PHYSICAL DIAGNOSIS

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PREFACE

In the preparation of this volume the author has had in mind the medical student and the busy practitioner, and it has been his purpose to incorporate in a brief work the principles of physical diagnosis, together with the physical findings in the commoner diseases of the respiratory and circulatory systems. In this connection anatomy and pathology have been considered from the clinical standpoint, emphasis being laid upon these subjects as they influence the physical manifestations of disease of the thorax and abdomen.

In addition to the physical examination of the thoracic and abdominal viscera, it has seemed proper and practical to include in the work the principal diagnostic signs referable to the head, neck, and limbs, together with a minimum examination of the nervous system.

The work has been profusely illustrated, in the belief that free illustration is the nearest approach to personal contact in the teaching clinic.

The author wishes to express his appreciation to Dr. C. E. Shinkle, whose diagnostic table on the Barany Tests is reproduced in the volume, for valuable assistance in preparing the section dealing with these tests. Many illustrations have been taken from other books, all of which have been credited in the text. He also wishes to thank Mrs. T. W. Marks for assistance rendered in the preparation of original drawings for the text; and the publishers for many courtesies during the preparation and publication of the volume.

W. D. Rose.

Little Rock, Ark.

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SECTION I

CHAPTER I

CLINICAL ANATOMY OF THE THORAX

The thorax, or chest, composed of the bony structures, the vertebral column and ribs, clothed with the associated soft structures, comprises two large chambers, one on either side of the median line, the *pleural cavities,* intervening between which is the *mediastinum.*

The *pleural cavities,* occupying the lateral regions of the thoracic cavity, extend upward into the base of the neck to the extent of one to one and one-half inches above the clavicle, and downward as low as the attachment of the diaphragm to the lateral thoracic walls. The pleural cavity is lined by a thin serous membrane, the *pleura,* which clothes its walls, in which situation it is termed the *parietal pleura.* The same membrane is reflected on to the lung at its root, clothing the external surface of the lung completely and dipping into the fissures, the *visceral pleura.* During health the pleural membrane is moistened with a small amount of serous fluid, which causes the visceral and parietal pleurae to glide noiselessly over each other during the movements of respiration. In inflammation of the membrane, however, the pleural surface becomes roughened and coated with a variable amount of fibrinous exudate, giving rise to a grating sound during the respiratory movements, the friction rub.

The *mediastinum,* the portion of the thoracic cavity intervening between the pleural cavities, bounded anteriorly and posteriorly by the chest walls, and laterally by the reflections of the parietal pleura from the anterior to the posterior wall of the chest, is divided into four parts, the superior, posterior, anterior, and middle mediastina.

The *superior mediastinum* is the portion of the mediastinal cavity situated above the lower border of the manubrium sterni anteriorly and the lower border of the fourth dorsal vertebra posteriorly. The principal structures occupying the superior mediastinum are the trachea, esophagus, thoracic duct, the arch of the aorta, and the great veins entering the base of the heart.

The *posterior mediastinum* is formed by the downward continua-

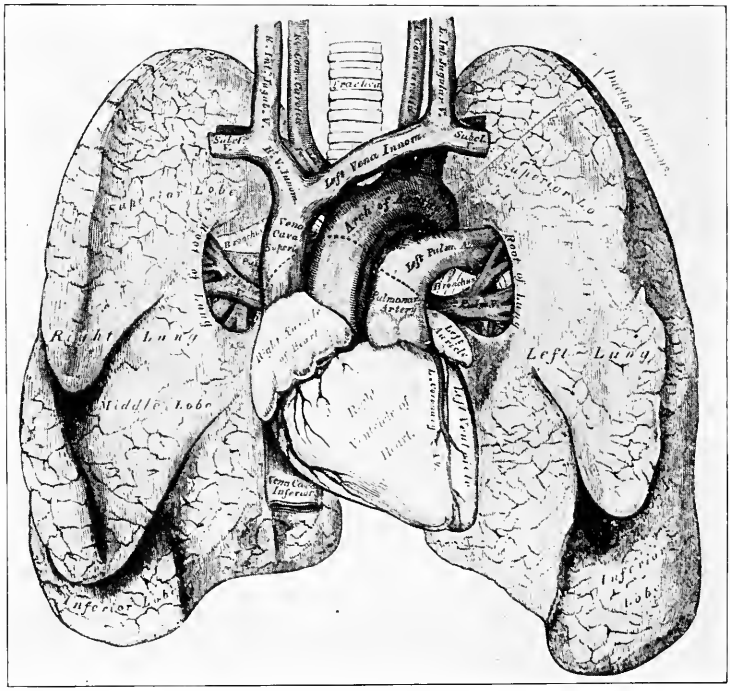


Fig. 1.—Front view of the heart and lungs. (From Gray.)

tion of the posterior portion of the superior mediastinum, extending downward to the upper diaphragmatic surface, having anterior to it the pericardium and heart. It contains the thoracic aorta, esophagus, thoracic duct, pneumogastric nerves, and the azygos veins, as well as the mediastinal lymphatic glands, which are sometimes the seat of enlargement or malignant disease.

The *middle mediastinum*, situated below the anterior portion of the superior mediastinum, and between the anterior and posterior mediastina, is the most important portion of the mediastinal cavity from the standpoint of the student of physical diagnosis, as it contains the pericardium and heart, the ascending aorta, the bifurca-

tion of the trachea, the bronchi and bronchial glands, the pulmonary artery with its two primary branches, the lower portion of the superior vena, and its junction with the azygos veins.

The *anterior mediastinum* is a narrow space situated behind the sternum with the pericardium and reflections of the parietal pleura posteriorly and laterally. It is of little diagnostic interest, save that it contains a few lymphatic glands which may become the seat of malignant disease.

The thoracic viscera comprise the trachea and bronchi, the lungs, the pericardium, and the heart, with the great vessels arising from it.

The *trachea* extends almost vertically downward from the lower border of the larynx, at the level of the sixth cervical vertebra, to

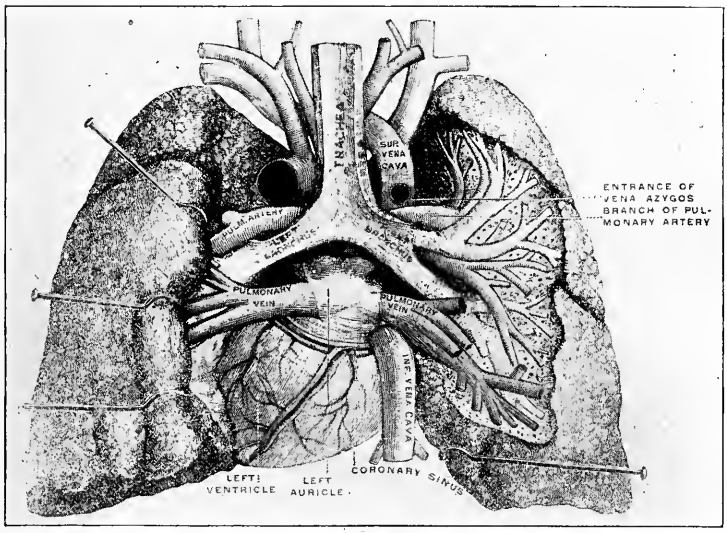


Fig. 2.—Pulmonary veins, seen in a dorsal view of the heart and lungs. The lungs have been pulled away from the median line, and a part of the right lung has been cut away to display the air ducts and blood vessels. (Gray, after Testut.)

the lower border of the fourth dorsal vertebra, where it divides to form the two primary bronchi.

The *bronchi,* right and left, pass obliquely downward and outward to enter the roots of their respective lungs, the left bronchus being slightly smaller than the right, and pursuing a more oblique course than does the right, which passes more directly downward. As the left bronchus is smaller and more deeply placed in the thoracic cavity than is the right, and as it forms a more acute angle with the trachea than does the corresponding bronchus of the opposite side, physical signs arising within it are not conducted to

the surface of the chest with the same degree of intensity as are similar sounds arising within the right bronchus.

The *lungs,* suspended by their respective roots, and covered upon their surfaces by the visceral pleura, hang free within the pleural cavities. The left lung has two lobes, separated by a fissure which extends well in toward the root of the lung and which is lined by a reflection of the visceral pleura. In inflammation of the pleura the portion of the membrane dipping into the fissure may be the only portion of the membrane involved, leading to a condition of interlobar pleurisy. The upper lobe of the left lung comprises a large portion of the external surface and the entire anterior border of the lung, while the lower lobe comprises the entire base and the greater portion of the posterior border of the lung. This is an anatomical fact of considerable clinical importance, as during physical examinations it is often desirable to ascertain whether a morbid process having its inception in the apex or upper lobe of the lung has progressed to the lower lobe.

The right lung has three lobes which are separated by two fissures. The upper lobe comprises the apex, a little more than half of the external surface and the portion of the anterior border of the lung above the level of the fourth costal cartilage. The lower lobe comprises the entire base of the lung, but only a small portion of its external surface. The middle lobe is a wedge-shaped portion intervening between the upper and lower lobes, comprising the anterior portion of the external surface of the lung below the fourth costal cartilage.

The external surface of each lung is convex, while the internal surface, which is in contact with the mediastinum, presents depressions corresponding to the mediastinal structures with which it is in relation. The internal surface of each lung is marked by a rather deep depression, which receives the pericardium with the heart, this depression being considerably deeper upon the left lung, owing to the projection of the heart toward the left side of the thoracic cavity. Just above and behind this depression each lung presents the hilus or pulmonary root for the entrance of the primary bronchus, blood vessels, lymphatics, and nerves of the lung, while extending downward from the hilus is a fold of the reflected pleura, the ligamentum latum pulmonis.

The internal surface of the left lung is traversed by a fairly deep groove for the lodgment of the aorta, which curves above the left bronchus and descends behind this tube. A second groove passes

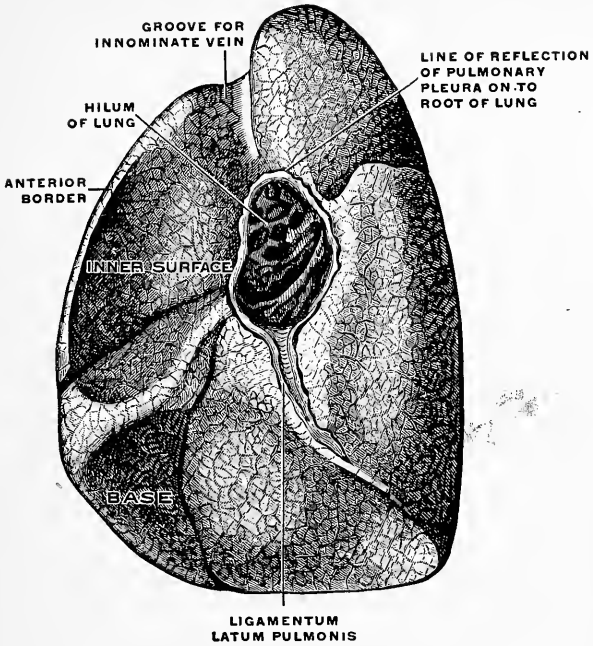


Fig. 3.—Mediastinal surface of right lung. (From Gray.)

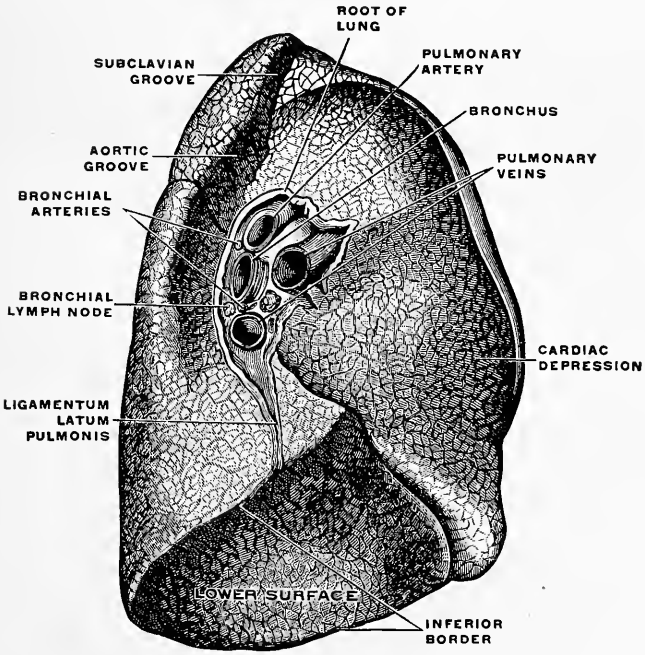


Fig. 4.—Mediastinal surface of left lung. (From Gray.)

upward from the aortic groove, where that vessel arches over the left bronchus, which lodges the subclavian artery.

The internal surface of the right lung is traversed by a groove, which, arching over the right bronchus, lodges the vena azygos major, while extending upward from this groove is a second depression, which lodges the superior vena cava.

The internal structure of the lung is very intimately related to many symptoms and signs arising during disease of the pulmonary organs. The bronchi, which enter the lungs at the hilus, branch dichotomously, until very fine branches, termed bronchioles, are formed.

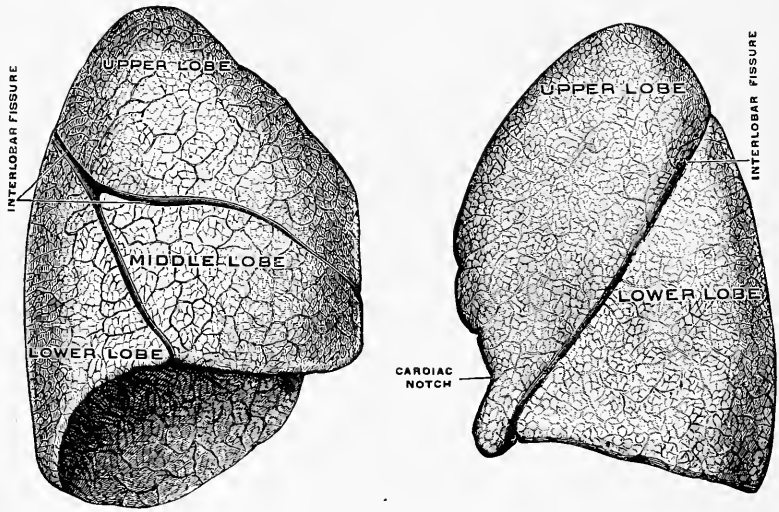


Fig. 5.—External surfaces of right and left lungs. (From Gray.)

In the bronchiole there is a gradual transition of the stratified columnar ciliated epithelium of the bronchi into simple columnar epithelium, which in turn, near the distal extremity of the terminal bronchiole gives place to small groups or islands of the flat, non-nucleated epithelial cells, *respiratory epithelium*. The epithelial lining of the terminal bronchiole rests upon a thin basement membrane, beneath which is a tunic containing numerous elastic fibers and circularly disposed smooth muscle fibers, spasm of which may play a part in the production of the paroxysm of bronchial asthma.

Each terminal bronchiole leads to an irregularly pyramidal chamber, the *infundibulum*, which constitutes "the blood-vascular unit" of the lung. The walls of the infundibula comprise a series

of blind pouches, the *alveoli,* which are lined with a single layer of flat respiratory epithelium resting upon a delicate basement membrane, containing many elastic fibers, which afford to the lung an elasticity or resilience, which plays an important part in the phenomenon of expiration.

The infundibula are invested by a dense capillary net-work

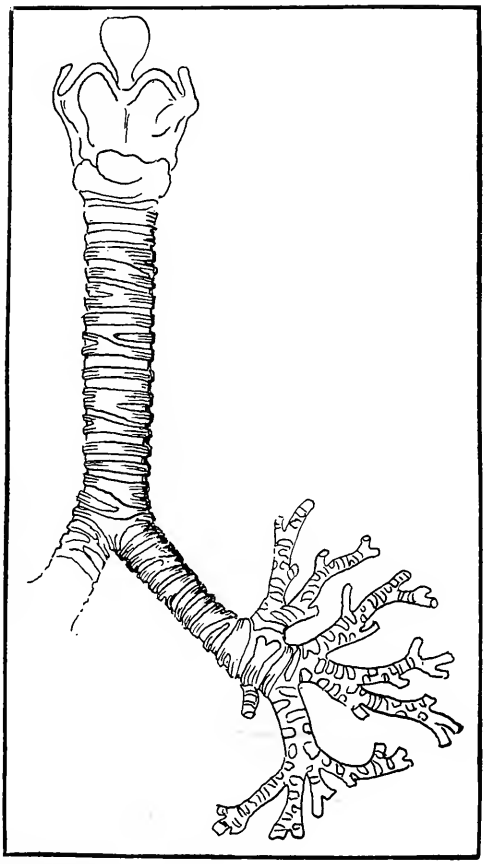


Fig. 6.—The bronchial tree. The walls of the bronchi contain cartilage in incomplete rings or plates distributed about their entire circumference. The cartilage and the elastic tissue make the tubes firm-walled; only the fine branches of one mm. or less in diameter have no cartilage, and are consequently collapsible. (From Brown.)

derived from branches of the pulmonary artery, which accompany the bronchi and bronchioles, the capillary plexus surrounding each infundibulum being disposed in a single layer, with no communication with the net-work of neighboring infundibula. The blood in these capillaries is very intimately exposed to the air

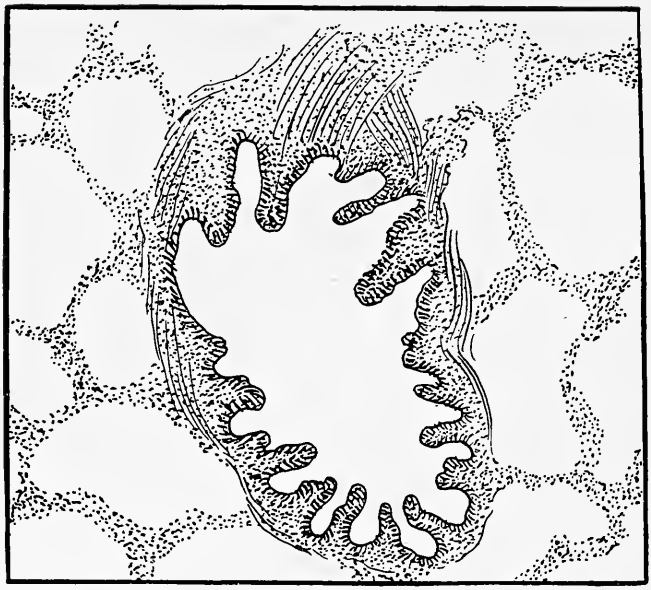


Fig. 7.—The bronchiole. (Highly magnified.) The bronchioles are irregular tubules of a diameter of one mm. or less. The walls consist of a thin flattened epithelium plus a thin layer of non-striated muscle. (Brown, after Böhm, Davidoff, and Huber.)

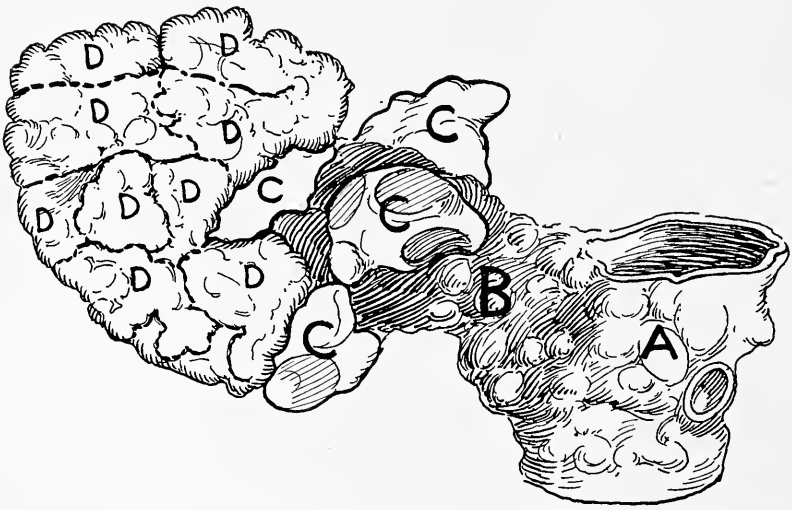


Fig. 8.—The branches of a bronchiole. Each bronchiole has numerous divisions; all are irregular. The final endings, the alveoli, are irregular air cells with thin walls. The cross section of the alveoli arising from one bronchiole is far greater than that of the bronchiole. *A*, respiratory bronchiole; *B*, alveolar ducts; *C*, atria; *D*, air sacs. The finer markings show the alveoli. (From Brown.)

in the infundibula, as they are separated only by three very thin membranes; namely, the endothelium of the capillary wall, the delicate basement membrane of the infundibula, and the single layer of flat epithelium lining the infundibula.

In hypertrophic emphysema, when the inter-alveolar septa are destroyed, as the capillary plexus of each infundibulum is distinct and has no connection with that of neighboring infundibula,

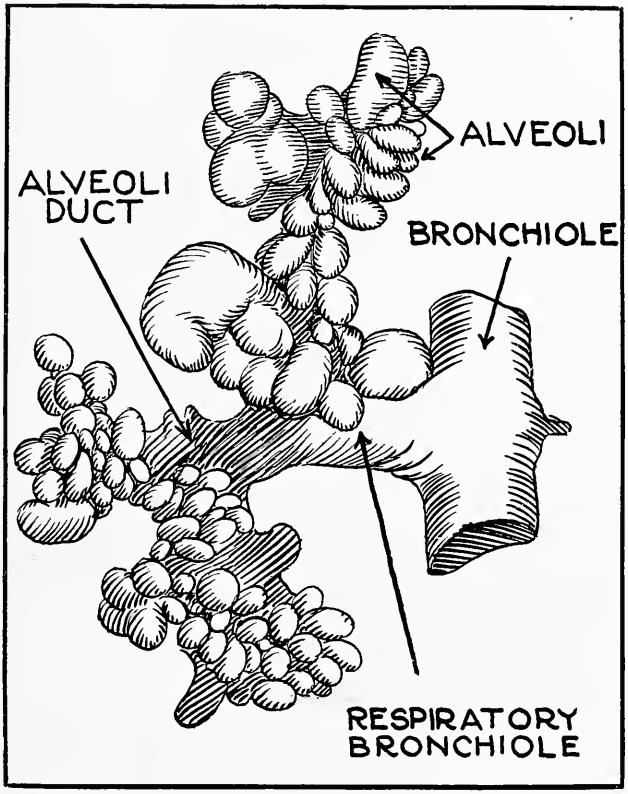


Fig. 9.—The branches of a bronchiole, showing irregularities and bulgings which air from a remote air cell traverses to get to the main bronchiole of a lobule. (Brown, after Gray.)

the amount of blood exposed to the air in the large cavities which are formed by coalescence of several infundibula is greatly reduced, leading to dyspnea on slight exertion in this class of patients.

The lymphatics of the lungs drain into the deep pulmonary lymph nodes and the bronchial and mediastinal glands, with the result that these glands are early involved in tuberculous infec-

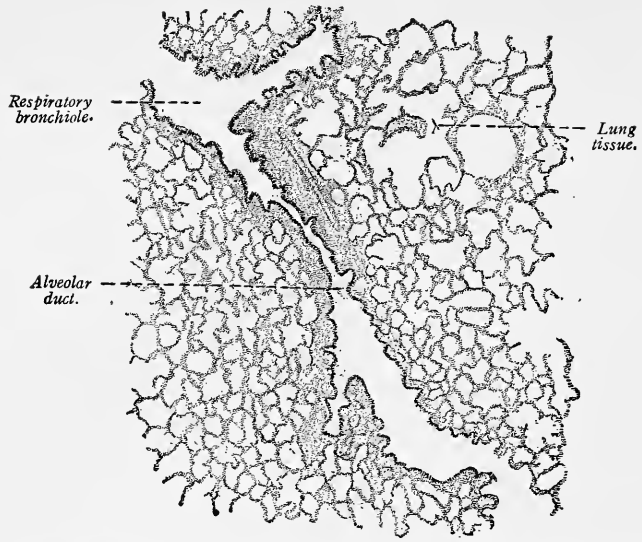


Fig. 10.—The respiratory bronchiole and alveoli in cross section. The broad expanse of alveoli comprises a maze of blind pockets in which air may wander around before reaching the respiratory bronchiole. Even after reaching the respiratory bronchiole, the bulgings and indentations of the walls offer additional pockets for air to enter and leave before reaching the bronchiole. (Brown, after Böhm, Davidoff, and Huber.)

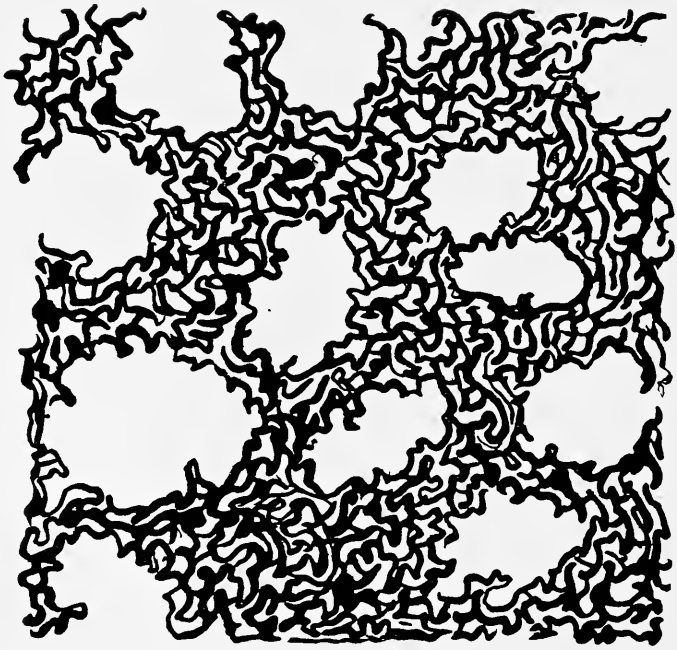


Fig. 11.—Pulmonary capillaries. The walls of the alveoli are thickly studded with capillaries; any marked alteration of alveolar air tension will therefore have a profound effect upon the circulation. (Brown, after Böhm, Davidoff, and Huber.)

tion of the lungs, or when the lung becomes the seat of malignant disease. Similarly these glands serve as filters for the irritant dusts which reach the finer bronchioles and infundibula in pneumonokoniosis.

The clinical anatomy of the heart and pericardium is discussed in a subsequent section.

SURFACE MARKINGS OF THE THORACIC VISCERA

The Pleura.—The surface markings of the pleura upon either side correspond to lines drawn upon the chest wall from either sterno-clavicular articulation to the transverse ridge which marks the junction of the manubrium and gladiolus of the sternum. Thence the anterior borders of the pleural membranes pass downward parallel with each other, slightly to the right of the mid-sternal line to the fifth intercostal space. At this point the two membranes separate.

The right pleura continues directly downward almost to the ensiform cartilage, whence it pursues an outward and downward course toward the spinal column, crossing the seventh rib in the mid-clavicular line, the ninth rib in the mid-axillary line, and the eleventh rib in the scapular line.

At the level of the fifth interspace the left pleura passes outward and downward to the posterior surface of the sixth costal cartilage; crosses this cartilage vertically; and passes downward and backward to the spinal column, occupying a slightly lower level than does the lower border of the right pleura.

The upper level of the supra-clavicular portion of the pleura is indicated by a line drawn obliquely upward and outward from the sterno-clavicular articulation on either side crossing the lower portion of the root of the neck in such a direction as to curve upward and reach the seventh cervical vertebra. The highest point of the curve, which corresponds to the apex of the pleural cavity, is one to one and one-half inches above the clavicle.

Upon the posterior surface of the chest the course of the pleura is represented by drawing a line along either side of the vertebral column, passing vertically downward from the seventh cervical vertebra to the articulation of the eleventh rib with the spinal column, whence the line is continued downward and outward in a gentle curve to meet the line of reflection of the anterior portions of the membrane.

The Lungs.—The borders of the lungs follow closely the line of reflection of the pleura, save that inferiorly the lower borders of the lungs fall short of the pleura by one interspace, being found at the sixth rib in the mid-clavicular line, the eighth rib in the mid-axillary line, and the tenth rib in the scapular line. The interval between the lower border of the lung and the pleura, representing one intercostal space on the surface of the chest, is the complementary sinus, into which the lower border of the lung

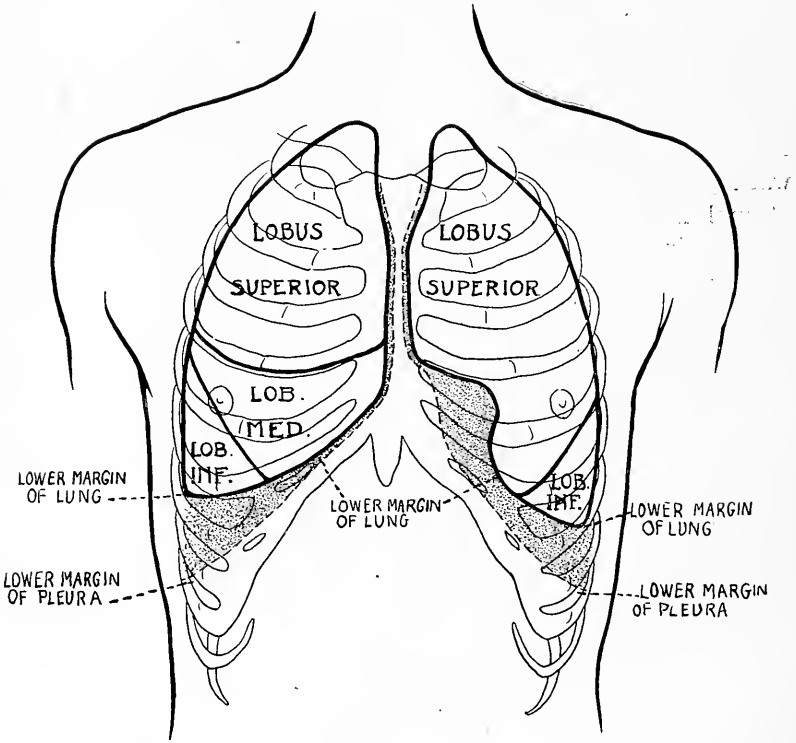


Fig. 12.—Illustrating the normal borders of the lungs and the location of the interlobular septi. Anterior view. (Pottenger, after Corning.)

descends during full inspiration. The surface markings should be borne in mind when determining the total expansion of the lungs by percussion.

Fissures of the Lungs.—The position of the great fissure which intervenes between the upper and lower lobes in both lungs is represented by a line drawn upon the chest wall from the spinous process of the third dorsal vertebra, obliquely downward and forward to the sixth rib in the mid-clavicular line. The fissure in-

tervening between the upper and middle lobes of the right lung is represented by a line drawn upon the surface of the thorax from the apex of the axilla almost horizontally forward to the sternum at the articulation of the fourth left costal cartilage.

Trachea and Bronchi.—The course of the trachea corresponds to a line drawn from the upper margin of the manubrium sterni to

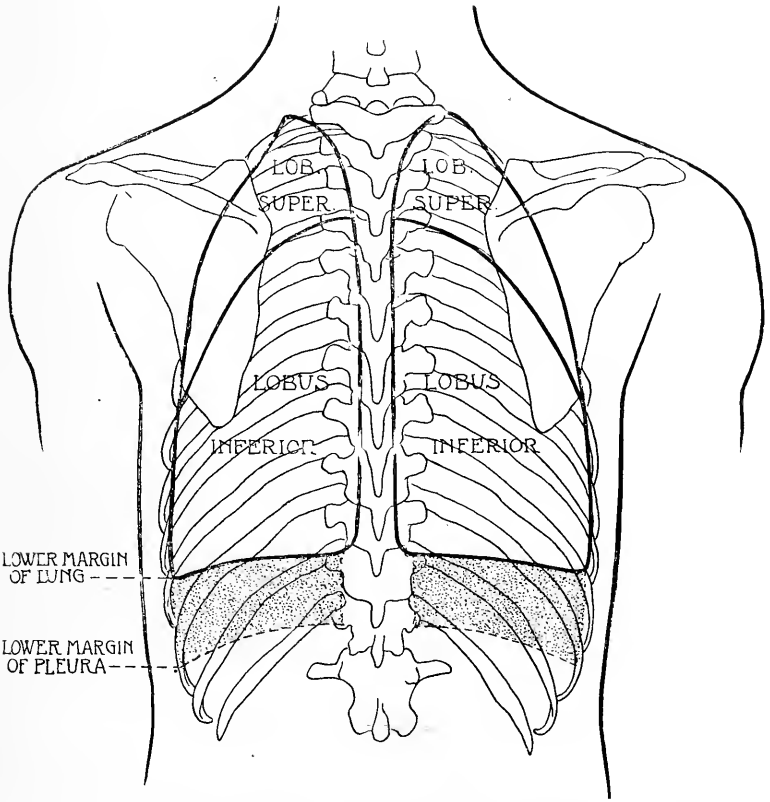


Fig. 13.—Illustrating normal borders of the lungs and interlobular septi. Posterior view. (Pottenger, after Corning.)

the level of the upper margin of the second rib in the median line. At this point the trachea divides into the two primary bronchi, which diverge from each other, passing downward and outward, the right bronchus inclining more directly downward than does the left.

Landmarks of the Normal Chest.—There are a number of normal anatomical landmarks which are visible or palpable upon the surface of the chest, which may be utilized in the description and

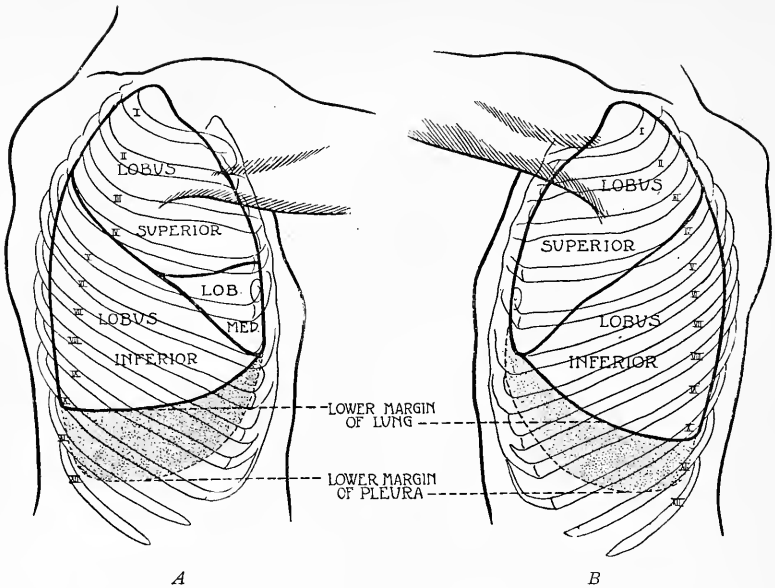


Fig. 14.—Illustrating the normal borders of the lungs and the location of the interlobular septi. Lateral view. *A*, right; *B*, left. (Pottenger, after Corning.)

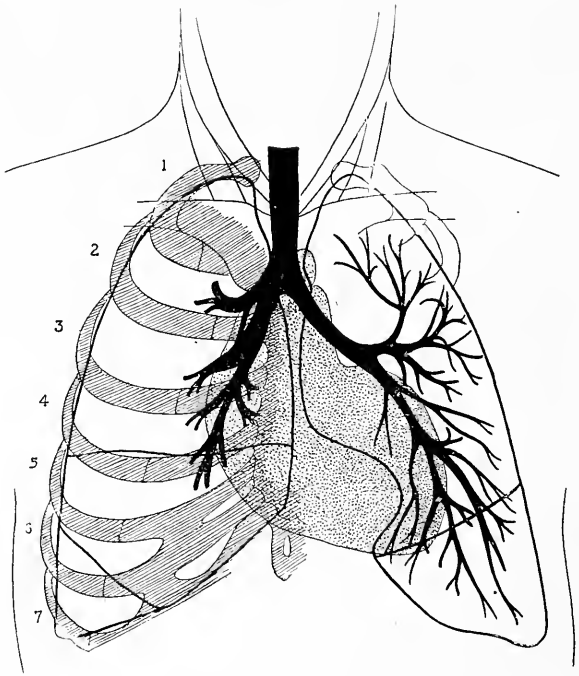


Fig. 15.—Showing the position of the bifurcation of the trachea and the peri-tracheal and peri-bronchial glands projected upon the anterior surface of the chest in a young adult. (Pottenger, after Gerhartz.)

localization of morbid conditions arising within the thoracic cavity.

At the upper border of the manubrium sterni a slight depression is visible, the *supra-sternal notch,* which is frequently the site of abnormal pulsations. During expiration the upper border of the sternum, which limits the supra-sternal notch inferiorly, occupies a position corresponding to the disk between the second and third dorsal vertebræ. The distance between the vertebral column and the notch is approximately two inches, representing the inlet of the thorax.

At the lower extremity of the sternum there is a second depression, the *scrobiculus cordis* or pit of the stomach, which corresponds to the midpoint of the body of the ninth dorsal vertebra.

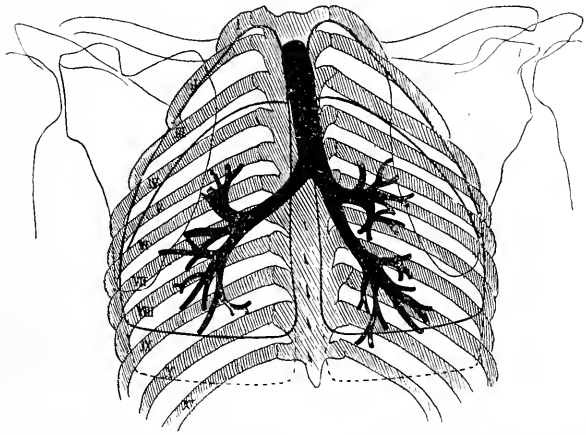


Fig. 16.—Showing the position of the bifurcation of the trachea with the peri-tracheal and peri-bronchial glands projected upon the posterior surface of the chest in a young adult. (Pottenger, after Piersol.)

The *sternum* occupies the median line of the anterior surface of the thorax, surmounted by the supra-sternal notch, with the scrobiculus cordis at its lower extremity. The average length of the sternum is six inches.

The *angulus ludovici,* or *angle of Louis,* is a transverse ridge upon the sternum, marking the junction of the manubrium and gladiolus. It is usually visible and is always palpable. This ridge corresponds to the level of the junction of the second costal cartilage with the sternum and is of service as a starting point in counting the ribs.

The *clavicle,* at all times a conspicuous landmark upon the

anterior thoracic surface, in apical lesions of the lungs becomes very conspicuous, contrasting markedly with the depressions above and below the bone.

Upon either side of the thorax there are twelve *ribs* and eleven *intercostal spaces.* The first rib lies rather deeply beneath the clavicle, but the remaining ribs are readily palpated. Each intercostal space is named in accordance with the number of the rib above it. Thus, the first intercostal space occupies the interval between the first and second ribs.

In *counting the ribs* one of several methods may be employed. In counting the ribs upon the anterior surface of the chest it is convenient to begin at Louis' Angle which, as stated, corresponds to the second chondro-sternal junction. In counting the ribs upon the posterior surface of the thorax the lower angle of the scapula may be used as a starting point, as the tip of this bone overhangs the seventh rib when the chest is in repose. Some clinicians prefer to count the ribs upward by locating the tip of the twelfth rib, which can be palpated in many subjects. In counting the ribs upon the lateral aspect of the thorax the highest digitation of the serratus magnus muscle overlies the sixth rib. The muscle may be rendered tense by directing the patient to raise the arm outward to a horizontal position.

The *mammary gland* in the male is rudimentary. In the female subject, however, it is well developed, extending from the third to the seventh interspace in the mid-clavicular line. In the male subject the *nipple* usually is a reliable guide to the fourth interspace, but in the female it is an entirely unreliable landmark, owing to the pendulous condition of the breast.

The *scapula* overlies the dorsal aspect of the bony thorax, extending along the vertebral column from the second to the seventh rib. Always visible, it becomes excessively so and stands out prominently in the phthisical thorax of pulmonary tuberculosis.

The *spine* in muscular individuals is represented by a median groove, and the spinous processes are hardly palpable. But in thin subjects and in children many of the processes are visible; and when the subject bends the trunk forward they are readily palpated and counted. The spine of the seventh cervical vertebra (vertebra prominens) is always prominent, and may be employed as a starting point in counting the spinous processes. Or the inferior angle of the scapula, which corresponds to the level of the seventh dorsal spinous process may be employed as a starting point

in the enumeration. Lateral or antero-posterior curvature of the spinal column is frequently noted, with or without pathologic significance.

Topographical Regions of the Thorax.—For purposes of clinical description and for convenience in localizing pathologic lesions arising within the thorax, a number of arbitrary regions may be outlined upon the surface of the chest by means of arbitrary vertical and horizontal lines.

Vertical Lines.—The *mid-sternal line* is drawn vertically through the center of the sternum from the midpoint of the upper

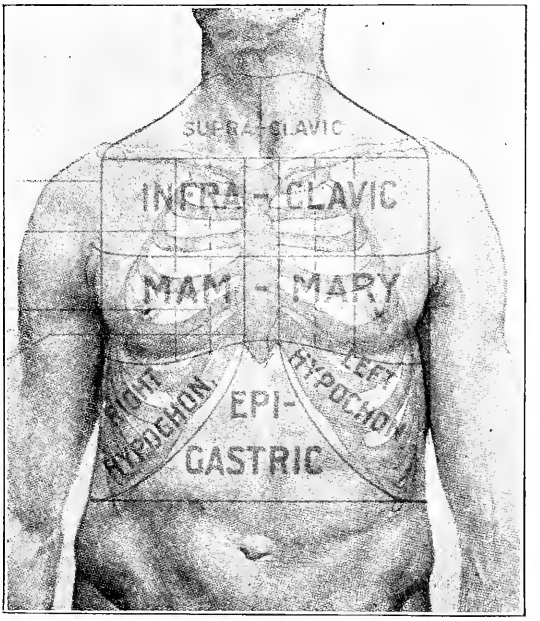


Fig. 17.—Topographical areas of the thorax. Anterior surface. (From Butler.)

border of the manubrium sterni to the tip of the ensiform cartilage.

The *sternal line* is drawn along the lateral border of the sternum from the sterno-clavicular articulation to the tip of the ensiform cartilage, and is continued downward and outward along the lower border of the costal arch.

The *mid-clavicular line* is a vertical line dropped from the midpoint of the clavicle. It often passes through the nipple and is hence termed the *nipple line*, or *mammary line*.

The *para-sternal line* is a vertical line midway between the sternal line and the mid-clavicular line.

The *anterior axillary line* is a vertical line dropped from the anterior axillary fold.

The *mid-axillary line* is a vertical line dropped from the apex of the axilla along the lateral thoracic wall.

The *posterior axillary line* is a vertical line let fall perpendicularly from the posterior fold of the axilla.

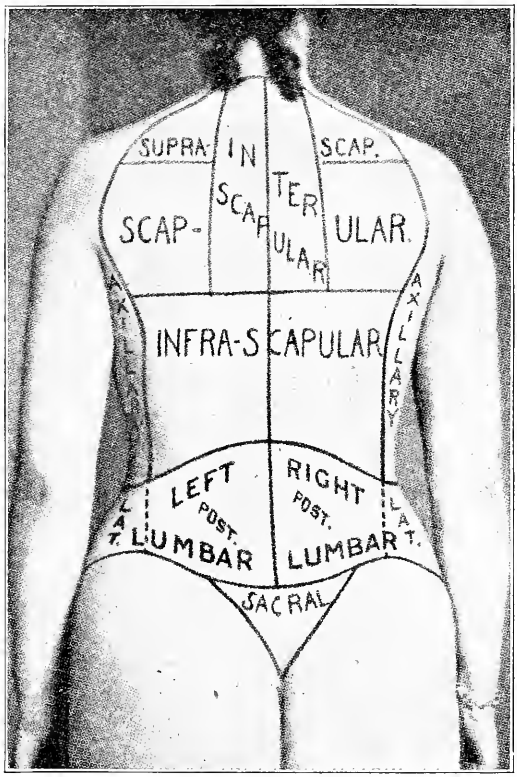


Fig. 18.—Topographical areas of the thorax. Posterior surface. (From Butler.)

The *scapular line* is a vertical line drawn upon the posterior surface of the thorax, passing through the inferior angle of the scapula.

The *mid-spinal line* is a vertical line drawn along the spinous processes of the dorsal vertebræ.

Horizontal Lines.—The *crico-clavicular line* is drawn from the

cricoid cartilage outward with slight inclination downward to the outer extremity of the clavicle.

The *clavicular line* is drawn outward with a slight inclination upward, following the course of the clavicle.

The *third costal line,* extends horizontally outward from the sternal line at the level of the articulation of the third costal cartilage with the sternum, to the anterior axillary line.

The *sixth costal line* extends outward from the sternal line at the sixth chondro-sternal articulation to the posterior axillary line.

The *scapular spinal line* is drawn along the course of the spine of the scapula.

The *infra-scapular line* is drawn horizontally between the inferior angles of the scapulæ.

The *twelfth dorsal line* is drawn from the spinous process of the twelfth dorsal vertebra outward and downward to the posterior axillary line.

Regions.—By means of these arbitrary lines a number of regions are marked off upon the chest wall.

The *sternal region* overlies the sternum, and is bounded above by the supra-sternal notch, below by the scrobiculus cordis, and laterally by the sternal lines.

The *supra-clavicular region* lies above the clavicle, bounded above by the crico-clavicular line, and below by the clavicular line.

The *infra-clavicular region,* lies immediately below the clavicle, and is bounded above by the clavicular line, below by the third costal line, internally by the sternal line, and externally by the anterior axillary line.

The *mammary region,* lies immediately below the infra-clavicular region, limited above by the third costal line, below by the sixth costal line, internally by the sternal line, and externally by the anterior axillary line.

The *hypochondriac region,* lies below the mammary region, between the sixth costal line, the anterior axillary line, and the downward and outward continuation of the sternal line along the line of the costal arch.

The *axillary region* occupies the lateral aspect of the chest, bounded above by the apex of the axilla, below by the sixth costal line, anteriorly by the anterior axillary line, and posteriorly by the posterior axillary line.

The *infra-axillary region,* lying below the axillary region, is

bounded by the anterior and posterior axillary lines, the sixth costal line and the downward continuation of the sternal line.

The *supra-scapular region* overlies the supra-scapular fossa, limited below by the scapular spinal line.

The *scapular region,* overlying the infra-scapular fossa, is limited above by the scapular spinal line, below by the infra-scapular line, externally by the posterior axillary line.

The *infra-scapular region* is limited above by the infra-scapular line, below by the twelfth dorsal line, externally by the posterior axillary line, and internally by the mid-spinal line.

The *inter-scapular region* occupies the interval between the scapular line and the mid-spinal line, being limited below by the infra-scapular line.

SECTION II

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS

CHAPTER II

INSPECTION

Objects and Technic.—In the study and analysis of disease of the respiratory organs inspection is employed to determine the state of the exterior of the thorax, various unilateral and bilateral variations from the normal contour and size of the thorax, the presence of local prominences and depressions, the character and frequency of the respiratory movements of the thorax, the degree of expansion of the two sides of the thorax, and the presence of abnormal pulsations visible upon the surface of the thorax.

During inspection of the thorax the patient's clothing should be removed to the waist, as a full and direct exposure of the chest is essential to a proper examination by all the methods employed in physical diagnosis. During the examination the erect or recumbent posture may be assumed by the patient, preferably the former. The light should fall directly upon the area under investigation, the source of the light passing over the examiner's shoulders. In the course of the examination the examiner should view the chest from the front, from the sides, and from behind. He should then stand above the patient and look downward over the shoulders; and, finally, with the patient in the recumbent posture the thorax should be inspected from below, the examiner standing near the feet of the patient.

During inspection of the thorax, as in all physical examinations, the attitude of the patient should be natural and unconstrained. During inspection of the front of the thorax the patient should sit erect with the arms hanging naturally at the sides. During inspection of the lateral regions of the thorax the patient's hands should be clasped behind the head, allowing a free exposure of the axillary regions.

After inspecting the thorax by direct light, the same procedure should be observed with the patient exposed to oblique illumination. This latter method of examination will often reveal a patch of deficient expansion so slight as to quite escape detection during the examination in direct light.

The Surface of the Chest.—Inspection of the surface of the thorax may reveal abnormalities in the condition of the skin, the subcutaneous tissues and musculature, changes in the superficial vessels of the thorax, or changes in the mammary gland or the ribs.

The *skin* of the thorax in health is smooth and glossy and is lubricated with the proper amount of sebaceous material. The skin of the male chest is frequently clothed with a greater or less abundance of hair, which may obscure the true state of the skin. Certain diseases not referable to the respiratory organs produce pigmentation of the skin, notably hepatic disorders, Addison's disease, chronic malarial poisoning, and the cachexia of malignant disease; whereas other conditions, as anemia and chronic pulmonary tuberculosis, are attended with an abnormally pale and dry skin. Moreover, various skin diseases produce eruptions upon the skin of the thorax, and the skin of this region may be the seat of scars from trauma or syphilis.

The *superficial veins* of the thoracic wall, scarcely visible during health, are engorged and tortuous when intra-thoracic lesions interfere with the return of the blood to the right heart. Thus engorgement or undue prominence of the superficial veins of the thorax may be significant of pressure exerted within the mediastinum by a tumor, aneurism, or enlarged heart. Engorgement of the veins over the lower region of the thorax, communicating with similarly distended veins over the abdomen, associated with the caput medusæ, is indicative of portal vein obstruction.

Edema of the chest wall occurs as a part of general anasarca. Localized edema of the chest wall points to suppurative disease within the thorax, as in empyema necessitatis.

Condition of the Subcutaneous Tissues and Muscles.—Wasting of the subcutaneous tissues and intercostal muscles accompanies the emaciation of chronic tuberculosis, diabetes, and paralysis of the intercostal muscles, rendering the ribs unduly prominent.

The Ribs.—Not particularly perceptible upon inspection save in the lower axillary region, the ribs in chronic wasting diseases become very conspicuous landmarks upon the thorax. In rickets

there is permanent deformity of the terminal ends of the ribs, constituting the Rachitic Rosary of this disease.

Mammary Gland.—In males a peculiar hypertrophy of the usually rudimentary mammary gland has been noted in some cases of pulmonary tuberculosis.

The Size and Contour of the Thorax.—The size and shape of the thorax are remarkably perverted from the normal by several diseases of the respiratory system, as well as in certain diseases of other origin, and occasionally as the result of occupation. In certain of these diseases the chest becomes permanently fixed in deformity, and the diagnosis may at times be made by a glance at the configuration of the chest.

The Normal Thorax.—The normal thorax is only recognized after long clinical experience. In the normal thorax the shoulders are usually on a level, the clavicles are not unduly prominent, al-

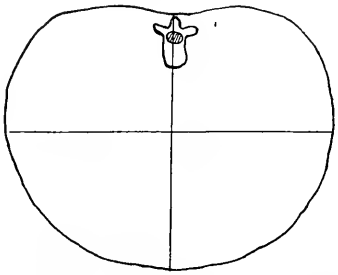


Fig. 19.—Cross section of normal chest. (Redrawn from Gee.)

though slight depressions in the supra- and infra-clavicular fossae are not incompatible with good physical health. The two sides of the normal chest are seldom exactly symmetrical. As a rule the right side is better developed than is the left. The sternum in the normal thorax presents a slight forward inclination from its top to its lower extremity, and usually a visible transverse ridge at the angle of Louis. The bony thorax is clothed with a healthy musculature so that the interspaces are neither shrunken nor protruding. The skin is smooth and moistened by a moderate amount of sebaceous material.

A cross section of the normal chest is elliptical, the transverse diameter exceeding the antero-posterior diameter by one-fourth. In the thorax of the child this relation between the transverse and antero-posterior diameters does not obtain, as the child's thorax is almost circular upon cross-section.

BI-LATERAL DEFORMITIES OF THE THORAX

The Emphysematous Thorax (Barrel Chest).—The emphysematous thorax, or barrel chest, occurs in subjects of hypertrophic emphysema. This type of thorax is increased in all of its diameters, but particularly in the antero-posterior, so that this diameter exceeds the transverse diameter of the chest. The chest on cross-section represents almost a circle, with the greatest degree of enlargement near the middle of the sternum. Thus, the chest in subjects of this disease is thick, the shoulders are raised and bowed forward, while the Angle of Louis is very prominent. The normal dorsal curvature of the spinal column is accentuated, and, with the drooping shoulders, gives to the patient a stooping attitude. The ribs meet the sternum at an abnormally obtuse angle, producing an abnormally wide costal angle. The scapulae are closely applied to the back of the bony thorax. The entire thorax appears to have become shortened and thickened.

The emphysematous thorax is rigid, permitting but little expansion; but in compensation for this deficiency, during respiration it rises and falls as one piece. Not infrequently there is visible retraction of the lower interspaces during inspiration, while expiration is always prolonged.

The Phthisical Thorax.—The phthisical thorax presents a marked contrast to the emphysematous chest. In the phthisical or paralytic thorax the antero-posterior diameter is markedly decreased, so that the thorax is long, and flat, presenting a picture of extreme emaciation, owing largely to wasting of the pectoral and deltoid muscles. The clavicles are very prominent, contrasting markedly with the recession of the supra-clavicular and infra-clavicular regions above and below.

The long, flat thorax is surmounted by an apparently abnormally long, tapering neck, in which the sterno-mastoid muscles and the larynx stand out prominently.

The scapulae are very prominent, standing out upon the posterior thoracic surface like wings, leading to the name "Alar Thorax" sometimes applied to this type of chest.

The ribs pursue a very oblique course downward from the spinal column and bend sharply upward again to meet the sternum, producing a very acute costal angle. The intercostal spaces are widened.

The Rachitic Thorax.—Rickets is accompanied by a character-

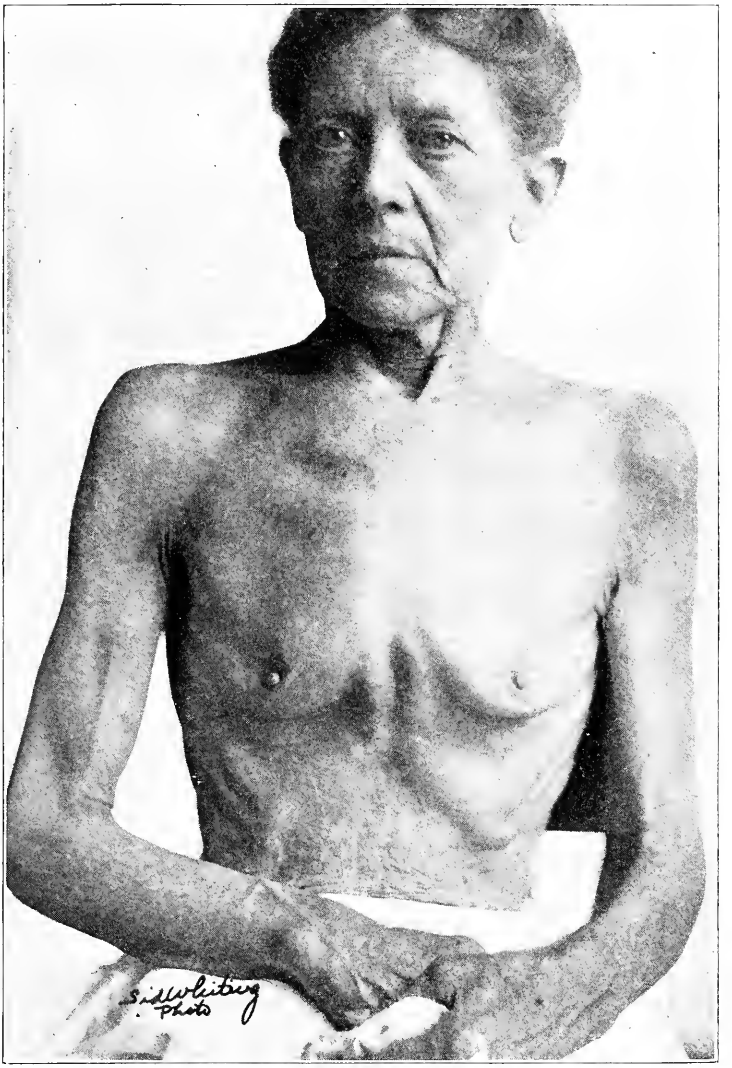


Fig. 20.—Emphysemic chest. (Front view.) With asthma, emphysema invariably develops. If the tissues of the alveoli are more resistant than those of the bronchi, asthma may become more permanent than emphysema; the reverse may also be the case. If asthma endures for years, however, there is sure to be more or less permanent emphysema. In an emphysemic chest the sternum is high, and antero-posterior diameter exceeds the lateral, and the ribs have a more nearly horizontal course than usually obtains. (From Brown.)

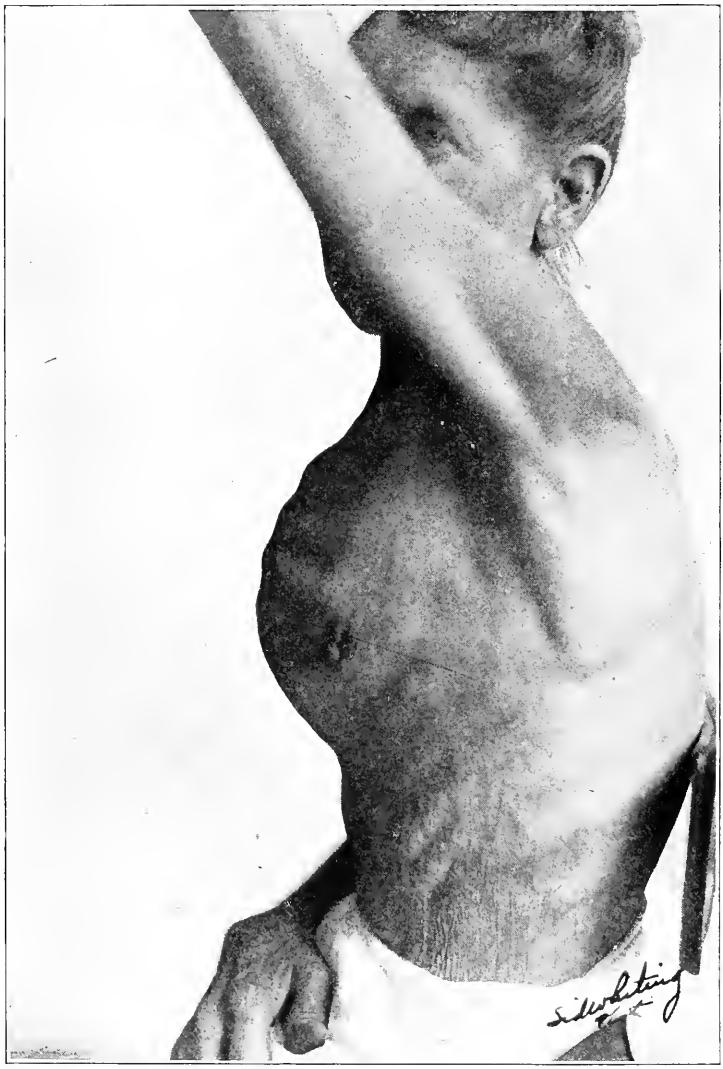


Fig. 21.—Emphysemic chest. (Lateral view.) The sternum and anterior part of the chest have been forced into the position of extreme inspiration. (From Brown.)

istic deformity of the thorax. In this type of thorax the antero-posterior diameter is increased, while the transverse diameter is decreased by a lateral compression of the soft ribs, causing the sternum to jut forward and become uncommonly prominent. A cross-section of the rachitic chest shows a marked increase in the antero-posterior diameter with an actual decrease in the transverse diameter of the thorax.

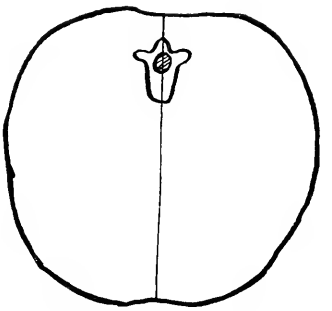


Fig. 22.—Transverse section of emphysematous thorax. (Redrawn from Gee.)

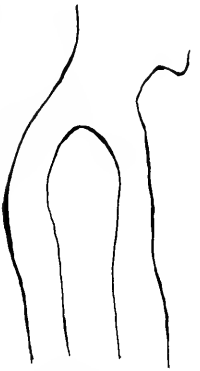


Fig. 23.—Lateral contour of phthisical thorax.

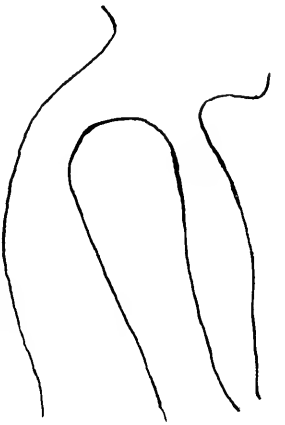


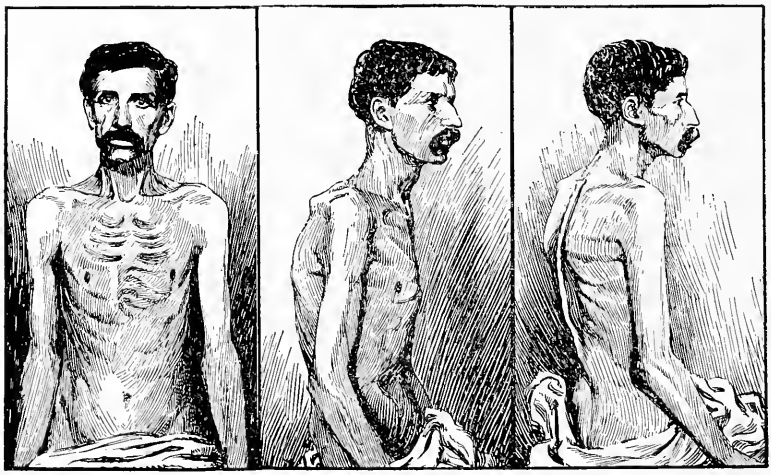
Fig. 24.—Lateral contour of emphysematous thorax.

At the junction of the ribs with their costal cartilages the rachitic thorax presents a series of nodular swellings, due to swelling of the osteo-cartilaginous junctional tissues, the rachitic rosary. As a result of the lateral compression of the thorax the costal angle is abnormally acute. It is not uncommon to find the lower ribs anteriorly flaring outward.

Various kinds of spinal curvature occur with the rachitic thorax, as kyphosis, lordosis, or scoliosis.

The rachitic chest is not significant of any disease of the respiratory organs; but the compressed thorax is too small for the lungs to properly expand and develop and hence it predisposes to disease of the organs of respiration.

Harrison's Sulcus.—Harrison's Sulcus is a depression or groove extending upon either side of the thorax from the ensiform process downward and outward toward the axillary regions. It is often a sign of early rickets; it almost invariably accompanies the rachitic thorax and it is also caused in early life by obstructive



A *B* *C*

Fig. 25.—Phthisical chest. *A*, anterior view; *B*, lateral view; *C*, posterior view. (From Pottenger.)

lesions of the upper air passages, in which event it is to be attributed to the external atmospheric pressure pressing upon the soft ribs which are not supported by full inflation of the lungs. The deformity is particularly common in negroes.

The Pigeon Breast (Keel Breast).—In this type of thoracic deformity the ribs are compressed and straightened in front of their angles, causing the sternum to jut forward and become unduly prominent. On cross-section the thorax is roughly triangular. The anterior portion of the thoracic cavity is encroached upon by the incurvation of the ribs, while the posterior portion of the cavity

is compensatorily voluminous. Harrison's Sulcus is often present upon the surface of the chest.

The pigeon breast occurs most frequently in cases of advanced rickets; but it may also be caused by the paroxysms of pertussis, or by greatly hypertrophied tonsils in early life.

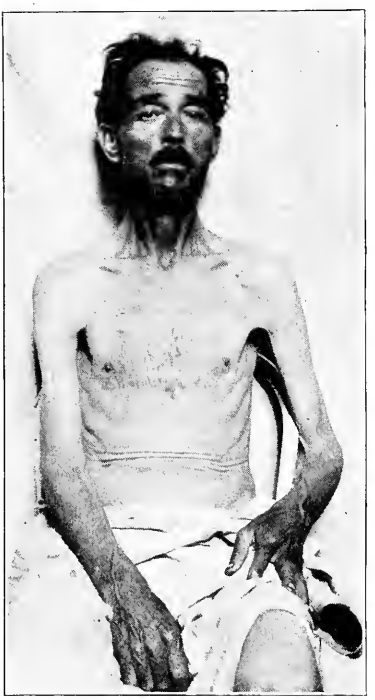


Fig. 26.—Phthisical thorax.

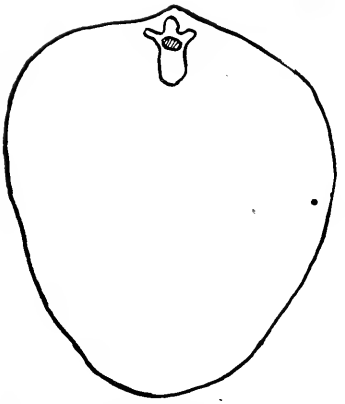


Fig. 27.—Transverse section rachitic thorax. (Redrawn from Gee.)

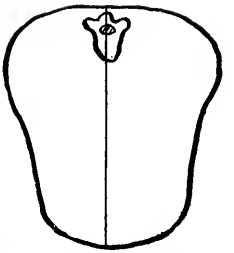


Fig. 28.—Transverse section of pigeon breast. (Redrawn from Gee.)

The Funnel Chest.—In this type of thoracic deformity the lower end of the sternum is depressed, the hollow or depression occasionally extending as high as the third rib. The funnel-chest is in some instances congenital; it has been noted in connection with rickets; and it sometimes develops as the result of an occupation which requires an instrument to be held constantly against the lower portion of the sternum. Thus, the deformity has been noted in cobblers, resulting from the continued pressure of the last. The funnel-chest is not a sign of pulmonary disease; but, by decreasing the capacity of the thorax, predisposes to disease of the broncho-pulmonary system.

UNI-LATERAL DEFORMITIES OF THE THORAX

Uni-lateral Enlargement, or bulging of one side of the thorax, is significant of fluid or gas in the pleural cavity or of intra-thoracic neoplasm. Thus, uni-lateral bulging may signify the presence of pleurisy with effusion, hemothorax, pyopneumothorax, or hydropneumothorax. Similarly, uni-lateral enlargement may be significant of compensatory emphysema, arising as a result of vicarious distention of the lung to compensate for crippling of the opposite lung by fibrosis, in which case the shrinking of the opposite side of the chest adds materially to the apparent bulging of the emphysematous side. When the cause of the bulging is fluid, it is most pronounced in the lower portion of the thorax, the costal angle is rendered more obtuse, and the interspaces are apt to bulge. The spinal column usually deviates toward the side of the effusion.

Uni-lateral Contraction of the Thorax.—Uni-lateral contraction of the thorax is indicative of a diminution of the intra-thoracic contents on the affected side. Thus, it may be significant of collapse of the lung following bronchial obstruction, destruction of pulmonary tissue in the course of pulmonary tuberculosis, chronic fibrosis of the lung, chronic adhesive pleurisy, in which adhesions obliterate the pleural cavity and draw the chest wall inward, or of chronic pressure exerted upon the lung by pleurisy with effusion.

All of these conditions are usually accompanied by compensatory emphysema of the opposite lung, thus causing accentuation of the disproportion between the two sides of the thorax. In addition to the diseased side being smaller than is its fellow, the shoulder of the affected side droops, and the spinal column is found bowed with its concavity toward the retracted side, while the interspaces on this side are narrowed or the ribs actually overlap.

LOCAL DEFORMITIES OF THE CHEST

Local Enlargement.—Local enlargement of the thorax possesses varying significance, depending upon the situation of the bulging. In the cardiac region local enlargement may be due to immense hypertrophy of the heart, pericarditis with effusion, pneumopericardium, aneurism of the ascending portion of the aorta, localized pleurisy with effusion, a mediastinal tumor pushing the heart forward, or an abscess of the lower portion of the sternum.

A local enlargement over the left hypochondriac region points to splenic enlargement; while a similar enlargement over the right hypochondriac region points to enlargement of the liver due to cyst, abscess or simple enlargement, or to low right-sided pleurisy with effusion.

Local Retraction of the supra-clavicular and infra-clavicular regions, with unduly prominent clavicles, usually point to apical pulmonary tuberculosis, cirrhosis of the lung, or to the traction of pleural adhesions. Local retractions over other regions of the thorax point to the presence of bronchiectatic or tuberculous cavities, or the traction of pleural or pericardial adhesions.

RESPIRATORY MOVEMENTS OF THE THORAX

The respiratory movements of the thorax comprise an inspiratory excursion and an expiratory recession, the two being followed by a slight pause. The inspiratory movement is an active process, initiated by muscular contraction, whereas the expiratory recession is a passive process, the chest collapsing with the relief of the muscular contraction. Of the two movements the expiratory recession is of longer duration than is the inspiratory excursion.

The *frequency* of the respirations in health varies with the age of the subject. In the healthy adult the number varies from 14 to 18 per minute, being slightly more rapid in women than in men. In the newly born the respiratory rate is 44 per minute, while at five years of age it is 26 per minute.

The *character* of the respiratory movement differs in the two sexes. Thus, in women the movements of the thorax are much more conspicuous than are those of the abdomen, the *costal type* of respiration. In the adult male, on the contrary, the abdominal muscles play quite a part in the respiratory movements, the *costo-abdominal type* of respiration.

However, exaggeration of either the costal or costo-abdominal type of respiration possesses diagnostic significance, and is pathological. Thus, purely costal respiration may be caused by immobilization of the diaphragm by paralysis or sub-diaphragmatic pressure due to ascites, abdominal tumor, peritonitis or tympanites. Similarly, exaggerated or purely abdominal respiration

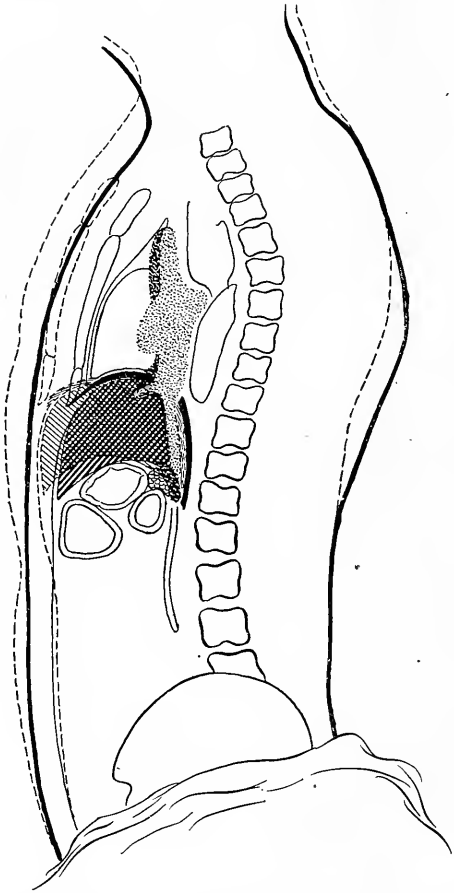


Fig. 29.—Illustrating the movements of the diaphragm and thoracic and abdominal walls, as well as the change in position of the intra-thoracic and intra-abdominal viscera, during respiration of the abdominal type. The movements are from the solid lines on expiration to the broken lines on inspiration. (Pottenger, after Hasse.)

points to disease in the thorax, as pulmonary tuberculosis, massive pleurisy with effusion, massive pneumonia, scleroderma of the chest wall, premature calcification of the costal cartilages, or inhibition due to the pain of pleurodynia or fracture of a rib.

Litten's Diaphragmatic Phenomenon.—During the movements of

the thorax in normal respiration a phenomenon may be observed which is of great diagnostic significance in the analysis of morbid conditions arising within the chest or abdomen. This is an undulation or "shadow" which may be observed upon the chest wall during inspiration and expiration, and which has received the name Litten's diaphragmatic phenomenon. The undulation or "shadow"

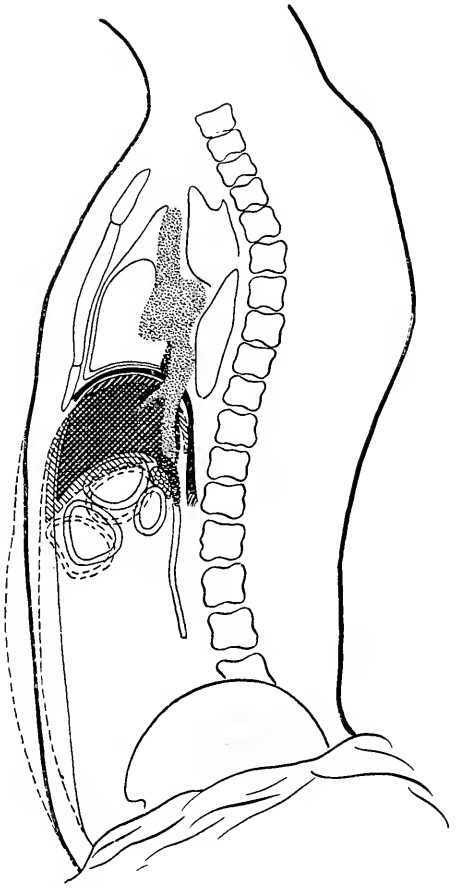


Fig. 30.—Showing the movements of the diaphragm and thoracic and abdominal walls, as well as the change in position of the intra-thoracic and intra-abdominal viscera, when combined thoracic and abdominal breathing are pronounced. The movements are from the solid lines on expiration to the broken lines of inspiration. (Pottenger, after Hasse.)

is initiated by the movements of the diaphragm during the respiratory movements of the thorax. During inspiration the diaphragm, which at the completion of expiration is closely apposed to the thoracic wall in its lower portion, becomes separated from the thoracic parietes in its descent. This separation of the two appos-

ing surfaces during inspiration causes a slight undulation or "shadow" to pass down the anterior, lateral, and dorsal surfaces of the thorax from the seventh to the tenth rib. During expiration, as the diaphragm adapts itself again to the chest wall, there is an ascending undulation, which, however, is not as readily perceptible as is the descending shadow which occurs during inspiration.

To elicit Litten's phenomenon the patient should be placed in the dorsal posture on a bed or table with the head slightly elevated and the arms stretched above the head, with the feet or head turned toward the source of light, in such position that the light falls obliquely upon the side under observation. The examiner should stand several feet from the patient with his back to the

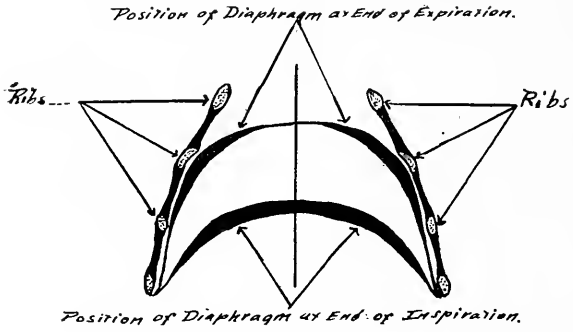


Fig. 31.—Basis of Litten's phenomenon. (From Cabot.)

light. Under these circumstances in a normal chest the shadow will be perceived to descend for the space of two inches or more during inspiration, and can usually be observed to ascend during expiration.

Litten's Shadow is a normal phenomenon. Interference with or abolition of, the shadow points to a pathologic condition in the thorax or abdomen, which inhibits the free movement of the diaphragm. In the thorax it may signify pneumothorax, pneumonia, hypertrophic emphysema, pleurisy with effusion, adhesions between the pleura and the chest wall, intrathoracic neoplasm, or pulmonary tuberculosis. Of intra-abdominal conditions it may be indicative of upward displacement if the diaphragm by an enlarged liver or spleen, ascites, large abdominal tumor, or subphrenic abscess.

Pathologic Respiratory Variations

Rapid Respiration (Polypnea).—Simple increase in the frequency of the respiratory movements of the thorax is observed after exercise and during strong emotion. It is also observed in pulmonary lesions which decrease the air space, as in the consolidations of tuberculosis and pneumonia; in diminution of the intrathoracic capacity by sub-phrenic pressure due to ascites or abdominal tumor, as well as mediastinal tumors compressing the lungs; in inflammations of the peritoneum; in congestion and edema of the lungs; and during the course of diabetes and uremia.

Slow Respiration (Oligopnea).—Slow respiratory movements, the rate falling below fourteen respirations in the minute, are observed during coma and collapse, and in cerebral pressure from tumor, apoplexy, abscess, or meningitis. The respiratory movements are also slowed during the course of infectious diseases associated with mental torpor.

Prolonged Inspiration.—Inspiration is unduly prolonged in cases of tracheal and laryngeal obstruction from any cause. The interspaces over the lower portions of the chest are apt to be retracted during the inspiratory effort.

Prolonged Expiration.—In hypertrophic emphysema and bronchial asthma the expiratory phase of respiration is prolonged, the muscles of respiration being called into play in the effort to expel the air from the lungs, substituting an active for a passive process.

Stertorous Respiration (Snoring Breathing).—This type of respiration is noted in the presence of large adenoid vegetations in the naso-pharynx, chronic tonsillar hypertrophy, quinsy, post-pharyngeal abscess, in paralysis of the palate, and during the coma of apoplexy, uremia, or diabetes.

Stridulous Respiration (Hissing Breathing).—Stridulous respiration is usually a sign of tracheal and laryngeal stenosis by tumors or foreign bodies. It may be produced by pressure upon these structures by enlarged glands, or an excessively hypertrophied heart. It also occurs in spasm and edema of the glottis, which may develop in connection with syphilis or tuberculosis of the larynx, during diphtheria, acute laryngitis, or as a complication of one of the acute infectious diseases. Stridor also occurs during an attack of laryngismus stridulus, and during the paroxysms of pertussis, the stridor in these instances being most pronounced or entirely confined to inspiration.

Cheyne-Stokes Respiration.—Cheyne-Stokes breathing is a type of respiration in which, following a period of apnea, the respirations become progressively deeper until a maximum depth is attained, whereupon they gradually become more shallow and finally terminate in another apneic period. The period of apnea lasts from ten to twelve seconds, and during this time the patient is likely to become unconscious. This type of respiration may persist for a few days or for several months, and is of grave prognostic significance. It is often a sign of impending dissolution. Cheyne-Stokes respiration is particularly associated with cerebro-spinal meningitis, apoplexy, brain tumor, the coma of uremia and diabetes, and with tuberculous meningitis; more rarely with fatty heart and valvular disease of the heart; and very rarely develops during typhoid fever and pneumonia.

Dyspnea.—Dyspnea, difficult or labored breathing, is recognized clinically by the increased frequency of the thoracic excursions and by the participation of the accessory muscles of respiration in these movements. Dyspnea may exist in varying

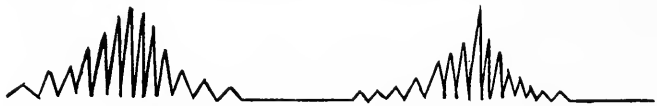


Fig. 32.—Cheyne-Stokes respiration. (From Cabot.)

grades, ranging from a slight increase in the frequency of the respirations to extreme difficulty accompanied by blueness of the lips and finger-tips (cyanosis).

The dyspnea may be limited to inspiration, as when the air passages are obstructed by a foreign body, or subjected to external pressure by a tumor, aneurism, or enlarged gland; or, on the contrary the difficulty may only involve expiration, as is observed in hypertrophic emphysema and asthma. More frequently, however, both phases of the respiratory cycle are involved.

The *causes* of dyspnea are varied. Thus, dyspnea may be an expression of a diminution of the air space in the lungs by the consolidations of pulmonary tuberculosis or pneumonia; or of external compression of the lung by a pleural effusion or intrathoracic neoplasm.

Dyspnea may be caused by circulatory disturbances of the lungs. If a tumor obstructs the free return of blood from the lungs to the right heart, pulmonary congestion or edema occurs, leading to dyspnea. More frequently, however, it is in valvular lesions of

the heart, which permit the blood to accumulate in the pulmonary circulation, that dyspnea referable to circulatory disturbances of the lungs is observed.

Diseases of the blood, associated with a diminution in the amount of hemoglobin or oxygen-carrying power of the blood as in chlororis, pernicious anemia, and the secondary anemias, occasion dyspnea.

Many acute infectious diseases associated with toxemia and fever produce a dyspnea arising from the action of circulating toxins upon the respiratory centers.

Orthopnea is an extreme grade of dyspnea in which the patient is able to breathe only in the erect or sitting posture. It is observed in asthmatic attacks and in regurgitant heart lesions.

ABNORMALITIES OF THORACIC EXPANSION

Abnormalities in the degree of expansion of the thorax may be bi-lateral, or uni-lateral, or circumscribed to certain areas of the surface of the thorax.

Bi-lateral Changes

Increased General Expansion of both sides of the thorax is observed following active physical exertion and during emotional states, without possessing pathologic significance. It is also observed in hypertrophic emphysema, during asthmatic attacks, and hysteria, when it has diagnostic significance.

Decreased General Expansion of both sides of the thorax occurs as a result of general muscular weakness, atrophic emphysema, bi-lateral pleurisy with effusion, paralysis of the respiratory muscles, obstruction of the upper respiratory tract, or from the pain of pleurodynia or intercostal neuralgia.

Uni-lateral Changes

Increased Expansion of One Side of the thorax occurs when one lung must compensate for a crippling of the opposite lung. Thus, in cirrhosis of the lung the opposite lung expands vicariously as a result of compensatory ephysema causing an increase in the expansion of the sound side of the thorax.

Diminution in the Expansion of One Side of the Thorax or uni-lateral diminution in the expansion of the chest, results from diminution of air space in the lung of that side, which may

signify the consolidation of tuberculosis or pneumonia, or the pressure of a pleurisy with effusion, or a crippling of the lung by pleural adhesions.

Local Changes

Local Increase in Expansion of the thorax is usually observed in the supra-clavicular regions in cases of hypertrophic emphysema, affecting chiefly the apices of the lungs.

Local Decrease in Expansion of the thorax often occurs in the supra-clavicular and infra-clavicular regions owing to apical tuberculosis. Local decrease is noted in other regions of the thorax when pleural adhesions bind the lung to the chest wall, the result of local pleurisies.

Wavy Expansion of the chest is sometimes encountered in connection with lobar pneumonia, when different areas of the chest appear to expand in an irregular and uneven manner, one portion filling or expanding before other portions.

Localized Areas of Pulsation.—Local areas of pulsation upon the thoracic surface possess various significance, depending upon the locality in which they are found. Thus, a pulsation near the base of the heart usually points to aneurism of the aortic arch. A pulsation arising on the left side of the thorax between the second and sixth ribs is frequently a sign of pulsating pleurisy. A pulsation over the lower left lung posteriorly, below the left scapula, may signify a pulmonary cavity containing fluid, to which the impact of the heart is transmitted during systole. A circumscribed pulsation in the lower portions of the chest anteriorly is often due to empyema necessitatis, in which event the chest wall is apt to be edematous or to present a localized area of discoloration.

CHAPTER III

PALPATION

Object and Technic.—Palpation is employed in physical diagnosis to confirm the findings of inspection as to the shape and size of the thorax, the respiratory movements and degree of expansion of the chest; and to detect slight deficiencies of expansion which are so slight as to escape detection during inspection. Palpation of the thorax also reveals the presence of certain vibrations (fremitus) arising within the chest. It also reveals the degree of resistance of lesions within the thorax, as also tenderness upon pressure, fluctuation, and local pulsations.

As commonly employed, palpation consists in applying the palms of the hands to the surface of the thorax for the purpose of appreciating and analyzing tactile impressions conveyed to the palpating hand. In examination of the anterior, posterior, and lateral regions of the thorax the entire palm is placed flat upon the thoracic wall, while in palpating the supra- and infra-clavicular regions, the finger tips are often employed alone. Certain clinicians prefer to employ the ulnar border of the hand in preference to the palm in palpation; but it seems unlikely that the tactile sensations appreciated by this portion of the hand could be as acute as those which can be detected by the more sensitive palms. During palpation of the thorax the clothing should be removed to the waist so that no fabric may intervene between the palms of the examiner and the thorax of the subject. As in all physical examinations the patient should assume an easy and natural position.

In testing the expansion of the apices of the lungs the examiner should stand behind the patient and with the index and middle fingers in the supra- and infra-clavicular spaces respectively gauge the degree of expansion of the apex during full inspiration. To detect deficient expansion of the lateral regions of the chest the examiner should stand facing the patient and apply the two hands to the lower portions of the thorax. To test the antero-posterior expansion he should stand beside the patient,

and place one hand on the sternum and the other between the scapulae and note the degree of expansion during quiet and full inspiration.

Fig. 33.—Palpation of anterior thoracic surface.

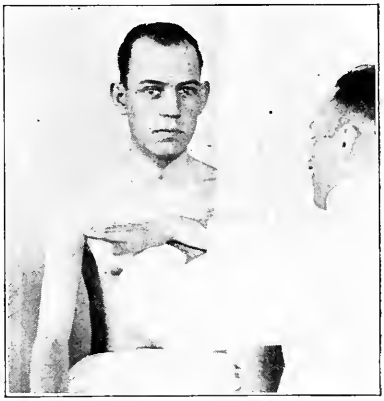


Fig. 34.—Ulnar palpation of thorax.

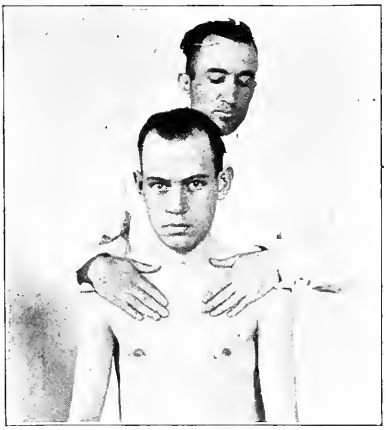


Fig. 35.—Palpation of upper anterior thorax.

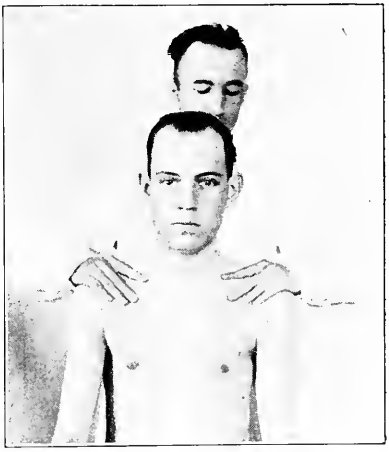


Fig. 36.—Palpation of pulmonary apices.

Palpation, to be serviceable, must be systematic, the entire surface of the thorax being palpated and corresponding regions upon the two sides compared. Hasty, purposeless palpation leads to erroneous conclusions.

Fig. 37.—Illustrating the method of detecting lagging at the apices. (From Pottenger.)

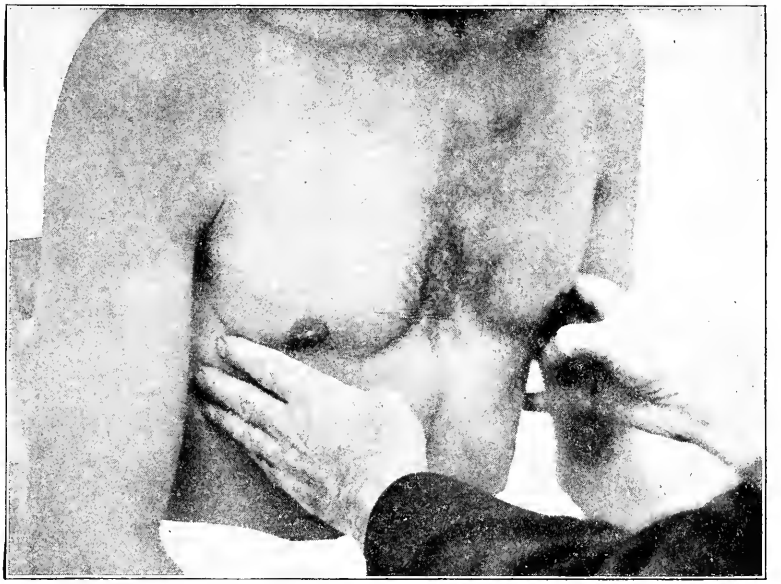


Fig. 38.—Illustrating the method of detecting lagging at the base. The hands should be laid gently on the chest so as not to interfere with the normal respiratory movements. Often a pressure no greater than one or two pounds will completely check the movements of the ribs. (From Pottenger.)

THORACIC VIBRATIONS

Vocal Fremitus.—Vocal fremitus is a palpable vibration which is conveyed to the palpating hand when applied to the surface of the thorax while the patient speaks. The vibrations originate in the vocal cords and are transmitted downward by the air columns of the trachea and bronchi and thence by the pulmonary parenchyma and chest wall to the hand of the examiner. Vocal fremitus is a normal physiologic phenomenon.

The intensity of the vibrations varies with the quality of the subject's voice, being more intense in men with coarse deep

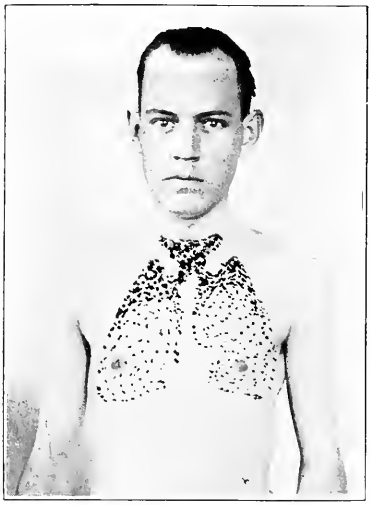


Fig. 39.—Normal variation of vocal fremitus.

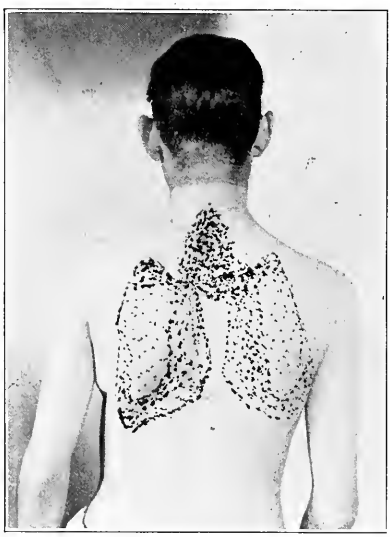


Fig. 40.—Normal variation of vocal fremitus.

voices than in women and children, whose voices are finer and less coarse. Similarly, the vibrations are more intense in thin chested individuals than they are in a subject with a thick chest with well developed musculature.

There are normal regional variations in the intensity with which the vocal fremitus is transmitted to the palpating hand. Thus, the fremitus is most intense immediately over the trachea. It is more intense over the infra-clavicular and mammary regions than it is over the lower regions of the thorax, becoming progressively more feeble as the palpating hand progresses downward. Its intensity is impaired over the scapulae posteriorly, and over the sternum anteriorly, as well as over the mammary

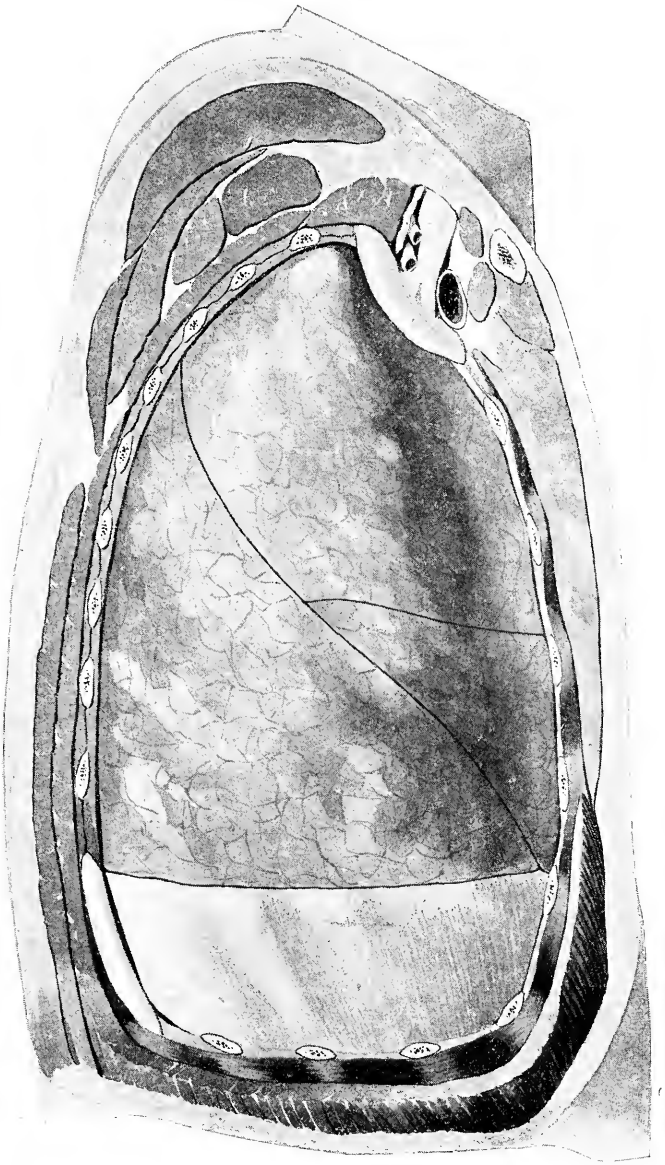


Fig. 41*A*.—Sagittal section through the body showing the thickness of the soft structures covering the apex from which, may be inferred the importance of the increased tone (spasm) or degeneration upon the findings on palpation, percussion, and auscultation. Anterior view. (Pottenger, after Corning.)

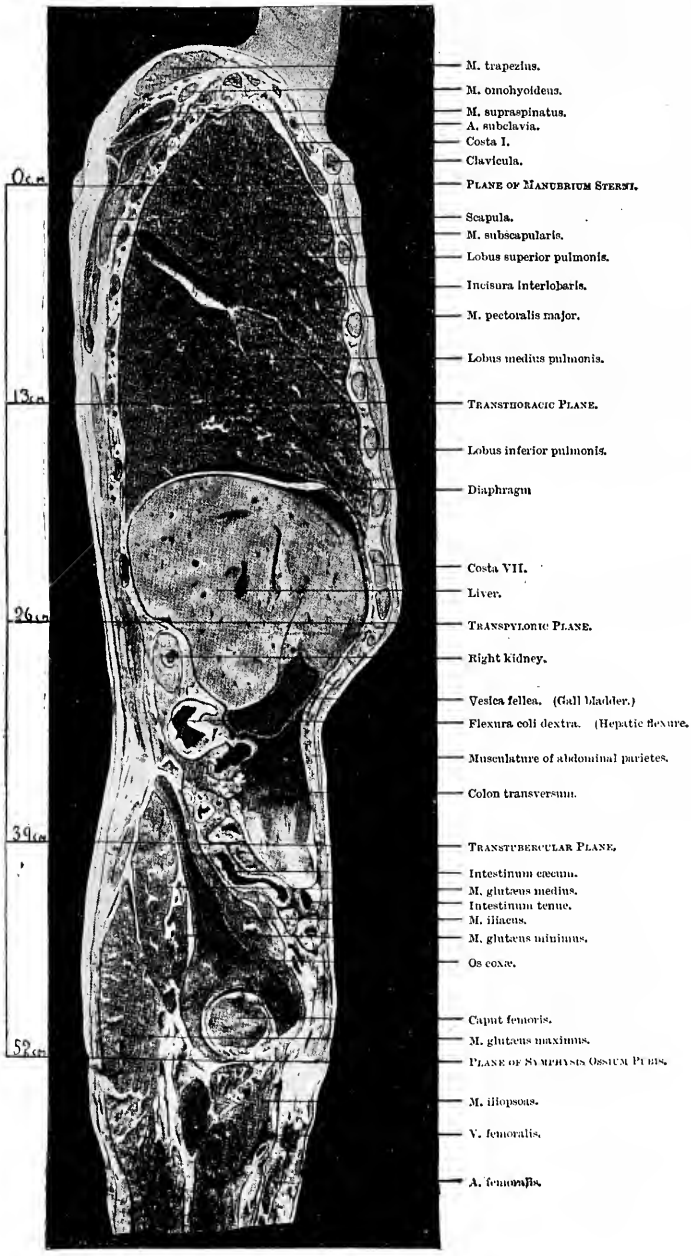


Fig. 41*B*.—Section through body 6 cm. to the right of the median plane, view from the right. Showing the importance of the soft tissues as influencing physical examination of different areas of the chest. (Pottenger, after Berry.)

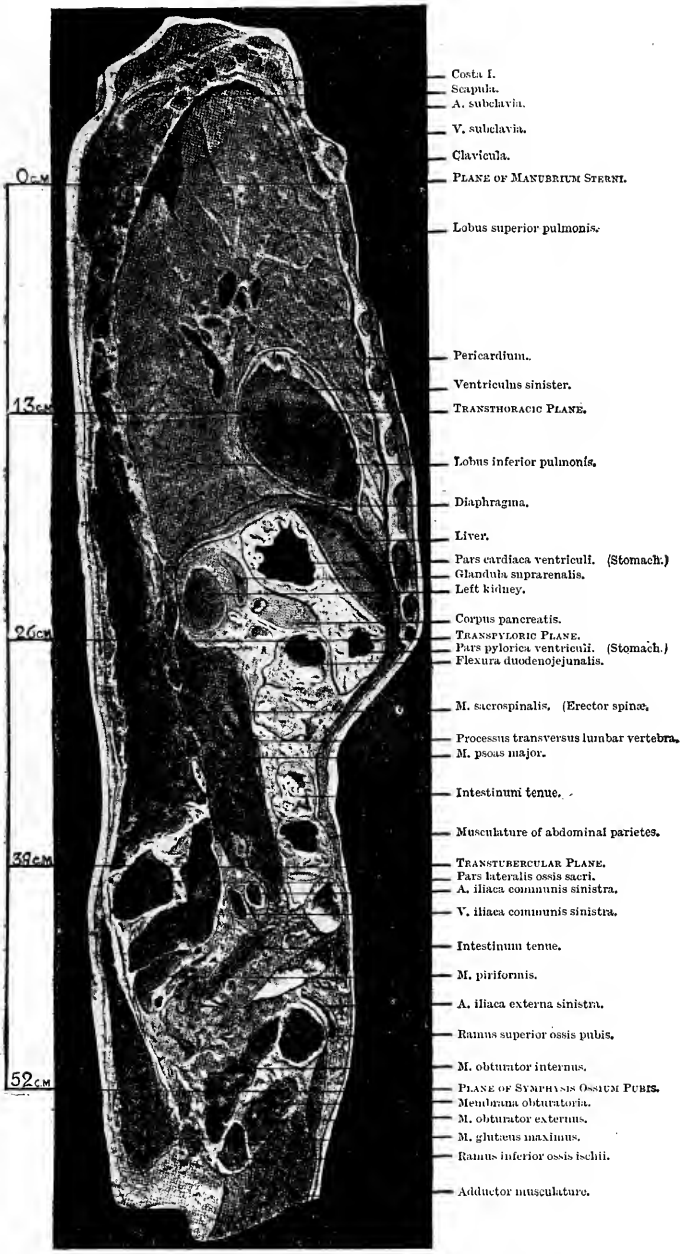


Fig. 41C.—Section through body 6 cm. to the left of the median plane viewed from the right. Showing the importance of the soft tissues as influencing physical examination of different areas of the chest. (Pottenger, after Berry.)

gland in the female subject. Moreover, vocal fremitus is more intense over the right side of the thorax than upon the left side, because the right bronchus is larger than the left, occupies a position slightly nearer to the anterior chest wall than does the left, and the eparterial bronchus, which is given off to the upper lobe of the right lung, leaves the trachea at a point nearer the larynx.

In eliciting vocal fremitus the examiner places the palms of the hands flat upon the thorax while the patient is directed to count "One, two, three," or to repeat the words "ninety-nine," the examiner meanwhile noting the intensity with which the vibrations are transmitted to the palpating hand.

Pathologic Variations.—Certain pathologic variations in the intensity of vocal fremitus are encountered in the investigation of morbid conditions arising within the respiratory organs. Thus, its intensity may be increased, decreased, or the fremitus may be absent, as a result of different diseases of the lung, pleura, or air passages.

Increased Vocal Fremitus.—The intensity of the voice vibrations is increased or exaggerated in those conditions in which the lung is rendered more solid and in which the air content of the tissue is reduced. That is, in all cases in which the air content is diminished by consolidation or pulmonary compression. This is because the vibrations are more readily transmitted by a continuous solid medium than by the normal pulmonary tissue in which the solid structure is interrupted by air-containing spaces. Hence, vocal fremitus is found exaggerated in the presence of the consolidations of pneumonia and tuberculosis and in the presence of a tumor of the lung or pleura. It is also increased over tuberculous and bronchiectatic cavities which have an open communication with the bronchial system. These cavities act as resonating chambers for the vocal vibrations.

Diminished Vocal Fremitus.—When the lung is separated from contact with the chest wall by a pleural effusion or a collection of gas or air in the pleural cavity as occurs in pneumothorax, the vocal fremitus is diminished. Its intensity is also diminished in hypertrophic and compensatory emphysema, owing to the rarefaction of the pulmonary tissues by the increased air content.

Absence of Vocal Fremitus.—Vocal fremitus is absent over a pulmonary cavity containing fluid. It is also absent over extensive pleural effusions, and over areas of the lung in which the

bronchus to the part has become obstructed from any cause. Excessive pleural thickening will abolish vocal fremitus.

Rhoncal Fremitus.—The term rhoncal fremitus is applied to the vibrations which are produced by the passage of air through mucus, pus, or blood in the bronchial tubes, the vibrations thus produced becoming appreciable to the palpating hand. Such a fremitus or vibration is encountered during the course of acute bronchitis, asthmatic attacks, in pulmonary tuberculosis, and in all catarrhal inflammations of the bronchial tubes.

These vibrations are occasionally referred to as *rhonchi* and are classified as large and small, as they arise in the larger or the smaller bronchial tubes respectively. Rhonchal fremitus is best demonstrated in children, whose chest walls are thin and resilient.

Friction Fremitus.—When the pleura becomes inflamed there is a palpable vibration caused by the rubbing together of the roughened surfaces of this membrane. It is demonstrable during inspiration, being usually most intense in the lower axillary region. It is a significant and pathognomonic sign of fibrinous and sero-fibrinous pleurisy. In the latter disease the friction fremitus is present prior to the development of the effusion; it disappears during the effusion; and it recurs later with the absorption of the fluid.

Tussile Fremitus.—Tussile or Tussive Fremitus is a palpable vibration produced upon coughing. It does not as a rule possess great diagnostic significance and is not frequently elicited during routine examinations; but it is of service in dealing with patients the subjects of aphonia from any cause, when it is not possible to elicit vocal fremitus.

Succussion Fremitus.—Succussion fremitus is a palpable vibration produced when a patient whose pleural cavity contains air and fluid is suddenly jarred or shaken. The impact of the fluid will be felt against the palpating hand under these circumstances. Large pulmonary cavities will give the vibration if they contain air and fluid. However, the sign is more frequently elicited in hydro- and pyo-pneumothorax, and rarely with pleurisy with effusion.

Crepitus.—In cases of surgical emphysema, when the subcutaneous tissues of the thorax contain beads of air, a fine crepitation or crepitus is often demonstrable upon palpating over the region with the finger-tips.

Tenderness and Pain on Pressure.—In disease of the pleura or

disease of the lung complicated with pleurisy, palpation often yields tenderness. The pain under these circumstances is elicited and is also defined by palpation of the intercostal spaces with the finger-tips. Pain elicited in this manner is suggestive of fibrinous pleurisy. Similarly, pain is elicited in intercostal neuralgia, pleurodynia, and in fracture or caries of a rib.

Local Pulsations.—The pulsation of a Pulsating Empyema or of aortic aneurism, or of a pulmonary cavity containing fluid and situated adjacent to the heart, the impact of the heart being transmitted to the fluid of the cavity, may be demonstrated by palpation.

Increased Resistance.—Upon palpating the intercostal spaces over a pleural effusion, over the consolidation of pneumonia or tuberculosis, and in cases of excessive thickening of the pleura, a sense of increased resistance is appreciated by the palpating fingers.

Fluctuation.—Fluctuation can only occasionally be elicited in affections of the thoracic viscera. However, in the case of empyema necessitatis about to rupture, pitting upon pressure or actual fluctuation can occasionally be demonstrated. It is elicited by placing the palpating hand over the suspected area and forcibly striking the opposite side of the thorax with the free hand.

CHAPTER IV

PERCUSSION

Objects and Technic.—Percussion is employed in the study of disease of the respiratory organs for the purpose of eliciting sounds normal to the pulmonary parenchyma and sounds only arising in diseased states of these organs; to determine the borders of the intra-thoracic organs; and also to note the degree of resistance offered to the percussion stroke by the tissues under examination.

Percussion, as commonly practiced, consists in striking the surface of the area under examination with a view primarily to eliciting sound, and secondarily for the determination of the degree of resistance offered to the percussion blow. During this maneuver the percussion blow may be directed with the finger or with a special percussion hammer, and either directly upon the part under examination, or upon an intervening medium, usually a finger of the opposite hand of the examiner, or in other instances plates of metal, ivory, or glass. The instrument with which the blow is struck, finger or hammer, is termed the *plexor;* the intervening medium, finger or plate, is termed the *pleximeter.* When an intervening medium or pleximeter is employed, the percussion is termed *mediate percussion;* whereas when no such intervening instrument is present, but the blow is directed directly upon the part which is under examination, the percussion is termed *immediate percussion.*

Immediate Percussion.—In this form of percussion the pleximeter is dispensed with, the blow being struck directly upon the part under examination. Immediate percussion is little employed except in tapping the clavicles to determine the presence of a possible consolidation of the apices of the lungs; or in slapping the two sides of the thorax alternately with the palm of the hand to determine the presence of dullness over a relatively large area, usually at the bases posteriorly.

Mediate Percussion.—In practicing mediate percussion, the left hand of the examiner is placed palm down upon the area under examination, the percussion blow being directed upon the

base of the nail or the second phalanx of the middle finger of this hand. To obtain the best results it is essential that the plexímeter finger be applied firmly and evenly upon the part; that the percussion blow be delivered quickly and in a vertical direction; that the plexor finger be raised at once and not per-

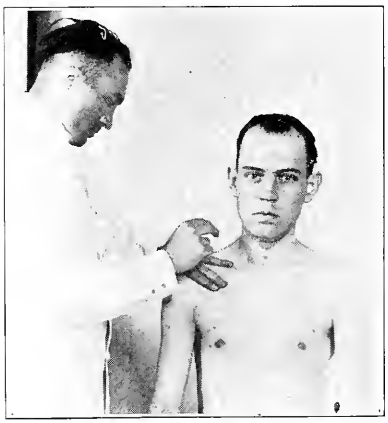


Fig. 42.—Percussion of pulmonary apices.

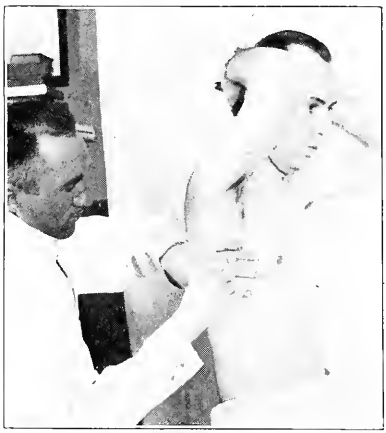


Fig. 43.—Percussion of lateral thoracic region.

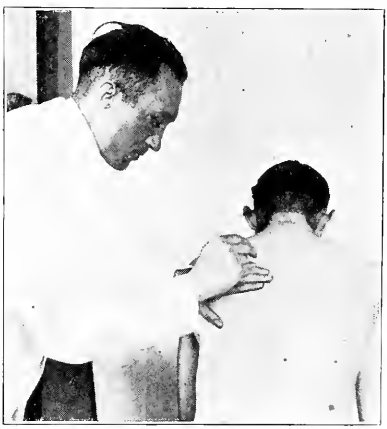


Fig. 44.—Percussion of posterior thorax.

mitted to remain in contact with the plexímeter; that the percussion blow be delivered entirely by a wrist movement, the forearm not participating; and that the blows be delivered with uniform intensity. In practicing percussion only a few strokes should be used in a given region. As much information can be

obtained by four or five strokes properly directed as by a greater number, which tend to deaden the nicety of the auditory appreciation of the sounds elicited. In comparing the sound elicited upon the two sides of the chest exactly corresponding points should be selected on either side, and the percussion strokes should be directed with the same force upon each side.

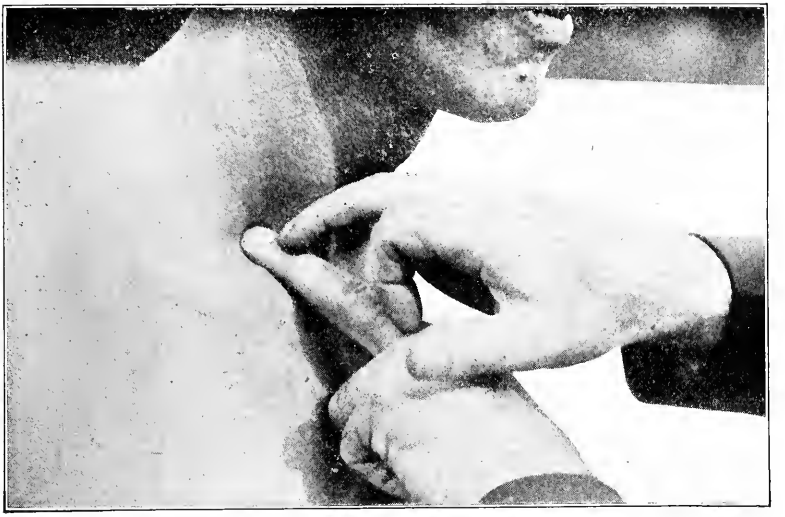


Fig. 45*A*.

Fig. 45*B*.

Fig. 45.—Illustrating a common error in percussing the apices. *A*. Proper position, showing percussion of the apices while the patient's head is erect and tension removed from the sterno-cleido-mastoideus and other neck muscles. *B*. Wrong position, percussing same when the head is turned and bent over toward the opposite side, thus putting the sterno-cleido-mastoideus and other muscles on tension, thereby raising the pitch of the percussion note and increasing the resistance to the percussion finger. (From Pottenger.)

Force of the Percussion Stroke.—The force to be employed in percussion depends upon the thickness of the chest wall and upon the object of the examination. In thin chested individuals and in children little force is required to elicit a clear sound. In stout persons, on the contrary, with thick thoracic walls, a greater degree of force is required for the purpose. The degree of force employed also varies with the location of the lesion which it is desired to study. In outlining lesions near the surface of the chest *superficial percussion* is employed, the stroke being light. In outlining deep lesions *deep percussion,* with heavy strokes must be employed.

Palpatory Percussion.—This mode of percussion is practiced by striking the plexímeter with a pushing movement rather than

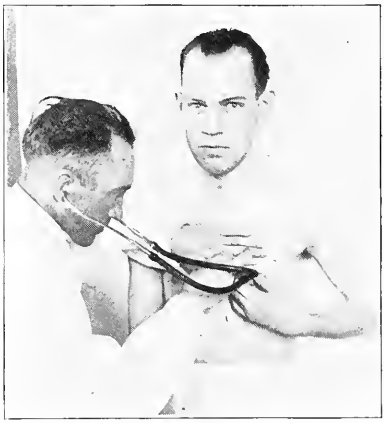


Fig. 46.—Auscultatory percussion.

with a sharp stroke, as in ordinary mediate percussion, and allowing the plexor to remain for an instant upon the plexímeter.

The chief object of this procedure is to determine the degree of resistance afforded by the underlying structures rather than the production of sound.

Auscultatory Percussion.—This mode of percussion is employed for the purpose of marking out the borders of organs, tumors, or collections of fluid. In practicing auscultatory percussion the chest-piece of the stethoscope is placed over the organ or structure to be outlined and retained in position by the patient or an assistant while the examiner, after first percussing near the bell of the instrument and fixing in his mind the quality of the note elicited, then percusses toward the instrument from several di-

rections upon the surface of the chest in the vicinity. The points at which the percussion note is observed to change are marked on the skin with a dermographic pencil; and, when connected by a line drawn through each of them, will represent the limits of the organ or structure under examination. As will be noted later, in percussing toward solid structures within the chest which are overlaid by lung tissue, the quality of the note is observed to undergo two changes.

Attributes of the Percussion Sound.—Upon percussion of the surface of the chest sound is elicited, which possesses certain attributes or properties; namely, *quality, pitch, intensity* or *volume,* and *duration.*

Quality.—Quality is the property or attribute by which a given sound is distinguished from a sound of different origin. It is by the quality of the sounds that the sound produced upon striking a piece of iron is distinguished from that produced upon striking a piece of wood. It is their quality which gives to the various sounds elicited upon percussion their individuality and their diagnostic significance.

Pitch.—The pitch of a sound is determined by the rapidity of the vibrations which produce the sound. When the surface of the thorax overlying the lungs is struck the air content of the pulmonary alveoli is thrown into vibrations. Pitch may be high or low, depending upon the rapidity of these vibrations, rapid vibrations producing a sound of high pitch, while slow vibrations produce one of low pitch. The rate of the vibrations is in turn influenced by the size of the cavity containing the air and by the force of the percussion blow.

Intensity.—Intensity, or volume, has reference to the loudness of the sound, this in turn depending upon the amplitude of the vibrations produced, the force of the percussion stroke, the thickness of the chest wall, and the amount of air in the area under examination. Thus, with heavy percussion over an area of lung containing much air and with a thin chest wall, an intense sound is produced; while a similar stroke over a region containing little air, overlaid by thick chest walls, would produce a sound much less intense.

Duration.—The duration of the percussion sound or the length of the sound possesses less diagnostic significance than do the other attributes. In general it may be stated that the clearer the note and the higher the pitch, the shorter the duration; the duller the note and the lower the pitch, the longer the duration.

Sense of Resistance.—Aside from eliciting sound percussion is employed to note the degree of resistance as appreciated by the plexímeter. In many instances the degree of resistance encountered affords as valuable information as does the sound elicited; and in instances where for any reason the sounds produced are not typical, it may be the sole guide of the examiner. An increased resistance to the percussion blow as appreciated by the plexímeter indicates a decrease in the air content of the region under examination and increase in solid structure or the presence of fluid. Thus, a high pitched note with well marked sense of resistance indicates that the air content is small, while the proportion of the solid material is excessive.

THE NORMAL PERCUSSION SOUNDS

The sound elicited by percussion of the normal chest is termed *resonance,* or *normal vesicular resonance.* The quality of this sound is distinctive and is only afforded by percussion of the normal pulmonary tissue containing its normal quota of air separated by the inter-alveolar septa.

Resonance in its greatest purity is obtained by percussion in the infra-clavicular and axillary regions, and at the bases posteriorly, below the angle of the scapula. In the supra-clavicular regions, upon percussing inward, the normal vesicular resonance of the pulmonary apices becomes mingled with the tympany of the adjacent trachea. In the mammary regions the mammary gland impairs the purity of the resonance, and upon percussion toward the sternum the *osteal resonance* of this bone blends with the normal vesicular resonance afforded by the pulmonary tissues. Also in the right mammary region in its lower portion the dullness of the liver blends with the normal vesicular resonance of the right lung; while on the left side the dullness of the heart blends with the resonance of the left lung. In the right axillary region, in the lower portion, the dullness of the liver impairs the resonance of the lung; while in the lower portion of the left axillary region the resonance of the left lung is impaired by both the tympany of the stomach and the dullness of the spleen. In the supra-scapular and scapular regions the resonance is markedly decreased by the intervention of the bony scapula overlaid by its muscles, while in the inter-scapular region and infra-scapular regions the resonance does not equal that of the anterior regions of the chest.

The Normal Limits of Pulmonary Resonance.—The normal limits or boundaries of pulmonary resonance correspond practically with the borders of the lungs anatomically, extending one to one and a half inches above the clavicle; as low as the sixth rib in the mid-clavicular line; the eighth rib in the mid-axillary line; and the tenth rib in the scapular line.

Variations in the Limits of Pulmonary Resonance.—In broncho-pulmonary disease any lesion which increases the extent of the lung in any direction will cause a corresponding increase in the limits or boundaries of pulmonary resonance; and any lesion which decreases the extent of the lung in any direction produces a corresponding limitation of the normal resonance to the extent of the lesion. It follows that in disease of the lung or pleura we may find the resonance generally increased or decreased in all directions, increased or decreased in certain directions; or in only one area of the lung.

A General Increase of normal resonance in all directions, all of the borders of the lungs extending further than they normally should, is noted in subjects of hypertrophic emphysema, during asthmatic attacks, in sufferers with fibrinous bronchitis, and in dyspneic patients with uncompensated cardiac lesions.

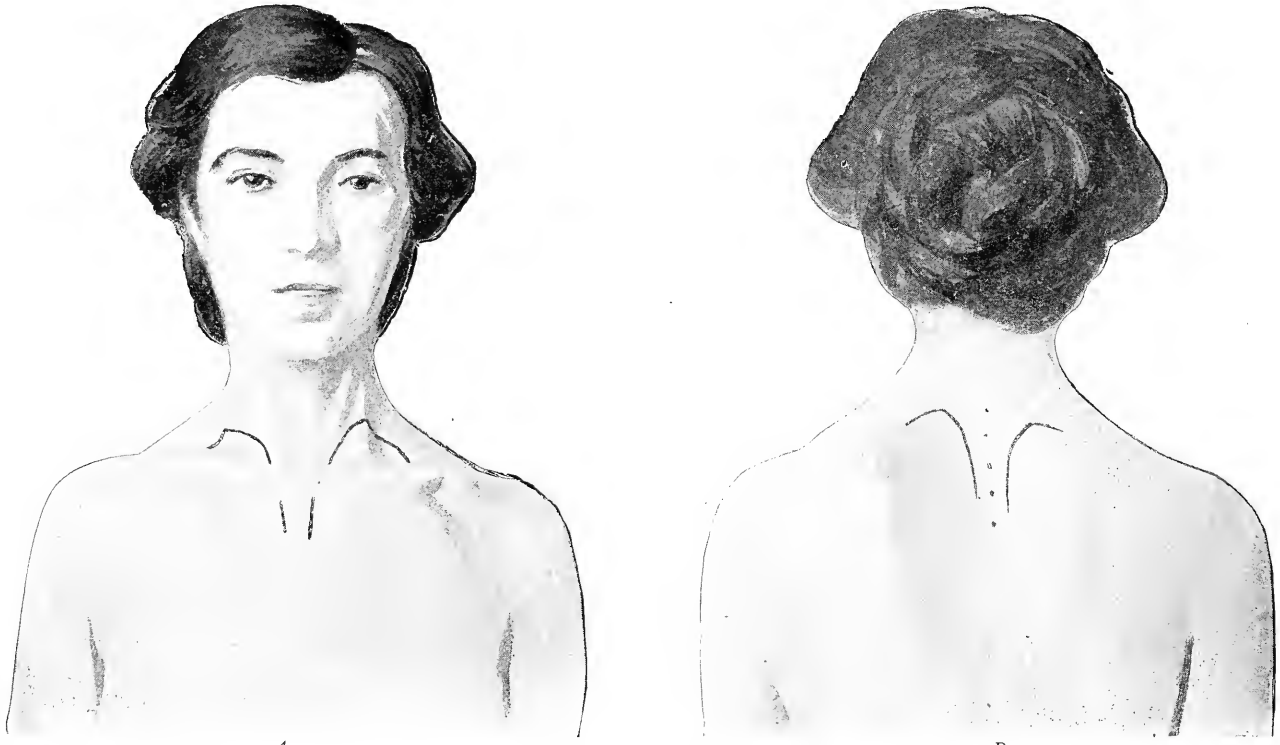
A General Decrease of pulmonary resonance, in which the borders of the lungs are generally retracted in all areas, is noted in atrophic emphysema, in which the lungs are greatly and symmetrically shrunken and atrophied.

Extension of Resonance at the Apices is practically only encountered in hypertrophic emphysema chiefly affecting the upper portions of the lungs and during asthmatic attacks.

Decreased Resonance at the Apices indicates apical tuberculosis, apical pneumonia, chronic adhesive pleurisy, or pulmonary collapse from bronchial obstruction. In apical tuberculosis the resonance is apt to be decreased at both apices; whereas in apical pneumonia or chronic adhesive pleurisy the retraction usually involves a single apex.

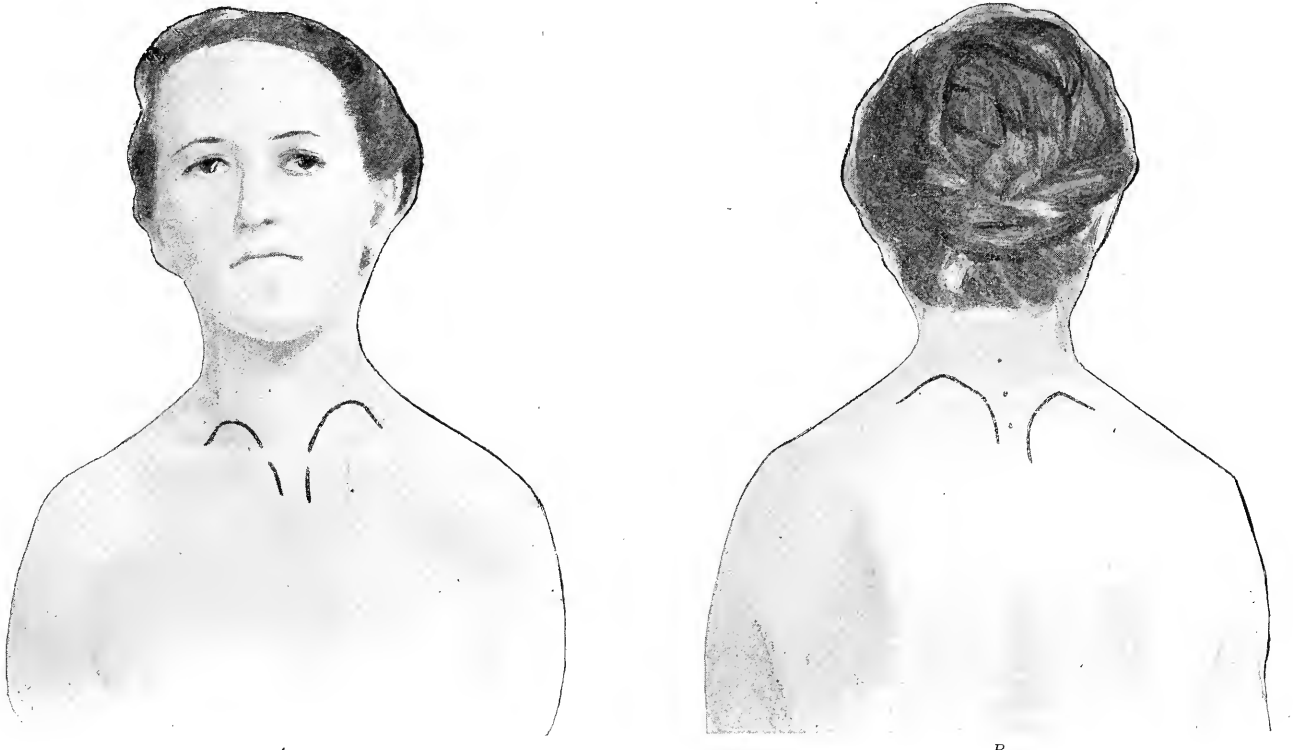
Extended Resonance of the Anterior Borders of the lungs, so that they overlap and obscure the normal limits of the heart, is indicative of hypertrophic emphysema or bronchial asthma.

Decreased Resonance of the Anterior Borders of one or both lungs is indicative of fibroid retraction of a lung from chronic interstitial pneumonia or fibroid phthisis, or displacement of the anterior border by pleural or pericardial effusion. In the case of fibroid retraction of the lung the cardiac impulse is diffuse, oc-



A *B*

Fig. 47.—Showing a patient ill of tuberculosis for seven years, the right upper lobe and left apex being involved. The lines indicate the apical outline. (Pottenger, after Brecke.) One may infer from observing the degeneration of the soft tissues in this patient, without mapping out the apices, that the right lung is the seat of an old chronic tuberculosis, and that if both apices are involved, the right is probably older and is much more extensive than the left.



A *B*

Fig. 48.—Showing patient actively ill of tuberculosis of the right apex for six months, with some involvement in the left. Lines indicate apical outline. (Pottenger, after Brecke.) From the degeneration of the muscles and soft tissues on the right side of this patient, barring occupational influences, one may be sure that the right apex has been involved over a prolonged period of time and that this lesion is chronic.

cupying a wide area in the second, third, and fourth interspaces; whereas in pleural or pericardial effusion the impulse is displaced or invisible.

Increased Resonance of the Lower Borders of the lungs is part and parcel of the general extension of resonance accompanying hypertrophic emphysema or bronchial asthma. It may also occur with fibrinous bronchitis and uncompensated cardiac lesions.

Decreased Resonance of the Lower Borders of one or both lungs points to fibroid retraction of the lung due to chronic interstitial pneumonia or fibroid phthisis; or to elevation of the diaphragm due to paralysis of that muscle or to the sub-phrenic pressure of ascites, abdominal tumor, peritonitis, hepatic enlargement or abscess. In atelectasis the lower border of resonance is elevated.

The Respiratory Excursion of the Lung.—In determining the respiratory excursion of the lung the lower borders of both lungs should be defined by percussion in the mid-clavicular, mid-axillary, and scapular lines, first during quiet respiration and then during forced respiration, the difference being noted. Usually measuring approximately one inch, in certain diseases of the thorax or abdomen it may be diminished markedly.

ABNORMAL PERCUSSION SOUNDS

Impaired Resonance.—The percussion note shows slight impairment of resonance, not amounting to dullness, which is the next gradation, when there is only a moderate increase in the solid over the normal structure of the lung. Impaired resonance is elicited particularly in the early stages of pulmonary tuberculosis at the apices of the lungs.

Impaired resonance is also a sign of moderate pleural thickening and incipient consolidation from any cause. It is the first step toward dullness, but is not so pronounced in its change of quality.

Dullness.—As in impaired resonance, dullness indicates a decrease in the air-content of the part and a corresponding increase in the solid elements of the area under examination. But the note is more materially changed than it is in impaired resonance. A dull note is elicited upon percussion in the presence of the consolidations of pneumonia and tuberculosis, infiltration of the lung with edema and hypostatic congestion, in carcinomatous infiltration, in the presence of considerable pleural thick-

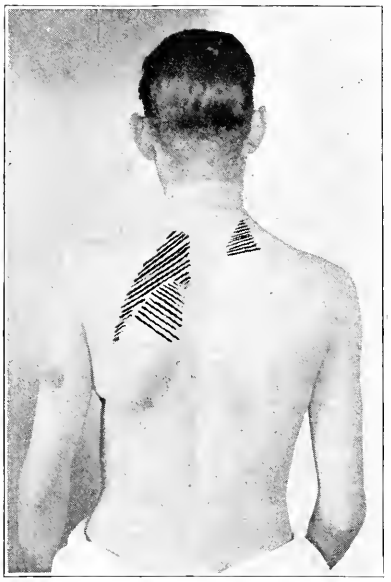


Fig. 49.—Dullness in apical pulmonary tuberculosis.

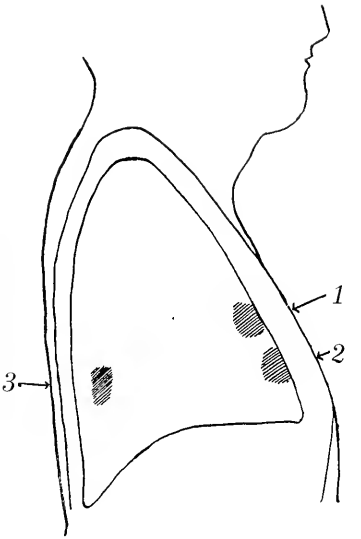


Fig. 50.—Physical causes of change in percussion note. (Redrawn from Le Fevre.) *1-2*, dullness on percussion; *3*, deep dullness masked by intervening lung.

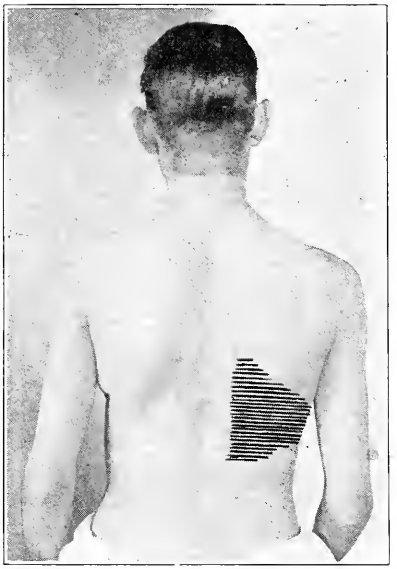


Fig. 51.—Area of dullness in moderate pleural effusion.

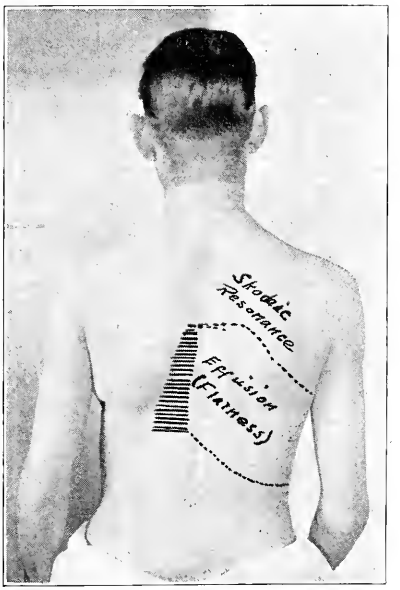


Fig. 52.—Grocco's sign in serofibrinous pleurisy.

ening, in the area of a lung which is compressed by a tumor, in atelectasis, and in pulmonary syphilis.

Dullness localized to a special region is seen in pleurisy with effusion, owing to compression or condensation of the mediastinal structures and their deflection toward the side of the thorax opposite to the effusion. Thus, in pleurisy with effusion there is often a triangular area of para-vertebral dullness opposite the side of the effusion, at about the level of the 12th dorsal spine, which constitutes *Grocco's sign* of this disease.

Flatness.—Flatness, or a percussion note which is entirely de-

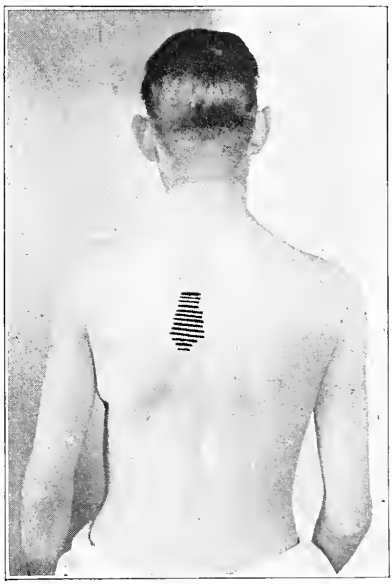


Fig. 53.—Dullness in aortic aneurism.

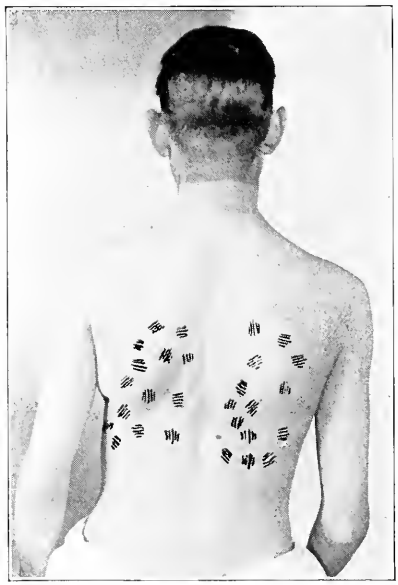


Fig. 54.—Schematic representation of multiple areas of consolidation in bronchopneumonia.

void of resonance, is indicative of the entire absence of air from the area percussed. It is elicited by percussion over a consolidated lobe in fibrinous pneumonia, over a pleural effusion, a tumor of the lung or pleura, a greatly hypertrophied heart, a cirrhosed lung, a hydrothorax, or a pulmonary cavity filled with fluid. A deeply seated consolidation, overlaid by normal lung often fails to give dullness or flatness owing to compensatory emphysema of the intervening lung and requires a deep percussion to elicit dullness.

Hyper-Resonance.—Hyper-resonance, or increased resonance,

is an abnormal clearness of the percussion sound, owing to an increase of the air content in the area percussed. Hyper-resonance may be bi-lateral, uni-lateral, or local. When bi-lateral hyper-resonance is elicited it usually indicates hypertrophic emphysema; when uni-lateral, compensatory emphysema is the usual underlying cause; while localized hyper-resonance may be elicited over a small portion of a lung which is the site of compensatory emphysema to compensate for an adjacent focus of consolidation. Such a localized area of hyper-resonance is often indicative of

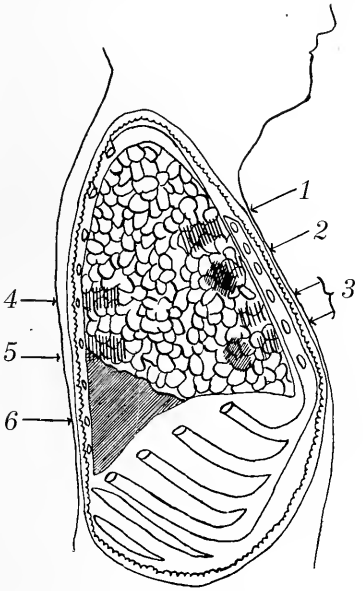


Fig. 55.—Changes in percussion note. (Redrawn from Le Fevre.) *1*, normal vesicular resonance; *2*, impaired resonance; *3*, normal resonance on light percussion, impaired resonance on strong percussion; *4*, normal vesicular resonance; *5*, dullness; *6*, flatness.

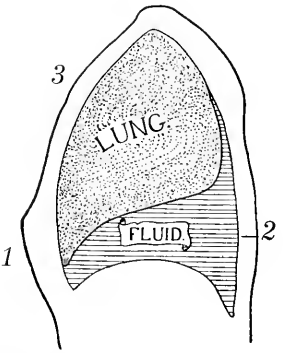


Fig. 56.—Percussion and auscultatory signs in pleurisy with effusion. (From Butler.) *1* and *2*, flatness on percussion, absence of respiratory sounds; *3*, skodaic resonance on percussion and puerile respiration.

a deeply seated patch of consolidation, which requires deep percussion to indicate its presence.

Skodaic Resonance is a variety of hyper-resonance, which is dependent upon relaxation of the pulmonary tissues, which are compressed by a solid tumor, an enlarged heart, or effusion in the pericardium or pleural cavity. Thus, skodaic resonance is elicited by percussion of the chest wall just above the level of a pleural effusion, or over the lung just adjacent to a greatly hy-

pertrophied heart or a large pericardial effusion, or over an area of the lung which is compressed by a neoplasm.

Tympany.—Tympany represents the acme of hyper-resonance, percussion yielding in this instance a tympanitic or drum-like note. Its prototype is found upon percussion over the distended stomach. The significance of tympany is a pulmonary cavity with tense walls, the cavity containing air and possessing a free bronchial communication. A bi-lateral tympanitic note is occasionally demonstrable in the presence of hypertrophic emphysema, in which instance it is to be attributed to the many thin-walled sacs containing an excess of air. A similar note is sometimes elicited in the early stages of lobar-pneumonia, when it is due to temporary relaxation of the pulmonary tension. A tympanitic note is elicited over tuberculous and bronchiectatic cavities with patent bronchial outlet, and over a pneumothorax.

SPECIAL SOUNDS

Amphoric Resonance.—Amphoric resonance is a tympanitic note, with a clanging, echoing quality, which is produced by forcible percussion over a rather large, superficial cavity with tense walls and free bronchial outlet. It is heard typically in large bronchiectatic or tuberculous cavities, and in pneumothorax with a pulmonary fistula. To elicit the sound the percussion

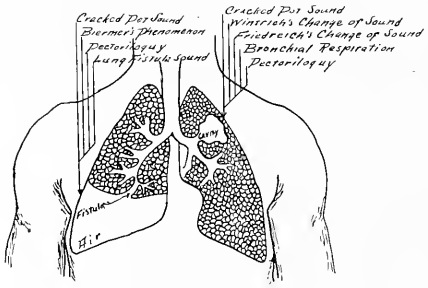


Fig. 57.—Anatomic and pathologic basis of physical signs in percussion and auscultation of the thorax. (Redrawn from Butler.)

stroke should be delivered rather forcibly with the patient's mouth held open.

The Cracked-Pot Sound (Money-Chink Resonance.)—The cracked-pot sound is a percussion note which resembles the sound produced by tapping the side of a cracked metal jar. The sound has also been compared to the muffled chink of coins, hence the name money-chink resonance, which is sometimes ap-

plied to it. The cracked-pot sound may be imitated by striking the clasped palms over the knee. To elicit the sound the percussion stroke should be delivered during expiration, with the patient's mouth open, the examiner's ear meantime being held near the lips of the patient.

The cracked-pot sound indicates a pulmonary cavity with free bronchial communication, or a pneumothorax with a communicating bronchial fistula. The cracked-pot sound may in rare instances be elicited by percussion above a pleural effusion or in pneumonia prior to consolidation. The mechanism of production of the sound seems to be the sudden expulsion of air from a cavity or area of the lung through a rather small opening.

Williams' Tracheal Tone.—This name has been applied to a somewhat tympanitic note elicited upon percussion of an apical

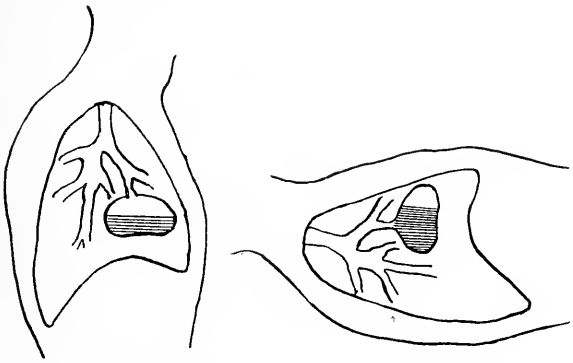


Fig. 58.—Wintrich's interrupted change of sound. (Redrawn from Da Costa.)

consolidation, shrunken pulmonary apex, or pleural effusion, which instead of yielding dullness gives forth tympany conducted from the adjacent trachea. The note is elicited by rather forcible percussion in the supra- and infra-clavicular regions.

Wintrich's Change of Sound.—Upon percussion over a cavity with a free bronchial communication, the note elicited is of lower pitch when the patient's mouth is closed than when the mouth is open. This variation of the pitch of the percussion note under these two circumstances constitutes Wintrich's Change of Sound, a sign which should be sought for in all cases of suspected cavity formation.

Interrupted Wintrich's Change of Sound.—This phenomenon consists in the alternate presence and absence of Wintrich's change of sound upon altering the posture of the patient. It is

indicative of a pulmonary cavity with free bronchial outlet, the cavity containing fluid, which so shifts with the change of the patient's posture as to alternately close and leave unoccluded the bronchial outlet to the cavity. When the patient's posture is such that the bronchial communication is open or is above the level of the fluid, Wintrich's change of sound is demonstrable upon percussion; whereas when with the change of the patient's posture the fluid shifts so as to occlude the bronchial opening, the sound cannot be elicited upon percussion.

Friedreich's Respiratory Change of Sound.—The percussion note elicited over a pulmonary cavity is of higher pitch at the completion of full inspiration than during expiration, owing to the increased tension of the walls of the cavity at this phase of the respiratory cycle, and because of widening of the glottis

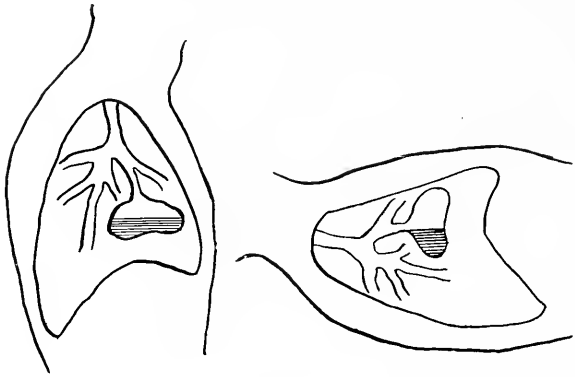


Fig. 59.—Gerhardt's sign. (Redrawn from Da Costa.)

during inspiration. This respiratory alteration in the character of the percussion sound constitutes Friedreich's respiratory change of sound.

Gerhardt's Change of Sound.—When the horizontal and vertical diameters of a pulmonary cavity with free bronchial outlet, the cavity containing fluid, are unequal, there is a change in the pitch of the percussion note elicited over the cavity with change in the posture of the patient. When the greatest diameter of the cavity is horizontal the percussion note is of lower pitch than it is when the greatest diameter is vertical, as a result of the change in the relative positions of the air and fluid in the cavity.

Biermer's Phenomenon.—This is a change of the percussion

note upon change of the patient's posture analogous to the change of pitch in Gerhardt's change of sound. Thus, in cases of hydro-pneumothorax the percussion note is of lower pitch when the patient is in the recumbent posture than it is when the patient assumes the upright position.

Gairdner's Coin Test (Bell Tympany).—In cases of pneumo-

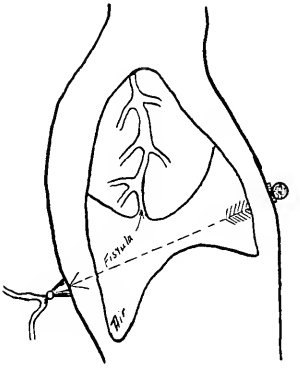


Fig. 60.—Mechanism of Gardner's coin test. (Redrawn from Da Costa.)

thorax, when the stethoscope is applied to the base of the chest posteriorly while an assistant percusses the front of the chest with two coins employed as plexímeter and plexor respectively, an echoing metallic ring is appreciated through the stethoscope, described as resembling the distant ring of a hammer upon an anvil.

CHAPTER V

AUSCULTATION

Object and Technic.—Auscultation is the act of listening to sounds arising within the thorax with the unaided ear, or with a special instrument, the stethoscope. When the unaided ear is employed it is termed *immediate auscultation* in contradistinction to instrumental auscultation, which is termed *mediate auscultation.* For obvious reasons the stethoscope with but few exceptions is preferable to the unaided ear; but the student of physical diagnosis should become proficient in both methods of auscultation.

Auscultation is employed in the study of sounds emanating from the intra-thoracic viscera and in the examination of certain vascular phenomena.

Stethoscopes are termed mon-aural and bin-aural, as they are equipped with one or two ear pieces respectively. The mon-aural stethoscope is little employed at the present time, having given place to the bin-aural instrument. Stethophones have been employed in auscultation, the object of the instruments being to magnify the sounds; but their use is not advisable, as they are poor conductors of high pitched vibrations, and from a clinical standpoint the clearness and distinctness of the sounds is of more importance than is magnification of the intensity of the sounds.

In the selection of a stethoscope care should be exercised to secure an instrument whose chest-piece or bell and tubing are sufficiently heavy to exclude all extraneous sounds, and whose ear-pieces fit snugly in the examiner's ears.

In practicing mediate auscultation the chest-piece of the stethoscope is applied firmly and evenly, but without exerting undue pressure upon the part under examination. The bell of the instrument is retained in position with the forefinger and thumb of the hand of the examiner. No article of wearing apparel should intervene between the bell of the instrument and the surface of the thorax. During the examination of the respiratory organs the examiner should note the character of the sounds produced during quiet, moderately deep, and forced inspiration, being ever on the alert for any deviation from the normal sounds.

The ear should be trained to disregard all extraneous noises, such as the friction produced by the rubbing together of the tubing of the instrument, and crepitations due to contact of the bell of the instrument with a hairy chest wall. This last annoying feature may be eliminated by moistening the bell of the stethoscope

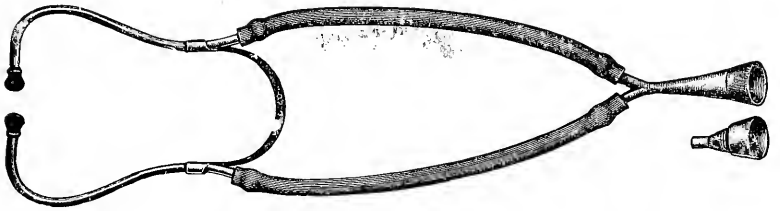


Fig. 61.—Stethoscope.

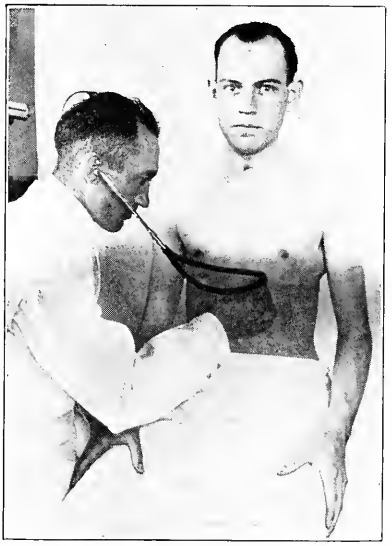


Fig. 62.—Auscultation of thorax.

before applying it to a hairy chest wall. The beginner in auscultation may find it difficult to separate the cardiac and pulmonary sounds; but concentration and practice will enable one to disregard the one while studying the other.

THE RESPIRATORY SOUNDS IN HEALTH

Upon auscultation of the various regions of the normal thorax three types of respiration are noted; namely, bronchial, vesicular, and broncho-vesicular.

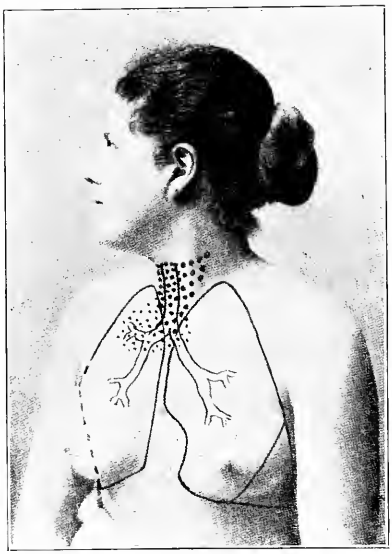


Fig. 63.—Normal areas of bronchial and broncho-vesicular breathing. Anterior view (From Butler.)

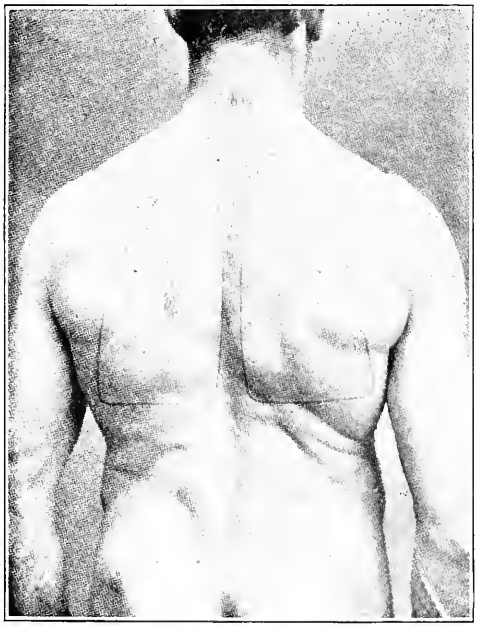


Fig. 64.—Normal areas of bronchial and broncho-vesicular breathing. Posterior view. (From Butler.)

Bronchial Breathing.—Bronchial breathing is audible upon auscultation over the lower portion of the trachea and over the inter-scapular regions between the seventh cervical and the fourth dorsal vertebræ. The sound is loud and blowing or tubular in quality, the two phases of the respiratory cycle being of equal length, though occasionally expiration is slightly prolonged. Inspiration and expiration are separated by a distinct interval; and expiration is of slightly higher pitch than is the inspiratory sound.

Bronchial breath sounds are produced by the vibrations which are set in motion in the inspired air as it passes through the glottis, these vibrations being transmitted downward chiefly by the air column in the trachea and bronchi, and partly by the walls of these passages.

Vesicular Breathing.—Upon auscultation in the infra-clavicular and mammary regions anteriorly, in the axillary regions laterally, and over the bases of the lungs posteriorly, the respiratory sounds have a soft, breezy sound of low pitch, which is termed Vesicular Breathing. The sound possesses or consists of two murmurs, inspiratory and expiratory, which are separated by an almost imperceptible interval, the inspiratory murmur being maintained three times as long as is the expiratory phase. The sound of vesicular breathing has been aptly compared to that produced by the passage of a gentle wind through the leaves of a tree.

Vesicular breathing is audible over portions of the lungs which are comparatively remote from the main bronchial tubes, the blowing sounds of the glottis being modified in their transmission through the lung, and are modified or mingled with the soft movements of the pulmonary tissues. Vesicular breathing is somewhat less intense over the scapulae posteriorly and over the mammæ anteriorly in women. In the right infra-clavicular region the expiratory phase is apt to be somewhat prolonged, owing to the closer proximity of the right bronchus to the anterior chest wall.

Broncho-Vesicular Breathing.—This type of respiratory sound combines the qualities of bronchial and vesicular breathing. It is audible over those portions of the thorax where the larger bronchial tubes are in fairly close proximity to the thoracic wall; but are nevertheless overlaid by air-containing pulmonary tissue. Broncho-vesicular breathing is normally heard over the lower

portion of the manubrium sterni anteriorly, and over the interscapular region posteriorly at the level of the third dorsal vertebra.

THE RESPIRATORY SOUNDS IN DISEASE

Bronchial Breathing.—When bronchial breath sounds are detected in a region of the thorax where they are not normally audible, it usually points to consolidation, the solidification of the lung conducting the murmur from the large bronchi to the surface of the chest. Other factors which produce the same result comprise pulmonary compression and collapse, hemorrhagic infarction, cirrhosis of the lung, enlarged bronchial glands, a tumor overlying a large bronchus, or a pulmonary cavity near the surface of the chest with a free bronchial communication.

Bronchial breathing which is produced by a cavity with a

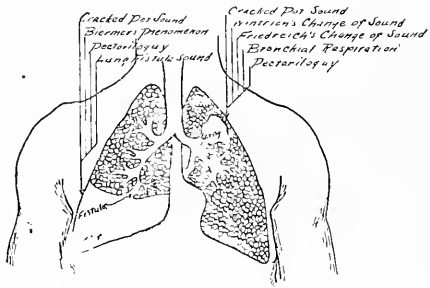


Fig. 65.—Anatomic and pathologic basis of physical signs in percussion and auscultation of the thorax. (Redrawn from Butler.)

patent bronchial communication frequently has a peculiar hollow quality engrafted upon it to which the term *cavernous breathing* is applied. In this type of bronchial respiration expiration is frequently of lower pitch than is inspiration. Simlarly a cavity with patent bronchial communication or a pneumothorax with an open bronchial fistula often gives rise to bronchial breathing of rather musical quality, closely simulating the sound generated by gently blowing across the mouth of an empty bottle, *amphoric breathing.*

Vesicular Breathing.—In disease of the respiratory organs the intensity or rhythm of the normal vesicular murmur may be so altered as to possess diagnostic significance.

Diminution in the intensity or entire abolition of the vesicular murmur may be encountered. Diminution in the intensity of the

murmur is normal in aged subjects and in subjects with very thick chest walls. The murmur is also diminished in painful diseases of the chest wall such as incipient pleurisy and pleurodynia, which cause the patient to inhibit the respiratory excursions of the chest. A similar diminution occurs with moderate pleural thickening, edema of the lung, the early stages of lobar pneumonia, and in presence of a closed pneumothorax.

Abolition of the vesicular murmur is noted over a region of the thorax in which the main bronchus of a lung is occluded, over a large pleural effusion, and over a pulmonary cavity which is filled with fluid.

Increased Intensity of the Vesicular Murmur (Puerile Breathing).—Exaggeration of the vesicular murmur is noted over a lung which is the site of compensatory emphysema due to crippling of the opposite lung, over a circumscribed portion of a lung which is expanding vicariously to compensate for consolidation in an adjacent focus, in catarrhal inflammations of the smaller bronchioles, and during the dyspnea of uncompensated heart disease.

Prolongation of the Expiratory Phase of the vesicular murmur accompanies hypertrophic emphysema and asthma. In these states the alteration in the phases of the sound is noted over both sides of the thorax; and in the case of asthma expiration is apt to be dotted with rales. Uni-lateral prolongation of the expiratory phase at an apex is suggestive of incipient pulmonary tuberculosis, particularly if noted at the left supra-clavicular region.

Cog-wheel Breathing.—In certain diseases of the respiratory organs the respiration, particularly inspiration, occurs in a series of short gasps or jerks, closely simulating the sound emitted by a sobbing child. This jerking or cog-wheel modification of the vesicular murmur is a valuable sign of incipient phthisis. It also occurs occasionally in hysteria, asthma, chorea, local catarrhal conditions of the bronchioles, in the pain of fractured rib, pleurodynia, or pleurisy. Although occasionally encountered in all of these conditions, cog-wheel breathing is a fairly reliable sign of pulmonary tuberculosis.

Broncho-Vesicular Breathing.—When encountered in an area of the chest where it is not normally audible, this type of respiration points to a moderate degree of the same pathologic changes which produce frank bronchial breathing. Thus, it is a sign of partial or incomplete consolidation, as in the early stage of

pneumonia or phthisis, or to a cavity or solid tumor which is overlaid by normal air-containing lung tissue.

VOCAL RESONANCE

Vocal resonance is the transmission of inarticulate sounds to the ear of the examiner during auscultation when the patient is directed to speak. To elicit the phenomenon of vocal resonance the chest-piece of the stethoscope is applied firmly and evenly to the surface of the thorax while the patient is directed to count "One, two, three," or to repeat the words "ninety-nine," with the face turned from the examiner. Under these circumstances the ear of the examiner appreciates certain rumbling, inarticulate sounds, which arise in the larynx and are transmitted downward into the lung and to the surface of the chest by the air columns of the trachea and bronchi and the pulmonary parenchyma. The intensity of the sound emanating from the chest varies in different regions of the thorax, being modified by the same factors which influence the intensity of vocal fremitus.

Variations of Vocal Resonance

Diminution or Absence.—The intensity of vocal resonance is impaired in the presence of hypertrophic emphysema or compensatory emphysema, owing to the rarefaction of the lung incident to these conditions. It is similarly diminished or abolished in the presence of pleural thickening, pleural effusion, and bronchial obstruction. A pulmonary cavity containing fluid abolishes vocal resonance over the area of the cavity.

Increased Vocal Resonance.—Vocal resonance is increased by the same lesions which cause increase of vocal fremitus; namely, consolidations, pulmonary compression, and cavities with free bronchial communications. Different grades of increased vocal resonance are designated by different names.

Bronchophony is a form of increased vocal resonance in which the transmitted voice sound is very audible, sounding as if it were very near the ear. However, the speech is not articulate as it is in the next ascending grade, pectoriloquy. Bronchophony points to consolidation, particularly consolidation overlying or superimposed upon one of the main bronchi.

Pectoriloquy.—Pectoriloquy, the transmission of the articulate voice upon auscultation, is evidence of a very dense consolidation

overlying a principal bronchus, or of a cavity or pneumothorax with free bronchial communication. More rarely pectoriloquy is elicited by auscultation above the level of a pleural effusion. If the voice is very distinct, it is very suggestive of a pulmonary cavity.

Whispering Pectoriloquy.—Whispering pectoriloquy, the transmission of the articulate whisper, represents the highest refinement of vocal resonance, and when elicited is almost conclusive evidence of the presence of a pulmonary cavity with bronchial communication or of an open pneumothorax. Normally the whispered voice is audible as such only over the trachea. In extensive consolidations and conditions of pulmonary compression and collapse the whispered voice is audible but is not articulate. Practically the only condition in which it is articulate is a pulmonary cavity with free bronchial outlet.

Baccelli's Sign.—The whispered voice is transmitted through a serous pleural effusion, but is not transmitted through a purulent effusion. This sign is utilized in differentiating between the two types of pleural effusion. While it is often a valuable means of differentiation, it not infrequently is not demonstrable; since the whispered voice often fails to be transmitted through a serous effusion of large extent.

Modified Vocal Resonance

Aegophony.—In the presence of pleurisy with effusion, upon auscultation immediately above the level of the fluid posteriorly while the patient speaks, a peculiar, quavering, nasal tone is sometimes audible, which has been compared to the plaintive bleat of a goat. This peculiar sound has been designated aegophony. Best detected posteriorly near the angle of the scapula, the phenomenon is sometimes audible over the anterior surface of the chest above the level of a pleural effusion.

Amphoric Vocal Resonance.—In the presence of large pulmonary cavities and pneumothorax the voice sound as appreciated by the usual methods frequently has engrafted upon it an echoing, metallic quality, analogous to the sound of the breath sounds under the same conditions, to which the term amphoric vocal resonance, or amphoric resonance has been applied.

NEW OR ADVENTITIOUS SOUNDS

The adventitious or new sounds which are called into being in diseased states of the respiratory organs comprise *rales,* the *metal*

lic tinkle, or *falling-drop sound,* the *succussion sound* or splashing, the *pleural friction sound,* and the *lung-fistula sound.*

Rales.—Rales are new or adventitious sounds which arise in the bronchi, bronchioles, or the pulmonary alveoli, depending upon interference with the free ingress and egress of air during respiration. The lesion which is responsible for the rales may be a diminution of the lumen of the bronchial tube by compression from without or swelling of the mucous membrane within, or spasm of the bronchial muscles; or it may be due to an obstacle imposed by the presence of mucus, pus, serum, or blood within the bronchial tubes.

Rales are classified as *dry rales* and *moist rales.*

Dry rales, or rhonchi, produced by diminution of the lumen

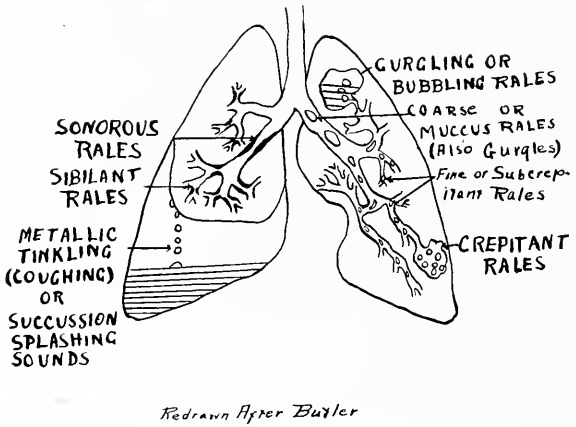


Fig. 66.—Anatomic and pathologic basis of auscultatory findings. (Redrawn from Butler.)

of the bronchial tubes, are divided into *sibilant rales,* when they reside in the smaller bronchial tubes; and *sonorous rales* which develop in the tubes of larger caliber. Sibilant rales are high in pitch and hissing in quality; whereas sonorous rales are of low pitch, and snoring, often musical quality.

Dry rales are heard in the bronchial tubes in the earliest stages of bronchial inflammations prior to the pouring out of secretions into the lumina of the tubes, and in bronchial asthma, owing to constriction of the bronchioles by muscular spasm. The *bruit de drapeau* is a dry rale heard during fibrinous bronchitis, which is caused by the flapping back and forth of a fragment of adherent secretion or mucosa during inspiration and expiration.

Moist Rales, produced by the passage of air through serum,

mucus, pus, or blood in the bronchial tubes, or to separation of the walls of the alveoli which have become adhered by tenaceous secretion, comprise the *crepitant rale,* the *subcrepitant rale,* and the *mucous rale.*

Crepitant Rale.—The crepitant rale is produced by the separation of the walls of the alveoli which have become adhered by tenaceous secretion. Hence it is heard at the completion of full inspiration. The crepitant rale is the finest of all rales and its quality may be simulated by rolling a small lock of hair between the thumb and forefinger held near the ear. Its quality has also been compared to the series of sharp cracklings produced by throwing a pinch of salt upon a hot stove. The crepitant rale is not infrequently confused with a slight pleural friction sound, which it closely resembles. But the crepitant rale is not accompanied by pain, it is more deeply seated, and it is not increased by pressure with the bell of the stethoscope as is the pleural friction sound.

The crepitant rale is heard during the first stage of lobar pneumonia, constituting the *rale indux* of this disease. It is also audible in catarrhal inflammation of the terminal bronchioles and alveoli as occurs in broncho-pneumonia or capillary bronchitis, and in pulmonary edema, hemorrhagic infarction, and partial atelectasis.

Subcrepitant Rale.—The subcrepitant rale is a moist rale, a trifle coarser than the crepitant rale. It is produced by the passage of air through serum, mucus, pus or blood in the terminal bronchioles, causing separation of the bronchiolar walls which have been adhered. It is audible during both inspiration and expiration. The crepitant rale occurs during the late stages of lobar pneumonia in which it constitutes the *rale redux;* during acute bronchitis, hemorrhagic infarction, broncho-pneumonia, and pulmonary edema.

Mucous Rale.—Mucous rales are generated in the larger bronchi and in pulmonary cavities. They are audible during both inspiration and expiration; and are best brought out by coughing or deep inspiration. Mucous rales are produced by the passage of air through a considerable accumulation of fluid.

Gurgling Rales are generated in a cavity containing fluid with a free bronchial outlet which is situated below the level of the fluid in the cavity. Mucous rales are encountered in the course of acute and chronic bronchitis, bronchiectasis, and phthisis.

The Metallic Tinkle (Falling-Drop Sound).—Occasionally during auscultation over a hydro- or pyo-pneumothorax, or a large pulmonary cavity a sound is perceived resembling the sound produced by drops of water falling upon fluid in a container. In the

condition mentioned the sign may often be brought out by shaking the patient, by change of posture, by deep inspiration or by coughing. Two explanations of the method of production of this phenomenon have been advanced; first, that it is to be attributed to the dripping of fluid from the retracted borders of the lung to the surface of an accumulation of fluid in the pleural cavity; second, that it is due to the bursting of bubbles on the surface of fluid in the pleural sac.

The Succussion Sound (Splashing Sound).—A splashing sound which is audible upon shaking the upper portion of the patient's body, is termed the succussion sound, and is a reliable sign of the presence of air and fluid in the pleural cavity. Hence it is

Fig. 67.—Usual site of pleural friction sound.

a sign of hydro-pneumothorax. The sound is not obtainable unless air is present with the fluid; a pleural effusion alone will not afford a succussion sound. Often the sound is audible at some distance from the patient. A succussion sound arising in the pleural cavity must not be confused with splashing sounds frequently arising in the stomach.

The Pleural Friction Sound.—Owing to the small amount of serous fluid which normally moistens the surfaces of the visceral and parietal pleurae, these membranes in health glide noiselessly over each other during the movements of respiration. During inflammation of the membrane, however, and as a result of the excessive extraction of the body fluids which accompanies prolonged diarrhea and profuse hemorrhage, there is produced a

pleural friction sound, which is audible upon auscultation of the surface of the thorax.

The pleural friction sound is usually best detected in the lower axillary region. It is accompanied by localized pain at its site of production, and is localized to this region. It is a superficial sound, audible at the completion of inspiration, more rarely during expiration, disappearing on suspending respiration. It is not influenced by coughing, and is increased in intensity by pressure upon the chest wall. Its quality sometimes resembles the crushing of snow under foot, while at other times it has been compared to the sound of the creaking of new leather. It is often distinguished from the fine crackling of the crepitant rale with difficulty. When marked, the pleural friction sound is attended by friction fremitus.

The Lung-Fistula Sound.—This term has reference to a sound which has been noted in cases of open hydro-pneumothorax, when the bronchial communication opens into the pleural cavity below the level of the fluid. During inspiration a series of bubbling gurgling sounds are generated in the pleural cavity to which the name lung-fistula sound has been given.

CHAPTER VI

THORACOMETRY AND CYRTOMETRY

Thoracometry, or mensuration of the thorax is employed to determine at consecutive examinations variations in the total circumference of the chest; to determine the presence of uni-lateral bulging or retraction of the thorax; and to estimate the total expansion of the chest.

In determining the total expansion of the thorax the difference between the circumference of the chest during complete expiration and during complete inspiration is taken, the difference between the two measurements taken at the nipples indicating the total expansion of the thorax.

In the determination of uni-lateral variations in the size of the two sides of the thorax it is customary to measure from the mid-spinal line to the mid-sternal line upon each side and note any discrepancies in the two measurements. Allowance must be made for the fact that the right half of the thorax is normally slightly larger than is the left half. In making all measurements of the thorax the common tape-measure is the best appliance.

In determining the antero-posterior and transverse diameters of the thorax the caliper is used. In determining the antero-posterior diameter one point of the instrument is placed over the mid-sternal line and the other over the mid-spinal line and the measurement read off on the scale of the instrument. The transverse diameter of the thorax is determined by applying a point of the caliper to each mid-axillary line and reading the diameter on the scale.

Cyrtometry, the determination of the curves of the surface of the thorax, is practiced by applying the cyrtometer accurately to the surface of the thorax. The cyrtometer consists of two pieces of flexible metal connected at one end by a spring. In practicing cyrtometry of the thorax the hinge is placed over the mid-spinal line, and the blades of the instrument are accurately moulded to the surface of the thorax. Upon removal of the instrument a tracing may be made showing the shape of a cross-section of the thorax, and revealing any uni-lateral variations in the two sides of the chest.

CHAPTER VII

ROENTGENOGRAPHY AND FLUOROSCOPY

The roentgen rays, by virtue of their power of penetrating anatomic and pathologic structures in direct proportion to their respective densities, are of very material aid in the diagnosis of many obscure intra-thoracic conditions; and, as they influence the photographic plate in the same manner as does light, permanent records of intra-thoracic conditions may be made. The roentgen ray, however, is only to be considered as an aid to diagnosis, serving often as a means of clearing up an otherwise obscure diagnosis; and its use should never supplant a careful physical examination of the patient. If fluoroscopy or roentgenography is to be used as a routine procedure, it should follow, and not precede, the physical examination of the patient. While many clinicians hold that the roentgen rays reveal tuberculous lesions of the lung before they can be demonstrated by the methods of physical examination at our command, other workers of no less experience hold that a skillful physical examination and a carefully prepared clinical history will bring out evidences of early tuberculous infection before they become demonstrable by the roentgen ray.

The intra-thoracic organs may be examined by the use of the fluoroscope, *fluoroscopy;* by the taking of single photographic plates; *skiagraphy, radiography,* or *roentgenography;* or by the taking of two exposures at different focus, thus affording accurate depth relations, *stereoroentgenography.* Each method of examination has its advantages and its special indications. While fluoroscopy can be utilized in studying the chest from various angles, it does not afford a permanent record of the case as does the roentgenogram. Stereoroentgenograms are of more value than are single plates, as they cause the different structures to stand out in their true relationship (Pottenger).

The Diaphragm (Williams' Sign).—The roentgen rays afford the most valuable means of studying limitations and variations in the movements of the diaphragm. For this purpose, fluoroscopy is superior to other methods of examination. Upon fluoro-

scopic examination, variations in the movements, position, and general outline of this important muscle may be noted. Limitation of the mobility of the diaphragm on one side is often significant of incipient pulmonary tuberculosis (Williams' Sign). However, similar limitation of movement of the muscle may be caused by subphrenic abscess, the traction of pleural adhesions, or by hydatid cyst or abscess of the liver.

While the position of the diaphragm in health is not absolutely constant, the upper border of the muscle on the right side usually corresponds to the upper border of the fifth rib, and on the left side with the upper border of the fourth rib. Fluoroscopic examination may show a depression or undue elevation of the muscle on either side, due to supra- or infra-phrenic pressure or paralysis of the muscle.

The general outline of the diaphragm is altered in diaphragmatic paralysis and in the presence of diaphragmatic hernia. In the case of the latter affection, straining or coughing will increase the herniation of the sac contents.

Pulmonary Tuberculosis.—In tuberculosis of the lungs the roentgenogram shows multiple patches of mottling in the area involved, a decrease in the transparency of the normal pulmonary tissues, not however, as dense or as sharply circumscribed as is the shadow cast in lobar pneumonia. On the contrary, in pulmonary tuberculosis there are multiple areas or impaired transparency, often superimposed, and frequently not exceeding one-fourth inch in diameter. In incipient tuberculosis this mottling is usually confined to the apical or axillary regions, the lower portions of the lungs remaining free at this stage of the morbid process.

Partially healed tuberculous lesions associated with calcareous deposit give a greater diminution of transparency and a greater density, an altogether more clear-cut picture than that afforded by recent or active tuberculous lesions. The peri-bronchial lymph-glands when involved afford shadows which must be differentiated clinically from similar shadows due to enlargements of these glands which are dependent upon the acute infectious diseases or syphilis. Cavities, when empty, are represented by transparent zones, usually surrounded by a darker zone corresponding to adjacent pulmonary consolidation. *Syphilis* of the lung gives a roentgenogram closely simulating that of pulmonary tuberculosis; so much so, indeed, that the course of the disease must be studied in the differentiation. *Abscess* and *gangrene* of the lung yield

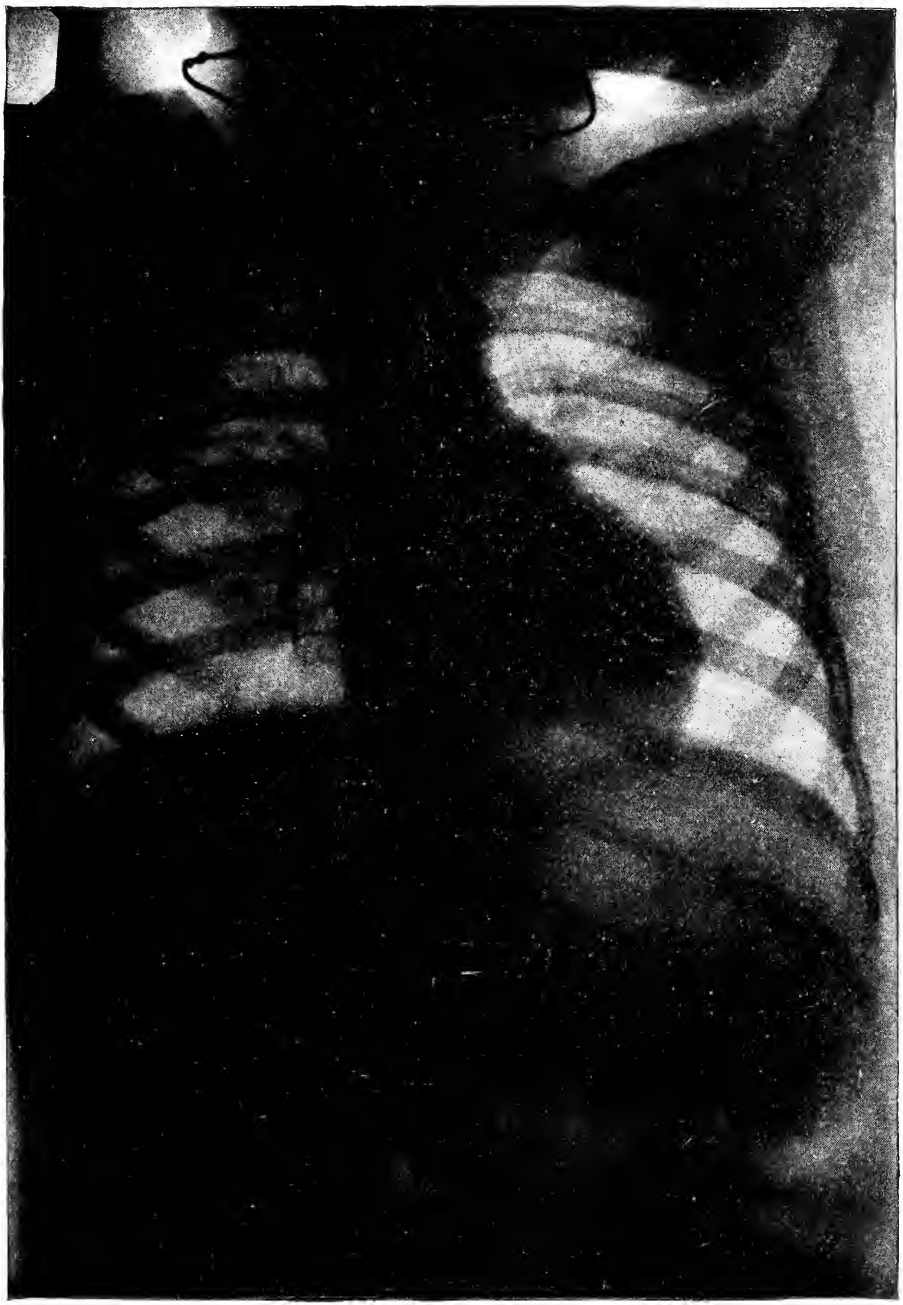


Fig. 68.—Peri-bronchial thickening in a child six and a half years of age. (From Pottenger.)

signs of cavity, the differentiation from tuberculosis resting upon the history and clinical manifestations of the case.

Pneumonia.—In lobar pneumonia, during the early stages with imperfect consolidation there is a diminution of the transparency of the normal pulmonary picture, usually having its inception around the bronchi, more rarely peripherally. When the consolidation is fully developed, a dark shadow with well-defined borders is cast, corresponding to the lobe or lobes involved in the disease. The shadow is occasionally so extremely dense as to obscure the shadows cast by the ribs overlying the area of consolidation.

Broncho-pneumonia yields multiple small shadows, often superimposed, and distributed over both lungs. The picture closely simulates that of miliary tuberculosis of the lungs.

Chronic interstitial pneumonia, or cirrhosis of the lung, gives a fairly characteristic roentgenogram. Shadows corresponding to fibrous bands of induration extend outward in various directions from the root of the lung toward the periphery.

Pulmonary Neoplasms.—A large, single tumor of the lung or pleura is represented roentgenographically by a dense shadow corresponding in extent to the area of distribution of the growth. Small, disseminated, metastatic growths, on the contrary, give a picture which is with difficulty differentiated from well developed tuberculosis of the lungs.

Pleural Thickening.—Thickening of the pleura produces a fairly dense, homogeneous shadow, the density corresponding to the degree of thickening present. A small area of excessively thickened pleura closely simulates a pulmonary neoplasm.

Pleural Effusion.—Sero-fibrinous pleurisy throws a shadow which is homogeneous; the opposite side of the chest should be studied for purposes of comparison. The disease is usually diagnosed by the displacement of the diaphragm downward, and the cardiac displacement toward the opposite side of the chest.

Pneumothorax.—Pneumothorax is distinguished from other intra-thoracic conditions by the uninterrupted transparency over the area involved, indicative of the absence of the lung tissue from this area. In pyo-pneumothorax this zone of transparency is bordered inferiorly by the dense shadow cast by the purulent collection, the upper border of which may be observed to undulate upon strong percussion of the chest wall during fluoroscopy.

Mediastinal Tumors.—In the case of tumors of the mediastinum the shadow of the tumor is fairly accurately reproduced, and its

size, extent, and relation to surrounding structures may be studied. A single large tumor casts a relatively dense shadow, whereas small, metastatic growths show multiple patches of impaired transparency within a fairly small space in the upper portion of the mediastinum.

The Heart.—The roentgenogram affords a valuable means of investigating the size, shape, and position of the heart and pericardium. Upon fluoroscopy, which is a ready and convenient method of studying cardiac conditions, during forced inspiration the transverse diameter of the cardiac shadow appears to diminish, to return again to its normal dimensions upon full expiration. Upward displacement of the diaphragm, due to increased intra-abdominal tension, causes an increase in the transverse shadow of the heart. Immense hypertrophy and dilatation also are indicated by an increase in the transverse diameter of the heart, whereas uni-lateral hypertrophy imparts an irregular contour to the cardiac shadow. Fluoroscopy and skiagraphy also reveal displacements of the heart toward the right or left by disease in the opposite lung or pleural sac; displacement upward by increased subphrenic pressure; or displacement downward from the weight of an aneurism of the aortic arch, or the pressure of the lungs in hypertrophic emphysema.

Pericarditis.—Acute fibrinous pericarditis yields no characteristic changes in the cardiac shadow. In sero-fibrinous pericarditis, however, the shadow cast by the precordial structures is increased transversely, particularly toward the right, encroaching upon the normal transparency in the cardio-hepatic angle of Ebstein. The shadow cast is not as dense as that produced by immense cardiac hypertrophy, and in addition it is roughly triangular, with the base resting upon the diaphragmatic shadow, not infrequently causing downward displacement of the left side of this muscle.

Aneurism of the thoracic aorta affords a shadow in the course of the vessel, with its size, location, and relation to surrounding structures. Upon fluoroscopy its pulsations may occasionally be made out.

SECTION III

DISEASES OF THE RESPIRATORY ORGANS

CHAPTER VIII

DISEASES OF THE BRONCHI

ACUTE BRONCHITIS

Pathology.—Acute bronchitis, an acute catarrhal inflammation of the mucous membrane of the medium sized and larger bronchi, occurs as a primary affection, and as a complicaton of many of the acute infectious diseases, notably the exanthematous fevers, influenza, typhoid fever, and malaria.

The disease is most prevalent during the sudden changes of early spring and late autumn. Among predisposing causes may be mentioned particularly acute coryza, affecting the upper respiratory passages; and passive congestion of the lungs incident to regurgitant heart lesions, acting upon the lower portions of the bronchial tree. The organism which is most frequently causative is the pneumococcus, alone or in conjunction with the staphylococcus, the colon bacillus, the micrococcus catarrhalis, or the bacillus typhosus.

During the early stages of the inflammation the mucosa of the bronchi is swollen and red, but is dry. During the further evolution of the disease, however, the congested mucous membrane becomes bathed with secretion, muco-purulent or purulent in character, containing large numbers of desquamated epithelial cells and bacteria.

Physical Signs.—Mild cases of acute bronchitis yield few physical signs, which are characteristic of the affection. In the more severe grades of bronchial inflammation physical signs are more in evidence, but often require a very careful examination to afford definite diagnostic data.

Inspection in mild cases usually reveals nothing abnormal, but in more severe cases there is dyspnea; while if there is a complicating or concomitant inflammation of the finer bronchioles

(capillary bronchitis), the condition is attended by a considerable degree of dyspnea and sometimes moderate cyanosis.

Palpation, negative in cases of moderate severity, in well developed cases may reveal slight rhonchal fremitus distributed over both lungs.

Percussion seldom elicits any alteration of the normal vesicular resonance in cases of frank acute bronchitis. Occasionally in very severe cases a slight impairment of resonance is evident over the bases posteriorly. The bases should be carefully examined daily in order that a complicating broncho-pneumonia may be recognized in its inception.

Auscultation during the early stage of the disease reveals the presence of sibilant and sonorous rales well distributed over both lungs. In a later stage of the affection, after secretion has become freely established, moist rales appear, the crepitant rale predominating.

Vocal resonance is not perceptibly altered. The respiratory murmur is harsh or puerile; but in uncomplicated acute bronchitis the breath sounds are never purely bronchial.

Diagnosis.—The diagnosis of acute bronchitis rests upon the absence of physical signs other than puerile breathing and a few rales distributed over both sides of the thorax, coupled with certain subjective symptoms, as an initial chill followed by moderate fever, a dry hacking cough which loosens with the establishment of the bronchial secretions; a feeling of rawness and pain beneath the sternum; and a general feeling of malaise and pains in the back and limbs.

In its abrupt onset the disease frequently is suggestive of *lobar pneumonia;* but this disease is eliminated by the absence of physical signs of consolidation; namely increased vocal fremitus and resonance, flatness, and blowing tubular breath sounds. Moreover, lobar pneumonia is almost always a uni-lateral affection, whereas acute bronchitis is bi-lateral in its manifestations. The constitutional toxemia and depression of lobar pneumonia far surpass that of acute bronchitis.

Broncho-pneumonia is usually gradual and insidious in its primary manifestations; and, in addition to the physical signs of a diffuse acute bronchitis, presents multiple areas of impaired resonance, over which the respiratory sounds are bronchial or at the least are broncho-vesicular. The constitutional disturbance accompanying broncho-pneumonia is more pronounced, and the dyspnea is of a more extreme grade.

Pertussis, during the first week or ten days, cannot be differentiated from acute bronchitis; but after the development of the first "whoop" the diagnosis is readily made. A history of exposure to pertussis may often be obtained.

CHRONIC BRONCHITIS

Pathology.—Chronic bronchitis is a chronic catarrhal inflammation of the mucous membrane of the medium sized and larger bronchi. Adults and elderly persons are the most frequent subjects of chronic bronchitis, the disease constituting the regular "winter cough" of many elderly persons.

Occasionally developing as a result of repeated attacks of acute bronchitis, chronic catarrhal bronchitis is much more commonly a sequence of chronic cardiac and real disease, gout, or chronic pulmonary disease, notably emphysema, phthisis and pneumonokoniosis.

In chronic bronchitis the bronchial walls are thickened from the deposition of fibrous connective tissue, while the bronchial musculature is atrophic. Frequently there is quite extensive proliferation of the mucous or goblet cells in the mucous membrane, which pour out a viscid grayish secretion containing innumerable desquamated epithelial cells. The lumen of the bronchial tube is in places diminished from hypertrophic thickening of the mucosa, while in other portions of the bronchial tree fusiform or saccular dilatations with atrophic mucosa and thin walls are present.

The secretion of the inflamed mucosa varies in quantity and character, being scanty and viscid in the Catarrhe Sec of Laennec, while in the opposite condition of bronchorrhoea serosa the secretion is abundant and serous, rarely mucoid or muco-purulent. In yet another type of the disease, the so-called putrid bronchitis, the bronchial secretion contains small yellowish bodies of very foul odor, Dittrich's Plugs.

Physical Signs.—*Inspection.*—The subject of chronic bronchitis is subject to attacks of shortness of breath or dyspnea upon moderate exertion. As the disease often occurs in emphysematous persons, the barrel chest of this disease with its limited degree of expansion is often seen.

Palpation, usually negative, sometimes shows the presence of rhonchal fremitus over both lungs.

Percussion.—The percussion note in chronic bronchitis is often

quite unchanged. In emphysematous subjects the note is hyperresonant, while over a large bronchial dilatation which is filled the note is dull, changing to tympany when the contents of the dilatation are expelled.

Auscultation.—The respiratory murmur is harsh, and in emphysematous subjects expiration is prolonged. Dry rales, sibilant and sonorous as well as moist rales are audible over both lungs, their character varying with the amount of secretion in the bronchial lumen. Vocal resonance is not perceptibly altered.

Diagnosis.—A history of chronic cough recurring every winter, with rales generally distributed over both lungs, and frequently an emphysematous thorax, render diagnosis not difficult.

The chronic cough of aortic aneurism is usually accompanied by stridulous respiration owing to uni-lateral vocal cord paralysis. The possibility of a tumor producing chronic cough by pressure should not be forgotten.

BRONCHIECTASIS

Pathology.—Dilatation of the bronchi is often the result of chronic bronchitis, the accumulation of the secretion in the weakened bronchial tubes producing dilatations. In other instances in chronic bronchitis the dilatations are produced by the pressure of the air in the bronchus during violent paroxysms of cough. Some dilatations are explained by the action of traction of peri-bronchial adhesions in chronic interstitial pneumonia or fibroid phthisis.

The bronchial dilatations are found most commonly in the right lung, in which they affect mainly the bronchi of the middle and lower lobes. Two principal forms of dilatation are found, namely saccular, and cylindrical. The saccular dilatations are usually surrounded by an area of indurated lung, the dilatation having been produced by the traction of the adherent peri-bronchial tissues. Cylindrical dilatation most commonly affects the smaller bronchial tubes, but this form of dilatation is sometimes encountered in the larger tubes. The two forms of dilatation, saccular and cylindrical, are often found in the same lung.

The state of the mucous membrane lining the dilatations varies. In some instances scarcely altered, in most cases it is thickened with polypoid elevations upon the surface; or, in dilatations containing abundant secretion, the mucosa is not infrequently ulcerated. The exudation from the walls of bronchiectatic dilatations

is usually purulent and abundant, occasionally thick and cheesy in consistence.

Physical Signs.—*Inspection.*—Small bronchiectatic dilatations do not produce characteristic physical signs. Advanced cases, in which the dilatations have attained considerable size, cause uni-lateral impairment of the expansion of the thorax, and sometimes retraction of the affected side with drooping of the shoulder.

Palpation.—In the presence of a large bronchiectatic cavity situated near the surface of the lung, with a patent bronchial com-

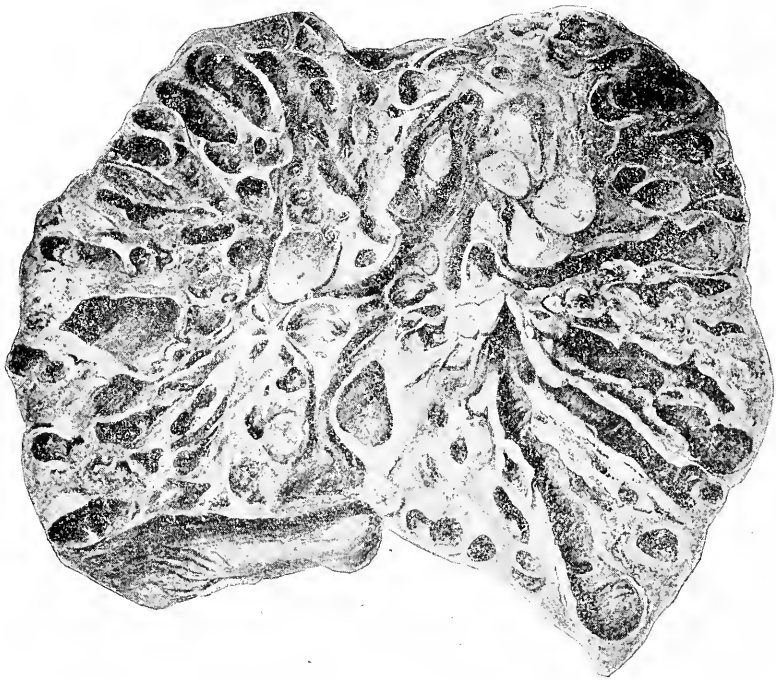


Fig. 69.—Sacculated bronchiectasis. (Pottenger, after Powell and Hartley.)

munication, vocal fremitus is very markedly increased when the cavity is empty, to become abolished when the cavity is filled with fluid.

Percussion.—The results of percussion in bronchiectasis vary, depending upon whether the cavity under investigation is empty or is filled with secretion. If the cavity or dilatation contains fluid, even though it has a patent bronchial outlet, the percussion note is flat; whereas, if the cavity is empty, it yields tympany or a cracked-pot sound upon percussion. In suitably situated cavities or dilatations all the signs of pulmonary cavity formation, such as

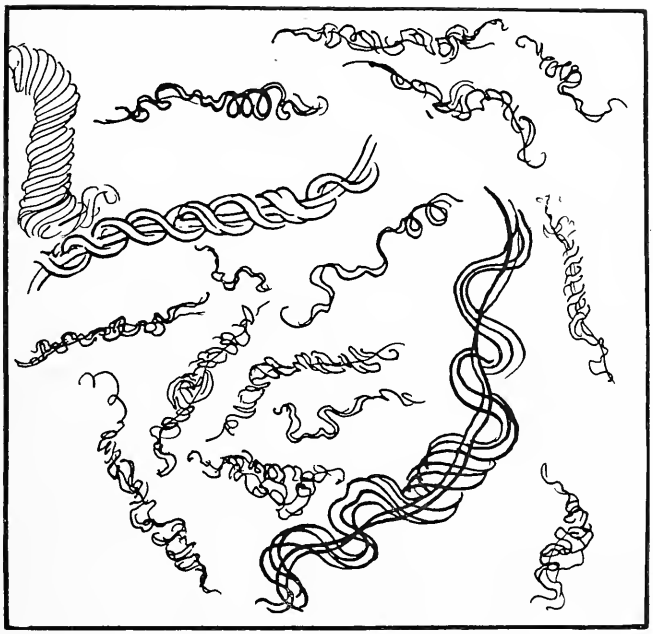


Fig. 70.—Curschmann's spirals. In chronic bronchitis, the cilia within the bronchi whip the mucus and other inflammatory detritus into various shaped masses. Some are large enough to be seen with the naked eye but many require magnification to be seen. They commonly exist in asthmatic sputum. (From Brown.)

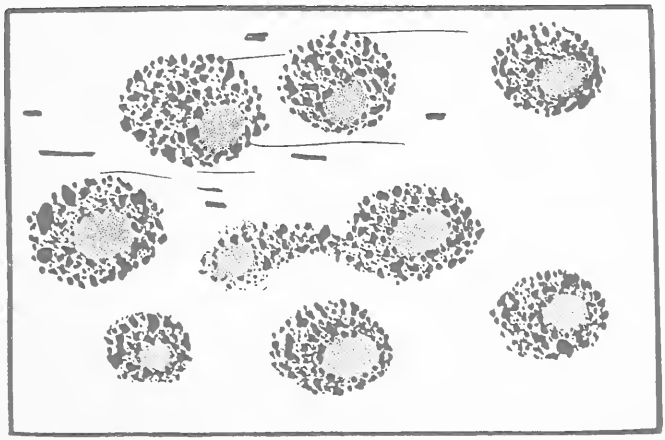


Fig. 71.—Eosinophiles. A considerable percentage of the pus cells of asthmatic sputum are eosinophiles. This is probably indicative of chronic intoxication. (From Brown.)

Wintrich's change of sound, Friedreich's respiratory change of sound, and the change of sound of Gerhardt, may be elicited.

In every case in which the signs of cavity are evanescent, present and absent at successive examinations, the possibility or probability of bronchiectasis should be borne in mind, as these cavities fill with secretion which masks all physical signs, and then the signs of cavity reappear when the contents have been evacuated.

Auscultation.—In cases of bronchiectasis which are of relatively short duration, auscultation yields only the signs of chronic bronchitis, puerile breath sounds and rales. If, however, a rather large dilatation be properly situated with reference to its bronchial outlet, amphoric breathing will be encountered. If a large dilatation is situated near the surface of the lung, and has a free bronchial outlet, vocal resonance is very much exaggerated, perhaps to the extent of affording bronchophony or pectoriloquy.

Diagnosis.—Cases of moderate bronchiectasis are sometimes difficult to distinguish from chronic bronchitis, of which disease it is often a sequel. In cases of longer standing, in which more extensive organic change has occurred in the bronchial system, the expectoration of a copious amount of sputum at one time, followed by an absence of expectoration for several hours, is suggestive; and when signs of cavity formation can be elicited at the base of the lung, the diagnosis is clear-cut and readily made.

A bronchiectatic cavity at the base of the lung must be differentiated from a tuberculous cavity in this locality. In tuberculosis in addition to signs of cavity, there is apt to be considerable deformity of the thorax, with fever and night sweats, while the course of the disease is steadily downward. In bronchiectasis, on the contrary, the physical signs of cavity persist for a long period of time, and the patient remains in comparatively good health.

BRONCHIAL ASTHMA

Pathology.—Bronchial or spasmodic asthma is a paroxysmal dyspnea which is almost entirely expiratory in type, the subject of the disease being unable to expel the air from the lungs. Bronchial asthma has nothing in common with the so-called cardiac asthma or renal asthma.

Numerous theories and hypotheses have been advanced in the attempt to explain the cause of bronchial asthma. Most authors agree that there is a marked neurotic element in these subjects. It has been suggested that the attack is caused by a sudden spasm

of the bronchial muscles; also that the obstacle to egress of the air from the lungs is due to narrowing of the lumen of the bronchioles by temporary and transient turgescence of the mucosa. Curschmann states that the underlying cause is a special form of inflammation of the smaller bronchial tubes, the so-called bronchiolitis exudativa of this author. Spasm of the diaphragm has been alleged to be the underlyng cause of the paroxysms.

Very little has been recorded in reference to the morbid anat-

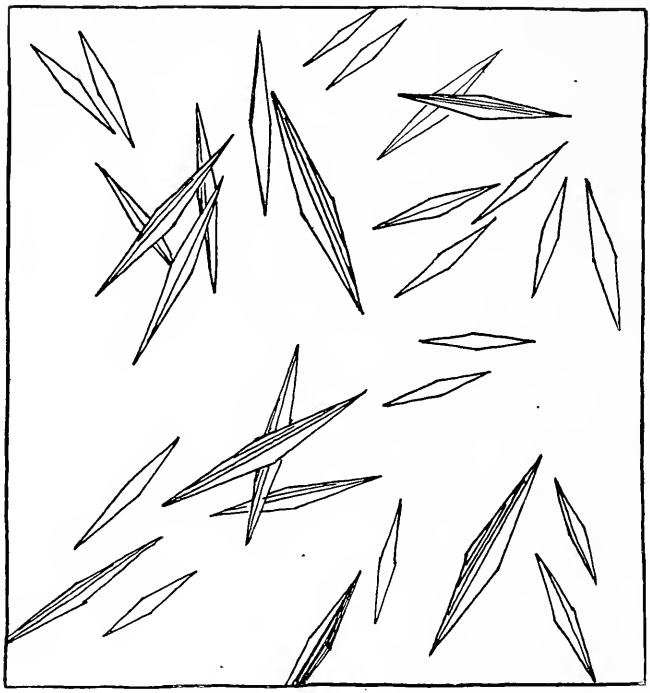


Fig. 72.—Charcot-Leyden crystals. These crystals are formed in sputum of chronic bronchitis, especially if asthma exists. They have been repeatedly found in other locations. They seem to indicate decomposition. (From Brown.)

omy of this disease, as but half a dozen autopsies are contained in the literature. In such cases as have been examined the ciliated epithelium of the bronchi has been found in a state of desquamation, with bronchial congestion and exudation rich in eosinophilic cells. The blood during bronchial asthma contains an excess of eosinophiles, these cells representing 25 per cent to 35 per cent of all the leukocytes.

The sputum in bronchial asthma is characteristic of the dis-

ease. In the early stages it is scanty and very tenacious, containing Curschmann's spirals and Charcot-Leyden's crystals. Macroscopically Curschmann's spirals are white or yellow, taking the form of twisted threads or small balls. The length of the spiral rarely exceeds half an inch, but may exceed two inches in some instances. Under the microscope they appear as mucous threads containing a clear central fiber, around which are wound many fine fibrils. Eosinophiles are often entangled in the meshes of the fibrils.

Charcot-Leyden crystals are colorless, pointed, octahedral crystals, the average length of which is about three times the diameter of a red blood cell. They are often absent in freshly expectorated sputum, but appear after it has stood for a short time.

During the later stages of the attack these two pathognomonic elements of the sputum disappear, the expectoration becoming more abundant and muco-purulent.

Physical Signs.—*Inspection.*—The paroxysm of bronchial asthma comes on suddenly, dyspnea of the expiratory type being the first and principal sign. In the course of the paroxysm the face and hands become cyanotic; the veins of the neck swell; the patient is obliged to sit upright in his efforts to empty the chest; the chest is large and fixed; the diaphragm is depressed; and there is a marked limitation of the degree of expansion of the thorax.

Palpation.—During the attack vocal fremitis, if it can be determined, is diminished owing either to the rarefaction of the pulmonary parenchyma by the increased air content or to bronchial obstruction by swelling of the mucosa or muscular spasm. In certain cases rhonchal fremitus is marked.

Percussion.—The note upon percussion is hyper-resonant, owing to the excess of air in the lungs which the subject is for the time unable to expel adequately.

Auscultation.—The normal vesicular murmur is replaced by numerous loud sibilant and sonorous rales distributed over both lungs. These rales are so pronounced that they may often be heard without the use of the stethoscope. Later in the attack, after the bronchial secretions have become freely established, the dry rales give place to moist and bubbling rales. The expiratory phase of the vesicular murmur is markedly prolonged, being dotted with rales.

Diagnosis.—Owing to the paroxysmal character of the asthmatic attack, its marked expiratory dyspnea, and the characteristic sputum, bronchial asthma is very readily diagnosed.

TRACHEO-BRONCHIAL STENOSIS

Pathology.—Stenosis of the trachea or bronchi may arise from causes acting from the interior of the trachea or bronchi, or from extraneous causes. Among intra-tracheal and intra-bronchial factors may be mentioned polypi and tumors, diphtheritic inflammation of the mucous lining, cicatrices from ulcers, the lodgment of foreign bodies, and rarely perichondritis. Among extraneous causes may be mentioned pressure upon the trachea by a thyroid enlargement, the pressure of an aneurism upon the trachea or bronchi, or pressure of a greatly hypertrophied heart or pericardial effusion.

The effect of the obstruction upon the lungs depends upon the site of the obstruction. Total obstruction of the trachea causes collapse of both lungs. Obstruction of a main bronchus causes collapse of one entire lung. Obstruction of a large bronchus in a lung causes collapse and airlessness of a large portion of the lung; while obstruction of a small bronchus has but little effect upon the lung, as only a small area of the lung is thus deprived of its free outlet.

When an obstruction occurs in any portion of the bronchial tree, the air in the infundibula supplied by this bronchus is absorbed and the pulmonary tissue collapses, constituting the so-called obturation atelectasis.

The tissues immediately surrounding a localized bronchial obstruction are emphysematous, the unobstructed portions of the lung expanding vicariously.

Physical Signs.—*Inspection.*—The physical signs of bronchial and tracheal obstruction vary with the site of the obstruction. In tracheal obstruction the signs are bi-lateral, affecting both lungs, while in stenosis of one of the primary bronchi the signs are uni-lateral, but affect an entire lung; while in minor grades of obstruction, that is, in obstruction of a small bronchus or bronchiole, only a small portion of the lung is affected and the physical signs arising from the obstruction are very slight or nil.

In most cases the patient is dyspneic; and, if the stenosis affects a large bronchus, there is some degree of cyanosis. In obstruction of a main bronchus the expansion of the lung supplied is negligible, the opposite side of the chest expanding vicariously. In tracheal obstruction or rather in partial tracheal obstruction, there is inspiratory dsypnea, the accessory muscles

of respiration acting vigorously but only a small amount of air entering the lungs, so that the patient becomes rapidly cyanotic. The inspiratory efforts are attended by depression of the supra- and infra-clavicular fossae, retraction of the intercostal spaces and of the epigastrium.

Palpation.—In obstruction of a main bronchus vocal fremitus is abolished over the entire area of the lung. In minor grades of obstruction the fremitus is little affected, owing to the small area of the lung involved.

Percussion.—In obstruction of a main bronchus the percussion note is dull over the area of distribution of the bronchus, or over the entire lung. In minor degrees of obstruction the note is scarcely changed, and may be even hyper-resonant owing to the compensatory emphysema of the adjacent infundibula.

Auscultation.—Upon auscultation over the area of an occluded principal bronchus the respiratory murmur is feeble or entirely absent, while over the opposite lung it is exaggerated or puerile. Coarse sonorous rales may sometimes be elicited over the site of the obstruction. Vocal resonance is abolished over the affected lung in obstruction of a main bronchus; but is little affected in minor grades of obstruction.

Diagnosis.—The presence of inspiratory dyspnea, sibilant and sonorous rales in a circumscribed area, and dullness over a large area of the lung or the entire lung is suggestive of bronchial obstruction. It is important to determine the site of obstruction, whether in the larynx, trachea, or bronchi. In laryngeal obstruction there is vigorous movement of the larynx, the attitude of the head is fixed and somewhat thrown back, and the respiration is stridulous. Finally, the use of the laryngoscope will reveal the constriction or obstructing body. Tracheal obstruction produces stridulous breathing, with limitation of the laryngeal movements, and orthopnea.

CHAPTER IX

CIRCULATORY DISTURBANCES OF THE LUNGS

PULMONARY CONGESTION

Pathology.—Congestion of the lungs occurs in two forms; namely as active congestion, and as passive congestion.

Active congestion of the lungs occurs in the early stages of inflammations of these organs, as in the period of engorgement of lobar pneumonia. But active congestion frequently occurs in conditions which do not become so grave as the first stage of pneumonia, in which event it is the result usually of the inhalation of irritating gases, of hot or cold air, and occurs as collateral congestion due to disease of only a portion of one lung, the congestion affecting an adjacent portion. Active congestion of the lungs, while not usually dangerous, has in rare instances ended fatally.

Post-mortem the lung in active congestion is enlarged, is deep red, its consistence is increased, yet the lung will float when placed in water.

Passive Congestion of the lungs occurs in two forms; namely as *mechanical congestion,* and as *hypostatic congestion.*

Mechanical congestion results from an obstacle interposed to the return of the blood from the lungs to the heart. The most common cause operating in this manner is mitral regurgitation, while a less frequent cause is a tumor pressing upon the great veins returning blood to the heart. Mitral stenosis and aortic insufficiency and stenosis operate similarly to produce mechanical congestion of the lungs.

In passive congestion of the lungs the vessels of the lung are dilated and the inter-alveolar septa distended with fluid, while the air vesicles contain hemorrhagic exudate. In the alveoli are found desquamated alveolar cells containing blood pigment, the so-called "heart-failure cells."

Hypostatic congestion of the lung is encountered in adynamic and asthenic states, particularly in elderly subjects who have been long in the recumbent posture during a continued fever or

chronic wasting disease. The congestion in this instance is localized to the posterior and inferior portions of the lungs and is largely dependent upon general asthenia and relaxation of the pulmonary vessels. That it is not entirely due to the supine posture is evidenced by the fact that it only occurs in subjects weakened by disease.

Physical Signs.—*Inspection.*—In active congestion of the lungs inspection reveals the fact that the patient is dyspneic, and perhaps cyanotic.

Palpation in active congestion will reveal slightly increased vocal fremitus, bi-lateral, most pronounced at the bases posteriorly, whereas in passive congestion the fremitus is less intense than normal.

Percussion.—In active congestion the percussion note is apt to be slightly hyper-resonant owing to the increased tension of the pulmonary tissues; whereas in passive congestion there is impairment of the resonance at the bases posteriorly owing to the gravitation of the blood to those regions of the pulmonary system.

Auscultation reveals in active congestion broncho-vesicular breathing; and, in cases of passive congestion will show in addition the valvular lesion which is responsible for the congestion. In both instances the pulmonic second sound is accentuated owing to the increased tension in the pulmonary circuit. In passive congestion moist or bubbling rales are not infrequently encountered at the bases posteriorly.

Diagnosis.—If the physical signs detailed in the preceding paragraphs occur abruptly without warning, active congestion of the lung may be inferred; while, if a valvular lesion of the heart is coupled with the respiratory signs, the diagnosis of pulmonary congestion is yet more probable.

EDEMA OF THE LUNGS

Pathology.—Pulmonary edema arises from a number of causes, and may be general, involving both lungs, or local, in which event it is circumscribed to a portion of one lung. The most prolific cause of pulmonary edema is insufficiency affecting the mitral valve, less frequently the aortic valve, thus heightening the blood pressure in the pulmonary circulation. Nephritis may be responsible for pulmonary edema, as may local disease of the lung, as infarction, phthisis, or pneumonia. Angio-neurotic edema has been considered a cause in certain instances, while in

other cases the edema has been attributed to vasomotor paresis.

The edematous lung is heavy, the alveoli filled with fluid, and the inter-alveolar walls are thickened and edematous. The lung readily pits upon pressure, and on section sero-sanguineous fluid exudes.

Physical Signs.—*Inspection.*—The subject of pulmonary edema is dyspneic, the dyspnea usually amounting to orthopnea, with a very anxious facial expression. Cough is frequent, the cough of the wet lung, and the expectoration is abundant, frothy, or sero-sanguineous.

Palpation reveals diminution of vocal fremitus, while rhonchal fremitus, the tactile equivalent of the numerous moist rales, is readily detected over both bases posteriorly.

Percussion.—The resonance of the percussion note is impaired over the bases posteriorly, whereas over the anterior surface of the chest in the infra-clavicular and mammary regions skodaic resonance is not infrequently encountered.

Auscultation.—Upon auscultation of the bases numerous mucous, bubbling rales are heard. The pulmonic second sound is accentuated.

Diagnosis.—The diagnosis rests upon the characteristic physical signs occurring in a patient with an obstacle to the return of blood to the heart, or in an asthenic state, and upon the expectoration of abundant frothy, sometimes blood-tinged sputum, which often is raised in gushes, which escape from the mouth and nose.

PULMONARY INFARCTION

Pathology.—Infarction of the lung occurs as a result of an embolus or thrombus plugging the end of one of the terminal arteries of the pulmonary system. The condition arises most frequently in connection with valvular lesions of the heart particularly those associated with acute or chronic endocarditis.

The infarcts are usually situated near or at the periphery of the lung; they are wedge-shaped, with the base of the wedge directed toward the surface of the lung. When recent, the areas of infarction are dark red in color, resembling a blood clot. Later, as partial organization occurs and the hemoglobin is partially removed from the mass, they assume a yellowish color. Eventually the infarct becomes organized, leaving a puckered scar at the site of the infarction. The pleura overlying an area of infarction usually shows signs of localized inflammation.

Microscopically the alveoli of the lung in the area of infarction are crowded densely with erythrocytes, which are also found in the inter-alveolar walls.

Infarcts of the lung are not infrequently multiple. In size they vary from that of a walnut to the size of an orange, but in rare instances may be extremely large, occupyng nearly an entire lobe of a lung.

An infarct of the lung may undergo several terminations. If, the embolus which plugged the terminal artery is non-infectious, the infarct becomes organized and eventually forms a puckered scar at the site of the infarction. If, on the contrary, the embolus be of septic origin, the infarction may be the starting point of a pulmonary abscess or of pulmonary gangrene.

Physical Signs.—The signs referable to pulmonary infarction vary, depending upon the number, size, and distribution of the areas of infarction.

Inspection reveals labored breathing, often amounting to dyspnea or orthopnea. The facial expression is anxious, and hemoptysis is not infrequent.

Palpation may show increased vocal fremitus if the area of infarction is of considerable extent and situated near the periphery of the lung, whereas, if the infarct is centrally placed, near the root of the lung, no alteration of vocal fremitus will be demonstrable. When a peripheral infarct overlies a main bronchus vocal fremitus is markedly exaggerated.

Percussion.—Over large infarcts dullness is encountered, whereas in a large infarct directly overlying a large bronchus, the tympany of the bronchus is transmitted to and engrafted upon the dullness of the percussion note.

Auscultation.—In suitably situated infarcts with reference to a main bronchus, loud, bronchial or tubular breath sounds are elicited, as well as rales transmitted from the bronchus. In cases of multiple small infarcts, or deeply seated infarction the breath sounds are broncho-vesicular or vesicular.

Diagnosis.—When the signs mentioned in the preceding paragraphs are elicited in a patient suffering with valvular heart disease, from which embolism might arise, infarction of the lung is suggested. As the infarctions are often situated in the lower lobes of the lung posteriorly, the physical signs arising from a large infarct may simulate rather closely those of lobar pneumonia.

An infarction of moderate size which is situated centrally produces few signs by which a diagnosis may be made, practically

the only sign elicited being slight embarrassment of the respiration.

A septic infarct naturally produces signs of septic poisoning with later development of the signs of pulmonary abscess.

PULMONARY NEOPLASMS

Clinical Pathology.—Tumors of the lung and pleura may be primary or secondary to tumor arising elsewhere in the body and implicating the pulmonary tissues as a result of metastasis. Of the two, the primary is decidedly rare, and the secondary more common.

The primary tumors of the lung comprise carcinoma, sarcoma, and endothelioma. Carcinoma in its evolution involves usually one lung, where it forms a large mass, and later breaks down, forming a cavity. But in other cases there develops a diffuse cancerous infiltration of the lung, simulating pulmonary tuberculosis.

The secondary tumors of the lung comprise all varieties of malignant growths. Secondary carcinoma of the lung rarely forms a single tumor, but is usually multiple, and not uncommonly involves the pleura. The cancerous nodules are diffusely scattered over both lungs. This represents metastases from a primary tumor which may be situated in the breast, the gastrointestinal tract, the genitourinary tract, or bone. Hodgkin's disease may affect the lung, traveling there by way of the mediastinal and bronchial lymphatic glands.

Carcinoma of the lung produces swelling of the bronchial glands and mediastinal glands and sometimes of the glands of the neck. Pleurisy is a common complication or accompaniment of pulmonary carcinoma, and may be hemorrhagic.

Men are afflicted more frequently with primary neoplasms of the lungs, while women are more often the victims of secondary tumors in this region.

Physical Signs.—The physical signs of tumor of the lung may be caused by the presence of the tumor or may be due to the accompanying pleural effusion, when this is present. In the latter event the signs of pleurisy with effusion will overshadow the other signs present.

The superficial veins of the thorax and the veins of the neck may be tortuous and overdistended, owing to compression of the superior vena cava in the chest. The contour of the chest is changed. In the case of a very large growth there is uni-lateral

bulging and widening of the intercostal spaces, whereas in the case of a small growth causing collapse of the adjacent pulmonary tissues, or to traction by adhesions, there will be restriction of the expansion and local depressions of the chest wall.

Vocal fremitus is sometimes exaggerated, at other times diminished. A hyper-resonant note is elicited if the tumor has broken down and formed a superficial cavity; while dullness or flatness is obtained over a large growth involving a large area of the lung.

The breath sounds may be suppressed, may be bronchial, or amphoric. The latter type of breathing is a sign of excavation of the lung by breaking down of the morbid growth.

Diagnosis.—In primary cases the diagnosis is always difficult but the presence of strictly uni-lateral signs, with enlarged glands is of assistance. In pulmonary carcinoma rarely carcinomatous tissue may be demonstrable in the sputum; and late in the course the growth may perforate the chest wall. Mediastinal tumors and aneurism of the aorta are hard to differentiate.

CHAPTER X

DISEASES OF THE LUNGS

LOBAR PNEUMONIA

Pathology.—Lobar, fibrinous, or croupous pneumonia is an inflammation of the lung, accompanied and attended by a variable degree of constitutional toxemia.

The cause of lobar pneumonia is the pneumococcus, or diplococcus pneumoniæ, first discovered in the sputum of pneumonia patients by Sternberg and Pasteur in 1880 and recognized as the cause of the disease by Fraenkel in 1884. The pneumococcus may be found in pure culture in the sputum of pneumonia patients or associated with the streptococcus, staphylococcus, or Friedlander's bacillus. Present in the oral and nasal secretions of many persons during health, it is a question whether lobar pneumonia is due to these strains of the pneumococcus or to a special strain.

Lobar pneumonia is very prevalent at the extremes of life, young infants and elderly persons being very susceptible to the disease. Most cases develop during the late winter or early spring months.

The morbid changes incident to lobar pneumonia pass through three or four rather well defined stages or periods; namely, the stage of engorgement, the stage of red hepatization, the stage of gray hepatization; and, if the patient recovers, the stage of resolution. However, these stages of the inflammation are not always recognizable as distinct and separate entities; and it is not uncommon to find one stage more or less blended with another.

The stage of engorgement is of brief duration, rarely exceeding 24 hours, as it is early followed by hepatization of the lung. During the period or stage of engorgement the lung is dark red in color, firm to the touch, feels boggy, but still crepitates, and the lung will float when placed in water. Microscopically during this stage the capillaries are distended with erythrocytes, the alveolar walls are thickened, and the alveolar spaces contain a

variable number of erythrocytes, leukocytes, and desquamated epithelial cells.

During the stage of red hepatizaton the pulmonary tissues in the diseased area of the lung are solid, firm, and devoid of air. The lung is enlarged, and may present indentations upon its surface corresponding to the ribs with which it is in contact. Upon section, the cut surface is dry, reddish or brown in color, and very friable. Upon scraping the cut surface with the knife, small fibrinous plugs may come away from the terminal bronchioles. The lung does not now crepitate; and sinks when placed in water. Microscopically the alveoli are observed to be filled with a dense, dry, fibrinous exudate, containing erythrocytes, leukocytes, and desquamated epithelial cells embedded and entangled in a matrix of fibrin, the erythrocytes predominating during this stage of the disease.

In the stage of gray hepatization the pulmonary tissue loses its reddish color upon section and becomes more or less gray or grayish-white. Section shows a moister surface than in the preceding stage, and but few fibrinous plugs can be scraped from the small bronchioles. Microscopically the polymorphonuclear leukocytes predominate, though some erythrocytes, as well as desquamated epithelial cells, are present in the fibrinous mass. However, in spite of this partial clearing of the alveoli, the lung is still not crepitant, and will sink when placed in water.

During the stage of resolution, if it occurs, the fluid is drained from the lung by the lymphatics, the debris is removed by phagocytes and also expectorated with the sputum, the lung gradually assuming its normal characters. The lung again becomes crepitant, and sections float when placed in water.

The expectoration during the active stage of lobar pneumonia is thick and viscid, and of a brownish color, the so-called "prune juice sputum." It is so viscid that it will not always separate from its container when it is turned upside down.

During the inflammation of the lung in lobar pneumonia the pleura is practically always involved over the area of consolidation, becoming roughened and not infrequently pouring out a moderate degree of serous fluid. However, in central pneumonia, in which the consolidation is deeply situated near the root of the lung the pleura escapes. It is, however, a question whether a pneumonia ever remains central, or whether every pneumonia which begins near the root of the lung eventually does not progress and affect the periphery of the lung.

Lobar pneumonia, as the name implies, usually involves an entire lobe of the lung. The disease is usually uni-lateral. In relative frequency the different portions of the lungs are involved in the following order: lower right lobe, lower left lobe, upper right lobe, an entire lung, or rarely both lungs.

According to the distribution of the disease several clinical types of lobar pneumonia have been described:

Apical Pneumonia affects only the apex of a lung.

Migratory Pneumonia successively involves lobe after lobe of the lung in regular progression.

Double Pneumonia involves both lungs.

Massive Pneumonia is a form in which, in addition to the alveoli, the bronchial tubes of an entire lobe or lung are plugged with fibrinous exudate.

Central Pneumonia is a form in which the disease is situated deeply at the root of the lung, and does not at once involve the peripheral portions.

Physical Signs.—*Inspection.*—The decubitus of the patient is often suggestive of lobar pneumonia. He may be found lying on the diseased side or may be found sitting up in bed with the spine curved toward the diseased side. Herpes labialis is a very common finding in lobar pneumonia, and a red spot or flush upon the cheek of the diseased side is a very frequent sign. The respiratons are short and accompanied by an expiratory grunt.

In a case of uni-lateral pneumonia, and the disease in the vast majority of cases is uni-lateral, inspection reveals restriction of the excursion of the chest on the diseased side, with exaggerated excursion of the sound side. The diseased side does not expand to its normal physiological capacity for two reasons: the air space in the lung is actually decreased; and, moreover, the pleurisy accompanying the pneumonic process causes the patient to inhibit the respiratory movements as much as possible. The sound lung, on the contrary, expands vicariously because it must as far as possible take on the work of the affected lung in order that the tissues may not suffer for want of their normal supply of oxygen.

Litten's diaphragmatic shadow is absent on the affected side of the chest.

In the cases in which the left lung is involved anteriorly, the cardiac impulse is very prominent, because the portion of this lung which overlaps the heart is enlarged and more or less firm and is pushed before the heart with each impact of that organ.

Palpation.—Upon palpating over the consolidated or diseased lobe or region of the lung, particularly in the stage of red hepatization the most striking finding is a marked increase of vocal fremitus. This increase of fremitus over the base posteriorly, where it is usually encountered in lobar pneumonia, is very striking since under normal conditions of the pulmonary parenchyma these vibrations are very indistinct or absent in this region. The fremitus is not altered during the period of engorgement, but appears during the period of red hepatization, disappearing again with the supervention of resolution.

There are two conditions under which the fremitus may be entirely absent over the consolidated area. If the main bronchus leading into this area becomes plugged with fibrinous exudate, as is frequently the case in massive pneumonia, the voice vibrations will not be appreciable to the palpating hand. Again, if there be extensive involvement of the pleura with effusion, the fluid masks the palpable vibrations.

During the period of engorgement and the early part of the stage of red hepatization the pulse is full and bounding, the heart acting powerfully as a result of the raised blood pressure in the pulmonary circulation. In the later stages of the disease, when the toxemia is well established, the heart is prone to undergo more or less severe parenchymatous myocarditis, the pulse becoming rapid, running, and feeble.

Percussion.—During the stage of engorgement, during the first twenty-four hours of the disease, the percussion note yields skodaic resonance. Percussion during the stage of red hepatization shows impaired resonance or actual flatness over the involved lobe, while percussion just above the consolidation, on the contrary, yields skodaic resonance, due to relaxation of the tissues, which are compressed by the increased size of the consolidated lobe.

In a case of central pneumonia percussion will reveal ordinary vesicular resonance; or, at most, only slight impairment of resonance, because the lung immediately beneath the chest wall is not consolidated, and yet such a patient will exhibit all the toxic symptoms of a severe lobar pneumonia.

During the latter portion of the stage of gray hepatization and during the stage of resolution, the lung shows a gradual return to the normal vesicular resonance over the diseased area.

Auscultation.—During the period of engorgement the breath sounds are quiet and partially suppressed, while at the completion of full inspiration there occurs a very valuable diagnostic

sign, namely a fine crepitant rale, the rale indux. This rale is produced by the separation of the walls of the infundibula which have been glued together with sticky, viscid exudate, and presents to the examining ear a series of sharp cracklings.

When the consolidation is well established, in the stage of red hepatization, this rale disappears, and is replaced by distinct bronchial or tubular breathing. During the latter portion of the stage of gray hepatization and during the period of resolution, when the consolidation becomes macerated and partially dissolved by serum, the bronchial breath sounds disappear and are replaced by a sub-crepitant rale, the rale redux, which is produced by the separation and approximation of the finer bronchiolar walls during expiration and inspiration.

The pulmonic second sound is accentuated owing to the increased load thrown upon the right heart due to the obstacle offered to the passage of the blood through the diseased lung. In prolonged and severe cases there is frequently a reduplication of the second sound of the heart, due to a-synchronous closure of the aortic and pulmonic valves, caused by the unequal tension in the general and pulmonary circulations.

Diagnosis.—In a case of frank lobar pneumonia in an adult with sudden onset with pain in the side, initial chill and rapidly rising fever, coupled with the development of a rusty tenaceous sputum, the diagnosis is not difficult. But in young children, in the aged, in alcoholic subjects, and in terminal or secondary pneumonias engrafted upon other conditions as cancer, nephritis or diabetes, the diagnosis is often reached with difficulty.

In a case of frank uncomplicated lobar pneumonia the physical signs are distinctive; lessened or deficient expansion of the diseased side of the thorax, exaggeration of vocal fremitus, dullness or flatness on percussion, bronchial breathing with the subcrepitant and crepitant rale on auscultation. But it should be remembered that in massive pneumonia the vocal fremitus may be lessened or absent over the diseased area owing to the main bronchus supplying the part being plugged with tenaceous exudate. Moreover, in ordinary pneumonia, in the routine case so to speak, the dullness is preceded and followed by a rather tympanitic note, occurring prior to and following complete and frank hepatization of the pulmonary tissue in the area of disease. So also the bronchial breath sounds are absent or a-typical in the presence of incomplete hepatization of the lung, in partial plugging of a bronchus, or in the presence of a complicating pleurisy

with effusion. Other data for diagnosis are that the disease usually terminates by crisis at the 7th or 9th day; that the onset is abrupt; that the pulse-respiration ratio is markedly altered; that there is usually labial herpes; and that there is usually a hectic flush upon the cheek upon the side of the disease.

The localization of the disease in the lung is prone to influence the physical signs and give trouble in diagnosis. Thus, in Massive Pneumonia the ordinary physical signs may be lacking over a large area of the lung, giving physical signs simulating pleurisy with effusion. In this class of cases it is occasionally possible to dislodge the plugging exudate by coughing.

Central pneumonia does not give the typical picture of frank lobar pneumonia. Here the lesion, starting deeply within the lung, overlaid as it is by normal pulmonary tissues, yields a rather broncho-vesicular respiratory murmur and only moderate impairment of pulmonary resonance. However, as a rule, these cases can only be said to be central in their incipiency, as they usually eventuate in an ordinary lobar pneumonia involving the peripheral portions of the lung.

Lobar pneumonia not infrequently is a source of pain in the right lower portion of the abdomen, thus simulating acute appendicitis. In other instances the disease is attended by constipation, abdominal pain and meteorism, simulating intestinal obstruction.

In drunkards the cerebral symptoms of pneumonia predominate; while in children cerebral symptoms are prominent and the rusty sputum may be absent throughout the course of the disease.

Lobar pneumonia must be differentiated from Acute Tuberculo-pneumonic phthisis, broncho-pneumonia, pulmonary infarction, pulmonary edema, pulmonary congestion from cardiac defects, and from pleurisy with effusion.

Acute Tuberculo-Pneumonic Phthisis.—In the early stages of the disease it is often impossible to differentiate between this disease and lobar pneumonia. This form of tuberculosis, often termed "galloping consumption," begins abruptly with chill, pain in the side, and cough attended by sputum which is at first mucoid, and later rusty. The physical signs are those of consolidation of one or more lobes, or possibly of an entire lung. The chill is followed by a rapid rise of fever and the picture is that of frank lobar pneumonia. But at the seventh or ninth day no crisis occurs; but, on the contrary, the fever persists, is attended by night sweats, while elastic fibers and tubercle bacilli appear in

the sputum. Evidence of softening now appears, with moist or gurgling rales and other signs of cavity formation.

Broncho-Pneumonia.—Broncho-pneumonia is usually gradual in onset and is usually secondary to other infectious fevers, as measles, influenza, scarlatina, or typhoid fever, or occurs as the result of the aspiration of blood from hemoptysis or the decomposition of the contents of bronchiectatic cavities.

The distribution of the disease is of aid in differentiating the two conditions, broncho-pneumonia being bi-lateral, whereas lobar pneumonia as a rule affects one lobe or one lung. Moreover, the age at which the two diseases are most prevalent differ. Broncho-pneumonia is most frequently encountered in young children, under three years of age, whereas lobar pneumonia is much more common after the third year.

The physical signs of broncho-pneumonia are diffuse, the patches of consolidation being scattered well over both lungs, so that the percussion note is never frankly dull or flat, but there is bi-lateral moderate impairment of pulmonary resonance. Similarly broncho-pneumonia is not attended by frank bronchial breathing, but rather by broncho-vesicular breathing.

Finally, broncho-pneumonia resolves by lysis, whereas lobar pneumonia terminates by crisis.

Pulmonary Infarction.—Infarction is abrupt in onset with dyspnea, cough, and the expectoration is not viscid, but fluid and often tinged with blood or almost pure blood. In simple infarction there is less fever than in lobar pneumonia; but in septic infarction a local pneumonia frequently develops at the site of the infarct, and leads to pulmonary abscess with its hectic temperature and sweats. There are often signs of an associated heart lesion. Infarction most frequently occurs in persons who suffer with chronic endocarditis.

Unless very large the infarcts produce very few distinctive signs, though large ones in the lower lobes produce broncho-vesicular or bronchial respiration when properly situated with reference to a large bronchus.

Pulmonary Edema develops in patients with valvular heart lesions, or nephritis, producing extreme dyspnea or orthopnea, with numerous liquid rales at the bases posteriorly. The respiratory sounds are weakened rather than purely bronchial. The sputum is abundant and characteristic; the condition is bi-lateral, affecting the bases of both lungs; and it is not accompanied by fever.

Pulmonary Congestion.—The variety of pulmonary congestion most closely simulating lobar pneumonia is the acute active congestion described as Woillez's disease, a form of active congestion of sudden onset, which really constitutes a larval type of pneumonia. Hypostatic congestion is bi-lateral in its manifestations, with moist rales at the bases of the lungs; but no fever, or sputum.

Pleurisy With Effusion may closely simulate lobar pneumonia, but there are sufficient diagnostic data to differentiate the two diseases.

In pleurisy with effusion the onset is gradual with chilly sensations rather abrupt with a distinct chill as in lobar pneumonia. Pleurisy in the early stage is almost always accompanied by a pleural friction sound, which disappears as the effusion accumulates in the pleural cavity. Pleuritic fever resolves by lysis, while the fever of pneumonia terminates by crisis. There is often a tuberculous history obtainable in connection with pleurisy. In pleurisy with effusion the vocal fremitus is abolished, instead of exaggerated as in pneumonia. There is flatness on percussion, whereas in pneumonia the note is dull, being preceded and followed by a rather tympanitic note during the stages of incomplete hepatization. Instead of the crepitant and sub-crepitant rales of pneumonia, in pleurisy with effusion there is a total absence of the respiratory sounds, or at the least a very great decrease in their intensity. Aspiration of the thorax reveals fluid in the case of pleurisy with effusion.

Herpes of the lips is rare in pleurisy, common in pneumonia. Egophony may be elicited below the clavicle and scapular angle in pleurisy with effusion, and the cardiac apex beat is displaced to the side opposite to the effusion. The intercostal spaces are more apt to be obliterated or to bulge in pleurisy with effusion than with pneumonia. Finally, visceral displacement is more pronounced in pleurisy than in pneumonia.

BRONCHO-PNEUMONIA

Pathology.—Broncho-pneumonia, catarrhal, or lobular pneumonia, is an acute inflammation of the terminal bronchioles, spreading secondarily to the adjacent alveoli, which become filled with inflammatory exudate from the terminal bronchioles.

Broncho-pneumonia is nearly always a secondary disease, following many of the infectious fevers, as pertussis, measles, influ-

enza, diphtheria, scarlatina, smallpox, or typhoid fever, diseases which during their course have been associated with a greater or less degree of bronchitis. In these instances the development of a broncho-pneumonia merely represents a downward extension of the acute bronchitis to the finer bronchioles. Broncho-pneumonia also follows the aspiration into the bronchi of particles of food, or secretions or blood from the upper respiratory passages, (aspiration or deglutition pneumonia). In apoplexy and other comatose states these particles are prone to be aspirated into the bronchi and set up a broncho-pneumonia. Aspiration pneumonia occurring in the newly born is due to the aspiration of secretions from the birth canal during labor.

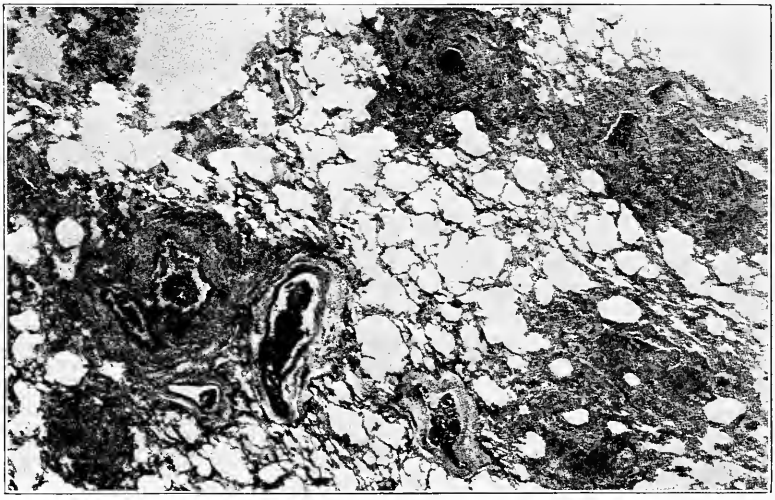


Fig. 73.—Consolidation of broncho-pneumonia. (From Delafield and Prudden.)

Broncho-pneumonia is very common in infancy, attacking many children under one year of age. In this class of patients the presence of rickets or severe diarrhea predisposes to the development of the disease.

A primary broncho-pneumonia occasionally develops in adults and children who are below par, in which the onset of the disease is abrupt, simulating lobar pneumonia.

The organisms most frequently responsible for broncho-pneumonia are Friedlander's bacillus, and the pneumococcus of Fraenkel. These organisms may occur alone or in association with the streptococcus, influenza bacillus, colon bacillus, or staphylococcus.

In broncho-pneumonia the lung presents upon its surface scattered patches of consolidation, separated by areas of compensatory emphysema. The consolidated patches are red or grayish, and slightly elevated above the surrounding surface of the lung. The disease is bi-lateral, affecting both lungs, which remain crepitant despite the multiple areas of consolidation, and the lung will float when placed in water.

When the lung is sectioned the pneumonic patches of consolidation are observed to be situated in and confined to the peripheral portions of the lung, just subjacent to the pleura.

The terminal bronchioles are filled with muco-purulent exudate; while the peri-bronchial tissues are consolidated in the immediate vicinity of the bronchus, and splenized for some distance beyond the area of consolidation. Microscopically the bronchial walls present swelling and desquamation of the lining cells, while the bronchial lumen is filled with muco-purulent exudate, composed mainly of desquamated bronchiolar cells and with very few erythrocytes or leukocytes. The bronchial walls and inter-alveolar septa in the consolidated areas show infiltration with leukocytes.

The distribution of the disease varies. As the name implies, the disease has a tendency to involve lobules rather than lobes of the lung. As a rule, this principle holds good in the distribution of the lesions, which usually assume one of two types. Thus in the disseminated form of broncho-pneumonia lobules are consolidated all over the two lungs, the areas of consolidation being separated by areas of crepitant lung. In the pseudo-lobar form, however, a number of lobules in one lobe are consolidated, perhaps the greater portion of a lobe of the lung.

Broncho-pneumonia may terminate in resolution, in abscess, in fibrosis, or in gangrene. In the cases which terminate by resolution the cellular exudate in the bronchioles and alveoli becomes liquefied, largely as a result of fatty degeneration, and is borne away by the lymphatics or is expectorated.

The pleura is frequently involved in broncho-pneumonia, but not to the same extent as in lobar pneumonia; but when pleurisy develops with broncho-pneumonia it is very apt to be purulent.

The expectoration in broncho-pneumonia is tenacious and glairy, containing no characteristic elements.

Physical Signs.—*Inspection.*—Broncho-pneumonia is attended by dyspnea, the accessory muscles of respiration coming into play with retraction of the lower interspaces and epigastrium

during inspiration. In children there are not infrequently cyanosis and suffocative attacks. There is cough, which with the dyspnea and cyanosis developing in a child convalescing from an acute infectious disease should direct the examiner's attention to the lungs. In many instances in children the disease is ushered in with convulsions.

Palpation may reveal increase in the vocal fremitus if there is a patch of consolidation of sufficient size and favorably located with reference to a bronchus. However, the intervening emphysematous portions of the lung tend to mask the fremitus, which may be actually diminished. Similarly, occlusion of a bronchus by secretion may abolish the fremitus. In the pseudo-lobar form of the disease there is always increase of vocal fremitus, whereas in the disseminated form such an increase is not to be expected.

Percussion may reveal multiple patches of impaired resonance or dullness, particularly over the bases posteriorly. However, the thorax of the young child, in whom the disease is very frequently encountered, is naturally rather resonant; and the areas of compensatory emphysema surrounding and separating the areas of consolidation tend to mask the dullness which would be evidenced by these areas and to impart a vesiculo-tympanitic quality to the percussion note. In the disseminated form of broncho-pneumonia dullness is not to be expected, as the areas of emphysema mask the dullness which might be caused by the areas of consolidation. In the pseudo-lobar form, on the contrary, there is frequently dullness at the bases posteriorly, which may be elicited by careful daily percussion of these areas. In demonstrating the areas of dullness it is necessary to employ very light percussion.

Percussion of the anterior regions of the thorax in these cases will yield hyper-resonance due to compensatory emphysema of these portions of the lungs.

Given any case of broncho-pneumonia, during the first twenty-four hours of the disease percussion will reveal no alteration in the percussion note; but the bases posteriorly should be percussed daily in a suspected case; and in most instances by the end of forty-eight hours dullness will have developed in these regions.

Auscultation.—Auscultation reveals upon consecutive examinations the downward extension of the original bronchitis. In addition to the rales arising in the bronchial tubes incident to the bronchitis, the crepitant rale is heard upon the completion of in-

spiration over the bases posteriorly, indicating the participation of the alveoli in the inflammatory process. At times the rales may be masked by plugging of the bronchi by exudate; but coughing usually serves to bring them again to the fore.

As the patches of consolidation form the respiratory sounds assume a broncho-vesicular character, but never become purely bronchial.

The pulmonic second sound is accentuated during the height of the disease; and if the right heart fails, edema of the lung supervenes, with the characteristic signs of that condition.

In the presence of considerable consolidation of the bases, bronchophony may sometimes be elicited just above the level of the consolidation. Over the anterior surface of the thorax, in the region of compensatory emphysema, the expiratory murmur is slightly prolonged, and accompanied by sibilant piping rales.

Diagnosis.—The physical signs of broncho-pneumonia, while bi-lateral, are seldom present to the same degree upon both sides of the thorax, one lung usually being involved to a greater extent than is the other. Whenever physical signs of consolidation are elicited over one lung, a careful search for similar signs should be made over the opposite lung.

Very often a diagnosis of broncho-pneumonia cannot be made upon the physical signs alone, as these are often misleading, and as the mode of onset of the disease is not infrequently atypical. The physical findings should be considered in conjunction with the fever, cough, dyspnea, and pain arising during the course of an acute infectious disease, or engrafted upon a previous acute bronchitis.

Occurring as the disease does during the course of or convalescence from some other disease, the picture is often modified for a time at least by the characters of the primary affection. But a history of fever, dyspnea, cough, and pain in the chest, arising during the course of an acute infection or engrafted upon a previous bronchitis is suggestive of broncho-pneumonia.

From *acute bronchitis* the disease is differentiated by the absence of areas of hyper-resonance and impaired resonance in the former, and by the finer quality of the rales in broncho-pneumonia. Moreover, with acute bronchitis there is less fever, and the disease is altogether milder in its manifestations.

From *labor pneumonia,* broncho-pneumonia presents many points of differentiation. Broncho-pneumonia is usually secondary to another disease, and of insidious onset; whereas lobar pneumonia is

usually a primary disease, with abrupt and stormy onset. Moreover, broncho-pneumonia is a bi-lateral pulmonary disease, producing many areas of impaired resonance over both lungs, rather than a single area of flatness over one lobe of the lung. Also, broncho-pneumonia terminates, or resolves by lysis, whereas lobar pneumonia terminates by crisis usually between the 7th and 9th days. As a rule broncho-pneumonia attacks subjects of impaired vigor who are below par, whereas lobar pneumonia usually affects robust persons, in the prime of health.

In the primary form of broncho-pneumonia, particularly if the disease assumes the pseudo-lobar form, in which the pneumonic patches fuse and involve the greater portion of a lobe, the differentiation from lobar pneumonia is attended with considerable difficulty. In this class of cases the physical signs are very similar if not identical; but in broncho-pneumonia there is nearly always apparent, if even to a slight degree, some involvement of the opposite lung.

In lobar pneumonia the fever is higher and the prostration more profound than in broncho-pneumonia; and the sputum is viscid and rusty, whereas in broncho-pneumonia it is tenaceous and glairy, often muco-purulent.

From the broncho-pneumonic form of acute tuberculo-pneumonic phthisis, broncho-pneumonia cannot be differentiated in the early stages. Involvement of the apices by the disease is suggestive of phthisis; but in certain cases of tuberculo-pneumonic phthisis the apical involvement is not particularly prominent. However, in phthisis the temperature is prone to remain more uniformly high, and to be interrupted by night sweats. There is usually obtainable a history of intimate association with a tuberculous person or evidence of a tuberculous focus at some point. Moreover, in the broncho-pneumonic form of acute tuberculo-pneumonic phthisis emaciation progresses rapidly, the course of the disease being progressively downward, until eventually tubercle bacilli can be demonstrated in the sputum.

Broncho-pneumonia in infancy is sometimes with difficulty differentiated from meningitis, as the disease in this class of patients is often accompanied by marked cerebral symptoms, as delirium or convulsions. Time and observation are essential to a differential diagnosis under these circumstances.

CHRONIC INTERSTITIAL PNEUMONIA

Pathology.—Chronic Interstitial Pneumonia, productive pneumonia, cirrhosis or fibrosis of the lung, is a chronic condition, the result of prolonged and continued irritation involving either smaller or larger areas of the lung. The predominant pathologic feature of the disease is the formation of an excess of fibrous connective tissue, which by contraction decreases the size and air space of the lung.

Chronic interstitial pneumonia may be a sequence of either

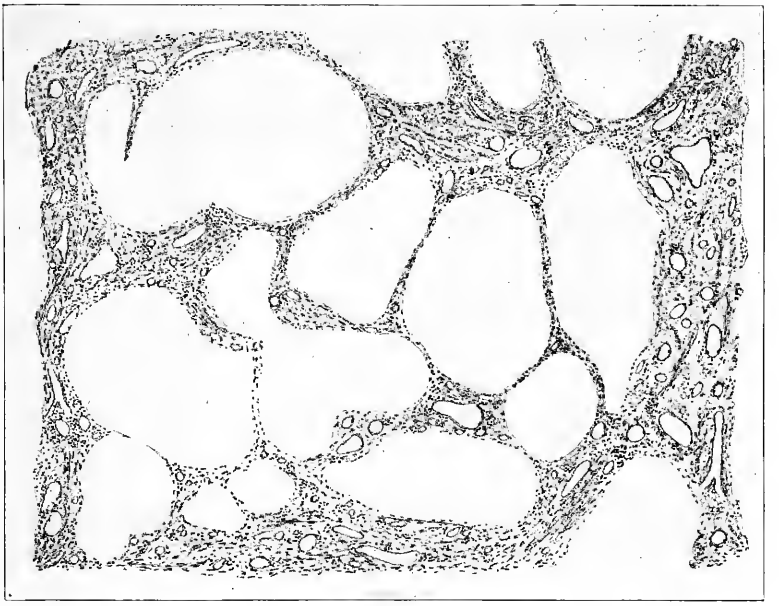


Fig. 74.—Interstitial pneumonia with emphysema. (From Delafield and Prudden.)

lobar or broncho-pneumonia, when instead of resolution occurring the disease terminates, in fibrosis or gray induration of the lung. In other instances the cirrhotic process has its point of inception in the pleura, the result of chronic pleurisy, in which event the connective tissue formation develops along the interalveolar septa. Localized areas of fibrosis of the lung accompany pulmonary tuberculosis, result from the continued inhalation of irritating dusts or vapors, and develop as a result of pulmonary syphilis.

Two forms of chronic interstitial pneumonia have been de-

scribed; namely, a massive or lobar form, and an insular or broncho-pneumonic form.

In the massive form of the disease one lung is involved. This lung is shrunken, the alveolar walls being much thickened and the alveolar spaces much smaller than is normal; the lung is drawn up into the pleural cavity, occupying a very small area near the spinal column. In cases resulting from pneumonia the pleura shows little change; but in cases of pleurogenous origin this membrane shows great thickening. The bronchial tubes show numerous dilatations, surrounded by masses of indurated lung tissue, the dilatations having resulted from the traction of peri-bronchial adhesions.

In the insular or broncho-pneumonic form of the disease the areas of fibrosis are small and are generally distributed throughout the base and lower lobe of the lung.

In both forms of the disease the opposite lung is found in a state of compensatory emphysema.

Physical Signs.—*Inspection.*—The affected side of the chest is deficient in expansion, immobile or moving but little with the respirations, retracted, with drawing downward of the shoulder. The cardiac impulse is displaced toward the affected side, and there is not uncommonly a very wide area of impulse in the 3rd and 4th interspaces. Owing to the retraction of the diseased side the nipple and scapula approach more nearly to the median line than is normal. There is spinal curvature, with the concavity toward the side of the disease.

The disease is uni-lateral; hence, if the left lung be involved the cardiac impulse is displaced toward the left, giving a wide area of visible impulse. If, however, the right lung is the seat of the disease, the cardiac impulse is often not visible, as it is not infrequently displaced to the right so that it lies behind the sternum. If the cardiac displacement accompanying right sided cirrhosis is not so extreme as to displace the impulse behind the sternum, it may still be invisible owing to overlapping of the apex of the heart by the compensatorily emphysematous left lung.

Palpation.—In the majority of cases of chronic interstitial pneumonia vocal fremitus is increased, although in cases of pleurogenous origin, associated with considerable pleural thickening, it may be diminished. In these cases a pleural friction fremitus can sometimes be appreciated. Over a bronchial dilatation which approaches the wall of the axillary region vocal fremitus is markedly increased.

Percussion.—Percussion over the retracted lung yields a dull or

flat note; while percussion over the opposite lung reveals hyperresonance due to compensatory emphysema. Percussion over the axillary region not infrequently yields a tympanitic note, due to the close proximity of a bronchial dilatation. The upper limit of pulmonary resonance is diminished on the side of the disease owing to shrinking of the pulmonary apex. Similarly in involvement of the right lung the lower border of pulmonary resonance is elevated, the liver pushing the diaphragm upward; whereas in left sided disease the tympany of Traube's semilunar space extends higher than normally.

Auscultation of the affected side reveals bronchial breathing over the retracted and shrunken lung, which, over dilated bronchi or bronchiectatic cavities frequently has an amphoric or cavernous quality engrafted upon it. Over the lower portion of the thorax of the affected side the breath sounds are feeble or entirely abolished. The same is true of cases of pleurogenous origin with thickening of the pleura.

Over the pulmonary apex and in the axillary region the respiratory murmur is usually broncho-vesicular, not infrequently amphoric. Over the opposite half of the thorax the respiratory murmur is harsh or puerile from compensatory emphysema. In pleurogenous cases a friction rub can often be heard over the cirrhosed lung.

The pulmonic second sound is accentuated; and late in the course of the disease, is apt to become weakened, the tricuspid systolic "safety-valve" murmur then becoming audible, betokening imminent right heart failure.

Diagnosis.—When a patient presents the physical signs detailed in the preceding paragraphs, with a history of chronic cough and shortness of breath, with good nutrition and fairly good health, despite the fact that he has been affected thus for a considerable length of time, sometimes for several years, chronic interstitial pneumonia is suggested. If, in addition, a history is obtained of these signs and symptoms supervening upon a previous lobar or catarrhal pneumonia or pleurisy, the diagnosis is still more probable. Slowly growing tumors of the lung or pleura may rather closely simulate the physical signs of chronic interstitial pneumonia.

It is to be borne in mind that the physical signs of fibroid phthisis are largely identical with those of chronic interstitial pneumonia. In tuberculosis, however, both lungs are apt to be involved in the fibrosis, and the sputum contains tubercle bacilli.

ACUTE TUBERCULO-PNEUMONIC PHTHISIS

Pathology.—Acute tuberculo-pneumonic phthisis occurs in two forms: The pneumonic form, and the broncho-pneumonic form of the disease.

In the pneumonic form of acute tuberculo-pneumonic phthisis only one lobe or the entire lung may be involved. The disease commonly starts from a tuberculous focus in one apex. The lung in this form of the disease is heavy, the affected portion devoid of air, and the pleura covered with a thin exudate. Upon section the picture closely resembles that of lobar pneumonia during the stage of hepatization. Males are attacked with this form of the disease more frequently than females.

The broncho-pneumonic form of acute tuberculo-pneumonic phthisis most frequently attacks children. Pathologically it is a caseous broncho-pneumonia which starts in the small bronchioles, which become blocked with cheesy material, while the alveoli are filled with the products of a catarrhal pneumonia.

By the fusion of several areas of the disease process almost an entire lobe may become involved; but in most instances the areas of disease are separated by air-containing lung.

Physical Signs of the Pneumonic Form.—*Inspection.*—The disease is characterized frequently by acute suffocative attacks of dyspnea with cyanosis. The sputum is mucoid and may or may not contain tubercle bacilli.

Palpation reveals increase of vocal fremitus over the consolidated area, which is usually chiefly confined to the upper lobe of the lung. Palpation may also detect lagging inspiratory inflation of the apices.

Percussion shows dullness over the area of consolidation; but very early in the disease the percussion note is often hyper-resonant.

Auscultation.—The first or earliest auscultatory findings are a suppression of the respiratory sounds, which later become broncho-vesicular or purely bronchial. This purely tubular breathing continues for a week or ten days, when, instead of clearing and disappearing as in lobar pneumonia, signs of cavity formation, indicating softening appear.

Physical Signs of the Broncho-Pneumonic Form.—*Inspection* shows that the child is dyspneic, with cough, emaciation, and hectic flush.

Palpation reveals multiple patches of increased vocal fremitus, most marked about the apices of the lungs.

Percussion.—The percussion note is seldom dull. It is rather vesiculo-tympanitic produced by the multiple small patches of consolidation with intervening emphysematous tissues.

Auscultation.—The respiratory murmur over the areas of consolidation is of the broncho-vesicular type, not attaining the purely bronchial character. There are crepitant and sub-crepitant rales. The auscultatory signs are largely those of a very acute bronchitis.

Diagnosis.—The pneumonic form of acute tuberculo-pneumonic phthisis must be differentiated from lobar pneumonia of the croupous type. In the early stages this is difficult; but it is to be remembered that this form of phthisis is prone to appear first in the apices, while lobar pneumonia is prone to involve the base of the lung posteriorly. Moreover, at the seventh or ninth day, instead of a crisis occurring with amelioration of the most acute symptoms, the disease in the case of phthisis runs on and becomes aggravated, sweats occurring, and the sputum developing the tubercle bacillus and elastic fibers.

The broncho-pneumonic form of acute tuberculo-pneumonic phthisis must be differentiated from non-tuberculous broncho-pneumonia, a problem which requires time and observation. A broncho-pneumonia having its inception in the apices of the lungs is suggestive of tuberculosis. Also the rapid emaciation of the subject points to the same disease; while the finding of the tubercle bacillus renders the differentiation positive.

CHRONIC ULCERATIVE PHTHISIS

Pathology.—Chronic ulcerative phthisis has its inception in a tuberculous focus in one or both apices and extends progressively downward in the lung. From an apical lesion infective material is aspirated into the bronchi of uninfected portions of the lungs and here sets up tubercle formation about the finer bronchioles. Thence the disease spreads to the infundibula, and, less commonly, ascending infection occurs, leading to infection of the bronchi above the smaller bronchioles.

Extension of infection also travels by continuity of tissue from a primary focus to the immediately surrounding portions of the lung. Infection frequently travels by the lymphatics or blood stream, infecting other portions of the pulmonary tissues. Through these avenues and these means the infection of the lung

when once started travels progressively downward, involving lobule after lobule and lobe after lobe of the lung.

In the further progress of the disease different portions of the lung show tubercles in different stages of infiltration, sclerosis,

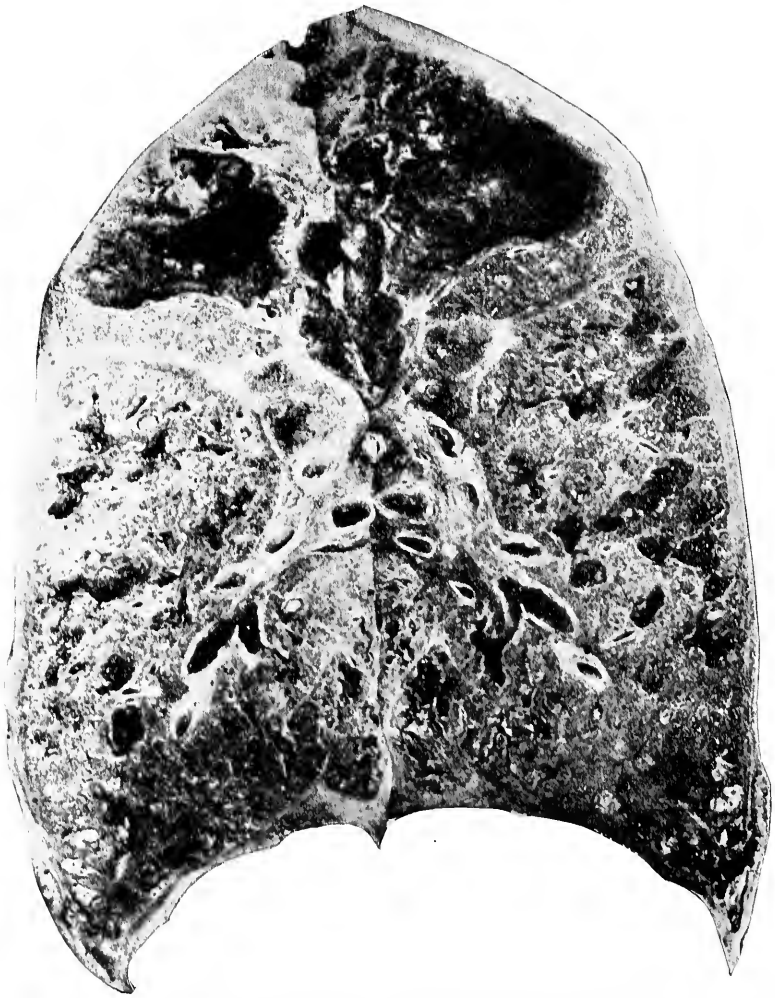


Fig. 75. Illustrating caseous tuberculosis. Large cavities at the apex and many small cavities throughout the lung. (Pottenger, after Tendeloo.)

or caseation and softening, leading eventually to cavity formation.

Ulceration of the walls of the bronchial tubes not infrequently permits stretching of these walls during paroxysms of cough or

from the weight of stagnating secretions, with the formation of dilatations or bronchiectatic cavities.

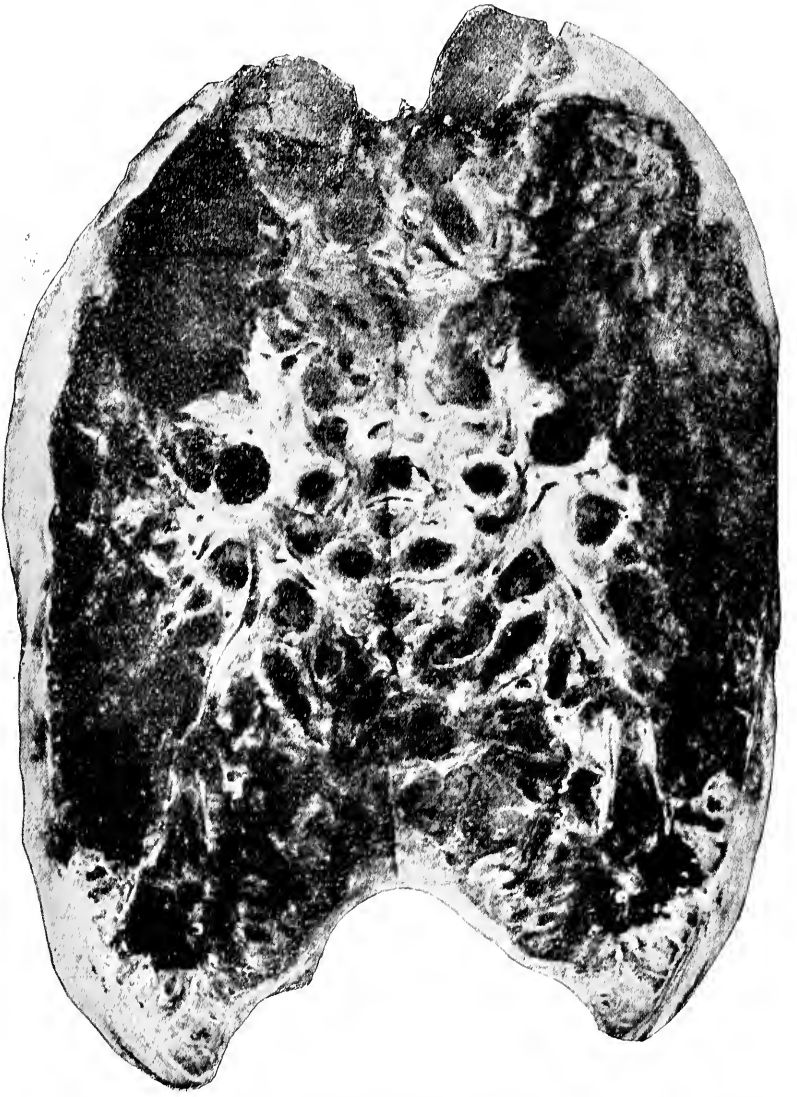


Fig. 76.—Illustrating pulmonary tuberculosis, with thickened pleura, many bronchiectatic cavities, and generalized cavity formation. (Pottenger, after Tendeloo.)

Aside from the bronchiectatic cavites, cavities may be formed in the pulmonary tissues apart from the bronchi. The walls on these cavities in some instances are smooth, while in other cases they are uneven and rugged. In these cavities blood vessels which

have not been destroyed may be found traversing the cavity; and by rupture may produce copious hemorrhage, which may prove fatal.

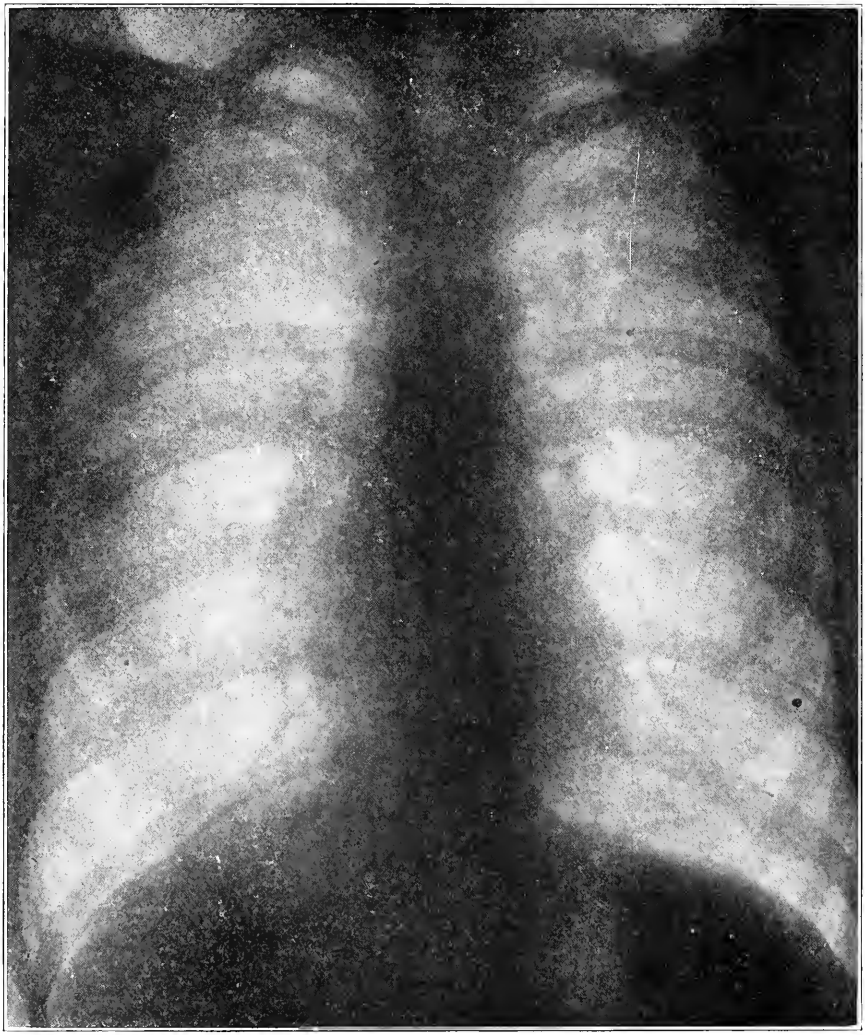


Fig. 77.—Roentgenogram. The special features of this picture are the prominent bronchi, showing induration; the diffuse shadows throughout the lungs, indicating tuberculosis; small tent-like raised areas in the diaphragm, indicating pleural adhesions and the large right heart. (From Brown.)

Cavities of moderate size by coalescence often lead to the formation of extensive excavations which, in exceptional instances may embrace the greater portion of a lobe.

When the peripheral portions of the lung are involved, a cavity may form just subjacent to the pleura, and by rupture through that membrane produce pneumothorax. In other instances, instead of rupturing with the formation of a fistulous communication between the lung and pleural cavity, adhesions may form

Fig. 78.—Lung. Chronic phthisis, showing a large irregular cavity in the upper lobe. In the lower lobe there are scattered acute nodules grouped in clusters around the small bronchi; and also several small more acute cavities. The bronchial glands are enlarged and caseous. (Edinburgh University Anatomical Museum.) (Woolley after Beattie and Dickson.)

between the visceral and parietal pleura, as the result of localized pleurisy overlying the tuberculous lesion in the lung. These pleural adhesions are frequently quite extensive, to a great extent serving to immobilize the lung.

Instead of undergoing caseation and softening with consequent cavity formation, tuberculous foci in the lung may undergo a process of sclerosis. Sclerosis is a reparative process, tending to inhibit the spread of infection; but it is rare for sclerosis to occur to an extent sufficient to save a tuberculous lung. Lime salts may be deposited in sclerotic and caseous foci and limit the spread of the infection temporarily; but an attack of a bronchial affection such as influenza is prone to "light up" these dormant or residual foci of infection.

The bronchial glands do not escape the tuberculous infection. Infiltration, caseation, abscess formation and rupture of the glands frequently are seen.

Physical Signs.—*Inspection.*—Chronic ulcerative phthisis produces a characteristic deformity of the thorax in its later stages, the phthisical or paralytic thorax. It may be noted that the degree of expansion of the chest upon the diseased side is deficient, this deficiency being first noted in the infra-clavicular region. If the left lung is diseased, the cardiac impulse shows a wide impulse in the second, third, and fourth interspaces. There is abolition of Litten's phenomenon on the diseased side. In not a few instances the pupil corresponding to the side of the disease is found dilated, probably owing to pressure upon the cervical sympathetic fibers. (See Fig. 26, p. 45.)

The subject of chronic ulcerative phthisis is dyspneic, which may be caused by the rapid extension of the disease with involvement of both lungs, to associated emphysema, or to right heart failure. Emaciation is a marked feature of chronic ulcerative phthisis, the muscles of the shoulder girdle wasting rapidly. (See Fig. 47, p. 72.)

Localized retractions of the chest wall are often noted, indicating areas of pulmonary collapse or the traction of pleural adhesions overlying tuberculous lesions of the lung. The finger-tips are often clubbed (Hippocratic fingers).

Many cases of chronic ulcerative phthisis, even in their incipiency will exhibit an abnormal flattening and mobility of the sternal angle (Rothschild's Sign); while in other cases there is early ankylosis and rigidity of the spinal column in the dorsal region (Lorenz's Sign).

Uni-lateral deficiency of expansion is noted first as a lagging

inflation of one or both apices, later affecting the infra-clavicular and mammary regions, the uni-lateral deficiency being compensated for by vicarious expansion of the undiseased lung.

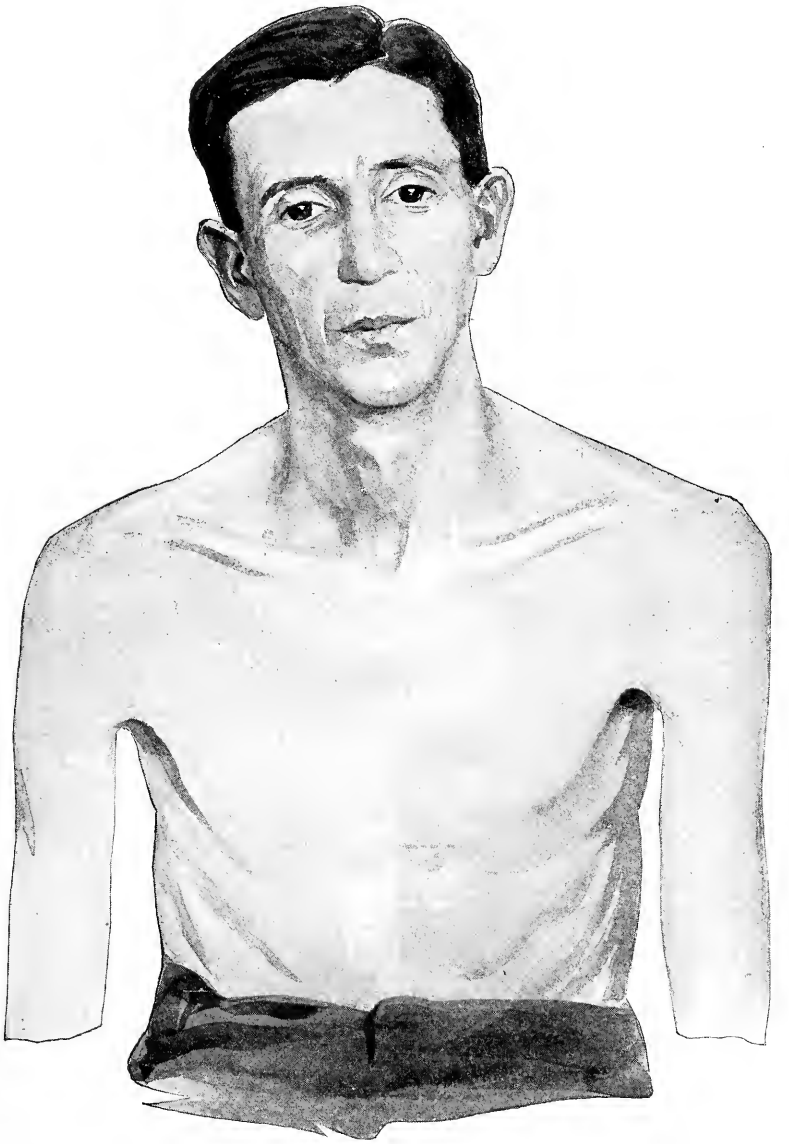


Fig. 79A.—Illustrating marked regional degeneration of the muscles and other soft tissues over the anterior surface of the chest as a result of chronic tuberculosis. The lesion is older and more extensive on the right side. The degeneration of the soft tissues on the right is particularly marked. The lowering of the angle of the trapezius is well shown both anteriorly and posteriorly; so is the degeneration of the right sterno-cleido-mastoideus as compared with the left. (From Pottenger.)

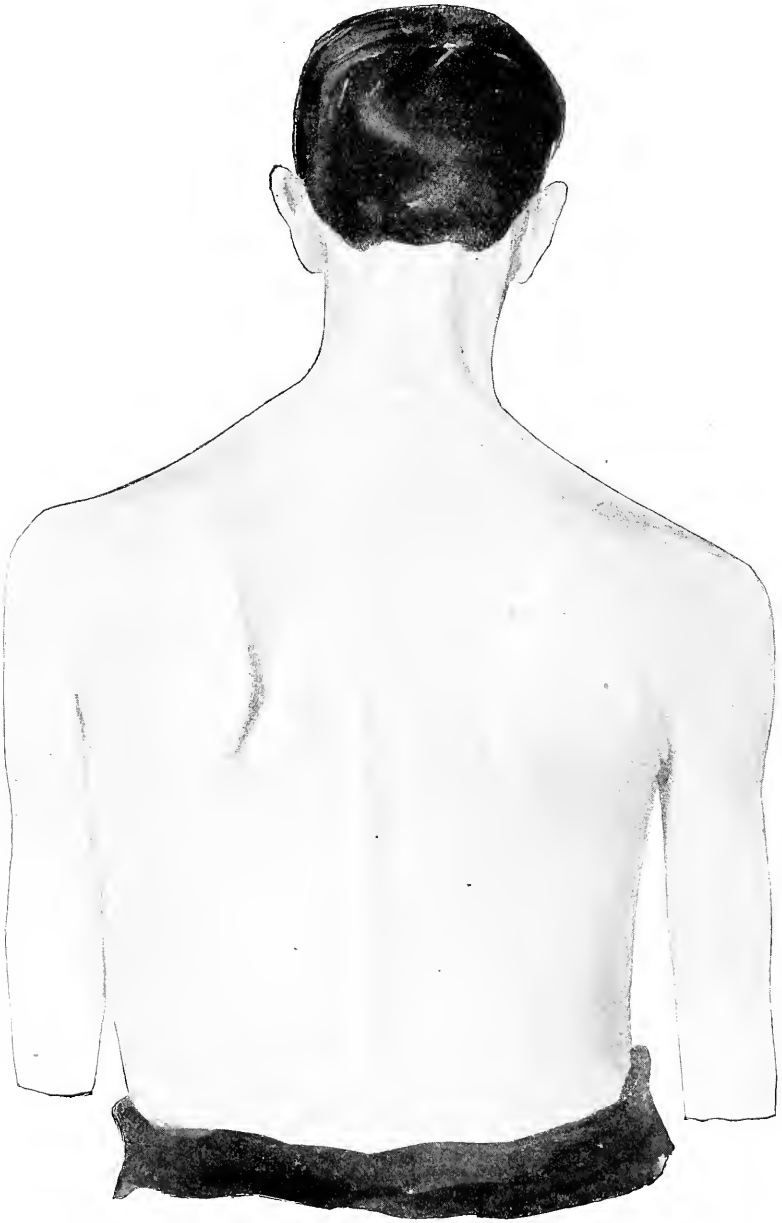


Fig. 79B.—Same as Fig. 79A, showing posterior view. The right trapezius and other soft tissues are wasted more than those on the left, and permit the shoulder to drop markedly. (From Pottenger.)

The sputum of chronic ulcerative phthisis is characteristic. Scanty and almost purely mucoid in the early stages of the disease, later the sputum becomes abundant, muco-purulent or purulent, containing leukocytes, epithelum, elastic fibers, the tubercle bacillus and various other bacteria. Small yellowish caseous

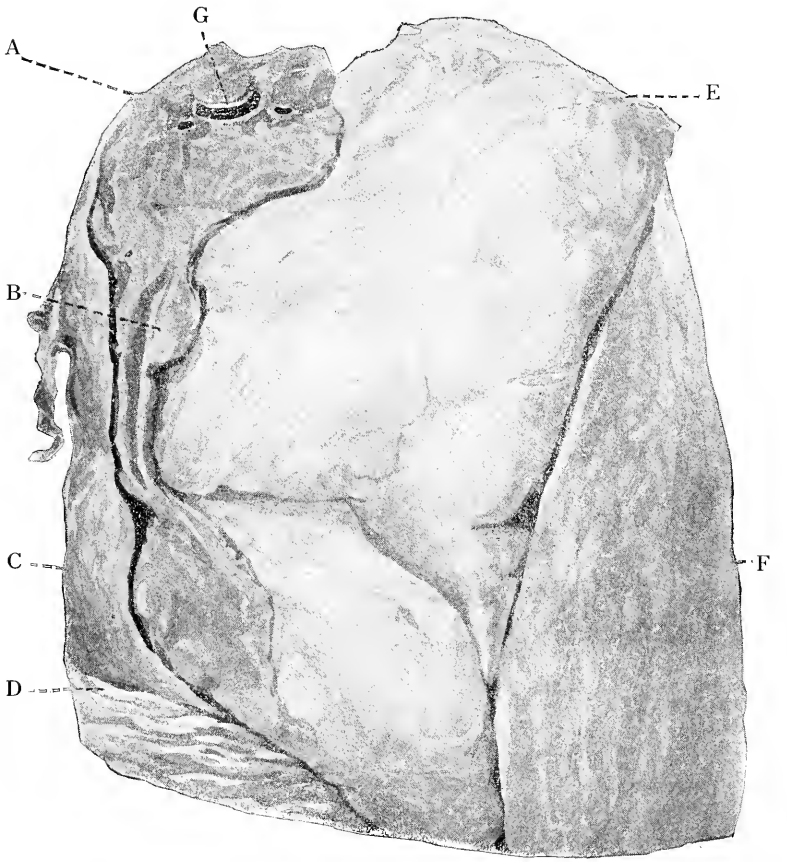


Fig. 80.—Illustrating the distortion of the thoracic viscera in a patient with a marked destructive lesion in the right lung, and marked compensatory changes in the left. *A,* the upper lobe on the right, is a small fibroid mass; *B,* the middle lobe, is only a fibrous string; *C,* the lower lobe, barely presents anteriorly, but posteriorly was the seat of emphysema; *D,* three-fourths of the heart lies to the right of the median line; *E,* the upper lobe on the left represents a large portion of the lung which presents anteriorly. A new lobe has been formed, pushing through the anterior mediastinum to a distance of three inches beyond the median line; *F,* the lower lobe is also markedly emphysematous; *G,* trachea. (From Pottenger.)

masses are frequently found in the sputum, which are most intimately associated with the tubercle bacillus. Occasionally the sputum is blood tinged or contains free blood. Hemoptysis is a valuable sign of the disease.

Palpation confirms the findings of inspection as to deficiencies of expansion of the thorax and may bring to light slight deficiencies which have escaped detection during visual examination. Palpation is particularly to be recommended in the detection of slight deficiencies of expansion at the apices in incipient cases. To detect lagging inspiratory filling of the apices the examiner should stand behind the patient and palpate the apices with the thumbs in the supra-clavicular and the fingers in the infra-clavicular fossae. (See Fig. 37, p. 57.)

Over densely infiltrated or consolidated areas of the lung vocal fremitus is exaggerated. It is particularly exaggerated over superficial cavities with bronchial communication. Great pleural thickening or exudate, or pleural effusion coexisting with the disease may obscure the fremitus or entirely abolish it. More rarely there may be pleural friction fremitus and rhonchal fremitus.

In view of the fact that vocal fremitus is normally more intense over the right apex than it is over the left, an equalization of the degree of vibration over both apices would point to consolidation of the apex of the left lung.

The *pulse* is usually moderately increased in rate, but not out of proportion to the fever.

Percussion.—In eliciting slight degrees of dullness in incipient cases the examiner should direct the patient to inspire to the full capacity and then to suspend respiration, while he carefully percusses the apices of the lungs. Dullness in this locality is indicative of apical consolidation. In eliciting dullness in the apices it is well also to employ immediate percussion by tapping the clavicle upon each side and noting any discrepancy in the notes elicited. Also in examining for dullness over the bases posteriorly immediate percussion is useful, the examiner slapping the sides of the chest alternately with the palm of the hand.

Percussion over a tuberculous cavity elicits a tympanitic or cracked-pot sound; and the signs of Wintrich, Friedreich, and Gerhardt can usually be elicited. (See Fig. 65, p. 86.)

Upon percussion of the pectoral muscles a sudden contraction of the muscle under examination may be noticed (Myoidema), which merely indicates that atrophy is progressing rapidly and is not pathognomonic of pulmonary tuberculosis.

In cases of suspected chronic ulcerative phthisis the interscapular regions around the level of the 5th dorsal spine should be percussed, and the quality of the note elicited on the two sides compared.

Percussion of a lung the seat of chronic ulcerative phthisis may yield tympany in the absence of cavity formation, provided that an area of firm consolidation is immediately under the percussion blow and reaches down to a large bronchus. In such a case the tympany of the bronchus is transmitted readily to the surface of the chest.

Auscultation.—Auscultation of the lungs usually affords the earliest information as to the presence of chronic ulcerative phthisis. In the very early stages of the disease the respiratory murmur is feeble, and there is recognized the jerky respiration or cog-wheel breathing, in which during inspiration the murmur is interrupted at several points, producing a sound somewhat similar to that of a sobbing child. Rarely in the early course of the disease a pleural friction sound is audible. (See Fig. 67, p. 92.)

One of the earliest signs of the disease is the crepitant rale. As the consolidation increases the breath sounds become bronchovesicular and finally bronchial, associated with increase of vocal resonance, bronchophony or pectoriloquy. A pulmonary cavity frequently is indicated by the development of whispering pectoriloquy, or cavernous or amphoric breathing. A cavity filled with secretion, or one whose bronchial communication is plugged gives forth no physical signs. But when such a cavity is only partially filled, there are moist and gurgling rales, with rarely the metallic tinkle or succussion sound, if the cavity be of sufficient size and favorably situated.

The presence of the lung-fistula sound indicates that pneumothorax has been produced by rupture of a sub-pleural cavity.

The mucous click, a sharp clicking sub-crepitant rale, may be heard in certain advanced cases, and is said to indicate rapid softening.

Diagnosis.—Chronic ulcerative phthisis in its evolution and course produces a multiplicity of physical signs; but in the diagnosis of the disease no sign, however slight or insignificant it may appear in itself, should be overlooked. The greatest difficulty in reaching a diagnosis arises in the incipient cases; and it is in just these cases, where the chance of recovery from the disease has not absolutely passed, that it is most important to diagnose the disease.

Signs and symptoms pointing to the presence of chronic ulcerative phthisis are emaciation, anemia marked in degree, fever, a symptom which is always present; night sweats, cough, pain in the chest, dyspnea and hemorrhage. Add to this the characteris-

tically deformed chest, the hectic flush of a well established case and the diagnosis is very probable. A history of the disease in the parents, or the discovery of a tuberculous focus somewhere in the body are very suggestive. The diagnosis is confirmed by the finding of the tubercle bacillus in the sputum. In young patients the tuberculin reaction may be of value.

In interpreting the physical signs especial emphasis should be laid upon areas of deficient expansion, dullness at the apices, harsh and prolonged expiration, and rales which persist and are repeatedly audible in the same area.

Malaria may be simulated by the fever of chronic ulcerative phthisis, with chills and sweats; but the blood is negative for the malarial plasmodium, and the sputum shows usually the tubercle bacillus; and there is usually a family history of the disease in some member of the family.

Consolidations of the lung due to lobar or catarrhal pneumonia are differentiated from that of tuberculosis by the absence of sputum containing tubercle bacilli, and the clinical course of these diseases.

Bronchiectatic cavities, pulmonary abscess, or *gangrene,* while they may give rise to physical signs suggestive of tuberculosis, are differentiated by the absence of the tubercle bacillus in the sputum.

FIBROID PHTHISIS

Pathology.—Fibroid phthisis is a chronic form of pulmonary tuberculosis, in the evolution of which the predominant feature is the formation of an excessve amount of fibrous connective tissue. The disease may arise in one of two ways. Thus, the tuberculous infection may be primary, and through the process of reparative sclerosis may assume the fibroid type. On the other hand, the tuberculous process may be engrafted upon a lung previously fibrosed, either by pneumonokoniosis or chronic interstitial pneumonia following upon a fibrinous pneumonia which has healed by sclerosis or a pleurogenous interstitial pneumonia.

In the cases which are primarily tuberculous, the disease starts in an apex of one or both lungs; and, as in other forms of the disease, progresses downward, involving the lower lobes successively. In this class of cases one apex may be sclerosed or the entire lung may be sclerotic and shrunken, showing cavities and bronchial dilatations either open or filled with caseous material.

In many instances the only way to determine whether a pul-

monary fibrosis is tuberculous or non-tuberculous, is by the presence or absence of tubercle bacilli in the sputum; but in other cases there are areas of tuberculosis demonstrable in various areas of the cirrhotic lung at autopsy.

The distribution of the fibrosis is largely influenced by the manner of inception of the disease. Thus, in cases arising from inhalation through the bronchi the formation of connective tissue is

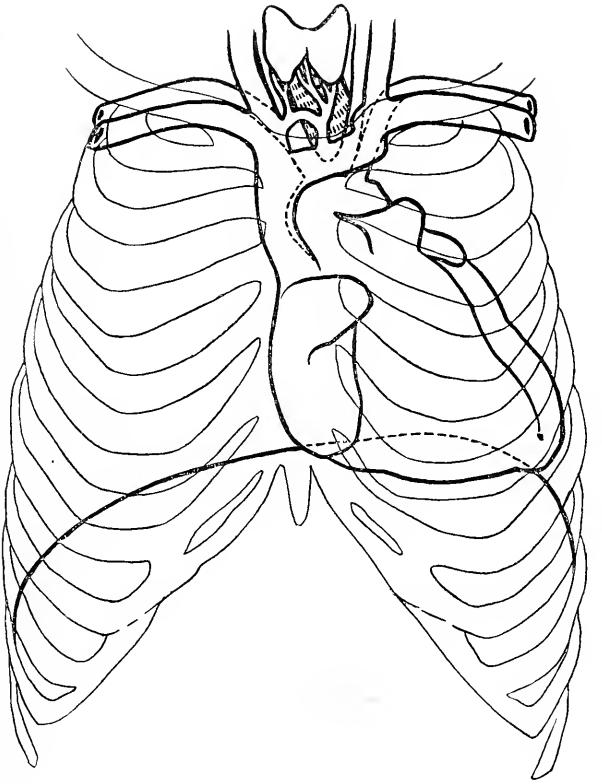


Fig. 81.—Illustrating schematically the displacement of the heart to the left. It will be seen that the left half of the diaphram is pushed upward, and the apex of the heart follows the fifth interspace. This displacement has a tendency to reduce the curve in the arch of the aorta. The trachea may be drawn entirely to the left of the median line as shown in the cut. (From Pottenger.)

most pronounced around the bronchi and bronchioles; whereas in the cases arising as a result of tuberculous pleurisy the peripheral portions of the lungs are most extensively involved.

The lung is frequently pigmented, is shrunken, and occupies a very small portion of the pleural cavity near the spinal column.

Physical Signs.—*Inspection.*—The signs of this disease are al-

most identical with those of chronic interstitial pneumonia. The affected side is shrunken, the shoulder droops, there is deficient expansion, and areas of local retraction of the chest wall are common. The cardiac impulse is displaced toward the side of the disease. In left-sided disease there is apt to be a wide visible impulse in the third, fourth, and fifth interspaces; while in disease of the right lung the impulse may be displaced behind the sternum. There is

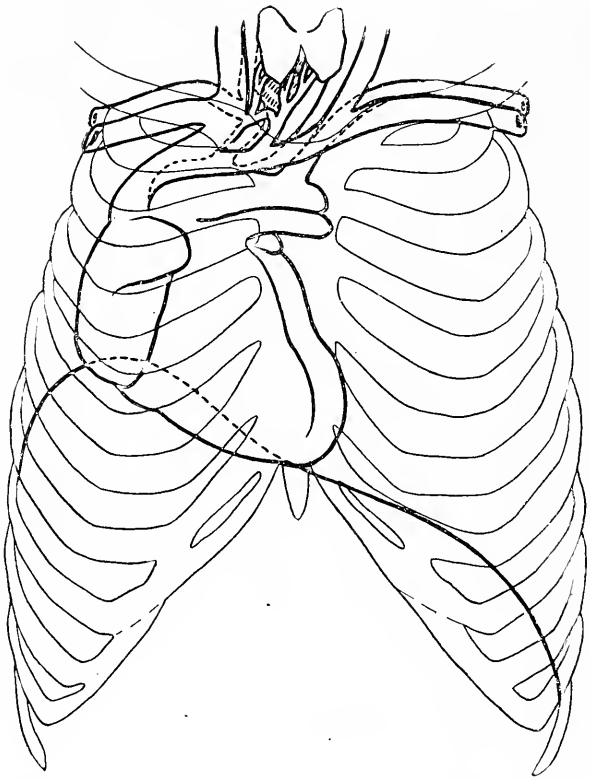
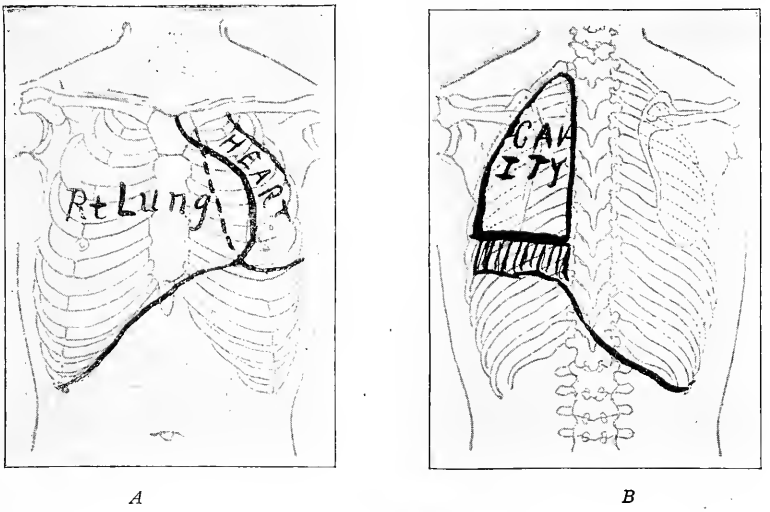


Fig. 82.—Illustrating schematically marked displacement of the heart to the right. It will be noticed that the heart is pushed upward and over. The right side of the diaphragm assumes a high position; while the left side assumes a low one in order to accommodate the left lung which is the seat of compensatory emphysema. When this displacement is present, the curve in the arch of the aorta is lessened with a tendency to pouching. (From Pottenger.)

frequently scoliosis, with the convexity of the curve toward the side of the disease. The intercostal spaces are narrowed, the ribs sometimes overlapping. The fossæ in the supra- and infra-clavicular regions are abnormally deep while the clavicles stand out prominently. The expansion of the sound side of the chest in uni-lateral disease is increased as a result of compensatory emphysema.

Palpation sometimes reveals slight degrees of deficient expansion which have escaped observation during inspection. The vocal fremitus varies in intensity with the condition of the lung and pleura, palpation over cirrhotic lung tissue adjacent to the chest wall and over cavities giving increased vocal fremitus; while pleural thickening or retraction of the lung yields a fremitus of diminished intensity. Similarly, if the fibrosis is centrally situated and overlaid by normal pulmonary tissue, the fremitus is diminished, or at least is not exaggerated. Over the sound side, in uni-lateral disease, the fremitus is diminished and the expansion increased.

Percussion.—Percussion over the apices usually gives a dull or flat note over the diseased side. A cavity in the apex is indicated by



A *B*

Fig. 83.—Showing schematically the compensation which has taken place between the two sides of the chest, and between the thoracic and abdominal cavities. *A*, anterior view; *B*, posterior view. (From Pottenger.)

hyper-resonance or tympany. A greatly thickened pleura or overlapping of the ribs gives a flat note accompanied by a marked sense of increased resistance. A small area of hyper-resonance or tympany at the base of the lung is indicative of a cavity or bronchial dilatation. Percussion of the sound lung, in uni-lateral cases, yields a hyper-resonant note.

Auscultation.—Over the apex there is bronchial, occasionally amphoric or cavernous breathing. At the bases there is often distinct bronchial breathing, provided the lung is not retracted up into the upper portion of the pleural cavity, in which event the breath

sounds are absent or feeble. Amphoric or cavernous breathing arising in any region of the thorax are indicative of pulmonary cavity or bronchial dilatation. The rales of chronic bronchitis are often encountered. The pulmonic second sound is accentuated, and in late cases is enfeebled, with the presence of the tricuspid relative systolic murmur.

Diagnosis.—The deformity of the chest, the wide area of the cardiac impulse, the bronchial and amphoric breath sounds over an apex with dullness or flatness, indicate fibrosis of the lung with cavity formation. Whether or not this is tuberculous is determined by the examination of the sputum for the tubercle bacillus. But in fibroid phthisis, in contra-distinction to fibrosis from other causes, the disease is apt to be bi-lateral, whereas in other instances it is apt to be uni-lateral. Hence careful examination of both lungs should be made for the purpose of detecting signs of disease in an apparently sound lung.

PULMONARY SYPHILIS

Pathology.—Syphilis attacks the lungs in two forms: as hereditary syphilis, and as acquired syphilis of the lung.

Hereditary syphilis of the lung was first described by Virchow as pneumonia alba. The lung in hereditary syphilis is enlarged, showing on its external surface indentations corresponding with the ribs with which it is in contact. The lung is white or slightly tinged with yellow, is firm, and upon section the cut surface resembles macroscopically a section of pancreatic tissue, a condition to which Lorain and Robin have applied the term "pancreatization."

Microscopically the inter-alveolar septa show an overgrowth of fibrous connective tissue, leading to thickening of the alveolar walls, the alveolar spaces being smaller than normal and densely filled with desquamated epithelium, cellular detritus, and fat. Hoffman called attention to a thickening of the vascular walls in the inter-alveolar septa, analogous to that which occurs in syphilitic fetal tissue elsewhere, the vessels often presenting evidences of hyaline degeneration.

The lesions of acquired syphilis of the lung may assume three types, one or all of which may be present in a given case; namely, gummata, interstitial sclerosis analogous to chronic interstitial pneumonia from other causes, and syphilitic broncho-pneumonia.

The gummata are situated deeply near the root of the lung,

varying in size from a hazelnut to a hen's egg or larger. They are apt to soften and break into a bronchus or undergo sclerosis and by traction lead to bronchiectatic dilatations. Gummata are the rarest of syphilitic lesions of the lungs. Wagner and Henop described gummata in both the upper and lower lobes of the lung, situated chiefly toward the root, usually containing in their center a dilated bronchus with chronically inflamed mucous membrane. The lung intervening between the gummata was partially crepitant while the apices and anterior borders of the lungs were in a state of compensatory emphysema.

The interstitial sclerosis attending acquired syphilis of the lung has its inception near the root of the lung, and extends thence in various directions between the alveoli. The patches of insular

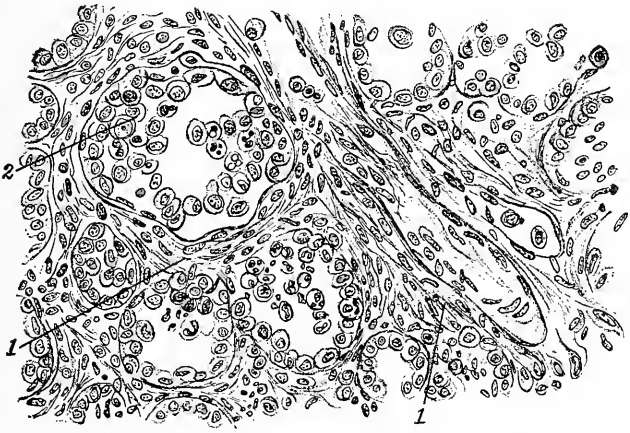


Fig. 84.—Pneumonia alba of newborn. (From McFarland.)

sclerosis often extend in all directions from gummata situated near the pulmonary root, dividing the lung into a number of divisions or lobules. Traction diverticula are formed and lead to bronchiectatic cavities.

The broncho-pneumonia of syphilis does not differ essentially from broncho-pneumonia from other causes, Pavlinoff describing a bi-lateral syphilitic broncho-pneumonia with patches of consolidation interspersed between areas of normal tissue in both lungs. The alveoli are filled with desquamated epithelium, leukocytes, and erythrocytes, the pneumonic patches being dull slate-colored on section.

Physical Signs.—Hereditary syphilis of the lung must in certain instances be differentiated from atelectasis, which it closely

simulates; but there are sufficient evidences of hereditary syphilis elsewhere upon the body as a rule to make the diagnosis clear.

The physical signs of acquired syphilis are not characteristic, the picture being often that of chronic interstitial pneumonia from other causes, chronic ulcerative phthisis, or ordinary bronchopneumonia. However, there are certain localizations of the principal signs emanating from pulmonary syphilis which are of considerable aid in diagnosis. Thus, the lesions are usually situated near the root of the lung, gummata and sclerosis in this situation giving rise to dullness on percussion and bronchial breath sounds upon auscultation along the lateral sternal borders, over the roots of the lungs, which signs decrease as the examiner passes outward, upward, and downward from these locations. Grandidier lays great emphasis upon the localization of the physical signs in these regions of the thorax, while Pankritius points out the importance of dullness in the interscapular regions at the same level.

The affection produces chronic cough, often attended by hemoptysis, and fever, simulating chronic ulcerative phthisis; but there can often be found evidences of syphilis in other regions of the body, and the specific tests for syphilis are positive.

PNEUMONOKONIOSIS

Pathology.—Pneumonokoniosis is a chronic induration of the lungs due to inhalation of various kinds of dusts and mineral particles. Depending upon the nature of the exciting cause the disease is subdivided into a number of different forms: *siderosis,* from the inhalation of iron filings; *chalicosis,* from the inhalation of stone particles; *anthracosis,* from the inhalation of coal dust. Similar pulmonary changes ensue upon the continued inhalation of fibers of wool, flax, cotton, tobacco, glass, bone, or horn.

Anthracosis, caused by the continued inhalation of coal dust, is termed "coal miner's disease." The minute amounts of this dust usually inhaled is absorbed by the leukocytes which reside upon the surfaces of the respiratory passages; are carried upward by the action of the ciliated epithelium of the tract, and expectorated. When the dusts are inhaled in larger amount, however, some of the coal particles penetrate the bronchial mucous membrane and find lodgment in the connective tissue, or enter the lymph stream and are carried to the smaller lymphatic glands around the blood vessels, the bronchi, the plura, or in the mediastinum. The lungs of all dwellers in cities are moderately pigmented from the in-

halation of coal dust and soot, while the lungs of persons who have lived all their days in the open country, remote from large manufacturing industries, are often pink in color and free from this pigmentation.

When the soot or coal dust is inhaled in very large quantities,

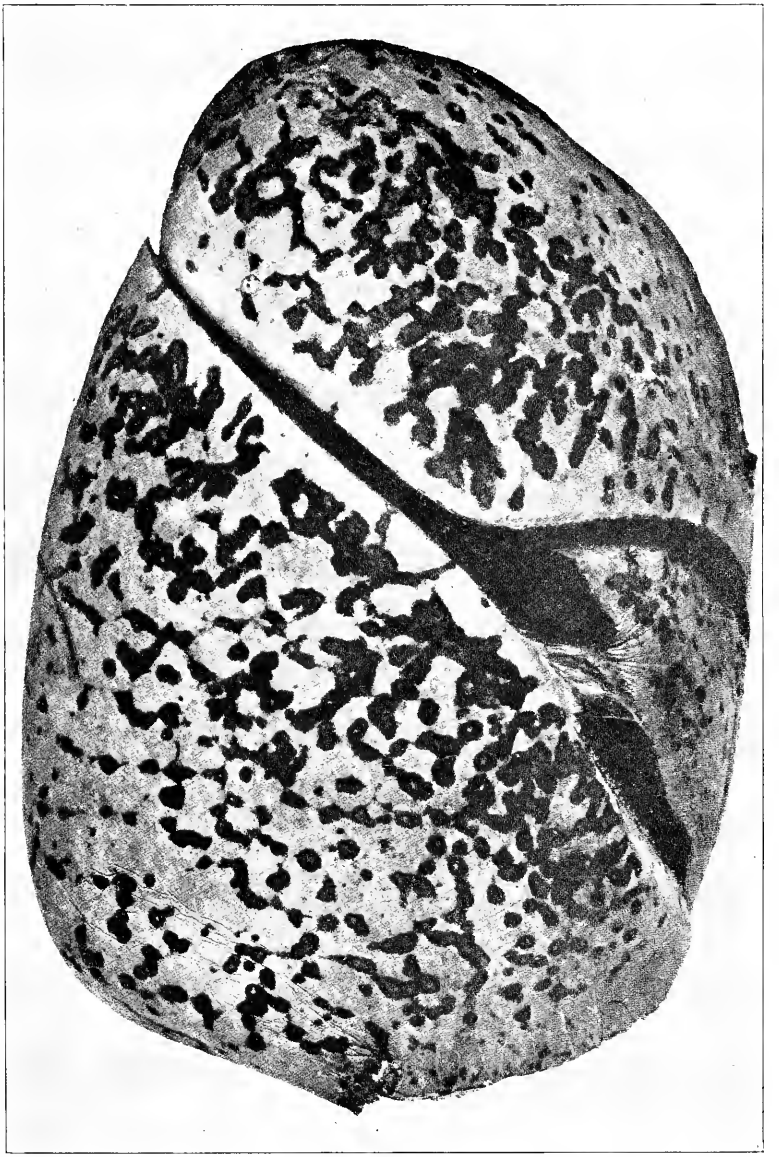


Fig. 85.—Anthracosis. (From Delafield and Prudden.)

a portion of it reaches the alveoli. In lungs the seat of this extreme grade of anthracosis the organs are distinctly black. The irritation of these grains of dust in the interstices of the tissues excites the growth of connective tissue and a connective tissue proliferation occurs leading to fibrosis in insular foci. On section these fibrosed areas are hard to the touch and exude a black fluid. Diffuse induration of an entire lung or the greater portion of a lung is common.

The bronchial and mediastinal lymph glands are indurated, and often the seat of periadenitis by virtue of which they adhere to the adjacent large vessels and by rupturing into the same distribute pigmentation to various organs of the body, as the liver, spleen, kidneys, and mesenteric lymphatic glands.

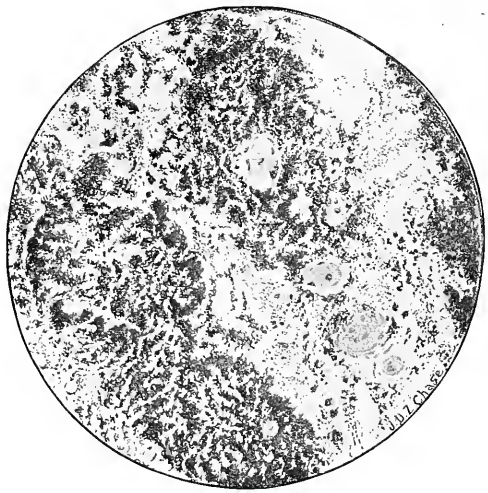


Fig. 86.—Pulmonary anthracosis. (From McFarland.)

Bronchial perilymphadenitis may lead to adhesions between the glands and the pericardium, producing a mediastinopericarditis. Adhesions may form between these glands and the esophagus, and lead to diverticulum of that tube. Adhesions may form between the glands and the aorta and cause erosion of that vessel with fatal hemorrhage. Adhesive bands may constrict the trachea or vessels and lead to tracheal or vascular stenosis or narrowing. They may lead to aspiration pneumonia when a gland erodes a bronchus and discharges its contents by that avenue. Finally, the inflamed mediastinal glands may produce vagus or recurrent laryngeal nerve paralysis by pressure upon these nerves.

Chalicosis, due to inhalation of alumina, quartz, or sandstone, is known as "stone-cutter's phthisis," "mill-stone maker's phthisis," "grinder's rot," or "potter's asthma." Chalicosis produces more induration of the lung than does any other form of pneumonokoniosis.

Siderosis is a fibrosis of the lung due to the inhalation of iron filings or dust. The changes in the lungs and mediastinal and bronchial glands are similar to those accompanying anthracosis. But the induration is more intense than it is in anthracosis.

The areas of localized induration which are formed throughout the lungs in some instances undergo softening and form pulmonary cavities. In other instances the softening is due to subsequent infection with the tubercle bacillus; though as a rule the pneumonokoniotic lung does not seem to prove a fertile field for the ravages of the tubercle bacillus. As a result of the chronic inflammation set up by the continual aspiration of the irritant dusts the bronchial tubes show a chronic bronchitis, which is followed by emphysema, while the lung slowly undergoes a slow insular sclerosis.

Physical Signs.—The physical signs of pneumonokoniosis are modified and influenced by the coincident chronic bronchitis, emphysema and interstitial sclerosis, with occasionally signs of cavity engrafted upon these signs. Signs of cavity suggest chronic ulcerative phthisis or bronchiectasis, and it should be remembered that both diseases are possible complications. In a typical case of pneumonokoniosis the physical signs are evolved with a fair degree of regularity. The earliest signs to become appreciable are those of a chronic bronchitis; then there appears the picture of a gradually developing hypertrophic emphysema; and finally signs of chronic interstitial pneumonia become evident, with or without signs of bronchiectasis or tuberculous cavities.

The sputum is of aid in diagnosis. In anthracosis it is black from the content of coal dust; in siderosis it is reddish or brown; while in chalicosis the shining particles of stone can be seen microscopically.

Diagnosis.—The diagnosis is made by obtaining a history of an occupation requiring the long continued inhalation of dusts, and physical signs of chronic bronchitis, emphysema, and cirrhosis of the lung, with or without evidences of cavity formation, and the characteristic sputum. Late in the disease the sputum may contain tubercle bacilli from tuberculous infection.

ATELECTASIS

Pathology.—Imperfect expansion of the lung or partial collapse of a lung which has become expanded may be congenital or acquired. In the congenital form of atelectasis the lung has never been properly expanded; while in the acquired form, which is a disease of later life, there occurs from various causes a partial collapse of the lung.

Congenital Atelectasis is a disease of the newly born, developing usually as a result of insufficient inflation of the lung due to the aspiration of meconium or mucus during parturition, or from weakness of the respiratory muscles subsequent to birth. In atelectatic children who survive, the anterior borders and upper lobes of the lungs are partially expanded, while the central and lower portions of the lungs are brownish red, vascular, and fail to crepitate upon manipulation.

As the child gains strength the anterior and upper portions of the lungs become emphysematous from compensatory emphysema, while the deeper portions are very slow to inflate. Congenital atelectasis is bi-lateral, involving both lungs, and usually to a similar degree.

It is probable that the central portions of the lungs of atelectatic children never attain to full inflation, as the tendency is rather for secondary changes to occur, leading to sclerosis and contraction of the deeper elements of the lung.

Acquired Atelectasis is often the result of compression of the lung by a pleural effusion, a tumor, aneurism, or deformity of the thoracic wall. The most fertile cause of the condition, however, is bronchial obstruction from foreign bodies, or external pressure of a tumor or aneurism. Similarly in severe cases of capillary bronchitis or broncho-pneumonia the terminal alveoli are obstructed by exudate and lead to circumscribed areas of atelectasis. In conditions of great debility the result of malnutrition or lying long in the recumbent posture with an exhausting disease sometimes induces areas of atelectasis.

The atelectatic area of the lung is darker than normal, is depressed, and is usually distributed in a number of areas corresponding to lobules of the lungs. On section the areas are usually dry, but may be moist from chronic passive congestion.

Physical Signs.—The physical signs of atelectasis are influenced and varied by the manifestations of the disease or condition which has given size to the atelectatic state of the pulmo-

nary tissues. The signs also vary in intensity and number with the volume of lung involved in the atelectasis and the condition of the undiseased portion of the pulmonary system. It is obvious that the physical signs arising from collapse of an entire lung will differ markedly in degree from those referable to a few scattered areas of collapse in one or both lungs.

Inspection.—Extensive atelectasis, involving a considerable area of a lung gives rise to dyspnea and sometimes to cyanosis, with inspiratory retraction of the intercostal spaces and epigastrium, with defective expansion of the side corresponding to the disease.

Palpation.—Vocal fremitus over the area of disease may be diminished, absent, or exaggerated, depending upon the state of the pulmonary parenchyma. Collapsed, toneless pulmonary tissue fails to conduct the vocal vibrations with the normal intensity, and if a main bronchus be completely obstructed, there will be an absence of vocal fremitus over the distribution of the bronchus. However, when secondary changes have set in in a case of atelectasis with partial broncho-stenosis the fremitus is transmitted with increased intensity. Pleural friction fremitus is occasionally demonstrable due to involvement of the pleura over the atelectatic area.

Percussion.—The dullness produced by small patches of atelectasis is masked by the hyper-resonance of the adjacent emphysematous lung tissue. An area of atelectasis must be large and superficially situated to give dullness upon percussion. A patch directly overlying a large bronchus yields the tympany of the bronchus upon percussion. Deeply seated areas of atelectasis fail to give dullness, owing to the intervention of the normal tissues between the area of disease and the thoracic wall.

Auscultation.—The vesicular murmur is feeble or abolished over an atelectatic area, unless the area overlies a large bronchus, when the murmur will be broncho-vesicular or purely bronchial. In non-extensive areas of atelectasis the only auscultatory phenomena may be a few rales upon deep inspiration.

In the atelectasis which develops at the bases of the lungs in patients who have long been in the recumbent posture full inspiration will reveal crepitant rales, owing to the fact that the alveolar walls which have become adhered with sticky serum are opened up and separated upon full inspiration.

The pulmonary second sound is accentuated in cases of extensive atelectasis.

In cases of congenital atelectasis the chief signs are marked dyspnea and cyanosis of moderate intensity, with inspiratory retraction of the lower interspaces, with cold extremities and attacks of syncope without apparent cause.

Diagnosis.—The diagnosis of pulmonary atelectasis is often for a time difficult or impossible. The finding of respiratory embarrassment and the location of some adequate causative lesion such as bronchial obstruction or pulmonary compression is suggestive. The physical signs are seldom clear-cut and distinctive, as small areas of collapse have their physical signs masked by the emphysematous condition of the surrounding lung. Moreover, as a larger area of collapse is apt to overlie a bronchus and have its tympanitic note engrafted upon the dullness of the atelectasis a pulmonary cavity may be suspected where none exists. The fact that the physical signs have a tendency to improve and to regress with changes of posture and deep inflation of the lungs is suggestive.

HYPERTROPHIC EMPHYSEMA

Pathology.—Hypertrophic emphysema, idiopathic or substantive emphysema, or the large-lunged emphysema of Jenner, is a condition of the lungs in which they are enlarged, their air cells greatly distended with air, and the inter-alveolar septa thinned and atrophic. It is also known by the names chronic or diffuse emphysema.

The disease is usually encountered in patients who suffer with chronic bronchitis or persons whose occupations require expiration with the glottis closed, as in glass-blowers, and players upon wind instruments. It is probable that impaired nutrition of the alveolar walls with the result that the elastic tissue is unable to contract and expel the air from the infundibula plays a part in the production of the disease. Freund's theory of the cause of hypertrophic emphysema assumes that it is primarily a disease of the costal cartilages; that there is a chronic hyperplasia of these cartilages, which ossifying prematurely, cause the chest wall to lose its elasticity, the emphysematous condition of the lungs developing as a result of lack of support.

Hypertrophic emphysema is bi-lateral, both lungs being involved to a similar extent. The distention of the lungs is general and universal in all directions, but it is most marked in the anterior borders, which, overlapping the heart, give rise to a

diminution of the area of that organ which is in contact with the anterior chest wall. The lungs are enlarged, are pale, are light and feathery to the touch and do not collapse readily when the chest is opened.

Under the microscope the air cells are observed to be very large, distended, and that in many instances the inter-alveolar septa have become atrophied and ruptured, forming larger cavities by the coalescence of several air spaces. With the destruction of the inter-alveolar septa the capillaries which they sup-

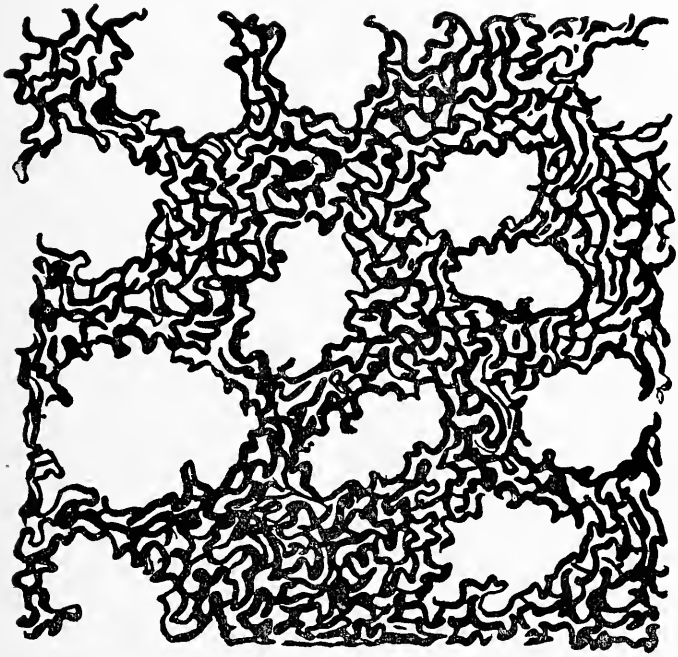


Fig. 87.—Pulmonary capillaries. The walls of the alveoli are thickly studded with capillaries; any marked alteration of alveolar air tension will therefore have a profound effect upon the circulation. (Brown, after Böhm, Davidoff, and Huber.)

ported are destroyed, and the quantity of blood exposed to the air in the lungs is commensurately diminished, resulting in deficient oxygenation of the tissues, cyanosis and dyspnea.

The pleura covering the lung loses its pigmentation in patches, a condition which was termed by Virchow Albinism of the lung. The bronchi and bronchioles show signs of bronchitis, and bronchiectatic dilatations are common. (See Fig. 74, p. 129.)

The right side of the heart is hypertrophied, due to the increased

load thrown upon it, and the tricuspid ring is usually enlarged, so that the valve segments often fail to close the orifice.

Rupture of the thinned lung may lead to the production of pneumothorax.

Physical Signs.—*Inspection.*—Hypertrophic emphysema produces a definite alteration in the shape of the chest, the barrel-chest of this disease. In this chest the antero-posterior diameter is increased often to such a degree that it equals or exceeds the transverse diameter. The expansion of the chest is minimal, the thorax rising and falling as one piece. The expiratory movement is much longer than is the inspiratory effort.

The cardiac impulse is often invisible, and there is often seen epigastric pulsation, while pulsations of the jugular veins are common. In the late stages with a failing heart, the patient shows dyspnea, and not infrequently is cyanotic. Not infrequently there is a delicate tracery of distended veinules over the lower portion of the thorax produced by intra-thoracic obstruction to the venous flow. (See Figs. 20 and 21, pp. 41 and 42.)

The facies of hypertrophic emphysema is fairly characteristic. The eyes are slightly prominent, the nose is somewhat thickened and cyanotic; while the head is thrown backward slightly in the effort to bring the accessory muscles of respiration into play. The neck is short and thick with prominent sterno-mastoids and trapezii.

Litten's diaphragmatic shadow is abolished; there is chronic cough; and the finger-tips are clubbed.

Palpation.—Vocal fremitus is diminished over both sides of the chest. In cases with marked bronchitis rhonchal fremitus is occasionally encountered. The cardiac impulse is rarely palpable, owing to the intervention of the emphysematous anterior borders of the lungs between the heart and chest wall. A systolic impulse in the epigastric region, owing to overaction of the right ventricle is readily palpable. The liver is seldom displaced sufficiently for its lower border to be freely palpated below the right costal arch; but in late cases when the right heart is failing, palpation of the liver reveals the systolic pulsation of tricuspid regurgitation.

The spleen is seldom displaced in hypertrophic emphysema. In late cases palpation of the abdomen may show the presence of moderate ascites.

Palpation reveals the deficient expansion of the thorax, the hard costal cartilages, and the rigidity of the muscles of the neck.

Percussion yields a hyper-resonant note over both lungs, the

limits of pulmonary resonance being increased in all directions. The area of cardiac dullness is restricted by the emphysematous anterior borders of the lungs. Percussion may be employed in detecting downward displacement of the liver, and the presence of ascites by eliciting dullness in the flanks with tympany in the median line of the abdomen.

The respiratory excursion of the lungs, determined by percussing out the lower borders of the lungs during expiration and inspiration, is very slight in hypertrophic emphysema, rarely exceeding half an inch.

Auscultation.—The respiratory sounds are feeble, with prolongation of the expiratory phase. The expiratory murmur is harsh, and not infrequently dotted with rales due to chronic bronchitis. The inspiratory murmur is always short, and may be entirely inaudible. Vocal resonance, like vocal fremitus, is impaired.

The heart sounds as a whole are diminished in intensity, owing to the intervention of the distended anterior borders of the lungs between the heart and chest wall. Of the individual sounds, the pulmonic sound is accentuated, owing to the obstacle to be overcome in the pulmonary circulation.

Diagnosis.—The diagnosis of hypertrophic emphysema can often be made at a glance. It rests upon the characteristic deformity of the chest, associated with dyspnea or cyanosis, persistent chronic cough, the short or absent inspiratory murmur, and the prolonged harsh expiratory murmur.

Chronic Bronchitis, with its chronic cough, shortness of breath, and slightly prolonged expiratory murmur, may resemble hypertrophic emphysema; but this disease does not produce the typic barrel chest, nor does it show general extension of the areas of pulmonary resonance, which forms so distinctive a feature of hypertrophic emphysema.

Pleurisy with Effusion is usually uni-lateral, and on percussion yields dullness or flatness instead of general hyper-resonance. It is usually accompanied by Grocco's sign, and aspiration shows the presence of fluid.

Pneumothorax, which in the incipiency may resemble hypertrophic emphysema, is a uni-lateral affection which develops rapidly, giving a hollow and tympanitic note on percussion, the succussion sound, the metallic tinkle, and the coin test.

ATROPHIC EMPHYSEMA

Pathology.—Atrophic emphysema, a state in which the total bulk of the lung is decreased, is a senile change, a part of the general wasting of the tissues of the body incident to advanced age. The disease, if such it may be styled, is associated with persistent chronic cough, lasting for many years, associated with chronic shortness of breath upon exertion.

In the subject with atrophic emphysema the thorax is abnormally small, the obliquity of the ribs is increased, and the excursion of the thorax with respiration is very much below normal.

The lung as a whole is smaller than normal, the pleura is deeply pigmented, while the pulmonary parenchyma shows evidence of pulmonary congestion, edema, or infarction. The bronchial tubes are dilated, the dilatations being surrounded by areas of induration.

Microscopically there is atrophy and rupture of many of the inter-alveolar septa, permitting the formation of larger or smaller chambers by the coalescence of several smaller ones. The capillaries are destroyed along with the inter-alveolar rupture, decreasing the quantity of blood exposed to the air in the infundibula.

Physical Signs.—*Inspection.*—The chest is small, the intercostal spaces are narrowed and pursue a more oblique course than normally, the supra-clavicular and infra-clavicular fossae are deeper than normal, while the thoracic excursion is slight. The dyspnea of atrophic emphysema, instead of being chiefly expiratory as in hypertrophic emphysema, is mixed, the duration of the two phases being approximately equal.

Palpation.—Vocal fremitus as a rule is slightly exaggerated owing to the increased density of the lungs, and the small amount of air contained.

Percussion.—The limits of pulmonary resonance are decreased in all directions. Even within the areas of resonance as determined by percussion there is moderate impairment of the normal vesicular quality of the resonance, attributable over the apices to fibrosis and condensation of tissue, and over the bases to edema of the lung. The area of cardiac dullness is extended owing to shrinking of the lung exposing a large area of the heart to the chest wall. The upper limits of hepatic and splenic dullness are higher than normal, owing to shrinking of the lung.

Auscultation.—The respiratory murmur is slightly impaired in

intensity, rarely attaining a broncho-vesicular character. In the presence of chronic bronchitis, which often coexists with the emphysema, there are dry and moist rales.

Diagnosis.—The diagnosis is easily made in an elderly subject with a small thorax, a generally "dried up" appearance, chronic shortness of breath, and chronic cough of long duration.

COMPENSATORY EMPHYSEMA

Pathology.—Compensatory emphysema is the name which is applied to a condition in which certain portions of a lung or an entire lung are over-distended with air owing to a decrease in the air space of the same or the opposite lung.

Compensatory emphysema may be transient or permanent. In inflammations of the terminal bronchioles with swelling of the mucosa, obliteration of the lumen of the tube is produced and the air in the infundibula is prevented from escaping during expiration and leads to atrophy and rupture of the inter-alveolar septa. If the obstacle to the egress of the air persists, large bullae or air spaces are formed from coalescence of several contiguous infundibula and a state ot permanent emphysema is produced.

A diffuse compensatory emphysema, involving an entire lung, usually results from massive pneumonia, chronic interstitial pneumonia, or a large pleural effusion, crippling the opposite lung. Localized areas of compensatory emphysema occur in a lung the seat of atelectasis, multiple patches of consolidation as that of broncho-pneumonia and tuberculosis, and localized fibrosis. (See Figs. 80 and 83, pp. 141 and 147.)

Physical Signs.—The physical signs of compensatory emphysema vary according to the extent of the emphysematous condition and its size, whether involving an entire lung or merely portions of a lung, and upon the extent of lung involved.

Inspection.—Upon inspection in a case where the entire lung is involved the side of the chest affected will be more prominent than the opposite side, which is usually shrunken owing to the crippling of the lung which occasioned the compensatory emphysema. Small areas of localized compensatory emphysema produce no alterations in the contour of the chest.

Palpation.—Vocal fremitus over a uni-lateral compensatory emphysema is unchanged or diminished in intensity, owing to the rarefaction of the pulmonary tissues.

Percussion yields a hyper-resonant note over an emphysematous lung; whereas in localized emphysema the note is scarcely altered.

Auscultation shows exaggerated or puerile breathing over the emphysematous lung, with slight prolongation of expiration.

Diagnosis.—A diagnosis of compensatory emphysema can seldom be made upon physical signs alone, as these are often few and obscure. The patient's history should be obtained, and some cause which would account for a compensatory emphysema elicited.

ACUTE VESICULAR EMPHYSEMA

Pathology.—Acute vesicular emphysema is a condition in which the air cells of the lungs are acutely distended from expiratory efforts or fits of coughing in the presence of an obstacle to the free egress of air from the lungs. It is apt to develop during bronchopneumonia, acute bronchitis, bronchial asthma, tracheal or bronchial stenosis, and during the extreme dyspneic attacks of cardiac failure.

The hyper-distention of the lungs occurs abruptly; but the air cells are merely distended; large cavities are not produced by rupture of the inter-alveolar septa as in hypertrophic emphysema; for recovery ensues or death occurs ere this change takes place.

Physical Signs.—The signs of acute vesicular emphysema are very similar to those of the hypertrophic form of the disease. There is a general extension of the limits of pulmonary resonance, the percussion note is hyper-resonant or tympanitic, and on auscultation there are sibilant rales distributed universally over both lungs, and a prolongation of the expiratory murmur. However, auscultation of the heart reveals no accentuation of the pulmonic second sound, as in hypertrophic emphysema.

INTERSTITIAL EMPHYSEMA

Pathology.—Interstitial emphysema is a condition in which air or gas is present in the interlobular or interlobar septa of the lung or beneath the pleura. Air may gain access to these regions as a result of traumatism or violent expiratory efforts, when rupture of the surface epithelium occurs and gives ingress of air to the deeper structures. It also occurs during convulsions, parturition, and while straining at stool. It may be caused by ulceration of the bronchi, or abscess or gangrene of the lung. Interstitial emphysema in the newly born has been caused during

violent efforts to mechanically inflate the lungs, and has resulted from spasmodic closure of the glottis.

When the air has gained access to the stroma of the lung it collects in the form of beads or bubbles varying in size. Some are very small, while others may attain the size of a walnut. These bubbles tend to make their way to the root of the lung and into the mediastinum and thence to pass upward along beside the trachea and to appear beneath the skin of the neck. Or the opposite sequence of events may be observed. Following tracheotomy wounds air may enter the tissues and burrow downward into the root of the lung and invade the stroma of these organs. Not infrequently the beads of air form bullæ just subjacent to the pleura; in which site rupture may cause pneumothorax.

Physical Signs.—Interstitial emphysema gives rise to few physical signs, and may escape detection entirely during a casual examination. When the air makes its way upward and appears subcutaneously it may produce a protrusion, which upon being palpated yields a crackling crepitus. Large beads of air beneath the pleura sometimes yield pleural friction sounds.

ABSCESS OF THE LUNG

Pathology.—Pulmonary abscess develops as a result of inflammations of the lung, as following lobar and catarrhal pneumonia, which do not terminate by resolution. A pulmonary abscess resulting from lobar pneumonia may occur in one of two forms. It may represent what is termed purulent infiltration, which merely represents an advanced stage of gray hepatization with tardy and incomplete resolution; or there may arise the formation of multiple abscesses throughout the lung. But pulmonary abscess more frequently follows catarrhal pneumonia, particularly the aspiration and deglutition forms of this disease. Abscess of the lung is an occasional complication of influenza.

The lodgment of foreign bodies in the bronchi sometimes produce ulceration and consequent abscess formation. Abscess of the lung may result from perforation of the lung from without or from within. Thus it may follow a perforating gunshot wound, the balls carrying in with them pieces of wearing apparel or other germ-laden material, or it may result from puncture of the lung by a fractured rib. Perforation of the lung from within may result from extension of esophageal carcinoma, suppura-

tion of contiguous mediastinal structures, or an abscess of the liver may rupture through the diaphragm.

Infectious embolism is responsible for a large percentage of pulmonary abscesses. This occurs in cases of pyemia and produces multiple abscesses usually situated beneath the pleura. These sub-pleural abscesses are conical in shape, with the bases directed toward the pleura. They are usually small, but may attain considerable size.

Pulmonary tuberculosis in the course of its evolution leads to pulmonary abscess. The tuberculous abscess is well circumscribed, occurs late in the course of the disease, and is associated with caseation and cavity formation.

From what has been said it is evident that pulmonary abscess is nearly always a secondary condition, secondary to disease or suppuration elsewhere in the body.

Owing to the characteristic structure of the lung suppuration in this organ does not always form a typical abscess with well defined walls; but may form a so-called purulent infiltration. In other instances, however, as in the tuberculous abscess, the pulmonary abscess has more or less well defined walls limiting the spread of the purulent focus.

In size the pulmonary abscesses may be small or may be quite large, involving the greater portion of a lobe. In rare instances an abscess may involve an entire lung. The pulmonary abscess is usually single, but in pyemic cases multiple abscesses are formed throughout the lung. The shape of the pulmonary abscess is very irregular, and the abscess is often divided into two or more loculi. The abscess is usually situated in the peripheral portion of the lung subjacent to the pleura, and usually occupies the lower lobe of the lung.

The contents of a pulmonary abscess is purulent, containing shreds of elastic tissue and necrotic debris. Evacuation of a pulmonary abscess may occur by rupture into a bronchus, the contents being partially expectorated, the shreds of elastic tissue in the sputum aiding materially in diagnosis; or, if the abscess be immediaately subjacent to the pleura, this membrane may be penetrated with the production of pyo-pneumothorax.

In unruptured abscess situated beneath the pleura that membrane is inflamed and covered with fibrinous or fibrino-purulent exudate.

Physical Signs.—The physical signs of pulmonary abscess vary with the type which the suppuration assumes. Multiple small

abscesses, distributed widely through the lung, and also a very diffuse purulent infiltration, give no distinctive physical signs. When the abscess is situated peripherally, just beneath the pleura, a friction fremitus and friction sound may be elicited in some instances, due to involvement of the pleura.

When a large abscess has formed the signs are those of cavity, these signs coming and going as the cavity is empty or fills up. When the cavity is filled there is impairment of the vesicular resonance in the area of the abscess, absence of breath sounds, and abolition of vocal fremitus and resonance. When, on the contrary, the cavity is empty there is tympany in this region with the usual signs of cavity, cavernous or amphoric breath sounds and exaggerated vocal fremitus and resonance, with the phenomena of Wintrich, Friedreich, and Gerhardt. In pyemic cases the general condition of pyemia may mask the true condition of the patient.

When an abscess ruptures into a bronchus the diagnosis may be established by the expectoration of pus containing shreds of elastic tissue. The sputum is often copious, and change of posture, by bringing the pus in contact with the healthy bronchial mucosa, may provoke an attack of cough with profuse expectoration.

The sputum in pulmonary abscess is often of rather offensive odor, but never has the stench of gangrene of the lung. The sputum contains shreds of elastic tissue and various pyogenic bacteria. In tuberculous abscess tubercle bacilli may occasionally be demonstrated in the expectoration. In abscess of long standing the sputum contains crystals of cholesterin.

Diagnosis.—Septic symptoms such as chill, fever, and colliquative sweats, with copious expectoration of purulent sputum, and signs of cavity formation usually in a lower lobe of the lung, following upon one of the conditions which may be provocative of pulmonary abscess, suggest the diagnosis. In pyemic cases the multiple small abscesses are often obscured by the general symptoms of pyemia. When a bronchus is penetrated and the copious expectoration containing elastic fibers occurs, the diagnosis is assured. When a patient with lobar pneumonia continues to have chills and fever after the crisis of the disease has passed, one should think of the possibility of a purulent infiltration supervening upon the pneumonic state.

Pulmonary abscess must be differentiated from *bronchiectasis* and *empyema*.

Bronchiectasis often leads to the copious expectoration of purulent or muco-purulent sputum, but this sputum does not contain elastic fibers, and the history of the two diseases is entirely different.

Empyema may be accompanied by chills and sweats, but in this disease there is an extensive area of flatness posteriorly and absence of breath sounds over this area. There is no copious expectoration with elastic fibers.

From *pulmonary gangrene*, which also produces cavities, abscess is differentiated by the absence of the extreme fetor of the former, which pervades a house or hospital ward and suggests the diagnosis.

GANGRENE OF THE LUNG

Pathology.—Pulmonary gangrene is an occasional sequence of lobar pneumonia, more commonly of catarrhal pneumonia, in patients debilitated by alcoholism, diabetes, or who suffer with chronic bronchitis. Gangrene of the lung may occur with pulmonary tuberculosis, pulmonary abscess, or tumor of the lung. Metastatic or embolic gangrene, following simple or infectious embolism is a common form of the disease. Gangrene of the lung may follow rupture into the lung of esophageal cancer, empyema, sub-phrenic abscess, or gastric ulcer.

Injuries of the lung, as gunshot wounds, frequently result in gangrene. Injury due to foreign bodies in the bronchi may be provocative of the condition. Gangrene of the lung may start from the putrid contents of a bronchiectatic or tuberculous cavity in the lung. Occasionally during convalescence from a protracted illness gangrene of the lung develops without apparent cause.

Pulmonary gangrene occurs in two forms, the diffuse, and the circumscribed forms. Of these, the diffuse is the rarer type of the disease. Diffuse gangrene of the lung occasionally follows pneumonia, but more commonly is the result of thrombosis of one of the large branches of the pulmonary artery. It is extensive, often involving the greater portion of a lobe of a lung. The affected area is dark in color, with a torn and ragged center, emitting an offensive odor.

In the circumscribed form of gangrene there are multiple areas of gangrene which are always separated by undiseased areas of the lung. The condition usually affects one of the lower lobes, and is usually confined to the peripheral portion of the lung. In the early stage the gangrenous area is dark, but firm;

later, softening occurs with the formation of a cavity which contains greenish, offensive fluid. Surrounding the gangrenous area is a zone of pulmonary tissue showing intense congestion, with splenization, and beyond this a zone of edematous pulmonary tissue. In rapidly spreading gangrene of the lung an artery may be eroded, leading to fatal hemorrhage. Perforation of the pleura is an infrequent complication, despite the peripheral distribution of the areas of gangrene.

Bronchitis is a concomitant condition, owing to irritation of the bronchial mucous membrane by the fetid contents of the gangrenous cavities. Gangrene of the lung is occasionally associated with abscess of the brain.

Physical Signs.—In a frank case of pulmonary gangrene with excavation of the lung the chief physical signs are those of cavity. When the gangrenous area is centrally situated, with healthy lung intervening between the site of disease and the chest wall, physical signs are obscure or lacking. There are present in nearly all cases characteristic signs of the coexisting bronchitis.

The breath is horribly fetid in pulmonary gangrene, this fact constituting a valuable sign of the disease when taken in conjunction with the sputum, which is characteristic. It is usually abundant, and upon standing separates into three strata; a lower of heavy greenish or brown sediment containing elastic fibers, granular material and pus, a median layer of brownish or greenish fluid, and a covering of grayish froth. Free blood may be present in considerable quantity; and hemoptysis may occur from erosion of an artery.

Diagnosis.—The foul breath, associated with the characteristic sputum, with signs of cavity formation, hemoptysis and signs of general debility and prostration, render diagnosis easy. In cases of latent pulmonary gangrene the breath is often not foul, nor is the sputum characteristic, owing to the fact that the gangrenous process is slow in progression and the area of gangrene is circumscribed by a fibrous wall. This form of the disease occurs particularly in diabetic and insane patients. In this class of patients an error in diagnosis is almost certain to arise. Mere foulness of the breath in any suspected case of pulmonary gangrene does not make the diagnosis, as a foul breath may result from putrid bronchitis, decomposition of the contents of bronchiectatic cavities, or carious teeth.

In *pulmonary abscess* the sputum is abundant and purulent, but

the breath is sweetish rather than foul, as in gangrene. Moreover, in the sputum of abscess shreds of elastic tissue are more abundant, and cholesterin crystals are more frequently encountered.

Pulmonary tuberculosis with cavity formation sometimes produces fetid breath and foul sputum containing elastic fibers; but the sputum in this disease contains tubercle bacilli, and the odor is never as foul as it is in pulmonary gangrene. Unfortunately, in gangrene acid-fast bacilli closely resembling in morphology the tubercle bacillus may be found in the sputum and lead to an error in diagnosis.

CHAPTER XI

DISEASES OF THE PLEURA

ACUTE FIBRINOUS PLEURISY

Pathology.—Acute fibrinous pleurisy, acute plastic pleurisy, or pleuritis sicca, is an acute inflammation of the pleura, which occurs in two forms, primary and secondary.

Primary acute fibrinous pleurisy occurs as the result of exposure to cold, particularly in patients debilitated by the use of alcohol or from other cause. Primary pleurisy has also followed contusions of the thorax.

Secondary acute fibrinous pleurisy is secondary to disease of the lung or to disease in more remote portions of the body. Thus, it follows or complicates many of the diseases of the lungs, as bronchitis, tuberculosis of the lungs or bronchial glands, lobar pneumonia and broncho-pneumonia, bronchiectasis, infarction, abscess, or gangrene of the lung. Acute fibrinous pleurisy arises as a complication of the acute exanthematous fevers and occasionally during the course of other acute infections.

Among primary lesions without the lungs which may be followed by acute fibrinous pleurisy may be mentioned endo-carditis, peri-carditis, tonsillitis, pyorrhea alveolaris, arthritis, and typhoid fever.

The disease usually involves the lower lateral and anterior portions of the pleura, in which site it may be localized to a very small area, or may involve the greater of the pleura covering the lung. The pleura becomes dull and lusterless, with a rather granular surface. The membrane is thicker than normal, and the surface is covered with one or more layers of fibrinous exudate. The exudate may be rolled into folds upon the surface of the pleura or may be thrown up into exuberant masses. There is a small amount of cloudy fluid exuding from the inflamed surface, but it never attains the degree which is seen in sero-fibrinous pleurisy or pleurisy with effusion.

Microscopically the pleura presents desquamation and degeneration of the covering endothelium in the seat of the inflamma-

tion, patches of the pleura being found entirely devoid of endothelial covering. The sub-serous connective tissue layer is edematous and shows a variable degree of leukocytic infiltration. The blood vessels in the zone of the disease are congested. Upon microscopic examination of the exudate which is thrown out it is found to contain fibrin, serum, and pus cells.

In very severe cases of long standing the visceral and parietal layers of the pleura often become adherent, crippling the movements of the lung to a variable extent during respiration.

Physical Signs.—*Inspection.*—The expansion of the thorax on the side of the disease is limited, and the respirations are slightly quickened. Litten's phenomenon is abolished upon the side of the disease.

Palpation yields the pathognomonic sign of acute fibrinous pleurisy; namely, pleural friction fremitus, caused by the rubbing together of the roughened surfaces of the visceral and parietal pleuræ. If the site of the disease is in the portion of the visceral pleura in contact with the pericardium, there is pleuro-pericardial friction fremitus.

Palpation confirms minor deficiencies of expansion on the side of the disease. Vocal fremitus is usually unchanged; but if there be considerable pleural thickening, its intensity is enfeebled over the area of the disease.

Percussion.—The note elicited upon percussion over an area of acute fibrinous pleurisy is little changed in quality; but percussion of the lower borders reveals limitation of the excursion of the diseased lung.

Auscultation over the site of disease reveals the pathognomonic pleural friction rub; and, in suitably placed lesions, pleuro-pericardial friction as well. The vesicular murmur is retained, but its intensity is slightly diminished. In great pleural thickening the murmur is abolished over the site of disease. Vocal resonance is as a rule unchanged; but in the presence of considerable pleural thickening, its intensity is diminished. (See Fig. 67, p. 921.)

Diagnosis.—The pleural friction when elicited is pathognomonic. The disease also produces darting stabbing pain in the side, which is increased by coughing and by deep inspiration, signs which also occur in other conditions.

In *pleurodynia* there is pain in the side, but this pain is continuous and made worse by movements of the trunk and by those of respiration. There is absence of the friction sound, and the localization of the pain is not as distinct as it is in pleurisy, the pain oc-

casionally leaving one side and appearing upon the opposite side of the chest. There is no fever as there is with pleurisy.

In *intercostal neuralgia* the pain is sharp and paroxysmal, exhibiting tender points over the exit of the nerves on the lateral and anterior chest walls. There is no friction sound and no fever.

SERO-FIBRINOUS PLEURISY (PLEURISY WITH EFFUSION)

Pathology.—Sero-fibrinous pleurisy, or pleurisy with effusion may follow exposure to cold or a wetting, which permits bacteria present in the bodily economy to attack the pleura; but in certain cases exposure is the only apparent cause of the disease.

Lobar pneumonia by involving the pleura over a consolidated lobe often causes sero-fibrinous pleurisy; but there is sometimes seen a primary pleurisy with effusion caused by the pneumococcus and arising independently of disease of the lung. Pleurisy with effusion is an occasional complication of nephritis or rheumatic fever, either due to the toxemia or to the bacteria associated with these disorders.

The most common organism associated with sero-fibrinous pleurisy is the tubercle bacillus. In these cases the tuberculous focus may be situated in the lung or in a distant portion of the body. The streptococcus is the cause of certain cases of sero-fibrinous pleurisy, with or without the coincident development of a streptococcic broncho-pneumonia.

In the female subject a sero-fibrinous pleurisy may be metastatic, resulting from tuberculous salpingitis.

Pleurisy with effusion attacks males more frequently than females, usually attacking persons between twenty and fifty years of age, though no age is exempt.

In sero-fibrinous pleurisy there is an initial dulling and loss of luster with roughening of the surface of the pleural membrane, but this is followed in a few hours by the exudation of a sero-fibrinous exudate. In a period varying from a few hours to several days there is more or less copious exudation of serous fluid from the surface of the inflamed pleura.

The fluid gravitates to the dependent portions of the pleural sac, and mounts up higher and higher as the effusion develops. In some cases the effusion becomes so great that it reaches the clavicle. The lung, compressed by the increasing fluid, is crowded

into the upper and posterior portions of the pleural cavity, occupying a comparatively small area near the spinal column.

The exudate consists of a straw colored fluid having a specific gravity of approximately 1020, containing flocculi of fibrin, epithelial and pus cells, bacteria, and blood cells. When the fluid is withdrawn spontaneous coagulation often is noted. The solid constituents of the exudate occupy the lower portion of the pleural sac, and the fibrinous material adheres to the surface of the pleuræ, where, often when the fluid is absorbed, it aids the formation of connective tissue adhesions between the visceral and parietal pleuræ.

These adhesions vary in extent in different cases. In some instances they are few in number and local; in other cases they are universally distributed over the entire pleura except for a pocket here and there; while in yet other instances they may obliterate the pleural sac entirely, resulting in a chronic adhesive pleurisy. When a patient with these numerous adhesions has a second attack of pleurisy, it is apt to involve only the non-adherent portions of the membrane, resulting in loculated or sacculated pleurisy.

In pleurisy with effusion the amount of the fluid in the pleural sac varies from one-half to four liters. After a variable course it tends to be spontaneously absorbed, often leaving after it the adhesions previously described.

In cases where the amount of fluid in the pleural sac is excessive there is visceral displacement. The liver or spleen is displaced downward and the heart is displaced to the side opposite the effusion.

Physical Signs.—*Inspection.*—The respiratory excursion on the side of the effusion is limited. If the effusion be large, there is uni-lateral bulging of the chest wall. Litten's sign is absent upon the side of the disease; the intercostal spaces are obliterated over the effusion or actually bulging.

In effusions of the right pleural sac the cardiac impulse is displaced toward the left and sometimes is elevated to the fourth interspace, in some instances being visible in the left mid-clavicular line, or even in the left axillary region. In left sided effusion the impulse is displaced to the right, often occupying a position behind the sternum. In extreme cases it may be visible to the right of the sternum in the third or fourth interspace.

The respirations are accelerated, owing to compression of the lung by the effusion, with consequent diminution of the air space. There is visible scoliosis, the spine deviating toward the side of

the effusion. The sound side of the thorax expands vicariously during respiration as a result of compensatory emphysema.

The decubitus of the patient is sometimes of aid in diagnosis. During the early, dry stage of the disease the patient is apt to lie on the sound side in the effort to protect the sensitive pleura from pressure; while, after the effusion has developed, he usually lies upon the side of the effusion in order to facilitate the full expansion of the sound lung. The shoulder upon the side of the effusion is on a slightly higher level than is its fellow. The nipple and scapula on the side of the effusion are farther from the median line than on the opposite side of the thorax.

Palpation.—In the first stage of the disease, prior to the development of the effusion, palpation reveals the presence in most cases of a pleural friction fremitus. If the lappet of lung overlying the heart is involved, there is pleuro-pericardial friction fremitus. The friction fremitus is present during the dry stage of the disease; it usually disappears with the development of the effusion; and it reappears upon its absorption. But not infrequently it can be detected during the effusion along its upper level, where the inflamed pleural membranes come in contact with the fluid. The friction fremitus not infrequently persists for years after recovery from the disease, as many patients can attest.

Vocal fremitus varies according to the degree of the effusion. In the presence of moderate effusion, which does not fill the pleural sac, vocal fremitus is normal above the level of the effusion; while over the effusion it is abolished. Posteriorly, near the vertebral column, the area occupied by the compressed lung, vocal fremitus is apt to be exaggerated. In considering modifications of the intensity of vocal fremitus, it must be borne in mind that dense pleural adhesions traversing a pleural effusion will and do transmit the vibrations to the palpating hand despite the presence of fluid in the pleural sac.

Palpation of the precordia confirms displacements of the apex beat, and palpation of the lateral thoracic regions shows deficient expansion of the diseased side. There is seldom edema of the chest wall in sero-fibrinous effusion, this sign being more commonly present in purulent effusions. In effusion of the right pleural sac palpation reveals the lower border of the liver at an abnormally low situation.

Mensuration.—Mensuration and comparison of the two sides of the thorax show an increase in the extent of the diseased side of

from one-half to one and a half inches. Allowance must be made for the fact that the right side is normally larger than is the left.

Percussion.—In the earliest stage of the disease the percussion note is unchanged. Later, as the effusion develops, there is a gradual impairment of the normal vesicular resonance, finally amounting to flatness over the effusion. Above the level of the effusion, the note is hyper-resonant, skodaic resonance. A similar hyper-resonant note is noted posteriorly above the level of the effusion. (See Figs. 55 and 56, p. 77.)

With the patient in the upright posture Ellis's line of flatness, indicating the upper limit of the effusion, can occasionally be mapped out. In pleurisy with effusion this line, representing the upper limit of the effusion, is not horizontal. It is higher posteriorly than anteriorly. In effusions of moderate extent the line begins low down in the posterior region of the thorax and proceeds upward and forward in a curve resembling the letter "S" to the axillary region, and thence proceeds in a gradual decline to the sternum.

Grocco's triangle of para-vertebral dullness is demonstrable in most cases of pleurisy with effusion. This triangular area, with a width of two to five centimeters, with its apex directed upward, occupies the side opposite to the effusion. It is probably due to displacement of the mediastinal structures by the pressure of the effusion. (See Fig. 52, p. 75.)

In right sided effusion the dullness of the fluid blends anteriorly and laterally with the dullness of the liver; whereas an effusion of the left side encroaches upon the tympany of Traube's semilunar space.

In sero-fibrinous pleurisy it is rarely possible to detect movable dullness upon change of posture. While not always present in this disease, movable dullness when elicited is an infallible sign of fluid.

Forcible percussion over the upper portion of the lung, above the level of the effusion, occasionally elicits a cracked-pot sound, produced by the sudden expulsion of air from the relaxed lung. Similarly, upon strong percussion over the infra-clavicular and mammary regions in very large effusions Williams's tracheal note may sometimes be elicited.

During absorption of the effusion the dullness gradually is superseded by normal vesicular resonance, save at the bases posteriorly, where the resonance is apt to remain impaired for

a long period. Areas of impaired resonance elsewhere point to areas of pleural thickening or encysted fluid.

Auscultation.—In the early stage of the disease, prior to the development of the effusion, a friction rub is often audible upon auscultation. It is usually most audible in the lower axillary region. The rub usually but not invariably disappears when the effusion develops to become once more audible when resolution or absorption begins. A pleuro-pericardial friction rub can be heard when the portion of the pleura overlying the pericardium is involved.

The breath sounds are abolished over the portion of the chest which overlies the effusion; while above the effusion the respiratory sounds are exaggerated or puerile. While the above statement as a rule is true, yet in very large effusions there is occasionally distant bronchial breath sounds audible over the effusion, due to the dense compression of the lung by the fluid which occupies the pleural cavity. The respiratory murmur over the sound lung is exaggerated or puerile owing to compensatory emphysema.

Vocal resonance over the effusion is abolished, unless a patch of pleura be bound to the chest wall by adhesions, in which event the resonance is audible in the area in question. While vocal resonance is as a rule absent over the effusion, yet in some cases bronchophony is encountered in this area. Along the inner border of the scapula and along the vertebral column egophony is found in some cases.

Baccelli's sign, the transmission of the whispered voice through a serous, but not through a purulent effusion serves to differentiate the former condition from the latter.

Upon auscultation of the heart, the sounds are often rather diffusely audible, owing to cardiac displacement. The pulmonic second sound is usually found accentuated, and a systolic murmur may be audible, which is produced by traction upon the vessels by cardiac displacement.

Diagnosis.—The diagnosis of sero-fibrinous pleurisy rests upon the deficient expansion and sometimes the bulging of the diseased side, the presence of the friction rub, which disappears with the advent of effusion, the absence of vocal fremitus over the effusion, the flat percussion note over the fluid, the absence of respiratory sounds over the effusion and puerile or bronchial sounds elicited above the level of the fluid, and presence of visceral displacement, with certain special phenomena as bron-

chophony, egophony and Baccelli's whispered voice in certain cases. The actual presence of fluid in the pleural cavity and its character are determined by aspiration. But aspiration may not in every case show the presence of fluid. Even if the aspirating needle is inserted in an area of flatness, it may penetrate a region where a thickened pleura is adhered to the chest wall and so fail to secure fluid, though it is present.

Pleurisy with effusion is often difficult to differentiate from lobar pneumonia. The points for differentiation between these diseases have been considered under lobar pneumonia (see page 123.)

From large pericardial effusion, sero-fibrinous pleurisy is sometimes differentiated with difficulty, particularly in the cases of left sided pleural effusion. But in pericardial effusion the base of the lung yields resonance instead of flatness; there is skodaic resonance over the adjacent portion of the lung compressed by the effusion; the cardiac impulse is not displaced to the right; the heart sounds are feeble; the pulse is the pulsus paradoxicus, trailing off toward full inspiration; and the degree of dyspnea is extreme, out of proportion to the extent of effusion. The area of dullness in pericardial effusion is pear-shaped with the base down.

Uni-lateral hydrothorax presents physical signs which are identical with those of sero-fibrinous pleurisy. But in hydrothorax there is absence of the initial friction rub; there is no primary pain in the side or fever; but instead a history of heart disease or nephritis. Hydrothorax is often accompanied by edema in other parts of the body.

Ellis's curve is not present in hydrothorax and upon aspiration the fluid of hydrothorax is more serous, is of lower specific gravity, below 1.014; it contains less albumin than 3 per cent; and does not coagulate easily; and contains no bacteria. In hydrothorax movable dullness is easily obtained, as the fluid in hydrothorax readily shifts with change of posture, while in pleurisy with effusion this sign is obtained with difficulty if, indeed, at all.

Intra-thoracic neoplasms may simulate pleurisy with effusion. They often produce displacements of the apex beat. The dullness, however, occupies the upper portions of the thorax and is of less extent, and is surrounded by a zone of compressed lung, yielding skodaic resonance. Vocal fremitus and resonance are increased rather than diminished. The breath sounds are often suppressed.

The growths often produce and coexist with moderate pleural effusion. Malignant neoplasms are prone to cause glandular enlargement in the supra-clavicular fossæ, and are eventually accompanied by cachexia. These growths also cause enlargement of the mediastinal glands, producing thus pressure paralysis of the recurrent laryngeal nerves. The physical signs are not influenced by change of posture.

Hepatic enlargement from abscess, echinococcus cyst, or enlargement from sub-phrenic abscess may and do simulate pleurisy with effusion. But the upper limit of dullness "is immovable, with its convexity upward and a friction sound is audible over the dull area, which would not be the case if the pleural surfaces were separated by fluid." These conditions are often complicated by moderate pleural effusion and hence coexist therewith. Upon aspiration, the pus from a hepatic abscess shows liver cells and bile and perhaps amebae. Grocco's sign is absent. An echinococcus cyst of the liver may produce hydatid fremitus.

Pneumothorax produces uni-lateral bulging of the chest wall with immobilization; but the percussion note is hyper-resonant or tympanitic; and the disease presents certain characteristic signs, as the coin test, the metallic tinkle, and the succussion sound.

LOCAL PLEURISY

Under the head of local or circumscribed pleurisy several conditions or forms of pleurisy are embraced, as diaphragmatic pleurisy, loculated, sacculated, or encysted pleurisy, and interlobar pleurisy.

Diaphragmatic Pleurisy

In this form of pleurisy the inflammation is limited chiefly to the parietal pleura covering the upper surface of the diaphragm and the visceral pleura in contact with it.

The inflammation, as a rule, assumes the dry, plastic type; but there is sometimes moderate effusion, which may be serous or purulent.

The Physical Signs are slight in comparison with the subjective symptoms, which are unusually severe in their manifestations. There is urgent dyspnea; the lower portion of the thorax is fixed, moving very little with respiration. A friction rub can sometimes be heard over the liver in right pleural disease, or over Traube's semilunar space in left sided inflammation.

There is tenderness upon pressure upon the lower intercostal spaces near the vertebral column, and extreme pain upon pressure over the insertion of the diaphragm at the tenth rib. There is often tenderness over the course of the phrenic nerve in the cervical region. Dysphagia is sometimes present, due to involvement of the esophageal orifice in the diaphragm; and hiccough and vomiting accompanying left sided diaphragmatic pleurisy. The vomiting and respiratory movements exaggerate the pain. The diaphragm and abdominal muscles are fixed, and the respiration is costal. The fever is often high. The pain is most intense in the epigastric region, simulating in this respect acute disease of the abdominal viscera.

The Diagnosis rests upon the very great severity of the subjective symptoms and the lack of physical signs. Gueneau de Mussy states that a pain extending from the tenth rib to the ensiform cartilage is pathognomonic of diaphragmatic pleurisy. Andral has noted cases attended with great dyspnea and attacks simulating angina pectoris.

Loculated, Sacculated, or Encysted Pleurisy

This type of pleurisy may be sero-fibrinous, but is more frequently purulent. In this form of pleurisy the fluid is circumscribed by adhesions between the visceral and parietal pleura into one or more pockets or loculi, which may or may not communicate with one another. While these loculi of effusion may develop in any portion of the pleural cavity, they are most frequently situated in the region between the mid-axillary line and the spine or upon the thoracic aspect of the diaphragm. In these cases the fluid may be bounded by adhesions, the result of a previous pleurisy; or an empyema may become limited and circumscribed by newly formed inflammatory adhesions.

The Physical Signs are slight and confusing. Areas of dullness may be found in certain cases; but this is not the rule; and vocal fremitus may be clearly transmitted by the adhesions. The free use of the aspirating needle is the surest means of diagnosis.

Inter-Lobar Pleurisy

In the evolution of sero-fibrinous or purulent pleurisy the pleura clothing the inter-lobar fissures of the lung are also inflamed, and often adhere, enclosing between the two pleural

layers a variable amount of sero-fibrinous or purulent effusion

Also in cases of lobar pneumonia and pulmonary tuberculosis an inter-lobar inflammation of the pleura may occur, with cohesion and retention of effusion. Inter-lobar pleurisy is usually purulent; and ofter simulates pulmonary abscess. Such a collection of pus may perforate and discharge into a bronchus and lead to the expectoration of purulent sputum.

Inter-lobar pleurisy usually develops near the root of the right lung, involving the pleura of the fissure between the upper and middle lobes of this lung.

The Physical Signs of inter-lobar pleurisy are often very confusing. As a rule there is little or no dullness upon percussion; but in certain cases a zone of dullness corresponding to the course of the fissure between the upper and middle lobes of the right lung can be found, limited above and below by a zone of skodaic resonance.

The x-ray is of aid in the diagnosis. Aspiration is dangerous, as the lung might be infected during withdrawal of the needle. The clinical picture often closely simulates that of pulmonary abscess.

PURULENT PLEURISY (EMPYEMA)

Pathology.—Purulent pleurisy, or empyema occurs rarely as a primary disease, chiefly in young infants; but in the vast majority of instances is secondary to disease or injury of the lung or thorax.

Purulent pleurisy follows infectious diseases as scarlatina, lobar or lobular pneumonia, and pulmonary tuberculosis. It may be the result of abscess or gangrene of the lung. It may be caused by penetration or perforation of the chest wall by a fractured rib or other penetrating wound. Perforation of the diaphragm by sub-phrenic abscess may cause empyema. Carcinoma of the esophagus may penetrate the pleura and be an exciting cause of empyema. Finally, a purulent pleurisy is a rare sequence of pertussis, measles, or typhoid fever.

A purulent pleurisy is usually purulent from the outset, it being very rare for an effusion which is primarily serous to become secondarily purulent. Empyema is especially frequent in young children, although no age is exempt from the disease.

The pneumococcus is responsible for the greater number of purulent pleurisies, either beginning as lobar pneumonia, or attacking the pleura primarily. Next in the order of their fre-

quency, come the pyogenic cocci, the tubercle bacillus, B. influenzæ, and the colon bacillus.

In purulent pleurisy the pleural cavity contains a variable amount of pus. The amount is usually quite large, often amounting to several liters. The solid constituents of the purulent fluid gravitate toward the dependent portions of the pleural sac, while the upper strata consist of fairly clear fluid. This fact may lead to an erroneous diagnosis; because, if the aspirating needle is entered above the level of the solid constituents, clear fluid may be withdrawn, suggesting the presence of a sero-fibrinous effusion.

The character of the pus varies in different cases. Usually it settles into two layers, thick below and clear above. In cases of considerable duration, it is thick and contains masses or shreds of fibrin. The pus may be odorless or fetid.

As the case progresses a greater or less degree of absorption of the exudate occurs. It is possible for the exudate to be entirely absorbed. Following absorption of the fluid elements, lime salts may be deposited in the remnants, imparting to them a gritty or calcareous quality.

The pleura in the presence of the purulent effusion is congested and covered with layers of sticky fibrin, and remnants of degenerated endothelium. Eventually the surface of the pleura becomes covered with a gray pseudo-membrane, which when stripped off, leaves erosions, indicating areas of endothelial degeneration and desquamation.

Pleural adhesions are frequently present in empyema. They are usually the result of a former pleurisy rather than of the empyema. They are mainly over the upper portions of the lung. This is probably because this portion of the pleuræ are in contact, while the portion lower down is separated by the fluid. The adhesions may be few, or they may extend well down to the surface of the fluid.

There is organization and thickening of the pleural membrane, tending to prevent the transmission of the sounds arising within the lung. The visceral layer of pleura is more thickened than is the parietal.

Changes in the Lung.—The empyema occupies space in the pleural sac previously occupied by the lung, so that this organ is compressed, and its expansion interfered with. This tends to produce a condition of atelectasis; and, in large effusions the

lung occupies a very small space. The lung becomes solid, airless, and of dark color.

The heart is displaced by the effusion, and the impact of this organ against the fluid causes "pulsating empyema." The liver is displaced downward.

Changes in the Thorax.—The collection of pus causes enlargement of the affected side of the chest. The intercostal spaces bulge from the pressure exerted upon them. The diaphragm is pushed down and with it the liver and spleen. "After evacuation the affected side collapses, the shoulder droops, the interspaces retract, and there is spinal curvature toward the affected side."

The pus of empyema may burrow beneath the costal pleura and point subcutaneously, constituting empyema necessitatis. Spontaneous evacuation of the pus will occur unless relieved.

The pus may perforate the visceral pleura and discharge into a bronchus, causing pyo-pneumothorax.

Physical Signs.—*Inspection.*—The physical signs of empyema on inspection are similar to those of pleurisy with effusion. But the diseased side bulges more than in sero-fibrinous pleurisy, and there is more extreme bulging of the intercostal spaces, particularly over the lower regions of the thorax. There is often edema of the chest wall. The apex beat is displaced and the diaphragm is depressed by the weight of the purulent effusion, producing downward displacement of the liver or spleen. Tortuous dilated cutaneous veins are often visible over the lower chest. Litten's sign is absent on the side of the disease. The diseased side exhibits no respiratory movement. There is visible pulsation in pulsating empyema, synchronous with the cardiac systole. In empyema necessitatis there is a protrusion of discolored integument, indicating the point where rupture is imminent.

Palpation.—Vocal fremitus over the effusion is absent, perhaps exaggerated above the level of the pus. Pulsating empyema yields a palpable systolic pulsation.

Percussion.—There is dullness amounting to flatness over the purulent effusion, and skodaic resonance above the fluid, as in pleurisy with effusion.

Grocco's triangular area of para-vertebral dullness is usually well marked.

Auscultation.—The whispered voice is often transmitted through a purulent effusion (Baccelli's sign). In children there is often blowing bronchial breathing above the level of the effusion.

Diagnosis.—Purulent pleurisy closely resembles a sero-fibrinous pleural effusion in the external manifestations. But in empyema the disproportion between the two sides of the chest is more marked; there is greater degree of intercostal bulging; and the visceral displacement is more extreme. Edema of the chest wall points to purulent rather than to serous or sero-fibrinous effusion. In empyema the dyspnea is greater, often amounting to orthopnea. There is less pain in empyema and there are signs of a septic state. Aspiration shows the presence of purulent effusion. Baccelli's sign is often of aid in differentiation from serous or sero-fibrinous effusion.

CHRONIC ADHESIVE PLEURISY

Pathology.—Chronic adhesive pleurisy, chronic plastic, or fibrinous pleurisy, usually is a result of sero-fibrinous effusion, more rarely of purulent pleurisy, and in rare instances develops as a primary or primitive affection.

When a pleurisy with effusion is absorbed or aspirated, the surface of the pleura is covered with an exudate rich in fibrin factors and frequently there are areas in which the surface endothelium has desquamated, exposing the subjacent connective tissue basis, of the pleural membrane. The surface of the visceral and parietal pleuræ, coated as they are with fibrinous exudate, have a tendency to adhere to each other, and, the fibrinous exudate having undergone organization, the two surfaces become bound together by fibrous adhesions. Between these adhesions, which vary In extreme cases the surfaces may adhere throughout their en- In extreme cases the surface may adhere throughout their entire extent, obliterating the potential pleural cavity.

Physical Signs.—The physical signs of chronic adhesive pleurisy vary with the duration of the disease and the extent of the adhesions, varying from moderate dyspnea to extreme embarrassment and thoracic deformity.

Inspection.—In cases with moderate adhesions slight dyspnea may be the only sign, or even this may be absent. But in more extensive cases, with many adhesions between the lung and chest wall local retraction or absolute immobilization of the side of the thorax is visible.

Palpation.—Pleural friction fremitus is demonstrable over sites of pleural roughening and thickening. Palpation may detect minor degrees of deficient expansion and retraction: Vocal fremitus is

as a rule diminished by the thickened pleura, occasionally exaggerated.

Percussion.—The percussion may be but little altered in cases of moderate pleural involvement; or the note may be strikingly dull and the resistance marked in cases with obliteration of the pleural cavity in large part or its entirety. The note over the sound lung is hyper-resonant.

Auscultation.—The pathognomonic pleural friction sound is audible, and the rales of an associated chronic bronchitis are often in evidence. The vesicular murmur is enfeebled or lost over a large portion of the thorax.

Diagnosis.—The diagnosis is made upon the presence of the pleural friction rub, the thoracic deformity and respiratory embarrassment, with a history of a previous acute pleurisy.

HEMOTHORAX

Pathology.—Hemothorax, a collection of blood in the pleural cavity, may result from rupture of an aneurism of one of the large intra-thoracic blood vessels or from erosion of an intercostal vessel in pleural disease; or it may occur as a result of trauma to the chest wall, as a perforating wound or perforation from a fractured rib. Gangrene of the lung may be responsible for the hemorrhage, or it may be a portion of a hemorrhagic diathesis. Rupture of an aneurism of the aorta usually produces hemothorax of the left pleura.

The onset of hemothorax is usually very abrupt. If one of the large vessels is the source, it is often rapidly fatal. In other instances the bleeding may be slowly continuous or may cease spontaneously after a variable length of time.

The amount of blood extravasated into the pleural sac is variable. If infection does not occur, following arrest of the hemorrhage complete absorption may occur; not, however, without leaving pleural adhesions.

Physical Signs.—These are the signs of internal hemorrhage; namely, pallor, dyspnea, rapid weak pulse, and collapse. Superimposed on these are signs of effusion in the pleural sac, and aspiration reveals the presence of sanguineous fluid.

CHYLOTHORAX

Pathology.—Chylothorax, the presence of chyle in the pleural sac, is encountered in rare instances. The chyle may be de-

rived from rupture of the thoracic duct or discharge by transudation from the lacteals. Thus, the thoracic duct may be ruptured by trauma to the thorax; or the duct may be obstructed by the pressure of an intra-thoracic tumor. Again, a chylous ascites may discharge into the pleural cavity by way of the lymphatics. Finally, occlusion of the left subclavian vein, into which the thoracic duct empties, may cause chylothorax.

Physical Signs.—The physical signs are those of pleural effusion. The nature of the effusion is determined by aspiration.

HYDROTHORAX

Pathology.—Hydrothorax, the presence of serous fluid in the pleural sac, develops as the result of transudation from the blood vessels in stasis of the blood stream, whether due to valvular heart disease, or to tumor pressing upon the large veins which convey blood to the heart. Blood changes, such as anemia and cachexia, may cause hydrothorax. Nephritis and carcinoma are occasional causes.

The Physical Signs are those of moderate fluid in the pleural sac. But no friction rub is present. Movable dullness is easily obtainable. The heart, liver and spleen may be displaced. Effusion is usually bi-lateral. In cases dependent upon regurgitant heart disease it is often uni-lateral, and usually affects the right side. The fluid is shown by aspiration. It is of low specific gravity, less than 1.014, contains little fibrin and albumin.

PNEUMOTHORAX (PYO- OR HYDRO-PNEUMOTHORAX)

Clinical Pathology.—Pneumothorax is an accumulation of air or gas in the pleural sac.

Pneumothorax is caused by perforating wounds of the chest wall by missiles or end of a fractured rib, or rupture of an empyema necessitatis.

Diseases of the lung which cause communication between the pleural sac and a bronchus will cause it, as for instance, following the rupture of a tuberculous cavity situated close under the pleura.

The development of the bacillus aerogenes capsulatus in the pleural sac will give rise to a primary pneumothorax. Perforation of the diaphragm by a sub-phrenic or hepatic abscess may be a cause. The commonest cause of the condition is the rupture of a tuberculous cavity.

The pleural cavity contains air or gas, which compresses the lung, which becomes shrunken and carnified. The heart, liver or spleen may be displaced. The air-containing pleural sac may be closed, with no opening to the exterior, or "open" with a communication with a bronchus or externally through the chest wall. There is usually a smaller or greater amount of serous fluid (hydro-pneumothorax), or of pus (pyo-pneumothorax) present in the dependent portion of the sac. The lung is compressed and small, and pushed upward and backward against the spinal column. The cardiac displacement is great, but its position is unchanged.

Physical Signs.—*Inspection.*—There is dyspnea and the facial expression is anxious.

The affected side of the chest bulges, and the intercostal spaces bulge. The apex beat of the heart is displaced. Expansion on the diseased side is absent, in marked contrast to the vicarious expansion of the sound side. The patient usually lies on the side of the pneumothorax in order to give the sound lung free play. There may be orthopnea. Litten's sign is absent on the diseased side.

Palpation.—Vocal fremitus is absent unless it is conducted to the chest wall by way of pleural adhesions.

Percussion.—The percussion findings depend upon the degree of tension of the air and the amount of fluid present therewith. If there is considerable fluid, it will give a dull note, with a sharp change into tympany when the upper border of the fluid is passed. The area of pulmonary resonance is increased upward above the clavicle and downward as a rule over the area of splenic and liver dullness. The coin test may be elicited. Movable dullness is demonstrable if fluid is present. (See Fig. 60, p. 81.)

Cases with bronchial communication will give a cracked-pot sound, and Wintrich's change of note. Percussion over the precordia may give tympany or resonance, owing to displacement of the heart to left or right. Also in cases with patent bronchial communication Biermer's phenomenon, an alteration in the pitch of the percussion sound with change in the patient's posture, may be elicited. The dullness of the liver or spleen extends lower than normally. Biermer's phenomenon is often demonstrable.

Auscultation.—The respiratory sounds are diminished or absent over the pneumothorax. In some cases distant amphoric breath sounds may be audible. The voice sounds are ringing and amphoric. Over the sound lung the breathing is puerile. The metallic tinkle, falling-drop sound, or gutta cadens is audible; also

the succussion sound upon shaking the patient. The lung fistula sound may be audible in cases with patent bronchial communication. The heart sounds are apt to have a hollow, echoing sound, due to the proximity of the air in the pleural sac. (See Fig. 57, p. 78.)

Diagnosis.—The uni-lateral bulging, with suppressed or absent breath sounds, tympanitic percussion note, the falling-drop sound, and succussion sound, with cardiac and visceral displacements make a characteristic picture.

The differential points between pneumothorax, pleural effusion, and hydrothorax, have been discussed in a previous section. (See page 176.)

SECTION IV

PHYSICAL EXAMINATION OF THE CIRCULATORY ORGANS

CHAPTER XII

CLINICAL ANATOMY

The Heart.—The heart, the great muscular pump by which the blood is propelled through the vessels, is roughly conical in shape, presenting for examination a base, three borders (right, left, and inferior), and an apex. The heart is situated rather obliquely in the middle mediastinum, with the base directed backward and toward the right side of the thorax, and the apex directed forward and toward the left side. The heart does not occupy the mid-point of the thoracic cavity; but projects farther to the left of the median line than to the right. Approximately one-third of the organ is found in the right half of the thoracic cavity, and two-thirds in the left half of this cavity.

The heart is divided by a longitudinal septum and a horizontal septum into four cavities, the right and left auricles and right and left ventricles. The auricles and ventricles are connected by the right and left auriculo-ventricular valves. The left auricle and ventricle contain arterial blood, while the right auricle and ventricle contain venous blood.

The left auriculo-ventricular, or *mitral valve,* intervening between the left auricle and left ventricle, consists of a ring and two segments or cusps, whose closure is limited by the chordæ tendineæ and papillary muscles. The right auriculo-ventricular or *tricuspid valve,* comprises a ring and three cusps or segments, similarly limited and controlled by papillary muscles and chordæ tendineæ. Arising from the left ventricle is the aortic orifice, comprising a ring and three segments, the segments being devoid of chordæ or papillary muscles; but the segments are reinforced near their center by a thin cartilaginous plate, the corpus aurantii. Opposite each segment the wall of the aorta has a small pouch or dilatation, the *sinus or Valsalva,* from two of which arise the coronary arte-

ries for the nourishment of the myocardium. The pulmonary valve, intervening between the right ventricle and the pulmonary artery, has the same structure as the aortic valve.

The cardiac *wall* consists of specialized involuntary muscle fibers, the cardiac muscle, or *myocardium,* consisting of several layers of muscle fibers, the fibers taking various directions. The myocardium is clothed internally by a serous membrane, the *endocardium,* which is reflected over the cardiac valves; and is covered externally by a similar serous membrane, the *epicardium,* which constitutes the visceral portion of the pericardium.

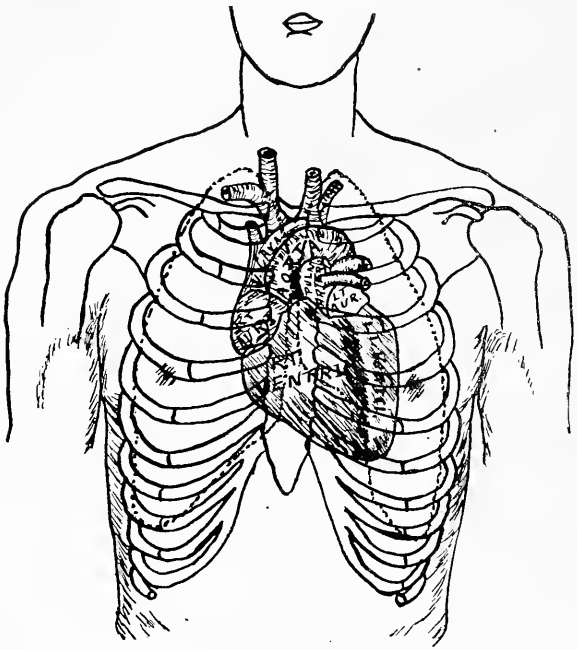


Fig. 88.—Relations of chambers of unopened heart to anterior chest wall.

Arteries and Veins.—Arising from the left ventricle is the aorta, the greatest artery of the body. The aorta passes upward and to the right behind the sternum to the upper border of the second right costal cartilage; thence backward and toward the left, arching over the root of the left lung, to reach the left side of the fourth dorsal vertebra, whence its course is downward along the left side of the vertebral column to enter the abdominal cavity through the aortic orifice of the diaphragm.

The *pulmonary artery,* arising from the right ventricle, courses

upward and backward to the inferior aspect of the aortic arch, where it divides into right and left branches which enter the roots of the corresponding lungs. It is about two inches in length, and is connected to the lower portion of the aortic arch by the ligamentum arteriosum, representing the obliterated ductus arteriosus.

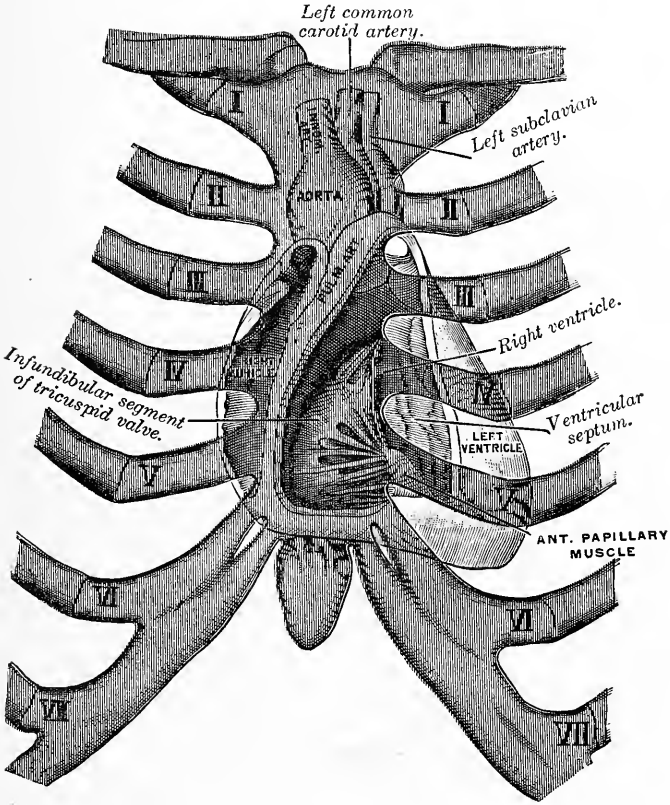


Fig. 89.—Relations of opened heart to anterior chest wall. (From Gray.)

Opening into the left auricle are the four *pulmonary veins,* returning arterial blood from the lungs; while emptying into the right auricle are the *superior vena cava,* returning venous blood from the upper portion of the general circulation, and the *inferior vena cava,* returning the venous blood from the abdominal cavity and lower extremities. (See Fig. 2, p. 19.)

The Pericardium.—The pericardium is a fibrous sac, covered on its external and internal surfaces by a thin serous membrane, the internal serous covering being reflected over the surface of the

heart as the epicardium. The pericardium secretes a small amount of serous fluid, enabling the parietal and visceral layers of the pericardium to glide noiselessly over each other during the cardiac contractions. The pericardium, with the heart, is pear-shaped with the base directed downward. The base of the pericardial sac is moored to the central tendon of the diaphragm by a little areolar tissue.

The pericardium and heart are separated by the diaphragm from the left lobe of the liver, and, on the extreme left, corre-

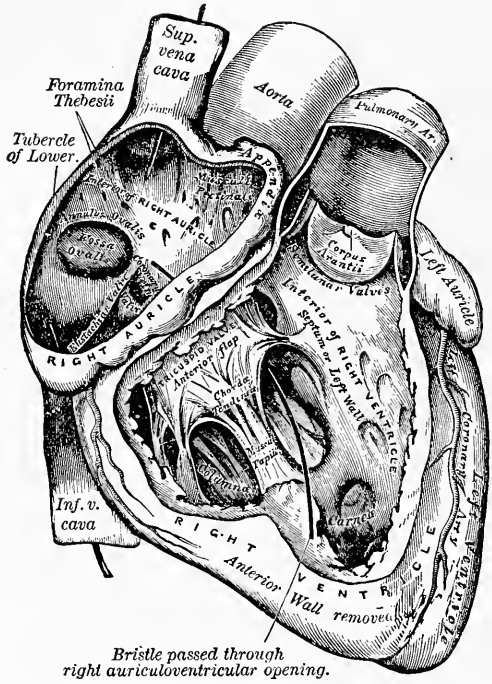


Fig. 90.—Interior of right auricle and ventricle. (From Gray.)

sponding to the apex of the heart, from the stomach. Upon either side the pericardium is covered by the mediastinal pleura, and is overlapped laterally and to a great extent anteriorly by the lungs. But anteriorly in the area of the Incisura Cardiaca a small portion of the right ventricle comes into direct contact with the chest wall at the inner extremities of the fifth and sixth costal cartilages. Superiorly the pericardium is continued upward for a short distance upon the great vessels which leave the heart.

Surface Topography of the Heart.—The *base* of the heart,

formed by the right and left auricles, corresponds to a line crossing the sternum obliquely from the lower border of the second left costal cartilage, about one-half inch to the left of its junction with the sternum to the upper border of the third right costal cartilage, approximately one inch beyond its sternal junction.

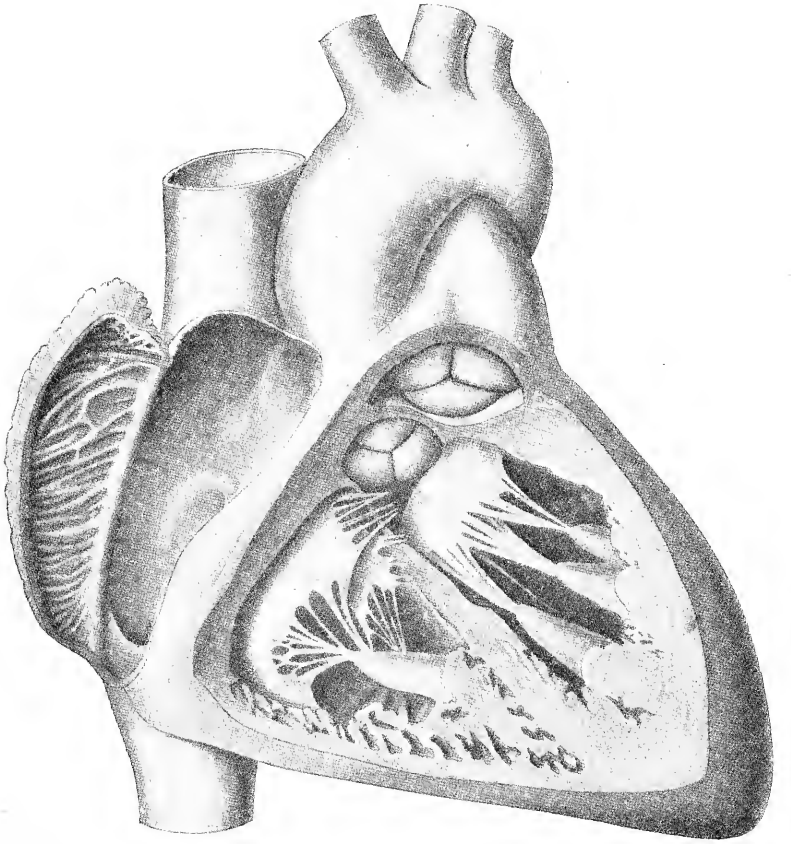


Fig. 91.—Interior of right auricle and both ventricles, showing mechanism of cardiac valves. (From Gray.)

The *right border* of the heart, formed by the right auricle, corresponds to a curved line, with its convexity directed to the right, extending from the upper border of the third right costal cartilage approximately one inch from its junction of the sternum, to the sixth right costo-sternal articulation. (See Fig. 89, p. 189.)

The *inferior border of the heart*, formed almost entirely by the right ventricle, and to a small extent by the left ventricle, corresponds to a line drawn from the sixth right costo-sternal articu-

lation to the position of the normal cardiac impulse in the fifth left interspace one-half inch internal to the mid-clavicular line.

The *left border* of the heart, formed by the left ventricle, is represented by a curved line drawn with its convexity toward the left, from the fifth left interspace one-half inch internal to the mid-clavicular line to the lower border of the second left costal cartilage, one-half inch to the left of its articulation with the sternum.

The *cardiac valves* all lie within a small ellipse extending from the third left costo-sternal articulation to the junction of the sixth right costal cartilage with the sternum. This area covers the anatomical site of the valves, but is not the area in which their sounds are best appreciated acoustically.

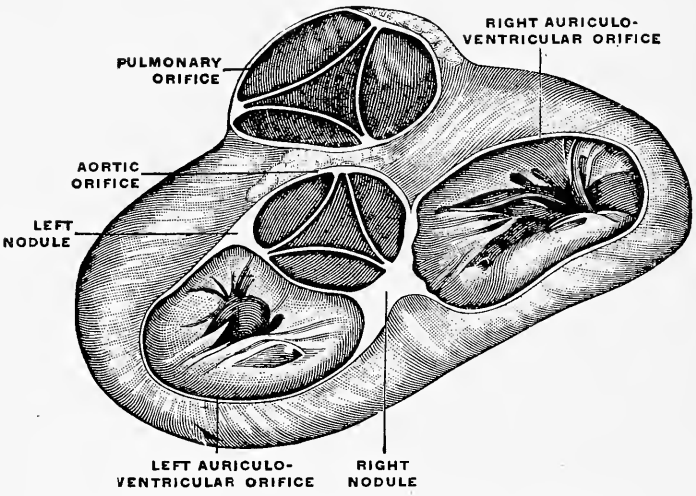


Fig. 92.—Fibrous rings at bases of cardiac valves. (From Gray.)

The Aorta.—The ascending portion of the aorta is represented by a broad line drawn from the third left condro-sternal articulation to the second right costo-sternal articulation; thence the vessel takes a course backward and to the left, the arch ending upon the left side of the body of the fourth dorsal vertebra.

The *pulmonary artery* corresponds to a broad line drawn from the second left intercostal space to the upper border of the second left costal cartilage, the point of bifurcation of the artery into its two main branches.

The Precordia.—The term precordia is applied to the region of the surface of the thorax which overlies the heart. The name does not refer simply to the limited region in which the heart

is directly apposed to the thoracic wall, but also to the region in which the anterior borders of the lungs intervene between the pericardium and heart and the thoracic parietes. The region embraces the areas of cardial dullness and cardiac flatness, to be described in a subsequent section. The precordia presents sharp margins or lines of division or borders above and to the left, but on the right side, it is continuous with the areas of hepatic dullness and flatness. (See Fig. 100, p. 213.)

CHAPTER XIII

INSPECTION

In the study of cardio-vascular disease inspection reveals undue prominence or recession of the precordia, abnormal pulsations in the neighborhood of the precordia, the position and character of the cardiac impulse and the presence of the capillary pulse in Corrigan's disease.

THE PRECORDIA

Undue prominence of the precordia is noted in pericarditis with effusion, cardiac hypertrophy and dilatation in patients with thin chest walls and in children. Aneurism of the aortic arch produces bulging in the upper portion of the precordial region. Precordial prominence may arise from causes not connected with the circulatory organs, as a tumor of the chest wall, lung or pleura; a cold abscess of the sternum, or a small localized pleural effusion.

Undue recession of the precordia is often the result of the traction of pericardial or pleural adhesions. A not infrequent cause of retraction in this region is fibrosis of the left lung in fibroid phthisis or chronic interstitial pneumonia. A similar recession of the lower region of the sternum is seen in the funnel-chest, sometimes as the result of occupation, in other instances occurring as a congenital defect.

ABNORMAL AREAS OF PULSATION

At the Base of the Heart.—A visible pulsation at the base of the heart over the manubrium sterni, accompanying and synchronous with the systoles of the ventricles is indicative of aneurism of the transverse portion of the aortic arch. As a rule, when a visible pulsation is present in this locality the aneurism has eroded the bone, and is accompanied by pain of a boring character.

To the Right of the Sternum.—A pulsation visible along the right margin of the sternum ranging from the second to the fifth

interspace, is indicative of right auricular dilatation, fibroid retraction of the right lung associated with cardiac displacement, or to cardiac displacement by left sided pleural effusion or pneumothorax.

To the Left of the Sternum.—Visible pulsation along the left sternal margin from second to sixth interspaces is indicative of aneurism of the descending portion of the aortic arch, dilatation of the left auricle or ventricle, fibroid retraction of the left lung, or displacement of the heart to the left by pressure of air or fluid in the right pleural sac.

In Epi-sternal Notch.—Visible pulsation in the epi-sternal notch sometimes follows the ingestion of stimulants, and often as a normal phenomenon in elderly subjects. Pulsation in this region also accompanies aneurism of the arch of the aorta, and develops when the subclavian artery is exposed by fibroid retraction of the lung. A pulsation in this notch sometimes accompanies anemic states.

Systolic Pulsation of the Liver.—A systolic pulsation of the liver is indicative of tricuspid regurgitation, and is usually accompanied by edema of the lower extremities. Often the pulsation is not visible, but is readily palpable upon bimanual palpation. A true pulsation of the liver occurring with regurgitant tricuspid lesion must be differentiated from the impulse which is frequently transmitted to the liver by an overacting right ventricle.

Epigastric Pulsations.—A systolic pulsation of the epigastrium is sometimes noted in healthy persons without possessing untoward significance. Moreover, a systolic epigastric pulsation may accompany the condition of Bathycardia an abnormal low position of the heart in the thoracic cavity. Pathologic shortness of the sternum causes a pulsation in the epigastrium, systolic in time. A systolic epigastric pulsation accompanies hypertrophy of the right ventricle, and occurs also when the heart is displaced to the right so that the apex lies behind the sternum.

A diastolic pulsation of the epigastrium accompanies states of anemia and neurasthenia and is noted in patients with chronic gastric indigestion. A diastolic pulsation in this region accompanies a tumor of an abdominal organ overlying the aorta, the pulsation being transmitted to the tumor at each pulsation of the vessel. A similar pulsation accompanies aneurism of the vessel. These pulsations are often not visible, but plainly palpable.

Systolic Pulsation of the Jugular Veins.—A systolic pulsation in these vessels is indicative of tricuspid regurgitation, the blood at each systole of the right ventricle regurgitating into the right auricle and causing a pulsation in the great vessels emptying their contents into this chamber. A second cause of such a pulsation is mitral regurgitation in the presence of an unclosed foramen ovale, in which event the back flow is felt through this abnormal opening upon the blood content of the right auricle, thus offering an impediment to the free discharge of the blood returning into the right auricle.

DIASTOLIC COLLAPSE OF THE JUGULAR VEINS (FRIEDREICH'S SIGN)

A diastolic collapse of these vessels is indicative of chronic adhesive pericarditis. In this disease the traction of the mediastino-pericardial adhesions draw the chest wall in during cardiac systole, the flexible thoracic parietes expanding during diastole, thus exerting a suction upon the blood in the great vessels at the base of the right auricle, aspirating the blood from these vessels and leading to their sudden collapse during diastole.

The Venous Pulse.—The venous pulse, as recorded in the internal or external jugular veins, may be encountered in either of two forms: (1) the *negative venous pulse* of health; and (2) the *positive venous pulse* of pathologic significance.

Negative Venous Pulse.—The negative, auricular, or presystolic venous pulse is represented by a sequence of presystolic pulsations, usually demonstrable in the external jugulars, and only rarely in the internal jugular veins. The negative venous pulse can only be demonstrated in thin patients. In this type of venous pulse the presystolic pulse wave is initiated by the systole of the right auricle, which, in addition to forcing the auricular contents onward through the right auriculo-ventricular valve, also causes, coincidently, an impulse which is transmitted or imparted to the blood column in the superior vena cava and innominate veins, resulting in a presystolic impulse which is visible or palpable over the external jugular vein, particularly on the right side.

These veins, during late diastole or just prior to ventricular systole, the time which corresponds to auricular systole, are full, owing to the increased intra-auricular tension; whereas, during ventricular systole, corresponding to auricular diastole, the veins

collapse, owing to lowering of the intra-auricular tension, which promotes the unimpeded flow of blood from the veins.

Sphygmograms from the external jugular veins of the negative venous pulse show often but a single wave, which corresponds to the auricular systole. But there may be two or even three waves, the second wave occurring during ventricular systole and corresponding to the closure of the right auriculo-ventricular valve; the third wave occurring during ventricular diastole, and corresponding to the closure of the pulmonary valve.

In determining the time of the negative venous pulse the examiner should palpate the jugular vein with the finger-tips of the left hand, while applying the tips of the fingers of the opposite hand to the opposite carotid artery or to the cardiac apex. This maneuver serves to distinguish the normal, negative venous pulse, which is presystolic, from a false venous pulse transmitted from the subjacent carotid artery, which is systolic. Moreover, if digital compression is applied to the vein near the middle of the neck, after it has been emptied by pressure applied from below upward to the point of constriction, in the case of the negative venous pulse the proximal portion of the vessel remains empty, while the distal portion, the portion beyond the point of compression, becomes overfilled and tortuous. Under these circumstances in a false venous pulse the carotid pulsation is not transmitted to the lower, collapsed portion of the vein; but it is increased over the portion of the vein above the site of compression.

Positive Venous Pulse.—The positive, ventricular, or systolic venous pulse is represented by a sequence of systolic pulsations of the internal jugular veins. This type of venous pulse is caused by direct regurgitation of blood into the right auricle from the ventricle during systole, as the result of incompetence of the tricuspid valve. Hence, it is a purely pathologic physical finding. Usually first demonstrable in the right jugular, owing to its closer proximity to the right auricle, the pulse eventually develops in the left jugular vein. In thoroughly competent jugular veins the impulse is interrupted at the supra-bulbar valve. Under these circumstances the impulse is appreciable in the inter-sterno-mastoid fossa, just above the sterno-clavicular articulation. But, as a rule, the valve above the bulb is not entirely competent and permits the impulse to be transmitted upward into the veins of the neck.

While the provocative lesion of the positive venous pulse is in the great majority of cases tricuspid insufficiency, such a pulse is

also produced in the rarer cases of mitral incompetence associated with patent foramen ovale. The positive venous pulse is usually accompanied by systolic pulsation of the liver.

The Centripetal Venous Pulse.—A visible pulsation, the centripetal, or penetrating venous pulse, is occasionally visible in the veins of the dorsum of the hand or foot or in the delicate mammary veins. This pulse is most frequently associated with aortic insufficiency or anemia, in which it represents an exaggeration of the capillary pulse of Quincke.

ABNORMAL RETRACTION OF THE THORAX (BROADBENT'S SIGN)

In chronic adhesive pericarditis, with each systole of the ventricles, traction is exerted upon adhesions extending between the pericardium and chest wall, producing a systolic retraction of the thorax, most noticeable posteriorly below the angle of the

Fig. 93.—Site of normal cardiac impulse.

left scapula in the 10th and 11th interspaces. There is not infrequently a similar systolic retraction of the anterior chest wall. Aside from chronic adhesive pericarditis, systolic retraction of a local area of the chest wall may arise from the traction of pleural adhesions, or may be an accompaniment of ventricular hypertrophy.

THE CARDIAC IMPULSE

With each systole of the ventricles there occurs a visible impulse upon the chest wall. This impulse, which overlies the apex of the heart, is usually visible; and in the rare instances in which it is not, is readily palpable. In the normal adult subject this impulse is located in the fifth left intercostal space one-half inch internal to the mid-clavicular line. In infants and young children the impulse occupies a higher level and is situated further from the median line, being found in this class of subjects in the fourth interspace three-eighths of an inch external to the mid-clavicular line. In the aged, on the contrary, the cardiac impulse is frequently very low, occupying the sixth or the seventh interspace.

The impulse covers an area of approximately one inch, and its force varies with the general physical development of the individual and with the state of the myocardium. A determination correctly of the exact position of the impulse affords very accurate information as to the position of the heart in the thoracic cavity.

The cardiac impulse should be studied with a view to the detection of displacement from its normal situation, its extent, and its strength.

Displacement of the Cardiac Impulse

The cardiac impulse may be displaced by pressure exerted upon the heart by adjacent viscera of the thorax or abdomen, by pathologic alterations in the myocardium, or by the collection of fluid in the pericardial sac, the direction and degree of the displacement often yielding a clue to the cause of the displacement.

Upward displacement of the cardiac impulse is indicative of cardiac atrophy and moderate pericardial effusion. The impulse is also displaced upward by increased sub-phrenic pressure occurring in diaphragmatic hernia, sub-phrenic abscess, ascites, large tumor of an abdominal organ, hepatic enlargement, and in peritonitis, and tympanites.

Downward displacement of the impulse occurs as the result of the growth of an aortic aneurism or a mediastinal tumor pressing upon the heart, or the pressure exerted by the over-inflated anterior borders and upper portions of the lungs in hypertrophic emphysema.

Displacement to the Left.—The cardiac impulse is displaced to the left by the action of an accumulation of fluid or gas in the right pleural sac. The impulse is displaced toward the left when pleuro-pericardial adhesions between the pericardium and left lung exert traction upon the heart. Fibroid retraction of the anterior borders of the left lung in fibroid phthisis cause left displacement of the impulse. (See Figs. 81 and 83, pp. 145 and 147.)

In left ventricular hypertrophy and dilatation the impulse is displaced to the left and downward. The pressure of a mediastinal tumor may cause a similar displacement.

Hepatic enlargement or distention of the stomach causes displacement of the impulse upward and toward the left. A similar displacement occurs in the presence of moderate pericardial effusion.

Displacement to the Right.—The cardiac impulse is displaced toward the right by the pressure of fluid or gas in the left pleural cavity, and is drawn to that side by the action of right pleuro-pericardial adhesions. Compensatory emphysema of the left lung when marked may push the impulse to the right. In right ventricular hypertrophy and dilatation of the heart the impulse is displaced toward the right, perhaps lying behind the sternum. A similar displacement is noted in the rare cases of congenital transposition of the thoracic viscera. (See Fig. 82, p. 146.)

Abnormal Extent of the Cardiac Impulse

Increased Extent.—The extent of the cardiac impulse is exaggerated in emotional states, and following physical exertion or excitement. The extent of the impulse is increased in cardiac over-action accompanying acute fevers or disorders of cardiac innervation. The area of impulse is increased by a mediastinal tumor pushing the heart forward, in cardiac hypertrophy and dilatation, in which latter it is seen over a very wide area. An increased area of impulse occurs when fibrosis of the anterior border of the left lung exposes an increased extent of the cardiac wall to the thoracic parietes, and when the left lung is drawn aside by pleural adhesions.

Decreased Extent.—In hypertrophic emphysema owing to the crowding of the anterior borders of the lungs between the heart and chest wall the area of impulse is decreased or abolished. In extreme grades of cardiac dilatation the impulse is frequently decreased or invisible. In deep chested subjects there may be a

very slight impulse or it may be absent. An absence of impulse may in some instances be explained by the fact that the apex is situated behind a rib.

Abnormal Strength of the Cardiac Impulse

Increased Strength.—In cardiac hypertrophy, in addition to being more diffuse than normally, the cardiac impulse is of increased force. Displacements of the impulse are usually found with hypertrophy. During active physical exertion, and strong emotional excitement there is a temporary increase in the strength of the impulse. Following the ingestion of cardiac stimulants, during the course of the acute fevers, and during acute myocarditis, the impulse is abnormally strong.

Decreased Strength of the cardiac impulse occurs when cardiac dilatation supervenes upon hypertrophy, in conditions of fatty change in the myocardium, and in cardiac atrophy. In the presence of edema or inflammation of the chest wall the strength of the cardiac impulse is diminished, and in pericardial effusion or hypertrophic emphysema it is weakened or abolished. In pericarditis with effusion the impulse will sometimes become visible when the patient bends forward, disappearing when the erect posture is resumed.

CAPILLARY PULSATION (THE CAPILLARY PULSE)

Systolic pulsation in the capillaries is sometimes a normal phenomenon, may be the result of temporary loss of vasomotor tone during anemia or febrile diseases; but it is a very valuable sign of Aortic Insufficiency or Corrigan's disease. There are several methods of demonstrating capillary pulsation. A good method is by blanching the finger nail by the exertion of slight pressure upon the tip of the nail when a systolic flushing and a diastolic blanching of the subungual tissues will be observed, the capillary pulse. Another method of demonstrating the phenomenon is by drawing the nail over the forehead producing a line, which is alternately red and blanched. A third method of detecting the capillary pulsation is by covering the lower lip by a glass slide and observing the systolic flushing and diastolic blanching of the lip compressed by the slide. The capillary pulse is frequently accompanied by visible pulsations in the veins of the dorsum of the hand or foot. (See page 198.)

CHAPTER XIV

PALPATION

In the study of cardio-vascular disease palpation is employed to locate the cardiac impulse when not visible. In performing this maneuver the whole hand should be placed flat palm down on the thorax over the apical area, not the fingers only. In palpating the apex beat information may be gained as to increase or decrease in the area of impulse, exaggeration or enfeeblement of its force, its quality, whether slow and heaving, as in hypertrophy of the left ventricle, or quick and slapping, as in cardiac dilatation; or whether the apex beat is regular or irregular. Shock from closure of the valves may be appreciated and, finally, friction fremitus and thrills may be detected. Palpation is also employed in study of the pulse. Palpation also confirms the findings of inspection as to prominences or retractions in the precordial region, and pulsations within or without the precordia.

Valve Shock.—This is due to closure of the valves of the heart, and can be appreciated when the hand is applied flat over the valve area. It may be felt over the auriculo-ventricular valves, but more distinctly over the semilunar valves. In both instances it is more intense in persons with thin chest walls. In the aortic and pulmonic areas it is more intense in cardiac hypertrophy.

The pulmonic shock is intensified by left sided valvular lesions and in obstruction to the pulmonary circulation, as occurs in cases of emphysema and cirrhosis of the lung, conditions which raise blood pressure in the pulmonary circulation.

The aortic shock is intensified in arterio-sclerosis, nephritis, and other conditions associated with increased blood pressure in the greater circulation.

The shock of the auriculo-ventricular valves is systolic; that of the semilunar valves is diastolic.

Pericardial Friction Fremitus.—This is a tactile vibration due to the rubbing of the parietal and visceral layers of the pericardium, which have become roughened by inflammation in the development of acute fibrinous pericarditis or the early stage of

pericarditis with effusion. The fremitus in the latter disease is likely to disappear with the development of the effusion, though it is not uncommon for it to persist at the base.

Pericardial friction fremitus does not extend beyond the precordia and is usually accompanied by some pain, which is aggravated by pressure over the lower end of the sternum.

Thrills.—Upon palpation of the precordia of a patient the subject of valvular heart disease, aneurism, or certain pathologic conditions of the blood, a vibration of the chest wall is noted which is not dissimilar to the sensation conveyed to the hand

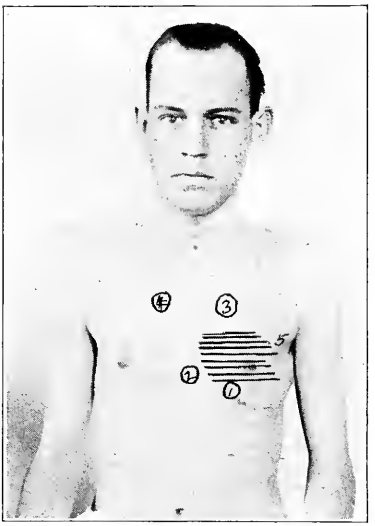


Fig. 94.—Sites of palpable thrills and pericardial friction fremitus. *1*, thrill at mitral valve; *2*, thrill at tricuspid valve; *3*, thrill at pulmonary valve; *4*, thrill at aortic valve; *5*, *pericardial friction fremitus*.

when placed upon the throat of a purring cat. This vibration is termed a thrill.

Thrills may be cardiac, vascular, or hemic; they may be presystolic, systolic, or diastolic. A cardiac thrill is produced by the same condition which is responsible for a murmur; namely, a narrowing of an orifice through which the blood stream is projected into a wider chamber beyond. So long as normal blood flows through normal vessels and orifices, of normal caliber, no sound is generated; but when the lumen at one point is decreased, the blood passing through this into the wider distal portion produces the so-called "fluid veins," the vibrations of

which are conveyed to the surface of the precordia as a tactile vibration, the thrill.

In feeling for a thrill the entire palm of the hand should be applied lightly to the precordia. Firm pressure may obliterate the thrill.

In general, a thrill at the base of the heart indicates aneurism of the aortic arch, but it may also occur in aortic or pulmonic valve lesions, particularly stenotic lesions, and in exophthalmic goiter.

A thrill at the apex of the heart, pre-systolic in time, is a very good sign of mitral stenosis. If systolic in time, it is a sign of mitral regurgitation.

A thrill at the second right costal cartilage indicates trouble with the aortic valve or aorta, usually aortic stenosis. It may be due to aortitis, or aneurism. This thrill is systolic. A diastolic thrill at this area would signify aortic regurgitation.

A systolic thrill at the second left costal cartilage is indicatve of pulmonic stenosis, while a diastolic thrill at the same area indicates pulmonary regurgitation.

A thrill may be elicited over the carotid artery in exophthalmic goiter, due to the enlarged thyroid compressing the artery and disturbing its lumen.

THE PULSE

By the term *pulse* is understood the expansion and contraction or rather retraction of an artery following each systole of the ventricles. Usually the radial artery at the wrist is the site selected for studying the pulse on account of its readiness of access, but other arteries, as the temporal, carotid, or femoral will serve the purpose. The pulse may be studied by digital examination, or by the sphygmograph.

Technic of Taking the Pulse.—In studying the pulse the patient should be in the sitting or recumbent posture with the arm extended and resting upon a table or supported by the left hand of the examiner. The physician, seated beside the patient, should place three fingers over the radial artery, the index finger being nearest the patient's hand. With the fingers in this position the examiner may roll the artery beneath them and can study the several factors which enter into the analysis of the pulse.

It is well in conditions where there is no cause for hurry to count the pulse for a full minute, as observed by the second hand of a watch. It can, however, be counted for 20 seconds and the

result multiplied by three; or counted for thirty seconds and the result multiplied by two.

In certain diseases the pulse becomes so rapid that it is impossible to count the beats. Under such circumstances an approximate estimate of the frequency of the beats may be made by endeavoring to count every other beat, or the examiner may

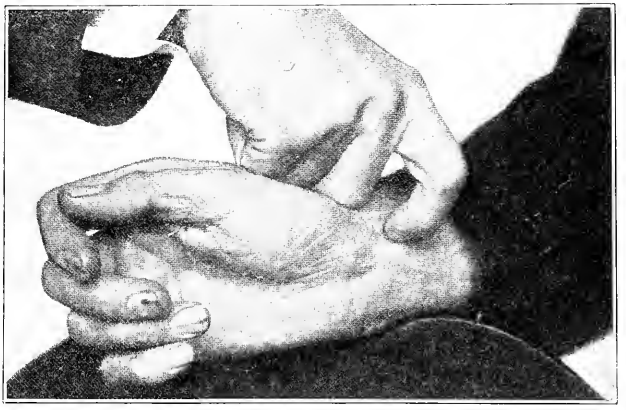


Fig. 95.—A method of finger-tip palpation of the radial artery. (Warfield, after Graves.)

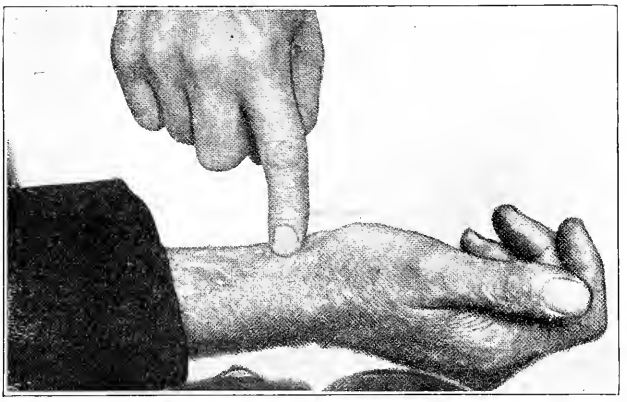


Fig. 96.—Another method of finger-tip palpation of the radial artery. (Warfield, after Graves.)

make dots with a pencil held in the unengaged hand and count the number of dots made during a minute.

In certain conditions in which the radial pulse cannot be felt, as well as in cases where it is suspected that every systole of the ventricles does not produce a radial pulsation, the exam-

iner may arrive at a conclusion by auscultating the apical area and counting the systoles.

The Sphygmographic Tracing.—A tracing of the normal pulse or a sphygmogram shows that the pulse wave consists of a sudden upstroke, the anacrotic limb, and of a gradual decline, the catacrotic limb. The last mentioned limb falls gradually to the base line and is interrupted by a distinct notch midway in its descent, the dicrotic notch, followed by an immediate second ascent to a variable extent, the dicrotic wave, which is followed by a second wave, the post-dicrotic wave on the descent to the base line.

The significance of the two principal strokes of the tracing is understood, the anacrotic limb being produced by the injection of blood into the already distended arteries by the ventricular

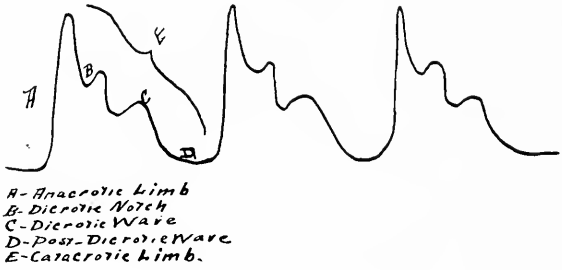


Fig. 97.—Normal sphygmogram.

systole, and the catacrotic limb being produced by the recoil of the elastic arteries to their normal caliber.

The cause of the dicrotic wave is uncertain. It occurs immediately following the closure of the aortic valves. It occurs in all pulse tracings, varying, however, in degree. In certain pathologic conditions, as the early stages of typhoid fever, it is so pronounced as to lead to the designation "dicrotic pulse." The accepted explanation is that the dicrotic wave is due to the rebound of the distended aorta at the time of the closure of the semilunar valves.

The second notch on the catacrotic limb and the undulatory oscillations of the fall are probably due to inertia of the instrument.

Variations in the Sphygmogram.—The most frequent pathologic variations in the sphygmogram are due to increase and decrease in the arterial tension respectively.

Thus, in conditions of high arterial tension, after the anacrotic

limb attains the maximum height, instead of receding immediately as in normal tension, the stroke is sustained, for a variable time, producing a "plateau," gradually falling to the base line.

In states of low arterial tension, on the contrary, the tracing presents a vertical anacrotic limb, with a quick fall to the base line with a rather marked dicrotic wave.

Changes in the Artery.—When the radial artery is palpated with the finger-tips, it will be observed that at each pulse wave the artery is changed from a flat, compressible tube into a circular one, and that the vessel lengthens or straightens out.

Analysis of the Pulse.—In analyzing the pulse the examiner should study the points noted below, variations in one or all of which possess diagnostic significance:

1. The condition of the artery wall.
2. Size of the artery.
3. Rate.
4. Rhythm.
5. Tension.
6. Volume.
7. Force.
8. Duration.
9. Bi-lateral symmetry of the pulses.

The Condition of the Artery Wall.—The wall of the normal radial artery in a person not advanced in age is soft and yielding, readily compressible, and cannot be distinguished from the surrounding tissues. Any departure from this elastic state is significant. In the aged and in arterio-sclerosis due to alcoholism, syphilis, gout, or other cause, the artery becomes hard and its walls unyielding. The vessel may often be rolled between the fingers and the lower end of the radius and feels like a pipe stem. Or the artery may be beaded and tortuous, with palpable nodules due to plaques of atheromatous degeneration. The artery may be merely stiff and compressed less readily than usual. This is normal in persons past middle age.

The Size of the Artery.—Variations in the size of the radial artery have little significance. The artery may be congenitally larger or smaller than normal. Temporary variations in caliber are due to increase or decrease in the blood content and are dependent on the amount of blood expelled at each systole of the ventricle.

The Rate of the Pulse.—The normal pulse rate in an adult male

is 70 to 75 beats per minute. The rate, however, is modified by many factors, as the age, sex, the size of the body, the position assumed by the patient, and the relations of the time of taking the pulse to the meals. Thus, at birth the pulse rate is 130 to 140 beats per minute. During the first year of life it is 115 to 130; at the seventh year it average 85 to 90; whereas in the aged it drops to 60 to 70 beats per minute. In women the pulse is usually more rapid than it is in male subjects.

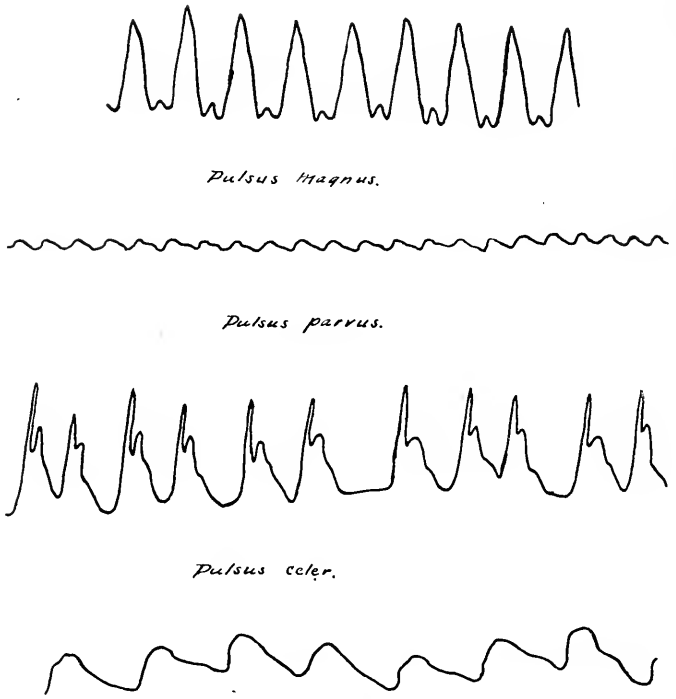


Fig. 98.—Sphygmograms of pathologic pulses. (After Da Costa.)

The size of the body has a slight influence on the pulse rate, the rate being slower in large subjects than it is in small persons. The position of the patient influences the pulse rate, the pulse being more rapid when counted in the upright posture than when counted while the patient is recumbent. The pulse rate is quickened for an hour or two following a full meal. Exercise and mental or emotional excitement temporarily increase the pulse rate, not infrequently doubling the normal rate for the

individual. Finally, the examiner should remember that the ingestion of many drugs influence the rate of the pulse and proper questions should be directed toward this point.

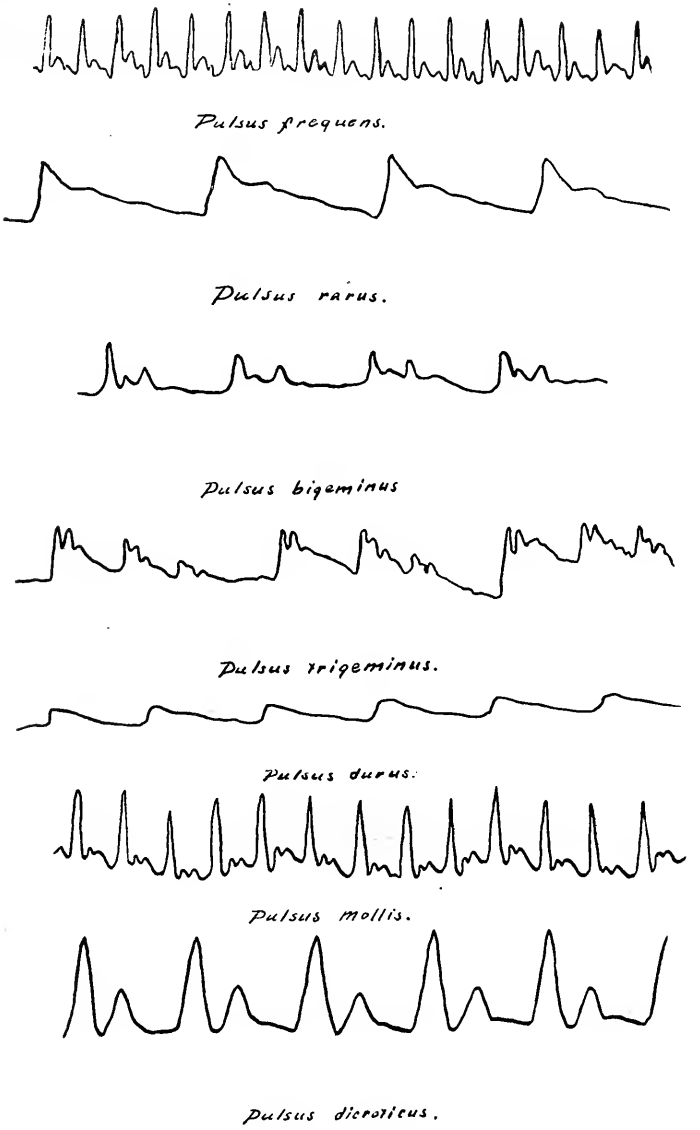


Fig. 99.—Sphygmograms of pathologic types of pulse. (After Da Costa.)

Physiologically the rate of the pulse is influenced largely by the degree of peripheral resistance. If the peripheral resistance is high, the heart will contract more slowly; whereas if the

peripheral resistance be decreased by relaxation of the arterioles, the heart will contract more rapidly. The rate is also influenced by the action of the vagus nerves, stimulation of these nerves slowing the heart, and depression permitting the heart to contract more rapidly.

As previously mentioned, the pulse rate varies with the age, sex, size, and position of the patient. An abnormally slow pulse rate is termed *bradycardia*, while the opposite condition, a very rapid pulse rate is termed *tachycardia*. Bradycardia occurs during convalescence from typhoid fever, pneumonia, and the acute infectious fevers, in cerebral tumors and hemorrhage, and after injuries to the cervical portion of the spinal cord, and in all conditions producing continuous stimulation of the vagus centers. The pulse rate is also decreased in aortic stenosis and in sclerosis of the coronary arteries and in general arterio-sclerosis, depressed fracture of the skull, and fibrous myocarditis.

Tachycardia occurs during fevers and vagus neuritis, exophthalmic goiter, during violent exercise and emotional states, as well as during failing compensation in valvular lesions of the heart.

Rhythm.—In health the pulse beats are of equal force and the beats are separated by uniform intervals. It follows that an irregularity of the pulse may have reference either to the force or the time sequence of the beats.

Arrhythmia is a deviation from the normal sequence of the beats without the omission of beats. It is observed in the course of acute fevers, valvular heart lesions, particularly mitral stenosis, digestive disturbances, following excessive indulgence in tobacco, in brain lesions, gout, myocardial degeneration, in which it may be the only sign, during mental excitement, and occasionally in elderly persons without pathologic significance.

Intermission is the occasional omission of a pulse beat. The omission may occur at irregular intervals or the omission may follow a regular sequence, every third, or every fourth beat being omitted. Intermission may persist throughout life without significance; or it may be merely a transient phenomenon. It is usually attributable to nervous depression or excessive use of stimulants or tobacco.

In analyzing a case of intermission it is important to determine whether the omission is due to an omission of ventricular systole, the *pulsus deficiens;* or whether due to a ventricular contraction

which is too feeble to produce a radial pulse, the *pulsus intermittens.*

When omissions of the pulse beats follow a regular sequence; when they are rhythmically irregular, it is termed an *allorhythmic pulse.* In this variety of intermission belong the *pulsus bigeminus,* where two beats occur in regular sequence, and are followed by an omission; and the *pulsus trigeminus,* in which three beats occur regularly to be followed by an omission of the pulse.

A very frequent form of irregularity is represented by the *pulsus intercidens,* a pulse in which after several regular beats the last regular beat is quickly followed by a weak one.

The *paradoxical pulse* is a pulse in which on completion of inspiration the beats become small and more rapid, and may become imperceptible at the wrist. It occurs in pericarditis with effusion and chronic adhesive pericarditis.

Irregularities in the force of the successive pulse beats is due to ventricular systoles which are not of equal force. This condition is seen in the *pulsus alternans,* a pulse of regular rhythm but in which the successive beats are irregular in force.

Volume.—The volume or size of the pulse is dependent upon the amount of blood expelled during ventricular systole, the ability of the aortic valve to prevent regurgitation, and the state of the vasomotor system. Thus, cardiac hypertrophy combined with vasomotor depression, permits relaxation of the arteries with the production of a full bounding pulse, the *pulsus magnus.* On the other hand, a weak or dilated heart, expelling at each systole a small quantity of blood, combined with vaso-constriction produces a small thready pulse, the *pulsus parvus.* This pulse is observed in inanition, mitral stenosis and regurgitation, and in marked aortic stenosis, conditions in which the ventricle cannot eject a large amount of blood into the aorta.

Force.—The force of the pulse depends upon the energy with which the ventricle contracts, and upon the elasticity of the arterial walls. If the ventricle is hypertrophied but the arterial walls have lost their elasticity, much of the heart's force in expelling the blood is wasted or lost by the absence of the elastic recoil of the arteries. In general, the force of the pulse is increased in conditions of cardiac hypertrophy and is decreased in conditions of cardiac debility. Moderate stimulation of the vagus nerve increases the pulse force by slowing the heart and increasing its energy; but if the stimulation is extreme, the heart beats are so few that the decreased blood content of the arterial system is not

sufficient for the powerful ventricular systole to act upon and the force of the pulse is diminished.

Tension.—The tension of the pulse depends upon the energy of the cardiac contractions and the degree of peripheral resistance offered to the blood stream. Thus, with a powerfully contracting heart and the peripheral resistance increased by angiospasm or arterio-sclerosis, the arterial tension is increased (hyper-tension). Hyper-tension occurs in all conditions of cardiac hypertrophy, arterio-sclerosis, nephritis, uremia, and apoplexy. On the contrary, when the output of blood from the ventricle is decreased by cardiac dilatation or valvular lesions, combined with vasomotor relaxation or decrease of the amount of circulating blood due to anemia, hemorrhage, or cachexia, the arterial tension is diminished (hypo-tension).

In hyper-tension the hard pulse is designated the *pulsus durus* in contradistinction to the soft, yielding pulse of hypo-tension, the *pulsus mollis.*

Often in the course of a continuous fever there is noted a pulse of low tension and rate, and full volume, in which there is a reduplication, appreciable to the palpating fingers as a minor beat, superimposed upon the principal beat, the *dicrotic pulse.* It is to be attributed to excessive elasticity of the arteries combined with a more or less general relaxation of the smaller arterioles. With the arterial system in this state, when the blood enters the arteries from the ventricles they are unduly distended, and the contraction of the vessels upon the blood column causes the secondary pulse wave.

Duration.—The duration of the pulse depends upon the degree of peripheral resistance, the elasticity of the arteries and the duration of ventricular systole.

The duration is increased in the slow sluggish *pulsus tardus,* which is always associated with increased peripheral resistance, due to constriction of the small arterioles, such as occurs in arterio-sclerosis, renal disease, and angina pectoris. The sphygmographic tracing of such a pulse shows a gradual upstroke, a well sustained plateau and a gradual fall to the base line. This slow pulse, the *pulsus tardus,* is observed also in aortic stenosis, in which case it is to be ascribed to the rather prolonged systole of the left ventricle and in which case the arterial pressure is low.

The duration of the pulse is diminished, producing a quick pulse, the *pulsus celer,* in conditions associated with diminished peripheral resistance, due to relaxation of the arterioles, such as

occurs in febrile states. A form of the pulsus celer is the *water hammer pulse,* or Corrigan Pulse which is observed in aortic regurgitation. This is a rapid pulse characterized by a sudden full expansion of the artery, followed by a sudden collapse of the vessel.

Bi-lateral Symmetry of the Pulse.—Normally the radial pulse is exactly alike at the two wrists both as to time and character. This bi-lateral symmetry of the pulses may, however, be disturbed,

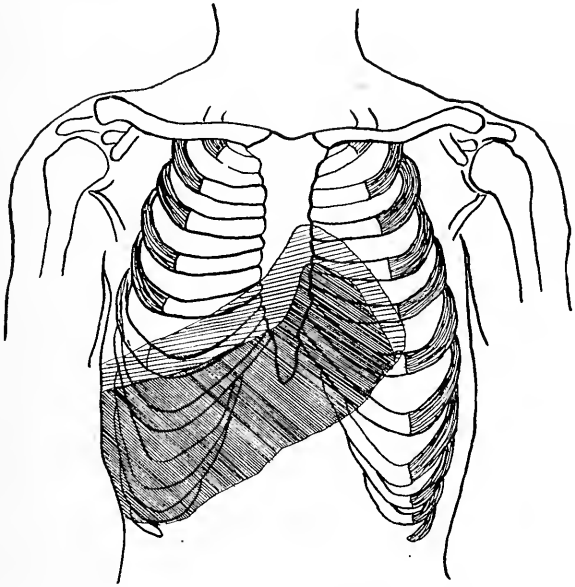


Fig. 100.—Areas of cardiac and hepatic dullness and flatness.

even to the total absence of the pulse at one wrist. Aneurism of the ascending aorta, or innominate artery, may retard the right radial pulse, while aneurism of the subclavian, axillary, or brachial arteries may cause retardation on either side.

Fracture of the bones of the arm or injuries producing cicatricial compression of the artery in the axilla or arm, as well as compression by tumors or enlarged glands will alter the character of the pulse at the wrist and cause asymmetry of the pulses. Pneumothorax or large pleural effusion by compressing the subclavian artery may cause a retardation or may alter the character of one radial pulse.

CHAPTER XV

PERCUSSION

Percussion is employed in the study of the circulatory organs chiefly to determine the position of the heart, and to detect alterations in its shape and size, as well as the presence of fluid in the pericardial sac. In the determinaton of the size, shape, and position of the heart, this information is gained by outlining the areas of cardiac dullness upon the surface of the thorax.

Areas of Cardiac Dullness.—When the examiner percusses toward the heart from various points in its vicinity upon the surface of the thorax, two changes in the quality of the percussion note are observed. As the borders of the heart which are overlaid by the anterior borders of the lungs are reached, the normal vesicular resonance becomes impaired, and finally, when the portion of the heart which lies in direct apposition with the chest wall, uncovered by the lungs is reached, the note becomes flat. Thus, there are and may be defined upon the thoracic surface two areas of cardiac dullness, the one within the other. The inner, representing the area in which the heart is uncovered by the lungs is termed the *area of absolute cardiac dullness* or the *area of cardiac flatness,* whereas the outer area, representing the portions of the heart which are overlapped by the anterior pulmonary borders is termed the *area of relative cardiac dullness.*

The area of absolute cardiac dullness, representing the small portion of the right ventricle which is directly apposed to the anterior thoracic wall corresponds to a roughly triangular area, bounded on the right by a vertical line drawn along the left border of the sternum from the level of the fourth costal cartilage to the upper border of the sixth costal cartilage, on the left by a line drawn downward with a slight inclination toward the left from the junction of the fourth left costal cartilage with the sternum to the fifth left interspace midway between the left para-sternal and left mid-clavicular lines, and inferiorly by a line connecting the lower extremities of these two lines. (See Fig. 100, p. 213.)

The area of relative cardiac dullness, representing the portions of the heart overlaid by the anterior borders of the lungs, is bounded on the right by a vertical line drawn upon the chest wall

from the upper border of the third costal cartilage near its junction with the sternum along the right sternal border to the upper border of the sixth costal cartilage, on the left by a slightly curved line having its convexity toward the left and upward, drawn from the third left chondro-sternal junction to the fifth intercostal space one-half inch internal to the mid-clavicular line, and inferiorly by a horizontal line connecting the lower extremities of these two lines, from the upper border of the sixth right costal cartilage to the fifth left interspace one-half inch internal to the mid-clavicular line. Thus these areas roughly represent a triangle within a triangle, the area of absolute cardiac dullness lying within the area of relative cardiac dullness, except inferiorly, where the boundaries are the same, and where the flatness blends with the flatness of the liver. Similarly the right border of the area of relative cardiac dullness joins the upper border of the area of hepatic dullness at almost a right angle, the angle of resonance in the fifth right interspace being termed *Ebstein's cardio-hepatic angle.*

Technic of Cardiac Percussion.—The areas of relative and absolute cardiac dullness, representing the size, shape and position of the heart, may be outlined by ordinary mediate percussion, or by auscultatory percussion. (See Fig. 46, p. 68.)

In mapping out these areas by mediate percussion, the examiner should begin to percuss from three directions in order to fix the upper and lateral boundaries of the region, employing both deep and superficial percussion. In fixing the right and left borders the examiner should percuss from the right and left axillary region upon either side in the third, fourth, and fifth interspaces toward the precordia, employing deep percussion, until impairment of the resonance indicates that the lateral borders of the area of relative cardiac dullness has been reached. Having marked these points upon the thoracic surface, the examiner continues the percussion, substituting superficial percussion for the deep percussion heretofore employed, in order to avoid eliciting the resonance of the anterior borders of the lungs, until the note elicited changes to flatness, indicating that the lateral margins of the area of absolute cardiac dullness have been reached representing the portion of the heart or right ventricle which is in direct contact with the chest wall. Having marked these points upon the chest wall, the examiner beginning in the left infra-clavicular region, percusses downward along the interval between the left sternal and para-sternal lines, employing first deep percussion, and when the upper limit of the area of relative

dullness has been reached and marked, continuing with superficial percussion, until the upper limit of the area of absolute cardiac dullness is attained, which is similarly marked upon the chest wall. By connecting the points marked out upon the chest wall, at which the first change in the percussion note was noted in each instance, the areas of relative and absolute cardiac dullness are graphically represented upon the surface of the thorax. and inferences as to the state of the heart may be drawn therefrom.

In outlining the areas of relative and absolute cardiac dullness by auscultatory percussion, the details of technic of which have been discussed in a previous chapter (see page 68), the chestpiece of the stethoscope should be applied to the chest wall a little above and slightly internal to the cardiac impulse while the examiner percusses toward the heart and marks the tonal changes as they are encountered. Auscultatory percussion is a very reliable method of outlining these areas, but for clinical practice mediate percussion is ordinarily sufficiently accurate.

VARIATIONS IN THE AREAS OF CARDIAC DULLNESS

General Increase.—A general increase in the area of cardiac dullness in all directions is indicative of cardiac hypertrophy and dilatation, pericarditis with effusion, or a tumor of the mediastinal structures which pushes the heart forward. (See Figs. 101 and 102, p. 217.)

General Decrease.—A decrease in all the borders of the area of cardiac dullness is indicative of cardiac atrophy, or pericardial adhesions drawing the heart under the anterior pulmonary borders. Hypertrophic emphysema, by interposing the voluminous anterior borders of the lungs between the heart and chest wall causes a general decrease in the area.

Displacement of the Area, as indicated by displacement of the apex beat occurs in pleurisy with effusion, traction of pleural adhesions, or sub-diaphragmatic pressure. The entire area of the cardiac dullness is displaced, but of normal dimensions.

Upward Increase.—An increase of the area of cardiac dullness in an upward direction accompanies pericarditis with effusion, and in the presence of aneurism of the ascending portion or arch of the aorta. In pericarditis with effusion the area becomes irregularly pear-shaped with its base downward, owing to the characteristic configuration of the pericardial sac.

Increase to Left.—An increase in the area of cardiac dullness toward the left occurs with hypertrophy and dilatation of the

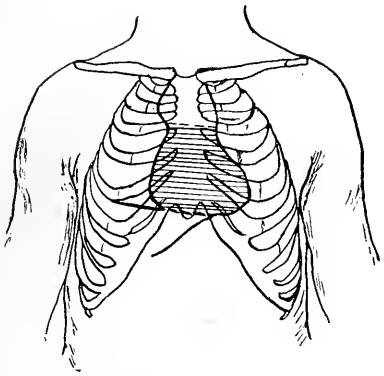


Fig. 101.—Right and left ventricular hypertrophy. (Redrawn from Butler.)

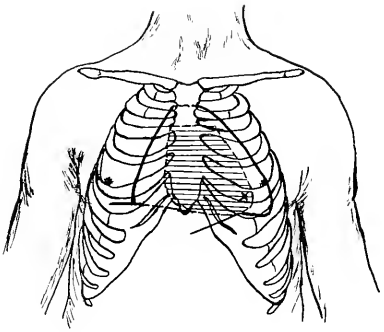


Fig. 102.—Large pericardial effusion. (Redrawn from Butler.)

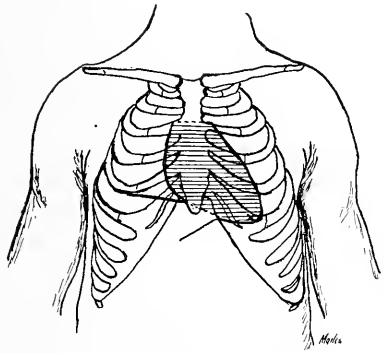


Fig. 103.—Heart in left ventricular hypertrophy. (Redrawn from Butler.)

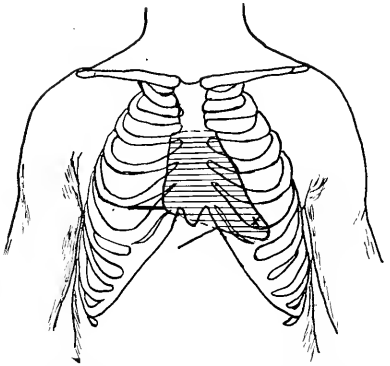


Fig. 104.—Right ventricular hypertrophy. (Redrawn from Butler.)

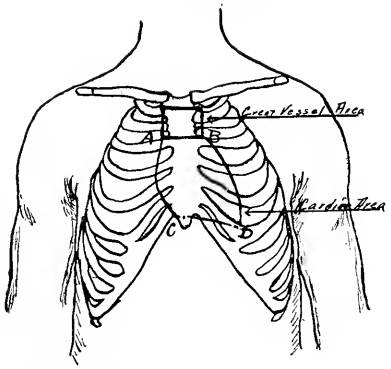


Fig. 105.—Areas of cardiac and vascular dullness. (Redrawn from Butler.)

left ventricle, and in cardiac displacement by mediastinal pressure. In left ventricular hypertrophy the area is increased both to the left and downward.

Increase to Right.—An extension of the area toward the right, the dullness of the heart encroaching upon the normal vesicular resonance of Ebstein's cardio-hepatic angle, occurs with right ventricular and auricular hypertrophy and dilatation, and in pericarditis with effusion, in which disease it constitutes Rotch's sign. A distended inferior vena cava may be responsible for a slight extension of the area of dullness to the right of the sternum. When the extension is the result of right ventricular hypertrophy there are often present epigastric pulsations, systolic in time, while right auricular dilatation is often accompanied by systolic pulsations in the jugular veins.

Vascular Dullness.—An area of dullness extending beyond the margins of the manubrium sterni, particularly if encountered upon the right side in the first or second interspaces, is usually a sign of aortic aneurism.

CHAPTER XVI

AUSCULTATION

Auscultation is utilized in the study of the circulatory organs to determine the character and intensity of the heart sounds, their rhythm, and the presence or absence of certain adventitious sounds arising in the heart, pericardium, and arteries.

The Normal Heart Sounds.—The heart in its action produces two sounds, which are termed the *first* and the *second* sound of the heart respectively. The first sound is heard most clearly as a rule in the region of the cardiac apex, while the second sound is most clearly audible at the base of the heart.

The *first* sound of the heart is lower in pitch and of longer duration than is the second sound. It has been compared to the sound of the word "lubb." It is most probably caused by the combined effect of the closure of the auriculo-ventricular valves, the contraction of the cardiac muscle, and the vibrations of the chordæ tendineæ following coaptation of the valve segments.

The *second* sound of the heart is of higher pitch and of shorter duration than is the first sound, being closely simulated by the sound of the word "dup." The second cardiac sound is unquestionably produced by the closure of the semilunar valves guarding the orifices of the aorta and pulmonary artery. The first sound of the heart is audible during cardiac systole, and is hence termed systolic; whereas the second sound occurs during diastole, and is termed diastolic. In health these two cardiac sounds follow each other in regular sequence or rhythm and are followed by a slight pause.

The cardiac contractions occur approximately 72 times per minute in the healthy adult male subject. However the rate of contraction is influenced by sex and age. In the female subject the rate is often somewhat higher than it is in the male. Age is attended by more definite and distinct variations in the heart rate. Thus at birth the rate is 130 to 140 contractions per minute, diminishing to 90 to 100 at the fifth year of life; while in the aged the rate is 60 to 72 per minute.

Auscultatory Valve Areas.—Each of the four valves of the heart has a corresponding area at which the sound produced by

the closure of the valve in question is more distinctly audible than elsewhere upon the surface of the chest. These areas do not correspond to the point of the thoracic wall which is nearest to the anatomic site of the valve. Thus, the mitral valve is most clearly audible over the *mitral area* which is situated over the apex of the heart, although the anatomical site of this valve is behind the left half of the sternum at the level of the fourth costal cartilage.

The aortic valve is best examined at the *aortic area,* just to the right of the right sternal border in the second intercostal space, although the anatomical site of this valve is posterior to

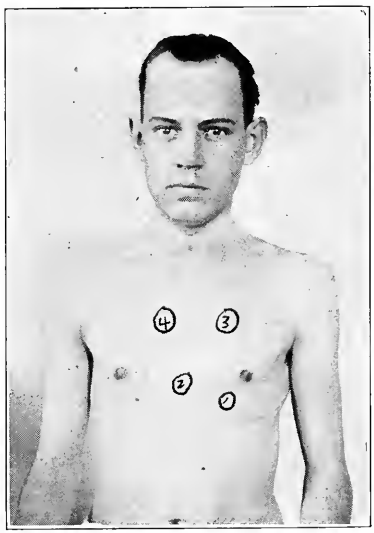


Fig. 106.—Auscultatory valve areas. *1,* mitral area; *2,* tricuspid area; *3,* pulmonary area; *4,* aortic area.

the left half of the sternum at the level of the third interspace.

The *tricuspid area,* at which sounds arising from the tricuspid valve are most distinctly audible, is situated over the lower end of the sternum, the anatomical site of this valve being behind the right portion of the sternum between the level of the fourth and sixth costal cartilages.

The *pulmonary area,* at which sounds generated by the action of the pulmonic valve are best appreciated occupies a point just to the left of the left sternal border in the second intercostal space, the anatomical site of this valve being posterior to the junction of the third left costal cartilage with the sternum.

Upon examination of the cardiac sounds of a normal subject it will be noted that the individual sounds arising at the different valve areas of the heart are not of uniform intensity. Thus, although the first sound of the heart is produced by the combined action of the two auriculo-ventricular valves, it will be observed that the first sound at the mitral area is lower in pitch and of somewhat greater duration than is the tricuspid first sound. Similarly, in examining the component valves concerned in the production of the second sound, it will be observed that in an adult subject the second sound at the aortic area is of greater intensity and duration than is the same sound generated by the pulmonary valve; whereas in a child the condition is reversed, the pulmonary sound being more intense than is the aortic second sound.

VARIATIONS IN INTENSITY OF THE CARDIAC SOUNDS

The intensity of the cardiac sounds as elicited at the various valve areas vary in intensity in different morbid states having their inception in the heart or in distant portions of the body. The intensity of both sounds or of one sound may be increased or diminished at one or more valve areas.

Accentuation of Both Sounds.—Both sounds of the heart are accentuated in the presence of cardiac hypertrophy, in cardiac over-action during exophthalmic goiter, acute febrile diseases, following the ingestion of cardiac stimulants, and as a result of violent physical effort. An apparent accentuation of both sounds is sometimes encountered in subjects with very thin chest walls, and also in patients in whom fibroid retraction of the anterior border of the left lung exposes the heart to the chest wall. A consolidation of the lappet of lung overlying the heart transmits the normal cardiac sounds to the surface of the thorax with exaggerated intensity, simulating a true accentuation of the tones.

Diminished Intensity of Both Sounds.—In robust patients with thick chest walls, in the presence of pericarditis with effusion, when a certain amount of fluid intervenes between the heart and chest wall, and in hypertrophic emphysema when the distended anterior borders of the lungs intervene between the heart and thoracic wall, the cardiac sounds are weakened, without possessing any reference to the state of the myocardium. In car-

diac dilatation, and myocardial degeneration the sounds are weakened from impairment of the integrity of the myocardium.

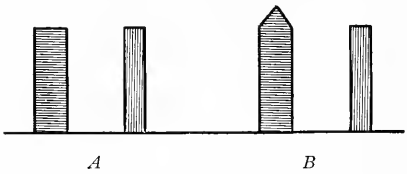


Fig. 107.—*A*. Normal first and second sounds. *B*. Accentuated first sound.

General asthenia from long continued wasting disease causes a weakening of both cardiac sounds.

Accentuation of the First Sound.—An abnormal intensity of the first sound at the mitral area, of slightly increased duration, followed by an abnormally intense aortic second sound is indicative of left ventricular hypertrophy. When dilatation is

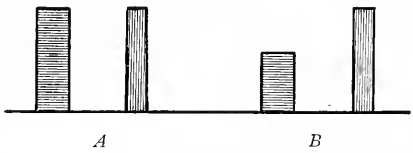


Fig. 108.—*A*. Normal first and second sounds. *B*. Diminished first sound.

about to supervene, the first sound as elicited at the apex is loud but of brief duration and has engrafted upon it the valvular quality of the normal second sound.

Diminished Intensity of the First Sound.—Weakening and degeneration of the myocardium of the ventricle which accompanies cardiac dilatation, the asthenia of chronic wasting disease, anemia, and prolonged fevers leads to a diminution in the

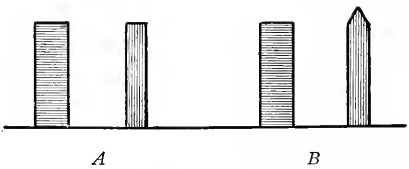


Fig. 109.—*A*. Normal first and second sounds. *B*. Accentuated second sound.

intensity of the first sound at the apex, the sound taking on a valvular quality analogous to that of the normal second sound.

Accentuation of the Aortic Sound.—The aortic sound is accentuated in all states of the body which are accompanied by an

increased tension in the greater circulation. Hence it is encountered in cases of angiospasm due to vaso-constriction, in arterio-sclerosis, chronic interstitial nephritis, uremia, and apoplexy. When the root of the aorta and the semilunar valves participate in general arterio-sclerosis, the aortic sound is accentuated, with a metallic, clinking quality. In hypertrophy of the left ventricle the aortic second sound is accentuated, to become weakened with the inception of dilatation.

Diminished Intensity of the Aortic Sound.—Weakening in the aortic sound accompanies lowering of the blood pressure in the greater circulation incident to hemorrhage, anemia, relaxation of the peripheral arterioles, and when, as a result of mitral or aortic stenosis or insufficiency, a diminished quantity of blood is ejected into the aorta during ventricular systole. Naturally the aortic sound is impaired or weakened in conditions which impair the integrity of the myocardium of the left ventricle as in cardiac dilatation and myocardial degeneration.

Accentuation of the Pulmonic Sound.—Conditions which raise the blood pressure in the pulmonary circulation, as in the case of obstructive pulmonary disease such as cirrhosis of the lung, pneumonia, phthisis, and emphysema, produce accentuation of the second sound at the pulmonary area. Similarly, regurgitant and stenotic lesions at the mitral or aortic valve, by permitting the blood to accumulate in the pulmonary circuit and thus raising blood pressure here lead to a similar accentuation. Tumors or enlarged glands pressing upon the great veins returning the blood from the lungs to the right heart operate in the same manner. In right ventricular hypertrophy from any cause the second sound at the pulmonary area is accentuated.

Diminished Intensity of the Pulmonic Sound.—Weakening or failure of the pulmonic second sound is indicative of failure of the right side of the heart. When a pulmonic sound which has been accentuated becomes weak, it indicates right ventricular dilatation or the development of tricuspid regurgitation.

Reduplication of the Heart Sounds.—Either or both sounds of the heart may under certain circumstances become doubled or reduplicated. Ordinarily the examiner encounters a reduplication of either the first or second sound, rarely of both. If the first sound is reduplicated the sound resembles the words "lur-rup-dup," whereas if the second sound is reduplicated alone, the sound resembles the spoken words "lub-durrup." In certain instances these sounds are so accented as to resemble the

gallop of a horse, under which circumstances the sound has been termed "gallop-rhythm" or "canter-rhythm," or the "bruit de galop."

Reduplication of the first sound of the heart is only rarely encountered, and the mechanism of its production is beset with difficulties. It has been asserted that the reduplication is due to a-synchronous closure of the mitral and tricuspid valves, owing to a-synchronous systole of the right and left ventricles. The phenomenon has also been referred to unequal tension of the

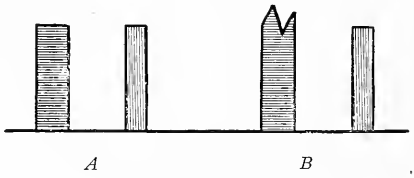


Fig. 110.—*A*. Normal first and second sounds. *B*. Reduplicated first sound.

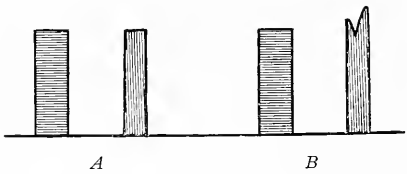


Fig. 111.—*A*. Normal first and second sounds. *B*. Reduplicated and accentuated second sound.

leaflets of the two auriculo-ventricular valves. The sound produced by reduplication of the first sound of the heart is often confused with a pre-systolic mitral murmur which is followed by a normal first sound. Reduplication of the first sound may be a sign of mitral stenosis, or it may develop on account of lesions of the auriculo-ventricular valves during failure of compensation.

Reduplication of the second sound of the heart is to be attributed to a-synchronous closure of the aortic and pulmonary valves as a result of unequal tension in the greater and lesser circulations. Hence, a reduplication of the sound is noted in all states which raise the pulmonary blood pressure, as in emphysema, cirrhosis of the lung, pneumonia, and left sided valvular heart disease; and in association with arterio-sclerosis and chronic interstitial nephritis, raising the pressure in the general circulation. Reduplication of the second sound has been noted in normal subjects during deep inspiration.

CARDIAC ARRHYTHMIA

Clinical Attributes of the Myocardium.—For a proper appreciation of the subject of cardiac arrhythmia certain inherent properties or clinical attributes of cardiac muscle per se must be considered. These properties or attributes comprise rhythmicity, tonicity, irritability, conductivity and automaticity. It must also be borne in mind that the cardiac contractions are always maximal, regardless of the strength of the stimulus which calls the contraction into being.

Throughout life the heart continues a series of rhythmical contractions, in health ventricular systole and diastole following similar phenomena in the auricles in regular and undisturbed rhythm. This rhythmicity involves both the strength and the time of the contractions. This series of contractions and relaxations, separated by definite and regular periods of repose constitute the cardiac cycle. Assuming that the regular series of events occurs 72 times per minute the time occupied by each of the elements of the cycle may be taken to be as follows: The entire cycle consumes 0.8 of a second. The contraction of the auricles consumes 0.1 of a second; the contraction of the ventricles, 0.3 of a second; and the period of repose of the entire heart 0.4 of a second.

The heart constantly remains in a state of partial contraction, constituting the *tone* of the organ. Possessed by all portions of the myocardium the widest variations in tonicity are encountered in the auricles, in which Botazzi has demonstrated that the variations of tone are periodic and wave-like.

The myocardium possesses an inherent *irritability* to stimuli reaching it from without, the degree of irritability varying in different portions of the organ. The irritability is markedly influenced by the state of nutrition of the cardiac walls. Unlike muscle from other organs or portions of the body, the strength of the response of the myocardium to a stimulus which is applied bears no definite relation to the degree of stimulation; but the response is maximal to all stimuli, strong or weak. Moreover, after a response has been excited by a stimulus reaching the heart, there is no response to a second stimulus reaching it after the initial response is started until its completion, the so-called Refractory Period. If, however, a contraction be excited earlier than it would automatically occur, the succeeding pause before

the next regular contraction is longer, constituting the so-called Compensatory Pause, a very important point for consideration in the study of arrhythmia of the extra-systolic type.

The myocardium possesses the property of conducting an impulse arising in the auricles to the ventricles by means of a specialized muscular bundle, the auriculo-ventricular bundle of His. The specialized fibers constituting this conducting bundle arise in the wall of the right auricle and interauricular septum, whence branches are sent upward as far as the great veins. The bundle passes downward to the ventricular septum and there divides, sending branches to each ventricle, which terminate in the Purkinje cells. By way of this bundle the contractions which are rhythmically initiated by the sinus node are transmitted downward over the auricles and ventricles, initiating the regular and rhythmical contractions of these chambers.

Erlanger has shown that by clamping the bundle of His the mammalian heart will exhibit the phenomenon of heart-block, which constitutes an occasional cause of arrhythmia.

The myocardium possesses the power of automatically contracting in regular rhythm, the automaticity residing in the myocardium whether it be assumed to be of neurogenic or myogenic character.

The cardiac action is influenced by and regulated by its nerve supply from the cerebro-spinal and sympathetic nervous system. Thus it is seen that arrhythmia may be due to alterations in the myocardium or to alterations in the nervous control of the heart; and that the arrhythmia may appertain to either the force of the contractions or to the time sequence of the contractions.

Types of Arrhythmia

Simple Arrhythmia.—This, the most common type of cardiac arrhythmia presents variations both in the frequency and force of the cardiac contractions. When the force of the contractions is affected the result clinically is the pulsus alternans. When the rhythm is the predominant feature influenced, it results in variations from the normal rhythm of the cardiac action, which in certain instances may be rhythmically irregular, producing the pulsus bigeminus or the pulsus trigeminus.

Intermittence.—In certain cases of cardiac arrhythmia the principal feature is the occasional omission of a contraction. In such cases it should be determined whether the omission

of a radial pulse beat is due to the absence or omission of a ventricular systole or whether due to a ventricular systole which is too feeble to produce a radial pulse.

Respiratory Arrhythmia.—An arrhythmia noticeable during expiration often develops as a post-febrile condition. During full inspiration, on the contrary, the pulse may be noticed to become progressively weaker and may become quite imperceptible at the wrist, the Paradoxical Pulse. This is noted in many young persons, and also occurs in sero-fibrinous and chronic adhesive pericarditis.

Tachycardia (rapid heart) accompanies mental and emotional disturbances, after violent physical exertion, during acute fevers, following the ingestion of large amounts of tea or coffee, or the excessive use of tobacco, and during various cardiac neuroses. The cardiac contractions become very rapid, but the patient is often not aware of the presence of the condition. Tachycardia is also noted in cases of exophthalmic goiter and when cardiac failure is imminent.

Paroxysmal Tachycardia, first described by Proebsting, is a form of rapid cardiac action characterized by the occurrence of paroxysms of very rapid heart action, beginning very abruptly, persisting for a variable length of time, and ceasing as abruptly as it began. The rhythm of the heart's action is rather markedly affected at the commencement and termination of the paroxysm. The rate may go above 200 beats to the minute. Usually brief, lasting a few seconds or minutes, the attack may persist for hours or days.

Palpitation.—Palpitation is a form of arrhythmia affecting chiefly the force of the cardiac contractions, though attended usually by disturbances of the rhythm of the heart. The principal feature of the condition is that the subject is painfully conscious of the presence of the tumultuous and irregular heart action. Palpitation is often noted at the onset of menstruation and at the climacterium, following excessive indulgence in coffee, tea, or tobacco, during attacks of indigestion, in neurasthenic patients, and in cases of threatened cardiac failure.

Bradycardia (Slow Heart).—In bradycardia the heart's action is abnormally slow, sometimes less than 50 beats to the minute. It is a normal phenomenon in some subjects; it occurs during convalescence from certain febrile diseases, notably after typhoid fever. It is also an accompaniment of increased intracranial tension in meningitus, cerebral abscess, or depressed fracture of

the skull. It forms a prominent feature of Stokes-Adams Disease, a syndrome in which the auricles and ventricles contract each with an independent rhythm, the ventricular contractions being greatly diminished in frequency. Associated signs comprise Cheyne-Stokes Respiration, apoplectiform seizures, unconsciousness, and transient paralyses.

Heart Block.—Heart block, due to diminution of the conductivity of the auriculo-ventricular bundle of His, may be partial or complete. In partial heart block only a portion of the auricular contraction waves are conducted to the ventricles, resulting in a diminution of the number of ventricular systoles, the ventricle contracting only with every second, third, or fourth systole of the auricle.

In complete heart block none of the auricular contraction waves are conducted to the ventricle with the result that the auricles and ventricles contract independently and rhythmically.

Extra-Systolic Arrhythmia.—Under certain conditions which are but imperfectly understood the auricular or ventricular systole is preceded by an extra-systole. As stated, the heart always contracts with maximum force regardless of the strength of the stimulus exciting the contraction; and a stimulus reaching the auricle or ventricle between the reception of the primary stimulus and the completion of the consequent contraction is without effect upon the myocardium, the so-called refractory period of the heart. When under these conditions an extra-systole is injected into the cardiac cycle, causing a diminution in the physiolotic refractory period, then the succeeding pause preceding the next regular systole is increased, the so-called compensatory pause. This gives rise to an apparent omission of the next regular ventricular contraction and produces arrhythmia, which may in certain instances be allorhythmic. The clinical significance of this type of arrhythmia is little understood; but it has not been demonstrated that it is indicative of any grave change in the myocardium.

Embryocardia.—In the course of prolonged continuous fevers, and in the presence of myocardial disease with imperfect compensation or advanced arterial sclerosis when cardiac failure is imminent, the rhythm of the cardiac action is altered, approaching the tones of the fetal heart. In this condition the first and second sounds approach each other in force and duration, and are separated by pauses of equal duration owing to prolongation of the first period of repose between auricular and ventricular

systole, and shortening of the second period of repose following ventricular systole.

Auricular Fibrillation.—In certain late cases of mitral stenosis the regular auricular systole is replaced by a series of incoordinated contractions, the different muscular bundles contracting independently instead of in unison with the result that the auricle remains in a state of diastole, permitting a systolic venous pulse to be registered in the jugular vein. This produces a type of arrhythmia which is so absolutely irregular as to defy description. The term delirium cordis has been applied to describe the condition.

Adventitious Sounds.—Adventitious sounds are abnormal sounds originating within the heart (endocardial); or outside the heart in the pericardium, lung, pleura, or vessels (exocardial).

ENDOCARDIAL MURMURS

Murmurs are abnormal sounds, arising within the chambers of the heart, which may be superadded to the normal cardiac sounds, or may entirely replace these sounds. The manner of

Fig. 112.—Physical basis of murmurs due to diminution of lumen. (From Cabot.)

generation of endocardial murmurs may be explained upon certain physical principles. They are produced by irregularities in the movement of the blood through the chambers and orifices of the heart by virtue of which the blood is set in vibrations, which when transmitted through the thoracic wall are audible as murmurs and palpable as thrills. As long as normal blood passes through a normal heart with normal endocardium and normal valves no sound is generated save the sound normally produced by the closure or co-aptation of the valve segments. But when the blood is forced through a narrowed or stenosed orifice into the wider chamber beyond, or when the blood is permitted by an incompetent auriculo-ventricular valve to regurgitate into an auricle the blood column is whipped into eddies the so-called "fluid veins" which throw the blood into rapid vibrations which are transmitted through the stethoscope to the ear as an appreciable sound.

The sound or murmur is ordinarily propagated in the direc-

tion of the fluid veins. Thus, in the case of the murmur of aortic stenosis the murmur is transmitted in the direction of the blood current, namely upward into the carotid arteries; whereas in the case of the murmur of aortic regurgitation the murmur is transmitted in the direction of the regurgitating blood stream, namely downward and toward the left.

The density of the blood influences the generation of murmurs. The thinner the blood, the greater the ease with which fluid veins or vibrations are produced in it, which serves to explain the great frequency of murmurs occurring in anemic states.

Moreover, a certain degree of blood pressure or endocardial pressure is essential to the generation of a murmur. This is evidenced by the fact that endocardial murmurs remain distinct and strong as long as cardiac compensation is maintained, to become indistinct or lost with cardiac dilatation.

Characteristics of Endocardial Murmurs

Endocardial murmurs possess certain characteristics or properties which are and remain characteristic of the murmurs arising at the several valves of the heart, and by a study of which the murmurs may be distinguished and their site of production may be determined.

Point of Maximum Intensity.—Every endocardiac murmur has a point of maximum intensity, the point at which it is most clearly audible upon the chest wall. These points correspond very closely as a rule with the points at which the closure of the normal valve in question is best heard, that is, in the four acoustic valve areas. Thus, a murmur which is generated at the mitral valve is usually heard with the greatest intensity at the mitral area, over the apex of the heart, whereas a murmur produced at the aortic valve is most clearly audible at the aortic valve area below the second right costal cartilage near the right sternal margin.

This selective transmission of the sound in the case of the different murmurs to a particular area of the thorax is accounted for by the fact that the sound is most likely to travel in the direction of the fluid vein, and by the difference in the conductivity of the component portions of the chest wall, and the distance of the cavity in which the murmur is generated from the chest wall.

Line of Transmission.—Most organic endocardiac murmurs are audible not only at their points of maximum intensity, but are transmitted or propagated thence in directions which vary in the individual murmurs, the line of transmission, or line of propagation of the murmur. The direction in which a given murmur is to be transmitted is determined by the direction of the blood current, and the relative conductivity of the adjacent thoracic structures.

Time of Murmurs.—Every murmur bears a definite relation to the events of the cardiac cycle. A murmur which is audible during systole is termed a *systolic murmur;* while one developing and audible during diastole is designated a *diastolic murmur.* A murmur which is audible just prior to systole is termed a *pre-systolic murmur.*

The Quality of Murmurs.—Murmurs are described as harsh and rasping, or as soft, blowing, and musical. The quality of a murmur possesses diagnostic significance and should in all instances be studied. As a general rule a harsh unmusical murmur accompanies stenotic lesions, while soft musical or blowing murmurs characterize regurgitant lesions of the valves. While studying the quality of the murmur the examiner should endeavor to determine whether the murmur is followed by the normal cardiac sound or whether it entirely replaces this sound. A murmur which merely accompanies or is added to the normal cardiac sound is not of as grave prognostic significance as is a murmur which entirely displaces the sound.

Intensity of the Murmur.—Just as a certain degree of endocardial pressure is essential to the development of a murmur, the intensity of a murmur is a good index to the endocardiac pressure and hence of the state of the myocardium. Thus, a loud murmur suggests the presence of cardiac hypertrophy, while a faintly audible murmur is very suggestive of cardiac dilatation. Moreover, a change in the intensity of a murmur during daily examinations affords an index to the reserve power of the heart, a change from a loud to a soft faint murmur suggesting a failing heart, while a steady increase in the intensity of a murmur from day to day is suggestive of cardiac improvement.

MITRAL MURMURS

Murmurs arising at the mitral valve are pre-systolic or systolic, as they are audible just prior to or during ventricular systole.

Mitral Pre-Systolic Murmur.—A pre-systolic murmur at the mitral area is indicative of mitral stenosis, the narrowing of the orifice whipping the blood stream into fluid veins which produce a murmur which is audible just prior to the first sound of the heart. The murmur is commonly followed by a sharp first sound; but as mitral stenosis and insufficiency frequently coexist, the regurgitant murmur frequently masks or impairs the first sound at the apex. The point of maximum intensity of the murmur is located at the mitral area over the cardiac apex; whence it is not transmitted. The mitral pre-systolic murmur is loud and harsh, crescendo in quality, increasing in intensity to

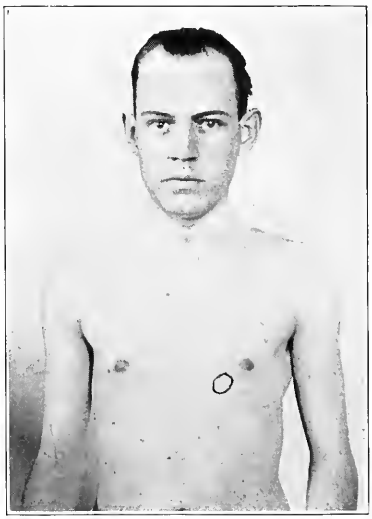


Fig. 113.—Point of maximum intensity of the mitral pre-systolic murmur.

its abrupt termination usually in a sharp first sound. The murmur is quite constantly accompanied by a thrill. The pulmonic second sound is accentuated as a result of increased pressure in the pulmonary circuit.

The murmur of mitral stenosis must be differentiated from the *Flint murmur,* which is also audible in the mitral area in cases of aortic regurgitation. The manner of production of this murmur is a matter of dispute, but the usual explanation is that in this disease the aortic cusp of the mitral valves becomes the target for two streams of blood, entering the ventricle from opposite directions, one entering from the left auricle the other regurgitating from the aorta, and is thereby thrown into vibra-

tions, which are audible as a late diastolic or pre-systolic murmur. The Flint murmur has its point of maximum intensity at the mitral area, is audible during late diastole or just prior to systole; is not transmitted therefrom; but it has not the ingravescent or crescendo quality of the mitral stenotic murmur; it is not followed by a snappy first sound; it is not accompanied by a thrill; and has associated with it other signs of aortic regurgitation, as pulsations in the arteries of the neck, the water-hammer or Corrigan pulse, and the capillary pulse of Quincke.

Mitral Systolic Murmur.—A mitral systolic murmur indicates incompetence or insufficiency of the mitral valve, due to organic

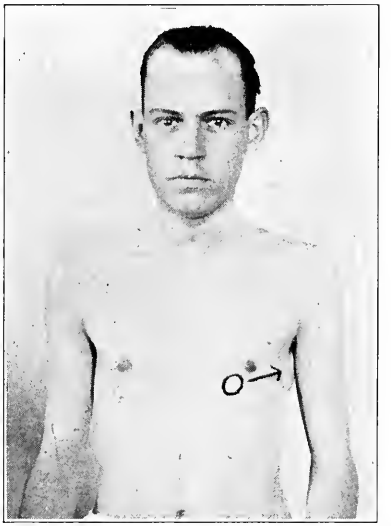


Fig 114.—Point of maximum intensity and line of transmission of mitral systolic murmur.

deformity of the valve segments, or stretching of the mitral ring, as a result of which the edges of the cusps cannot be brought into close co-aptation during ventricular systole, and reflux of blood occurs into the left auricle. The point of maximum intensity of the murmur is situated at the mitral area over the cardiac apex, whence it is transmitted toward the left axillary region, not infrequently as far as the angle of the scapula. The murmur occurs during ventricular systole, masking or replacing the first sound of the heart at the apex. In quality the murmur is soft and sometimes musical, or blowing, and of low pitch. The intensity of the mitral systolic murmur varies

with the state of the ventricular musculature, remaining strong as long as compensation is maintained, becoming weak or disappearing when dilatation supervenes.

The pulmonic second sound is accentuated owing to right ventricular hypertrophy or raised arterial pressure in the pulmonary circulation; and, in long-standing cases with organic change in the mitral valve, a safety-valve leak develops at the tricuspid valve.

The murmur of mitral regurgitation is less constantly accompanied by a thrill than is the murmur of stenosis of this valve.

AORTIC MURMURS

Murmurs arising at the aortic orifice are systolic and diastolic, as they are occasioned by an obstruction to the free flow of blood from the ventricle into the aorta during ventricular systole, or by lesions of the valve which, by impairment of its integrity, permit a portion of the blood expelled during systole to regurgitate into the ventricle during diastole.

Aortic Systolic Murmur.—A systolic murmur at the aortic valve area is usually indicative of an obstacle to the passage of blood into the aorta during ventricular systole. This obstruction is usually a sequence of ulcerative endocarditis, which causes adhesions to form between the edges of the cusps, thus narrowing the orifice. A similar murmur may be caused by relative stenosis, in which event the aortic ring and cusps are normal, but there is a dilatation or aneurism of the aorta immediately beyond the valve. A systolic murmur at the aortic area may be indicative of aortic roughening.

The murmur is most clearly heard at the aortic area in the second right interspace close to the sternum, whence it is transmitted upward into the great vessels of the neck. The murmur develops during ventricular systole, and may or may not be followed by a clear second sound. In murmurs due to deformed valve cusps the second sound is usually replaced by a diastolic murmur due to aortic regurgitation; but in cases in which the systolic murmur is caused by dilatation or roughening of the aortic wall the second sound is clear.

The murmur is loud, harsh, and unmusical, the intensity depending upon the degree of contractile power of the ventricular musculature.

Aortic Diastolic Murmur.—A diastolic murmur at the aortic

area indicates aortic insufficiency or Corrigan's disease. The underlying lesion may be a shrinking and thickening of the cusps, rupture or perforation of a cusp, or the presence of warty vegetations upon the cusps which prevent their close and accurate co-aptation during diastole. Or again, the murmur may arise as the result of the inability of normal valve cusps to close an abnormally large aortic orifice (relative insufficiency).

The murmur is most distinctly audible at the aortic area, being propagated therefrom downward along the right border of the sternum. While this murmur is usually most intense at the aortic area, in certain instances it is heard most clearly over the

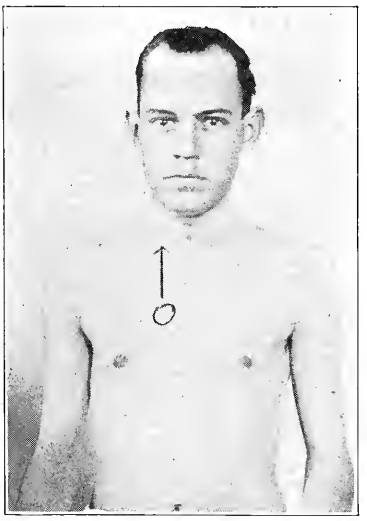


Fig. 115.—Point of maximum intensity and line of transmission of aortic systolic murmur.

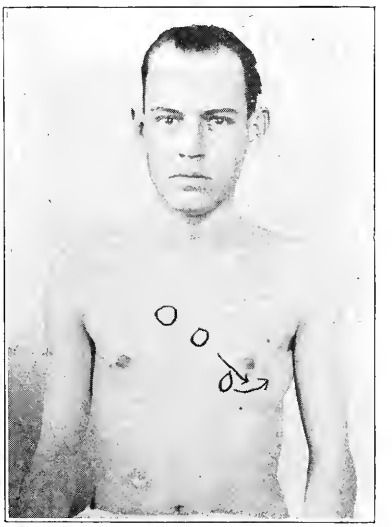


Fig. 116.—Points of maximum intensity and lines of transmission of aortic diastolic murmur.

gladiolus, just below the Angle of Louis, or over the cardiac apex, whence it is transmitted downward and toward the left axilla. The murmur occurs during diastole, masking or replacing the second sound at the aortic area. The murmur is loud and blowing but not harsh or unmusical.

While the diastolic murmur of aortic insufficiency may occur alone, it is not infrequently accompanied by a systolic aortic murmur, due to aortic stenosis, a deformity of the aortic valves underlying both conditions and causing both stenosis and insufficiency. In such event there is a double murmur generated at the aortic valve, harsh during systole, and less so during diastole.

Such a double murmur must not be confused with a pericardial friction rub with its to-and-fro rhythm.

TRICUSPID MURMURS

Murmurs at the tricuspid area are infrequent; but when present they are pre-systolic and systolic.

Tricuspid Pre-systolic Murmur.—A pre-systolic murmur arising at the tricuspid valve is indicative of tricuspid stenosis. The lesion is usually a congenital condition, very rarely encountered in an acquired form.

The murmur is best appreciated at the tricuspid valve area

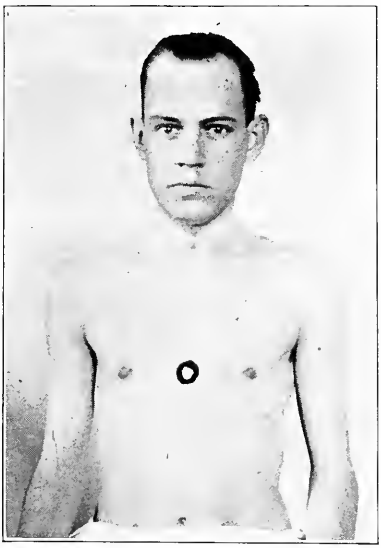


Fig. 117.—Point of maximum intensity of tricuspid pre-systolic murmur.

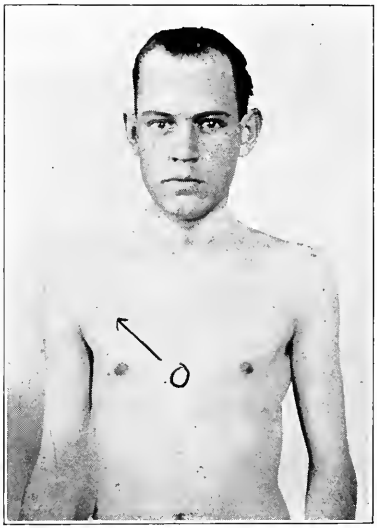


Fig. 118.—Point of maximum intensity and line of transmission of tricuspid systolic murmur.

over the lower portion of the sternum, whence it is not transmitted, In quality, time, and intensity it is the counterpart of the murmur of mitral stenosis. The murmur is usually associated with a thrill over the tricuspid area together with an enfeebled pulmonic second sound and dyspnea.

Tricuspid Systolic Murmur.—A systolic murmur at the tricuspid area occurs with tricuspid regurgitation. The causative lesion may be a deformity of the cusps of the valve the sequence of acute endocarditis. More commonly a systolic murmur at this valve is relative, the result of increased blood pressure in the

right ventricle and pulmonary circulation the sequence of an obstructive disease of the lung or mitral insufficiency.

This murmur is best heard at the tricuspid area, whence it is transmitted upward and toward the right. It corresponds to the systole of the ventricle, replacing or masking the first sound at the tricuspid area. It is a soft, blowing murmur of moderate intensity. It is accompanied by systolic pulsation in the jugular veins and pulsation of the liver.

PULMONARY MURMURS

Murmurs at the pulmonary area are of very frequent occurrence, but organic disease of this valve is very rare. Most of

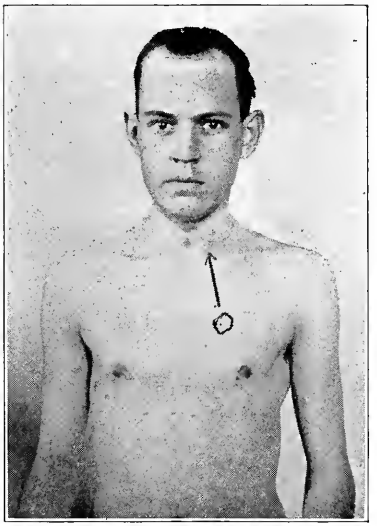


Fig. 119.—Point of maximum intensity and line of transmission of pulmonic systolic murmur.

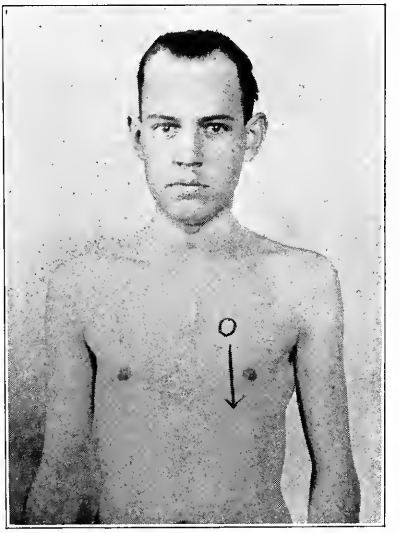


Fig. 120.—Point of maximum intensity and line of transmission of pulmonic diastolic murmur.

these murmurs are functional, and will be discussed in a subsequent section. The murmurs generated at the pulmonic valve are systolic and diastolic.

Systolic Pulmonic Murmur.—A systolic murmur at the pulmonary valve is rarely encountered and when it is found, it signifies pulmonary stenosis, a congenital lesion. The murmur occurs during ventricular systole, is most intense over the pulmonic area in the second left interspace adjacent to the sternum, is harsh and unmusical and is transmitted upward into the root of the neck.

Diastolic Pulmonic Murmur.—A diastolic murmur at this valve represents pulmonary regurgitation, due to alteration in the integrity of the cusps of the valve, incident to acute endocarditis or due to dilatation of the orifice so that the normal cusps cannot close the opening (relative insufficiency).

The point of maximum intensity of the murmur is situated at the pulmonic area, whence the murmur is propagated downward along the left sternal border. The murmur occurs during diastole, masking or replacing the second sound at the pulmonic area. The murmur in quality resembles the murmur of aortic regurgitation, being not unmusical and soft.

FUNCTIONAL MURMURS

Functional murmurs, also known as inorganic, accidental, or hemic murmurs, are endocardiac murmurs arising in a heart which is not the seat of any structural change. Functional murmurs are not caused by valvular deformity, but are due to excessive fluidity of the blood incident to anemia, to temporary myocardial weakness during the course of acute fevers, to cardiac neuroses, or to stretching of the valvular orifices due to great physical exertion. These murmurs are audible most frequently at the pulmonic area, and least frequently at the aortic area. They are more common at the mitral than at the tricuspid area.

Functional murmurs are transient, coming and going, but not persisting for any great length of time. They are always systolic and are not transmitted beyond the precordia. They are encountered most frequently in association with states of anemia and asthenia. Functional murmurs are soft and of low pitch; and when due to anemia are often accompanied by the humming top murmur in the jugular veins.

MULTIPLE MURMURS AND THEIR DIAGNOSIS

While the organic and functional murmurs arising at the various valves and orifices of the heart are separate entities, it is to be borne in mind that two or more of these murmurs may coexist in the same patient, the differentiation of which is often attended with not a little difficulty.

When two distinct murmurs are encountered at two heart valves as for instance at the aortic and mitral valves, the mere presence of two murmurs at two valves does not necessarily sig-

nify organic disease of both valves, as one murmur may be relative on account of the dilatation of the left ventricle as the result of the aortic lesion. The mitral murmur in this instance is functional, and the aortic murmur is organic. Similarly, an organic mitral insufficiency is frequently followed by a functional tricuspid regurgitation.

An accurate diagnosis of multiple murmurs is difficult. When two murmurs occur at two phases of the cardiac cycle, one systolic, and the other diastolic, this fact is of great assistance. When two murmurs at the same period of the cardiac cycle are discovered, the differentiation must rest largely upon the points of maximum intensity and the lines of transmission of the different murmurs. The quality of the murmurs is of some assistance, remembering the general rule that stenotic murmurs are harsh, while regurgitant murmurs are usually musical. If, in the case of two murmurs one be harsh and the other musical, there are certainly two murmurs. If, on the contrary, both are alike in quality, it is possible that there is only one murmur, which is transmitted from the orifice where it is produced to a second orifice.

Moreover, murmurs arising in the heart must be differentiated from a possible cardio-respiratory murmur by directing the patient to suspend respiration, whereupon the latter will disappear.

Finally, in the differentiation of multiple murmurs the auscultatory findings must be correlated with the general appearance of the patient, and such accessory signs as edema, dyspnea, and cyanosis.

Cardio-Respiratory Murmur.—The cardio-respiratory murmur is a systolic blow or whiff audible in the mitral area over the cardiac apex, closely simulating an endocardiac murmur. The sound is produced by the impulse of the heart against a portion of lung anchored in front of the heart by a pleural adhesion or hypertrophic emphysema. The sudden expulsion of the air from the portion of lung by the impact of the heart generates a sound closely simulating an endocardial murmur.

Pericardial Friction.—During inflammations of the pericardium the surfaces of the visceral and parietal membranes, which glide noiselessly over each other in health, become roughened and produce a friction sound, which in some instances closely simulates an endocardial murmur. The pericardial friction sound has a to-and-fro rhythm dependent upon the contractions of the

heart, which are not precisely synchronous with the heart sounds as are endocardial murmurs, but last longer than do the heart sounds. The sound is very variable, being exaggerated by moderate pressure with the stethoscope and being abolished by firm pressure with this instrument. It is transient, perhaps present at one examination and absent a few hours later. The rub is altered by change of posture, often disappearing when the patient assumes the dorsal decubitus, to reappear upon his return to the sitting posture. (See Fig. 94, p. 203.)

The pericardial friction sound is audible all over the precordia, but is most intense in the fourth interspace to the left of the sternum. The two phases of the sound are of equal intensity, but not of equal duration. The sound seems very superficial, and is not abolished upon suspension of respiration as is the pleural friction rub.

In pericarditis with effusion, as the fluid accumulates in the pericardial sac the friction rub usually disappears, though it is not uncommon for it to persist at the base of the heart.

Pericardial Succussion Sound.—Splashing or succussion sounds arising within the pericardium indicate the presence of air and fluid, or hydro-pneumo-pericardium. The sounds may be splashing, bubbling, or gurgling, and have been compared to the sound produced by a water-wheel. They are not abolished during suspension of respiration.

VASCULAR MURMURS

Arterial Murmurs.—Auscultation may reveal the presence of murmurs in the aorta, the carotids, subclavian, brachial and femoral arteries. In auscultation of an artery the examiner should apply the stethoscope over the vessel lightly but firmly, yet without exerting sufficient pressure to diminish the lumen of the vessel. He should gradually then apply sufficient pressure to partially occlude the vessel. During the first maneuver vascular phenomena may or may not be elicited. In the second examination even in a normal artery, a systolic murmur will be generated by the partial occlusion of the vessel. This murmur is produced by vibrations caused by "fluid veins" which are initiated by vibrations set up by the passage of the blood through the constricted portion of the vessel into the wider portion beyond.

The Aorta.—Upon auscultation of the aorta in the left interscapular region a systolic murmur in the vessel is a sign of aneu-

rism of the aorta. The murmur is accompanied by concomitant signs of aneurism; namely, dullness on percussion, a palpable thrill, pulsation of the chest wall in many instances, and pressure symptoms. Frequently there is tracheal tugging, or Oliver's sign.

The Carotids.—Upon auscultation of the carotid artery the first and second sounds of the heart may sometimes be audible, although the first sound is often absent. These sounds are not to be confounded with murmurs; they are merely the normal sounds of the heart transmitted along the course of the circulating blood. Endocardiac murmurs are similarly transmitted, a harsh systolic murmur audible over the vessel signifying aortic stenosis, aortic roughening, or aneurism of the arch. The transmitted second sound of the heart may be replaced by the diastolic murmur of aortic regurgitation.

The Subclavian Artery.—In certain cases of apical pulmonary tuberculosis a systolic murmur is audible in the subclavian artery. The murmur in this instance is due to constriction of or bending of the lumen of the vessel by the traction of adhesions. A diastolic murmur is occasionally audible in the subclavian artery in aortic regurgitation or Corrigan's disease.

The Femoral Artery.—In many cases of aortic regurgitation a double murmur, systolic and diastolic, may be elicited by auscultation over the femoral artery, the systolic murmur resulting from the sudden injection of blood into the artery, and the diastolic murmur resulting from the reflux of the blood stream which the incompetent aortic valve is incapable of sustaining in the aorta. This double murmur in the femoral artery constitutes Duroziez's sign of aortic regurgitation or Corrigan's disease.

Venous Murmurs.—A continuous murmur may be elicited over the jugular vein in health by tightly applying the stethoscope to the vein. A similar murmur may sometimes be generated by turning the head far to one side. Hence, in auscultation of this vessel the stethoscope should be lightly applied and the head maintained in a symmetric and unconstrained position.

Venous Hum.—The principal diagnostic sign afforded by auscultation of the venous system is the venous hum, humming-top murmur, nun's murmur, or bruit de diable. This murmur is usually to be elicited over both jugulars, but is more intense over the right vein. The murmur is usually loudest or most intense just at the inner third of the clavicle. It is more intense when the patient sits upright, during inspiration, and diastole, factors which increase the rapidity of the flow of blood through the

veins. The mechanism of production of this murmur has been disputed. Occasionally audible in normal subjects, it is almost pathognomonic of anemia, particularly chlorosis and pernicious anemia.

In these conditions the increased fluidity of the blood possibly plays a leading role in the production of the murmur; and, moreover, the diminished nutrition of the vascular wall permits relaxation which favors vibration.

BLOOD PRESSURE

Definition.—It is obvious that for the maintenance of a constant flow of blood through the vessels a certain degree of force must be exerted upon the blood column. This force, which governs the onward course of the blood, constitutes blood pressure, and is derived from three principal sources.

At each contraction of the left ventricle a variable quantity, from 80 to 100 c.c. of blood is forced into the aorta. But this volume of blood is not injected into an empty vessel. The aorta is filled with blood at the commencement of ventricular systole, as not sufficient time elapses between the ventricular systoles for the blood to flow from the large arteries into the capillaries and veins. As a consequence, when the systolic discharge of blood from the ventricle occurs, the walls of the large arteries yield or are stretched by virtue of the elastic elements which they contain. As a result of this elasticity, the vascular walls immediately contract upon the contents of the vessels and force the blood column onward.

However, the flow of the blood through the vessels encounters a distinct resistance to its onward progress, when the capillaries are reached, an obstacle to which the term "peripheral resistance" is applied. When it is recalled that the sectional area of the capillaries is many hundreds of times as great as that of the larger arteries, it is obvious that a considerable degree of friction is generated by the passage of the blood through these minute vessels.

Moreover, the arterioles, the immediate precursors of the capillaries, are supplied with a special nervous mechanism through the vaso-motor nervous system, whereby the calibre of these vessels may be constricted or dilated, thus producing variations in the degree of peripheral resistance.

Thus, the term *blood pressure* refers to the interaction of these

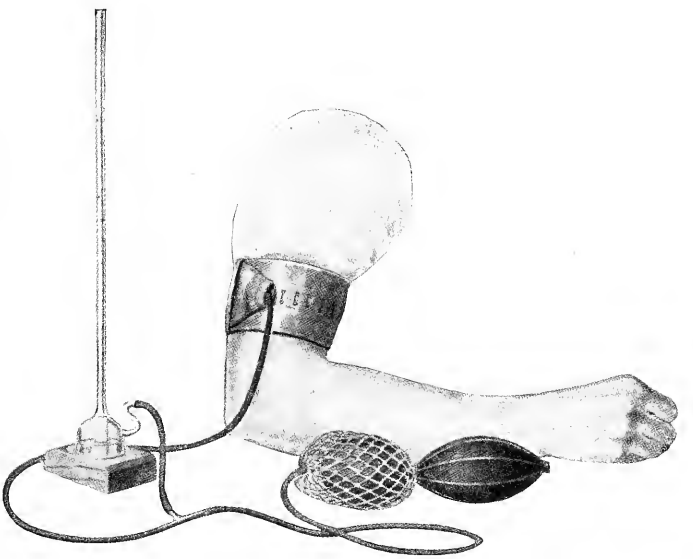


Fig. 121.—Cook's modification of Riva-Rocci's blood pressure instrument. (From Warfield.)

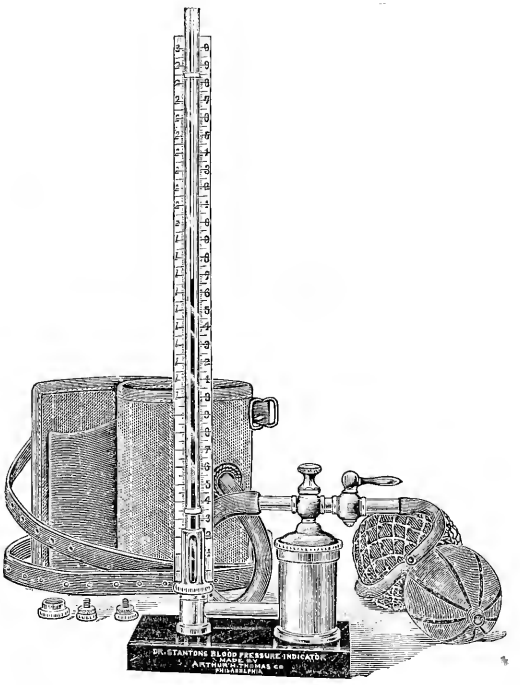


Fig. 122.—Stanton's sphygmomanometer. (From Warfield.)

three factors (ventricular contraction, elasticity of the arteries, and peripheral resistance), and clinically it represents the total pressure exerted by the heart and blood vessels.

Determination of Blood Pressure.—Clinically it is desirable to

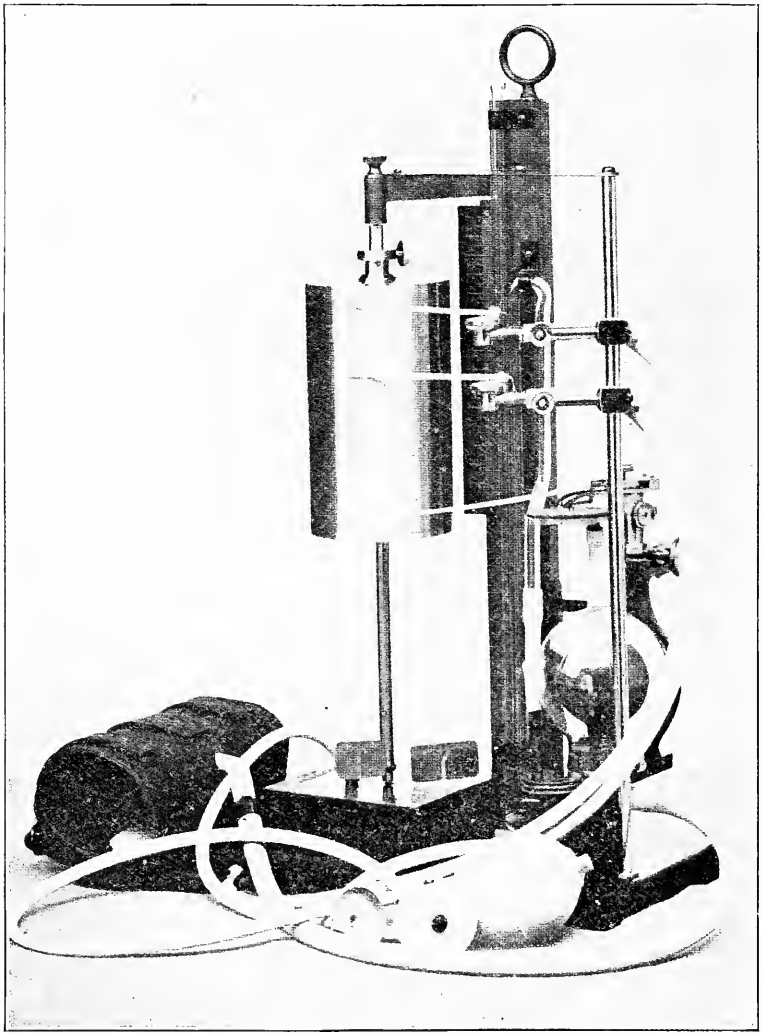


Fig. 123.—The Erlanger sphygmomanometer with the Hirschfelder attachments by means of which simultaneous tracings can be obtained from the brachial, carotid, and venous pulses. (From Warfield.)

determine the blood pressure during ventricular systole (systolic pressure); during ventricular diastole (diastolic pressure); and the difference between these determinations, (pulse pressure).

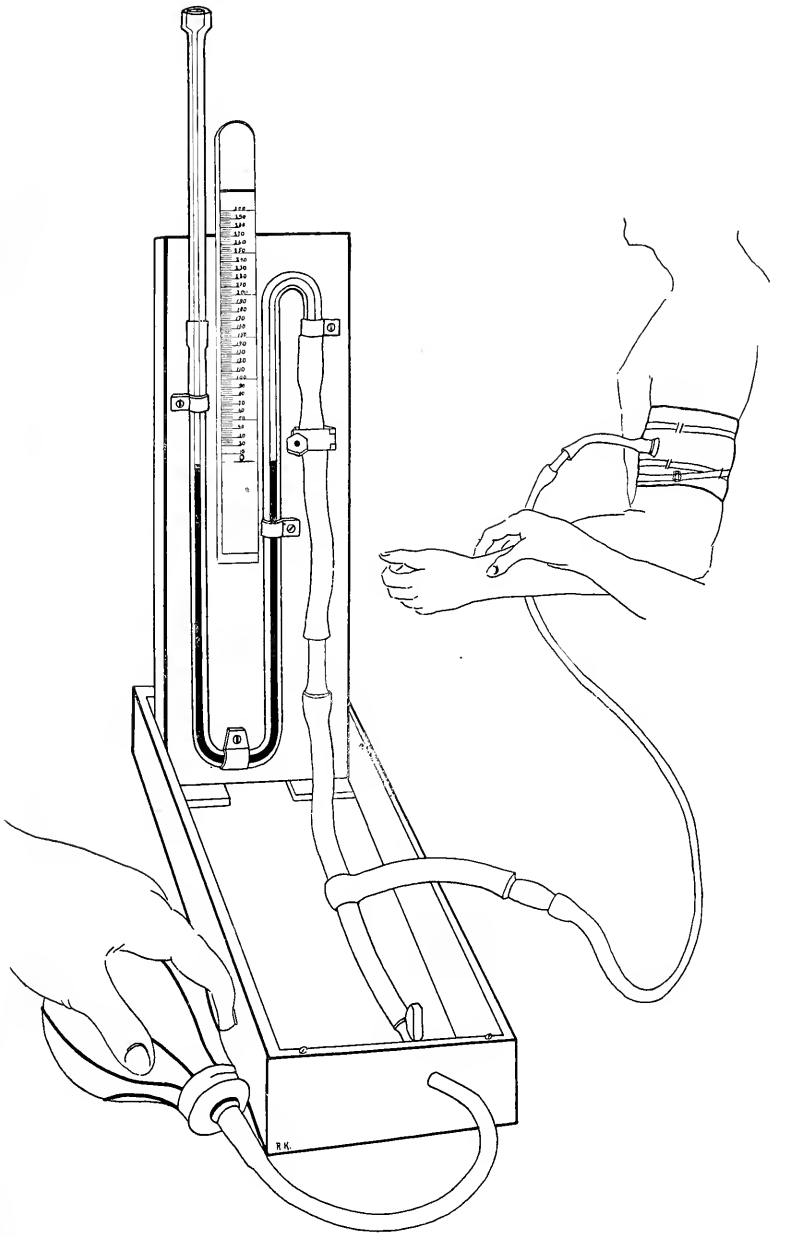


Fig. 124.—The Janeway sphygmomanometer which has been found a convenient and practicable instrument. The scale can be pushed below the level of the top of the box, the long arm of the mercury tube is disjointed and placed in the bottom of the box, the lid is then closed, and the instrument takes up but little space in the physician's bag. (From Warfield.)

Blood pressure is determined by an instrument, the sphygmomanometer, the procedure being termed sphygmo-manometry. The first really accurate and practical sphygmo-manometer was devised by Riva-Rocci in 1896. This instrument has been modified by Cook, Stanton, Erlanger, Janeway, and Faught, the basis of all in-

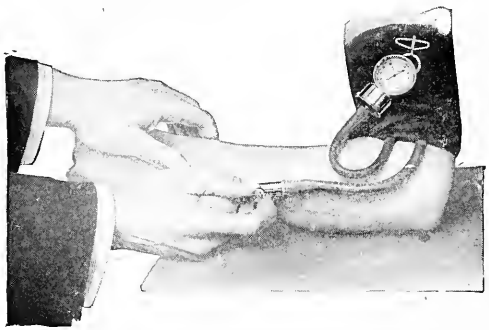


Fig. 125.—Rogers' "Tycos" dial sphygmomanometer. (From Warfield.)

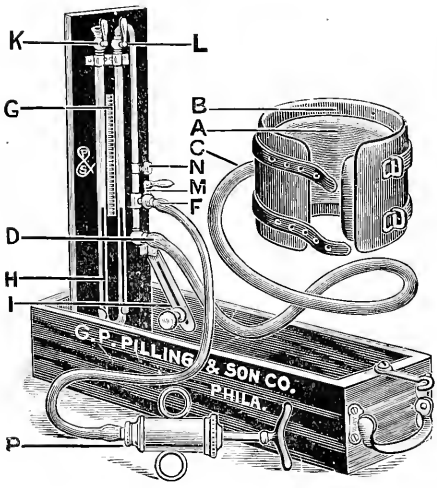


Fig. 126.

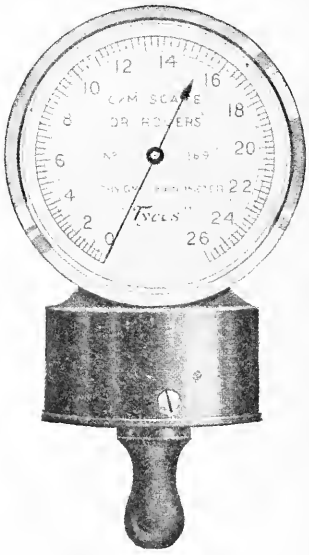


Fig. 127.

Fig. 126.—The Faught blood pressure instrument. An excellent instrument which is quite easily carried about and is not easily broken. (From Warfield.)
Fig. 127.—Detail of the dial in the "Tycos" instrument. (From Warfield.)

struments of this type consisting of an inflatable rubber bag, contained in an inelastic leather cuff so that during inflation of the bag the entire pressure is exerted upon the encased arm; a mercury manometer; and an air pump so connected by rubber tubing

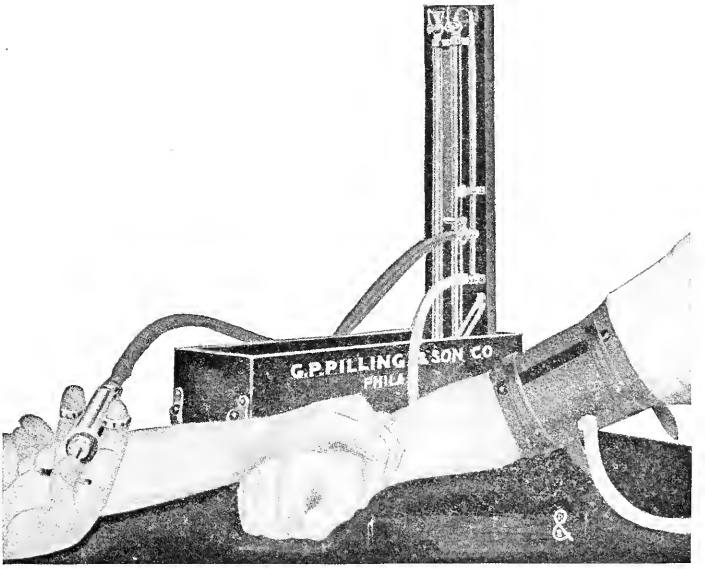


Fig. 128.—Method of taking blood pressure with a patient in sitting position. (From Warfield.)

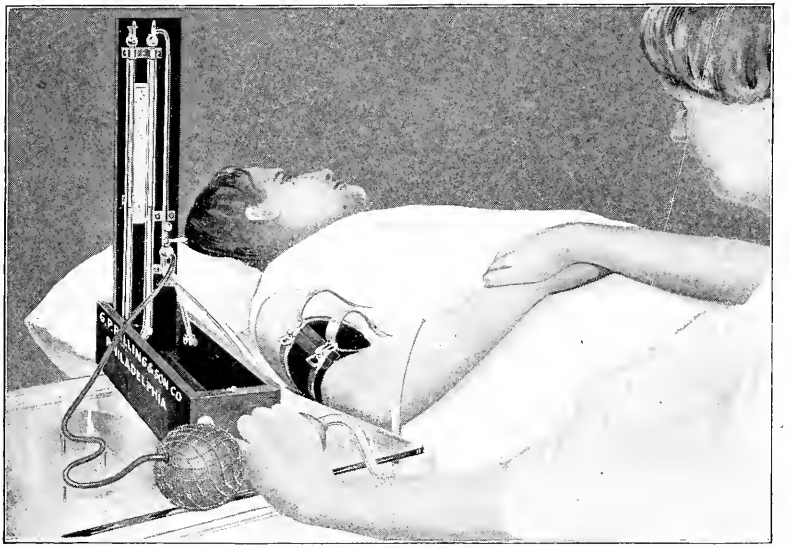


Fig. 129.—Method of taking blood pressure with patient lying down. (From Warfield.)

that the air which is pumped is distributed with uniform pressure to the cuff and the manometer.

A recently devised instrument, which dispenses with the use of the mercury manometer, and which instead of recording the pressure in millimeters of a mercury column records the pressure upon a dial, is the Rogers Tycos Instrument.

With either type of instrument in recording the blood pressure two methods may be employed; namely the palpatory method; or the auscultatory method. Whichever method is employed, certain details of the technic must be observed in order

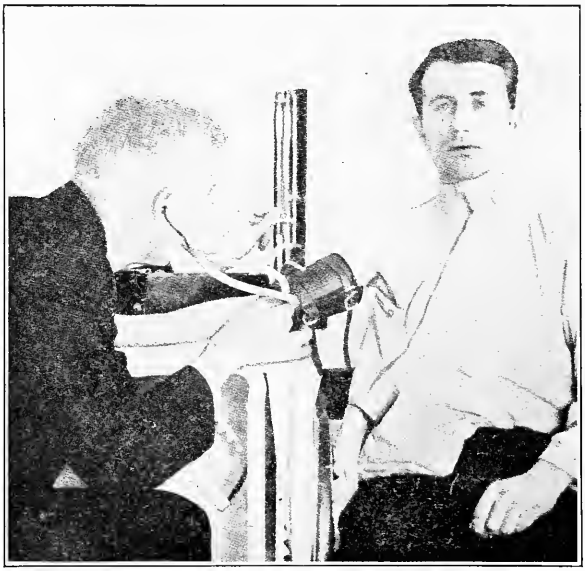


Fig. 130.—Observation by the auscultatory method and a mercury instrument. One hand regulates the stopcock which releases air gradually. (From Warfield.)

to obtain satisfactory results. The cuff should be placed at least two inches above the bend of the elbow; the connections of the tubing to the different portions of the instrument should be air-tight; the dilatable rubber bag should be adapted to the inner portion of the arm, overlying the brachial artery; the cuff should be snugly applied, but not with sufficient force to interfere with the venous return; and the lower portion of the cuff should fit the arm more loosely than the upper portion.

Palpatory Method.—When the cuff has been properly fitted to the arm and the tubing to the recording instrument, manom-

eter or dial, air is pumped into the cuff until the pulse becomes inappreciable to the finger palpating the radial artery. When the pulse disappears the mercury is pumped up 10 or 15 mm. above this point; and the screw is turned and allows the mercury to drop very slowly. At the instant that the pulse becomes again appreciable at the wrist the release valve is closed and the *systolic pressure* is read upon the scale or dial.

The principle involved in this procedure is that it requires an amount of external pressure to obliterate the pulse in the artery, which is commensurate with the intra-vascular pressure during systole.

Having determined the systolic pressure by the method described, the release valve is again slowly rotated, and the column or needle allowed to descend upon the scale very slowly, the undulations of the column or needle being closely observed. The descent is attended by oscillations; and at one point in the descent these oscillations become very pronounced, this point corresponding to the *diastolic pressure,* and as a rule being accompanied by a larger pulse wave than normal at the wrist.

By deducting the diastolic pressure from the systolic pressure, as recorded, the *pulse pressure* is obtained. In a healthy adult male the systolic blood pressure usually ranges between 120 and 135 millimeters, though a systolic pressure of 140 in such a patient is not necessarily pathologic. The diastolic ranges between 90 and 110 millimeters, the pulse pressure usually ranging from 25 to 35 millimeters.

Auscultatory Method.—The auscultatory method of determining the blood pressure is more accurate than is the palpatory method, and, in addition, shows wider ranges of pressure in the individual case. The systolic pressure, as determined by the auscultatory method, is always about 5 millimeters above that registered by the palpatory method, whereas the diastolic pressure often ranges 10 to 15 millimeters below that obtained by the palpatory method.

In determining blood pressure by this method the bell of a stethoscope is applied over the brachial artery just above the bend of the elbow, and the cuff inflated until all sound disappears. Having attained this point, the air is allowed to slowly escape from the cuff, whereupon a series of sounds are heard which have been divided into five phases. The *first phase* is represented by the first sound which is heard, which is the proper

point at which to record the *systolic pressure.* The first phase is quickly followed by a peculiar murmuring sound as the tension in the cuff is lowered, the *second phase;* this in turn is followed by a sharp, ringing note of increased intensity, the *third phase.* The sharp murmur of the third phase gradually gives place to a less intense sound, the *fourth phase;* this phase lasts until all sound ceases, the *fifth phase.* The *diastolic pressure* may be recorded at the beginning of the fourth phase or at the beginning of the fifth phase, the time at which all sound ceases. There is a difference of approximately 5 millimeters, as the record is made at the fourth or the fifth phase; but as it is often difficult to say just when the fourth phase begins, and as it is relatively easy to determine when all sound ceases, it is a safe rule to record the diastolic pressure at the commencement of the fifth phase, bearing in mind the discrepancy between the reading at the two phases.

Normal Variations.—Before drawing conclusions from variations in blood pressure, certain normal variations must be eliminated. Thus, the pressure varies with the attitude assumed by the patient, being higher when he stands, and lower when the sitting or recumbent attitude is assumed. Clinically the blood pressure may be estimated with the patient either in the sitting or recumbent posture with equally satisfactory results; but whichever attitude is assumed at the first estimation should be employed in all subsequent examinations. After a full meal the blood pressure is slightly higher than it is several hours after a meal; and during sleep it is normally lower than during waking hours. Exercise, nervous excitement, and the ingestion of stimulants increases the blood pressure temporarily. Similarly, high altitudes raise the blood pressure temporarily.

Pathological Variations.—When a high systolic pressure is encountered in a patient who is and has been at rest, it usually points to cardiac hypertrophy, the causes of which are varied; to arterio-sclerosis, to nephritis, or brain tumor, or apoplexy. In female subjects, it may point to threatened eclampsia. In cardio-vascular disease a high systolic pressure which is accompanied by an increased pulse pressure usually is indicative of adequate compensation; whereas a normal systolic pressure with a decreased pulse pressure points to threatened cardiac failure.

A decrease in the systolic pressure accompanies conditions of shock and collapse, internal hemorrhage, and the vascular asthenia of Addison's disease. In the course of typhoid fever a sud-

den drop in the systolic pressure is suggestive of perforation. A low systolic pressure is often an early sign of phthisis.

The importance of variations in the *diastolic pressure* has come to be generally recognized. The diastolic pressure represents the degree of peripheral resistance which must be overcome before the left ventricle may discharge its contents; and if this pressure is raised, as it usually is in arterial fibrosis and chronic interstitial nephritis, an added burden is thrown upon the heart before the blood can begin to circulate. Moreover, when a high diastolic pressure is encountered, it is of great importance from the standpoint of prognosis and treatment to determine whether this increase is due to functional angiospasm or to organic disease of the arterial system. In aortic regurgitation the diastolic pressure is low, while the pulse pressure is increased to a corresponding degree.

The *pulse pressure* deserves careful study in all cases in which sphygmomanometry is practiced. The pulse pressure represents the contractile power of the left ventricle in excess of the diastolic pressure; or, in other words, the power of the left ventricle over and above the peripheral resistance to the circulation of the blood. Thus, in the course of lobar pneumonia, when the heart is laboring under an increased load, a daily record of the pulse pressure gives valuable information as to the state of the myocardium, and affords a valuable prognostic and therapeutic index. As the overtaxed heart gradually fails, the systolic pressure gradually approximates the diastolic pressure, the pulse pressure steadily diminishing until it is nil, the point at which life becomes impossible.

Venous Pressure.—Hooker, who has devised an apparatus which permits the recording of the venous pressure, states that there is a progressive rise of venous pressure from youth until old age. He also finds that just before death there is a rapid rise of the venous pressure. Clark* states that a venous pressure of 20 centimeters of water represents the limit between adequate compensation and decompensation of the heart, and that a rise above this point is apt to be followed by cardiac failure.

A rough estimate of the venous pressure may be made by observing the superficial veins upon the back of the hand when the hand is raised above the level of the heart. Normally these veins

*Arch. Int. Med., Oct., 1915.

should collapse when the hand is raised above the level of the heart, and they should practically collapse with the hand at the level of the cardiac apex. But in the presence of increased venous pressure the veins fail to collapse when the hand is raised even above the level of the heart. Oliver states that the venous pressure may be estimated in millimeters of mercury by multiplying by 2 each inch above the level of the cardiac apex in which the veins collapse.

SECTION V

DISEASES OF THE CIRCULATORY ORGANS

CHAPTER XVII

DISEASES OF THE PERICARDIUM

PERICARDITIS

Inflammation of the pericardium occurs primarily and as a secondary disease. Primary pericarditis may be caused by trauma to the pericardium from without or from within the thorax. Certain cases of so-called idiopathic pericarditis develop in children without assignable cause.

Secondary pericarditis may be a sequence of rheumatism, tonsillitis and other septic states, acute fevers, gout, tuberculosis, and in the course of arterio-sclerosis.

Pericarditis is commonly classified as: Acute fibrinous pericarditis; sero-fibrinous pericarditis, or pericarditis with effusion; and chronic adhesive pericarditis.

Acute Fibrinous Pericarditis

Pathology.—In this form of pericarditis, which is also termed pericarditis sicca, the surface of the visceral pericardium and later of the parietal pericardium loses its normal glistening smooth appearance, and becomes roughened. It is the seat of a fibrinous exudate, which may be circumscribed, involving only a small portion of the membrane, or may be universal, involving the entire pericardial surface. There is usually a small quantity of fluid admixed with the exudate; but in tuberculous cases it may be quite dry. The roughened, exudate-clothed pericardium does not glide noiselessly as is the case in health; but instead produces a friction rub.

The appearance of the pericardial surfaces varies in different stages of the disease. Thus, in a fairly early case the surfaces

appear like two pieces of bread and butter which have been apposed and pulled asunder, the "bread and butter" stage. In other instances the exudate is rolled into irregular folds upon the pericardial surface, constituting the cor villosum.

The subjacent myocardium is involved to a variable extent in the inflammatory process, presenting infiltration with leukocytes. Endocarditis is a frequent accompaniment, but does not arise by extension of the pericardial inflammation.

Physical Signs.—*Inspection.*—In acute fibrinous pericarditis inspection is usually negative, though it may reveal the presence

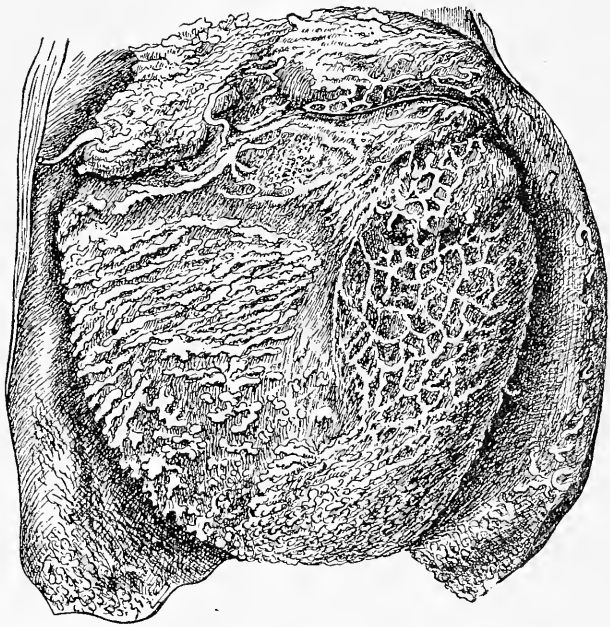


Fig. 131.—Acute fibrinous pericarditis. (From McFarland.)

of dyspnea and perhaps this will amount to orthopnea. The cardiac impulse is accentuated in force, and perhaps in extent. The facial expression is anxious.

Palpation reveals the presence of pericardial friction fremitus, which is increased by having the patient bend forward. The pulse is accelerated, and is apt to be of the pulsus paradoxus type.

Percussion reveals no deviation from the normal, as the heart is not enlarged in acute fibrinous pericarditis.

Auscultation shows the presence of the pericardial friction rub. As stated in a previous section, this rub is very variable, being

increased by moderate pressure and abolished by strong pressure with the stethoscope; also varying in intensity upon change of posture, and varying from day to day in intensity; also, as stated in a previous section, corresponding roughly with the systole and diastole of the heart, but not as accurately as do endocardial murmurs, *lasting longer than do the heart sounds.* Although its systolic and diastolic phases have the same intensity, they are usually of unequal length. It is confined to the precordia and is not transmitted thence, as are endocardial murmurs. If the associated effusion becomes great, the friction rub disappears, but not infrequently remains audible over the base of the heart. The quality of the friction rub has been compared to the creaking of the leather of a new leather saddle. These friction sounds are usually best heard to the left of the sternum in the fourth and fifth interspaces or near the aortic valve area. In other instances it is most audible at the apex area. The friction sound seems very superficial, as if it were just beneath the ear. The pericardial friction rub usually presents two phases, systolic and diastolic; sometimes only a single phase; in other instances three phases, simulating a sort of canter rhythm or gallop rhythm

Diagnosis.—The diagnosis of acute fibrinous pericarditis is very easy when the disease is suspected and the friction rub, the pathognomonic sign is sought for. But, arising as it does, during some acute infection it is often masked by the other symptoms of the disease and is not suspected nor sought for, hence is overlooked.

A mistake may arise in failing to differentiate acute fibrinous pericarditis from organic disease of the aortic valve, or from pleuro-pericardial friction in pleurisy arising during pneumonia or tuberculosis. Thus organic disease of the aortic orifice producing a double murmur, systolic and diastolic in time, with a palpable thrill may be easily mistaken for the two phases of the pericardial friction rub. But this double murmur corresponds more closely with the cardiac systole and diastole; the murmurs are transmitted from the precordia in different and definite lines; and produce alterations in the character of the pulse, as for instance the water-hammer pulse in Corrigan's disease, and the small, wiry pulse of aortic stenosis. The close localization or circumscription of the murmur to the aortic orifice is of aid in differentiation, and, moreover, aortic disease causes hypertrophy and perhaps ultimately dilatation of the heart. The pericardial friction sound is more superficial and variable than is the aortic

murmur, and is influenced by pressure with the stethoscope, which does not influence the murmur of organic disease of the aortic valve.

The *pleuro-pericardial friction rub* somewhat resembles the pericardial friction rub, but it disappears during suspension of the respiration following full inspiration.

Acute fibrinous pericarditis is accompanied by pain over the precordia or around the xyphoid appendix, and moderate fever.

Sero-Fibrinous Pericarditis (Pericarditis with Effusion)

Pathology.—Sero-fibrinous pericarditis begins as a dry pericarditis, the pericardial layers being covered with fibrinous exudate. Soon, however, there is an exudation of sero-fibrinous fluid, which fills the pericardial sac more or less completely. This fluid is often turbid, containing flocculi of fibrin. The quantity of the fluid varies from 200 cubic centimeters to two liters or more. In some cases there are only a few cubic centimeters of the fluid. The fluid may be completely absorbed with little permanent damage to the pericardium; but occasionally adhesions form between the visceral and parietal layers, and in other cases there are left localized areas of pericardial thickening, the so-called milk spots, or soldier spots. The subjacent myocardium is inflamed to a variable extent, and endocarditis, is sometimes a concomitant condition, but never by extension of the inflammation through the myocardium.

Physical Signs.—*Inspection.*—Sero-fibrinous pericarditis produces a variable extent of precordial bulging, most pronounced in young children and thin chested individuals. The cardiac impulse may be invisible, or there may be a wavy impulse in the third and fourth intercostal spaces to the left of the sternum. In large effusions the epigastrium bulges. The expansion of the left lung is diminished by pressure of the lung by the effusion. The diaphragm and left lobe of the liver are depressed, adding to the epigastric prominence. Precordial bulging is usually confined to children, whose thoracic parietes are resilient, the rigid chest walls of adults not bulging readily. If the apex beat is present, it is enfeebled. In extensive effusion there is epigastric bulging which constitutes Auenbrugger's sign. There is dyspnea, sometimes orthopnea. Precordial bulging is noted in women as well as in children. When the cardiac impulse is invisible it may in some instances be made visible by having the patient bend forward. Tortuosity of the superficial veins some-

times occurs as a result of intra-thoracic pressure of the effusion.

Palpation confirms displacement of the apex, which is sometimes abnormally low. Early in the disease a friction rub is palpable, which disappears as the effusion develops, though it sometimes persists at the base, to reappear with absorption of the effusion. As to intensity, the cardiac impulse is feeble, and gradually decreases in intensity as the effusion develops, to finally disappear altogether. Ewart has noted that the first rib is palpable at its chondro-sternal articulation in pericarditis with effusion, the "first rib sign." Fluctuation can be obtained in only rare instances of large effusion. Epigastric tenderness upon palpation is frequent.

The *pulse* is usually weak and of small volume, often irregular, and of the pulsus paradoxus type, gradually becoming progressively weaker during full inspiration.

Percussion.—The area of heart dullness is increased, and this increases gradually and progressively as the effusion progresses. The shape of the area of dullness is pear-shaped with its base directed downward toward the diaphragm. In the fifth interspace the dullness extends one or two inches to the right of the sternum constituting Rotch's sign. On the left side the dullness may extend outward beyond the apex of the heart or even into the axilla. Also the dullness of a large effusion may encroach upon and obscure the normal gastric tympany in Traube's semilunar space. The diaphragm and left lobe of the liver are depressed. Gerhardt has pointed out that when the patient is upright the area of dullness is broader than when he is recumbent. A co-existing emphysema may serve to obscure the dullness of a fairly large pericardial effusion.

The increase in heart dullness usually occurs in all directions, but in many cases it is increased only to the left side and upward. Rotch's sign is an extension of the dullness into Egstein's cardio-hepatic angle, and this is often an early sign of the disease.

Auscultation.—In the early stages of the disease prior to the development of the effusion, a friction sound is audible over the precordia. When the effusion develops this friction rub disappears gradually, although frequently being still audible at the base. If inaudible when the patient is in the recumbent posture, it sometimes appears when he is in the sitting posture. The cardiac sounds become gradually weakened and muffled as the effusion develops. The pulmonic second sound is apt to be ac-

centuated. The crowding of the left lung may cause bronchial breath sounds in the axillary region.

Diagnosis.—The pear-shaped area of increased cardiac dullness, the initial friction rub, disappearing with development of the effusion, the muffled and faint character of the heart sounds, with pain over the precordia, aggravated by pressure over the lower end of the sternum, dyspnea, cyanosis, the paradoxic pulse, and fever, point to effusion into the pericardial sac.

Dilatation of the heart produces increase in the area of heart dullness and must be differentiated from pericardial effusion. Concerning these two conditions Osler lays down six rules of differential diagnosis:

1. The character of the impulse, which in dilatation is commonly visible and wavy.

2. The shock of the cardiac sounds is more distinctly palpable in dilatation.

3. The area of dullness in dilatation rarely has a triangular form; nor does it, except in cases of mitral stenosis, reach so high along the left sternal margin or so low in the fifth and sixth interspaces without visible or palpable impulse. An upper limit of dullness shifting with change of position speaks strongly for effusion.

4. Rarely in dilatation is the distention sufficient to compress the lung and produce the tympanitic note in the axillary region.

5. In dilatation the heart sounds are clearer, often sharp, valvular, or fetal in character; gallop rhythm is common, whereas in effusion the sounds are distant and muffled.

6. The x-ray picture may be very definite and unlike any form of dilatation of the heart. (Osler.)

Pleurisy with effusion sometimes requires to be differentiated from pericardial effusion when this is very large. In left sided pleural effusion, unless the effusion assumes the encysted form, it is apt to be mistaken for a large pericardial effusion; and similarly a large pericardial effusion may be difficult to differentiate from a left sided pleural effusion. But in pleurisy with effusion the cardiac impulse is displaced; the flatness extends around the side of the chest; the compressed lung gives skodaic resonance in the infra-clavicular and mammary regions; Traube's semilunar space of tympany is obliterated; and the spleen is apt to be displaced downward. In pericarditis with effusion, on the contrary the effusion is apt to extend well to the right of the sternum (Rotch's sign), which is of aid in the differentiating the two diseases.

Chronic Adhesive Pericarditis

Pathology.—In chronic adhesive pericarditis adhesions are left as the result of a previous acute pericarditis of the fibrinous or serofibrinous type. The adhesions may be localized to a limited extent of the visceral and parietal membranes or may be universally distributed over these membranes. The adhesions, moreover, may exist between the external surface of the pericardium and the adjacent pleura, constituting pleuro-pericarditis or mediastino-pericarditis.

The internal form of chronic adhesive pericarditis, in which the adhesions exist between the visceral and parietal layers often

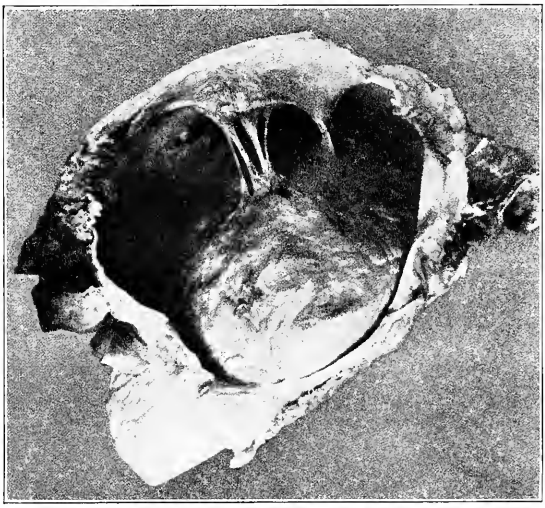


Fig. 132.—Pericardial adhesions. (From Delafield and Prudden.)

does not embarrass the cardiac action, and often gives rise to no symptoms and few physical signs. But if the adhesions are abundant there is more or less embarrassment of the cardiac action, with consequent hypertrophy and dilatation of that organ.

In external pericarditis the adhesions bind the outer surface of the pericardial sac to the costal pleura anteriorly or to the diaphragm, or the esophagus, or the spinal column, or the great vessels arising from the base of the heart. In this form the action of the heart often causes systolic retractions of the thoracic parietes.

As stated, the condition of the myocardium depends upon the degree of interference with the heart's action. The heart is

often unchanged in simple adhesions within the pericardial sac; but shows hypertrophy and dilatation in extensive adhesions.

Physical Signs.—*Inspection.*—With cardiac hypertrophy there may be undue prominence of the precordia with the apex beat displaced from its normal site in the fifth left interspace. There is often a systolic retraction of the chest wall anteriorly near the apical area. In cases with diaphragmatic adhesions there is often a systolic retraction of the tenth and eleventh interspaces posteriorly below the left scapula (Broadbent's sign).

Friedreich's sign, diastolic collapse of the jugular veins may be noted in some cases; and Kussmaul's sign, inspiratory overfulness of the jugulars is sometimes to be noted. The mobility of the diaphragm is often interfered with and limited in extent by adhesions.

The cardiac impulse may be wavy and increased very much in extent. Upon changing the patient's position, laying him on his side, the apex beat or cardiac impulse often does not move with this change of posture as it normally does.

Palpation.—"There may be a diastolic shock, a sudden rebound of the heart walls during diastole, after having been drawn together during systole against the resistance of the adhesions." (Butler.) The pulsus paradoxus is often present. Palpation confirms displacement of the apex beat when this is present. Adhesions between the pericardium and diaphragm often prevent the normal epigastric excursion during inspiration.

Percussion.—Percussion reveals a considerable increase in the transverse diameter of the heart. But this is not a constant finding, as the heart may be neither hypertrophied nor dilated. There are often adhesions existing between the pleura and the pericardium so that the border of the cardiac dullness above and to the left of the heart is not diminished by full inspiration, as the anterior border of the left lung, is prevented from intervening between the pericardium and the anterior chest wall. But this too is not a constant or reliable sign. The gastric tympany of Traube's semilunar space is often encroached upon.

Auscultation.—The character of the heart sounds depends upon the state of the myocardium. If the heart is hypertrophied, they are accentuated; whereas, if cardiac dilatation has supervened, they are diminished. There may be present the murmurs of coexisting valvular disease, not dependent upon the condition itself. A friction rub may rarely be heard along the left border of the

sternum. The pulmonic second sound is sometimes reduplicated, and sometimes there is a systolic murmur at the mitral area.

Diagnosis.—With a history of pericarditis, the finding of signs of adhesion such as fixation of the cardiac impulse, overfulness of the cervical veins during inspiration, and diastolic collapse of these vessels, with systolic retractions of the thoracic walls anteriorly or Broadbent's sign posteriorly, point to chronic adhesive pericarditis. It is true that a systolic retraction of the thoracic wall in the region of the apex may be due to atmospheric pressure, in cases of hypertrophy of the heart, when the anterior borders of the lungs are tardy in closing in between the heart and anterior chest wall, but the systolic retraction of chronic adhesive pericarditis is altogether more forcible than is this phenomenon.

In cases where there are only a few simple adhesions between the epicardium and visceral pericardium, causing little or no embarrassment to the action of the heart, a diagnosis is extremely difficult or impossible.

HYDRO-PERICARDIUM (HYDROPS PERICARDII)

Hydro-pericardium, the accumulation of serous fluid in the pericardial sac, is usually a sequence of the general dropsy of nephritis, or of valvular heart disease, more rarely being due to thrombosis of the cardiac veins. The pericardial sac contains a variable amount of clear, serous fluid.

The physical signs are those of fluid in the pericardial sac. There is, however, no friction rub, pain, or fever, which excludes pericarditis with effusion. Coupled with a history of cardiac or renal disease, these signs are suggestive. The condition is often overlooked entirely.

Hydro-pericardium produces dyspnea by compression of the lungs.

HEMO-PERICARDIUM

This is the presence of blood or blood-tinged fluid in the pericardial sac. The fluid of sero-fibrinous pericarditis is sometimes tinged with blood. It also follows stab wounds or penetrating wounds from other causes. It may be due to rupture of the ascending part of the aorta before it leaves the pericardial sac, or it may be due to rupture of a coronary artery.

The physical signs are those of fluid in the pericardial sac,

with in some cases signs of internal hemorrhage, as pallor, rapid weak pulse, dyspnea and collapse. Death occurs early as a result of pressure upon the heart.

PNEUMO-PERICARDIUM

Pneumo-pericardium, the presence of gas in the pericardial sac is usually associated with the presence of fluid (hydro-pneumo-pericardium), blood (hemo-pneumo-pericardium), or pus (pyo-

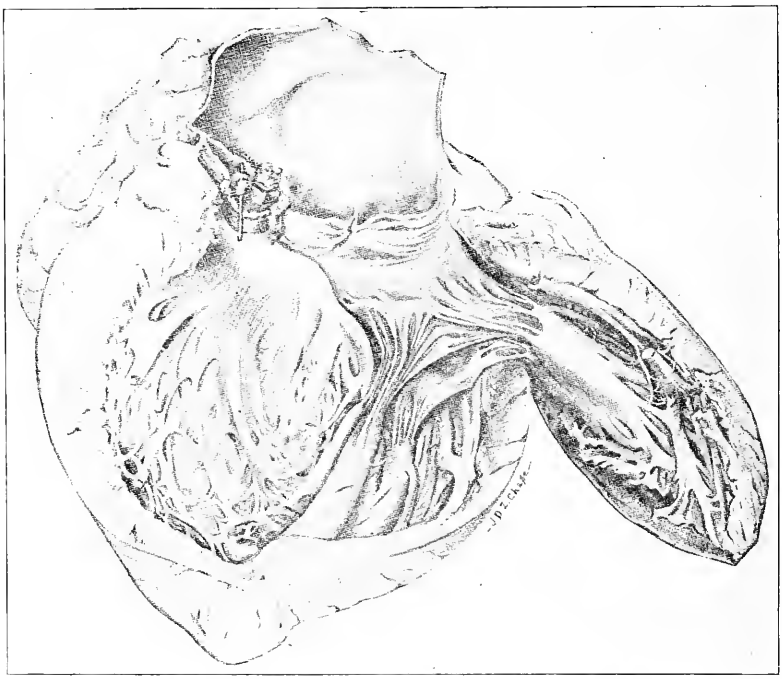


Fig. 133.—Malignant endocarditis of aortic valve. (From McFarland.)

pneumo-pericardium). It is a disease which is only rarely encountered.

Pneumo-pericardium may be the result of penetrating wounds; perforation of the pericardium by a tuberculous cavity, gangrene of the lung or pneumo-thorax; or it may be spontaneous, owing to the development of the bacillus aerogenes capsulatus of Welch. Malignant disease of the esophagus or stomach rarely causes perforation of the pericardium and pneumo-pericardium.

Physical Signs.—Upon *inspection* there may be precordial bulging with absence of the visible apex beat. Upon *palpation* a fric-

tion rub can sometimes be felt, and in other cases associated with fluid succussion fremitus. Upon *percussion* the normal area of cardiac dullness will yield hyper-resonance, while the level of the fluid in the pericardial sac will give dullness.

Upon *auscultation* the heart sounds are feeble being obscured by the pericardial succussion sound, which is a churning sound which has been likened to the sound produced by a water-wheel in motion. A friction sound may often be heard. The succussion sound is distinctive and pathognomonic.

Diagnosis.—The succussion sound of the condition is pathognomonic and when demonstrated makes the diagnosis clear. Pneumo-pericardium may be confused with left sided pneumothorax or gaseous distention of the stomach. In left pneumothorax the area of cardiac dullness is not obliterated, and the cardiac impulse is usually visible though often displaced toward the opposite side of the chest. The same principle applies to distention of the stomach; and, moreover, the tympanitic note of a distended stomach disappears when a stomach tube is passed and the gas is liberated from the viscus.

CHAPTER XVIII

DISEASES OF THE ENDOCARDIUM AND VALVES

ACUTE ENDOCARDITIS

Pathology.—Acute endocarditis occurs in two forms, simple acute endocarditis, and malignant or infective endocarditis.

Acute simple endocarditis is nearly always secondary to disease elsewhere in the bodily economy. Probably the most frequent cause is rheumatism or acute rheumatic fever. Next in frequency comes tonsillitis, followed by scarlatina, and chorea. Many acute infections are liable to become complicated with acute endocarditis. Wasting diseases, as carcinoma, diabetes and nephritis, are frequently associated with acute endocarditis.

Recurrent endocarditis is a form of endocarditis in which valves which are the site of chronic lesions suddenly light up with acute attacks of endocarditis. Endocarditis occurs in association with the exanthematous fevers occasionally and is a not infrequent complication of erysipelas.

Acute endocarditis occurs more frequently in males than in females, and is most frequent about the third and fourth decades.

Simple endocarditis often develops during the course of tuberculosis or gout.

The lesions of acute simple endocarditis very rarely affect the endocardium lining the walls of the chambers of the heart (mural endocarditis); but this number of cases is so small as to be negligible. In the vast majority of cases the changes are in the valves themselves (valvular endocarditis).

The earliest changes from normal in the valves is noted at a point a little distance from the free edge, the point where the valves come into contact. It is probable that the earliest changes are a decrease in the size of the individual endothelial cells with a tendency to assume a cuboidal form, leaving crevices between the cells by way of which bacteria circulating in the blood are enabled to penetrate and affect the deeper structure of the valve.

In the simplest type of change there is necrosis of the valvular

tissue where the bacteria penetrate, and beyond this hyaline change in the valvular tissue.

Fibrin is deposited upon the denuded area layer after layer, forming excrescences. This constitutes the verrucose type of simple endocarditis. If these verrucosities are torn loose by the circulating blood they float free in the blood stream as emboli. In course of time the excrescences become organized and scar tissue replaces the verrucose excrescence, which by contracting causes deformity of the valve and incompetency.

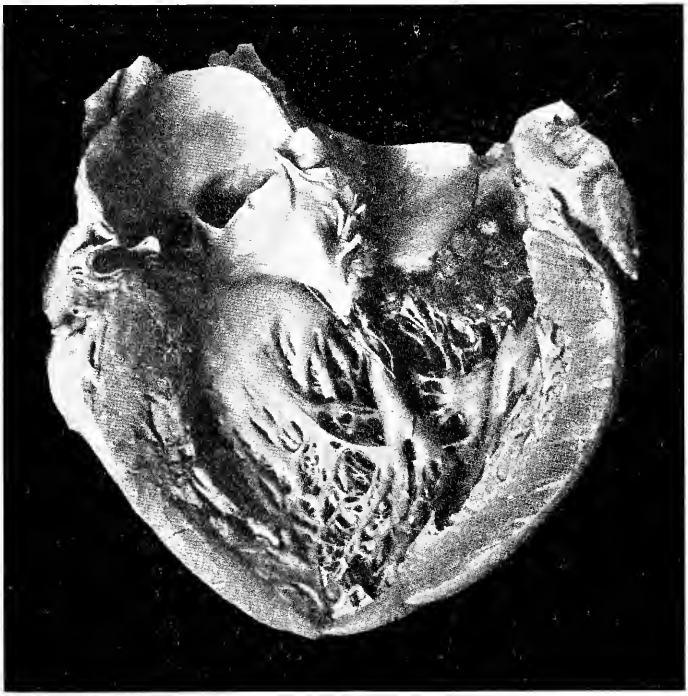


Fig. 134.—Endocarditis, verrucose form. (From Delafield and Prudden.)

Malignant or *infective endocarditis* occurs in rare instances as a primary inflammation of the endocardium. Usually it is secondary to puerperal sepsis, osteo-myelitis, rheumatic fever, or as a progression of the simple acute form into the malignant. It may be secondary to erysipelas or gonorrhea. Malignant endocarditis is often engrafted upon valves the seat of chronic endocarditis.

In this form of endocarditis purulent collections form in the connective tissue stroma of the valve, the vessels dilate and new

vessels grow out into the valve stroma. There is an attempt at repair by connective tissue formation, but the diseased surface of the valve does not heal with a smooth surface, but with irregular villous processes consisting of granulation tissue and fibrin. Perforation of a valve often occurs and the new connective tissue always leaves the valves incompetent. Emboli formed of fragments of the diseased villous processes on the valves may be carried to remote organs, and, being infectious, cause metastatic abscesses. (See Fig. 133, p. 262.)

Physical Signs.—*Inspection.*—In simple acute endocarditis inspection usually reveals nothing. In malignant endocarditis if dilatation has supervened the apex beat is weak and diffuse and perhaps displaced. The signs upon inspection in both forms are few.

Palpation.—Palpation yields little or no information in both forms. If valvular lesions are present there may be a palpable thrill; and if dilatation is present the apex beat may be feeble and slapping and displaced from its normal position.

Percussion.—Percussion will add no information in acute simple endocarditis. In the malignant form the percussion findings are similarly lacking unless dilatation is imminent or has supervened, when there will be increase in the area of cardiac dullness in one or more directions.

Auscultation.—In simple acute endocarditis there is often a systolic murmur over the apex or at the aortic area; but these are not pathognomonic as they might arise as a result of anemia or valvular lesion of other causation. In the malignant form murmurs of pre-existing valvular disease are often present. If it is possible to detect changes in the quality of these murmurs in daily examinations, an acute endocarditis may be indicated as being engrafted upon a previous valvular lesion.

Diagnosis.—In the diagnosis of acute simple endocarditis the history is very important. A history of rheumatic fever, tonsillitis, or an acute infectious disease coupled with the meager physical findings may suggest a diagnosis. Of the physical signs the most important probably is a systolic murmur at the mitral area, particularly if this develops upon a roughened somewhat prolonged first sound. Of course, these murmurs must be distinguished from a murmur due to anemia or a pre-existing valvular lesion. But murmurs of anemia are more prone to involve the pulmonic valve and very rarely indeed do they involve the aortic valve, over which in endocarditis a murmur may some-

times be audible. It is difficult or impossible to differentiate an acute simple endocarditis supervening upon a chronic valvular lesion, as the difference in the murmur is slight if any.

The *diagnosis* of *malignant endocarditis* when there is a history of previous septic infection and evidences of metastatic involvement in other parts of the body is fairly easy. Regurgitant diastolic murmurs are suggestive in a measure, as functional murmurs are mostly systolic in time. There is leukocytosis and precordial distress and fever of a septic type. It should be remembered that malignant endocarditis presents a very varied symptomatology, sometimes occurring in a cardiac type in which the murmurs of chronic valvular lesions predominate the picture; in a pyemic type with symptoms of metastatic involvement; in a typhoid type closely simulating typhoid fever; and finally a type in which cerebral symptoms predominate, as delirium or coma.

From *typhoid fever* it is differentiated by the more abrupt onset of endocarditis, the absence of the step-ladder ascent of the fever in the first week, and the presence of precordial distress and dyspnea early in endocarditis, with chills and leukocytosis in marked contrast to the leukopenia of typhoid fever.

CHRONIC ENDOCARDITIS

Pathology.—Chronic endocarditis is usually secondary to acute endocarditis, particularly to that form occurring in association with acute rheumatic fever and tonsillitis. Other cases of chronic endocarditis arise in persons who have not previously had acute endocarditis, as a result of the constant circulation in the blood of the toxins of lead, alcohol, syphilis, gout, and diabetes. Excessive and prolonged muscular exertion in laborious occupations may initiate the sclerotic process in the aortic valve segments. These patients also present sclerosis of the arteries, of which the sclerosis of the valves is merely a part. Arterio-sclerosis and nephritis by raising blood pressure tend to cause sclerosis of the aortic valves.

The changes in the valves consist of a progressive sclerosis with ultimate deposition of calcium salts. The valves are thickened, inelastic, and their edges often coalesce. This produces a permanent condition of incompetency or stenosis. Moreover, the chordæ tendineæ are shortened and thickened so that they no longer permit of close co-aptation of the edges of the valve segments. The ventricles hypertrophy to compensate for the val-

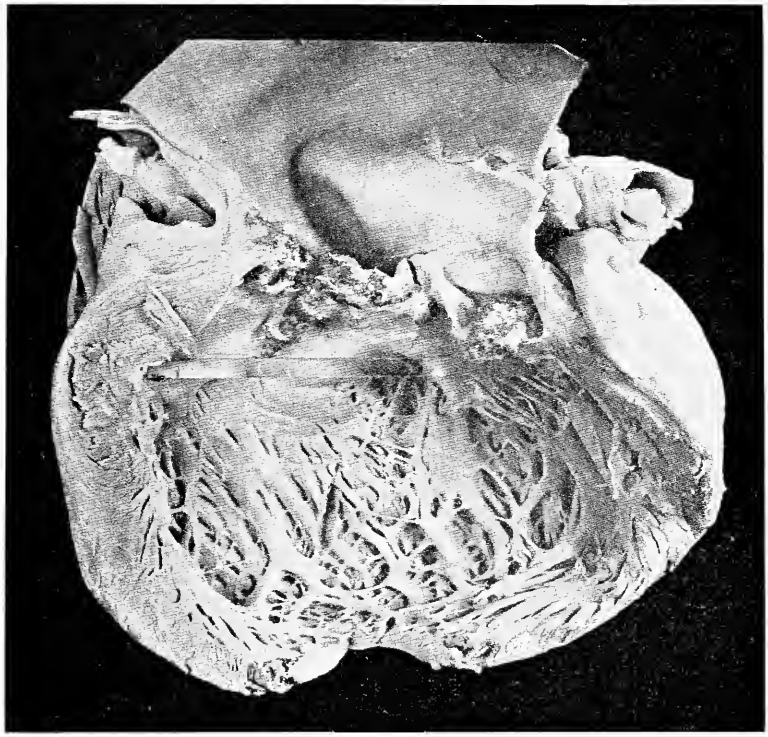


Fig. 135.—Chronic endocarditis. (Delafield and Prudden.)

vular deficiency; and ultimately degeneration of the myocardium and dilatation supervene.

The Physical Signs of chronic endocarditis are those of chronic valvular disease, varying in their manifestations and results with the valve affected or valves affected, if more than one, as is often the case.

CHRONIC VALVULAR DISEASE

Chronic valvular lesions of the heart are of two types: *stenotic,* produced by narrowing or partial obstruction offered at the valvular orifice to the onward flow of the blood; and *regurgitant,* produced by the inability of the valve cusps to prevent the backward flow of the blood, owing to deformity of the valve cusps or to a temporary stretching of the valvular orifice so that the normal segments are incapable of closing the abnormally large orifice. Not infrequently a lesion of a valve which produces stenosis also results in regurgitation, incompetence, or insufficiency.

The valves of the left side of the heart are much more frequently the sites of chronic lesions than are those of the right side. Acquired lesions of the valves of the right side of the heart are very rare, the vast majority of lesions on this side depending upon congenital malformations. However, acquired lesions do occur in this portion of the valvular mechanism of the heart, usually as the end result of left sided valvular lesions. Generaly speaking, mitral lesions are more frequent than are any other valvular lesions; aortic lesions coming second in frequency, while lesions of the valves on the right side are very rare.

Effects of Valvular Lesions.—The immediate effect of a valvular lesion is to decrease the amount of blood in front of the

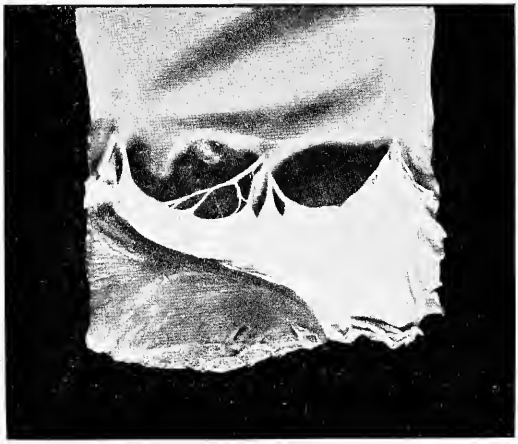


Fig. 136.—Fenestration of semilunar valves. (From Delafield and Prudden.)

lesion and to increase the amount of blood behind the lesion; hence, to lower the blood pressure in the arterial system and to raise the blood pressure in the venous system. This is well illustrated by the sequence of events following aortic stenosis. The first result of a stenotic lesion at this orifice is to produce dilatation of the left ventricle, the ventricle being unable to expel its increased blood content. The ventricle then hypertrophies in response to the increased demand for work. So long as this hypertrophy is maintained, or so long as compensation is not broken, no change is noted in the general circulation. But the time comes when the ventricle is unable to sustain the extra burden and dilatation of the left ventricle gradually supervenes.

The dilatation of the left ventricle, affecting the mitral ring

permits stretching of this ring, the cusps of the valves are unable to properly close the orifice, with the result that a certain amount of blood regurgitates into the auricle during ventricular systole. This extra load thrown upon the left auricle produces dilatation of the left auricle, which in turn hypertrophies to compensate for the regurgitation. This hypertrophy in course of time yields to dilatation; when, by virtue of the regurgitation of blood from the left ventricle which the dilated auricle is unable to expel during its systole, the blood pressure in the pulmonary circulation is raised, as evidenced by accentuation of the pulmonic second sound.

The continuous high tension in the pulmonary circuit leads to engorgement of the pulmonary venous system, resulting in edema of the lung, with bronchitic symptoms, or in more extreme grades, to hydro-thorax.

The increased strain which is thrown upon the right ventricle by the accumulation of blood in the lesser circulation produces dilatation of the right ventricle, which hypertrophies for a time to compensate for the extra burden, and then dilates. This dilatation of the right ventricle produces enlargement of the right auriculo-ventricular ring, which the segments of the tricuspid valve are unable to close. Hence the right auricle goes through the same cycle of dilatation, temporary hypertrophy and permanent dilatation, producing a permanent tricuspid regurgitation with systolic pulsation of the liver, and general venous stasis with edema of the lower extremities, ascites, or general anasarca.

AORTIC INSUFFICIENCY (CORRIGAN'S DISEASE)

Pathology.—Aortic insufficiency, aortic incompetency, aortic regurgitation, or Corrigan's disease may result from pathologic alteration of the valves due to endocarditis, whether shrinkage of the cusps, adhesions between the cusps, or perforation of one or more cusps; dilatation of the aortic ring in syphilitic aortitis; enlargement of the aortic ring due to aneurism of the root of the aorta, or dilatation of the heart; or congenital defects in the valves, as absence of a cusp or fenestration of the cusps.

The valves, deformed from any of the above mentioned causes, are unable to prevent the reflux of blood from the aorta into the ventricle during diastole, hence the left ventricle becomes the target for this reflux which produces a murmur as it passes backward through the incompetent valves. The left ventricle, in response to the increased demand for work in expelling

a greater quantity of blood at each systole hypertrophies and compensates for the valvular deficiency. This hypertrophy becomes very great, constituting the Cor Bovinum. After this compensation has been maintained for a variable time, the ventricle dilates and stretching of the mitral ring permits a regurgitant lesion at this site. This leads to heightening of the blood pressure in the pulmonary circulation and to accentuation of the pulmonic second sound and hypertrophy of the right ventricle.

Physical Signs.—*Inspection.*—In women and children, in whom

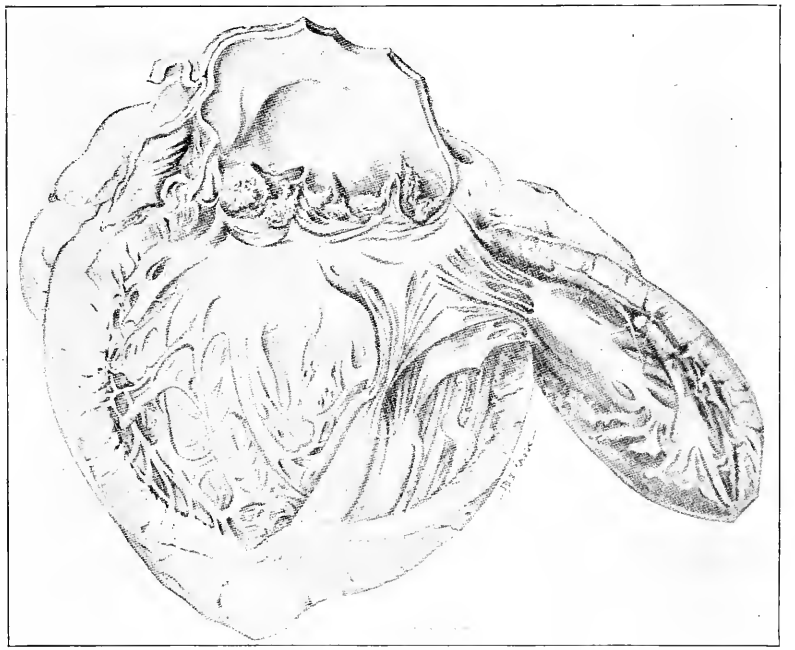


Fig. 137.—Chronic verrucose endocarditis of aortic valves. (From McFarland.)

the chest wall is elastic, there is apt to be a certain degree of precordial bulging. The apex beat is displaced downward and toward the left owing to the left ventricular hypertrophy. The capillary pulse of Quincke is present. There is dyspnea, sometimes orthopnea and cyanosis when the heart is beginning to fail. The carotid arteries may pulsate and there is apt to be systolic epigastric pulsation. The area of the cardiac impulse is increased and it is displaced sometimes as low as the seventh interspace and as far outward as the anterior axillary line.

Palpation.—The impulse during cardiac or left ventricular hy-

pertrophy is firm and forcible and heaving; and later, when dilatation is imminent is weaker and more slapping. A diastolic thrill is often present at the aortic valve area or over the middle of the gladiolus.

The pulse is of the water-hammer or Corrigan type.

Percussion.—The area of cardiac dullness is increased in extent, mainly downward and toward the left. In rare cases the dullness is increased to the right of the sternum. Aortic incompetence produces greater increase in the area of cardiac dullness than does any other valvular lesion.

Auscultation.—There is a diastolic murmur with its point of maximum intensity at the aortic valve area near the junction of the second right costal cartilage with the sternum, or over the middle of the gladiolus or in the neighborhood of the apex of the heart. The point of maximum intensity of this particular murmur is thus very variable. Perhaps in the greatest majority of cases it is near the middle of the gladiolus. The line of transmission is downward and toward the left. The murmur may and usually does replace the second sound of the heart at the aortic area; but sometimes both murmur and second sound can be heard. (See Fig. 116, p. 235.)

The quality of the murmur is blowing and not unmusical. The pulmonic second sound is unaltered, unless there is, as is frequently the case, an associated regurgitant lesion at the mitral area, in which event the pulmonic second sound is accentuated. The first sound of the heart is unusually loud and somewhat prolonged, owing to the large volume of blood to be expelled at each systole of the left ventricle. If there is an associated mitral regurgitation the first sound at the apex will be replaced by a systolic murmur.

A diastolic murmur at the apex in Corrigan's disease has been described by Austin Flint, the mechanism of which has been explained in a previous section. A diastolic murmur is often audible upon auscultation over the carotid artery; and Duroziez's sign, which has been described, is often demonstrable over the femoral artery.

Diagnosis.—The diagnosis of aortic regurgitation is as a rule not difficult. The diastolic murmur at the aortic area, transmitted downward and toward the left axilla, the signs of hypertrophy or dilatation of the left ventricle, the pulsations in the carotids, the water-hammer pulse, and Duroziez's sign, when present, make the diagnosis.

AORTIC STENOSIS

Pathology.—True organic stenosis of the aortic valve is encountered but rarely; but roughening of the valve segments or of the aortic lining just beyond the valve, generating a murmur very similar to that caused by aortic stenosis is rather frequent.

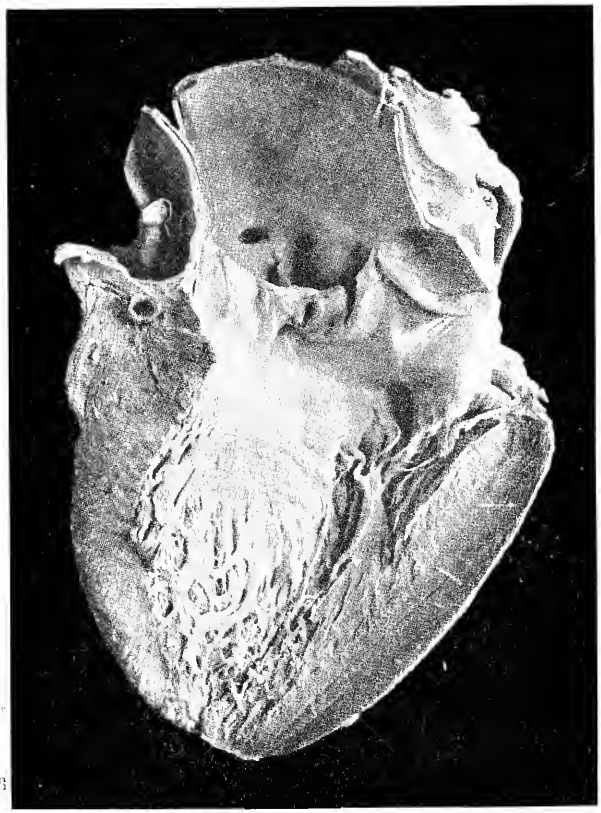


Fig. 138.—Chronic endocarditis with coalescence of two aortic cusps. (From Delafield and Prudden.)

Again, a relative stenosis of the aortic valve may be present arising as the result of dilatation of the aorta distal to the valve, a state which also produces a murmur simulating the murmur of organic aortic stenosis.

Stenosis of the aortic valve arises as a result of endocarditis, which leads to adhesion of the valvular cusps, associated with or followed by fibrosis and calcification of the valve leaflets. In

other instances aortic stenosis is part of a general arterio-sclerosis, which extends to and involves the valve segments.

Congenital deformity of the valve, there being merely a button-hole slit in a solid membrane instead of the three valvular cusps, is an infrequent cause of aortic stenosis.

In elderly persons aortic stenosis is usually the result of arteriosclerosis, while in children and young patients it is generally a sequence of rheumatic endocarditis attacking the aortic valve along with other cardiac valves.

Whatever the cause of obstruction, the effect upon the myocardium is in each case very much the same. An increased load is thrown upon the left ventricle, which hypertrophies in the effect to overcome the obstacle to the propulsion of its contents into the greater circulation. That is to say, the ventricle compensates for the valvular lesion. So long as compensation is adequate, so long as compensation is maintained, there are no signs of embarrassment of the circulation. But when the left ventricle begins to fail the left auriculo-ventricular ring stretches, the mitral valve becomes unable to close the abnormally large orifice, the pressure is raised in the pulmonary circulation, causing accentuation of the pulmonic second sound and leading eventually to hypertrophy of the right ventricle, with all the sequence of changes detailed in the section on the results of valvular lesions (see page 269).

The left ventricular hypertrophy of aortic stenosis differs from that of aortic regurgitation in that in the former condition the walls of the ventricle are thickened without any increase in the size of the ventricular chamber; whereas in the hypertrophy of the latter the chamber is increased in size, a condition to which the term eccentric hypertrophy has been applied.

As a rule the lesion which produces aortic stenosis also renders the valve segments incapable of completely closing the orifice during diastole, so that aortic stenosis and incompetency not infrequently co-exist.

Physical Signs.—*Inspection.*—The cardiac impulse is displaced to the left and downward, owing to the left ventricular hypertrophy. As cardiac dilatation supervenes the lateral displacement increases. The impulse is of increased extent; and its character is strong and heaving so long as compensation is maintained, becoming weak and indistinct when the left ventricle fails. The intervention of the borders of emphysematous lungs

between the heart and chest wall in elderly patients tends to mask the character of the cardiac impulse or to obscure it entirely.

Palpation.—A systolic thrill is nearly always demonstrable at the aortic valve area. As stated, the cardiac impulse may be strong and heaving or weak and indistinctly palpable, its character varying with the state of the myocardium.

The *pulse* is slow and small, particularly in marked organic stenosis. It is regular as long as compensation is maintained; later showing signs of cardiac dilatation.

Percussion.—The transverse dullness of the heart is increased to a variable extent. This increase, however, particularly in elderly patients, may be masked by an emphysematous condition of the lungs.

Auscultation.—There is a systolic murmur at the aortic area. The murmur is usually harsh, sometimes musical, well sustained. It is transmitted into the great cervical vessels. Quite frequently there is a double murmur at the aortic area, systolic and diastolic, for the reason that the lesion which causes stenosis also permits regurgitation of blood into the ventricle during diastole. The aortic second sound is rarely audible. The systolic murmur of relative mitral insufficiency is audible at the apex in most advanced cases. (See Fig. 115, p. 235.)

Diagnosis.—A systolic murmur at the aortic area, harsh in quality, well sustained in duration, transmitted upward into the cervical vessels, and accompanied by a thrill and a small slow pulse, is very suggestive of organic stenosis of the aortic valve.

However, the mere presence of a systolic murmur at this area is not conclusive, as such a murmur may have a different origin.

The murmur generated by *aortic roughening* is systolic in time and heard at the aortic valve area; but it produces no change in the character of the pulse, is not accompanied by a thrill, and is not characterized by ventricular hypertrophy.

Similarly, the murmur of *relative aortic stenosis* which is caused by dilatation of the aorta just distal to the valve, is systolic in time, but produces no alteraton in the pulse, and no organic change in the myocardium.

Moreover, in both these murmurs the aortic second sound is unimpaired.

Functional murmurs are rarely audible at the aortic area. They

are musical, are transient, are not transmitted beyond the precordia, are usually encountered in young persons, or in the anemic.

MITRAL INSUFFICIENCY

Pathology.—Mitral insufficiency is due to shrinking and sclerosis of valve segments and chordæ tendineæ, the sequence of acute or chronic endocarditis, of rheumatic origin, or a part and parcel of arterio-sclerosis. Also in left ventricular dilatation the mitral ring is stretched and is unable to permanently and completely close the left auriculo-ventricular orifice, resulting in relative insufficiency.

The results upon the heart and circulation of mitral insufficiency are as follows. During ventricular systole the mitral segments are unable to withstand the backflow of blood, a portion of which regurgitates into the left auricle. Here it combines with the blood entering the left auricle from the great veins, leading to dilatation of the left auricle, and eventually to hypertrophy. The pressure is thus raised in the lesser or pulmonary circulation with resulting hypertrophy of the right ventricle. The left ventricle hypertrophies owing to the lack of the valvular action of the mitral valve. More or less edema and congestion of the lungs occurs leading to symptoms of catarrhal inflammation of the bronchial tubes. In course of time the tricuspid valve develops a "safety-valve" leak and there is more or less extensive venous engorgement of the general circulation.

Physical Signs.—*Inspection.*—The cardiac impulse is displaced downward and toward the left, owing to the associated left ventricular hypertrophy. The character of the impulse varies with the state of the myocardium. In hypertrophy it is strong and forcible; in cardiac dilatation it is weak, slapping, and undulatory. Pulsations are often visible in the veins of the neck. The cardiac impulse may be displaced far to the left, sometimes past the left nipple line, sometimes as far as the anterior axillary line. In children and thin chested women, a moderate degree of precordial bulging not uncommonly attends the condition. In these cases there is commonly a wide area of impulse. Systolic pulsation in the epigastrium is often present in cases associated with right ventricular hypertrophy. Pulsation over the liver, when present, signifies tricuspid regurgitation.

Palpation.—A systolic thrill over the apex is rarely present.

The impulse is strong and heaving so long as hypertrophy is maintained, to become weak and uneven when this is lost.

The *pulse* is normal or regular in rate and rhythm and of full volume as long as compensation is maintained. Later when cardiac failure is imminent it is irregular in force and rhythm. Not every ventricular contraction may produce a radial pulse.

Percussion.—The transverse dullness of the heart is increased to the left and slightly downward. In cases associated with right ventricular hypertrophy the cardiac dullness often extends well to the right of the sternum.

Auscultation.—Auscultation over the apex reveals a systolic murmur, with its point of maximum intensity located over the apex, and transmitted toward the left axilla, or even as far as the angle of the left scapula. The murmur is blowing and rather musical, partially or totally obscuring the first sound at the apex. The murmur varies in intensity with changes of posture. Thus, it may be absent in the recumbent, and audible in the erect posture. When present it is well brought out by muscular exertion. The pulmonic second sound is accentuated, or there may be a systolic tricuspid safety-valve leak. The aortic second sound is also accentuated due to hypertrophy of the left ventricle. Often this accentuated sound is clearly audible over the apex during diastole. (See Fig. 114, p. 233.)

Diagnosis.—The diagnosis of mitral insufficiency rarely presents any great difficulty. The presence of a systolic murmur at the apex, which is blowing and musical, transmitted toward the left axilla, associated with accentuation of the pulmonic second sound, and often of the aortic second as well, make a clear picture.

A *functional murmur* sometimes occurs at the mitral valve, though they are more frequent at the pulmonic valve. But still the mitral valve is the second place in frequency for these murmurs. But, as noted previously, these murmurs do not produce ventricular hypertrophy, nor are they propagated beyond the precordia, and usually occur in the anemic and debilitated. Moreover, functional murmurs are transient.

MITRAL STENOSIS

Pathology.—Stenosis of the mitral valve is usually a sequence of acute endocarditis arising during the course of rheumatism, tonsillitis, scarlatina, chorea, or other acute infectious disease. But there is another group of cases in which a slow sclerosis of

the cusps of the valve occurs without any history of infective disease.

As in other stenotic lesions, the valve cusps are sclerosed and shrunken, the edges often adherent, sometimes leaving a mere slit or chink, the "button-hole" orifice of Corrigan. In the slowly sclerotic cases the valves are seldom found adherent, but there is considerable thickening and shortening of the chordæ tendineæ, preventing proper co-aptation of the valve segment edges. In yet other cases segmental deformity is absent, but the ring is rendered smaller by the projection of calcareous masses from the margin of the ring, or as a result of congenital defect.

In mitral stenosis, when compensation is maintained the myocardium of both left auricle and left ventricle is thickened. The ventricular hypertrophy develops as the result of the aspirating action of this chamber in drawing its proper amount of blood through the narrowed orifice and because it must contract powerfully upon the smaller amount of blood to force it into the aorta. The auricular hypertrophy develops with the efforts of the auricle to expel its increased contents through a narrowed orifice.

Physical Signs.—*Inspection.*—In thin chested patients and in young children there is visible bulging of the lower and right portions of the precordia the result of right ventricular hypertrophy. The cardiac impulse is strong, but its area is diminished. It is displaced to the left, but seldom downward. Systolic pulsation of the epigastrium is commonly seen, as the result of the powerful contractions of the hypertrophied right ventricle.

Palpation.—Over the mitral valve area over the apex there is a pre-systolic thrill which is pathognomonic of the disease. The pulmonic valve shock is accentuated at the left second interspace near the sternum.

The *pulse* is small and of low tension, regular during compensation, irregular when dilatation is imminent.

Percussion reveals an increase in the transverse diameter, particularly to the right of the sternum owing to right auricular and ventricular hypertrophy, and to a minor extent to the left of the normal cardiac outline, as a result of left ventricular hypertrophy.

Auscultation reveals a pre-systolic murmur over the apex, which is not transmitted beyond the precordia. This murmur is harsh, ingravescent or crescendo in quality, increasing steadily in intensity to its termination, and is commonly followed by a snapping first sound at the apex. This murmur must be differentiated

and distinguished from the Flint murmur occurring during aortic regurgitation. The pulmonic second sound is accentuated, and occasionally a "safety-valve" systolic murmur is audible over the tricuspid area. The second sound of the heart at the apex is feeble, either from imperfect filling of the aorta by the smaller content of the left ventricle, or because the hypertrophied right ventricle intervenes between the aortic valve and the chest wall. (See Fig. 113, p. 232.)

Diagnosis.—A pre-systolic murmur at the mitral area, ingravescent or crescendo in quality, followed by a snappy first sound, and an enfeebled second sound; an accentuation of the pulmonic second sound; extension of the transverse dullness of the heart; a pre-systolic thrill at the apical area; and the small pulse of low tension, suggest the diagnosis of mitral stenosis. The murmur of Austin Flint must be eliminated before the case is pronounced one of mitral stenosis.

PULMONARY INSUFFICIENCY

Pathology.—Pulmonary insufficiency or regurgitation is a very rare condition. When found, it is usually due to a congenital deformity of the pulmonary valve, only in rare instances being due to chronic endocarditis, the sequence of rheumatism or other form of sepsis.

Physical Signs.—*Inspection.*—There is moderate precordial bulging chiefly to the right of the sternum or over the ensiform cartilage. The impulse lies behind the sternum and is invisible, but in some instances may be displaced to the right of this bone.

Palpation rarely reveals a thrill over the pulmonary valve area; more frequently it shows a systolic pulsation to the right of the sternum from the second to the sixth interspaces. The pulse is as a rule quite unchanged.

Percussion shows an increase in the transverse dullness of the heart to the right of the sternum, with seldom any increase toward the left and downward.

Auscultation reveals a diastolic murmur with its point of maximum intensity at the pulmonary area, transmitted downward along the left border of the sternum. The murmur is blowing and not unmusical. It masks or replaces the second sound of the heart at the pulmonary area. (See Fig. 120, p. 237.)

Diagnosis.—The diagnosis rests upon the physical signs described; but it must be borne in mind that while murmurs are

frequently audible at the pulmonary area, they are usually functional and systolic. Functional murmurs must be excluded.

PULMONARY STENOSIS

Pathology.—Stenosis at the pulmonary valve is nearly always due to a congenital defect. Very rarely the lesion is the sequence of ulcerative endocarditis attacking the pulmonary valve.

Physical Signs.—*Inspection.*—The precordia in children and thin chested individuals is apt to bulge, the cardiac impulse is diffuse and feeble, and there is often a systolic pulsation to the right of the sternum.

Palpation reveals usually a systolic thrill at the pulmonary area. The pulse is unaltered. (See Fig. 94, p. 203.)

Percussion reveals an increase in the transverse dullness of the heart to the right of the sternum, with little or no increase to the left and upward.

Auscultation.—A systolic murmur is audible at the pulmonary area, which is transmitted upward into the great vessels of the neck. The murmur is usually harsh and unmusical. The first sound at the tricuspid area is often replaced by the safety-valve leak of tricuspid insufficiency. Occasionally a double murmur, systolic and diastolic is audible at the pulmonary area, due to a lesion which produces both stenosis and insufficiency.

Diagnosis.—The physical signs described when encountered in a patient who is not the subject of valvular disease of the left side of the heart, suggests the probability of a true and isolated pulmonary lesion. (See Fig. 119, p. 237.)

TRICUSPID INSUFFICIENCY

Pathology.—Tricuspid insufficiency occurs very rarely as a result of congenital lesions of the valve, the vast majority of the cases being instances of relative insufficiency due to lesions of the left side of the heart. The relative insufficiency results from increased tension in the pulmonary circuit, usually produced by regurgitant lesions of the left heart; though a similar condition may arise as the result of heightening of the pressure in the pulmonary circulation by obstruction to the flow of blood offered by obstructive pulmonary lesions, as emphysema and tuberculosis.

Physical Signs.—*Inspection.*—Systolic jugular pulsation is a

very reliable sign of tricuspid insufficiency, as is also a systolic pulsation of the liver. The cardiac impulse is rather diffuse and its intensity is decreased.

Palpation shows the weak apical impulse, and bi-manual palpation detects a hepatic systolic pulsation and differentiates it from the transmitted impact of a hypertrophied right ventricle. The *pulse* is weak, but regular.

Percussion shows extension of the area of cardiac dullness to the right caused by right auricular and ventricular hypertrophy or dilatation; and not infrequently an associated increase in the transverse diameter to the left, owing to hypertrophy or dilatation of the left ventricle associated with the left sided valvular lesion which has resulted in the relative lesion at the tricuspid orifice.

Auscultation reveals a systolic murmur over the tricuspid area at the lower border of the sternum. The murmur is not harsh and is transmitted upward and toward the left. The pulmonic second sound is enfeebled because of the small output of the right ventricle during systole. There may be associated left sided valvular lesions which were responsible for the tricuspid lesions. (See Fig. 118, p. 236.)

Diagnosis.—The presence of the systolic murmur at the tricuspid area, the extension of cardiac dullness to the right, the weakened pulmonic second sound, and often signs of left sided valvular lesions, make the diagnosis not difficult, particularly in the presence of systolic jugular pulsations and hepatic pulsation.

TRICUSPID STENOSIS

Pathology.—Tricuspid stenosis is a very rare lesion, which is very seldom diagnosed during life. It is usually due to congenital narrowing or stenosis of the valve.

Physical Signs.—If there are any physical signs, there will be a palpable thrill at the tricuspid area, pre-systolic in time. The patients are commonly dyspneic and cyanotic; there is a pre-systolic murmur at the tricuspid area, which is not transmitted thence; while there is extension of the area of cardiac dullness to the right of the sternum. (See Fig. 117, p. 236.)

CHAPTER XIX

DISEASES OF THE MYOCARDIUM

ACUTE MYOCARDITIS (ACUTE MYOCARDIAL DEGENERATION)

Pathology.—Acute myocarditis occurs clinically in two forms: as (1) acute parenchymatous myocarditis; (2) acute interstitial myocarditis.

Acute parenchymatous myocarditis occurs as a complication of acute infectious febrile diseases, notably pneumonia, typhus and typhoid fevers, scarlatina, and diphtheria. An acute myocarditis arising during these diseases usually has its inception during the active febrile stage of the disease at which time it may be the cause of sudden death; but in other instances its incidence is deferred, the condition developing and sometimes causing death during late convalescence. Acute parenchymatous myocarditis also occurs as a complication of endocarditis and pericarditis, but not by direct extension of the inflammatory process to the myocardium.

While the name myocarditis implies an inflammation, the signs of acute inflammation are not in evidence, the characteristic changes in the myocardium being those of a slow degeneration or metamorphosis, ranging in severity from cloudy swelling in the favorable cases to fatty degeneration in the grave cases.

Grossly the heart presents a varied picture, being pale or grayish-red in the presence of cloudy swelling; yellowish in fatty degeneration, and gray in hyaline degeneration. It is probable that the earliest retrogressive change in acute parenchymatous myocarditis, in those cases which yield the best prognosis, is cloudy swelling; and that the ultimate change is a fatty degeneration, in which the prognosis is extremely grave.

Acute Interstitial Myocarditis also develops during the course of acute fevers, Leyden first describing the disease in connection with scarlatina. Rhomberg has noted the changes characteristic of the disease in diphtheria, typhoid fever, acute rheumatic fever, and variola. The disease occurs in two grades or degrees of se-

verity: (1) the transient *non-suppurative interstitial myocarditis;* and (2) the more grave *suppurative interstitial myocarditis.* In the former the intermuscular spaces of the myocardium are infiltrated with leukocytes, the coronary capillaries are dilated, and the muscle bundles present areas of vacuolation, nuclear multiplication, and pigmentation; but the morbid condition often eventuates in resolution without the formation of fibrous connective tissue between the muscle bundles.

The suppurative form of the disease, of more grave prognostic significance, is usually the sequence of infectious embolism of the coronary arteries. When one of the terminal vessels of this arterial system becomes occluded by a simple, non-infectious embolus, anemic or hemorrhagic infarction of the myocardium is apt to ensue; but if the occluding embolus contains infectious material, if its source happens to be a vegetation from the cusp of a cardiac valve the seat of infective endocarditis, or an area of infectious osteomyelitis, a more serious lesion of the myocardium is produced. At the sites of obstruction circumscribed areas of leukocytic infiltration and bacterial colonization develop, leading to minute purulent infiltrations between the muscle bundles, weakening the heart wall and rendering rupture imminent. The rupture may occur externally into the pericardial sac with the production of a purulent pericarditis; or the pus may discharge into a cavity of the heart, the organisms being distributed to various organs of the body by the blood stream with the production of metastatic abscesses.

Instead of terminating by resolution, as in the case of acute non-suppurative interstitial myocarditis, the areas of purulent infiltration result in the formation of fibrous patches, weakening the cardiac musculature and predisposing to aneurism of the cardiac wall.

Physical Signs.—Physical signs in this disease are often lacking and when present are not clear and distinctive. The cardiac impulse as a rule shows a primary accentuation, with a subsequent weakening in its force and area. In certain cases sudden death may occur either during the height of an acute infectious disease or even late in convalescence. A fairly reliable diagnostic sign is an equalization of the intensity of the first and second cardiac sounds, both sounds assuming a valvular quality, with coincident signs of pulmonary stasis. Late in the course of the disease, when cardiac dilatation has supervened, the area of

cardiac dullness is extended and a relative mitral regurgitant murmur is commonly audible.

Diagnosis.—The diagnosis must be reached through the discovery of a causative factor rather than upon the physical signs, which are often few and confusing. But when during the course of an acute infectious disease the heart tones become enfeebled, equalized, and valvular, with alterations in rhythm as embryocardia or gallop-rhythm, the incidence of an acute myocarditis may be suspected.

Many cases simulate closely acute endocarditis; but in the latter disease the cardiac weakness is not so rapid and extreme, nor is the cardiac rhythm disturbed to an equal degree.

CHRONIC MYOCARDITIS (CHRONIC FIBROUS MYOCARDITIS; CHRONIC INTERSTITIAL MYOCARDITIS)

Pathology.—Chronic myocarditis is a slow sclerosis of areas of the myocardium, developing secondarily to changes in the coronary arteries. The essential lesion of the coronary circulation productive of chronic myocarditis is a narrowing or obliteration of the lumen, the result of obliterative endarteritis or embolic occlusion. Obliterative endarteritis of the coronary circulation is merely part-and-parcel of generalized arterio-sclerosis.

When an uninfectious embolus lodges in a terminal branch of a coronary artery an infarct of the myocardium is apt to form. The infarct is surrounded soon by a zone of infiltrating leukocytes. The infarct is wedge-shaped with the apex of the wedge directed toward the site of the embolism. The infarct, which may assume either the anemic or hemorrhagic type, leads to an area of softening of the myocardium, to which the term Myomalacia Cordis has been applied by Ziegler. This area is a point of lowered resistance to the endocardiac pressure and is liable to lead to rupture with sudden death. If rupture does not occur, the area of infarction is gradually replaced by fibrous connective tissue.

The portions of the myocardium usually attacked are usually the lower two-thirds of the anterior wall and the upper portion of the posterior wall of the left ventricle; the interventricular septum; or the bases of the papillary muscles.

Associated changes occurring in the heart the seat of chronic myocarditis comprise: (1) hypertrophy; (2) dilatation; (3) valvular disease. In cases of moderate involvement there is sufficient sound myocardium to compensate for the cardiac impair-

ment by hypertrophy. In more extensive fibrosis, however, there is little intact myocardium; the cardiac wall yields under the normal or increased endocardiac pressure; and dilatation becomes inevitable. Valvular disease develops in association with moderate cases of fibrosis when it involves the papillary muscles, even in the presence of cardiac hypertrophy. In cases in which the sclerotic process is extreme and associated with cardiac dilatation valvular disease is a constant accompaniment.

Physical Signs.—The physical findings in chronic myocarditis are not characteristic and distinctive, as they vary with the stage of the disease and the state of the myocardium. As in the acute form of the disease, the heart sounds are usually less muscular and more valvular in quality; they are accentuated in the presence of compensatory hypertrophy; and they are enfeebled after dilatation has supervened. The aortic second sound is frequently accentuated, as the patients often have arterio-sclerosis. The pulse is hard and tense, with stiff, unyielding arterial wall, and is often disordered in rhythm. The area of cardiac dullness is often found extended from cardiac hypertrophy or dilatation. Murmurs are encountered in the purely valvular cases and in cases associated with cardiac dilatation.

Diagnosis.—The diagnosis of chronic myocarditis is based partially upon the physical signs, but mainly upon the finding of arterio-sclerosis in a subject past middle life who presents such physical signs. Chronic dyspnea and signs of pulmonary congestion aid in suggesting the diagnosis.

CARDIAC HYPERTROPHY

Pathology.—Cardiac hypertrophy, an overgrowth of the musculature of the heart, with maintenance of its nutrition, may involve a single chamber of the heart, one side of the heart, or the entire organ. The portion most commonly involved is the left ventricle.

Thickening of the wall of the heart with enlargement of the chamber is termed eccentric hypertrophy. A similar mural change with decrease in the size of the chamber is termed concentric hypertrophy, a condition which has not been demonstrated to the satisfaction of many clinicians.

The cause of cardiac hypertrophy is increased work thrown upon the heart while its nutrition is maintained. The causes of this overwork may reside within the heart; or without the viscus.

Among the commoner causes of cardiac hypertrophy may be mentioned:

1. Continued excessive muscular exertion, as noted in athletes and laborers.

2. Interference with the cardiac action by pericardial adhesions.

3. Over-eating habitually, and excessive drinking of beer.

4. Diseases of the nervous supply of the heart leading to cardiac over-action.

5. Arterio-sclerosis and nephritis, which by raising the blood

Fig. 139.—Enormous hypertrophy of left ventricle due to prolonged increased peripheral resistance. Note that the whole anterior surface of the heart is occupied by the left ventricle. The right ventricle does not appear to be much affected. (From Warfield.)

pressure in the general circulation cause hypertrophy of the left ventricle.

6. Disease of the lung such as hypertrophic emphysema and chronic interstitial pneumonia, and left sided valvular lesions, which by raising the blood pressure in the pulmonary circuit cause hypertrophy of the right ventricle. Also congenital heart disease results in cardiac hypertrophy.

7. Hypertrophy of the left auricle is caused by mitral regurgitant or stenotic lesions.

8. Right auricular hypertrophy is caused by tricuspid stenosis or regurgitation.

9. Pregnancy causes some hypertrophy of the heart.

The hypertrophied heart is increased in size, sometimes to such an extent that it constitutes the cor bovinum.

The contour or shape of the hypertrophied heart varies in different degrees and varieties of hypertrophic change. In total hypertrophy of the heart the organ is round. In left ventricular hypertrophy and right ventricular hypertrophy respectively these portions of the heart are abnormally large.

Physical Signs.—*Inspection.*—In **left ventricular hypertrophy** the cardiac impulse is forcible and heaving and displaced downward and to the left. There is precordial bulging especially notable in children and women, and often systolic pulsations in the carotid arteries. The extent or area of the cardiac impulse is increased.

Palpation confirms the displacement of the apex beat and its firm heaving quality. The valve shock over the aortic valve is increased. The pulse is regular, full, and tense. The cardiac impulse is not fast; it is slow and heaving; does not occur quickly, but slowly and in a heaving manner. The impulse may be found in the sixth or even in the seventh interspace.

The cardiac impulse may not be either visible or palpable if hypertrophic emphysema causes the anterior borders of the lung to come between the heart and chest wall.

Percussion.—The transverse dullness of the heart is increased to the left and downward. It may extend past the mid-clavicular line and well beyond the right of the sternum.

Auscultation shows accentuation and sometimes reduplication of the aortic second sound. The first sound at the apex is also accentuated. There is in some cases a systolic murmur audible at the mitral area. A tinkling sound may be heard sometimes to the right of the cardiac apex. A cardio-repiratory murmur may be encountered in some instances due to the strong impact of the hypertrophied ventricle against a portion of lung anchored anterior to the heart by pleural adhesions. When the valves are not diseased the first sound of the heart in addition to being accentuated is rather prolonged. When the hypertrophy is caused by valvular lesions these are audible, or when dilatation is about to supervene, relative murmurs may be heard due to stretching of the mitral ring.

Right Ventricular Hypertrophy

Inspection.—There is undue prominence or bulging of the lower sternum and epigastric pulsation, systolic in time. There is also

systolic impulse to the right of the sternum in the sixth and seventh interspaces. Pulsation is often noted above these levels; the apex beat is displaced to the left; but there is little if any downward displacement.

Palpation reveals the presence of the pulsation at the lower end of the sternum and in the epigastrium; but the thrust is not as strong and as distinct as it is in the case of left ventricular hypertrophy. The valve shock over the pulmonic valve is stronger than is that over the aortic area. There is occasionally a palpable impulse transmitted to the liver by the over-acting heart, which should not be confused with the systolic pulsation of that organ occurring with tricuspid regurgitation.

Percussion.—Percussion shows that the area of cardiac dullness is increased toward the right, sometimes extending an inch to the right of the sternum or more.

Auscultation.—The tricuspid first sound is accentuated and somewhat prolonged, and a systolic "safety-valve leak" is sometimes demonstrable when ventricular failure is imminent. The pulmonic second sound is accentuated. Reduplication of the second sound is not infrequent.

The *pulse* is of small volume but regular, unless dilatation is imminent, in which case it is arrhythmic.

Left Auricular Hypertrophy

Left auricular hypertrophy can seldom be ascertained by physical means. An extension of cardiac dullness to the left of the sternum in the second and third interspaces is suggestive when found, and, if the hypertrophy is due to mitral stenosis, the pre-systolic murmur of this condition may be heard. Or, if it is due to mitral regurgitation, this murmur may be audible.

Right Auricular Hypertrophy

There is apt to be an increase in the area of heart dullness to the right side of the sternum in the third and fourth interspaces. Pre-systolic pulsation to the right of the sternum in this region sometimes is seen. These signs with the signs of right ventricular hypertrophy and a systolic murmur at the tricuspid valve are very suggestive of right auricular hypertrophy. Systolic pulsations in the jugular veins are common and there are usually signs of engorgement of the general venous system.

Diagnosis.—Cardiac hypertrophy is indicated by a well defined set of physical signs. These are:

1. The heaving character of the cardiac impulse.
2. The increase in the area of cardiac dullness.
3. Accentuation of the second sounds of the heart.
4. The hard tense pulse of full volume.

Certain other conditions simulate cardiac hypertrophy and require differentiation.

Thus fibroid retraction of the left lung may cause a wide impulse simulating cardiac hypertrophy. But the physical signs of cirrhosis of the lung or chronic interstitial pneumonia are quite sufficient to render a differential diagnosis comparatively easy.

Neurotic conditions such as the over-use of stimulants or tobacco, or exophthalmic goiter, cause vigorous heart action occasionally simulating true cardiac hypertrophy, but are differentiated with comparative ease. In these conditions the impulse is less diffuse and heaving, and there are ameliorations and aggravations of the attacks.

In pericardial effusion the increased cardiac area is triangular or pear-shaped with the base directed downward toward the diaphragm, and the heart sounds are feeble, and the pulse paradoxic.

In cardiac dilatation the heart sounds are feeble, but distinct, and the pulse is feeble and irregular, and cardiac murmurs may be detected. There are also signs of general venous stasis.

CARDIAC DILATATION

Pathology.—Cardiac dilatation is an enlargement of the chambers of the heart, due to stretching of the cardiac walls, in which the chambers are unable to expel their contents during systole. Clinically cardiac dilatation may be said to have its inception when the heart is no longer able to empty itself during systole.

The cause of cardiac dilatation resides in increase of intraventricular tension, or weakening of the cardiac wall. In many instances there is a combination of these two factors at work in the production of cardiac dilatation.

Among the factors which tend to produce cardiac dilatation may be mentioned:

1. Prolonged muscular exertion, notably in mountain climbing and foot racing.

2. Sudden exertion in the presence of a valvular lesion or chronic myocarditis.

3. Valvular heart lesions. In stenosis or regurgitation at the aortic valve the left ventricle first hypertrophies and then dilates; a mitral regurgitation develops from stretching of the auriculo-ventricular ring. The left auricle hypertrophies in the effort to compensate for the insufficiency and then dilates. The blood is

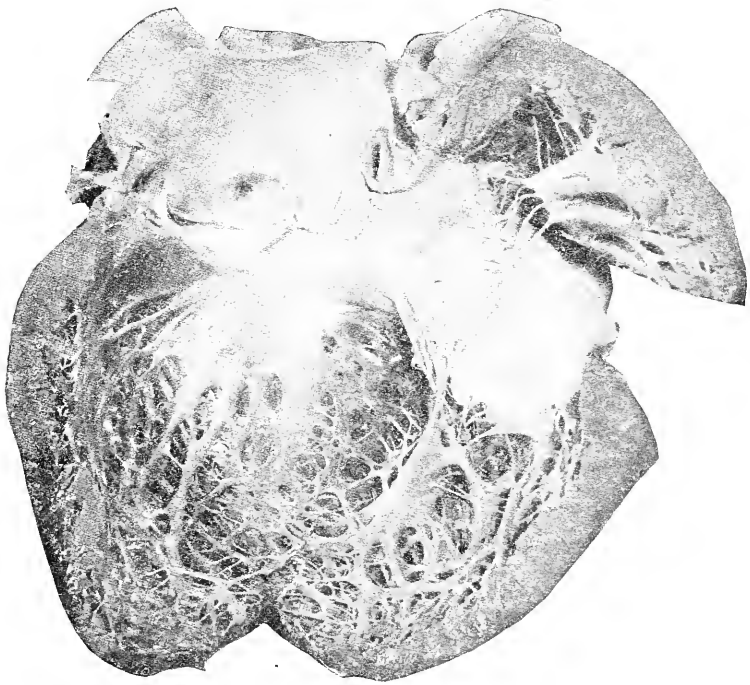


Fig. 140.—Aortic incompetence with hypertrophy and dilatation of left ventricle, the result of arteriosclerosis affecting the aortic valves. Note how the valves have been curled, thickened, and shortened, the edges of valves being a half inch below the upper points of attachment. The anterior coronary artery is shown, the lumen narrowed. (Reduced one-half.) (From Warfield.)

dammed back into the pulmonary circuit and the pressure therein raised, leading to catarrhal condition of the bronchial tubes and to right ventricular hypertrophy; and later dilatation and insufficiency; and then the right auricle hypertrophies and dilates, with a leak at the tricuspid valve, the ultimate result being venous congestion of the general circulation, with pulsation of the liver and edema of the lower extremities or anasarca.

4. The toxins of infectious diseases, notably influenza, typhoid fever, pneumonia, and erysipelas, affecting the myocardium produce dilatation or yielding of the weakened cardiac walls.

5. Conditions of malnutrition and anemia weaken the myocardium and lead to dilatation.

6. Causes raising arterial pressure in the general circulation, as arterio-sclerosis and nephritis.

7. Conditions raising arterial pressure in the pulmonary circulation as hypertrophic emphysema, chronic interstitial pneumonia or other obstructive lung disease.

Thus it is apparent that cardiac dilatation is most commonly the result of valvular lesions, regurgitant or stenotic, and is usually preceded by cardiac hypertrophy.

Cardiac dilatation is associated with relative insufficiency caused by stretching of the auriculo-ventricular rings, so that although the valve leaflets are themselves not diseased, they are unable to co-aptate and close the abnormally large orifice. Moreover, the chordæ tendineæ and papillary muscles do not share in the dilatation, do not stretch, but being of normal length, do not permit the co-aptation of the valve edges.

As in cardiac hypertrophy, the contour of the heart varies with the type or degree of dilatation present. When dilatation involves all four chambers of the heart, the heart is rather round or spherical. When a single chamber, or one side of the heart is dilated, the contour is irregular. The right ventricle is anatomically liable to a greater degree of dilatation than is the left ventricle; and the left auricle to more than is the right auricle.

The myocardium is thinner than normal and shows different stages of fatty or albuminoid degeneration.

Physical Signs.—*Inspection.*—The cardiac impulse is diffuse and often undulatory in type. It is often displaced to right or left. In extreme dilatation, when the auricles are involved, pulsation to the right of the sternum in the third interspace is occasionally visible.

Palpation.—The cardiac impulse is hard to define and is weak in strength, or is altogether impalpable. A visible diffuse cardiac impulse which is not palpable is of great diagnostic significance. Epigastric tenderness can usually be elicited in right ventricular dilatation. The *pulse* is weak and irregular.

Late in the course of right sided cardiac dilatation the systolic pulsation of the liver becomes palpable.

Percussion.—The area of cardiac dullness is increased to the

right and left, and, in auricular dilatation, along the left border of the sternum as high as the second interspace. If emphysematous lung intervenes between the heart and the thoracic wall, it may obscure to a great extent the cardiac enlargement.

Auscultation.—The cardiac sounds upon auscultation of the precordia are enfeebled. The first sound is not infrequently reduplicated, as also may be the second sound. The pulmonic sound is strong if the left side of the heart is dilated while the right is hypertrophied. This sound is weak or feeble if the right heart participates in the dilatation. Gallop-rhythm or canter-rhythm is not infrequent in the late stages of the disease. Murmurs due to mitral or tricuspid regurgitation, as well as aortic and pulmonary murmurs are usually audible.

Diagnosis.—The character of the heart sounds, the increased extent of the cardiac dullness, the diffuse, wavy and displaced cardiac impulse, and signs of systemic venous engorgement, render the diagnosis not difficult.

The enlarged heart of *cardiac hypertrophy* is differentiated by the strong cardiac impulse, distinctly visible and displaced, the accentuated second sounds, the full and regular pulse, and the absence of signs of cardiac failure as venous congestion.

From *pericarditis with effusion* the differential points are mentioned under that disease (page 258).

Increased dullness to the left of the heart due to consolidation of the lung is usually differentiated by the presence of bronchial breath sounds and rales.

The cardiac area may be broadened by pushing of the heart forward by the pressure of a mediastinal tumor, but these tumors produce pressure symptoms which are not present in cardiac dilatation.

Encysted pleurisy may be confused with cardiac dilatation by broadening the area of dullness around and adjacent to the heart; but there is usually the friction rub, and the heart sounds are not changed and there are no signs of cardiac or circulatory failure.

The presence of hypertrophic emphysema by interposition of the anterior borders of the lungs between the heart and chest wall may mask the cardiac enlargement to a variable degree, and require careful percussion to bring out the increased size of the organ.

CONGENITAL HEART DISEASE

Pathology.—Pulmonary stenosis is the most frequent and clinically the most important of the congenital lesions of the heart. The stenosis may be complete, the orifice of the vessel being closed by a fibrous membrane, or may permit all grades of patency. In addition to obstruction at the valve there may be narrowing of the conus arteriosus of the right ventricle, or the

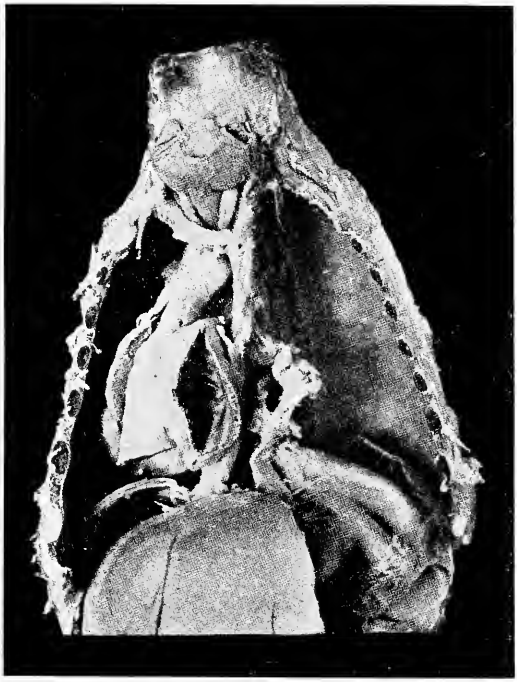


Fig. 141.—Reptilian heart. (From Delafield and Prudden.)

pulmonary artery may be congenitally narrow beyond the valvular opening.

Probably the second most common congenital lesion of the heart is the patent foramen ovale, which normally closes during the first week of extrauterine life, but which may remain partially open to adult life in from 2 to 5 per cent of persons.

The interauricular septum may be found absent, resulting in the reptilian heart or cor triloculare. In other instances both the interauricular and interventricular septa are absent, the heart consisting of only two chambers, the cor biloculare.

In a certain number of cases the ductus arteriosus, which usually closes during the first month of extrauterine life remains patent to give rise to signs of congenital heart disease.

Congenital lesions of the aortic, mitral and tricuspid valves are rarely encountered. Of the various valves of the heart, there may be supernumerary cusps or a diminution of the number of cusps, or adhesions between them, or merely a button-hole slit in a membrane closing a valve.

In certain rare instances the aorta is found to arise from the right ventricle and the pulmonary artery from the left ventricle; while in conditions of visceral transposition the heart lies chiefly in the right side of the thorax.

Physical Signs.—The physical signs of congenital heart disease are early apparent, the most striking sign being extreme blueness or cyanosis of the child. However, cyanosis may be absent in the presence of congenital cardiac disease. The fingertips are often clubbed, the so-called Hippocratic fingers. Dyspnea is always present.

As pulmonary stenosis is the most frequent underlying lesion there is in most cases a systolic blowing murmur to the left of the sternum in the pulmonic area, with signs of hypertrophy of the right ventricle. On the other hand, a patent ductus arteriosis yields a rather prolonged systolic murmur in the same area which is, however, more distinctly audible in the third left interspace.

Diagnosis.—A diagnosis of congenital heart disease can be readily made in most instances on the extreme cyanosis, dyspnea, clubbed fingers, and loud blowing murmurs. However, it is often very difficult to say with certainty just what the underlying lesion is, as the signs are often confusing and often two conditions coexist in the same case. The murmurs of congenital disease are very difficult to differentiate from functional murmurs in anemic children; but it should be borne in mind in this connection that functional murmurs are not transmitted and do not produce alterations in the myocardium, as hypertrophy. From acquired heart disease, congenital disease is usually differentiated by the fact that it is present from birth, the baby often having been from birth very blue (morbus coeruleus); that the child is under two years of age, at which time acquired lesions are practically unknown; and that the murmurs are atypical in location and transmission.

PART II. THE ABDOMEN

SECTION I

GENERAL EXAMINATION OF THE ABDOMEN

CHAPTER XX

CLINICAL ANATOMY OF THE ABDOMEN

The abdomen, the portion of the trunk which is limited above by the ensiform cartilage and costal arch, and below by the pubic crest and Poupart's ligaments, has a roughly oval form, the shape varying, however, with the age and sex of the subject. Thus, the abdomen of a child is roughly conical with the apex inferiorly; while in the adult female, owing to the broad pelvis in this sex, it is roughly conical with the apex above; whereas in the adult male the abdomen is oval or barrel-shaped, with a moderate antero-posterior flattening.

The *abdominal cavity* is limited above by the lower surface of the diaphragm, and inferiorly by the levator ani, assisted by the coccygeus, these two muscles constituting the pelvic diaphragm. The more roomy upper portion of the abdominal cavity, above the brim of the pelvis is termed the *abdomen proper,* while the smaller portion below the pelvic inlet is termed the *pelvis.*

The abdominal cavity is not limited above by the costal arch, which forms its upper boundary upon the surface of the abdomen, but by the diaphragm, extending upward into the bony thorax for some distance. On the right side its upper limit is on a level with the upper border of the fifth rib in the mid-clavicular line; while on the left side its upper limit is approximately one-half inch lower in the same line.

The *abdominal wall* is composed largely of muscle and soft structures, reinforced in certain regions by bony structures. Anteriorly and laterally the wall is formed of the abdominal muscles, the lower ribs, and the iliac bones. Posteriorly it is formed by the

posterior abdominal muscles, the quadratus lumborum and psoas on either side, and in the median line by the vertebral column. The anterior abdominal wall and the lateral walls between the last rib and the iliac crest are devoid of bony support and are subject to distention and retraction, depending upon the state of the abdominal contents.

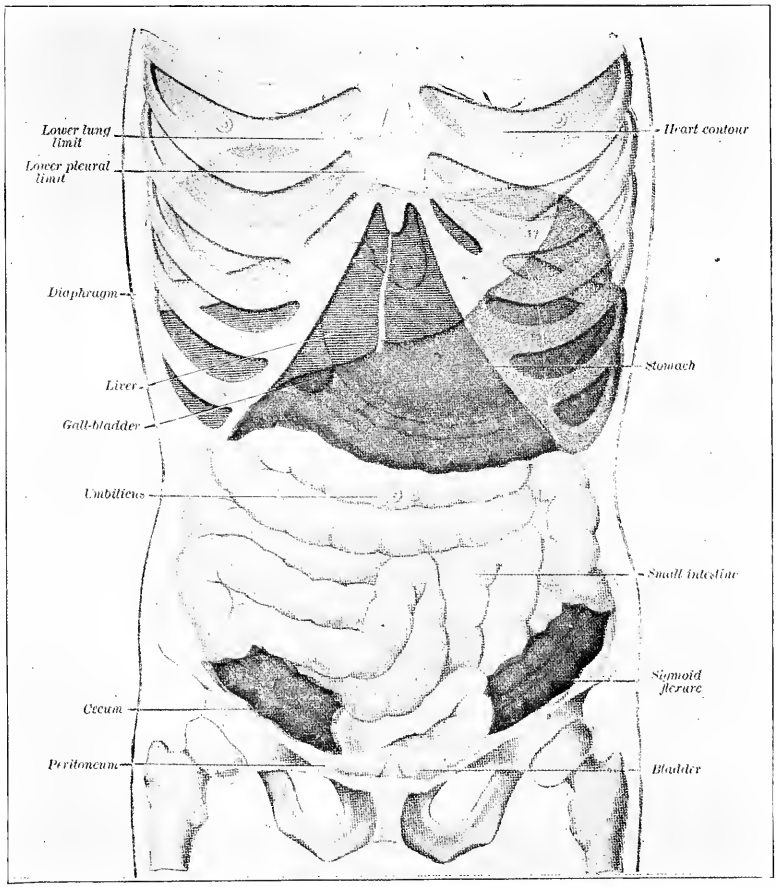


Fig. 142.—Relations of abdominal and thoracic viscera. (From Gray.)

Anatomical Landmarks of the Abdomen.—At the upper limit of the anterior abdominal surface in the median line is the *ensiform cartilage*, with the *costal arch* descending from it upon either side. The anterior extremities of the seventh, eighth, ninth and

tenth costal cartilages are palpable, and, in thin patients, the free extremities of the eleventh and twelfth ribs as well.

At the lower limit of the abdomen the *symphysis pubis* with its *pubic spines* are found, and extending from them in an upward and outward direction *Poupart's ligaments* on either side.

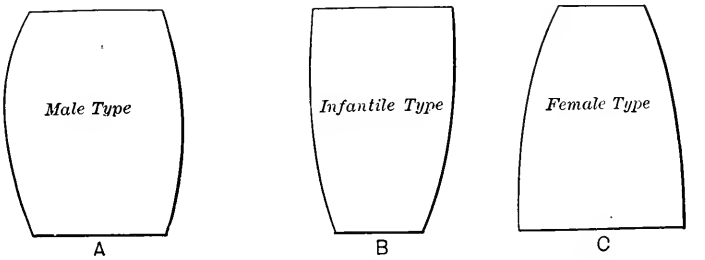


Fig. 143.—Schematic outlines of abdominal contour. (From Gray.)

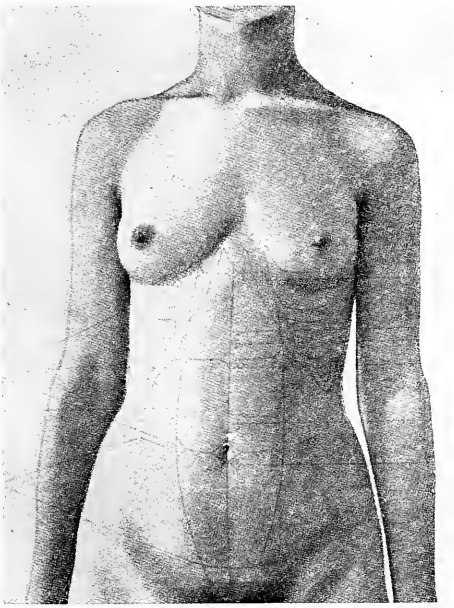


Fig. 144.—Showing the surface and bony landmarks of the abdomen and the location of the abdominal aorta and its more important branches. (From Butler.)

At the lower and lateral regions the *iliac crest*, terminating anteriorly in the *anterior superior iliac spines* are encountered, the latter being plainly palpable in very obese subjects.

In the lower central region of the abdominal surface the *umbili-*

cus is noted. It corresponds to the disc between the third and fourth lumbar vertebræ.

The *linea alba* extends in the mid-line from the ensiform cartilage to the symphysis pubis. It is indicated by a slight groove in the median line of the abdomen above the umbilicus, and by a line of hair or of brown pigment (Linea Nigra) below the umbilicus.

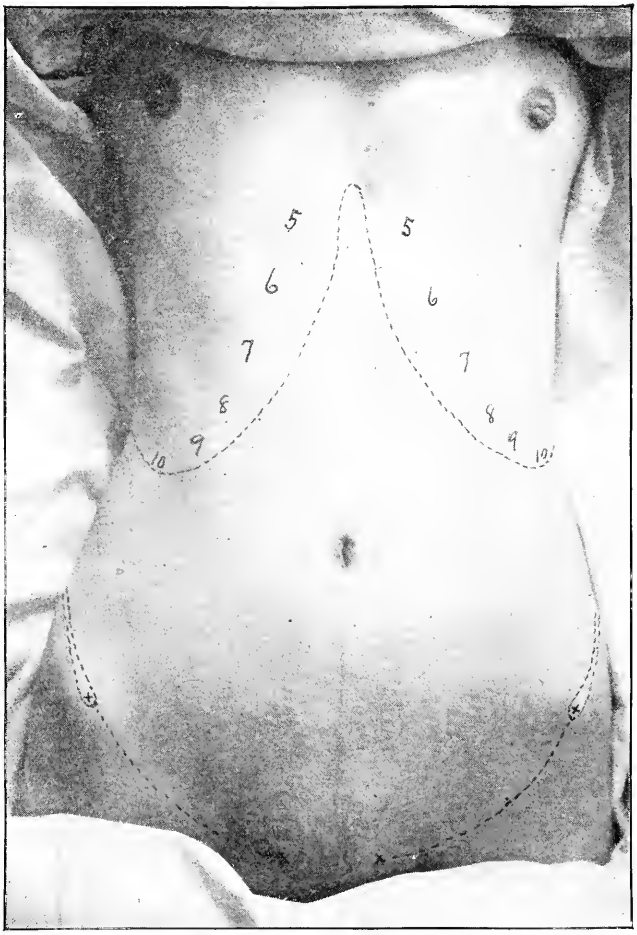


Fig. 145.—The abdominal surface with the rib margins and the iliac crests outlined. (From Crossen.)

The *linea semilunaris,* on either side of the abdomen extends with a slight convexity outward from the junction of the tip of the ninth costal cartilage with the outer border of the rectus

muscle to the pubic spine. It corresponds accurately to the outer limit of the sheath of the rectus muscle.

Lineæ transversæ are to be noted in subjects of good muscular development. They are transverse constrictions in the recti mus-

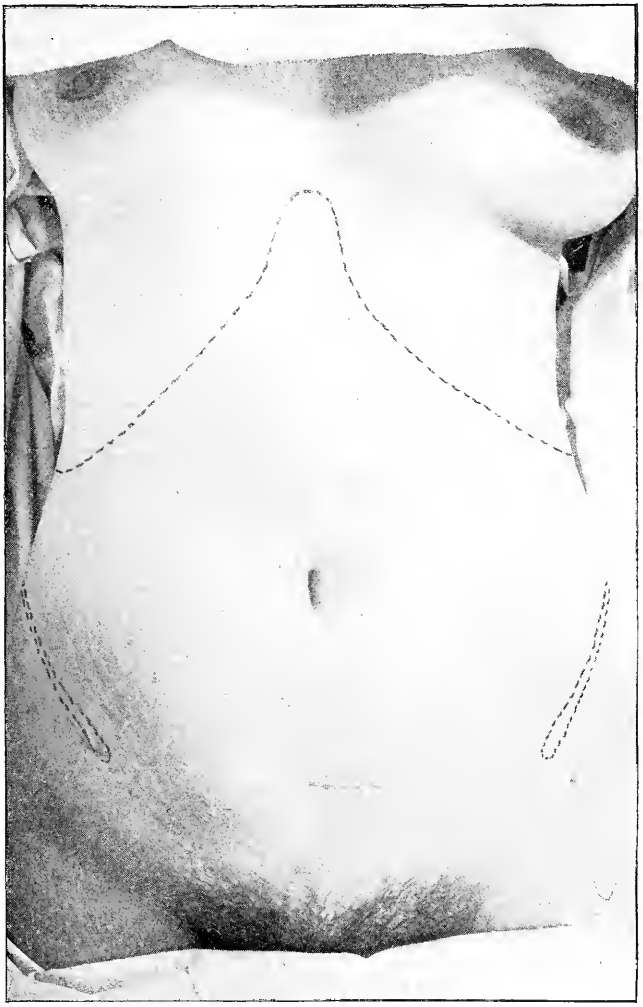


Fig. 146.—Another abdominal surface, with the ribs and crests outlined. This patient is rather stout. Notice how much the landmarks differ from those in Fig. 145. (From Crossen.)

cles. They are three in number, as a rule, although a fourth may sometimes be found. One is located at the ensiform cartilage, another at the umbilicus, and a third midway between the two. When

a fourth is present, it is located midway between the umbilicus and symphysis pubis.

Cutaneous flexion folds are encountered in obese subjects. They are usually two in number, one at the level of the umbilicus and the other just above the symphysis pubis.

Surface Markings.—The course of the *abdominal aorta* corresponds to a vertical line upon the surface of the abdomen extending from a point a little to the left of the ensiform process down-

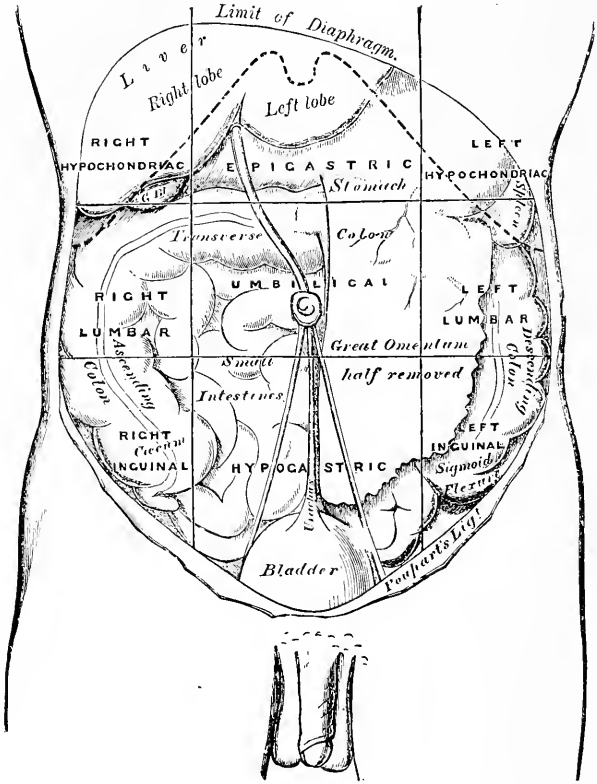


Fig. 147.—Anterior view of abdominal viscera *in situ*. (From Gray.)

ward to a point three-fourths of an inch below and a little to the left of the umbilicus, where the vessel bifurcates to form the common iliac arteries. (See Fig. 229, p. 368.)

The course of the *common iliac* and *external iliac* arteries corresponds to a line drawn from the point of bifurcation of the aorta to a point midway between the anterior superior iliac spine and the symphysis pubis.

The course of the *deep epigastric artery* is represented by a line drawn from the mid-point of Poupart's ligament upward and inward to the umbilicus.

The course of *inferior vena cava* is represented by a vertical line

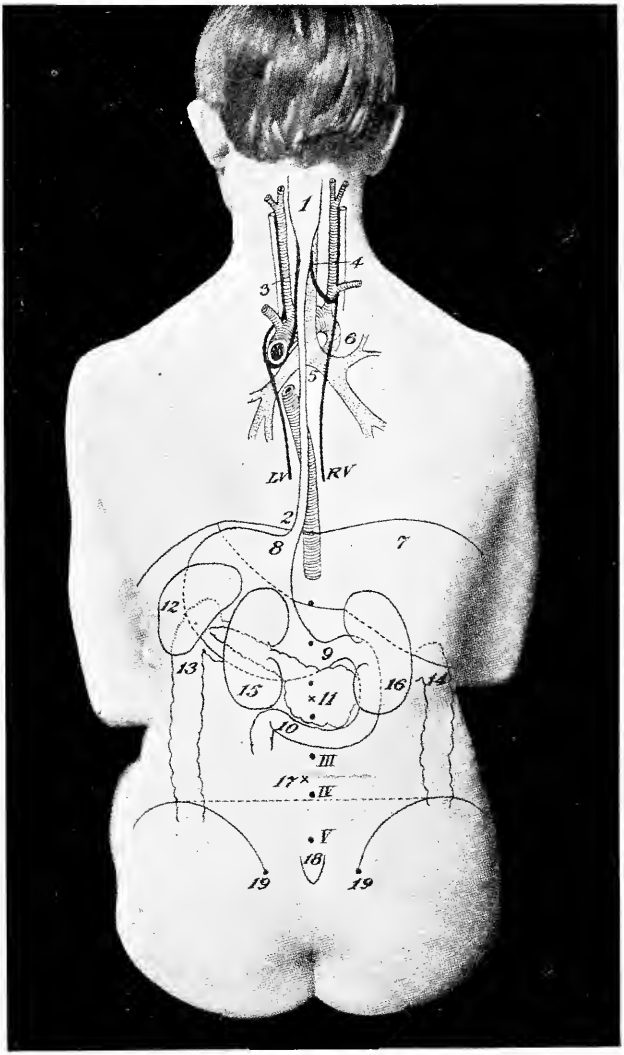


Fig. 148.—Surface markings of chief thoracic and abdominal viscera. Posterior view. (From Eisendrath.)

1, pharynx; *2*, esophagus; *3*, left recurrent laryngeal nerve; *4*, right recurrent laryngeal nerve; *5*, bifurcation of trachea; *6*, arch of aorta; *7*, liver; *8*, cardiac portion of stomach; *9*, and *10*, duodenum; *11*, head of pancreas; *12*, spleen; *13*, descending colon; *14*, ascending colon; *15*, left kidney; *16*, right kidney; *LV*, left vagus nerve; *RV*, right vagus nerve.

drawn along the line representing the course of the abdominal aorta, a little to its right side.

The *common* and *external iliac veins* are indicated by lines upon the abdominal surface slightly below and to the right of and corresponding in direction to the lines of the arteries of the same name.

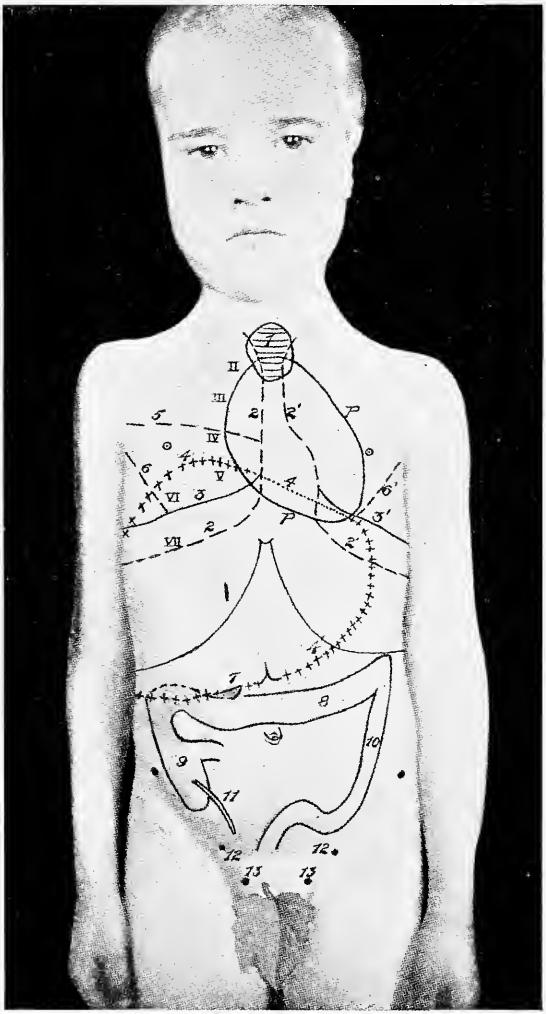


Fig. 149.—Relations of thoracic and abdominal viscera in the child. (From Eisendrath.) *1*, thymus gland; *2*, outline of right pleura; *2'*, left pleura; *3*, lower border of right lung; *3'*, lower border of left lung; *4*, upper border of liver (note large size of this organ in child); *4'*, lower border of liver; *5*, interlobar fissure between right upper and right middle lobes; *6*, interlobar fissure between right middle and right lower lobes; *6'*, fissure between left upper and left lower lobes; *7*, gall-bladder; *8*, transverse colon; *9*, ascending colon and cecum; *10*, descending colon; *11*, appendix; *12*, internal abdominal ring; *13*, external abdominal ring; *P*, pericardium.

The surface markings of the various abdominal organs are given in the sections dealing with the organs.

Topographical Anatomy of the Abdomen.—For purposes of description and to facilitate the localization of morbid conditions

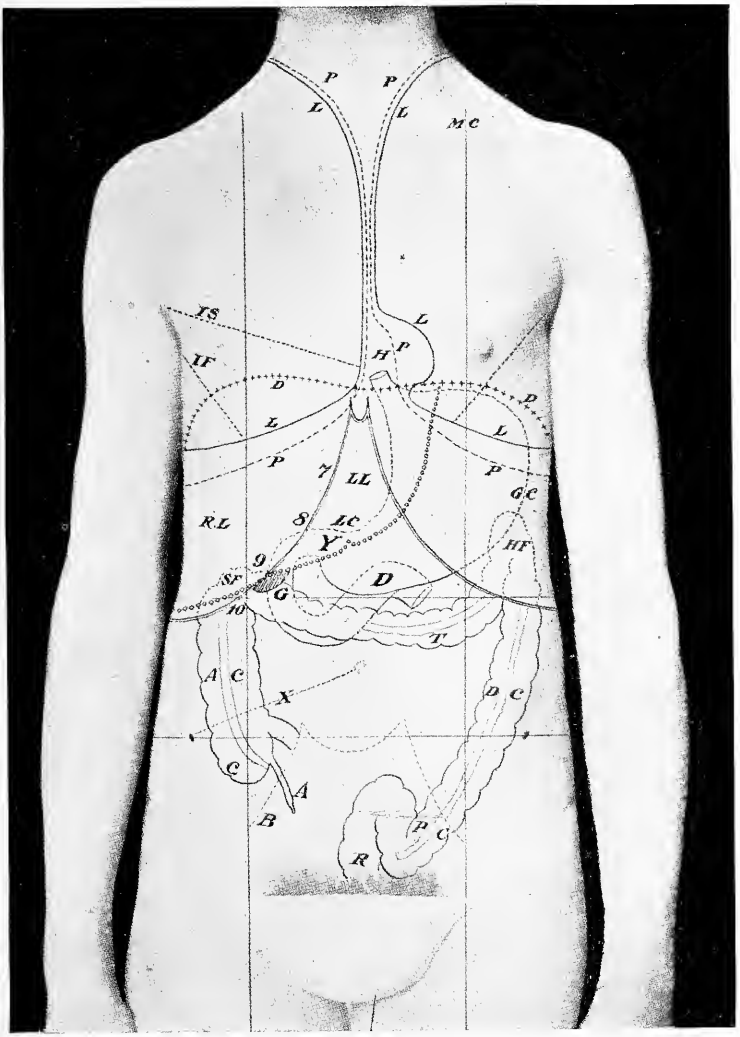


Fig. 150.—Surface markings of chief thoracic and abdominal viscera. Anterior view. (From Eisendrath.) *P*, pleura; *L*, lung; *MC*, midclavicular line; *D*, upper level of diaphragm; *RL*, right lobe of liver; *LL*, left lobe of liver; *LC*, lesser curvature of stomach; *GC*, greater curvature of stomach; *Y*, pylorus; *G*, gall-bladder; *SF*, hepatic flexure of colon; *HF*, splenic flexure of colon; *D*, ascending portion of duodenum; *Y* to *D*, horizontal and vertical portions of duodenum; *C*, cecum; *A*, appendix; *B*, pelvic brim; *X*, McBurney's point; *T*, transverse colon; *DC*, descending colon; *PC*, pelvic colon, or sigmoid flexure; *R*, rectum; *IS*, fissure between upper and middle lobes of right lung; *IF*, fissure between middle and lower lobes of right lung; *H*, portion of pericardium which is not covered by pleura.

arising within the abdominal cavity, the abdomen is divided into nine regions by four lines drawn upon the surface of the abdomen. Two of these lines are drawn around the body in a horizontal direction, while two are erected perpendicualrly upon its anterior surface. The upper horizontal line, the *sub-costal line* is drawn around the body at the level of the most dependent portion of the tenth costal cartilage. The lower horizontal line, the *inter-*

Fig. 151.—The usual anatomic division of the abdomen into nine regions by two transverse lines and two vertical lines. The upper transverse line is at the level of the cartilages of the tenth ribs, and the lower with the highest points of the iliac crests. The two parallel vertical lines pass through the cartilages of the eighth ribs and the middle of Poupart's ligaments. (From Crossen.)

tubercular line, encircles the trunk at the level of the tubercle which is found upon the iliac crest two inches behind the anterior superior iliac spine. The two perpendicular lines are projected upward from the mid-point of Poupart's ligament upon either side, blending with the mid-clavicular lines of the thorax.

By means of these lines the abdominal cavity is divided into nine

arbitrary regions. The *epigastric region,* bounded inferiorly by the sub-costal line, and superiorly and laterally by the costal arch, overlies the stomach, duodenum, liver, gall-bladder, pancreas and kidneys.

The *left hypochondriac region,* limited inferiorly by the sub-costal line and internally by the line of the costal arch, overlies

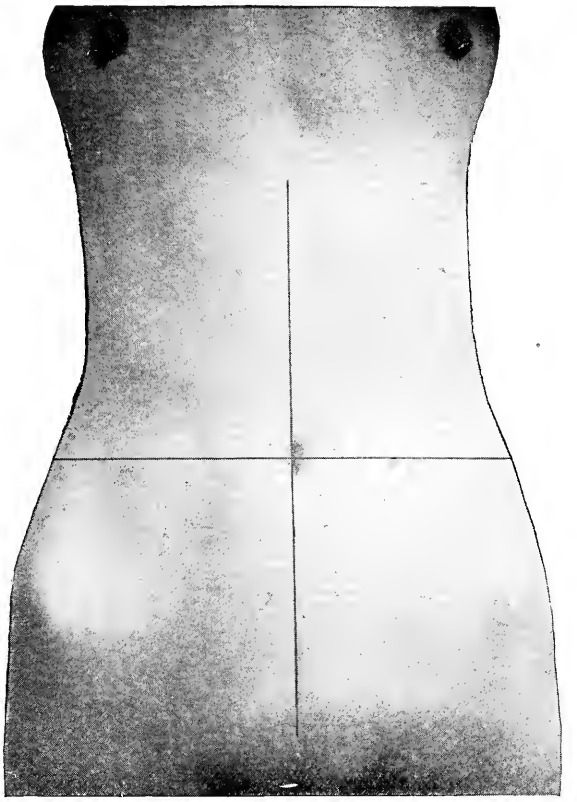


Fig. 152.—The abdominal surface divided into quadrants. (From Crossen.)

the fundus of the stomach, the spleen, and the splenic flexure of the colon.

The *right hypochondriac region,* limited inferiorly by the sub-costal line and internally by the line of the right costal arch, overlies the portion of the abdominal cavity which is occupied by the liver and right kidney.

The *umbilical region,* limited above by the sub-costal line, inferiorly by the inter-tubercular line, and laterally by the right and

left mid-Poupart lines, overlies the small intestines, the mesentery, the great omentum, the kidneys, and the transverse colon.

The *left lumbar region*, bounded above by the sub-costal line, below by the inter-tubercular line, and internally by the left mid-Poupart line, overlies the left kidney, the descending colon, and small intestine.

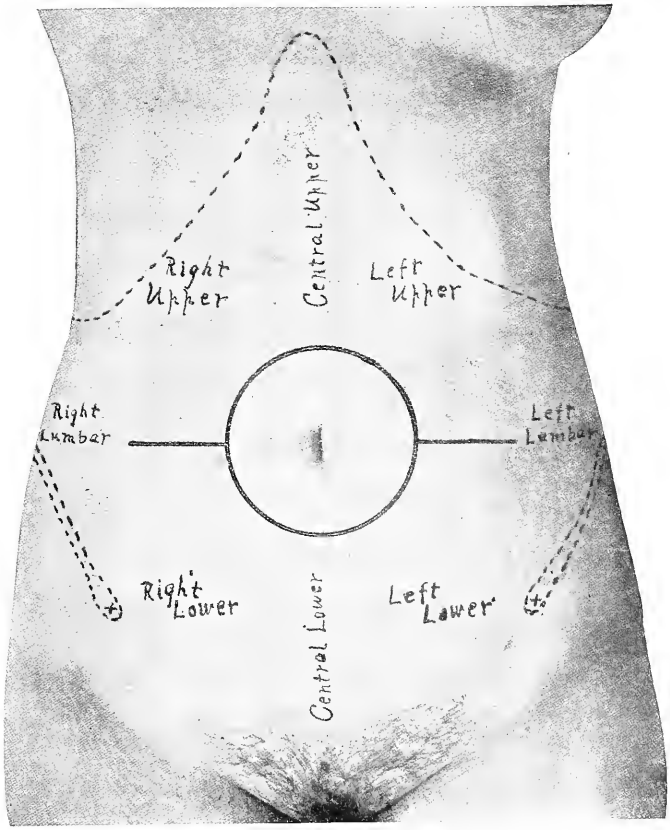


Fig. 153.—Another abdomen divided with the circle and short horizontal lines, and showing the names on the primary regions. The area within the circle carries the usual designation, "umbilical region." (From Crossen.)

The *right lumbar region*, limited by the sub-costal, inter-tubercular, and right mid-Poupart lines, overlies the right kidney, the ascending colon, and small intestine.

The *hypogastric region*, lying below the inter-tubercular line and limited laterally by the mid-Poupart lines and inferiorly by the pubic bone, overlies the distended bladder, the small intestine, the

sigmoid flexure, the cecum and occasionally the vermiform appendix, and the pregnant uterus.

The *left iliac region,* limited above by the inter-tubercular line, inferiorly by Poupart's Ligament, and internally by the left mid-Poupart line, overlies the sigmoid flexure.

The *right iliac region,* limited by the inter-tubercular line, the right mid-Poupart line and Poupart's ligament, overlies the cecum and vermiform appendix.

Instead of mapping out nine regions of the abdomen by means of two horizontal and two vertical lines, the abdominal cavity may be divided into four regions, or quadrants, by a horizontal and a vertical line passing through the umbilicus. In this mode of subdivision the four regions are termed respectively, the right upper quadrant, the left upper quadrant, the right lower quadrant, and the left lower quadrant of the abdomen. While this is a very practical and convenient division of the abdominal cavity and suffices for clinical description in many instances, the divisions are not sufficiently circumscribed for use in the description and localization of small tumors or masses arising within the abdominal cavity.

An eminently satisfactory subdivision of the abdominal cavity is that devised by Crossen, in which the natural landmarks of the abdomen are utilized, the only artificial lines employed being one encircling the umbilicus, and a horizontal line drawn from either side of the circle. By this method the abdomen is subdivided into regions which are respectively designated as right upper, left upper, central upper, right lower, left lower, central lower, umbilical, right lumbar, and left lumbar. This method of subdivision is very serviceable in the preparation of clinical charts.

CHAPTER XXI

INSPECTION OF THE ABDOMEN

Technic.—During inspection of the abdomen the patient should first be directed to assume the dorsal decubitus, the position in bed or on the examining table being absolutely symmetric and unconstrained. The subject should be covered by a sheet which may be turned down, exposing the abdomen to within a short distance of the pubic bone. The abdomen should be inspected in direct light, and then in oblique light, the latter often revealing slight pulsations or enlargements or vermicular movements which escaped detection during the examination in direct light.

For the purpose of detecting certain phenomena such as visible peristalsis and visceral ptosis an additional inspection should be made with the patient in the standing posture.

Finally, the patient should be examined in the knee-chest posture which allows movable tumors within the abdominal cavity to fall forward and become more clearly visible.

The Skin of the Abdomen.—The abdominal skin is lax and loose in the emaciation of chronic wasting disease, and is tense and glistening in the presence of abdominal distention from acites. abdominal tumor, or pregnancy. Whitish or silvery streaks, lineae albicantes, over the lower portion of the abdomen and the upper portions of the thighs are indicative of past abdominal distention, either from tumor, ascites, or pregnancy.

Scars upon the abdomen may result from surgical operations, the eruption of syphilis or other skin disease. A scar in the groin is suggestive of suppuration of an inguinal gland which has opened spontaneously or has been opened by the surgeon.

The Linea Nigra, a line of brown pigment in the median line below the umbilicus accompanies pregnancy and chronic abdominal distention from other cause.

The abdomen is the usual site of the cutaneous eruption of typhoid fever, the rose spot. These spots are small, hyperemic, slightly elevated spots, which disappear upon pressure. In certain instances the papule is surmounted by a small vesicle. They are not confined to the abdomen, although most commonly en-

countered there, but may appear upon the back, arms, or thighs. The rose spots appear in successive crops, disappearing in two or three days, sometimes leaving a brown stain.

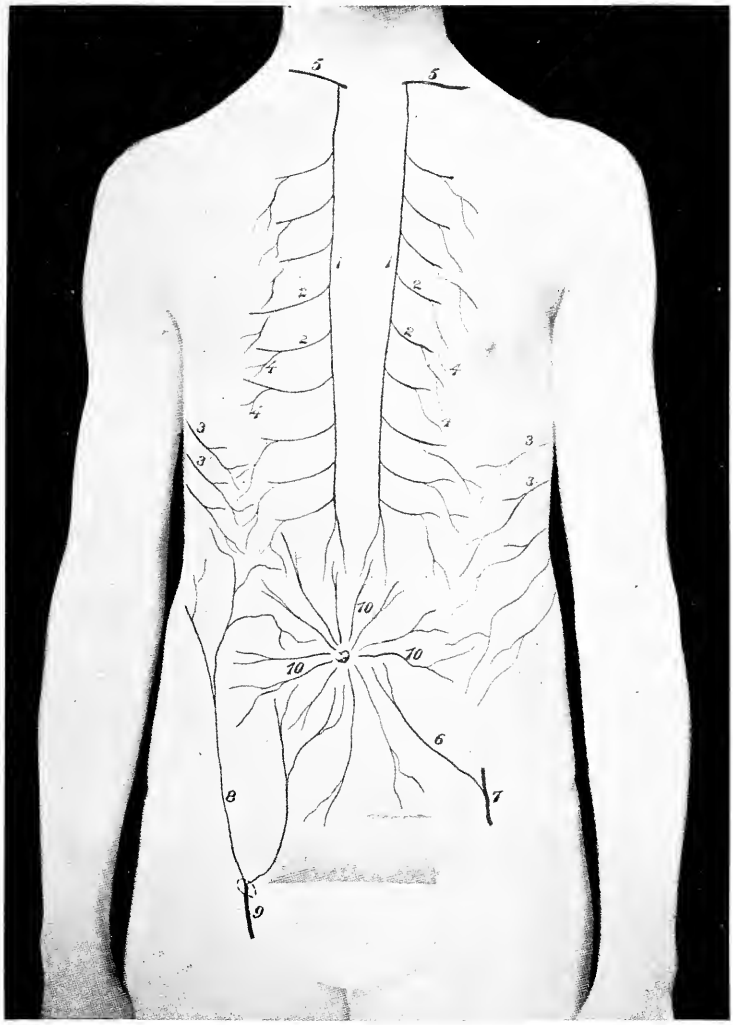


Fig. 154.—Establishment of collateral circulation in portal vein obstruction and mediastinal tumor.

1, internal mammary veins; *2*, anterior intercostal veins; *3*, posterior intercostal veins; *4*, radicles of *2*; *5*, subclavian vein; *6*, deep epigastric vein; *7*, external iliac vein; *8*, superficial circumflex iliac vein; *9*, internal saphenous vein; *10*, caput medusae.

During pregnancy the abdominal skin sometimes shows brownish areas of discoloration, chloasma.

Enlargement of the Superficial Veins of the Abdomen.—In portal obstruction from cirrhosis of the liver there is present upon the abdominal wall a series of distended superficial veins extending

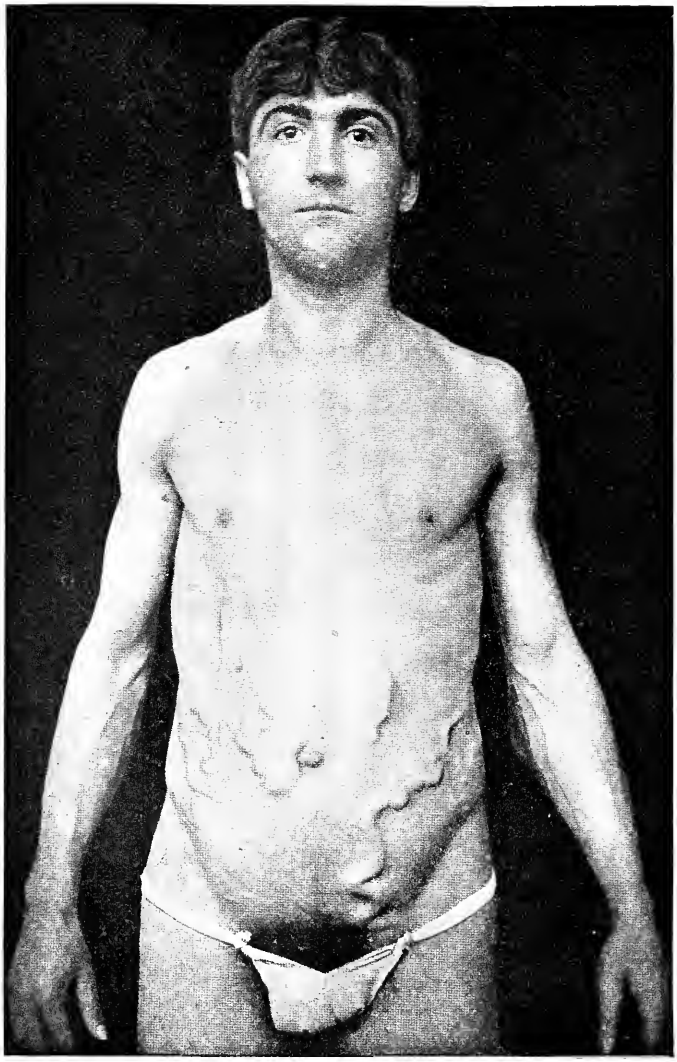


Fig. 155.—Abdominal arteries in a case of double iliac thrombosis of typhoid origin. (Woolley, after Thayer.)

outward from the umbilicus in a radial manner, constituting the caput medusæ. A distention of the superficial veins over the abdomen, communicating with similarly distended veins over the

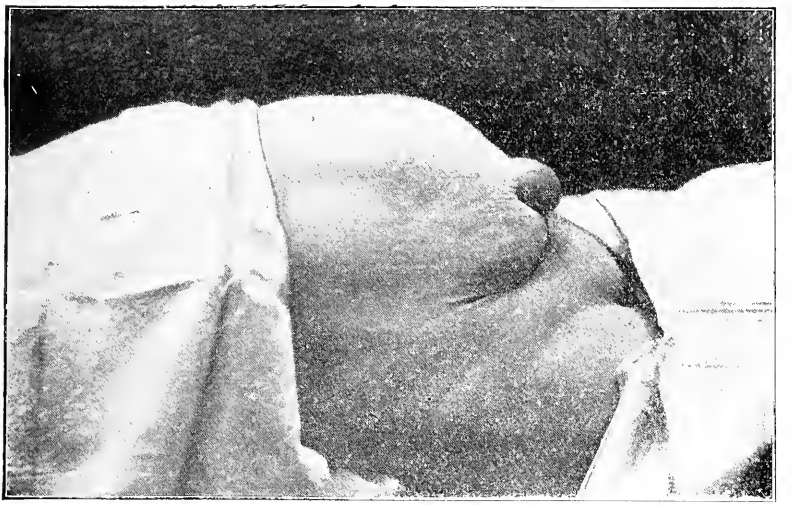


Fig. 156.—A small umbilical hernia, with a relaxed abdominal wall. (Crossen, after Hirst.)

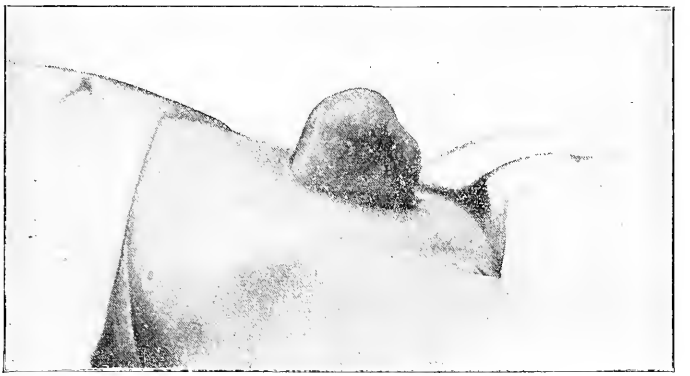


Fig. 157.—A large ventral hernia at the site of an operation scar. (Crossen after Hirst.)

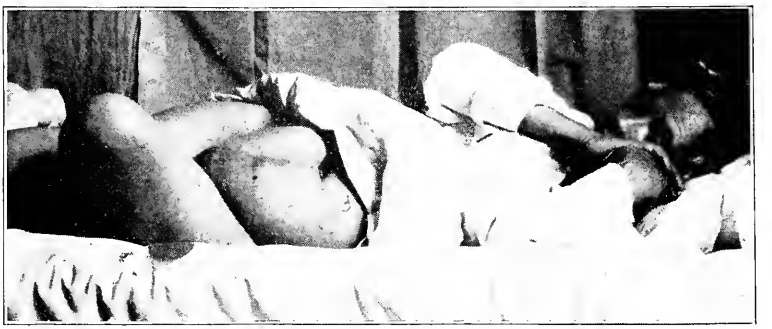


Fig. 158.—Ventral hernia.

thorax is indicative of portal vein obstruction by hepatic cirrhosis or tumor, chronic ascites, or pressure upon the inferior or superior vena cava by abdominal or mediastinal tumor. The direction of the blood flow in the distended vein is an index to the

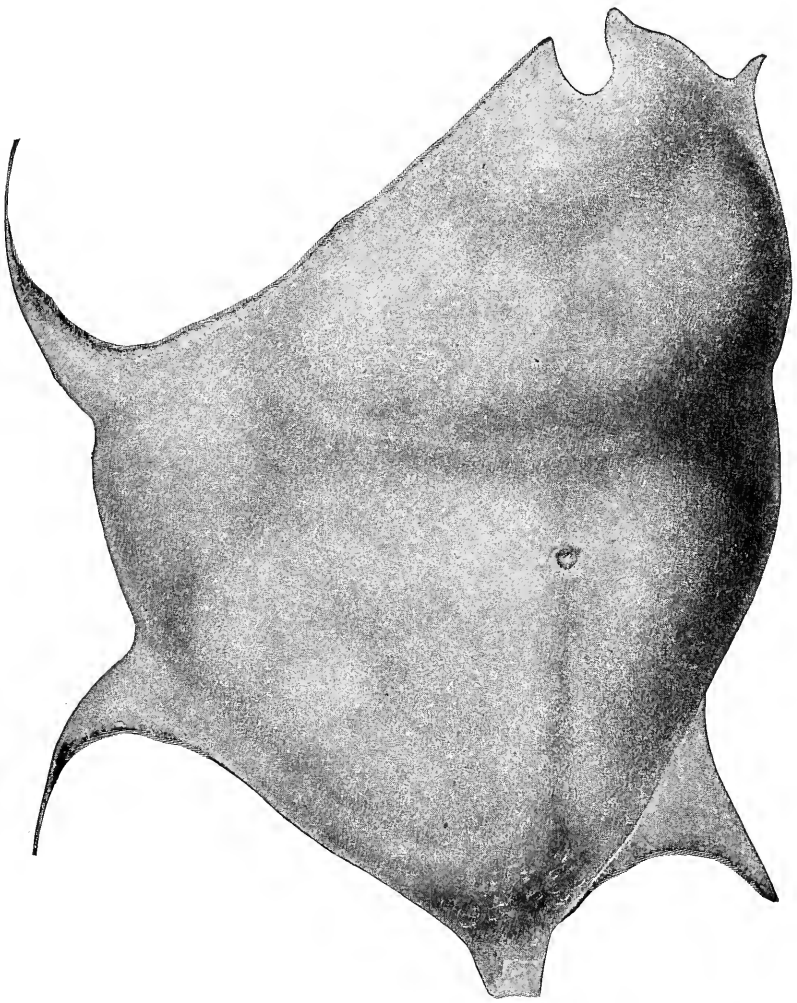


Fig. 159.—Stenosis in the vicinity of the splenic flexure. (Austin, after Nothnagel.)

site of the obstruction. If upon compressing the vein it is found that the direction of the blood current is upward the obstruction is in the portal vein or inferior vena cava; whereas, if the current is downward the obstruction is in the superior vena cava.

This venous distention is an evidence of the effort at the establishment of collateral circulation in the presence of obstruction of the usual channels.

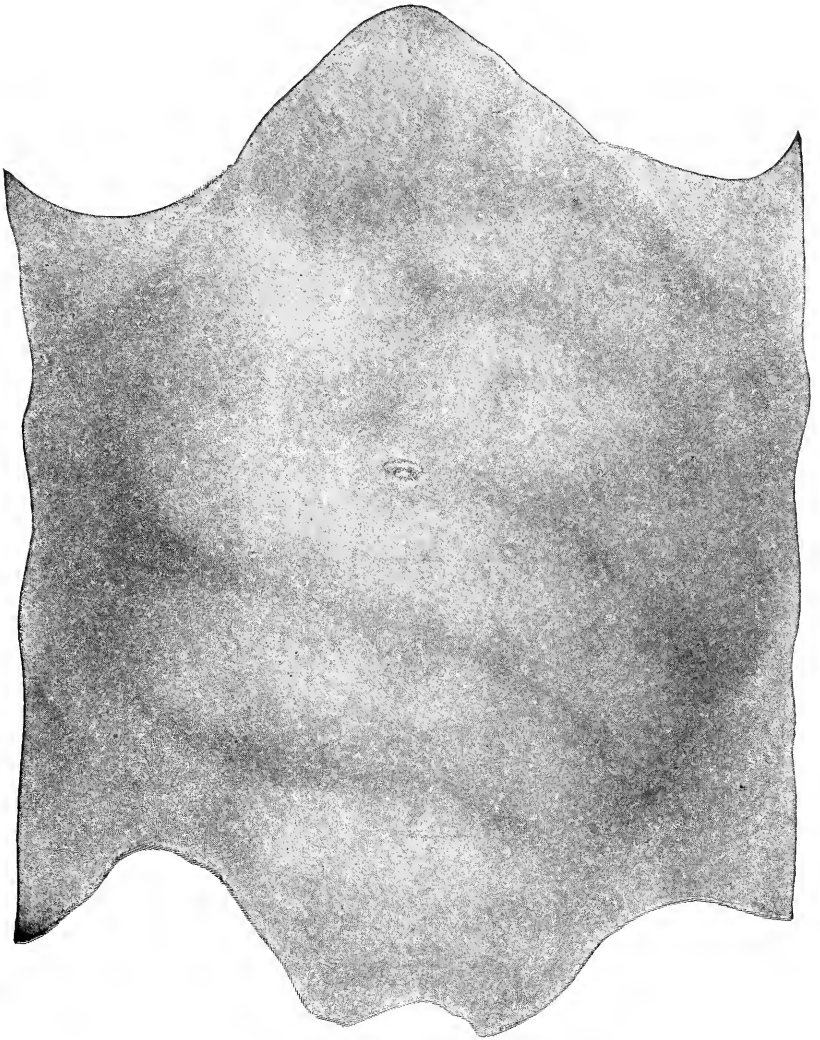


Fig. 160.—Stenosis of the lower ileum from peritoneal adhesion. (Austin, after Nothnagel.)

The Umbilicus.—The umbilicus should be examined for protrusion, retraction, skin eruptions, and inflammation. A protruding umbilicus is noted in umbilical hernia, the latter months of pregnancy, portal vein obstruction, ascites or abdominal dis-

tention due to large tumor of an intra-abdominal organ. The umbilicus is retracted in the obese subject.

Enlarged Glands.—The glands in the groin are enlarged as a result of venereal infection or localized non-specific inflammation resulting from abrasions about the external genitalia of the lower extremities. The character of the enlargement, whether hard or soft and fluctuating, and whether the glands remain separated and discrete or are matted together should be determined.

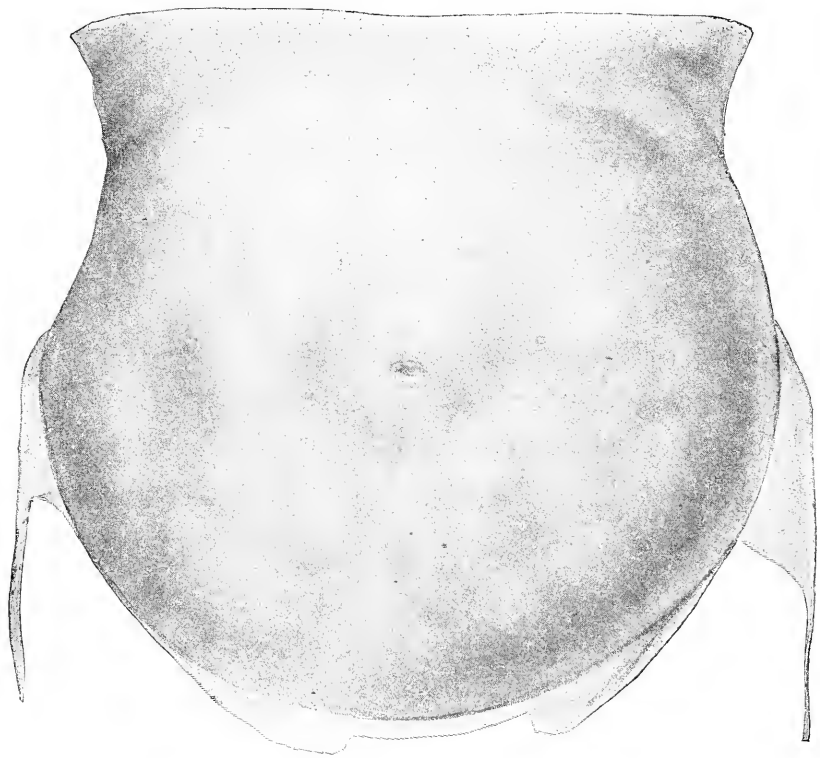


Fig. 161.—Normal intestinal peristalsis. (Austin, after Nothnagel.)

Visible Peristalsis.—The peristaltic movements of the stomach, small intestine, or large intestine, at times become visible as a vermicular movement upon the abdominal wall. In its exaggerated form it is indicative of obstruction located at the pylorus, in the small intestine, or in the colon. The peristaltic movement of the stomach is visible under these circumstances in the upper portion of the abdomen, pursuing a direction from left to right and slightly downward. Visible peristalsis of the small intestine is

chiefly confined to the umbilical region, whereas that occasioned by obstruction in the large intestine is observed over the course of the colon. When not very pronounced this peristaltic movement of the abdominal wall may often be accentuated by applying a cold hand to the surface of the abdomen or by flicking the abdominal surface with a towel wet in cold water. When the site of the obstruction is in the ilium just proximal to the ileocecal valve, the visible peristalsis assumes a "ladder pattern" the waves lying one above the other in the umbilical region.

Visible peristalsis observed in extremely emaciated patients

Fig. 162.—Median grooving of the abdominal wall where there is separation of the recti muscles. The woman is represented as lying on her back. (Crossen, after Webster.)

with very thin abdominal walls, or in women in whom repeated pregnancies have caused a diastasis of the rectus muscles possesses little, if any, significance.

Abolition of the Respiratory Movements of the Abdomen.—Fixation of the abdominal wall with inhibition or abolition of the respiratory movements is indicative of pain arising within the abdominal cavity, usually due to peritonitis.

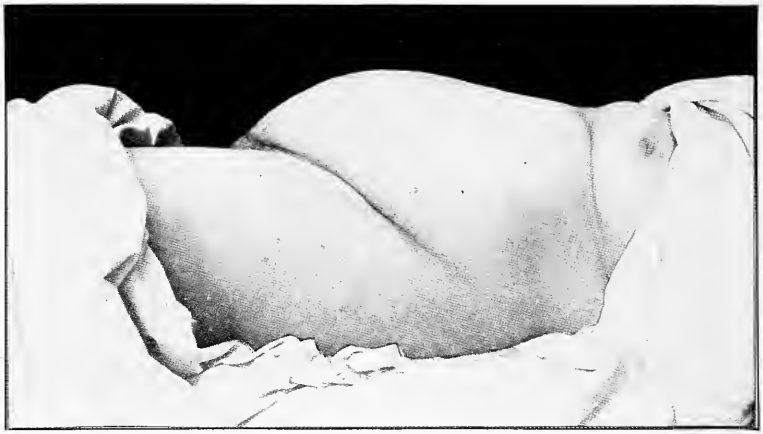


Fig. 163.—Obesity. The most prominent feature in this case is the marked obesity—see Fig. 164. There is also a fibroid tumor of the uterus and a small amount of ascitic fluid. (From Crossen.)

Fig. 164.—Obesity. Patient standing. Same patient as shown in Fig. 163. Notice the thick roll of subcutaneous fat that drops down below the general contour of the abdomen.

VARIATIONS IN THE CONTOUR OF THE ABDOMEN

The normal abdomen is symmetrical with a moderate degree of antero-posterior flattening in the male, is of uniform tension, and a moderate bulging of the lower portion in female subjects. The umbilicus is nether unduly depressed nor protrudng.

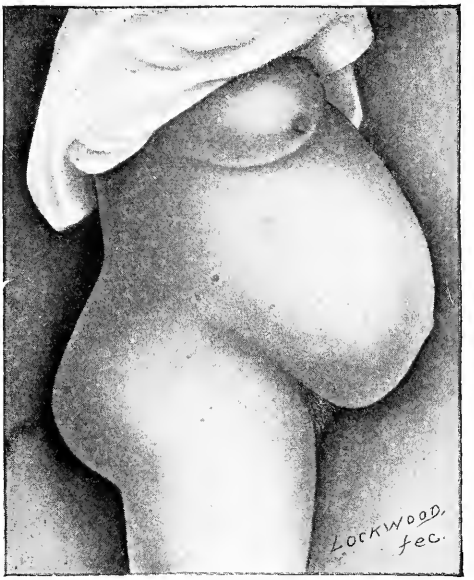


Fig. 165.—Obesity, mistaken for pregnancy by patient. (Crossen, after Williams.)

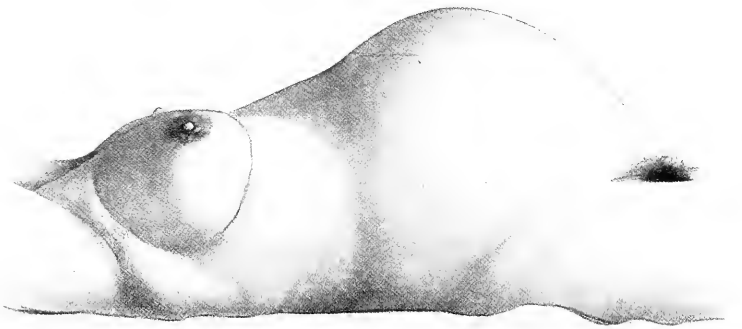


Fig. 166.—Contour of the abdomen in pregnancy with patient recumbent. (Crossen, after Edgar.)

Symmetric Enlargement

Obesity.—In the obese subject the abdomen is symmetrically enlarged, the cutaneous flexion folds are accentuated, the umbilicus is depressed, and the lower portion of the abdomen is pendulous, encroaching to a variable extent upon the thighs and pubes.

Pregnancy.—The abdominal enlargement accompanying pregnancy is progressive, increasing gradually as the uterus rises out of the pelvis and occupies the abdominal cavity. In its fully developed state the umbilicus protrudes, the abdominal skin presents the lineae albicantes, and the abdominal distention is accompanied

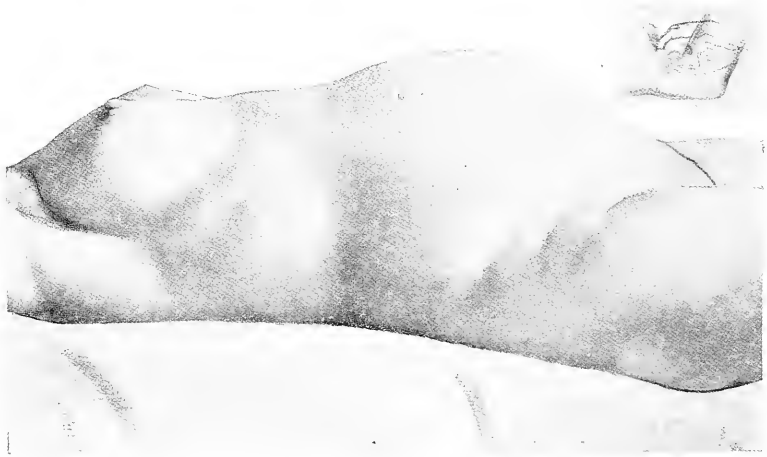


Fig. 167.—Tympanites, mistaken for pregnancy by the patient. The small figure in the upper corner shows the internal condition as determined by bimanual examination, the uterus being of normal size. (Crossen, after Edgar.)

by changes in the breasts and the positive signs of pregnancy. The abdominal distention is much greater in multiparous women than it is in primiparae.

Meteorism.—Meteorism or tympanites produces symmetrical abdominal enlargement, the walls being tense, smooth and shiny, affording upon percussion a distinctly tympanitic note, which extends high up and decreases the area of normal dullness of the liver. The umbilicus protrudes.

Ascites.—The degree of abdominal enlargement accompanying ascites varies with the amount of fluid in the peritoneal cavity. With the development of the ascitic fluid there is a gradual and uniform enlargement of the abdomen. The contour of the abdo-

Fig. 168.—Extreme ascites. In the patient from which this photograph was taken, the abdomen was so distended with fluid that the wall was raised higher than the mesentery would permit the intestine to float, giving dullness about the umbilicus as well as elsewhere. The rise of the wall from below is rather abrupt. There is also edema of the wall, as shown by the persisting groove where the skirts were tied about the waist.

Fig. 169.—Showing the area of dullness in moderate ascites, with the patient lying on her back. (From Crossen.)

men is characteristically altered, the antero-posterior diameter increasing with moderate flattening of the lateral regions. In the

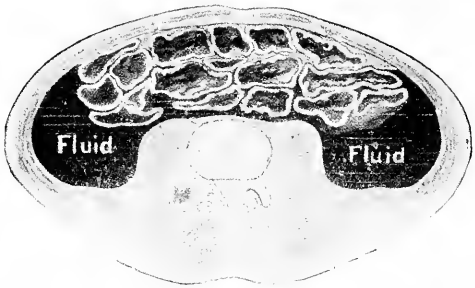


Fig. 170.—Showing the reason for the disposition of the dull and resonant areas in a case of moderate ascites. (Crossen, after Butler.)

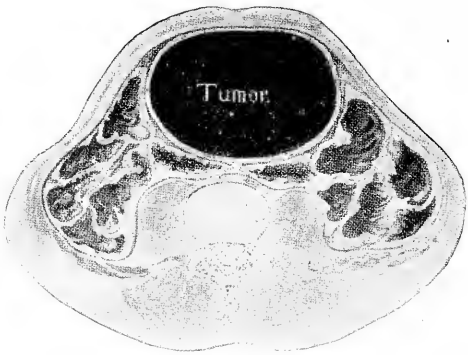


Fig. 171.—Indicating the relation of the dull and resonant areas in the case of a tumor occupying the central lower abdomen. (Crossen, after Butler.)

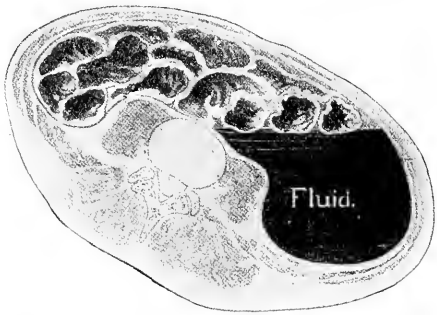


Fig. 172.—Ascites. Representing the patient turned on one side. The fluid gravitates to the under side, leaving the upper flank resonant. (Crossen, after Butler.)

presence of a large effusion the skin is smooth and tense, not uncommonly presenting lineae albicantes. Enlarged tortuous superficial veins are often present.

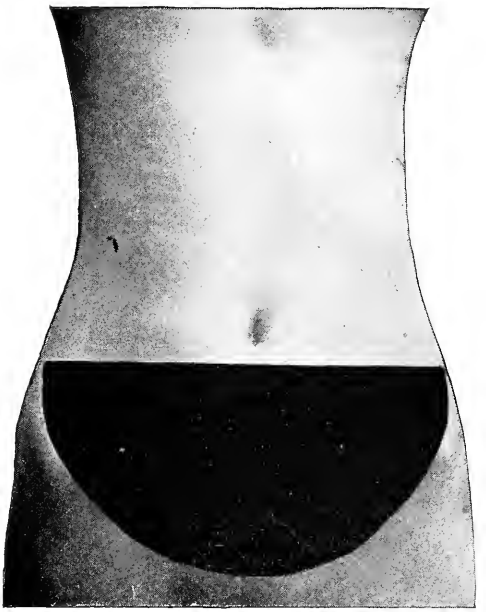


Fig. 173.—Indicating the area of dullness in moderate ascites, with the patient standing. (From Crossen.)

Fig. 174.—Indicating the area of dullness in a case of moderate ascites, with the patient turned on the left side. (From Crossen.)

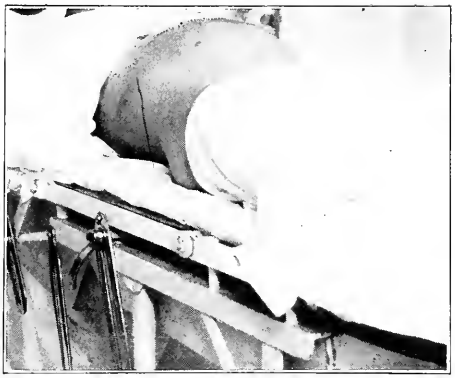


Fig. 175.—Abdominal enlargement due to ovarian cyst.

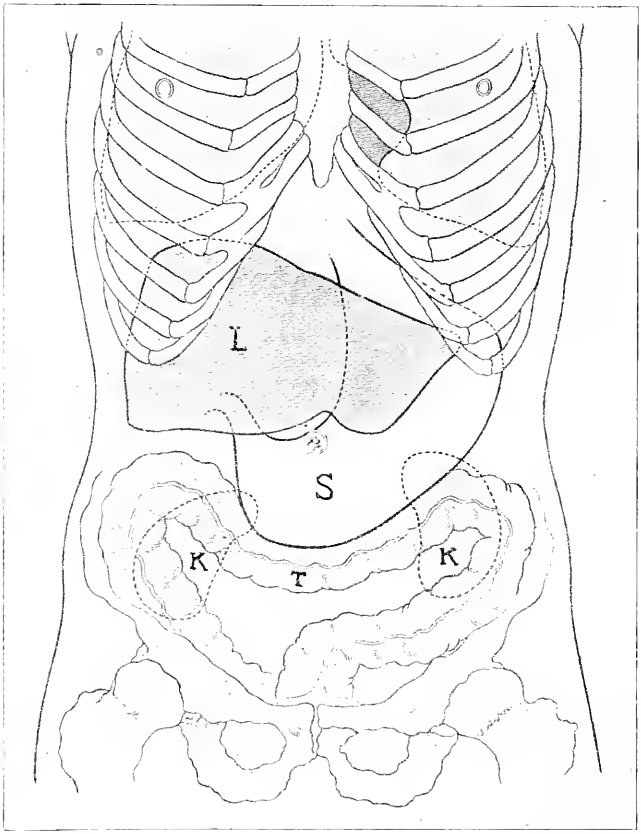


Fig. 176.—Front view of general enteroptosis. (Eisendrath, after Coffey.)

L, liver outline on surface; *S*, stomach (lesser curvature on line with umbilicus, greater curvature midway between umbilicus and symphysis); *KK*, right and left kidneys, showing marked downward displacement; *T*, transverse colon also markedly prolapsed.

When a patient with ascites assumes the dorsal decubitus the percussion note is tympanitic in the median line and flat in the flanks, owing to the fact that the intestines float upward in the fluid, which gravitates to the dependent portions of the abdominal cavity. When the patient is placed in the lateral decubitus there is dullness upon the under side and tympany upon the uppermost side for the same shifting of the intestines. Finally, upon placing the patient in the knee-chest position there is flatness in the umbilical and hypogastric regions, and tympany in the flanks.

Visceroptosis.—Ptosis of the abdominal organs produces rather characteristic alterations in the abdominal contour with the patient in the erect attitude. Thus, in gastroptosis, there is an abnormal flattening of the upper region of the abdomen with undue prominence of the central region; whereas, in enteroptosis the upper and central regions of the abdomen are flattened while the lower region symmetrically bulges; the contour of the abdomen resembling that of a gourd. (See Fig. 176, p. 322.)

Abdominal Retraction.—In chronic wasting disease, prolonged diarrhea, and cardiac or pyloric stenosis the abdominal wall is generally retracted, the bony landmarks standing out very prominently. The abdomen appears to be "scooped out" like a boat, the scaphoid abdomen.

Asymmetrical Variations

Local bulging of the upper region of the abdomen in the median line and laterally may be due to distention of the stomach, or enlargements of the liver or spleen, the causes of which are discussed in the section upon the examination of these organs.

Undue prominence or bulging of the umbilical region is significant of umbilical hernia or tumor of an abdominal organ.

Localized bulging in the hypogastric and iliac regions may be due to a distended bladder, a pregnant uterus, uterine myoma, or ovarian cyst.

CHAPTER XXII

PALPATION, PERCUSSION, AUSCULTATION, AND MENSURATION OF ABDOMEN

PALPATION

Technic.—During palpation of the abdomen the patient should assume the dorsal decubitus with the head slightly elevated by a small pillow and the knees drawn up and supported by a pillow, which relieves the tension of the abdominal wall. In bed-ridden subjects a similar state of abdominal relaxation may be attained by propping the patient's shoulders up with pillows and drawing up the knees and supporting them. Under certain circumstances it is desirable to palpate the abdomen with the patient in the knee-chest position.

The patient having been placed in a natural and unconstrained attitude, he should be directed to refrain from the natural tendency to hold the breath during the examination.

The hands of the examiner should be warm, as a cold hand applied to the surface of the bare abdomen will cause a local muscular rigidity which combats and frustrates the object of the examination. The examining hand should be first applied very gently to the abdominal surface with the palm down and fingers extended, avoiding any sudden pressure or punching movements. During the course of the examination the examiner should first palpate a region which is supposed to be normal before proceeding to the suspected site of disease, as by so doing he gains the confidence and co-operation of the patient.

The abdomen should be palpated systematically, the examiner examining the state of the abdominal wall, palpating any local bulging or retraction which was noted during inspection, and endeavoring to determine whether it is located in the abdominal wall or arises within the abdominal cavity, and the state of the various solid organs of the abdomen, the technic of palpation of which are discussed in their appropriate sections.

The Abdominal Wall.—An estimate of the thickness of the abdominal wall may be made by pinching up the wall between

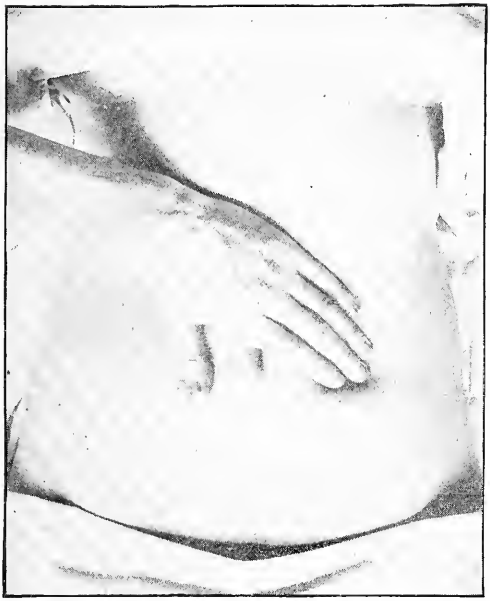


Fig. 177.—Palpation of the abdomen. First step. Hand flat on abdominal surface. (From Crossen.)

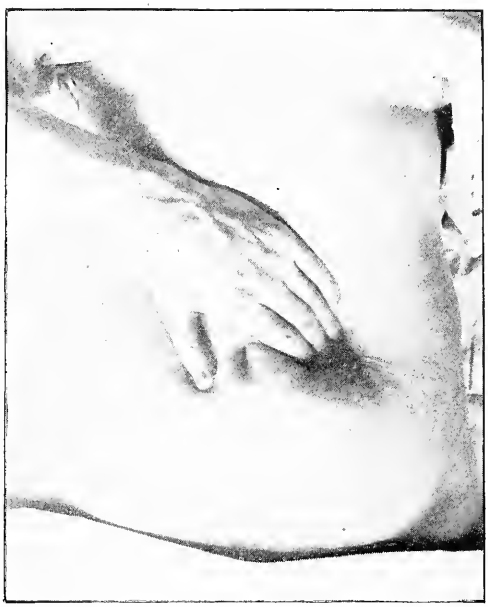


Fig. 178.—Palpation. Depressing the wall with the fingers of one hand, in various situations. (From Crossen.)

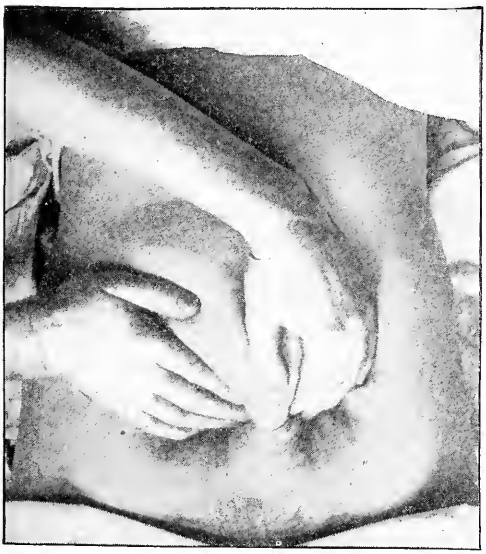


Fig. 179.—Palpation with both hands. (From Crossen.)

Fig. 180.—Deep palpation with both hands. (From Crossen.)

the forefinger and thumb, or by approximating the two hands placed palm downward upon the abdominal surface. Increased thickness of the abdominal wall indicates, an excess of fat, the presence of edema, or suppuration of the wall. If the increase be due to excessive deposition of fat in the wall, a fat wave will be obtained upon bimanual palpation; if due to edema, the wall will pit upon pressure; and if due to a localized or extensive suppuration of the wall, there will be accompanying signs of inflammation, as discoloration of the surface, and elevated temperature.

Rigidity of the abdominal wall with possibly spasm upon attempts at palpation, indicates inflammation of the peritoneum or of an abdominal organ. Muscular rigidity is most commonly en-

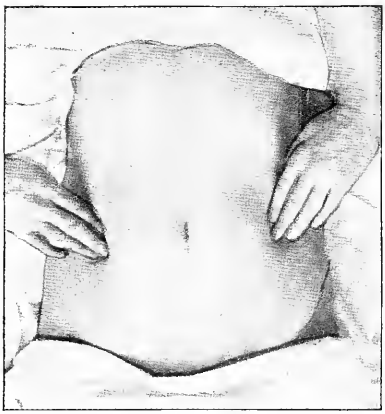


Fig. 181.—Testing the thickness of the abdominal wall. (From Crossen.)

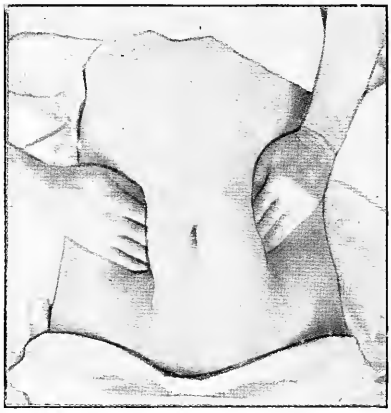


Fig. 182.—Testing the thickness of the abdominal wall.—Second step. The fingers carried beneath the wall. (From Crossen.)

countered in the rectus muscle. Rigidity of the right rectus alone occurs with acute appendicitis, whereas bi-lateral rigidity of the recti accompanies acute peritonitis.

Tenderness.—When tenderness is elicited upon palpation of the surface of the abdomen, if not due to hyper-esthesia of the parietes, it points to a diseased abdominal organ. The tenderness is most apt to be encountered over the gall-bladder, stomach, spleen, kidney, appendix, and sigmoid flexure. In acute peritonitis there is general or diffuse tenderness.

Fluid Wave.—In the presence of ascites a fluid wave can be demonstrated upon bi-manual palpation. In palpating for fluid in the peritoneal cavity, one hand of the examiner is applied flatly

over one lumbar region, while the opposite side of the abdomen is tapped with the other hand of the examiner, the finger-tips being used. An impulse or wave is thus created in the fluid, which is appreciable to the palpating hand.

Fat Wave.—Excessive deposition of fat in the abdominal wall

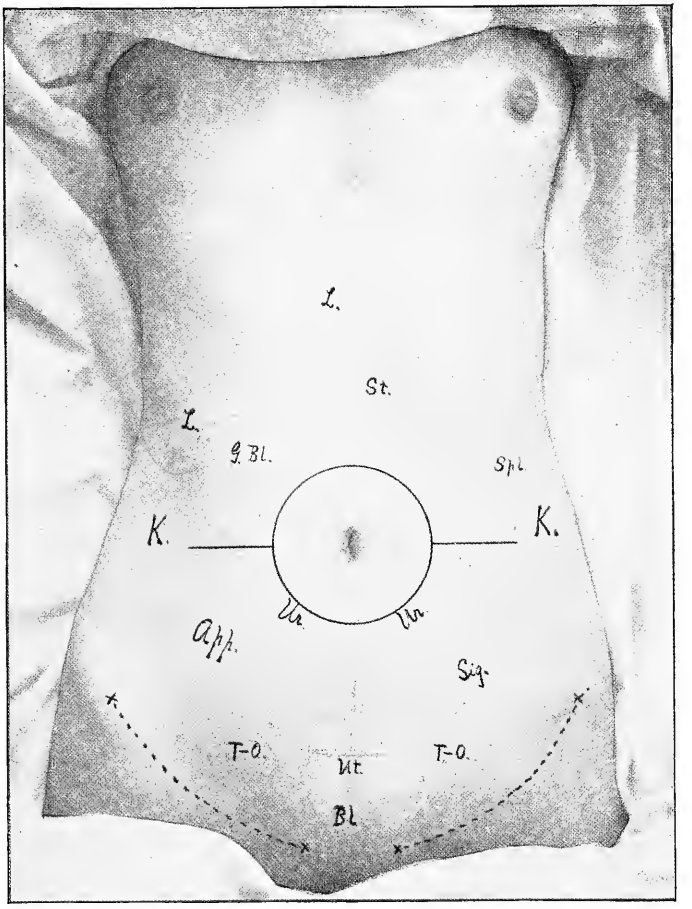


Fig. 183.—Various areas of significant point-tenderness. These are the areas to be investigated during the course of an abdominal examination. (From Crossen.)

gives a wave upon bi-manual palpation closely simulating the fluid wave. To exclude such a fat wave during bi-manual palpation an assistant should apply the ulnar side of the hand to the median line of the abdomen while the examiner practices bi-manual palpation as in eliciting the fluid wave, when the fat wave is inter-

Fig. 184.—Trying for a fluid wave across the abdomen. (From Crossen.)

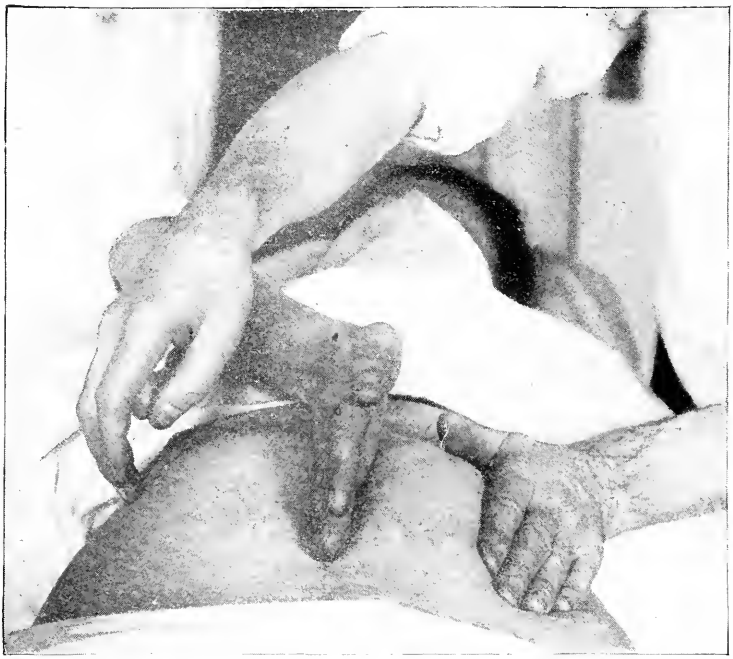


Fig. 185.—Differentiating a fat-wave from a fluid-wave. The fat-wave is stopped by the pressure in the median line. (From Crossen.)

rupted by the intervention of the assistant's hand and is not transmitted to the palpating hand of the examiner.

Intra-Abdominal Tumor.—An intra-abdominal tumor may be so large as to entirely fill the abdominal cavity; but as a rule careful bi-manual palpation enables the examiner to determine its origin,

its size and shape, whether it is fixed or movable, and finally whether it moves with respiration. In examining for respiratory mobility of an intra-abdominal tumor the examiner should place both hands palms downward flat upon the abdomen, with the fingers directed toward the costal arch, while the patient is directed to breathe deeply. At the commencement of expiration, the finger-tips are pressed downward firmly and with uniform pressure, when the lower margin of the tumor or enlarged organ is encountered. The most commonly encountered movable abdominal tumor is a movable or displaced kidney; but tumors of the liver, spleen, or stomach, are movable with respiration.

Peritoneal Friction Fremitus.—Upon palpating over the upper regions of the abdomen a friction fremitus analagous to pleural or pericardial friction fremitus is sometimes encountered. The vibration is produced by roughening of the peritoneal surfaces in peritonitis. This fremitus is most commonly encountered over the hypochondriac regions, in the presence of peritoneal involvement in the course of peri-hepatitis or peri-splenitis.

PERCUSSION

In the examination of the abdomen ordinary mediate finger percussion may be employed, although in delimiting the various solid viscera and tumors of the abdomen auscultatory percussion is more serviceable and reliable. In general, the percussion note is tympanitic over the hollow abdominal viscera, changing to dullness and flatness over the solid organs. A similar flat note is elicited over solid intra-abdominal tumors and fluid in the peritoneal cavity.

During percussion of the abdomen the patient should assume the dorsal decubitus with the abdomen exposed. In outlining the borders of solid organs and tumors and in estimating the relative tympanicity of the adjacent hollow viscera, auscultatory percussion, using a light percussion blow or a stroking movement upon the abdominal surface is most serviceable.

The details of technic of outlining the various abdominal viscera are discussed in their appropriate sections.

AUSCULTATION

Auscultation is seldom employed in the examination of the abdomen and its viscera. Upon auscultation over the spleen or

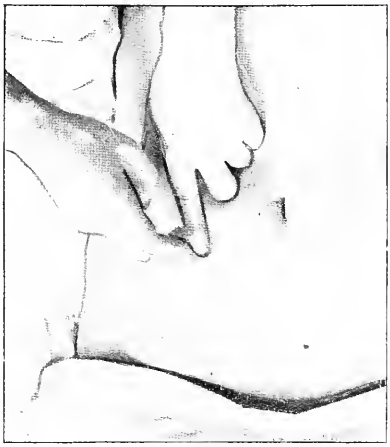


Fig. 186.—Ordinary percussion, which is usually rather superficial. (From Crossen.)

Fig. 187.—Deep percussion. Notice how the left index finger is pressed into the abdomen, so as to thin out the wall and get closer to deep structures. (From Crossen.)

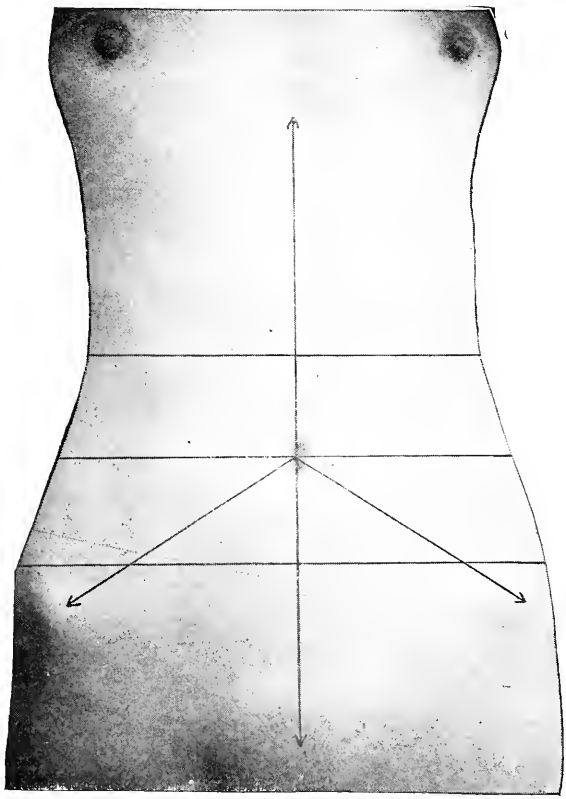


Fig. 188.—Showing the lines for mensuration. (From Crossen.)

liver a friction rub may occasionally be audible in presence of peri-splenitis or peri-hepatitis, due to peritoneal involvement. Similarly, in cases of aortic aneurism a valvular murmur may be detected by auscultation over the course of the vessel. In cases of suspected pregnancy, again, auscultation is available in the search for the fetal heart sound as well as the umbilical or uterine souffle.

MENSURATION

Mensuration is employed in determining the approximate dimensions of intra-abdominal tumors. Successive mensuration is practiced to determine progressive enlargement of the abdomen from tumor or ascites. In practicing mensuration of the abdomen three measurements are commonly employed; one at the level of the umbilicus; one three inches above this point; and a third a similar distance below the umbilicus.

SECTION II

SPECIAL EXAMINATION OF THE ABDOMINAL VISCERA

CHAPTER XXIII

THE STOMACH, INTESTINES, AND PANCREAS

EXAMINATION OF THE STOMACH

Clinical Anatomy.—The stomach occupies the epigastric and left hypochondriac regions of the abdomen when the organ is normal. The *cardiac orifice* of the stomach is located behind the seventh left costal cartilage, at a point one inch from the sternum. The position of the *pyloric orifice* is somewhat variable, its site being modified by the condition of the stomach. When the stomach is empty it occupies the median line at a point midway between the epi-sternal notch and the symphysis pubis. When the stomach is moderately distended the pylorus occupies a position approximately one inch to the right of the mid-line at the same level, this displacement to the right being increased to two or three inches in extreme distention of the organ.

The *fundus* of the stomach is in contact with the inferior aspect of the diaphragm, behind and below the apex of the heart, in which position it extends as high as the sixth rib. The *lesser curvature,* the upper limit of the stomach, is covered by the left lobe of the liver, passing downward and to the right from the cardiac orifice to the pyloric orifice of the stomach. The *greater curvature,* the lower limit of the stomach, crosses the left costal arch at the level of the ninth costal cartilage, the most dependent point of the normal organ being approximately two inches above the umbilicus.

The *anterior surface* of the stomach, largely overlapped by the left lobe of the liver, lower border of the left lung, and left costal arch, is exposed to the anterior abdominal wall in a very limited portion of its extent. *Traube's semilunar space,* the area in which

the anterior wall of the stomach is in direct contact with the anterior abdominal wall, affording upon percussion pure gastric tympany, is limited above by the left lobe of the liver and lower border of the left lung, and externally by the spleen.

The *posterior surface* of the stomach, looks backward and downward, reposing in the so-called "stomach bed" formed of the

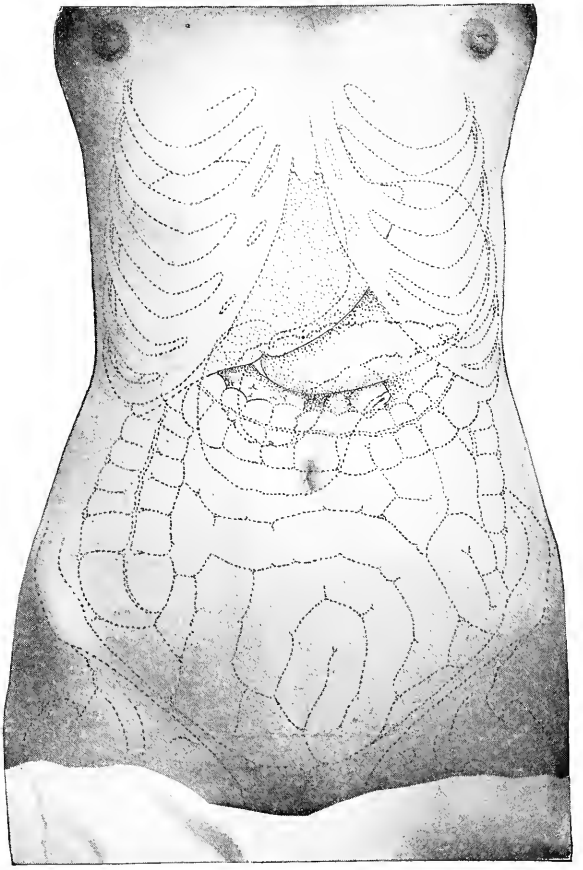


Fig. 189.—The central upper abdomen. Showing in outline the liver and stomach and pancreas. (From Crossen.)

transverse mesocolon, the pancreas, left kidney, and supra-renal capsule.

Inspection.—The normal stomach produces no alteration in the normal contour of the abdomen; but in persons with diastasis of the recti the greater curvature of the stomach which is the seat of dilatation or *gastrectasis* will cast a shadow by oblique il-

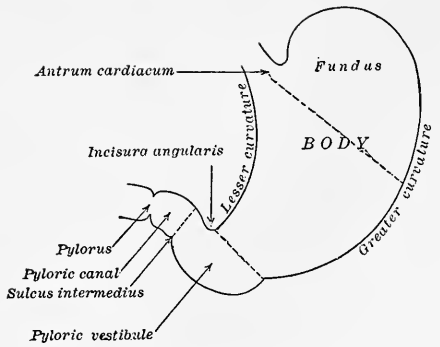


Fig. 190.—Anatomic subdivisions of stomach.

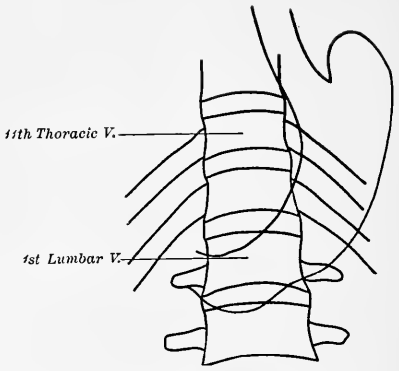


Fig. 191.—Form and surface topography of empty stomach. (From Gray.)

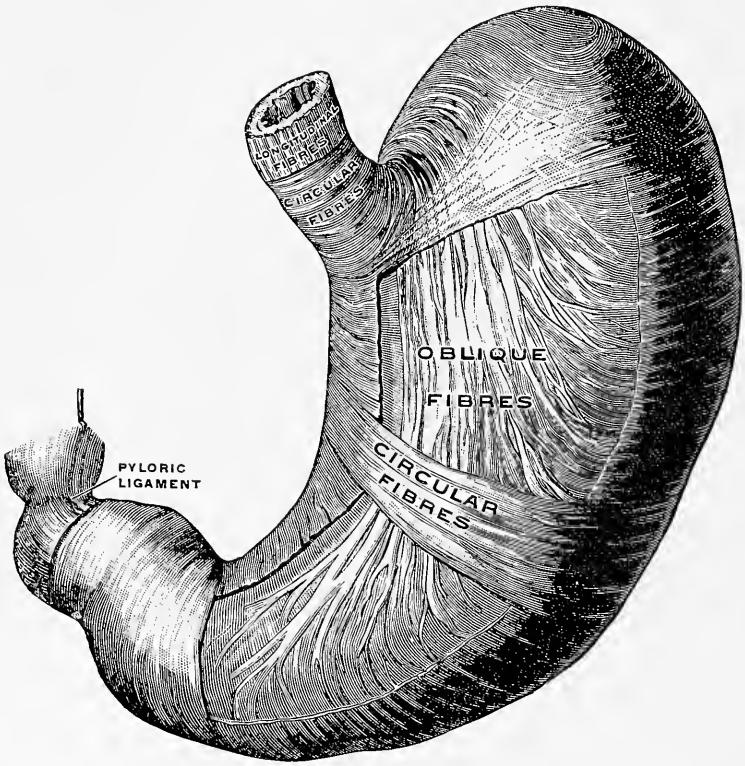


Fig. 192.—Musculature of the stomach. (From Gray.)

lumination, which ascends and descends with the movements of respiration. In gastrectasis the greater curvature of the stomach occupies a very low position, often below the umbilicus.

Fig. 193.—Traube's semilunar space. (From Butler.)

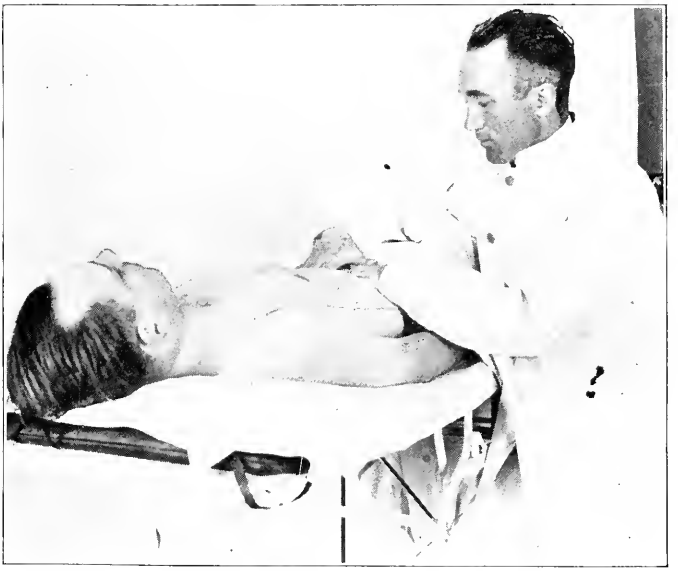


Fig. 194.—Palpation of the epigastrium.

In *gastroptosis* the epigastrium is flattened with more or less prominence in the umbilical region.

Visible peristalsis of the stomach is often present in cases of

pyloric obstruction, the waves being recognized as a vermicular movement upon the abdominal wall which travels from the left to the right and downward.

Palpation.—In practicing palpation of the stomach, the patient should assume the dorsal decubitus with the head comfortably elevated by a pillow and the legs drawn up and properly supported. The examiner, seated on the left side of the patient should apply the tips of the fingers of the right hand to the epigastrium, the fingers being separated and extended.

Palpation of the stomach may reveal the presence of tender-

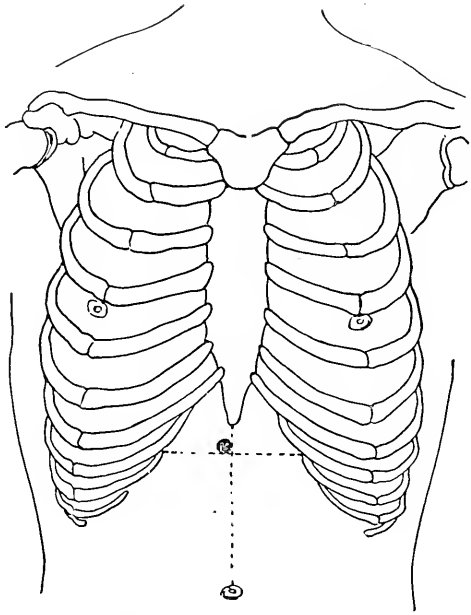


Fig. 195.—Epigastric pressure point. (From Austin.)

ness, confirm visible peristalsis, or a tumor of the stomach. or succussion sounds.

Tenderness in the area of the stomach is usually due to gastritis, but may be a sign of gastric ulcer or carcinoma, in which latter event the tender point adjacent to the tenth dorsal vertebra will be present.

A *tumor* of the stomach may occupy the anterior wall, the posterior wall, or the pylorus. A pyloric tumor is accompanied by visible peristalsis and in addition there is usually a palpable tumor at the pyloric orifice. The stomach is usually the seat of gas-

trectasis and upon proper examination reveals succussion sounds. A tumor upon the anterior wall of the stomach may be palpable by ordinary methods; but a tumor of the posterior wall may be confused with such a tumor. To differentiate the site of the tumor, whether on the anterior or posterior wall of the stomach

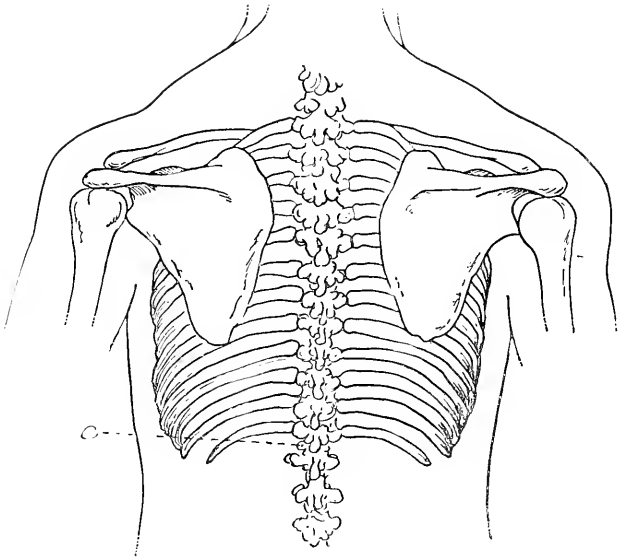


Fig. 196.—Dorsal pressure point in gastric ulcer, indicated at *a*. (From Austin.)

Fig. 197.—Showing the region for tenderness or a mass from disease of the stomach or pancreas. (From Crossen.)

the stomach may be filled with gas by the ingestion in solution of a drachm of sodium bicarbonate, to be followed immediately by the ingestion of a similar amount of tartaric acid. The two compounds generate carbon dioxide which distends the stomach. When so distended a tumor situated upon the posterior wall is not palpable.

Succussion sounds arising in the stomach are elicited by palpating the epigastrium with short pushing movements with the fingertips, beginning below the greater curvature of the stomach and proceeding upward until this point is reached, when the splashing sounds will be audible.

Glenard's Belt Sign.—In gastroptosis and gastrectasis, when the examiner standing behind the patient, places his hands upon the lower portion of the abdomen and lifts upward and backward, the patient with gastroptosis or gastrectasis experiences a sensation of relief from the dragging sensation which accompanies these states.

Percussion.—Percussion of the stomach is employed to determine the size, shape, and position of the viscus. The results of the examination vary with the state of the organ, whether empty or distended with fluid or gas. Normal gastric tympany is only obtained in Traube's semilunar space, where the stomach is in apposition with the anterior abdominal wall. The fundus of the stomach is determined with difficulty, owing to its situation beneath the left costal arch.

The boundaries between the stomach and the liver and the stomach and left lung, the upper limits of Traube's space, can be determined by mediate percussion from an area of frank gastric tympany in the center of this space toward the liver and lung respectively. To delineate the lower border of the stomach or greater curvature it is necessary to introduce fluid into the stomach, since otherwise the tympany of the colon and stomach could not be distinguished. Upon percussing upward under these conditions, the colonic tympany will give place to dullness, when the greater curvature is reached.

But the size, shape, and position of the stomach are better determined by auscultatory percussion. In this method of examination the chest-piece of the stethoscope is placed at the center of gastric tympany in Traube's space. The examiner delivers a few blows and fixes in his mind the character of the sound elicited. Without moving the position of the stethoscope he then begins at several points and percusses from several regions of the

abdomen toward the bell of the stethoscope. In each instance a change in the quality of the percussion note will indicate when the stomach border has been reached.

An extension of gastric tympany beyond the normal limit upward indicates gaseous distention of the stomach, or it may be due to cirrhosis of the liver or fibroid change in the left lung; or it may be a sign of gastrectasis resulting from pyloric stenosis.

Diminution of the area of normal gastric tympany may be due to enlargement of the liver or spleen, or left sided pleurisy with effusion, or cardiac obstruction.

An hour-glass constriction of the stomach may be demonstrated by the introduction of water into the stomach; when, upon percussion over the viscus, it is observed that the stomach is not uniformly distended; but that the cardiac portion is distended, while the pyloric portion remains empty; and, moreover, that in a short time fluid passes into the pyloric portion, which in turn becomes distended. If, during this time, the stethoscope be applied over the central portion of the stomach, it may be possible to hear the water gurgle through the constricted portion of the stomach.

Auscultation.—Apart from eliciting succussion sounds, the significance of which has been mentioned in the section upon palpation, auscultation of the stomach yields little diagnostic data.

THE SMALL INTESTINE

Clinical Anatomy.—The small intestine, the section of the gastro-intestinal tract extending from the pyloric orifice of the stomach to the junction with the large intestine at the ileo-cecal valve, lies within the frame formed by the course of the large intestine, slightly overlapping the ascending and descending colon, and extending for a variable distance below the brim of the pelvis. The *duodenum,* the proximal 12 inches of the small intestine pursues a course resembling the letter "C" from the pylorus to the duodeno-jejunal flexure at the left of the second lumbar vertebra, embracing in its course the head of the pancreas and the common bile duct which empties its contents into this portion of the small intestine. The *jejunum,* the second division of the small intestine, comprising approximately 8 feet of the tube, lies in the umbilical and right and left lumbar regions, and is freely movable. The *ileum,* the distal 12 feet of the small intestine, which terminates at the ileo-cecal valve, lies in the umbilical, hypogastric, and lum-

bar regions, in which it is freely movable. Only the great omentum intervenes between the jejunum and ileum and the anterior abdominal wall.

Inspection.—Visible peristalsis of the small intestine may be seen in obstruction of this portion of the alimentary tract. If the site of the obstruction is in the lower portion of the ilium the peculiar "ladder pattern" is noted occupying the umbilical region.

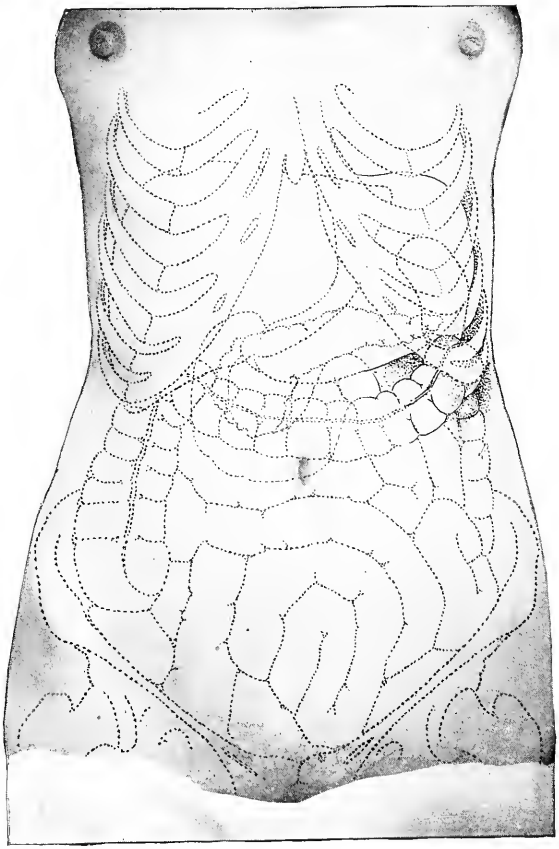


Fig. 198.—The left upper abdomen. The site of the spleen and of the splenic flexure of the colon, the organs in this region most commonly affected, are shown by the stippling. When normal, the spleen lies considerably higher in the abdominal cavity than is generally supposed. Its anterior projection is shown here in dotted outline, with the lower end in contact with the splenic flexure of the colon. (From Crossen.)

Palpation.—The coils of the normal small intestine are not palpable. But in patients with moderately thin abdominal wall, hard fecal lumps, scybala, enteroliths, or large gall-stones are occasionally palpable. A hard tumor palpable in the region of the

small intestine may be malignant or may be the result of intussusception, volvulus, or fecal impaction. Or it may be the result of tabes mesenterica, due to matting of the omentum in tuberculous peritonitis. When an intestinal solid tumor pulsates, the patient should be placed in the knee-chest posture in order to determine whether the pulsation is transmitted to the tumor from the abdominal aorta. When palpation is practiced in this posture, the tumor falls away from the aorta, and the pulsation of this vessel is no longer imparted to the tumor.

Percussion.—The normal intestine yields tympany upon per-

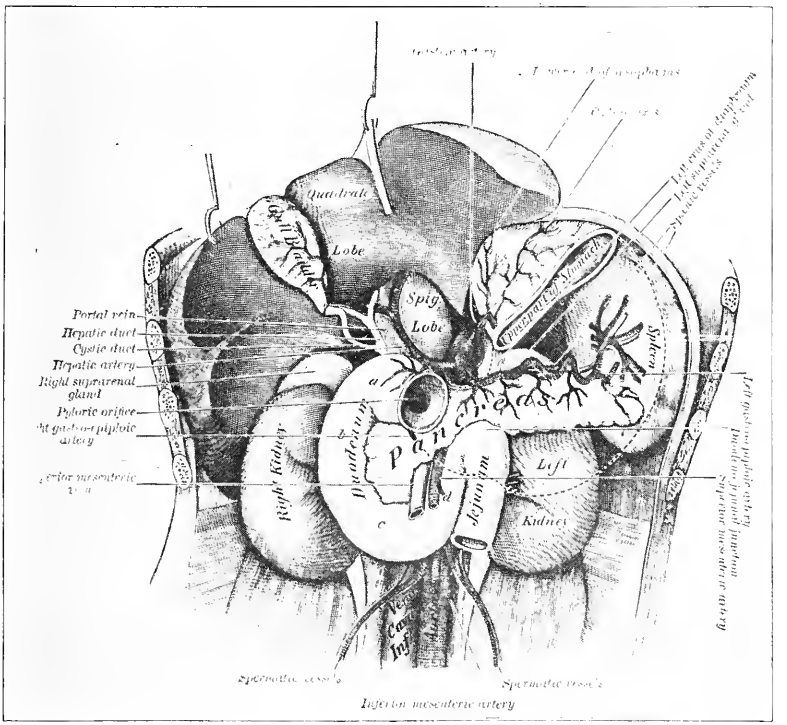


Fig. 199.—The duodenum, its four parts marked *a*, *b*, *c*, *d*. The liver is lifted up; the greater part of the stomach is removed, broken lines indicating its former position. (Gray, after Testut.)

cussion, the presence of dullness or flatness usually indicating a tumor. When the intestine is greatly distended with gas in peritonitis or intestinal obstruction the percussion note is slightly lowered in pitch, but, is still tympanitic. A similar note may be produced by the presence of a solid tumor surrounded by resonant intestine.

Auscultation.—The gurgling sounds or borborygmi which are frequently audible in the intestine have no significance. But in

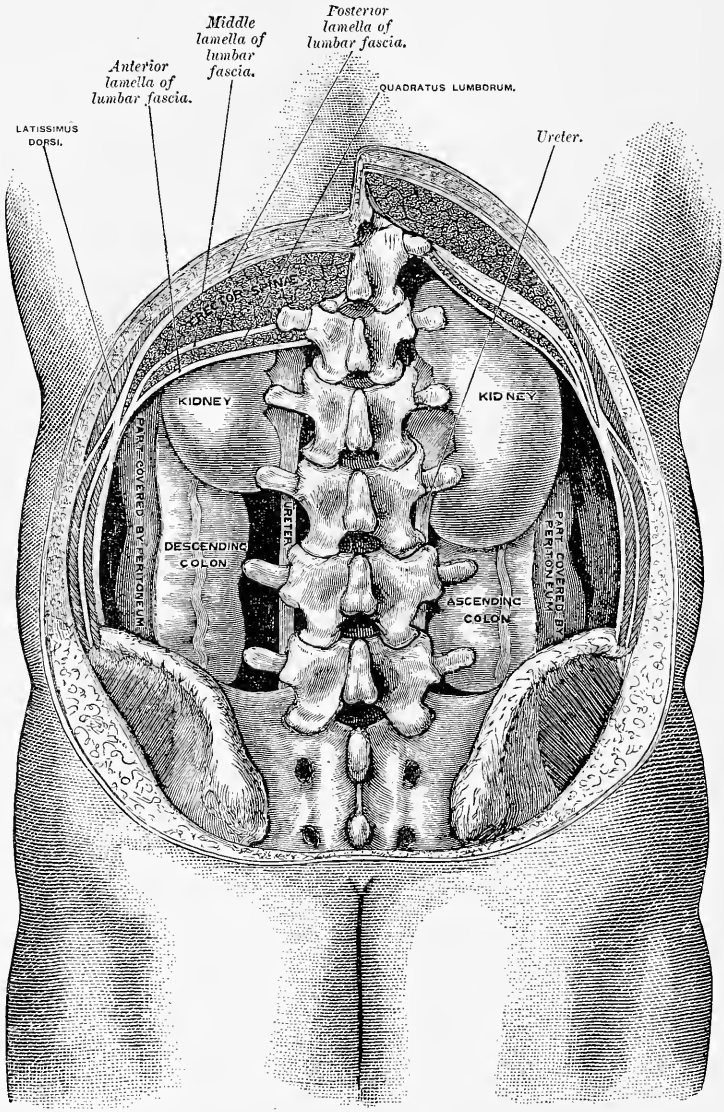


Fig. 200.—Relations of large intestine to kidneys. (From Gray.)

auscultation over a partial obstruction a gurgling sound may often be audible, which is due to the passage of fluid through the constricted lumen of the gut.

EXAMINATION OF THE LARGE INTESTINE

Clinical Anatomy.—The large intestine, comprising the cecum with the vermiform appendix, ascending colon, transverse colon, descending colon, and sigmoid flexure, is arranged in the form of a frame enclosing the coils of the small intestine on the right,

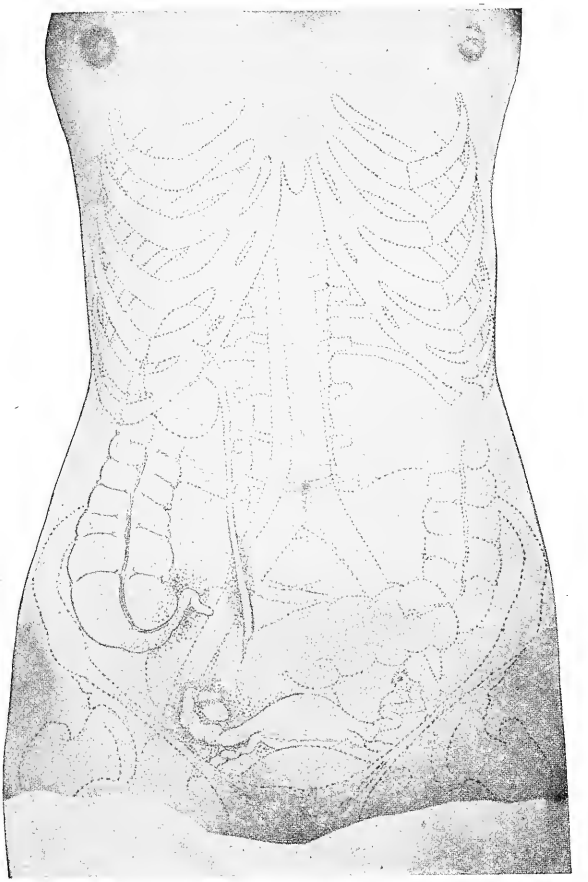


Fig. 201.—The right lower abdomen. The organs commonly affected, and the areas accordingly of particular interest, are indicated by the stippling. (From Crossen.)

superiorly, and upon the left. The cecum with the appendix occupies the right iliac and hypogastric regions. The ascending colon passes vertically upward in the right lumbar and right hypochondriac regions to the tenth right costal cartilage. The transverse colon passes across the abdomen, descending from the right

Fig. 202.—Indicating the point to seek for appendix tenderness. (From Crossen.)

Fig. 203.—Palpating for tenderness or a mass in the appendix region. (From Crossen.)

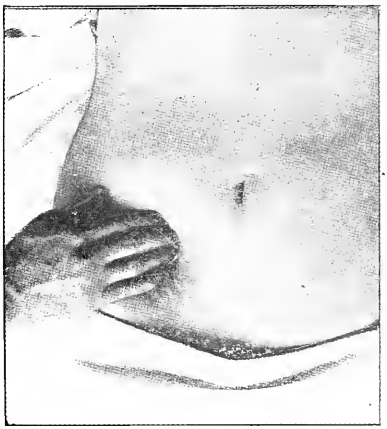


Fig. 204.—Palpating for the appendix itself, to determine whether or not there is any appreciable infiltration and thickening of it. When thickened, the appendix is felt as a small tender roll, deeply placed. (From Crossen.)

Fig. 205.—Another method of palpating the appendix. Beginning near the umbilicus, the fingers are carried in deeply and then brought slowly outward toward the anterior superior iliac spine. As the appendix passes under the examining fingers, it is felt as a small roll between the fingers and the posterior abdominal wall. (From Crossen.)

hypochondriac region to the upper part of the umbilical region; thence ascending obliquely into the left hypochondriac region to the lower extremity of the spleen. The descending colon passes vertically downward in the left lumbar and iliac regions, terminating in the sigmoid flexure in the hypogastric region.

Inspection.—In cases of gaseous distention of the large intestine

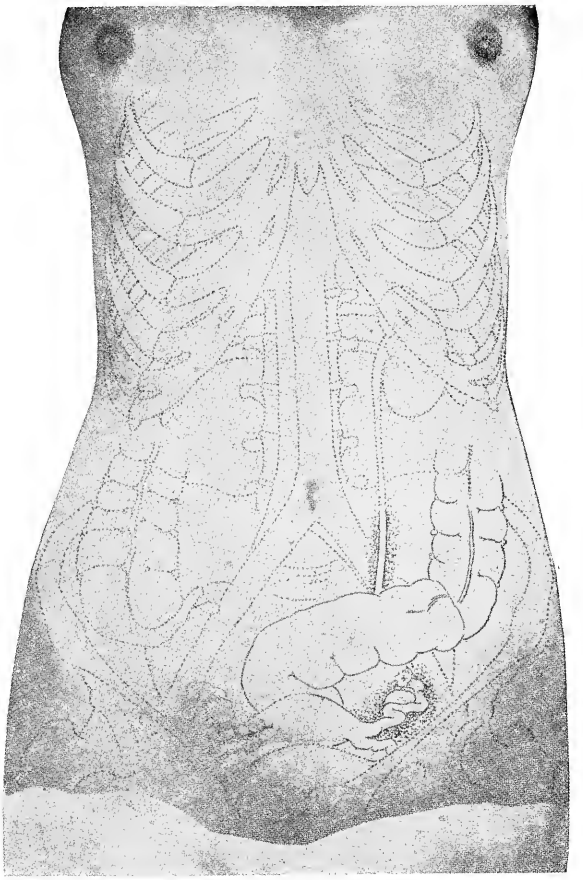


Fig. 206.—The left lower abdomen. The organs commonly affected, and the areas accordingly of particular interest, are indicated by the stippling. (From Crossen.)

there is often a visible tumefaction in the right and left lumbar and in the hypogastric region, which corresponds with the course of the ascending and descending colon. Gaseous distention of the transverse colon is apt to produce a protrusion just above the umbilicus in the umbilical region. (See Figs. 159, p. 312.)

Palpation.—In palpating the cecum the examiner, seated at the

right side of the patient applies the right hand over the gut and presses downward with a rolling movement, endeavoring in this manner to outline the gut. The gurgling murmur which is usually set up by this maneuver possesses little or no significance. During this examination an attempt should be made to palpate the vermiform appendix, which is often to be felt along a line from McBurney's point to the symphysis pubis as a cord about the size of a lead pencil, and about the length of the little finger. In this manner any tenderness or thickening of the appendix is to be determined if possible.

Fig. 207.—Palpating for tenderness or a mass in the umbilical region. (From Crossen.)

In palpating the ascending colon the patient should assume the left lateral decubitus, while the examiner applies the right hand to the abdomen at right angles to the course of the colon and by means of a rolling movement determines whether the colon is contracted or distended, and whether it contains fecal masses. Fecal accumulation is more common in the cecum and ascending colon than elsewhere. As mentioned, gurgling is particularly common in the cecum with little significance.

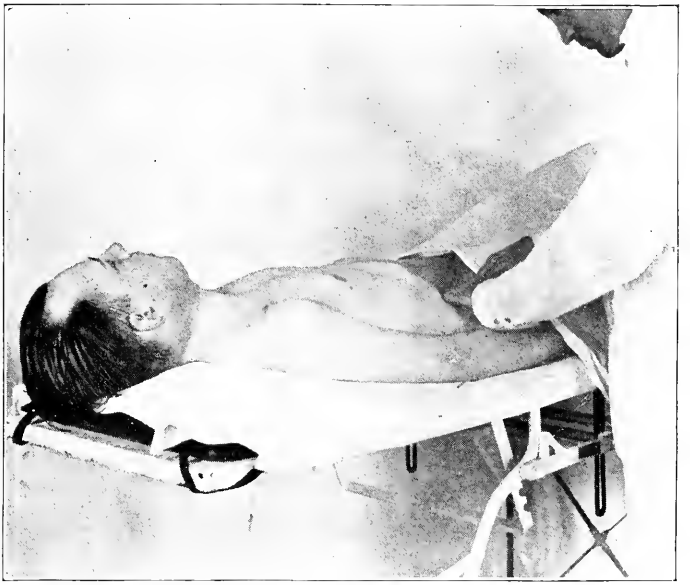


Fig. 208.—Palpation of ascending colon.

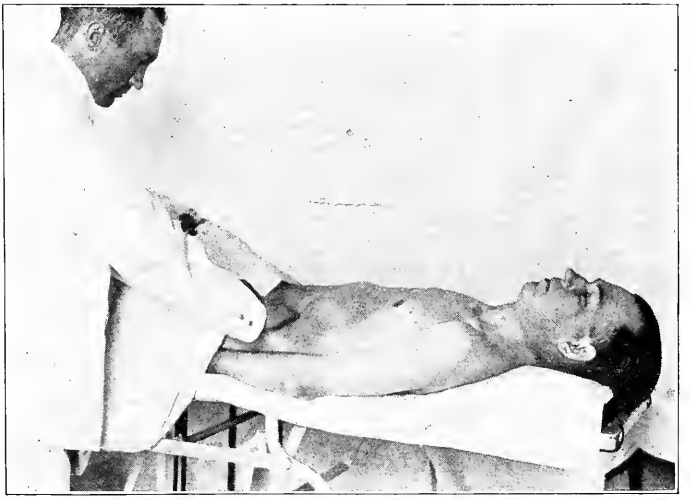


Fig. 209.—Palpation of the descending colon.

In palpating the *transverse colon* the patient lies upon the back while the examiner places both hands flat upon the abdomen in the umbilical region with the finger-tips extending somewhat above the umbilicus. The patient is instructed to breathe deeply and at the commencement of each expiration the examiner makes downward pressure with the finger-tips and endeavors to determine the position and state of this portion of the intestine. In palpation of this portion of the colon, as in other portions, the hand should be applied at right angles to the course of the intestine. Therefore, if the transverse colon pursues an arched course with the convexity downward, as it frequently does, the right and left halves of this portion of the intestine must be palpated in different directions. While the normal transverse colon is found at or above the umbilicus, in enteroptosis it may be encountered as low as the symphysis pubis.

The *descending colon* is palpated with the patient in the right lateral decubitus, the method corresponding in all respects with that employed in palpating the ascending colon.

Palpation of the *sigmoid flexure* is performed with the patient in the dorsal position, the examiner placing the fingers of the right hand upon the abdomen at right angles to the course of the gut, rolling the intestine between the finger-tips and the iliac fossa, searching for tenderness, muscular rigidity, tumor, or fecal impaction. As a rule, impacted feces will pit upon pressure, whereas a hard tumor will not.

EXAMINATION OF THE PANCREAS

Clinical Anatomy.—The pancreas is an elongated solid organ, comprising a head, neck, body, and tail, situated deeply in the epigastric region, its tail, however, extending into the left hypochondriac region.

The pancreas measures approximately six inches in length and its location in the abdominal cavity corresponds to the level of the first and second lumbar vertebræ. The head of the pancreas is encircled by the second and third portions of the duodenum, the common bile duct intervening between the two structures, while the tail extends toward the left and is in relation with the hilum of the spleen.

The pancreas is covered anteriorly by the peritoneum of the

lesser sac, this surface being in relation with the posterior wall of the stomach, the transverse colon, and a few coils of the small intestine. Posteriorly the pancreas rests upon the abdominal

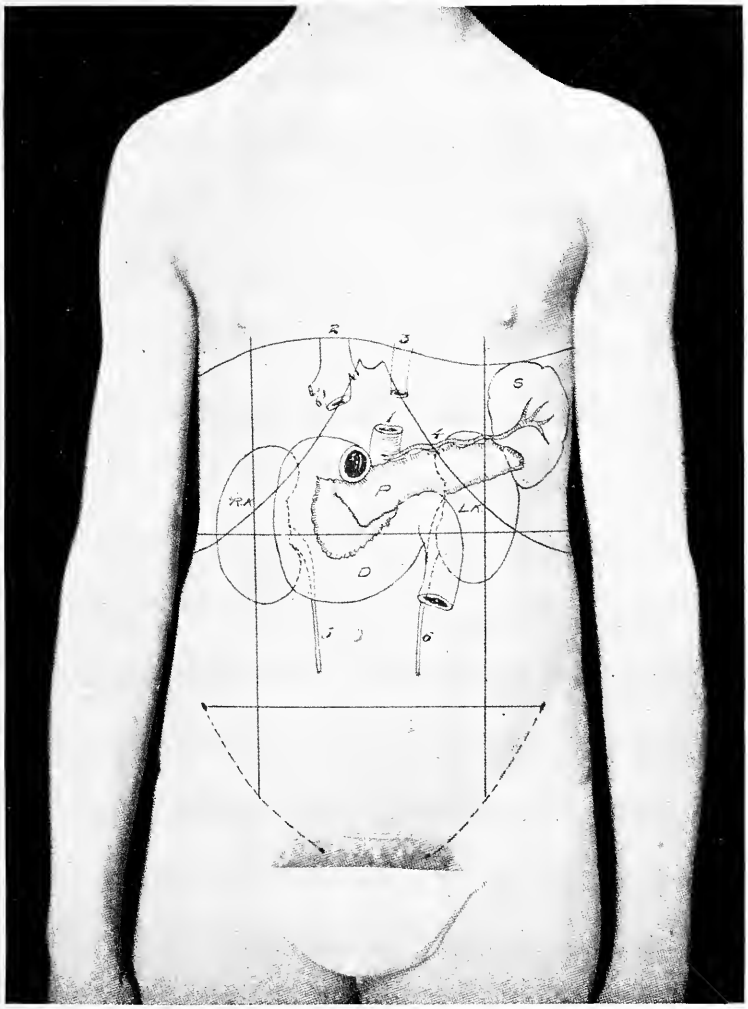


Fig. 210.—Relations of pancreas to adjacent viscera.
1, aorta; *2*, inferior vena cava; *3*, esophagus; *4*, splenic artery; *5* and *6*, uretus; *RK*, right kidney; *LK*, left kidney; *D*, duodenum; *P*, pancreas; *S*, spleen.

aorta and inferior vena cava, the right crus of the diaphragm, the renal and portal veins, and the left kidney.

The pancreas crosses the lower portion of the epigastric region three inches above the umbilicus.

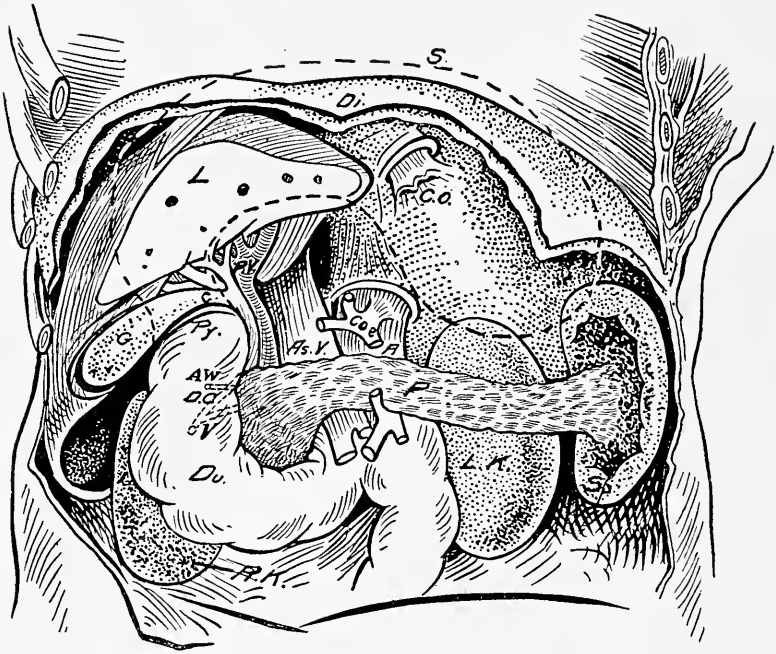


Fig. 211.—Topographical relations of liver, bile passages, and pancreas. (From Eisendrath.)

S, stomach in dotted outline; *L*, cut edge of right lobe of liver; *PV*, portal vein; *As.* V, ascending vena cava; *DC*, common duct; *G*, gall-bladder; *Du*, duodenum; *Sp*, spleen; *RK*, right kidney; *LK*, left kidney; *Coe*, celiac axis; *A*, aorta; *CO*, cardiac orifice of stomach; *Di*, diaphragm; *P*, pancreas; *Py*, pyloric end of stomach; *V*, duct of Wirsung, joining with common bile-duct; *AW*, accessory duct of Wirsung.

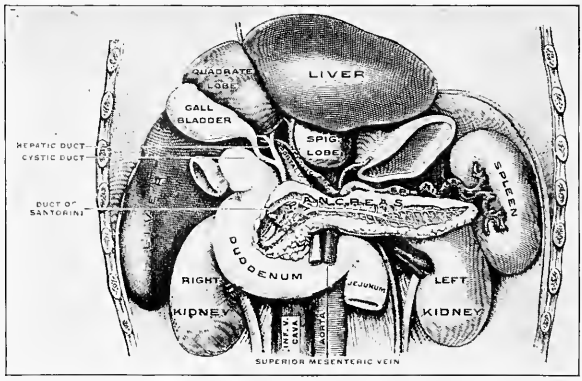


Fig. 212.—Position and relations of pancreas. (From Gray.)

Physical Examination.—Owing to the deep location of the pancreas within the abdomen, physical examination of the organ is not very satisfactory, being limited to palpation.

The normal pancreas is seldom palpable save in a very emaciated patient with diastasis of the rectus muscles.

When a mass is encountered in the deeper portion of the epigastric region it is apt to be a cyst or solid tumor of the pancreas. Pancreatic tumors in the great majority of instances are carcinomata.

A tumor arising from the pancreas may be mistaken for a

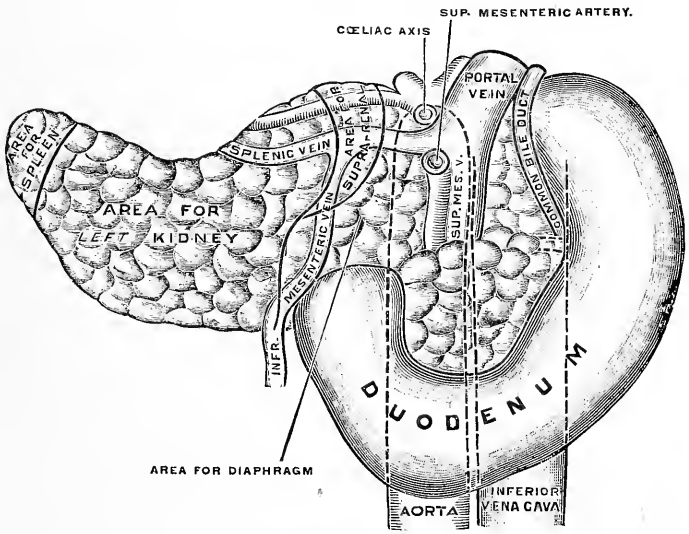


Fig. 213.—Pancreas and duodenum. Posterior view. (From Gray.)

tumor of the pylorus, a distended gall-bladder, aortic aneurism or a tumor of the transverse colon. A pyloric tumor is always more superficial than is a pancreatic growth. Moreover, a tumor at the pylorus is freely movable and produces pyloric stenosis with a consequent dilatation of the stomach. A distended gall-bladder exhibits lateral mobility, is more superficial than is a tumor of the pancreas, and is not accompanied by glycosuria and fatty stools as is a pancreatic new-growth.

A pancreatic tumor may be raised with each pulsation of the aorta, simulating aortic aneurism; but while the pulsation of aortic aneurism is expansile and exerted in all directions, that of

an overlying pancreatic tumor is not, the mass merely being raised with each pulsation of the subjacent aorta.

A tumor of the transverse colon is very near the surface of the abdomen, it is freely movable, is prone to cause constipation, and blood is frequently found in the stools with such growths.

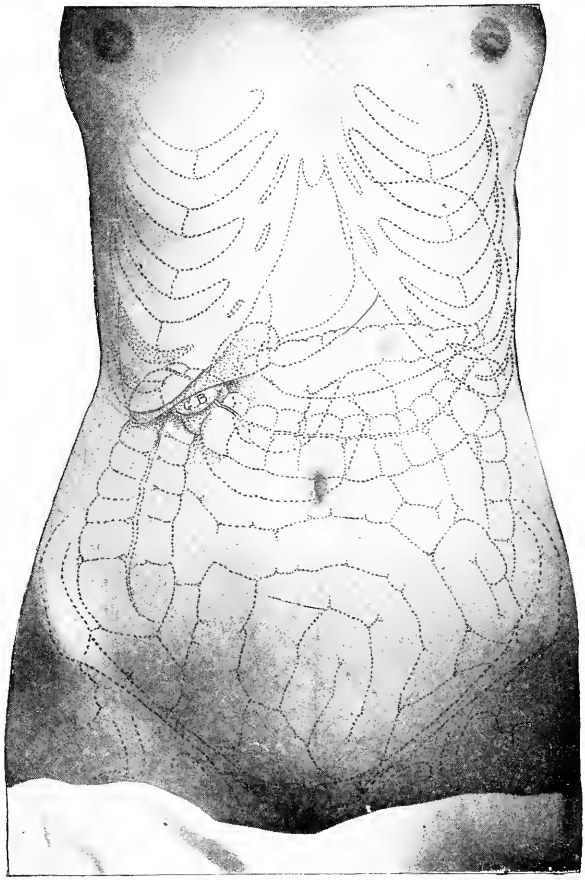


Fig. 214.—The right upper abdomen. The site of the gall-bladder, the area of particular interest in this region is indicated by the letters *G.B.* (From Crossen.)

CHAPTER XXIV

EXAMINATION OF THE LIVER AND GALL-BLADDER

Clinical Anatomy.—The liver, the largest gland of the body, occupies the upper and right portion of the abdominal cavity, lying in the right hypochondriac region, a larger portion of the epigastric region, the thin extremity of the left lobe extending into the left hypochondriac region, and the lower extremity of the right lobe invading the right lumbar region for a short distance. The liver is roughly wedge-shaped, with the wide base directed toward the right, and the thin sharp edge of the wedge directed toward the left side of the abdomen. The normal adult liver measures eight to nine inches transversely, six to seven inches vertically at the base of the wedge, and four to five inches antero-posteriorly at a point on a level with the upper border of the right kidney. (See Fig. 214, p. 352.)

The liver is divided into two unequal portions, the right and left lobes, by the falciform ligament and longitudinal fissure, the right lobe greatly exceeding the left lobe in size. At the point where the falciform ligament joins the inferior margin of the liver there is a small notch, the umbilical notch, which is situated at the level of the ninth right costal cartilage one inch to the right of the median line. Slightly beyond this notch the liver presents a second notch in which is lodged the fundus of the gall-bladder, corresponding to the junction of the ninth rib and the right border of the rectus muscle. (See Fig. 149, p. 300.)

The *upper surface* of the liver, smooth and convex, is closely applied to the concave under surface of the diaphragm. Upon its central portion the superior surface of the liver presents a shallow depression, the *cardiac depression,* corresponding to the position of the heart upon the upper surface of the diaphragm.

The *anterior surface* of the liver is applied to the under surface of the diaphragm which separates it from the lower ribs and their cartilages upon the right and left sides, while in the median line it comes into direct contact with the anterior abdominal wall in the costal angle.

The *right* and *posterior surfaces* of the liver are in contact with

the under surface of the diaphragm, which separates the liver from the right pleural cavity and lower border of the lung.

The *under surface* of the liver, directed backward and toward the left, is in relation with the stomach, the hepatic flexure of the colon, the right kidney and supra-renal, the second portion of the duodenum and the gall-bladder.

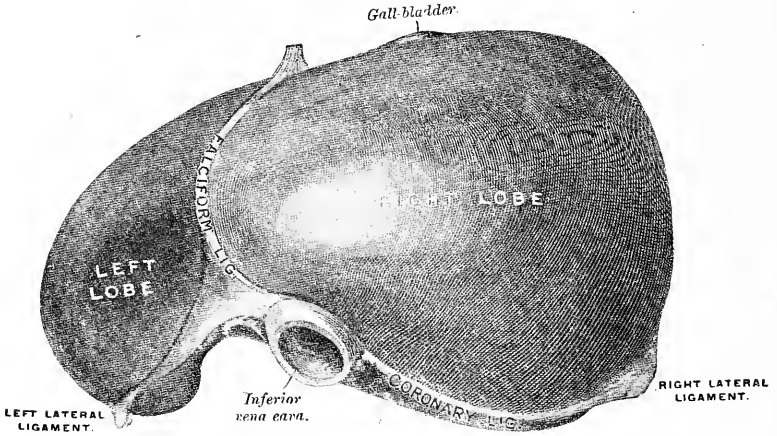


Fig. 215.—Superior surface of liver. (From Gray.)

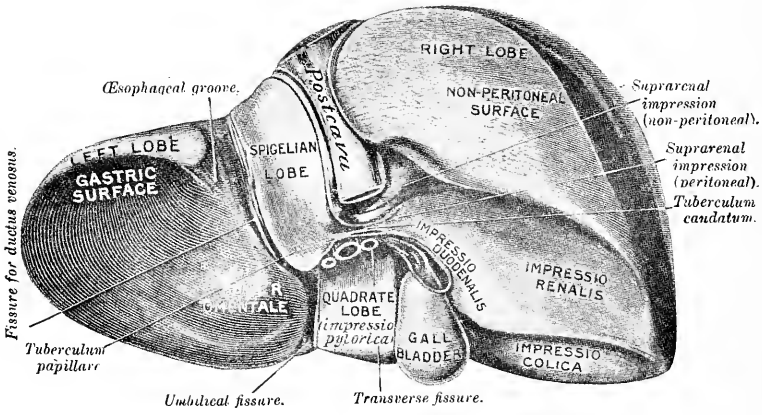


Fig. 216.—Inferior surface of liver. (From Gray.)

Surface Topography.—The *upper border* of the liver corresponds to the level of the lower border of the sixth rib in the mid-clavicular line, the lower border of the eighth rib in the mid-axillary line, and the lower border of the tenth rib in the scapular line.

The *lower border* of the liver corresponds to a line drawn downward and to the right from the lower border of the sixth rib in the left mid-clavicular line, the point on the surface corresponding to the left extremity of the organ, the line crossing the left costal arch at the eighth costal cartilage, the median line four inches below the ensiform cartilage, the right costal arch at the ninth costal cartilage, the lower border of the tenth rib in the mid-axillary line, and the lower border of the eleventh rib in the scapular line.

Inspection.—Enlargement of the liver produces prominence of the right costal margin and fullness in the right hypochondriac and epigastric regions. Rarely it is possible to perceive the lower

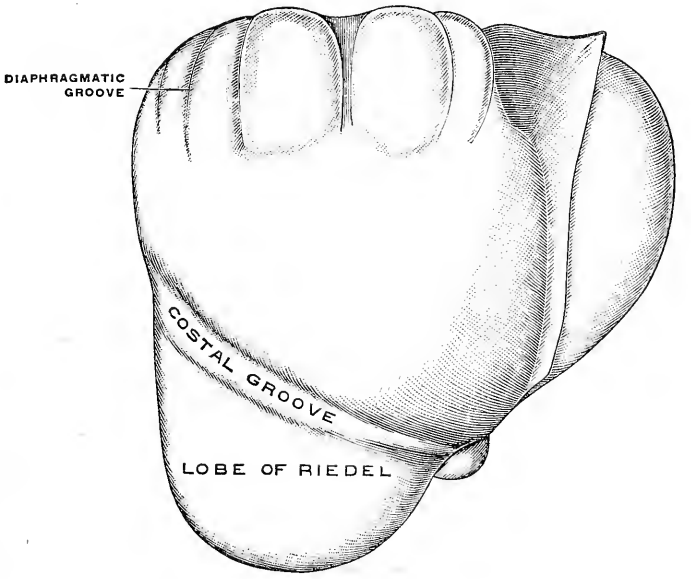


Fig. 217.—Corset liver. (From Gray.)

border of the enlarged liver rise and fall with the respiratory movements. A small local enlargement of the lower portion of the liver is more readily perceived by inspection than is a moderate symmetrical enlargement. As a rule, in examination of the liver inspection is inferior to palpation, impressions based upon inspected signs alone often leading to error. (See Fig. 220, p. 357.)

Systolic pulsation of the liver is occasionally visible in tricuspid regurgitation, though usually the pulsation requires bimanual palpation for its recognition. A transmitted impulse

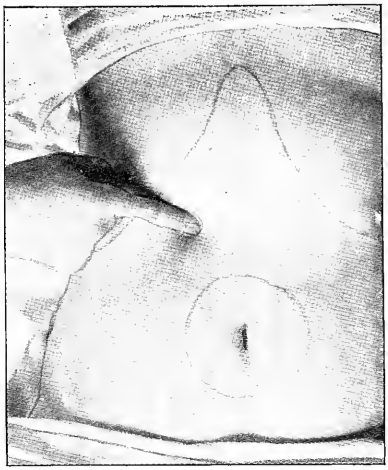


Fig. 218.—Indicating the site for tenderness or a mass due to disease of the gall-bladder. It may be found anywhere from the point indicated downward and outward to the margin of the ribs on the right side. (From Crossen.)

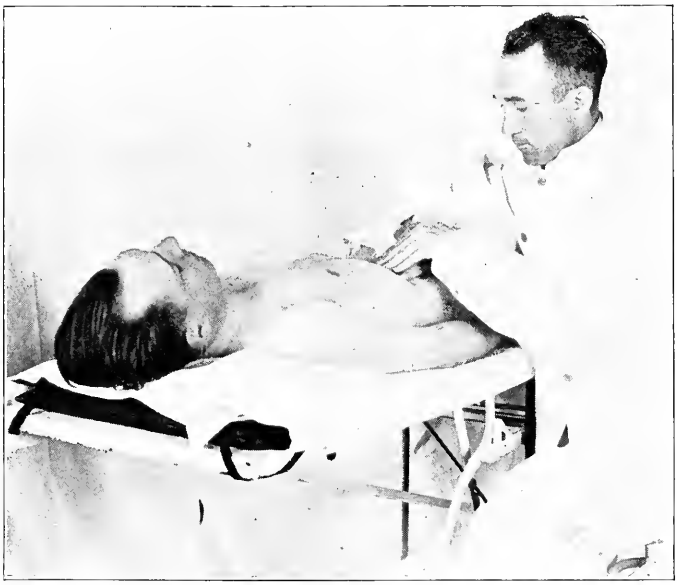


Fig. 219.—Palpation of liver.

conveyed to the liver by the impact of an over-acting heart should not be mistaken for a true pulsation of the organ.

Palpation.—*Technic.*—The patient should assume the dorsal decubitus with the knees drawn up and supported. The examiner, seated by the right side of the patient, should feel with the finger-tips of the right hand for the lower margin of the liver just below the lower costal margin. If it be found that the liver pro-

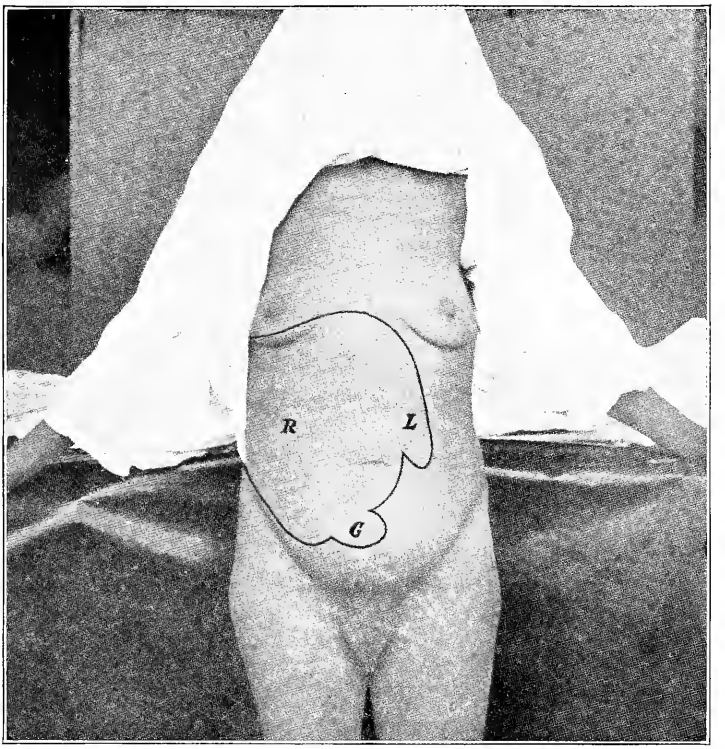


Fig. 220.—Hepatic enlargement due to carcinoma of head of pancreas. *R*, right lobe of liver; *L*, left lobe of liver; *G*, distended gall-bladder. (From Eisendrath.)

jects below the costal margin, the examiner places both hands flat upon the abdomen and by downward pressure causes the finger-tips to glide over the exposed portion of the liver, searching for any abnormality in contour.

If the lower margin of the liver is not encountered below the costal margin, the examiner should place the finger-tips of both hands just below the costal arch and press inward as far as possible. The patient is directed to inspire deeply, when during in-

spiration the liver will be felt to descend with the descent of the diaphragm and become palpable at the completion of inspiration.

By the first maneuver an enlarged liver will be revealed projecting below the costal arch; while, by the second maneuver a normal or contracted liver may be palpated.

Tenderness upon palpation of the liver may arise from disease of the liver or gall-bladder. Hepatic tenderness is present in hepatic congestion, peri-hepatitis, acute yellow atrophy, and hypertrophic biliary cirrhosis. Cholecystitis and cholelithiasis are attended by tenderness having its point of maximum intensity at

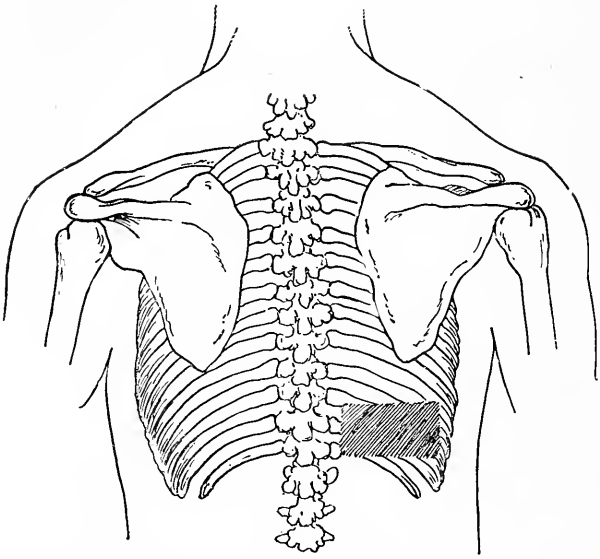


Fig. 221.—Dorsal pressure point in chololithiasis. (From Austin.)

the junction of the ninth costal cartilage and the outer border of the right rectus muscle.

Systolic Pulsation of the liver, recognized by bi-manual palpation of the liver is a sign of tricuspid regurgitation.

Enlargement of the liver accompanies fatty infiltration of the organ, chronic passive congestion of the organ and amyloid disease of the liver; Weil's disease, hepatic abscess, carcinoma or gumma or leukemia, and hypertrophic cirrhosis, echinococcus cyst or Banti's disease, or acute infectious fevers.

Reidel's lobe, a congenital anomaly of the liver, sometimes palpable below the right costal arch as well as a similar deformity of the liver incident to prolonged tight lacing in female subjects,

may be mistaken for a tumor of the liver. But when a tumor is suspected it should be borne in mind that the liver comes third in the order of frequency as the site of abdominal carcinomata. Carcinoma of the liver produces considerable degree of enlargement of the organ, often associated with carcinomatous nodules projecting from the surface of the organ. These nodules are hard and often umbilicated.

A kidney enlarged as the result of hydro-nephrosis, tuberculosis, or malignant disease, may be mistaken for an enlargement of the liver. In this connection the examiner should remember that the enlargements springing from the kidney occupy a more

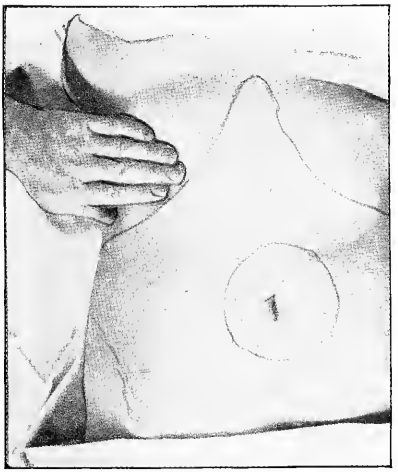


Fig. 222.—Palpating for general tenderness of the liver. (From Crossen.)

Fig. 223.—Showing the site for tenderness of the left lobe of the liver. (From Crossen.)

lateral position in the abdomen; also that the hepatic flexure of the colon is united to the lower pole of the kidney with the result that when the kidney enlarges it carries the colon in front of it, whereas an enlargement of the liver descends in front of the hepatic flexure, the colon lying external to the tumor. (See Fig. 200, p. 342.)

Percussion.—While palpation reveals the presence of localized enlargements of the liver and of the organ as a whole, percussion is usually more serviceable in determination of changes in the size of the organ and displacements.

The Areas of Hepatic Dullness and Flatness.—Upon percussing

downward over the surface of the thorax and abdomen in the mid-clavicular, mid-axillary, and scapular lines from an area of frank vesicular resonance, the percussion note will become impaired or dull when the point is reached where the upper limit of the liver is overlaid by the pulmonary structures. The point of change in the note indicates the upper limits of the area of hepatic dullness. The upper limits of this area are encountered in the fourth interspace in the mid-clavicular line, the seventh interspace in the mid-axillary line, and at the eighth interspace in the scapular line. (See Fig. 100, p. 213.)

Upon continuing the percussion downward along these lines, substituting light for forcible percussion a point is reached in

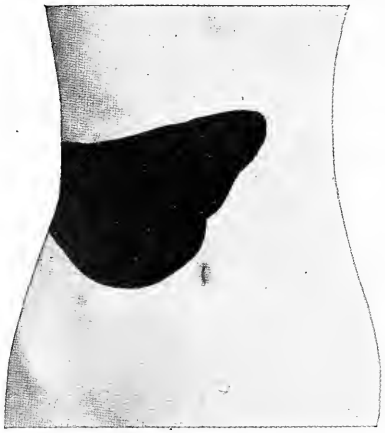


Fig. 224.—Indicating the region for dullness from enlarged liver. (From Crossen.)

which the dullness gives place to flatness, indicating the upper limit of the region where the liver is in direct contact with the adbominal wall, the upper limit of the *area of hepatic flatness.* The upper limits of this area are found in the normal subject at the sixth rib in the mid-clavicular line, the eighth rib in the mid-axillary line, and the tenth rib in the scapular line.

If the percussion is continued downward along the same lines the flat note will be replaced by intestinal tympany when the lower limit of the liver is attained. The points of change indicating the lower limit of the area of hepatic dullness are encountered at the ninth rib in the mid-clavicular line, the tenth rib in the mid-axillary line, while in the scapular line the flatness of the liver is continuous with that produced by the kidney.

The lower limit of the area of hepatic flatness in the epigastric region lies three inches below the ensiform cartilage. Thus, it is observed that the area of hepatic dullness and flatness extends across the right upper portion of the abdomen, in a direction downward and toward the right; that posteriorly it is continuous with the flatness of the right kidney; and that anteriorly it blends with the right border of the area of cardiac flatness.

Enlargement of the Liver, the principal causes of which have been detailed under palpation, is indicated by an extension of the area of hepatic dullness in one or more directions. An apparent enlargement of the hepatic area or an apparent increase in the area of hepatic dullness may be caused by enlargement of the right heart, a right sided pleural effusion or sub-phrenic abscess.

Decrease of the Size of the Liver, occurring in atrophic cirrhosis or acute yellow atrophy is indicated by a decrease in the extent of the area of hepatic dullness upon percussion. An apparent decrease in the size of the liver may be caused by hypertrophic emphysema encroaching upon the hepatic area superiorly or by coils of the intestine in tympanites encroaching upon the area inferiorly.

Displacement of the Liver

Downward Displacement of the liver or hepatoptosis may be occasioned by the pressure upon the upper diaphragmatic surface by hypertrophic emphysema, pneumothorax, right sided pleurisy with effusion, cardiac hypertrophy or pericardial effusion. The liver may be pressed downward by a sub-phrenic abscess. Finally, the liver may participate in a general visceroptosis and occupy a lower level in the abdominal cavity than is normal. Downward displacement of the liver is separated from increased size of the organ by demonstrating by percussion that the upper limit of hepatic dullness occupies a lower level than is normal.

Upward Displacement of the liver occurs as a result of pressure exerted upon the under surface of the organ by ascites, tympanites or abdominal tumor. Upward displacement may, however, be the sequence of decreased intra-thoracic pressure as occurs with fibroid retraction of the right lung; or may be the

result of paralysis of the diaphragm. In upward displacement of the liver it may be demonstrated by percussion that both the upper and lower limits of hepatic dullness are elevated, not the upper limit alone.

Auscultation.—Auscultation over the hepatic area may elicit occasionally a friction sound in connection with peritoneal involvement in peri-hepatitis. In cases of cholelithiasis, gall-stone crepitations have been described.

CHAPTER XXV

EXAMINATION OF THE SPLEEN, KIDNEYS, BLADDER, AND URETERS

EXAMINATION OF THE SPLEEN

Clinical Anatomy.—The spleen is a solid organ situated deeply in the left hypochondriac region, its upper and inner extremity crossing the left mid-clavicular line into the epigastric region. It is placed obliquely in the abdominal cavity, between the fundus of the stomach and the left kidney and diaphragm, its long axis corresponding to the course of the tenth rib. The spleen moves with the movements of respiration. (See Fig. 210, p. 349.)

The spleen is separated by the diaphragm from the ninth, tenth, and eleventh ribs. The pleural cavity intervenes between the portion of the organ in contact with the diaphragm and the chest wall. The upper portion of the spleen also has the lower border of the left lung overlying it. The spleen measures five inches in its longest diameter, and three inches in width.

The spleen corresponds to an area on the surface of the body from the ninth to the eleventh ribs, inclusive, its inner end being one and one-half inches from the mid-spinal line, its outer end reaching as far forward as the mid-axillary line.

Inspection.—The normal spleen gives no visible signs of its presence. When enlarged it produces bulging of the abdominal wall in the right hypochondriac and epigastric regions, which may be moderate, or as in the case of the leukemic spleen, may be extreme.

Palpation.—*Technic.*—With the patient in the dorsal decubitus the examiner should apply the hand flat upon the abdomen so that the finger-tips can be inserted beneath the left costal arch. If the spleen is enlarged, no difficulty will be experienced in palpating the lower border. However, the normal organ is not palpable. If the spleen is not encountered by the procedure described, the patient is directed to inspire deeply. Upon making pressure at the commencement of full inspiration the lower border of the organ

will be felt as it descends with the diaphragm during respiration.

Some difficulty may arise in determining whether a palpable tumor below the left costal arch is the spleen or kidney. In this connection it should be recalled that the spleen moves with respiration whereas the kidney does not. Moreover, the kidney is overlapped by the large intestine, an enlargement of the kidney pushing the tympanitic gut before it, whereas the spleen occupies a position in front of the intestine. Finally, the shape of the two

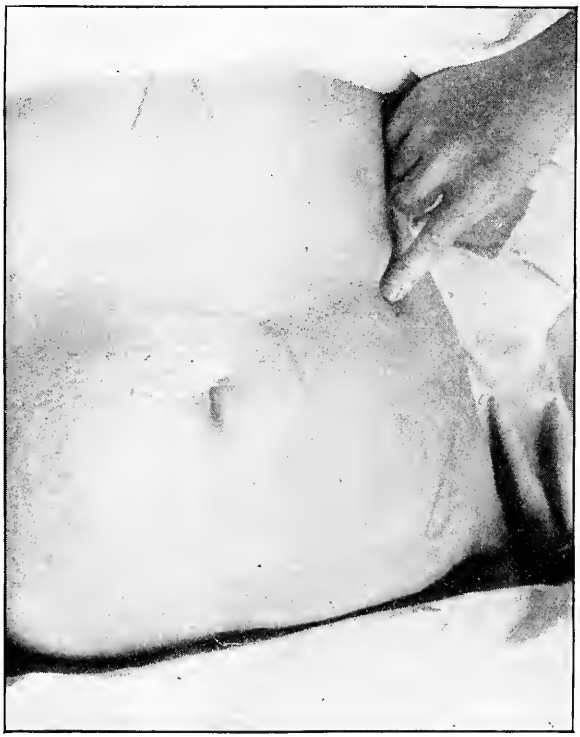


Fig. 225.—Indicating the area to search for splenic tenderness or enlargement. When the spleen is diseased it usually becomes enlarged and heavy and sinks below the margin of the ribs at the point indicated. (From Crossen.)

organs differs, the spleen being more or less tetrahedral with a sharp edge, while the kidney is smooth and reniform.

Aside from leukemia, splenic enlargement occurs in the course of many acute infections, in cirrhosis of the liver, and Banti's disease, and when the seat of a tumor. The organ is also enlarged in acute splenitis, in which the area of splenic dullness extends rapidly, accompanied by local pain and tenderness. In chronic

malaria the spleen is enlarged, constituting the "ague-cake" of this condition. Amyloid infiltration produces symmetrical enlargement of the spleen, of firm consistence. In movable spleen the organ is first larger than normal, and later small.

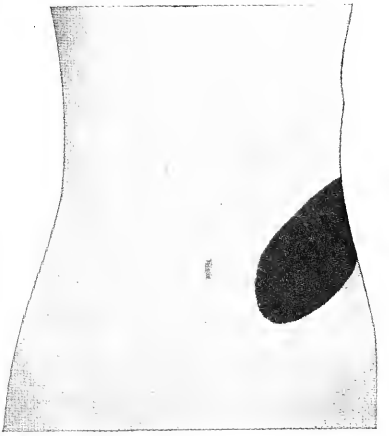


Fig. 226.—Indicating the region for dullness from enlarged spleen. (From Crossen.)

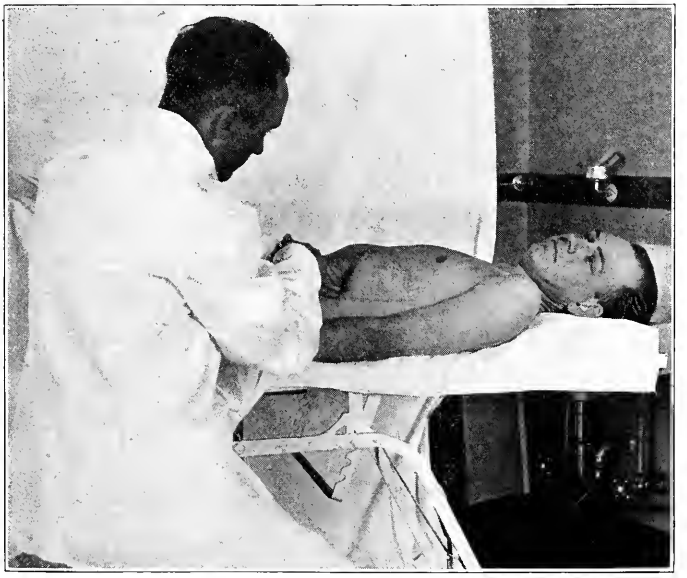


Fig. 277.—Palpation of the spleen.

Tenderness on palpation of the spleen may be due to acute congestion, peri-splenitis, or infarction of this organ. Signorelli's

Point, to which pain is referred in splenic disease is situated just below the junction of the fifth left costal cartilage and the mid-clavicular line.

Downward Displacement of the spleen may be caused by increased sub-phrenic pressure by emphysema, left sided pneumothorax or pleural effusion, massive pericardial effusion, or thoracic neoplasm. The spleen may drop downward owing to relaxation of its ligaments, constituting the so-called movable or wandering spleen, which may be encountered in the pelvis. If the pedicle becomes twisted, the spleen becomes enlarged and later contracts, becoming smaller than the normal organ. The spleen also occupies a lower level than normal in Glenard's disease. A

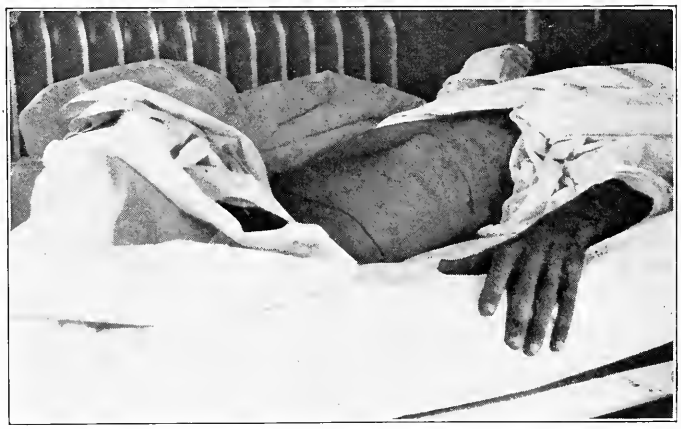


Fig. 228.—Splenic enlargement in leukemia.

floating or movable spleen should not be mistaken for an abdominal tumor. The shape of the spleen, if it is possible to palpate it accurately, the presence of the notch, and its absence from its normal position as indicated by resonance in the splenic area, aid in differentating it from tumor.

Upward displacement of the spleen occurs when it is pressed upon by ascites, tympanites, or large abdominal tumor. Fibroid retraction of the left lung or paralysis of the diaphragm will cause it to occupy a higher level in the abdominal cavity than is normal.

Percussion.—In percussion of the spleen the patient may lie upon the right side, stand, or sit. To delineate the superior and inferior borders of the organ the examiner percusses downward from the area of frank pulmonary resonance in the axillary re-

gion, until the dulling of the note indicates that the upper border of the spleen has been reached. Upon continuing the percussion downward, the dullness will give place to the intestinal tympany when the lower border is reached. The anterior border is located by percussing outward from the median line in the tenth intercostal space. The posterior border is defined by means of percussion carried outward from the left side of the vertebral column. In delimiting the spleen light percussion is necessary on account of the proximity of the organ to the stomach and colon, which serve to add a tympanitic quality to the note elicited upon forcible percussion.

Percussion of the spleen confirms the palpatory signs as to enlargement and displacement of the organ. As in the case of other solid organs auscultatory percussion may be employed to advantage in outlining the position of the spleen.

Auscultation yields little data concerning the spleen, except to determine a friction sound in cases of peri-splenitis.

EXAMINATION OF THE KIDNEYS

Clinical Anatomy.—The kidneys are situated in the posterior portion of the abdomen, behind the peritoneum, on either side of the vertebral column. The upper extremities of the kidneys correspond to the level of the upper border of the twelfth dorsal vertebra; the lower extremities are on a level with the third lumbar vertebra. Owing to its relation with the right lobe of the liver, the right kidney occupies a slightly lower level than does the left.

The kidney is approximately four and a half inches in length, two and a half inches in width, and two inches in thickness. The left kidney is somewhat longer and narrower than is the right.

Posteriorly the kidneys rest upon the muscles of the posterior abdominal wall, the psoas, quadratus lumborum, fascia of the transversalis, and the diaphragm. The anterior surface of the right kidney is in relation with the visceral aspect of the liver, the descending portion of the duodenum, and the hepatic flexure of the colon and adrenal. The anterior surface of the left kidney is in relation with the posterior surface of the stomach, the spleen, the body of the pancreas, the jejunum, adrenal and splenic flexure of the colon.

The kidneys are located in the epigastric, umbilical, and hypochondriac and lumbar regions on either side. The superior pole

extends as high in the epigastric region as a transverse line drawn about two inches below the ensiform process. The inferior pole extends below the sub-costal line, only slightly in the case of the left; and to a greater degree in the case of the right kidney.

In the female subject the kidneys are placed lower than they are in the male. In both sexes the upper pole is nearer the mid-

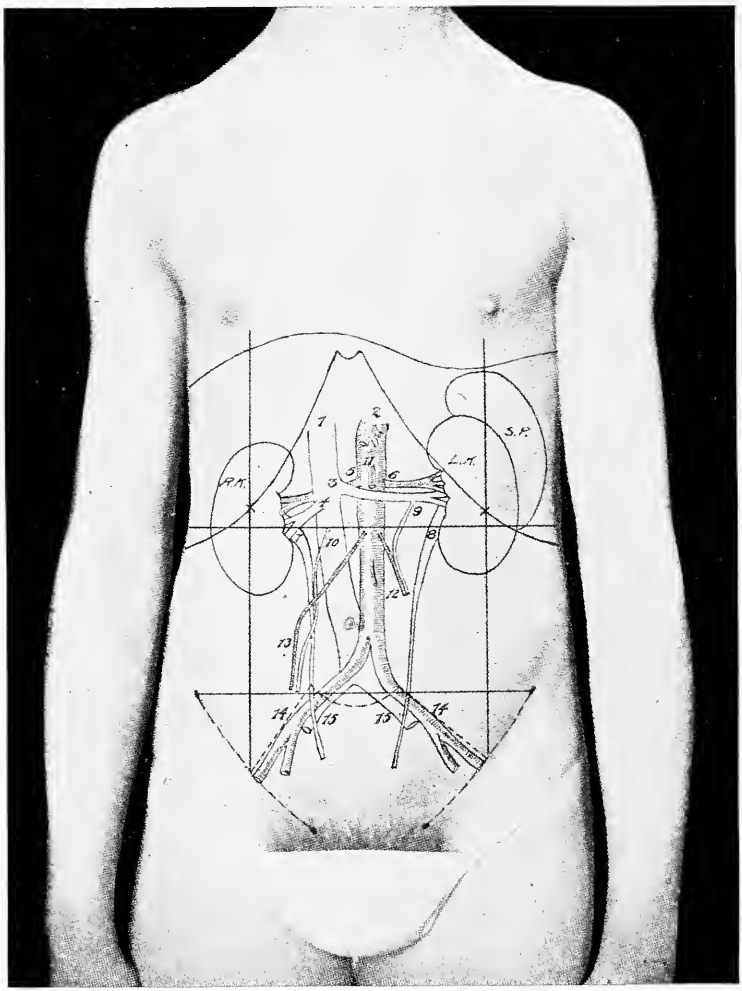


Fig. 229.—Surface markings of kidneys, uterus, and abdominal vessels. Anterior view. (From Eisendrath.)

1, inferior vena cava; *2*, aorta—celiac axis just below *2*; *3* and *4*, right and left renal veins; *5* and *6*, right and left renal arteries; *7* and *8*, right and left ureter; *9*, left spermatic vein; *10*, right spermatic vein; *11*, superior mesenteric artery; *12* and *13*, right and left spermatic arteries; *14*, external iliac arteries; *15*, external iliac veins; *RK*, right kidney; *LK*, left kidney; *SP*, spleen.

dle line than is the inferior. In the infant the kidneys are relatively larger than in the adult. The relative weight of the kidney in the adult to the entire body weight is 1:240, whereas in the infant it is 1:120.

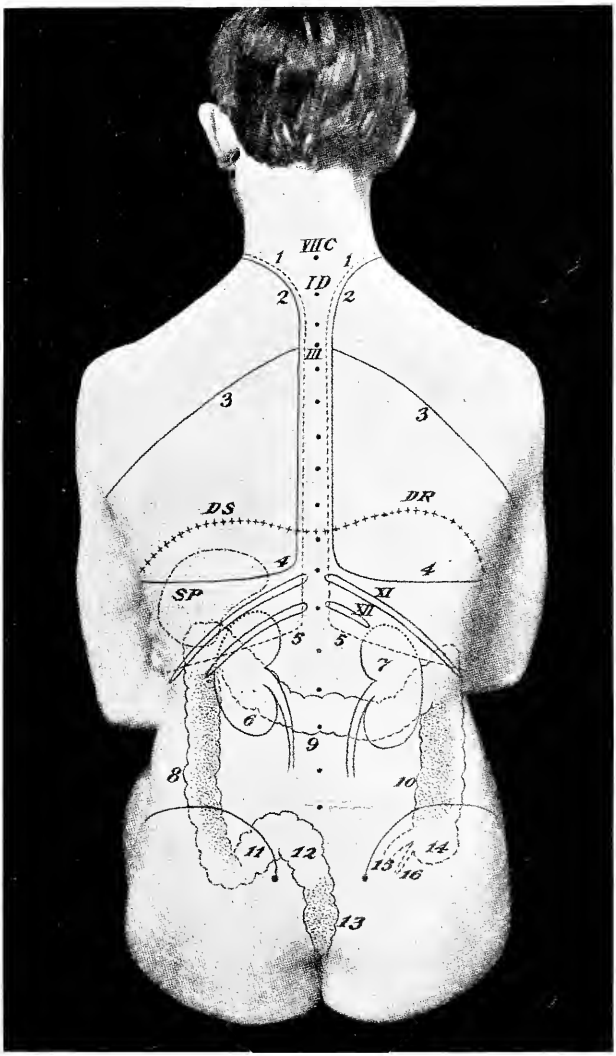


Fig. 230.—Surface markings of pleura, lungs, interlobar fissures, and relations of pleural cavities to kidneys. (From Eisendrath.)

1, apical and mediastinal pleura; *2*, upper and posterior margins of lungs; *3*, interlobar fissure between upper and lower lobes on each side; *4*, lower border of lung; *5*, lower border of pleura; *6*, left kidney; *7*, right kidney; *8*, descending colon; *9*, transverse colon; *10*, ascending colon; *11* and *12*, sigmoid flexure; *13*, rectum and anal canal; *14*, cecum; *15*, termination of ileum; *16*, appendix; *DS*, left dome of diaphragm; *DR*, right dome of diaphragm; *SP*, spleen.

Inspection.—The normal kidney yields no evidence of its presence on inspection. However, tumors of the organ, hydro-nephrosis, or large cystic kidney produces bulging in the umbilical and lumbar regions. The costal arch is pressed forward. The mass does not move with the movements of respiration. It is to be remembered that a tumor of the kidney in a child is probably sarcoma. Peri-nephric abscess may produce bulging in the costo-vertebral angle posteriorly. Concomitant signs are edema of the skin, and the presence of fluctuation with septic symptoms.

Palpation.—*Technic.*—The patient assumes the dorsal decubi-

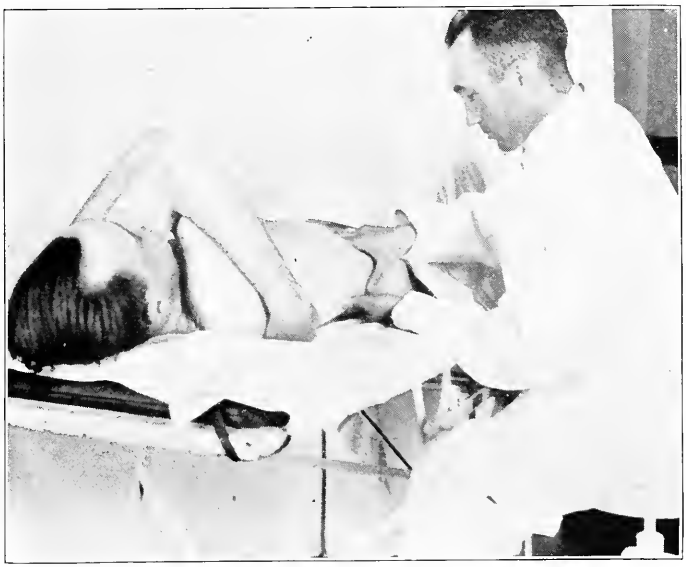


Fig. 231.—Palpation of the kidney.

tus, with the knees drawn up and supported, the arms hanging loosely at the sides.

The examiner sits by the side to be examined. Bi-manual palpation is employed. The examiner makes pressure with the index and middle fingers of the left hand in the costo-vertebral angle, the interval just below the twelfth rib near the spinal column. The examiner places his right hand upon the anterior abdominal wall one inch external to the linea semilunaris, his fingers directed upward, just below the costal arch. The patient breathing deeply, the examiner makes downward pressure with the fingers of the right hand, at the same time making pressure

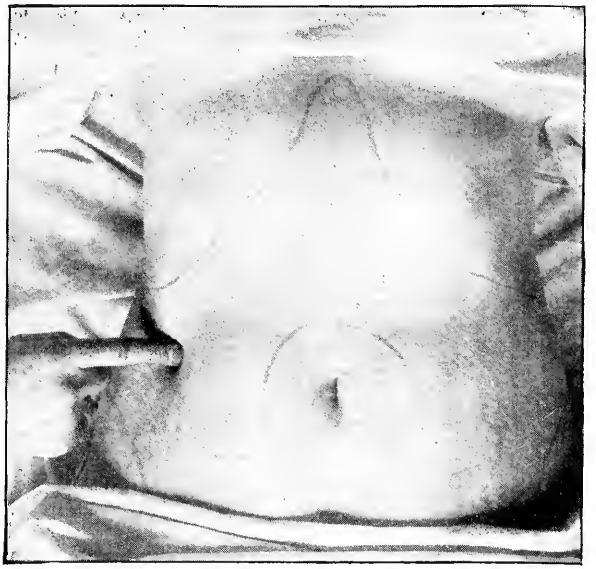


Fig. 232.—Indicating the region for kidney tenderness in front, on the right side. (From Crossen.)

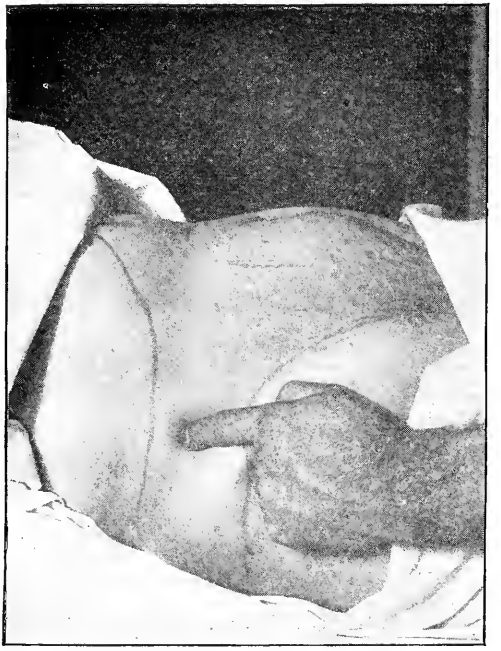


Fig. 233.—The point for kidney tenderness laterally. (From Crossen.)

Fig. 234.—The point for kidney tenderness posteriorly. (From Crossen.)

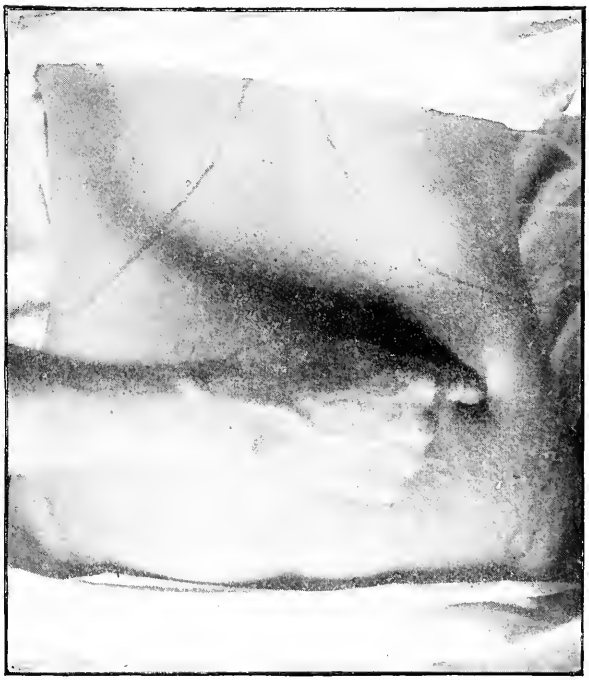


Fig. 235.—The area for left kidney tenderness in front. (From Crossen.)

with the left. If in this manner the kidney be felt at the completion of inspiration, but glides back into place during expiration the condition constitutes *movable kidney.*

Three degrees of movable kidney are recognized. In the first degree, only the lower pole of the kidney is palpable; in the second degree the lower half of the kidney is palpable; while in the third degree the entire organ can be palpated.

If, on the contrary, the kidney fails to glide back into its normal position during expiration, if during this period the entire kidney remain palpable, and can be pushed about in the abdominal cav-

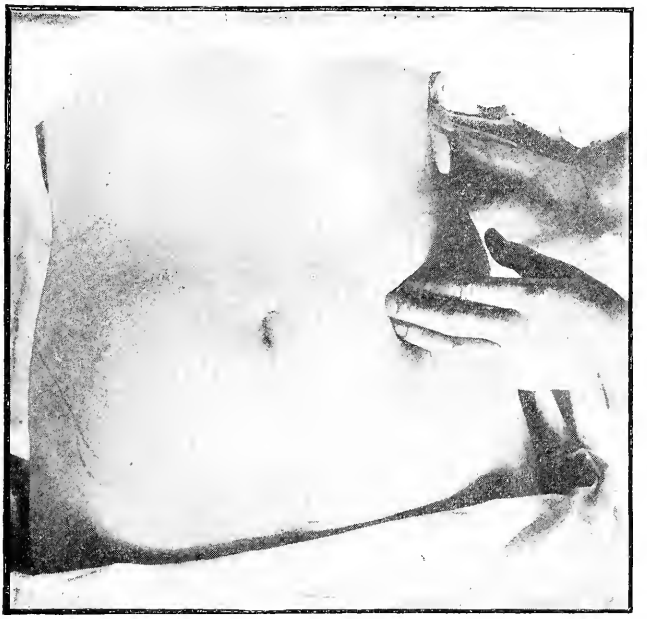


Fig. 236.—Method of palpating for a mass in the kidney region. The structures are caught between the hand behind and the one in front. (From Crossen.)

ity, the condition is termed a *displaced kidney,* or *floating kidney.*

The right kidney is frequently movable in girls and women, particularly in those with enteroptosis; less frequently movable in men.

While the normal kidney is palpated with difficulty, and often is not palpable, a kidney enlarged by malignant disease is readily palpated. A kidney the seat of hydro-nephrosis, pyelo-nephrosis, and surgical kidney, often yields fluctuation on bi-manual palpation.

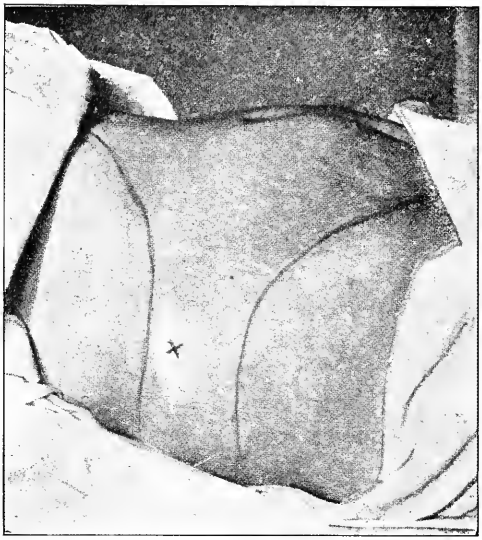


Fig. 237.—Point for kidney tenderness laterally. (From Crossen.)

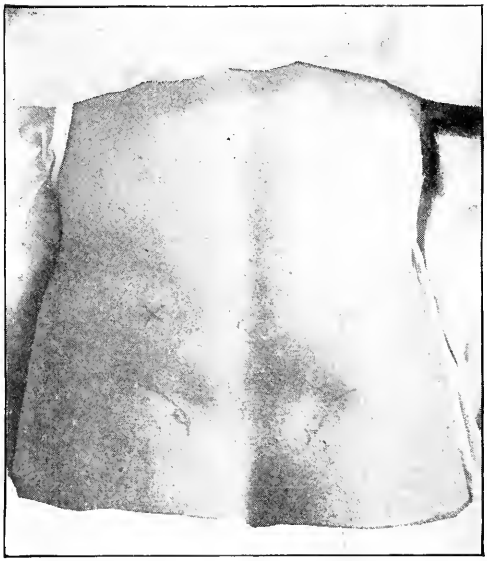


Fig. 238.—Points for kidney tenderness in the back. (From Crossen.)

When a mass is felt in this region doubt may arise as to whether or not it is an enlarged kidney or whether it is a gall-bladder or spleen, or pancreatic in origin, or of the pylorus. A tumor of the kidney is situated more laterally than a distended gall-bladder, and shows its radius of mobility is backward into the loin, while that of the gall-bladder is transversely below the margin of the liver. As stated in a previous section, the hepatic flexure of the colon is attached by areolar tissue to the lower extremity of the right kidney so that a renal enlargement or tumor carries the tympanitic colon before it, while tumors of the gall-bladder, pylorus, or pancreas reach the abdominal wall nearer the median line, without the intervention of the tympanitic colon.

In the case of the left kidney, however, owing to the fact that

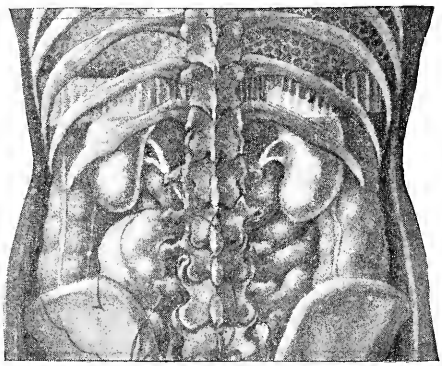


Fig. 239.—Relation of the kidney to the lower margin of the last rib. (Crossen, after Butler.)

there is no attachment to the splenic flexure, the colon is pushed internally, allowing a renal tumor to meet the abdominal wall. However, the displaced colon will yield a tympanitic note along the inner border of the solid mass.

Tumors or enlargements of the *spleen* approach the abdominal wall above the transverse colon, which is displaced downward.

A *poly-cystic kidney* is studded with roundish masses, which may be appreciated by palpation.

Malignant tumors of the kidney develop anteriorly, where they form palpable tumors, whereas a peri-nephric abscess is most plainly palpable posteriorly.

If palpation of the kidney yields no enlargement of the organ, but tenderness over the renal area, renal inflammation or inflam-

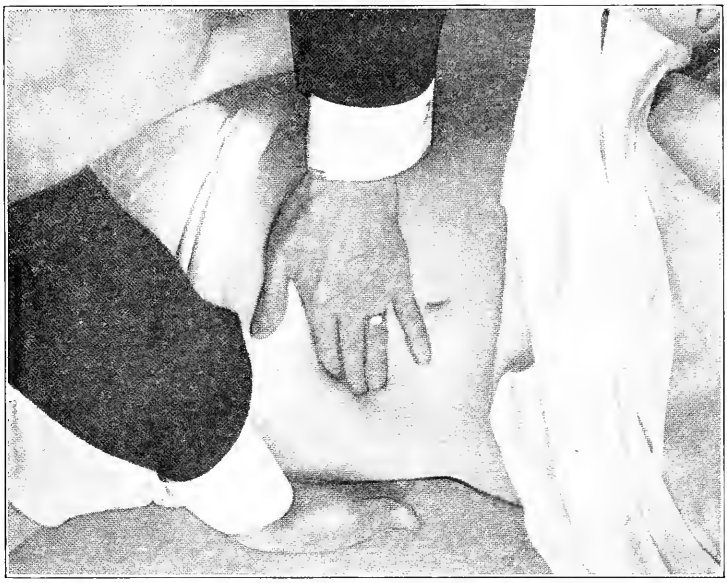


Fig. 240.—Showing technic of physical examination for nephroptosis. First position of examiner's hands in both positions of the patient. (From Longyear.)

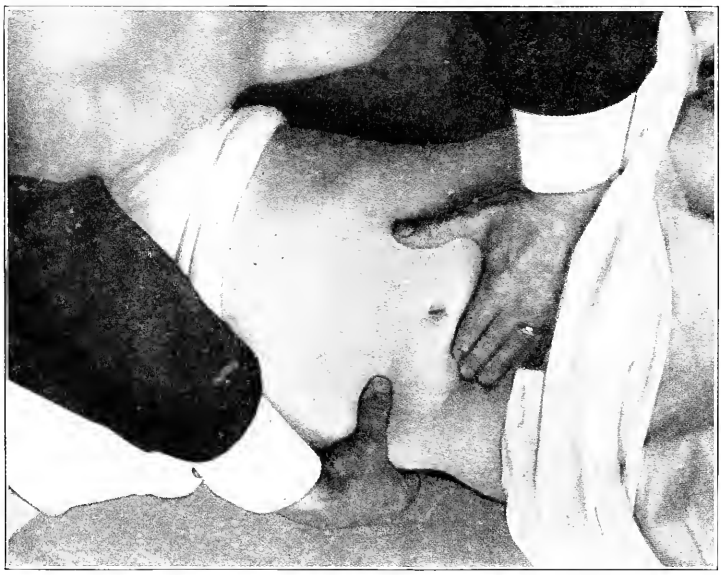


Fig. 241.—Showing technic of physical examination for nephroptosis. Second position of examiner's hands in both positions of the patient. The kidney is held in ptosis by deep pressure of the thumb under the costal margin and palpated by the tips of the fingers of the left hand. (From Longyear.)

mation of the surrounding structures may be assumed to be present.

In certain cases of movable or wandering kidney, the kidney may be more readily palpated with the patient in the sitting posture.

Percussion.—In examination of the kidney percussion is much inferior to palpation. The normal kidney cannot be mapped out anteriorly; but posteriorly the inferior pole and outer convex border can be marked out from the tympanitic colon which surrounds it, the upper limit of dullness blending with that of the liver or spleen.

When, however, the kidney is much enlarged from tumor or other cause, so that the organ approaches the anterior and lateral abdominal walls, percussion is useful in delineating the boundaries of the enlarged organ; but even in this event percussion is inferior to skillful and careful palpation. Floating kidney may be indicated by the presence of tympany in the renal area where the note should be dull.

EXAMINATION OF THE BLADDER

The bladder is a hollow viscus, lying posterior to the symphysis pubis. A pelvic organ in the adult, in the infant the bladder is situated in the abdominal cavity above the pubic symphysis.

Inspection.—The normal bladder is not visible; but when distended it produces bulging in the hypogastric region which, in extreme cases, may extend into the umbilical region. The cause of such distention may be prostatic hypertrophy, a lumbar cord lesion, or the comatose state of an acute infection.

Palpation.—The moderately distended bladder cannot be palpated through the abdominal wall; when, however, distended fully, it may be felt as a tense spherical mass in the hypogastric region.

Percussion.—Percussion is only available in cases of extreme distention, when a flat note in elicited surrounded by intestinal tympany.

Auscultation is not employed in the physical examination of the bladder.

EXAMINATION OF THE URETERS

The normal ureter cannot be palpated, and an enlarged one only in an emaciated subject with very lax abdominal walls. However, palpation over the ureter may elicit tenderness due to inflammation of the tube, which on the right side must not be mistaken for inflammatory disease of the vermiform appendix or gall-bladder. (See Fig. 229, p. 368.)

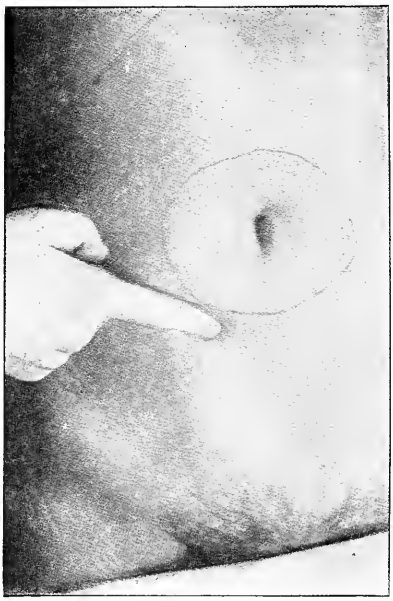


Fig. 242.—Indicating the site to search for tenderness of the right ureter. This may be found anywhere from the point indicated to some distance inside the circle, towards the umbilicus. (From Crossen.)

Fig. 243.—Palpating for tenderness or thickening about the right ureter. (From Crossen.)

The course of the ureter is indicated on the surface of the abdomen by a line drawn almost vertically from a point in the umbilical region about two inches from the median line at the level of the anterior extremity of the twelfth rib to a point a little below the umbilicus, thence converging toward the median line as the symphysis pubis is approached.

PART III. THE HEAD, NECK AND EXTREMITIES

SECTION I

THE HEAD AND NECK

CHAPTER XXVI

EXAMINATION OF THE HEAD

In the examination of the head the following points should be noted by the examiner:

1. Size and shape.
2. Condition of the fontanelles and sutures (in children).
3. Condition of the bones.
4. Condition of the hair.
5. Position of the head.
6. Movements of the head.

Size and Shape.—The head may be abnormally small (microcephalia) with premature closure of the fontanelles and sutures, a condition usually associated with idiocy.

A very large head is encountered in hydrocephalus, while moderate enlargement occurs in connection with rickets and cretinism and hypertrophia cerebri.

In *rickets* the circumference of the head is increased two or three inches, the enlargement being chiefly due to thickening of the cranial bones. The shape of the head is rather square (box head), owing to the presence of osteoid bosses upon the frontal and parietal regions. The rachitic head is flattened at the vertex and over the occiput. Soft, compressible areas, *craneotabes,* are often present; the fontanelles are widely open and the sutures are tardy in closing.

Concomitant signs of rickets are the rosary, the chicken or pigeon breast, spinal curvature, tumid belly, and changes in the extremities of the long bones.

Hydrocephalic Head.—In hydrocephalus the head is enlarged, the circumference sometimes reaching 32 inches at the eighth month of life. The large prominent forehead is in marked contrast with the small face. The fontanelles and sutures are widely open, and the veins of the scalp are prominent and distended. The skull is very thin and may be translucent to candle light. The child has difficulty in holding the head up.

While the head in hydrocephalus somewhat resembles the rachitic head, there are differences. In hydrocephalus the shape is globular rather than square as in rickets; also in hydrocephalus the sutures and fontanelles are wider and the fontanelles bulge, which is not true of rickets.

In *cretinism* the head is large, flattened at the vertex, with open sutures and fontanelles, but without bulging. The facial expression is dull, the nose flat, the face large, with puffy eyelids. The extremities are short and thick; the tongue is large, often protruding from the mouth. There are pads of fat in the supra-clavicular regions.

Fontanelles and Sutures.—The posterior fontanelle normally closes about the end of the second month, while the anterior fontanelle closes between the eighteenth and twentieth months of life.

Tardy closure of the fontanelles occurs most frequently in connection with rickets. Closure is also delayed in hydrocephalus and cretinism. In rickets the fontanelles may remain open beyond the fourth year of life.

Bulging fontanelles indicate increased intra-cranial pressure, and are noted in hydrocephalus, cerebral hemorrhage, meningitis, brain tumor, sinus thrombosis, meningeal hemorrhage, and during acute fevers.

Depressed fontanelles are noted during chronic wasting diseases, in pulmonary diseases attended by dyspnea, after severe diarrhea, during the early stages of meningitis, and in cholera infantum.

Enlargement of the fontanelles, the anterior fontanelle exceeding one inch in diameter, is suggestive of rickets, hydrocephalus, cretinism, and may be a hereditary condition.

Open Sutures.—The sutures of the child's head normally close between the sixth and eighth months. Open sutures after this time are significant of rickets, cretinism, or hydrocephalus.

Condition of the Bones of the Head.—A number of changes in the bones of the head possess diagnostic significance.

Craniotabes, the presence of thin, compressible areas in the cranial bones, is symptomatic of rickets, infantile syphilis, or chondro-dystrophy.

Osteoid bosses on the frontal and parietal bones in infants are symptomatic of rickets.

Soft, nodular swellings on the skull, which become harder with advancing age, are symptomatic of syphilitic periostitis of the cranial bones.

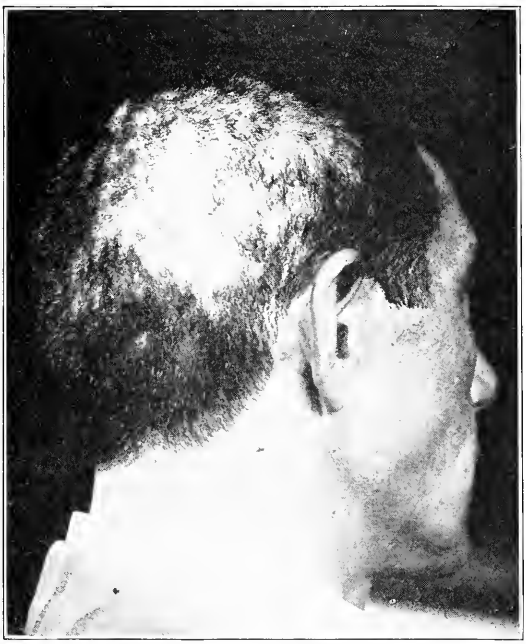


Fig. 244.—Alopecia areata. Numerous small patches have coalesced, forming a rather unusual picture, inasmuch as the baldness is not as complete as usual. (From Hazen.)

Tenderness over the mastoid process, with fever, deep seated pain, and, if pus has formed, fluctuation, is symptomatic of inflammatory disease of the mastoid cells.

The Condition of the Hair.—*General falling* of the hair follows many acute febrile conditions, notably typhoid fever. General loss of hair also occurs in gout and myxedema.

Circumscribed falling of the hair, producing local areas of baldness, results from tinea tonsurans, scarring from local trauma, and neuralgia of the trigeminal nerve, and syphilis.

In a child baldness in the occipital region, with excessive sweating of the head suggests rickets.

Color of the Hair.—The color of the hair may be altered by local application of chemicals, or as a result of metallic poisoning. Thus, hydrogen dioxide bleaches the hair, while the hair assumes a green color in chronic copper poisoning.

Canities, whiteness of the hair, a physiologic change in persons past middle age, is observed in connection with syphilitic endoarteritis involving the scalp and accompanying trophic nervous disturbances.

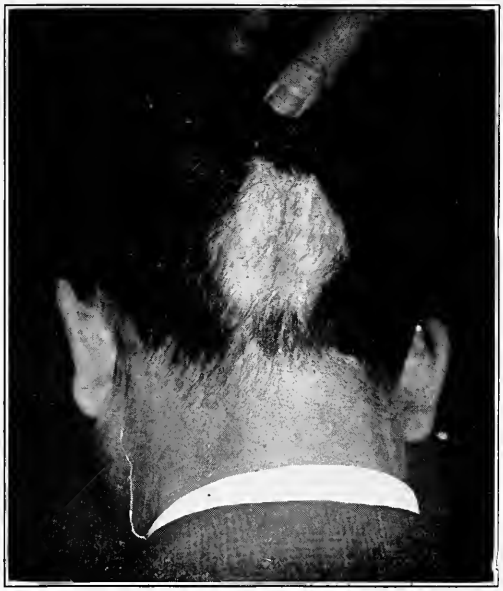


Fig. 245.—Alopecia areata. A patch that is not as yet completely denuded. (From Hazen.)

Position of the Head.—*Retraction* of the head occurs in tetanus, strychnine poisoning, meningitis. In children retraction of the head may occur during attacks of acute indigestion, the significance of which is slight; but it should not be mistaken for retraction in connection with grave affections.

Lateral deviation of the head is observed in connection with *wry-neck.* due to spasm of the sterno-mastoid muscle, in rheumatic torticollis, in which there is painful contraction of the sterno-mastoid and trapezius, in hematoma of the sterno-mastoid, which is attended by an oval tumor in the belly of the muscle. In young

children lateral deviation of the head may be due to injury to the muscles of the neck during parturition.

Abnormal fixation of the head is observed in connection with retro-pharyngeal abscess, cervical adenitis, rheumatism, arthritis deformans, extensive scars from burns, and in Pott's disease.

Movements of the Head.—*Nodding spasm,* a rhythmical up and down movement of the head, is observed in patients suffering with hysteria, and occasionally in connection with rickets. The movement may be continuous; or may be absent during quiescence and only brought out by excitement.

Arrhythmic, purposeless movements of the head occur in Sydenham's Chorea.

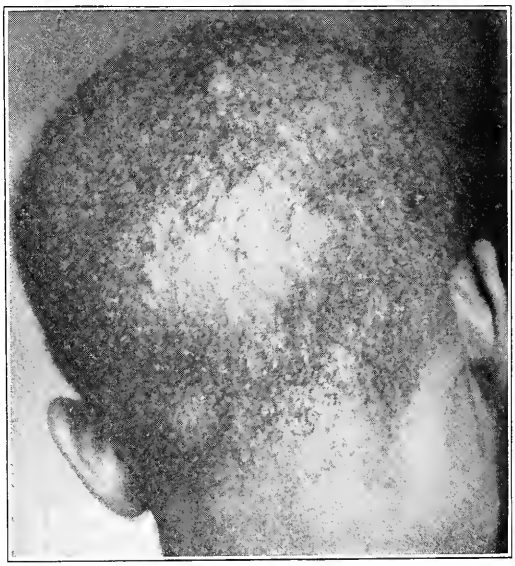


Fig. 246.—Syphilitic alopecia. (From Hazen.)

Spasmodic movements of the head, in which the head deviates laterally, occur in spasmodic torticollis.

Inability to move the head occurs in connection with the flaccid paralysis of acute anterior polio-myelitis, in caries of the cervical vertebræ, and in the late stages of cerebro-spinal meningitis, and during comatose states from any cause.

The Ear.—Congenital Defects.—Among the congenital defects of the auricle may be mentioned entire absence of this portion of the auditory apparatus; excessive development or defective development of the auricle, macrotia and microtia respectively; the

presence of more than one auricle, or polyotia; malformation or absence of the lobule, helix, or antihelix.

Fistula auris congenita, a rare defect, consists of a short blind canal, lined with epithelium, with its orifice either in front of or below the tragus.

Hematoma auris, or *othematoma*, is a bluish-red swelling involving the concha and fossa of the antihelix and helix, the lobule escaping. It is a tropho-neurosis, and the condition is observed most commonly among insane patients, in whom it was formerly attributed to ill-treatment. A similar bluish discoloration, involving the entire auricle may follow trauma, due to effusion of blood beneath the peri-chondrium.

Tophi, small, hard nodules of sodium urate, are frequently found in the helix in gouty patients.

Cysts of the auricle, small, non-inflammatory tumors, containing clear fluid, are sometimes encountered about the auricle. They are differentiated from peri-chondritis by the absence of pain and other inflammatory signs.

Sebaceous cysts, due to blocking of the ducts of the sebaceous glands and accumulation of the secretion, produce roundish tumors situated usually in the skin behind the lobule or in the lobule.

Blueness of the auricle occurs as a sign of cyanosis, also in the early stages of frost-bite, in which it becomes later yellowish-white.

Keloid may be encountered on the lobule, due to piercing the lobule for earrings, most commonly in the negro race.

Otomycosis.—In otomycosis, due to the growth of the Aspergillus Niger in the external auditory canal, the canal is studded with black spots, which under the microscope reveal the presence of the fungus.

Discharge of blood from the external auditory meatus is indicative of fracture of the base of the skull or otitis media. In the case of fracture of the base of the skull the blood is mixed with cerebro-spinal fluid, which prevents coagulation; while in otitis media there is admixture with pus. Discharge of pus unmixed with blood indicates purulent otitis media or abscess.

CHAPTER XXVII

EXAMINATION OF THE FACE

CONTOUR OF THE FACE

The contour of the face is altered by many diseases, chief among which may be mentioned acromegaly, hydrocephalus, osteitis deformans, leontiasis ossium, leprosy, and facial hemiatrophy and hemi-hypertrophy.

Acromegaly.—In acromegaly the face assumes an oval or elliptical shape, due to the enlargement of the frontal and malar bones, and the mandible, which become massive. Owing to this growth the teeth are separated by intervals, the lower teeth projecting beyong those of the upper jaw. The ears are large, the nose thickened, and the superciliary ridges are prominent. The tongue is large, sometimes protruding from the mouth. The eyes are unchanged; and, by contrast with the massive features, appear abnormally small.

Cretinism.—In cretinism the face is broad and flat, presenting a bloated appearance. The eyes are wide apart, the eyelids are thickened, the nose is broad, and negroid. There is pouting of the lips and protruding tongue, the child presenting a picture of imbecility.

Myxedema.—In myxedema the lines of expression in the face are obliterated by swelling in the subcutaneous tissue. The contour of the face has been likened to a "full moon." The nostrils and lips are large and thick, the mouth is enlarged, and there is usually a reddish patch over the cheek. Other signs of myxedema are the dry rough skin, the increase in bulk of the whole body, the inelastic swelling of the subcutaneous tissue, which does not pit upon pressure, and local deposits of subcutaneous tissue in the supra-clavicular fossae.

Hydrocephalus.—The face in this disease is triangular with the base of the triangle above. The features, which are of normal size, present a marked contrast with the enormous forehead.

Osteitis Deformans.—The face in this disease is triangular

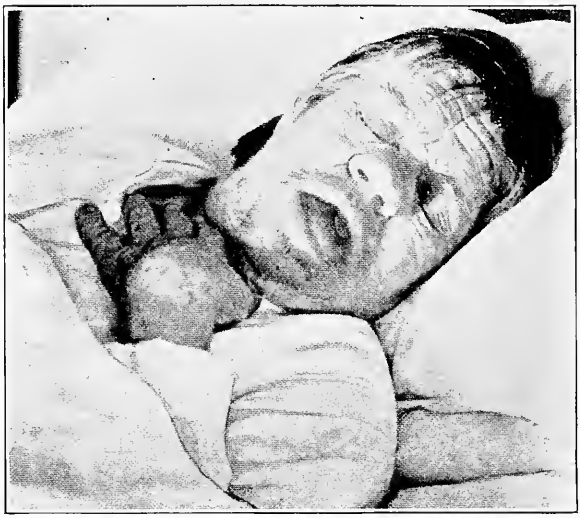


Fig. 247.—Face of acromegaly. (Butler, after Worchester.)

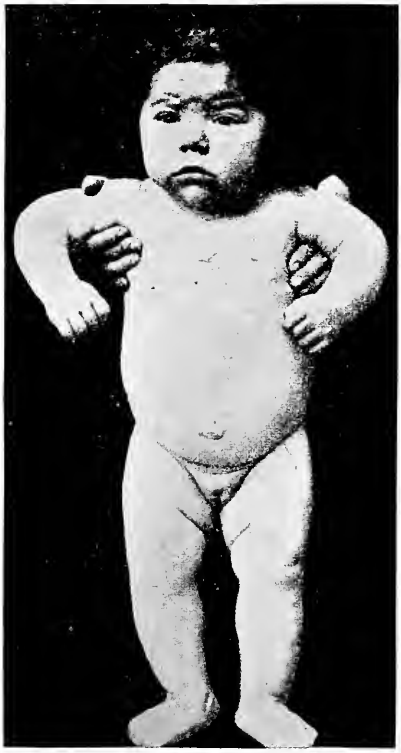


Fig. 248.—A case of congenital myxedema. (Woolley, after Kassowitz.)

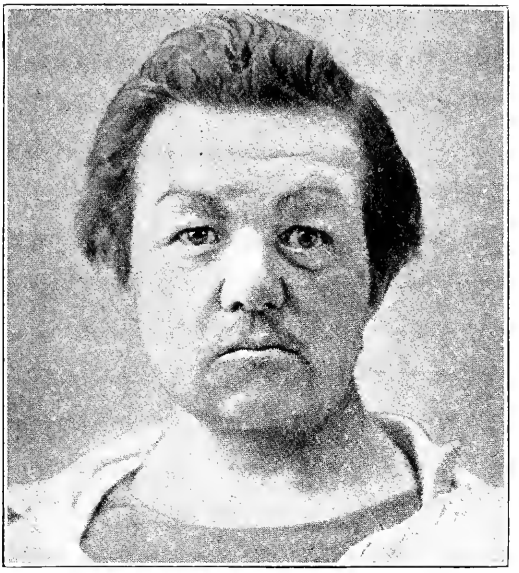


Fig. 249.—Face of myxedema. (Butler, after Gordinier.)

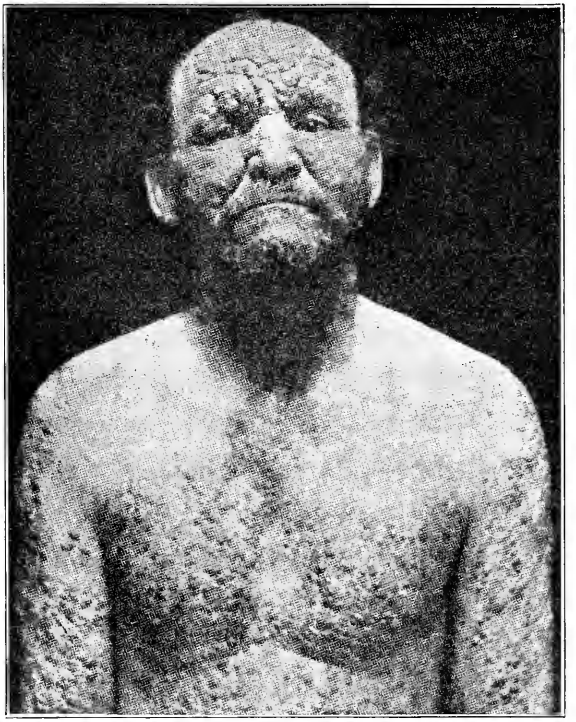


Fig. 250.—Leprosy. (From McFarland.)

with the base directed upward, owing to the thickening of the bones of the cranium. The head is carried in a position of forward inclination. The disease is associated with bowing of the bones of the upper and lower extremities, kyphosis, and not infrequently anchylosis of the spine.

Leontiasis Ossium.—This disease is characterized by progressive enlargement of the bones of the cranium and face, beginning usually in the superior maxillary bones. Blindness occasionally develops from pressure upon the optic nerves.

Fig. 251.—Facial hemi-atrophy. (From Butler.)

Leprosy.—When the nodes of leprosy develop in the face they produce thickening of the skin of the forehead and cheeks. The nose is flat and thick; the lips are thick; the ears are thick and large, while the eyebrows, eyelashes, and beard are shed, constituting the Facies Leontina.

Facial Hemi-atrophy.—In facial hemi-atrophy one-half of the face is smaller than the opposite half, with a sharply defined vertical line of junction. The condition usually begins during childhood in one or two spots on one side of the face. The skin begins to undergo atrophic changes, followed by a similar involvement of the underlying subcutaneous tissue, muscles and bones.

The skin of the affected half of the face becomes wrinkled, the teeth become loose, the eyebrows fall out. The secretion of the sebaceous glands is diminished or abolished. The face is drawn toward the sound side, rendering the contrast between the two sides striking.

A similar facial asymmetry is encountered in children as a developmental defect, often in association with congenital torticollis.

Facial hemi-hypertrophy, the opposite condition, in which one side of the face is enlarged, occurs as an anomaly in the development of the face, sometimes associated with hemi-hypertrophy of the entire half of the body.

THE COLOR OF THE FACE

Pallor of the face occurs in anemia, ischemia, the edema of Bright's Disease, and transiently as the result of sudden fright.

Flushing of the face may be transient and due to vasomotor disturbance, or may be persistent, notably in the early stage of acute fevers, as yellow fever. The flushed cheek of pneumonia and the bi-lateral flushing of tuberculosis have been described. A flushed face accompanies excessive cardiac hypertrophy, and in one form of essential anemia, namely, Chlorosis Rubra, is a marked feature of the disease. In apoplectic attacks and in the early stages of alcoholic intoxication the face is flushed.

Cyanosis, or bluish discoloration, particularly noticeable in the lips and ears, occurs in uncompensated heart disease. A similar bluish discoloration of the face is symptomatic of poisoning with coal tar products.

Yellowish discoloration of the entire face is suggestive of the cachexia of malignant disease, syphilis, or chronic malaria. A similar hue accompanies chronic constipation with inactive liver, certain cases of exophthalmic goiter and Addison's disease. A lemon yellow color of the face and body, with maintenance of the subcutaneous fat of the body, occurs with pernicious anemia.

Bluish discoloration, or argyria, occurs in cases of chronic silver poisoning.

Brownish, muddy patches upon the face, termed *chloasma,* frequently develops in pregnant women and in women with uterine or ovarian disease.

SPASM OF THE FACE

Spasm of the facial muscles occurs as a result of functional or organic disorders. It may be tonic or clonic, uni-lateral, or bi-lateral. It is more frequently encountered in women than in men. Among the conditions in which facial spasm possesses diagnostic significance may be mentioned:

Habit Spasm.—This spasm occurs in neurotic children, particularly in young girls. It is intensified by excitement or examination. It may consist in the rapid winking of an eye or the drawing up of one corner of the mouth. The neck muscles are frequently involved, the head being given a quick shake at the time of the winking.

Convulsive Tic.—This is a very sudden spasm of the facial muscles, frequently involving the brachial muscles as well. The spasmodic movements may be almost constant or may occur in paroxysms. In extreme cases the spasm may involve all the muscles of the body, the movements being very irregular and violent. The spasm is often accompanied by explosive utterances, echolalia and coprolalia.

Blepharospasm.—This is a sudden tonic contraction of the orbicularis palpebrarum muscle, causing partial or complete closure of the eye. More commonly the spasm affects the lateral facial muscles also, producing constant twitching of the side of the face. Usually uni-lateral, blepharospasm may be bi-lateral. The spasm is increased by emotional excitement and voluntary movement of the muscles of the face. If not reflex from irritation of the conjunctiva or cornea by a foreign body, it indicates involvement of the facial nerve.

Chorea.—Chorea produces arrhythmical jerking contractions of the facial muscles. It is accompanied by the other symptoms of the disease, as purposeless movements of the hands and feet.

Exophthalmic Goiter.—Spasm of the levator palpebræ superioris muscle, causing rapid movements of the upper lids occurs occasionally in exophthalmic goiter, in which it constitutes Abadie's sign of this disease.

Tetanus.—Tetanus or lockjaw produces tonic spasm of the facial muscles, with the risus sardonicus, or sardonic smile which is characteristic of the disease.

Uni-lateral clonic spasm of one or more facial muscles points to irritation of the facial region of the cortex of the brain or to irritation of the facial nerve trunk in its course or at its exit by tumor or aneurism of the vertebral artery.

THE FOREHEAD

The forehead should be examined for scars, skin eruptions, and nodular swellings.

Scars upon the forehead may be indicative of former traumatism or of the eruption of syphilis.

Eruptions.—The forehead is subject to many cutaneous eruptions, notably those of measles, smallpox, and syphilis, in which last named disease it constitutes the so-called corona veneris.

Nodular swellings of the forehead may be indicative of glanders, trichinosis, syphilitic periostitis, or tumor of the cranial bones.

THE EYES

The Eyelid

Edema.—Edema of the lids with puffiness, occurs in connection with the edema of nephritis and in anasarca due to cardiac disease or hepatic cirrhosis. Edema of the lids is also noticed during the active stage of pertussis, severe coryza, erysipelas, cerebral thrombosis, and in arsenic and iodine poisoning. Slight puffiness and edema of the lids upon arising in the morning is noted in certain persons as a normal phenomenon.

Duskiness of the lids and the infra-orbital region is symptomatic of uterine and ovarian disease, pregnancy, anemia and exhausting disease, the molimina of menstruation, and it is said masturbation.

Xanthoma is a small, slightly elevated, flattened lipomatous new-growth which is occasionally encountered on the eyelids of diabetic patients.

Ptosis.—Ptosis of the upper lid is usually due to syphilitic paralysis of the oculo-motor nerve. Usually uni-lateral, ptosis may be bi-lateral. A bi-lateral ptosis of brief duration sometimes is seen in anemic and overworked women. Ptosis of the lid occurs with acute encephalitis and as a congenital condition.

Hordeolum.—A hordeolum or stye is a small abscess in the lid margin, situated at the root of an eyelash upon the anterior margin of the lid. Styes are acute, run a short course, but are prone to recur repeatedly.

Blepharitis Marginalis.—Blepharitis marginalis, inflammation of the lid margin is characterized by the formation of a series of scales or crusts along the lid margin, which upon removal expose a red, glazed surface. As the scales adhere to the lashes, they are sometimes mistaken for the eggs of pediculi.

Chalazion.—A chalazion or meibomian cyst is a small hard tumor of the upper eyelid, imbedded in the tarsal plate. It results from obstruction of a meibomian gland, and is prone to become inflamed and suppurate.

Epithelioma.—Epithelioma usually is seen upon the lower lid in persons past middle life. A history of long duration is usually obtainable.

Chancre.—The initial lesion of syphilis rarely occurs upon the lids.

Lagophthalmos.—Lagophthalmos, imperfect closure of the eyelids occurs with the exophthalmos of Graves' disease, and as a result of partial facial nerve paralysis.

The Conjunctiva

Pallor.—Pallor of the conjunctiva is a sign of anemia and calls for rather than replaces a blood examination.

Yellowness of the conjunctiva accompanies jaundice, and points to hepatic disorder.

Sub-conjunctival hemorrhage may occur during paroxysms of cough in pertussis or asthma, or as the result of local trauma.

Conjunctivitis.—In inflammation of the conjunctiva the membrane is red and bathed with muco-purulent or purulent discharge. Conjunctivitis may result from local infection, or may accompany the acute infectious diseases.

The Globe

Exophthalmos.—Exophthalmos, or protrusion of the globe of the eye, may be indicative of hemorrhage into the orbit, paralysis of the ocular muscles, thrombosis of the superior longitudinal sinus, tumor of the orbit or superior maxillary bone pushing the globe forward, or of exophthalmic goiter. Exophthalmos may be

uni-lateral or bi-lateral, the latter constituting one of the cardinal symptoms of exopthalmic goiter.

Von Graeffe's sign of exophthalmic goiter consists in the inability of the upper lid margin to accurately follow the sclero-corneal junction downward during downward rotation of the globe of the eye.

Enophthalmos.—Recession of the globe of the eye into the orbit, enophthalmos, occurs in exhausting diseases, particularly those which are associated with the loss of tissue fluids, as cholera. Enophthalmos is also caused by absorption of the orbital adipose tissue during chronic wasting disease, notably in tuberculosis, diabetes, marasmus, and the cachexia of malignant disease.

Position of the Globe.—During epileptic seizures and hysterical coma the globes of the eyes rotate and turn upward. In hydrocephalus the globe looks downward, while following cranial injuries both globes look toward the side of the injury (conjugate deviation).

Oculocardiac Reflex.—Gentle pressure upon the eyeball of a normal subject produces a perceptible slowing of the pulse through vagus inhibition. This is a true reflex, the afferent impulse incited by pressure upon the globe of the eye being transmitted through the ophthalmic division of the trigeminal nerve to the Gasserian ganglion and thence through the larger root of the fifth cranial nerve to its root of origin. Thence the impulse is transmitted downward to the nucleus of origin of the vagus nerve, resulting in tonic efferent impulses from this center causing inhibition of the cardiac rate.

Abolition of this reflex indicates a break in the reflex arc at some point. Such abolition is noted in cerebro-spinal syphilis and paresis. Abolition of this reflex is one of the earliest signs of syphilitic involvement of the central nervous system, and it is a sign which is readily elicited by the general practitioner. (E. Murray Auer: Jour. Am. Med. Assn., Mar. 24, 1917.)

Cornea and Sclera

Arcus Senilis.—The arcus senilis is a grayish line at the sclero-corneal junction which partially encircles the cornea. Present in many elderly persons the arcus senilis is particularly frequent in arterio-sclerosis and chronic nephritis.

Interstitial Keratitis.—Inflammation of the interstitial tissue of the cornea, leading to partial opacity of this structure or to

small pinkish "Salmon Patches" is nearly always a sign of hereditary syphilis. The condition is usually bi-lateral, affecting children between 5 and 15 years of age, girls being more frequently attacked than boys.

Ulceration.—Comparatively large ulcers of the cornea are apparent as losses of the surface epithelium; while minute ulcers may require the instillation into the eye of a few drops of Fluorescin, which stains the ulcer a bright yellow-green. Corneal ulceration frequently develops during the exposure of the cornea as a result of the exophthalmos of Graves' disease; and in cases in which the cornea is insensitive owing to disease of the ophthalmic division of the fifth cranial nerve. Corneal ulcer is prone to develop during prolonged fevers when the patient lies long with the eyes only partially closed.

Opacity.—Corneal opacity may result from the repair of a corneal ulcer, from interstitial keratitis, or as a result of Pannus. Corneal opacity sometimes develops during the course of scrofula or chronic malaria.

Staphyloma.—Staphyloma, a bulging of the cornea, usually is a sequence of weakening of the cornea by deep ulceration, particularly in connection with gonorrheal ophthalmia.

Yellow Sclerotics.—Yellow discoloration of the sclerotics occur in jaundice from hepatic disorder. Small, circumscribed, yellowish patches, pinguecula, are innocent growths springing from the ocular conjunctiva.

Bluish Sclerotics occur in connection with chlorosis, in which they contrast markedly with the greenish discoloration of the skin; also in nephritis, and Addison's disease.

Scleritis.—In inflammation of the sclerotic coat of the eye small bluish or purplish elevations are left upon the sclerotics.

THE NOSE

Shape.—The shape of the nose is altered by a growing tumor within the nasal cavities, or from the adjacent bones of the face. In cretinism and myxedema the nose is flattened and negroid. In syphilis in certain instances the nasal bridge is destroyed with the production of a characteristic deformity, the saddle nose.

Redness.—Redness of the nose, aside from being commonly associated with a history of chronic alcoholism, is observed in

lupus erythematosus, in circulatory disturbances, chronic digestive disorders, and in amenorrhea.

Epistaxis.—Discharge of blood from the nose is frequently a sign of incipient typhoid fever. A discharge of blood mixed with cerebro-spinal fluid occurs with fracture of the base of the skull. Discharge of blood from the nose may signify foreign bodies in the nose, acute catarrh, local hyperemia from cardiac disease, local ulceration which may be simple, carcinomatous, or syphilitic, or hemorrhagic diseases as hemophilia, scurvy, or purpura hemorrhagica.

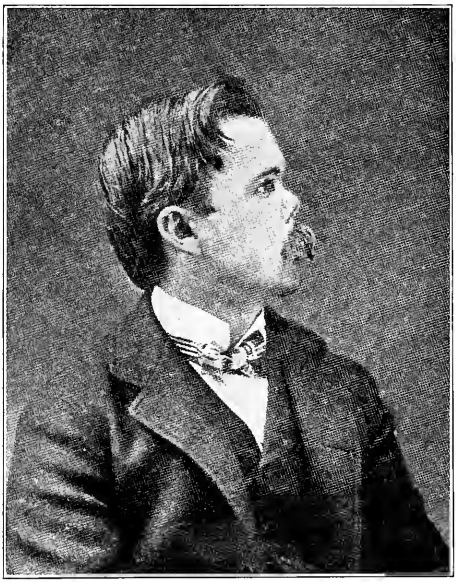


Fig. 252.—Saddle-nose. (From Eisendrath.)

Pseudo-Membrane.—A pseudo-membrane develops in the nose in nasal diphtheria, a condition which is associated with considerable swelling of the associated lymph glands. The pseudo-membrane may spread to the skin of the face, the conjunctiva or the antrum of Highmore.

Adenoid Vegetations.—In the presence of adenoid vegetations in the naso-pharynx the nose is but poorly developed, the nostrils appearing small and pinched.

Ulceration.—A chronic ulcer on the ala of the nose may be tuberculous, carcinomatous or syphilitic.

THE LIPS

Pallor of the lips suggests but does not prove the presence of anemia.

Cyanosis, or blueness of the lips, if not due to the ingestion of

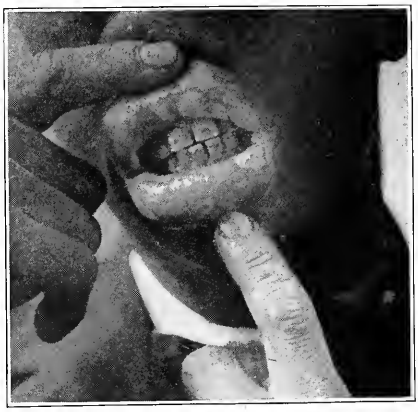


Fig. 253.—Mucous patches. (From Hazen.)

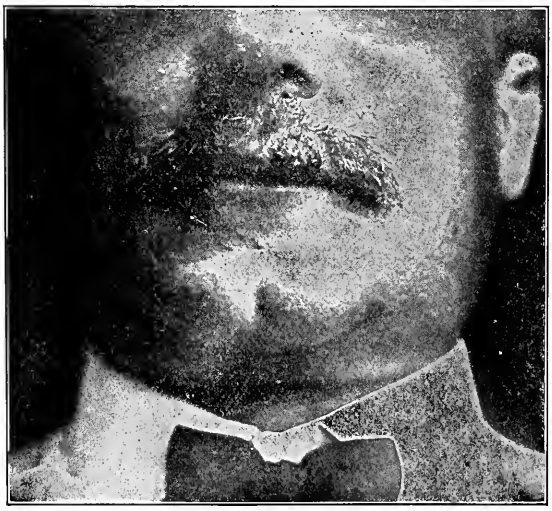


Fig. 254.—Chancre of the lip of one month's duration. (From Hazen.)

large doses of coal-tar products, is indicative of regurgitant heart disease or pulmonary disease of an obstructive nature as emphysema and pneumonia.

Parted lips, when dry and cyanotic indicate the dyspnea of cardiac or pulmonary disease. Parted lips in a child with a small, pinched nose is suggestive of adenoid vegetations in the naso-pharynx.

Loose, pendulous lower lip accompanies chronic bulbar palsy, and less frequently is seen with diphtheritic palsy.

Herpes of the lips, herpes labialis, occurs with pneumonia most frequently, less frequently with malaria, and typhoid fever, and other febrile affections.

Enlargement of the lips accompanies angio-neurotic edema,

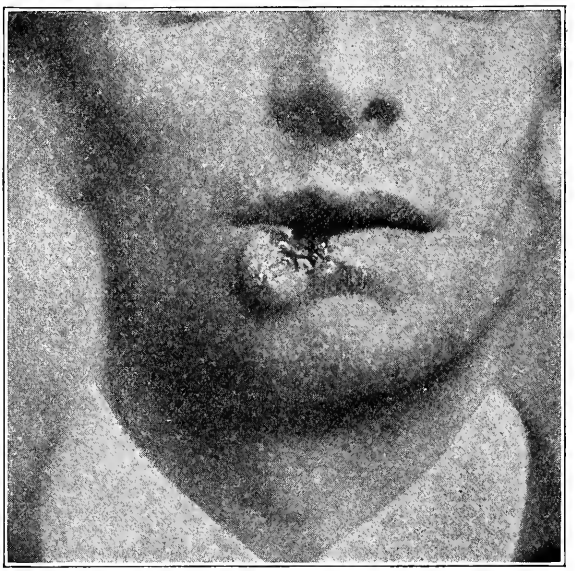


Fig. 255.—Prickle-celled carcinoma of the lower lip in a young man, which arose after treating a clinically benign lesion with caustic pastes. (Gilchrist's collection.) (From Hazen.)

local abscess formation, and phlegmonous inflammation, and obstruction of the lymphatics draining the lips, *macroheilia.*

Rhagades, or fissures of the lips, usually affect the lower lip near its center, in cold dry weather. Similar fissures developing upon the lips near the angle of the mouth in a child are good signs of hereditary syphilis.

Mucous Patch.—Flat, whitish sores near the angles of the lips with sharply defined borders, are mucous patches of syphilis.

Chancre.—An indurated sore on the lip, particularly when de-

veloping in a young person, and associated with enlargement of the associated lymph glands suggests the initial lesion of syphilis.

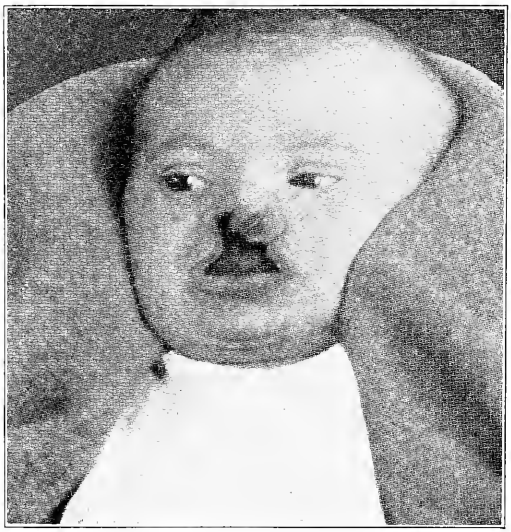


Fig. 256.—Double harelip and cleft palate. (From Eisendrath.)

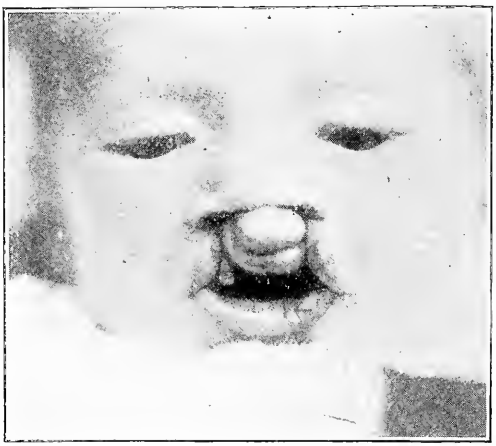


Fig. 257.—Case of complete double cleft in which at birth a tooth hung from the lateral margin of the alveolar cleft by a thin pedicle of soft tissue. (From Blair.)

Epithelioma.—A chronic irregular ulcer at the muco-cutaneous junction of the lower lip in a person past middle life with enlargement of the lymph glands at the angle of the jaw, is suggestive of epithelioma.

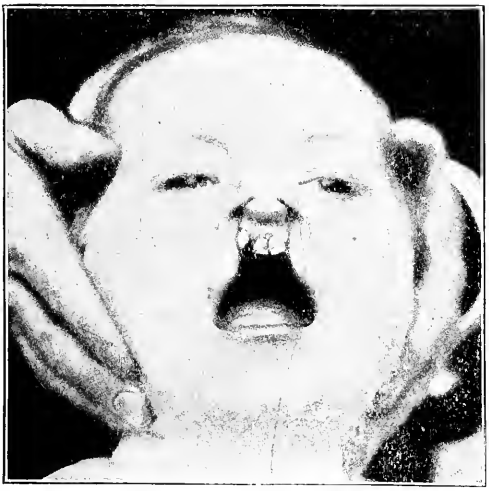


Fig. 258.—Complete double cleft of the lip. This is here accompanied by a double cleft of the palate. The intermaxillary bone carries three incisors. (From Blair.)

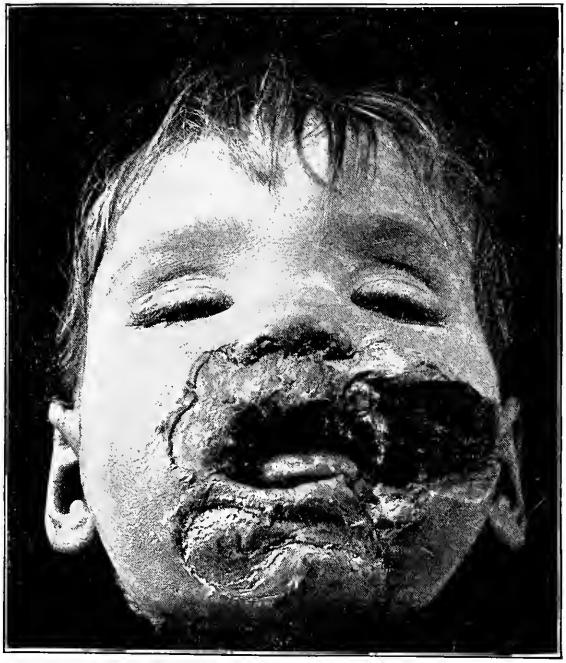


Fig. 259.—Noma. A piece has been removed from the left cheek for examination. (From the Hunterian Museum, London.) (From Blair.)

Hare-lip is recognized as a vertical slit or cleft in the upper lip on one or both sides of the median line. The cleft may be small and confined to the lip, or may be associated with cleft-palate, club-foot or other deformity.

Noma, or cancrum oris, is recognized as a gangrenous mass of tissue involving the lip and adjacent surface of the cheek accompanied by a very foul odor. Occurring after measles and diphtheria, it is frequently a sequence of ulcerative stomatitis.

THE BREATH

Foul Breath.—A foul breath may be caused by carious teeth, diseased gums in pyorrhea alveolaris or mercurial poisoning, follicular tonsillitis, ulcerative or gangrenous stomatitis, or gangrene of the lung.

Uremic Breath.—In uremia the breath has a urinous or ammoniacal odor.

Diabetes.—Diabetes mellitus imparts a sweetish, fruity odor to the breath, the acetone breath.

THE TEETH

Premature and **delayed** dentition possesses diagnostic significance. The former suggests hereditary syphilis, while the latter accompanies rickets, cretinism, and disorders of nutrition.

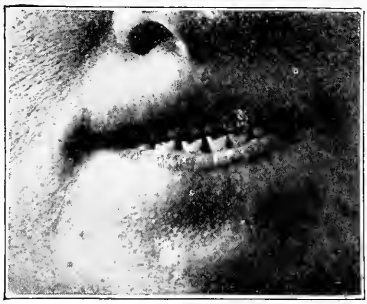


Fig. 260.—Hutchinson's teeth. (Courtesy of Drs. Fordyce and MacKee.) (From Sutton.)

Early Decay.—In children early decay of the teeth occurs in association with rickets and gastro-intestinal digestive disorders. In adults carious teeth occur in pregnancy and diabetes mellitus as well as in chronic phosphorus poisoning.

Loosening of the teeth, with spongy bleeding gums, occurs in

scurvy and mercurial poisoning. In pyorrhea alveolaris the teeth are loosened.

Hutchinson Teeth.—In hereditary syphilis the upper central incisors may present each a notch in its free border. These teeth are small and separated by distinct intervals. It affects the permanent and not the deciduous teeth.

Grinding of the teeth during sleep in children is observed in connection with rickets and derangements of digestion.

THE GUMS

Blue Line.—A blue line on the margin of the gum is indicative of chronic lead poisoning. In the early stages of the intoxication the line is not continuous, but occurs as a series of blue dots at the base of the teeth. The line may extend along the entire length of the gum, or may be limited to the bases of a few of the front teeth in either jaw.

In chronic copper poisoning a blue or greenish line develops along the roots of the teeth.

Red Line.—A red line along the gingivo-dental margin occurs with pyorrhea alveolaris, gingivitis, frequently in diabetes, and it is said in tuberculosis.

Spongy Gums.—In ulcerative stomatitis the gums are swollen, spongy, of deep red or purple color, with a line of ulceration adjacent to the incisors, sometimes extending to all the teeth.

In scorbutus the gums are spongy, bleed easily, and the teeth are loosened. In mercurial poisoning the gums are spongy, there is excessive salivation, and fetid breath.

In pellagra the gums are spongy, and assume a cerise color.

Epulis.—An epulis is a small, soft tumor springing from the gums or alveolar process of the superior maxillary bone. It is usually a giant-cell sarcoma.

THE TONGUE

Size of the Tongue.—*Hypertrophy* of the tongue occurs in infants as a congenital condition, the tongue reaching such enormous size that it cannot be contained in the mouth. *Acquired hypertrophy* of the tongue is seen in acromegaly, myxedema, and cretinism, in acute glossitis, and as a result of lymphatic obstruction, macroglossia.

Atrophy of the tongue occurs as a part of glosso-labio-laryngeal

palsy. Uni-lateral atrophy of the tongue may accompany facial hemi-atrophy and as a result of hemorrhage or tumor developing in close proximity to the hypo-glossal nucleus.

Movements of the Tongue.—The manner in which the tongue is protruded upon request as well as the integrity of its movements during mastication and speech should be carefully noted. Thus in nervous and neurasthenic subjects the tongue is protruded quickly upon request, whereas in typhoid states the protrusion is very slow and tardy.

In uni-lateral paralysis of the tongue, accompanying hemiplegia or uni-lateral hypo-glossal palsy the tongue deviates from the median line when protruded. In bi-lateral paralysis of the tongue, as a result of bulbar paralysis, or symmetrical lesions of the cortex or supra-nuclear tracts, the tongue lies upon the floor of the mouth and cannot be protruded.

Inability to perform the finer movements of the tongue concerned in mastication and speech is an early sign of glosso-labio-laryngeal paralysis, or true bulbar palsy. A similar impairment of the movements of the tongue accompanies pseudo-bulbar paralysis, a state in which the central lesion is not situated in the medulla, but in the lingual fibers from the cortex above the medulla. This condition of pseudo-bulbar palsy is not accompanied by atrophy of the tongue, which only occurs in true bulbar palsy, or glosso-labio-laryngeal paralysis. The absence of lingual atrophy in the former indicates that the causative lesion is in the upper neurone, above the nucleus.

Tremor.—A coarse or fine tremor of the tongue upon protrusion accompanies many organic nervous diseases and typhoid states, and exhausting fevers. In organic nervous involvement the tremor is constant, whereas in typhoid states it only develops upon protrusion of the tongue.

Spasm.—Tonic spasm of the tongue accompanies Thompson's disease, or myotonia congenita. A similar tonic spasm of the tongue occurs from reflex irritation of the trigeminal nerve. Clonic spasm of the tongue is noted in connection with chorea, epilepsy, puerperal melancholia, multiple sclerosis and paresis.

Ulceration.—Ulceration of the dorsum of the tongue may be indicative of simple ulceration, tuberculosis, syphilis, or carcinoma.

Simple ulceration results from local trauma or irritation. Not infrequently in young children an ulcer of the frenum is noted, resulting from the irritation of the sharp edges of the lower central incisor teeth.

A *tuberculous ulcer* of the tongue may be oval, linear, or stellate. The surface of the ulcer is pale and uneven, covered with grayish exudate, presenting no evidences of acute inflammation. It is usually accompanied by tuberculosis of the cervical lymphatic glands.

A

B

C

Fig. 261.—Illustrating tuberculous lesions of the tongue, *A* and *B* in the same individual, healing after two and one-half years' treatment; *C*, on the dorsum of the tongue of an individual suffering from active advanced tuberculosis; no signs of healing shown, patient dying of the disease a few months later. (From Pottenger.)

A *syphilitic ulcer* of the tongue produces a chronic dissecting glossitis characterized by multiple fissures situated principally upon the lingual edges, but crossing the dorsum of the tongue in various directions. The cervical lymph glands are commonly enlarged.

A *carcinomatous ulcer* of the tongue is solitary, with induration of the surrounding tissues, somewhat simulating a chancre; but the ulcer does not disappear under anti-syphilitic therapy. The carcinomatous ulcer is apt to develop in elderly persons, and the accompanying glandular enlargement appears more tardily than in the other lingual ulcers.

Geographical Tongue.—In the geographical tongue there are one or more patches upon the dorsum in which the surface epithelium has desquamated, the patches extending at the periphery while healing at the center, pursuing a circinate course

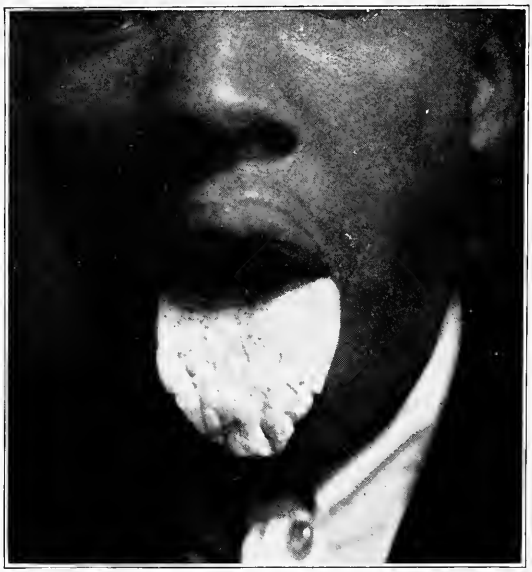


Fig. 262.—"Cobblestone tongue" due to gummous deposits two years after infection. (From Hazen.)

over the dorsum, two or more patches frequently coalescing. Occurring in under-nourished children and adults, the geographical tongue possesses little significance.

Leukoplakia.—In leukoplakia irregularly shaped plaques of thickened epithelium appear upon the dorsum of the tongue. The plaques are smooth, pale, slightly elevated above the surrounding surface of the tongue, one to two centimeters in diameter, and non-ulcerative. Their recognition is important in that they have become the starting point of carcinoma of the tongue.

Smoker's Patch.—In persons who use tobacco to excess a round or oval patch is sometimes encountered upon the dorsum of the

tongue near the tip, slightly elevated, red or pearly in color, smooth, and with no tendency to ulceration.

Cysts.—Mucous and blood cysts occasionally develop in the tongue. Rarely the cysticercus cellulosæ, the larva of the taenia solium may produce a cyst upon the under surface of the tongue, or an echinococcus cyst may be encountered in this region. Also a Ranula, due to obstruction and dilatation of the ducts of Blandin-Nuhn's glands may be found on the under surface of the tongue near the tip.

Thrush.—In thrush the dorsum of the tongue is covered or studded with small, white flakes, resembling closely deposited flakes of coagulated milk, but differing from them in that they cannot be wiped off; and, if removed, leaving bleeding points.

Indentation of the edges of the tongue by the teeth, is noted during prolonged fevers when the hygiene of the oral cavity is not properly practiced. In pellagra there are similar indentations in the deep red border of the tongue occurring with this disease.

Pellagra.—In pellagra the tongue presents a fiery red border and tip, showing indentations corresponding to the teeth with which it is in contact. Frequently small circumscribed sloughs are encountered upon the borders of the tongue, corresponding to areas of epithelial denudation. The gums are spongy and of a cerise color, while the mouth is the site of a stomatitis of variable intensity. The condition of the tongue and buccal mucosa is intensely painful.

Dryness of the Tongue.—The tongue in health is kept moistened with the salivary secretions and the buccal secretions. When this secretion is inhibited during acute fevers, the administration of atropine, or from excessive loss of body fluids incident to prolonged diarrhea and profuse hemorrhage the tongue is abnormally dry, and not infrequently covered with a thick brown coat.

Color.—The ingestion of various chemicals and drugs and certain diseases alter the color of the tongue. The tongue is white following the ingestion of mercuric chloride, ammonia, sulphuric acid and phenol. The ingestion of caustic potash or soda causes reddening of the tongue with evidences of destructive action of these substances. Hydrochloric and nitric acids color the tongue yellow.

In Addison's disease and purpura hemorrhagica the tongue not infrequently exhibits small non-elevated purple spots; while xan-

thelasma produces yellowish, slightly elevated dots along the margins of the tongue.

In scarlatina the tongue is bright red, the filiform papillae contrasting sharply with the slight white furring of the tongue, the *strawberry tongue.*

THE BUCCAL CAVITY

Color.—The buccal mucosa is pallid in anemic states, is excessively red during local inflammation as in catarrhal stomatitis, and is bluish in cyanosis and argyria.

Moisture.—Excessive moisture of the buccal cavity, incident to over-activity of the salivary and buccal glands accompanies local inflammation, following the ingestion of massive doses of the iodides and mercurial salts, during the early stages of smallpox and typhus fever, occasionally during pregnancy, and during convalescence from typhoid fever.

Dryness of the buccal cavity, or xerostomia, owing to temporary arrest of the salivary and buccal secretions occurs during acute febrile diseases, diabetes mellitus, in mouth breathers and lesions of the pons and medulla affecting the integrity of the nervous mechanism of the salivary glands.

Eruptions.—In variola and varicella vesicles may appear upon the buccal mucosa, similar vesicular eruption accompanying herpes buccalis and aphthous stomatitis. Measles is accompanied by pathognomonic lesions upon the mucosa, *Koplik's spots.* These are minute red spots with a bluish-white center, occurring upon the inner surface of the cheek opposite the molar teeth. The number of the spots varies; there may be only one or two or the mucosa may be fairly studded with them. The spots occur early, disappearing with the inception of the exanthem.

Mucous Patch.—The mucous patch of syphilis is frequently encountered upon the buccal mucosa. In all suspicious cases a careful search of the mucous lining of the cheek should be made for these lesions.

Noma, cancrum oris, or gangrenous stomatitis, develops as an indurated spot upon the mucous lining of the cheek near the angle of the mouth, later involving the entire thickness of the buccal wall, the gangrenous tissue emitting an especially foul odor.

THE PHARYNX

In examining the pharynx the tongue should be gently depressed with a wooden spatula or other type of tongue depressor, while the patient is instructed to utter the word "AH," which lowers the base of the tongue, permitting a good view of the posterior pharyngeal wall.

Redness.—Abnormal redness of the pharyngeal wall accompanies acute inflammation of the pharynx, which may be primary, or occur with the acute exanthematous fevers, or acute infectious disease, as influenza and erysipelas.

Eruptions.—The eruptions of variola, varicella, and of herpes buccalis are often distributed generally over the pharyngeal wall.

Ulceration of the pharyngeal wall is indicative of tuberculosis, syphilis, or typhoid fever.

Bulging of the posterior pharyngeal wall, either in the median line or laterally, occurs in post-pharyngeal abscess, which is often due to tuberculous disease of the cervical vertebræ.

Elongated Uvula.—Elongation of the uvula may occur from inflammation of the adjacent pharyngeal mucous membrane, but it may also be a part of the general edema incident to cardiac or renal disease.

Perforation of the soft palate is usually of syphilitic origin.

Paralysis of the soft palate may be uni-lateral or bi-lateral. Paralysis of the palate is detected by observation of its movements while the patient speaks, at which time the normal palate moves upward. If this normal mobility of the palate is lost on one side, the paralysis is uni-lateral; if both sides remain immobile, the paralysis is bi-lateral. Bi-lateral palatal paralysis is not infrequently attended by regurgitation of fluids through the nose upon the attempt to swallow. Paralysis of the palate may be part and parcel of glosso-labio-laryngeal paralysis, may depend upon cervical caries, or may be due to diphtheritic paralysis.

THE TONSILS

Inflammation.—A moderate grade of tonsillar inflammation resulting in painful deglutition, accompanies most of the acute exanthematous fevers. In acute follicular tonsillitis the tonsils are moderately enlarged, red, and studded with minute yellowing dots, corresponding to plugs of mucus, epithelium and bacteria which can be squeezed from the tonsillar crypts.

Chronic simple enlargement of the tonsils, in which the two bodies may almost meet in the median line, occurs occasionally in childhood.

Pseudo-membrane.—A pseudo-membrane upon the tonsil, perhaps involving the pharyngeal wall as well, if not diphtheria, is apt to be due to streptococcic inflammation or scarlatina.

Ulceration.—Ulceration of the tonsil is due to tuberculosis, syphilis, or, if in an elderly person, to carcinoma, or in a younger subject to sarcoma.

Peri-tonsillar Ulceration (Vincent's Angina).—In this disease which is a uni-lateral affection, there is ulceration of the peritonsillar tissues, with a variable amount of yellowish exudate covering the tonsil. There is marked swelling of the submaxillary lymph glands.

CHAPTER XXVIII

EXAMINATION OF THE NECK

Shape.—Certain variations in the shape of the neck characterize certain diseases. Thus, a short, thick neck suggests hypertrophic emphysema, and is a constant accompaniment of the barrel chest of this disease. Similarly in plethoric patients the neck is short and thick. A long, slender neck, on the other hand, is frequently observed in phthisical patients.

Rigidity.—This may be caused by tuberculous disease of the cervical vertebræ or rheumatism of the muscles of the neck. Tender, enlarged cervical glands or boils or carbuncles may cause the patient to hold the neck rigid. As previously stated, retraction and rigidity of the neck occur in meningitis, tetanus, and strychnine poisoning. Rigidity of the neck with fixation of the head is also observed as a result of arthritis deformans, spasmodic torticollis, and due to scars from extensive burns of the neck.

Prominent Sterno-mastoids.—Abnormal prominence of both sterno-mastoid muscles is usually a sign of long continued dyspnea, due to pulmonary or cardiac disease. An undue prominence of one sterno-mastoid may be caused by spasmodic torticollis, a tumor, cyst, or abscess of the muscles.

Torticollis.—This is a spasm, usually tonic, rarely clonic, of the sterno-mastoid and trapezius muscles. Its cause is occasionally irritation of the spinal accessory nerve which supplies these muscles, by cicatrices or enlarged glands. Most cases, however, occur without assignable cause. Congenital torticollis is caused by congenital shortness of one sterno-mastoid and is not due to spasm in any sense.

Deflection of Larynx and Trachea.—The larynx and trachea, the latter overlaid by the thyroid gland, occupy the median line of the neck. Deflection of these structures to one or the other side may be due to atrophy of the muscles on one side of the neck, to tumor or aneurism in the adjacent tissues, or to disease of the thoracic viscera. Of the last named factors, fibroid phthisis is very important, the structure being deflected toward the side of the cirrhotic lung.

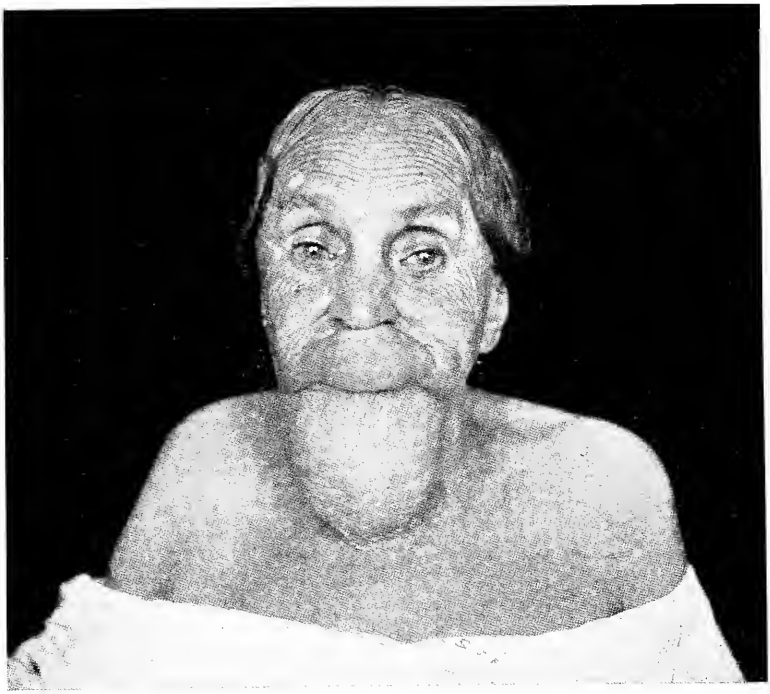


Fig. 263.—Large cystic goiter. (From Eisendrath.)

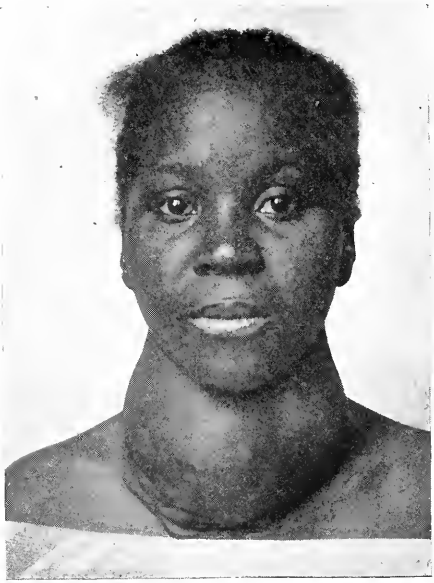


Fig. 264.—Goiter. (From Woolley.)

Movements of the Larynx and Trachea.—Marked inspiratory descent of the larynx occurs in laryngeal stenosis. Normally the larynx descends slightly during inspiration and rises to a similar degree during expiration. When this mobility is abolished in a dyspneic patient the cause of the dyspnea is below the larynx as, for instance, pressure on a bronchus by enlarged glands or aneurism.

Tracheal Tug.—This is an important sign of aneurism of the thoracic aorta, and has been discussed in a previous section.

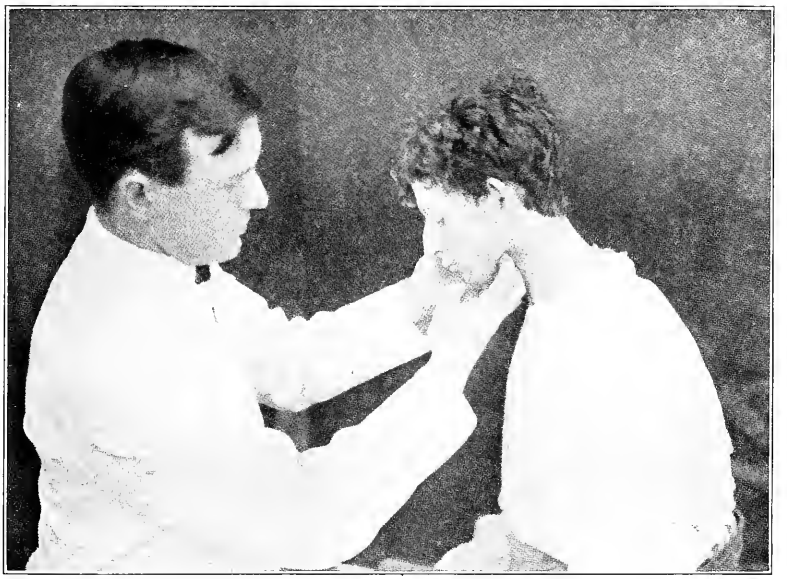


Fig. 265.—Palpation of submaxillary and submental glands. (From Eisendrath.)

Thyroid Gland.—This gland may be increased in size or it may be diminished in size.

Enlargement of the thyroid may involve one or both lobes. The degree of enlargement varies. There may be a small localized swelling at one point, or the entire gland may be found greatly enlarged, exerting dangerous pressure upon the trachea, carotid arteries, and nerves. The consistence of the enlarged gland varies. In the fibrous forms of goiter the gland is hard, while in the cystic form it is soft and may fluctuate. Sometimes a thrill may be detected on palpating the gland, accompanied by a systolic murmur, due to the increased vascularity

of the gland. An enlarged thyroid gland moves with the trachea during deglutition.

The significance of a thyroid enlargement varies with the cause. It may be due to abscess following an infectious disease, or to malignant growth. If due to simple hypertrophy of the gland, the tumor will usually appear during pregnancy, and disappear spontaneously after labor. If fluctuation is detected, it is probably a cystic goiter or an abscess of the gland. If the enlargement be due to exophthalmic goiter it will be associated with the cardinal symptoms of this disease, as tachycardia, exophthalmos, and tremor.

Atrophy of the thyroid gland, revealed by the presence of a

Fig. 266.—Congenital hemangioma of neck. (From Eisendrath.)

depression in the normal position of the gland, occurs in cretinism and myxedema.

Enlarged Glands.—Enlarged lymph glands in the cervical region may have a varied significance, the significance varying with the location of the glands involved and with the state in which they are found; namely, whether hard or soft and fluctuating, whether single and individual or matted together in a mass. Among the causes of glandular enlargement in this region may be mentioned the following conditions.

The lymph glands at the angle of the jaw may enlarge from follicular tonsillitis, diphtheria, scarlatina, measles, German

measles, varicella and smallpox; also in erysipelas, glanders, whooping cough, and retro-pharyngeal abscess. In these conditions the glands are acutely tender for a period and usually undergo resolution without abscess formation.

The sub-maxillary glands, just below the chin may enlarge as a result of carious teeth, stomatitis, syphilis, mumps, cancer of the lower lip or anterior portion of the tongue. This group of glands are often enlarged in cases of actinomycosis.

Fig. 267.—Hodgkin's disease.

The parotid lymph glands enlarge in mumps, and diseases of the upper pharynx and skin of the face, as well as in malignant disease of the parotid gland.

The occipital glands are enlarged in German measles, often the only group attacked in this disease. Enlargement of this group of glands is also a valuable sign of syphilis, and occurs also in cases of pediculosis of the scalp.

Tuberculosis of the cervical glands causes glandular enlargement, particularly in the glands under the jaw. The glands tend to become adherent to the cutaneous structures and often suppurate.

Hodgkin's disease causes glandular enlargement in the lymph glands of the neck, of long standing, involving also the glands of the axilla, groin, and showing slight splenic enlargement.

Lymphatic leukemia is a cause of enlargement of the cervical lymph glands.

Enlargement of the lymph glands above the left clavicle points to cancer of the stomach.

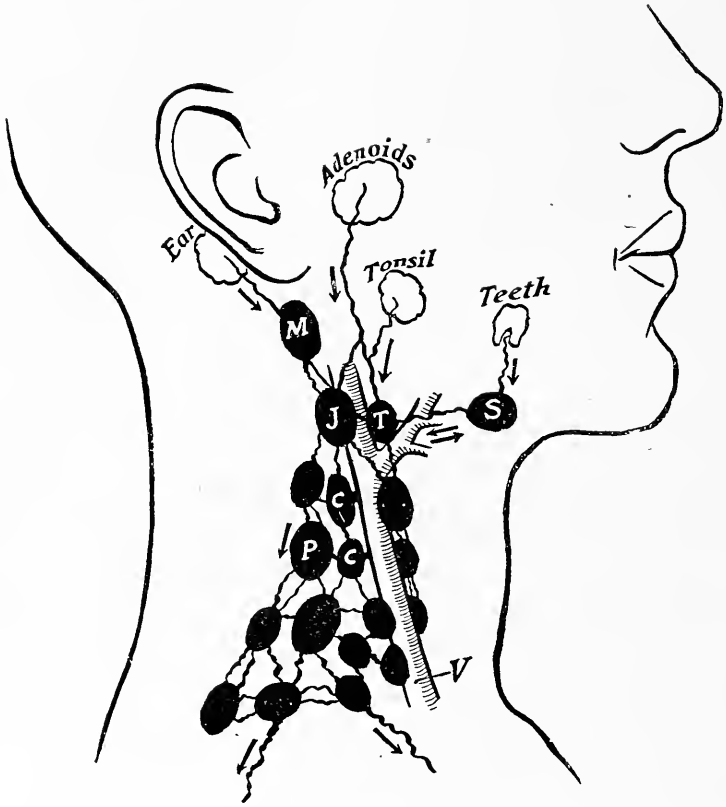


Fig. 268.—Cervical glands commonly involved in tuberculosis. (From Eisendrath.)

The condition of the glands and the duration of the enlargement possess diagnostic significance. Thus, acute painful cases of short duration are probably due to a tonsillitis, or the exanthemata. Chronic cases, of long standing, may be due to tuberculosis, syphilis, or Hodgkin's disease. In tuberculosis the glands are matted together with tendency to suppurate. In syphilis they are hard and small. In Hodgkin's disease the glands are enlarged, but remain separate, and do not tend to suppurate.

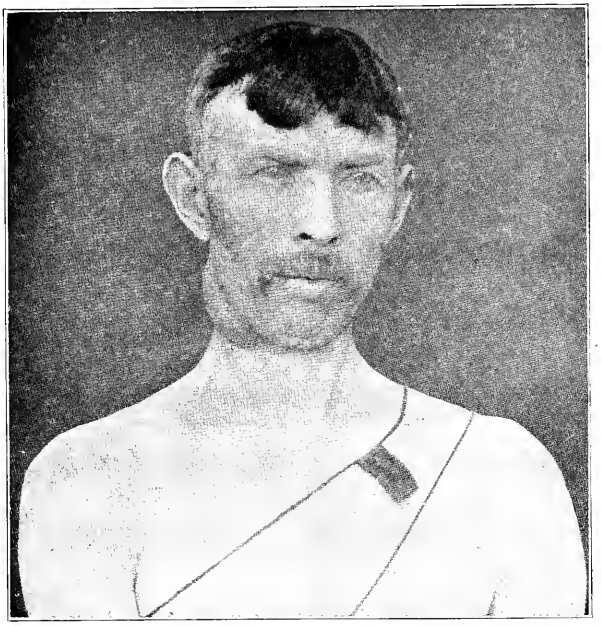


Fig. 269.—Bronchial cyst. (From Eisendrath.)

Fig. 270.—Bronchial cyst. (From McFarland.)

Abscess.—An abscess in the cervical region is almost certainly of tuberculous origin, being the result of tuberculosis of the cervical lymphatic glands. Also Pott's disease high up in the neck may be complicated by abscess (Cabot).

Scars.—Scars in the neck are usually the result of cervical tuberculosis (Cabot). Traumatism and wounds may of course leave scars in this region.

Branchial Cysts and Fistulae.—Branchial cysts and fistulae, resulting from imperfect closure of the embryonic branchial clefts, are encountered in rare instances. A branchial cyst is formed by closure of the pharyngeal and cutaneous surfaces of the cleft without closure of the intervening mesoblastic tissues. Branchial fistulae may be complete or incomplete, depending upon the degree of fusion of the embryonic clefts, the incomplete being represented by diverticula, either external or internal, opening into the pharynx.

Ludwig's angina, a painful indurated swelling beneath the angle of the jaw, due to septic infection of the tissues surrounding the submaxillary gland, is an occasional cause of dyspnea and dysphagia which may become alarming.

Woody or ligneous phlegmon, an insidious induration of the subcutaneous tissues of the neck, involving the lateral or anterior aspect of the cervical region, is occasionally encountered. In some instances the structures are indurated from the jaw to the clavicle; the condition is attended by little pain and no fever.

SECTION II

EXAMINATION OF THE HAND AND ARM

CHAPTER XXIX

THE HAND

THE NAILS

Pallor.—Pallor of the nails is a sign of anemia, and it is well to bear in mind the rule laid down by Stephen McKenzie, that when pressure upon the tip of the finger completely drives the blood from beneath the nail, the red corpuscles are present in only half their normal number.

Cyanosis.—Cyanosis or blueness of the nails is a sign of deficient aeration of the blood, either due to a failing heart, an obstructive pulmonary lesion, or the ingestion of coal tar products. As has been stated, cyanosis appears very early under the nails and about the lips.

White spots in the nails are usually significant of trophic changes in the nail; less commonly they are due to injury of the matrix by picking at the base of the nail.

Capillary Pulse.—The capillary pulse has been described under the section upon the examination of the circulatory organs; and as stated, is a valuable sign of aortic regurgitation or Corrigan's disease.

Transverse Groove.—A transverse groove on the back of a nail is a sign of a recent acute illness. The groove has its inception at the base of the nail and its distance from the base when observed may indicate when convalescence from the illness in question commenced. Thus it requires six months for the groove to progress from the base to the free edge of the nail; hence, if it be encountered half way between the matrix and the

free edge, it is an indication that convalescence began approximately three months previously.

Longitudinal ridges in the nails are said to be a reliable sign of gouty diathesis. Certainly the ridges are encountered in many gouty patients.

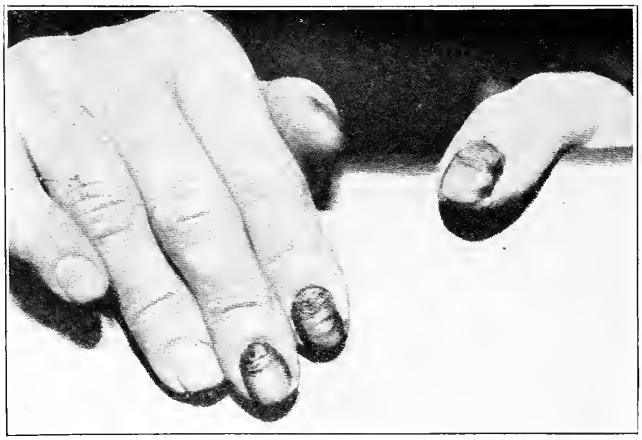


Fig. 271.—Hypertrophy of the nails. (From Hazen.)

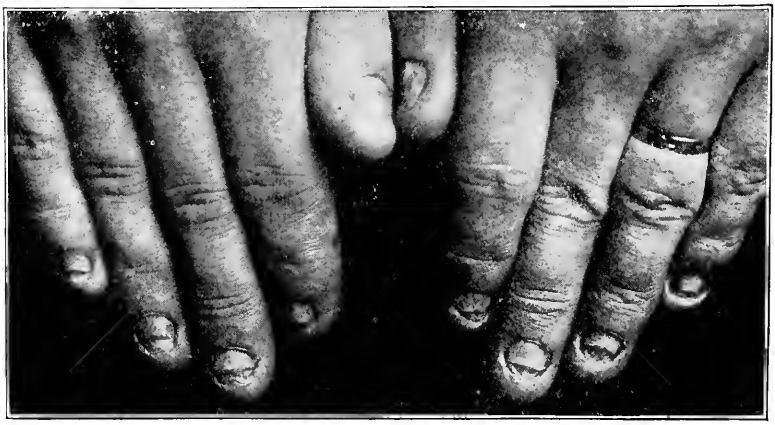


Fig. 272.—Symmetrical atrophy of nails. (Courtesy of Dr. J. C. E. King and Dr. H. G. Parker.) (From Sutton.)

Incurvation of the nails, with or without clubbing of the finger-tips is a sign of chronic disease of the heart or pulmonary tissues, such as cardiac failure, aneurism, phthisis, or emphysema.

The incurvation may be lateral or longitudinal, or may occur in both directions.

Hypertrophy of the nails, particularly in the transverse direction associated with thickening and sometimes with twisting, occurs after acute fevers, particularly following typhoid fever, in connection with syphilis, and in sclerodactyly. A similar hypertrophy of the nail may result from eczema, may be encountered in a subject with Raynaud's disease, and in pulmonary osteo-arthropathy. The nail may be simply hypertrophied without any defect in its structure (megalonychosis); or in addition to hypertrophy the nail may be twisted spirally (onychogryposis).

Atrophy of the nails, with ulceration at the base, occurs in Morvan's disease, a syndrome which develops as a sequence of neuritis and syringomyelia. Atrophy of the nail may follow psoriasis of the fingers.

Arrested Growth.—The growth of the nails is impaired or ceases on the paralyzed side in hemiplegia. A similar arrest of growth of the nails of the paralyzed limb occurs in infantile paralysis. Arrest of growth of a nail may be detected by staining the nails at identical points upon the two hands and observing any discrepancy between the growth of the nails.

Excessive brittleness of the nails is noted in persons of gouty diathesis, the nails frequently presenting the longitudinal striations which have been described.

Onychia, ulceration of the nail matrix, occurs in children with hereditary syphilis, or scrofula, and it is said in persons who are addicted to the chloral habit.

Paronychia, or whitlow, an acute inflammation of the tissues surrounding the matrix of the nail, may be a sequence of local trauma or may be caused by lateral hypertrophy of the nail.

Indolent Sore.—An indolent sore near the root of the nail, if indurated and associated with enlargement of the epitrochlear lymph glands, is usually a chancre; but may be due to tuberculosis.

THE FINGERS

Tophi.—Tophi are concretions of sodium biurate which occur in the joints of the fingers in gouty subjects. They are more prominent on the dorsal surface of the joints, and may break through

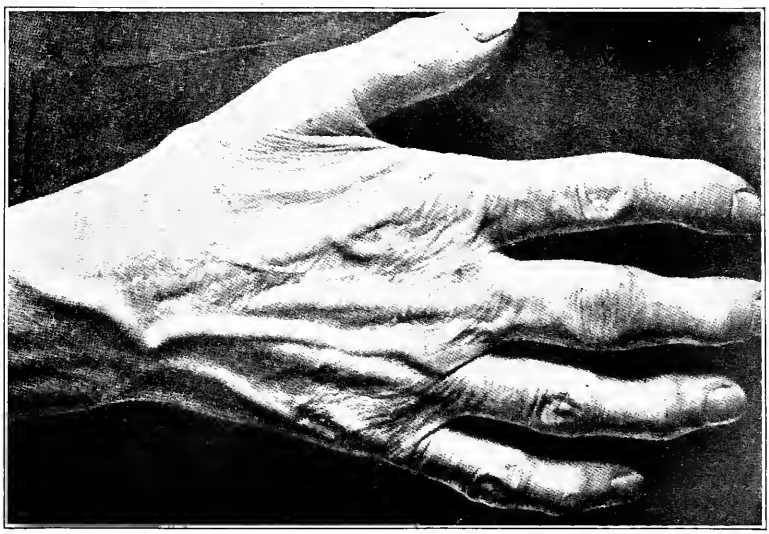


Fig. 273.—Heberden's nodes. (From Butler.)

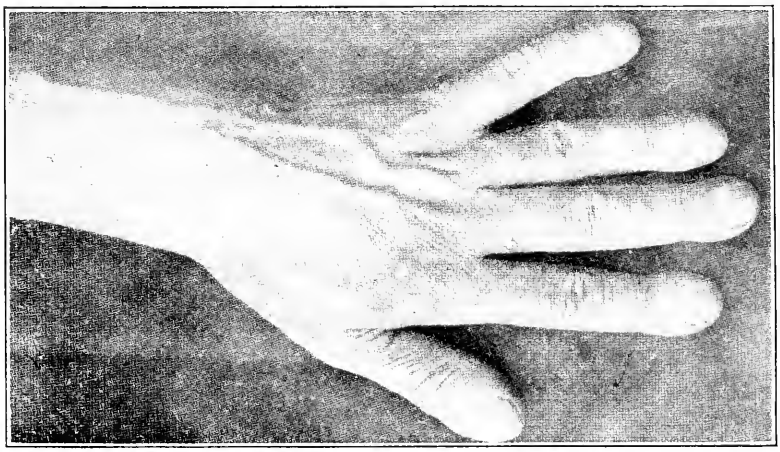


Fig. 274.—Pulmonary osteo-arthropathy. (From Butler.)

the skin, when they constitute the "chalk stones" of the disease.

Enlarged Joints.—Enlargement of the joints of the fingers is seen in connection with gout and chronic rheumatism. In rheumatism the enlarged joints are often hot and painful.

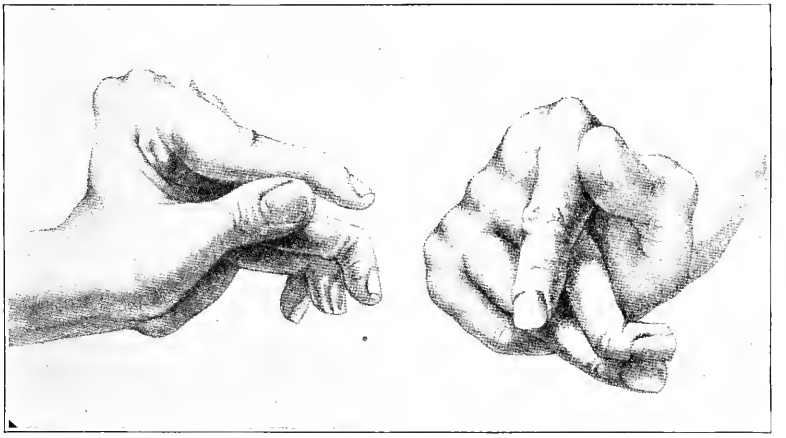


Fig. 275.—Arthritis deformans. (From Butler.)

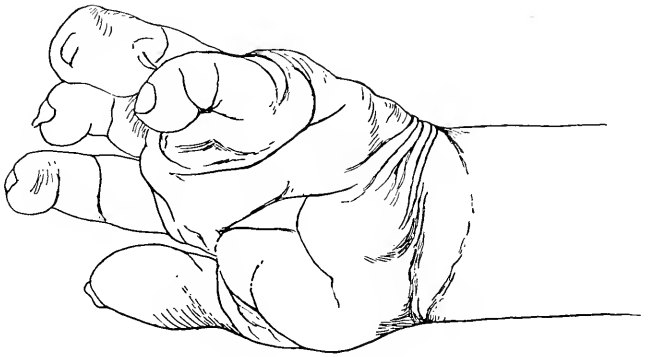


Fig. 276.—Morvan's disease. (From Butler.)

Heberden's Nodes.—These nodes, also termed Haygarth's Nodosities, are knobby enlargements of the proximal ends of the terminal phalanges. They are noted in gout and in arthritis deformans, in which diseases they are said to be of good prognostic significance.

Clubbed Fingers (Hippocratic Fingers).—Clubbing of the terminal phalanges accompanies many chronic diseases of the heart and lungs, notably chronic bronchitis, emphysema, phthisis, and chronic pleurisy, and uncompensated cardiac disease. The nails are commonly incurved. An exaggeration of this condition with swelling of the carpal joints is noted in pulmonary osteo-arthropathy.

Distortions of the fingers accompany gout, chronic rheumatism, and arthritis deformans. The distortions are not produced merely by fixation of the fingers in abnormal positions, but are produced by organic changes, in gout by the deposition of sodium biurate in the joints, in arthritis deformans by absorption of bone and the growth of exostoses. The fingers are most frequently deflected toward the ulnar side of the hand.

Dactylitis.—Dactylitis is usually a sign of hereditary syphilis, less frequently of tuberculosis. In the evolution of the deformity a fusiform purple swelling, which is prone to undergo ulceration with sinus formation, appears upon one or more of the fingers, most frequently involving the proximal phalanges.

Raynaud's Disease.—In Raynaud's disease, or "dead fingers" the fingers are bluish-black or livid, gangrene occurring in spots and leading in many instances to spontaneous amputation of the fingers.

Morvan's Disease.—In this disease the fingers are the site of painless, destructive whitlows, which have their inception adjacent to the base of the nail, leading to necrosis of the terminal phalanges and marked swelling of the fingers.

SHAPE OF THE HAND

The shape of the hand varies in different subjects and under varying conditions of age and occupation. The broad, heavy hand is said to be indicative of a sanguine personality, while the slender hand is said to indicate a nervous temperament. Bluish dotting of the hand of a coal miner points to the possibility of anthracosis, while in old age and in malignant disease and phthisis the hand is small and withered.

Spade Hand.—In acromegaly and myxedema the hand is large, with thick fingers and broad nails. In myxedema the hand is boggy, but does not pit on pressure; whereas in acromegaly the hand is hard, as the basis of the hypertrophy is osseous.

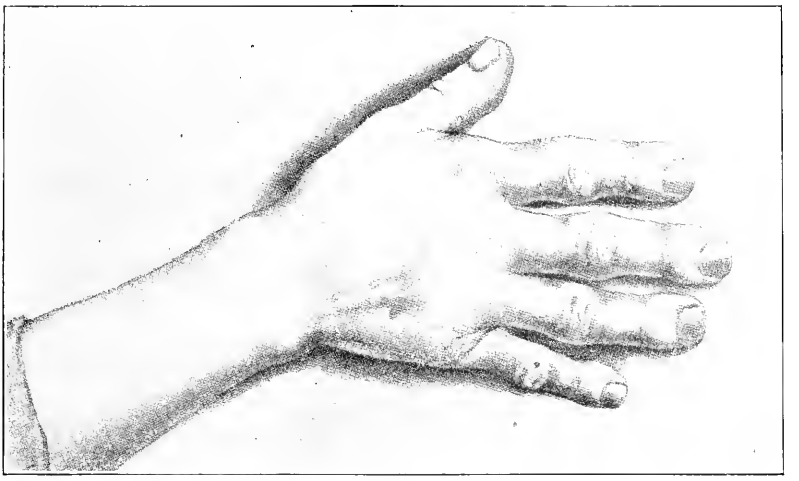


Fig. 277.—Spade hand. (From Butler.)

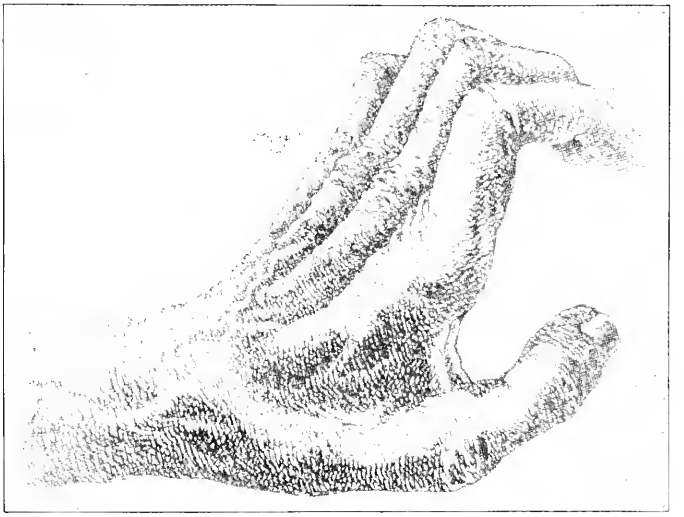


Fig. 278.—Claw hand. (Main-en-griffe.) (From Eisendrath.)

Claw-Hand (Main-en-griffe).—In amyotrophic lateral sclerosis and progressive muscular atrophy the proximal phalanges are drawn backward toward the wrist, while the second and third phalanges are flexed toward the palm. The underlying cause

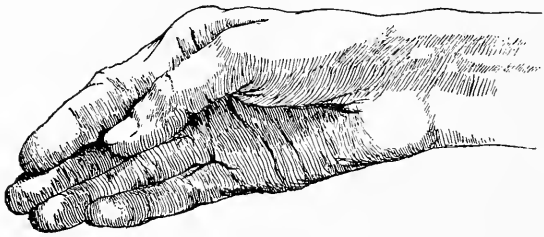


Fig. 279.—Accoucheur's hand. (From Butler.)

Fig. 280.—Wrist-drop. (From Eisendrath.)

of this deformity is a paralysis of the lumbrical and inter-osseous muscles, causing the proximal phalanges to assume a state of dorsal extension, while the distal phalanges are flexed.

Hemiplegic Hand.—In hemiplegia the contractures of the arm

and hand are replaced in course of time by permanent deformity. The fingers in this deformity are flexed upon the palm, the wrist is flexed upon the forearm, while the elbow is retained in a state of permanent flexion and applied closely to the side of the body.

Seal-Fin Hand.—In chronic gout and rheumatoid arthritis the entire hand is deflected toward the ulna as a result of spasm of the extensor muscles, imparting to the hand a fancied resemblance to the fin of a seal.

Ape Hand.—This type of manual deformity is the result of wasting of the thenar and hypothenar muscles in progressive muscular atrophy, causing the hand to assume a position in which the fingers and thumb are on one parallel plane.

Accoucheur's Hand.—In this type of manual deformity, which occurs in Tetany, the thumb is flexed into the palm of the hand, while the fingers, flexed at the metacarpo-phalangeal joints and first inter-phalangeal joints, are extended at the second inter-phalangeal articulations and pressed closely upon the thumb.

Dupuytren's Contracture.—This is a permanent painless flexure of one finger of one or both hands into the palm. Most commonly the little finger alone is involved, but sometimes the ring finger or other fingers are flexed also. Dupuytren's contracture results from burns or other injuries to the palmar fascia.

Ganglion.—A ganglion is recognized as a localized swelling upon the dorsum of the hand. It is presumably caused by cystic degeneration of a synovial fringe within a tendon sheath. Ganglia are not infrequently tuberculous in origin.

Wrist-Drop.—In wrist-drop the hand hangs powerless from the wrist. It is significant of neuritis or paralysis of the musculospiral nerve.

TREMOR OF THE HAND

Intention Tremor.—Intention tremor is a tremor of the hand which is converted into coarse shaking movements when the patient endeavors to perform any act, such as bringing a glass of water to the lips, or holding a pen to write. Intention tremor is a cardinal sign of multiple sclerosis and is sometimes noted in hysteria.

Paralysis Agitans.—In paralysis agitans the patient is subject to a constant tremor of the hands, in which the thumb and index finger are held in close proximity to one another or in actual

contact, describing a rolling movement as if they were rolling a pill (Pill-rolling tremor). The tremor in marked contrast to that of multiple sclerosis disappears completely during voluntary movements of the hands.

Professional Spasm.—Writers, violin players, and others who constantly employ one set of muscles are often troubled with painful spasms in the muscles used, incapacitating them for their usual occupations.

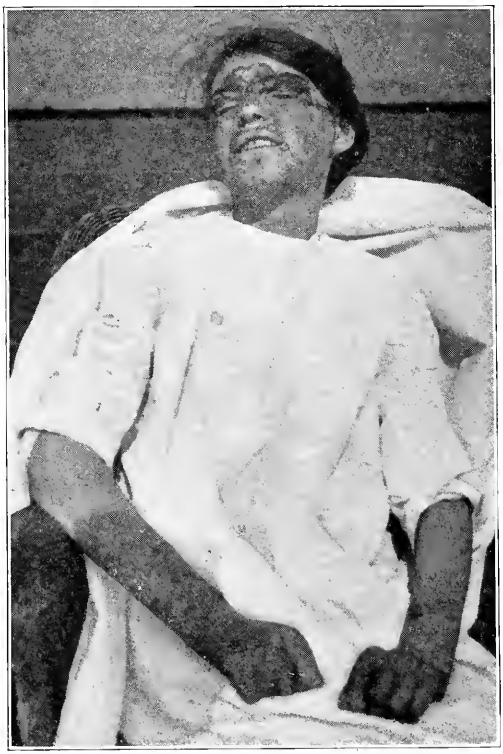


Fig. 281.—Pellagra.

Athetosis.—This term refers to certain slow and purposeless movements of the fingers which are encountered in patients suffering with organic disease of the central nervous system.

Pellagra.—The cutaneous manifestations of pellagra involve the extensor aspect of the hand and forearm, producing an eruption of an erythemato-squamous type. In incipient cases, in the stage of initial erythema, the eruption resembles closely ordinary sun-

burn or solar erythema. In the latter evolution of the disease the superficial epithelium takes on a brown pigmentation, and desquamates in scales. Sensation is lost in the areas of des-

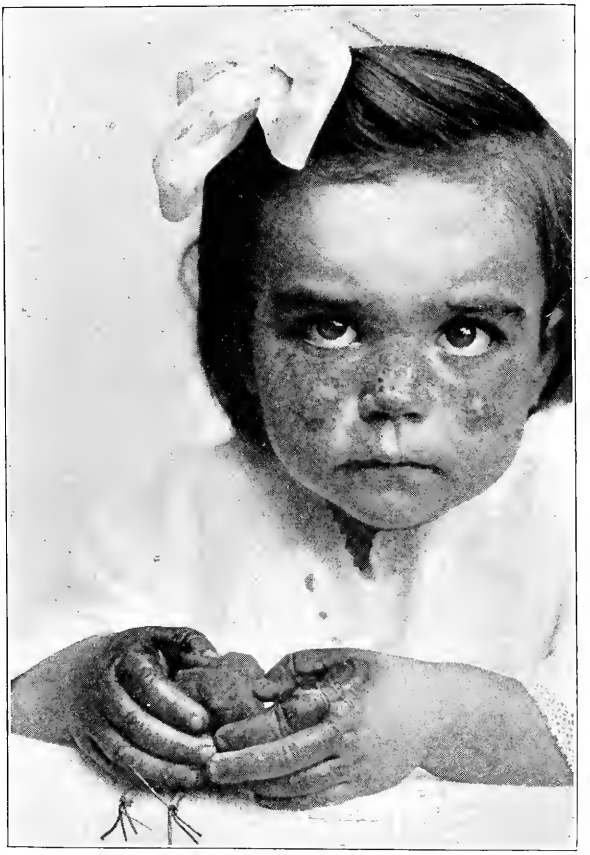


Fig. 282.—Pellagra in child less than 3 years old.

quamation. The skin covering the elbows should always be inspected for the eruption. The eruption may involve the dorsum of the foot or the face, and rarely the neck or chest.

CHAPTER XXX

THE FOREARM AND ARM

EXAMINATION OF THE FOREARM

Epiphyseal enlargement of the forearm bones at the wrist is indicative of rickets. It is usually accompanied by the rachitic rosary and other signs of the disease.

Enlargement of the lower end of the radius with clubbing of the fingers occurs in pulmonary osteo-arthropathy the result of chronic pulmonary or cardiac disease.

Enlargements or nodes along the shafts of the radius or ulna are usually due to syphilitic periostitis.

Erythema nodosum occasionally occurs upon the forearm, manifesting itself by the appearance of elevated, red, shining nodular swellings, which are very painful upon pressure.

Edema of the forearm, usually affecting the arm as well, results from thrombosis of the axillary vein, or from the pressure of mediastinal tumors upon the subclavian vein.

EXAMINATION OF THE ARM

Tumors.—A superficial tumor arising in the arm is most apt to be lipomatous. It is often lobulated. A ruptured biceps produces a sharp tumor over the lower portion of the arm. A deeply seated tumor of the arm is most likely to prove sarcoma of the humerus. An acute painful swelling of the humerus following typhoid fever or scarlatina is due to acute periostitis.

Small Nodular elevations upon the humerus are usually the result of syphilitic periostitis.

Paralysis.—Paralysis of one arm may be total, the arm hanging limply, without power of movement, or may be partial. Partial brachial paralysis may assume one of two types; namely, the upper arm type of Duchenne-Erb, or the lower arm type of Klumpke. Brachial paralysis may be the result of trauma at

birth, or may result from compression of the brachial plexus by a tumor or by a crutch. Some cases are due to a faulty position of the arm during anesthesia. The upper arm type of Duchenne-Erb involves the deltoid, brachialis anticus, triceps, supinator longus, supinator brevis, and the infra-spinatus muscles. The patient is unable to adduct the arm, and the forearm remains in a position of extension and pronation. The lower arm type of Klumpke involves the small muscles of the forearm and hand, with inability to move the hand or fingers.

Rigidity and Contracture.—In hemiplegia the paralysis of the arm is spastic and is followed in the course of time by permanent contracture. The elbow is maintained in a state of semi-flexion,

Fig. 283.—Lipoma of arm.

the wrist is flexed upon the forearm, while the arm is often closely apposed to the trunk. Spastic rigidity of the arm is often one of the earliest signs of chronic hydrocephalus.

Movements.—In Sydenham's chorea the arms participate in the purposeless movements of the head and face. In this form of chorea there is no motor weakness; whereas in the so-called *hemiparalytic chorea,* which is attended by similar purposeless movements of the arms, the muscular power is usually impaired. Pregnant women occasionally exhibit similar purposeless movements of the arms in the so-called *chorea gravidarum.*

In *paramyoclonus multiplex* there is frequently noted a symmetrical, bilateral, clonic spasm of the muscles of the arms. The biceps, triceps, and deltoid muscles are involved. The paroxysm

is characterized by a series of very rapid clonic contractions of symmetric groups of muscles in the two arms, the contractions often exceeding a hundred in a minute. Usually of very brief duration, the paroxysm may in some cases last for several moments.

Atrophy.—Atrophy of the muscles of the arm follows the paralysis of acute anterior poliomyelitis and brachial palsies, conditions which involve the lower motor neurone.

Miner's Elbow.—In this condition there is a swelling overlying the olecranon bursa, produced by chronic bursitis of this structure, which sometimes yields fluctuation on palpation.

SECTION III

EXAMINATION OF THE LOWER EXTREMITIES

CHAPTER XXXI

THE FOOT, LEG, AND THIGH

THE TOES

Gangrene of the toes is usually significant of diabetes, arteriosclerosis, or Raynaud's disease. Gangrene of the toes is less frequently a sequence of frost-bite, local trauma, ergotism, or embolism in connection with cardiac disease.

Perforating Ulcer.—The perforating ulcer, or Mal Perforante,

Fig. 284.—Gangrene of toes.

occurring with locomotor ataxia and rarely with diabetes, is a deep circular ulcer, usually situated upon the under surface of the great toe.

Gout produces hot tense swelling of the metatarso-phalangeal articulation of the great toe, which is very sensitive to pressure.

THE FOOT

Flat-Foot, pes planus, is a flattening or giving way of the normal arch of the foot as a result of muscular paralysis, or ligamen-

tous weakness from long standing or traumatism. Flat-foot is a sequence of rickets and infantile paralysis. Flat-foot is recognized by painting the sole of the foot with a colored fluid and causing the patient to stand upon a piece of paper, and noting whether an impression of the entire sole is left upon the paper.

Club-foot or talipes is a permanent fixation of the foot in deformity. In *talipes equinus* the heel is drawn up in such a manner that the patient walks upon the ball of the foot or the toes. In *talipes varus* the foot is inverted, the patient walking upon its outer border. In *talipes valgus* the foot is everted and the patient walks upon the inner border of the foot.

Enlargement of the foot with more or less distortion occurs in acromegaly, myxedema, and pulmonary osteo-arthropathy.

Erythromelalgia.—In this condition the sole of the foot is very red and the seat of burning pain, which is made worse by walking and is relieved by elevating the limb.

THE LEG

Bowing of the tibiae is most commonly due to rickets, but may also be noted in connection with osteitis deformans, mollities ossium, and cretinism.

Nodes.—Red, shining nodes situated over the tibiae, which are very painful upon pressure are indicative of erythema nodosum, which is more frequently encountered here than in any other locality.

Deep nodular swellings, situated upon the tibia are due to syphilis periostitis, while painless, non-inflammatory indurated areas distributed over the leg may be gummata.

Leg Ulcers may be due to varicose veins, but are often due to tertiary syphilis, especially if there are multiple annular ulcers situated nearer the knee than the ankle.

Swelling of the Calves in children, associated with loss of muscular power and difficulty in rising to the erect posture, is indicactive of pseudo-hypertrophic muscular palsy.

Atrophy of the muscles clothing the anterior and outer aspects of the leg is a sign of progressive muscular atrophy.

Varicose Veins.—Varicosities of the veins of the leg are indicative in some instances of prolonged standing, or the pressure of a pregnant uterus or tumor within the abdomen upon the vessels returning blood from the lower extremity.

Kernig's Sign.—In acute meningitis it is impossible to fully extend the leg upon the thigh. To elicit Kernig's Sign the patient should be placed upon the back with the thigh flexed to a right angle with the body. An effort is then made to extend the leg, bringing it in a line with the thigh. In the presence of meningitis it is difficult or impossible to extend the leg because of the

Fig. 285.—A case of rickets. (From Woolley.)

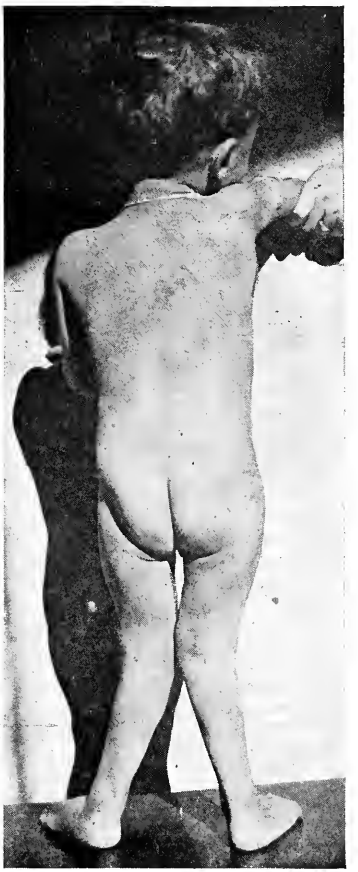


Fig. 286.—A case of rickets. (From Woolley.)

marked flexor contracture of the hamstring muscles. In diagnosing meningitis by means of this sign it is necessary to exclude sciatica, old contractures, myositis, and tuberculous disease of the knee joint.

Charcot's Joint.—In the course of locomotor ataxia not uncommonly as a result of trophic disturbance the knee-joint assumes

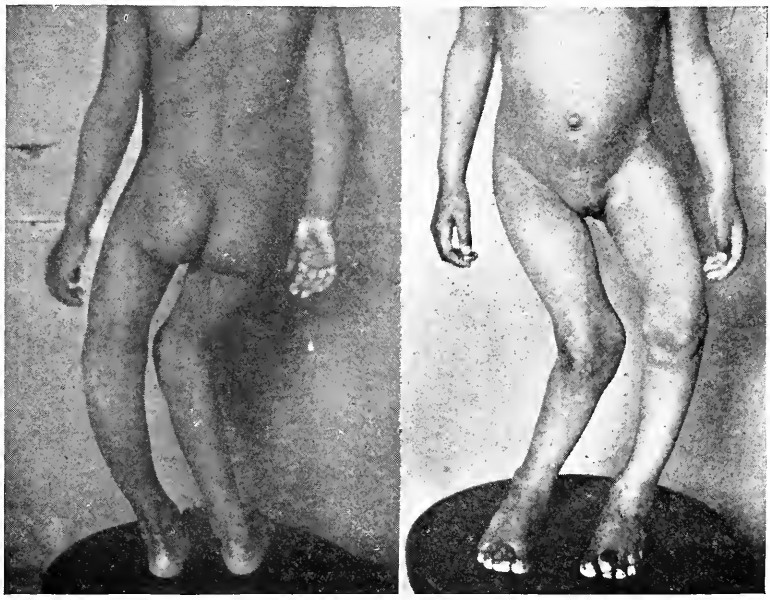


Fig. 287.—A case of rickets. (From Woolley.)

Fig. 288.—Showing extreme case of bow-legs. (From Woolley.)

an enormous size, due to chronic inflammation of the synovial lining of the joint, which later progresses to the bone itself. The enlargement of the joint is always considerable and may become enormous. Early in the case the enlargement is due to effusion in the joint, but later it is produced by true osseous overgrowth. Pain is slight or is entirely absent. Usually affecting the knee-

Fig. 289.—Varicose ulcer of leg. (From Eisendrath.)

joint, the condition may involve the hip-joint, and less commonly the smaller articulations.

Housemaid's knee, produced by chronic bursitis of the prepatellar bursa as a consequence of persistent pressure upon the bursa incident to occupation, is characterized by effusion into the knee-joint, the effusion pushing the patella upward before it. Fluctuation can sometimes be obtained.

THE THIGH

Edema of the thigh, affecting the leg and foot as well possesses definite significance depending upon whether it is uni-lateral or bi-lateral. Thus, edema of one lower extremity may result from varicose veins or thrombosis of the femoral vein. Bi-lateral edema points to cardiac insufficiency or hepatic disease producing general anasarca.

A chronic swelling of the lower end of the femur is often due to osteo-sarcoma of that bone.

Intermittent Claudication.—In subjects of arterio-sclerosis an intermittent lameness may result from deficient circulation to the muscles of the thigh.

Inguinal Adenitis.—Enlarged glands in the inguinal region may indicate venereal disease. In gonorrhea and chancroid the

Fig. 290.—Osteosarcoma of femur.

glands are matted and tend to suppurate, whereas in syphilis the glands are only moderately enlarged, are hard, and discrete. Inguinal adenitis of long standing is suggestive of tuberculous disease of the hip or knee, or Hodgkin's disease. In malignant disease of the genitalia there is early inguinal adenitis.

Swelling in Scarpa's Triangle.—A swelling in this portion of the thigh may be due to femoral hernia, or psoas abscess, the latter always occupying a position external to the femoral vessels.

Osteitis Deformans, (Paget's Disease).—Osteitis produces bowing of the bones of the thighs, with a consequent diminution of the stature. The head in this disease is characteristically deformed, and the contour of the thorax and abdomen is altered.

Osteomalacia, in its evolution is characterized by bowing of the bones of the lower extremity, produced by softening and rarefaction of the osseous structures peculiar to this disease.

Rickets.—In advanced rickets there is usually notable bowing of the bones of the lower extremities, leading to "bow-legs" or "knock-knees." In addition there are symmetrical swellings at the epiphyses of the long bones.

Pulmonary Osteo-Arthropathy.—In this disease the extremities of the long bones of the lower extremity, particularly of the tibiae, participate in the chronic enlargement which characterizes the disease.

Phlegmasia alba dolens, resulting from thrombosis of the femoral vein, produces swelling and edema of the thigh, with marked tenderness upon manipulation. The usual cause is puerperal sepsis, but this condition is also a not infrequent complication of typhoid fever.

Paralysis.—Paralysis of one leg if spastic is usually a part of a hemiplegia, but may rarely be due to a cortical lesion involving the leg center. Flaccid paralysis of one leg is the result of pressure neuritis, chronic lead poisoning, or anterior poliomyelitis.

Paralysis of both legs, paraplegia, may result from a cerebral lesion, as is the case in Little's disease, or may be due to transverse myelitis, disseminated sclerosis, or the late stages of locomotor ataxia.

PART IV

EXAMINATION OF THE NERVOUS SYSTEM

SECTION I

MOTOR AND SENSORY PHENOMENA

CHAPTER XXXII

STATION, GAIT, AND MUSCULAR POWER—TREMOR

Introduction.—The sources of the nervous impulses which initiate muscular movements reside in certain specialized cells of the cerebral cortex lying anterior to the fissure of Rolando, in the nuclei of the cranial nerves at the base of the brain, and in the anterior horns of the spinal cord. The experimental work of Hughlings Jackson, Hitzig, Ferrier, and Horsley has demonstrated that the motor path from the cerebral cortex to the voluntary muscles comprises two segments, or neurones; namely, the *upper motor neurone,* extending from the cerebral cortex to the anterior cornual cells, and forming synapses with the cells of certain of the nuclei of origin of the cranial nerves; and the *lower motor neurone,* which extends from the anterior cornual cells to the muscle in question.

The axis cylinders of the *upper motor neurone,* arising from cells of the cerebral cortex in the motor area pass downward into the white substance of the brain to form the corona radiata. They are collected into a compact bundle of fibers which traverse the internal capsule between the basal ganglia, constituting the genu and anterior two-thirds of the posterior limb of this structure. Emerging from the internal capsule, the upper motor neurone enters the crus cerebri, some fibers at this point crossing to the opposite side to form synapses with the cells of the nucleus of origin of the oculomotor nerve. The upper neurone traverses the crus and pons, distributing fibers to all of the motor cranial

nerves of the opposite side and a few fibers to the same nerves on the same side, and enters the anterior portion of the medulla oblongata to form the pyramid. In the medulla the greater number of the fibers constituting the upper motor neurone cross to the opposite side, forming the decussation of the pyramid. These fibers enter the lateral portion of the spinal cord as the crossed pyramidal tract, while the smaller number of fibers, which did not cross at the decussation, pass down the anterior portion of the cord as the direct or uncrossed pyramidal tract. The fibers of the crossed and direct pyramidal tracts terminate at various levels of the cord by forming synapses with the anterior cornual cells, the direct pyramidal fibers crossing in the anterior white commissure before forming this junction. Thus the upper motor neurone terminates by effecting a junction with the cells of origin of the lower motor neurone. It is to be noted that impulses arising in the cerebral cortex are all transmitted to the opposite side of the spinal cord by the upper motor neurone.

The axis cylinders of the *lower motor neurone* arise in the anterior cornual cells and emerge as the anterior spinal nerve roots to form the peripheral nerves which supply muscles on the same side of the body. They do not cross.

The sensory conducting system comprises three neurones. The *first sensory neurone* is derived from the ganglia upon the posterior nerve roots, the axis cylinders of which divide in a T-shaped manner, the longer division going to the peripheral sensory nerve, while the shorter branch enters the posterior horn of the spinal cord and divides into a long ascending and a short descending branch. The longer, ascending branches from this source ascend in the posterior columns of the cord to terminate in cells of the gray matter of the same side of the cord or to ascend to the nucleus gracilis and nucleus cuneatus of the medulla.

The *second sensory neurone* arises from the medullary cells or the medullary nuclei, form the arcuate fibers, and terminate in synapses about the cells of the median and lateral nuclei of the optic thalamus of the opposite side.

The *third sensory neurone* takes origin from the nuclei of the optic thalamus and terminates in the sensory areas of the cerebral cortex.

Gross lesions involving the integrity of the upper motor neurone in any portion of its course from the cerebral cortex to the

anterior horns of the cord produce spastic paralysis of definite portions of the muscular system; since the regulating or governing impulses descending from the cerebral cortex are in abeyance and the constant tonic impulses from the anterior cornual cells are uncontrolled. Lesions of the lower motor neurone, on the contrary, produce flaccid paralysis, with atrophy of the muscle, as trophic impulses have their origin in the anterior cornual cells.

The Station.—The station is the attitude of the patient when standing at ease in the erect posture. In testing the station the patient should be directed to stand with the feet closely approximated, and the test should be made first with the eyes open and then with the eyes closed. A normal person while undergoing this examination will frequently sway slightly from side to side, and in cases of muscular weakness, either from exhausting disease or from neurasthenia, the swaying is more marked. But when the swaying movement becomes so extreme that the patient is in danger of falling if not supported, the station becomes pathological. Thus in tabes dorsalis the patient with feet closely approximated and the eyes closed sways excessively and if not supported is apt to fall (Romberg's sign).

The Gait.—In many nervous diseases the gait is characteristic and gives at once a clue to the correct diagnosis. In observing the gait of a patient who is suffering with an organic nervous disease the clothing should be removed from the lower extremities so that the phenomena attending locomotion may be clearly observed.

The Spastic Gait.—In spastic diplegia due to lesions in the lateral pyramidal tracts the lower limbs are stiff owing to an inability to bend the knees, so that the patient progresses by means of short steps, the toes scraping along the floor. The toes of the shoes are worn excessively. The presence of a marked ankle clonus on both sides communicates a general tremulousness to the entire carriage of the patient, who is apt to stumble over slight obstacles and fall.

The Hemiplegic Gait.—The hemiplegic gait is merely a unilateral spastic gait, the spastic limb during progression describing an arc of a circle while the sound limb supports the weight of the body. In spastic cerebral paraplegia, or double hemiplegia, both limbs describe the arc of a circle during progression, each foot in turn being swung outward and planted in front of the other with the production of the cross-legged or

"scissor" gait; the trunk and upper limbs meanwhile being jerked about from side to side in the effort to move the spastic members forward.

The Steppage Gait.—Patients with multiple neuritis with foot drop, or with lesions of the lumbo-sacral region of the spinal cord exhibit the steppage gait, a mode of progression in which

Fig. 291.—Little's disease. (Infantile spastic diplegia.)

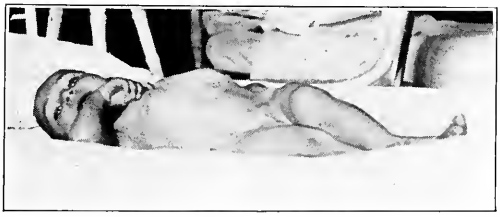


Fig. 292.—Little's disease. (Infantile spastic diplegia.)

each foot is alternately raised high, the toe thrown upward, the foot striking the ground forcibly, as if the patient were continually stepping over obstacles in his path. A uni-lateral steppage gait accompanies paralysis of the external popliteal nerve.

The Ataxic Gait.—The ataxic or tabetic gait occurs typically in tabes dorsalis, a very similar gait being observed in Fried-

reich's ataxia, and in tumor of the posterior columns of the cord. The patient walks on a very broad base, swaying from side to side. The foot in progression is raised suddenly from the floor, is thrown forcibly forward, and thrown forcibly down "in flail-like fashion," the heel usually striking the floor first. The patient keeps the eyes fixed steadily upon the floor before him in the effort to guide his onward course. He is unable to suddenly stop or start on command or to turn suddenly and retrace his course. Similar ataxia in the upper limbs is demonstrable in the inability of the patient to touch the finger-tip to the nose, or to accurately approximate the finger-tips with the arms before the body.

The Festinating Gait.—In paralysis agitans the patient moves forward with the body inclined somewhat forward, advancing with short, shuffling steps which become progressively faster as he crosses the room. When ordered to turn, the entire body is turned *en masse*. This type of locomotion constitutes the so-called *propulsion*. *Retropulsion* may often be elicited in these patients. If the patient is quickly pulled backward, and, indeed sometimes on merely looking upward, he tends to run backward with short, shuffling steps, although the body is invariably inclined forward.

The Cerebellar or Vertiginous Gait.—In cerebellar disease associated with severe vertigo the patient progresses in a very irregular course, often lurching from side to side. Quite frequently the patient exhibits a tendency to reel in a fixed direction, forward, backward, or to one side. This type of progression occurs with uni-lateral cerebellar lesions. In uni-lateral cerebellar tumors the head is not infrequently inclined toward opposite side, while the face is turned slightly toward the side of the lesion.

Muscular Power.—A rough estimate of the muscular power may be made by the "resistance method," the patient being directed to perform the functon of a given muscle, while the examiner endeavors to resist the movement and gauges the amount of power required in the effort. Variations in muscular power range from simple weakness to complete loss of power or paralysis. Paralysis may be complete or partial, in which latter event it is termed paresis. Paralysis may be *spastic*, when the paralyzed limb is rigid and the muscles unyielding to passive movement, or *flaccid*, when the muscles are soft and pliable. Paraylsis may affect one limb, when the condition is termed *monoplegia*; it

may affect one entire side of the body, when it is termed *hemiplegia;* or all four limbs may be involved, when the condition is designated *diplegia.*

Tremor.—Coarse shaking movement of the muscles of the hand upon voluntary muscular effort (intention tremor), accompanies disseminated sclerosis. On the contrary, the fine "pill-rolling" tremor of paralysis agitans is inhibited by voluntary movement. Convulsive tremors involving a small or limited group of muscles are observed in Jacksonian epilepsy, while fibrillary twitchings accompany progressive muscular atrophy. A hemichorea may persist for years as a residual sign of cerebral hemorrhage.

CHAPTER XXXIII

SENSORY PHENOMENA—THE REFLEXES

Tactile Sensation.—The acuity of tactile sensibility is tested by gently touching the cutaneous surface in various regions with a feather or a small wick of cotton, while the patient's eyes are closed. A normal person can state the instant when the skin is touched and, in a general way, the nature of the fabric with which it is in contact. While the readiness with which the tactile sensations are registered is a reliable index to the integrity of the sensory pathway, allowance must be made in certain instances for the degree of natural intelligence of the patient under examination. Tactile sensibility is impaired (hypesthesia) in compression or partial lesion of the sensory pathway; it is abnormally acute (hyperesthesia) in functional irritability of the sensory tract; and it is abolished (anesthesia) in organic disease of the dorsal columns of the cord.

Pressure Sense.—The pressure sense is investigated by noting the ability of the patient to appreciate minor variations in pressure, when cubes of uniform size, but of varying weight, are placed upon the surface under examination. During this examination muscular sensation must be eliminated by placing the limb upon a firm, unyielding surface; and temperature sense must be excluded, as extremes of temperature have a tendency to impair the nicety of the pressure sense. Variations in the acuity of the pressure sense have the same significance as have variations in the tactile sense.

Sense of Temperature.—The entire cutaneous surface is supplied with specific "heat spots" and "cold spots," which are supplied by nerve endings for the appreciation of these varieties of sensation. Hence the power of discriminating variations in temperature may be retained, while tactile sensation is temporarily abolished. Thus, compression of the ulnar nerve, which causes a marked diminution of tactile sensibility over the distribution of the nerve, does not involve the temperature perception in this area. As a general rule, the portions of the body which are habitually clothed are more sensitive to thermic variations than are the exposed portions of the integument.

The sense of temperature may be tested by applying to the area under examination alternately test-tubes containing water at temperatures slightly above and below that of the body. In organic disease of the spinal cord, as syringomyelia and in lesions of the medulla and pons, as hemorrhage, tumor, or softening, the perception of temperature may be impaired (thermo-hypesthesia); may be intensified (thermo-hyperesthesia); or it may be abolished (thermo-anesthesia) in the area under investigation.

Sense of Pain.—It is generally agreed that there are specialized "pain spots" distributed universally over the cutaneous surface, which are supplied with special nerve endings; and that the sensation of pain is not merely due to over-stimulation of the nerve endings of tactile or temperature sensation. The sense of pain is tested by pricking the skin with a fine needle. In certain instances the pain sense is perverted, a stimulus applied to one limb causing a painful sensation in the opposite limb. Diminution of the perception of pain (hypalgesia) is encountered in compression and partial lesions of the sensory nerves; increased sensibility to pain (hyperalgesia) accompanies functional irritation of these tracts; while abolition of the pain perception (analgesia) signifies a total destruction of the sensory tract.

Muscular Sense.—Muscular sense is the sense by means of which judgments are formed as to the weight of articles which are lifted, by which the patient is aware of the position of certain portions of the body without the aid of the eyes, and by which he is enabled to maintain the standing posture without conscious effort.

The muscular sense may be examined by directing the patient with the eyes closed to place the finger upon a certain portion of the body, as for instance, the tip of the nose; it is also tested by directing the patient to stand upright with the feet closely approximated and with closed eyes. Thus, in organic disease of the nervous system the disturbance of the muscular sense, with similar tactile sensory disturbance, is responsible for Romberg's sign.

The muscular sense is also investigated by noting the perception of active and passive movements of the limbs. Thus, the patient is directed to perform various movements with the limbs, such as describing a semi-circle on the floor with the toe, or touching the knee with the ankle of the opposite limb. In testing the perception of passive movements, the limb of the patient is slowly moved while the eyes are closed, and he is asked to

indicate the range of the movement and the new position of the limb.

Stereognostic Sense.—Stereognostic sensibility is the faculty by which objects placed in the hand are recognized by their palpable shape and consistence. An abolition of this sense (astereognosis) is often indicative of a lesion involving the superior parietal lobule of the brain.

The Reflex Arc.—The reflex arc comprises an afferent or sensory neurone, which conducts impulses to an intermediate cell station, and an efferent neurone over which the cell station or medullary center discharges motor impulses in response to the sensory stimulus which is conveyed to it by way of the afferent sensory neurone. The entire sequence of changes constitutes a reflex act. In the case of the spinal reflexes the afferent neurone is represented by peripheral spinal sensory nerve with its root ganglion, the intermediate cell station by the gray matter of the spinal cord, and the efferent neurone by the motor nerve arising from the cells of the anterior horn of the cord. Spinal reflexes occur without any intervention on the part of the cerebrum, the different segments of the cord acting independently; but there are governing fibers descending in the pyramidal tracts from the cerebral cortex which modify and may voluntarily abolish or inhibit the spinal reflexes.

The Patellar Tendon Reflex (Knee-jerk).—If the patient is in a sitting posture, to elicit this reflex the leg is flexed at a right angle with the thigh, while the patellar tendon is struck a rapid light blow with a percussion hammer. If the patient is bed-ridden, the reflex may be elicited by raising the leg from the bed by means of a hand placed beneath the knee-joint while the blow is delivered. During the examination every effort should be made to distract the attention of the patient from the procedure, in order to prevent cerebral inhibition of a normal reflex. If the patient is very self-conscious and the reflex consequently is elicited with difficulty, Jendrassik's reinforcement may be resorted to. The patient is directed to lock the hands and pull, meanwhile keeping his eyes fixed upon the ceiling.

Exaggeration of the patellar tendon reflex is indicative of disease between the level of the reflex arc and the cerebral cortex, whereby the governing impulses from the cerebrum are interrupted. Such a condition arises in spastic spinal paraplegia, amyotrophic lateral sclerosis, cerebral hemorrhage, and disseminated sclerosis.

Abolition of the patellar tendon reflex is indicative of a break in the reflex arc due to disease of the sensory neurone, posterior root zone, or anterior root cells. Such lesions arise during tabes dorsalis, anterior poliomyelitis, peripheral neuritis and trauma to the cord at the site of the reflex arc.

Tendo-Achilles Reflex.—This reflex is elicited by directing the patient to kneel upon a chair, and, rendering the tendon taut by moderate dorsal flexion of the foot, whereupon the tendo-Achilles is struck sharply, when normally sudden extension of the foot is produced. The significance of variations in the response are the same as those for the knee-jerks.

The Plantar Reflex.—When the sole of the foot is stimulated by stroking with a match or probe, plantar flexion of all the toes ensues. In upper motor neurone involvement, instead of the uniform plantar flexion of the toes, the great toe is extended while the remaining toes are flexed (Babinski's sign).

Ankle Clonus.—To elicit this clonus the examiner grasps the calf of the leg in the palm of the left hand, while with his right hand he exerts pressure upon the fore part of the sole of the foot, thus maintaining the foot in a position of dorsal flexion. In disease of the upper motor neurone, as disseminated sclerosis, cerebral hemorrhage, or spastic paraplegia, a series of regular, rhythmic contractions of the calf muscles ensue, which continue until the muscles are temporarily exhausted.

Patellar Clonus.—The patellar clonus is elicited by placing the limb in a position of full extension and grasping the patella between the thumb and fingers and exerting strong downward pressure upon the quadriceps extensor tendon. In disease of the upper motor neurone a series of rhythmical contractions are set up in the quadriceps extensor analogous to that obtained in the ankle clonus.

CHAPTER XXXIV

THE CRANIAL NERVES

The twelve cranial nerves are paired nerves, resembling in this respect the spinal nerves. The first two cranial nerves, however, the olfactory and optic, differ so markedly in their anatomic and physiologic features from the other cranial nerves as to have been compared to accessory lobes of the brain. The centers of the cranial nerves lie in a mass of gray matter along the floor of the fourth ventricle, the aqueduct of Sylvius and the floor of the third ventricle, representing an upward continuation of the central gray matter of the spinal cord.

Lesions involving the cranial nerves may be situated in the cerebral cortex or the fibers descending from the cortical cells to the deep origin of the cranial nerves (supranuclear lesions), may involve the nucleus alone (nuclear lesions), or may involve only the peripheral portion of the nerve (infranuclear lesions). While supranuclear and infranuclear lesions not infrequently manifest themselves in derangements of a single cranial nerve, the nuclei of origin of these nerves are so closely aggregated beneath the floor of the fourth ventricle and sylvian aqueduct that a lesion in this situation usually involves the nuclei of several cranial nerves, with the consequent production of more general manifestations.

THE OLFACTORY NERVE

The center for the olfactory nerve is probably situated in the uncinate and hippocampal gyri, with communicating fibers to the cerebral cortex, optic thalamus, and internal capsule. The terminal branches of distribution of the nerve are distributed to the superior turbinated bodies and the upper portion of the septum, whence they pass upward to the dilated anterior extremities of the optic tracts, the optic bulbs.

The integrity of the olfactory nerve is tested with familiar odorous substances, such as the oils of peppermint or cloves, cologne water or cinnamon. Ammonia or acetic acid should not be employed, as they are known to affect the trigeminal nerve. In

applying the test the substance is applied to each nostril separately and in turn, with the eyes of the subject meanwhile closed.

The sensibility of the nerve may be diminished or abolished by local or central conditions. The most frequent cause for loss of the sensibility of the nerve lies in local nasal conditions, as coryza or polypi. In the aged there is often a normal diminution in the acuity of the perception and differentiation of odors. Moreover, after prolonged or excessive stimulation the sense of smell becomes blunted or diminished for the time being.

Marked diminution in the acuity or abolition of the olfactory sense, *anosmia,* is significant of many intracranial conditions. In congenital absence of the olfactory nerves it is a natural sequence. Compression of the nerve trunk by aneurism of the middle cerebral artery, by chronic hydrocephalus, by a cerebral tumor or abscess, or irritation by a meningitis chiefly localized to the anterior fossa of the skull results in anosmia. Destructive lesions of the bulb or tract, caries of the cranial bones, or injury incurred during basal fracture, cause anosmia. Similar loss of the olfactory sense is noted in tabes dorsalis and paresis.

Perversions of the olfactory sense, *parosmia,* are not infrequently met with in cases of tabes dorsalis, during the aurae of epileptic seizures, and in various mental disorders.

Hyperacuity of the sensibility of the nerves, *hyperosmia,* occurs in neurotic and insane patients. The acuity of this special sense is often markedly increased in persons following certain occupations and in blind patients.

THE OPTIC NERVE

The optic nerve and retina have been aptly called an accessory lobe of the brain. The visual fibers of the optic nerve take origin from centers upon the mesial aspect of the occipital lobe of the cerebrum in the region of the calcarine fissure and the cuneus on either side. These are the higher centers of vision. From these centers the right and left optic radiations respectively pass forward and form synapses with fibers terminating in the external geniculate bodies and the corpora quadrigemina of the two sides of the brain. From these centers fibers arise which form the optic tract, a band of fibers which courses around the crura cerebri on either side to meet anteriorly and form the optic chiasm, where a partial decussation of the fibers occurs, the right optic tract distributing visual fibers to the right half of each retina,

and the left tract supplying similar fibers to the left half of each retina. Lesions involving different portions of these tracts produce characteristic lesions which aid in localizing the individual lesion.

The light fibers, the fibers of the optic nerve and retina which react to light stimuli, arise in the retina, whence they pass backward in the optic nerve, undergoing partial decussation at the chiasm, and proceed along the optic tracts to the external geniculate bodies and corpora quadrigemina, whence they pass to the oculomotor nucleus beneath the floor of the aqueduct of Sylvius by way of the fasciculus sublongitudinalis. Thus the reflex arc of the light reflex is composed of an afferent limb, a substation in the midbrain, and an efferent limb, which will be considered in detail under the examination of the third cranial nerve.

Vision.—The acuity of vision normally is tested with the ordinary Snellen Test Type.

Amblyopia.—Amblyopia, dimness of vision, which is not due to errors of refraction, may result from the excessive use of tobacco or alcohol. Amblyopia may also arise during diabetes mellitus, or it may signify impending uremia in a nephritic patient. The ingestion of certain drugs, as quinine or the salicylates, may induce amblyopia.

Hemeralopia (Day-Blindness).—Hemeralopia, a condition in which the vision is impaired during the day, but improves on dark days or at night, is often part-and-parcel of tobacco amblyopia. It may also signify chronic optic neuritis from intracranial causes or intoxications, or chronic retinitis from a similar cause.

Nyctalopia (Night-Blindness).—Nyctalopia, characterized by imperfect vision in subdued light, is often the result of frequently repeated exposures to strong illumination. In other instances it is a congenital defect of the visual apparatus.

Color Vision (Color-Blindness).—The inability to differentiate between differences in the gradation of colored fabrics is in most instances an inherited defect. Acquired color-blindness occurs, however, as the result of toxic amblyopia, optic neuritis, or as a rare result of trauma to the cranium. In testing for color-blindness, the Holmgren or Thomson test should be employed.

Holmgren Test.—In applying this test the patient is given a skein of wool of a light-pink color and directed to select from a mass of similar skeins of various colors and shades of colors all those which nearly match the color of the selected test skein.

If the color vision is impaired, skeins of varying colors, gray, green, pink, and brown, will be selected indiscriminately. If the subject fails on the pink skein, a pure green skein is selected for a control.

Thomson's Test.—In this test a stick to which numerous bundles of yarn of various colors are attached is employed. The colors have corresponding numbers, the odd numbers being green and the even numbers corresponding to the confusing colors. The color vision is tested with a pale green test skein, the patient being required to match it with ten tints on the rod. The selection of skeins with even numbers reveals the patient's inability to discriminate between the different shades and colors. A control test should be made with red and old-rose skeins as test colors.

Field of Vision.—The dimensions of the field of vision in each eye is best determined by a perimeter; but as this instrument is usually not available, other methods of testing the field of vision must be employed. A rough but sufficiently accurate estimation of the size of the field of vision may be made by the following simple procedure.

The patient is seated in a straight chair with his back toward the source of illumination, the examiner occupying a chair facing the patient, and approximately three feet from him. In testing the left eye, the right eye of the patient is covered with a bandage; the examiner closes his right eye, at the same time fixing his left eye upon the pupil of the left eye of the patient. The examiner, beginning well beyond the limits of vision for both patient and himself, slowly moves his hand inward until the patient first sees the finger-tips. This maneuver is repeated in all the meridians of the visual field; and if the finger-tips become visible to the patient at the same instant they are apprehended by an examiner with a normal visual field, the patient's visual field is of normal extent; that is, is not contracted. If, on the contrary, the hand of the examiner must be brought nearer the visual axis than is required for the normal examiner, the visual field of the patient is contracted.

Contractions of the visual field may be *concentric* or *irregular*. Concentric contraction is noted in many cases of hysteria, and also in glaucoma. Irregular or asymmetric contractions, represented by scotomata and hemianopia possess a varied significance.

Scotomata are to be detected by passing small pieces of white and colored cardboard across the axis of vision while the patient

fixes the eye under examination upon a designated objective point. Under these circumstances the patient is directed to state the point in the progress of the cardboard at which it becomes temporarily invisible. It is to be remembered that there is a physiological scotoma for light and color, corresponding to the blind spot of Mariotte, which must be eliminated in ocular examinations. An *absolute scotoma,* betrayed by the inability of the patient to recognize in the scotomatous field a white cardboard or light stimuli, is significant of grave destructive lesions, as optic neuritis or a lesion involving some portion of the optic tract. A *relative or color scotoma,* revealed by the inability of the subject to appreciate red and green cards in certain portions of the visual field, is usually the result of the excessive use of tobacco or alcohol, and gives a distinctly better prognosis than do the absolute scotomata.

Hemianopia, obliteration or darkening of one-half of the visual field, is tested for clinically by the maneuver used for determining variations in the extent of the visual field; which, in the presence of hemianopia reveals a darkening of one-half of the visual field.

Hemianopia may be horizontal or vertical, homonymous or heteronymous, bi-temporal, bi-nasal or mixed, as the case may prove.

The significance of hemianopia is a lesion involving the optic nerves, optic chiasm, or optic tract; and the site of the intracranial lesion is determined by the distribution of the hemianopic changes. In homonymous hemianopia the corresponding halves of the visual fields are obliterated; as, for instance, the temporal half of the right retina and the nasal half of the left retina. Such an ocular finding constitutes right lateral homonymous hemianopia, the significance of which is a lesion involving the right optic tract alone. Similarly a bi-temporal hemianopia signifies a lesion involving the central portion of the chiasm, whereas a bi-nasal hemianopia is produced by lesions at both extremities of the chiasm, but sparing the central portion of this structure, a condition which rarely occurs. Transitory hemianopia sometimes occurs with hysteria and migraine without anatomic change in the tract.

Wernicke's Pupillary Reaction.—If, in a case of hemianopia, with the patient seated in a darkened room, a thin ray of light from an ophthalmoscopic mirror is projected into the orbit upon the hemianopic retinal area at an angle of 40 to 60 degrees from the visual axis, myosis may or may not result. In hemianopias

in which the causative lesion is situated in the optic tract anterior to the corpora quadrigemina no pupillary reaction will occur, as the reflex arc for the light reflex is broken; but if the lesion is situated posterior to the corpora quadrigemina, the myosis occurs, as the reflex arc in this instance is not disturbed. This test, depending partially on the action of the third cranial nerve, is employed to further localize lesions productive of hemianopia.

THE THIRD, FOURTH, AND SIXTH CRANIAL NERVES

These nerves, which control the pupillary reactions, and the movements of the ocular muscles, are more profitably examined in unison than singly and individually. All three nerves arise from nuclei situated beneath the floor of the fourth ventricle and the aqueduct of Sylvius. The third cranial nerve (oculomotor) supplies fibers to the sphincter of the pupil and all of the ocular muscles except the external rectus and the superior oblique. The fourth cranial nerve (trochlear) supplies the superior oblique muscle of the eye. The sixth cranial nerve (abducent) supplies the external rectus muscle of the eye.

Pupillary Reflexes.—*Light Reflex.*—The normal pupil when exposed suddenly to light stimuli responds by a reflex contraction of the iris. The light reflex may be elicited by shading the eyes with the hands whereupon, on suddenly uncovering one eye the pupillary contraction may be noted. This method of examination, however, is apt to prove fallacious, inasmuch as a reaction to accommodation is likely to be mistaken for a normal light reflex. This source of error may be avoided by throwing a beam of light from an ophthalmoscopic mirror upon the shaded eye, or by testing similarly with the illumination from a small electric flashlight. In the absence of these instruments, the reflex may be elicited by exposing the pupil to the light of a burning match.

The reflex arc involved in the light reflex consists of an afferent limb consisting of the optic nerve and tract, the corpora quadrigemina and fasciculus sublongitudinalis, a station represented by the third nerve neucleus, and an efferent limb comprising the third cranial nerve, the ciliary ganglion and ciliary nerves to the sphincter pupillæ.

A sluggish reaction to light or total abolition of the light reflex signifies optic atrophy, partial or complete paralysis of the third cranial nerve, or degenerative changes in the ciliary ganglion. It may signify compression of the optic tract or the fas-

ciculus sublongitudinalis, which forms the connecting link between the corpora quadrigemina and the third nerve nucleus.

Consensual Light Reflex.—If during the examination for the light reflex in one pupil, the pupil of the opposite eye is observed, while shaded and protected from the light stimuli applied to the opposite retina, it will be observed to react along with the pupil of the exposed eye. This phenomenon constitutes the consensual light reflex, and is due to the transmission of an impulse across the fibers which connect the two third nerve nuclei.

Reaction to Accommodation.—When the range of vision is suddenly transferred from a distant objective point to an object near at hand, the pupils will be observed to contract and the eyes to converge, the reaction to accommodation. This reaction may be quickly tested by directing the patient to fix the gaze on a distant portion of the room, and then quickly to transfer the gaze to the finger of the examiner held near the face of the patient. Abolition of this reflex is due to third nerve paralysis.

Argyll-Robertson Pupil.—Abolition of the light reflex in one or both eyes with retention of the reaction to accommodation constitutes the Argyll-Robertson pupil, which is occasionally found in disseminated sclerosis, and very frequently in tabes dorsalis and paresis. Marina has shown this type of pupillary reaction to be caused by degenerative changes in the ciliary ganglion. In this pupil the pupillary margins are very frequently irregular, while the pupils are often somewhat myopic (spinal myosis) from disease of the cervical cord. Ultimately in tabes and paresis the pupil becomes immovably fixed, reacting neither to light nor to accommodation.

An opposite pupillary reaction, the pupil reacting to light, but failing to react to accommodation is often seen as a sequence of post-diphtheritic paralysis.

Hippus.—Rapid, rhythmic, clonic contractions of the sphincter pupillæ producing winking movements of the iris which are so gross as to be visible to the unaided eye (hippus) are frequently demonstrable in disseminated sclerosis, more rarely in hysteria, incipient acute meningitis, and epilepsy.

Pupillary Unrest.—This phenomenon, which is a normal physical finding, consisting of a regular narrowing and widening of the pupil, is so fine that it can only be demonstrated by means of the aid of a magnifying lens with the pupil brilliantly illuminated. Abolition of this normal pupillary unrest is one of the earliest signs of tabes dorsalis and paresis.

Myosis.—Contraction of the pupil may result from irritative or destructive lesions. Irritative myosis is noted in the early stages of cerebral hemorrhage, in incipient brain tumors before sufficient pressure has been exerted upon the third nerve to cause paralysis, and in early acute meningitis and encephalitis. Paralytic myosis is seen in tabes dorsalis, the late stages of tabes of the cervical cord, and syringolyelia of this portion of the cord, leading to destruction of the pupil-dilating fibers.

Mydriasis.—Irritative mydriasis is often due to irritation of the pupil-dilating center in the cervical cord from congestion, spinal meningitis, or tumor. Paralytic mydriasis may signify paralysis of the sphincter pupillæ, caused by disease of the third cranial nerve or ciliary ganglion, increased intracraneal pressure from brain tumor, or glaucoma.

Strabismus (Squint).—In paralysis of one or more ocular muscles the normal axis of the eyeball deviates from its normal position, with the production of double vision or diplopia. A simple rule in the differentiation of the various ocular paralyses is that the affected eye is displaced by the unopposed antagonists to the side opposite to the usual traction of the paralyzed muscle, while the false image, the result of diplopia, is displaced in the direction of the line of traction of the paralyzed muscle (Purves Stewart).

Nystagmus.—Nystagmus is a rapid oscillation of the globe of the eye upon voluntary motion, usually in a horizontal direction, more rarely in a vertical direction, and very rarely it is rotary. It is a sign of value in disseminated sclerosis, epilepsy, chorea, brain tumor, tabes dorsalis, Friedreich's ataxia, and in some cases of chorea. Nystagmus may be the result of errors of refraction and may be noted in albinos. Miners are subject to a form of nystagmus, probably caused by the constant excursion of the eyes while working in the recumbent or stooping posture.

Aural nystagmus, which may be produced experimentally by syringing the membrana tympani with water either above or below the temperature of the body, is regarded by Barany as the result of convection currents produced in the endolymph by the warming and cooling of the labyrinth. This "thermic nystagmus" is of value in testing the integrity of the vestibular nerve.

Conjugate Deviation.—This comprises a concomitant deviation of both eyes toward the right or left, its significance being a lesion in the cerebral cortex, corona radiata, or internal capsule, above the crossing of the motor fibers. Thus, in cerebral hemorrhage

the eyes are turned toward the side of the lesion and opposite to the side of the paralysis (Prevost's sign). In interpreting the sign it is to be remembered that the lateral movements of the eyes are governed by impulses arising in the cerebral cortex and passing by way of the corona radiata and internal capsule to the sixth nerve nucleus of the corresponding side, and thence across the posterior longitudinal fasciculus to the subdivision of the opposite oculomotor nerve nucleus which presides over the internal rectus muscle. Thus the conjugate lateral deviation of the eyes is caused by the simultaneous stimulation of the external rectus muscle on the side of the lesion and of the internal rectus muscle on the side opposite to the lesion, causing the patient to "look at his lesion."

Ptosis.—Ptosis of the upper eyelid is revealed by the inability of the patient to elevate the lid. It is due to a lesion of the oculomotor nerve or nucleus. Isolated paralysis of the fourth cranial nerve is very rarely encountered, as this nerve usually participates in the palsies of the third and sixth nerves. In the rare instances of simple trochlear paralysis there is inability to rotate the globe downward and outward. Cerebral syphilis is the usual cause of the paralysis.

Abducent Paralysis.—Isolated paralysis of the sixth cranial or abducent nerve is revealed by the inability of the patient to rotate the eyeball outward beyond the midpoint. Upon endeavoring to follow the finger of the examiner the external rotation of the globe is interrupted at this point.

TRIGEMINAL NERVE

The trigeminal nerve has an extensive origin from the floor of the fourth ventricle, beneath the aqueductus sylvii, and the cervical spinal cord as low as the second cervical nerve. The fifth cranial is a mixed nerve, containing both motor and sensory fibers. The fibers constituting the sensory trunk have developed upon them the Gasserian ganglion which rests in a small fossa upon the petrous portion of the temporal bone. The motor root of the nerve supplies the masseters, the temporals, pterygoids, internal and external, mylohyoid, anterior belly of the digastric, the levator and tensor palati and tympani; and the azygos uvulæ. The sensory trunk and Gasserian ganglion terminate in three trunks, the superior and inferior maxillary, and the ophthalmic, which distribute sensory fibers to the anterior two-thirds of the

tongue, the mucous membrane of the buccal and nasal cavities, the salivary glands and teeth, the infraorbital and mandibular portions of the face, and the anterior portion of the scalp.

Motor Paralysis.—Motor paralysis of the fifth cranial nerve is tested for by palpating the masseter and temporal muscles while the patient is directed to clench the teeth. In uni-lateral paralysis there is loss of the prominence with which the muscles stand out on the normal side. The patient is then directed to open the mouth and protrude the lower jaw. In uni-lateral paralysis the jaw is deviated toward the paralyzed side by the action of the sound external pterygoid muscle.

Irritative lesions of the motor trunk or centers produces *trismus,* a mild form of tetanic spasm of the muscles of the lower jaw. Severe tetanic spasm of these muscles accompanies tetanus and strychnine poisoning.

Sensory Paralysis.—Sensory paralysis involving the inferior maxillary division of the nerve produces anesthesia of the infraorbital region, which is tested for by drawing lightly across the face a small pledget of cotton loosely rolled or a camel's hair brush.

Implication of the sensory fibers and the Gasserian ganglion are recognized by the very painful spasm, tic douloureux.

In testing the sense of taste over the anterior two-thirds of the tongue the patient is directed to protrude the tongue and the examiner places on it various substances, such as quinine, sugar, salt, and citric acid, in powdered form. While the tongue is protruded the patient is required to point out on a printed card whether the sensation appreciated is sweet, sour, bitter, salty, or negative. The patient should not be allowed to make his decision after the tongue has been returned to the oral cavity as the flavors may be carried by the saliva to the posterior portion of the tongue which is supplied by the glossopharyngeal nerve.

THE FACIAL NERVE

The nucleus, or origin, of the facial nerve lies in the lower portion of the pons near the medullary junction, the root fibers of the nerve emerging at the lower border of the pons just internal to the point of emergence of the auditory nerve. In company with the auditory nerve, the facial nerve enters the internal auditory meatus of the temporal bone, transverses the aqueductus fallopii of that bone, and emerges from the stylo-mastoid foramen. In

the aqueductus fallopii the nerve receives the chorda tympani, which contains taste fibers from the anterior portion of the tongue. After emerging from the stylomastoid foramen the nerve divides

Fig. 293.—Facial paralysis.

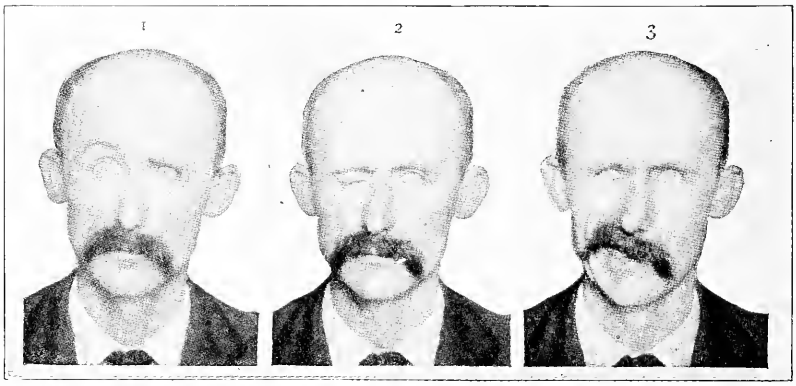


Fig. 294.—Facial paralysis. (Church.) *1*, bilateral attempt to raise eyebrows; *2*, bilateral attempt to close eyes; *3*, smiling. (From Eisendrath.)

into a number of diverging branches to supply the majority of the muscles of the head and face.

Facial Paralysis.—The facial nerve is purely a motor nerve, except for the fibers it receives from the chorda tympani, destructive

lesions in its center, or origin, or along its course through the aqueduct producing facial paralysis. In this form of paralysis the normal flexion folds disappear from the affected side of the face, the patient is unable to close the eye, which remains open and staring, is unable to whistle or smile, the angle of the mouth droops on the paralyzed side, while the opposite angle is drawn toward the healthy side. These changes constitute the typical Bell's palsy, which is due to a lesion of the nerve after its exit from the stylomastoid foramen, and which is often due to exposure to cold.

If the lesion be situated in the aqueductus fallopii, in which situation the nerve is very susceptible to pressure from disease of adjacent structures, in addition to the signs of uni-lateral facial paralysis, the sense of taste is abolished over the distribution of the chorda tympani.

If the lesion involves the nucleus of origin of the nerve, or the root fibers or trunk prior to its entry into the internal auditory meatus, there is usually in addition to the other signs, hyperacuteness of hearing due to paralysis of the stapedius muscle, which receives a branch of supply from the facial nerve as it traverses the aqueduct.

In the case of a supranuclear lesion, a lesion involving the corona radiata and affecting only the supranuclear fibers, the main evidences of paralysis will be seen over the lower portion of the face, the muscles of the upper portion being affected to a minor degree, owing to the fact that the muscles of this upper region always act in unison and derive a nerve supply from both cerebral hemispheres.

THE AUDITORY NERVE

The auditory nerve is composed of two distinctly differentiated sets of fibers: (1) cochlear fibers, which subserve the function of audition; and (2) vestibular fibers, which supply the semicircular canals and preside over equilibrium.

Deafness.—Impairment of the function of audition may be the result of local disease of the middle ear or of disease of the cochlear division of the auditory nerve. The acuity of hearing in the two ears may be determined by means of a watch, each ear being alternately occluded while the opposite ear is under examination, or by means of the vibrations of a tuning fork. In differentiating between middle ear deafness and nerve deafness

the tuning fork should be applied to the midline of the forehead. Under these conditions, if the cause of the deafness lies in middle ear disease, the vibrations are most clearly audible in the diseased ear; whereas if it is a case of nerve deafness due to a lesion of the eighth nerve, the vibrations are audible only on the side of the sound ear.

Tinnitus.—Tinnitus aurium, or ringing in the ears, occurs with intracranial tumors and aneurism, temporary obstruction of the eustachian tube during acute colds, and during disease of the labyrinth.

Vertigo.—Vertigo, or dizziness, when not of gastro-intestinal origin, signifies a cerebral or intracranial lesion such as tumor or aneurism acting upon the cerebellar centers of coordination or the afferent paths of the vestibular division of the auditory nerve. Tumors of the cerebellum are characterized by extreme vertigo and incoordination. Vertigo may be due to Meniere's disease or aural vertigo.

The Barany Tests

The tests of Robert Barany, which are based upon the experimental work of Flourens and Ewald, afford a valuable means of diagnosing and localizing intracranial lesions involving the integrity of the vestibular division of the auditory nerve. The causative lesion may operate upon the labyrinth, as in acute or chronic suppuration in this apparatus; upon the cerebellum, as in cerebellar abcess or tumor; upon the nucleus or origin or upon the trunk of the vestibular division of the auditory nerve.

The tests consist in observing the character of the nystagmus, the manner in which the patient performs certain pointing tests, and the tendency on the part of the patient to fall in certain directions, (1) in the absence of external stimulation or depression of the labyrinth; (2) when the labyrinth has been stimulated or depressed by the application of water at temperatures above or below that of the body or by the electric current; and (3) after the patient has been rapidly rotated in a revolving chair either toward the right or toward the left.

The technic of the Barany tests has been variously modified, chiefly by Barany himself, to meet different clinical needs, the principles underlying the tests in all cases being the same. A very satisfactory routine method of examination is the following:

First Test.—*Direct Objective Examination.*—Without apply-

ing any stimulus to the vestibular apparatus the nystagmus is noted and its character recorded. The nystagmus which is encountered may be of labyrinthine or cerebellar origin. Labyrinthine nystagmus comprises two components: a quick snap of the globe in one direction, which is followed by a slower return to the resting position. Cerebellar nystagmus consists of two components, which are almost equal in time, but the primary movement is slightly more rapid than is the secondary return to the resting state. In recording nystagmus the quick motion is always recorded.

In the interpretation of nystagmic movements, it is to be borne in mind that the direction of the nystagmus in any given case conforms to the law of Flourens and Ewald that nystagmus arising from excitation of a single semicircular canal occurs only in the plane of that canal; that the relation between the movement of the endolymph in any canal and the direction of the consequent nystagmus is definite and constant; and that reversal of the direction of the movement of the endolymph causes a reversal of the direction of the nystagmus. Thus by a study of the nystagmic movements the extent of a labyrinthine lesion may be determined. Vestibular nystagmus may be horizontal, vertical, oblique, or rotary; thus, since irritation of a single semicircular canal can produce nystagmus only in the plane of the canal, and as the nystagmus caused by acute disease of the labyrinth does not occur in one direction, but in various directions without corresponding exactly to the plane of any canal, we assume that all of the canals are involved in the acute suppurative process.

Having studied the nystagmus occurring without external stimulation of the labyrinth, the patient is directed to stand or is supported, with the eyes closed and the feet close together, and any tendency to fall and the direction of the fall, is recorded. According to the rule of Barany, "a person exhibiting vestibular nystagmus tends to move within the plane of the nystagmus, and to fall in the direction opposite to the quick nystagmic movement."

The patient is next required to place the tip of the index finger in contact with the index finger of the examiner, to close his eyes and lower the arm to the side, and to replace his finger in contact with that of the examiner, the pointing test. Whereas a normal person can perform this test with a fair degree of accuracy, a patient with labyrinthine or cerebellar disease is liable

to err in the direction of his lesion. The results of the first test in the presence of labyrinthine and cerebellar disease, as well as in the normal patient, are tabulated in the appended table, which has been compiled by Dr. C. E. Shinkle.

Second Test.—*The Caloric Tests.*—The caloric or thermic tests consist in alternately stimulating and depressing the labyrinthine apparatus by irrigating the external auditory canal with water above and below the temperature of the body. The tests are applied to the supposed side of the disease and to the supposedly normal side on alternate days to obviate the lingering effects of a former test. Instead of employing water, the same result may be obtained by the use of a galvanic battery, the anode being used for depression and the cathode for stimulation. In using the galvanic battery the patient retains one electrode in the hand while the other is applied to the mastoid process, or the wall of the external auditory canal.

The Cold Water Test—It has long been known that irrigation of the external auditory canal with water at the temperature of the body rarely produces discomfort, whereas irrigation with water at higher or lower temperatures produces nystagmus and often violent vertigo and vomiting. In the application of the cold water test the external auditory canal is irrigated with water below the body temperature, usually at a temperature of 86° F. In the presence of a congested tympanic membrane, however, water of a lower temperature is required to produce a reaction. The water should be directed into the canal with but moderate force, preferably from a fountain syringe or a small glass funnel attached to a piece of catheter tubing.

The Warm Water Test.—In this instance the external auditory canal is irrigated in a similar manner with water slightly above the body temperature, each meatus being tested on alternate days. The technic is the same as for the cold water test.

The physical basis underlying the caloric tests consists in the establishment of convection currents in the endolymph under the influence of the cool or warm external applications. In the production of caloric nystagmus the horizontal and anterior vertical canals are principally influenced, as they are situated in close proximity behind the median tympanic wall, and are hence easily influenced by external thermic influences; whereas the posterior vertical canal, which is situated medially and internally to them, is less exposed to such stimuli. When, under the application of the cold and hot water tests, the specific gravity of the

endolymph is raised or lowered, a relative change in the position of the contained endolymph of the semicircular canals is induced. Naturally, the principal direction of the shifting of the endolymph, depending on variations in its specific gravity, is upward and downward. Thus, with the head erect, during the application of the cold water test, the cooling of the endolymph in the horizontal semicircular canal, which is first exposed to the action of the external stimulus, does not lead to any endolymphatic movement, as such movement is prevented by the horizontal postion of the canal. But, in the case of the anterior vertical canal, the outer or ampullary end, which points directly downward, is exposed to the cool water and a downward movement of the endolymph toward and through its ampulla is induced, leading to a rotary nystagmus toward the opposite side.

If now the position of the head is reversed, the head being inclined directly downward toward the floor, immediately after the application of the cold water test, producing in this manner a reversal of the current of the endolymph, the direction of the rotary nystagmus is naturally reversed, the nystagmus in this instance occurring toward the side of the ear which is syringed. Moreover, if the head immediately after irrigation of the external auditory canal is bent forward so that the face looks directly toward the floor, the nystagmus produced is entirely horizontal, for the reason that in this position of the head the chief endolymphatic movement occurs in the horizontal canal with a consequent purely horizontal nystagmus.

The usual reactions to the caloric tests in labyrinthine disease, cerebellar disease, and in the normal person, are tabulated in the annexed table.

Third Test.—*The Rotation Tests.*—In these tests, the patient, seated on a revolving chair, is turned rapidly toward the right or left, or from and toward the side of the suspected lesion. Ten revolutions of the chair, which should consume about twenty seconds, are best adapted to elicit the reaction. At the completion of the last turn the patient and chair are abruptly stopped. When we speak of turning the patient from left to right, we mean in the direction from the tip of the patient's nose to his right ear and vice versa.

When a patient is thus rotated—let us say toward the right—there is developed a nystagmus (primary nystagmus) in the direction in which he is turned; namely, toward the right. When

the patient is suddenly stopped, a nystagmus develops in the opposite direction (after-nystagmus), the average duration of which is about forty seconds in a normal person. While turning the patient toward the right the quick component of the nystagmus produced is toward the right, and vice versa. This nystagmus is increased in intensity if the eyes are voluntarily turned toward the quick component, and is diminished or abolished when the eyes are turned toward the slow component of the nystagmus. For this reason Barany advises the use of smoked spectacles, which render fixation of vision in any direction impossible, while testing for horizontal nystagmus by rotation tests.

The physical basis underlying this horizontal nystagmus upon rotation of the patient with the head erect lies in the movements which are generated in the endolymph of the two horizontal semicircular canals. Thus, when the patient is rapidly rotated in a horizontal plane—let us say toward the right—the primary movement of the endolymph in the right horizontal canal is toward its ampulla, whereas in the left canal it is toward the small end of the canal, the very movements which according to the work of Flourens and Ewald are calculated to produce horizontal nystagmus toward the right. When, on the other hand, the rotation of the patient is suddenly terminated, the endolymph by virtue of its momentum is displaced in the opposite direction, with a resulting reversal in the direction of the nystagmus.

It follows naturally that by altering the position of the head during rotation different canals may be brought under investigation and the integrity of the several elements in the labyrinthine system can be tested by variations in the nystagmus produced.

It is very important to compare the relative duration of the after-nystagmus obtained by rotation in opposite directions. As has been stated, the average duration of this nystagmus after ten turns is about forty seconds. While a difference of three or five seconds between the records of after-nystagmus following alternate rotations to right and left has little significance, any discrepancy above this figure is evidence of vestibular impairment.

Having determined the nature and degree of the nystagmic movements as influenced by the rotation tests, the pointing tests and standing tests should be examined just subsequent to rotation of the patient, the significance of the variations being recorded in the accompanying table.

TABLE SHOWING CLINICAL FACTS UPON WHICH EQUILIBRIUM TESTS ARE BASED*

		In Labyrinthine Disease	*In Cerebellar Disease*	*Normal Individual*
Direct Examination of the Patient	Falls	Toward the affected ear. Direction can be changed by rotating head on shoulders.	Either way; most often toward the side of lesion.	Does not fall.
	Nystagmus	Jerky toward side opposite to that of the lesion.	Steady; hard to determine direction; most marked away from the side of the lesion.	No nystagmus.
	Points	Towards the side of the lesion with either hand.	Toward the side of lesion with hand on that side; may point normally with other hand.	Points normally.
After irrigating the ear with cold water (or applying the anode of a galvanic battery to same) — On the side of the lesion	Falls	Toward the affected internal ear no matter how head is placed.	Any way; most likely toward side of the lesion.	Toward the ear tested.
	Nystagmus	Jerky away from the side of the lesion.	Steady; most marked away from side of the lesion or may be jerky away from the side of the lesion.	Away from the side of the ear tested, jerky.
	Points	Toward the side of the lesion with either hand.	Points to side of lesion with hand on side of lesion; hand on well side shows no change or points to side of lesion.	Toward the side of the ear tested with either hand.
After irrigating the ear with cold water (or applying the anode of a galvanic battery to same) — On the side opposite to the lesion	Falls	Either way; most likely to side of the lesion.	Away from the side of the lesion.	Toward the ear tested.
	Nystagmus	Jerky, both directions; most marked away from side of lesion.	Jerky toward the side of the lesion.	Away from the side of the ear tested, jerky.
	Points	Either way with either hand, most likely towards the side of the lesion with either hand.	Toward the side of lesion with hand on that side; away from side of lesion with other hand.	Toward the side of the ear tested with either hand.

*Courtesy of Dr. C. E. Shinkle.

TABLE SHOWING CLINICAL FACTS UPON WHICH EQUILIBRIUM TESTS ARE BASED*
(Continued.)

			In Labyrinthine Disease	*In Cerebellar Disease*	*Normal Individual*
After irrigating the ear with hot water (or applying the cathode of a galvanic battery to same)	On the side of the lesion	Falls	Toward the affected internal ear most often.	Any way; most often away from the side of lesion.	Away from the ear tested.
		Nystagmus	Jerky toward side opposite to that of the lesion.	Steady; perhaps jerky towards the side of lesion.	Toward the side of the ear tested, jerky.
		Points	Confused; most often toward the side of lesion with either hand.	Toward the side of lesion with hand on side of the lesion; other hand does not vary or points away from lesion.	Away from the side of the ear tested with either hand.
	On the side opposite to the lesion	Falls	Toward the affected internal ear, no matter how head is placed.	Toward the side of lesion.	Away from side of ear tested.
		Nystagmus	Jerky toward side opposite to that of the lesion.	Jerky toward the side opposite to that of the lesion.	Toward the side of ear tested, jerky.
		Points	Toward the side of the lesion with either hand.	Toward the side of lesion with either hand.	Away from the side of the ear tested with either hand.
After Rotating the Patient	Toward the side of the lesion	Falls	Toward the side of the lesion.	Toward the side of lesion.	In same direction as that of rotation.
		Nystagmus	Jerky away from side of lesion.	Jerky away from the side of the lesion.	Jerky in direction opposite to that of rotation.
		Points	Toward side of lesion with either hand.	Toward the side of lesion with either hand.	In same direction as that of rotation.
	Away from the side of the lesion	Falls	Toward the side of the lesion.	Either way; most likely away from side of lesion.	In same direction as that of rotation.
		Nystagmus	Jerky away from the side of the lesion.	Steady or perhaps jerky; if the latter, direction is toward the side of lesion.	Jerky in direction opposite to that of rotation.
		Points	Towards the side of the lesion with either hand.	Toward the lesion with hand on side of lesion; away from lesion with other hand, if any change at all.	In same direction as that of rotation.

*Courtesy of Dr. C. E. Shinkle.

THE GLOSSO-PHARYNGEAL NERVE

The glosso-pharyngeal nerve supplies sensory fibers to the mucous membrane of the pharynx and for the posterior third of the tongue. It is also the motor nerve of the middle constrictor of the pharynx and the stylo-pharyngeus.

Paralysis of the nerve is evidenced by loss of taste sensation for the posterior third of the tongue and abolition of the pharyngeal reflex. Lesions of the nucleus of the nerve do not affect the sensation of taste owing to communications of the taste fibers with the trigeminal nerve.

THE PNEUMO-GASTRIC NERVE

The pneumo-gastric, or vagus nerve, arises from a nucleus beneath the floor of the fourth ventricle along with the nucleus of origin of the glosso-pharyngeal nerve. The nerve has a very extensive distribution, supplying motor fibers to the palate, pharynx, and larynx. It also sends fibers to the esophagus, stomach, heart, lungs, and through the sympathetic system to the intestines and spleen.

Paralysis of the pneumo-gastric nerve produces uni-lateral paralysis of the palate. This paralysis is demonstrated by observing the excursion of the palate while the patient pronounces a syllable, such as the word "Ah," when only half of the palate rises in the normal manner. In a patient with palatal paralysis fluids which are ingested have a tendency to regurgitate through the nose. The speech is impaired, assuming a nasal quality, owing to impairment of the innervation of the vocal cords. In unilateral recurrent laryngeal paralysis phonation is impaired but not abolished; but in bi-lateral recurrent laryngeal paralysis phonation becomes impossible.

THE SPINAL ACCESSORY NERVE

The spinal accessory nerve consists of two divisions: (1) the spinal, which arises from the anterior horns of the cervical cord as low as the fifth cervical nerve; and (2) the accessory, which arises from a nucleus situated near that for the pneumo-gastric nerve. The fibers of the spinal division of the nerve are distributed to the sternomastoid and trapezius muscles, while the

fibers arising from the accessory nucleus are distributed to the pharyngeal and superior laryngeal nerves.

In testing the spinal portion of the nerve the patient is directed to rotate the head and to shrug the shoulders. In paralysis of this division of the nerve paralysis of the sterno-mastoid on the corresponding side causes difficulty in turning the head toward the sound side. Paralysis of the trapezius muscle is revealed by inability to shrug the shoulder.

THE HYPOGLOSSAL NERVE

The hypoglossal nerve takes origin from a center in the lower portion of the floor of the fourth ventricle. The nerve trunk emerges in a series of fascicles in the interval between the anterior pyramid and the olivary body.

The hypoglossal nerve supplies motor fibers to the tongue and sends motor fibers to all the muscles attached to the hyoid bone with the exception of the digastric, middle constrictor of the pharynx, mylohyoid and stylohyoid.

Uni-lateral hypoglossal paralysis is demonstrated by directing the patient to protrude the tongue, when it will be observed to deviate toward the paralyzed side. There is or is not atrophy depending on whether the lesion causing the paralysis is supranuclear or infranuclear.

In bi-lateral hypoglossal paraylsis the patient is unable to protrude the tongue.

APPENDIX

CASE HISTORY

Case Number_____ Date_____

PERSONAL DATA

Name _____ Address _____
Sex _____ Age _____
Occupation _____ Nativity _____
Civil state _____ Number of children_____
Pathologic labors _____ Miscarriages _____

FAMILY HISTORY

Health of parents_____ Age and cause of death_____
Health and age of sisters_____
Health and age of brothers_____
Age and cause of death of sisters_____
Age and cause of death of brothers_____
Special history of tuberculosis, syphilis, epilepsy, insanity, gout, and diabetes in family _____

PREVIOUS HISTORY

User of tobacco, alcohol or narcotic drugs_____
When and for what period_____ Dates of treatment for same_____
Venereal disease (gonorrhea and syphilis)_____
_____ Dates of treatment_____
Diseases during childhood (Varicella, scarlatina, mumps, pertussis, diphtheria)
_____
Infectious diseases (Pneumonia, tuberculosis, diphtheria, otitis, cerebro-spinal meningitis, typhoid fever)_____
_____ Dates_____ Complications_____
Former attack of present complaint_____
Date_____ Mode of onset_____
Former injuries_____ Residual deformities_____
Recent loss of weight_____

PRESENT ILLNESS

Date and mode of onset_____ Treatment_____
Evolution of disease to date_____
Subjective symptoms_____
_____

PHYSICAL EXAMINATION

Height_____ft._____in. Weight_____lbs.

RESPIRATORY SYSTEM

Inspection:

Herpeslabialis_____State of alae nasi_____
Cervical veins_____ Contour of chest_____
Clavicles_____ Supra- and infraclavicular fossae_____
_____ Uni-lateral enlargement_____
Uni-lateral retraction_____
Local bulging_____ Local retraction_____
Type of respiratory expansion (Costal or abdominal)_____
Respiration sterterous, stridulous or jerky_____
Dyspnea_____ Inspiratory_____ Expiratory_____
Mixed_____ Cyanosis_____
Cheyne-Stokes breathing_____ Litten's Phenomenon_____
Present_____ Absent_____
Expansion of thorax: General increase_____ General decrease_____ Uni-lateral increase_____
Uni-lateral decrease_____ Wavy breathing_____

Palpation:

Expansion of apices_____ Expansion of bases_____
_____ Antero-posterior expansion_____
Vocal fremitus: Normal_____ Exaggerated_____
Diminished_____ Friction fremitus_____
Rhonchal fremitus_____ Tussile fremitus_____
Succussion fremitus_____ Sense of resistance: Normal _____ Increased_____ Decreased_____
Local tenderness_____ Fluctuation_____
Crepitation_____ Local pulsations_____

Percussion:

Normal limits of resonance_____ Increased resonance: Upward_____ Downward_____ Anteriorly over heart_____
Dullness or impaired resonance_____ Sites_____
Flatness_____ Sites and dimensions_____
Hyper-resonance_____ Skodaic resonance_____
Tympany_____ Signs of cavity or excavation: Cracked-pot sound_____ Wintrich change of sound_____ Interrupted Wintrich change of sound_____ Gerhardt's change of sound_____
_____ Friedreich's change of sound_____
Amphoric resonance_____ Grocco's sign_____
Resistance_____ Increased_____ Decreased_____

Auscultation:

Breath sounds: Vesicular_____ Increased_____ Decreased_____
Bronchial_____ Site_____ Increased_____ Decreased_____
Broncho-vesicular_____ Site_____ Amphoric_____ Site_____

Cavernous_____ Site_____ Puerile_____ Sites_____
Prolonged expiration_____ Cog-wheel breathing_____
Vocal resonance_____ Increased_____ Diminished_____
Bronchophony_____ Pectoriloquy_____ Whispering pectoriloquy_____
_____ Aegophony_____ Absent respiratory and voice sounds
_____ Sites_____ Rales_____ Sibillant_____
Sonorous_____Sites Crepitant_____ Subcrepitant_____
Mucous_____ Sites_____ Pleural friction_____
Metallic tinkle_____ Succussion sound_____
Mensuration_____ Cyrtometry of chest_____
Examination of sputum_____

CIRCULATORY SYSTEM

Inspection:

Precordial bulging_____ Precordial retraction_____
Abnormal pulsations_____ Cardiac impulse: Site_____
Extent_____ Force_____ Displacement_____ Upward_____
Upward and to left_____ to left_____ to left and downward_____
to right_____ Absent or invisible_____
Systolic retraction (Broadbent's sign)_____
Overfullness of jugulars (Kussmaul's sign)_____ Diastolic
collapse of jugulars (Friedreich's sign)_____
Tortuous cutaneous veins_____ Systolic jugular pulsations
_____ Hepatic pulsation_____ Capillary pulse_____

Palpation:

Thrills_____ Sites_____ Time_____
Pericardial friction_____ Valve shock_____
The pulse_____ Condition of artery_____ Rate_____ Rhythm_____
Volume_____ Tension_____ Omission_____ Intermission_____
Bi-lateral symmetry_____ Dicrotism_____

Percussion:

Cardiac borders_____ Increase to right_____ To left_____
To left and downwards_____ General increase_____
Vascular dullness_____ Increased to right_____

Auscultation:

Heart sounds: General accentuation_____ General diminution_____
Accentuation first sound_____ Accentuation second_____
Reduplication first sound_____ Reduplication second sound_____
Endocardiac murmurs_____ Site_____ P.M.I_____
Line of transmission_____ Time_____ Quality_____
Intensity_____ Pericardial friction_____
Pericardial succussion_____ Flint murmur_____
Venous hum_____ Aortic murmurs_____
Examination of the blood: Hemoglobiu_____ Leukocytes: Numerical
_____ Differential_____ Erythrocytes_____ Numerical____
_____ Parasites_____ Polychromatophilia_____
Blood pressure_____ Systolic pressure_____ Diastolic pressure
_____ Pulse pressure_____

THE ABDOMEN

Inspection:

Distended veins_____ Caput Medusae_____ Diastasis_____
Visible peristalsis_____ Tumor_____ Pigmentation_____
Scars_____ Umbilicus_____ Hernia_____

Palpation:

Thickness of wall_____ Tumor of wall_____ Fluid wave_____
_____ Fat wave_____ Muscular rigidity_____
Muscular spasm_____ Tenderness_____ Site_____

SPECIAL ABDOMINAL ORGANS

The Stomach:

Position greater curvature_____ Lesser curvature
_____ Gastrectasis_____ Gastroptosis_____
Visible peristalsis_____ Pyloric tumor_____
Hour-glass constriction_____ Tenderness_____
Succussion _____ Transillumination_____
Gastric contents: Free HCl_____ Combined HCl_____
Lactic Acid_____ Blood_____

The Intestines:

Tenderness_____ Tumor_____ Impacted feces_____
_____ Gaseous distention_____ Visible peristalsis_____
Examination of feces_____ Parasites_____ Ova_____ Blood_____
Color_____ Consistence_____ Undigested food_____

The Pancreas:

Tenderness_____ Tumor_____ Cyst_____
Fat indigestion_____ Cammidge Reaction_____

The Liver and Gall-Bladder:

Enlargement_____ Diminution_____
Ptosis_____ Tenderness_____ Systolic pulsation
_____ Consistence_____ Fluctuation_____
Nodules_____ Reidel's lobe_____ Gall-bladder tumor____
_____ Gall-stone crepitus_____ Peritoneal friction_____

The Spleen:

Enlargement_____ Diminution_____
Displacement_____ Tumor_____ Peritoneal friction_____

The Kidney:

Tenderness_____ Ptosis_____ 1st degree_____
2nd degree_____ 3rd degree_____ Cystic_____ Tumor_____
Urinalysis_____

THE HEAD AND NECK

The Head:

Microcephalic_____ Megalocephalic_____ Rickets_____
Hydrocephalus_____ Cretinism_____ Fontanelles: Depressed_____

Bulging_____ Sutures_____ Craneotabes_____
Condition of hair: Color_____ General loss_____ Localized loss_____
The face: Contour_____ Pallor_____ Cyanosis_____ Jaundice_____
Chloasma_____ Spasm_____ Scars_____ Eruptions_____
The eyes: Edema of lids_____ Exophthalmos_____ Enophthalmos_____
Strabismus_____ Hippus_____ Nystagmus_____ Argyll-Robertson pupil_____ Ocular mobility_____
The Lips: Pallor_____ Cyanosis_____ Epithelioma_____
Chancre_____ Fissures_____ Parted lips_____
The teeth: Premature decay_____ Delayed dentition_____
Hutchinson teeth_____ Sordes_____
The Gums: Spongy_____ Lead line_____ Copper line_____
Red line_____ Epulis_____ Gingivitis_____
The Tonsils: Chronic hypertrophy_____ Tonsillitis_____
Diphtheria_____ Vincent's angina_____
The Ear: Deformity or injury_____ Tophi_____ Discharge_____

The Neck:

Cervical glands_____ Thyroid_____
Tracheal tug_____ Sternomastoids_____ Branchial cysts_____ Torticollis_____ Retraction_____

THE HANDS AND ARMS

Nails: Pallor_____ Cyanosis_____
White spots_____ Incurvation_____ Capillary pulse_____
Fingers: Tophi_____ Nodes_____ Clubbed fingers_____
Enlarged joints_____ Gangrene_____ Manual deformities_____
Tremor_____ Wrist-drop_____ Onychia_____
Forearm: Rickets_____ Pellagra_____ Erythema multiforme_____
Arm: Ruptured biceps_____ Tumors_____ Paralysis_____

THE FOOT AND LOWER EXTREMITY

Gangrene_____ Foot-drop_____
Edema_____ Ulceration_____ Joints_____
Babinski's reflex_____ Knee-jerk_____

THE NERVOUS SYSTEM

Headache_____ Vomiting_____
Paralysis_____ Choked disk_____ Convulsions_____
Disorders of sensation_____ Disturbances of speech_____
Disorders of organs of special sense_____
Pain, tremor_____ Station_____ Gait_____ Reflexes_____
Diagnosis _____
Complications _____
Prognosis _____
Treatment _____
_____
Result _____
*Discharged*_____

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