the inner or outer aspects of the cranial bones.

All benign tumors are of slow growth, encapsulated and painless, except when they press upon nerve trunks. Malignant tumors, on the contrary, are of rapid growth, somewhat painful, not encapsulated, and frequently lead to erosion of the cancellous tissue with resulting spontaneous fracture.

The *myxofibromata* form a mid-class between the benign and malign tumors. They grow rather rapidly, are somewhat painful, and tend to relapse locally after removal. They do not break down and ulcerate, and do not give rise to metastases in the other organs.

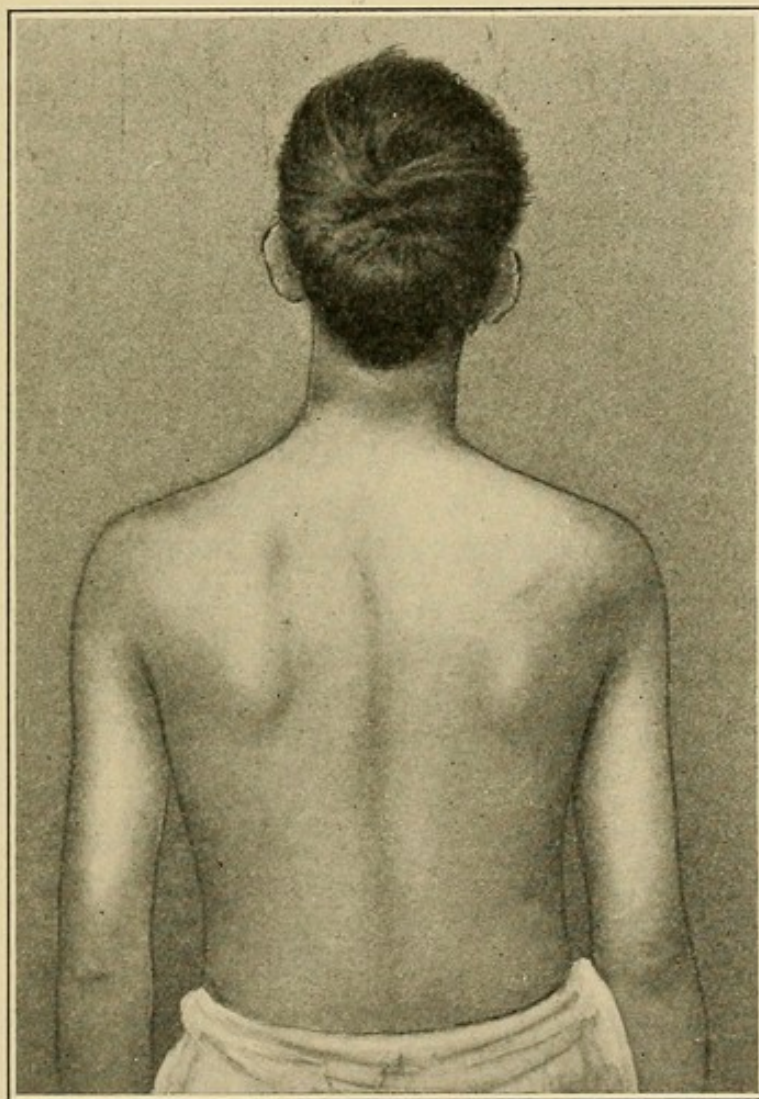
Sarcomata may develop either from the periosteum or from the medullary substance. The *periosteal tumors* usually start on one side of the bone, but they soon surround its entire circumference and spread rapidly along its surface; they may be very vascular and pulsate and they give rise to metastases in the glands and internal viscera. The superficial cutaneous veins in the skin overlying the tumor are dilated. The *medullary sarcomata* usually commence near the end of one of the long bones, but seldom encroach on the articular cartilage or joint; they gradually expand the surrounding cancellous tissue, thinning it out and causing it to crackle when it is palpated (the egg-shell crackle), and frequently cause spontaneous fracture.

The round and spindle-celled medullary sarcomata are of very rapid growth and give rise to metastases; the giant-celled ones, most frequently situated on the lower ends of the femur and radius, the upper ends of the tibia and humerus, the horizontal ramus of the lower jaw and in the diploë, are

of very slow growth, frequently encapsulated, never give rise to metastases, and do not spread along the bone.

Carcinomata of the bones are always metastatic tumors. The first indication of their presence is often a spontaneous fracture, or severe shooting pains in the affected region.

FIG. 164



Sarcoma of the right scapula.

As a rule, no difficulty is experienced in distinguishing between the enlargement of bone due to chronic inflammation and that due to neoplasm, for with the former the enlargement is much more diffuse and irregular, and the history points to an infection of the bone.

Similarly the differentiation between malign and benign tumors is easy; the giant-celled sarcomata belong to a mid-class between the malign and benign growths.

The enlargements of bone due to *arthritis deformans* are distinguished from those due to neoplasm by the symmetrical distribution of the lesions, their multiple character, and by the fact that they usually develop after middle age.

Pulsating sarcomata of the bones resemble aneurysms, from which, however, their incompressibility, their non-disappearance on compression of the afferent vessel, and their diffuse character serve to distinguish them.

Hydatid Cysts of the Bones.—*Echinococcus* cysts are rare in their occurrence; they develop in the medullary canal, expand it and cause its absorption, with resultant spontaneous fracture.

CHAPTER XLVIII.

GENERAL REMARKS ON THE DIAGNOSIS OF JOINT INJURY AND DISEASE.

THE trend of modern surgery is toward conservatism, and while it aims to thoroughly eradicate what is diseased, it strives more and more to retain what is healthy and preserve the function of the diseased part. In chronic diseases of the joints there is as yet no unanimity of opinion as to whether this conservative end is better attained by the bloody or bloodless method of treatment, but this much is acknowledged by all—viz., that either method will achieve its best results if the disease is recognized early and treatment immediately instituted.

The diagnosis of the acute inflammatory joint diseases is not difficult, for the local lesions and the constitutional symptoms clearly indicate the nature of the malady. Similarly the presence of traumatic lesions is readily recognized, though until the *x*-ray enabled us to view the articular structures it was often impossible to determine their exact character. The recognition of chronic joint disease, especially in its initial stages, is, on the other hand, always difficult and calls for repeated, careful, systematic physical, and *x*-ray examination.

The joints are made up of the articular ends of the bones, covered on their articular surfaces by cartilage, and held together by capsular ligaments, which latter may be strengthened by accessory bands and ligaments. The interior of the capsule is lined by a synovial membrane, which may have diverticula and pouches (*bursæ*) between the tendons and muscles passing over the joints, and whose function it is to secrete a lubricating fluid for the articulating bones. Additional cartilaginous structures are present within some of the joints, to equalize or deepen the articular surfaces. Any

one or all of these structures may be involved in disease. The process usually starts in the synovial membrane or bone, and either remains confined to these structures or extends to the other parts of the joint, resulting in a panarthrititis.

In dealing with joint diseases it is necessary first of all to obtain a good family and personal history of the patient, especially as to family or personal tuberculosis, syphilis, or hemophilia. The mode of onset, the course and duration of the symptoms should also be ascertained. The examination should determine the contour of the joint, the position in which the affected limb is held, the direction of its axis, its cutaneous aspect, the presence of fluid within its capsule, the relation of its own bony prominences to each other and to neighboring fixed bony points, shortening or lengthening of the limb, the range of active and passive motion in *all directions*, whether a reflex muscular spasm is elicited in passively moving the joint, and finally the *x-ray* should determine the position of the bones, their contour, their density, and the condition of the epiphyseal cartilage. In every detail of the examination we should always compare the healthy with the supposedly diseased side.

Contour.—The contour of a supposedly diseased joint is very significant. If an enlargement thereof is sharply circumscribed and strictly limited to the joint-capsule it is indicative of the diseased process being intracapsular; but if, on the other hand, the enlargement extends in a fusiform fashion up and down the limb with the joint at its widest part, the disease is no longer intracapsular, but involves the periarticular structures as well, an indication of a panarthrititis.

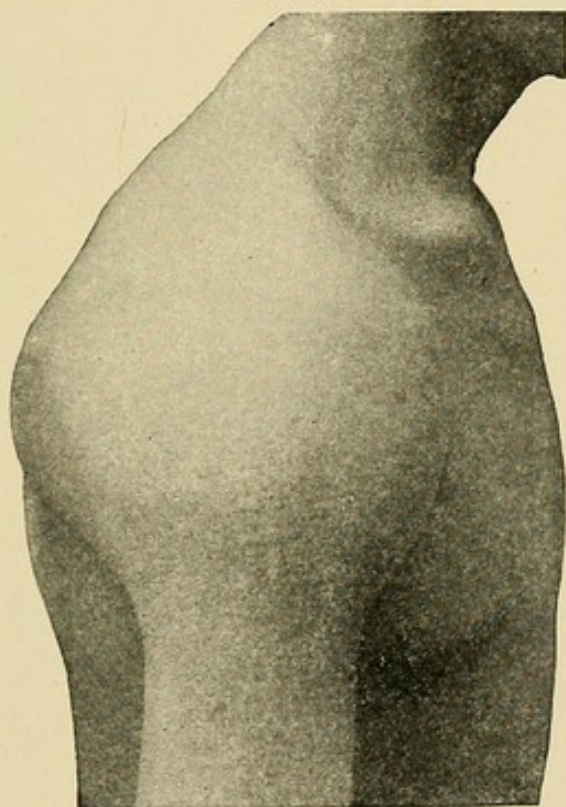
Position.—The position in which a joint is held is important. In the flexed position it can accommodate the largest amount of fluid within its capsule, consequently this is the position that is assumed when a considerable amount of exudate is present within the joint. Other factors, such as destruction of the bone with displacement of the joint surfaces and reflex muscular contraction from bone disease, may account for abnormal positions in which the joint is held.

Color and Vascularity of Cutaneous Covering.—The color and vascularity of the cutaneous covering of a diseased

joint are likewise significant. Thus the pale, waxy, marbled aspect of the skin in tuberculosis has given to this disease the name of white swelling. With acutely inflamed joints the skin is reddened and hot. The enlargement of the cutaneous veins bears testimony to long-standing congestion of the part from chronic inflammation or neoplasm.

Atrophy.—The atrophy of muscles above and below a joint results from disuse as well as from organic disease of the joint. In hysterical joint affections the muscular atrophy

FIG. 165



Contour of shoulder-joint in inflammation of subdeltoid bursa.
(Von Bruns.)

is sometimes very great and this may mislead us into thinking that organic disease of the joint is present. The absence of all other signs of organic disease will enable us to make the correct diagnosis. Furthermore, in organic joint disease the atrophic muscles frequently show the electric reactions of degeneration; this never occurs in hysterical affections.

Presence of Fluid.—The presence of fluid within the capsule of the joint is readily recognized from the filling up and rounding out of the normal depressions around the joint. Thus in the *shoulder* the normal curvature is increased and

the deltoid is expanded by a fluid swelling beneath it; this is especially noticeable at the anterior border of the latter along the bicipital groove, and sometimes posteriorly, and in the axillary space. Such capsular distention is distinguished from distended bursæ around the shoulder-joint by the fact that the swelling in the former instance is to be seen and felt in the axilla, and in front of and behind the deltoid muscle, whereas distended bursæ occupy one or the other of these regions.

FIG. 166



Olecranon bursitis. Note the central location of the swelling and the normal hollows on either side of the olecranon process. Compare with Fig. 167, of contour of elbow-joint when fluid is present within the joint capsule.

In the elbow the hollows on either side of the olecranon and tendon of the triceps are replaced by soft, fluid swellings, the outer of which also extends down to and masks the head of the radius. It is readily distinguished from distention of the olecranon bursa by the fact that in the latter condition there is a central fluid prominence over the bone, whereas in the former the swellings are placed on either side of and above the bony projection.

In the wrist there is a general fulness, both on the anterior and posterior aspects of the joint; fluctuation may be detected between the dorsal tendons, which are slightly separated and displaced. It is distinguished from fluid swellings of the tendon sheaths by the fact of its strict limitation to the neighborhood of the joint, and by absence of crepitus that is usually to be felt in these latter conditions.

FIG. 167



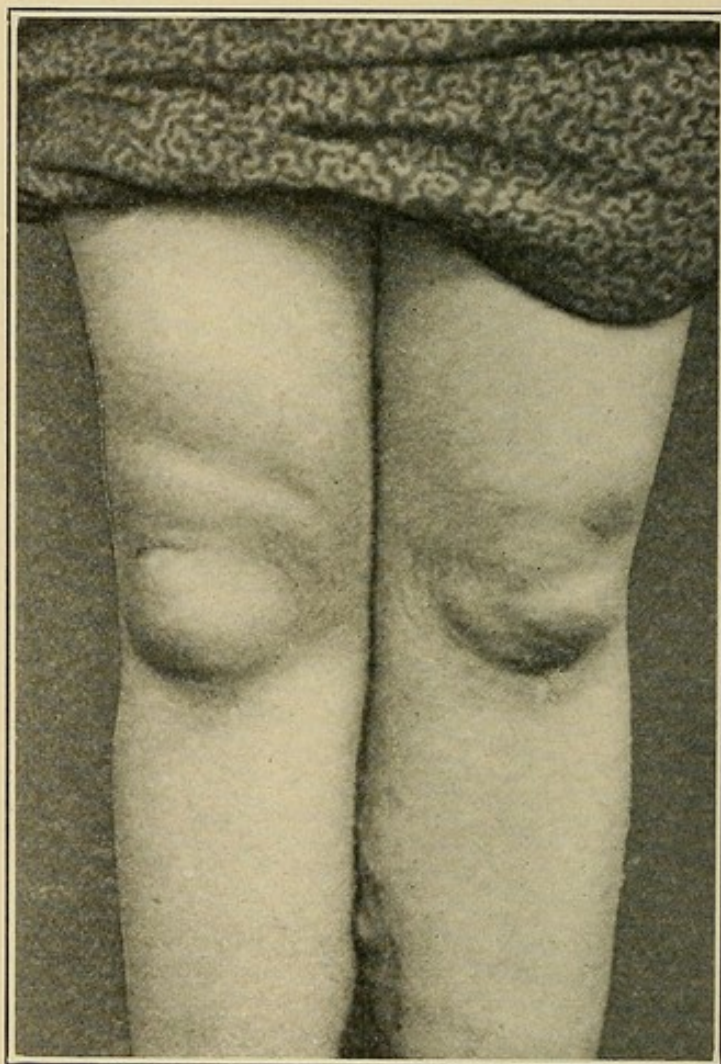
Contour of elbow-joint in distention of its capsule. Note obliteration of all the normal furrows. (Von Bruns.)

In the hip there may be a little fulness in the gluteal region or in the upper and outer part of Scarpa's triangle. The limb is held in abduction, flexion, and eversion.

In the knee the normal hollows, especially those on either side of the patella and ligamentum patellæ, disappear. There is also a swelling corresponding to the subcrural pouch, more marked on the inner than on the outer side,

and extending three or four inches above the patella. When the effusion is large in amount the patella is felt to float, and on crowding down the fluid from the subcrural pouch with the one hand and pressing the patella sharply backward with the other it can be felt to tap against the intercondyloid

FIG. 168



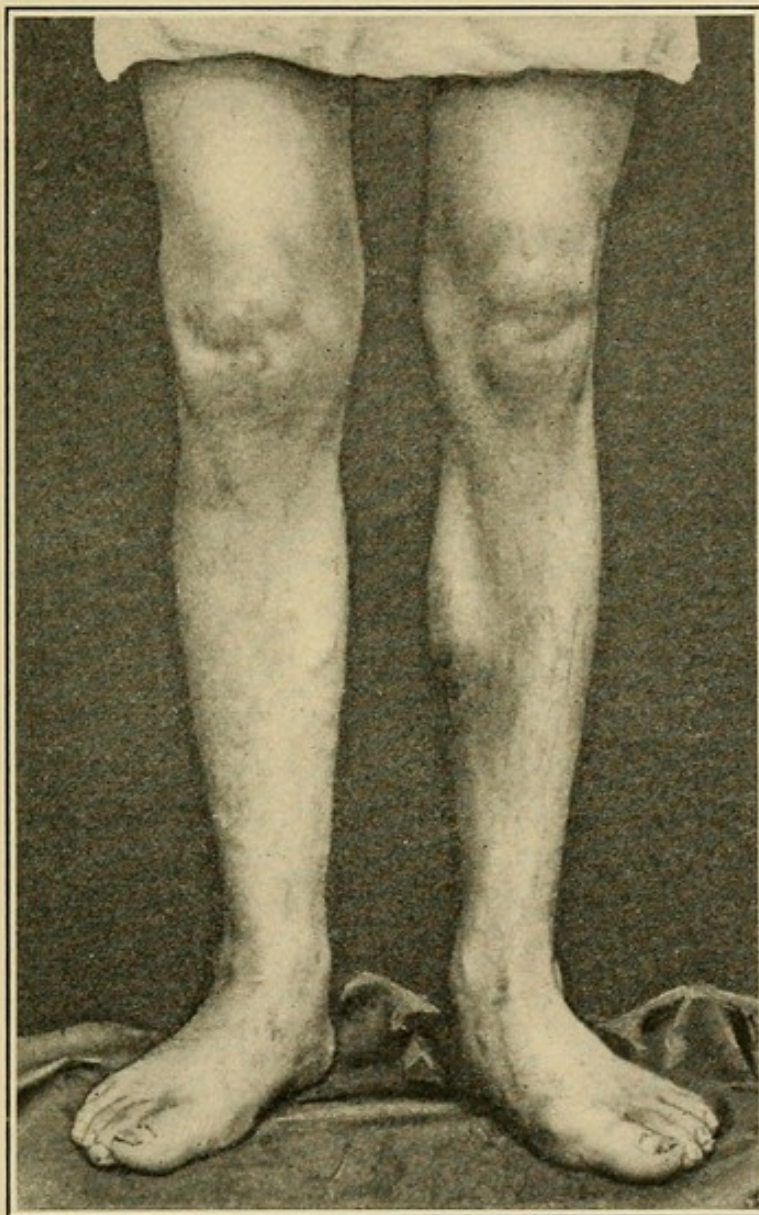
Bilateral distention of the patella bursa. Note the central location of the swellings and the persistence of the normal furrows to either side of the patella. These characteristics distinguish these swellings from those due to the presence of fluid with the joint. Compare with Fig. 169.

notch of the femur. Enlargement of the patella bursa can be distinguished from intracapsular joint effusions by the central location of the swelling that it occasions.

In the ankle the hollows between the tendo Achillis and the malleoli are replaced by fluctuating swellings, the dorsal tendons are displaced forward, and a fluid swelling appears

in front of each malleolus. Enlargement of the bursa beneath the tendo Achillis is distinguished by the fact that it is confined to the back of the joint.

FIG. 169



Distention of the right knee-joint with fluid in simple inflammation. Note the absence of all the normal furrows, especially those on either side of the patella as seen in the healthy left knee. Compare with Fig. 168.

Relation of Fixed Points.—The relation of fixed points on the articular ends of the bones to each other and neighboring landmarks is important as an indication of the normal or abnormal position of the articulating surfaces. These relations may be altered by fracture, or by dislocation, or by

destructive disease of the bones and capsule which permit of pathological dislocation.

These relations are readily ascertained and the cause of a disturbance in their normal condition is determined from the anamnesis, by careful palpation, and by *x-ray* examination. In the *hip* the upper border of the great trochanter should just touch a line that passes from the anterior superior iliac spine to the lowest point of the ischial tuberosity; furthermore, the distance of the anterior margin of the upper border of the great trochanter from a line that passes vertically downward from the anterior superior iliac spine, the patient lying horizontally, and the distance of the great trochanter from the median line of the body should correspond on both sides.

In the *elbow* the two epicondyles and the upper border of the olecranon should be on a straight line when the forearm is extended.

In the *wrist* the tip of the radial styloid process should be a little lower than the tip of the styloid process of the ulna.

Measurement.—Measuring the length of a limb that has been injured or is thought to be diseased should always be a matter of routine procedure. If no tape-measure is at hand, a rough but very good estimate of the comparative length of the lower limbs may be obtained by placing the patient *flat on his back* on a level couch or bed and then comparing the level of the malleoli, the limbs being in symmetrical positions. The more accurate method is tape-measurement. In the upper extremity the measurement should be made from the tip of the acromion process to the tip of the styloid process of the radius; in the lower extremity from the anterior superior iliac spine to the tip of the internal malleolus. Care should be taken that the disease or injury is not bilateral—*e. g.*, coxa vara, congenital dislocation, etc.

Motion.—The extent of and direction in which active motion is possible should be accurately determined. In dislocations active motion will be possible only in the direction of the deformity. It is likewise important to determine the extent of passive motion, and, in connection therewith, the presence or absence of *reflex muscular spasm*, a most important early sign of articular disease.

The spasm is usually not elicited by all passive movements; but with movements in certain directions, either in flexion or extension, abduction or adduction, inward or outward rotation, it is suddenly provoked and further movement in that direction is then impossible.

X-ray.—The *x*-ray has added vastly to our means for the detection of injuries and diseases of joints, and especially has

FIG. 170

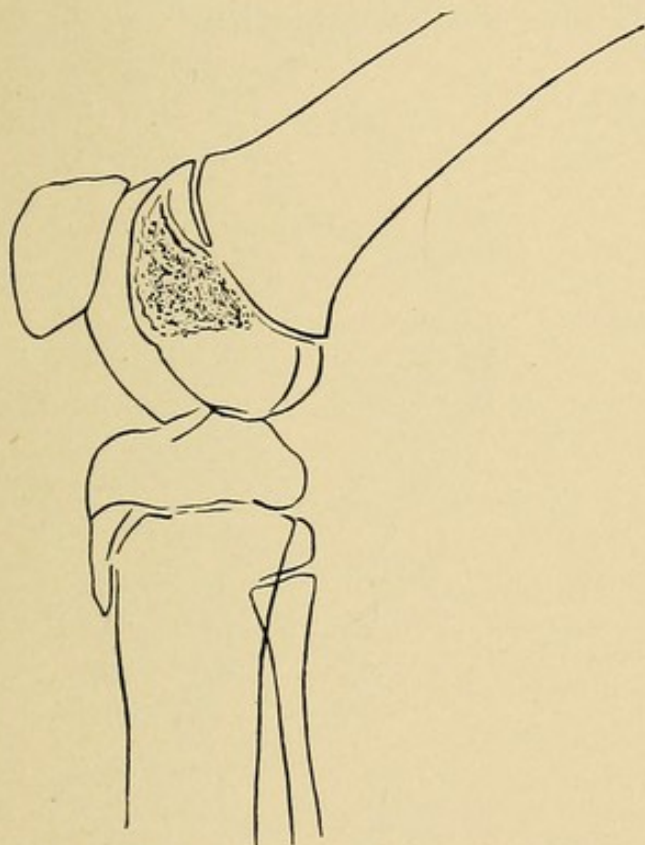


FIG. 171

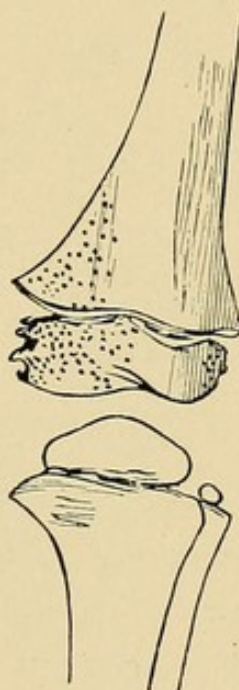


FIG. 170.—X-ray tracing of normal knee-joint (lateral view), showing the ossification nucleus of the femoral condyles. This is a fine network in structure and persists up to the sixteenth year of life. (Ludloff.)

FIG. 171.—X-ray tracing of normal knee (anteroposterior view), showing the protuberances on the medial aspect of the inner femoral condyle). These are present only from the second to the fifth years of life. (Ludloff.)

it proved of value in a field where early diagnosis is particularly important—viz., in tuberculous disease. Thus in *tuberculosis of the knee-joint* occurring in children under five years of age the *x*-ray shows in the initial stages a diminution or disappearance of the normal protuberances which are to be seen on the bony, cartilaginous junctions, especially of the internal condyle, an irregular bone formation on the

under surface of the condyles, an enlargement of the bony or ossified parts of the femoral condyles, tibia, patella, and fibula head, and an enlargement and rarefaction of the ossification nucleus in the epiphysis. The last is the most valuable because it can be seen up to the fifteenth or sixteenth years of life. In the *hip-joint* the *x-ray* does not afford as much information as is desirable because of the thick overlying muscles. The joint line, instead of showing as a light, half-moon-shaped zone, appears very dark or obscure or shows dim, finger-like projections and irregularities

FIG. 172

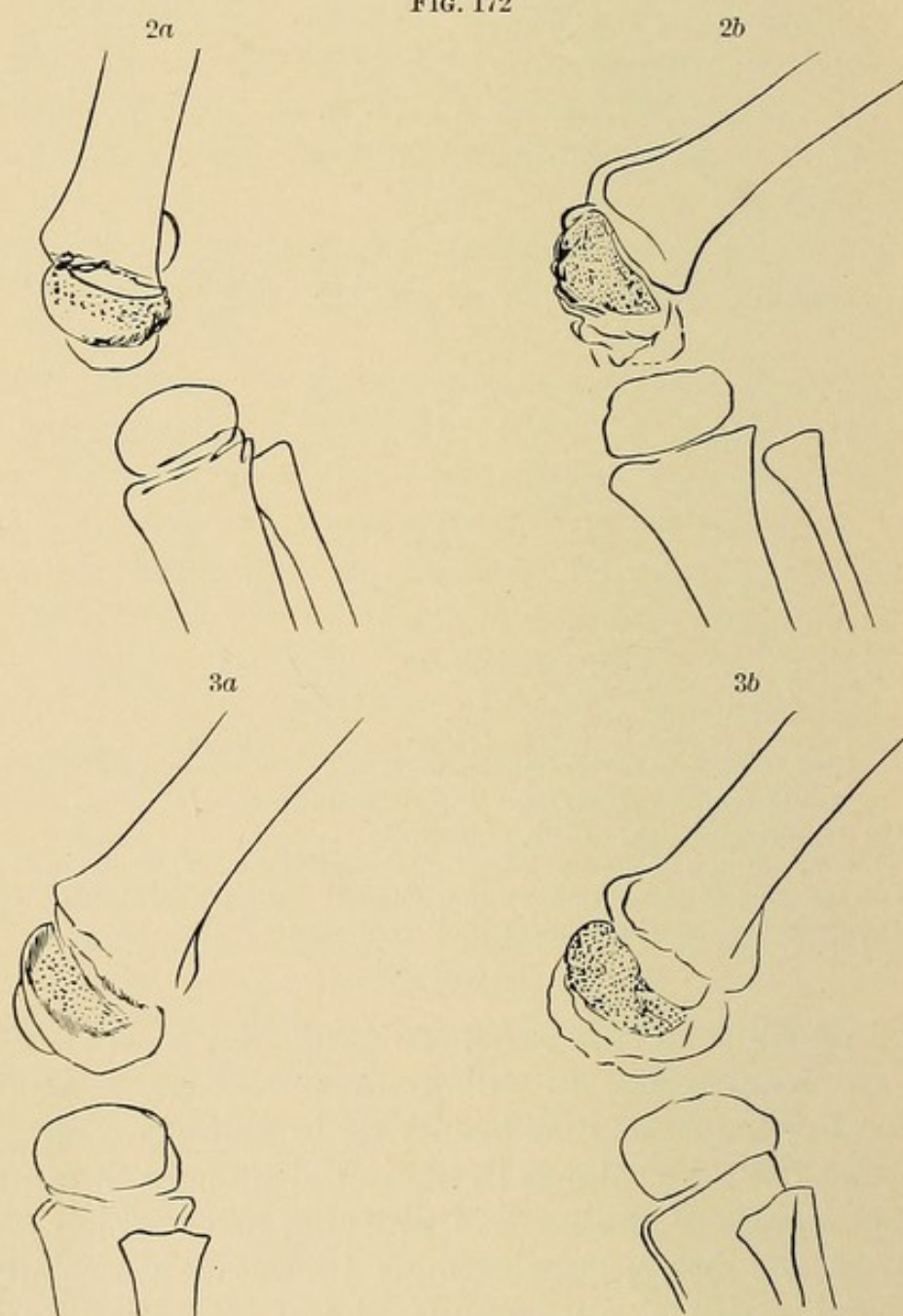
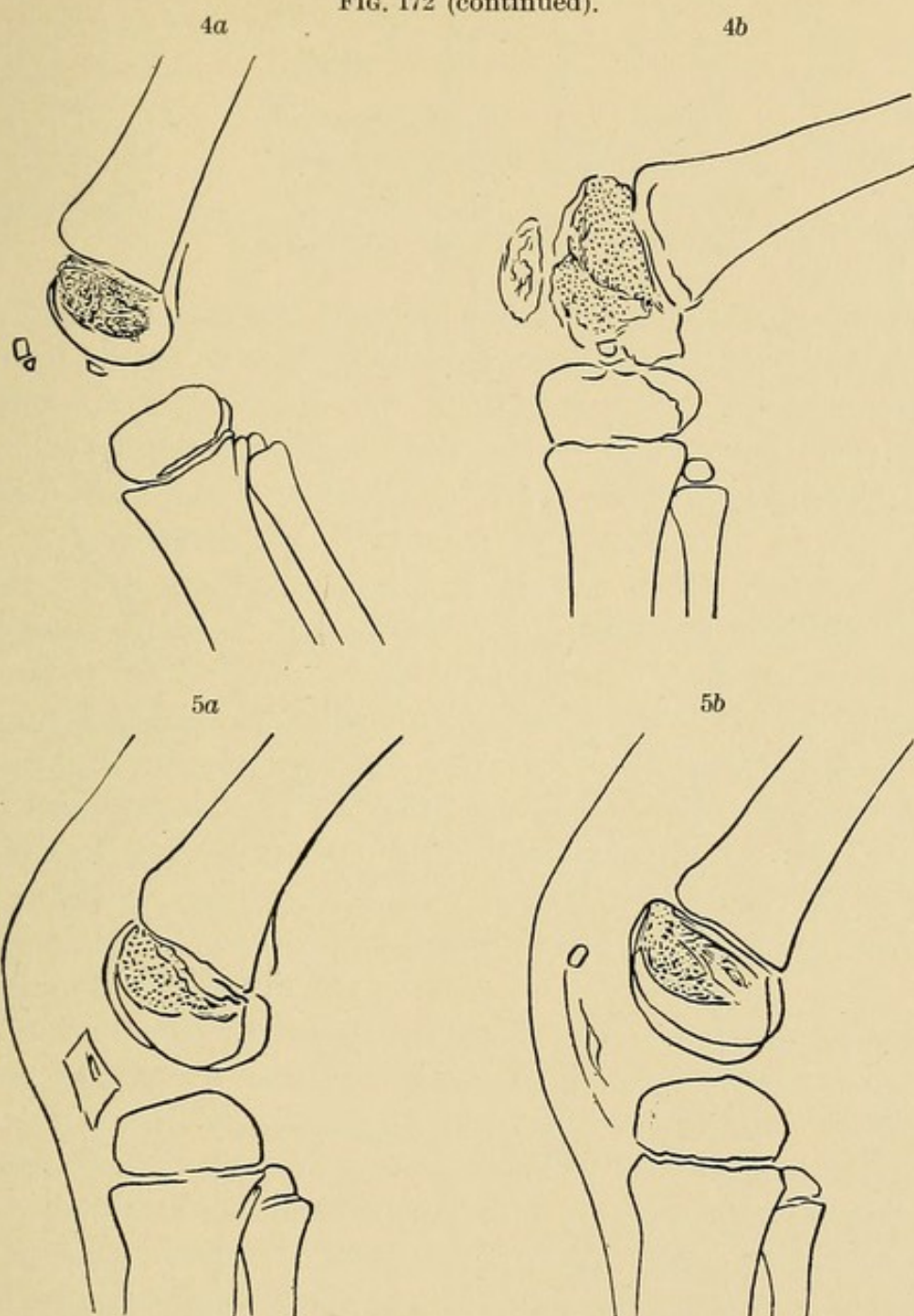
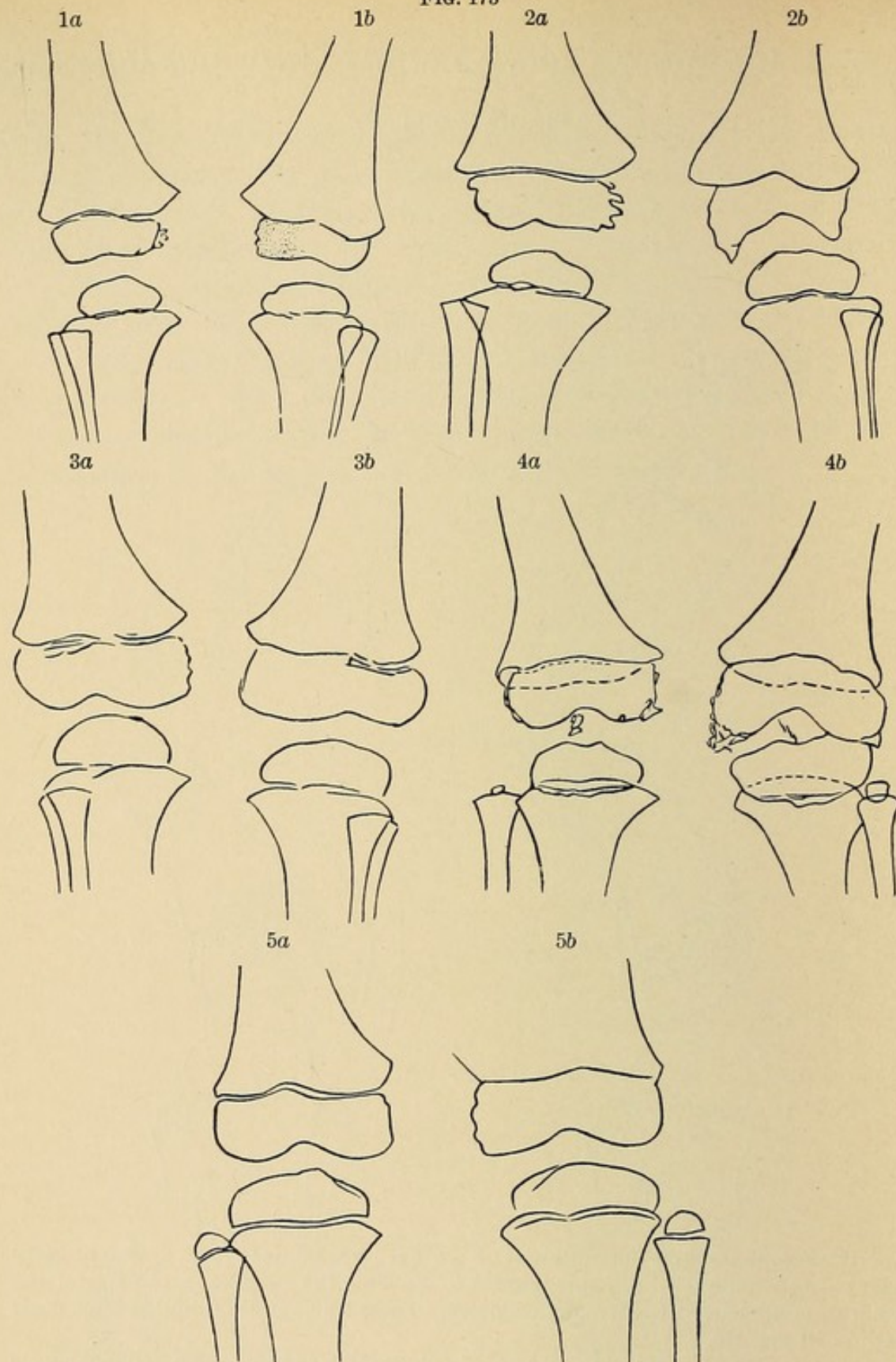


FIG. 172 (continued).



Series of *x*-ray tracings (lateral view) of normal knee-joints (figures *a*) and of early stages of tuberculous knee-joints (figures *b*), showing the changes in the size of the ossification nucleus of the femoral condyles. These tracings are from the same cases as those in Fig. 173.

There is no lateral view of 1*a* and 1*b*. In 2*b*, 3*b*, and 4*b* the ossification nucleus is much larger than in the corresponding healthy knee-joints. In 4*b* and 5*b* the ossification nucleus of the patella is much enlarged. In 2*b* and 4*b* there is abnormal irregularity on the under surface of the external condyle. These changes can be appreciated up to the sixteenth year of life and hence are of value in the diagnosis of more cases of early knee-joint tuberculosis than are the changes in the protuberances of the inner condyle, for these latter disappear normally after the fifth year of life (Ludloff.)



Series of x-ray tracings (anteroposterior view) of normal knee-joint (figures a) and of early stages of tuberculous knee-joint (figures b), showing the changes which are produced in the protuberances on the femoral condyle, in the early stages of tuberculous disease of the part. All the tracings of the healthy limbs, except 5a, show more or less well-marked protuberances on the medial aspect of the inner femoral condyle. In 5a they are missing because this is a tracing of the knee in a child of seven years of age, when the protuberances have already disappeared. (See legend, Fig. 171.) The tracings of the diseased knees shows these protuberances almost disappeared. In some especially (4b) there are marked protuberances on the lower aspect of the internal condyle and an enlargement of the internal condyle tibial tuberosity, patella, and fibula head. (Ludloff.)

CHAPTER XLIX.

INJURIES OF THE JOINTS.

DIRECTLY and indirectly applied traumatism, and irregular or too sudden or too vigorous action of the muscles attached to the bony prominences around a joint may occasion a fracture of the articular ends of the bones, rupture of ligaments, dislocation of the joint ends, evulsion of bony tubercles or processes, hæmarthrosis or synovitis.

Fractures are characterized by the same signs as attend similar lesions of the shafts of bones; *dislocations* by the deviation of the axis of the limb, abnormal position of the head of the bone, and change in the length of the limb; *sprains* by pain in and tenderness over the joint, some loss of function, ecchymosis and the absence of signs pointing to fracture or dislocation; *synovitis* and *hæmarthrosis* by swelling and distention of the capsule and pain in the joint, the swelling being plainly fluctuating in the former instance and soft and doughy in the latter.

It would appear from the above that the diagnosis of the nature and exact location of the lesion is comparatively easy, and so it would be but for the obliteration of all the landmarks around the joint which is occasioned by the extensive swelling of the periarticular structures and for the excruciating pain that usually renders examination of the parts without the aid of an anæsthetic exceedingly difficult. The *x-ray* has materially aided our diagnostic resources and at the present day no joint lesion should be treated without its employment. In making an examination of an injured joint we should always compare the supposedly healthy side with the injured one.

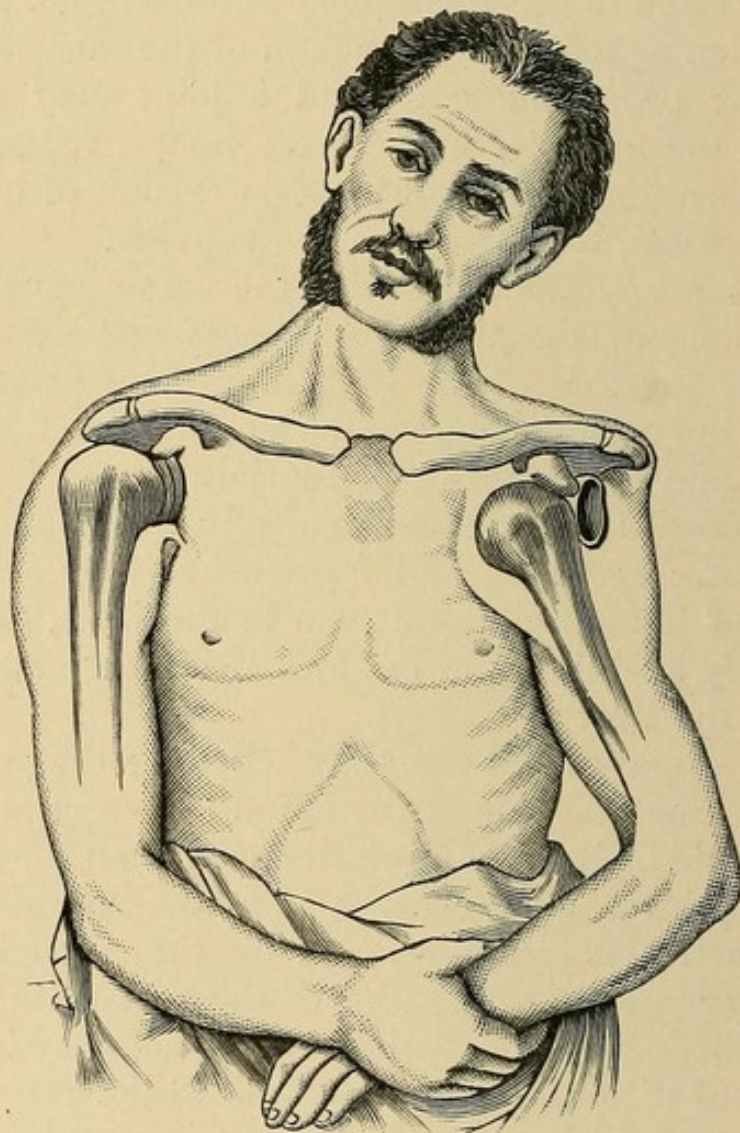
CLINICAL AND DIAGNOSTIC FEATURES OF THE INJURIES OF THE LARGE JOINTS.

Shoulder-joint.—In the examination of an injured shoulder it is important to note the contour of the joint (whether it is rounded or flattened), the direction of the axis of the

limb, the presence of any deformity, the position of the head of the humerus in reference to the glenoid cavity, and whether the humeral head moves with the shaft.

With the most common form of *dislocation* the subcoracoid, the patient's attitude is characteristic. The affected

FIG. 174



Subcoracoid dislocation of the humerus. Note the flattening of the shoulder, the position of the arm and forearm, and the position of the head of the humerus in relation to the other bony landmarks. (Hoffa.)

forearm is held flexed with the elbow away from the side and the arm rotated inward. The anterior axillary fold is lowered; the long axis of the humeral shaft is inclined inward; the shoulder is flattened; the acromion process is prominent. The head of the humerus is out of the glenoid cavity; it rotates with the shaft, and most often lies under the

PLATE XIII.



Subcoracoid Dislocation of the Shoulder-joint. Note the inward displacement of the humeral head, which lies just below the coracoid process.





PLATE XIV.



Fracture of Greater Tuberosity of the Humerus, with Downward Displacement thereof. The usual signs of fracture permitted a ready diagnosis to be made.

coracoid process. The elbow cannot be brought in toward the median line, nor can the hand of the injured arm be placed upon the opposite shoulder. Active and passive movements are greatly restricted, and a soft crepitus is elicited on manipulating the arm.

Fracture of the *anatomical neck of the humerus* is rare and occurs chiefly in elderly people. The usual signs of fracture are present. Unless the fragments are impacted the shoulder is flattened and there is a sharp deformity over the anterior aspect of the joint.

Fracture of the *surgical neck of the humerus* affords the usual signs of fracture.

Separation of the upper humeral epiphysis occurs only in young people, never after the twentieth year, and usually between the ninth and seventeenth. The forward and inward displacement of the upper end of the lower fragment which usually attends this condition produces a characteristic angular deformity, the prominence being below the glenoid cavity and at the inner side of the arm. The age of the patient, the characteristic deformity, the soft crepitus, and the fact that the head does not rotate with the shaft indicate the nature of the injury.

If after injury to the shoulder the joint is tender, painful, and swollen, and no positive evidences of fracture or dislocation are present, a sprain must be assumed. With hæmarthrosis the swelling of the joint is soft and doughy, with synovitis fluctuating.

Elbow-joint.—In the examination of the elbow-joint it is important to note the relation of the epicondyles and tip of the olecranon process, the position of the radial head, the carrying angle, and the range of motion.

In the normal joint the two epicondyles and the tip of the olecranon process should be on the same horizontal plane when the forearm is extended; deviations therefrom are due to fractures and dislocations of the articular ends of the bones.

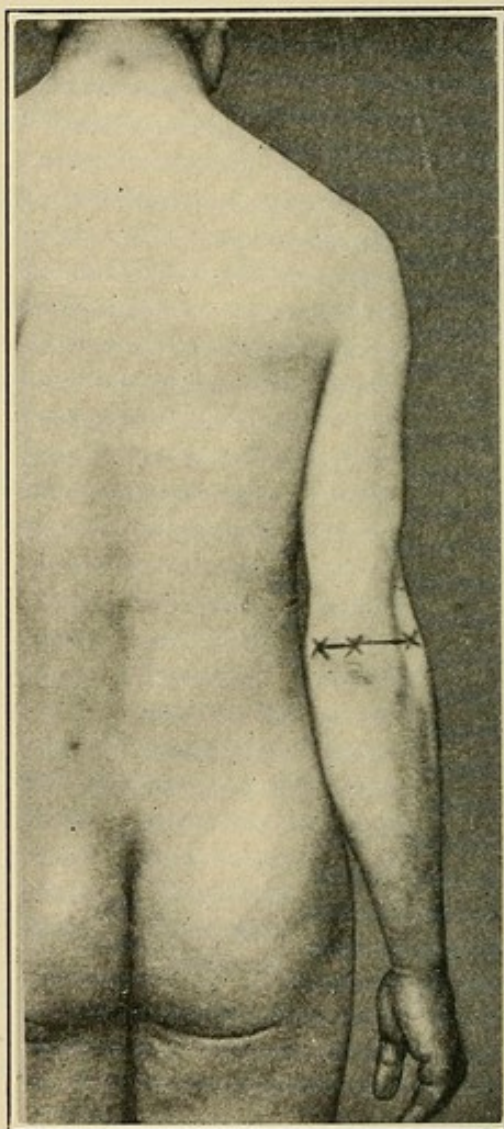
The *head of the radius* should lie below the external condyle of the humerus, and should move with its own shaft in pronation and supination of the hand.

The *carrying angle* is the lateral angle that the supinated

forearm makes with the upper arm. It varies normally within very wide limits and may be entirely absent.

As regards movement, it is important to remember that in the extended position of the forearm no lateral motion is possible.

FIG. 175



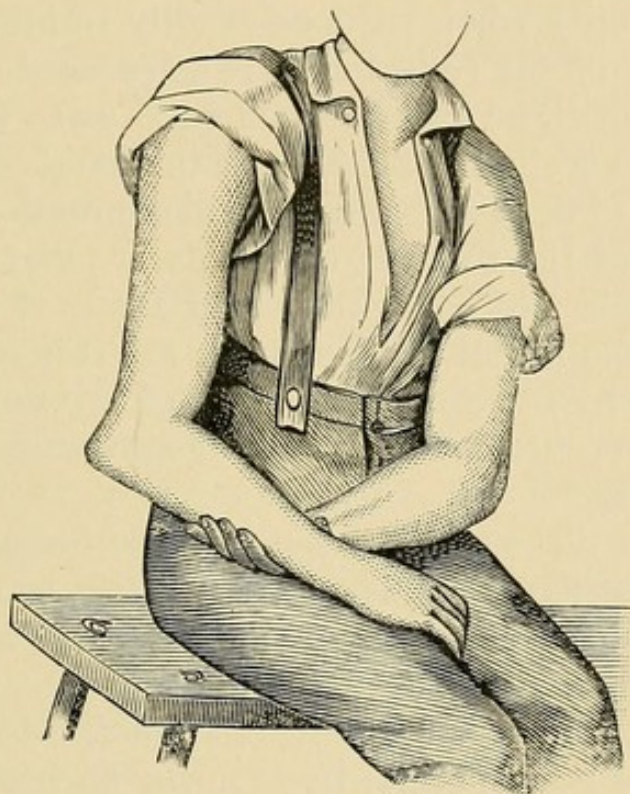
The normal relation of the two epicondyles and the tip of the olecranon process. In the extended position of the forearm these three points should be on the same horizontal plane.

Measurements of the joint are made between the two epicondyles and from the tip of the acromion process to the external epicondyle of the humerus.

Dislocation of *both bones* of the forearm backward, with or without fracture of the coronoid process of the ulna, is characterized by a *disturbance in the relation* of the three

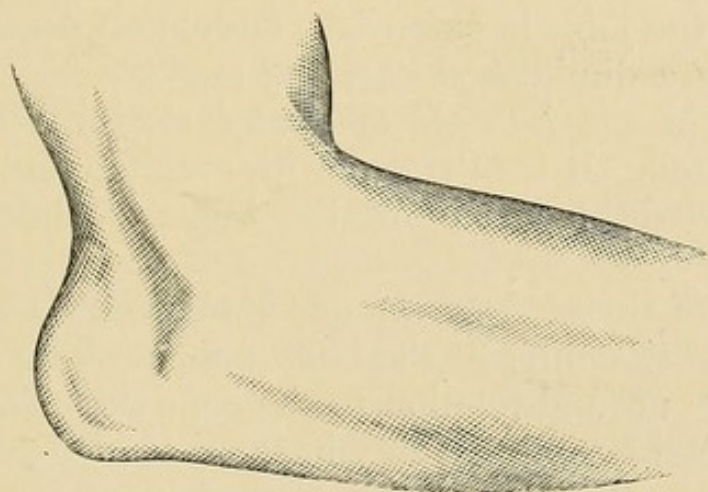
bony points, the olecranon tip being on a higher level than the epicondyles; by *undue prominence of the olecranon*

FIG. 176



Supracondyloid fracture of the humerus. The visible deformity is similar to that in posterior dislocation of both bones of the forearm as represented in Fig. 177. (Hoffa.)

FIG. 177



Posterior dislocation of both bones of the forearm. (Hoffa.)

process behind, by *displacement of the radial head*, and by *the condyles being far in front of the olecranon process*.

The visible deformity occasioned by supracondylar fractures resembles that which is produced by this dislocation; but in these fractures the condyles and olecranon maintain their normal relation, the radial head is in its normal position and the deformity when reduced readily returns.

Subluxation of the radial head is a frequent occurrence in young children. The arm hangs slightly away from the side, the elbow-joint is slightly flexed, and the hand semi-pronated. The extremes of flexion, extension and supination are painful. The relation of the three bony points is preserved, but the radial head does not lie below the condyle of the humerus. Fractures of the radial head or neck are differentiated from the preceding by the normal position of the radial head, the presence of crepitus, and the lack of movement of the head with the shaft.

In *backward* dislocation of the ulna the forearm is fixed in complete extension, the olecranon process is elevated, and the forearm forms an obtuse angle with the arm, the apex being directed away from the median line.

Fracture of the *olecranon* is characterized by disturbed relation of the bony points, by crepitus and mobility of the olecranon process.

Fracture of the *internal condyle* of the humerus is characterized by crepitus, abnormal mobility of the condyle, upward displacement of the internal epicondyle, lateral mobility of the joint in extension, adduction being especially free, and by a diminished carrying angle.

Fracture of the *internal epicondyle* is quite common in young children. It is attended with crepitus and downward and forward displacement of the inner of the three bony points.

Fracture of the *external condyle* is marked by upward displacement of the outer of the bony points. The radial head maintains its normal relation to the condyle. There is, as a rule, abnormal lateral mobility in the extended position, and the transverse diameter of the elbow-joint is increased.

With *supracondylar fracture* there is a marked fulness in front of the joint and a prominence of the point of the elbow behind, just as is the case with backward dislocation of both bones of the forearm. The bony points, however, maintain

their normal relation. There are crepitus and abnormal lateral and anteroposterior mobility above the elbow, and the deformity readily recurs after reduction.

T-shaped fractures are recognized by the crepitus and abnormal mobility which are to be elicited by rocking the condyles upon one another, by the disturbed relation of the bony points (one or both condyles being displaced), by the increased diameter between the condyles, and by abnormal range of abduction and adduction.

Separation of the *lower epiphysis* is fairly common in children under ten. The bony points maintain their normal relation. The diagnosis is made from the age of the patient, the history of an injury to the elbow, the abnormal mobility of the lower end of the humerus, the soft crepitus, the increased width between the condyles, and the abnormal lateral and anteroposterior mobility.

Disturbances in the relation of the *three bony points* may be classified as follows:

The *midpoint* is displaced upward in backward dislocations of both bones of the forearm, or of the ulna alone, and in fractures of the olecranon.

The *outer point* is displaced upward in fractures of the external condyle.

The *inner point* is displaced upward in fractures of the internal condyle, and downward and forward in fractures of the internal epicondyle.

In T-shaped fractures both lateral points are displaced.

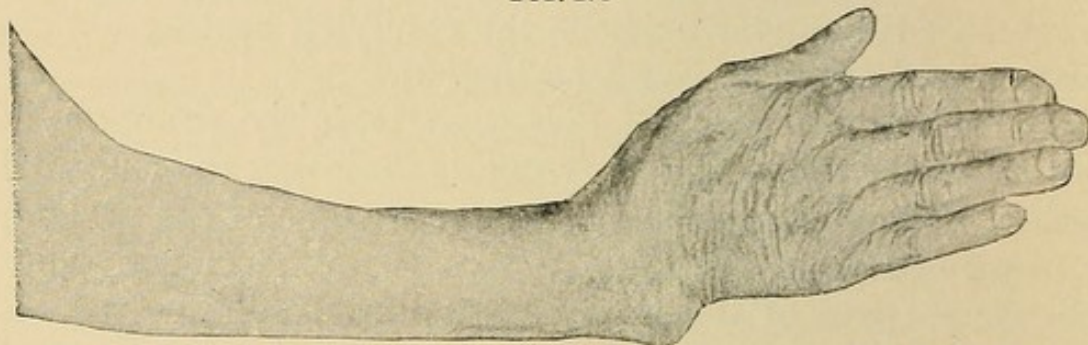
In all other injuries the normal relation of the bony points is maintained.

Wrist-joint.—In the examination it is important to note the contour and shape of the joint, the relation of the styloid processes to each other, the measurement between the two styloids, and the range and direction of active and passive mobility. In the normal joint the tip of the *radial* styloid is lower than the tip of the *ulnar* styloid, and the base of the *thenar* eminence is consequently lower than that of the *hypothelar*.

Fractures of the *lower end of the radius*, which are most of them Colles' fractures, are characterized by a higher relative position of the thenar eminence, as a rule by antero-

posterior and lateral deformities (the wrist presenting the well-known silver-fork deformity—*i. e.*, a flattened prominence in front just above the joint and a corresponding depression behind), by elevation of the radial styloid process, and, unless the fragments are impacted, by crepitus and false point of motion.

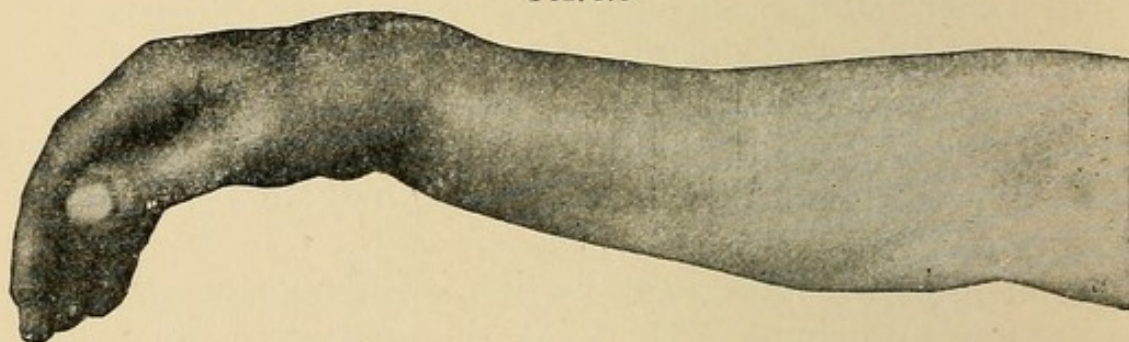
FIG. 178



Marked abduction (bayonet deformity) in Colles' fracture. (Von Bergmann.)

With the *very uncommon dislocation backward* of the carpal bones the styloids maintain their normal relation, but the distance between these processes and a fixed point on the hand is shortened. The displaced carpal bones can be readily felt, and the deformity when reduced does not tend to return.

FIG. 179

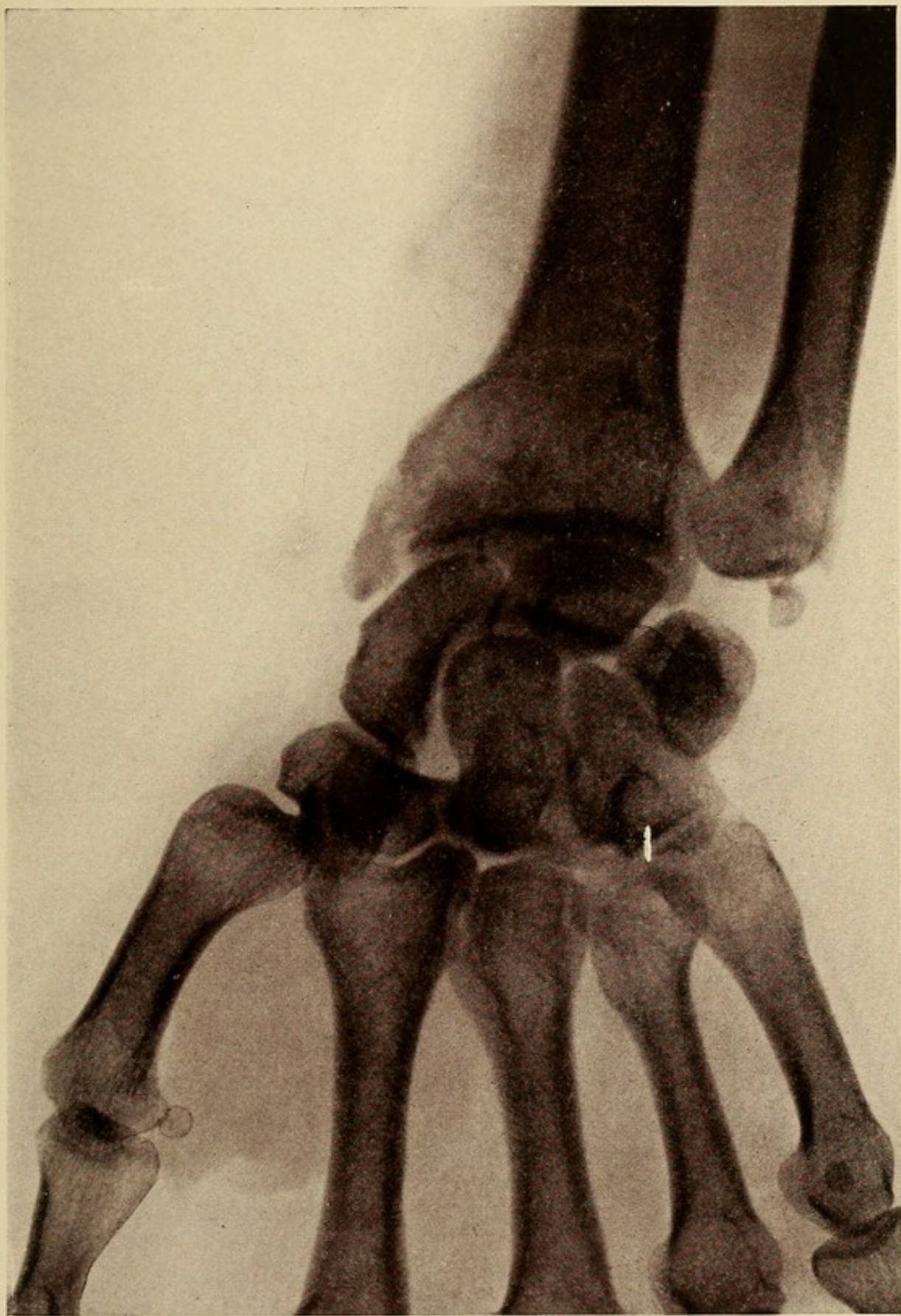


Backward displacement (silver-fork deformity) in Colles' fracture. (Von Bergmann.)

Separation of the *lower radial epiphysis* may be confounded with Colles' fracture, but it is distinguished therefrom by the soft character of the crepitus, the slighter deformity which it occasions, and by its occurrence in young subjects.

Sprains and contusions, though not as common as radial fractures, are recognized by the absence of all the evidences of fracture or dislocation.

PLATE XV.

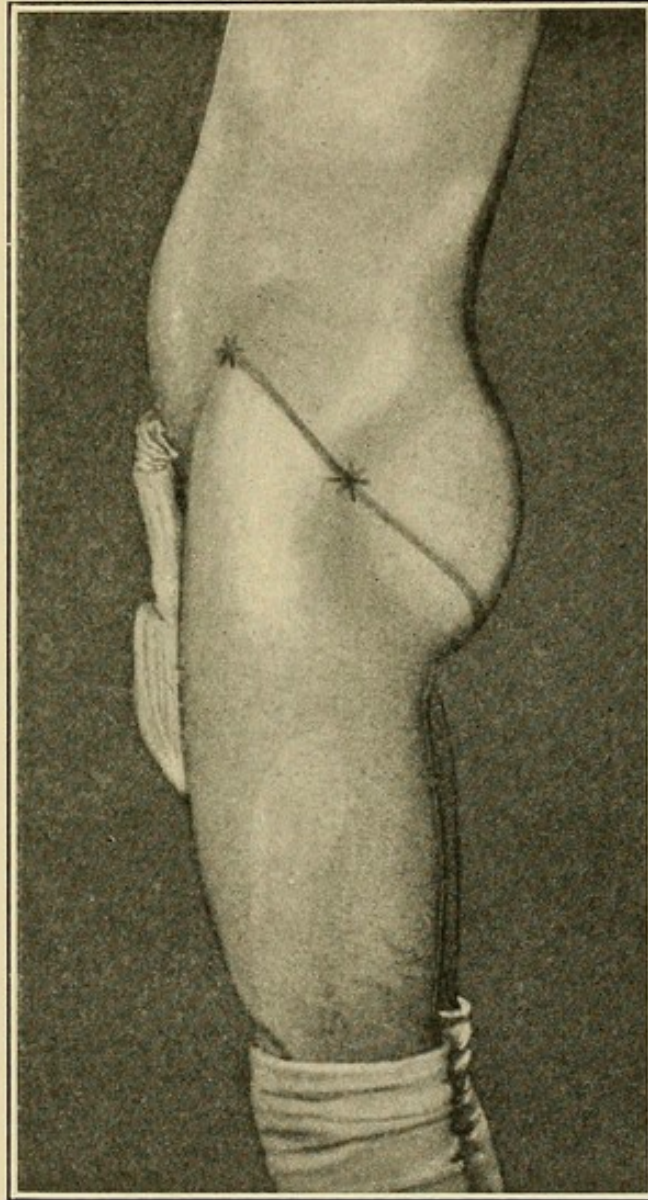


Colles' Fracture of the Radius and its most frequent complication—viz., Fracture of the Tip of the Ulna Styloid, which latter permits of the bayonet deformity. (Fig. 178.)



Hip-joint.—In examining an injured hip notice should be taken of the position of the foot—*i. e.*, whether it is inverted or everted. The length of the limb, the relation of the upper

FIG. 180



Nélaton's line passing from the anterior superior spine along the upper border of the greater trochanter to the lower margin of the ischial tuberosity. If a vertical line is let fall from the anterior superior spine, and then a horizontal line is drawn from the upper border of the great trochanter to meet it, Bryant's iliofemoral triangle will be constructed. The length of the base line of this triangle as compared with the healthy side is important for the recognition of fractures and dislocation of the hip.

border of the great trochanter to Nélaton's line—*i. e.*, a line passing from the anterior superior iliac spine to the lower margin of the tuberosity of the ischium, the length of the Bryant base line, and the distance of the great trochanter

from the median line of the body should all be carefully measured and compared with the sound side. Any marked *difference* in the relations or measurements between the injured and healthy side is due to fracture or dislocation.¹

FIG. 181

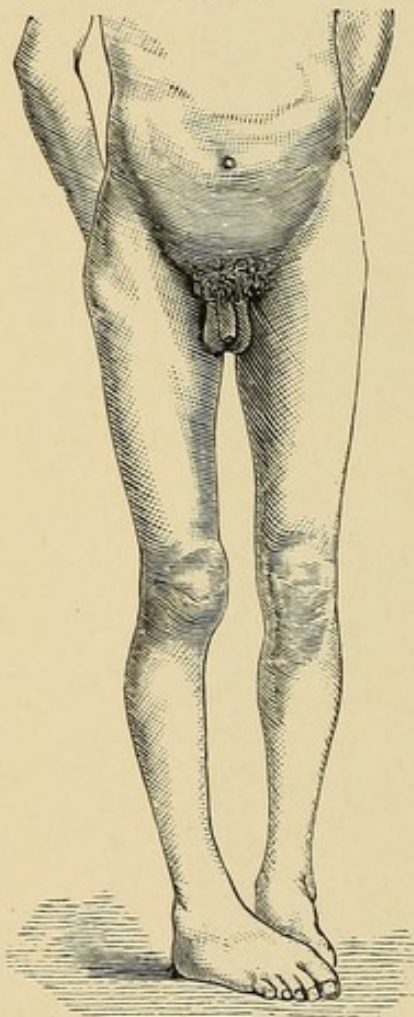


FIG. 182

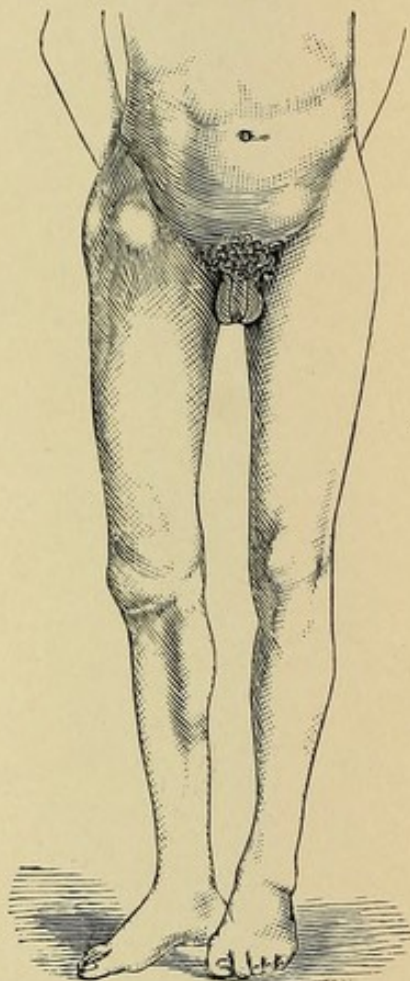


FIG. 181.—Iliac (backward) dislocation. Note position of the foot, and the shortening of the limb. (Bigelow.)

FIG. 182.—Pubic (anterior) dislocation. Note position of the foot, and the prominence over the pubic ramus. (Bigelow.)

Backward dislocations of the femoral head are characterized by inward rotation of the foot, adduction, flexion, and shortening of the limb, and, as a rule, by displacement of the upper trochanteric margin above Nélaton's line.

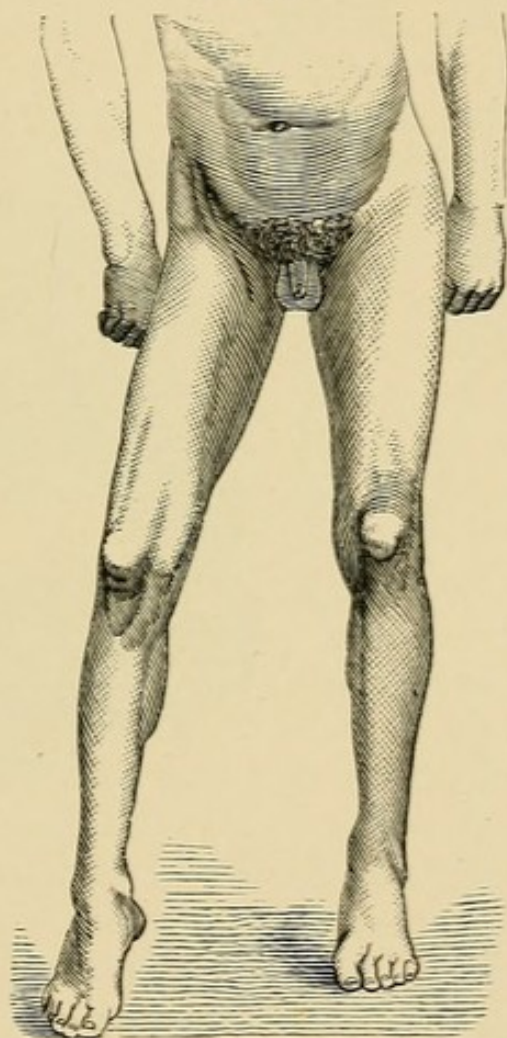
In *anterior pubic dislocations* the foot is rotated outward,

¹ Slight differences in the measurements of the two sides are sometimes present even when the parts are perfectly normal.

the limb is shortened and extended at the hip, and the dislocated head forms a visible or palpable prominence over the horizontal pubic ramus.

In *internal obturator dislocations* there is little apparent shortening and no palpable dislocated head, but corresponding to the normal trochanteric position is a hollow.

FIG. 183



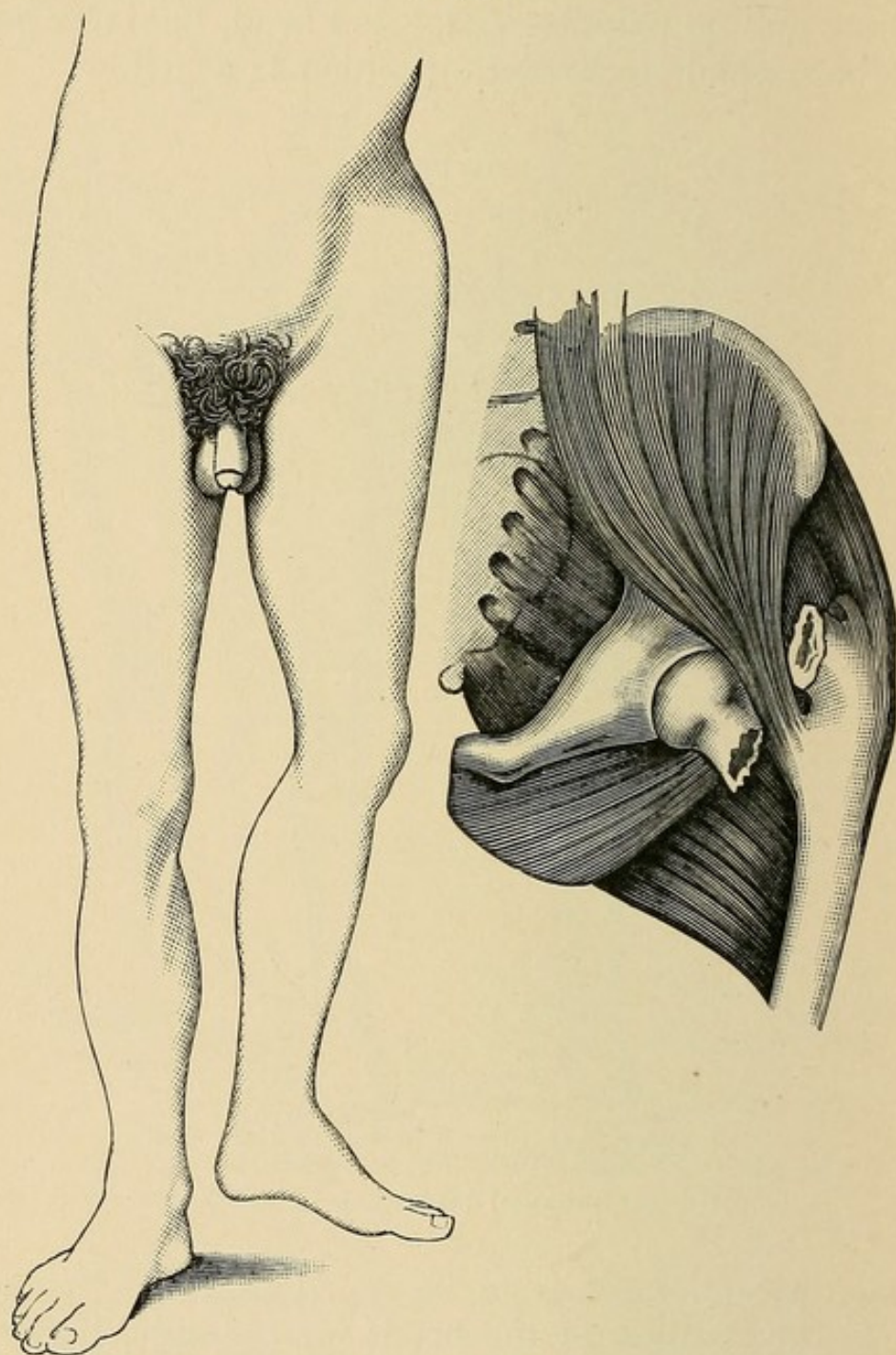
Obturator (internal) dislocation. (Bigelow.)

In all dislocations it is to be noted that the trochanter points to the position of the head; for example, if the trochanter is in front the head must be behind.

Fractures of the *femoral neck* are often attended by the same position of the limb and foot as are backward dislocations of the hip, and are to be distinguished from the latter by their greater frequency of occurrence in elderly subjects, by crepitus, the absence of flattening in the gluteal region,

by the inability to feel the head of the femur in an abnormal position, and by the head not moving with the shaft of the bone.

FIG. 184



Fracture of the femoral neck ; note position of the foot. Compare with Figs. 181, 182 and 183.

Knee-joint.—The superficial situation of the knee-joint renders it easily accessible to palpation. In the examination attention should be directed to the relation of the tibial

tuberosities, the femoral condyles, the fibular head, and the patella; to the patient's ability to raise the extended leg; to the range of active motion in flexion and extension and to the presence of abnormal mobility, whether in flexion, extension, ab-, or adduction, or rotation. It is important to remember in connection with the normal mobility of the joint that extension is possible only to a straight line, and is checked by the lateral and posterior ligaments and the anterior crucial ligament, that flexion is possible only to 135 degrees, and is checked by the forepart of the capsule, ligamentum patellæ and posterior crucial ligaments, that abduction and adduction are not possible at all, and that in the partly flexed position slight rotation inward and outward is possible, the former being checked by the anterior crucial ligament, the latter by the lateral ligaments.

It is most essential to remember these natural ranges of active mobility and the limiting factors thereof, for rupture of the restraining ligaments permits of abnormal mobility of the joint in that direction, in which it is limited by the torn ligament.

Supracondylar femoral fractures likewise permit of abnormal mobility of the leg, but these are readily recognized by the usual evidences of fracture—fulness in the popliteal space, and shortening of the limb.

Fracture of the *patella* and *rupture* of the *quadriceps* and *patellar tendons* result in more or less complete inability to elevate the extended leg. Palpation readily differentiates these conditions, for the line of the patella fracture and the furrow between the ruptured ends of the quadriceps or patella ligaments are easily palpable.

Separation of the *femoral epiphysis* is frequent in children, and is marked by a soft crepitus, by which characteristics it is distinguished from supracondylar fractures, which it otherwise resembles.

Dislocation of the *knee-joint*, a rare occurrence, is readily recognized by palpation. If the swelling is not excessive and the patella does not lie upon and fill up the protruding tibial head the diagnosis can be made by inspection.

Rupture of the *lateral ligaments* permits of abnormal abduction, adduction, and rotation, depending upon whether

the internal or external ligament has been torn. Rupture of *the crucial ligaments* permits of hyperextension and abnormal rotation.

Ankle-joint.—In the examination note should be taken of the integrity of the malleoli and of the fibula above the tibiofibular joint, of the relation of the malleoli to the astragalus, and of the function and position of the foot.

Fractures of the fibula anywhere between the tip of the malleolus and three inches above it afford the usual evidences of broken bones. Should there be a coincident rupture of the internal lateral and inferior tibiofibular ligaments, or a fracture of the internal malleolus, an abnormal mobility of the foot, and in some cases a backward displacement of the foot, results. In the latter instance the foot is shorter (as measured from the front of the ankle to the cleft between the first and second toes) and everted.

Simple dislocations of the ankle are rare, but are readily recognized, the tibia being either in front of or behind the astragalus. In the former instance the foot is in plantar flexion, the heel is prominent, the Achilles tendon concave posteriorly, and the anterior aspect of the foot is shortened. In the latter the foot is in dorsal flexion, the heel disappears, and the anterior aspect of the foot is lengthened.

CHAPTER L.

ACUTE INFLAMMATIONS OF JOINTS.

THE same exciting causes of joint inflammation at one time attack the synovial membrane alone, with resulting acute or chronic synovitis, and at another time all the structures of the joint with resulting acute or chronic panarthrititis.

The acute inflammations are distinguished from the chronic ones by the presence of more or less severe constitutional disturbances—*i. e.*, high fever, rapid pulse, and increased leukocyte count. It is to be remembered that an acute inflammatory condition may subside into a chronic one. The participation of all the joint structures in an inflammatory process readily distinguishes a panarthrititis from a synovitis.

Acute Synovitis.—The acute forms of synovitis are due to traumatism, rheumatism, gout, pyæmia, gonorrhœa, and the acute infectious diseases, and are characterized by a moderately severe grade of constitutional symptoms, and by a distention of the capsule with serum, or seropus, or pus. The severity of the constitutional symptoms depends entirely on the grade and severity of the synovial inflammation. The joint is painful and tender; if it is a superficial one the overlying skin is hot, and the limb is usually held in slight flexion. The muscles may undergo atrophy. The evidences of capsular distention have been described on page 457.

The nature and cause of the synovitis must be ascertained from the history and from evidences of disease in other organs.

Thus in rheumatism there are profuse acid sweats, highly colored urine, and multiple joint involvement. In gonorrhœa the joint symptoms generally commence after the third week of the urethritis, though they occasionally develop during the

chronic stage of the disease; one or many joints may be involved. In *pyæmia* there is a primary focus for the systemic infection, though cryptogenetic forms of infection do occur; the constitutional symptoms are more severe and the exudate is purulent. With *traumatic synovitis* the history of an injury is always present.

The chronic forms of synovitis are either the remains of an acute inflammation or they are chronic from the outset, in which case they are for the most part due to typhoid, or syphilis, or tuberculosis. The constitutional manifestations are very mild or altogether absent. The capsule becomes distended with serous or seropurulent or purulent fluid, the synovial membrane becomes thickened, its fringes at times considerably hypertrophied, and motion of the joint often elicits crepitation. Usually there is no pain nor is there much restriction of motion; the chief complaint is that the limb becomes easily tired and does not feel as strong as before. In some instances the exudate is very slight, but the thickening of the synovial membrane and its fringes is very much in evidence, causing pain and marked crepitation. As in the acute forms of the disease, the diagnosis of the exciting cause of the chronic synovitis must be determined from the anamnesis, from the physical examination and by a process of exclusion. The *tuberculous forms* of the disease are characterized by their persistence in spite of the usual methods of treatment, by the greater amount of synovial thickening, the greater frequency of crepitation, and a yellowish, flocculent exudate.

Acute Arthritides.—The acute arthritides are nearly always due to infection of the joint with bacteria, the infection taking place through open wounds or from perforation of a neighboring suppurating focus into the joint, or by the medium of the blood stream. They are thus for the most part secondary lesions and accordingly are found to develop after penetrating wounds of the joints, with acute infectious osteomyelitis or epiphysitis, with pyæmia, typhoid fever, pneumonia, the acute infectious diseases, and gonorrhœa.

The constitutional symptoms attending these acute arthritides are usually severe; the temperature is high, the pulse rapid, the leukocyte count high, and the patients look and

feel very sick. The joint is painful and swollen, the skin red and hot, the periarticular structures infiltrated and thickened, the capsule distended with seropus or pus, the articular ends of the bones and the ligaments tender, active and passive motion exceedingly painful and the limb is held in a more or less flexed position.

The cause of the disease is determined from the clinical history, the presence of clinical evidences of disease in other organs—*e. g.*, acute osteomyelitis, pneumonia, acute infectious diseases, gonorrhœa, etc. It is to be especially noted that a joint inflammation may be the first objective sign of an acute infectious osteomyelitis, and consequently in young children in whom no other cause for the acute arthritis can be discovered such acute infection of the bone-marrow or epiphysis should be suspected and looked for.

In some of the arthritides following acute osteomyelitis the course is subacute, the constitutional symptoms being considerably less severe, the joint inflammation less intense, and the exudate within the joint of a ropy, viscid character. These cases belong to the class described by Volkmann as "catarrhal synovitis."

Joint exudates and periarticular effusions of a serous, seropurulent, or purulent character sometimes accompany severe forms of osteomyelitis and periarthritides, even though no real joint inflammation exists. Such collateral exudates are similar to the exudates which form in the soft parts around a virulent focus of infection. If they are purulent in character it may be very difficult to differentiate them from those which are due to a true arthritis. When the joint is opened the synovial membrane is seen to be bright and shiny and not coated with fibrin, and spreads made from the exudate show no bacteria. All such exudates disappear spontaneously when the primary focus of disease is incised and drained.

An arthritis and a collateral exudate into a joint secondary to acute infectious osteomyelitis are sometimes mistaken for rheumatism, and especially is this likely to happen when the bone disease gives no physical evidences of its presence. In favor of osteomyelitis are the presence of pain and tenderness at the articular ends of the bones, a periarticular exudate,

and the frequency of this disease in children in whom rheumatism is comparatively infrequent. The tendency for rheumatism to affect a number of joints also distinguishes this malady from the acute arthritis secondary to bone disease.

Gouty and Rheumatic Arthritides.—Gouty and rheumatic arthritides are special forms of acute joint inflammation and do not end in suppuration. The gouty attacks frequently commence in the middle of the night, the metatarsophalangeal joint of the big toe and the metacarpophalangeal joint of the thumb being the favorite sites of the disease. The joints become swollen and acutely painful; the overlying skin is red, shiny, and œdematous. The symptoms pass off in a few days, but the attacks recur from time to time. Each one leaves a slight deposit of biurate of soda in the articular cartilage, ligaments, and ends of the bones, which finally forms well-marked swellings (tophi) around the joint. Similar deposits are found in the external ear and other cartilaginous structures.

CHAPTER LI.

CHRONIC DISEASES OF THE JOINTS.

TUBERCULOSIS OF THE JOINTS.

THE cure of tuberculous arthritis, whether by the bloody or bloodless methods of treatment, will be the more probable the earlier the diagnosis is made. With the truth of this statement acknowledged by all, it becomes the duty of every practitioner to carefully study and examine his cases of joint disease so as to be able at the earliest possible opportunity to institute such curative methods as are most applicable. It must not be imagined, however, that such early diagnosis is easy, for in its initial stages tuberculous arthritis is very often attended by few symptoms, and these are elicited only after careful examination.

The majority of the patients with this ailment are children who have a tendency to hide their malady rather than expose it, and only by careful observation of their previous and present actions and habits can a clue be obtained that something is wrong. Such careful observation, combined with repeated, painstaking physical and *x*-ray examination, will, in most cases, enable us to make an early diagnosis of tuberculous joint disease.

If the patient is a child the mother will, as a rule, attribute its slight limp or the fact that it easily tires at play, or refuses to use its arm as before, to an injury; a little cross-questioning, however, will soon reveal the fact that the supposed traumatism was very slight and was only thought of when the child commenced to complain. The patient, although previously a sound sleeper, is now said to wake up at night with a cry and then fall asleep again. Pain may be complained of in the affected joint or it may be referred to another joint—*e. g.*, to the knee in hip-joint disease.

If the individual is examined at this stage there may be no objective signs. This should not lead the examiner to dismiss the case as one of minor or insignificant importance, but should prompt him the more to make repeated examinations. Sooner or later *pathognomonic* signs of joint disease are manifested. Such evidences are, firstly, a restriction in the *free passive motion* of the joint in some or all of the directions in which it normally moves, such restricted movement being associated with *reflex muscular spasm*; and, secondly, the abnormal position in which the joint is held when at rest. The *x-ray* may show changes in the ossification nucleus of the epiphysis and in the osteocartilaginous junctions of the articular ends, for which see pp. 463-465.

These early objective evidences of disease are, as stated above, pathognomonic, and their presence should be repeatedly sought for in patients presenting the subjective symptoms detailed above.

As the disease progresses the objective manifestations become more numerous and evident. The joint if a superficial one becomes swollen, its contour obliterated, its shape fusiform, the periarticular structures and capsule thickened and infiltrated, and the articular ends of the bone enlarged. The skin becomes stretched, pale and waxy, the cutaneous veins distended and enlarged, motion more restricted, and *reflex* muscular spasm more marked; the limb is held in a vicious position and the pain is more intense. The general health also commences to suffer; the pain interferes with sleep, and the progressing tuberculous infection causes anæmia, loss of appetite, and loss of weight.

If the malady continues to advance the tuberculous tissue in the bone and within the joint undergoes caseation, breaks through the periosteum and capsule into the soft parts, and gives rise to cold abscesses which perforate through the skin in the neighborhood of the joint, or burrow along the muscular planes and perforate through the skin at a distance from the original site of the disease. The destruction of the joint structures leads to dislocation. If now a mixed pyogenic infection through the fistulous opening occurs profuse suppuration results. The general health suffers materially; the constant discharge and fever sap the strength, the pain inter-

feres with sleep, and the appetite, digestion, and assimilation are bad.

Differential Diagnosis.—*The initial symptoms of tuberculous arthritis bear some similarity to the pains that attend growth and to the manifestations of hysterical joints. Growing pains, by some attributed to very mild forms of osteomyelitis, are usually in the shafts of the long bones, more rarely in the joints. They disappear with rest in bed, and are not associated with restricted joint movement or reflex muscular spasm.*

Hysterical and neuralgic joint pains are characterized by their inconstancy, now better, now worse, now here, now there, and by their association with other hysterical symptoms. Though the joints may be held in vicious positions that resemble those which result from real disease, even muscular atrophy from disuse being associated with such malpositions, the physical examination reveals no abnormalities and in narcosis there is no restriction of joint movement and no reflex muscular spasm. The physical sufferings are entirely disproportionate to the actual findings, and with massage and general hygienic and electric treatment the neuralgic and hysterical pains as a rule subside entirely.

In the stage of effusion with swelling of the joint and periarthrititis, tuberculous arthritis bears some resemblance to monarticular rheumatism, to acute and chronic synovitis, to gonorrhœal and syphilitic arthritis, to the arthritis which is secondary to acute osteomyelitis and to malignant disease of the articular ends of the bones.

Monarticular rheumatism is distinguished by its tendency to repeated recurrence, and by the increased severity of the pain in wet weather. Joint crepitus, greater pain, and stiffness characterize the rheumatic affections, which furthermore are favorably influenced by antirheumatic remedies.

Chronic synovitis is not attended with as much synovial thickening nor with the periarthrititis.

Acute synovitis is always secondary to a constitutional disease—e. g., rheumatism, gout, infectious diseases, septicæmia, osteomyelitis, etc. It is of sudden onset in a previously healthy joint, whereas the acute form of tuberculous synovitis, which is due to the perforation of a tuberculous bone focus

into the joint cavity, is preceded by symptoms which are very suggestive of bony tuberculosis—*e. g.*, pain, limping, easy exhaustion, etc.

The acute initial stages of *gonorrhæal arthritis* are readily distinguished from the chronic manifestations of tuberculous joints. The ankylosis to which it gives rise in the later stages differs from the ankylosis in tuberculous disease by its greater firmness. Such joint lesions usually follow directly upon an acute gonorrhœa or occur during its chronic stage.

Syphilitic arthritis is likewise distinguished by the intensity of the pain which attends it, and the firmness of the ankylosis to which it gives rise. The subjective symptoms are relieved by antisyphilitic treatment.

The arthritides which follow the acute infectious diseases result in ankylosis, which may be falsely interpreted as due to tuberculosis; the anamnesis affords the basis for the differentiation.

The severe initial period of acute osteomyelitis and epiphysitis readily differentiates the joint affections to which they give rise from those which are due to tuberculosis. If the acute process subsides, leaving a thickened joint with peri-arthritis, and especially if sinuses form and lead down to bare bone, tuberculosis may be suspected; the anamnesis in such cases is more valuable for differentiation than are the physical findings.

The pain and enlargement of joints due to *arthritis deformans* may give one the impression of tuberculosis. The intensity of the pain, the rigidity, the cartilaginous and fibrous thickening, the multiple character of the lesion, the more chronic course and the non-suppurative character of arthritis deformans will serve to distinguish these joint affections from those due to tuberculosis.

Sarcoma and carcinoma of the articular ends of the bones may bear some resemblance to tuberculosis. If there is a primary carcinomatous neoplasm in some other organ the diagnosis of the secondary bone deposit is readily made. In primary sarcoma of the bones it is well to remember that these growths occur later in life, whereas tuberculosis is more frequent in childhood. Primary periosteal neoplasms grow more rapidly and to larger dimensions than do tuberculous

lesions; they infiltrate the surrounding tissues and more often give rise to secondary deposits in the glands and internal organs. Primary medullary sarcomata expand and erode the bone and often first signify their presence by spontaneous fracture.

Injuries to the joints resulting in fracture of the articular ends of the bones, or epiphyseal separation, may simulate tuberculous disease thereof. A little cross-questioning will elicit the history of a severe injury in the former case, which, with an entire absence of all other evidences pointing to tuberculous disease, should readily enable us to make the diagnosis.

Enlarged and inflamed bursæ or hernial protrusions of the synovial membrane through weak spots in the fibrous capsule may cause swelling of the joint and vicious positions thereof that harmonize somewhat with those due to joint tuberculosis. These bursæ sometimes communicate with the joint, and when inflamed the process may extend from them into the joint cavity, or, *vice versa*, the disease may extend into the bursæ from the joint cavity. If the bursæ alone are diseased, the position of the swelling to which they give rise at once distinguishes them from joint disease *per se* (see p. 457); but if the joint is involved coincidently with its surrounding bursæ, and if there is much periarthrititis, the enlarged bursæ will be recognized with considerable difficulty.

CLINICAL AND DIAGNOSTIC FEATURES OF TUBERCULOUS DISEASE OF THE SPECIAL JOINTS.

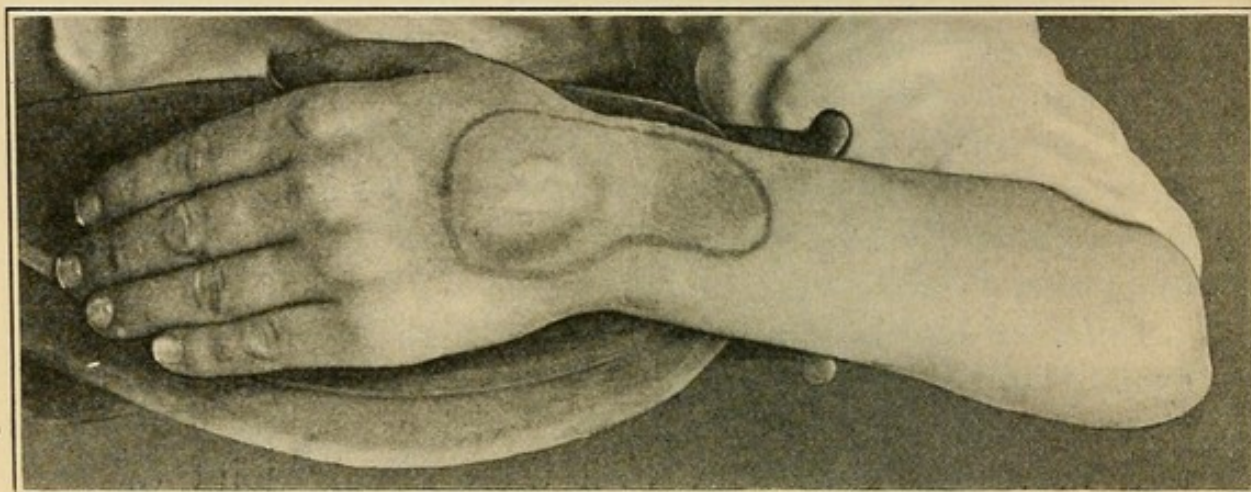
Shoulder-joint.—This is rarely affected in children and but seldom in adults.

Clinically it manifests itself either as a dry caries of the bones, the humerus being primarily involved, or as a fungus degeneration of the joint structures. The former type of the disease is characterized by intense pain, which in the absence of definite physical abnormalities may lead one to the erroneous diagnosis of joint neurosis or joint rheumatism. The fungus forms of the disease may simulate and must be differentiated from neoplasms of the articular ends of the bones.

Elbow-joint.—Elbow-joint tuberculosis is most commonly met with in young adults.

Wrist-joint.—Wrist-joint tuberculosis is most frequent in young adult and middle life. The disease usually commences

FIG. 185

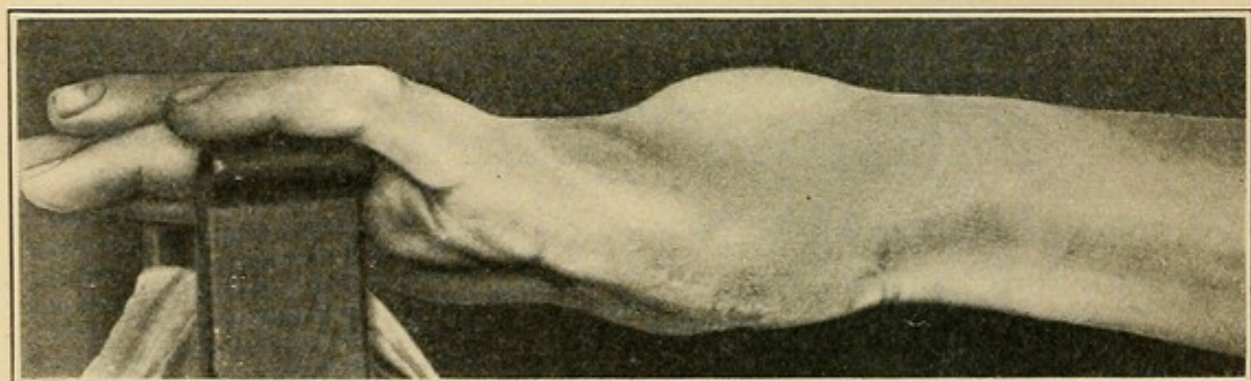


Tuberculous tenosynovitis around wrist-joint. Note the figure-of-8 shape.

with disturbed function of the joint or a slowly increasing exudation into the joint cavity, to which atrophy of the arm muscles succeeds.

Tuberculous tenosynovitis may be mistaken on superficial examination for wrist-joint disease, an error that should be

FIG. 186

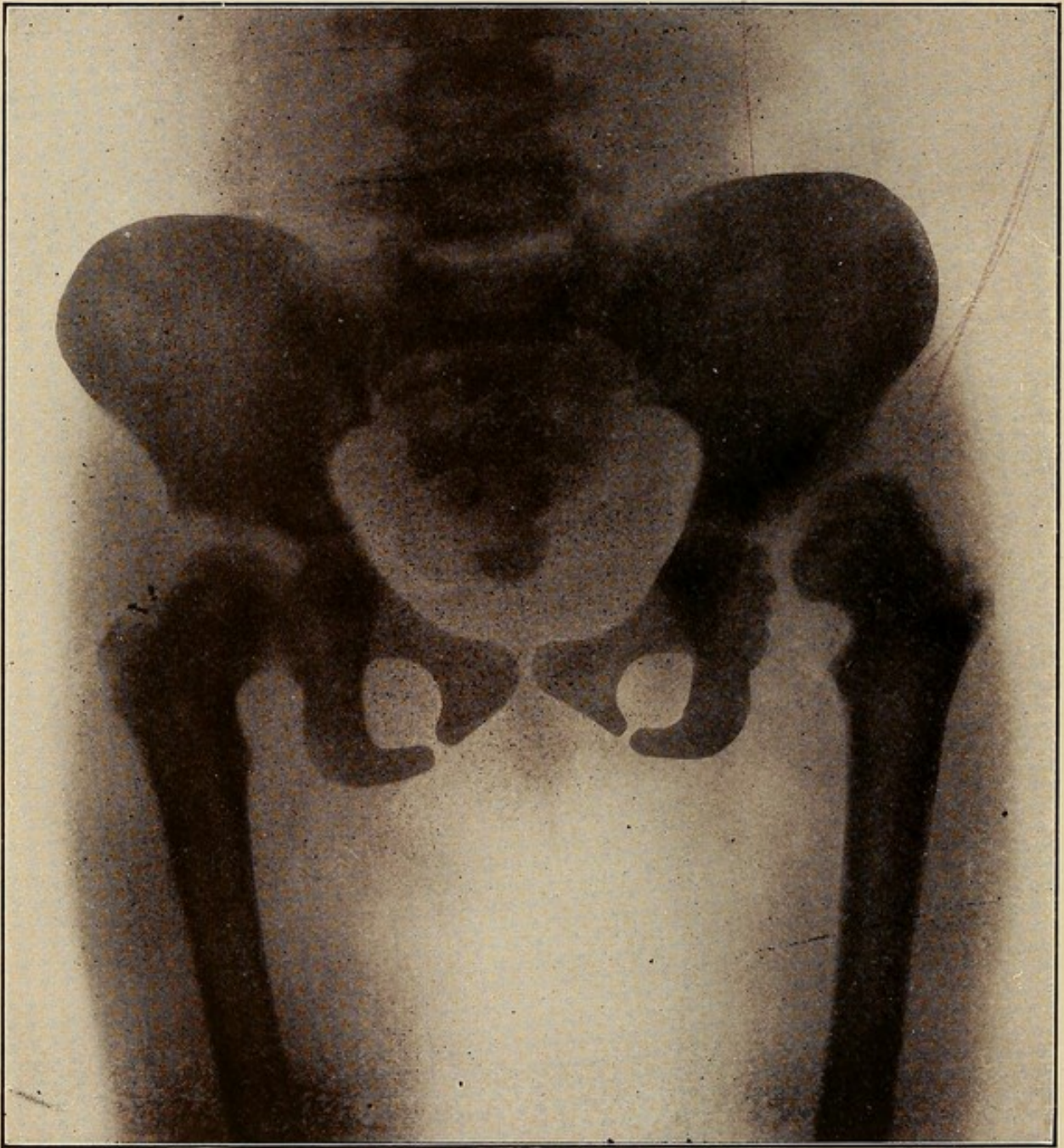


Tuberculous tenosynovitis around the wrist, seen in profile. Note that the swelling is not spindle-shaped nor limited to the wrist-joint, as is the case of tuberculosis of the wrist-joint.

easily avoided with careful examination and a little reflection. For the swelling which is due to a tenosynovitis is strictly limited to the course of the affected tendon sheaths, and usually has a figure-of-8 shape, the constricted portion



PLATE XVI.



Early Stage of the Disease of the Left Hip-joint (to the right in the picture) of the Synovial Type, showing Irregularity in the Shape of the Acetabulum. (Whitman.)

corresponding to the annular ligament, beneath which the tendons pass. The swelling in wrist-joint disease, on the other hand, is limited to the wrist-joint and has a spindle shape.

The frequent occurrence of rice bodies in tendon-sheath tuberculosis, and the fact that motion in this malady is restricted only in those directions in which the affected tendons are put on the stretch, and that such limitation of motion is not accompanied by reflex muscular spasm further distinguish this disease from wrist-joint tuberculosis.

Hip-joint.—Hip-joint tuberculosis is especially frequent in children. Its clinical course varies but little from that which has been pictured above as characteristic of tuberculous joint disease. During the early stages of the malady the affected limb appears to be lengthened, and is usually held in a position of flexion, abduction, and eversion, more rarely in adduction, flexion, and inward rotation. The flexion and abduction are, however, not always in evidence; they may be and usually are masked by a forward curvature of the spine (lordosis) and by tilting of the pelvis, the latter being the cause of the apparent lengthening. By placing the patient flat upon his back with his spine absolutely horizontal the deception is unmasked, for the diseased limb at once flexes at the hip-joint. Similarly, if while the patient is flat and horizontal a straight rod is laid across the anterior superior iliac spines, the tilting of the pelvis is at once demonstrated, for the rod does not lie at right angles with the median line of the body as it should; it crosses it obliquely and its deflection downward on the diseased side indicates the angle of abduction.

In the *later stages* of the disease the position in which the limb is held changes to adduction, flexion, and inversion; the pelvis is tilted upward on the diseased side, causing apparent shortening of the limb. A lateral spinal curvature with the convexity to the sound side in the lumbar region follows upon the abnormal position of the limb.

When *cold abscesses* form they point most frequently in front of and internal to the great trochanter; sometimes they point in the gluteal region or below Poupart's ligament to the inner side of the femoral vessels. Sometimes the tuber-

culous process involves the psoas bursa, and the caseation of the latter gives rise to an abscess in the lower part of Scarpa's

FIG. 187

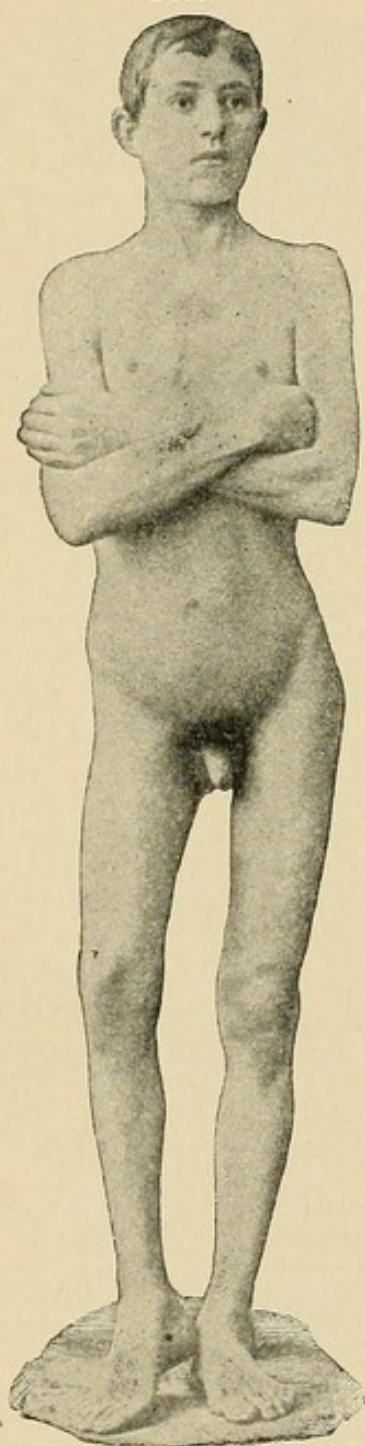


FIG. 188

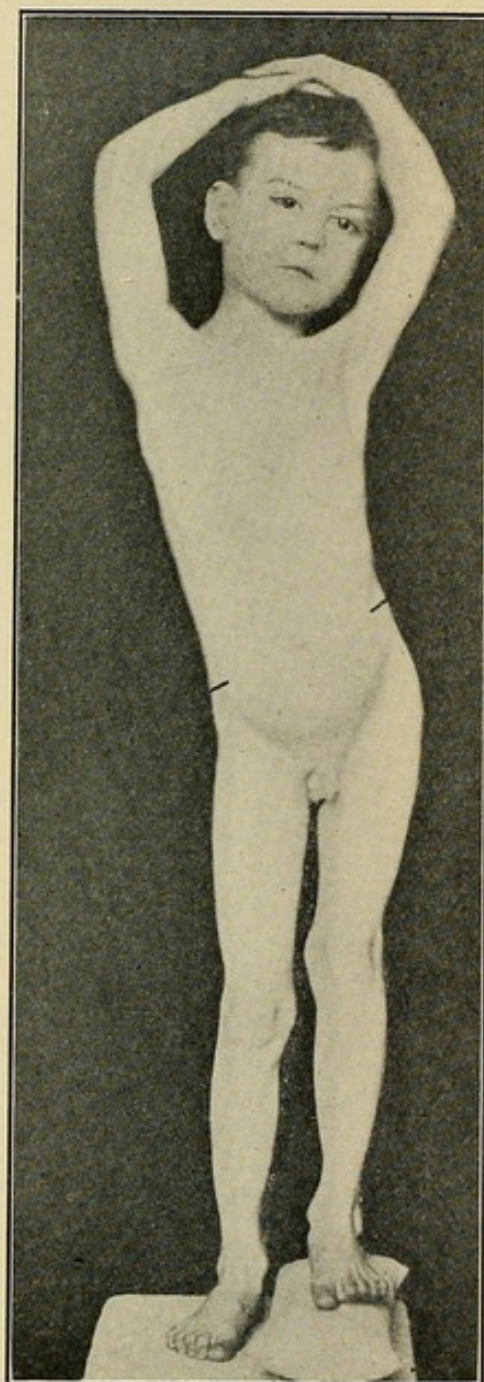


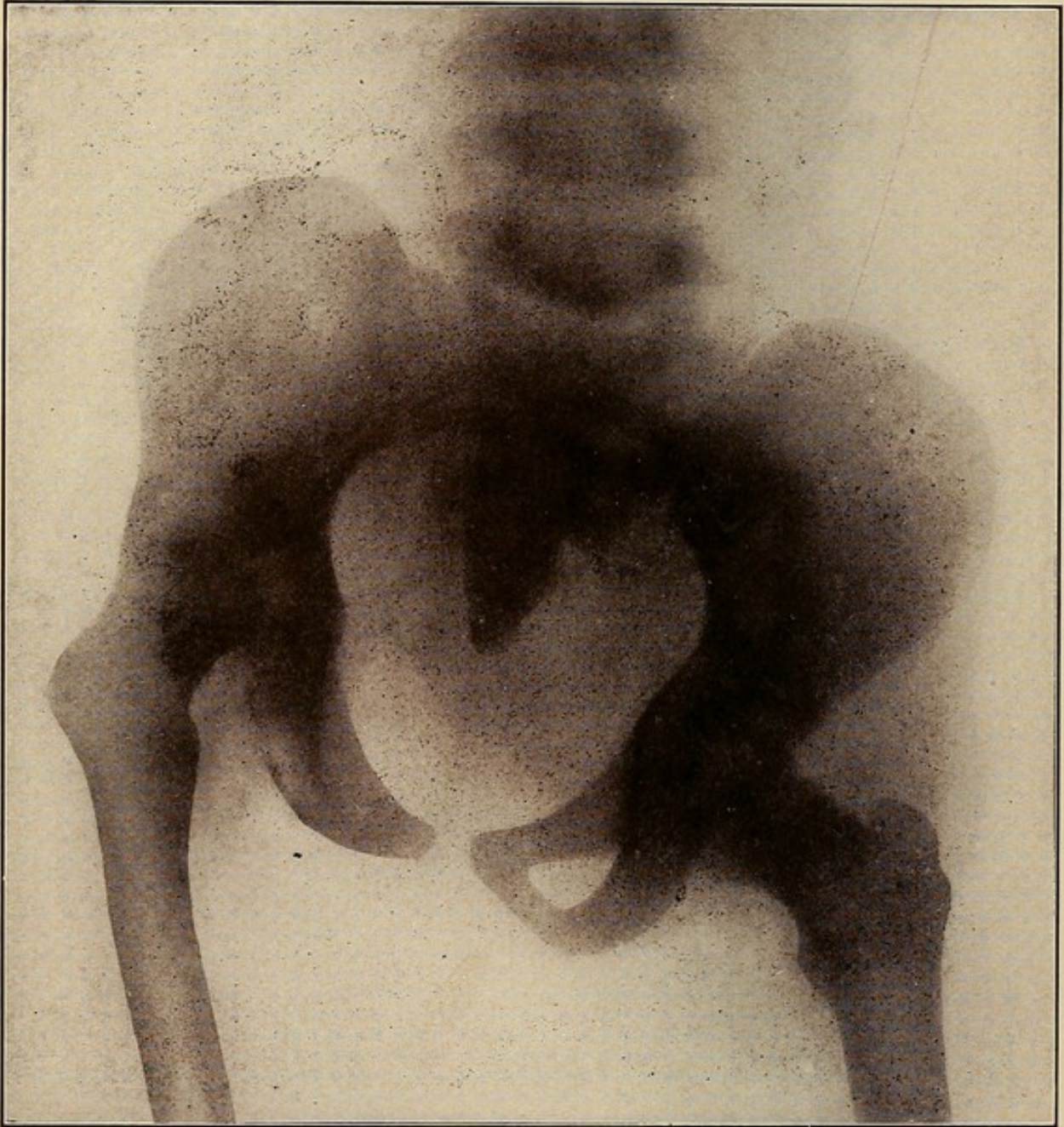
FIG. 187.—Coxitis in the stage of flexion, abduction, and outward rotation. (Von Bergmann.)

FIG. 188.—Apparent lengthening. When the distorted limb is brought to the median line the pelvis is so tilted that the abducted leg seems longer. (Whitman.)

triangle, or in the psoas muscle. If the acetabulum is perforated by the tuberculous process a pelvic abscess develops.



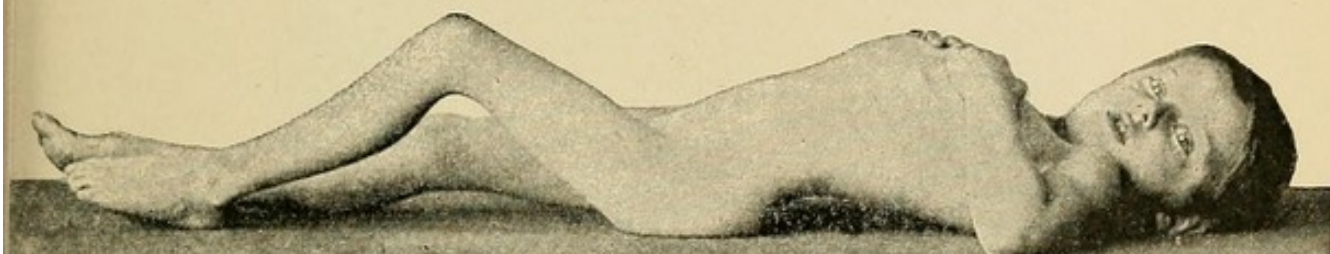
PLATE XVII.



Advanced Disease, showing Wandering of Acetabulum and the Obliquity of the Pelvis due to Adduction. Actual shortening one inch, apparent shortening three inches. (Whitman.)

In the *final stages* of the disease the head of the femur, the posterior margin of the acetabular cavity, and the joint ligaments have been eroded and destroyed, permitting a backward dislocation of the femoral head and causing a *real*

FIG. 189

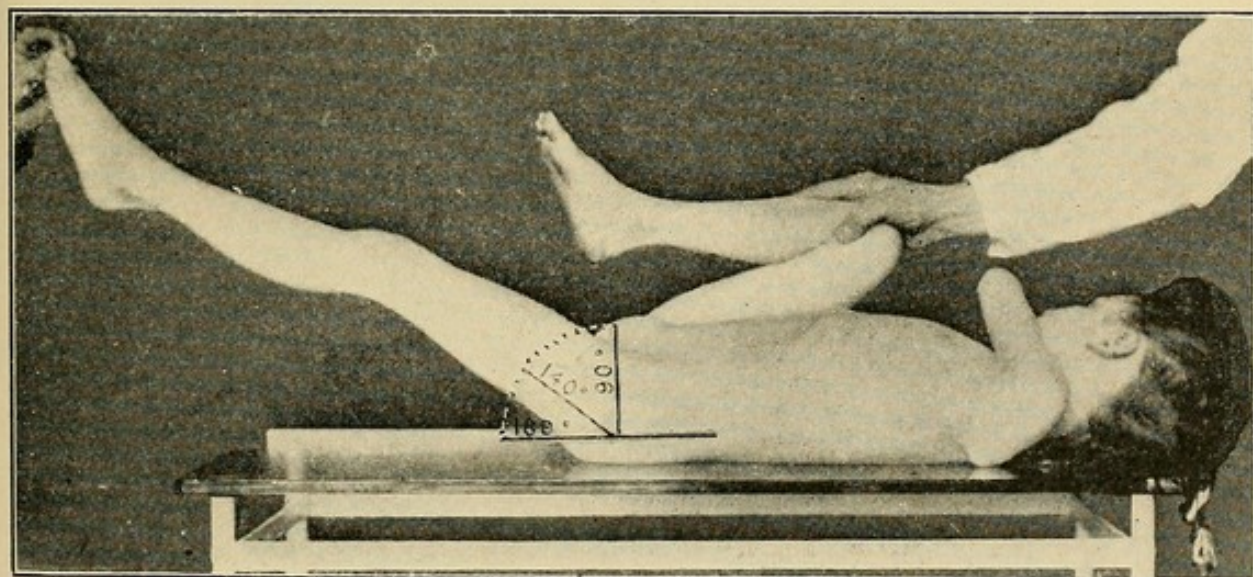


Flexion of the hip, lordosis of the lumbar vertebræ. (Von Bergmann.)

shortening, the limb being adducted, flexed, and rotated inward.

Some difficulty may be experienced in differentiating tuberculous spondylitis from tuberculous coxitis, for the cold abscesses which result from the former may point and open

FIG. 190

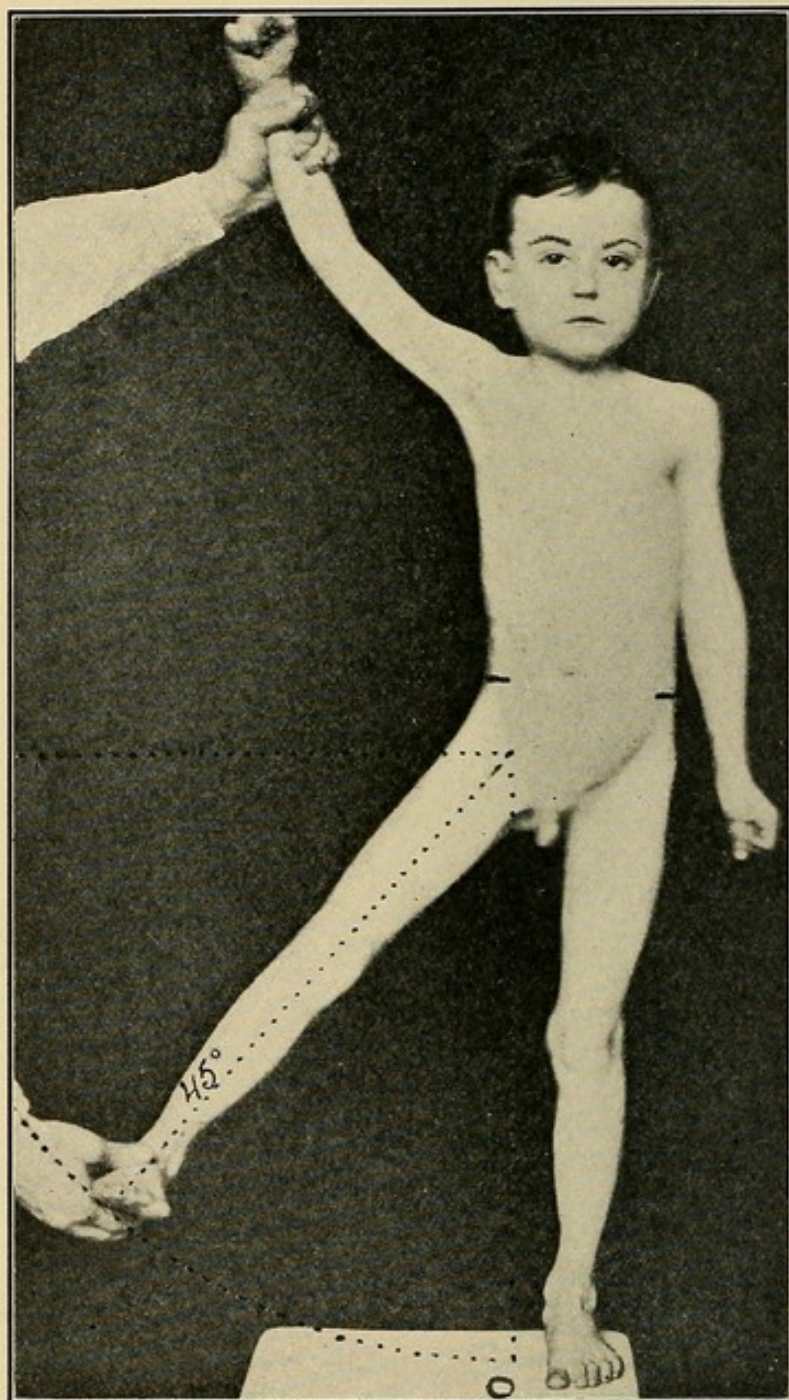


The degree of flexion is shown when the lumbar spine is held in contact with the table by flexing the other thigh. (Whitman.)

in the same regions as those resulting from coxitis, and the reflex spasm of the ileopsoas muscle which frequently accompanies vertebral disease occasions the same vicious position of the lower extremity—viz., flexion, abduction, and outward

rotation. Examination, however, will readily clear up the doubt in the diagnosis, for with spondylitis there is tenderness over the vertebral spines or transverse processes, and usually

FIG. 191



Showing how to determine degree of abduction. (Whitman.)

some deformity (kyphosis, or lordosis, or lateral curvature) with rigidity of the spine; the movements of the hip-joint, on the other hand, are free, and there is no reflex muscular

spasm excited during its passive motion. The *x*-ray will give further evidence for differential diagnosis.

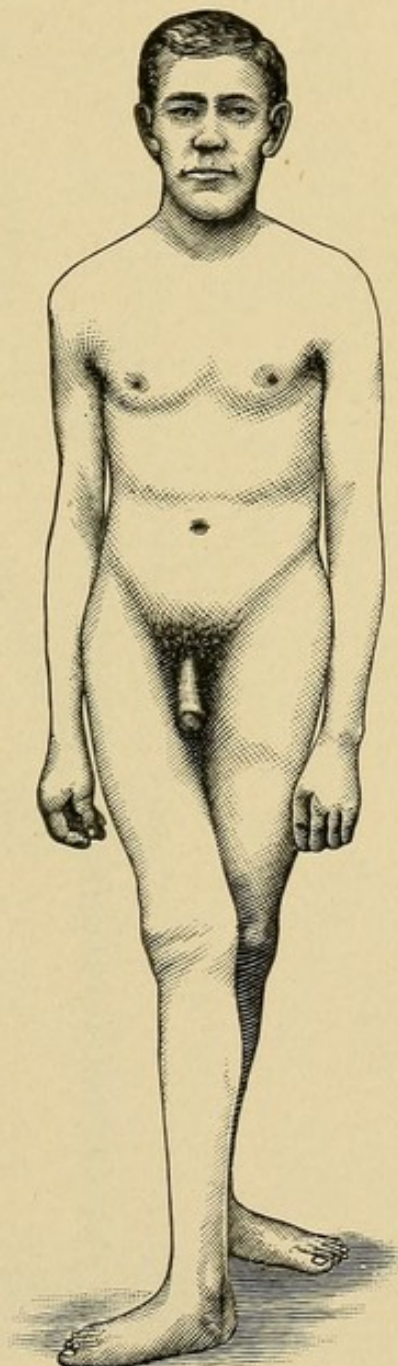
The pathological dislocation of the *femoral head* attending the advanced stages of hip-joint disease may possibly cause

FIG. 192



Coxitis in the stage of flexion, adduction, and inward rotation. (Von Bergmann.)

FIG. 193

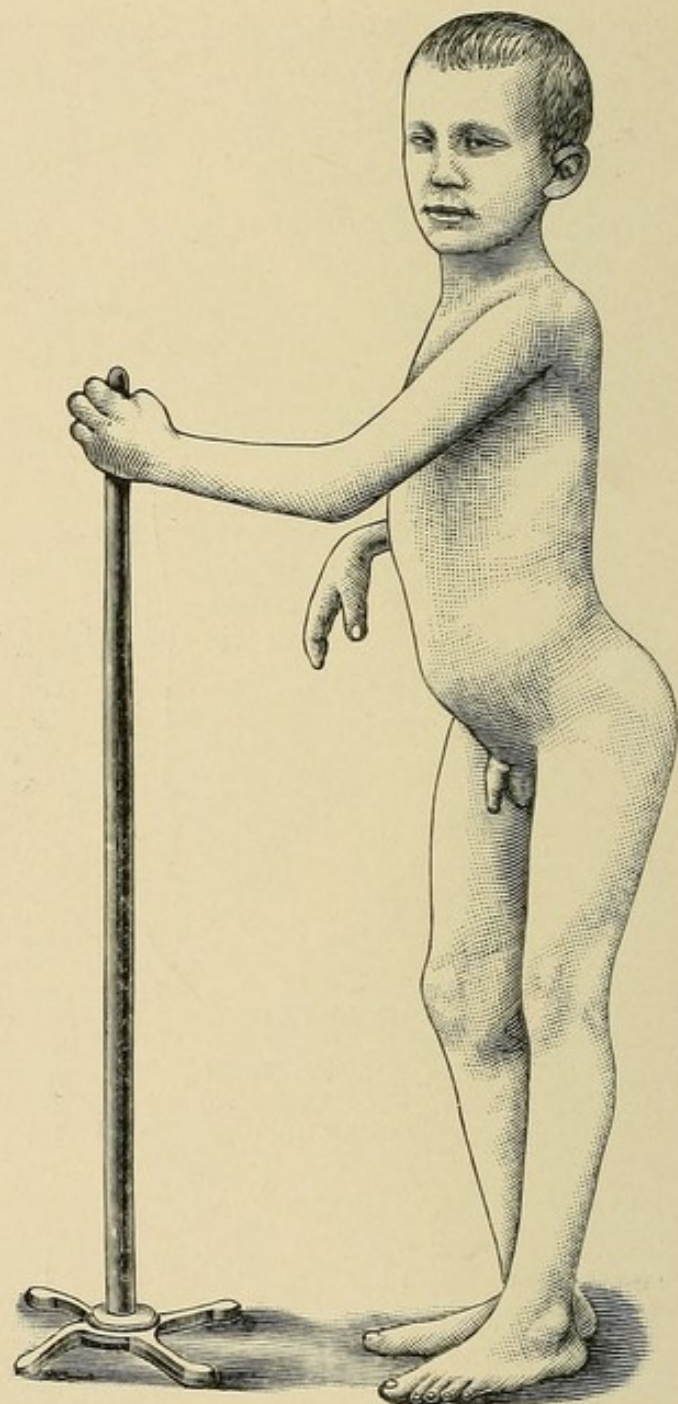


Coxa vara. (Kocher.)

this affection to be confounded with coxa vara, congenital hip-joint dislocation, and fracture of the neck of the femur. The *x*-ray will, as a rule, afford sufficient data for the recog-

dition of the last-named diseases, but in addition there is with fractures and dislocations the history of a severe traumatism. In congenital dislocation there is no pain on motion of the

FIG. 194



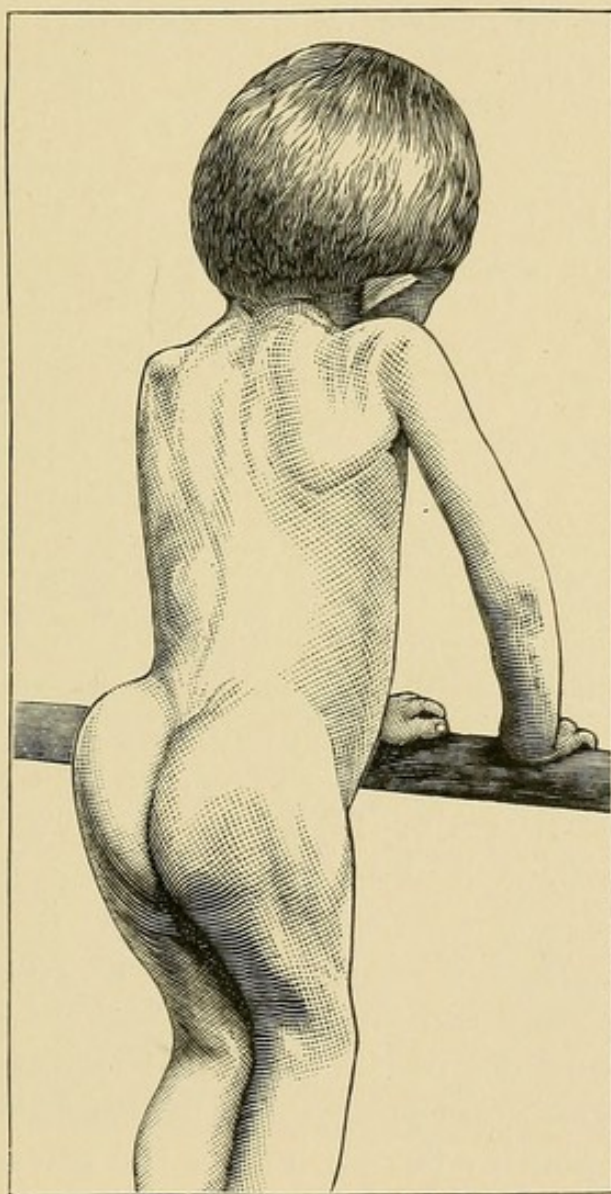
Unilateral congenital dislocation of the hip. (Von Bergmann.)

hip nor reflex muscular spasm; the affection dates from birth in contradistinction to tuberculosis, in which the disease develops after the child has been able to walk naturally and

normally, and finally the gait is waddling and the head of the bone can be felt to slide upward on the dorsum of the ilium as the patient brings the weight of the body upon the leg.

With *coxa vara* there is likewise no pain nor reflex muscular spasm elicited on moving the joint, though the limping,

FIG. 195



Bilateral congenital dislocation of the hip. (Von Bergmann.)

shortening and vicious position of the limb in adduction, extension, and outward rotation may strongly suggest hip-joint tuberculosis.

Knee-joint.—Knee-joint tuberculosis is common. Though it usually commences in the femur or tibia, the earliest

manifestations are from the diseased synovial membrane. As a rule there is first of all an effusion into the joint, with thickening of the synovial membrane, thus resembling in every respect a chronic hydrops. The differentiation is to be made by finding evidences of tuberculosis in *other organs*, by the absence of any etiological factor to account for the hydrops—*e. g.*, trauma, rheumatism, or gonorrhœa—by the tendency of the effusion to persist and to recur in spite of the usual methods of treatment, by the greater thickening of the synovial membrane (especially to be appreciated at the sides of the patella and in the space between the articulating bones), by the crepitation which is to be elicited on moving the fluid around within the joint, and by the yellowish, flocculent character of this fluid. In these early stages the function of the joint is but little disturbed, only the extremes of flexion and extension being limited.

This effusion may after a time gradually become less, and be replaced by a fungous thickening of the synovial membrane. In some cases the fungous degeneration of the joint structures is the first evidence of the disease. As a result of such fungous degeneration the joint gradually becomes enlarged and either assumes a spindle shape, to which the atrophy of the quadriceps contributes, or when only one part of the synovial membrane is thickened the enlargement resembles that due to a sarcoma. The consistency of the joint is firm, and no fluctuation or dancing patella is present.

Ankle-joint.—Ankle-joint tuberculosis is marked in its early stages by pain and swelling, the latter being especially noticeable at the sides of the extensor tendons, in the region of the malleoli, and on the posterior aspect of the joint. The foot is held in plantar flexion, and if the astragalocalcanean joint is involved the foot is likely to be fixed in adduction and supination.

The pain and swelling of the ankle due to flat-foot are readily distinguished from that due to tuberculous disease of the ankle by the presence of the sunken arch, the associated pains in the calf, and the absence of limitation of movement in the ankle with reflex muscular spasm.

Tuberculous tenosynovitis is distinguished from tuber-

PLATE XVIII.



Tuberculosis of the Knee-joint, advanced stage, engrafted upon an old gonorrhœal inflammation.

Note the fungous masses in the joint cavity, the enlargement of the tibial head and patella, and the irregularity of the cartilages covering the articulating surfaces.



culous arthritis of the ankle by the same characteristics as distinguish tenosynovitis in the hand from wrist-joint disease. (See p. 490.)

Astragalocalcanean Joint.—Tuberculosis of the astragalocalcanean joint is characterized by a lower site of the swelling than in ankle-joint disease and by the absence of swelling to the side of the extensor tendons.

Tuberculosis often attacks several of the metacarpal or metatarsal bones simultaneously. The shafts usually become cylindrically enlarged, forming the characteristic *spina ventosa*. (See Fig. 163.)

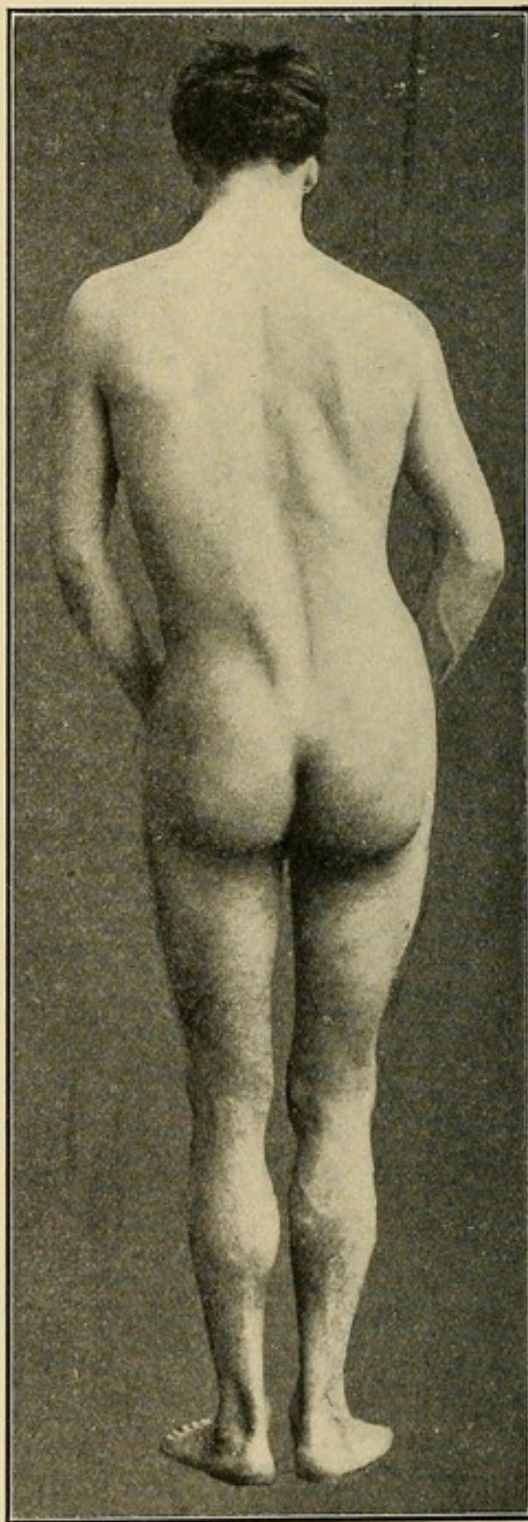
Sacroiliac Joint.—Sacroiliac tuberculosis is especially important because of the semblance its clinical picture bears to tuberculous coxitis. In both affections there is limping and apparent lengthening of the diseased limb. The former is distinguished by the fact that when the pelvis is supported and fixed the hip-joint movements are not at all restricted nor accompanied by reflex muscular spasm. The pain which is due to sacroiliac disease is referred to the gluteal region and leg, and is often enough ascribed to sciatica. The error will be avoided if the area of distribution of the pain is carefully noted, for in sacroiliac disease the pain is not felt in the area supplied by the sciatic nerve. Furthermore the pain of sciatica is sharp and neuralgic in character, whereas in sacroiliac disease it is dull and aching, and, finally, with sciatica there is no apparent lengthening of the limb, nor local pain on pressure over the diseased joint, nor pain on pressing together the iliac crests. Vertebral disease is easily differentiated from sacroiliac disease if the spinal deformity, tenderness over the vertebræ, and limitation of the movements of the spine are taken into consideration.

Vertebral Tuberculosis.—Vertebral tuberculosis (Pott's disease) occasions in its early stages *local pain* over the diseased bones, *referred pain* to the region which is supplied by the nerves whose roots are compressed by the affected vertebræ, *painful rigidity of the spine* at the site of the disease, and an *abnormal attitude*. When the disease is located in the dorsolumbar region its early manifestations are a "loss of walk" and a refusal to stand, and when the

upper dorsal region is involved embarrassed respiration is one of the first symptoms.

The location of the referred pain naturally varies with

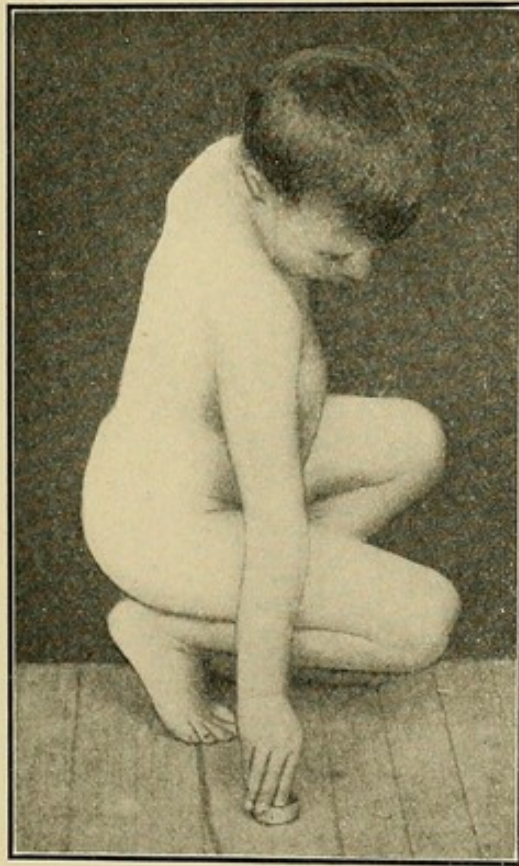
FIG. 196



Deformity caused by persistent sciatica of the right side. This attitude is similar to that symptomatic of sacroiliac disease. (Whitman.)

the site of the disease. If the latter is in the lumbar region the pain is referred to the legs; if it is in the dorsilumbar region the pain is referred to the lower part of the abdomen and the gluteal region; if it is in the dorsal region the pain is referred to the epigastrium; if it is in the cervicodorsal region the pain is referred to the arms, and if it is in the upper cervical region the pain is referred to the region supplied by the cutaneous branches of the cervical plexus.

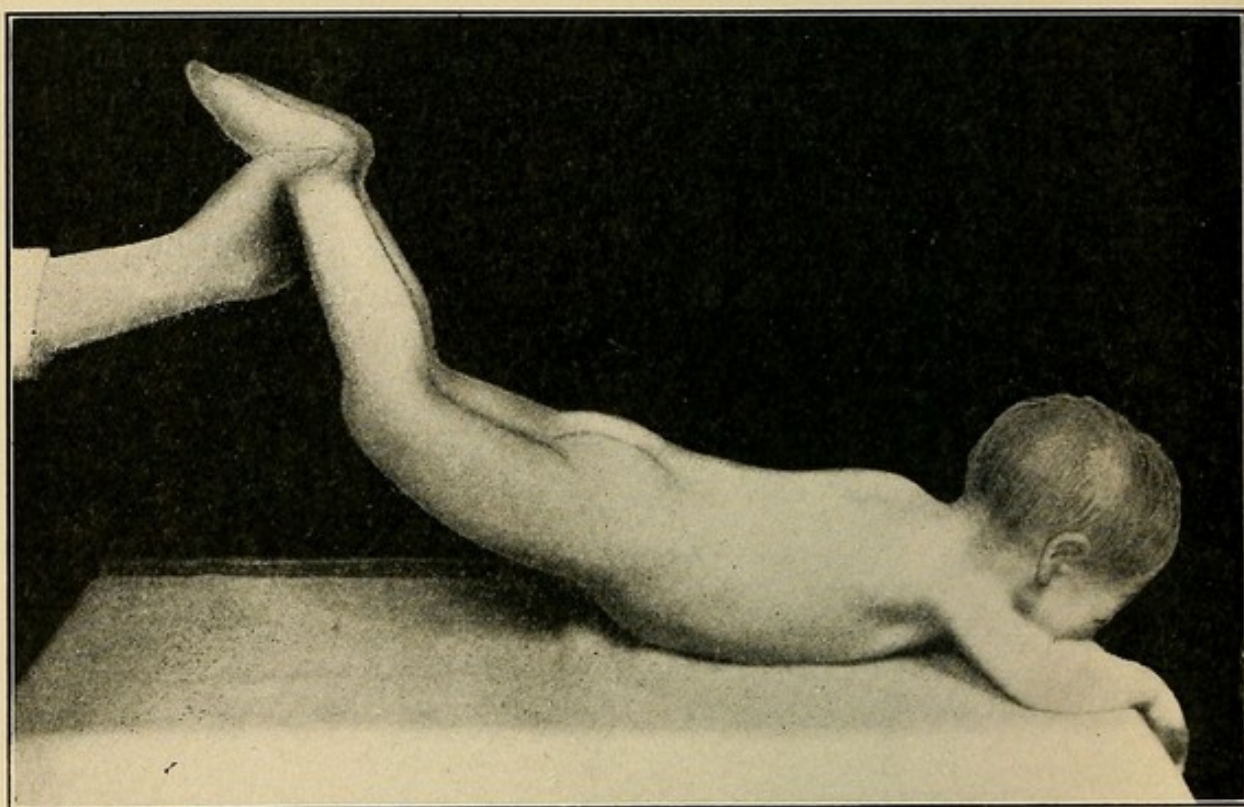
FIG. 197



Lumbar disease. Painful rigidity of the spine illustrated by the manner of picking up an object. (Whitman.)

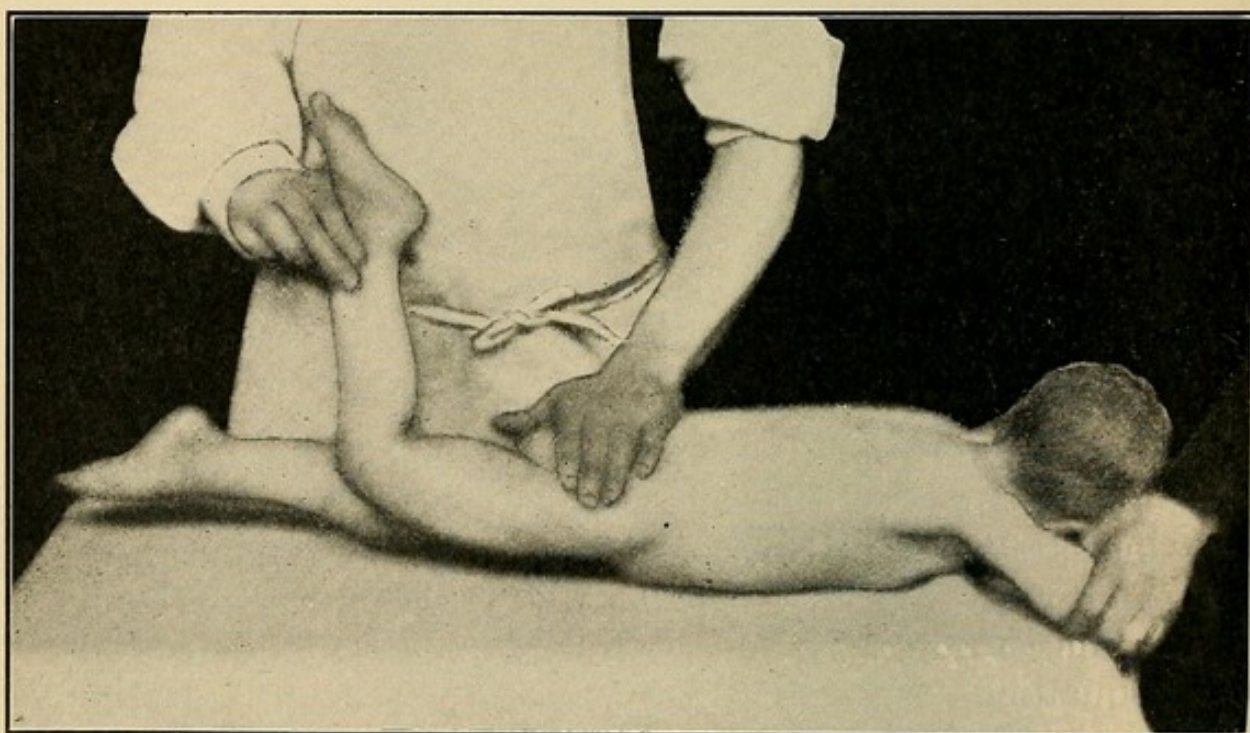
The painful rigidity of the spine is chiefly due to reflex muscular spasm. In the dorsilumbar region it can be demonstrated in two ways: (1) By asking the patient to pick up an object from the floor; in order to do this he gradually lets himself down with an absolutely rigid back into a sitting or squatting position, and then raises himself up by resting his hands upon his thighs, thus climbing with extended arms up his own legs; and (2) by placing the patient on a level

FIG. 198



Showing the rigidity of the spine before appearance of deformity. (Whitman.)

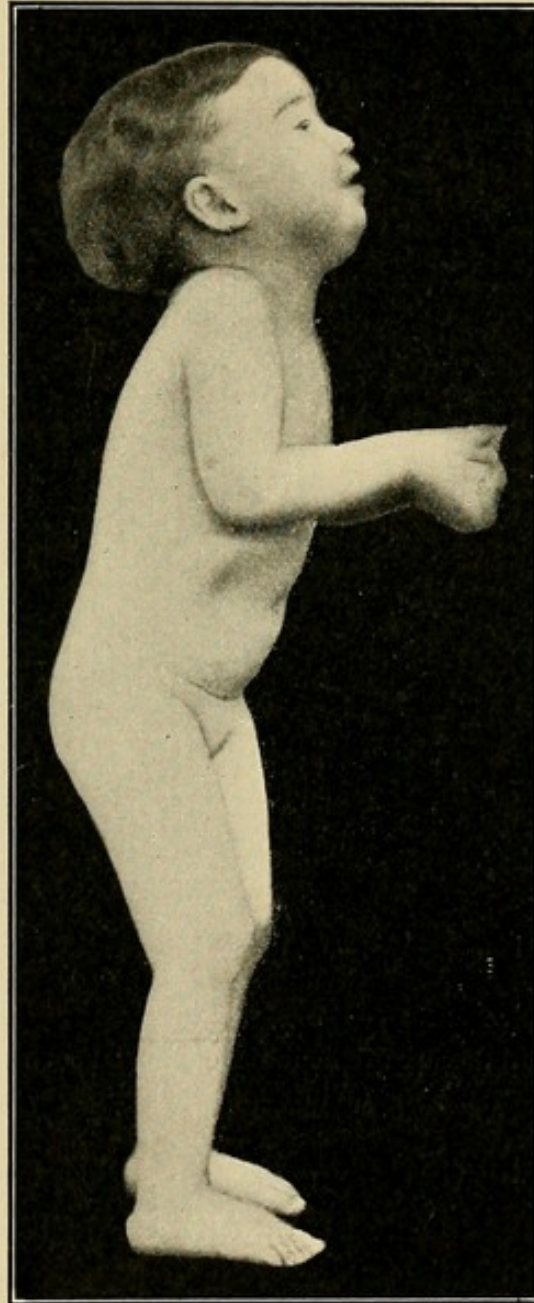
FIG. 199



Test for psoas contraction. (Whitman.)

couch with his face downward, and then grasping the ankles and lifting the legs from the table and from side to side. In a healthy subject the spine can thus be bent back in the dorsilumbar region to 60 degrees, while lateral mobility to

FIG. 200



Disease of the upper dorsal region. Characteristic attitude. (Whitman.)

30 degrees or 40 degrees on either side of the median line is possible. A reflex psoas spasm is an early sign of lumbar Pott's disease.

The attitude in the early stages of a lumbar or lower dorsal Pott's disease is overrectness, the abdomen being prominent;

the walk is swaggering or waddling. With an upper dorsal Pott's disease there is a slight forward inclination of the body, the head being tilted backward or inclined to one side, the shoulders being elevated, shrugging, and square. With lower cervical disease the head is inclined backward or toward one shoulder, while with upper cervical disease the chin is tilted to one side, simulating the attitude of torticollis; the neck is stiff and the patient refuses to rotate the head, and usually supports the chin on his hand.

FIG. 201



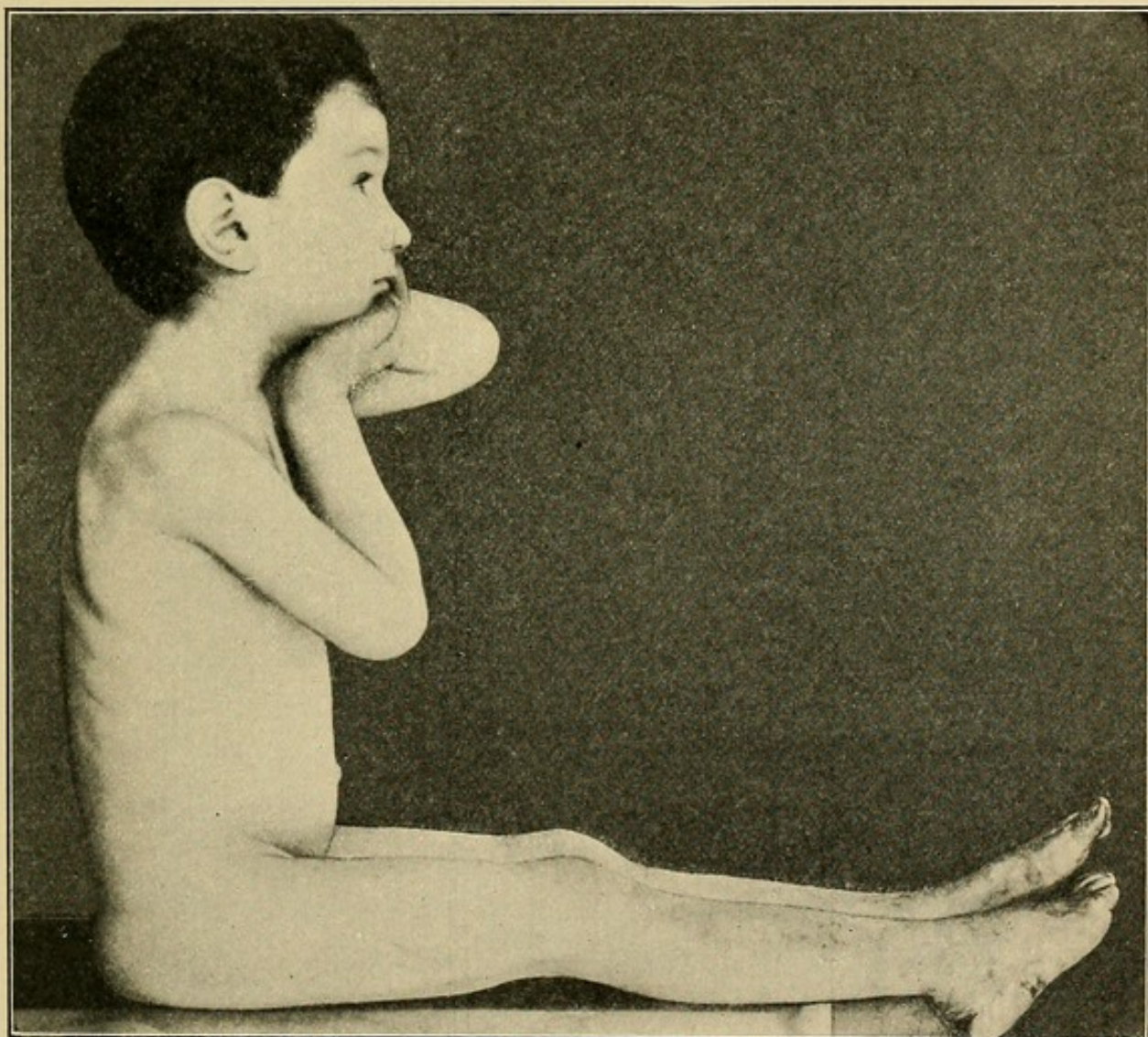
Characteristic attitude in disease of the middle cervical region at an early stage.
(Whitman.)

Angular or kyphotic deformity of the spine, cold abscess formation, ankylosis of the spine and in some cases evidences of cord compression are the additional signs of spinal tuberculosis in the late stages of the disease.

The site of the abscess depends upon the location of the disease. With cervical Pott's disease a chronic retropharyngeal abscess is first formed. This may burrow downward into the mediastinum, or outward into the posterior or anterior triangle of the neck, or downward and outward into the axilla.

In the dorsal region the abscess may point directly backward, or it may burrow forward along the intercostal spaces, or upward into the neck or downward into the psoas muscle. In disease of the dorsilumbar or lumbar regions the abscess may point in the lumbar region and present superficially in Petit's triangle, or it may form in the psoas muscle, and

FIG. 202



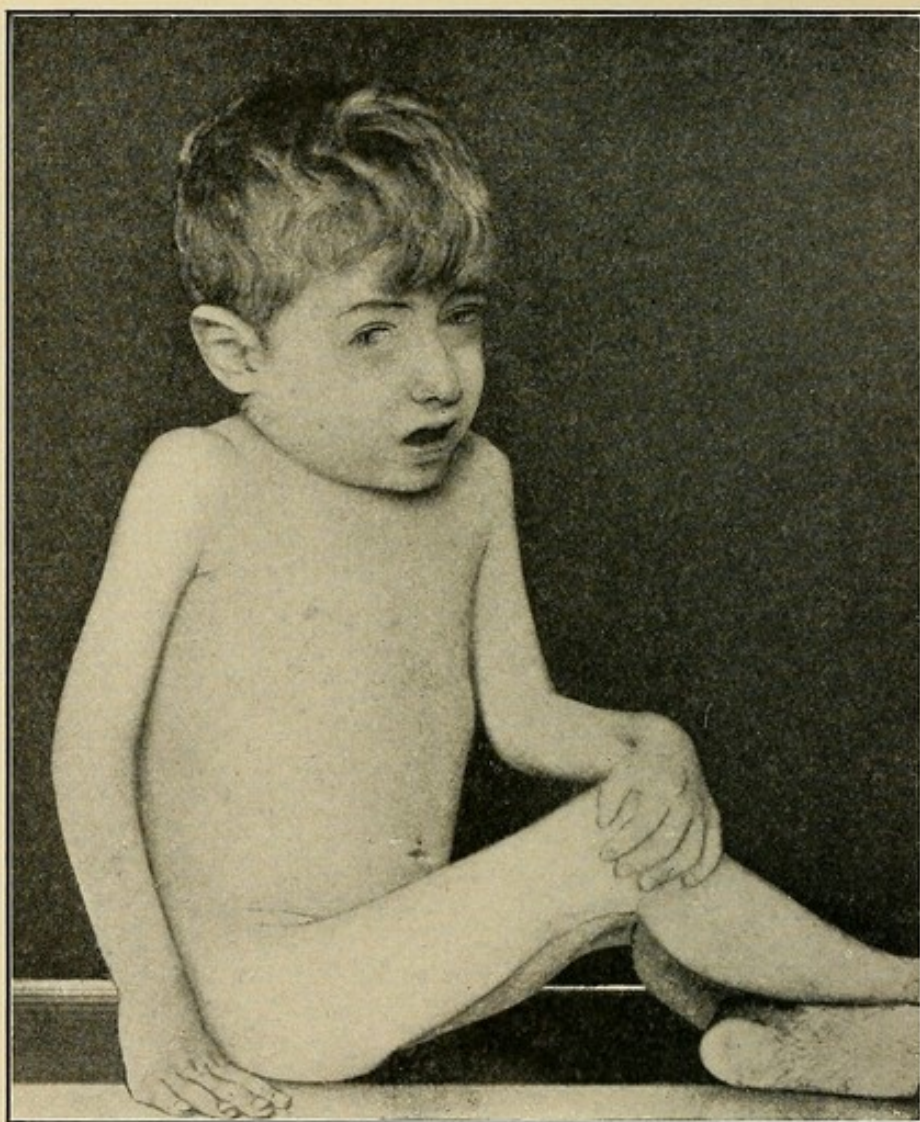
Cervical disease. A characteristic attitude. (Whitman.)

thence burrow downward, presenting superficially above Poupart's ligament to the outer side of the femoral vessels or below Poupart's ligament to the inner side of the vessels.

The referred pains that attend early Pott's disease will lead to frequent mistakes in diagnosis unless a careful examination is made. In children especially we should not

be too quick to attribute pains in the legs or in the abdomen to neuralgia or stomach ache, etc., but we should in every instance, where there is no palpable cause for the pain, carefully examine the spine for a possible explanation of it. The pain and stiffness that go with the early stages of lumbar

FIG. 203

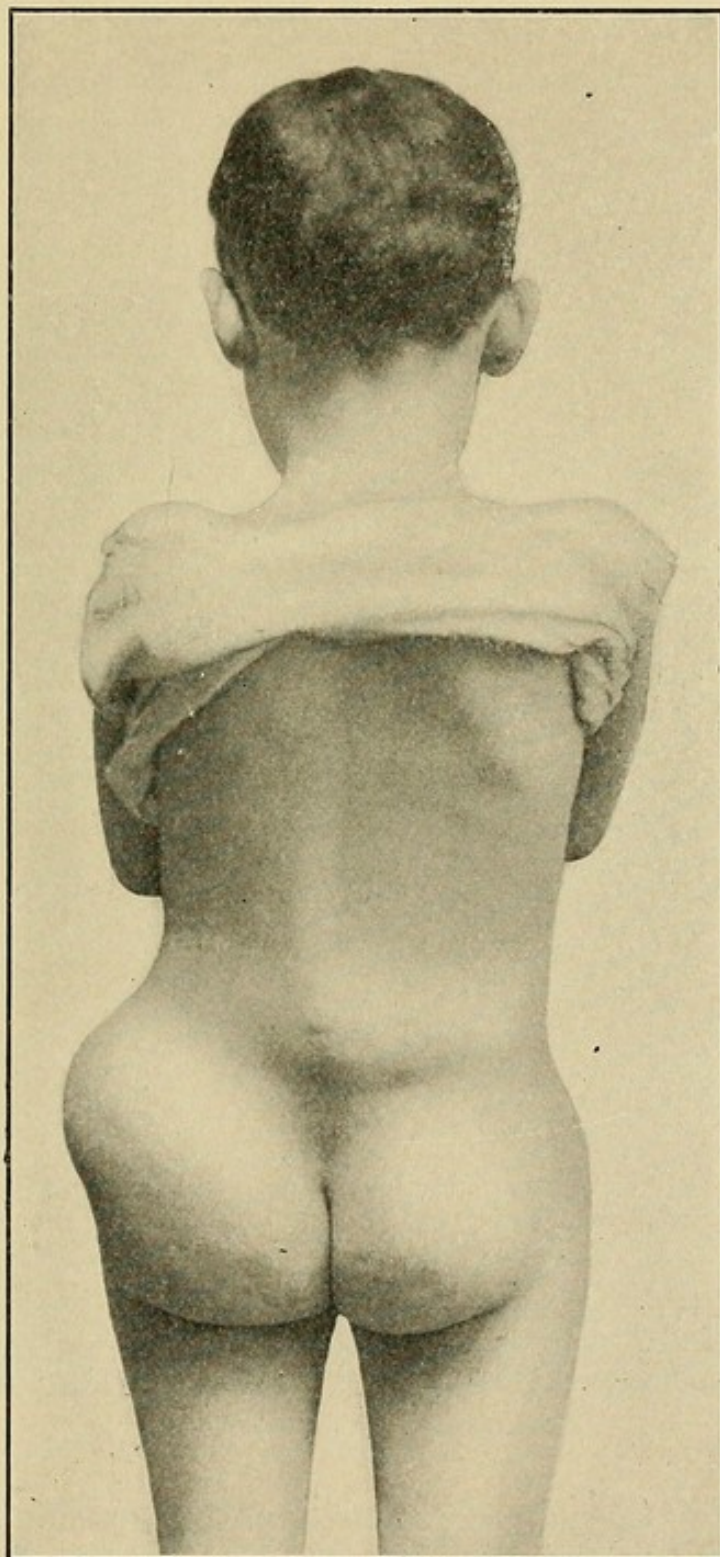


Cervical disease with abscess. Characteristic attitude. (Whitman.)

Pott's disease may be mistaken for that which is due to lumbago and sciatica; and the attitude and gait that attend vertebral tuberculosis of this region may suggest sacroiliac disease, bilateral congenital dislocation of the hip, progressive atrophy of the back muscles, hip-joint disease, and rachitical spine. *Lumbago* is an acute affection of sudden

onset, and is usually accompanied by local pain and sensitiveness of the muscles themselves. In sciatica the pain

FIG. 204



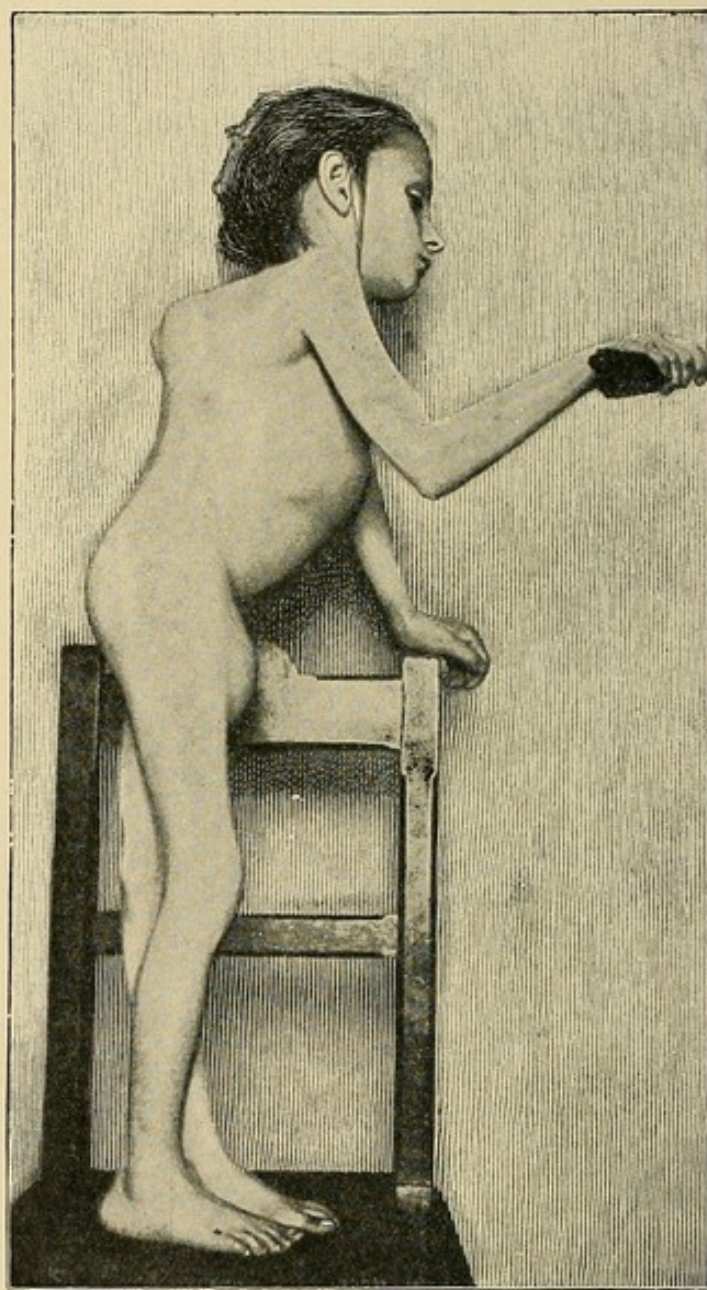
Pott's disease of lumbar vertebræ with cold abscess. (Von Bergmann.)

is most often unilateral and is confined to the distribution of the nerve, which is frequently sensitive to pressure through-

out its course, and the motion of the spine is free or but slightly restricted, all of which are the reverse of the symptoms of Pott's disease.

In *sacroiliac disease* the movements of the spine are not

FIG. 205



Psoas abscess. (Von Bergmann.)

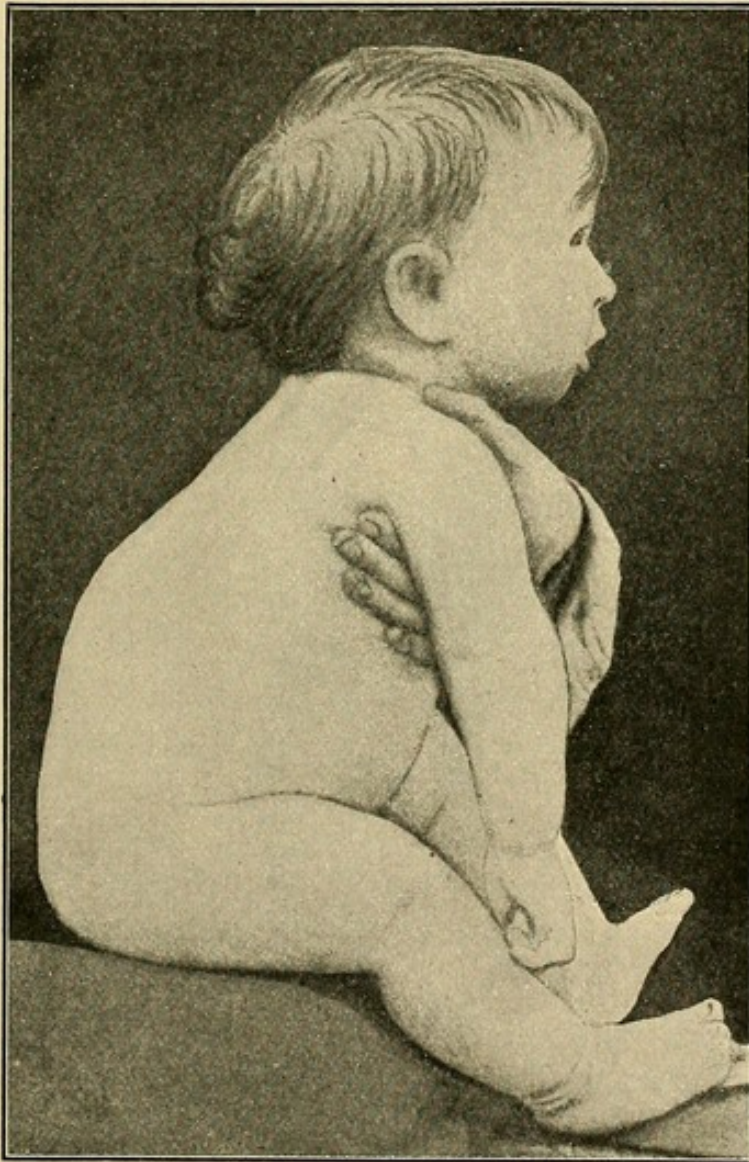
restricted, and the pain and sensitiveness are usually localized to this joint.

In *bilateral congenital dislocation of the hip* the gait and attitude have existed since the child began to walk and the symptoms of bone disease are absent.

In *progressive muscular atrophy* there are no evidences of bone disease.

The *psoas contraction* and the *limp* which accompany some cases of lumbar Pott's disease are late manifestations, and are readily ascertained as not due to *hip disease* by flexing

FIG. 206



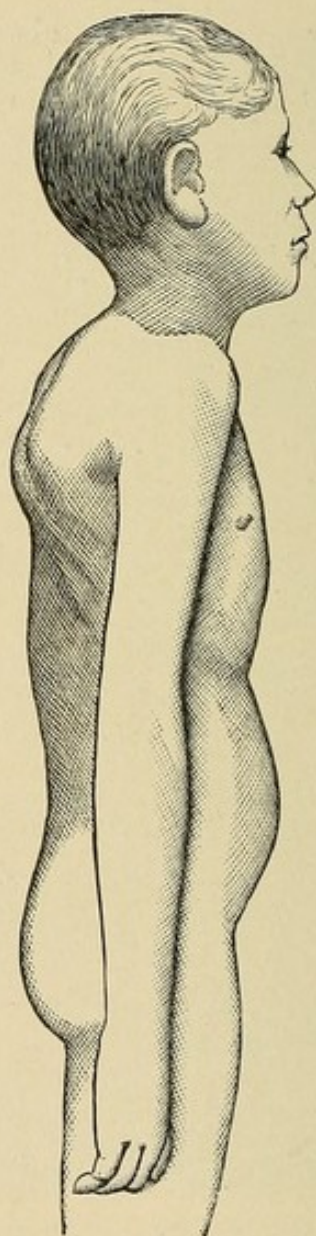
Rachitic kyphosis. Compare with Fig. 207. (Whitman.)

the thigh, in which position there is no limitation to free movement of the hip-joint in all directions. The possible complication of hip-joint and lumbar Pott's disease should be borne in mind in those cases in which a large psoas abscess is present.

The evidences of *general rachitis*, the absence of painful rigidity of the spine, and the rounded character of the spinal curvature readily enable us to differentiate rachitical deformity of the spine from that due to tuberculous disease.

The stiffness and distortion of the neck that attend cervical

FIG. 207



Kyphosis from vertebral tuberculosis. Compare with Fig. 206. (Von Bergmann.)

Pott's disease at once suggest the different varieties of *torticollis*. In *typical torticollis* the distortion of the head is almost invariably caused by contraction of the muscles supplied by the spinal accessory nerve—viz., the sternomastoid and the trapezius; thus the chin is slightly elevated

and turned away from the contracted muscles. In the wryneck of Pott's disease the chin may be tilted down or up or laterally to an exaggerated degree and is turned toward the contracted muscle, and the painful contraction is relieved if the head is supported. *Congenital torticollis* exists from birth and is not painful.

Rheumatic torticollis is of sudden onset and the affected muscles are sensitive to pressure. Acute torticollis accompanied by muscular spasm and by local tenderness sometimes accompanies enlarged or suppurating cervical glands, ear disease, tonsillitis, etc. But this form of wryneck is sudden in onset; and if the tension be relaxed by inclining the head toward the contracted muscles, motion of the spine will be found free and painless.

Rheumatic affections of the spine are differentiated from the tuberculous by their sudden onset, the participation of other joints in the disease, and by their more painful character.

Tumors of the spine, such as cancer or hydatid cysts, *syphilitic disease*, and *aneurysmal erosion* produce symptoms somewhat resembling those of tuberculous disease. A careful consideration of the general history and of the onset of the symptoms and a careful physical examination, aided by the test of treatment, will, in most cases, enable us to make a diagnosis.

Abscesses in the lumbar region resulting from lumbar Pott's disease are to be differentiated from perinephritic abscesses by the symptoms of spinal disease and the absence of evidences of renal affection.

Psoas abscesses are differentiated from chronic appendicular abscesses by the presence of symptoms of spinal disease, and from the clinical history of the malady.

Abscesses due to disease of the pelvic bones or pelvic cellular tissues are not attended with the evidences of spinal caries.

CHAPTER LII.

CHRONIC DISEASES OF THE JOINTS (*Continued*).

THOUGH tuberculosis is the most frequent type of chronic joint disease, the occurrence of syphilitic, neuropathic, neuralgic, osteoarthritic, and hæmophilic joint affections must not be forgotten.

SYPHILITIC JOINT DISEASE.

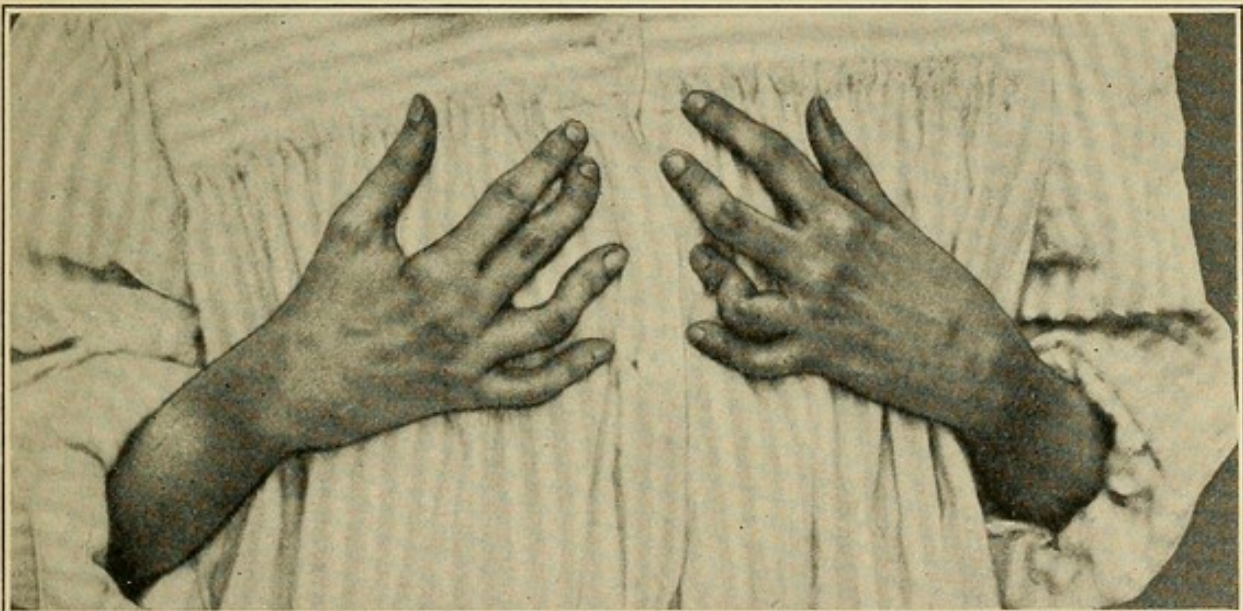
The chronic synovitis due to syphilis differs in no way from that due to other causes, and it can be recognized only from the anamnesis, the presence of other syphilitic lesions, and by the exclusion of other etiological factors. In the tertiary period of syphilis localized elastic nodules (gummata) may form in the perisynovial fibrous tissue; these resemble fibromata and occasion a sense of painful weakness in the articulation. Diffuse nodulation with much thickening and infiltration of the capsule and moderate serous effusion into the joint may also occur in the tertiary stage of syphilis, and the cicatricial contraction to which such a process gives rise causes considerable impairment in the function of the joint and often leads to a firm ankylosis thereof. The ankylosis may suggest tuberculous disease of the joint, but it differs from that due to tuberculosis in its firmer and more painful character, and its more rapid development. The presence of other specific lesions aids in making the diagnosis.

Softening and erosion of the articular cartilages, with eburnation of the bone, is a rare result of syphilitic disease; the condition bears a superficial resemblance to osteoarthritis, from which, however, the absence of pain and of the characteristic overgrowth of bone and the slighter grades of the destructive process serve to differentiate it.

OSTEOARTHRITIS.

The joint lesions due to osteoarthritis (*arthritis deformans*) are characterized by their occurrence usually in elderly subjects, their constant progression, and by the absence of suppuration and caries. The clinical picture is so typical that errors in diagnosis are rarely made. Either a single large joint—*e. g.*, the hip, the knee, the elbow, the shoulder—or the vertebral articulations, or multiple small joints—*e. g.*,

FIG. 208



Arthritis deformans. Note enlargement and thickenings of the joints and their irregular deformities.

the fingers and toes—become painful and stiff and crepitate on motion, the stiffness being especially marked after the joint has been at rest. The disease in the monarticular variety seems to follow injury; in the polyarticular variety it occurs spontaneously. The large joints become deformed, somewhat swollen, especially if there is an effusion into the capsule; the ends of the bones become enlarged and the overlying muscles atrophic. The smaller joints likewise become swollen, with marked overgrowth and eburnation of the articular ends of the bones. In both varieties acute exacerbations occur from time to time, marked by fever,

increased pain, and joint effusion, and finally the affected joints become crippled and deformed.

The painful character of the affection; the stiffness of the joint, especially after it has been at rest; the crepitus and slow enlargement of the bones; the effusion, and the absence of suppuration and caries should readily enable one to make the correct diagnosis.

Chronic synovitis is not attended with as much pain or stiffness as is arthritis deformans, and the effusion is always more copious.

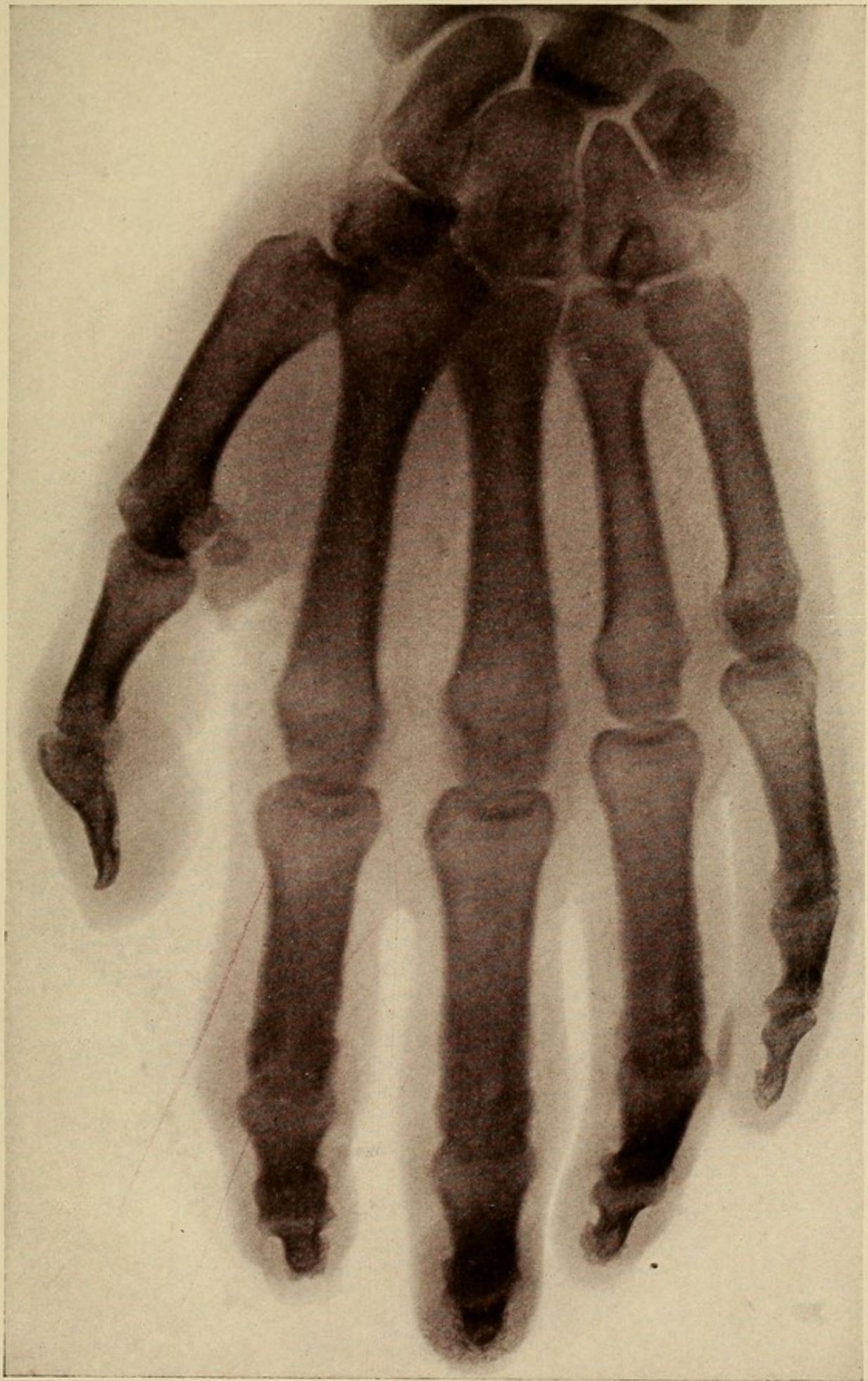
Chronic rheumatism is not marked by the deformity of the bones, nor the crepitation, nor the constantly progressive character of arthritis deformans.

Gout is characterized by acute attacks of joint inflammation of entirely different onset and clinical history.

Charcot's disease is not painful; it is attended with other signs of tabes dorsalis, and it does not occasion as much bony overgrowth as does arthritis deformans.

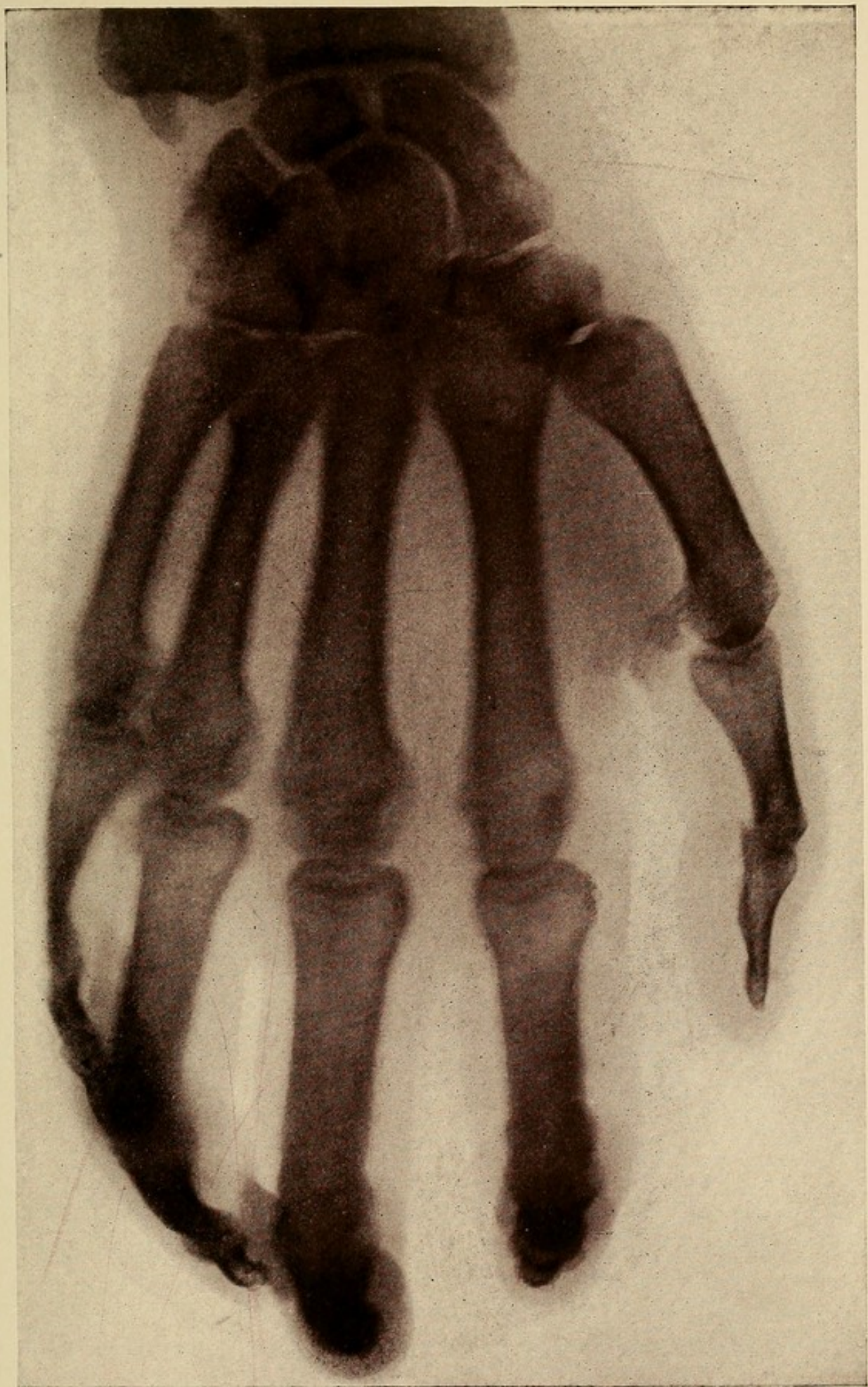
In the hip-joint the disease results in a depression of the femoral neck, with consequent shortening of the limb and limitation of abduction. These symptoms may lead to a diagnosis of coxa vara, or if, as is frequently the case, an injury precedes the onset of the malady or occurs during its course, the diagnosis of fracture of the neck of the femur may be made. The marked and continued pain of arthritis deformans, its constantly progressive character, and the muscular atrophy readily differentiate it from coxa vara, and the movement of the head with the shaft of the bone excludes fracture unless it be impacted.

The pain evoked by movements of the hip in sciatica may possibly suggest arthritis deformans. The differentiation is readily made if the patient is asked to stand up and spread his legs apart. The limitation of abduction which accompanies arthritis deformans interferes with this action; further evidence of sciatica is afforded in the distribution of the pain along the posterior aspect of the thigh and outer side of the leg and foot; and of arthritis deformans by the crepitation and stiffness of the joint, especially after rest.



Companion plate to Plate XX. Arthritis Deformans at very early stage, the joint thickenings and enlargement of the articular ends of the bone being very slightly in evidence.





Arthritis Deformans.

Note the enlargement of the articular ends of the metacarpal and phalangeal bones and the thickenings around the joints. Compare with Plate XIX., representing the other hand of the same individual, in which the lesions were less marked.

the seat of recurring attacks of *rapid, painless distention*, usually provoked by slight trauma, with gradual atrophy of the bones and relaxation of the ligaments, in virtue of which they become weakened, flail-like and dislocated, the diagnosis of neuropathic joint disease is justified. The above characteristics at once distinguish these joint lesions from those due to arthritis deformans; it is to be noted, however, that instead of atrophy of the bones, new osseous formation may sometimes occur, leading to large overgrowths and fixation of the joint. The painless character of the joint disease and the presence of the primary affection will always suffice to distinguish these joint diseases from the other chronic arthropathies.

HÆMOPHILIC JOINTS.

The *hæmophilic joints* are characterized by their sudden onset in individuals having a family and personal history of bleeding. They are frequently multiple, tend to recur, and arise spontaneously or after slight injury. The joints become swollen, are soft and doughy at first, but later become hard and firm; motion is painful and the skin is hot.

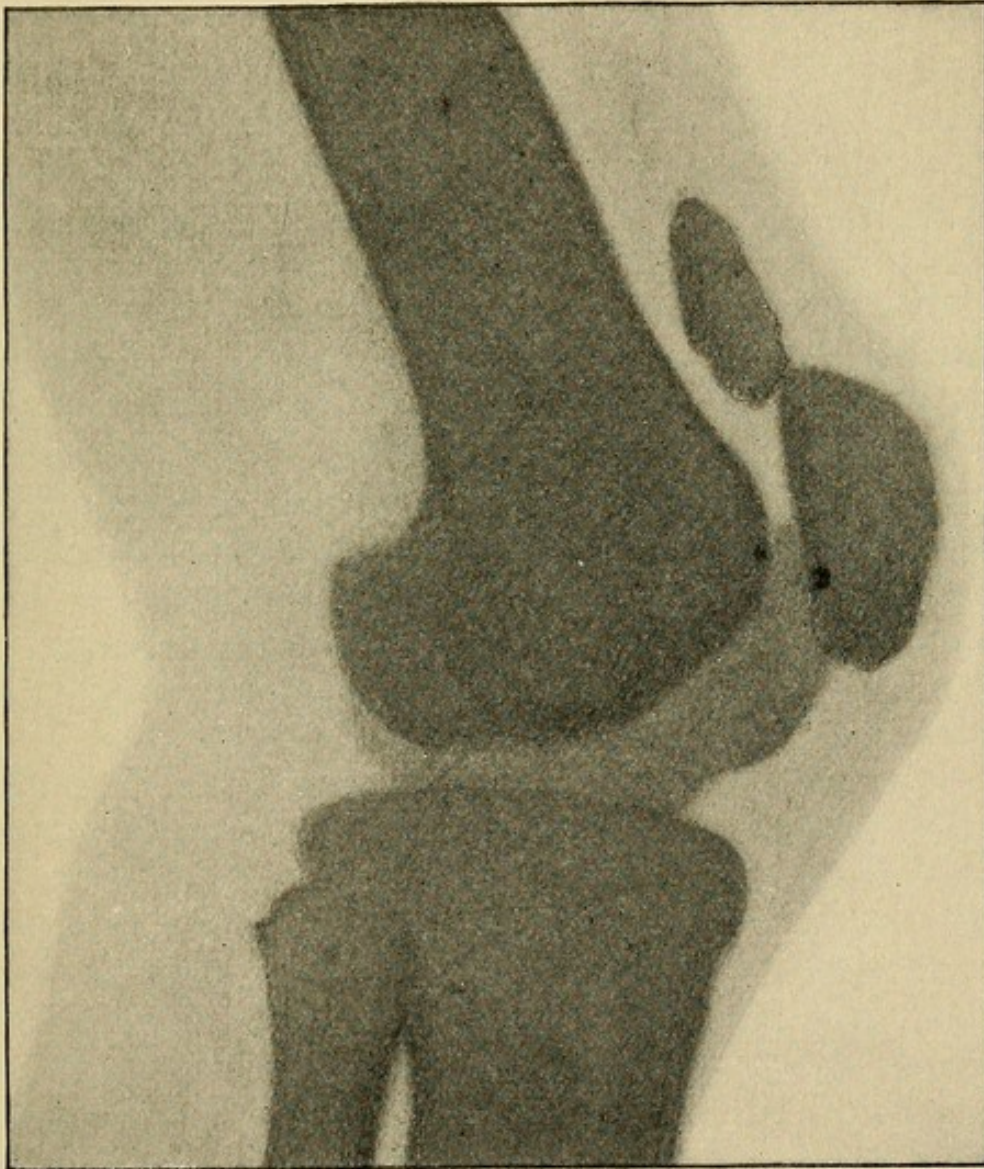
NEURALGIC JOINTS.

The *neuralgic joints* are encountered chiefly in neurotic individuals, especially young women. The deformity, pain, and wasting of muscles which accompany them may strongly simulate chronic joint disease. On local examination, however, either no organic lesions are found or only such as are entirely disproportionate to the severity of the subjective symptoms. Under anæsthesia the joint moves freely in all directions, and there is no reflex muscular spasm. A close observation of the patient will sometimes reveal the fact that the vicious position in which the limb is maintained is not a constant one, but varies from time to time. It is to be noted that no other hysterical symptoms may be present, and that the individual may appear to be entirely beyond the pale of suspicion of *being possessed* of such a *malady*.

FOREIGN BODIES IN JOINTS.

Recurrent, sudden attacks of intense pain in a joint (the knee is the one that is most frequently affected) with its momentary locking, followed by a subacute synovitis, are

FIG. 210



Free body in the knee-joint. (Von Bruns.)

characteristic of loose bodies in the joint. The diagnosis is verified by feeling the loose bodies, which is best done by crowding down the patella and quadriceps bursa and then palpating along the sides of the patella.

The attacks are very similar to those provoked by a loose or displaced semilunar cartilage. The latter condition is distinguished by a longer duration of the joint pain and fixation (in fact, these last until the meniscus is reduced), by the palpation of the displaced meniscus and by the history of a severe injury.

CHAPTER LIII.

INJURIES OF THE SPINE.

WHAT has been said in reference to the diagnosis of injuries of the bones and joints in general applies equally well to these conditions of the vertebræ. The latter have an especial significance only because they may be complicated by injury to the spinal cord, its membranes and nerves, which complications when they exist far outweigh in importance the lesions of the osseous and articular structures.

Fracture.—It is a common belief that fractures of the vertebræ are always attended with deformity of the spine and injury to or compression of the cord, its enveloping membranes and nerves. As a matter of fact, the former is present only when the fractured bones are displaced, and the latter only when the cord is compressed or destroyed or the seat of concussion. Even in extensive fractures there may be no displacement of the fragments and no disturbances of the cord; in such cases the diagnosis of fracture rests upon a history of trauma and the presence of pain, tenderness, crepitus, and abnormal mobility over one or more vertebræ. In some cases of vertebral fracture spinal deformity alone is present. This is recognized by a deviation of the spinous or articular processes from their normal positions and by angulation of the spine. If such cases are first seen some time after the injury has been sustained the deformity and the local pain and tenderness may suggest tuberculous or other organic disease of the vertebræ.

The differentiation, however, is readily made from the history, the patient having been entirely well prior to the reception of the injury. In still other cases of fracture there are symptoms of spinal-cord involvement without any spinal deformity. If these symptoms develop at once or in the course of the first twenty-four hours and grow gradually worse, they are due to compression of the cord by blood; if they develop

three or four days after the injury they are due to inflammatory exudate, and if they first appear some time after the reception of the injury they are due to cicatricial adhesions or callus.

Dislocation.—Instantaneous death after a trauma to the upper part of the neck, or after hyperextension, or hyperflexion, or hyperrotation of the head suggests a complete *dislocation of the atlas or axis* with compression of the cord or medulla.

FIG. 211



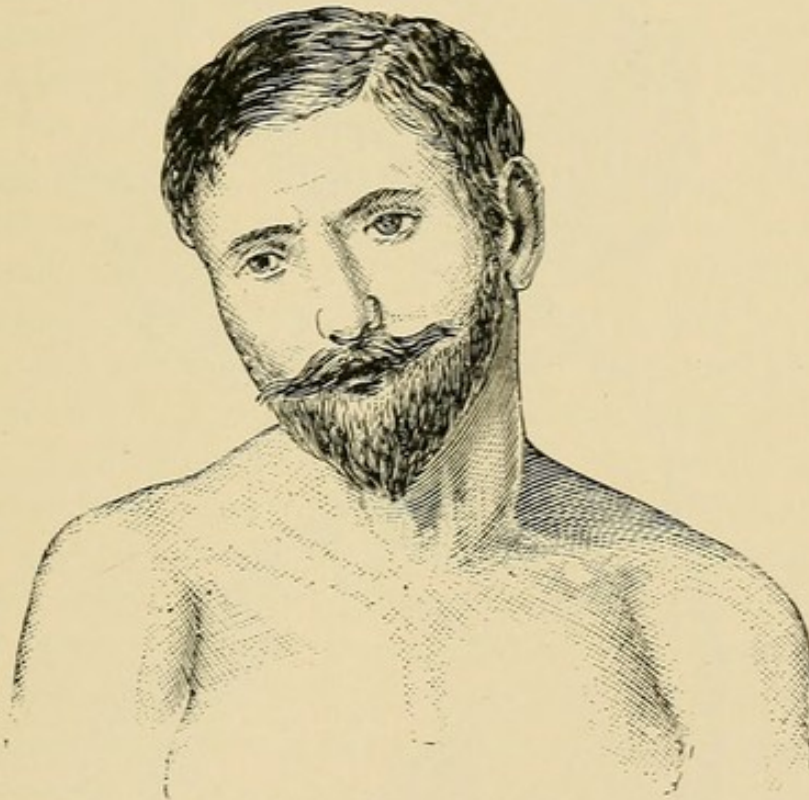
Attitude in complete rotary dislocation of the left cervical vertebræ. Note that the neck is shorter on the dislocated side, and that the head is flexed to the dislocated side and rotated to the opposite side.

Dislocation of the dorsal and lumbar vertebræ is only possible when combined with fracture of the articular processes. This lesion is readily recognized from the displacement of the articular and transverse processes and from the evidences of compression of the cord.

Bilateral dislocations, complete and incomplete, and unilateral dislocations are possible in the lower five cervical vertebræ. The former are usually attended with evidences of cord compression, which with the spinal deformity and a sinking of the head downward and forward between the shoulders indicate the nature of the injury. More or less fixation and rotation of the head to one or the other

side, the neck being convex on one side and concave on the other, together with displacement of the spinous and lateral processes, point to a unilateral dislocation upon the side of the convexity of the neck. The cord in these unilateral dislocations usually escapes compression, though a tingling and neuralgic pain along the course of the nerves may arise from pressure upon and stretching of the nerve trunks in the intervertebral notches.

FIG. 212



Attitude in incomplete rotary dislocation of cervical vertebræ (left side). Note that the neck is longer on the dislocated side and the head is flexed to the healthy side. (Hoffa.)

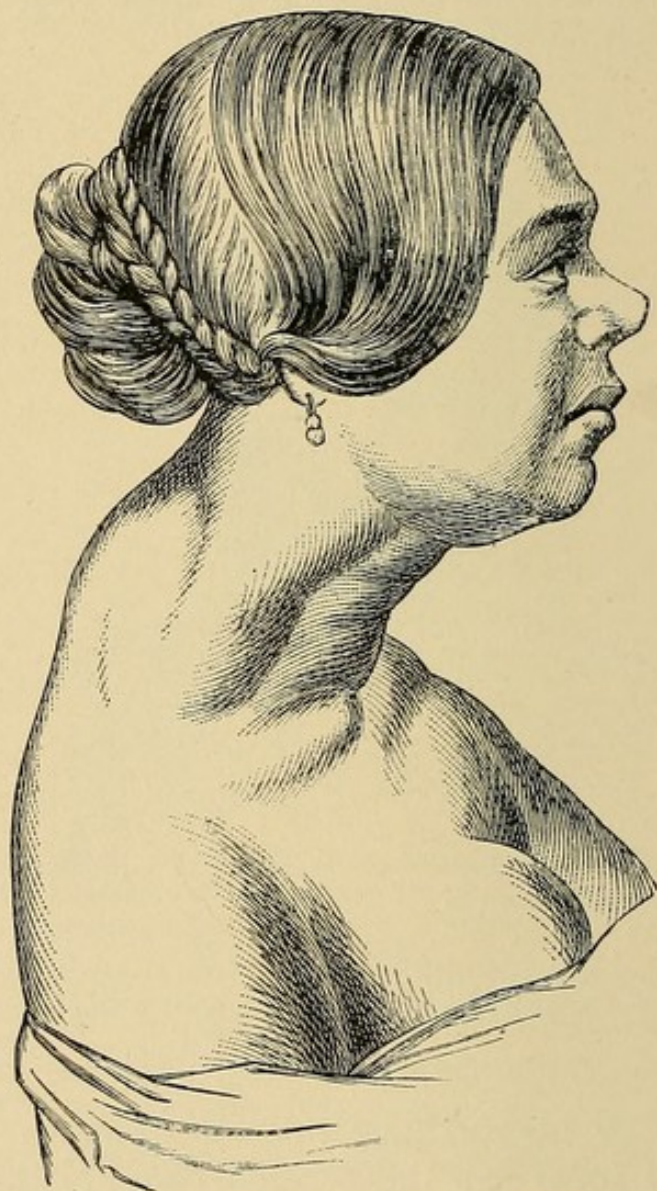
Injuries of or hemorrhage into or upon the spinal cord and its nerve roots are the serious complications of spinal injuries. They may complicate fractures or dislocations, or occur independently of these conditions. Every spinal injury should therefore be viewed with suspicion as regards its possible effect upon the cord, and even in the absence of spinal deformity and other signs pointing to fracture we should be very cautious in expressing an opinion as to the ultimate consequences.

Just as in brain injuries so in those of the cord, the severity,

extent, and permanency of the symptoms depend upon the character, site, and cause of the lesion.

Spinal Concussion.—Spinal concussion is, like cerebral concussion, transient in its immediate effects. Its manifestations are muscular weakness or paralysis, paræsthesiæ,

FIG. 213



Attitude in complete forward dislocation of the cervical vertebræ. (Hoffa.)

hyperæsthesiæ, or anæsthesiæ. If these symptoms persist there is present something more than concussion. Concussion may give origin to a chronic myelitis (railway spine), the evidences of which are a gradually developing and slowly but continuously progressing pain in the back, psychical disturbances, such as irritability, lack of energy, and weakness of

memory; anæsthesiæ, hyperæsthesiæ, convulsions, paralysis, etc., the cerebral symptoms being probably due to a coincident chronic encephalitis. The absence of any physical evidences of fracture or dislocation or of compression of the cord may lead to such patients being mistaken for simulants.

Compression of the Cord.—Compression of the cord is lasting in its effects. It may appear immediately after the injury, as when it is due to displacement of bones, penetrating foreign bodies, or severe intramedullary hemorrhage, or it may develop after a shorter or longer interval, as when it is determined by a slow extramedullary hemorrhage, or an inflammatory exudate, or callus, or cicatricial adhesions around the cord and its membranes. The symptoms of compression are very similar to those of destruction of the cord, and their character and extent depend upon the nature and site of the lesion. Paralysis of the penile and perineal muscles and of all the muscles of the legs except those supplied by the anterior crural, the obturator, and the superior gluteal nerves; anæsthesia of the penis, scrotum, perineum, lower half of the gluteal region, and the whole of the legs except the front and outer part of the thigh, and loss of bladder and rectal control point to total transverse lesions at the upper end of the sacrum, involving the cauda equina and causing paralysis of the sacral plexus. The patient lies with feet extended (drop-foot), but can move the thighs and legs.

Complete paralysis of the muscles of both limbs, including those passing to them from the trunk; total anæsthesia of the legs, gluteal and perineal regions and possibly the lower part of the abdomen, and complete loss of control over the bladder and rectum indicate a total transverse lesion of the lumbar enlargement of the cord which corresponds to the twelfth dorsal and first lumbar vertebræ. The patient lies with the thighs, legs, and feet extended.

The same phenomena with a more extensive region of anæsthesia, limited above by a hyperæsthetic zone which feels like a tight, painful girdle around the waist, and paralysis of the abdominal muscles indicate a transverse lesion in the mid-dorsal region. There is no tendency to drop-foot and the limbs offer some resistance to passive motion, the tendon reflexes being exaggerated and the muscles somewhat rigid

unless there has been a total destruction of the cord, under which circumstances the muscles are relaxed and the tendon reflexes lost.

The same phenomena with paralysis of the spinal and intercostal muscles, with anæsthesia of nearly the whole trunk, the hyperæsthesia possibly involving the arms, and with priapism, indicate a total transverse lesion in the cervicodorsal region. The hands are in a position of *main en griffe*, but the elbows and shoulders can be freely moved.

The same phenomena with the paralysis and anæsthesia extending to the arms indicate a lesion in the lower cervical region. If the seventh cervical segment is destroyed the forearms are partially flexed and lie upon the body with the hands pronated. Voluntary movements of the wrist are impossible, but the elbow and shoulder can be moved. If the sixth cervical segment is involved the arms are abducted from the side, the forearms are supinated, and the wrist and fingers are paralyzed. If the fifth cervical segment is involved the arms lie extended and relaxed at the side of the body, all motion being impossible.

Partial paralyses in the area of distribution of the spinal nerves point to incomplete transverse lesions; and motor paralysis on the injured side with sensory paralysis on the opposite side—*i. e.*, Brown-Séquard paralysis—points to hemitransverse lesions. With the lesser degrees of compression or destruction the motor paralyses are more in evidence than the sensory; in fact, the latter may be altogether absent.

In determining the exact level of the lesion in the cord we are guided chiefly by the area of anæsthesia. It is important to remember in this connection that the level of the lesion should always be placed one segment higher than appears to be the case from the area of anæsthesia. The muscles which are the seat of flaccid paralysis and atrophy, and which show reaction of degeneration and loss of reflex action, also guide us to the level of the lesion. The character of the lesion is often very difficult to determine. If the symptoms come on gradually during the first twenty-four hours after the injury the lesion is probably a compression by a blood clot. Whether in the other cases the lesion is a compression or destruction can only be determined by operation.



PLATE XXI.



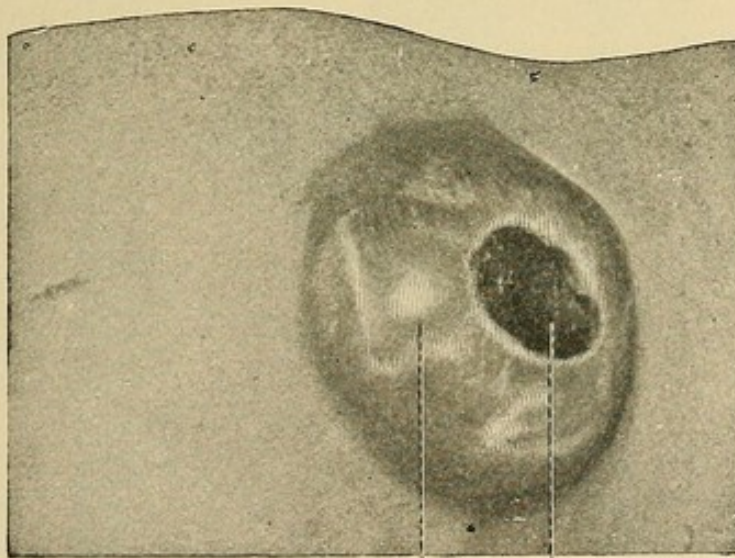
Defect in the Vertebrae, allowing a Protrusion of the Spinal
Cord and Membranes.

CHAPTER LIV.

TUMORS IN THE SPINAL REGION.

A TUMOR in the middle line of the back, most commonly at the lower end of the spine in a newborn babe, that can be made smaller by compression, that has an impulse on crying and straining, and at whose base the outlines of a defect in the vertebræ can be made out is a spina bifida—

FIG. 214



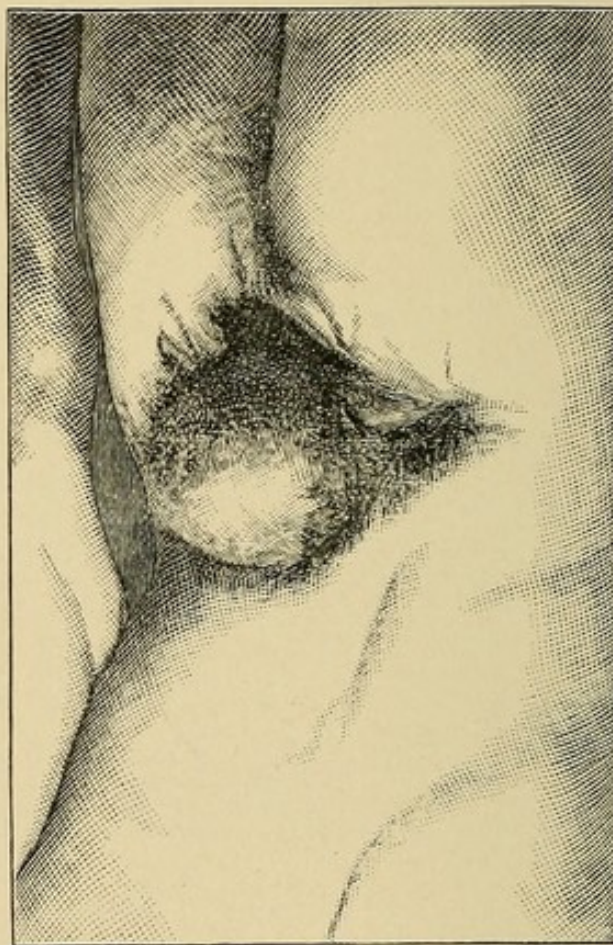
Vascular medullary area.
Serous epithelial zone.

Myelomeningocele. (Von Bergmann.)

i. e., a hernial protrusion of the cord and its membranes. If the tumor has a broad base, and if at the centre of its surface there is an elongated, rounded white area without skin (the medullovascular zone) surrounded by a pearl-gray, glistening zone of irregular shape which merges into a zone of normal skin covered by fine hairs, it is a *myelomeningocele*. Such tumors are frequently associated with paraplegia and

other congenital malformations, such as club-foot, hernia, etc. If the tumor is of mushroom-shape and is covered by normal or thickened skin, beneath which there may be a lipoma, and if it is fluctuant and translucent, it is a *meningocele*, a rather infrequent form of spina bifida. If the tumor has a broad base and is covered with skin that appears normal at the margins of the tumor, and becomes thinner and thinner toward its centre, where it is translucent, glistening,

FIG. 215

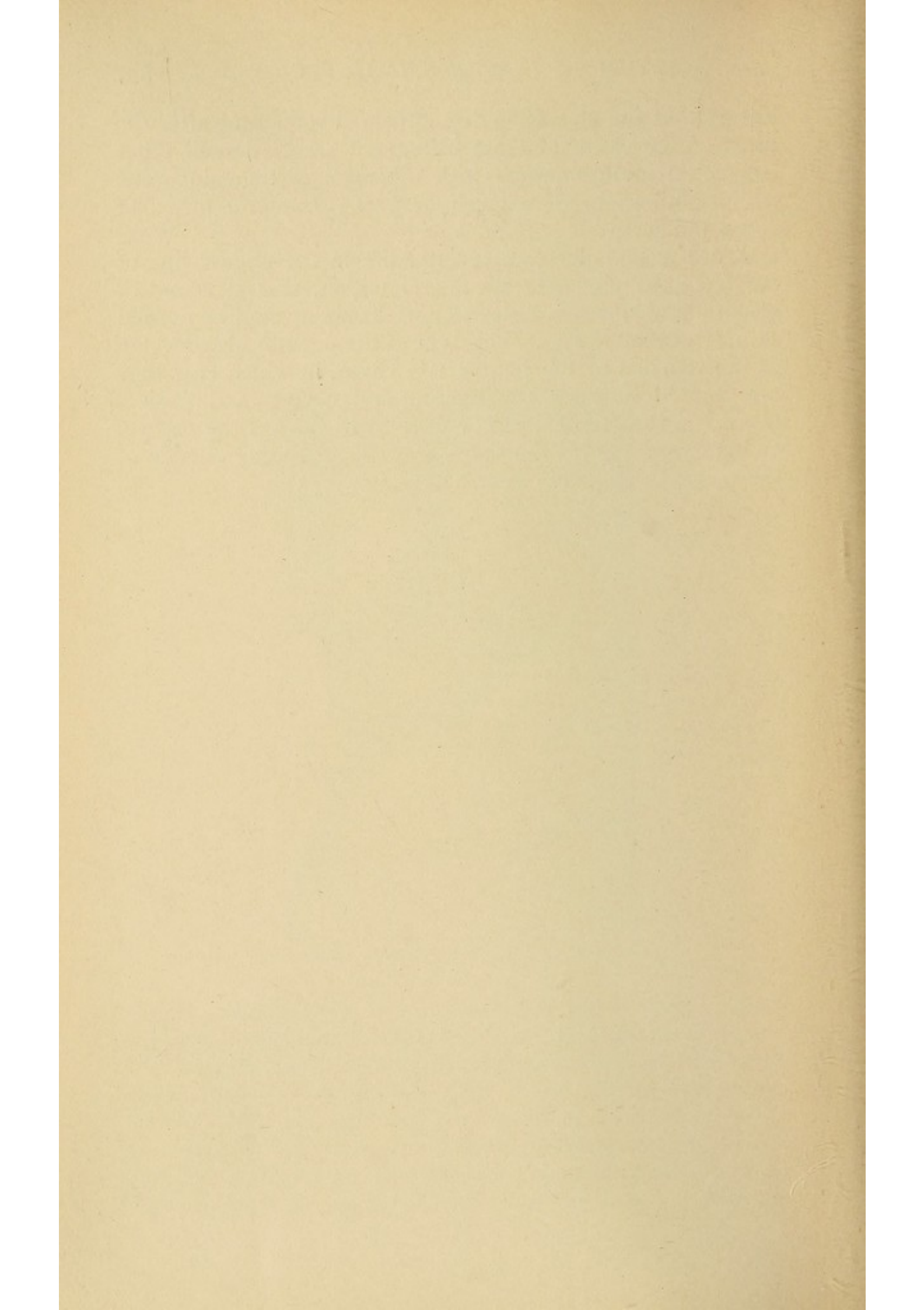


Meningocele. Before operation. (Von Bergmann.)

and of grayish color, it is a *myelocystocele*; if such a tumor contains considerable fluid it is a *myelocystomeningocele*. The absence of a medullovascular zone distinguishes these latter forms of spina bifida from the myelomeningocele. Should the thin cutaneous covering of a myelocystocele ulcerate, the ulcerated area will have some resemblance to the medullovascular zone of a myelomeningocele, and so lead us to a wrong classification of the tumor. But the

history that the ulceration first appeared some time after the birth of the child, and the infrequent association of other congenital malformations with the myelocystomeningocele, will enable us to distinguish between these two forms of spina bifida.

A tumor that develops in later life in the middle line of the back, usually over the sacral region, that is rounded, smooth, and somewhat elastic, and that is covered by normal skin, is a dermoid cyst. This type of tumors may also develop in the remains of the neurenteric canal, in which case they are located between the rectum and coccyx and project inward toward the rectum or downward toward the coccyx, or backward toward the sacrum, even protruding outside of the pelvis through the sciatic foramina.



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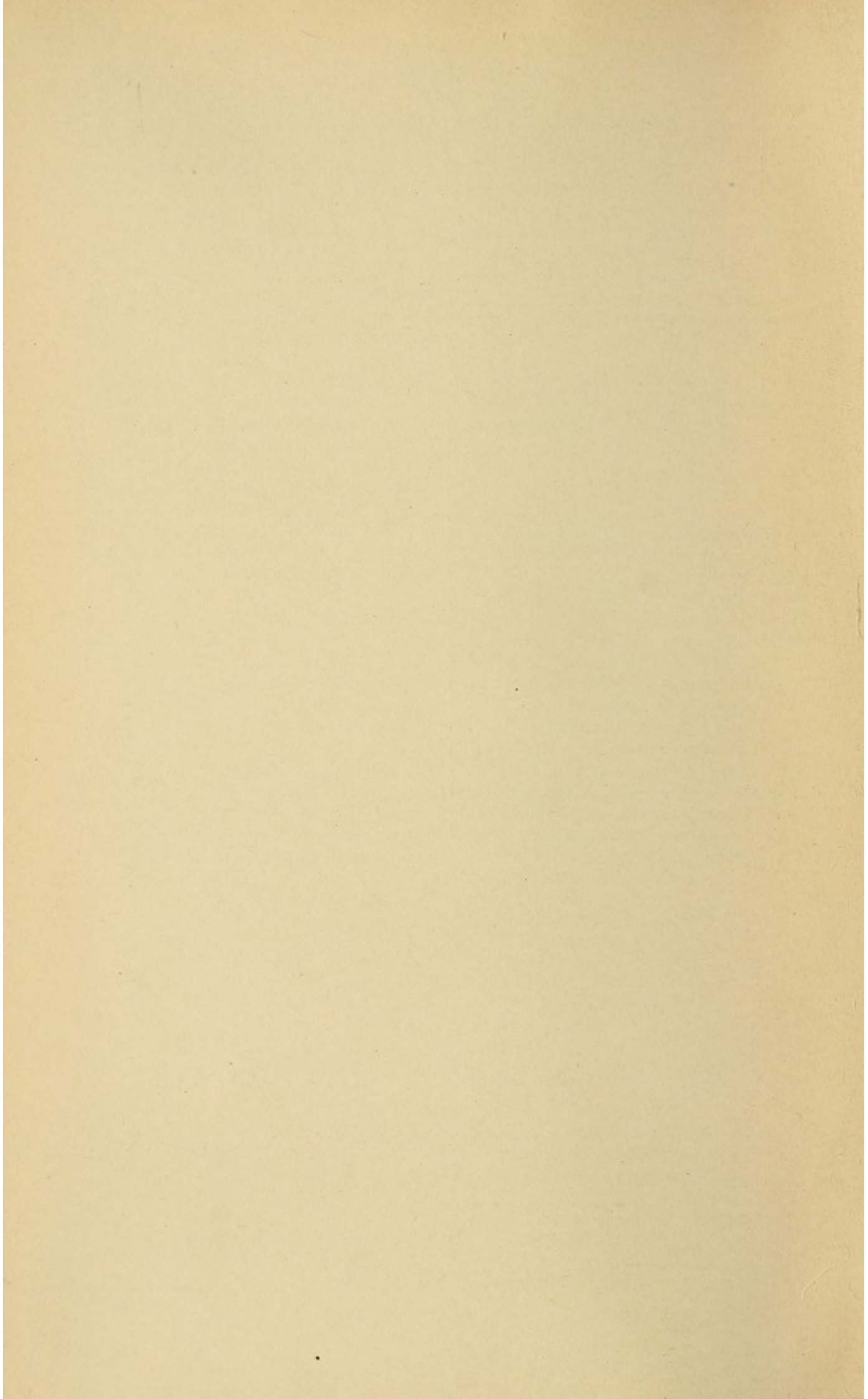
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