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DISEASES OF THE HEART AND CIRCULATION

KEATING AND EDWARDS

DISEASES

OF THE

Heart and Circulation

IN

INFANCY AND ADOLESCENCE.

BY

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ILLUSTRATED WITH PHOTOGRAPHS AND WOOD-ENGRAVINGS.

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PREFACE.

THE many excellent text-books on children's diseases have failed to give a satisfactory account of the diseases of the heart, and, indeed, as far as we know, the work now presented by us is the only systematic attempt that has been made to collect in book-form the abundant material which is scattered throughout medical literature in the form of journal articles, clinical lectures, theses, and reports to societies. We have endeavored —though the difficulty of the task has rendered our work, doubtless, in many respects incomplete—to collect these valuable materials and place them within easy reach of those who are interested in this important subject.

This work has been published in monthly instalments in the "Archives of Pediatrics" for the past year; encouraged by its kind reception, we place the matter now in more convenient form, with many changes and additions, and hope for our labor a kind recognition.

We have endeavored to avoid as far as possible all unsettled matters of controversy, and to quote authorities on both sides when necessity made their entrance unavoidable.

To French literature we are especially indebted for many references without which we feel that this work would have been a much more difficult one to write.

PHILADELPHIA, 1888.

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DISEASES

OF THE

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INFANCY AND ADOLESCENCE.

CHAPTER I.

METHODS OF STUDY, INSTRUMENTS, FŒTAL CIRCULATION.

A CORRECT knowledge of the anatomy, physiology, and general topography of the heart is essential to an intelligent study of its diseases.

The heart is a hollow viscus, whose function is to force the blood to all parts of the body. It is enclosed in the fibrous sac of the pericardium, which gives it fixation, as the cardiac apparatus is simply suspended in this sac by the large vessels which go to and arise from its base.

The pericardium is in shape an irregular pyramid, attached by its base to the centre of the diaphragm; tendinous bands indirectly attach it to the vertebral column; aponeurotic fibres

also fix its apex to the sternum, vertebral column, and the hyoid bone.

This pericardial sac is situated in the median line of the thorax, between the two lungs and their covering, the pleuræ. The heart lies obliquely in this membrane, with its long axis downwards and towards the left. Its posterior boundary is the posterior mediastinum, being separated from the vertebræ by the trachea, descending aorta, intercostal artery and veins, greater vena azygos, thoracic duct, lymphatic glands, œsophagus, the two pneumogastrics, and both sympathetic nerves.

Anteriorly the heart and its coverings are separated from the sternum by connective tissue and the borders of the pleuræ. The heart in a child is somewhat higher in the thorax than that of an adult; its situation, based on several careful dissections, is about as follows: The auricles are on a line with the second intercostal space, the right extending beneath the sternum and almost to its right border. The right ventricle is beneath the sternum and to its left; its lower border is on a line with the head of the sixth costal cartilage. The left ventricle is situated between the third and fourth intercostal spaces and beneath the fourth rib. The position of the apex-beat differs materially from that in an adult, as will be seen by the accompanying table, based upon a series of examinations made by us in the Philadelphia Hospital.

The apex is much higher and nearer the nipple than is the case in an adult thorax; in a certain number of cases the nipple pulsates synchronously with the apex-beat; our investigations lead us to locate the apex-beat in the fourth interspace. A higher location of the apex may possibly be accounted for to some extent by the distention of the alimentary canal so often seen in young life, and also from the somewhat relatively larger size of the liver at this period of life. An examination of ninety-six boys for admittance to Girard College, made by one of us, served to further illustrate the fact that the apiceal impingement has a higher location in proportion to the decrease in the age of the child,—that is, in boys of six years it was generally found to be close to the nipple, whereas in boys of ten years its location was found to be from a half to an inch lower. The base of the heart cor-

Topography of the Heart; Apex-Beat.

Name.	Age.	General Characteristics.	Previous Diseases.	Present Condition.	Pulse-Rate.*	Position of Apex-Beat.	Heart-Sounds.	Remarks.
John Putney.	2 years.	Light hair, eyes, and complexion.	None.	Healthy.	145	On nipple line; ¾ in. below.	Clear and healthy.	Stomach and intestines distended.
Agnes Gallagher.	16 mos.	" "	"	Healthy; slight bronchitis.	140	On nipple line; 1 in. below.	" "	Stomach and intestines slightly distended.
Walter Clarke.	15 mos.	Dark hair, eyes, and complexion.	Malnutrition.	Anæmic.	Unable to count; restless.	Just below nipple.	Very valvular.	Nipple pulsates synchronously with apex-beat.
Frank Murry.	16 mos.	Light hair, eyes, and complexion.	Hereditary syphilis; disease of knee-joint; phimosis.	Weak and anæmic; syphilis in abeyance.	136	On nipple line; 1 in. below.	At apex first sound not healthy.	Spleen and liver enlarged.
Joseph Batty.	13 mos.	" "	Scrofula.	Glandular abscess.	160	⅛ in. to right of nipple line; ⅜ in. below.	Healthy (?)	
William Dresser.	13 mos.	" "	Anæmic.	Improved.	142	1 in. below nipple; ⅛ in. to right.	Healthy.	
Mary Jillard.	3 years.	" "	Scrofula.	Scrofula.	140	Immediately below nipple.	Healthy.	Nipple pulsates synchronously with apex-beat.

* The rapidity is in part accounted for by the excitement incident to the examination.

responds posteriorly to about the fifth dorsal vertebra.* Much of the anterior surface of the heart is removed from the chest-

* In confirmation of the above outlines, we quote the following observations, which came to our notice four months after our own investigations (Arch. Pediatrics, June, 1887, p. 364). Wassilewski, "The Position and Limits of the Heart in Children," Arch. f. Kinderh. [abstracted], viii. 3: "The heart has a more horizontal position in children than it has in adults. The apex-beat during the first four years of life is to be felt in either the fourth or fifth left intercostal space, or in both at the same time, from one to two centimetres to the left of the *linea mammillaris*. Of eighteen hundred and twenty children between the day of birth and the twelfth year of life who were examined by the author (only normal hearts being included in the table), in only 0.6 per cent. of the cases was the apex-beat internal to the *linea mammillaris;* in 1.5 per cent. of cases it was upon the *linea mammillaris*, and in the remaining 98 per cent. of cases it was external to it. In 43.3 per cent. the apex-beat was felt in the fourth intercostal space, in 21.5 per cent. in both the fourth and fifth spaces, and in 35 per cent. only in the fifth. The horizontal position of the heart in children is dependent upon the high situation of the diaphragm, and also the relation which the vertical diameter of the heart bears to its transverse, these diameters being the same during the first few years of life. The author agrees with Rauchfuss in attributing to the heart a small (absolute) area of dulness, but relatively a large one. The dimensions of the absolute area of dulness vary in children of the same age, and are especially dependent upon the relation of the borders of the lungs to the heart, and upon the presence or absence of pulmonary emphysema, deviations in the vertebral column, etc. Also, the so-called relatively large area of dulness depends upon the relation of the lungs to the heart, for it may not be possible to define the lower half of the left border of the heart if that portion is covered by a thick layer of lung-tissue. This area also varies in children of the same age, but varies less than the area of absolute dulness. In order to determine the entire superficial extent of the heart—that is, of that portion which lies next the anterior thoracic wall—palpation and palpatory percussion must be practised. Attention is called to the fact that in about half of all children there is more or less pronounced dulness under the left clavicle, and extending almost to the second rib, being more perceptible at the inner than at the outer border of the bone, and also to the presence of moist subcrepitant râles at the same place. This dulness is explained by the presence of a small section of lung-tissue, belonging to the left apex, in the vicinity of the heart. Other conditions which contribute to produce dulness over this area are the presence of the thymus gland, relative narrowness of the left bronchus, and a relatively small volume of the apex of the left lung. This explanation is the more rational since the dulness at the part in question disappears after deep inspirations, and because it becomes more pronounced if the heart becomes enlarged towards the left or assumes a relatively high position. The subcrepitant rhonchi which may be heard in this location are probably associated with atelectasis, and they disappear after deep inspirations have been taken. Similar auscultatory phenomena are also sometimes observed in children whose lungs are quite normal at the apex, in connection with enlargement of the lymphatic glands above the left clavicle, and in febrile conditions associated with rapid and superficial breathing. It is also sometimes observed in children who are entirely healthy, especially after sleeping. Similar physical signs are sometimes observed in connec-

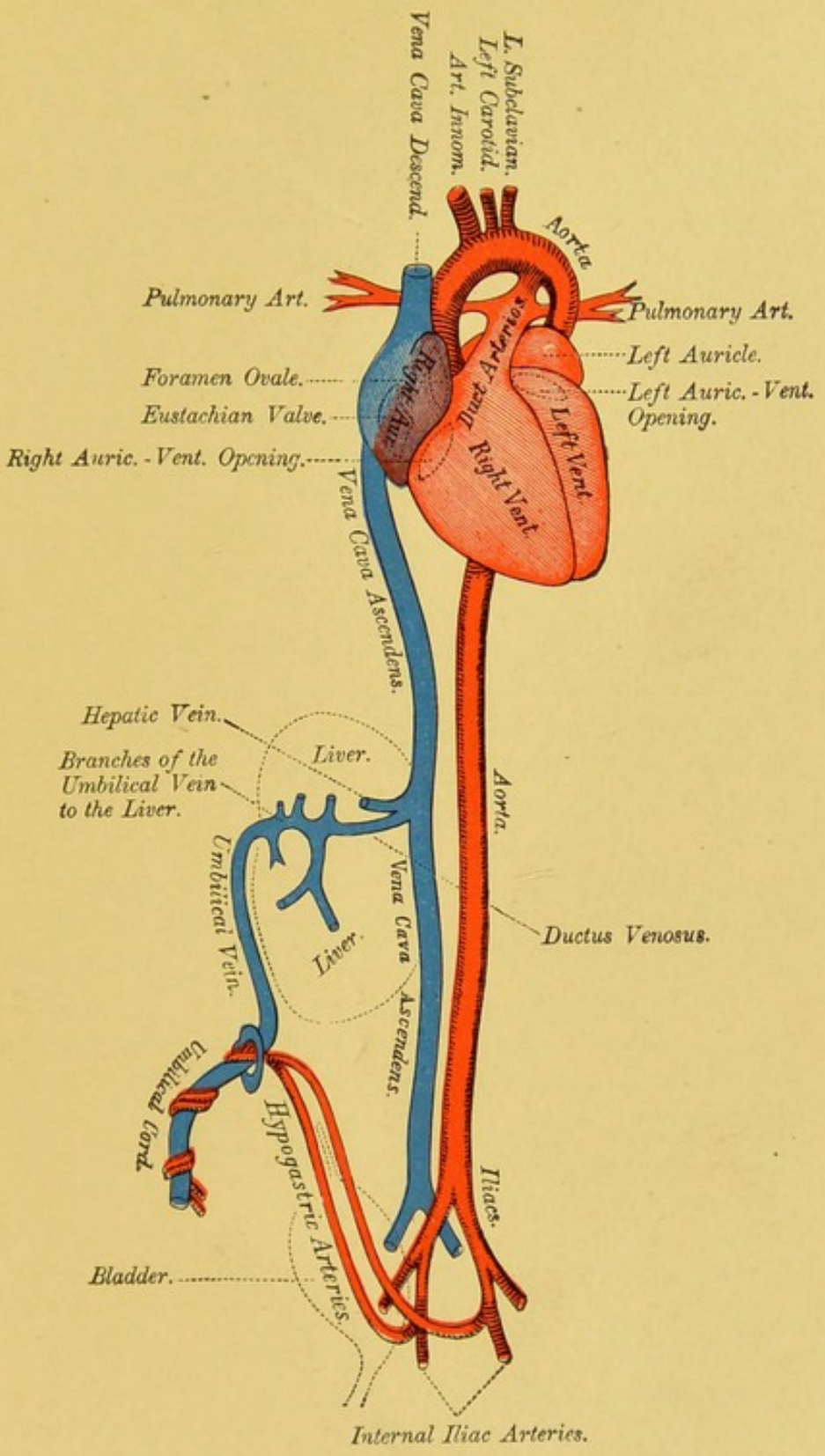

DIAGRAM OF THE FŒTAL CIRCULATION. FROM CAZEAUX & TARNIER'S OBSTETRICS.

(*See page 20 for explanation.*)


Established
1773
11, CHANDOS St. CAVENDISH

wall by the lungs; the right lung extending to the mid-sternal line, and the left encroaching on a large proportion of the left ventricle,—in fact almost all of it except the extreme point,—hence but the tip of the left ventricle and the lower part of the right ventricle are accessible to the physical diagnostician, unembarrassed by the presence of pulmonary tissue. This topography constitutes an irregular triangle, and in order to describe a triangle three points are essential, and these are, first, the apex-beat, which is situated immediately below the nipple; second, the junction of the sternum with the xiphoid cartilage; third, the junction of the third left costal cartilage with the sternum. The heart is divided into four cavities, having four valvular openings. The latter all lie in close proximity to each other in a child, within a space less than half an inch square. The determination of the position of the valves is of paramount importance. From examinations of the cadaver we conclude that the mitral valve will usually be found at the left border of the sternum, on a level with the upper border of the third costal cartilage. More under the sternum, slightly in front, and a little lower, lies the tricuspid valve.

The valves of the pulmonary artery are situated opposite the lower margin of the second interspace; slightly lower, and in an oblique direction, is to be found the aortic opening. As will be seen by comparison with the known seat of these valves in adults, children present a slight upward and outward deviation of the entire cardiac apparatus.

Sounds.—The rhythmic action of the heart is divided into a systolic and a diastolic period. During the former active cardiac movements occur; during the latter the heart itself is quiescent, the systole being an action, the diastole a pause in such action. During this rhythmic action the sounds of the heart are made audible to the auscultator. It is during the systole that the *apex-beat* is to be seen and felt, that the ventricles are contracting, and the blood is streaming out of the aortic and pulmonary arteries, and that the mitral and tricuspid valves are closed, thus forming the *first sound*,—the

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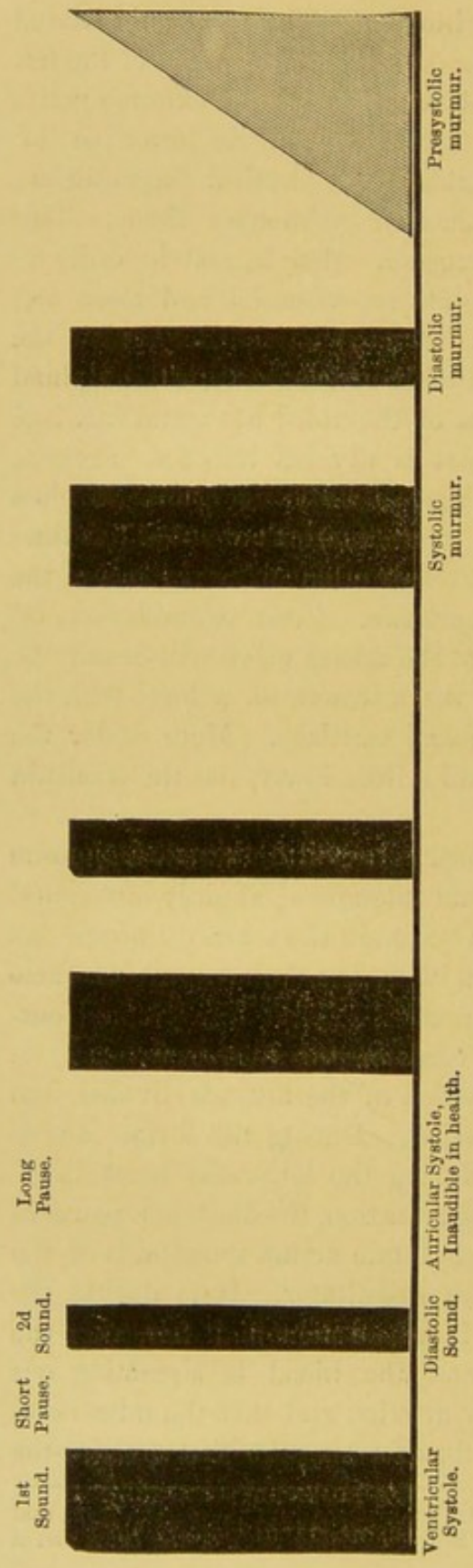
tion with enlargement of the heart, exudations into the pericardium, and in cases in which a portion of the lung covers the apex of the heart and a portion of the left ventricle."



complex sound,—the musculo-valvular sound; the muscular element being made apparent by the stroke of the heart against the chest-wall and by the audible contraction of the cardiac muscle. It is the long, low, dull boom. The *second*, or valvular, sound is short, clacking, abrupt, and ringing; it is almost purely valvular, and is due to the sudden closure of the semilunar valves, the aortic and pulmonary. While this is happening the blood is slowly finding its way from auricles to ventricles through the opened mitral and tricuspid valves in order to produce another systolic action. The aortic and pulmonary arteries at their origin, the chordæ tendineæ, and the whirl of blood in the ventricles severally assist in the production of the cardiac sounds. Just how much of it they contribute is as yet a moot question.

The sounds and pauses of the cardiac rhythm will perhaps be made clear by the accompanying diagrammatic representation with which we have been accus-

tomed to illustrate the matter to our classes. The heavily-shaded lines represent the first sound, the lighter the second; the long pause is represented by the greater length of the line, the short pause by the shorter line.

To recapitulate: the first, or systolic, sound represents the period during which the blood streams from the left ventricle to the aorta, and from the right ventricle to the pulmonary artery (closure of mitral and tricuspid valves, apex-beat, and carotid pulse).

The time elapsing between the first sound and the occurrence of the so-called diastolic sound,—the second,—is designated by the term *short pause.*

The *long pause* is the time during which the blood is finding its way through the pulmonary artery and lungs, by way of the pulmonary vein, into the left auricle, and from there into the left ventricle. It also represents the time required by the blood to traverse the systemic circulation and find its way back into the right auricle, and from there into the right ventricle. The diastole commences with the closure of the semilunar valves, aortic and pulmonary, and is terminated by the contraction of the two auricles, thus emptying themselves of blood and filling the ventricles, thereby aiding in producing another systole. The action of the auricles, their systole, is inaudible in health.

METHODS OF STUDY.

Suspected disease of the heart is to be determined by conjointly *inspecting, palpating, percussing,* and *auscultating* the præcordia and the superficial circulatory vessels.

Inspection.—In commencing the study of disease in the young, the little patient will at once present an obstacle by his unwillingness to be examined. While endeavoring to quiet the patient and allow his circulation to establish its equilibrium, it is well to note the history of the case. Has your patient had scarlatina, measles, rheumatism, or diseases with a known tendency towards the endo- or peri-cardium? Note the appearance of the capillary circulation, especially about the lips and finger-nails, the expression of the face, the presence of dropsy or anasarca, the respiratory functions, the existence

of cough, the character of the respiratory action, and the condition of the alæ nasæ, together with the color of the mucous membranes. In children the præcordia is somewhat more bulging and apparent than in adults, as is also the depression at the lower part of the præcordial region. Neither of these signs, however, merit much consideration. A decided bulging would perhaps point towards hypertrophy or pericardial effusion, as a decided retraction would to a dense pericardial adhesion.

Inspection is of great utility in locating the position of the apex-beat. Its situation is, as has already been stated, somewhat higher than in the adult. All things being equal, it is more distinct in a lean child than in a plump one. Its point of impingement is not constant, changing somewhat with the movements of the patient and the condition of the alimentary canal.

A boy aged fourteen in the medical wards of the Hospital of the University of Pennsylvania, who was an enormous eater and suffered from chronic slowness of digestion, with flatulent distention of the whole tract, would several hours after a full meal show an upward displacement of the apex-beat of one or more interspaces. It is also modified by the respiratory act, inspiration causing it to descend, expiration to become more distended and apparent. Many diseases also displace it, viz., hypertrophy, dilatation, pericardial effusion, pleural effusions, enlargements of the liver and spleen, and congenital abnormalities.

Palpation, on the whole, is perhaps more valuable to the clinician than is inspection. As stated when considering the subject of inspection, the apex-beat must be located as a landmark. This is generally best accomplished with the tips of the fingers, which are more sensitive than the ball of the thumb. In a healthy subject the apex-beat will be limited in area, well defined, and punctuated; that is, giving evidence of the first and second sound; the former a long, dull vibration, the latter a short and distinct impulse against the palpator's hand. Over the right ventricle, in a healthy subject, the finger should detect no considerable impulse, except after sudden muscular exertion or mental emotion; then the action is more of

a rhythmic heave than a beat, augmented by the left lobe of the liver which adjoins the right ventricle of the heart through the interposing diaphragm. The apex-beat may have its rhythm interfered with by many conditions both within and without the circulatory apparatus: for example, in hypertrophy the extent and force of the beat is much augmented; in dilatation the beat is apparent over a much larger area than in health, but its force is very deficient. In pericardial effusion it is irregular, feeble, wavy, with a more or less upward displacement. A feeble apex-beat accompanies diseases of the brain, a vitiated condition of the blood, and general asthenic conditions. The to-and-fro motion of a roughened pericardium, sometimes styled cat's-tongue pericardium, will transmit to the hand a rubbing movement, as a valvular murmur will produce a peculiar thrill, styled by Laennec the purring tremor.

Percussion.—To percuss the heart of a child with accuracy is no easy task; it requires a certain amount of skill and a great deal of care. The general directions for eliciting cardiac percussion-dulness are about as follows: with the little patient preferably sitting up in bed, as the recumbent position would interfere with the correct approximation of outlines, the observer commences to percuss at the left mid-clavicular line, and proceeds downwards until a dull sound, together with a peculiar sensation transmitted to the finger, which will be appreciated at once by all who have attained any skill whatever in physical diagnosis, tells us that we have reached the upper border of the heart, which in a child will be about the upper border of the fourth costal cartilage, or possibly in the third interspace. This is the uppermost portion of the heart that is left free from lung encroachment. The transverse diameter of the heart is to be determined by percussing from the *right* side of the sternum, on a level with the fourth rib or interspace, directly across the bone. At about the left edge we meet with resistance and elicit a dull sound; we continue percussing until a clear note is once more brought out, in this way demonstrating the transverse cardiac dulness. Locating the apex-beat by palpation and inspection determines the lower margin of the heart, and we then have but the inferior surface

to map out. This, however, is probably the most difficult and perplexing study that the physical diagnostician has to combat. It is only to be accomplished by appreciating the difference between hepatic and cardiac dulness, as at this point one merges into the other. The dulness due to the presence of the heart is not so absolute as that from hepatic tissue.

Percussion of the præcordia in children is, on the whole, unsatisfactory, as the space over which dulness may be elicited is so extremely small that were we to trust to percussion alone we could but conclude in many cases that the heart was altogether wanting. Many extraneous conditions alter the cardiac dulness. During inspiration and distention of air-vesicles the heart is covered over and somewhat removed from the chest. On the other hand, expiration enlarges the area of dulness very materially, especially upwards and laterally. In emphysema the dulness is permanently diminished, and is permanently increased by diseases of the heart and pericardium. Later in our study we will compare the adult præcordia with that already given.

Auscultation.—In the young this is perhaps the most satisfactory manner of examining the heart. Auscultation may be practised either by the *mediate* or the *immediate* method, in the former using the stethoscope, and in the latter applying the ear directly to the chest. The stethoscopes before the medical

man of to-day are legion, as are their form, shape, design, and applicability. On the whole, however, the most generally satisfactory instruments are those that are simple in construction and light in texture. The light gun-metal mon-aural instrument, with detachable ear-piece, introduced by Hawksley, is undoubtedly the best for every-day use; and if the auscultator possesses a double stethoscope, as seen in the accompanying figure, and described by one of us in the *Medical News*, November 7, 1885,* he will be fully prepared, as far as

* A New Binaural Stethoscope, by William A. Edwards, M.D.

instruments go, to elucidate the most intricate problems in physical diagnosis. In the auscultation of children it is well to remember the following aphorisms: Your patient and yourself are to be in a position that will not be constrained or uncomfortable, and will permit of a ready application of the ear or stethoscope to the region under investigation. The chest should be bare, and the stethoscope applied closely and accurately to the integument. The auscultator's head must not be too low. This is absolutely essential.

Instruct your patient, if old enough, to inspire, expire, and hold the breath; also auscult during full-held inspiration and expiration.

Following the foregoing rules, one will be able to detect two sounds very dissimilar in character; one will be low, dull, booming, and giving the impression as occurring close to the ear,—the so-called first sound; the other is short, abrupt, ringing or flapping,—the second sound. These two sounds in children are audible over the entire præcordia, and in fact over most of the costal framework. Certain regions of the chest have been selected at which the individual component elements of these sounds may be heard with maximum intensity, viz., the play of the mitral valve is best heard just over the apex-beat, no matter what its situation; the tricuspid sound is best made out in the vicinity of, and slightly above, the ensiform cartilage. In this region diseases of the right ventricle are also to be studied. The second costal cartilage on the right of the sternum has been designated "the aortic cartilage." At this point the aortic valves are to be auscultated.

The sounds of the pulmonary artery are to be sought for in the second intercostal space, near the left edge of the sternum. As before stated, the cardiac apparatus is situated somewhat higher in the very young, hence we, in these cases, place our landmarks somewhat higher. A point worthy of remembrance, more particularly to the young auscultator, is the fact that both the first and the second sounds are to be heard at each of the points above stated; but the sounds vary with the different situations. The practical fact to remember is that the sounds have their maximum intensity and clearness over the seat of production. The sounds are variously modified

by many conditions. They may be changed in rhythm, in character, and in transmission. The latter element, even in the most healthy states, is very diversified.

Full inspiration lessens the sounds very materially, as full expiration increases the extent over which they may be heard. The first sound is probably more liable to undergo change than the second, as in children the general systemic conditions which aid in altering the second sound are not so apt to be present; as, for instance, the altered blood conditions of lithiasis, or gout, with increased arterial tension. Both of the sounds may be obscure or distant, or the periods of silence, the long and short pause, may be changed in rhythm. Again, the sounds may intermit, or, what is more unusual in early life, a reduplication of the sounds may occur; one or both may be double, or one alone may be doubled over a certain part of the præcordia, and not over another. In some rare cases the heart presents four sounds, more usually, however, three, and the second sound is probably the one most frequently reduplicated.

This anomalous action is of little practical value to the diagnostician. Its cause is the want of synchronous action between the left and the right heart.

THE FŒTAL CIRCULATION.

Certain differences exist between the fœtal and adult heart, a correct understanding of which is essential before congenital diseases and malformation of the heart can be appreciated.

The two sides of the fœtal heart communicate one with the other. In the adult all the venous blood is carried from the right ventricle to the lungs *via* the pulmonary artery, in order to eliminate effete matter and receive oxygen. In the fœtus this mechanism does not pertain, as only enough blood is passed through the pulmonary arteries to maintain their calibre, so that they will be in condition to carry the blood to the lungs immediately after birth.

In examining the fœtal auricles an inter-auricular communication will be found to exist, arranged in such a manner as to permit the blood running into the right auricle to pass into the left, but not in a contrary direction. This *foramen*

ovale plays an important rôle in congenital heart-disease. By reflecting a moment we will see that an appreciable quantity of the fœtal blood, after reaching the right auricle, does not, as in the adult, find its way into the right ventricle, but through the foramen ovale into the right auricle.

Notwithstanding this arrangement to prevent the right ventricle from receiving more blood than enough to keep the pulmonary artery patulous, it still receives too large a quantity; hence nature has provided a vessel peculiar to the fœtus, the *ductus arteriosus*, arising from the point of bifurcation of the pulmonary artery and opening into the arch of the aorta, in this way allowing but a small proportion of the blood to find its way along the pulmonary artery and into the lungs.

In order that the fœtus may get rid of its impure blood, nature has prolonged the fœtal hypogastric arteries into two large arterial trunks, which pass into the cord, and are then styled *the umbilical arteries*, and carry the impure blood to the placenta for renewal. The *single* umbilical *vein* receives the pure blood and carries it to the under surface of the liver, where it is turned into another special fœtal vessel, the *ductus venosus*, and emptied into the ascending vena cava, and from this vessel into the right auricle.

To secure a more correct understanding of the fœtal circulation, it is perhaps better to trace it from the entrance of the blood through the umbilical vein to the under surface of the liver. This viscus receives but a small part of it; the great mass is thrown into the vena cava through the ductus venosus. The cava also receives the blood which has circulated in the lower extremities of the fœtus, and that blood which has circulated through the liver. This blood, mixed in quality, is carried to the right auricle, and the major part of it is at once carried to the left auricle *via* the foramen ovale. From the auricle it is directed into the left ventricle, whose systole sends the greater part of it into the head and upper extremities through the aorta; but a small portion is sent to the lower extremities. The blood which has been distributed to the upper part of the body is returned to the superior vena cava, and is then emptied into the right auricle, and from here the largest quantity of the blood is thrown into the right ventricle,

whose systole propels it into the pulmonary artery and through the ductus arteriosus into the descending aorta.

It is thus seen that some of the blood in the descending aorta, which is to be conveyed to the lower extremities, has already circulated through the head and upper extremities; but the greater part of the blood in the descending aorta is carried for purification to the placenta through the umbilical arteries.

At birth the circulation must at once change from the conditions that were adaptable to the uterine environment to that which will support the life and vitality of a separate organism.

The *establishment of independent circulation* takes place as soon as the child is born. The first act of the new-born babe is a lusty cry which inflates the lungs, and, in consequence, dilates the pulmonary arteries. As a sequence, the greater part of the blood in the right ventricle is at once distributed to the lungs, where it becomes changed from venous to arterial blood, and is returned through the pulmonary veins to the left auricle. The left auricle now receives more blood than it has been accustomed to, the right less, and, owing to arrest of the placental circulation, the umbilical veins are inactive. We now find that the pressure of the blood in the two auricles is equalized, which aids in the closure of the foramen ovale. The blood no longer finds its way from right to left auricle, but into the right ventricle, and thence to the pulmonary artery. The ductus arteriosus becomes impervious, and soon collapses. The blood in the descending aorta does not find its way into the hypogastric arteries, but directly into the lower extremities, and adult circulation is established.

CONGENITAL DISEASES OF THE HEART, MALFORMATION, CYANOSIS.

The circulatory apparatus at the beginning of embryonic life is represented simply by a few cells; pulsations in the human embryo appear towards the fifth day (Paul).

Coste has had the good fortune to study the human heart at the second week of uterine life. Authors differ as to whether the heart is first simply a straight tube or spindle-

shaped pouch; at all events its earliest trace is simply a thickening of the intestinal fibrous layer of the fore part of the alimentary canal. At two weeks the heart has become curved like the letter S, the posterior part of the tube rests on the dorsal surface of the anterior part; the upper or anterior extremity forms connection with the arterial branches, and will ultimately furnish the aortic arches; the posterior part receives the omphalo-mesenteric veins. As growth progresses the curves in the letter S increase, and shallow indentations become more apparent. These mark the first division of the organ into auricles and ventricles. The first indentation represents the auricles into which the veins are running, the middle the ventricles, and the lower or third portion the common arterial trunk. Before the cardiac apparatus assumes the S-like form the primitive auricles are the larger; but a little later the ventricles become much the largest. At the end of the second week the heart is still simply a hollow, twisted tube, the blood entering by the veins, and finding its exit by the arteries, the auricles and ventricles simply forming a common chamber. The exact method by which the auriculo-ventricular septum is evolved is as yet not clearly demonstrated. At the fourth week the heart is divided into halves, a left, or arterial, and a right, or venous, half. The common arterial trunk is now separated into what will eventually form the aortic and pulmonary artery. At about the third month the auriculo-ventricular septum is completely formed, as is also the septum ventriculorum dividing the right and the left ventricles. The septum dividing the auricles is now entirely completed except that portion which will form the foramen ovale, and will be closed in a manner to be described hereafter. The fœtal heart, after about the first month, has a more or less rectangular outline, the auricular appendages overhanging the ventricles. The manner of the formation of the aorta and pulmonary artery cannot better be described than in the words of Rokitansky, quoted by Longstreth: "These two vessels have their origin in the common arterial trunk, which divides into the permanent aorta and pulmonary artery, to be completed about the eighth week. A little swelling appears in the common trunk, which does not grow in a straight line through the lumen of the

common trunk, but in such a manner that the forming septum makes a concavity posteriorly towards the aorta, and a convexity anteriorly towards the pulmonary; thus on cross-section the aorta has the outline of the gibbous moon, the pulmonary fitting into it separated by the septum of a new moon. The septum ventriculorum, starting at the base of the ventricles from the fibrous ring of the auriculo-ventricular orifice (having already been built upward from the future apex of the heart), originates at a point on the posterior wall of the common ventricular cavity in exact correspondence with the starting-point of the little swelling on the inner surface of the common arterial trunk. The two septa are thus formed in apposition, and the structure of the septum ventriculorum is so far advanced that by the eighth week the vascular trunks are connected with the proper ventricles, but the septum ventriculorum does not close completely until about the twelfth week."

Having hastily reviewed the development of the human heart, let us for a moment consider the changes which take place in the vascular arrangement of the fœtus after birth, as it is to an abnormality or arrest of these changes that many cases of so-called congenital heart-disease are due. The ductus arteriosus collapses soon after birth; the vessel contracts by its own inherent contractility, and from the loss of the column of blood which formerly passed through it, the mass of the blood now going to the lungs. Some interesting observations on this structure were made by J. Collins Warren before the Philadelphia Pathological Society in October, 1885.

His investigations lead him to conclude that the ductus arteriosus at the time of birth in certain important respects differs in structure from the aorta and pulmonary artery. The media is much thicker than in either of these vessels; it is thrown into irregular folds, which are increased at the time of birth. The outlines between the different layers are less marked than in the walls of other vessels. The lamina elastica is indistinct, and in places apparently wanting. The media consists chiefly of longitudinal layers of muscular fibre, a few circular bundles existing in the outermost layers. A few weeks after birth the greater portion of the walls of the ductus undergo hyaline degeneration, the outer or circular fibres of the media alone

remaining. There is at this time an active growth of long, spindle-shaped cells, with staff-shaped nuclei at the edge of the media bordering on the opening into the aorta; there is also thickening of the intima. Eventually the hyaline tissue becomes absorbed, and is replaced by a band of fibres continuous at each end with the media of the large vessels. At the aortic end, in a longitudinal section, the media is seen slightly separated at the point of the cicatrix, and between the two and continuous with them are the longitudinal fibres of the ligamentum arteriosum. In the centre of the depression marking the site of the cicatrix a small vessel is given off into the axis of the ligament, where it either loses itself in a capillary network or becomes continuous with a similar vessel coming from the pulmonary artery.

Dr. W. S. Forbes would add as a cause of closure of the ductus arteriosus the traction exerted by the descent of the diaphragm upon the fibrous bands extending from the tendinous centre of the muscle to the aortic arch.

The foramen ovale soon becomes permanently closed, probably by contracting adhesions to the edges of the aperture. The umbilical arteries and veins and the ductus venosus speedily collapse and become impervious. Any one of these structures may remain pervious and constitute some of the circulatory anomalies due to arrested development or want of proper completion in the stages of change from fœtal to adult circulation.

Attempts have been made for the last thirty years to classify the circulatory anomalies and malformations into a convenient working form, so that cases may be arranged under this or that heading, with, however, but meagre success. Peacock's classification, a fairly good one, is as follows:

1. Arrest of development early in fœtal life (fourth to sixth week; heart with two or three cavities; single or imperfectly divided arterial trunk).

2. Arrest at a later period (sixth to twelfth week; imperfect auricular or ventricular septum; imperfect or misplaced vessels).

3. Those after the third fœtal month (closure and patency of fœtal passages; irregularities of valves, cavities, etc.).

We agree with Longstreth, that could a classification be based on the seat of the disease alone it would simplify the study of these cases to a great degree,—in fact, be as simple as the study of adult valvular disease; but this, unfortunately, if carried out, would, owing to the varieties in the morbid processes, lead us to greater chaos than ever, so that on the whole it is perhaps wiser to divide the subject under two grand headings,—(1) the primary malformation, (2) and their secondary effects.

The foramen ovale.—As stated above, the auricles shortly after birth are no longer intercommunicating, owing to the closure of the valve. This, however, is not always the case, as the valve may fail to adhere, there may be perforations or slits in its leaflets, or the opening may be so disproportionate in size that the valves will not make it impervious. At times this opening is so large that but little inter-auricular septum exists at all. Some observers state that the valve is never completely obliterated until the eighteenth month or second year of extra-uterine life. We occasionally meet with cases on the post-mortem table in which there is an anatomical defect in the foramen, but in which there is perfect physiological action. Such a case was examined by us in the Philadelphia Hospital. The child was aged four, and had presented during life no evidence of cardiac or circulatory disturbance. Death was caused by acute entero-colitis. In front of and slightly above the fossa ovale was seen the so-called valve of Vieussens, which is a simple muscular projection, with its concavity looking posteriorly and inferiorly. This was continuous below with the Eustachian valve. The proper adherence of these two structures would have closed the foramen ovale. The two arches in our case crossed like the blades of scissors, and admitted the passage of a small, flat instrument from the right to the left auricle; but at the moment of auricular systole the blades of the scissors, so to speak, were accurately applied one to the other, and, consequently, no admixture of the venous and arterial blood was possible. It seems to be a fact that defects of the foramen ovale are more common in females than in males. It also seems to be established that most cases of patency of foramen ovale are to be

classified under the second division of our subject,—*i.e.*, secondary effects. This, of course, excludes those cases presenting slits or perforations in the valves.

In many cases the patulous foramen is secondary to defects in the mitral valve, allowing regurgitation or obstruction in the large arterial trunks, aorta, and pulmonary artery. A patulous foramen is more frequently associated with obstruction or narrowing of the pulmonary artery. Narrowing of the tricuspid orifice would also be a direct cause of patulous foramen ovale, but primary defect in the tricuspid orifice, causing narrowing or stenosis, is very rare; in fact, it is rare that we see it even in combination with other defects. It may be, and generally is, due to narrowing of the pulmonary artery. As a rule the direction of the blood-current in cases of patulous foramen ovale is the same as that during fœtal life,—*i.e.*, from right to left auricle,—but cases have been noted in which the direction of the blood-current was directly opposite from that which pertains during intra-uterine life,—*i.e.*, from left to right.

Premature closure of the foramen ovale.—This is of very rare occurrence. In these cases, as one would expect, the right side of the heart is unduly developed and the left atrophied. The blood during fœtal life is of necessity all transmitted through the right cavities, the pulmonary artery, and duct. To Vieussens is due the credit of first recording such a case.

Patent septum ventriculorum.—An abnormal communication may also exist between the two ventricles greater or less in degree; in some cases almost the entire septum may be absent; as a rule, however, a small opening is found in the *pars membranacea*, or *undefended part*, which is that portion of the septum that is practically simply a membranous partition between the ventricles. The openings may be multiple. Three have been recorded.

This again is rarely a primary malformation, but usually follows defects in the pulmonary circuit; the septum normally closes a little before the twelfth week, hence a patulous condition would indicate an abnormality early in fœtal life. The blood-current is usually from left to right; it, however, may be reversed, as we saw in cases of auricular communication.

The pulmonary artery and valves: narrowing stenosis or atresia (with closed ventricular septum).—This is a somewhat frequent defect; the atresia varies in degree from simple narrowing to complete closure; as a rule, the greater the stenosis and thinning of arterial wall the earlier did the defect arise.

The foramen ovale, as above stated, is generally patulous, as is also the ductus arteriosus. In cases of atresia the right ventricle will be found markedly decreased in size; in cases of stenosis the ventricle is generally in a condition of dilated hypertrophy.

Stenosis and atresia (with open septum ventriculorum).—This condition is considered by many competent authorities to be the most frequent malformation of the heart. The narrowing or stenosis may be observed at any point from the valves to the bifurcation of the artery; it is, however, generally most noticeable at the orifice. The vessel-walls may be extremely thin, and the vessel itself somewhat shrunken.

In complete atresia one of two conditions will be met with: either the valves alone are closed, accompanied by a narrowing of the vessel calibre, or the vessel itself, from orifice to bifurcation, is changed into a cord. Many secondary changes are to be noted. Right auricular and ventricular hypertrophy, with dilatation and altered tricuspid leaflets, is not infrequently combined with the stenosis and atresia; the left ventricle being smaller than normal, the shape of the heart is changed.

The aorta may be increased in size and the mitral valves altered by morbid changes.

The fact that the septum ventriculorum remains open explains the mechanism by which the circulation is maintained, the blood finding its way through the patulous septum into the left ventricle.

The foramen ovale and ductus arteriosus in some cases are closed, in others open. In about thirteen per cent. of all cases the ductus arteriosus was found to be entirely absent.

The pulmonary artery valves may present congenital numerical abnormalities, being either excessive or deficient in number and conformation.*

More than the normal number of valves has but little clinical significance; but a lessened number is decidedly im-

* Heart showing anomalous arrangement of the leaflets of the pulmonary valve, these being four in number. Wilson, Trans. Path. Soc. Phil., vol. vii. p. 57.

portant in prognostic significance of later life, as, if there be but two valves, there is great probability of insufficiency, permitting regurgitation, arising during adult life.

If there be but *one* valve, with ill-defined markings, showing attempts at division, the probabilities are that obstruction will arise.

The valves may be entirely absent, or there may be simply a diaphragm separating the orifice from the ventricle.

The tricuspid valve and orifice.—This, again, is a situation in which we rarely encounter primary abnormalities, although it is often indeed difficult to say whether they are primary or secondary.

The valve may be very imperfect, allowing regurgitation, or there may be stenosis or atresia, due to adhesion of the leaflets or contraction of the connective tissue. In some cases the valve has been seen stretched across the orifice like a little diaphragm; there is usually a small opening in its centre, through which the blood is able to find its way; in other cases the orifice and valve have failed to develop, appearing shrunken or shrivelled; the entire right ventricle in these cases has not undergone the proper amount of development. The pulmonary artery is also usually insufficiently developed, with narrowing of the pulmonary conus. The foramen ovale and the ductus arteriosus may both remain patulous. Having now considered the right side of the heart and its defects, we will consider *defects in the left side of the heart.* Congenital primary defects are relatively much less frequent in the left heart than in the right. Defects in the aorta and its valves are perhaps more frequently met with than defects in the mitral valve or orifice.

The aorta has been noted in a condition much narrower than normal; this is a true primary defect, due to unequal division of the truncus arteriosus communis.

Narrowing of the aortic conus may originate late in fœtal life from an endocarditis, similar to that seen in extra-uterine life. In these cases the mitral curtains will generally be implicated in the process, as will also the aortic valves.

The left ventricle may be either in a condition of simple hypertrophy or one of dilated hypertrophy, or it may be

shrivelled and shrunken, as was the right ventricle in similar affections of its outlet.

Should the aortic stenosis arise early in the fœtal life other secondary effects will be noted, not, however, as marked as were those in the right heart. The auricular or ventricular septa may be patulous; the aortic valves may be but two in number, or firm bands may form beneath the aortic orifice. The mitral orifice is much less frequently affected than the aortic. This is the rule also in the right side of the heart, the pulmonary being more frequently affected than the tricuspid.

Transposition and malformation of the great vessels.—Unequal division of the common trunk has already been noted under pulmonary and aortic stenosis or atresia.

The transposition may be of two varieties: the vessels may communicate with the wrong ventricle but their relative positions to each other may be normal, or they may communicate with the wrong ventricle and be transposed in their relations to each other; this latter defect arises very early in fœtal life, and is attended with many secondary defects.

The common trunk at its beginning may fail to completely divide, and the blood of the aorta is allowed to mingle with that in the pulmonary artery; in all these cases we are likely to note a deficiency in the ventricular septum and other abnormalities. In these cases the heart is not infrequently *displaced* in the chest.

Transposition of the aortic and pulmonary arteries has been a well-recognized congenital defect for a number of years; indeed, the first case was recorded so early as 1797, by Dr. Baillie, and the specimen still exists in the Royal College of Physicians.

The descending aorta may be given off from the pulmonary artery through the ductus arteriosus. This is generally caused by imperfect development of the aorta between the origin of the left subclavian artery and the duct, as a consequence sufficient blood is not carried from the ascending to the descending aorta. These cases are seen on the post-mortem table, advanced in years, with an obliteration of the aorta beyond the left subclavian artery, the circulation being maintained through collateral channels. Such a case is recorded by Steidelle.

Transposition of the venous trunks.—In these anomalies the foramen ovale remains pervious of course, allowing the venous and arterial blood to mingle; this to a certain extent overcomes the anomalous arrangement of the venous trunks, as persons have reached a fairly advanced age under conditions of transposed venous trunks.

The ductus arteriosus.—The closure of this structure commences normally at the aortic extremity, and is usually, according to Billard, not entirely closed until fourteen days after birth.* It may not close until twenty-one or thirty days after birth. The pulmonary extremity may remain pervious for a longer time; if the closure commences at the pulmonary extremity, it is as a rule due to cardiac abnormality and reverse blood-current.

Cases are recorded in which the duct is totally impervious, others where it is partly open, and again others in which the entire structure is wanting, and in other cases two ducts have been found; a distinct duct has been recorded as arising from the right ventricle. It has been noted in a state of great dilatation.

Premature closure of the duct is in reality a defective development, or absence, of the duct. In many cases the pulmonary orifice is so narrowed that the duct is apparently closed. If the pulmonary orifice is closed the duct remains patulous. The blood from the right heart, to reach the lungs, must pass through the foramen ovale or through the ventricular septum. A patulous duct is usually seen in case of transposed vessels, as above recorded, or when the descending aorta is markedly narrowed.

Numerical auriculo-ventricular anomalies.—A supernumerary ventricle may exist on the right side. They have been met with of various sizes. The defect is in all probability due to inflammation of the fœtal endo- and myocardium, together with hypertrophy of the muscular bands close to the conus arteriosus dexter. Fenton records a heart with five cavities. The *cor biloculare* is a heart consisting of but two

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* For manner of closure see observations of J. Collins Warren.

cavities, one auricle and one ventricle. The *cor triloculare biatriatum* is the association of a single ventricle with two auricles. On the other hand, the *cor triloculare biventriculare* is a double ventricle and a single auricle.

As an interesting example of these numerical abnormalities we would cite the case reported by Brewer in *Boston Med. and Surg. Reporter* (*Weekly Med. Review*, October 17, 1885). At the Columbia Lying-in Hospital in Washington a healthy colored woman was delivered of a male child. At the time of birth the child was cyanosed, and it was only after the employment of artificial respiration and various other stimulating measures that respiration was established. The child lived fifty-four hours, during which time embarrassment of respiration, rapidity of pulse, and great restlessness were constantly observed. The efforts at nursing were feeble and without result.

At the autopsy the heart alone was removed and preserved for subsequent examination. The lungs and abdominal organs were examined *in situ*. The former were well aerated, and the latter presented nothing abnormal. Upon later investigation the heart was found to consist of three cavities, two auricles and one ventricle. The auricles were well formed, but of unequal size, the left being considerably enlarged. The septum was present, and exhibited nothing abnormal except the large size of the foramen ovale, which admitted the tip of the little finger. There was but one, the left auriculo-ventricular, opening. In place of the tricuspid valve there was a slight depression, at the bottom of which was a minute fibrous ring, three mm. in diameter. This was impervious, and an opening made through it in search of a rudimentary right ventricle revealed nothing but the dense muscular tissue of the ventricular wall.

The ventricular portion of the heart did not differ in size and external appearance from normal specimens of the same age. The walls were somewhat hypertrophied, measuring nine mm. in thickness. Its cavity was spacious, and presented no trace of a septum; from it was given off one large arterial trunk, the aorta. A small vessel, blindly originating at the junction of the anterior wall of the aorta with the ventricle, measuring three mm. in diameter, bifurcating eight mm. above

its origin, was observed and considered by Dr. D. S. Lamb, of the Army Medical Museum, to be the rudimentary pulmonary artery. As the autopsy was necessarily hurried, further investigation, with a view of ascertaining the origin of the vessels supplying the lungs, was not undertaken.

"Although the specimen exhibits three distinct cavities, considered from a physiological point of view, it should be classed as a bilocular heart, inasmuch as the right auricle, having no connection with the ventricle, serves only as a common venous trunk.

"Examples of trilocular hearts are by no means common. Peacock, in 1858, had collected but eleven, and the American and British journals published since that time furnish an additional record of seven cases. Most of these, however, are examples of an arrest of development taking place at a period of fœtal life much later than the case which is now under consideration. Evidence of this is to be found in the fact that in nearly every instance a rudimentary ventricular septum is present, and the pulmonary artery, in a more or less complete state of development, can generally be found to communicate with the ventricle.

"Of the few cases which closely resemble this one can be mentioned the one reported by C. Bernhard, in which there was entire absence of auricular septum, right ventricle, and pulmonary arteries; two cases (reported by Owen and Vernon) in which there was absence of left ventricle and aorta, the auricular septum being defective; and the case described by Heinman in the *Medical Record* of 1878, which consisted of two auricles and one (the left) ventricle, the aorta being well formed and the pulmonary artery rudimentary and impervious."

These three latter numerical defects are, as a rule, associated with abnormalities of the pulmonary artery or orifice. That presenting a single auricle is in reality a deficient closure of the foramen ovale; but the hearts thus deformed, as a general rule, present many other abnormalities.

The same band-like hypertrophy of muscular fibre which assisted in forming a supernumerary ventricle on the right side may also be met with in the left ventricle, producing a similar anomaly.*

* Note.—See Addenda, No. I.

Symptoms.—Many children at birth present an intensely blue discoloration, which more or less speedily passes away, depending upon the voluntary or artificial establishment of respiration, whereby the circulatory organs are rendered active and the cyanosis rapidly disappears. If, however, the discoloration continues, other conditions being excluded, congenital malformation of the heart or great vessels must be looked to as the causative agent. This is also the case in a child who weeks after birth develops cyanosis, which is then almost proof positive of congenital defect, provided, of course, that the cyanosis is not due to an acute disease, as acquired valvular disease, atelectasis, or pulmonary affections. Atelectasis and other pulmonary affections are to be diagnosed by their special respiratory symptoms, and malformations by their special pulse- and heart-symptoms.

If due to congenital malformation, a murmur may be heard, together with quickened pulse and rapid heart-action. If atelectasis and cardiac defect should be associated, the diagnosis is extremely difficult, and can only be verified by post-mortem examination, as the case in all likelihood will rapidly terminate in death.

Should the malformation be of such a character as is compatible with life, the child will present but few symptoms which are diagnostic, except the physical, and these unfortunately are not very satisfactory: the little patient grows and passes through the various stages of development in a feeble manner, is poorly nourished, and incapable of the usual exertions of early childhood and adolescence. One case, a girl, remained in bed until she was sixteen years old. Cyanosis may or may not be present. It is the rule that during the first week of life cyanosis, greater or less in degree, appears for the first time. It may then remit or entirely disappear, or it may remain permanent throughout the entire life of the individual. Dyspnœa may be a very exacting symptom, usually aggravated by exercise or excitement, at which time the cyanosis, if absent, may again become apparent, or if present is greatly intensified. The body temperature presents great variations in different cases. Some cases, by surface thermometry, will show a normal registry, but will complain

of chilliness or of actual cold; others will show a subnormal temperature, possibly of half a degree, particularly after a paroxysm of dyspnœa, or cyanosis. If the temperature is materially lowered for any length of time, the case is approaching dissolution. The temperature is said to rise as high in these children when affected by febrile disease as it does in those with normal hearts. Palpitation is generally evident. In some cases it only occurs on exertion; in others when there is dilated hypertrophy and obstruction in the blood-current. It is then apt to be excessive, distressing, and alarming to the patients and their friends.

Cough is almost always present, aggravated by pulmonary congestion, to which these cases are liable on account of venous stasis in the bronchial and pulmonary mucous membranes. There may be bloody expectoration. Besides bronchitis they are prone to congestion of liver, spleen, general or local dropsy, and albuminuria. Clubbing and rounding of the fingers and arching of the nails was by the older observers considered characteristic of malformation of the heart. Later it was thought to be pathognomonic of tubercular pulmonary phthisis. Now, however, we recognize it in many other affections, as in acquired valvular disease, chronic pleurisy, and in chronic pulmonary diseases.

It is worthy of note, however, that frequently tubercular phthisis is associated with congenital stenosis of the pulmonary artery. This has been the experience of many observers. Whether it is a true relation between cause and effect, it is, in view of our knowledge of bacillary phthisis, indeed difficult to say, as it does not seem obvious why cases with congenital stenotic pulmonary arteries should present a soil that would favor the development of the bacillus tuberculosis.

There may be ulcerations about the toes, anus, vulva, or finger-nails, due to deficient circulation, or a peculiar skin mottling, thought by some to be characteristic. The physical signs are on the whole not as characteristic as we would desire; indeed, there exists no sign or sequence of signs by which a congenital murmur can be definitely differentiated from an acquired lesion.

By conjointly inspecting and palpating we may determine

the fact that the heart is not in its normal position, and remembering the fact that displacement of the heart is usually associated with transposition of the principal trunks, we may in this way throw some light on the problem. Palpation not infrequently determines the presence of a purring thrill. Percussion may, or may not, show increased area of dulness, as a heart may be extensively malformed with but little, if any, alteration in its size. Auscultation will in all likelihood reveal a low, blowing, basal, systolic murmur, usually single. This murmur is generally caused by a patulous foramen ovale, or imperfect septum ventriculum. Of course the murmurs may be multiple, or they may be rough, rasping, and diastolic in time.

The murmur due to a patent foramen ovale may vary in time and intensity. Tilbury Fox considered that a murmur due to patent foramen ovale should be *presystolic* in time. George Johnson is of a similar opinion. The fact of the matter is, that the murmur may be either systolic or presystolic. Systolic, says Foster, when there is high pressure in the venous system, and the direction of the current is from right to left auricle, or is due to *vis a tergo* of a powerful right ventricle, the current is then from left to right auricle. Presystolic when from unusually strong *left* auricle there is a flow through the foramen into right auricle. Congenital disease of the pulmonary artery, or orifice, will always present cyanosis, in some cases marked blueness, in others sallowness or the whiteness of profound anæmia alternating with cyanotic attacks. The murmur is usually systolic, roaring or rolling, superficial, and apt to be rough. In two of Sansom's cases tactile thrill was appreciable.

Aortic stenosis is of such extreme rarity either as a congenital affection, or one of infancy, that we may almost exclude it from consideration. If murmurs occur at this region they are generally hæmic in origin. West, however, says that even these are rare before the seventh year.

The following summary of congenital murmurs, after Sansom, is, we think, of sufficient interest to be inserted here:

(1) Cyanosis with no murmur; *patent foramen ovale.*

(2) Cyanosis with systolic or presystolic murmur, varying

in intensity over sternal ends of third and fourth costal cartilage or third intercostal space; probably *patent foramen ovale.*

(3) Cyanosis with *loud,* unvarying systolic murmur at apex, and heard in back between scapulæ. There is probably imperfection in the *inter-ventricular septum.*

(4) Cyanosis or *marked* anæmia, with superficial systolic murmur at base; constriction at pulmonary orifice, possibly complicated with hæmic murmur.

(5) With congenital affection of heart and dilatation of left cavities; probably endocarditis of valves has been added.

Without an accurate and reliable clinical history, and a general consideration of all the symptoms, a diagnosis cannot be made by the physical examination, which, in itself debarred of this knowledge, is of little avail in elucidating the problem.

Prognosis.—It seems to be an established fact in congenital defects, as it is in acquired disease of the heart, that the prognosis does not depend so much upon the valve that is diseased, or upon the amount of stenosis and obstruction, or insufficiency and regurgitation, as it does upon the ability of the cavities to perform the work allotted to them. This secondary compensating alteration is the keynote in the prognosis.

It is said that less than eight per cent. of infants with malformed hearts die within the first week, and only thirty-six per cent. within the first year.

Of the immediate cause of death in infancy, about twenty-five per cent. die of dyspnœa, fifty per cent. of convulsions, and about twenty-five per cent. die of acute intercurrent disease, as hæmoptysis, affections of the brain, tuberculous disease, or intestinal and portal hemorrhage. Certain defects allow of a longer maintenance of life than others, as, for instance, if the ductus arteriosus remains pervious, and is the sole abnormality, life has lasted for the following number of years: nineteen, twenty-three, thirty-two, forty-eight, fifty-two. Sanders records a case which, however, succumbed at the fourth and a half month.

Stenosis of the aorta exerts but little influence upon the life of the individual; the left ventricle is so well able to hypertrophy, and thus overcome the stenosis, that the patient is

hardly aware of its existence. Not so, however, if the pulmonary artery is narrowed, as life is here jeopardized from the inability of the right ventricle to sufficiently hypertrophy, and from the fact that stenosis of the pulmonary artery is liable to be accompanied by other congenital defects. However, cases of complete stenosis of the pulmonary artery have been recorded, in which the subjects reached the ages of sixteen and twenty-one years. Life has been maintained until thirty-seven years with stenosis and atresia of the pulmonary artery, with, however, patent septum ventriculorum. That cases may even grow old is shown by the case recorded in the *Peninsula J. M.*, Ann Arbor, Michigan, 1853–54, i. 213, in which a man with a patulous foramen ovale reached the age of sixty-six years. A woman with stenosis of the pulmonary artery, and patulous foramen ovale, with complete obstruction of the ductus arteriosus, reached the age of fifty-seven years, and eventually died of cerebral apoplexy.

In cases of stenosis of the pulmonary artery, with incomplete ventricular septum, Kussmaul has observed the following duration of life. From birth to 1 year, 8 cases; 1 to 5 years, 14 cases; 5 to 10 years, 19 cases; 10 to 20 years, 14 cases; 20 to 30 years, 9 cases.

Kussmaul has also shown that in almost complete stenosis or obliteration of the vessel the expectancy is markedly less: from birth to 6 months, 10 cases; 6 months to 1 year, 4 cases; 1 year to 5 years, 5 cases; 5 years to 10 years, 3 cases; to 21 years, 1 case; to 37 years, 1 case.

In transposition of the main trunks life is not liable to be long maintained; the subjects rarely reach even early adolescence. Four cases are recorded in which the little patients reached between two and three years of age.

Treatment.—We can expect to have but little, if any, effect upon grave cases of congenital cardiac defects; certain abnormalities are totally incompatible with life, and these cases speedily succumb to the unequal contest.

In other cases, by the maintenance of rest, both of body and mind, when we fear that the compensation is about to break, the exhibition of nutritious, easily-digested food, with attention to the gastro-intestinal tract and the larger abdominal glands,

together with a carefully selected climate, with protection against cold or sudden chilling of the circulation, is about all that we are able to do in these cases.

CYANOSIS.

CYANOSIS, or morbus cœruleus, sometimes styled the "blue disease," is to be considered as among the most prominent and characteristic symptoms of congenital heart-disease. It may also, however, be met with in the acquired variety, whether it happens to be an endo- or a pericardial inflammation; it may also be present in hydrops pericardii or accompany an active pericardial effusion, so that we conclude that cyanosis is better considered as a symptom than as a disease *per se.*

Cyanosis is in all probability caused by stasis in the venous current independently, to a certain extent, of the admixture of venous and arterial blood, though should this mingling of the two forms of blood occur the blue discoloration will probably be more marked and persistent. Many cases prove, however, that there is no positive relation between the amount of this admixture and the degree of discoloration. For example, cases are recorded in which the aorta arose from the right ventricle, and others in which the heart had only two cavities, the cyanosis not being constant or very well marked. In most congenital cases the conditions which cause venous stasis are present in their most favorable aspect, accompanied by a very faulty venous return. Some authorities (Morgagni, Stillé) consider that cyanosis is alone due to the first mentioned cause,—*i.e.*, venous stasis; others attribute to the latter—admixture of blood (Hunter and Gintrac), the main causative agent in the blue discoloration; it would seem, however, that both are right, and that it is the association of the two causes in most congenital cases that renders the symptoms the more evident, although, as above mentioned, cases are on record in which there was complete admixture of the blood without any cyanosis whatever. Another factor, in making the symptom the more appreciable, is the extreme pallor and the thin skin which these cases usually present, thus making the blue color the more evident. Again, the entire quantity of blood receives less oxygen and, per consequence, is much darker than normal,

assisting in making the patient even more livid. In so far, then, as the blood is in a pathological state may cyanosis be considered as a separate disease, and, if we consider that the blood is deficient in oxygen, and contains an excess of carbonic acid with carbonaceous products, we may classify cyanosis with the blood-diseases. Most cases of persistent cyanosis in the early years of life are due to congenital defects in the centre of the circulation,—that is, the heart or the great vessels. *Sex* appears to offer a direct predisposing factor, as most cases are recorded as occurring among males. Of 134 cases collated by Lewis Smith, 78 were males and 56 females. Gintrac reports 28 males and 16 females; Stillé, 41 males and 31 females; and Aberle, in reporting 180 cases, says that two-thirds were males.

Upon examining the mortuary statistics we find, for example, that New York reports 207 deaths in a year from cyanosis, 117 males, 90 females; and that the city of Philadelphia, for the year 1876, returns a total of 100 deaths from cyanosis, 53 males, 47 females: 94 of these were under one year, 5 at one year, and 1 at two years. The report of the same city for the year 1882 shows, however, a nearly equal distribution between the two sexes: total deaths 139; males 69, females 70; 135 under one year, 2 at one year, 2 at two years. England presents for two years 691 deaths from this cause, 418 males and 273 females.

A point worthy of note is the fact that the later statistics do not show such an excess of males as are reported by the earlier writers.

Most cases of cyanosis will be met with in large cities and among the so-called lower classes. Those who have a perpetual struggle for an existence, in damp, ill-ventilated apartments, with scanty food and deficient clothing, together with wretched hygienic surroundings, are most apt to give birth to cyanotic babes. Smith cites the following statistics in proof of the fact that cities return the largest proportion of cyanotic cases. In New York City, for six years, there was 1 death from cyanosis to 436 deaths from all causes. In Brooklyn the proportion was about the same. The converse of the proposition is shown from the fact that the whole State of Kentucky, for a

period of five years, reports but 1 death from cyanosis to 2469 from all causes. This line of argument could be carried on to great length, but enough has been cited to prove the action of agencies which enervate the system and destroy the health, in causing the birth of children with cardiac malformation and cyanosis. As we stated when considering the symptoms of congenital heart-disease, the cyanosis depending on congenital deformity may not show itself for some time after birth. Smith presents an interesting table, showing the time of appearance of cyanosis in 41 cases of congenital defect: in 3 at two weeks, 1 at three weeks, 2 at one month, 7 from one to two months, 5 from two to six months, 5 from six to twelve months, 3 from one year to two years, 6 from two to five years, 1 from five to ten years, 6 from ten to twenty years, 1 from twenty to forty years, 1 over forty years.

How well does this table illustrate the fact, to which we have already called attention, that the growing heart possesses a certain inherent power to conform itself to a disordered blood-circulation, and that a congenitally crippled heart may in certain instances carry on life to full adult age, giving the subject but little if any discomfort until some seemingly trifling incident aids in "breaking the compensation." On the other hand, the developing cause may be such a profound impression on the system as to give rise to the question as to whether it has not in itself crippled the heart independently of any congenital malformation that may have existed; for example, the case of Waters,* in which cyanosis developed in a child aged six, during an attack of measles, and remained persistent. Here it is to be decided whether the morbilli itself did not attack the endo- or pericardium, and give rise to a case of acquired cyanosis. Another example is the case of Napper,† in which a six-months' babe received a severe fall, and was cyanotic forever afterward. Steadman‡ records a case of an infant, æt. ten weeks, who presented coincidently convulsions and cyanosis. Cases could in this way be indefinitely cited, but enough has been advanced to show that when cyanosis develops we must

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* Phila. Med. Exam., June, 1850.

† Lond. Med. Gaz., 1841.

‡ Lancet, London, 1842.

decide whether it is congenital in origin or due to an acquired disease; after congenital cyanosis once appears, there is then but little improvement in the patient, as the probability is that the compensation has become wholly inadequate, and the cavities are dilated or dilating.

The symptoms, prognosis, modes of compensation, and treatment have already been considered under the head of congenital heart-disease; let us then conclude by citing a few illustrative cases from our hospital and private practice.

CASE I. J. W. was fourteen months old when he first came under observation; face œdematous, hands and feet pitting on pressure, extreme anæmia. On auscultation a loud, rough, systolic murmur or bruit, most marked in pulmonary area, but heard also with great clearness at the apex and in the left axilla. The child developed marked cyanosis six weeks after birth coincidently with a sharp attack of bronchitis, this latter becoming chronic, and persisting until death.

The liver and spleen were enlarged when the child first came under observation, and continued so throughout. Ten months after we first saw the child, or at its twenty-fourth month, the little patient succumbed to an attack of measles, the immediate cause of death being capillary bronchitis, "suffocative catarrh."

Post-mortem, eight hours after death.

Heart. Right heart in a condition of dilated hypertrophy. Ductus arteriosus obliterated. The right auricle markedly dilated, foramen ovale patulous and valves very deficient; indeed, the intra-auricular septum was almost absent, the orifice was quite the size of a twenty-five-cent piece; nature had evidently attempted to close this unduly large orifice by membranous bands, and had been partially successful. The tricuspid orifice was widely dilated. Right ventricle dilated, pulmonary valves almost normal, orifice slightly dilated.

Left auricle was small, walls thin, mitral valve decreased in proportion to size of left heart, barely admitting tip of little finger; the left ventricle was also small, walls thin; aorta normal.

Lungs. Signs of capillary bronchitis, with here and there foci of recent catarrhal pneumonia.

Case II. The following case was observed on the post-mortem table of the Philadelphia Hospital: Baby S., a foundling, æt. three weeks. It was stated that the babe was blue when born, and when it came under observation was markedly cyanotic; lips, tongue, mucous membrane, and finger-nails all blue. In this case we were unable to detect a murmur with absolute certainty after repeated careful examinations. The child remained cyanotic nine weeks after admission; it died during a convulsion.

Post-mortem, twenty-four hours after death.

Heart. Left ventricle increased in size, cavity much larger than normal, walls thicker; aortic valves and aorta healthy. The left auricle was also somewhat increased in size, the mitral orifice dilated, admitting the index-finger very readily.

Right auricle in a condition of marked dilated hypertrophy, coronary sinus dilated, foramen ovale patulous, and the orifice of the tricuspid valve a mere slit, admitting simply a small probe. The right ventricle was enormously hypertrophied, with decrease in containing capacity, the walls were in some places three-quarters of an inch in thickness; on this side of the tricuspid orifice the leaflets were found to be adherent and imperfectly developed.

The orifice of the pulmonary artery was obliterated by the adherence of the semilunar valves one with the other. The ventricular septum was perfect, no communication existing between the right and left ventricle.

The ductus arteriosus was patulous, and would allow the point of a penholder to pass.

Lungs. Crepitant; scattered throughout both lungs were areas of pulmonary apoplexy.

Case III. This boy, æt. three years, well illustrates the effect of an incomplete septum ventriculorum. During life the child presented a bulging præcordia, with impulse noticeable over a very large area, cardiac dulness extending from lower border of second rib to upper border of seventh. Palpation revealed a marked systolic thrill and auscultation a loud, rough murmur, systolic in time and heard over the entire præcordia.

Post-mortem.—Heart enormously enlarged, weighing almost

as much as an adult heart. The foramen ovale was patulous; in the septum ventriculorum, at the part most remote from the apex, was an opening about the size of a ten-cent piece.

The tricuspid orifice was dilated, admitting first and second fingers to second joints. The mitral valve and orifice normal. The muscular structure of the left heart was much thickened, of the right much thinned.

That nature endeavors to effect a mode of cure in these cases was well proven by the post-mortem that we are now considering; the abnormal communication between the two ventricles was surrounded by an irregular corrugated arrangement of tissue as though an effort had been made to supply a bridging tissue or a series of little valves in order to correct the primary defect; this corrugated membrane was attached all around the opening except at one small part near the tricuspid valve, here were to be noted a series of cords seeming to interdigitate with the tricuspid leaflets. The child died of acute pneumonia.

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CHAPTER II.

ACUTE AND CHRONIC ENDOCARDITIS—ENDOCARDITIS IN THE FŒTUS AND NEW-BORN.

Endocarditis is an inflammation of the lining of the heart. This membrane, the endocardium, is a continuation of the membrana interna of the arteries, is non-vascular in character, and lies upon a connective-tissue basis, over the auriculo-ventricular valves; its internal coat or that over which the blood courses is composed of a layer of flat cells lying upon a fibro-elastic layer and connected to this layer and also to the basement membrane by connective-tissue fibres. It is through this net-work of connective tissue that the vasa vasorum find their way. The fibro-elastic tissue and the flat epithelial cells are most highly developed on the auricular surface. As regards the blood-vessels, the capillaries, which course along the cardiac walls beneath the endocardium, cease to exist in the sigmoid valves, and but few are found in the layers of the mitral.

Ætiology.—Acute, so-called idiopathic, endocarditis is probably extremely rare as a primary disease; indeed, it is doubtful if it occurs at all. Most of the cases that have been considered as idiopathic depend upon a primary alteration in the quality of the blood, either affecting its chemical composition, altering the normal constituents, or else depending upon some external morbific material which has gained entrance by contagium or is due to deficient elimination and excretion, allowing excrementitious matters to circulate over the endocardium in the blood-current.

Acute endocarditis is, however, proven to arise as a secondary affection in the course of certain diseases, as, for example, it is seen in certain diatheses; notably the lithæmic or in the course of essential zymotic fevers, as measles, variola, varioloid, and typhoid fever. It occurs often in pneumonia and pleurisy; it may also be met with as a manifestation of pyæmic invasion, producing the intercurrent variety and leading to ulcerative changes.

Mild attacks of endocarditis, possibly amounting to mere congestion and slight proliferation of the endothelial elements, directly predispose to more severe attacks.

Blache, however, believes that in the child primary endocarditis may arise independently of any other disease.

Authorities are not agreed as to the number of cases of endocarditis which complicate or are caused by rheumatism. It is our opinion that rheumatic endocarditis is more frequent in the child than in the adult. Vernay has found that of twenty-one cases of rheumatism between the ages of fourteen and twenty, only *one* escaped endocarditis.

D'Espine and Picot found in forty-seven cases of rheumatism in children only ten cases in which the sounds of the heart were perfectly normal.

Senator is responsible for the statement that the younger the patient the greater the risk of the heart becoming affected, as this organ is implicated in fully one-third of all the cases of rheumatism occurring before puberty.

Roger and Jaccoud have observed cases where the symptoms of endocarditis preceded for fifteen days the articular manifestations of rheumatism.

Scarlatina is frequently associated with rheumatic manifestations; hence endocarditis is not infrequently seen in connection with the two diseases. The acuteness of the attack of rheumatism, either simple or complicating scarlatina, will probably give evidence of endocarditis in cases where a less rapid onset and a milder sequence of symptoms would not direct attention to the heart; indeed, it is a well-established fact that chronic rheumatism may acutely invade the heart as well as the more acute rheumatic manifestations, and Gubler has seen it associated with muscular as well as articular rheumatism. It may be expected in all cases when the blood is altered in its characteristics or when it is contaminated by excrementitious or septic matter; in the latter we would include the whole field of bacteriology.

The association of endocarditis with erythema nodosum has been twice noted in children aged seven by Martineau. Chorea may be associated in an ætiological relation with the production of endocarditis.* Should endocarditis arise during typhoid fever, it is apt to be masked by the more grave symptoms of the primary disease as illustrated by the case of Magnan, who observed a case of typhoid fever in a girl æt. fourteen,

* Note.—See Addenda, No. II.

of an adynamic type; death occurred on the fifth day of the disease. During the progress of the disease the heart presented no symptoms, but at the autopsy some pericarditis was noted with effusion, the endocardium was affected around the mitral valve. Ulceration, however, had not yet arisen. As we see by this case, endocarditis may be caused by or associated with pericarditis, the same ætiological factor being the causative agent in both affections.

The association of endocarditis with phlegmonous periostitis or coxalgia in the young has been recorded. This Kirk noted in St. Bartholomew's Hospital in a boy æt. fourteen. The mitral and aortic valves were attacked, many emboli were found in distant organs. It may also be associated with osteomyelitis. Post-mortem examinations have definitely proven this association.

Classification.—As we shall see, the disease presents symptoms more or less marked in proportion to its severity, and these symptoms are due not to a different classification of the varieties but simply from classifying stages as separate and distinct forms of the same disease. Accordingly, then, we might classify endocarditis under three headings, (1) acute, (2) chronic or interstitial, (3) ulcerative. The second may be simply a chronic stage of the first, or may be developed primarily as a subacute or chronic form resulting from constitutional vice, as syphilis or rheumatism. The ulcerative form is in many cases almost totally independent of a well-definable cause.

Symptoms and Diagnosis.—The disease is peculiarly prone to be latent in the child, and the diagnosis of endocarditis—and by diagnosis we do not mean simply the detection of an inflamed endocardium, and the localization of its site—has been the subject of an almost endless amount of literature, a tangled mass of material found in text-books and journals, affirmations and denials, that probably have led to more confusion in the medical mind than has any one subject in the literature of medicine. The result is that the practitioner who has not been taught at the bedside is as much at sea as regards the diagnosis, prognosis, and treatment of the affection, as was the physician of the last decade. We must always remember that

in studying cardiac diseases we have also to do with a vital fluid, capable of undergoing changes daily, yes, even hourly, in its composition and texture, such as its fluidity, coagulability, and plasticity, a total change in its constituents as regards their physical and chemical properties, and also by the addition of adventitious matters which may be taken up by it in its course through the vessels or by development within the fluid itself,—bacilli. We must also remember the character of the flow, its pressure, its force, and resistance, the regularity or intermitting of its rhythm, and also certain vibratory movements, which are transmitted by continuity and contiguity; also bear in mind that the cavities of the heart and blood-vessels are lined by vital structures, that they are variable in size, and that they are capable of being influenced through that subtle system which controls vitality, and to which we attribute trophic changes.

Taking all these matters into consideration, and bearing in mind that the subjective symptoms are few indeed, we are to rely alone upon our own sense of observation for a diagnosis, gathering what we can obtain from the general constitutional condition of the patient or from the subjective symptoms.

The *temperature* is apt to be extremely irregular, in fact is almost intermittent in type, fluctuating between 103° or 104°, and 99° or 100° during the acuteness of the attack.

Local Signs.—If the child is old enough, it will complain of a pain in the left axillary region; most young children will, however, locate the pain at about the ensiform cartilage; they seem to place their hand on this region instinctively. If the aorta is involved, we note shooting pains along its tract, increased by position. The further local symptoms will depend upon the character of the inflammation; in the mild forms, or those of simple hyperæmia and slight exudative changes, the symptoms seem to be in proportion to the depth of the inflammation. If the myocardium is at all involved, palpitation becomes a marked symptom, together with præcordial distress; as the disease advances the heart's action becomes tumultuous, *delirium cordis.*

Percussion in the early stages of acute endocarditis is indefinite; in fact, it is not of much value as a physical sign in

acute endocarditis, where the cavities have undergone no changes.

The *pulse* is at first somewhat accelerated; later, however, when the cardiac muscle becomes involved in the process, it becomes feeble and dicrotic.

Respiration is markedly affected, dyspnœa arising very early in the case; indeed, in children suffering from disease with a known tendency toward the endocardium, the intervention of sudden and alarming dyspnœa, arising independently of pulmonary affection, may be the first indication that the attendant will have of cardiac implication. Cough may or may not be a prominent symptom, depending, as it does, to a great extent, upon the presence of dilated cavities, with inefficient systole and venous stasis.

Nausea and vomiting may arise early in the case or toward the close may constitute an alarming symptom, threatening life by exhaustion and inanition.

The patient may rapidly sink into an adynamic state, deepening into a profound typhoid condition, and death occurs from asystole. As a rule, great irregularity and tumultuous action of the heart show increased severity of the disease, the extent of the impulse and the impingement of the apex-beat becoming more marked as the case progresses. The obstruction in the circulation is further evidenced by the condition of the neck-circulation, the veins being distended and the arteries throbbing. In cases of endocarditis complicating rheumatism or in the exanthemata, the subjective symptoms may not be at all well marked. It must be borne in mind that the subjective symptoms in children have not the same significance as when they occur in an adult.

Physical Signs.—These are the class of symptoms upon which we are more apt to base our diagnosis, and are discoverable by conjointly inspecting, palpating, and auscultating the region under consideration. In all cases of scarlatina, kidney-disease, acute or chronic rheumatism, chorea, measles, and typhoid fever, the examination of the heart by auscultation should be made with care, as it is said that in about fifty per cent. of the cases of rheumatism in childhood the mitral valve will be affected.

Auscultation will generally reveal a systolic bruit or murmur at the apex. The murmur is sometimes heard with startling distinctness in children. At this age it is also more apt to be permanent. The abnormal sound may not alone be localized to the apex, but may be transmitted in various directions, even into the arterial system. It may be difficult to distinguish and correctly interpret this murmur, as in children a pericardial bruit often bears a striking resemblance to an endocardial murmur. Again, hæmic murmurs in the young child are at times differentiated only with the most extreme care. The character of the murmur in acute endocarditis will depend greatly upon the rapidity of the circulation, the character of the blood, and the condition of the endocardial surface over which the fluid passes, and will be further influenced by the degree of the inflammation.

Palpation reveals to us an irregularity in the heart's impulse, a more or less violent throbbing, and, possibly, should the inflammation be extensive, the presence of a vibratory thrill. It is also to palpation that we are indebted for evidences of involvement of the heart's structure, the myocardium, and for points in connection with the differential diagnosis of peri- or endocarditis; or where these two affections are associated it also gives us the exact location of the apex-beat, which is not only changed but frequently disappears altogether in pericarditis with effusion. In endocarditis we are able to recognize great diminution in the force of the apex-beat, or its change in position when severe endocarditis has attacked the lining membrane of the left ventricle, with involvement of the muscular structure and engorgement and over-distention of the cavities.

Inspection not alone will show us irregularity and tumultuous action of the heart, but will also advise us in regard to the condition of the circulation generally. In children the veins of the neck become turgid more readily than in adults; indeed, the presence of endocarditis affecting the mitral valve will, in a large number of cases, so impede the circulation through the pulmonary artery as to produce engorgement of the right heart and a tricuspid insufficiency to such a degree as to produce a murmur at this valve. Engorgement of the

systemic veins would naturally be a result, the damming back of the blood-current through the lungs accentuating the second pulmonary sound, and either actually or relatively masking the intensity of the first sound. Indeed, the relation of this secondary effect of the damming back of the circulation upon the right heart is said to give us a tricuspid murmur in fifty per cent. of all cases where recent mitral disease exists. This murmur is generally heard over the body of the heart, and is accompanied by vibration. As will be seen farther on, mitral disease occurring most frequently in childhood, and at this period of life causing much interference with the action of the valves, giving rise to obstruction and regurgitation, the large number of secondary tricuspid murmurs heard at this period of life is accounted for. It is a rare clinical fact for endocardial inflammation to localize itself at the aortic orifice during childhood. In children one is apt to find a soft pericardial friction that may be mistaken for an endocardial murmur.

Prognosis.—The prognosis is not necessarily fatal; it, however, must be guarded. Most children recover from the first attack, with, however, in all probability a chronic endocardial change, making the patient prone to subsequent attacks, or the subject of chronic valvular disease in later life. This permanent injury to the valves is always to be dreaded. When exudative changes are rapid in onset, the carrying of emboli with splenic engorgement, hemiplegia, pulmonary disturbance, and kidney-infarction will show an acute ulcerative process. This complication is especially apt to occur in all forms of septic disease, with endocarditis as a concomitant, for example, in scarlatina or diphtheria. Children, however, are more apt to be carried off by pulmonary complications, as engorgement or catarrhal pneumonia. Simple endocarditis complicating rheumatism or measles or scarlatina, barring its tendency to permanently affect the valves, is very rarely of itself a cause of death.

Post-mortem Lesions.—In making examinations of the heart in the young for suspected endocarditis, it is necessary to distinguish post-mortem imbibition from the slighter degrees of endocardial injection; the color generally aids us, as that produced by inflammation is bright red or scarlet and that

from imbibition is violet; furthermore, the right heart usually presents the imbibition most clearly, and the left the signs of endocarditis. The cavities generally contain clots. Endocarditis usually selects primarily the edge of a valve, and the first change is simply one of consistence, proportionate to the degree of inflammation. Rarely do we find that the process has been arrested at this stage, but we may find that the case has been one of simple congestion with abnormal injection of the endocardium, most evident around the orifices of the left side. This process, as we have already stated, may be arrested here; but, if it goes on, vascularization and infiltration soon set in and the exudative stage is established. Blache has had the good fortune to study the initial stages of this disease on the post-mortem table at a very early date. In the infant the presence of exudate is most characteristic; this is accompanied by hyperæmia, increase in the size of the valve, whose surface becomes dense and rough and probably studded with vegetations. A more or less clear serum will be noted infiltrated into the surrounding tissue, the epithelial cells increase in size with infiltrated nuclei, the cells proliferating and becoming detached. Should a fibrous exudate occur, which, however, is more rare in children, the changes in the valves, of course, will be more marked and recognizable.

The exudate speedily becomes organized and vegetations form; those at the base consist essentially of the same form and structure, but they differ toward the periphery, as, for example, granular epithelium with hyaline nuclei or subendothelial nodosities, which look not unlike Paccinian corpuscles, or we may note fibroid growths sessile or pedunculated, condylomatous or papillomatous excrescences, or again callosities, which may be large and diffused. The membrana interna may take part in the process and become hyperplastic; this may extend into the aorta and produce atheroma, which, however, is somewhat unusual in the early years of life.

Ulcerative or purulent endocarditis may be noted in the heart of an infant, in the centre of a fibroid concretion, about the valve pus may be noted. In the infant, as in the adult, the lesions of endocarditis may be entirely localized to the left heart; they may, however, be met in the right heart also.

CASE IV. Mary A., æt. eight and a half,—this case occurred in our private practice,—had been under observation for some time, had irregular and ill-defined rheumatism of no definite type; child was somewhat anæmic.

Suddenly the signs of acute endocarditis were developed,—a loud apex-murmur, systolic in tone and audible in the axilla, was to be heard with great distinctness. The temperature rose to 102°, with morning remissions, presenting the characteristic type of intermittent temperature-range so often seen in these cases; face was puffy and feet œdematous.

Breathing became much embarrassed; hebetude developed, deepening into coma, from which she was aroused only with great difficulty; paralysis of the face developed; the child passed into convulsions, during which she died, four weeks after the endocardium was first affected. Percussion revealed an enlarged liver and spleen. The urine contained albumen, but no tube-casts were found at any time.

Post-mortem showed the evidences of recent endocarditis about the mitral valve and over the auricular endocardium. The case presented further lesions in the arterial system, which we will refer to under the appropriate heading.

ENDOCARDITIS IN THE FŒTUS AND NEW-BORN INFANT.

Rauchfous, of St. Petersburg, makes the statement that in several years he has encountered three hundred cases of fœtal endocarditis. Of this number the right heart was affected in one hundred and ninety-two cases, the left alone in but fifteen instances. The relatively greater frequency of fœtal endocarditis in the right chambers has been well recognized by all observers; it is stated in explanation that the increased blood-pressure on the pulmonary leaflets is the cause of the localization of fœtal endocarditis to the right heart. It is a fact that most congenital cardiac diseases are located at the pulmonary orifice.

The endocardial hyperplasia in this class of cases is generally a soft, red, pedunculated vegetation arranged at or about the tricuspid valve. Sometimes they are met with at the mitral valve, but rarely do we see them on either the aorta or pulmonary artery.

In the Hospital Necker, Blache has observed two cases of fœtal endocarditis in twins dead just after birth. In both hearts he noted soft red globular vegetations at the mitral valve.

Most of these cases of endocarditis have a connective-tissue proliferation as a base for the vegetations. Arising during fœtal life, they may, if the child survives, disappear, but endocarditis of intra-uterine life is, unfortunately, only too apt to result in organic changes giving rise to cyanosis at birth.

The diagnosis of endocarditis in the new-born is of course a difficult task. Cyanosis will be the most marked and characteristic symptom. Over the entire body may be noted a bluish discoloration; dyspnœa will be present, and the extremities cold, due to deficient circulation. Auscultation will possibly reveal a systolic bruit transmitted in the direction of the circulation; extreme palpitation and tumultuous cardiac action will not infrequently be noted over the entire præcordia. Death as a rule takes place within a few days after birth, when the diagnosis may be verified by the presence of the characteristic lesions. Hayem records the following case: Child died three days after birth; the mother had pneumonia at the time of confinement. Examination of the child's heart showed lesions of the tricuspid valves; the mitral and pulmonary were also affected, but the aortic was free. Right auricle was distended by clots; in the ventricle they were adherent to the columnæ carnæ. The meninges were infiltrated, the vessels ruptured, and hemorrhage had taken place into the cavities; left choroid plexus was thickened and covered with clots; the internal jugular and the sinuses also contained clots. Kidneys and liver were congested.

ULCERATIVE ENDOCARDITIS.

This disease has received various names by different writers, for example, *infectious*, *septic*, *arterial pyæmia*, *diphtheritic*, *mycosis endocardii*, and taking its name from the disease with which it is intercurrent, we cite the terms *scarlatinal* and *rheumatic* endocarditis as examples.

The appellation *acute ulcerative* is, however, in general use, and is perhaps as applicable as any that we could select;

malignant, however, could with propriety be associated in the title of many cases. Some authorities designate those cases in which the bacteria play an important rôle as malignant bacterial endocarditis.

As a rule, in childhood, this condition, which is the result of purulent emboli or necrotic changes in the interior of the heart, follows such diseases as diphtheria, the infectious diseases, or suppurative disease of the bone or joints. According to Osler, ulceration of the endocardium may be due to any of the following conditions: (1) rapid exudative process which cuts off the circulation at the apices of the papillary elevation; (2) degenerations of neoplastic tissue with softening of the villi or efflorescences which will be swept into the current, leaving ulcers; (3) minute abscesses in the valves beneath the endocardium which rupture and leave ulcers. The possibility of a primary protopathic endocarditis must, however, be recognized as, at least, a possible occurrence. Of course, we may have all stages and all degrees of severity of these vegetations and ulcerations, from the soft and friable vegetation to the purulent slough which teems with bacteria. In childhood this condition is usually secondary, being similar in this respect to tubercular nodules or ulceration into the endocardium, which are probably never primary at this site.

We cannot better describe the microscopical and macroscopical appearances of the parts attacked in ulcerative endocarditis than by quoting from the admirable article of Osler:*

"The study of a small fresh endocardial vegetation shows it to be made up of cells derived from the subendothelial layer, round and fusiform, which by their proliferation have produced a small nodular projection on the surface of the endocardium. Varying with the rapidity of the growth, the mass will present the characters of a soft granulation tissue or a tolerably firm fibrous outgrowth. Usually the round cells predominate, but there may be many elongated, spindle-formed cells with three or four processes. What part the endothelium plays in this growth has not been determined. Tiny out-

* Med. News, March 21, 1885.

growths may be seen in which the process appears to be entirely subendothelial, but usually before the mass attains any size the smooth surface is lost, and there is deposited upon it a cap of fibrin in the form of a granular, sometimes stratified, material, of variable thickness. Though this resembles an ordinary coagulable exudation, it is probably deposited directly from the blood, and is of the nature of a thrombus. Upon and in this layer may be found, sometimes in large numbers, those remarkable little bodies which have so long been known, when collected together, as Schultze's granule masses, and which have of late become so prominent as the blood-plates of Bizzozero and the hæmatoblasts of Hayem. Occasionally they are very abundant, and I have seen soft warty vegetations composed (superficially) in great part of them.

"The larger vegetations, more characteristic of malignant endocarditis, consist of a granular material composed of altered and dead tissue elements, fibrinous exudation, and colonies of micrococci; the deeper parts present the appearance of a granulation tissue, while at the attachment in the valve there is always more or less infiltration and increase of the cell elements. The granular substance is structureless, and resembles diphtheritic exudation, the resemblance at times being so close that one can readily understand the application of the term "diphtheritic" to the inflammation. It may be distinctly laminated, and with a high power fine filaments can be seen, though usually the granules conceal all appearance of structure. Strands of translucent material may occur throughout the mass, as if portions had undergone a sort of hyaline transformation. In some instances it is very marked. Pale spheres filled with granules also occur, and may be very abundant. They have been described as colonies of micrococci, but some regard them as altered endothelial elements. I have seen them too numerous to be explained on this view. At the attachment of the vegetation there is a zone of tissue deeply infiltrated with leucocytes, and deeper still the tissue elements of the valve present an increase of nuclei and cells. The destruction of tissue appears to result in two ways: first, a gradual extension inwards of the necrotic process, doubtless

induced by the micrococci; secondly, the softening and separation of valve-tissue caused by the rapid development of leucocytes at the base of the vegetation.

"The micrococci are constant elements in the vegetations. All granules of a uniform size met with in the sections are not microörganisms, nor, indeed, are all which stain by some methods recommended for the detection of these bodies. By far the most satisfactory method is that of Gramm, in which the section, after staining in gentian violet, is transferred for a few minutes to a dilute solution of iodine and iodide of potassium, and then to the alcohol, when it is found that the color has been extracted from all tissue elements and nuclei, leaving only the microörganisms stained. They vary a good deal in number and arrangement, and may be scattered singly in the granular substance or arranged in groups. They are usually very numerous at the deeper part of the vegetations, just where the structureless material joins the granulation tissue, and they may penetrate deeply into the substance of the valve. Sometimes the smaller vegetations seem made up exclusively of them. Several of my specimens appear to confirm the view of Klebs, that the micrococci lodge first on the endocardium and penetrate into the substance, often as distinct columns. In their immediate vicinity there is a zone of necrosis, and beyond this an accumulation of leucocytes and signs of reactive inflammation. The microörganisms found in connection with malignant endocarditis are not all of the same kind. Klebs distinguishes two forms, one met with in septic and the other in rheumatic cases. In some instances the micrococci are all arranged in zoöglœa-like masses, in others, particularly the septic cases, they are in chaplets. Some present distinct capsules. Small elongated bacilli have also been found; I have seen them in one instance—short, stout rods often joined in pairs. Delafield and Prudden have recently noted the presence of bacilli in the vegetations of a very acute case of malignant endocarditis. Cornil, in a recent lecture, stated that the bacillus tuberculosis had been found in the vegetations on the valves in cases of phthisis, and expresses the opinion that before long we should have accurate knowledge of a variety of microörganisms in endocarditis, depending

upon the nature of the primary disease. By culture experiments alone can we hope to have the question settled.

"Briefly stated, the theory of acute endocarditis which at present prevails, and the only one to which I shall refer, is that it is in all its forms an essentially mycotic process; the local and constitutional effects being produced by the growth on the valves, and the transference to distant parts of microbes, which vary in character with the disease in which it develops. This very attractive theory can be adjusted to meet every requirement of the case, though as yet lacking certain of those substantial data so necessary for full acceptance, but which, having been furnished of late years in other diseases, we may reasonably hope will in time also be forthcoming for this."

It is Prudden's opinion that the great frequency with which the acute ulcerative disease is engrafted upon an old endocardial lesion would seem to indicate that in the human subject the absence of endothelium, or the roughness of the surface of the thickened endocardium, affords conditions of predisposition for the lodgment, and vulnerability toward the incursions of the bacteria, when once they gain access to the blood, similar to those produced experimentally in the rabbit by mechanical or chemical means.

Ulcerative endocarditis is not a frequent disease in childhood; out of two hundred and nine cases but four were under ten years of age.

The history of these cases usually begins with some evidence of embolic infarcta, either into the spleen, kidneys, or brain. Such a case was observed by Professor Osler: the boy was aged eleven, had two attacks of chorea. He rapidly improved under five-minim doses of Fowler's solution, given every four hours. The first sound was murmurish; eight months afterward the patient suffered a return of the chorea, a week after its return he had a febrile reaction, the temperature reaching 104° F. Child became unconscious, slight paresis of left side was noted, and death occurred six days after the commencement of the disease; a temperature of 106° was recorded before death. An examination of the heart was made, revealing irregular, soft, grayish-white vegetations in

the mitral valves, infarcts in the spleen and kidneys, and a small spot of red softening in the right corpus striatum.

The symptoms of ulcerative endocarditis are peculiarly prone to be masked by the primary disease, or they may assume types and forms akin to other maladies. Recognizing this fact, authorities have named certain types of the affection as the *pyæmic*, *typhoid*, *cardiac*, and *cerebral* types.

Homolle's* case of a boy whose disease simulated typhoid fever, and then cerebro-spinal disorder, is of extreme interest. The child lived five days after admission to the hospital. Post-mortem revealed suppurative meningitis of the brain and cord, pneumonia of one lung, with marked recent ulcerative endocarditis engrafted upon old valvular disease. Heineman records a case in the *New York Medical Record*, ii., 1881, well worthy of notice here. The boy, æt. fourteen, was admitted to the hospital November 19, and it was stated that since the 17th he had suffered with pains in back and legs, chills, fever, anorexia, and vomiting. The bowels were constipated, tongue coated, temperature 105.2°; was rational on admission. An examination of the heart and lungs was entirely without diagnostic result. The next day the temperature in the morning was 103.4°; evening, 105.6°; fæces and urine voided involuntarily. In twenty-four hours an eruption appeared, which was purpuric, and first noticed on the face, extending to the legs and arms. Temperature was now 104.8°, pulse feeble, delirium marked; two convulsions followed each other rapidly, and the child died during the second seizure. At the autopsy recent vegetations were seen on the mitral, and at the apex on the anterior wall of the left ventricle a small cavity, probably due to necrosis and loss of tissue. Pericardium contained a sero-purulent fluid; a purulent exudation was noted in the brain, the meninges of cord were congested, opaque, and inflamed. Lower segments of both lungs were congested; kidneys showed abscesses, probably embolic in origin.

Heslop† records an interesting case to which one of us has

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* Bull. Soc. d'Anat., 1873.

† Med. Times and Gaz., vol. ii., page 245, 1856.

already called attention.* The girl presented no heart-murmur on admission, but the action of the heart was tumultuous and irregular. On patient's admission to hospital, she stated that she had had rigors, followed by flushes of heat, but never any rheumatism. She was a weak, anæmic girl, and died in convulsions. The part of the endocardium immediately beneath the semilunar valves presented an irregular, ecchymosed surface, and had the appearance of being undermined, leading to the base of the aorta. At the attachment of the middle and anterior segment of the valves was a mass of fibrinous deposit the size of a small walnut, surrounding a cavity, containing a recently-formed coagulum, pus, etc.; it did not perforate. It was noticed that two days before death a continuous blowing sound was heard, accompanying and masking both first and second sounds. The patient had vomited at the commencement of the attack, and there was epigastric tenderness; rigors set in early, and the skin was jaundiced.

Moxon's † case was that of a child admitted to Guy's Hospital with suppurative periostitis, death resulting from multiple cardiac emboli, causing abscesses; the kidneys were also affected.

Chance‡ speaks of a boy aged thirteen who, apparently in perfect health but with a scrofulous diathesis, was attacked one day immediately after eating with nausea and vomiting. The next day he became drowsy, and complained of feeling very sick, with pain in the stomach. Finally, complete coma set in with rapid and fluttering pulse, and occasional convulsive movements. He died in two days from the beginning of the attack. The autopsy revealed multiple cardiac ulcerations and perforations with pericarditis.

The consideration of our subject would, indeed, be incomplete without referring to the case reported by Drs. Tuckwell and Harris: § H. E., a boy, aged four years, was admitted to the Rad-

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* "Ulcerative Endocarditis." J. M. Keating, M.D., Trans. Col. of Phys., 1879.

† Medical Times and Gaz., vol. ii., 1872.

‡ Lancet, London, vol. i., 1846.

§ Ibid., 1885, pp. 516, 517.

cliffe Infirmary on November 25, 1884, and died on the 30th of that month. The child was so ill that no very detailed examination could be made. Child had been previously healthy, in fact had only suffered from an attack of measles. From this he did not entirely regain his previous robust health. He seemed, however, well until three weeks before admission, when he lost appetite, coughed, and had a hot, flushed face. On admission, pulse, 180; temperature, 101°; respiration, 54. The next day child became pale and cyanotic; temperature, 103°; respiration in the evening, 66; pulse, about 180; exact rate could not be determined. During the next twenty-four hours a faint apex murmur, systolic in time, was audible; remained about in this condition until death; the murmur, however, having become louder, the temperature more fluctuating, pulse irregular, coma, lung consolidated, and extreme diarrhœa; death closing the scene five days after admission.

The post-mortem, made seventeen hours after death, revealed extensive ulcerative endocarditis, affecting the orifice of the pulmonary artery, a patent foramen ovale, and a communication between the right and left ventricles; numerous pulmonary infarcts were also noted. The heart was considerably hypertrophied, weighing four ounces; apex was formed entirely by right ventricle. Pulmonary artery was completely blocked by a firm thrombus, which was markedly adherent to the walls of the vessel; when this thrombus was torn away it left visible an extensive, ragged, irregular ulcer, which extended from the origin of the pulmonary artery upward a distance of an inch and a quarter into the vessel; it was not limited to one side of the vessel, but extended all around it. The semilunar valves appeared to have been destroyed completely. No tendency to perforation of arterial walls was evident.

Tricuspid valve was healthy; orifice dilated; endocardium of right auricle and ventricle healthy. Foramen ovale admitted a No. 12 English catheter; the two ventricles communicated by an opening in the upper part of the septum; these two openings were congenital. Mitral valve healthy, as were also the aorta and its valves. Pleuræ contained fluid, over their diaphragmatic surface were several punctiform hemorrhages.

Both lungs contained numerous recent infarcts. Spleen presented an infarct; weighed three ounces. Liver and kidneys healthy. No ulceration in any part of intestines, either large or small. Brain and membranes normal. The observers consider this case as one of *primary* ulcerative endocarditis of the pulmonary valves. The case during its course did not resemble in any way either the pyæmic or the typhoid forms of the disease.

The following case, recorded by T. Mitchell Prudden in the *American Journal of Medical Sciences* for January, 1887, p. 55, is well worthy of reference, particularly on account of the additional value of a microscopical examination in association with the clinical symptoms:

Mary C., aged fourteen years, had convulsions at eighteen months, scarlatina early in life, but no rheumatism, and was apparently well and strong. She was admitted to hospital February 22, 1886, for operation on club-foot. Cuneiform osteotomy was performed "antiseptically" on February 26, and for the first few days patient did well, but complained of pain in the foot. On March 6 there was an erysipelatous redness about the wound, and the temperature rose, but was reduced by antipyrin. She became delirious at times and unconscious; had twitching of muscles on left side, and died on March 10.

Autopsy.—Brain: meninges apparently normal; ventricles not dilated. Numerous small subpial hemorrhages, mostly quite superficial, on cerebral convolutions. A few hemorrhages were at the bottom of the sulci, and some of these involved small areas of the gray matter, which about them were soft and red. *Heart:* pericardium normal. Aortic and pulmonary valves normal. Scattered along the edges of the tricuspid and mitral valves, and nearly completely investing them, were irregular rows of larger and smaller white and red, firm, and closely adherent excrescences, some of them covered with loosely hanging red and white thrombi. On one of the papillary muscles of the left ventricle was a small eroded area loosely covered with a thin red fibrinous pellicle. In the left ventricle were also numerous small subendothelial petechiæ and one small abscess. Lungs normal. Spleen soft and large, and presented an irregular, grayish, friable area about one

centimetre in diameter. Kidneys moderately large, capsule free, studded with numerous small, yellowish-white spots surrounded by zones of diffuse redness. On section, the cortices were thickened, light in color, markings obscure. Everywhere, both in cortex and medulla, the cut surface was thickly besprinkled with small yellowish-white spots and streaks. The spots were from one to three millimetres in diameter, and many of them were surrounded by a sharp red zone. There were also numerous sharply circumscribed red spots. Liver presented several larger and smaller white, irregular areas, some of them very soft, having the appearance of infarctions. Other organs appeared normal.

Microscopical Examination.—The tricuspid and mitral valves are irregularly thickened, the new tissue being mostly dense and firm, and consisting largely of basement substance. In the superficial portion, however, the cells are more abundant and fusiform, stellate, and spheroidal. The surfaces of these—for the most part apparently old vegetations—are irregularly bestrewn with larger and smaller masses of very small spheroidal bacteria, arranged in pairs or in large or small irregular clusters, or lying singly. They stain readily by Gram's or by the simple fuchsin method. These bacterial masses are in part covered, in part intermingled with granular matter, a few leucocytes, fibrin, and a few red blood-cells. Beneath the mass of bacteria there is in most cases a larger or smaller area of necrosis of the vegetation in which the nuclei remained unstained, and the basement substance presents a translucent, finely granular or structureless appearance. In many places there is an irregular zone around the areas of dead tissue in which there is a greater or less accumulation of leucocytes. In general, the necrotic process in this case is not advanced, involving only little spots here and there on the surface of the vegetations where the tiny masses of bacteria have found lodgment.

The lesions of the other internal organs were those common to pyæmic abscesses and infarctions. The kidney presented microscopically a variety of phases in its lesions corresponding to the varied gross appearance. In some places the larger, but particularly the smaller blood-vessels were plugged by masses of bacteria with little or no reaction of the tissue about

them. In other places the bacterial masses were surrounded by a zone of dilated blood-vessels. Again, there was localized necrosis around the bacterial plug, while around this necrotic area there was sometimes simply a zone of enlarged blood-vessels or an accumulation of leucocytes, or both. In some cases the bacterial mass was closely surrounded by an accumulation of leucocytes so dense that the kidney-tissue for some distance around was entirely concealed, or completely broken down. The liver and spleen presented the usual effects of infectious emboli. The brain showed microscopically a plugging of the smaller blood-vessels in the hemorrhagic areas of bacteria, while around these plugs were either a simple zone of dilated blood-vessels or extravasation, or a localized breaking-down of brain-tissue; or, in a few cases, an extravasation of blood surrounded by a zone of purulent infiltration. Finally, in some places, the brain-tissue surrounding the bacterial embolus appeared to be simply necrotic, without hemorrhage, and without inflammatory reaction. The author concludes that the bacteria were the Staphylococcus pyrogenes aureus; these, he says, are the same species which Wyssokowitsch found in his one case, Weichselbaum in two of his four cases, and Zeigler in one case of this disease.

Meigs has recently exhibited an extremely interesting case of ulcerative or mycotic endocarditis to the Philadelphia Pathological Society.* The specimen was removed from the body of a girl æt. 17, a prostitute. The destruction of tissue was rapid and well marked.

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* October 13, 1887.

CHAPTER III.

ACUTE AND CHRONIC PERICARDITIS.

PERICARDITIS is an inflammation of the pericardium, or serous covering of the heart. As before noted, this covering is composed of two layers, the visceral and the parietal; either of the layers or both may be inflamed; as a rule it becomes general and extends over the greater part of the pericardium. It is usually, in fact almost always, the result of an inflammatory process more or less well defined, and generally associated with other lesions either of the myocardium or of the endocardium that are inflammatory in character, especially those that are dependent upon some morbid condition of the circulating medium. Hughes is of the opinion that the disease is more common in the infant than in the adult, and considers it a general rule that the younger the child the more chance there is for this affection to occur.

Etiology.—That pericarditis in the young is frequently unrecognized is attested by the fact that the post-mortem often reveals the disease of characteristic type in cases where the practitioner had not considered it existing at all.

Rheumatism is beyond doubt the principal causative agent in producing the disease; pericarditis is not rare in the newborn.

McLoud had one-half of his cases of rheumatism attacked by pericarditis; of those under fifteen years of age a little more than one-third presented pericardial complications. Out of forty-one cases of rheumatism in the young, twenty-three had pericarditis.

West considers idiopathic pericarditis a possibility, arising independently of any antecedent disease or of affections of the lungs or pleuræ, and cites the case of a child of eleven years who had no previous disease, but who presented well-marked pericarditis. That primary idiopathic pericarditis ever occurs seems hardly reconcilable with our present knowledge of pathology; traumata or constitutional dyscrasiæ, as gout, rheumatism, or syphilis, are sometimes extremely difficult of detection in their incipiency, and possibly many cases of so-called idiopathic origin may be traced to one or other of these causes. Blache also doubts its existence as a primary disease; it is generally seen in association with other diseases or with endocarditis.

Continuity and contiguity of structure becomes an important producing agent, consequently we frequently see it associate with or complicating pulmonary diseases, or left-sided pleurisy. It seems probable that we often have a sort of selective affinity for this serous membrane in certain forms of constitutional disease; for example, the pericarditis arising in the course of typhoid fever, rheumatism, or scarlatina. In rheumatism the occurrence of pericarditis is by no means in accordance with the severity of the type, nor can the fact that it will arise in any given case be determined by the condition of the other serous membranes. Virchow and Bamberger say that in children pericarditis is peculiarly prone to be coincident with pneumonia. All of the eruptive or exanthematous fevers do not tend equally towards the pericardial membrane; this tendency is less in variola than in measles, less in measles than in scarlatina. Frank has recorded cases of measles attended by pericarditis, and Kirby reports the case of a child aged two with fatal pericarditis following varicella; Stokes a case aged five years of acute dry pericarditis which followed an undetermined eruption. To Robert Mayne* is due the credit of first calling attention to the fact that scarlatina is the one eruptive fever in which we note the greatest number of cases of pericarditis: he records an example of the disease in a child aged twelve years. Scott Allison† has noted

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* Dublin Jour. Med. Sc., No. xx., 1835. † Lond. Med. Gaz., 1845.

three cases following scarlatina. The memoir of Thore (*Arch. Gén. de Méd.*, 1856) relates a case of hydropericarditis following scarlatina. This known tendency of the poison of scarlatina to localize itself upon the serous membranes is to-day well recognized. The pericardium is most apt to become involved during the second or third week after the appearance of the disease; at this time an effusion may be rapidly poured out into the pericardium, the lungs at the same time becoming œdematous. The general constitutional condition during an attack of scarlatina seems to directly predispose to inflammation of the peri- and endocardium; the condition of the kidneys during the latter part of a case of scarlatina, and the consequent saturation of the blood with morbific products, may have some causative relation to the pericarditis which is only too apt to arise at this time. In certain cases of albuminuria in children without any history of kidney-trouble, pericarditis has been observed to arise; it has also arisen as a sequela of chronic infantile diarrhœa. Whether it here bears any relation to the antecedent bowel-trouble, or its origin is simply a coincidence, we are unable to say. The association of pericarditis with pyæmia has been well shown by Kirkes, who, in the *London Medical Times*, 1862, reports three cases of the disease coincident with pyæmia; these cases, where pyæmia and purulent deposits are noted in connection with pericarditis, are probably due to extension from the myocardium.

In the adult, pericarditis is apt to follow or accompany meningitis; so it becomes of interest to inquire whether the same association of diseases exists in the child, this being a period of life during which meningitis is a not uncommon affection, but unfortunately clinical evidence is as yet not entirely satisfactory upon this point. Stanley, Burrows, and Latham have, however, noted in the child the association of acute cerebral affections and inflammation of the pericardium; it is a question whether the cerebral disease was not due entirely to the pericarditis, and not *vice versa*.

Perichondritis, or necrosis of the ribs in the pericardial region, may give rise to a pericarditis; Kirkensteiner has met such a condition in a child aged three years. The deposition of tubercles in the pericardium may give rise to an acute affec-

tion of that membrane. Rillet and Barthez, in three hundred and twelve instances of death by tuberculosis in children, observed ten cases of the deposition of tubercles in the pericardium with acute inflammation of the sac.

The visceral layer of the pericardium is most apt to be affected. A case reported by Liton, *Bull. Soc. Anat.*, 26, 221, showed the tubercles deposited on the anterior ventricular wall, of the size of a bean.

Age.—Sibson's experience shows that out of three hundred and twenty-six cases of rheumatism, sixty-three had pericarditis, and twenty-five of these were from sixteen to twenty years of age. All the fatal cases were under twenty years of age.

Post-mortem appearances; morbid anatomy.—The appearances in childhood are much the same as in the adult, except that this is the age at which we meet a great tendency to effusion with extreme rapidity in the formation of an exudate; the exudate is also liable to be plastic. Pericarditis presents another difference in childhood: the adult may present a dry pericarditis, this in a child is not at all frequent. A slight bloody tinge to the fluid is not at all uncommon in the early years of life.

The amount of liquid that the pericardium may contain is very variable. One hundred or one hundred and twenty-five grammes have been noted; it may contain as much as two hundred grammes.

The physical and chemical characteristics of the fluid are very similar to those in the adult; in the child as in the adult, it may be pus and constitute purulent pericarditis.

The pseudo- or false membrane may be disseminated over the entire sac, or may be located only over a very limited portion of one or the other surface; the membrane does not seem to be so apt to deposit on the visceral pericardium.

This false membrane may cause adhesion between the two layers by bridges or bands, or may totally obliterate the cavity of the pericardium. In some cases milk-like patches may be noted of about the size of a ten-cent piece scattered here and there over the visceral pericardium; organization may have taken place in these patches to an advanced degree, presenting blood-vessels with distinct walls; these patches constitute the

"plaque laiteuse" of the French writers, who have remarked that these plaques are apt to occur in cases of secondary pericarditis following wounds or septic absorption. Jenner has, however, attributed the origin of these milk-like deposits to the impingement of the heart against the thorax, considering that to attrition they owe their origin. We have noted these in rachitic children with depressed thorax, and can but conclude that constitutional vice does seem to have a great deal to do with their arising in any particular case.

We occasionally on the post-mortem table meet with cases of *hemorrhagic pericarditis* in the young. Bamberger and Lacrozille have particularly dwelt upon this condition; these observers conclude that the pathology is similar to hæmatoma of the dura mater, and as neoplastic formations are frequent in children, we find that a tendency to this form of the disease is well marked during childhood. The false membrane will be found of extreme delicacy, in which the vessels have very thin and delicate walls and are easily ruptured; the effusion, however, seems to depend as much upon a bloody exudation as it does upon direct rupture of the vessels; this pericardial condition has an analogue in hemorrhagic pleurisy.

Dr. Four records a case in a child aged fourteen, the symptoms simulating closely those of typhoid fever.

In these cases the evidence of a grave blood-disease will usually be found at the post-mortem examination. Kibber observed hemorrhagic pericarditis in a child aged four, who died of diphtheria. Eight to ten disseminated hemorrhages were seen on the visceral and parietal pericardium. They were made up entirely of infiltrated blood, which was just beneath the serous tissue.

When examining cases dead of pericarditis, certain *concomitants* may be noted. Most frequently we will see endocarditis associated with the pericardial disease. The cardiac cavities will almost always be found to be dilated. In adults this is probably due to a great extent to mechanical causes, as adhesions, but in children the myocardium is more apt to be affected to a greater extent, aiding materially in the early dilatation of the cavities. This is further evidenced by the fact that the myocardium not infrequently presents purulent foci, and that

we almost always find pericarditis and myocarditis clinically associated.

Symptoms.—Pericarditis, like endocarditis, may be either acute or chronic, primary or secondary, and the symptoms will be manifested accordingly.

The symptoms of acute pericarditis in the child are peculiarly prone to be masked, latent, and ill defined during the early stages. This latency of the early symptoms is a peculiarity of the disease when occurring in early life. A symptomatic diagnosis in the child is indeed a difficult task. The marked local pain complained of by the adult is not so well defined in children. Beyond doubt the child suffers, but is unable to definitely locate the seat of pain. When treating children suffering from disease the known tendency of which is to present pericardial complications, we must constantly be alert and on the watch for those symptoms which would point towards pericardial involvement, as the disease is only too apt to complicate some already existing morbid action. Especially is this true of pleurisy or pleuro-pneumonia of the left side. The presence of high temperature, disturbance of the circulation, and even pain, may all form but an integral part of the primary disease, of which pericarditis is but a complication. Possibly the physical signs, as presented by inspection, palpation, percussion, and auscultation, will give us some points by which a correct diagnosis can be arrived at during the early stages; but certainly, as soon as sufficient lymph has been exuded to roughen either the visceral or reflected pericardium, or an effusion has formed, with its undoubted characteristic symptoms, the diagnosis at once becomes apparent.

Physical signs; local symptoms.—We may then rely first upon the friction-murmur, and later upon the muffled heart-sounds, which may eventually almost entirely disappear, especially at the apex, the sounds at the base being heard until the fluid completely distends the pericardial sac; the friction-sound or murmur will also linger until this condition pertains. This friction-sound is to and fro,—that is, synchronous with the systole and the diastole, the former causing the inflamed and roughened surfaces to closely approximate, the latter to recede. We must bear in mind that the heart of a child is much nearer

the auscultator's ear than that of an adult; forgetting this point, auscultation is apt to be very confusing and misleading in the young. The friction-sound, if it exists, will rapidly become more apparent, as in the child the membrane is formed with great rapidity. In pericarditis the bruit or murmur which is heard over the præcordia may have two sources of origin,—it may be due either to an intercurrent endocarditis or to pericarditis alone and uncomplicated. A pericardial murmur in a child may closely simulate an endocardial bruit.

The special and diagnostic characters of a pericardial friction murmur are as follows:

It is usually basal, or directly over the body of the heart.

The murmur is almost always double, or to and fro.

It is not transmitted into the vessels and circulation, but may be heard in a child over a much larger præcordial area than in the adult. It is but rarely, however, heard over the posterior left thorax.

The murmur, particularly in the young, will be altered by the position of the patient, in being intensified as the subject leans forward, and rendered less audible during full inspiration or in the reclining posture.

The effusion is apt to arise somewhat rapidly, and by inspection we may note a pericardial bulging, which, in children, is marked, and arises early. The ribs being flexible and the thorax small, the bulging becomes the more apparent. A rachitic deformity of the chest must be differentiated from the bulging due to an effusion.*

The distention of the pericardium will cause upward displacement of the apex-beat. This is coincident with the formation of the fluid, and is proportionate to its quantity. In cases where any amount of effusion has been poured out, the apex may be displaced one or more interspaces. The cardiac impulse, like that of an adult under similar conditions, will be materially diminished. The symptoms on palpation are about the same in both the child and the adult.

The acuteness of the disease and the rapidity of the effusion will be productive of a series of symptoms, which are in themselves almost diagnostic.

We shall style them the *general symptoms*.

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* NOTE.—See Addenda, Nos. III. and IV.

The disease may be ushered in by a chill, fever, and some ill-defined cerebral symptoms, or choreic movements, followed by somnolence or delirium. These, however, may be but the symptoms of the primary disease, and the pericarditis for a time remain unrecognized. The *pulse* at first may be regular, but as the cardiac muscle becomes weaker the circulation becomes irregular and the radial pulsation is feeble and intermittent. This is particularly the case should it arise in the course of acute articular rheumatism or the eruptive fevers. As the disease advances the pulse becomes small, irregular, and intermittent; respiration is embarrassed, or mild dyspnœa may arise. If the case pursues a downward course the dyspnœa becomes apnœa, and asphyxia is marked, with grave cerebral symptoms, or an intercurrent cerebral affection may complicate the case; for instance, rheumatism may conjointly attack the pericardium and the cerebral meninges. From the fact that pericarditis is nearly always secondary, it is apt to pursue a slow and tardy course, if the case is to terminate favorably. The disease, as a rule, becomes in children an illustration of chronic pericarditis, associated with organic cardiac disease in later life. If the disease primarily originated in a rheumatic subject, we are apt to have recurrent pericarditis.

Chronic pericarditis may exist, and present to the diagnostician few, if any, reliable signs, and no pathognomonic signs whatever. From the fact that chronic pericardial adhesions produce dilatation of the cardiac cavities, we most usually note diffusion of the apex-beat. The adhesions, furthermore, have a tendency to drag the heart towards the epigastric region. Consequently the apex-beat may be noted to have been displaced in that direction. Chronic pericarditis in the child does not often present præcordial retraction, nor are the cardiac sounds diminished as much as in the adult.

Reduplication of the cardiac sounds coincident with pericardial adhesions has been observed by Rauneau in a child aged six years and six months. The autopsy showed complete dense pericardial adhesions, with a calcareous plate. The child had no symptoms of rheumatism at all. Friedrich has attributed to reduplication of the cardiac sounds a pathognomonic significance in pericardial adhesions.

The so-called ossification of the pericardium, which is really a calcareous degeneration, is of extreme rarity in childhood.

Dr. Bosissio, of Milan, states that pressure by the auscultator's head or stethoscope will increase sounds that exist in the pericardium due to adhesions; this is the more marked the younger the child. We have never as yet been able to convince ourselves of the accuracy of this observation. In those cases which it has been our good fortune to meet, pressure seemed to have but little, if any, appreciable effect in intensifying the sounds. Tumors of the heart are so rare in early life that it is sufficient for us to simply state that they have been recorded, and may confuse the diagnostician to differentiate between these neoplastic growths and chronic pericarditis.

Prognosis of pericarditis.—Usually in children the acute form tends toward recovery. At all events, death is not liable to occur directly from the disease itself, as we so often find at the autopsy of children the presence of pericardial disease that had existed during life, with but few, if any, symptoms.

PERICARDITIS IN THE FŒTUS AND NEW-BORN BABE.

Billard has gone so far as to say that at this age pericarditis was most frequently met with. Later investigations have, however, shown that this is not the case. To Billard, at all events, is due the credit of first calling attention to the disease at this period.

We have noted in a child dying thirty-six hours after birth well-marked adhesions of the pericardial surfaces.

Tardieu, in making medico-legal investigations, has remarked its presence in the fœtus and the new-born. The pathological changes in the affection are about the same as in the adult, but its etiology is more than difficult to decide. It has been recorded as being intercurrent with puerperal disease of the mother. Its association with early vaccination is also not unrecognized.

Webber states that pyæmic pericarditis may follow inflammation of the umbilical cord. This author also remarks that in children who die a few days after birth chronic pericarditis

is more commonly observed than is the acute variety, and that they also frequently have a concomitant pleurisy.

Kirkenstein recognizes two forms of pericarditis in the new-born, circumscribed as the plaque laiteuse, and general or diffuse inflammation of the entire pericardium. At birth the pericardium may contain from eight to twenty grammes of fluid exudate. The quantity, however, varies, as does its character. All grades are noted, from clear serum to a purulent accumulation; it is rarely seen to be hemorrhagic.

Blache is of the opinion that at birth the effusion is peculiarly prone to purulent change, and that if the effusion is non-fibrinous yellowish serum, it always dates from uterine life, and is due to disease of the mother.

Occasionally the pericardium may show petechial spots or sero-sanguinolent clots.

When the liquid is a yellow-colored effusion it is apt to be large in amount, constituting hydrops pericardii.

Symptoms.—The physical signs by which we recognize the disease at a later period of life cannot be clinically recognized in young infants; in the new-born we cannot appreciate the presence of the disease at all.

In some cases a tumultuous heart's action with great acceleration of the pulse, may be noted. Dyspnœa is apt to be a prominent and well-marked symptom. The features are said by some to present a peculiar pinched appearance. The interference with the circulation is further made evident by the condition of the fontanelles, which are somewhat distended and pulsate synchronously with cardiac systole.

Prognosis.—Owing to the difficulty in recognizing the disease at birth, it is almost impossible to give an accurate prognosis.

The course of the disease is, however, apt to be rapid, and generally tending towards a fatal termination. In Bednar's case of an infant aged three weeks, the disease lasted from four to eleven days.

The following case of pericarditis occurred in our practice

Mary E., aged two and one-half years, was a fairly healthy child until eight weeks before death, when she contracted a left-sided pleurisy, followed by a serous effusion, which eventually became purulent. When it was aspirated a few ounces of ill-

smelling pus were removed, and the child improved for a short time, when symptoms indicative of the reformation of purulent matter demanded better drainage, which was secured by an incision and the introduction of a tube.

The liver and spleen were enlarged. A diarrhœa set in, which was totally uncontrollable, to which the child succumbed six weeks after we first saw her.

Post-mortem.—Examination was made a few hours after death, and as we report the case solely for the condition of the pericardium, suffice it to say that the left pleural cavity contained several ounces of pus, and presented the usual appearances in such cases.

Heart and pericardium.—The former much enlarged, and the latter adherent to the adjacent structures. The parietal pericardium was much thickened and covered by a pyogenic membrane; the visceral layer was thickened and covered by pus; the layers were adherent over left ventricle.

The following case, recorded by Broxholm,* is of extreme interest on account of the fact that the child complained of no pain, and, although effusion was present in marked degree, did not present either dyspnœa or orthopnœa. The child, a boy aged five, expired suddenly before medical aid could reach him. He had, however enjoyed seemingly good health.

Sectio cadaveris twenty-four hours after death.

Child was rickety.

Meninges were vascular and congested, no effusion; brain was very vascular. No ventricular effusion, none at base of brain. The lungs were adherent by lymph-deposits, some old, some recent. Tubercles infiltrated throughout lungs.

Pericardium.—Much distended, contained one pint of straw-colored effusion, with numerous flakes of greenish-colored lymph: the interior of pericardium, all over its surface, presented deposits of fibro-plastic matter, rendering its surface exceedingly rough and granular. Heart-muscle hypertrophied, and presented over the centre of the ventricles two patches of a light-brown color, feeling very firm and hard, thinner than the surrounding tissue, and cutting like cartilage.

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* Lancet, London, vol. i., 1856, p. 343.

One segment of the mitral valve was calcified, and in the chordæ tendinæ attached to the same segment, a little removed from their point of attachment, were also noted calcareous degenerations. The tricuspid valve was much thickened, having fibrinous deposits on its edges.

Cavities all dilated and filled with coagulated blood. The stomach, liver, spleen, and other organs appeared healthy, but the mesentric glands were enlarged.

The child had never had rheumatism, as far as could be determined by the history and the post-mortem appearance.

Kirby's case,* before referred to, well illustrates the disease as intercurrent in the eruptive fevers. A previously healthy boy, aged one year and eleven months, had been ill for five or six days with varicella; child developed cough, high temperature, and œdema of leg; sank lower and lower, lying on his back in his mother's lap, countenance pale, pupils dilated, and surface cold, pulseless at wrist; respiration labored; over præcordial region increased dulness; normal sounds of heart scarcely audible, but a friction-sound, although faint, was distinctly heard; child died.

Post-mortem.—Pericardium very vascular and much distended by fluid; the sac was nearly full of yellow flaky serum, and its serous lining, throughout its whole extent, was coated with rough villous-looking lymph. In the pleura, also on both sides of the thorax, where it covers the upper portion of the diaphragm, there were patches of coagulable lymph.

To illustrate the pyæmic form of pericarditis we will select the case recorded by W. S. Kirkes.† A boy, aged sixteen, referred to on page 206 of this article, was admitted to St. Bartholomew's Hospital, having received a violent kick on the shin, about an inch below the knee. Four days after receipt of injury the boy applied for admittance to hospital; there was then a red tense swelling over the injured part, the child presenting a peculiar haggard, anxious look. Auscultation revealed a well-marked pericardial friction-sound; the patient complained of general pains in the limbs, and of feeling very

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* Lancet, London, 1860, vol. i. p. 87.

† London Med. Times and Gaz., Nov. 1, 1862, p. 461.

ill. There was, however, no swelling, redness, or particular tenderness of any joint; no symptoms of rheumatism whatever. The skin was hot, the pulse about 120, and the tongue coated with a yellowish fur. On the day after admission a few small pustules were detected on the front of the chest and abdomen. The swelling over the left shin was incised, and much puriform matter evacuated, and the patient was supported with bark, wine, and good nourishment. The nervous agitation increased, the symptoms became more typhoid, signs of copious pericardial effusion ensued, and the boy died exhausted three days after admission, seven days from the receipt of the injury.

On post-mortem examination the pericardial cavity was distended with turbid serum, while flakes and curdy masses of recent lymph were spread over the surface of the membrane, which was intensely vascular. The muscular tissue of heart was softened, especially about the left ventricle; several diffluent, purulent-looking masses, varying in size from pins' heads to split peas, were scattered through the myocardium, especially at base and in left ventricle. All valves of heart were healthy.

Lungs showed numerous pyæmic deposits. The tibia was much affected and infiltrated with purulent matter.

CHAPTER IV.

THE TREATMENT OF ENDO- AND PERICARDITIS.

As primary endocarditis is of extreme rarity, the treatment of endocardial inflammation is mainly that of the disease with which it is intercurrent. As we have already noted, it is prone to occur as an accompaniment of rheumatism, scarlatina, nephritis, measles, variola, varioloid, erysipelas, diphtheria, and typhoid fever; it has arisen during the course of coxalgia, or in those subject to the diatheses; its association with chorea and erythema nodosum has also already been noted.

In the first-mentioned affection endocarditis may appear

early in the case and be the sole manifestation of the rheumatic disease; or, on the other hand, it may be the first local manifestation of a general rheumatic outbreak that in a few days will involve the joints. Cases of endocarditis should be placed at perfect rest: all excitement of any kind whatever is contra-indicated. There should be but little bright light admitted to the child's room, nor should conversation be indulged in about the little patient's bedside. Cool acidulated drinks will be gratifying to the patient and will assist in reducing temperature, as lemonade sweetened with glycerin, or neutral mixture. Locally, the præcordia may be irritated by iodine in the following combination:

℞ Tr. iodin., ʒiii;
Spts. chloroformi, ʒi;
Tr. aconit. rad., gtt. xxv. M.
Apply with a brush twice daily.

This mixture will not cause pain by its application and will not be so likely to strip off the epidermis early in the case. The application of tr. iodine alone to the delicate skin of a child will cause undue irritation and make the child very restless, thus exciting cardiac action.

In older children a blister may be applied as large as the præcordial surface. To this raw surface Bouillard has applied daily forty to sixty centigrammes of powdered leaves of digitalis, the irritant action of which maintained the vesicant action. We would, however, sound a word of caution in this method of administering digitalis, as we have noted alarming symptoms from the absorption of digitalis by the skin.

In the rheumatic cases much benefit may be derived from salicylic acid, given as the salicylate of soda, lithia, or quinine. Some authorities administer large doses of nitrate of potash or tartrate of soda and the sulphate of quinine.

The salicylates may be pushed if they appear to act favorably upon the rheumatism and if the cardiac action is not extremely irregular, agitated, or intermittent, and if the pulse is regular and not too frequent.

Digitalis may be exhibited as the tincture in six- to eight-drop doses three times a day for a child of five years, the dose being gradually and cautiously increased as needed or decreased

if necessary, as when its effect has once been secured we can maintain the impression with much smaller doses. It must be borne in mind that digitalis is not one of the quickly acting cardiac stimulants, an opinion that our intimate association with students and young practitioners has taught us is very prevalent. In many cases we much prefer the infusion of digitalis flavored with cardamom, in drachm doses.

We have never derived any appreciable benefit from convallaria in any way whatever, and, indeed, it seems to us that the drug merits no place in our consideration.

Veratrum and aconite are to be administered only with the extremest care, and are then to be carefully and intelligently watched. Opium, if given at all, must be in small doses, and is also to be watched most carefully, especially should there be any tendency to cyanosis. Bromide of potassium has been of service in our hands in quieting the patient and allowing the heart an opportunity of establishing its equilibrium. If the cardiac muscle is weakened, possibly the sodium salt would be the better drug. We consider that under no circumstances would it be justifiable to administer chloral.

Upon the whole, the most benefit will probably be derived by an early supersaturation of the blood by the alkalies. The ordinary fever mixture of spts. ætheris nit. and liq. ammoniæ acetatis is alkaline and sedative.

During convalescence great care is to be exercised, as in all probability permanent injury to the valves has been the result of the acute process. A well-selected dietary is to be advised, —one that will be nutritious, but will not demand undue exertion of the gastro-intestinal tract or the large abdominal glands in its assimilation, and above all else will not cause flatulent distention of the stomach and reflexly irritate the heart. This subject has been fully elaborated by one of us in a recent publication.*

The importance of guarding against the evil effects of high temperature merits our serious consideration. The diseases during the course of which endo- and pericardial inflammations are usually associated are apt to have exceedingly high temper-

* "Maternity, Infancy, and Childhood." J. B. Lippincott Company.

atures, such as rheumatism, pleuro-pneumonia, pyæmia, etc., and the question often arises what effect the treatment such as is in vogue will have upon the cardiac condition. The treatment is usually cool bath, cold sponging, with pack, or evaporation by surrounding the child with a moistened sheet. In judging of this the strength of the heart and the freedom of the circulation have to be taken into consideration, and the activity and character of the treatment arranged accordingly. For instance, in a case of pleuro-pneumonia, if the heart be implicated and weakened, a cold douche or sudden shock should be most certainly avoided. In cases of recurrent rheumatic endocarditis engrafted upon some old valvular lesion the sudden shock might fatally affect a weakened heart, and cardiac paralysis from over-distention or visceral congestion be the result. In no case should a persistent temperature of over 104° be permitted; it is not the question of the necessity of the reduction to which we call attention, but to the method by which it must be accomplished.

We do not, however, at all advocate the indiscriminate knocking down of temperature simply because the condition of hyperpyrexia is present, a custom that to-day appears to be too much in vogue. First we should endeavor to determine the cause and effect of the high temperature. It would be no more rational to exhibit antipyretics or a cold pack to reduce a high temperature without first ascertaining its cause and endeavoring to remove it than it would be to treat any disease by a name; for example, the administration of digitalis in "heart-disease" without first determining the lesion and the condition of the cardiac cavities.

It is well, then, to first use the milder means before we resort to the more heroic measures, now so much the fashion.

The simple fever mixture already referred to in combination with drop-doses of aconite, say every four hours for a child aged five, together with iced lemon-juice and the carbonated waters, will frequently accomplish our object. We may sponge the extremities with cool water containing an evaporating volatile substance, as vinegar or alcohol; should the thermometer again show a registration 104°, or above, the cautious application of ice-cloths to the head and the nape of the neck

would be justifiable, or even an ice-cap in children above twelve years of age. Quinine in suppository may be administered as the temperature begins to fall. If, however, the temperature should persistently remain high and show its well-known effects upon the heart and circulation, then we may exhibit either antifebrin, antipyrin, or thallin.

The former is probably the most reliable, its action usually being well marked within an hour after administration. The dose for a child of five years may be from three to six grains, repeated several times during the twenty-four hours if necessary. As the temperature falls the pulse will be reduced in frequency. In some cases slight cyanosis may supervene. Sweating is almost always present, and in some cases may be exhausting or even alarming. Atropine has but little if any effect in controlling this ephidrosis. An increased amount of urine will usually be voided following the administration of the drug. Antifebrin may occasionally cause vomiting, although it is not so likely to as either thallin or antipyrin. Rarely will the patient complain of chilliness, which we sometimes note after the administration of the two latter.

Iron and arsenic can with great propriety be administered during this convalescent stage.

The treatment of *acute ulcerative endocarditis* is very unsatisfactory. As a rule, in childhood, the disease is met with as a concomitant or as an intercurrent affection in the course of diphtheria, the infectious diseases, or suppurative disease of the bone or joints. The cases are almost invariably fatal.

It has been suggested to apply an ice-bag to the præcordia, with the internal administration of antiseptics, as salicylate of soda, benzoate of soda, and sulphate of quinine.

The tincture or infusion of digitalis may be administered in large and increasing doses.

The exhibition of camphor in an emulsion made with the yelk of an egg would perhaps mitigate the symptoms. Musk would also be indicated, and carbonate of ammonia as a rapid general diffusible stimulant. Recognizing the fact that the endocardium is teeming with micro-organisms, authorities have advised the exhibition of the following, known as Van Swieten's solution:

℞ Hydrarg. chlor. cor , grm. 1;
Alcoholis, grms. 100;
Aquæ, grms. 900. M.
f ʒi contains about $\frac{1}{15}$ grain of the bichloride.

In the TREATMENT of PERICARDITIS we must also remember that the disease is apt to be intercurrent. Should it occur with rheumatism, the treatment of this affection must be pushed actively. Locally, we may apply heat and moisture by poultices, or the præcordia may first be painted with iodine and the poultice applied.

Some writers advise the application of an ice-bag to the region under consideration. Gendrin is of the opinion that it immediately diminishes the local pain, calms the tumultuous pulsations of the heart and the extreme anxiety of the patient; as a rule, also, its refrigerant action diminishes the febrile movement in a little while and lowers the frequency of the pulse.* In our opinion it should be tried only with great caution, and then only in children who are very robust. Its application should be limited, in order not to depress the patient. Pulse and temperature are to be watched, and as they return to normal the ice is to be removed. Within the last few years, coiled rubber tubing, through which water of varying temperature may be passed, has been suggested, and is, in our opinion, far better than the use of ice. We consider that mercurialization is never allowable and that venesection is only occasionally admissible.

Aconite, veratria, and tartar emetic are to be looked upon as dangerous remedies, except in the hands of experienced practitioners. Undoubtedly there are sthenic cases where the first-mentioned drug may be used with benefit. The application of a large blister to the præcordia is sometimes accompanied by very happy results in pericarditis. It would perhaps be safer to restrict this treatment to children over ten years of age. To Corvisart is due the credit of first calling the attention of the general profession to this mode of treatment, but it should be limited to cases where effusion is suspected. Much nourishment is required, together with alcoholic stimulants. Should marked depression arise with impending asystole, brandy and digitalis may be administered. The patient must

* Valker, Bull. Médical, Nov. 20, 1887, is of a similar opinion.

be at absolute rest, and not allowed to sit up under any circumstances if there are evidences of syncope. This depression of the vital energies may be further combated by quinine, musk, champagne, carbonate of ammonia, hypodermic injections of brandy, ether, or camphor, and wine whey should be included in the dietary.

If an *effusion* forms and is not readily absorbable, we may make applications of flying blisters or strong iodine, with iodide of potassium internally. We must also endeavor to raise the general health to as high a state of efficiency as is possible, thus aiding absorption of the exudation. Tonics are very useful, particularly the tincture of the chloride of iron, as mist. ferri et ammoniæ acetatis (Basham's Mixture).

Diuretics, as nitrate, acetate, or citrate of potassium, scoparius, and caffeine, have been employed. We prefer the acetate and citrate of potassium.

Paracentesis pericardii.—In rare instances in children's practice will paracentesis be demanded. When we fear heart-failure, a purulent effusion or distortion, and permanent displacement of the heart, we may consider ourselves justified in tapping the pericardial sac. The presence of albuminuria and tube-casts which seem to depend upon the pericardial effusion would also be an indication for tapping.

It is stated that Schuh, in the service of Professor Skoda, was the first to puncture the pericardium. Although paracentesis seems to have been proposed by Riolan* in 1649, and performed by Romero, of Barcelona, who operated successfully, and reported his cases to the Faculty of Medicine of Paris, Mérat says that the faculty would not allow the report of Romero's successful cases to be printed in their Transactions, fearing that by so doing they would sanction an operation which as yet had not met with their approval.

After having determined the expediency of paracentesis it is not wise to wait too long, as delay is apt to set up a low grade myocarditis, with fatty degeneration of the muscular wall of the heart and dilatation of its cavities.

In performing the operation it is best to use an aspirator with a vacuum jar, a delicate double canula: the innermost

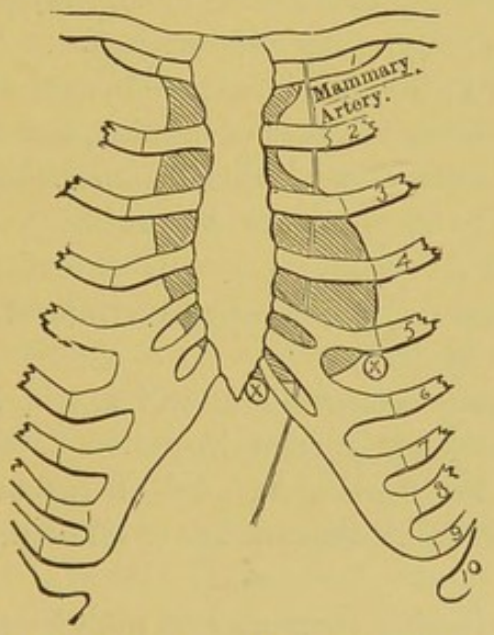
* Enchiridion Anatom., lib. iii. c. 4 (1649), quoted by Roberts.

portion may be either a solid needle or a needle-pointed tube, either of which are to be withdrawn, the former entirely, and the latter until its point is sheathed.

Should the fluid reaccumulate, a second or third tapping may be resorted to, as in the case recorded by Gooch, referred to in table, in which a child aged thirteen was tapped six times, and Bouchout's case, in which a girl aged eleven and a half was tapped eight times. The danger does not seem to increase any in proportion to the number of tappings.

We do not favor the injection of any substance into the pericardium either with a view of antisepsis or of washing out the sac, although Aran's case, recorded in Trousseau's Clin. Med., iii. 386, in which iodine was injected twice, shows that the procedure may not always be attended with fatal results.

Should pus accumulate, and after several aspirations fail to resolve, there can be no objection to an opening being made to secure perfect drainage, as purulent pericarditis if left to nature will almost inevitably prove fatal.



It is essential to definitely settle the point at which we are to aspirate with the least danger of wounding the heart-muscle, and with the greatest certainty of reaching the effusion.

These objects can perhaps be best fulfilled by introducing the needle either in the left costo-xiphoid angle and pushing upwards towards the heart, or by inserting the trocar at the

fifth interspace, about where the apex should normally be situated when it is not displaced, remembering that in the very young the heart is situated slightly higher in the thorax.

Care should be taken not to wound the mammary artery.

The accompanying figure will serve to illustrate the parts mentioned. The ⊗ indicates points for puncturing the pericardium.

In selecting the first-mentioned site for puncture it is well to introduce the needle as close to the ensiform cartilage as possible, thus avoiding the artery. It also should be inserted as high as possible in the fossa, so as to be sure of entering the pericardial sac and not the abdominal cavity, as it may pass below the diaphragmatic arch. The presence of fluid will, however, so distend the sac that there is little if any danger of not reaching the fluid or of wounding surrounding structures. We have been present at many examinations of the cadaver in conjunction with Dr. Osler in which a needle introduced as above always readily perforated the pericardial cavity. There is little danger of reaching the pleura, as it curves to the left before reaching the xiphoid cartilage.

The position in the fifth interspace over the location of the normal apex-beat is safe, easy of location, and has always been attended with satisfactory results in our hands as far as the mechanics of the operation are concerned.

The skin in the young is usually so tender that it is not necessary to make a preliminary skin puncture, as we sometimes do in an adult.

As the effusion decreases it is well to withdraw the needle somewhat in order to prevent laceration or wounding of the heart-muscle; the final withdrawal of the needle is accomplished when we think that enough or all of the fluid has been removed.

A simple adhesive strip may be placed over the point of puncture.

In cases of sacculated pericardial effusion the operator will of course introduce the needle at that point where the signs of effusion are most marked and which presents the least evidence of the presence of the heart-muscle. Occasionally, after the introduction of the canula, there will be no flow. This may

depend upon two conditions: either a thickened pericardium has been pushed in front of the needle, or the fluid is too thick or flaky to admit of a flow through the calibre of the particular instrument that is inserted. If the operator is satisfied of the correctness of his diagnosis in regard to the presence of fluid, it is well to reintroduce in the case of thickened membrane, and in the latter instance to select a larger trocar and canula.

Certain *dangers* are to be met with in paracentesis pericardii. Great care is to be exercised in order to prevent the entrance of air, and the consequent change of a serous effusion into a purulent one teeming with bacteria. Albert Wilson has recorded a case of pericarditis in which he considers a specific micro-organism was found independently of any outside contamination, which will be referred to later in our study.

The operator must also avoid wounding or perforating either the pleura, diaphragm, or heart-muscle, but above all must he guard against wounding the internal mammary artery and producing alarming hemorrhage. Anatomists tell us that it arises from the subclavian, and takes a downward course parallel with the edge of the sternum until the sixth costal cartilage is reached. At its lower edge it divides into two branches, the superior epigastric, running on downwards, and the musculophrenic, which runs along the sixth interspace.

Cruveilhier and Sappey (Roberts) give as the average distance of the artery from the sternum at the fifth or sixth interspace four to five millimetres, but Roger has found in children that the distance is only two or three millimetres.

That wounding or perforation of the heart may and does occur, witness the case recorded by Roger,* of a child æt. eleven, in whom one hundred grammes of blood was removed by wounding the ventricle, and his second case,* of a boy æt. five, in whom two hundred grammes of venous blood was removed from tapping the right ventricle. The case recovered.

Wounding of the heart may usually be avoided by selecting a sufficiently low point for puncturing. There is much greater danger to life in inadvertently tapping the auricle than there is in perforation of the ventricle.

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* Referred to in table.

*Table of Cases of Paracentesis Pericardii.**

*Operator.	Sex and Age.	Mode and Site of Operation.	Recovery.	Death.	Time that Patient survived the Operation.	Remarks.	Complication.	Reference.
Roger.....	M. 5	Aspiration. 6th interspace.	1			At first no fluid, then blood and serum, followed by pure blood, apparently venous and not in jets. 200 grammes. Child became pale. Improvement followed. Pericarditis did not return. Death occurred five months after operation. Author says right ventricle was wounded.	Dilatation and valvular disease of the heart.	Bull. de l'Acad. de Méd., 1875, t. xl. p. 1276.
Villeneuve.....	M. 5	Aspiration. Most projecting point.	1			Two syringefuls of serum. Puncture did not close, fluid became purulent and flowed for nearly six months. Finally closed spontaneously.		Lond. Med. Rec., September 15, 1875. p. 532; from Marseille Méd.
Lubric.....	M. 6	Trocar. 5th interspace, four centimetres outside nipple.		1		Intended to tap left pleura, as diagnosis was pleuritis and pericarditis. f℥xvi purulent serum flowed, but autopsy showed it had come from pericardium, and that pleura was adherent. Reporter says that death must not be attributed to the tapping.		Bull. de l'Acad. de Méd., 1875, t. xl. p. 1216; Id., t. xxxvii. p. 658.
Juergensen.....	M. 6	Tiersch's syringe at first (aspiration). Trocar second operation. Site not given.		1	7 days.	First tapping, ℥iii pus. Second operation, two days after, ℥vi½ pus. Left canula in sac and washed out with salt solution. Died with cerebral symptoms. Followed pneumonia of left side.	Cerebral meningitis.	Ziemssen's Cyclopædia of Med., Amer. edit., vol. v. p. 113.
Duncan.....	M. boy.	Trocar.		1	Few hours.			Edinb. M. J., October, 1872. p. 376.
Heath.....	M. 6	Aspiration. 3d and 4th interspaces.		1	50 days.	Tapped pericardium twice. f℥iii¾ and f℥vi. Tapped abdomen twice.	Phthisis and tubercular peritonitis.	Practitioner, xi. 265.
Roger.....	F. 11	Trocar. 5th interspace.		1	35 days.	Tapped twice. First, 100 grammes of blood; second time, 500 grammes of serum, five days after.	Ascites.	Bull. de l'Acad de Méd., 1875, p. 1274.
Bouchout.....	F. 11½	Aspiration. 5th interspace.		1	34 days.	Tapped eight times at same place. Serous effusion becoming hemorrhagic. Two punctures of the heart without accidents. Death followed three days after eighth operation. Intervals between the operations were four	Left pleuritis six weeks previously; endocarditis.	Gaz. des Hôpitaux, 1873, p. 1130.

						days, three days three days, four days, seven days, seven days, three days. Autopsy showed nearly 800 grammes of chocolate-colored fluid in pericardium. No syncope or aggravation seemed to follow the punctures of the heart, though 80 grammes and 30 grammes of blood were withdrawn.		
Roger	F. 12	Trocar. 6th interspace.		1	1 day.	Removed 780 grammes. At first no flow; introduced lower down.	Myocarditis and heart clot.	Bull. de Acad. de Méd., 1875, p. 1284.
Saundby	M. 13	Aspiration. 4th interspace.		1	Few hours.	Removed pus, f℥xxx; probably from rupture of pulmonary abscess.	Pleurisy and abscess of lung.	Edinb. M. J., March, 1875, p. 799.
Gooch	M. 13	Aspiration. 5th interspace.		1	38 days.	Tapped six times. Purulent fluid, f℥xxi; f℥xxxv; f℥xl—iodine injected; f℥l—iodine; f℥xxx; f℥xx—iodine.	Peritonitis.	Brit. M. J., June 19, 1875.
Smith, J. L.	— 13	Aspiration.		1		F℥xxx pus. Autopsy showed f℥xxxvi in pericardium. Pericardial effusion probably developed by admission of pleuritic effusion through a minute opening.	Pleurisy.	New York Med. Record, February 12, 1876, p. 110.
Jowett	F. 14	Not stated.		?	Life prolonged.	Hope of recovery.		Günther, Blutigen Operationen, iv. 3, 102.
Norris, Herbert	F. 14	Bowditch's pump and canula. 4th interspace.		1	22 hours.	F℥iiss serum.	Acute rheumatism, acute endocarditis, and pleuritis.	Agnew's Surgery, i. p. 348.
Jobert	M. 16	Incision and trocar. 5th interspace.	1			Removed 400 grammes. Tapped pleura also. Under notice for three months.	Phthisis.	Trousseau, Clin. Med., iii. 370.
Hunt (?)	F. 16	Aspiration. 4th interspace.		1	2 days.	Few drachms. At autopsy pericardium contained f℥xiv. Near puncture firm adhesion to heart, which may account for small amount of fluid obtained.	Rheumatic fever.	Lancet, March 10, 1877, p. 343.
Pepper	F. 17	Aspiration. 5th interspace.		1		f℥viii serum. Able to get out of bed twenty-six days after operation. Pleuritis and ascites three and a half months subsequently. Death occurred fifteen months after operation. Autopsy showed complete adhesion, no valvular lesion, fatty degeneration of muscular structure.	Albuminuria and casts; due to effusion in pericardium.	Medical News and Library, March, 1878, and Amer. Journal Med. Sci., April, 1879.
Heger	M. 19	Trocar. 5th interspace.		1	69 days.	Tapped twice. 1500 grammes and 400 grammes. Drainage-tube left in six hours.	Phthisis.	Arch. Gén. de Méd., November, 1854.
West	M. 16	Aspiration.	1			Pericardium tapped twice and laid open. Temperature neither before nor after the operation was above normal.		Brit. M. J., April 2, 1887.
Stevenson	F. 5 yrs. 4 mos.	Small trocar and aspirator ½ in. in diameter. 3d left intercostal space ⅔ in. from sternum.		1	12 days.	f℥vss thin turbid serum. A friction rub heard immediately afterward. Paracentesis thoracis performed three times.	Double pleurisy and peritonitis.	St. Barth. Hosp. Repts., vol. xvii., p. 217.
Barlow	F. 19 mos.	Hypodermic needle.		1	½ hour.	f℥i black blood.	Cardiac and renal disease.	Ibid., p. 222.

* Collated in the main from J. B. Roberts's Paracentesis of the Pericardium (J. B. Lippincott & Co., 1880).

It is never well to draw off large pericardial effusions rapidly, hence we would suggest aspiration in these cases by a capillary tube. If a larger tube is used there is great danger of cardiac arrest.

The treatment of the complications or concomitants of pericardial effusion is in the main governed by the factors present in each individual case, as, for example, should renal complications arise, the physician is to determine at once whether the pericardial effusion itself is not giving rise to the abnormal renal action. Frequently do we meet the converse of the proposition,—*i.e.*, the hydropericardium of Bright's disease. Pleurisy and pneumonia arising with or antedating the pericardial effusion must be treated on general principles, recognizing, however, the additional gravity in the prognosis.

CASE.—The case reported by Albert Wilson,* referred to above, is of much interest on account of the apparent pathogenic significance of the bacillus.

E., a girl, æt. seventeen, had a regurgitant mitral murmur for five years. Contracted acute rheumatism. Acute pericarditis, with a pericardial effusion, rapidly arose, complicated with left pleural effusion. Patient died twenty-one days after being first attacked by the disease.

Autopsy.—Face dark blue. Pericardium and heart were distended. Right lung retracted, solid, non-crepitant; was in stage of red hepatization. Left lung was pale, crepitant, and adherent to the pericardium. Left pleura contained three pints of clear serum. No adhesions posteriorly. The pericardium was about one-fourth to three-eighths of an inch thick, strongly adherent to the heart, and impossible to separate it. Even the large vessels were matted with organized lymph.

Mitral valve showed old lesions, left ventricle greatly hypertrophied, etc.

Microscopic examination.—Scrapings of pericardium showed in fresh and dry state very small, short, non-nucleated rods, which take the pink stain. They are micro-bacilli.

Inoculation experiments.—He used three sterilized preparations:

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* Edinb. Med. Journ., vol. xxx., 1885, p. 1105.

First, carrot infusion, two months old; second, calf jelly; third, ascitic serum, collected antiseptically, three weeks old, and perfectly clear.

Pericardium experiments.—Under the spray, and with strict antiseptic precautions, a piece of the pericardium was removed, little pieces of lymph were scraped off, and a flask of serum was inoculated and placed in an incubator at 90° F. The upper part of the fluid remained clear, but a deposit formed at the bottom. The germs were anærobic. Examined microscopically, they were found to contain the same micro-bacilli above referred to, without nuclei. Similarly two other sterilized flasks of serum were inoculated from the flask with the same results.

Microscopic section of heart.—The section of the pericardium showed wavy layers of fibrin and lymph more or less organized. Strewn in the meshes were small clusters of micro-bacilli.

Wilson concludes that the pericarditis was undoubtedly associated with a micro-bacillus, which was the primary germ or parasite.

Dr. T. M. Rotch of the Harvard Medical School has kindly recapitulated for us his views on the percussion outline of pericardial effusion and the selection-site for paracentesis pericardii, making his deductions from experiments upon sixteen infants; we take great pleasure in incorporating them in this little work:

"The following remarks concerning pericardial effusion are a brief *résumé* of the observations which I have made on this subject since publishing my article in 1878. These observations, which are mostly confirmatory of the points suggested in the original article, have been made at the Boston City Hospital from its large number of acute cases, amounting to nearly three thousand ward-patients in a year of all ages, from the Children's Hospital, Infant Hospital and the Boston Dispensary children's clinic, the clinical observations also being tested in various ways in the anatomical and physiological laboratories of the Harvard Medical School.

"Owing to the acknowledged latency of the symptoms in

many cases of acute pericarditis in infancy and childhood,* the diagnosis of effusion must often rest on the physical signs, and of these signs, as I have already stated,† percussion is the only one that can in the majority of cases be relied upon at the stage of the disease when it is especially important to know whether an effusion is present or not for purposes of differentiation from dilated heart, with the question of tapping to be decided upon. Again, this physical sign of percussion can only be of value when applied after precise anatomical relations have been established and physiological and mechanical laws recognized; and this at once brings us to the anatomical consideration of the subject.

"In the various articles written on pericardial effusion the authors are found to copy each other year after year, expounding, repeating, and perpetuating views which have never been distinctly proved and are often manifestly erroneous, their lines of percussion not being exactly stated and their diagrams, from improper methods of experimenting, being open to criticism, as representing hypothetical conditions which do not exist in the human subject.‡

"Hughes states that infantile pericarditis occurs more frequently than the adult disease, and the younger the child the more chance there is for the appearance of the disease.§ Keating and Edwards speak of the association of endocarditis and pericarditis as a frequent concomitant, and of the dilatation of the cardiac cavities being caused, to a great extent, by adhesions in the adult, and by the myocardium being affected in the child.‖ Keating and Edwards have also given a most valuable description of the anatomy, physiology, and general topography of the heart and pericardium.¶

"Assuming that the observations of these gentlemen are correct, they have a significant bearing on what I consider of the first importance for a proper appreciation of the subject—

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* Keating and Edwards, Archiv. Pediat., April, 1887, p. 209.

† Transactions Mass. Med. Soc. 1878.

‡ A notable example of this statement can be seen in the pericardial-effusion plates in Reynolds' System of Medicine, copied by Roberts and others.

§ Archiv. Pediat., April, 1887, p. 204. ‖ Ibid., p. 208.

¶ Ibid., February, 1887, p. 65.

namely, the methods of the anatomical investigations. The older the child and the nearer to adult age that it approaches, the greater is the chance that adhesions from some slight and perhaps almost overlooked inflammation of parts or organs adjacent to the pericardium have taken place, and this, in connection with the natural absence of adhesions mentioned by Keating and Edwards, emphasizes the fact that the primary experiments regarding the shape of a distended pericardium should be made on the infant or young child, where the result is more likely to show a correct figure on percussion, both clinically and in the laboratory.

"The number of clinical observations on infants is not yet large enough to provide us with sufficient data, but the experiments from which my diagrams are drawn were made on sixteen infants, in none of whom did adhesions exist, while in the case of an adult at the city hospital—where, although the pericardium was evidently much distended with fluid, the percussion failed to show absolute dulness to the right of the sternum—the autopsy revealed adhesions of the middle lobe of the lung holding it tightly to the right edge of the sternum in the 5th interspace, with the effusion behind it, thus giving resonance where the uncomplicated case would have given dulness; hence it is evident that we must first study and acquire a knowledge of the uncomplicated cases before we are prepared to elucidate cases complicated by adhesions. And yet there is a strong probability that many of the clinical observations made on adults by various competent clinical observers, and established by them as rules for diagnosis, are, from the presence of adhesions—sometimes in one place and sometimes in another—rendered of little practical value, as shown by the difficulty experienced in making a diagnosis by these rules in new cases, and even delaying the final solution of the problem by, through not knowing where or whether, and hence not stating where or whether, adhesions existed, perpetuating erroneous views.

"Assuming, then, for the present that the infant's (first) and the child's (second) pericardium is most likely to be the best for studying the uncomplicated pericardial-effusion outlines, and allowing that experiments made as directed in my

article in 1878 are correct until they are disproved—which so far has not been done—the deductions which can be made from the results of these experiments are that the fluid accumulates at the bottom of the pericardial sac, where it collects on either side of the arched diaphragm like a saddle,

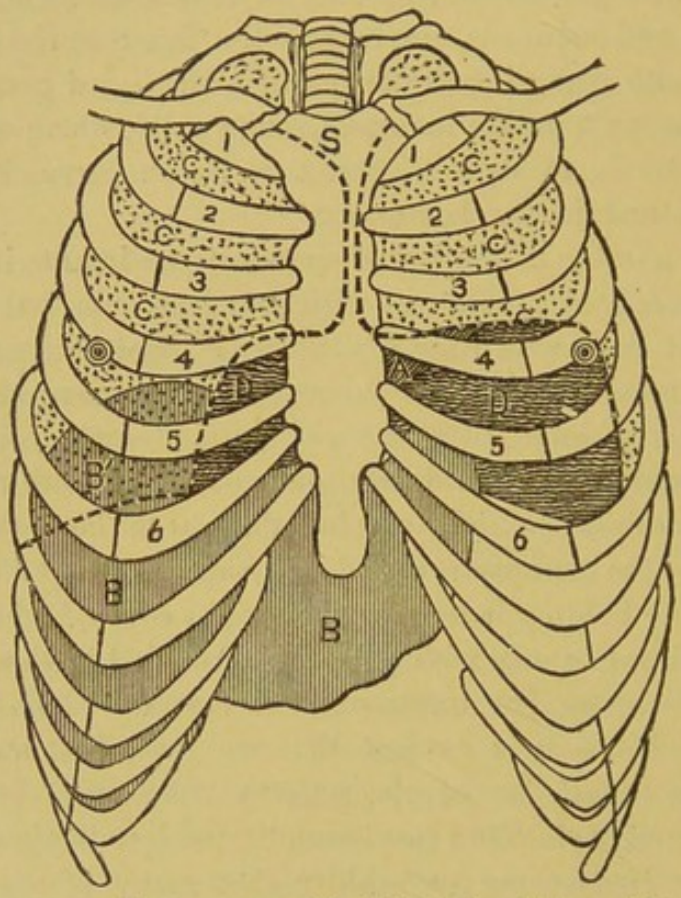

DIAGRAM I.—Small amount of liquid introduced into sac (Rotch).

A, The portion of the area of absolute dulness which is still caused by the physiological dulness of the heart.

B, Liver.

B', That portion of the liver which is covered by the right lung.

C, Lung.

D, Effusion.

A + D, Area of percussion dulness found when the effusion is small.

S, Sternum.

◎, Nipple.

1, 2, 3, 4, 5, 6, Ribs.

- - - Broken line, Border of lung.

extending to the right and to the left of the sternum; that the layer of fluid is a little the thickest in the left diaphragmatic depression, almost as thick in the right, and then that it gradually grows thinner as it ascends in the vertical line, and in a small effusion leaves a portion of the heart uncov-

ered in the region of the 4th left costal cartilage, as seen in Diagram II.

"The lungs gradually recede before the fluid, as seen in Diagram I., and the absolute dulness is found to the right of the sternum. Of this area of dulness, that portion which

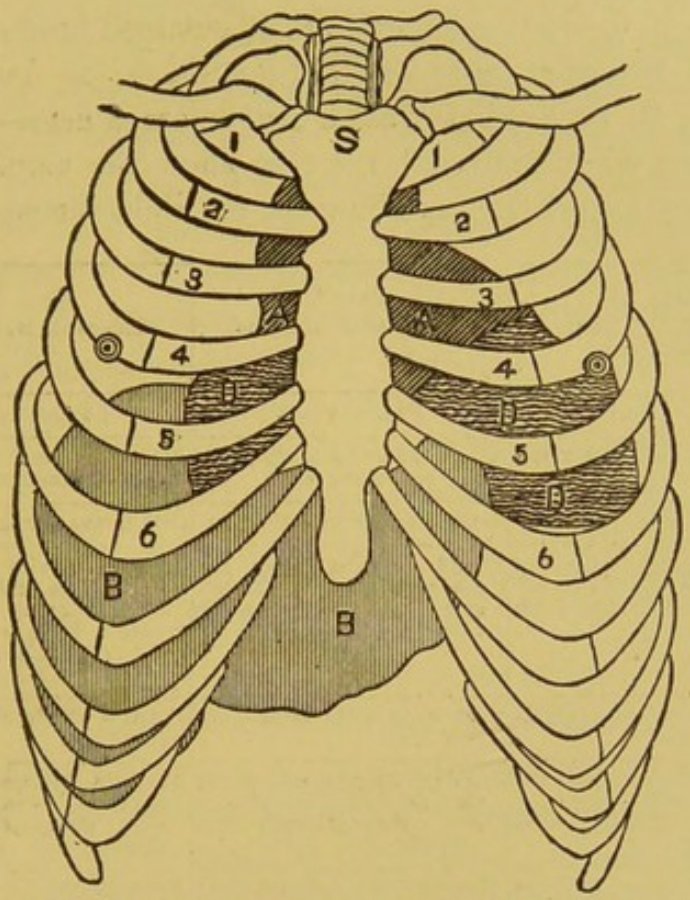

DIAGRAM II.—Represents Diagram I. with the lungs removed (Rotch).

A, A portion of the normal heart enclosed in the pericardium.

B, Liver.

D, Effusion as it appeared in the sac, the cocoa butter being in small amount, and the lungs having been removed after the butter had hardened.

S, Sternum.

, Nipple.

1, 2, 3, 4, 5, 6, Ribs.

is found in the 5th right interspace is of diagnostic value, first, because, unless there is consolidation of the middle lobe or a right pleural effusion, absolute dulness is extremely rare in this interspace, as proved by my having had hundreds of infants and children percussed in this interspace, with a resulting resonance; and second, because this interspace is not

encroached upon by even a largely-dilated heart more, perhaps, than one or two centimetres. In fact, it is doubtful whether the heart's absolute dulness ever extends beyond the right edge of the sternum as low down as the 5th interspace —an observation which is not alone the result of my own examinations, but that of many competent observers and authorities on the percussion outlines of enlarged hearts.

"The following cases—1. Dilated heart; 2. Pericardial effusion; 3. Pericardial effusion and enlarged heart—will be of interest when compared, for they show the value of this anatomical knowledge regarding the 5th right interspace.*

CASE 1. Endocarditis; Dilated Heart.	CASE 2. Pericarditis; Effusion.	CASE 3. Endocarditis; Enlarged Heart; Pericardial Effusion.
Girl. 11 years.	Boy. 6 years.	Girl. 8 years. Aug. 3, 1887.
Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.
Orthopnœa; præcordial pain.	Orthopnœa; præcordial pain.	Orthopnœa; præcordial pain.
Heart's impulse feeble, but perceptible a little to left and below left nipple, 5th interspace.	Heart's impulse feeble, but perceptible a little to left and below left nipple, 5th interspace.	Heart's impulse feeble, but perceptible, and felt all over cardiac area, with apex-beat a little below and to left of left nipple, 5th interspace.
Vertical absolute dulness not increased.	Vertical absolute dulness not increased.	Vertical absolute dulness not increased.
Absolute dulness under the sternum and to left of sternum: identical with cases 2 and 3.	Absolute dulness under the sternum and to left of sternum; identical with cases 1 and 3.	Absolute dulness under the sternum and to left of sternum; identical with cases 1 and 2.
Absolute dulness did not extend to right of sternum.	Absolute dulness in 5th right interspace 2 or 3 cm. from edge of sternum.	Absolute dulness in 5th right interspace 3 or 4 cm. from edge of sternum.
Systolic murmur at apex.	Pericardial friction rub at base.	Soft systolic murmur at apex, transmitted to axilla. Pericardial friction rub at base.
Recovery.	Recovery.	Aug. 6: Less dulness in 5th right interspace; apex murmur much louder and harsh. Aug. 11: Dulness only to right edge of sternum. Aug. 18: Dulness only to middle of sternum; friction rub ceased. Dec. 1, 1887: Physical examination the same as on Aug. 18, showing enlarged heart and mitral systolic murmur.

* 1 and 2 have been cited in my original paper; 3 is now under my care. In all the cases my observations and physical examinations have been verified by competent diagnosticians. The term "absolute dulness" is used as synonymous with "flatness."

"We have then three cases in children with, according to the combined report of a number of physicians who saw them, almost identical rational symptoms and physical signs, excepting that, while an effusion of any significance was present, dulness persisted in the 5th right interspace in cases 2 and 3, with complete disappearance of the dulness in case 2 and leaving the dulness of an enlarged heart in case 3.

"The next point of importance is the position of the apex-beat. It has been stated by certain writers that the apex-beat is displaced upward, but direct proof of this is wanting.

"Unless in some way intrathoracic pressure should cause displacement, it would be impossible for the heart not to sink rather than to be floated up, unless the specific gravity of the effusion was greater than 1.050, which seems highly improbable as likely to occur in an ordinary pericardial effusion, for the specific gravity of a purely purulent fluid is only about 1.032. There may, of course, be a lateral displacement.

"How, then, can we explain the clinical phenomena of the heart-beat at a higher point, say the 4th rib or 3d interspace? By referring to what I have said above, it will be seen that the heart's apex is masked by the mass of fluid in the left diaphragmatic depression, while the ventricular wall of the heart is either covered by a very thin layer of fluid and often is entirely uncovered at the 4th interspace, 4th rib, and 3d interspace, where, irritated by the abnormal effusion, it strikes against the thoracic wall and simulates an apex-beat, as was well exemplified in case 3, where it is recorded that the impulse was felt throughout the whole cardiac area, while the apex was still perceived in the 5th interspace. Now, in this case, if there had been a still greater effusion, the apex-beat would have been lost, but the impulse of the ventricular wall would have continued to simulate an apex-beat; and I believe that this is the case in all these so-called misplaced apex-beats.

"Now, if the apex remains in position in the 5th left interspace, and if the effusion is also in the 5th right interspace, where no part of the heart is present, is it not more rational to choose the 5th right interspace as the point for tapping? and this I have already done on the cadaver a number of times."

HYDROPERICARDIUM.

Dropsy of the pericardium is probably always a secondary disease. The pericardial sac normally contains a small amount of serum,—about half a drachm,—but it is usually increased during the last days of a patient's life, so that most usually on the post-mortem table we note a greater or less quantity of fluid in the pericardium.

Pericardial dropsy is met with in cases of general dropsy, in Bright's disease, emphysema following pertussis in the child, in thoracic deformities causing pressure, and in organic heart-disease causing blood-stasis.

The process is entirely passive, hence we do not encounter any fever, and no traces of inflammation can be noted upon the pericardial surfaces. The fluid is not particularly prone to become abundant, consequently bulging is not well marked. Friction-sounds never occur.

Hydropericardium may arise as the last scene in grave diseases. In such the differential diagnosis between muscular degeneration, its consequent asystole, and hydropericardium is indeed a difficult task.

In most cases of dropsy of the pericardium we may note a small pulse, dyspnœa, venous stasis, and cyanosis, scanty urine, and compression of the lungs, a train of symptoms that are not at all characteristic. Owing to the nature of the structures in early life we may observe depression of the diaphragm if the fluid is at all abundant.

Paracentesis may possibly be indicated in order to relieve symptoms or prolong life. The further treatment of the case is simply that of the disease with which it is intercurrent, and of the general anasarca.

HÆMOPERICARDIUM.

This is a rare disease in early life, except when due to rupture of the delicate vessels in cases of acute pericarditis in which the exudate is commencing to organize. It may also be due to either the rupture of a cardiac aneurism, an aortic aneurism of the first part of the arch, or even to a rupture of an aneurism of the coronary arteries.

Children may present pericardial hemorrhage in cases of scurvy or purpura. We may also call attention to our previous remarks on hæmorrhagic pericarditis.*

PNEUMOPERICARDIUM.

A pericardium containing gas is an extremely rare condition. Some authors state that they have observed it when the pericardium contains a decomposing fluid. The gas or air may be admitted from an abscess of adjacent tissues, which has perforated the pericardium. Death always rapidly occurs, except, however, in traumatic cases, in which recovery may and does sometimes occur.

Tumors may invade the pericardium. They are rarely primary, but most usually due to secondary involvement by carcinomatous growths which have their primary seat in the mediastinum or œsophagus. This condition is rare in early life.

CHAPTER V.

MYOCARDITIS.

MYOCARDITIS, or carditis, is an inflammation of the walls of the heart, and usually is a concomitant of endo- or pericarditis, the continuity of structure permitting an extension of the inflammation from the endocardium to the subendocardium and intermuscular connective tissue, or, on the other hand, may be an extension inwards from a primary pericardial focus of inflammation. In some instances the process undoubtedly has its primary seat in the muscular tissue itself.

Dietrich and Friedrich are of the opinion that youth predisposes to this affection, and cite as proof the eighteen cases of carditis recorded by Valex, six of which were children. In Bernheim's forty-six cases of myocarditis, twelve were between eight and twenty years. If we are to consider as myocarditis all those cases of softening or hardening, perforation of the septum, and partial, acute, or chronic aneurism that are in infants more than at any other age of life the result of direct

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* NOTE.—See Addenda, No. V.

inflammatory lesion of the muscular tissue of the heart, we can but agree with Dietrich and Friedrich, or even go further and say that the embryo and infant present the most favorable subjects for this disease. We may recognize two forms of the disease,—*indurative*, either local or general, and *purulent*. The indurative form may be further subdivided into the *acute* or *chronic*, either of which may occur during uterine life. In both the indurative and purulent forms leucocytes are infiltrated in the wall of the heart; the former, the indurative, causes thickening and hardening, which may occur in bands or patches of fibrous tissue. The endocardium is apt to show similar changes due to connective-tissue proliferation. Ziegler says that not infrequently some of the muscular trabeculæ are transformed into fibrous cords, and that the new fibrous tissue, when fully developed, is dense and almost free from cells; if the inflammatory process is kept up it may still contain infiltrated leucocytes in some spots. The muscular cells in many places may disappear; in other places, especially near the periphery, they remain, but show evident signs of atrophy. When the inflammation is more recent the tissue is grayer or more reddish, and richer in cells and vessels. At first nothing is seen but an infiltration of small cells, amid which the muscle-cells appear in various stages of disintegration, and sometimes hemorrhage occurs. The disintegrating muscle-cells are not afterwards replaced. If these sclerotic patches are large they seriously affect the heart. This hardened tissue is unable to dilate and contract with the rhythmic action of the heart; in its earlier stages the blood-pressure may cause bulging, and thus set on foot the changes which will ultimately terminate in a cardiac aneurism. Rokitansky reports such a case in a child aged nine. The connective tissue may constrict a portion of the cardiac wall and cause it to project within the auricular or ventricular cavity as a pedunculated fibroid polyp.

The production of cicatricial tissue may also have a serious tendency, as its contraction may cause valvular disease; two such cases are reported by Lösner showing rupture of the septum following endo- and myocarditis. This oc ırred in a poorly developed and cachectic child aged four, who was taken sick with scarlatina. The chest examination revealed the presence

of a pleural effusion with bulging; on the left side near the præcordia was heard a friction fremitus. Heart-beats were violent; auscultation over centre of right ventricle revealed a prolonged and well-marked presystolic bruit. No signs over the left ventricle or aortic region.

Autopsy.—Acute endocarditis localized in the left ventricle. Thickening of the endocardium, especially at the mitral and aortic valve. At the aortic region and just below the orifice was found a communication between the two ventricles, which opened in the right ventricle beneath the tricuspid valve. The opening was cicatricial and hard.

The other case was also aged four. The child presented a systolic and diastolic murmur.

Autopsy.—Tubercles of lungs and glands; in the left ventricle the endocardium was thickened; at the mitral orifice an opening was seen beneath the aorta, which opened in the right ventricle beneath the tricuspid valve. These two cases probably had their death accelerated by the occurrence of scarlatina, as the communications had probably long existed and may have been congenital.

Ziegler pursues the subject somewhat further, and describes *myomalacia cordis* as a peculiar softening of the heart due to arterial anæmia, the usual cause of this anæmia being sclerosis of the nutrient arteries of the heart, or the coronary arteries and their branches. It has been noted to have arisen after coronary embolism. This softening may be very slight, or so extensive as to include almost the entire thickness of the heart-wall, rupture of the heart resulting, the blood escaping into the pericardial sac and death rapidly closing the scene.

Clinicians have most usually confounded myomalacia cordis with myocarditis, or fatty degeneration, but Ziegler concludes that the affection is really anæmic necrosis, but that fatty change and inflammation do at times accompany it, but not as a chief or primary condition. He compares this cardiac softening with cerebral softening, or encephalomalacia.

Purulent myocarditis has already been referred to while considering ulcerative endocarditis. This condition is usually seen in connection with pyæmia, and is due to bacteria deposited in the heart-muscle from the blood-current. One or many

small, grayish-yellow abscesses may form and rupture either internally or into the pericardium. Leyden* says that diphtheria often causes myocarditis without an attendant endocarditis, and that myocarditis may be seen in association with variola, epidemic meningitis, and relapsing fever.

Rosenbach† has recorded granular and waxy degeneration of the heart-muscle in conjunction with diphtheria. Blache records the following case: a child, aged thirteen, was attacked by acute articular rheumatism twice within a year, and developed an aortic murmur. A year later it died, and the heart, on post-mortem examination, was hypertrophied, with thick and rigid walls. On the right side were noted disseminated spots of inflammation throughout the muscle. The left ventricular muscle was inflamed and transformed into fibroid tissue; it was hard, resisting, and bleached-looking, not unlike cicatricial tissue. Here it was supposed that the myocarditis was the primary affection and the endocarditis and valvular disease secondary. The myocarditis was probably due to infarcts, as atheroma is rare at this age. Paul also notes the association of myocarditis with typhoid fever and rheumatism, and remarks that the selective sites of myocarditis in their order of frequency are first the apex of the left ventricle, then the base, posteriorly near the aortic valves, then the septum near the base, and more rarely the papillary muscles and fibres of the right ventricle.

Rokitansky believes that the wall of the left ventricle is the most frequent seat; Dietrich, the septum, thus explaining the cases of patulous septum. It is sometimes, however, difficult to say whether these fistulous tracts are congenital or acquired.

Blache classifies as follows the diseases with which myocarditis may be associated:

General diseases.	General illness.
	Grave fevers—variola.
	Cachexiæ.
Local causes.	Diseases of pericardium.
	" endocardium.
	" vessels of heart.
	Abscess or tumors of heart.

* Zeitschr. f. Klin. Med., iv., Ziegler. † Virch. Arch., vol. lxxix.

Alteration in circulation.
- Embolism.
- Thrombosis.
- Atheroma.
- Œdema.

Symptoms.—The symptoms of myocarditis depend, of course, on its form and the extent to which the disease has progressed. When the lesion is small and limited, few, if any, symptoms exist at all by which a clinical diagnosis can be made. If, however, the lesion is extensive, then we meet those symptoms which we all recognize as characteristic of heart-disease, as dyspnœa, palpitation, dropsy, visceral derangement, or præcordial discomfort; nervous symptoms are peculiarly liable to arise early in these cases. Friedrich cites such a case, in which the nervous symptoms were so severe in a child aged eight that the disease was alone discovered at the autopsy, when an abscess was found between the auricle and the right ventricle. Rarely do we meet a murmur, and when it does arise it is in all probability due to infiltration and thickening of one or more chordæ tendineæ, thus interfering with their normal action.

Most cases of myocarditis pursue a long course; should, however, an aneurism develop, the case will be more rapid. In other cases sudden death may occur from cardiac arrest. The most usual termination is, however, by dropsies, pulmonary complications, or by exhaustion.

Traumatism may cause myocarditis and rapidly prove fatal. A case is recorded of a child aged twelve who was kicked over the heart and died shortly afterwards, with abscesses in the heart-muscle.

The *diagnosis* is indeed difficult, and in many cases quite impossible; this is particularly true of the so-called cerebral form of myocarditis, which is especially noticeable during early life. Burnheim reports several cases in children. A child aged twelve, with febrile symptoms, delirium, agitation, and dilated pupils, died four days after admission. Child never had a pain in the chest, or heart-irregularity, or cardiac palpitation. At the post-mortem the heart was of a deep red color, softened, and easily torn; in the walls of both ventricles a number of abscesses were found, with quite a number be-

neath the visceral layer of the pericardium; auricular muscle softened. Heart-clots were numerous. It is a clinical nicety to differentiate between cases of subacute or chronic myocarditis and the heart that accompanies emphysema, or that seen with renal disease, or again, the heart that is altered by fatty degeneration.

As a further illustration of the difficulty in diagnosis we cite the case of a child aged six, seen by Labric. The child had a left-sided pleurisy, and died soon after the effusion had formed.

Autopsy.—Pleura thickened and covered by false membrane. The pericardium was distended with seropurulent fluid and false membrane around the entire heart. The heart-muscle was softened, pale, and in patches discolored; near the pericardium the muscle was infiltrated with embryonic cells; the pericarditis evidently had caused the inflammation to extend to the myocardium. The endocardium was found to be hyperæmic, and at the valves the early stages of endocarditis could be noted.

Prognosis.—If we are able to make the diagnosis the prognosis is unfavorable, because cases which are far enough advanced to diagnose are almost in the last and fatal stage of the disease.

Treatment.—It is hardly necessary to add that treatment is of little avail. Should the patient present evidence of syphilis, either hereditary or acquired, iodide of potassium may be administered in moderately large doses. In some cases, independently of any specific action, the drug seems to be of benefit. Trinitrin (nitroglycerin) and adonidine may also prove efficacious. Digitalis is to be exhibited with the greatest care and caution. Rest, regimen, and supporting measures directed to the general well being of the individual about complete the plan of treatment.

TUMORS, NEW GROWTHS, AND PARASITES OF THE HEART.

The heart-muscle, as we noted in cases of myocarditis, may undergo a sclerosis. This sclerotic change has been noted to be so localized that the term *fibroma* could with propriety be applied to it. A fibroma as large as an English walnut has

been remarked in the thickness of the left ventricle of a boy aged six years by Luschka,* and Wagstaffe,† in a girl of three months, noted a fibroma situated within the septum and filling the greater part of the cavities of the heart.

Myomata, or muscular tumors of the heart, may not infrequently be noted. They are almost always congenital. Virchow‡ and Recklinghausen§ have recorded several instances of this new growth. The following true tumors may also invade the heart primarily, but they are rare: sarcoma, lipoma, and myxoma.

Tubercles in acute miliary tuberculosis may be deposited in the heart. On very rare occasions we may find cheesy masses, the result of chronic tuberculosis of surrounding organs; almost invariably are they secondary to caseous nodules in the pericardium.

Secondary carcinomata is probably the most frequent new growth of the heart; *syphiloma* is more rare; gummata are extremely rare; when they occur they will be found in the myocardium, imbedded in dense hyperplastic fibrous tissue; if they are recent, will appear as soft grayish-red patches; if older, as a dry, yellowish, cheesy node.

B. Teisser‖ records an interesting case of cardiac syphilis in a young prostitute, who was in the third year of syphilis, which, however, had only manifested itself in the form of buccal mucous patches. She was suddenly seized with dyspnœa, followed by asphyxia and death within twenty-four hours. The autopsy showed extensive involvement of the anterior walls of the right ventricle in its upper half, the muscular tissue of which seemed to have become entirely transformed. The thickness of the cardiac wall appeared about normal, but it seemed of a peculiar light-gray color, and its consistence much firmer. Section showed numerous milk-white lentil-sized nodules, both in the cardiac walls and elsewhere. These presented a caseous appearance, but were in reality of quite

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* Virch. Arch., Bd. viii. † Trans. Path. Soc., xii., p. 121, 1871.

‡ Virch. Arch., Bd. xv., xxx.

§ Myoma cordis.—*Monatschr. f. Geburtsk.*, Bd. xx., 1862.

‖ Annals de Derm. et de Syph., 2me sér., t. iii., No. 6; Gaillard's M. J., March 17, 1883.

firm consistence, showing no trace of softening even in the centre.

In addition to the interstitial myocarditis and the gummatous deposits, there were considerable vascular alterations in the form of peri-arteritis and endo-arteritis.

Ziegler remarks that simple inflammatory indurations of the heart-muscle occur as a consequence of congenital syphilis, and that this condition is much more common than are gummata.

Calcification of certain parts of the heart near the valves is not infrequently noted. At times concretions may be found in an abscess of the myocardium which has been absorbed.

Parasites.—Cysticercus, echinococcus, and trichinæ are found in the heart. According to Cobbold, 3.5 per cent. of all cases of hydatids in man occur in the heart.

Heart-clot will be considered in a special chapter.

CHAPTER VI.

VALVULAR DISEASE.

HAVING considered the anatomy of the heart, the rationale of its sounds, the fœtal circulation, and the congenital anomalies of the heart and circulatory vessels,* we are fully prepared to correctly interpret the murmurs which occur in the heart and great vessels. Heart-murmurs are modifications in, or replacement of, the normal heart-sounds by certain new and adventitious sounds which we designate murmurs. These new sounds are either produced within the heart (valvular murmurs), on its surface (friction-murmurs), or in the blood-current within the heart or great vessels at its base (hæmic or functional murmurs).

We also find murmurs in the heart unconnected with any structural lesion, to which we will refer later on in our study.

It will be serviceable for a moment to consider the distribution of the blood-supply in the valves, as it has an important bearing upon valvular disease.

E. Coen† has experimented on the human heart, and the heart of the various domestic animals, in order to study the blood-supply of the cardiac valves. In order to obtain complete injections he studied the hearts of the fœtus and new-born children. His injections were made with gelatin and Berlin blue. He found (1) in the pulmonary and aortic semilunar valves no vessels in the substance of the valves.

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* See Chapter I.

† Blood-Supply of the Cardiac Valves; Bullet. delle Scienze Med., Sept. 1886; London Med. Rec., March 15, 1887, p. 116.

The vessels that come from the cardiac parietes with the muscular fibres stop at the margin of implantation of the valve itself, and then form a very distinct and fine net-work, sharply limited. (2) In the auriculo-ventricular valves blood-vessels are always present, which starting from the margin of implantation of the valves extend to them in a fine net-work. Three or four principal branches extend into the valve, and subdivide into an irregular net-work. This net-work is prolonged to the point of implantation of the chordæ tendineæ, either in closed loops or with very fine terminal branches. The thickness and compactness of the net is greater towards the point of implantation of the valve than towards its free margin. Numerous vessels start from the papillary muscles, and extend for a certain distance along the chordæ tendineæ; but Coen has never seen these little vessels pass through the whole length of the chordæ tendineæ, nor their anastomosis with the capillary net-work of the valves, as Luschka, Henle, and Krause assert.

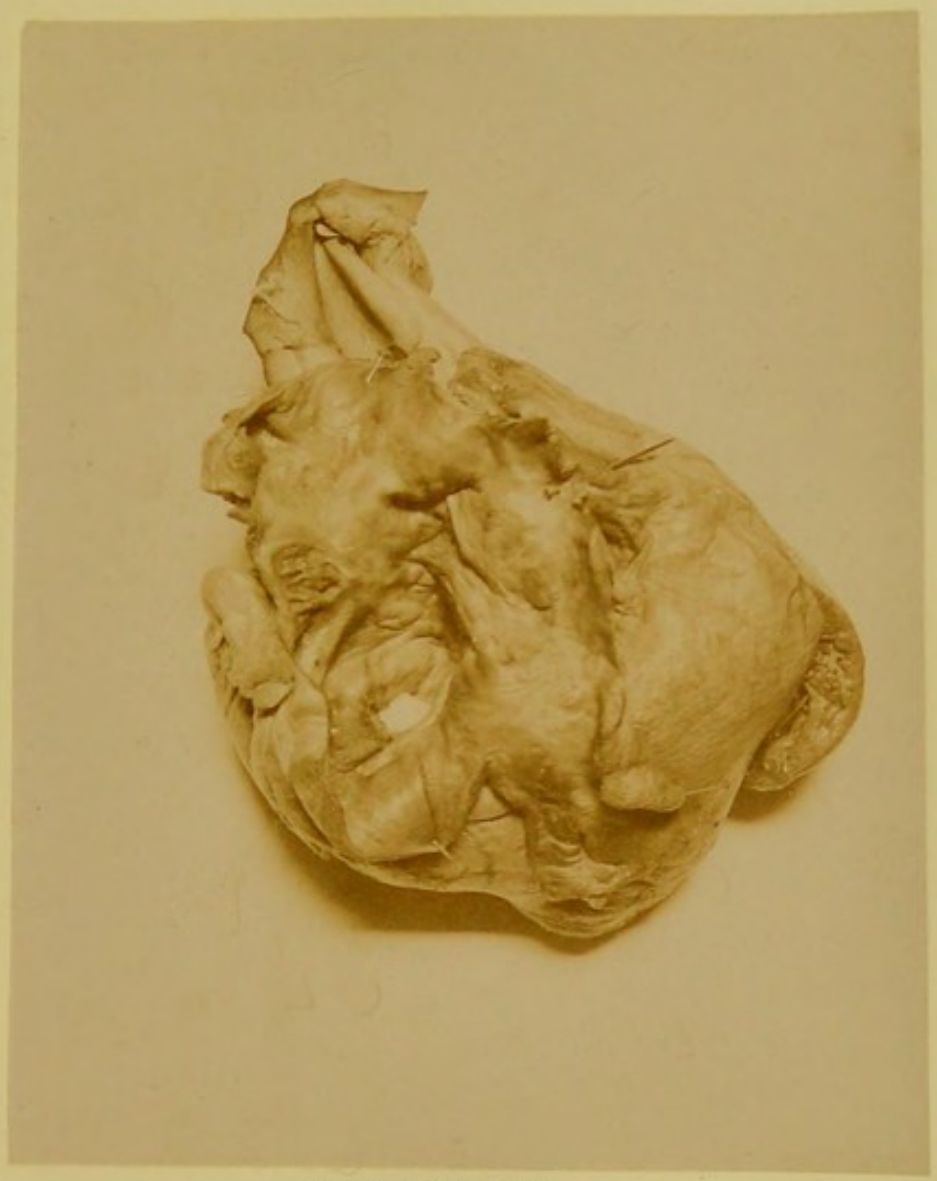
The vascularity of the auriculo-ventricular valves must, therefore, be admitted, but not that of the semilunar valves.

Let us first, then, consider endocardial murmurs, valvular disease, as at this situation we are able to detect the disease by the physical signs.

These murmurs have a common quality, they are all blowing; yet the sound itself may present all variations in the musical scale. Much time has been spent by writers in endeavoring to classify them by their relation to some familiar sound, as rasping, filing, sawing, blowing, cooing, and sighing, all of which are, however, irrelevant, as they teach us but little as to the real source of a murmur.

Most endocardial or valvular murmurs are due to a change at the valvular orifices, either a narrowing or stenosis or an insufficiency, with inability to close the aperture, permitting regurgitation. The possible valvular murmurs in the heart (exclusive of certain congenital defects, as slits and perforations) may be represented as follows:

Mitral valve . . { Regurgitation. / Obstruction.



MITRAL STENOSIS.

Tricuspid . . .	{ Regurgitation. Obstruction.
Aortic . . .	{ Regurgitation. Obstruction.
Pulmonary . .	{ Regurgitation. Obstruction.

We thus see that it is possible to have two murmurs at each valve, or eight in all. Some of these are, however, of extreme rarity as primary murmurs; it is rare, indeed, to meet with a tricuspid stenotic murmur; so, also, are the murmurs at the pulmonary orifice and valve rare except as congenital disease, as we saw earlier in our study. So of these eight murmurs we can practically exclude a tricuspid narrowing and consider the murmurs at the pulmonary area as congenital, with, however, certain exceptions, which we will note under the appropriate heading.

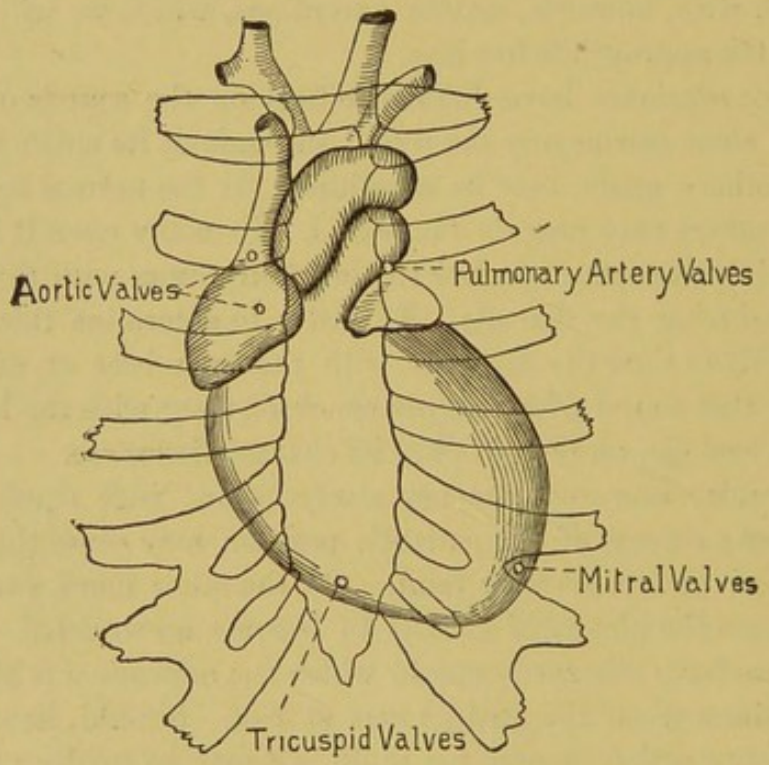
These murmurs have different effects on the sounds of the heart: some accompany the sound throughout its entire duration; others, again, may be substituted for the normal sound; again, others may precede the sound. In many cases it is indeed difficult to say whether the murmur occurs with the systole or during the diastole. In order to determine this fact, it is well to time the murmur with the apex-beat or carotid pulse; that sound which occurs synchronously with the heart-beat is systolic, no matter what its characteristics are.

Valvular murmurs are not always heard with equal distinctness; change of the patient's position may cause them to disappear or become very faint. On the other hand, exercise will cause the abnormal sounds to become accentuated. Indeed, we have observed cases in which the murmur was absent at all times when the patient was at rest. Should, however, the cardiac action become too rapid, we may be totally unable to differentiate the murmur, especially with children who normally have a quick circulation.

Sometimes the patient may himself recognize the murmur, or it may be audible to those around. Such a case is recorded by Osler of a child, æt. eleven years, in which the murmur could be heard with great distinctness several feet from the patient.

Murmurs are sometimes heard in the recumbent position which are totally inaudible when the patient is erect. Hutchinson* believes that this is a good diagnostic point, as anæmic murmurs are best heard with the patient recumbent. We are unable to satisfy ourselves of the fact stated by many authors, that pressure of the stethoscope will intensify valvular murmurs.

Having determined the existence of a murmur, we must now definitely locate its seat and its character, whether it is an obstruction in the flow or a regurgitation. This is, of course, determined by the fact that a sound has its maximum intensity near its seat of production, and, all things being equal, the closer we approach this seat the louder will the murmur become.



The figure, taken from DaCosta, serves to illustrate the four points at which we endeavor to isolate sounds produced in individual valves, remembering always that the points marked in the illustration are not the anatomical seats of the valves, but are the situation at which our ear will most closely ap-

* Am. J. Med. Sci., April, 1872.

proach the valve; the valves themselves lie about opposite the third intercostal space at the left border of the sternum and slightly beneath the bone. Placing our ear or stethoscope at the apical impingement and hearing a murmur at or near this apex-beat, we conclude that it is produced at the mitral orifice; if its intensity is greatest at the ensiform cartilage or a little above it, then we conclude that the tricuspid orifice is at fault; if, on the other hand, we determine that the murmur is heard with greatest distinctness at or near the second costal cartilage on the right side ("aortic cartilage") or a little farther down the sternum, about opposite the third intercostal space, we are to decide that we are dealing with a case of aortic valve disease.

The pulmonary artery and valve are so rarely the seat of disease, except in congenital cases, which we have already considered, that it is sufficient to call attention to the cut for the manner of determining its site. Mitral murmurs are occasionally heard with startling distinctness in this so-called pulmonary artery region.

After having determined the existence of a murmur and its relation to the cardiac rhythm, we are then to seek out its line of transmission, as it is a well-recognized law in physical diagnosis that a murmur is carried or propagated in the direction of the blood-current; for example, an aortic obstructive murmur will be heard at its seat of production and also in the neck circulation, sometimes in the subclavian or brachial, or even in the external iliac.

Mitral regurgitation.—The valve is usually rendered incompetent by rheumatic endocarditis, which may have been congenital or post-natal in origin, or by the other morbific actions considered under the head of endocarditis. Cases are recorded in which regurgitation has occurred owing to imperfect development of one or more leaflets of the valve, although this is a relatively much less frequent congenital anomaly than in any of the other valves; again, several of the chordæ tendinæ may be either congenitally shortened or contracted following endocarditis, and pulling the curtain from its normal position, thus causing irregularity in its action or adhesion of the point of the valve to the ventricular wall, may cause abnormal action.

In order to determine the existence of a mitral murmur we may proceed as follows: Listening over the base of the heart and determining the existence of a murmur, we would then place our ear or stethoscope over the apex-beat, and if the valve is permitting regurgitation of the blood we will realize the fact that the abnormal sound is here heard with maximum intensity, and that it is systolic in time. Furthermore, we will also determine the fact that the murmur may be heard in the left axilla and at the point of the left scapula, remembering, of course, that the murmur in this case is not following the law of transmission above noted, because there is no blood carried in the direction under consideration, but that we simply hear the murmur at these points because during systole the heart rotates upon its axis, the left ventricle more nearly approaches the bony chest-encasement, and at the points named is covered by a smaller amount of lung-tissue and a small amount of chest-wall, and consequently is nearer the observer's ear; hence the sound becomes more intensified at this time. Sometimes the murmur will not be carried beyond the axilla, failing to reach the scapula; again will we meet a regurgitant murmur, which will be localized entirely to the apical region.

In certain cases of mitral regurgitation, the murmur is heard with startling distinctness in the so-called pulmonary artery region. Naunyn's explanation of this has always seemed to us to be the correct interpretation; he first says that the situation of maximum intensity in these cases, in the second left intercostal space, does not correspond exactly to the place where the sound of the pulmonary artery is heard; that is to say, not quite at the edge of the sternum, but a little distance from it, or at a point which almost corresponds with the situation of the left auricular appendix, which winds around the pulmonary artery and lies in front of it. Now in a case of mitral insufficiency the abnormal current of blood flows into the auricle, and as the appendix freely communicates with the auricular chamber, we can readily see how it is that in certain cases, when the appendix is long enough to approximate its tip closely to the anterior wall of the thorax, we hear a mitral regurgitant murmur in the pulmonary area. We may state that localization of murmurs in young children is

MITRAL REGURGITATION.

rendered more difficult by anatomical conformation, the small relative size of the heart, and the proximity of the ear to the heart itself.

The diagnosis of a mitral regurgitation is not completed until we have recognized the *local* and *general changes* consequent upon its existence. It is also upon the gravity of these concomitants that we in great part determine the significance and prognosis of the affection.

In most cases there will be found to be a slight mitral stenosis coexisting, which, in a measure at all events, tends to compensate the insufficiency. During systole the blood finds its way into the left auricle through the insufficient valve, the leak detracting from the amount which the ventricular systole should have expelled into the aorta. We thus see that, on the one hand, the aorta and the general circulation receives too little blood, whereas the left auricle receives too much, as it is now supplied by the left ventricle in addition to its normal supply from the pulmonary veins. This increased supply rapidly sets on foot hypertrophic changes in the auricular wall, which almost as rapidly gives way to dilatation. Synchronous with the hypertrophy is to be noted a rise in tension, starting in the auricle and being propagated into the pulmonary veins, which will give evidence of increased nutrition in the hypertrophic condition of their walls, with here and there foci of fatty degeneration. The increased tension does not, however, stop at the veins, but extends into the capillaries of the pulmonary artery and on into the right ventricle, then the auricle, and eventually into the entire venous system. A moment's reflection will show us that this general reversal of pressure will produce diminished tension in the aorta and increased tension in the venous system. Hence will we find, in cases of mitral insufficiency permitting regurgitation which has been of any duration, that the tension is highest in the pulmonary veins and venæ cavæ, and at its minimum in the aorta.

The chambers of the heart become speedily deranged, some by dilatation and others by hypertrophy, or by a combination of both. The left ventricle is usually found in a condition of hypertrophy, as it receives the blood under high pressure from the pulmonary veins, and furthermore, it performs all its func-

tions under high pressure also. The left auricle will usually, in fact always, be dilated; the right ventricle hypertrophied.

The early alteration in the cavities occurs without the individual's knowledge; it is only when the compensation is deranged and the cardiac equilibrium is interfered with that the patient becomes aware of any discomfort. This change in the cardiac action is usually due to alteration in the myocardium, allowing dilatation; should the pericardium be adherent, this dilatation the more speedily arises. Now it is that palpitation arises, owing to the dilated chambers endeavoring to expel their unduly large contents, more noticeable, of course, during active exercise, when the circulatory apparatus receives undue stimulation. Shortness of breath will also become an exacting symptom, as the system at large demands increased oxygen, which even the rapid breathing is unable to supply; local or general dropsy is apt to arise, and, when occurring in the serous cavities, constitutes a grave element in the prognosis. Dry cough, in many cases due to the congestion of the bronchial vessels, will to the patient be the sole symptom that leads him to consult medical aid, and then the condition of the mitral valve is revealed. By this time the alteration in the chambers has so far progressed that the cardiac impulse will be seen and felt lower down than usual, even as low as the sixth or seventh interspace and outwards to the axillary line, or, as we have noted it, in the axilla. Inspection will also reveal marked undulations, even as far to the right as the epigastrium; a purring sensation, the purring tremor of Laennec, will be recognized by placing the hand over the præcordia, synchronous with the systole. Percussion will show a greatly increased area of dulness, which will still, however, maintain the shape of an irregular triangle, to which we called attention earlier in our study.

The degree to which the chambers in cases of mitral disease may undergo dilated hypertrophy and thus alter the percussion outline is well illustrated by the accompanying copy of a photograph of a child under the care of Dr. Hare,* whose history is as follows:

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* We are indebted to Dr. Hare for permission to publish this case.

Lizzie D., æt. twelve, presented herself at the Children's Dispensary of the University of Pennsylvania.

+ Mark shows point of main apex-beat; the broken line indicates approximation of cardiac and hepatic dulness.

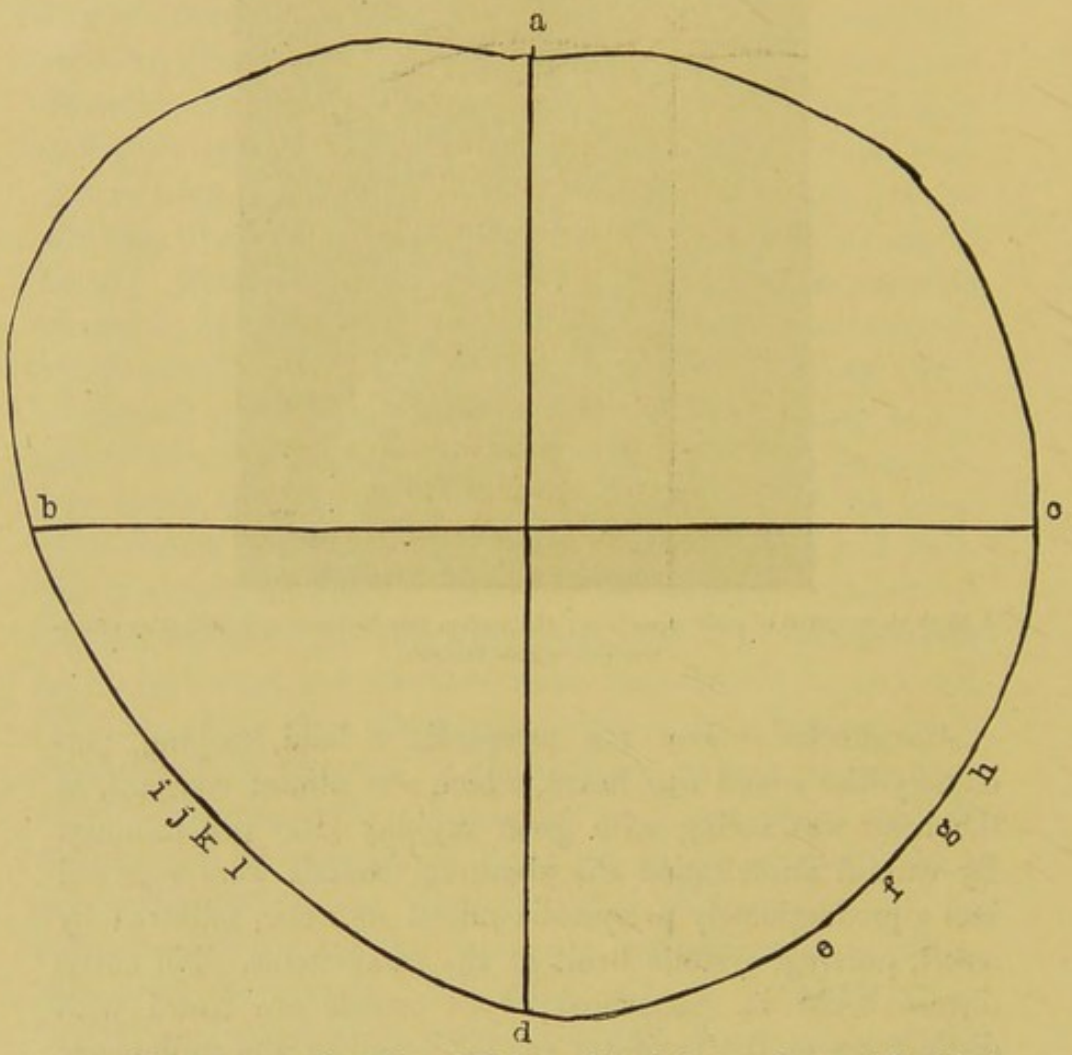
Auscultation.—Over the præcordia a loud, rasping, machinery-like sound was heard, which was almost constant, as the heart was acting with great rapidity (150 per minute). By careful auscultation the abnormal sounds were resolved into a predominately presystolic mitral murmur, followed by a soft, purring, systolic bruit at the same valve. No aortic disease could be discovered; both sounds are heard with distinctness at the ensiform cartilage, and at the pulmonary area the second sound is markedly accentuated. The mitral murmur has a wide distribution, is just heard immediately to the left of the sternum in the second and third interspaces, increasing in intensity as the apex is approached and passed and carried on to the angle of the left scapula.

There is no arterial pulsation visible at the wrist.

Cardiac dulness began an inch to the right of the sternum, and extended to the eighth interspace at the posterior axillary line. The distance from the nipple to the extreme left point

of dulness was three and a half inches. The greatest area of dulness from right to left was eight inches. These points are indicated by the iodine line on the skin seen in the photograph.

Palpation reveals a thrill and apex-beat in the sixth, seventh,



a, Spinous process of vertebræ. *b*, Extreme right of chest. *c*, Extreme left of chest. *d*, Sternum. *e*, *f*, *g*, *h*, Abnormal anterior body of chest. *i*, *j*, *k*, *l*, Normal anterior body of chest.

and eighth interspaces, the maximum intensity being in the sixth and seventh.

The bony chest-encasement, in cases of extreme increase in cardiac volume, will, in the young, be much altered, as may be appreciated by noting the chest tracing taken from the above case with a lead-tape.

The child presented but few, if any, symptoms of cardiac

disease, except the physical; there was no dropsy or other concomitant, except shortness of breath, for which symptom she sought medical advice. She and her friends can recollect no attack of acute disease, and state that the child has hardly been sick a day in her life.

TRICUSPID DISEASE.

PRIMARY disease of the tricuspid valve is somewhat unusual if we exclude cases having a prenatal origin, which have already been considered, consequently primary murmurs are rare in this situation. When heard they will most usually be found to be dependent upon mitral disease which has weakened the right heart.

Balfour is of the opinion that mitral stenosis is the most frequent cause of serious tricuspid regurgitation, and the earlier in life the stenosis occurs, the more rapidly, as a rule, the tricuspid regurgitation follows, and the more serious the prognosis. As we noted in the mitral, so in the tricuspid, the murmur originates almost always from endocarditic disease, or, as Loomis says, from syphilis. The disease most usually begins during fœtal life. It seldom occurs as an independent affection in adults. It, however, does occasionally attack this valve primarily in an adult, as a case which occurred in our hospital wards last winter will illustrate. Rosenstein says that out of a hundred cases of valvular disease scarcely two occur in the tricuspid, and even in these two cases the tricuspid is not the only affected part, being nearly always accompanied by disease of another valve, usually the mitral.

Tricuspid insufficiency.—The insufficiency is more apt to be relative than absolute; that is, we are more apt to find that the ventricle is dilated so that the ring of insertion of the tricuspid valve has become so large that the flaps, while almost

histologically intact, are unable to close the opening, than we are to note shrinking and thickening at the border of the valves. Friedreich has definitely demonstrated the fact that simple dilation of the ventricle will be sufficient to stretch the valves far enough apart to allow regurgitation.

Symptoms and physical signs.—The valve being incompetent, each systole causes a backward flow of blood from the right ventricle to auricle, and to the veins which communicate with it. The auricle speedily becomes dilated and hypertrophied, the latter in proportion to its muscular tissue, which is slight at best. The backward flow causes increased tension in the vena cava and a decrease in the pulmonary artery and the aorta. At about the fourth rib may be noted an increased area of dulness, which may extend well over to the right and below the ensiform cartilage. The ventricle becoming dilated and hypertrophied encroaches upon the left heart, and eventually finds its way in front of the left chambers; as a consequence the apex-beat becomes wavy and ill-defined. We usually note a condition of overfulness of the cervical veins, which may present a wavy impulse, systolic in time. This is much more apt to be marked in children than in adults. The portal system becomes much congested, the liver enlarged, its left lobe often receiving an impulse from the over-distended ventricle. Hemorrhoids may arise, and constitute an annoying symptom. The urine may become scanty or suppressed. Œdema and anasarca may arise late in the case, as Day* remarks in a boy, aged 11 years, under his care, in whom œdema and anasarca of the lower extremities came on a month before death. Loomis remarks that in tricuspid regurgitation dropsy of the genital organs rarely arises. All the general constitutional symptoms of cardiac disease are apt to arise early in tricuspid affections, and are almost never absent.

The physical signs in most cases of tricuspid disease will be somewhat obscure, and give evidence of a multiple cardiac lesion.

Auscultation will reveal a blowing or cooing murmur at about the ensiform cartilage, systolic in time, having its point

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* Med. Press and Circular, Jan. 10, 1881, p. 47.

of maximum intensity a little to the right of the bone. The second sound over the right heart is apt to be weak, on account of the deficient circulation in the pulmonary artery. From the fact that mitral disease most usually coexists we are apt to note a small radial pulse, other than this it gives no indication of disease.

In severe cases we may hear the "sound of the jugular valves," first described by Bamberger, who considered it of great diagnostic value. The sound is produced in the veins in the neck, more particularly the jugular, by the fact that the reflux of venous blood is confined to the bulk of the vein, as the valves are unimpaired, and a sound is produced by their closure. When the valves are insufficient the pulsation occurs along the entire vein. Theoretically, liver pulsation should arise very early in a case, on account of the valveless condition of the veins in that organ, but clinically it is a very unusual symptom. However, we should bear in mind that the venous pulse may appear in both the liver and neck, and disappear several times in the course of the disease. Geigel has demonstrated the fact that under favorable circumstances the vena cava inferior may be seen and felt as a pulsating vessel, even in the right mesogastric space.*

Diagnosis.—The fact that we are not dealing with a single murmur but usually with a multiple lesion renders the diagnosis somewhat obscure. Bearing in mind the topography of the cardiac apparatus, we will at once see that a tricuspid regurgitation is always heard at the lower and right border of the sternum, never rising as high as the third rib. Furthermore, we find no accentuation of the second sound, but, on the other hand, find jugular and epigastric pulsation, with possibly a venous hum in the neck circulation. From mitral disease we differentiate tricuspid regurgitation by following out the transmission of the murmur, noting the condition of the cardiac cavities, and recognizing the accentuated second sound which is apt to accompany mitral regurgitation. Both of the murmurs are systolic in time. The further differential points have been considered under the heading of Mitral Disease.

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* S. S. Rosenstein, Cyc. Proc. Med., vol. vi. p. 149.

Tricuspid stenosis.—This lesion is so rare that we hesitate to formulate any rules for its recognition. A stenosis confined to this valve *alone* has, so far as we know, not yet been recorded. Loomis, however, mentions one case recorded by Bertin* which we have not had the opportunity of verifying.

Its causes, independently of congenital abnormalities, already considered, are the same as those that we have laid down for the other valvular lesions.

Bedford Fenwick,† however, takes exception to the statement that tricuspid stenosis is rare, except as a congenital condition, thus differing with Flint, Niemeyer, Peacock, and Rosenstein.

Fenwick, in his history of forty-six cases, most of which are in adults, remarks that the disease is almost always acquired in after-life, and that it is but very rarely, perhaps never, when found in adults, one of congenital or intra-uterine origin. The youngest age at death, of Fenwick's cases, from tricuspid stenosis, was a girl aged seventeen. The history of the case, dying at seventeen, shows definitely that the lesion was not of congenital origin. The child was in good health until she suffered an attack of acute rheumatism; after some years attention is directed to her heart, and it is found slightly diseased. Five years later she is found to have mitral stenosis and commencing disease of the aortic orifice; this by the end of the next year is found to have progressed into a state of stenosis; finally, in another year, a fresh attack of acute rheumatism has exhibited fresh inflammatory power, and she is now found to have the tricuspid orifice stenosed. Fenwick asks, Is there here a trace of a fact to support the theory of a congenital origin? His conclusions are so apt that we will quote verbatim:

1. Tricuspid stenosis is not "extraordinarily rare."

2. It is more common by far in females than in males, perhaps in the proportion of seven or eight to one.

3. It is always associated with stenosis of the mitral orifice.

4. In about twenty-five per cent. of cases the aortic orifice is also stenosed.

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* Traité des Maladies du Cœur, obs. 17.

† Trans. Lond. Path. Soc., vol. xxxii. p. 44.

5. In almost exactly fifty per cent. there is antecedent history of acute or subacute rheumatism.

6. Tricuspid stenosis, when found in adults, is due to acquired disease, and very rarely, perhaps never, of congenital origin.

In further illustration of the fact that tricuspid stenosis is occasionally met with, we cite the following cases:

Sex.	Age.	History.	Weight of Heart.	Size of Tricuspid.	Concomitants.	Reference.
M.	14	Rheumatism two years before. Second attack six months before death.	13 oz.	Only admitting little finger.	Right ventricle dilated. Right auricle vegetations. Left ventricle dilated—hypertrophy. Mitral stenosis.	A. Boye Barrow, Trans. Lond. Path. Soc., vol. xxxii. p. 74.
F.	12	Rheumatic at eight years. Second attack at twelve.	12½ oz.	Tips of two fingers; buttonhole.	Adherent pericardium. Left auricle dilated. Mitral stenosis. Died at age of twenty-three.	P. Horrocks, Ibid. p. 76.
F.	Young.	.....	.....	One finger.	.....	Flint, Diseases of Heart.
F.	19	No rheumatic history.	.....	Contracted, rigid, and ossified.	.....	Burns, quoted by Peacock; Fenwick (Ibid.).
F.	17	No rheumatic history, but much exposure.	11 oz.	Two fingers.	Mitral stenosis. Right cavities dilated. Left cavities dilated—hypertrophy.	Goodhart, Brit. Med. Jour., Sept. 23, 1871.

AORTIC DISEASE.

DISEASE of the aortic orifice is relatively less frequent in the child than in the adult; it is also less frequently met with than disease of the auriculo-ventricular orifices.

Aortic insufficiency, arising primarily in infants, is among the uncommon clinical observations, excluding cases having congenital origin; in older children it is not at all rare. The etiology of aortic insufficiency is much the same as we have already considered in treating of the general subject of valvular disease. At this valve, however, we are more likely to

have an insufficiency suddenly produced during sudden exertion from the rupture of a segment of the valve, occurring usually at the free border, sometimes, however, at the insertion of a leaflet; it is difficult to believe that a healthy valve would rupture under these circumstances, and we must admit the existence of a previous valvular disease, which has weakened its structure.

Endocarditis, sclerotic in structure, is perhaps more often met with at this orifice than in all the others; occasionally the inflammation causes adhesion of one or more leaflets, or a portion of them, to the wall of the vessel. Cases have been noted in which insufficiency was caused by a perforation due to a local valvular aneurism. A large number of cases of aortic disease are secondary to mitral endocarditis, which has extended by continuity and contiguity of structure. In older children the valves may become diseased by an extension of an atheromatous degeneration of the aorta; in other instances insufficiency may be produced by dilatation of the aorta independently entirely of valvular disease. Paul has demonstrated the fact that in youth the semilunar valves have a greater area than is necessary to cover the section of the aorta, but as the aortic orifice enlarges after manhood, while the valves no longer increase in size, the latter finally become smaller than the area of the section of the aorta. Beneke* has made a number of measurements to demonstrate this progressive dilatation of the aorta with age, showing that the aorta gradually increases in size from birth until the age of twenty-one years. The dimensions of the body and of the aorta then remain stationary until the age of forty to forty-five years, after which the relation changes as the aorta increases in size.

The changes thus shown in the heart of a child will throw light upon the fact that a growing heart possesses a certain inherent power to overcome morbid changes which is not seen in the adult, thus rendering the prognosis of cardiac disease in the young much more favorable.

The observations are so interesting and valuable that we append an abstract in tabular form.

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* F. W. Beneke, Ueber das Volumen des Herzens. Cassel, 1879. (Paul.)

Dimensions of the Circumference of the Aorta at its Origin, according to Age and Sex. Relations of this Quantity to the Length of the Heart (the figure marked indicates the relation of this quantity to one hundred centimetres in length of body).

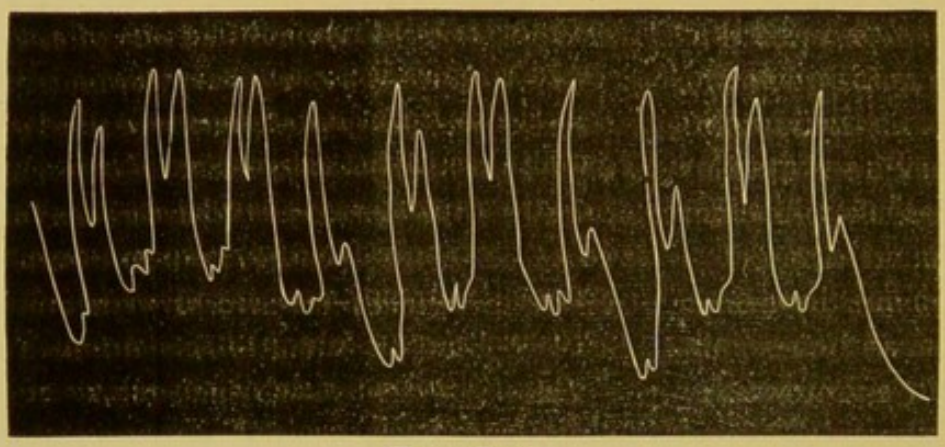
Age.	Male Sex.				Female Sex.			
	Number of Cases observed.	Internal Circumference of Aorta in Millimetres.	Relation to 100 Centimetres of Length.	Average Length of the Body in Centimetres.	Number of Cases observed.	Internal Circumference of Aorta in Millimetres.	Relation to 100 Centimetres of Length.	Average Length of the Body in Centimetres.
Fœtus	1	9.5	30.6	31.0	...	.....	.....	.....
Fœtus	1	11.0	29.7	37.0	...	.....	.....	.....
Fœtus, six to seven months	1	12.5	30.9	40.5	...	.....	.....	.....
Fœtus, seven months	1	14.0	33.7	41.5	...	.....	.....	.....
Fœtus, seven to eight months	1	13.5	33.7	40.0	...	.....	.....	.....
Fœtus, seven months	...	.....	.....	.....	1	11.0	30.5	36.0
Still-born	6	18.7	37.5	50.0	1	17.5	35.0	50.0
From one to eleven days	4	20.0	39.8	50.4	4	19.0	38.1	50.2
From eleven days to three months	17	23.7	44.2	53.7	14	23.5	42.4	55.4
From three months to one year	10	36.0	48.6	74.0	14	28.3	45.4	62.3
From one to two years	11	33.9	46.9	72.7	9	33.2	44.1	75.4
From two to three years	12	40.0	48.8	82.0	9	34.6	41.5	83.5
From three to four years	4	39.3	42.5	93.1	2	39.3	42.5	93.1
From four to five years	1	38.0	38.0	100.0	4	40.1	41.3	97.0
From five to six years	5	40.3	38.9	103.7	3	40.0	36.4	119.6
From six to seven years	6	43.0	37.0	116.1	2	39.5	40.0	104.5
From seven to nine years	6	46.6	38.3	121.4	3	43.0	35.9	119.5
From ten to eleven years	8	47.3	38.8	122.4	2	44.0	35.1	125.5
From eleven to thirteen years	5	50.8	36.9	137.4	4	48.5	34.8	139.5
From thirteen to fourteen years	4	46.2	32.3	143.5	5	49.6	36.3	136.9
From fourteen to fifteen years	7	49.0	33.9	144.3	3	49.6	33.7	147.3
From fifteen to sixteen years	9	51.9	33.2	157.0	3	56.3	36.9	153.6
From sixteen to seventeen years	3	55.1	35.3	156.6	5	49.6	32.5	152.5
From seventeen to eighteen years	7	53.5	33.1	161.3	5	55.4	34.9	159.0
From eighteen to nineteen years	5	57.8	34.6	166.8	3	53.8	32.3	164.6
From nineteen to twenty years	11	57.8	34.3	168.6	4	53.5	34.1	156.7
From twenty to twenty-one years	3	62.8	36.0	172.0	5	56.2	36.7	156.7

Symptoms and physical signs.—Of all cardiac diseases this is certainly the most easily recognized, both on account of its characteristic general symptoms and the acuteness with which the physical signs are defined.

Corrigan first recognized this fact by describing the "visible pulse," so marked in this disease. The pulse is sudden and bounding, giving an impression of seeming strength, but it is elevated suddenly, and falls immediately. In the words of Corrigan, "the arterial trunks of the head, neck, and upper limbs at once attract the eye by their peculiar pulsations; at each diastole the subclavian, carotid, temporal, humeral, and sometimes even the palmar arteries are projected forcibly from

their bed and bound under the skin." In order to better observe these points we are in the habit of having the patient strip one arm and preferably one side of the chest, separating the arm from the body, semiflexing and supinating the forearm; the observer at a glance will be able to note the entire course of the arteries under consideration.

The character of this pulsation is well shown by the cardiograph tracing upon a case, æt. seventeen, with aortic insufficiency. Corrigan's original explanation, which has stood the



(From Paul.)

test of years, cannot be improved upon: "When the semilunar valves are healthy they are closed by the pressure of blood immediately after each ventricular contraction. When the occlusion is complete the blood propelled from the ventricle is retained in the aorta, and the large vessels remain distended. These vessels then maintain almost the same calibre in systole as in diastole. But when the valves no longer close the aortic orifice, a certain amount of blood flows back into the ventricle after each systole; it follows that the ascending aorta and the arteries supplied by it allow a certain amount of the blood contained in them to escape; they become flaccid after each ventricular contraction and their diameter diminishes. At this moment a fresh contraction of the ventricle rapidly forces into the vessels a quantity of blood which dilates them forcibly and suddenly. The arterial diastole is then marked by such a sudden increase in the calibre of the vessel that it produces a visible pulsation, constituting one of the signs of the disease." It is not necessary that the

backward flow should be directly into the left ventricle, as in certain congenital cases, in which the flow has been from the aorta to the pulmonary artery by an abnormal communication, or from the aorta to the right ventricle, all the phenomena of aortic valvular insufficiency have been noted. It is necessary and important to bear this in mind when observing young children.

The murmur is heard at the second right costal cartilage, "the aortic cartilage;" it is heard as high as the upper border of the second intercostal space, also slightly to the right of the sternum, and descending downwards throughout the extent of the bone, inclining a little to the left as the ensiform cartilage is approached. The murmur is sometimes heard with maximum intensity opposite the third right costal cartilage. Again this intensity is occasionally best marked at the extreme tip of the sternum, the ensiform cartilage, or xiphoid appendix. The bone, or in the very young the cartilaginous structure, in some cases seems to act as a tuning-fork and amplify the bruit by its vibrations. Aortic insufficiency is accompanied by a murmur which is diastolic in time, replacing the click of the semilunar valves and commencing with the cardiac diastole, consequently occupying the greater portion of the period of silence; it terminates with the diastole, or better the murmur is cut short by the next systole. The systole will be found to be shorter than normal, with a rapid subsidence. As the heart becomes hypertrophied the murmur becomes more distinct and presents greater areas of transmission.

Its character is somewhat superficial, sometimes soft and cooing, at others hard and rasping; it is heard best, as a rule, with the patient standing.

Renaut says that in cases of great insufficiency with an hypertrophied auricle, a sphygmographic tracing of the carotid pulse will show an elevation of the line corresponding to the auricular systole.

The general symptoms in young patients are apt to be latent for a long time, and the sole manifestations are the physical signs. Later, hypertrophy of the left ventricle arises, and the apex becomes lower than normal, producing an epigastric impulse, and displacing the left lobe of the liver downwards.

Potain and Rendu are of the opinion that this pulsation is due to the backward flow of blood, and that it occurs during the diastole; they state that it always precedes the apex-beat.

As the case advances the cardiac chambers become dilated; it is then that we note an increase in the vertical diameter of the heart; the tricuspid may become insufficient by a process of simple dilatation of the orifice. The apex impingement may be less noticeable to the eye, although the hand will appreciate the active and laborious cardiac movements; as the heart hypertrophies it extends downwards and more to the left, becoming more parallel to the anterior chest-wall and burying itself beneath more dense lung-tissue, which prevents the apex-beat from being so well marked.

The patient, if perfectly quiet, will be in comparative comfort, but the slightest exertion will produce palpitation and distress, with a feeling of anxiety and oppression. Dyspnœa becomes an exacting symptom, which muscular exercise or mental worry will increase to apnœa; it is the duty of the physician in these cases to warn the parents or guardians of the necessity of correcting the child in a mild manner, and to especially caution them against the danger of violent whipping or secluding the child in a lonely or dark room; parents should see that these children are not unduly excited by their nurses reciting "ghost stories" or tales of reckless daring, culled from the unfortunately prevalent poor literature of the day. Among the most alarming cases that we have been called upon to treat have been examples of night-horror in young children with valvular disease, whose nurse or elder brother or sister has conned to sleep by some story gleaned from the cheap weekly papers scattered so broadcast throughout our country.

Occasional attacks of angina pectoris may arise, alarming the patient greatly, and introducing a new and serious element in the prognosis.

The natural course of the disease in the young is slow; when dilated hypertrophy arises we then note the symptoms of venous tension, congestion of the portal and pulmonary veins, with œdema of the extremities.

AORTIC STENOSIS.

Unlike the tricuspid valve, the aortic is peculiarly prone to present a double lesion. The disease which crumples and stiffens the leaflets of the semilunar valve almost always renders it also unable to close the opening, so that we clinically often note the association of insufficiency with stenosis or narrowing of the aortic orifice. However, cases do occur in which the systolic stenotic murmur persists alone throughout the case.

Symptoms and physical signs.—From the fact that the lesion is rarely single, but most usually accompanied by a certain degree of insufficiency, thus producing a slight confusion in the diagnosis, we must be on our guard in summing up the case.

When the valve is injured by sudden muscular effort, which, as we before stated, does sometimes occur in the semilunar valves, it is rarely that a stenosis or obstruction is produced, the result usually being an incompetency permitting regurgitation.

Peacock, in 1865, had collected seventeen such cases, four of which were under his observation.

All these cases had previously enjoyed good health, never having had rheumatism or, as far as known, previous cardiac disease. Out of these seventeen cases the aortic valves were injured ten times, mitral four times, tricuspid three times.

W. L. Axford more recently reports a case of a previously healthy boy who, upon sudden exertion, ruptured the aortic valve, the history being as follows:

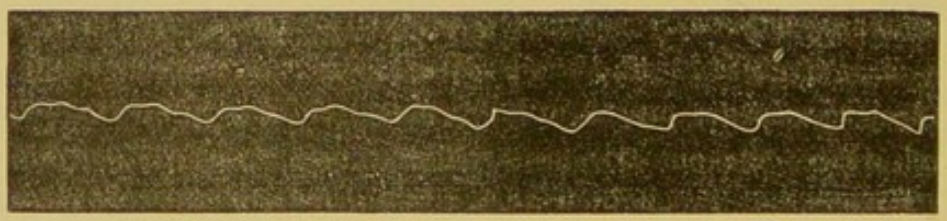
J. G., aged sixteen, well developed, while carrying a heavy weight made a sudden and severe muscular effort; soon complained of considerable pain in the centre of the sternum; also of an uncomfortable feeling in the left arm. Expectorated some blood and had a short, dry cough. Before seeking medical advice had been treated for dyspepsia.

His condition soon after the rupture was: Pulse, 100; temperature, 97½°; respiration, 26 and irregular; skin tinged a peculiar brown; complained of a dull, rumbling sound in his ears; urine loaded with urates. Auscultation revealed an aortic regurgitant murmur, partially obscuring and following the second sound of the heart. After treatment for some time

the subjective symptoms disappeared, but the murmur was still to be heard. At the present time the boy is up and about, but unable to do manual labor.

The *murmur* produced by stenosis at the aortic orifice is systolic in time, and carried upward to the point at which the aorta becomes most superficial, consequently its maximum intensity is at or a little above the second right costal cartilage, as marked in the diagram. The murmur occurs synchronously with the apex-beat and the carotid pulse. The bruit of an aortic stenosis is perhaps the most widely distributed of all the valvular murmurs; it is carried outwards by the blood-current to the carotids, subclavian, and axillary. In some cases we have heard it in the femoral, and even in the popliteal. Some authors state that they have recognized the murmur even as distant as the dorsalis pedis arteries. They also have a very wide distribution within the chest encasement, as, for example, we have noted them over the sternum, at the apex, over the left lobe of the liver. and in puny, thin children at almost any part of the chest.

The *pulse* in aortic obstruction depends greatly upon the degree of stenosis: small if this is extreme; usually, however, it is full and strong, giving evidence of ventricular hypertrophy, which arises early. Its characters are well illustrated in the accompanying tracing:



Child six years. Aortic stenosis, with autopsy. (Blache.)

It is necessary to differentiate an aortic systolic murmur from a mitral systolic. In order to distinguish them we first locate the point of maximum intensity. Furthermore, the systolic murmurs are rarely of the same timbre and quality. The aortic most usually is the rougher and more accentuated. Again, mitral systolic murmurs are not transmitted into the arterial circulation, but into the axilla.

The effect on the cardiac cavities is much the same as already described for aortic regurgitation, as are the general constitutional symptoms, so that a repetition would be superfluous, particularly as sooner or later the two murmurs will almost always be found to coexist.

DISEASE OF PULMONARY ARTERY VALVES.

Beyond question post-natal disease of the pulmonary valves or orifice is a clinical rarity. We have already considered, at some length, the congenital diseases and defects at this situation; when meeting a murmur whose maximum intensity corresponds to the upper margin of the second left intercostal space, we must first determine whether this is the true primary seat of the disease, remembering how unusual it is to meet a bruit at this point; and furthermore, we must, if possible, determine whether we are dealing with a congenital or an acquired affection, bearing in mind that many competent authorities consider stenosis and atresia of the pulmonary artery and orifice with open septum ventriculorum as the most frequent malformation of the heart; this narrowing is generally best marked at the orifice. It is possible in these cases to maintain life even up to the age of thirty-seven years; we have already reported the case of a man who survived until the age of sixty-six years, so that the physical diagnostician may upon rare occasions meet with either a pulmonary regurgitation or stenotic murmur.

Paul, in 1871, reported an undoubted case of acquired or extra-uterine pulmonary stenosis, and in 1882 Vimont records fifty-two cases of acquired disease of the pulmonary artery, among which are included cases of stenosis and insufficiency. In our own hospital wards we have met one case of ulcerative endocarditis which the post-mortem examination showed was confined entirely to the pulmonary artery valves.

Stenosis of the pulmonary artery valves produces a striking alteration in the cardiac topography. Hypertrophy of the right ventricle arises, which may attain the thickness and capacity of the left ventricle, giving the heart a sac-like shape with two equal-sized cavities; in some instances the right ventricle is much the larger of the two. The pulmonary artery itself will

be found to be in a state of dilatation with thinning of the walls; the later stages of the process will, of course, be one of cardiac dilatation, as we have already seen when studying the other murmurs.

According to Paul, the diagnosis of *stenosis* of the pulmonary artery depends upon the following signs: "Measurement of the heart shows depression of the hepatic angle of the cardiac triangle, and consequently a more horizontal position of the lower border. In certain cases palpation may reveal a purring thrill, which is more marked in the second left intercostal space. Auscultation discloses a peculiar blowing murmur. This murmur occupies particularly the left second intercostal space from the sternum to four or five centimetres from the edge of the bone. If the murmur is intense it is transmitted readily by the thickened wall of the ventricle, and then covers the entire anterior surface of the heart; but the maximum intensity remains very distinctly in the second left intercostal space." The murmur is systolic in time just as its analogue at the aortic orifice; it is, perhaps, a little more superficial.

Paul further states that the murmur has its maximum intensity when the patient is ausculted in the recumbent position; it diminishes considerably and sometimes disappears entirely in the sitting posture.

A fact which should be of considerable aid in diagnosing this class of murmurs is the relation which they bear to the respiratory function. As the patient holds his breath the murmur becomes weaker and weaker until during a forced expiration it almost entirely disappears, then allowing the patient to inspire several times and free the pulmonary circulation, the murmur will again return; these characters are said to be extremely well marked in pulmonary stenosis. When the ventricle becomes much hypertrophied the respiratory auscultation is not so characteristic, as the force of gravity will then not overcome the force of the heart; it is now that arrested respiration will cause the murmur to become more plain.

Potain and Rendu state that their patients complained of a sensation of peripheral coldness despite the integrity of the arterial circulation. Paul has failed to note this symptom in

his cases who suffered from the acquired variety of the disease, but found it well marked in those cases who presented cyanosis, depending upon congenital defects in the valve and orifice.

Insufficiency of the valves of the pulmonary orifice extra-uterine in origin is beyond all question the rarest form of valvular disease. Its etiology is similar to all other valvular affections.

A consideration of the disorders of the pulmonary orifice and valves would indeed be incomplete without extended reference to the case reported by Bruen* of a young girl who at the age of twenty years presented well-marked signs of cardiac derangements. The patient was a syphilitic, and was under observation from November, 1878, until July, 1882.

Post-mortem.—Heart, left side: Slight ventricular hypertrophy; mitral valves somewhat thickened at the margins, with roughening of their auricular aspect; valves competent; the left auricle is normal, as are also the aorta and the aortic valves.

Right side: Two of the semilunar leaflets at the mouth of the pulmonary artery are nearly destroyed by atheromatous changes, while the third segment is much thickened and projects as a leaf-like fold, roughening the mouth of the pulmonary artery. This vessel is dilated to twice its normal size, forming nearly an aneurismal dilatation; the vessel-walls are covered with a fringe of vegetation, of inflammatory origin, or due to atheromatous changes. The right auricle is very small and imperfectly developed, the bulk of its cavity being formed by the auricular appendix.

The tricuspid valve was much thickened, but appeared to be competent.

An intra-ventricular communication large enough to admit the forefinger was noted directly beneath one of the two tricuspid leaflets; it was lined with endocardium, and must have allowed free interchange of blood between the two ventricles. Walls of right ventricle were thickened and its cavity dilated.

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* Dilatation and Atheroma of the Pulmonary Artery, etc., Dr. E. T. Bruen, Trans. Path. Soc. Phila., 1883, p. 78.

Dr. Bruen's conclusions are as follows: The case well illustrates the fact that an admixture of venous and arterial blood may occur without cyanosis occurring. Pulmonary artery disease is consistent with a fair amount of general health, and compensation by the right heart may occur, just as in cases of aortic disease. In descriptions of pulmonary artery disease attention is called to bronchitis, pneumonia, and hydrothorax as sequential states. In Bruen's case no such complications were present until just before death, when the patient finally succumbed to congestion of the lungs added to the cardiac state.

The aneurism of the pulmonary artery formed a pulsating tumor on the left side of the sternum, between the second and fourth ribs, extending outward from the border of the sternum and including an area covered by a silver dollar. Over the tumor a post-diastolic and a presystolic, bruit-like murmur could be heard at a point between the second and fourth ribs, while close to their junction with the sternum a hoarse systolic murmur could be heard. The bruit was localized; the heart systolic murmur was carried out into the entire arterial system.

Dr. E. O. Shakespeare, remarking upon the specimen, said that he had been struck with one point of great interest in connection with inflammation of the lining coats of the pulmonary artery as evinced by the vegetations. These growths are very rarely found in the venous current; arterial blood seemed a requisite for such diseased action. Evidently the site of the perforation being just below the aortic and pulmonary valves brought about just this necessary prerequisite,—viz., abundance of arterial blood within a vessel which normally carries venous blood.

CHAPTER VII.

GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OF VALVULAR DISEASE.

DIAGNOSIS.—In making up our diagnosis of valvular heart-disease it is necessary to proceed in a systematic manner, bearing certain well-established rules in mind. First, we should determine whether there is any actual organic disease present, or if it is not simply a functional disturbance; then we should

consider the alterations, if any, in the size or capacity of the cardiac chambers, together with the changes in its walls.

Most important is it to determine the etiology of the lesion. In order to accomplish this desirable object we should carefully inquire into the antecedent history of our patient, noting well the presence or absence of any family predisposition to heart-disease, also the previous history in regard to certain diseases which have a known cardiac tendency, as rheumatism, scarlatina, and morbilli.

The symptoms present, more particularly those indicating interference in the circulation, are to be carefully chronicled. All-important in this respect are the *physical signs*. Indeed, it is a good rule to examine the heart in all cases that consult us. In the physical examination we note,—

1. The position of the apex-beat and the character of the impulse.
2. The contour of the præcordia.
3. The presence or absence of a tactile fremitus or thrill.
4. The percussion outline of cardiac dulness.
5. Auscultation of the heart-sounds over all the different parts of the præcordia, together with the lines of known transmission.
6. The presence or absence of a murmur, together with the characters of such murmur.
7. The condition of the general circulation, the pulse, and the venous system.

We have already indicated the conditions which cause the præcordia to become either retracted or unduly prominent, and those that displace the apex-beat.* We must always bear in mind that in children organic disease, sometimes of a very serious nature, may exist with but few or any signs, and that even the physical signs in the early stages of the process may be ill-defined and difficult to interpret. On the other hand, patients may complain bitterly of cardiac disorder, and present some objective symptoms, but we will be unable to take a more serious view of the case than by considering it one of mere functional disorder.

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* See chapter on "Methods of Study" and Addenda, No. VI.

Prognosis.—With increasing knowledge in diagnosis our prognosis is becoming much more favorable. Indeed, it seems patent to us that the prognosis in the young is extremely favorable, and even in adults "heart-disease" is not the awe-inspiring death-knell in the public ear that a few short years ago placed it in the popular mind in the same category that "cancer" occupies to-day.

The first question asked by friends and relatives is, What is the danger of sudden death, and is a cure possible?

The probable duration of the case should, if possible, be determined. This, of course, depends greatly upon the seat and extent of the lesions and their concomitants.

Simple roughening of the endocardium is *per se* of little consequence, although it will produce a murmur, if it does not extend and involve the valves, which renders the case more serious, but not of equal gravity in all the valves. For example, sudden death is perhaps most likely to ensue in cases of *aortic regurgitation*, the least likely in *mitral regurgitation*, although it is said to have occurred in rare instances. Cases of aortic obstruction may last for a long time, their main effect being damming back of the blood-current, thus affecting the heart and lungs. Mitral stenosis becomes serious on account of the inability of the auricle to undergo simple hypertrophy, unaccompanied by early dilatation; the lungs are more speedily affected in cases of mitral disease than in any other valvular lesion.

Tricuspid regurgitation is at the same time one of the most distressing and most serious of the valvular disorders; the venous system speedily becomes overloaded and the symptoms exacting. Furthermore, the patient is apt to have multiple cardiac lesions, the tricuspid leak being one of the last of the series. The last hours in the life of a patient with this lesion are among the most trying scenes of a practitioner's experience.

All extensive or double lesions—*i.e.*, obstruction and regurgitation—of course increase the gravity of the prognosis.

A cardinal question in the prognosis, Is valvular disease ever curable? From our own experience we would certainly answer this in the affirmative, as we have met cases in the young in which distinct mitral murmurs due to organic dis-

ease have disappeared under our observation, although we do not think that the valve is ever restored to its normal condition. Still, it seems probable that the inflammatory deposits may be partly absorbed or removed after the lapse of several years.

Hypertrophy in the majority of cases is to be considered nature's method of protecting herself, and is not to be interfered with. It alone becomes dangerous when excessive, as it may cause the blood to tear through diseased arteries, or, on the other hand, if the right heart is hypertrophied, the lungs are supplied with an over-abundance of blood, and, per consequence, are constantly in a state of active congestion.

Dilatation of the chambers is always dangerous, and is never to be considered in any light but as one of evil import. Its gravity is in proportion to its degree and its relation to the hypertrophy; if in excess, the prognosis is serious indeed. It appears to us that the degree of dilatation is the keynote of the prognosis. Many cases of sudden death in valvular disease are due to a weak, flabby, dilated heart, which will be found to be in a condition of asystole.

Dilatation adds much to the patient's distress, throwing increased labor upon the circulatory apparatus, and predisposing to local or general dropsy.

Should fatty *degeneration* of the heart-wall arise, sudden death may occur at any time; pericardial adhesions tend to set this change on foot, and hence should be considered in making up our prognosis.

A summing up of all *symptoms* present will throw considerable light upon the case; for instance, great irregularity or intermittency in the heart's action, serious interference in the venous circulation, dropsies, apoplectiform or epileptiform seizures, render the outlook somewhat alarming, remembering, however, that patients often survive a long time after being "water-logged;" the intervention of pulmonary disorders will cause serious symptoms to arise, also increasing the existing dropsy or causing a general anasarca, all of which may subside with the subsidence of the lung-symptoms. In all cases we must carefully investigate the condition of the kidneys, lungs, and arteries, as upon their integrity to a great extent depends

the prognosis. To conclude, then, it is only in the young that curative changes can be expected, and then only under the most favorable conditions: they must lead a quiet, tranquil existence without worry or excitement, and be provided with a suitable diet, sufficient hours for rest, and the avoidance of all injurious habits.

Sir Andrew Clark, at the last meeting of the British Medical Association, remarked so pertinently upon prognosis of valvular disease:

"A lad, æt. sixteen, with an enlarged heart, loud systolic bruit in the mitral area. There was a direct and a regurgitant aortic murmur; impulse of heart was diffuse and heavy. The cervical veins were rather full, and the pulse was somewhat jerking and collapsing. The boy said he suffered nothing, but felt quite well. The family had been told that he was the subject of grave heart-disease, and the consultation had been sought for merely to ascertain by what means his life could be prolonged as much as possible. They were advised to follow out their intention of giving the lad a university education, which they did. This was fifteen years ago, and now the subject of the consultation is the incumbent of one of the largest parishes in England, and continues to pursue an active, useful, and comfortable life."

Sir Andrew Clark expresses himself to the effect that organic murmurs of the heart, although firmly established and lasting for some years, may eventually disappear, and cites several cases illustrating the fact. What are the conditions which justify a favorable prognosis in a given case of valvular heart-lesion? "According to the author, they are the following: (*a*) good general health; (*b*) proper habits of living; (*c*) no essential liability to rheumatic or catarrhal affections; (*d*) an origin of the valvular lesion independent of degeneration; (*e*) an existence of the valvular lesion for over three years without change; (*f*) sound ventricles of moderate frequency and general regularity of action; (*g*) sound arteries, with a normal amount of blood and tension in the smaller vessels; (*h*) a free course of the blood through the cervical veins; (*i*) freedom from pulmonary, hepatic, or renal congestion." To these must be added obedience to prop-

erly adjusted rules of health, which, however, need not interfere with the performance of the usual duties of life. The author sums up as follows: 1. "There are many persons with long-standing disease of the heart engaged in the active business of life, who, without any symptom of heart-disorder, have enjoyed good health and have reached an advanced age. 2. The mitral regurgitant murmurs so often encountered in chorea disappear for the most part within eight or nine years of the attack. 3. Valvular inflammations and their effects arising in the course of rheumatic fever do sometimes disappear and leave behind no clinical evidence of their former existence; this occurs, for the most part, in the young, but also sometimes in the middle-aged. 4. The signs of valvular defects arising out of degenerative changes of middle life do also, on rare occasions, disappear, and, when circulatory and respiratory disturbances accompany their beginning, they sometimes subside and admit of apparently complete readjustment. 5. As there must be, in the histories, habits, occupation, and surroundings of patients with valvular disease, conditions which in one case bring about secondary disorders, and in another exempt from them, these differences should be searched for and made capable of application in practice." *

Francis† relates the case of a boy æt. seventeen, attacked by rheumatic fever followed by adherent pericardium, who has lived by following the rules required to promote this condition to the age of sixty-three and is now well.

He epitomizes the rules for living as follows: 1. Rational living. 2. Drinking but little. 3. Avoiding alcohol altogether, as it causes muscular debility and promotes fatty degeneration.

Treatment.—Very seldom can the physician cure disease; at best he can simply guide the case to either a happy termination or through the various stages that inevitably end in dissolution. In chronic or subacute cardiac disorder we may undoubtedly prolong life, render the patient much more comfortable, and in many instances protect him from unpleasant or dangerous symptoms, which, should they arise, will be

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* N. Y. Med. Jour., March 12, 1887.

† British Med. Jour., April 2, 1887, p. 725.

greatly relieved by judicious treatment. Parents should appreciate the necessity of children with damaged valves being constantly under medical supervision, although it is not necessary for them to be constantly under the influence of drugs.

At the outset a plan of living should be laid down and rigidly adhered to; all laborious occupations must be given up, severe exercise, particularly such as involves a sudden output of energy, must be looked upon as the most hazardous stimulation that the circulation can be subjected to; indeed, we have seen the most decided and permanent benefit follow complete and absolute rest enforced for some time. Let us not, however, err upon the side of too little exercise, as many cases are much benefited by an open-air existence, with moderate walking and carriage-riding. This question of the amount of exercise must be determined for each particular case by the medical attendant, always bearing in mind that in proportion to the degree of dilatation or degeneration present is the capacity for exertion diminished. Loomis very aptly remarks that in aortic incompetence patients in sleeping should assume as nearly as possible a horizontal posture; by lying on their backs they lower the height of the distending column of blood, and thus relieve both the cardiac circulation and the tendency to pulmonary congestion. It is of paramount importance that the mental faculties should be as little disturbed or worried as possible; we would sound a note of warning in regard to excessive study, long hours in school, with but little or any ventilation, together with a stooping and cramped position at the desk.

These patients must be warmly clad, but care is to be taken that there is no constriction about the neck or chest of the growing child; impress upon the mother the necessity of postponing the adoption of the corset for her daughter just as long as possible, also the danger of late hours and the abuse of tea or coffee. Boys must not be permitted to use tobacco in any form, and as they grow older the danger of venereal excesses should be presented to them.

Occasionally we will meet cases which simply cannot exist in the particular climate in which their lot has been cast; then will the change to a fairly warm and bracing climate

often prove of extreme and permanent benefit. Such a climate, for instance, is offered by Southern California all the year round, or extreme South Central Florida during ordinary winters, the Bahamas, Cuba, or among those not so far south, Thomasville, Georgia, and Asheville, North Carolina.

Sponging the skin with cold or tepid water will often result in much benefit and comfort. This may either be applied at home or, if the patient is of the wealthier class, a winter visit to some of the many springs in either our own Southern or Western United States or the more elaborate and elegant establishments of Europe, particularly the baths of Southern France, to which we have elsewhere called attention.

Virginia seems particularly rich in mineral springs: more than fifty are places of resort and fashion, and over twenty are used commercially. West Virginia presents two well-known and much-used spring resorts: the Berkeley Springs is one of the oldest resorts in the United States, having been laid out in 1777, and the White Sulphur Springs have been used medicinally since 1778. North Carolina presents quite a number of hot springs, one of which reaches a temperature of 117° F.,—that is, the Warm Springs of Madison County,—which are used as a resort. Georgia as yet has not developed to any great extent her natural springs. Florida is notable for the great size of her springs, but little literature is available upon the subject, and few analyses have been made; most of them are thermal springs.

Utah, Colorado, New Mexico, California, Oregon, and Alaska present many beautiful and health-giving springs, which space forbids us to mention more fully.

The *diet*, the *digestive* organs, and the large abdominal glands must ever and always receive a considerable share of our attention. Gastric or intestinal dyspepsia will render the patient extremely uncomfortable by the attendant flatulence, which offers a mechanical interference to the heart's action. In a general way a very nutritious, easily digestible diet, containing abundance of protein, will throw the least strain upon the heart, and most easily nourish the patient. Milk and cream should be a constant element in this dietary. Alcohol is contraindicated as an article of diet, and is only to be used as

a medicine under the advice of the physician, and then only when definite symptoms call for its exhibition. Sugar, sweet vegetables, and animal fat must be used in moderation. Constitutional diatheses, if they exist, are to be corrected in so far as our means will allow, particularly the rheumatic and syphilitic.

In all cases of cardiac disease the blood should be examined by the hæmacytometer, a study made of its hæmic unit, together with the condition of the corpuscles, as frequently the most marked benefit will arise from the administration of agents to correct the alteration of the blood-crasis.

Drugs, or medicinal agents.—Digitalis, of course, is the first in the category. It is unnecessary for us to dwell upon the effects of the drug, as they are a matter of every-day experience to all who in any way have to do with the sick.

Digitalis is almost a specific in those cases presenting rapid, irregular, or embarrassed cardiac action, with a weak, compressible pulse. Under its action the heart-beat becomes regular, calm, and efficient; the pulse becomes at the same time slower, stronger, fuller, and more regular. Intermittency has by some been considered a contraindication for the administration of digitalis; but our own experience has agreed with that of Fothergill, who is of the opinion that this very condition often demands an increase in the dose. If, of course, the drug appears to produce this condition, its discontinuance at once is imperative.

Under the use of the drug the urine will increase in quantity. This increase, according to Ringer, is more marked if dropsy is present. Diminution in quantity passed is an indication for discontinuing the medicine. Digitalis does not invariably reduce cardiac dropsy; but usually, however, its effects are most happy.

Digitalis is stated by many to have a cumulative action, thus causing toxic symptoms to arise suddenly. We must confess, however, after a somewhat extended experience with the drug, that we have never experienced this action. Osler has arrived at a similar opinion from deductions of a large series of cases in his practice. Böhm,* however, reports a

* Bartholow, Med. News, April 23, 1887.

fatal case of digitalis-poisoning in a girl who died suddenly on attempting to get out of bed. Let us review for a moment cases in which the drug is either indicated or, on the contrary, is contraindicated. Simple hypertrophy of the left ventricle is a distinct contraindication, except when the hypertrophy is insufficient to compensate the dilatation. Great care is to be then used in its administration. Should the observer incorrectly interpret the amount of dilatation and overestimate it, giving the drug in proportion to his idea of this dilatation, disastrous results may speedily follow.

It is in cases of mitral regurgitation that the drug plays its most important rôle, the heart's action being rendered more regular. The musculi papillares consequently act with more determination and regularity, and in this way is corrected an active element in mitral insufficiency, as undoubtedly irregular action of the papillary muscles does much to assist in the already imperfect action of the valve. The general and pulmonary symptoms attendant upon the lesion will also be greatly relieved.

In mitral stenosis we would exhibit the drug only with extreme care, and in cases which can be under our almost daily supervision.

Again, in aortic disease digitalis must be used intelligently, recognizing that the state of the ventricle must be accurately determined before its administration. In tricuspid disease and affections of the right heart digitalis accomplishes but little except correcting the irregularity in the systoles.

Fatty degeneration of the heart-muscle requires extreme caution in the use of digitalis, as does also atheroma, if at all extensive; in bronchitic attacks of cardiac origin digitalis will often prove almost a specific, particularly if associated with strychnia.

It is not our province to dwell upon the various preparations of the drug; suffice it to say that in many cases in children's practice we obtain the best results from the fresh infusion, combined with either tr. cardam. comp., or an alcoholic extract of the fresh bruised seeds of cardamom.

Within the last few years many new drugs and preparations have been added to our armamentarium, some of which have

maintained their place in our lists, others being doomed to a short career, failing in the practical trial of bedside utility.

Strophanthus has by many competent observers been placed as an equal to or even a peer of digitalis. Fraser,* of Edinburgh, was the first to introduce it and recommend it to the profession. Upon our own side of the world Dana and Smith, of New York, report favorable results from the use of strophanthus hispidus.

Dr. Emil Pins,† of Vienna, in the June and July, 1887, numbers of the *Therapeutische Monatshefte*, also reports clinical observations in its favor.

Fraser and Brunton show by their studies that this drug is nearly identical with digitalis in its action on the heart and blood-vessels, and in its influence on the secretion of urine.

Pins has found that the tincture of the seeds acts admirably in those cases of cardiac failure depending more particularly on valvular disease, and produced the most desirable results when the lesions caused dropsy and consequent need of increased renal action. Under the use of strophanthus the pulse, heretofore weak, rapid, irregular, and flurried, became slower and more powerful, while the anasarca steadily decreased. In cardiac dyspnœa, both direct and indirect in its causation, it relieved the patient, and in every way proved itself to be the equal of digitalis.

J. Higham Hill‡ has found the tincture of strophanthus of great value in a case of mitral disease; under its use the pulse became slower, fuller, and much stronger; the renal secretion increased in three days from thirty ounces to eighty ounces; it was used in five-minim doses in half an ounce of water three times a day. Francis§ also speaks highly of its value.

Strophanthus is a powerful muscle-poison, increasing the contractile power of all striped muscles, and rendering their contractility more complete and prolonged; as a result of this action on the muscles the heart is early and markedly affected; the heart receives at once a larger quantity than any other

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* Brit. Med. Journ., Nov. 14, 1885.

† Med. News, Aug. 13, 1887.

‡ Brit. Med. Jour., April 2, 1887. § Ibid.

muscle in the body; consequently its action on the heart is first made evident, so that by regulating the dose a powerful effect may be produced upon the heart, leaving the other muscles unaffected.

Fraser's experiments show that strophanthus exerts a more powerful action on the heart and a less powerful action on the blood-vessels than digitalis. In this it possesses a decided advantage over the latter drug.

Bartholow* suggests trinitrin (nitroglycerin) and arsenic with a strictly regulated diet for those cases of heart-disease in which the muscle has been altered, fatty heart or weak heart, the cavities commencing to dilate and the patient experiencing attacks of angina or pseudo-angina.

Trinitrin may be given in drop doses of a one-per-cent. solution, increased by one drop until the characteristic effects are produced. Some cases are readily affected, others requiring a commencing dose of from five to ten drops. The drug may be given in tablets, which is a very acceptable form.

Bartholow considers nitroglycerin to possess the following advantages:

1. It lowers the vascular tension by dilating the arterioles. 2. It increases the rate of the heart's movements. 3. It lessens the irritability of the nervous system, which finds expression in spasms, especially of the nervous system of organic life; the arterioles of the cardiac muscle under these circumstances receive, of course, more blood, and per consequence nutrition is increased.

He is in the habit of combining with trinitrin the arseniate of soda or Fowler's solution, and is firmly of the opinion that incompetence of the valves caused by yielding of the weakened walls of the heart may be recovered from entirely under the influence of these remedial measures combined with a proper diet and exercise.†

Da Costa‡ speaks favorably of adonidine in one-tenth grain doses, considering that its powers as a heart-tonic are not inferior to digitalis, while it is free from the disturbing effects of the latter; he concludes as follows:

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* Med. News, April 23, 1887.

† Note.—See Addenda, No. VII.

‡ Med. Times, May 28, 1887.

"There are so many claims made in favor of new drugs, and especially heart-tonics, that it makes one suspicious of them all; but having used adonidine for some time, I consider it a valuable addition to our therapeutics of heart-affections, and have not observed from it anything resembling the cumulative effect which at times, though rarely, follows the administration of digitalis. In this case the contractions of the heart, while they became more regular, were reduced to fifty-six per minute. He had slight vertigo and headache, which disappeared upon resuming the infusion of digitalis, while the adonidine was continued. This drug will never supersede digitalis in the treatment of cardiac dropsy, from its want of diuretic action, but in other cases this might prove a decided advantage. Without discussing the effects of the agent upon the cardiac dilatation and hypertrophy, and merely referring you to the relief from the dyspnœa and the increase in the strength of the pulse, I point out to you that all this was associated with a very considerable amount of cardiac dilatation or stretching, with very little hypertrophy. There is another patient in the ward with very decided hypertrophy in whom the adonidine produced disagreeable results: the heart's action became intensified, and attacks of palpitation came on upon slight exertion. I recall another case in private practice where the same result occurred: the heart was strengthened too much. This all shows that it is a decided heart-tonic, and also indicates its line of usefulness. In a given case of weak and disordered circulation, the nearer it approaches a condition of dilated heart the more benefit from the adonidine; the more it approximates hypertrophy and over-action of the heart, the more is this agent contraindicated. In cases of weak digestion with weak heart, it is especially suitable."

The fruit of the kola-tree, the kola nut, contains about two per cent. of caffeine, but the action of kola seems to be different from pure caffeine.

"Its principal therapeutic application seems to be in cases of diminished renal secretion in cases of heart-disease, and Monnet refers to a number of instances in which the administration of this drug increased the general blood-flow and vascular tension, and so promoted diuresis. In some patients the

urinary secretion was increased from a pint a day to two and one-half and even three quarts. In such cases not only does the increased diuresis tend to remove dropsy which may be a sequence of the heart-disease, but the nutritive and tonic principles contained in it also serve to render it a general tissue-stimulant. Kola also seems to lessen the irritability of the nervous system."

Pribam and Piering* have studied the value of agaricine in reducing perspiration in several cases of heart-disease; the administration of the drug in one-twelfth grain, in pill form, almost always proved efficacious.

Chloride of barium, in one-sixth-grain doses night and morning, raises arterial tension and thus acts as a diuretic.

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* Zeitsch. für Therap., March 15, 1885.

SPARTEINE.—Lauggaard reports the following formulæ, which he found useful in eighteen cases of heart-disease:

℞ Spartëine sulph., gr. vi;
Pulv. rad. liquirit.,
Succ. liquiritæ, āā q. s. M.
Ft. pil. No. 20.
S.—One or two pills, two to four times daily.

℞ Spartëine sulph., gr. iii to vii;
Aq. destil., fʒiiss. M.
S.—Twenty drops, from two to four times daily, in sweetened water or wine.

℞ Spartëine sulph., gr. iii to vii;
Syr. aurant. cort., fʒi, fʒivss. M.
S.—ʒi in water, two to four times a day.

—*Therapeutische Monatshefte*, June, 1887.

Citrate of caffeine: Jaccoud,—

℞ Caffeine, gr. c;
Sod. benzoat., ʒii;
Aq. destil., fʒviii.

He prescribes from three to six grains in twenty-four hours.

Tauret gives the following formulæ for hypodermic use:

℞ Caffeine, gr. xxxv;
Sodii benzoat., gr. xv;
Aq. destil., fʒii¾.

Also,

Caffeine, ʒi;
Sod. salicylat., gr. xlv;
Aq. destil., ʒii¾.

—*L'Année Médicale*, 1887. Prior: "Clinical Importance of Sulphate of Spartein," Edin. M. J., Dec., 1887, p. 567.

Oliver * has administered barium in cases of aortic regurgitation, and noted a reduction in the pulse-rate, which was also rendered fuller and firmer, at the same time the amount of urine was increased.

Aconite and belladonna have powerful and beneficial action in calming the heart when its action is tumultuous and excited; veratria, caffeine, and strychnia are all valuable to fulfil special indications. Convallaria maidis for a time attracted great attention; its tincture in large doses appears to be a true heart-tonic; in no way, however, does it compare with digitalis. Bromide of potassium in certain cases is very beneficial,—in small doses it slows the circulation and allows the heart to establish its equilibrium; it is particularly applicable in cases of fatty heart.

The question now arises as to whether we have any means at our command to cure the cardiac disorder. It is a waste of time to attempt to influence valvular disease by any therapeutic measures; we must simply encourage hypertrophy up to a certain point and then direct our attention to the dilatation, if possible not allowing it to overcome the former condition.

Palpitations, cardiac distress, and dragging, angina or pseudo-angina, may often be relieved by the application of a belladonna plaster to the entire præcordia; a freshly-made belladonna ointment is sometimes more serviceable.

Dyspnœa and asystole may be combated by capsules of ether or by its inhalation; nitrite of amyl is often of extreme benefit in advanced cases.

Alcohol in some of its many forms will prove a valuable stimulant to the circulation and respiration.

Cases of dyspnœa combined with definite pulmonary symptoms are much relieved by a combination of digitalis and tincture of nux vomica. Position will do much to relieve this distressing symptom; proper treatment and a healthy condition of the gastric intestinal tract will also do much to add to the patient's comfort and well-being.

Hæmoptysis must not always be looked upon as a serious symptom, as in many cases it proves to be of extreme benefit;

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* Lancet, July 30, 1887.

if the loss of blood is not sufficient to do the patient an injury, it is well to allow nature to take its course unaided by any therapeutic agents. In cases of over-distention of the right chambers, it is often advisable to practise venesection, cupping, or the application of leeches before resorting to these measures. We must remember that anæmia may be produced by our meddlesome practice, consequently we should well consider all aspects of the case before removing any blood; dry cupping, hot stupes, or fomentations containing turpentine or sinapisms will ofttimes tide a case over a dangerous period.

Sooner or later anasarca or dropsy will arise in most all cases of heart-disease, and is to be combated first by digitalis, which acting upon the heart will increase arterial tension in the kidneys and thus spur them on; diuretics and purgatives are also to be resorted to. In cases that can stand them, Turkish baths will be of inestimable benefit; Roberts advises local baths to the extremities, wrapping the legs up in warm fomentations along their whole extent and covering them with mackintosh. The skin may be used as an eliminate by the administration of jaborandi or pilocarpine; Hay's method of administering concentrated draughts of sulphate of magnesia upon an empty stomach is often followed by marked improvement; some cases, however, will not tolerate the remedy, it should then be at once abandoned, as disastrous results may result in its perseverance. The removal of local effusions, as hydrothorax and abdominal dropsy, has been considered earlier in the work.

Insomnia in advanced cases becomes a wearying and exhausting symptom, and one that will tax the attendant's skill to overcome; opiates, chloral, and bromides are dangerous on account of the condition of the heart; furthermore, they induce a condition during which the voluntary efforts necessary to carry on respiration may cease; still in most cases we must at some time have recourse to these drugs, always, however, recognizing their dangerous character.

Two new and reliable drugs have, however, been added to our list for the treatment of the insomnia of heart-disease, paraldehyde and urethran,—the former in doses of four to eight minims, the latter in doses of three grains and upwards.

The former is somewhat unpleasant to the taste; the latter is probably the best, as it sometimes produces a calm, restful sleep when opium has failed, and when chloral is contraindicated on account of the condition of the heart.

Frequent examinations should be made of lungs, kidneys, and liver, as these organs invariably present sequential lesions.

In conclusion, we repeat Finny's* deductions from a number of carefully studied cases of heart-disease: 1. That too much dependence is not to be placed on the presence of the physical signs of mitral regurgitation as evidence of organic disease. 2. That such signs may be due to purely functional derangement and weakness of the heart, or to an altered condition of the blood. 3. That blood-murmurs produced in the heart and large vessels may be louder than the murmurs due to valvular lesions. 4. That the danger of valvular diseases is enormously increased by, if not directly due to, weakness of the cardiac walls. 5. That increased action and force of the ventricular contraction in the presence of valvular disease is not to be considered a disease, but rather a symptom of disease, and is directly proportionate to the amount of regurgitation or obstruction. 6. That mitral regurgitation is not to be considered in the light of a "safety-valve function" in cases of aortic obstruction, but as an element of increased danger to life. 7. That lowering treatment of the heart's force is rarely, if ever, required in disease of the organ. 8. That indications for treatment in diseases of the heart should be sought from the evidence of the condition of the muscle of the heart, and not that of the valves.

NOTE.—It is to be understood that the doses suggested are for an individual of about the age of fifteen years. In graduating the dose for a younger child it is well to use the well-known formula of adding twelve to the age and dividing by the age of the patient; for example, let us suppose that the dose of say carbonate of ammonia for an adult is five grains, what will be the dose for a child æt. three? $12 + 3 = 15$,—$\frac{3}{15}$ of the ordinary dose, or, reducing the fraction to its lowest denomination, $\frac{1}{5}$, and as the adult dose was five grains, that for a child æt. three will be one-fifth as much, or one grain.

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* Dublin J. Med. Sci., Feb., 1883.

CHAPTER VIII.

ANEURISM.

Aneurism and atheroma of the aorta.—For many years the existence of this condition in infants and young children was totally denied; however, Roger,* in 1863, recorded a case of aneurism of the arch of the aorta in a girl of ten years of age, and Herveux remarks that out of the five hundred and fifty-six cases of aneurism reported by Crisp, five alone appeared in individuals of several days up to twenty years.

Again, of ninety-eight cases of aneurism, but one was found in a person under twenty years of age.

One case of abdominal aortic aneurism is recorded as occurring in a child aged fourteen years,† and Broca, in a treatise on aneurism, relates one case of aneurism of the aorta in an infant of one month.‡

A case is reported (Phaenomenow, *Arch. für Gynæ.*, 1881) of a fœtus who presented an aneurism of the abdominal aorta ten centimetres wide and eleven centimetres long. It was situated between the origin of the renal arteries and the bifurcation of the iliacs. Microscopic examination showed its walls to be composed of the layers of the arterial coats.

Sanné (ibid.) made an autopsy in a child, aged thirteen: the arch of the aorta was increased in size and very atheromatous; on the convex superior portion (transverse arch) was to be seen

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* Bull. de la Soc. Méd. des Hôp., Paris, 1863, p. 499.

† Bull. Thérap., 1835, p. 393, vol. ix.

‡ Sanné, Rev. de Méd. L'Enfance, February, 1887; also Edinb. Med. Jour., August, 1887, p. 188; also Med. and Surg. Reporter, October 22, 1887, p. 549.

a sacculated aneurism whose orifice of communication with the aorta was partly closed by cretaceous deposits; the aortic valves were insufficient, stenotic, and atheromatous. The pericardium contained several ounces of serum, and the left ventricle was hypertrophied. The same author has seen two cases, aged respectively two and three years, with marked general atheroma.

Moutard-Martin* contributes the clinical and post-mortem notes of a child, aged two, who presented chronic aortitis, with contraction and stenosis of the aortic valve, hypertrophy of the heart, chronic pericarditis, and loud murmur. The aorta was two or three centimetres higher in the chest than normal, and was very atheromatous. Norman Moore† exhibited to the London Pathological Society a specimen of aortic aneurism taken from the body of a child aged five years. *Sex* in early life does not bear such a direct relation to the production of aneurism as it does in adults, in whom the disease is more frequent in males than in females; this is to be accounted for in great part by the fact, as stated by Beneke, that the blood-pressure during childhood is about the same for both sexes, but from puberty onward it is greater in the male.‡

Sufficient cases have now been cited to show that aneurism and atheroma of the aorta may occur at any period from fœtal life upwards, nor does youth protect the *cerebral circulation* from atheromatous degeneration and aneurismal dilatation, as the case of aneurism of the middle cerebral artery in a boy, aged twelve, recorded by West, will show:

Boy had mitral insufficiency. At eight years of age had had scarlet fever and dropsy, afterwards some rheumatic joint-pains. For two years had dyspnœa. Four days after admission to hospital was suddenly seized with headache and vomiting; shortly afterwards became drowsy and had several fits, though none of them were severe. No paralysis was detected, but the drowsiness gradually deepened into coma, and the boy died about twenty-four hours after the commencement of head-symptoms.

Post-mortem.—All the loose tissue at the base of the brain

* Bull. Soc. Anat., 1875, p. 775.

† Trans. Path. Soc., 1882, quoted by Shattuck, Boston Med. and Surg. Journal, Sept. 22, 1887, p. 280.

‡NOTE.—See Addenda, Nos. VIII. and IX.

was distended with blood, which tracked in all directions upward towards the surface of the brain, and downward along the medulla and cord, and on both surfaces of the cerebellum. All the ventricles, the fourth included, were filled with recent blood-clot. The source of the hemorrhage was an aneurism of the left middle cerebral, of the size of a small pea, about an inch from its origin. The walls of the vessel were very atheromatous and brittle, although elsewhere they were perfectly healthy. The aneurism had contracted dense adhesions all around, and had ruptured into the lip of the descending cornu of the left lateral ventricle. The blood had filled both the lateral ventricles, and had then spread by the transverse fissure to the exterior of the brain, chiefly along the base, around the crura cerebri, over the cerebellum, and round the crura cerebelli to the fourth ventricle. There was no evidence of embolism in the diseased artery.

The pericardium was universally adherent, and the mitral valve much thickened and covered with numerous recent vegetations. The other organs were healthy.

Osler* records an aneurism of a branch of the anterior cerebral in a boy six years of age, being one of the youngest cases on record. The boy was brought to hospital unconscious, with feeble pulse, pale face, eyes and head turned to the right, and left hemiplegia; death in six hours. He had fallen from a hay-loft three weeks before, but he recovered rapidly from the effects. There was meningeal hemorrhage at base and in the longitudinal fissure. An aneurismal sac was found imbedded in the calloso-marginal fissure just where it turns vertically upwards. The rupture was on the meningeal surface, but hemorrhage had extended into contiguous portions of the brain; the arteries were not atheromatous, presumably altogether normal, and the heart was healthy.

The symptoms, physical signs, modes of termination, and treatment of aneurism in the child are precisely similar to those in an adult, and it would be superfluous to recapitulate here, as the subject is so fully covered in the various standard text-books.

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* Canada Med. and Surg. Journal, 1886. This case is referred to by Sachs, Journal of Mental and Nervous Diseases, Aug., 1887.

Care, however, must be exercised in the diagnosis of aneurism, as mistakes are by no means uncommon. Hare* has recently recorded an interesting case of spurious aneurism of the innominate artery in a young girl aged seventeen. In this case the most definite signs of true aneurism were present, yet the post-mortem proved the entire absence of any lesion of the blood-vessel. The physical signs were characteristic and supposed to be very diagnostic, as several expert physical diagnosticians saw the case, one of whom lectured upon it as a text of aneurism in the young. The vessel, when examined at the section, seemed to be a little relaxed, and somewhat more elastic than normal. The aortic valves were extensively diseased, all the others healthy; the girl was weak, anæmic, and hysterical. The thyroid gland, during the early developmental stages of exophthalmic goitre, presents some signs which simulate an aneurism. A further diagnosis must be made between the enlargement of a tubular peri-bronchitis and an aneurismal dilatation—a point fully elaborated by Roger (*ibid.*).

HYPERTROPHY AND DILATATION.

We are not at all apt to confound these conditions, but it is better to consider them together, as they so generally exist at one and the same time in a given case.

Between the ages of three and eight years the difference in the size of the heart is marked. Guersant states that the left heart is the larger, in the proportion of one to three. At this early period more work is required of the left side in order to send the blood actively to the periphery of the body; we also note the fact that the arteries are equal in size with the veins, whereas in an adult the veins are the larger.

Three forms of hypertrophy have been described, as simple hypertrophy, eccentric hypertrophy, and concentric hypertrophy; the former being a simple increase in the thickness of the muscular wall, the second an hypertrophy accompanied by dilatation, the former being in excess, however; the third is supposed to be a condition of hypertrophy in which the cavities are contracted.

Hypertrophy and dilatation is somewhat frequent in early

* Med. News, Oct. 1, 1887, p. 388.

life as a consequence of the various lesions of the valves or pericardium; in other words, the same disorders which bring about hypertrophy in an adult will also set the process on foot in a child.

Aortic and mitral regurgitation are peculiarly prone to give rise to a dilated hypertrophy, as the cavity or cavities are receiving two streams of blood under increased pressure. Dilatation is often established by the many pulmonary affections of early life. Rillet and Barthez say that under these circumstances we are much more apt to have dilatation than hypertrophy. Displacement of the heart from a pleuritic effusion and deformity of the thorax are potent causes of the condition in the young. Some authorities consider that permanent enlargement or a hypertrophy may supersede the condition of cardiac degeneration seen in systemic fevers, or the softening of a myocarditis, which was secondary to peri- or endocarditic inflammation. The latter is well illustrated by the case of a child eleven years old, reported by Guersant, who was attacked by acute rheumatism, accompanied by hyperpyrexia, soon developed extreme dyspnœa and marked palpitation; pulse irregular and intermittent; extremities cold and œdematous, bulging præcordia, excessive action of the heart; bruit at apex and in axilla.

The autopsy revealed a right pleural effusion with false membrane, an adherent pericardium, and an enormous cardiac hypertrophy. Bamberger also reports the case of a child aged eleven, with acute rheumatism and pericarditis, in which the hypertrophy was well marked, and arose with great rapidity. A definite cause of hypertrophy which all who treat the young must clearly recognize and guard against is repeated violent effort or exercise with the arms, as gymnastic exercises, rowing particularly. This change in the cardiac walls is brought about in two ways. An important factor is the excessive cardiac action suddenly produced; secondly, the constriction of the arteries and the obstruction to the circulation caused by the rigid muscles crossing the arteries and opposing the passage of blood.

The right cavities are very apt to be affected in swimming or running contests or in excessive diving. All these sports

are becoming a part of the regular college curriculum, fortunately, however, in most cases under the direction of skilled instructors, many of whom are at the same time physicians; nevertheless, it is well for us, in those families who are under our care, to from time to time inquire into the condition of the circulatory apparatus of the young student who is ambitious to place his college colors at the head of a race. In young boys hypertrophy may follow the smoker's heart; indeed, the irregularity produced by tobacco may lead to more serious disease, even to organic murmurs and cardiac failure. The simple hypertrophy which may be caused by the strain of excessive exercise tends to fatty degeneration of the cardiac fibres when the habits become more sedentary, and may lead to weakness, which evil habits, excessive tobacco indulgence, or venereal excesses will eventually develop into dilatation, irregularity, and finally organic disease. Dilatation is the more apt to arise early in those cases where there is great internal pressure on the cardiac walls during diastole, or when the obstruction arises suddenly. Again, dilatation is only too apt to follow an exhausting illness. Dilatation and hypertrophy seem in some cases to more or less depend one upon the other, therefore the rapidity with which they become established depends much upon the cause. Occasionally in compensatory hypertrophy dilatation is salutary, and does not allow the hypertrophy to become excessive; on the other hand, in some instances the dilatation rapidly overcomes the hypertrophy and the case speedily terminates. The nervous energy that animates the heart is a direct aid in producing a certain degree of compensatory hypertrophy, and when it is enervated dilatation and loss of contractile power will rapidly follow.

Dilatation is only too apt to accompany acute or chronic disorders of the pulmonary tissues, the right heart being first affected, but eventually both sides will dilate; dilatation is produced somewhat suddenly in asphyxia from croup, diphtheria, capillary bronchitis, pneumonia, and pertussis. Gourraud also notes it in cases of disturbance in the blood-making apparatus, in which the heart and nervous system are supplied with poor and ill-nourishing blood.

Pitt* has noted that at puberty, in rapidly-growing children, dilatation of the heart is apt to arise unless great care is used to prevent undue circulatory strain at this time. He points out that, according to the observations of Beneke, the annual increase in the size of the heart between seven and fourteen years of age is only eight per cent., whilst during the development of puberty the increase varies between eighty and one hundred per cent.,—that is, the heart nearly or quite doubles in size during the development of puberty. When the changes of puberty are accomplished in one year the heart doubles in size during that year; when the changes are spread over two years the annual growth is fifty per cent.; when over five years, twenty-two per cent.; so that in each case the same change—that is, doubling of the size—takes place. The importance of these changes to the individual may be inferred from the fact that the heart may in this one year grow three times as much as it did in the preceding year, but when such a task is demanded of an organ, it is only in the minority that it can take place in one year without unfortunate results. Clinical experience appears to show that the great demands made at this time on the heart may lead to slight cardiac dilatation and to the lack of reserve cardiac energy to meet emergencies so common at this age, especially in those who have grown very rapidly. The symptoms of cardiac weakness are usually not met with in those in whom puberty has developed rapidly, but in children in whom it is retarded or is still incomplete. Pitt cites the case of a girl, aged sixteen, who had grown nearly three inches during the preceding year, and was the average height. She had noticed for some time previously that she easily became short of breath, and after playing tennis suffered from severe dyspnœa for the rest of the day, with a feeling of cardiac discomfort sometimes amounting to pain. More recently she had suffered from dyspnœa at night, occasionally so severe that she was unable to lie down; also from palpitation, languor, or partial syncope, and anæmia. Menstruation had occurred once six months previously, and was then only scanty. The cardiac impulse was found diffused over an area of an inch and a half, extending in the fifth interspace to the nipple-line. There was

* British Medical Journal, November 27, 1886.

slight epigastric pulsation. The first sound at the apex was prolonged, the second over the pulmonary area accentuated, and the first over the aortic area weaker than normal.

Physical signs and symptoms.—In the young hypertrophy will cause a yielding and bulging of the præcordia; this is very marked in young cases. Schrötter thinks that pericardial adhesions are always associated with this condition, but such does not seem to be the fact. Dilatation alone does not cause bulging. The apex-beat and the cardiac impulse is much changed; hypertrophy causes a downward displacement with a marked left lateral obliquity. We have frequently noted the apical impingement in the seventh interspace at the anterior axillary line; its beat was forcible, powerful, and well defined. Dilatation as it arises will increase the visible cardiac movements in a transverse direction, but will not cause them to become much, if any, lower; they will be diffuse, extensive, but ill-defined; sometimes they are undulatory, irregular in rhythm, or they may be even intermittent; occasionally under these circumstances the cardiac impulse can be seen but not felt,—a fact first recorded by Walshe,—at other times it is not perceptible at all, either to touch or sight. These alterations in the cardiac action depend much, of course, upon the relation of hypertrophy to dilatation; the particular part of the heart involved will have much to do with the seat and character of the impulse. Either dilatation or hypertrophy or both will cause marked and constant changes in the area of cardiac percussion-dulness. Osler records the case of a young man under his care for several years who was very emotional and hypochondriacal, and was addicted to excessive venery, whose left ventricle became strongly hypertrophied and beat without the nipple-line. We have already recorded cases in which the hypertrophy and dilatation were enormous.

Upon auscultation, when the valves are not diseased, hypertrophy will present a prolonged dull first sound; should dilatation exist the sound may be very clear and sharp; not infrequently do we note reduplication of the sounds. In other cases the *tintement métallique*, described by Bouillaud, may be heard; this sound is a peculiar clink, which must be heard to be appreciated: it is impossible to represent it in words or

symbols. The second sound is most usually clear and loud or ringing.

When dilatation becomes excessive the first sound is short, sharp, and valvular, resembling very closely the second; later it becomes very weak; reduplication is now, perhaps, more likely to occur, and murmurs may arise from simple incompetency of the valves, produced by the great dilatation. The pulse in hypertrophy unassociated with valvular lesions is full, regular, strong, and of high tension; its rate is generally about normal, although it may be increased; as dilatation arises the pulse becomes irregular and intermittent; indeed, these constitute the earliest signs of cardiac failure. As the degeneration advances the pulse becomes quick, small, thready, and weak.

The *prognosis* depends much upon the cause and the relation of dilatation to the hypertrophy.

In the fevers and in anæmia temporary dilatation often arises, but passes away as the general health is restored; but when we are unable to reduce the dilatation we base our prognosis upon its degree. Most cases of dilated hypertrophy are accompanied or caused by valvular disease. If the compensation is maintained, but few or any symptoms are presented. However, should the compensation fail, and our measures for its re-establishment be futile, the case at once assumes a serious aspect. In hypertrophy unassociated with dilatation the prognosis also depends much upon its cause. For example, the hypertrophy of acute Bright's disease is very apt to disappear with the subsidence of the primary disease; but should it be associated with irremediable disease, as chronic valve-disease, the prognosis presents an entirely different outlook, as here the welfare of the individual depends largely upon the ability of the cardiac muscle to maintain a sufficient degree of hypertrophy to compensate for the valvular leak, or obstruction, as the case may be. Under these circumstances a careful consideration of the general and local nutrition of our case becomes important. Another important factor is the seat of the valve-disease, as mitral stenosis* and regurgitation have a

* Osler believes that mitral stenosis, like the regurgitant murmur at the same valve, may exist for many years without exciting symptoms of heart-disease.

fair prognosis in relation to longevity. Such is also the case in aortic stenosis. Cases of aortic insufficiency do not allow perfect filling of the coronary arteries. Hence they do not favor hypertrophy, consequently their chance of establishing a compensatory hypertrophy is poor at best.

A dangerous form of hypertrophy is that which is attendant upon general arterial degeneration, on account of its liability to produce rupture, as illustrated in the case we mentioned when considering aneurism.

TREATMENT: *hypertrophy.*—Here the treatment is, as a rule, to maintain an hypertrophy which shall just be sufficient, and no more, for the extra work which the heart has to do. As in organic disease, while we are unable to remove the root of the trouble, still we can by extreme care and the intelligent co-operation of our patient or their parents maintain that nice balance which, while the hypertrophy is sufficient, still it is not excessive; and, on the other hand, we are able to prevent the occurrence of dilatation, which, as we have already learned, is an untoward event for whose appearance we must be ever on the watch.

In hypertrophy the diet, exercise, and habits are to be carefully regulated. Those little patients who present arterial lesions also, together with renal disorder and high tension, must particularly be guarded against the danger of rupture of the vessels. Cardiac irregularity and unduly forcible contraction can often be met by rest and the administration in the latter instance of cardiac sedatives, as aconite or veratrum, and in the former by small "steadying" doses of digitalis.

As we have already stated, boys, as they grow older, must be made to understand the danger of over-exertion, the use or abuse of tobacco and alcohol, or of sexual excesses.

Dilatation.—The treatment of this condition has been somewhat elaborately alluded to when treating valvular heart-disease, as the treatment of dilatation is practically that which is applicable to chronic valve-disease.

As the symptoms arise, the work of the heart must at once be reduced by enjoining absolute rest, as, for instance, in the dilatation of puberty referred to by Pitt (ibid.) the child was ordered rest in the recumbent position, which, in connection

with a regulated diet, arsenic, and iron, was sufficient to completely restore the heart to its normal condition.

Rest, in the majority of the cases of heart-failure in its early stages, is nothing more nor less than a specific, notwithstanding Oertel's* opinion that exercise, especially climbing, forms an important element in the treatment, as he considers that severe exercise stimulates the heart-muscle and favors the restoration of hypertrophy. Osler considers his suggestion of a reduction in the amount of liquids ingested as a happy method of reducing the volume of blood to be circulated by the heart.

Digitalis becomes a true heart-food in these cases; by its exhibition we render the irregular, wavy, feeble, and frequent contractions regular, slower, and stronger, at the same time the overloaded, embarrassed circulation is markedly relieved. The medicinal substitutes for digitalis were considered in the last chapter.

In the final asystolism which will eventually arise, our sheet-anchor—digitalis—seems to have lost its most desired action, and we will be obliged to resort to alcoholic stimulation, together with hypodermic medication of rapid diffusible stimulants, as ether, carbonate of ammonia, or brandy.

ATROPHY OF THE HEART.

Cardiac atrophy occurs either congenitally or as an acquired affection; this condition was formerly termed phthisis of the heart. The heart is most usually decreased both in size and weight; in many of the degenerations of the heart-muscle there is localized atrophy of the fibres.

Congenital atrophy is almost entirely confined to the female sex, and is associated with anomalies or defective development of the arteries and the sexual organs.

Virchow has called attention to this condition of cardiac and circulatory atrophy in cases of chlorosis. Gowers† mentions a case seen by Allen Burns, in which an adult presented a heart corresponding in size to that of a child of six or seven years.

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* Ziem. Handbuch der Allgemeine Therapie, Bd. iv.; Osler, American System of Med., p. 635.

† Osler, ibid.

Paul describes an atrophic sclerotic myocarditis which he states may occur during intra-uterine life. Phthisis seems to cause a marked and persistent atrophy of the heart. Quain has shown that in 54.4 per cent. cases of phthisis the heart is below the normal size in regard to weight; typhoid fever, marasmus, and diseases of a subacute character may lead to wasting of the heart. Locally, pressure by pericardial adhesions, by mediastinal growths or deformities, and interference with the circulation in the coronary arteries may cause atrophy.

Post-mortem examination in simple atrophy presents a general diminution in size and weight; in local atrophy one or more of the cavities or portions of the walls of the heart may be found below the ordinary standard. The atrophied heart may be normal in color, or it may be very pale; it has been noted of a deep reddish-brown. The pericardium presents a somewhat peculiar appearance, which Laennec has aptly styled a "withered-apple" aspect; it is due to the fact that the pericardium, not shrinking with the heart, presents a puckered, opaque, and œdematous appearance; the coronary arteries become prominent and tortuous for the same reasons. An important fact to remember in making these post-mortem examinations is that the acquired variety of atrophy alone presents the appearances just described; they are never seen in the congenital forms.

Symptoms.—These are not at all diagnostic; they are simply those that of necessity arise from diminished power of the heart; the sounds are feeble, the pulse is weak, and anæmia or chlorosis is generally present.

Diagnosis.—Atrophy of the heart is rarely diagnosed during the patient's life.

The *treatment* consists in combating the primary disease which has caused the atrophy.

CHAPTER IX.

CARDIAC NEUROSES.

FUNCTIONAL disorders of the heart's action, irrespective of inflammation or structural lesion of any kind whatever, constitute a frequent and an important class of cardiac diseases in

the growing child; they are usually paroxysmal in their manifestation, and may or may not be accompanied by pain.

Palpitation.—A violent or tumultuous action of the heart is a personal experience through which most of us have passed, excited by some intense mental emotion; we, however, have observed persons in whom the cardiac equilibrium could not be disarranged no matter what the provocation, either mental or physical. In these cases it has seemed to us that the heart possessed a peculiar organization, which maintained its rhythm under all and every condition; the pneumogastrics and the sympathetic ganglia in these individuals appear to be perfectly phlegmatic or callous, if we may so apply the term. Some children are undoubtedly endowed with pneumogastric and sympathetic nerves that are slow to carry impulses to the circulatory apparatus, while others have neurotic fibres which are always alert and active, responding but too readily to the slightest irritation. Da Costa has most happily expressed these thoughts in a terse manner by the term "irritable heart," which is peculiarly apt. This condition is met with under many diverse circumstances and conditions, and presents all degrees of severity, from the mildest disorder, often described by the patient as a "fluttering," to a most severe functional disorder accompanied by a fear of impending death.

Irritable heart in the young is generally a concomitant of neurasthenia, over-exertion, sexual excesses, and the abuse of certain articles, as tobacco, tea, coffee, or alcohol,—the so-called toxic cases. It is also seen in some anæmic and leukæmic cases and in malarial poisoning. The immediate cause is, of course, an undue excitability of the muscular wall of the heart, in all probability brought about by derangements in the cardiac ganglia, the vagus, and in the filaments of the ganglia of the great sympathetic, which are distributed to the heart. This condition is what is described by the parents as a nervous and excitable temperament in their child; Thomas Shapter considers early age a direct predisposing factor to this condition.

The irritable heart from over-exertion is met with in young lads who are gymnasts, runners, jumpers, etc.; the disorder is here sometimes styled heart-shock when due to a single great

and continued effort; in all probability, as Osler* states, there has been an acute dilatation caused by the sudden strain, and the heart possibly never returns to its normal condition. The other form induced by continued over-exertion is the condition to which Da Costa first called attention as met with in young soldiers during the war of the Rebellion, and now frequently seen in civil life in young persons exposed to occupations requiring strength beyond their years; some authorities have styled this form of palpitation as idiopathic dilated hypertrophy; Osler is of the opinion that syphilis may enter into its causation.

A fruitful source of palpitation or irritable heart in young boys is masturbation, or in older individuals excessive coitus; in many instances in which we have been consulted the cardiac symptoms speedily disappear, without other active treatment, when the youth was shown the moral and physical dangers of his habits.

Tobacco must often be considered when seeking the etiology of cases that desire our opinion.

James G., aged sixteen, presented himself at our service in the University Hospital; the boy worked in a cigar-manufactory,† and stated that he habitually smoked eight to ten strong cigars a day, and desired our advice in regard to the condition of his heart. Examination showed a somewhat pale, anæmic boy, with an anxious expression, some dyspnœa, which he stated was greatly increased by exercise, marked palpitation, considerable cardiac distress or pain. The heart was acting at the rate of one hundred and eighty beats per minute, was very irregular, sometimes intermittent, but did not present any

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* Toronto Med. Soc , April 14, 1887.

† The examination of one thousand workers in tobacco compared with a series of control experiments on animals resulted in finding among the workers dilatation of the pupils, cardiac neurosis, exaggerated tendon and vaso-motor reflexes, trembling hands, dyspnœa. Headache, gastralgia, and nervous cough were also present. The respiratory organs were most frequently attacked after the nervous system. The same symptoms were produced by injecting one-fortieth to one-tenth drop of nicotine in rabbits, one-twentieth to one-third in dogs, and keeping them also in a tobacco-impregnated atmosphere. (Bull. Gén. de Thérap., July 15, 1887; Med. News, Oct. 8, 1887.)

evidences of increase in size either by dilatation or hypertrophy, —usually these cases present some slight enlargement. The patient was unable to assume the recumbent position but for a few minutes, was totally unable to lie upon the left side, for as soon as he assumed a left lateral decubitus a severe attack of angina would arise. This pain in many cases is very characteristic of the toxic action of tobacco upon the heart; we have notes of many cases in the young in which it was always present.

The pulse in irritable heart may be extremely irregular, varying from 75 or 80 to 150 or 200, depending much upon the patient's environment either before or during our examination; position will exert a decided influence upon the rate, usually slower when the patient is recumbent.

A peculiar flushing or lividity of the cutaneous surface, due to vaso-motor change or innervation, is sometimes noted.

Graves's disease and tachycardia, with pulsation at the rate of 180 to 200, are considered by Osler as forms of irritable heart.

Treatment.—The removal of the cause is absolutely necessary to attain success; this, with perfect rest, preferably in the recumbent position, will often suffice to obtain a cure. In treating chronic tobacco-poisoning, in addition to the essential point of total abstinence, which, unfortunately, often cannot be obtained, Favarger* aptly advises that smoking be never indulged in on an empty stomach. Inasmuch as nicotine has been found in the perspiration and urine, he considers it not unreasonable that means to promote its elimination be used in treatment, such as packs, diuretics, and diaphoretics. Galvanism has been used by some authorities in cases of tobacco-heart with marked benefit; we have in a recent case obtained happy results from trinitrin, one one-hundredth-grain doses, increasing to full physiological tolerance, together with a regulated diet and absolute rest.

Palpitation, independent of the toxic causes, may often be relieved by bromide of potassium, or, in weak individuals, the bromide of sodium; strychnia is peculiarly efficacious in many

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* Therap. Gaz., Oct. 15, 1887, p. 689.

instances; ammonia in other patients produces speedy relief; arsenic and cod-liver oil will often fulfil special indications. It has been our practice for some time to cover the præcordia with a freshly-made belladonna-plaster.

The digestion must receive especial care. We have all met with cases in which starchy or saccharine foods in stomachs unable to digest them have caused distressing and alarming symptoms by over-distending the stomach, thus affecting the heart both by a direct mechanical obstruction and by a reflex irritation. In those cases of cardiac irritability which are gastric in origin, small doses of carbolic acid and soda will often prove of inestimable benefit. Patients of a gouty or rheumatic diathesis are particularly liable to have irritable or irregular cardiac action, which will be markedly increased by errors in diet, especially the exhibition of alcohol.

Prompt relief must often be obtained for the more severe forms of palpitation; we generally select for this purpose the antispasmodics, opium, ether, or well-diluted alcohol, or Hoffmann's anodyne. The German writers recommend the application of ice to the præcordia, which may be accomplished either by an ice-bag or by means of Littre's tubes. Flint has arrested a paroxysm by mechanical means, as pressure upon the abdomen, holding the breath after a deep inspiration, and compression of the vagus and sympathetic nerves in the neck.

Clemens* agrees with Stokes and Oertel in recommending enforced exercise for the so-called nervous palpitation.

Infrequency of the heart's action.—Cases of diminished frequency are recorded in which the individual presented a normal pulse-rate of 60, 40, and, under certain conditions, as low as 32 per minute; these are congenital peculiarities of the circulation. With the acquired variety in later life, a not infrequent condition, we have in this treatise nothing to do. Children with infrequent heart's action are apt to present some form of cerebral disturbance; these may be of the nature of epileptiform seizures or great mental excitability.

Flint calls attention to a curious form of functional disorder which would lead to the error of inferring infrequency of the

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* All. Med. Central Zeitung, Sept. 10, 1887.

heart's action from the pulse alone. This condition is characterized by the regular alternation of a ventricular systole, giving rise to a radial pulse, with one too feeble to be appreciated at the wrist. For example, Flint assumes the number of the ventricular systoles to be 70 per minute; in such a case the radial pulse would be 35 per minute. He has met with several cases of this disorder; the carotid pulse, however, accurately represents the heart's systole,—so that with auscultation we would note four sounds to each radial pulse. In this wise we may fall into the mistake of considering the case as one of reduplication of both the first and second sound. We have not as yet met such a case in young children, but have noted them in patients of eighteen years or over.

REDUPLICATION OR DOUBLING OF THE HEART-SOUNDS.

Reduplication of the first or ventricular sound may be heard in a perfectly healthy individual; it is, however, under these circumstances not constant, heard to-day and inaudible to-morrow.

It is also noticed in connection with heart-disease, though here, again, it may not be constant. Doubling of the second sound—arterial sound—is met with as the next most frequent abnormality of this kind.

Various explanations have been advanced in explanation of reduplicated sounds; some considering, as Da Costa does, that they are caused by an arrest of synchronous action in the right and left hearts; others that it originates in non-synchronous tension of the individual segments of the auriculo-ventricular valves.

That the former explanation in some cases is the correct one we are able to testify from clinical observation, as in a few instances in children, with their thoracic walls, we have been enabled to note a double impulse accompanying each systole. Bamberger, Leyden, and Skoda give similar testimony; also endorsed by Malbrane, Gerhardt, Freidrich, Rosenstein, and Roy. One of our cases was far advanced in the sequential lesions of mitral insufficiency. It is interesting to note the fact that with the abnormal beat there is no pulsation in the arteries. Paul endeavors to explain the non-simultaneous

action of the ventricles by stating that when the mitral valve is markedly incompetent, the overfilled right ventricle is unable to empty itself completely during the systole, and the next instant, during the diastole, is again distended with blood, and so excited to renewed contraction; on the other hand, he states that the left ventricle takes no part in this second or abnormal contraction of the right heart,—that is, at least none that is appreciable to our hearing or our sense of touch.

Stearn,* however, takes exception to the statement that both ventricles act simultaneously with the first of the double sounds, while the right acts alone with the second; he states that neither anatomical structure nor nervous supply will allow such a theory to be accepted, citing against it also the fact that there is no hemi-systole in the dying heart.

Further, in one of his cases there was a pulsation with each of the systolic sounds in the carotid artery, although there was none in the radial, and he is therefore obliged to conclude with Bozzolo that there are two complete systoles, one following the other very rapidly, as the cause of the phenomena. He explains the absence of the radial pulsation with the second beat by the fact that systole recurs so rapidly that there is no time for the left ventricle to refill, this being the more difficult as there was in his two cases—which he believes to be always present in such—tricuspid regurgitation; this would still further diminish the quantity of blood going to the left side, while the systemic veins and right auricle would be surcharged and ready to pour their contents into the right ventricle. A further point militating against the statement that reduplication of the first sound is due to a want of simultaneous contraction of the ventricles, is the fact that the second arterial sound is not also doubled, for if the ventricles do not act together, the diastolic closure of the semilunar valves —aortic and pulmonary—should also be non-synchronous.

A rare form of reduplication is that in which the first sound is split into three parts, the *trommelschlag*, or drum-beat, of the Germans.

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* Deutsches Arch. für Klinische Med., October 15, 1884; also Edinburgh Medical Journal, December, 1884.

Potain has reported in cases of cardiac hypertrophy dependent upon "granular atrophy of the kidneys" a variety of reduplication of the first sound which he has designated *bruit de galop*, in which besides the normal sounds an additional sound preceding the first was noted; this was considered to be due to contraction of the hypertrophied auricles.*

As we have already stated, reduplication of the first sound may occur entirely independent of any appreciable disease, so also may we meet *reduplication of the second sound;* most usually, however, it is an evidence of some cardiac disorder. Its cause seems to be alone want of synchronous closure of the aortic and pulmonary valves, consequently the two sounds do not correspond with each other. It seems possible to us, however, that the tension of the leaflets might occur in two separate and distinct movements, and thus occasion division of the second sound. We have met cases in which the division occurred over but one of the arterial orifices, most usually the aortic, which we are totally unable to explain except upon this hypothesis. We have the notes of two cases in which reduplication of the second sound was associated with organic mitral disease; we have also been enabled to verify its association with adherent pericardium, a condition that is met with even in the very young, as Behier† noted an infant of eleven months with an adherent pericardium, the result of chronic pericarditis.

ANGINA PECTORIS.

This name, like many others in medicine, was first used to designate a group of symptoms, but has now crept into our nomenclature as the title of a disease.

Angina pectoris may be considered a paroxysmal neuralgia, having its maximum intensity in or about the præcordia, from there extending upwards to the left shoulder and down the arm to the tips of the fingers. This, however, is not the invariable course of the pain: a young girl under our care

* Those who desire to pursue this subject further are referred to a recent article by Cuffer and Barbillion, in the Gaz. des Hôp., No. 36, March 24, 1887, p. 284.

† J. M. Da Costa, Amer. System Med., vol. iii. p. 786; from Constantin Paul, Mal. du Cœur, Paris, 1883.

suffers most intensely in the right shoulder and arm, and other cases have been recorded in which the pain extended down the legs.

These paroxysmal attacks are almost always accompanied by a fear of impending death,—a fear which we are able to testify is sometimes most unfortunately realized in those cases who present an angina as secondary to organic disease of the heart or larger vessels; in its severe form it is perhaps the most excruciating pain that we will be called upon to allay; associated with this intense suffering is the sensation that death is about to take place; a choking sensation, together with restrained respiration, and disturbed circulation all add to the patient's distress.

Sometimes we have noted decreased action of the pulse at other times in the same patient, or in different patients we have noted marked increase in the pulse-rate; the heart's action is also irregular and occasionally intermittent; arterial tension is generally increased at first, but diminished later in the attack; the face is most frequently pallid. One of our cases, however, had great lividity of the face throughout the attack; the expression is one of extreme anxiety, afterwards becoming haggard or depressed. The cutaneous surfaces are cold and usually bedewed with perspiration; the condition of the circulation will depend much upon the degree and character of the coexisting organic heart-disease, if it is present, together with the functional disturbance excited by the paroxysm. Angina pectoris is proven to be associated with fatty heart; also, as Flint tells us, with obstruction of the coronary arteries. The paroxysms are apt to arise suddenly and disappear in a similar manner, to be followed by a discharge of pale watery urine; their duration is but a few moments, although they may be protracted into minutes; their severity may be most intense, or, on the other hand, they may be extremely slight, with but a momentary heart-pang and but transient disturbance of the circulation, the entire paroxysm being but a passing annoyance.

The *exciting cause* in some individuals is very slight; one of our cases would be awakened by the first twinge of pain, another was unable to face a strong wind, in still another it was caused

by swallowing a large bolus of food. As the cases progress the attacks appear to arise without any exciting cause; the intervals between the attacks may be long or short, some cases not having more than one a year, others one every month or so.

There is little difficulty in making the *diagnosis* if the case is at all carefully observed. Mistakes have arisen in the differential diagnosis between this disease and gastralgia or intercostal neuralgia.

Many attempts have been made to explain the pathology of this affection. We agree with Flint that most cases are due to ischæmia of the heart: the nerves concerned in the excruciating pain are probably the sensory fibres of the pneumogastrics. Their connection with the brachial plexus causes the pain to radiate down the arm. Flint, in endeavoring to explain the occurrence of pain in parts which have no direct connection with the cardiac nerves, states that a centripetal influence conveyed to the nervous centres may occasion pain referable to different situations.

When angina pectoris arises as a purely functional cardiac neurosis unassociated with organic disease, the pathology and indeed the etiology are difficult of interpretation; fortunately, however, these cases are rare. Some authorities have considered tobacco, gout, hysteria, and exposure to cold as etiological factors.

Age.—We have never met a case of angina pectoris in a patient under seventeen years; most cases occur after middle life, and principally in men.

Prognosis.—A patient the subject of this disease must be considered as subject to sudden death during any paroxysm; the fact that previous paroxysms have been mild is no assurance that a severe and fatal attack may not arise at any time; on the other hand, the attacks may recur for years and the patient ultimately succumb to another disease. A single paroxysm may alone occur, with possibly a return after many years, or perhaps without recurrence at all.

A most important factor in the prognosis is the existence of organic heart-disease; recovery is, of course, out of the question under these circumstances. Indeed, the danger of sudden death from asystolism must always be considered, especially should

the aortic valves be incompetent or the cardiac muscle fatty; on the other hand, if there is no organic disease and the heart and circulation are not materially disarranged during a paroxysm, the prognosis in relation to life is fairly good; indeed, the prospects of cure may be here favorably entertained.

Treatment.—The treatment is to be divided into that which is appropriate to the attack and the measures which are to be adopted during the intervals with a view to preventing a recurrence.

During the paroxysm we must not alone combat the intense pain, but we must also direct our attention to the ever-present danger of speedy dissolution from asystolism. These indications are best met by the administration of morphia, together with a quick diffusible stimulant, as ether, Hoffmann's anodyne, or alcohol in small doses, frequently repeated; but, unfortunately, we are not apt to be present during an attack, so that it is well to instruct our patients in the management of their own cases; nitrite of amyl inhaled at the outset will in some cases produce the most happy results; counter-irritation of the præcordia will, in a certain number of cases, prove beneficial.

During the interval we must caution our patients to avoid all known exciting causes of a paroxysm; exercise or work, either mental or physical, is never to be carried to the point of fatigue. The young are to be particularly cautioned in regard to sexual intercourse and the use of tobacco, tea, or coffee.

The diet is to be carefully regulated, and so balanced that while nutrition shall not suffer there will still be no excess to tax the digestive powers and reflexly irritate the heart or cardiac ganglia. Certain constitutional dyscrasiæ are, if possible, to be corrected, notably the gouty and rheumatic. An anæmic condition appears to be both an exciting and predisposing cause, which is to be removed if at all possible.

Many drugs have been advanced as specifics in this disease, but, unfortunately, like most specifics, they have been of value but in the hands of a few observers. Nux vomica is worthy of trial, and in some cases digitalis or trinitrin may prove efficacious. Beard and Rockwell suggest the use of electricity.

EXOPHTHALMIC GOÎTRE.

Exophthalmic goître, *Graves's* disease or *Basedow's* disease, the *maladie de Graves, goître exophthalmique* of the French, and the *glotzangenkropf, Basedow'sche krankheit* of the Germans, is an enlargement of the thyroid body, accompanied by protrusion of the eyeballs, palpitation or increased cardiac action, dyspnœa, and anæmia.

We have met several cases of this disease in young girls, developing at or about the first menstruation, or following a severe mental strain or exhaustion at this time. All of our cases were anæmic and presented some premonitory symptoms before the disease was fully established,—the circulation was disturbed and the heart very irritable, later the face would flush and a sensation of fulness in the eyes and throat would be complained of; as the disease became fully developed, the heart became irregular and tumultuous in its action, the eyes would protrude, and the thyroid gland became prominent.

The cases presented no marked disturbance of vision; but there was a want of synchronism between the motion of the lids and that of the eyeballs; later the lids are usually unable to cover the balls, even during sleep. During the menstrual flow all the symptoms are increased, particularly the sensation of over-distention in the neck and eyes.

The thyroid gland is not always equally enlarged, and it may increase or decrease in size; it pulsates synchronously with the heart's beat, and in its earlier development has been mistaken for aneurism. The heart usually presents the earliest signs of commencing disturbance in cases of exophthalmic goitre; generally it is the first symptom noted by the patient. Its action becomes irregular, tumultuous, and rapid, in the young producing a prominence of the præcordia. The sounds are altered, becoming loud and accentuated; not infrequently a soft cooing murmur is audible at the base of the heart and in the larger arteries; the entire circulation seems to be unduly stimulated. We have noted pulsation in the small arteries.

Constitutionally the patients are generally in an anæmic, debilitated condition, with poor appetite and faulty digestion; occasional attacks of diarrhœa may arise. The temperature is

apt to be slightly elevated, although this is by no means a constant symptom.

The increased size of the thyroid is apt to produce some difficulty in breathing, and may cause an alteration in the voice, or indeed complete aphonia.

Prognosis.—Exophthalmic goitre pursues a somewhat variable course; it usually increases from several months to years, then reaches a stationary period, from which it may either decline, with re-establishment of normal menstrual function and subsidence of cardiac and ophthalmic symptoms, or, on the other hand, the disease may progress and death occur from organic heart-disease, asthenia, or by intercurrent disease.

Some cases present alarming symptoms due to pressure on the trachea.

There are cases on record in which the protrusion of the eyeball was absent throughout the case; all the other symptoms, however, were present in a characteristic manner.

Pathology.—The enlargement of the thyroid gland is due to dilatation of its vessels, secondary to the persistent increased cardiac action, which is probably due to a stimulation of the accelerator nerves of the heart, the root of the trouble being probably situated in the sympathetic and vaso-motor nerves. An increased amount of connective tissue and a diminution of the ganglionic cells has been noted in the lower cervical sympathetic ganglia.

Lauder Brunton considers that "the protrusion of the eyeballs is due either to dilatation of the vessels in the orbit or to contraction of the involuntary muscular fibres in the orbital membrane which cover the spheno-maxillary fissure, or possibly to both causes combined." *Sex* and *age* are decided predisposing factors. Most of our cases were in females, and none were younger than twelve years; we have never seen the disease in a girl before puberty nor in a male under twenty years of age.

Treatment.—Pathologists not having as yet arrived at a consensus of opinion regarding the cause or the lesions present, our treatment must be somewhat palliative and expectant; a radical curative agent is not yet in our possession. We must content ourselves for the most part in securing a healthful

abode, an occupation or mode of living that will not cause emotional disturbance or mental and physical fatigue, and an adequate diet with the avoidance of all dietetic errors or excesses. The use or abuse of alcohol or tobacco must be especially explained to the patient; it is also well to regulate the sexual indulgence.

Iron, in many cases, will prove very beneficial by correcting the attendant anæmia; digitalis should be tried to correct the accelerated cardiac action. Quinine, digitalis, and belladonna in combination often produce marked benefit.

Tri-weekly applications of a mild galvanic current to the neck and the thyroid tumor should be tried; indeed, in our own practice this has been most satisfactory.

Some writers recommend aconite and cardiac sedatives, or the application of an ice-bag to the præcordia. Care should be taken that the eyes do not become affected from the inability of the lids to close and protect them. Deep corneal ulcers sometimes result.

CHAPTER X.

DISEASES OF THE BLOOD.

THE blood consists of two primary elements, cells floating in an albuminous menstruum; the former constitutes a little less than one-half by weight of the blood; the menstruum, or blood-plasma, is almost entirely water,—about ninety per cent.,—the remaining portion consisting of proteids, the elements of fibrin, salts, principally sodium chloride and sodium carbonate, a trace of sugar, some fat and creatin, hypoxanthin, and urea.

Of recent years four forms of corpuscles have been described,—red, white, nucleated red, and the hæmatoblasts,* or blood-plates,† or invisible corpuscles.‡ The red blood-corpuscles are present in each cubic millimetre of blood to the

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* Hayem. † Bizzozero. ‡ Norris.

number of about five millions; they have a fairly uniform size, stated by Hayem to be about 7.5 μ*; this is about $\frac{1}{3200}$ of an inch.

The colorless corpuscles are about 10 μ, or $\frac{1}{2500}$ of an inch, in diameter; they are nucleated, and have a granular protoplasm; at the temperature of the body in healthy blood they present amœboid movements. They are present in a cubic millimetre of blood in the proportion of about one to three hundred, or one to five hundred red corpuscles. The nucleated red corpuscle is seen in the blood of the fœtus and infant; at the fourth year they have entirely disappeared, so that the blood of a healthy adult will be without them; they are normal constituents of the marrow of the long bones. Their nuclei may be single or multiple; three have been noted; they are colorless; in some cells grouped together, in others not, again in others they may protrude from the cell. The corpuscles are about $\frac{1}{1800}$ of an inch, some being colored quite as the ordinary red corpuscle.

Lastly, the hæmatoblasts† are small, colorless corpuscles, varying in size, but of an average of 3 μ in diameter. They are found in perfectly healthy blood. The hæmatoblast is a separate blood-element, and not the result of any disintegrating process in any of the other forms of corpuscles; it is intimately connected with the production of fibrin.

Having now considered the four blood-elements, it becomes interesting to inquire into their origin. The manner of formation of the blood-plates, or hæmatoblasts, is to us as yet a sealed book; we know absolutely nothing about them. Osler remarks that they occur under the most diverse conditions,—in the young organism, just commencing life, and, on the other hand, in the old, worn-out, cachectic individual, just about giving up life. Nor is the manner of the development of the red corpuscles settled beyond question; it is said that they originate from the colorless corpuscles and the lymph-cells, or leucocytes; that the thymus gland, the gland-tissue of the

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* μ is used as a symbol for a micro-millimetre, or $\frac{1}{1000}$ of a millimetre.

† Synonymes: *Blutplättchen*, *microcytes*, *elementary corpuscle* (Zimmermann), *globulins* (Donné), *grains sarcodiques* (Vulpian), *granulations libres* (Rauvier), *Schultze's masses*.

tonsil, the spleen, the adenoid, or lymph, tissue of the intestines, all are situations in which the red cells may develop from the leucocytes there created. That the red corpuscles may develop from the nucleated red corpuscles is at least established as far as the embryo is concerned; as the child grows the nucleated red cells disappear, except in the red marrow.

Osler* has observed that the nucleated cells appear to originate from colorless marrow-cells, which gradually become more homogeneous with hæmoglobin developing in the protoplasm; eventually the nucleus will disappear or degenerate, and the cell has the appearance of an ordinary red corpuscle.

The same observer has seen and sketched cells that were budding from the marrow-cells.†

Bizzozero states that the nucleated red corpuscles are independent blood-elements, developing by fission, and with the disappearance of the nuclei they constitute the ordinary red corpuscles.

There seems to be a unanimity of opinion in regard to the origin of the colorless corpuscles which develop from the cells of the follicular cords in the lymph-glands and adenoid tissues; the stages and manner of this development have not, however, as yet been fully worked out.

Space forbids us pursuing the extremely interesting study of the relation of the cytogenetic organs to the blood-formation, or as to what ultimately becomes of the red corpuscles.

PLETHORA.

In the healthy infant at birth there is always a condition of comparative plethora, as at this time the blood by weight amounts to one-eighteenth of the body, while in the adult the relation is one-twelfth to one-fourteenth. The so-called plethora of the older writers is scarcely admitted to our classification of to-day. If the organs of secretion and excretion are normal, they maintain the quantity and quality of the blood at about its normal standard.

Osler considers that those individuals with red faces, full

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* Centralblatt f. d. Med. Wissench., 1878.

† Trans. Am. Assoc. Adv. Sci., 1882; Am. Syst. of Med.

vessels, and bounding pulses, who give the impression of a greatly distended circulating system, present this appearance more as the result of an active blood-distribution than of an actual increase in the total volume; he doubts if the evidence will support the view that a rich and abundant diet, without much exercise, will permanently increase the amount of blood.

Cohnheim coincides in this view by stating that, "except as a transitory state, polyæmia does not occur under any circumstances."

In those individuals who are popularly known as "plethoric" we probably have a condition of distention of the superficial vessels, in the face notably, and not a general increase in the blood-mass, cells, and plasma; these persons may, however, have a superabundance of red corpuscles, polycythæmia rubra; observers have noted cases in which the red corpuscles were markedly increased in number. Of course in those patients who have suffered a great loss of fluid there will be found an increase in the cells.

ANÆMIA*

Is a systemic disorder dependent upon a definite lesion, which, thanks to recent observers, can be demonstrated with the utmost precision and satisfaction. The causes of anæmia may be conveniently considered under two headings, predisposing and exciting. Under the former we may mention sex, age, and constitutional peculiarities; and of the latter the most prominent are undoubtedly hemorrhage and the general fevers. Anæmia may be congenital, as in the following case reported by F. P. Henry:† Mary C., born at 5.20 A.M., November 5. Count made at 2.30 P.M., November 6. Child weighed six and three-quarter pounds at birth; labor normal. Number of red corpuscles per cubic millimetre, three million six hundred and twenty-five thousand; proportion of white cells to red, one to one hundred and forty-five. This case was undoubtedly one of congenital anæmia. The child's only appearance of malnutrition was a shrivelled state of the integument of the

* Synonymes: *Spanæmia, oligæmia, oligocythæmia, anémie, anämie.*

† Anæmia, Phila., 1887, p. 18.

feet and a less rosy color of the skin than normal. For a new-born child it was decidedly pale. This shrivelled state of the skin emphatically negatives the idea of a relative anæmia from excess of fluid. The blood was probably deficient in quantity (oligæmia) as well as defective in quality (oligocythæmia). There was also a decided increase in the number of the white cells. There had been no hemorrhage from the umbilical cord. As other causes it is well to bear in mind sexual excesses, deficient food or faulty assimilation, or bad hygienic surroundings, a long-continued albuminous drain, as by prolonged suppuration or chronic Bright's disease. A severer form of anæmia is of course produced by a large hemorrhage; but it is worthy of note that the human organism is able to withstand the loss of a very large quantity of blood and ultimately make a complete recovery, as the process of blood-reformation commences and is carried on with astonishing rapidity, requiring, however, many months before the cellular elements have regained their normal state. In other cases it may never be restored, and a condition to which we will refer more fully will arise, and the patient never recover. Even as the corpuscles increase in number the hæmoglobin does not also increase; so that the corpuscles while normal in number have a lowered hæmoglobin percentage.

Profound anæmia may be produced by the action of poisons upon the blood, as by lead, arsenic, or mercury. Anæmia is also a concomitant of syphilis and malaria. Recent investigations* seem to show that in the latter disease a micro-organism may be the active agent in destroying the red corpuscles, these bodies occupying the interior of the red corpuscles and occurring free in the plasma; some are ciliated, or at least have prolonged, wavy terminations that are seen with difficulty, others are crescentic.

Anæmia may be produced by a disturbance in the functions of the organs upon whose integrity the blood-elaboration depends. We approach this subject with some hesitancy on account of our want of knowledge of the process of hæmatogenesis. Our study has, however, progressed sufficiently

* Welch, Osler, Councilman, and Italian and East Indian observers.

for us to state with a certain degree of assurance that the spleen, the bone-marrow, and the general lymphatic tissues are the primary seats from which the matured red corpuscle finds its way into the general circulation; the marrow and the spleen seem also to possess the power of blood-destruction; as Osler aptly puts it, "they consume their own smoke, using the waste-products for the purpose of further manufacture."

A somewhat peculiar fact is that with impaired activity in the blood-making tissues we note an increase in size in these structures. How different, then, is this change, as we are accustomed to associate decrease in the size of an organ as synonymous with want of functional activity! We are able to conclude, however, that a progressive increase in size of the blood-making organs will sooner or later cause or be attended by a more or less profound anæmia. This change pertains whether one or all of the blood-making organs or tissues are affected. Furthermore, all the most important and essential symptoms of anæmia will be present, even if but one organ is affected, as the spleen or bone-marrow, or if the entire series are impaired. But the diseases caused by affections in the various blood-making tissues have received different names, and may be classified, as Osler states, in the following manner:*

Primary or cytogenic anæmia.

- Leucocytic. { Splenic. / Lymphatic. / Medullary. } Leukæmia.
- Non-leucocytic. { Splenic, anæmia splenica. / Lymphatic, Hodgkin's disease. / Medullary, idiopathic anæmia (certain cases). }

To consider this little chart more carefully we will start with—

Simple anæmia (primary or cytogenic anæmia).—This is a frequent form; sometimes its etiology is extremely difficult to interpret, at others its association with profound malarial intoxication is marked; the spleen is usually much enlarged;

* Henry (*ibid.*) makes a more elaborate classification under the head of *primary anæmias*, *secondary anæmias*, *toxanæmias*, and *parasitic anæmias*.

rarely are the lymph-glands noticeably increased; some authorities style it the splenic form of Hodgkin's disease, or pseudo-leukæmia.

Lymphatic anæmia (enlargement of the lymph-glands accompanied by anæmia) is the condition styled Hodgkin's disease, or pseudo-leukæmia. In this disease the spleen and marrow are not often affected, but there may be a general increase in size of the lymphatic elements of the body, with nodular enlargements in the organs; to differentiate this disease from lymphatic leukæmia recourse must be had to the microscope.

Medullary anæmia.—This form of anæmia is as yet an undecided problem in the study of hæmatogenesis, some observers regarding the changes in the marrow as a consequence of the anæmia, others as its cause. Cohnheim and Pye-Smith regard medullary anæmia as an established fact, Pepper thinks that the so-called idiopathic anæmia may have its starting-point in changes in the bone-marrow.

Osler considers, however, that we have not as yet arrived at a true solution of the condition, as alteration and hyperplasia in the bone-marrow has been noted in wasting diseases, and, furthermore, cases of idiopathic anæmia are recorded in which the marrow was normal. As we see by the chart, we may also have a splenic, lymphatic, or medullary *leukæmia*,—that is, an anæmia attended by an increase of the colorless corpuscles.

As in simple anæmia the splenic form was the most frequent, so in leukæmia.

Splenic leukæmia, in all features save the excess of colorless corpuscles, is identical with splenic anæmia.

Lymphatic leukæmia.—This is a less common affection than lymphatic anæmia, or Hodgkin's disease; rarely is it met as a primary uncomplicated disease. It is usually seen in association with enlargement of the lymph-glands, or of the tonsils, or Peyer's glands.

Medullary leukæmia.—A myelogenous form of leukæmia is a well-recognized clinical fact. Neumann regards the marrow changes as the most essential and important, and would relegate to the lymph-glands and spleen a secondary part in the process.

Whether it is advisable for us to clinically divide cases of anæmia and leukæmia is as yet among the undetermined problems of medicine, but, to quote the words of Osler, "It seems questionable whether such a variable feature as increase in the colorless corpuscles should be permitted to separate diseases which have all essential characters in common. We shall probably, however, continue for a long time to speak of these conditions as separate and distinct, but it is evident, as time goes on and our knowledge of the diseases and of blood-development increases, the identity of many of them will be acknowledged, and we shall find that here, as so often the case in natural history, the multiplication of species has been the result of imperfect information, and that as points of resemblance in essential characters and development are studied minor differences disappear."

The characters of the blood of a new-born child.—The appearance of the blood of a new-born child as it issues from the capillaries is almost that of venous blood; this condition continues until about twelve days after birth.

The red cells are much more unequal in size than are those seen in the blood of a person after adolescence, the larger being larger and the smaller smaller than in the adult; their intimate composition, however, differs very slightly from those of an adult. The corpuscles osmose and deform more rapidly when in contact with reagents or with water, the small cells notably being transformed into spherical globules.

The number of red cells contained in a cubic millimetre is almost as great immediately after birth as in the most vigorous adult, and is nearly always greater in number than that found in the blood of the mother. Out of seventeen children whom Hayem examined the largest was six million two hundred and sixty-two thousand, and the smallest four million three hundred and forty thousand.

The result shown by these numbers appears to be influenced by the manner in which the cord is tied. Out of six children who had the cord tied immediately, the average number was five million eighty-seven thousand. Of eight children in whom the cord was not tied until the umbilical artery had ceased pulsating the average was five million five hundred and seventy-

six thousnad, a difference of four hundred and eighty-nine thousand in favor of the latter. This difference persisted more or less for forty-eight hours, though at this time it was not greater than four hundred and thirty-two thousand.

The coloring-matter, judged by the chromometric examination, is about the same in a child as in the adult.

At the moment of birth the same variety of white cells exist as in the adult.

Frequently the cells are a little smaller, the smallest being most abundant.

(Hæmatoblasts also exist in the blood of the infant, as we have stated earlier in this chapter.) During the first two or three days the number of white cells is greater than in the adult; the average of Hayem's count for the forty-eight first hours after birth were eighteen thousand white cells to a cubic millimetre, whereas in adults they are five thousand.

After birth the blood of the child shows some modification. During the period which corresponds to diminution of weight in the child, the number of red and white cells remains stationary or gradually increases until the child arrives at its minimum weight; that is usually in about three days. There is then a sudden and abrupt diminution in the number of white cells: from eighteen thousand it descends to six or four thousand; and an elevation in the number of red cells, which at this time generally reach their maximum.

Henry presents the following counts:

Case I.—Healthy girl; weight, eight and a half pounds; blood venous-looking two and a half hours after birth; number of red cells, six million four hundred and ten thousand per cubic centimetre; second count, twenty-four hours later, five million eight hundred and ten thousand; third count, forty-eight hours after last, five million six hundred and eighty thousand.

Case II.—Male; weight, eight pounds; first count, twenty-four hours after birth, five million nine hundred and twenty-five thousand per centimetre; second count, twenty-four hours later, five million five hundred and twenty thousand; third count, forty-eight hours later, four million eight hundred and seventy thousand.

CASE III.—William, born November 6, 5 A.M.; weight, six and a half pounds; November 7, four million five hundred and twenty thousand; white to red, one to nine hundred and four; second count, November 8, 11 A.M., red cells, five million three hundred and thirty-five thousand; white to red, one to seven hundred and eleven.

CASE IV.—Sela, female, born November 17, 8.15 A.M.; weight, seven pounds; rapid, normal labor; healthy child; November 19, 10.30 A.M., five million one hundred and eighty-five thousand; white to red, one to three hundred and fifty; second count, November 21, 4 P.M., number of red, five million four hundred and ninety-five thousand; white to red, one to six hundred and twenty-eight.

The diminution in number of white cells is a constant phenomenon, but in some children the minimum number of these cells is not reached until twenty-four hours after the body-weight has reached its minimum.

The rate of increase in the number of red cells is variable, from one hundred thousand to six hundred thousand.

The increase of the red globules does not depend altogether upon the loss of serum, but is due to the production of new elements.

At the time when the child begins to gain weight the number of white cells increases a little, usually averaging from seven thousand to nine thousand per cubic millimetre.

The number of red cells is less than in the adult, and in the course of the second week there is usually a difference of about half a millimetre from the first count (above recorded).

The changes in the histological character of the blood and in the diameters of the cells differ much day by day, and it is in this particular that the child's blood differs from the adult.

For a normal child these differences at the end of the third day are totally independent of body-weight; they appear to be due to the formation of new elements,—that is to say, the number of cells is in inverse ratio to the size of the cells.

As we see then the blood of the new-born presents certain characteristics, together with certain cellular forms, which belong peculiarly to itself; the daily fluctuations which we have noted above are due to the process of evolution. The

*specific gravity** of the blood is highest at birth, 1.066. It is lowest between the ages of ten weeks and two years,—1.048 in boys and 1.050 in girls.

CHLOROSIS.

Chlorosis is a form of anæmia presenting certain special features which will necessitate its separate consideration. It is pre-eminently a disease of the female sex, most usually associated with the period of puberty, or, at all events, with disturbance in the menstrual function. Cases are recorded as occurring among young children.

The predisposing factors seem to be deficient personal and public hygiene, poor food, and overwork, and those who have to do much stair-climbing.† On the other hand, girls surrounded by all the luxuries that wealth and refinement can provide are by no means exempt. In these cases the want of proper exercise and a healthy occupation, together with pernicious literature and sometimes masturbation, seem to be potent causes in producing the complete symptoms. Our own experience would lead us to believe that this is frequently a family idiosyncrasy. In some patients the neurotic element of the disorder is so marked and prominent that writers have sometimes regarded the disease throughout as a neurosis.

Symptoms.—The peculiar complexion and the symptoms of profound anæmia constitute the most prominent manifestations of the malady. The complexion is perhaps the most striking feature: its peculiar yellow-greenish tinge has caused the disease to be designated the "green sickness;" as in most (idio-

* Three years ago (1884), Professor Charles S. Poy communicated to an English physiological society a method of determining the specific gravity of the blood by the use of only a few drops of that liquid. In the *Journal of Physiology* (February, 1887), E. Lloyd Jones, of the Cambridge Pathological Laboratory (*New York Medical Journal*, April 9, 1887, p. 409), further elaborates the subject. His observations extend to three hundred and sixty-two persons of all ages and both sexes. The ingestion of food, particularly if accompanied by much water, causes a fall. If alcohol is used there is a rise; sleep will have the same effect. Gentle exercise causes a fall, but prolonged exercise accompanied by perspiration will cause it to rise.

† Osler, ibid.

pathic) anæmic persons the subcutaneous fat is not diminished, so also do we find in this disorder that the contour of the body may be maintained, or in some cases there may be a superfluous deposit of fat. The digestion suffers early in the case; a depraved appetite is prone to be present; hysterical outbursts are by no means uncommon; the menses are deranged, and the anæmic condition is further evidenced by venous and cardiac murmurs, palpitation, and syncopal attacks or even coma; breathlessness is often a prominent symptom. In advanced cases the patients appear waxy, they are lethargic, listless or absent-minded.

Pathology.—The corpuscles are not reduced so much in number as they are in their contained amount of hæmoglobin. Investigations have definitely demonstrated the poverty of the separate corpuscles in hæmoglobin, a further change is noted in the varying size of the corpuscles. Large, giant red cells and microcytes are sometimes noted. The changes, however, are not as characteristic or as well marked as in idiopathic anæmia, but young, poorly-formed corpuscles, deficient in hæmoglobin, may be said to be the microscopic diagnostic feature of chlorosis. Hayem and Willcocks further add the fact that the average corpuscular diameter will be found to be lower than normal. The blood-serum is about normal in quality, except that the solids are slightly reduced in amount. As we have already stated when considering cardiac atrophy, Virchow has pointed out the fact that in chlorosis there may be a congenital deficiency in the size of the heart and the activity of the circulation: the heart and vessels may remain almost infantile in size. These alterations are not always present, nor are abnormities in the female sexual apparatus always present. Some, however, consider that these alterations in the heart and reproductive organs are the primary change which cause the blood-crasis. Others, however, do not grant this hypothesis, stating in rebuttal that children who have passed through an absolutely healthy childhood may develop chlorosis, and ultimately recover. These cases are within the experience of all clinicians.

The blood-making organs present no lesions that aid us in diagnosis.

Treatment.—Iron may here be considered almost a specific, hence some writers have endeavored to show that the etiology of the disease is to be sought in the gastro-intestinal tract, and that the failure of assimilating the traces of iron which are to be found in the every-day dietary, accounts for its absence in the corpuscles. Be this as it may, the fact remains that our main reliance is to be placed in iron. Osler has found Blaud's pill* the most efficacious, stating that under its use he has frequently noted a doubling of the number of the red corpuscles per cubic millimetre in a fortnight. Any preparation of iron that the practitioner may elect will prove beneficial. It does not make such a great difference which one is selected so long as a sufficient quantity is given.

The vegetable acids will often be of decided benefit. Indeed, one writer has gone so far as to say that the disease is due to an absence of hydrochloric acid in the gastric juice, considering that upon its presence depends the dissolving of the iron compounds of the food and their consequent assimilation by the tissues.

Diet and hygiene must receive careful attention and supervision.

PERNICIOUS ANÆMIA.†

This form of anæmia arises apparently idiopathically, at least we are unable to satisfactorily associate its origin with any definite and specific organic lesion.

Etiology.—We can no longer consider this as one of the rare diseases, as the current literature of the day contains many examples of pernicious anæmia; it is, on the whole, perhaps more frequent in Germany and Switzerland than it is in either England or America.

Those who are overworked, poorly nourished, and dwell among unhygienic surroundings, are on the whole most prone

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* ℞ Ferri sulph.,
Potass. carb. et tart., āā ℥ss;
Tragacanth., q. s.
M. et div. in pil. No. xcvi.

† Synonymes: *Perniziöse Anämie*, Quincke; *Progressive perniziöse Anämie*, Biermer; *Idiopathic anæmia*, Addison; *Essentielle Anämie*, Lebert; *Anémie progressive*, Lepine; *Essentielle Maligne*, or *Essentielle febrile Anämie*, Immermann; *anæmatosis*, Pepper.

to the disease; this, however, does not always pertain, as, for instance, the disease is not at all frequent among the poorer classes of Ireland, who are notoriously ill fed and housed. Osler's Montreal cases were among the upper or higher mechanic classes.

Age.—It is undoubtedly a fact that the adult is more prone to the disease than the child; however, cases do occur among children, as in Pye-Smith's selected table of one hundred and three cases we find six below the age of fifteen. Recently Schapiro has recorded in the Russian journal *Vratch** a case in a boy thirteen years old, Quincke† in a child eleven years old, and Kjellberg‡ in a child of only five years, being the youngest case on record.

Sex.—Excluding, as we must in the present treatise, all cases occurring in the parturient female, pernicious anæmia is more frequent in the male than in the female.

As etiological factors may be mentioned chronic diarrhœa or discharges, ulcers, loss of blood, mental worry, or fright.§

Gastric and intestinal disorders have by many been considered a direct cause of the blood-changes. As early as 1860 Flint endeavored to show the possible relation between pernicious anæmia and degenerative disease of the gastric tubules. Fenwick‖ more recently reported cases in which the two conditions were associated, as have also Henry and Osler;¶ and still more recently Kinnicutt** reports three cases, in which he considers as proven that extensive destruction of the secretory structures of the stomach may be regarded as causal in a certain number of cases of pernicious anæmia.

Runeberg, in 1886, suggested that pernicious anæmia may sometimes be due to the presence in the intestine of a tapeworm (the bothriocephalus latus), and Schapiro†† has very

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* Med. and Surg. Rept., Nov. 12, 1887, p. 656.

† Am. J. Med. Sc., Jan. 1885, p. 253.

‡ Nordiskt Medicinskt Arkiv., Bd. xvi. Hft. 13, quoted in Am. J. Med. Sci., January, 1885, p. 253.

§ Curtin, Med. Times, Phil., April 4, 1885.

‖ Lancet, Lond., vol. ii., 1877.

¶ Am. J. Med. Sc., April, 1886.

** Ibid., October, 1887, p. 419.

†† Ibid.

recently recorded an instance of genuine pernicious anæmia in a boy of thirteen years, who made a fair recovery after the expulsion of a number of segments of the bothriocephalus.

After having considered all these causes, we will still find a large number of cases that appear to arise spontaneously, and to which we can but apply the term idiopathic; indeed, one of the most marked characteristics of the disease is its development without assignable cause,—many modern writers do not consider the disease idiopathic, or pernicious anæmia, if they are able to definitely determine its cause in a given case. It seems to us that its etiology is to be determined perhaps by a careful study of the chemico-vital changes—if we may be allowed the term—of the blood, and it is not to the pathologist and microscopist that we are to look for a solution of the problem, but rather to the laboratory of the chemist and the physiologist.

Symptoms.—The blood, when drawn, is about the color of a light claret; it flows slowly, and sometimes it is difficult to procure a drop; hydræmia is not marked, as the corpuscles appear to fill the drop completely.

Broadbent says that the blood in these cases will stain the finger like nitric acid; some compare it to muscle-washings or weak coffee. The blood remains uncoagulated for a long time; its specific gravity may be as low as 1.030.

Microscopic examination will reveal certain alterations in the blood-cells which are by many considered pathognomonic.

Red corpuscles.—Size: A great variation may be noted, some markedly increased, constituting the large, giant forms, or megalocytes; others are normal in size; and a third variety, or small, round cells, the microcytes, are deeper in color than the other varieties, which may even be paler than normal.

Shape: Alterations in form and shape are in this disease most characteristic, and, according to Osler, this irregularity is never met with to the same extent in other conditions. These irregularities of form may be various, so that they have lost all resemblance to the normal red disk; their biconcave form has been replaced by flat, oval, or tailed varieties, or they may be elongated and rod-like. This change in shape may not extend to the entire corpuscle, as one end may be normal while

the other is pointed, tailed, or blunted. Balloon and reniform shapes have been noted.

But little tendency is shown by the corpuscles to form into well-marked rouleaux.

The large cells are often ovoid with sinuous margins. These changes in shape are believed by Mackern and Davy to be due to an abnormally soft stroma, by others to an altered state of the serum.

Nucleated red corpuscles are stated by Ehrlich to be present in all cases. Osler found them but in two cases, and then they were scanty.

Granular masses—Max Schultz's granule-masses—may or may not be present. Quincke and Leube have each recorded a case in which they were abundant. Osler could in some cases find but a trace of them, and in others they were less abundant than in health. These masses are composed of the hæmatoblasts or blood-plates; in cachexiæ and leukæmia they are sometimes very abundant. The white or colorless corpuscles may be increased relatively; usually, however, they are normal. In some cases they have been noted larger than normal, and somewhat less granular. Litten* has observed a case of pernicious anæmia pass into leukæmia. The amœboid movements were active in the cases observed by Osler, Pye-Smith, and Gardner.

After all, however, the most characteristic alteration in the blood is the great reduction in number of the red corpuscles. Considering the normal number per cubic millimetre to be five million, we may find under these circumstances but one million two hundred and fifty thousand, or even as few as five hundred thousand.† This reduction is even more extensive than that following a severe hemorrhage or the exhausting process of phthisis.

The reduction is not constant, but varies from time to time.

The hæmoglobin is markedly deficient, which accounts for the light and peculiar color of the blood; many believe that there is no proportion between the reduction of the corpuscles

* Berlin. Klin. Wochenschrift, 1877.

† Quincke reports a reduction of red corpuscles to but one hundred and forty-three thousand per c.m.

and their deficiency in hæmoglobin. Osler agrees with the statement of Laache, that the relative coloration of the corpuscles is increased. This seems as marked a feature in pernicious anæmia as the relative reduction is in chlorosis. From this fact we understand why the anæmia is never quite so intense as the number of corpuscles would appear to indicate.

Quinquand* states that there are one hundred and twenty-five grammes of hæmoglobin in one thousand granules of normal human blood, and considers that a reduction to 26.5 grammes, or one-fifth, would be invariably fatal.

General symptoms.—Again must the classical description of Addison be quoted: "It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness and breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless; the flabbiness of the solids increases; the appetite fails; extreme languor and faintness supervene; breathlessness and palpitation are produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles; the debility becomes extreme; the patient can no longer rise from his bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."†

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* Compt. Rend., xxvii., No. 6, quoted by Musser, Reference Handbook Med. Sci.

† Monograph on Diseases of Supra-renal Capsules, p. 3; Osler, Amer. Syst. Med.

As we see, then, the symptoms of pernicious anæmia are simply, as Coupland remarks, those of simple anæmia aggravated and intensified.

The complexion of these cases assumes a peculiar tint which must be seen to be appreciated, variously styled as a light lemon tint, a straw-yellow, or a grayish-yellow, or it may even assume the hue of a mild icterus.

Slight œdema may or may not be present; it is usually apparent about the feet and legs during the latter part of the case; cutaneous hemorrhages may appear on the legs and arms; epistaxis is not uncommon, and melæna may arise.

Examination of the eye-ground may detect retinal hemorrhages, but these changes in the eye are not particularly characteristic, as they are not different from those found in other diseases, for example, in the secondary anæmias and in Bright's disease. Optic neuritis and œdema of the disk and retina have been noted.

A distinct basal hæmic or anæmic murmur may be present with the venous hum, or *bruit de diable*, in the veins of the neck.

These hæmic murmurs have a variable distribution; sometimes it is with difficulty that we can decide their points of maximum intensity; occasionally we could almost conclude that there was a murmur at each valvular orifice; at our next examination all abnormal sounds may have disappeared to reappear again to-morrow, or upon exertion or change of position, as from the recumbent to the erect or *vice versa*. All the visible arteries may pulsate, and auscultation may reveal a systolic arterial murmur.

The *pulse* is usually rapid, soft, and compressible.*

Derangement of the gastro-intestinal system is a prominent and almost ever-present cause of complaint, accompanied by nausea and vomiting with almost complete anorexia.

Fever may or may not be present; the urine is pale and acid; hæmaturia rarely occurs. Cerebral symptoms are sometimes prominent.

Prognosis.—This is essentially unfavorable, most cases pur-

* Henry, 100 to 120; Osler, 80 to 100 or more.

suing a progressively downward course, to terminate, after a lapse of, at the most, two to three years, in death, usually by asthenia; however, undoubted evidences are presented by writers of cases of recovery from this disease.

Diagnosis.—We consider anæmia pernicious when the corpuscles have been reduced below two million per cubic millimetre, and present the alterations in size and shape that we have already described. The percentage of hæmoglobin is not reduced, or it even may be above normal. The blood presents *megalocytes* (large red corpuscles), *microcytes* (below the normal size), and *poikilocytes* (distorted cells).

Post-mortem appearances.—The blood-changes have already been described.

The heart is flabby, and has usually undergone fatty degeneration; normal hearts have, however, been recorded; the interior of the blood-vessels has undergone similar change. The gastric membrane is much affected (see *etiology*). The spleen is usually normal, as are the lymph-glands; nucleated red corpuscles have been noted in the spleen and lymph-glands.

The fatty tissue of the long bones may be replaced by a red marrow resembling the osseous system of the infant. These changes, however, are not peculiar to pernicious anæmia, and, as Henry states, are not primary. The sympathetic system is by some considered normal; others have remarked a fatty change, and an increase in the insterstitial tissues with pigmentation of the cells has been recorded.

Several instances of decrease in size of the supra-renals have been noticed.

Treatment.—Arsenic is undoubtedly our main reliance in combating this almost hopeless malady. Full and increasing doses of Fowler's solution may be administered with occasionally a favorable result. Iron is of secondary importance; in this disease it has seemingly lost its almost specific powers, which are so well shown in cases of secondary anæmia or in chlorosis.

Transfusion of blood or milk may be tried. Inhalation of oxygen has been recommended, but medicinal or therapeutic measures should be assisted to their utmost by careful hygienic and dietetic regulations.

LEUKÆMIA.*

An increased number of colorless corpuscles, associated with enlargement of the spleen, lymph-elements, and bone-marrow, constitutes an example of the disease leukæmia.

Changes in the hæmatopoëtic organs have caused writers to describe the disease under three headings, according as one or the other organ was affected, as splenic, lymphatic, and medullary or myelogenous leukæmia. Rarely, however, is but one of the organs affected alone; most often is the spleen affected, and with it the bone-marrow. Cases are recorded in which the thymus and thyroid bodies were affected, and others in which the tonsils and intestinal glands first presented abnormalities, and Kaposi† reports a case in which the lymphatic elements of the skin were first involved.

Etiology.—Age: The disease occurs at all ages. Trousseau and Mosler have recorded cases at fifteen and sixteen months, Osler one in a suckling of eight months, Goodhart, six cases under two years, and one of us in a child of four and a half years.‡

Sex.—Males are undoubtedly more liable to the disease than females.

Prolonged exposure to cold is said to be an exciting cause. Poverty, with all its attendant ills and worries, presents many predisposing factors. The hemorrhagic diathesis appears sometimes as a cause, notably in Howard's§ case of a boy whose mother and sister were bleeders, he having also suffered much from nose-bleed when a young child. Traumatism of the spleen and bones has been accorded a place in the etiology. Syphilis and malaria have also been attributed predisposing and exciting powers.

Symptoms.—The blood: As the blood is removed from the finger it is reddish-gray or brown, pale-red, grayish-red, or chocolate-brown ("milch chocolade"), according as it appears to the eyes of the various observers.

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* Synonymes: *Leucocythemia*, Bennett, Henry.

† Wiener Med. Jährbucher, 1885; Osler, ibid.

‡ Medical News, November 21, 1885.

§ Montreal General Hospital Reports, vol. i., 1880.

Great increase in the number of colorless corpuscles is at once apparent, until they may bear the proportion to red of one to twenty, or one to ten, or one to four. Rarely does it go below this, although it has been reported as low as one to two.*

The corpuscles are no longer the normal white cells, but they vary much in size. Their amœboid movements are slow and sluggish. Some of the cells bear a close resemblance to the marrow-cells, and Damon† has observed in the blood of a boy suffering from leukæmia certain crystals different from any before observed, to which he gave the name "Leucocrystallin;" the crystals seen in the sputum of asthmatic cases, so widely known as Lyden's‡ crystals, have also been observed in the blood of these patients.

The red corpuscles are but little affected. Microcytes and large corpuscles sometimes occur, but we do not see the many characteristic alterations that occur in pernicious anæmia.

General symptoms.—Insidious in onset, the disease rarely comes under our observation until the lesions are well advanced, the spleen enlarged, and the abdomen prominent, when we will probably note extreme shortness of breath, marked pallor, and possibly œdematous extremities; the patient will complain of dragging in the side, dizziness, and possibly nose-bleed, or, as in the boy reported by Howard, a sudden and fatal hæmatemesis, the same observer reporting the case of a girl in whom severe hemorrhage from the stomach was an early and an alarming symptom; in fact, gastric symptoms are rarely absent, not always, however, of such an alarming character as those just referred to. Diarrhœa is an almost invariable concomitant; melæna may occur.

The spleen particularly, and possibly the lymphatic glands, are enlarged, the liver may be and is most usually towards the close of the disease; ascites is usually present to a greater or less degree; it may either be due to pressure from the enlarged abdominal organs or to a general hydræmic condition of the blood. The profound anæmia may cause dizziness, syncope,

* Osler.

† Boylston Prize Essay on Leucocythæmia, 1864; Henry, ibid.

‡ Sometimes styled Charcot's crystals.

and possibly a mild delirium; headache is sometimes complained of. Osler has never noticed that the cases were sad or morose, as stated by some writers on the subject.

Hyperpyrexia is present at some time during the history of the case. The eye presents some alterations in the retina; hemorrhages even may occur; these hemorrhages are peculiar in that they are made up principally of white corpuscles, which on ophthalmoscopic examination present a yellowish-white color.

The præcordial region will usually reveal, on auscultation, hæmic murmurs which are so often heard in anæmic conditions; the pulse is quick, soft, and compressible.

The spleen.—Pain and distress in the splenic region is very often present; the organ may be markedly enlarged and occupy the larger portion of the abdominal cavity or even the pelvic; pressure from the spleen may cause interference in the venous return, or it may even cause intestinal obstruction; a murmur has been heard in the spleen, and Gerhardt has known one to pulsate.

The *lymph-glands* are not affected to the same extent as in Hodgkin's disease, nor do we often note them as causing severe pressure-symptoms. As the gland increases in size there may be local tenderness or pain; the case which we have reported (*ibid.*) presented marked enlargement and induration of the cervical glands. Perhaps on the whole lymphatic leukæmia is more frequent in children than in adults.

The *course of the disease* is usually slow and chronic; however, in children it is apt to be very rapid. Fortunately, this acute leukæmia is happily very rare; most cases succumb to a gradual asthenia; a sudden, fatal hemorrhage may arise at any time.

Treatment.—In the early stages, before profound alterations have occurred in the spleen or lymph-glands, and while the leucocytosis is but moderate in degree, some prospects of a cure may be entertained; later, however, we must disabuse our minds of such comforting thoughts and consider that the most we can do is to simply palliate symptoms.

Iron, arsenic, and quinine are, perhaps, the drugs from which we will derive the most satisfactory results; they must,

however, be administered in full doses and be continued for several months; improvement will sometimes occur totally independent of any and all form of treatment.

The symptoms to which we have called attention require their appropriate symptomatic treatment as they arise.

HODGKIN'S DISEASE.*

The majority of the cases of this disease occur among the young. Its characteristics are anæmia and hyperplasia of the lymph-glands, with secondary lymphatic growth in other parts of the body.

A proportionately larger number of males are affected in this disease than we noted in leukæmia; an hereditary predisposition has been claimed for the disease, and syphilis, bad food, exposure of all kinds, and local lymphatic irritation have been looked upon as predisposing factors.

The blood.—There is, of course, anæmia present, but the red corpuscles are not reduced in number to the same extent as in pernicious anæmia; they are usually uniform in size; nor does poikilocytosis exist to any marked extent; numerous microcytes have been noted in a few instances. Osler has not seen the nucleated corpuscle in any of his cases.

The white corpuscles are not very much increased, although a fair amount of leucocytosis may be present. Schultze's granules are present.

General symptoms.—The earliest symptoms are usually referable to the lymphatic glands, which in their incipiency present a painless enlargement, usually attracting the patient's attention more by their inconvenience than by actual pain. The cervical glands are generally first affected, then the axillary. Should the bronchial or mesenteric glands be first affected, and produce mechanical interference with the functions of the organism, great difficulty in correctly interpreting the case may arise. Writers mention examples of this disease in which the early symptoms were marked dyspnœa, œdema of the extremities, thoracic pains, or even in one case paraplegia.

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* Synonymes: *pseudo-leukæmia, adenoid disease, anæmia lymphatica,* and many others.

The anæmia seems to increase in proportion to the glandular hypertrophy. Hemorrhage is not so liable to occur as in leukæmia. The heart and circulation present the ordinary symptoms and murmurs depending upon anæmia, and are usually in proportion to its severity; these symptoms may be further increased by pressure of the glands upon the nerves.

In this disease fever is a marked and prominent symptom. Osler considers that an irregular hectic type, with morning remissions, is the most common; he has, however, noted ague-like paroxysms, with rigors, a hot and a sweating stage. During the height of the fever the glands may become much swollen. Respiratory symptoms of two varieties arise,—those dependent upon the anæmia, as breathlessness, and those depending upon pressure from the enlarged glands; even pleuritic effusion has thus been caused (Osler), due to pressure on the azygos and intercostal veins.

Dyspepsia, dysphagia, gastro-intestinal troubles, constipation, enlargement of the liver and spleen, jaundice, and ascites may all arise; the nervous system and the special senses become deranged; albumen may be present in the urine.

Prognosis.—This depends much upon the situation of the lymphatic enlargement and the rapidity with which it takes place. The disease may either prove rapidly fatal in from two to four months or it may extend over a period of years, death eventually taking place from asthenia; death may occur, however, at any time from pressure on the vital structures by the enlarged glands.

Treatment.—As we would expect from our experience in other blood-diseases, arsenic is of much benefit; it must, however, be given in full doses until its early toxic effects are noticeable, when the dose may be reduced but not discontinued. Under this plan of treatment we will occasionally have the satisfaction of seeing the glands become smaller and smaller; but rarely, however, will we meet with a complete recovery. Phosphorus, iron, quinine, cod-liver oil, and general tonics may be exhibited; local frictions or inunctions to the enlarged glands have but little if any effect; substances injected into these glands do not seem to have any appreciable effect.

If the glands are small, isolated, and accessible, they may be removed. Tracheotomy is sometimes demanded for occlusion of the glottis or trachea.

HÆMOPHILIA.*

This is peculiarly a congenital or hereditary abnormality, whose full recognition and accurate description is due to the acumen of American physicians. While most cases present undoubted evidences of heredity, still the disease may arise in a child whose ancestors present not a trace of the disorder. Most of these children succumb to the disease; some, however, have lived, and in turn procreated bleeders.† Males are more prone to be affected than females.

Osler‡ presents many cases, and an interesting family genealogy showing the strong hereditary tendency of the disease.

Age.—Grandidier§ has shown that the large proportion of cases occur from the first year to the end of the second year; rarely does the first bleeding occur after the twelfth year, and in only one case has it occurred after the fifteenth year. Bleeders are seen under all conditions of life, although climate seems to have some effect in determining the disease; most cases are seen in cold, damp localities, still warm, equable climates present a few illustrations of hæmophilia. Families who are the subject of this disease are apt to be very prolific.

Symptoms.—Marked symptoms appear during the early years of life; in fact, the condition is usually well established before the fifth year.

Osler agrees with Legg and Grandidier in that the symptoms may be grouped under three divisions: external bleedings, spontaneous and traumatic; interstitial bleedings, petechiæ, and ecchymoses; and the joint-affections. The latter observer cites the following localities which were affected in the order named: epistaxis; from the mouth, stomach, bowels, urethra, lungs, skin of head, tongue and finger-tips,

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* Synonymes: *Hereditary hemorrhage, hemorrhagic diathesis, hæmatophilia, Blutterkrankheit, idiosyncrasia hemorrhagica.*

† Bleeder is the term applied to one who is the subject of hæmophilia.

‡ Ibid., p. 933.

§ Die Hämophilie, Leipzig, Zweite Auflage, 1877.

tear-papilla, female generative organs, external ear, cerebral hemorrhage, navel (long healed). A case is also recorded in which a child bled to death from the scrotum.

Severe and even fatal hemorrhage may result from traumatism or from the most trivial operations, as, for instance, the extraction of a tooth, leeching, blister, or vaccination. The bleeding in these cases is always simply a capillary oozing, which may last for hours, days, or weeks.

The interstitial hemorrhages—hæmatoma, petechiæ, etc.—may either arise spontaneously or be the result of trauma; the extremities are usually affected, less commonly the mucous and serous membranes.

Joint-affections are commonly met with; they may present simply slight pain or intense inflammation and redness, resembling rheumatism. The large joints are generally affected, particularly the knees.

Treatment.—When hemorrhage occurs the ordinary means for its arrest must be tried, both locally and internally; ergot and iron have been of service.

During the absence of bleeding all our measures should be directed to prevent its recurrence; the patients must be guarded against trauma or the most trivial operations; teeth should not be extracted. Females of a bleeder family should not be allowed marriage, as some of their male children will inevitably be affected. A suitable warm and equable climate should be selected for winter residence.

THROMBOSIS AND EMBOLISM.

The two great generators of blood-clots—the paraglobulin is contained mainly in the white corpuscles and the fibrinogenous material found in solution in the plasma—uniting are acted upon by a fibrin ferment, possibly contained in the white corpuscles, and cause clotting, or the formation of fibrin, to take place; but this requires another element, in many cases equally important,—namely, an abnormal condition of the endothelium of the blood-vessels. According to Weigert the death of the white corpuscle is essential, and according to the experiments of others the destruction of the red corpuscle is equally important. Indeed, the investigations of Virchow have shown

that the disease of the blood-vessel is not essential in the formation of a clot; in all probability destruction of the blood-cells with stagnation of the circulation are to be looked upon as the essential elements. This probably accounts for the fact that we see so many very diseased hearts capable of sustaining life and permitting blood which is healthy to pass through them, and again heart-clots, or infarcta, forming in acute disease in childhood when the endothelium is normal but when stagnation has taken place in the circulation and the blood itself disorganized.

What is understood as thrombosis is a clot formed in a vessel and remaining *in situ*. When it becomes detached and enters the current it becomes an embolus, though under the latter definition would come any foreign body that is carried through the circulation. Of course the effects of such plugging of the vessels or stoppage of circulation are purely mechanical, but infectious emboli may carry disease to distant parts of the body.

In children's practice the question of thrombosis and embolism becomes an extremely interesting and important one; we find embolism resulting from thrombosis in the peripheral veins, such as the renal, the umbilical, the diploic, but more especially of the veins in the more dependent part of the body, as the femoral, the internal saphena, or the cerebral sinus, and lateral sinuses, owing to the tendency of clot-formation, on account of the *vis a tergo* and thoracic aspiration, but we may also have autochthonous clots in the pulmonary artery from pressure of enlarged glands of the neck or bronchial glands. We may also have clots formed from stasis of the circulation due to mitral disease of long standing.

Thrombosis may occur also in diseases of the cytogenetic organs, as in lymphadenoma, leucothythæmia, and we may also have it in typhoid fever, diphtheria, pyæmia, and certain surgical cases; indeed, in all forms of febrile disease where great destruction of corpuscles has taken place blood-clotting is imminent, and the seat of the thrombus will be mainly dependent upon local causes. The lodging of an embolus, whether venous or arterial, will be purely a matter of accident. Venous emboli may be carried from any of the venous trunks

into the right heart and lodged in the pulmonary artery or in its ramifications; the symptomatology will be mainly dependent upon the mechanical obstruction caused by this.

The division made by Beverley Robinson is eminently correct,—into the sudden fatal, the grave, the benign. In the first form the main trunk or both divisions are suddenly obstructed. Respiration becomes rapid, and exaggerated attempts are made at breathing. There is no obstruction to the entrance of air, but the fault lies with the supply of venous blood, which cannot reach the radicles of the pulmonary artery, and the current is dammed back upon the right heart and venous system. There is intense dyspnœa, pallor of face, rapid and irregular heart-action; there is difficult oxygenation, and death is the result of asphyxia. But where the embolus is lodged in the right heart, sudden stoppage to the circulation may take place and a fatal syncope result.

In the second form the secondary division of the pulmonary artery is plugged, the same series of symptoms, still with the suddenness, take place. We have the gasping respiration, deep and frequent, absence of all evidence of pulmonary disease, no dulness on percussion, no râles, but a rough respiratory murmur; there may be a venous pulse through tricuspid regurgitation; there is gradual cyanosis. Death gradually occurs, and probably preceding it, sanguinolent sputa, and local physical signs of infarcta exist. Some suppose that if the branches of the pulmonary artery are only partially plugged, permitting the current of blood to surround the embolus, reabsorption may take place.

In the third division minute infarcta occur in the minor ramifications of the pulmonary artery. These are apt to be carried to the right and left lungs, into the median and lower lobes, and as these emboli soften in the centre and disintegrate, they may be absorbed, and few symptoms may be the result. When these radicals of the pulmonary artery are obstructed no collateral circulation can be carried on through the bronchial artery, as the circulation between the two is absolutely independent; the bronchial arteries are those of nutrition; the pulmonary artery keeps up the function of the lung.

Of course it is readily recognized that some forms of pulmon-

ary embolism, depending greatly at which point the embolus lodges, may give rise to pulmonary consolidation, or extension to the pleura may take place, with effusion or plastic deposit, which may be either unilateral or bilateral.

The importance of emboli as carriers of septic matters should always be recognized; in this way tubercular infection may be carried, or hydatids in embryo may be localized in the pulmonary tissue.* From what we have seen, pulmonary thrombosis is by no means a rare occurrence, and the only means of differential diagnosis between pulmonary embolism and thrombosis during life are the suddenness of the symptoms in the former case, with the pre-existence of disease, to which could be attributed the formation of a thrombus, and in the latter case to the evidence of a more gradual plugging of the pulmonary artery.

CARDIAC THROMBOSIS.

Blood-clotting, which may fill the heart with coagula, is brought about by so many causes that naturally it has led to much discussion in medical literature. The same condition that will produce clots elsewhere will have more or less tendency to cause a coagulum to form in the cavity of the heart, independent of any lesion affecting the endocardium itself. The anatomical arrangement of the cardiac cavity is such as to intercept the column of blood which flows through it. An embolus carried from a distant venous trunk and lodged in the reticulated surface of the cavity of the right heart will often become a predisposing cause to thrombosis, as much so as would a roughened endocardium or diseased blood checked in its course by cardiac weakness; the slow precipitation of coagula is by no means uncommon in the febrile affections of childhood, and especially those diseases which are accompanied by disorganization of blood, with increase in the fibrogenetic elements and corpuscular death. The appearance of

* An extremely interesting case has just occurred in Dr. Osler's wards in the University Hospital. A boy, ætat. 13, with mitral disease and its many concomitants, presented on the post-mortem table a pneumothorax of peculiar origin. A hemorrhagic infarct had ruptured through the periphery of the lung and torn through the visceral pleura, forming a lung fistula and greatly distending the pleural sac with air.

such a clot, whitened by the precipitation of its fibrin and the washing out of its coloring-matter, its close adherence to the papillary muscles, its firm seat in the net-work, and the prolongation of its tendrils in the direction of the blood-current, has given rise to the theory of a polypoid degeneration, the formation of concretions due to inflammation of the lining membrane of the heart.

To Virchow, who followed in the footsteps of Van Sweeten, we are indebted for a clear understanding of the causation of infarcta, which may have formed in any part of the circulation, either arterial or venous, carried into the venous or arterial trunks through the heart, and lodged in the narrower vessels of the arterial or venous system. But we also know that the same result will follow a clot formed in the heart itself; through detachment of a calcareous valve, dilatation of the heart, pericarditis, roughened walls, a similar result may be brought about; but it requires more than this simple slowing of the circulation. Disease of the heart itself will not always produce cardiac thrombosis; a change must occur in the character of the blood itself to bring this about. This latter has been termed the vital or pathological cause, and is not dependent entirely upon the death-agony. In proof of this we have the statements of Harley in the early febrile stage of scarlet fever. We find it in certain inflammatory diseases, we have noted it ourselves frequently in measles, and it is one of the causes of early death in diphtheria. In rheumatism, of course, we have, as a frequent complication, endocarditis, which aids in its formation by adding a roughened endocardium.

The *symptoms* depend greatly upon the rapidity of formation, also naturally upon the size of the clot. They are usually brought about by the presence of the foreign body within the cardiac cavity and the obstruction to the circulation. They are more or less dependent upon the strain thrown upon the heart, and are frequently greatly exaggerated by excitement, by the febrile condition, sudden movements, or displacements of the heart by pressure from existing pericarditis, or large amounts of gas in the stomach. When they occur in the right heart, as they most usually do, the sounds in the left heart are unchanged, while those of the right become distant and muffled.

Some authors note a soft or possibly a rough and harsh bruit in the præcordial region, which may be transmitted into the aorta, with a doubling of the first sound; but this, indeed, is a difficult matter upon which to base a diagnosis. We should take into consideration the irregularity and intermittency of the cardiac action,—the word tumultuous seems to describe it better. In connection with abnormal heart-sounds, the rapidity of onset, a modification of the normal sounds by which they become muffled and indistinct, all complicating some disease, which we recognize as predisposing to cardiac thrombi, are sufficient for a diagnosis. Should the right heart become engorged, as it naturally will do, percussion-dulness will be marked over an extended area. There will be turgescence in the venous trunks, hyper-resonant pulmonary percussion, and the symptoms before noted, showing the plugging of the pulmonary artery. There would be a sudden and marked asphyxia in the first instance, and a more gradual evidence of venous stasis in the latter.

To those who have witnessed at the bedside the sudden plugging of the pulmonary artery, with its frightful consequences, no description is necessary to aid them in forming a diagnosis. The same sequence of symptoms, dispossessed to a certain extent of the horrors of suddenness of onset, will follow the formation of a cardiac thrombus as a complication of acute disease.

Prognosis.—The question is often asked after a cardiac thrombus has formed, Can reabsorption take place? and though it seems almost impossible theoretically that such a fortunate result can be attained, reports of cases prove that occasionally a clot has formed sufficiently large to produce a series of symptoms that make the diagnosis positive. Post-mortem examinations and clinical experience have shown that small clots out of the way of the direct circulation may exist without marked disturbance, and concretions form and absorption take place, or disintegration result in emboli which may cause pyæmic symptoms or disturbances in distant organs. Of course we must bear in mind the distinction between the cadaveric clot, those occurring during the death-agony, and those that are ante-mortem; to the latter we have

called especial attention. As a complication of acute disease of childhood the question of heart-clots becomes a matter of importance. Attention has been called to it* by one of us in diphtheria, scarlet fever, and measles, and at that time the thrombus was attributed to an increase in fibrin due to the rapid tissue-change, the importance of pushing an alkaline treatment from the start was urged, and again in reporting the death of a number of cases of malignant measles.† The mode of death was peculiar. The fatal signs came on suddenly and with frightful intensity: the gasping breathing, the frantic efforts to obtain air, the imploring look, with consciousness not impaired but seemingly unduly acute, until the final convulsion or gradual cyanosis brought the end.

The turgid veins, venous engorgement, the feeble pulse, the fluttering heart, pointed unmistakably to but one cause,—the gradual-forming right-sided heart-clot; and the post-mortem appearances as demonstrated at the section gave us a large, tough, chicken-fat clot obstructing the venous circulation, firmly planted in the right heart and its tributaries, too often exhibited to be disputed. One of the earliest symptoms of this impending danger was undue rapidity of respiration. The child seemed to be doing well, but its eruption irregular, probably incomplete, or dark and mottled and in blotches, when attention would be called to the great rapidity of respiration, with a peculiar, gasping inspiration, fish-like in character. The other fatal symptoms would follow rapidly, and within twelve hours the child, despite carbonate of ammonia, warm baths, digitalis, etc., would die of heart-clot.‡ In some cases pneumonia was absent; where it existed the fatal issue might have been brought about by stasis in the circulation. The immense number of micrococci found in the blood was looked upon as a possible cause in the determination of the clot; but the fact remains the same, whether we adopt this view or not,

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* Journal of American Sciences, January, 1882.

† Philadelphia Medical Times, August 12, 1882.

‡ The blood-clotting and manner of closure of the ductus arteriosus and hypogastric artery is fully described and illustrated by J. Collins Warren, "The Healing of Arteries," etc. New York: William Wood & Co., 1886, pp. 111–137.

that in all of the diseases of childhood that are accompanied by a febrile state with blood-disorganization, where the pathological and mechanical elements are combined, cardiac thrombosis of the left heart deserves serious consideration.

ADDENDA.

I. See page 33.

Cardiac ectropion—fissure of the sternum—is among the recorded anomalies of the thoracic walls which become of interest to us in our present studies. Martin* records an instance occurring in a female child who presented at birth an almost complete absence—or, at all events, a marked fissure—of the sternum. There was a breach of about two inches in continuity of the bony thoracic wall, in the space usually occupied by the sternum, in its whole extent. The space was covered only by soft structures, which flapped inward and outward with each respiratory act. The heart lay within this space, and could be readily grasped through its thin covering. There was a similar deficiency in the abdominal wall, the recti abdominis being separated or non-developed. There was also an umbilical hernia. The child was then four and a half months old. The anomaly of itself rarely proves fatal; most cases reach advanced adult life. Our friend Dr. G. E. de Schweinitz has noted the condition in a male adult.

II. See page 49.

Osler† states that "there is no known disease in which endocarditis is so constantly found *post-mortem* as chorea;" it is exceptional to find the heart healthy. He doubts that even acute articular rheumatism, which is so prone to endocardial complications, can be compared to chorea in this respect.

III. See page 74.

The percussion outline is much altered when an effusion arises; the area of dulness is markedly increased. By some it is said to assume a pyramidal or pear shape with the base downward;‡ others state that the effusion always increases the lateral area of dulness and produces

* N. Y. Med. Rec., Sept. 24, 1887.

† "The Cardiac Relations of Chorea," Am. J. Med. Sc., Oct., 1887, p. 375.

‡ Da Costa, Flint, Sibson, Bauer, and others.

an absolute flatness in the 5th right intercostal space, at from two to three centimetres from the right edge of the sternum.*

IV. See page 74.

Rotch, however, concludes from experiments upon the pericardium of sixteen infants and four adults that the apex remains in its normal position, and that the higher impingement noted in these cases is not due to the apex, but to the tumultuous action of the side of the heart.

V. See page 101.

Latent pericarditis—sometimes styled *idiopathic*—is occasionally met with in the young. For instance, Sturges† records the following case: A boy æt. seven suddenly fell down dead after running a short distance; child had always enjoyed good health, and there was no history of any previous illness. *Post-mortem* showed marked pericardial adhesions, with some recent lymph, but no serum. No cause could be assigned as to the origin of the pericarditis. Da Costa‡ reports another case, in an Italian boy aged fourteen, remarking that it was a case of purely idiopathic pericarditis, and that he could recall other cases from hospital and private practice.

VI. See page 135.

Sansom§ relates the case of a lad who consulted him solely for the symptoms of incontinence of urine, and in whom well-marked aortic disease, with hypertrophy of the left ventricle, was found to be present.

VII. See page 145.

Children show a wonderful immunity from the toxic action of arsenic; more particularly is this seen in the anæmia of heart disease. Consequently, the dose may be relatively very large. Some recent observations on this subject have been made by Osler.‖

VIII. See page 152.

Hutchinson¶ has observed an aneurism of the arch of the aorta in a girl aged four years, who died, after ten days' illness, of acute peri-

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* Rotch, Trans. Mass. Med. Soc., 1878.

† Lancet, London, July, 1885, p. 153.

‡ Med. Times, Phila., Dec. 15, 1887, p. 161.

§ Lancet, London, Aug. 13, 1877.

‖ Thera. Gaz., Nov. 15, 1886.

¶ Trans. Path. Soc., Lond., v. 104.

carditis; the sac hung from the arch of the aorta into the pericardium, somewhat compressing the pulmonary artery. Keen* has recorded ten cases of aneurism in girls between the ages of eight and eighteen years, together with several other cases which more properly come under the province of the surgeon.

IX. See page 152.

An aneurism of the *ductus arteriosus* has been observed by Billard and Thorè,† and Leullich‡ has observed a case in an infant ten weeks old, the aneurism measuring 15 mm. in diameter, filled with clots, and opened 3 cm. farther down in the descending aorta. Martin§ has recorded an aneurism of the ductus arteriosus in a child a month old; it was the size of a small nut, filled with clots, and impervious.

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* Med. News, Phil., Dec. 24, 1887, p. 725.

† Archiv. gen. de Med., 1850.

‡ Arch. d. Heilkunde, 1876; quoted by Paul (ibid.).

§ Bull. Soc. Anat., 1827, ii. 17.

INDEX.

CLINICAL STUDIES

ON

THE PULSE IN CHILDHOOD.

A CAREFUL and systematic study of the conjoint action of the heart and arteries—the pulse—in childhood is as yet an uncultivated field in pediatric literature; that, however, it has not been wholly neglected is evidenced by the fact that as early as 1822, Mayor, of Geneva, endeavored to derive practical benefit from the already ascertained knowledge of the fœtal heart-beats, which he had accidentally discovered in 1818. Frankenhäuser, somewhat later, further tried to utilize this knowledge in diagnosing the sex of the fœtus by the rapidity of the heart-beats, a diagnostic guide which has recently received the sanction of Jacobi, who states that he was seldom mistaken in predicting the sex when based on Frankenhäuser's principle. It is necessary, however, to make the examination when the pulse is not disturbed by causes due to change in either the mother or the fœtus; the field opened by the earlier observers has not received much further attention, and to those engaged in the study of the maladies of infancy it is particularly noteworthy that, notwithstanding the importance which is attached to the pulse in adult life, its characteristics in infancy are almost unrecorded. Trousseau and Valleix have recorded their observations in relation to the pulse of the healthy infant, but, unfortunately, these gentlemen do not agree in their statistics.

The pulse in health.—To collect and collate a series of observations upon the pulse of the healthy infant from the moment of birth to the end of the first year is a task beset with difficulties; it is frequently most difficult to correctly

record the pulse for the first hour after birth; indeed, it is often impossible, for at least the first ten days of the infant's life, to count the pulse at the wrist at all in most cases, or locate the pulsations at the præcordia; indeed, Lederbérder was only able to count the radial pulsations in six infants during the first minute of life.

Immediately after birth the pulse becomes much less frequent than during fœtal life, when it ranges between 124 to 150 or more. Our own observations in the main agree with Smith and Jacobi, that within an hour after birth the heart's action becomes more regular and settles down to about an average beat of 136 per minute. Other observers do not agree with these figures. Valleix estimates the number of pulsations per minute, between ten days and twenty-one days, at 87; Trousseau places the figures for the first week of life as between 78 and 150, Gorham in the main agreeing with him. These figures, to us, seem too low, especially if we consider their disproportion to the fœtal beat, and, furthermore, the fact that the new-born babe's heart must now carry on its independent circulation and, per consequence, must find exacting demands upon its contractile power, so that we can but conclude that the higher figures are more nearly correct. During the first eighth- to quarter-minute after birth the heart pulsations are not discernible, then they commence slowly, so that by the first half-minute they are probably not more than 10 or 12 per minute, in the second half-minute a vigorous child will cry and the pulse will become rapidly accelerated, even as high as 160, to shortly settle down to between 136 and 140. Immediately after birth the pulsations are alone to be ascertained by placing the hand over the præcordia; as the child becomes a little older,—one week,—the femoral or carotid is reliable to estimate the pulse-beats, or we may observe the basilar through the open fontanelle. Jacobi states that the beats of the fontanelle or the carotid can be distinguished and counted easily, up to a frequency of 240 per minute. The following table is prepared in order to present the relative frequency of the pulse at different ages and under different conditions.

It will be noted that sleep has the most remarkable effect in reducing the number of pulse-beats; it will also be noted that

the figures in the main agree with Trousseau and Smith. The former states that the average pulse of the healthy infant between the first and second months is 137 per minute, from the third to the sixth month 128, and from the sixth to the twelfth month 120. (See Table, page 4.)

We also learn from these observations that the pulse is more rapid while awake,* particularly if sitting or standing, and that muscular exertion or mental excitement may cause the pulse to become as rapid as in disease. In feeble children this acceleration is more marked; as the child grows the pulse is much less susceptible to all these influences, and we find the child of six years with an average pulse of 100, which at thirteen has become reduced to 88, closely approximating the adult rate of 72.

The frequency of the child's pulse is, however, not its sole difference from that of the adult, but it has certain other characteristics which are worthy of study.

Irregularity.—The pulse of the young child is very apt to be irregular; this occurs whether the child is at rest, asleep, or undergoing active movements; hence the results furnished by an examination of the pulse do not offer any very definite characteristics and are not as pathognomonic as they are in the adult. With growth, this irregularity becomes less marked; it is, however, noticeable throughout the entire period of childhood. Conditions which will hardly perceptibly affect the pulse of the adult render the infant's pulse very irregular. Particularly will derangements of the digestive system, so common in infancy, show marked effect upon the pulse-rhythm. Constipation, diarrhœa, intestinal worms, dentition, meningitis, and anæmia all produce irregularity in the pulse-wave. Jacobi remarks that this irregularity becomes a puzzling factor in the differential diagnosis of incipient meningitis, with its pneumogastric irritation and anæmia.

Much may be learned by the graphic study of the pulse, notwithstanding the comparatively feeble beat, small volume,

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* Although Goodhart recently (third edition, London, September, 1888, p. 583) stated that in several cases Newnham noted it to be three or four beats quicker during sleep.

Infantile Pulse in Health—Asleep and Awake.

	AGE										
	First week.		From close of 1st week to close of 4th week.		From close of 4th week to close of 12th week.		From close of 12th week to close of 6th month.		From close of 6th month to close of 12th month.		Total.
	Awake but quiet.	Asleep.	Awake but quiet.	Asleep.	Awake but quiet.	Asleep.	Awake but quiet.	Asleep.	Awake but quiet.	Asleep.	
Number of observations	20	20	12	12	16	18	20	14	15	10	157
Extremes	100–150	106–138	126–156	108–140	110–150	100–132	110–140	106–118	114–142	108–120	
Mean	125	122	141	124	130	116	125	112	128	114	

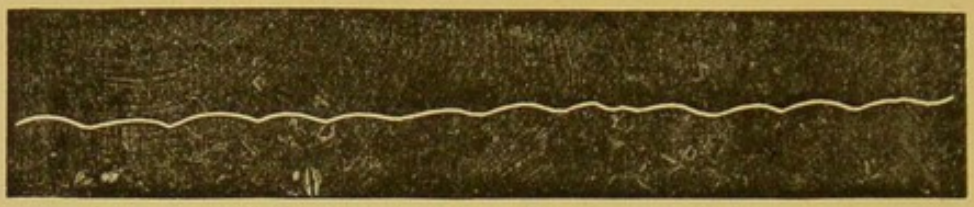
Pulse during Active Muscular Movement or Mental Excitement.

	AGE.					
	During 1st week. No child younger than two days.	From close of 1st week to close of 4th week.	From close of 4th week to close of 3d month.	From close of 3d month to close of 6th month.	From close of 6th month to close of 12th month.	Total.
Number of observations	5	10	6	9	4	34
Extremes	146–162	146–162	142–176	140–160	138–188	
Mean	154	154	159	150	163	

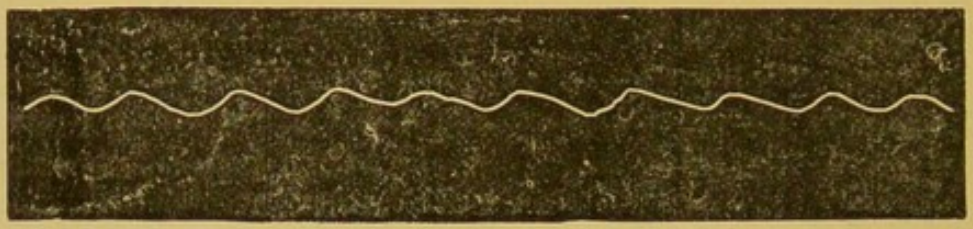
and less amplitude of the child's pulse; the cardiograph and sphygmograph present many interesting points worthy of study, although occasionally the sphygmograph will be very unsatisfactory. Goodhart goes so far as to state that with the sphygmograph he has met with little but disappointment in children. The most important peculiarity of the child's pulse revealed by tracings is that *dicrotism is absent*, and does not appear until the age of ten or fourteen years is reached. This peculiarity is well illustrated in the accompanying sphygmographic tracings taken from the thesis of Blache, which show these transitions in the pulse-wave and the age at which dicrotism commences to appear. In endeavoring to account for this absence of dicrotism we must remember that in a child there is not the same relation existing between blood-tension and arterial resistance; the child's pulse has not the same recoil,—that is, the expansion—the systolic wave—is more marked and perceptible than the contraction.

To Hofmann belongs the credit of establishing the fact that the blood-pressure in the newly-born animal is very small; for example, in the dog at birth it is but ninety millimetres, whereas in the grown dog it has risen to one hundred and sixty or one hundred and eighty millimetres. It has also been stated by way of explanation that the young child presents greater strength of the vessels to a relatively less strong heart, with a much shorter arterial circuit; for example, the common carotid in the newly-born is half the length of the descending aorta, but much less later in life, when the individual has attained its growth. Under these circumstances the blood is unable to distend the arteries sufficiently to allow a good recoil, hence the absence of dicrotism. In confirmation of the fact that the short arterial circuit is an element in reducing the dicrotic wave we have but to remember that Marey has shown that the longer the vessel the greater the dicrotism. We now know that dicrotism of the pulse is produced by the elasticity of the great vessels, especially when combined with low tension of the peripheral circulation and a sharp contraction of the heart, conditions which we also know do not exist in the young child. For example, it has been experimentally demonstrated that the adult kidney is much more permeable than that of the child;

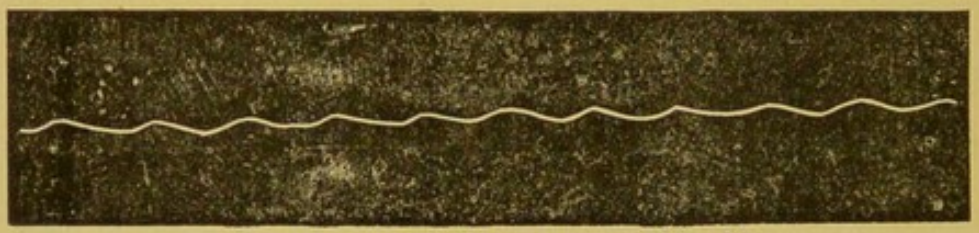
so we at once see that here is a decided interference to low tension of the peripheral circulation, as a marked resistance is offered to the arterial current.



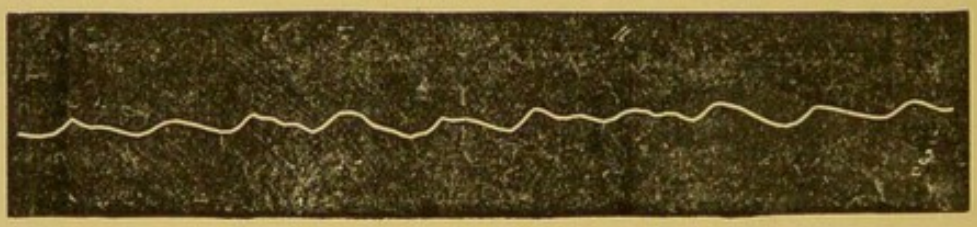
Normal pulse, child æt. three. Pulse 120.



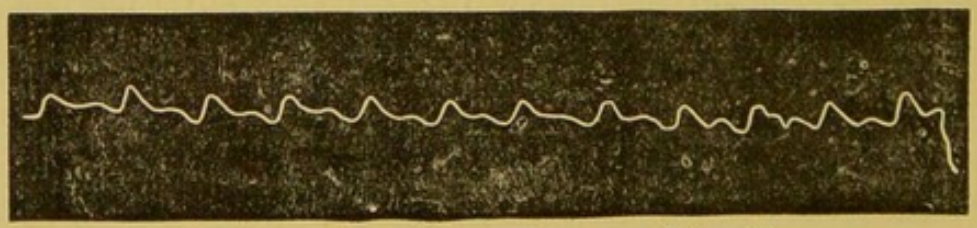
Normal pulse, child æt. four. Pulse 92.



Normal pulse, child æt. seven. Pulse 80.



Normal pulse, child æt. eleven. Pulse 90.



Normal pulse, child æt. fourteen. Pulse 88.

In some of the so-called diatheses the relation of heart to blood-vessels is much altered; in rickets we find large arteries and a normal-sized heart. Under these conditions blood-

pressure will be reduced—large arteries always have this effect—and low tension will be present.

Having now considered the two most marked characteristics of the pulse in early childhood—irregularity and absence of dicrotism—and thus established our text, we will now proceed to study the pulse in *health* and *disease*. In order to do this systematically we may adopt the classification of Broadbent, somewhat modified to suit the character of the cases under consideration.

1. *The number of beats per minute, their regularity and equality.*—This we have already fully considered in the opening pages of our paper.

2. *The size of the artery*,—an important consideration in childhood.—The size of the artery is very variable in different children. All things being equal, a large artery will present a more decided impulse, but at the same time it is more compressible; in a smaller artery compression frequently renders the pulse-wave much stronger. Undue smallness of the arteries is among the recorded abnormalities of the arterial system. Particular attention has been called to this condition by Virchow, to which we have already referred;* Meckel, Morgagni, Rokitansky, Lanceraux, and Bamberger record similar conditions. Cases of congenital smallness of the arteries often reach adult life; Gowers and Jacobi have so recorded them, and very recently Fraentzel† reports several cases of congenital narrowness of the aortic system, in two cases confirmed by autopsy.

The subjective symptoms are those of heart-disease, but the heart-sounds are clear, the second sound often accentuated, and the heart exhibits some dilatation. The symptoms are especially like those of cardiac overstrain, but develop in persons who have undergone no, or but slight, exercise. The hypertrophy of the heart begins in youth, is followed by dilatation, and finally by the signs of insufficient compensation. The arteries of the body are small, their tension high, and the face often strikingly pale.

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* "Diseases of Heart in Infancy," Keating and Edwards, 1888.

† Deutsche Medicin. Wochenschr., 589, 1888.

3. *Tension.*—A healthy pulse of average tension stands out during the systolic wave and subsides gradually during diastole; should the tension be decreased, it is only at the apex of the systolic wave that we are able to feel the pulse in a young child. On the other hand, should the tension be increased, the pulse-wave is almost as marked as in the adult.

4. *Character of the pulse.*—The character of the pulse cannot be better described than by quoting from two Greek physicians,* Rufus, who considered it small and yielding, no perceptible difference between systole and diastole, and Herophilus, who states that it is deprived of sense or rule or proportion; they also in a very realistic manner illustrated its characteristics by metric measurements. Those who desire to pursue this subject further will find Duremberg's "History of Medicine," Paris, 1879, i. p. 224, worthy of perusal, and will be convinced of the fact that we have made but few advances in the study of the character of the pulse in childhood.

5. *The strength of the pulse* in childhood is never a constant quantity until about the age of fourteen is reached; before that time it has not established its equilibrium and is easily affected by the most trivial departure from health,—even a slight accumulation of flatus, for example, will convert the perfectly normal pulse of the infant into a rapid-running pulse that at an older period of life would be indicative of grave disease.

We may, in an off-hand manner, estimate the strength of the pulse by placing several fingers on the radial artery and one or two nearer the heart, and thus estimate the degree of pressure that will be required to obliterate the radial pulsation.

6. *The state of the arterial walls.*—Insomuch that atheromatous arteries are rare in children, it is hardly necessary to study the pulse as affected by atheromatous changes in the arteries. When atheroma does occur in children it is apt to be localized in the cerebral vessels, and does not extend to the entire arterial system, consequently it cannot have the same effect on the pulse as it does in the adult. The extent to which the child's vessels

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* "Heart and Blood-Vessels in the Young," Jacobi; Brooklyn Medical Journal, March, 1888.

are liable to atheroma has been already fully elaborated by us, and space forbids a repetition.

The pulse in disease.—The pulse of the young is affected to a marked degree by the so-called functional disorders, and of these abnormalities in its action alterations in the rhythm are by far the most frequent. As we have before remarked, functional disorders of the heart's action, irrespective of inflammation or structural lesion of any kind whatever, constitute a frequent and an important class of cardiac diseases in the growing child.

Alterations in the rhythm.—A persistent frequency of the pulse is usually due to cardiac overstrain from continuous exertion; it is also a concomitant of neurasthenia and the abuse of certain articles, as tobacco, tea, coffee, or alcohol,—the so-called toxic cases. A fruitful source of palpitation or irritable heart in young boys is masturbation; persistent frequency is seen in anæmic and leukæmic cases, also in malarial poisoning. Alterations in the blood crasis, anæmia, leucocythæmia, melanæmia, and pernicious anæmia have as a constant attendant great irregularity of the heart and pulse; the younger the child the greater the irregularity.

Paroxysmal palpitation may occur and the pulse be very irregular,—from 75 to 200 within a few minutes. During the remissions of a paroxysm the heart may regain its normal rate. Position will exert a most decided influence upon the pulse-rate; while recumbent it will be much slower. Some of these cases present a peculiar flushing of the skin, due to vaso-motor change or innervation. Most typical examples of paroxysmal palpitation occur in Graves's disease and tachycardia: it is not at all unusual to meet a pulse-rate of 180 or 200, although the former condition rarely occurs in the young. We have recorded cases of exophthalmic goitre in young girls at puberty in whom paroxysmal palpitation was a distressing attendant manifestation of the disorder. We must, however, always bear in mind that a child may present extreme rapidity of the pulse and circulation, either constantly or paroxysmally, almost independently of organic disease, and that we must not attach too much importance to acceleration of the pulse-rate unassociated with conditions that in themselves merit careful study.

Neurotic influences often produce extreme palpitation of the heart, with a pulse-rate far above the normal; the pulse under these influences gives one the impression of but little onward movement of the blood; it seems more to vibrate than to pulsate. At a later period of life this condition is alarming, and sometimes is the precursor of death.

We do not find in children the same intense lividity and general capillary congestion of the face and extremities as we do in adults, but, on the contrary, intense pallor of the skin and mucous membrane exists; for a short time preceding death the pallor may become a light violet hue.

Palpitation in childhood sometimes produces angina pectoris, but it is not of the same variety as in the adult. It perhaps should be called pseudo-angina; but as the nervous system of the child is so easily impressed, we sometimes note the association of encephalic troubles and cardiac irregularity. We also note anæmia of the nerve-centres as a result of this irregularity, lowered arterial tension, engorgement of the venous circulation, sleeplessness, somnolence, sometimes torpor or even coma.

Infrequent, intermittent, and irregular pulse, reduplication or doubling of the heart-beats.—The former condition may be congenital. We have now under our care a lad, aged sixteen, whose normal pulse is but 40 to the minute; during an attack of typhoid fever one year ago, the highest pulse-rate recorded in this case was but 60. Individuals with infrequent pulse enjoy robust health; indeed, infrequent pulse is perfectly compatible with the most vigorous health Jaundice, renal disease, and some nervous disorders occasionally present an infrequent pulse. The literature presents cases of diminished frequency in which the children presented normal pulse-rates of 60, 40, and 32 per minute. Children who are the subjects of infrequent pulse are apt to present some form of cerebral disturbance; these may be of the nature of epileptiform or syncopal seizures or great mental excitability.

As we have before remarked, Flint calls attention to a curious form of functional disorder which would lead to the error of inferring infrequency of the heart's action from the pulse alone This condition is characterized by the regular alternation of a ventricular systole, giving rise to a radial pulse,

with one too feeble to be appreciated at the wrist. For example, Flint assumes the number of the ventricular systoles to be 70 per minute; in such a case the radial pulse would be 35 per minute. He has met with several cases of this disorder; the carotid pulse, however, accurately represents the heart's systole,—so that with auscultation we would note four sounds to each radial pulse. In this wise we may fall into the mistake of considering the case as one of reduplication of both the first and second sound. We have not as yet met such a case in young children, but have noted them in patients of eighteen years or over.

The *bigeminal pulse*—that is, a pulse in which the heart-beats and pulse-waves are in couples, a strong beat being followed by a weaker one—never, in our experience, occurs in childhood, although we have noted it several times in adults.

Dropped beat is rare except in association with organic valvular disease; its most frequent association is with mitral stenosis. Broadbent agrees with the now generally accepted statement that there is never want of synchronous action of the ventricles, but that in these cases of dropped beat the right heart contracts effectually in both beats, while the left heart succeeds in raising the aortic valves in the first beat only. One can readily appreciate the value of this additional right ventricular action in mitral stenosis.

Intermittent pulse does not occur as frequently in childhood as it does in later years, principally because its main cause—fatty degeneration of the heart-muscle—does not often arise during the early periods of life; however, we will sometimes meet an intermittent pulse in a child of apparently perfect health; it also occurs after exhausting diseases. This very day we have examined a child, aged ten, convalescing from typhoid fever, in whom the pulse is markedly intermittent.

Irregularity may almost be said to be one of the normal characteristics of the infant's pulse, giving place to the regular rhythm as the child grows. Mitral regurgitation, affections of the respiratory apparatus, as bronchitis, pertussis, and emphysema, all produce great irregularity of the pulse. Nervous influences play a marked rôle in its causation, as do also certain toxic agents, as tea, coffee, and tobacco. In conclusion, we

would draw attention to the fact that the irregularity of the pulse that accompanies mitral regurgitation is more marked than at any period of life, probably due to the fact that irregularity is so common at this early period of life.

THE PULSE IN VALVULAR DISEASE.

Mitral regurgitation.—Great irregularity of pulse is almost diagnostic.

Mitral stenosis.—Until heart-failure arises the pulse is small and regular, but as soon as compensation fails irregularity at the wrist arises. Although for a time the præcordial pulsations may continue to be regular, eventually extreme irregularity both in cardiac and arterial action arises, as Broadbent most aptly remarks; finally, the irregularity of the pulse and its lack of correspondence with the irregular heart defy description.

Aortic stenosis.—Should a child present this pulse in its typical form it is most characteristic, the wave is gradual in onset and long in duration, due to the narrowed condition of the valves; it is also small, and at once gives to the observer the impression of want of impact or strength. But rarely, however, do we meet this pulse in its purity, as aortic stenosis is so apt to be complicated by regurgitation at the same valve. We have elsewhere presented a case of pure aortic stenosis with pulse-tracing in a child, aged six, on whom an autopsy was held.

Aortic insufficiency.—If we exclude cases of congenital origin, primary regurgitation at the aortic valve is rare in childhood. Corrigan has made this pulse almost historic in medicine, and it is not necessary for us to dwell upon its characteristics now, particularly as we have fully elaborated its peculiarities in another publication.

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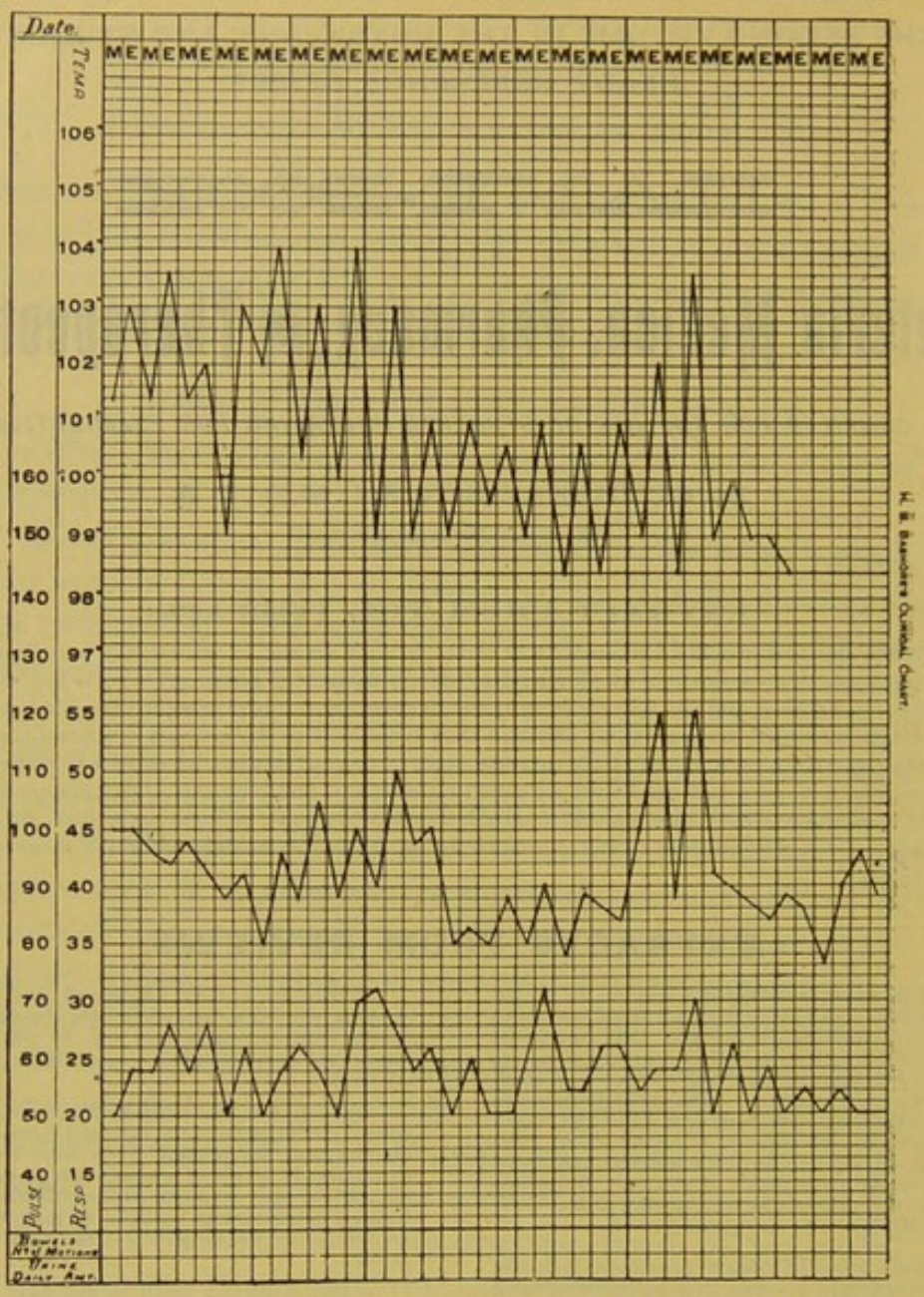

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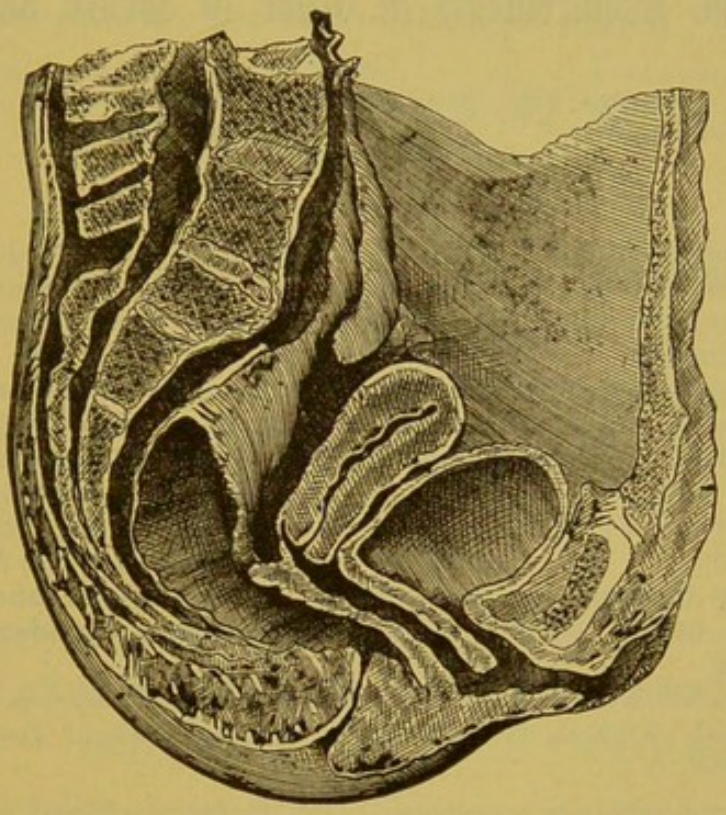
FIG. 44.



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(a) Aconitine.	Narcotic and Apyretic.	1-500 gr.	1-16 gr.

Following this, Preparations of the Pharmacopœia, each tabulated. For example:

TINCTURAL.

TINCTURA.	DRUG.	AMOUNT.	ALCOHOL.	DOSE.
* Aconiti.	{ Aconite. { Tartaric Acid, 60 † P.	5½ oz. to 24 gr.	100	1 to 3 drops.

* 60 Fineness of Powder as per U. S. P.
† P. Macerate 24 hours. Percolate, adding Menstruum to complete (1) pint tincture.

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oughly macerated and soaked in fluid, and from which they are forced into the œsophagus during rumination or into the honey-comb bag during the intervals of rumination. It is evident, therefore, that the food contained in this pouch may undergo changes due to the movements to which it is subjected, the temperature, and the action of saliva and other fluids. The changes are, therefore, physical and chemical. The walls of the rumen, by their contractions and resulting movements, may exert a considerable amount of mechanical force on the aliments contained within it, although this has been greatly exaggerated. Nothing like trituration takes place, but simply thorough mixing of the new and old food together

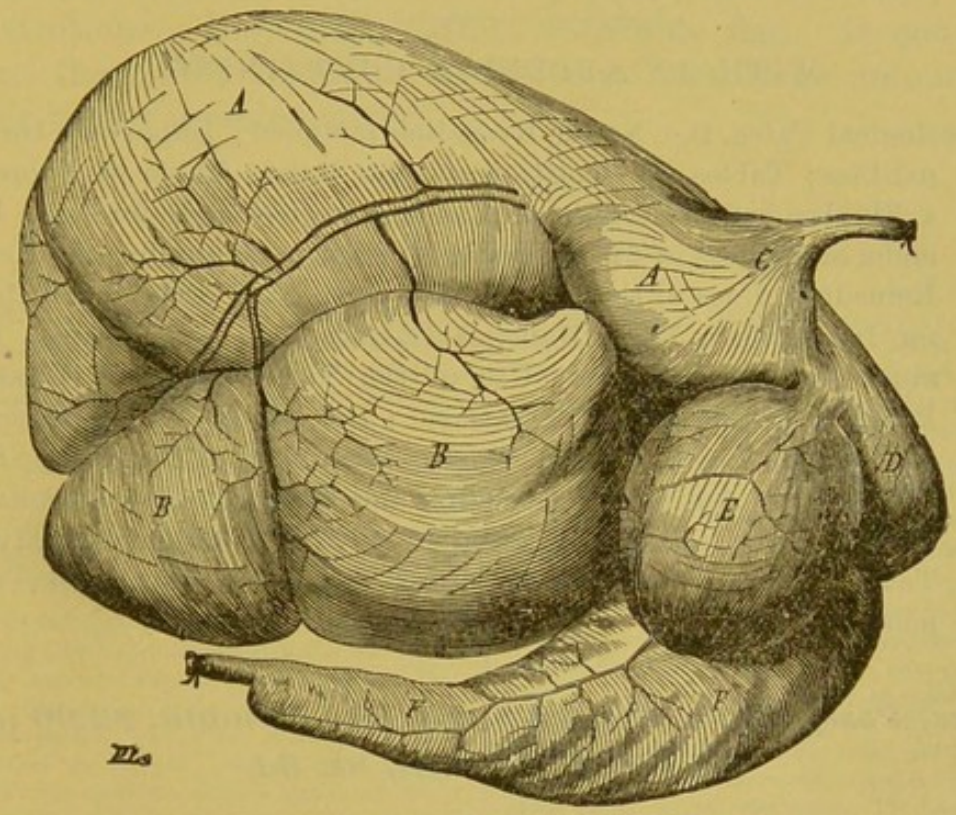

FIG. 153.—STOMACH OF THE OX. (*Colin.*)

A, rumen (left hemisphere); *B*, rumen (right hemisphere); *C*, insertion of the œsophagus; *D*, reticulum; *E*, omasum; *F*, abomasum.

and with fluid; consequently, it is not necessarily the portion of food which first enters the paunch which is the first to leave. The maceration which the food undergoes in the fluids of the paunch is especially marked in the case of grain and dry fodder, and is greatly assisted by the temperature of the organ.

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Following these will be found two sample pages from the body of the work, showing style of type and presswork.

GENERAL INDEX.

THERAPEUSIS.

AUTHORS QUOTED.

GENERAL INDEX.

THERAPEUSIS.

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attached by vessels to several small placentæ succenturiatæ. The clinical importance of multiple placentæ lies in the fact

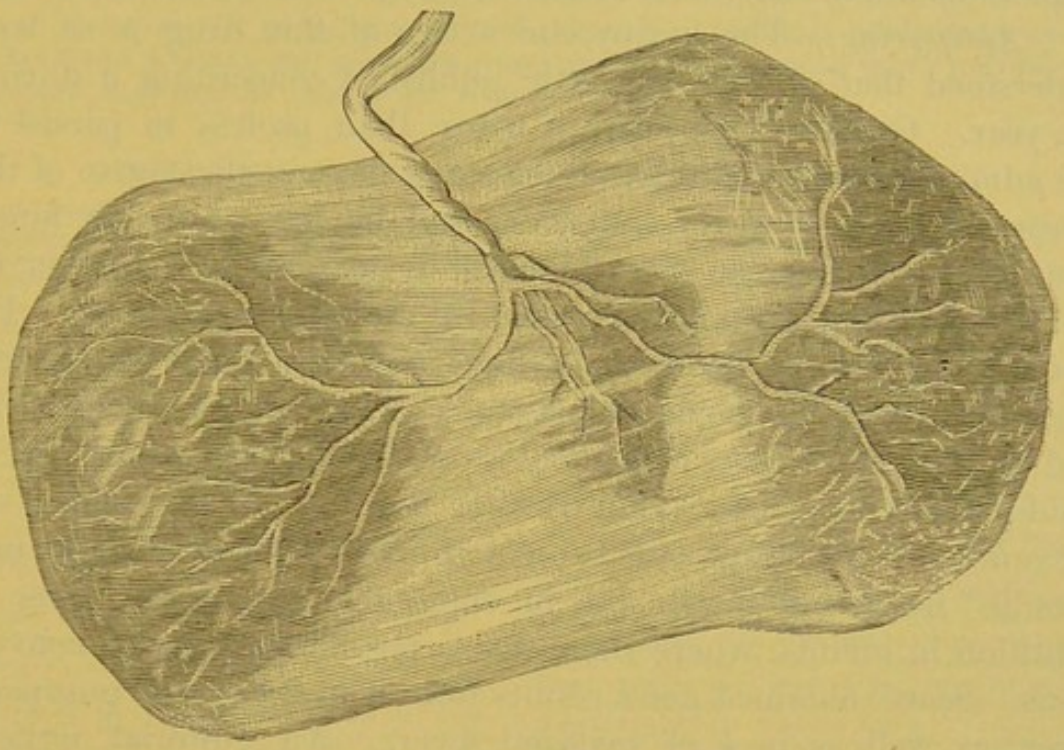

FIG. 2.—(*Annales de Gynécologie.*)

that one of these lobes may be retained in the uterus, while the other is expelled; and this accident is apt to occur if the attendant

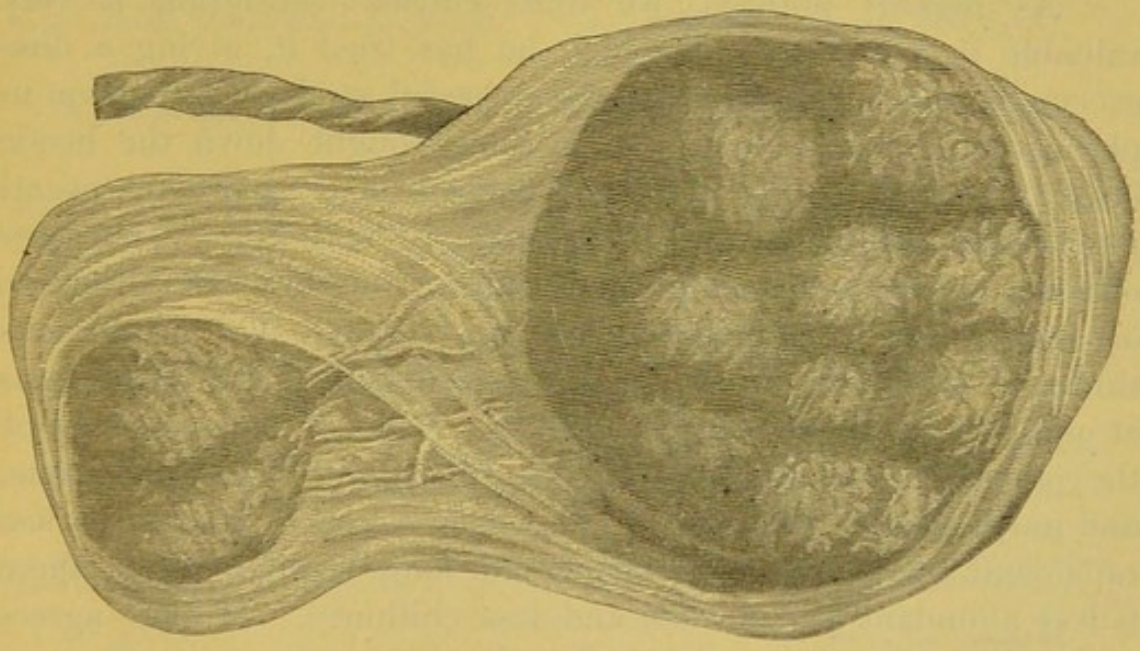

FIG. 3.—(*Annales de Gynécologie.*)

is in too great a hurry to remove the placenta. The practitioner should always carefully examine the placenta,—not only to see

under certain conditions; and it does not yet seem to have been satisfactorily demonstrated that the cyanosis is not due to some deleterious alteration of the blood.

Antipyrine.—The antipyretic action of this drug is so well understood that we find but little published concerning it during the year. Guitéras[38] considers it worse than useless to persist in the administration of antipyrine for many days in the course of the continued fevers of warm countries; and he has frequently found that its discontinuance was followed by a regular reduction of the temperature, with improvement of the other symptoms. He also noticed that the heart was weaker and the arterial tension less while antipyrine was being administered. Minot[38] gave antipyrine and thalline in 24 cases of typhoid fever, finding that 20–30 grs. of the former was needed. (For further notes of Minot's paper, see "*Thalline.*") Robison[39] failed to cut short typhoid fever by its use. Gradle[39] has found it valuable in doses of 4 grs. in the fever of dentition in infants, where there was a possible danger of convulsions. Scott[21] obtained good results from it in 2 cases of puerperal fever, as well as in 4 of malarial fever. An editorial writer[12] mentions the rapid reduction of a temperature 107.5° F. in a case of malarial fever, though he does not consider that the drug is any thing more than a simple antipyretic in this disease.

As regards phthisis, we find Patrick[39] declaring it very valuable in a few cases in which he has tried it, giving a dose every afternoon. Laache[35] made a special study of its action in phthisis, and found that it, as a rule, brought down the hectic fever very satisfactorily. Olikoff[35] confirms the reports in several journals concerning the hæmostatic action of antipyrine. In 6 cases of hæmoptysis he has employed a solution of 90 grs. in 6 ounces of water, used by inhalation, and has ordered 5–6 inspirations through the inhaler every ½–1 hour, diminishing the hæmoptysis at once, and soon arresting it entirely. Snyers[22] has made trial of the comparative value of antipyrine, antifebrine, thalline and kairine, and much prefers the former. The defervescence, he says, is less rapid, and a subsequent rise slower and more regular; hence there is less abundant perspiration and less chilliness. Müller[22] agrees with this statement, but claims that the slower rise after antipyrine does not always take place, and is sometimes even more abrupt than after antifebrine.

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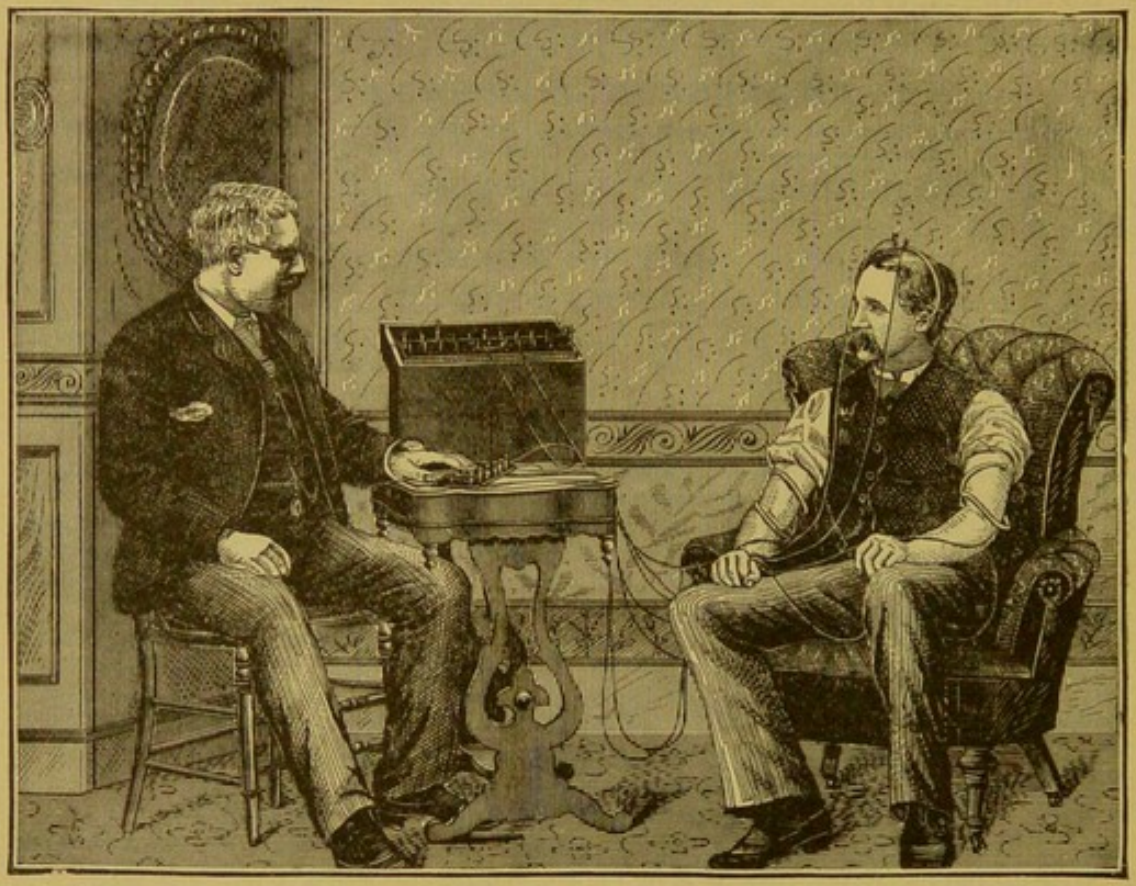

FIG. 61.—THE AUTHOR'S DIAGNOSTIC KEY-BOARD AS APPLIED IN ACTUAL USE.—The spring electrodes are represented in the cut (for the purpose of illustration) as applied to the facial, ulnar, and musculo-spiral nerves of each side. If he so chooses, the operator can have his case-book on a stand at his right, for recording his observations as they are made.

supplied by it should be tested afterward. The strength of the current employed should be ascertained by throwing a galvanometer into the circuit (when extreme accuracy is desired); by so doing, a comparison of the nerve- and muscle-reactions of the two sides can be based upon conditions which are exactly alike.

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"A notable and worthy feature of the work is a number of colored plates, illustrating the appearance of various stages and conditions of throat disease. We do not know of a more thorough, careful, or reliable work on the subject."—*California Medical Journal.*

"The drawings from life are excellent, and their reproduction by the chromo-lithographic process leaves nothing to be desired. The work is especially adapted to the needs of the general practitioner, and, without giving a detail of its merits, it is one which we can commend without reservation."—*Medical Age.*

"It is intended to furnish the general practitioner not only with a guide for the treatment of diseases of the nose and throat, but also to place before him a representation of the normal and diseased parts as they would appear to him were they seen in the living subject. As a guide to the treatment of the nose and throat, we can cordially recommend this work."—*Boston Medical and Surgical Journal.*

"This is one of the most excellent works we have seen, treating of the important and troublesome diseases of the upper air-passages, and makes a most excellent guide for the general practitioner in the treatment of diseases of the nose and throat."—*Southern Practitioner.*

The above represent but a small proportion of the testimonials received from journals from all parts of the world.

The next page is a specimen from the work itself.

Of the three acids mentioned, chromic acid is by far the most satisfactory for posterior applications. Nitric acid is not sufficiently safe, while glacial acetic acid requires too many applications.

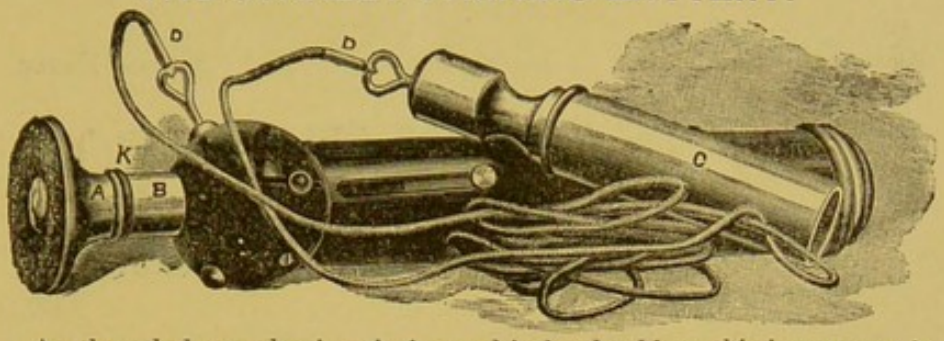
When an application is to be made, the instrument is adjusted so that the curved tip will take the proper direction on emerging, and the end of the rod is protruded. The tip is heated slightly to the fire of a match, and dipped among the crystals of the acid, then allowed to re-enter the tube. Enough of chromic acid will have adhered to the rod for the application. The tube being passed through the nasal cavity as far as the hypertrophy, the rhinoscope, held with the left hand, is placed in position, and the parts are illuminated. The location of the tube being ascertained, its point is placed against the side of the growth, and the spring is pressed upon. This forces the acid-covered point to emerge, the bend causing it to apply itself against the growth. By now drawing the instrument out a short distance, the application can be made more effective, the point thus parting with all its acid on the hypertrophied membrane as it rubs against it. The pressure on the spring being then released, the point disappears in the tube, and the instrument can be withdrawn. A solution of bicarbonate of soda, used posteriorly with the atomizer, is always indicated after this operation, to neutralize any excess of the acid that might have remained on the membrane, and to limit absorption. Four or five applications of this kind generally cause marked shrinkage of a moderate-sized growth.

Galvano-cautery can also be used in the same manner by introducing the cautery knife *d* (Fig. 33) instead of the acid application. The loop is introduced cold and applied against the side of the growth. Its position being ascertained with the rhinoscope, the circuit is closed, the handle being at

SURGICAL INSTRUMENTS,

TRUSSES, ETC.

THE STANLEY FARADIC BATTERY.



As the whole mechanism is incased in hard rubber, this instrument is much less liable to get out of order than the ordinary battery; in fact, it will, with ordinary care, last a lifetime.

The galvanic cell being tightly closed by a rubber stopper, there is no leakage, and, consequently, no danger of soiling the hands or clothing.

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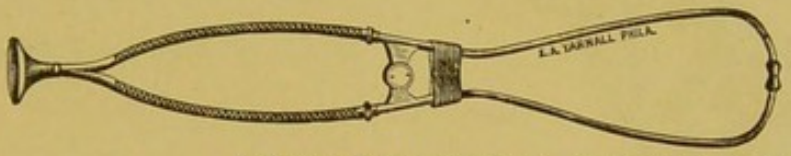
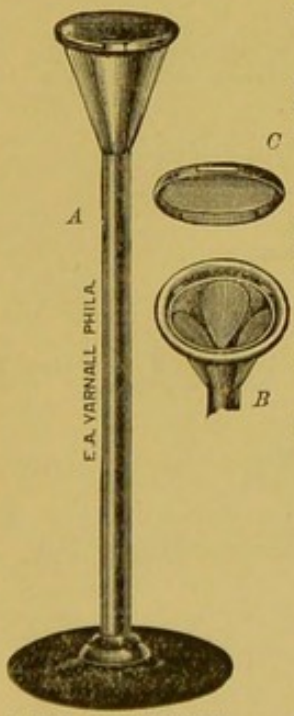
THE "UNIVERSITY" STETHOSCOPE.





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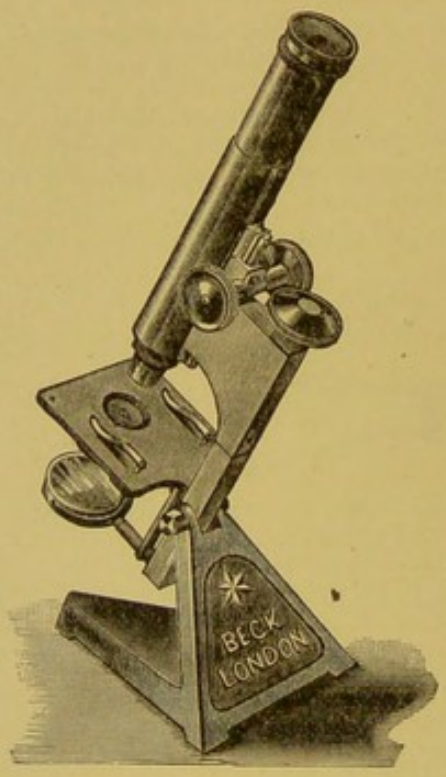

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