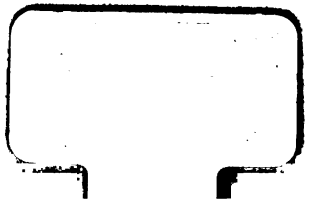
DIAGNOSIS OF CARDIO-VASCULAR DISEASES

BERGER



DIAGNOSIS
OF
CARDIO-VASCULAR DISEASES

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PREFACE

THE contents of this brochure represents a condensed **"epitomized"** treatise on the subjects of the principal diseases of the heart and blood vessels. The text is simple, concise and destitute of ambiguity. It is, however, resplendent with good common sense, true clinical facts and useful drawings: —all of which considered together forms a very useful, handy and authentic reference on the subject.

Not only will the practitioner of medicine find this brochure to be a valuable aid in **Diagnosis,** but the life insurance examiner will also appreciate its worth after an hour's reading. The subject on blood-pressure is remarkably interesting, both from a diagnostic and from a financial standpoint (in life insurance companies). Theory is banished from this work and it may be verily said admirably suitable for **"practical purposes."**

It is the sincere hope that this little monograph will attract the attention of the entire medical fraternity, and that they will appreciate the efforts of the Sultan Drug Co. in their manifest desires to co-operate with the medical profession towards medical progress. This complimentary contribution to medical literature represents an outlay of a considerable sum of money, exhaustive clinical study and references from the leading authorities on the subject.

HENRY I. BERGER, M. D.

Indianapolis, Ind.

ANATOMICAL REMINDERS IN THE DIAGNOSIS OF THE DISEASES OF THE HEART

1. The **pericardium** is a closed sac capable of being distended by fluid. The average quantity of fluid that can be contained within the sac is one pint. The displacement of the heart in pericardial effusion is directly due to the distensibility of the sac and also to the extent of its attachment to the base of the heart and the great vessels.

2. The **position of the heart** changes with position of the patient, except in cases where the pericardium is involved by adhesions.

3. The heart being attached to the central tendon of the diaphragm, causes the heart to go lower with each inspiratory (downward) movement of the diaphragm. On standing, the heart is also on a lower level. It can be easily proven by a fluoroscopic examination.

4. The **size of the heart** is determined by percussion. We recognize "superficial dulness" as the dulness obtained by percussion over that portion of the heart not covered by lung, while percussion over that portion of the heart covered by lung gives the "deep cardiac dulness." We use the combined areas in mapping out the heart.

(a) The **apex** normally lies in the fifth left intercostal space, three inches to the left of the median line and one half inch internal to the nipple line, and only in an area about the size of a fifty cent piece (if heart is normal). The apex may be displaced in many directions. For differential diagnosis of apical displacements, see page 49.

(b) The **right side of the heart:** from a point one half inch from the right sternal border on the upper end of the third costal cartilage draw a line convex to the right to the sixth right costal cartilage.

(c) The **left side of the heart:** from a point one inch from the left sternal border, a little above the third rib, draw a line convex to the left to the apex.

(d) The **lower border of the heart:** from the sixth right costal cartilage draw a line crossing the base of the xiphoid cartilage and thence to the apex.

5. The **heart valves** are located within an ellipse drawn from the upper left corner of the cardiac area to the lower right corner; the order of arrangement being from above downward: P-A-M-T. The initials refer to the pulmonary, aortic, mitral and

tricuspid valves respectively. The **areas of maximum intensity** of **valvular murmurs** are NOT over the anatomical position of the valves. These areas seem to be one in each corner of the cardiac area. See the schematic representation on page 8.

6. The **coronary arteries:** These vessels supply the heart muscle, hence, in cases where a limited amount of blood reaches the heart muscle, as for example, when the coronary arteries are narrowed and hardened by sclerotic changes, we must then expect pathological changes to develop in the myocardium. These are chiefly manifested by **degenerative processes** (see pages 35-36) and by a frequent clinical syndrome called **Angina Pectoris.**

Another important anatomical fact in connection with the nutrient arteries of the heart is the fact that these arteries arise from the Sinuses of Valsalva which are located at the very beginning of the aorta, hence it can be readily seen that inflammatory conditions in the aorta (an aortitis) may readily extend into the open coronary vessels. An aortitis may therefore secondarily involve both the semilunar valves and the coronary vessels.

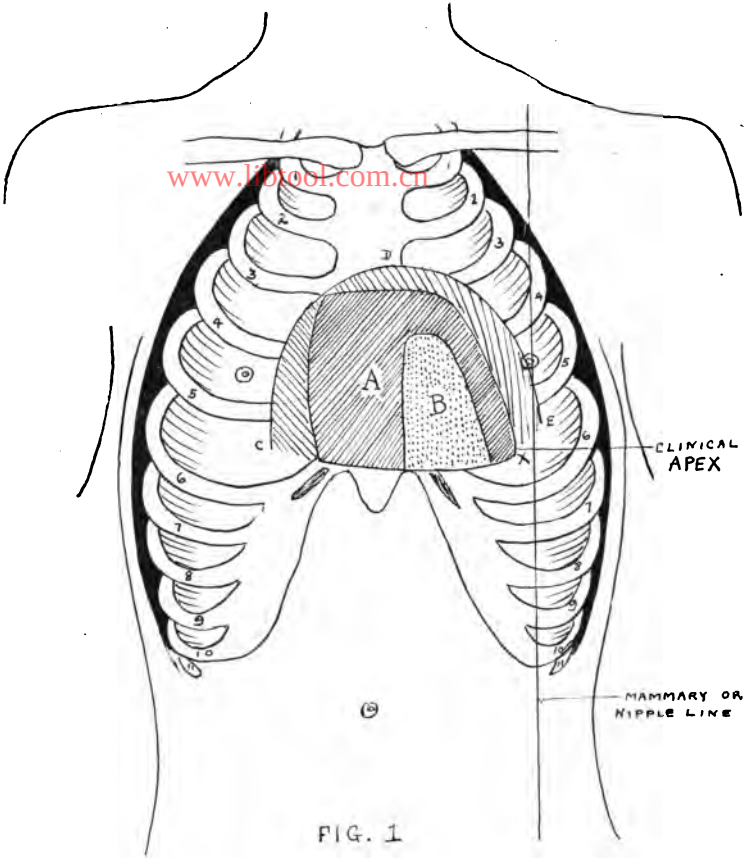

FIG. 1

TOPOGRAPHY OF HEART IN A NORMAL CHEST

A—Deep cardiac dulness.
B—Superficial cardiac dulness.
C D E—area of dulness in a child 7 years old.
X— apex in adult. In children up to 10 years it is in the 4th interspace. Note that the apex is about ½ inch internal to nipple line.
Note: the cardiac dulness below merges into liver dulness.

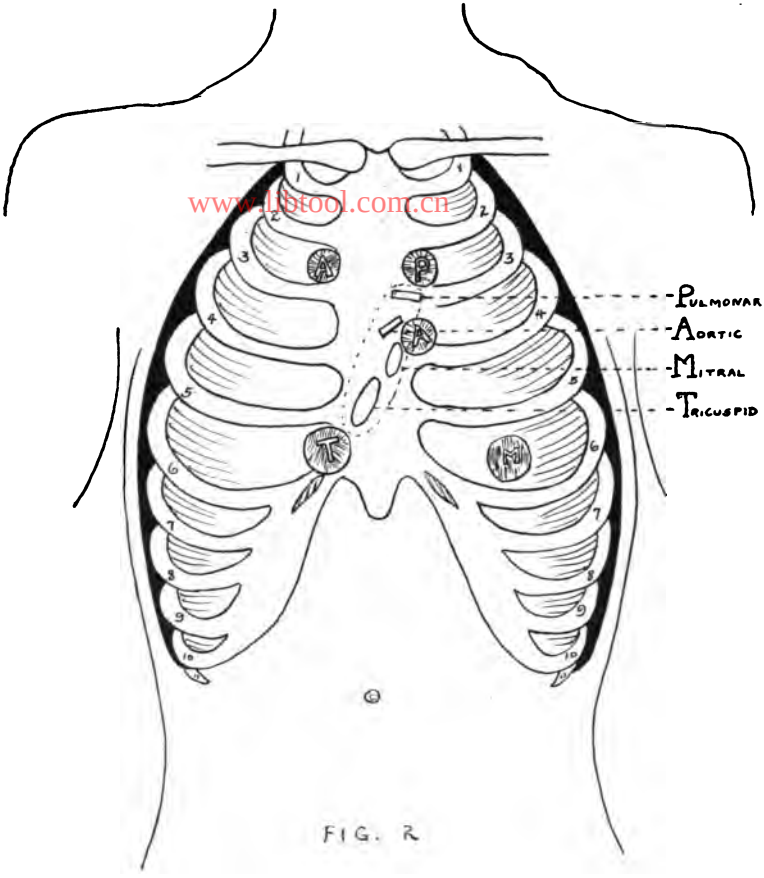

FIG. R

NORMAL POSITION OF HEART VALVES
WITHIN THE DOTTED ELLIPSE
AND
THE AREAS OF MAXIMUM INTENSITY
IN VALVULAR LESIONS
WITHIN THE SHADED CIRCLES

Upper (A) = Aortic stenosis.
Lower (A) = Aortic regurgitation.
(schematic)

PERICARDITIS

Definition: An inflammation of the protective sac-like covering of the heart, in which sac, a variable amount and a variable kind of fluid may occur and a small percentage of which may undergo organization with the formation of adhesions.

Clinical Classification:

1. **Acute Dry Plastic Pericarditis.** (It may become chronic.)
2. **Pericarditis with Effusion:** (Either acute or chronic.)
   (a) Sero-fibrinous effusion.
   (b) Purulent.
   (c) Hemorrhagic:
   This latter form is rare and usually occurs in cancer, tuberculosis, pernicious anemia, and in scurvy.
3. **Adherent Pericarditis:** (Always a chronic process.)

Etiology:

1. Rheumatism, in all ages, furnishes the largest percentage of cases. (Usually the subacute and chronic forms only.)
2. Septic processes anywhere in the body, as **erysipelas,** acute necrosis of bone, and including the diseases of childhood, as scarlet fever and diphtheria. The septic processes usually give rise to a purulent pericarditis.
3. Pneumonia, especially a left-sided pneumonia.
4. Extension of contiguous inflammation:—from spinal column, pleura, heart, œsophagus, mediastinum, etc.
5. Injury to the precordial area, especially those caused by penetrating pointed instruments.
6. As a terminal event in chronic diseases:—kidney disease, diabetes, cancer, arteriosclerosis, etc.

(A) ACUTE DRY PLASTIC PERICARDITIS

Clinical History:

1. The disease sets in with severe **pain.** The characteristic features of the pain are:
   (a) Always limited to the precordium or around xiphoid.
   (b) Is increased by full inspiration.
   (c) Seldom lasts longer than three days for the reason that at least one ounce of fluid is secreted into the pericardial sac, lubricating the walls

and thus the pain disappears entirely or else is greatly ameliorated.

2. **Dysphagia:** Painful swallowing may occur in this form but it is never as conspicuous as in the sero-fibrinous variety.
3. The **constitutional symptoms** are "as a rule" **mild.** The higher the temperature the greater the constitutional disturbances.
4. The only reliable **physical sign** is the **pericardial friction rub.** It presents the following characteristics:
   (a) It is never heard beyond the precordial area.
   (b) It is very superficial and not very intense.
   (c) It disappears on deep pressure with the stethoscope.
   (d) Is intensified by deep inspiration and upright posture.
   (e) Its time with reference to the cardiac sounds is not fixed or definite. It may be systolic, diastolic or to-and-fro.
   (f) Its disappearing acts: that is to say, even after having been once discovered the friction rub may be absent at the next examination.
   (g) It is best heard at the base in the third and fourth left intercostal spaces and corresponding half of the sternum.
5. The tendency is to recovery and resolution. Rheumatic cases recover in 7 to 10 days. Some cases may become chronic.

DIFFERENTIAL DIAGNOSIS

PERICARDIAL FRICTION RUB	ENDOCARDIAL MURMUR
Time: Has no relation to heart sounds. May be single or double. May change daily. May disappear and reappear.	It has a fixed relation to cardiac cycle, i. e., presystolic, systolic or diastolic. It never disappears completely.
Quality: Soft and superficial; i. e., lacking in intensity.	Deep and distant.
Pressure: Slight pressure intensifies. Firm continued pressure obliterates the friction.	No effect.
Change in position: Upright posture intensifies.	As a rule no effect.
Full breath: Deep inspiration intensifies.	No effect.
Transmission: Never transmitted beyond the precordial area. It is heard best at the base.	Transmitted in definite directions beyond the borders of the heart. They have fixed points of maximum intensity and are accompanied by changes in the heart, as well as in the valves.
In the average case the heart is never dilated. Chronic adhesive pericarditis may lead to dilatation.	

(B) PERICARDITIS WITH EFFUSION

Clinical History:

1. It begins as a dry pericarditis. If it develops insidiously it denotes an underlying chronic process and the prognosis is not very favorable.
2. In addition to the features of the dry form, we have **"mechanical symptoms" due to pressure** of the fluid in the distended pericardial sac. They are:—
   - (a) **Dyspnea:** due to interference with heart action and to compression of adjacent left lung.
   - (b) **Cough:** due to pressure on trachea and bronchi.
   - (c) **Dysphagia:** due to pressure on œsophagus.
   - (d) **Venous congestion:** pressure on large veins causes damming back of the blood, therefore there is headache (sometimes delirium), and flushed face, etc.
   - (e) **Aphonia:** due to pressure on the recurrent laryngeal nerve as it winds around the arch of the aorta. It is, however, more characteristic of mediastinal tumor and of thoracic aneurism.

(f) **Enlarged liver, ascites** and **edema:** occurs in long standing cases.

(g) **Muffling of the heart sounds** while the radial pulse has good qualities.

3. The **Constitutional Symptoms** are variable:

If the effusion is purulent we get signs of septic poisoning: i. e., chills, septic fever, sweat, anemia and exhaustion.

They are more marked in this form than in the dry form and both varieties are dependent upon the underlying cause.

"Walking cases" are also occasionally found.

Physical Signs:

Inspection: Bulging precordium and epigastric fullness. As fluid increases patient must sit up.

Palpation: Apex cannot be definitely located. Left lobe of liver felt depressed in epigastrium.

Percussion: Increased dulness in the width with its broadest base below. The dulness is therefore pear-shaped. Upper limit of dulness changes with position.

Auscultation:

1. Muffled and distant first sounds.
2. If effusion is very large the second sound may disappear.
3. The first sound with its decreased intensity is heard with maximum intensity in the fourth interspace.
4. We infer therefore that the heart is displaced upward and to the left.
5. Signs of compression of adjacent left lung which gives signs of pneumonic consolidation.
6. "Pulsus Paradoxus:" A difference of the pulse with inspiration and expiration.

DIFFERENTIAL DIAGNOSIS

PERICARDIAL EFFUSION	DILATATION
Inspection: Apex not visible.	Diffuse apex beat.
Palpation: Failure to locate apex.	Diffuse precordial wave.
Percussion: Largest area of dulness below. Adjacent left lung tympanitic. The borders of the dull area are sharply delineated.	Largest area of dulness above. No tympany, hence no sharp demarcation between pulmonic resonance and heart dulness.
Auscultation: Heart sounds distant and muffled. First sound decreased in intensity. Second sound may be absent. Apex displaced upward and to the left and is within the dull area. Signs of consolidation at adjacent left lung.	Heart sounds clear and valvular. If apex is displaced it will be downward. No signs of bronchial breathing in portion of lung touching heart on left side.

(See also page 20.)

(C) ADHESIVE PERICARDITIS

Clinical History:

Note: This form of pericarditis is more apt to be the sequel to pericarditis with effusion (especially the purulent variety) than to a dry plastic pericarditis.

It is essentially a **chronic process** and the onset is very obscure. The usual symptoms are:—

1. **Dyspnea:** patient cannot exert himself without causing shortness of breath. It becomes progressively worse.
2. **Undermining of health.**
3. **Constant cough** without much expectoration.
4. **Cyanosis:** especially marked if adhesions are over the right ventricle.
5. Precordial pain very rare. However, **anginal attacks** occur in many cases.
6. Patients may live 10-15 years without other serious manifestations. However, sooner or later, hypertrophy and dilatation must ensue (q. v., pages 17-18).

Physical Signs:

Inspection: Broadbent's Sign: i. e., systolic retraction of the intercostal spaces: only the lower five intercostal spaces in the left hypochondriac region and extending to the back. The retraction is synchronous with the radial

or carotid pulse. The epigastric pulsations are very marked. Retraction over the xiphoid may also be seen at times.

Palpation: The adhesions firmly fix the apex beat. Diastolic shock or rebound.

Percussion: Increased dulness.

Auscultation: If dilatation has supervened we will get some of the signs on page 19. Pulsus paradoxus may be present.

ENDOCARDITIS

Definition: An inflammation, either simple or septic, acute or chronic of the inner lining membrane of the heart. The heart valves become the seat of vegetations which may

(a) undergo organization;

(b) necrose and slough off, the fragments entering the blood stream causing embolic process;

(c) become sclerotic and thickened and mechanically interfere with the working of the heart valves.

Classification:

1. **Simple** or **benign** endocarditis.
2. **Septic-malignant-ulcerative-infective** endocarditis.

Etiology:

1. All varieties of rheumatism.... { articular. muscular. gonorrhoeal.
2. Auto-intoxication, due to some chemical poison in the blood.
3. Infectious fevers: scarlet fever, diphtheria, pneumonia.
4. Syphilis, gout, Bright's Disease and cancer.

   Syphilis first causes an aortitis and then the inflammation extends to the valves.
5. The septic form occurs in cases of septic poisoning anywhere in the body; e. g., puerperal infection, septicemia, pyemia, etc.
6. It is very common in pneumonia.

(A) ACUTE SIMPLE ENDOCARDITIS

Clinical History:

The majority of the cases occur in connection with an attack of acute articular rheumatism, and when the heart does become involved not more than 3 days will have elapsed after rheumatism appeared.

The only **physical sign,** and this is very dubious, is the appearance of a new systolic murmur over the body of the heart, especially over the mitral area. The murmur is apt to be systolic in time and not likely to be transmitted. If the murmur is once discovered it is very apt to become more intensified with the progress of the disease and the latter is a characteristic feature.

(B) SEPTIC ENDOCARDITIS

Clinical History:

As stated before, this form of endocarditis occurs most often in connection with pneumonia, septicemia, and puerperal sepsis. There are four varieties of this disease and the first mentioned is the most important.

1. Typhoid.
2. Cardiac.
3. Cerebral.
4. Early septicemic.

All forms of septic endocarditis are characterized by an "iceberg" temperature chart:—the remittency being very pronounced.

The **typhoid form** is best described in the differential diagnostic table on page 16.

In the **cardiac form** we find irregularity and rapidity of the heart action. It usually occurs in the course of valvular disease and if so, the already existing murmurs may become more intensified. A murmur independent of the valvular defect is difficult to detect and seldom occurs. The coronary artery may become plugged (see angina pectoris).

In the **cerebral form** the most prominent early manifestations are:—headache, restlessness and delirium. This form must be distinguished from cerebro-spinal-meningitis.

In the **early septicemic variety** prostration, anemia and emaciation occur early and progresses rapidly to a fatal termination. Embolic processes may occur. Usually a nephritis complicates. Death occurs as a rule in two weeks.

DIFFERENTIAL DIAGNOSIS

SEPTIC ENDOCARDITIS	TYPHOID FEVER
Invasion: Abrupt with marked prostration which progresses daily.	Gradual with lassitude, nosebleed, anorexia, brow headache, bronchial catarrh. There may be wakefulness but no prostration.
Temperature: Iceberg septic temperature, diurnal variations, remittency and irregularity very marked.	Remittency only in first and third week and never as great. Step ladder ascent and decline in first and third week respectively. During the fastigium the fever is continuous and very slight remissions.
Spleen: Not enlarged, but is severely painful, due to perisplenitis.	Enlarged in the second and third weeks.
Eruption: Embolic processes in the skin cause petechial hemorrhagic eruptions.	Appear in crops, between the 7th and 14th days. In exceptional cases in the third week may a petechial rash occur. The rose spots disappear on pressure and do not remain after death.
Intestinal Symptoms: Absent. If toxemia is marked we may get a septic diarrhoea. Stools not characteristic.	Abdominal distension. Catarrhal enteritis causes loose movements of a pea-soup granular consistency.
Embolic processes: Very important. In skin: petechial rashes. In brain: hemiplegia. In joint: septic arthritis. In kidney: hematuria and nephritis.	Absent.
Sweating: Very conspicuous.	Not conspicuous.
Leucocyte count: Above normal: leucocytosis.	Below normal: leucopenia.
Widal Test: Absent.	Present.

Note: Although the heart is the seat of the lesion in septic endocarditis there is nothing distinctive about the organ. The presence of the elements of septicemia masks the cardiac picture. Blood cultures should be taken in suspicious cases.

(C) CHRONIC ENDOCARDITIS

Explanation: As stated under the discussion of acute endocarditis, punctate vegetations occur in the vicinity of the valves, but they do not interfere with the action of the valves. However, when the process has become chronic these vegetations, which are inflammatory in nature (consisting of R. B. C., W. B. C., blood plates and fibrin entangling these formed elements), tend to organization with ultimate sclerosis and thickening of the valves. In the septic conditions, necrosis of the valves leads to deformities of these structures, hence interference with their normal functionation. The ultimate pathological changes that must take place in the heart are hypertrophy and dilatation (q. v. pages 17-18).

The **causes** of the chronic form of endocarditis are the same as those of the acute. In addition to those enumerated on page 14, we may add the poisons of lead and alcohol and those occupations requiring prolonged and heavy muscular work.

In order to better understand and to interpret the changes occurring in valvular disease the author has deemed it expedient to present first the subjects of hypertrophy and dilatation, followed by a short discourse on the mechanism and interpretation of murmurs.

HYPERTROPHY OF THE HEART

Definition: A conservative process marked by an increase in the thickness of the heart muscle and consequently increased power. As long as hypertrophy predominates over dilatation the patient is not in imminent danger. If there is much change in the arterial wall (arteriosclerosis), then hypertrophy is not a desirable process.

Etiology:

1. Chronic endocarditis (chronic valvular disease).
2. Adhesive pericarditis.
3. Exopthalmic Goiter.
4. Chronic Nephritis especially the interstitial form.
5. Gout.
6. Myocardial degenerations.
7. Any obstruction to the onward flow of blood in the arterial system:—
    (a) Mediastinal tumor pressing on arch of aorta.
    (b) Thoracic aneurism.
    (c) Atheroma or arteriosclerosis causing a deficient elasticity of the arterial walls.
8. Prolonged muscular strain: e. g., in longshoremen and athletics.

Symptoms:

Aside from the congestive headache, dizziness and palpitation, we have no others as long as hypertrophy is the predominating factor. The great danger is dilatation. Such persons showing signs of hypertrophied heart must be cautioned against sudden exertion as it may result in death. Hypertrophy is a slow process and is in proportion to the demands made upon the heart.

In speaking of hypertrophy we usually refer to the left ventricle. The **right ventricle,** however, may also hypertrophy in the following diseases:

1. Cirrhosis of the lung.
2. Pulmonary emphysema.
3. Right side valve lesions.
4. Mitral disease (very common).
5. Pericardial adhesions.

It is clinically recognized by:—

(a) an accentuated pulmonic second sound.
(b) slight increase of cardiac dulness to the right of the sternal border.
(c) a marked tendency for dilatation to occur early.

Physical Signs of **Hypertrophy** of the **Left Ventricle:**

Inspection: A forcible apex beat which can be accurately located. Epigastric pulsations. Apex beyond nipple line.

Palpation: Apical displacement downward to the sixth left intercostal space. In aortic disease it may be in the seventh interspace ("Cor Bovinum").

Percussion: The heart is enlarged as evidenced by an increased area of dulness. The enlargement in the long axis of the heart is proportionately greater than in the width.

Auscultation: First sound increased in intensity therefore more booming in quality. Aortic second sound also accentuated.

DILATATION OF THE HEART

Definition: Dilatation of the heart represents an advanced stage of hypertrophy, hence the same factors operate in the production of the latter as in the former. The symptoms usually appear gradually, but cases of acute dilatation may occur: e. g., Grave's Disease, beer drinkers, and in acute fevers in which the myocardium degenerates. When compensation breaks then dilatation ensues.

Symptoms:

1. **Dyspnea** and **chronic cough:**

   Dyspnea is very urgent in acute cases. It leads to pulmonary engorgement and this in turn may cause chronic bronchitis, pulmonary edema, etc. The cough is present all the time.

2. **Engorgement** of the **abdominal viscera** will cause:
   (a) enlargement of liver, spleen, and kidney.
   (b) ascites.
   (c) edema of the legs.
   (d) catarrhal gastritis and enteritis, causing diarrhea due to venous congestion of the alimentary tract.
   (e) urine decreased in quantity and its sp. gr. is high.

3. While that part of the body below the neck is engorged, the head is anemic, and it results in an anemic headache, dizziness, vertigo and ringing in the ears. At times, mania and delirium may develop. Attacks of syncope may occur especially in fatty heart.

4. **Palpitation** is very prominent. The pulse as a rule is rapid and feeble.

Physical Signs:

Inspection: Diffuse irregular impulses are seen to strike the chest wall and the epigastrium. It is for this reason that the apex cannot be accurately located.

Palpation: Diffuse apex beat corroborated. Diffuse tremors or thrills are sometimes felt. They occur more often with aortic than with mitral disease. The thrills indicate enfeeblement of the muscle and dilated cavity. The thrills must be distinguished from the presystolic thrill of mitral stenosis.

Percussion: Increased dulness (greatest in the width), due to an enlargement of the heart. The greatest breadth of dulness is above, while in pericarditis with effusion it is below.

Auscultation: Irregular, intermittent heart. First sound decreased in intensity and may be duplicated. Galloping rhythm and embryonic cardiac sounds may also occur. Accentuation of pulmonic second sound. Any of the valvular murmurs.

Differential diagnosis: from:—

1. Hypertrophy.
2. Pericardial effusion.
3. Aneurism of the aorta.
4. Mediastinal tumor.

DIFFERENTIAL DIAGNOSIS

HYPERTROPHY	DILATATION
Inspection: Apex beat, forcible and localized. Epigastric pulsations.	Diffuse precordial wave. Apex diffuse. Marked epigastric pulsations.
Palpation: Apex can be definitely in sixth left intercostal space. Heart is regular. Thrills are absent.	Difficult to locate apex. Irregular heart. Systolic thrill sometimes present.
Percussion: Dulness greater in long diameter than in width.	Dulness greater in width than in long diameter.
Auscultation: First sound increased in intensity. Second aortic accentuated.	First sound decreased in intensity. Second pulmonic accentuated. Foreign sounds heard and also a duplication of sounds.

PERICARDIAL EFFUSION	DILATATION
Apex: Displaced upward and to left fourth interspace within nipple line. It cannot be well palpated.	Displaced downward and to the sixth left interspace beyond nipple line. Diffuse pulsation over the precordial area.
Pulse: Very good quality as compared to the muffled and distant heart sounds.	Pulse is feeble and irregular.
Enlargement of heart: Increased dulness in width, but is greatest below. Upper limit of dulness changes with position.	Increased dulness in the width, but greatest above. Unaffected by posture.
Compression of adjacent left lung: Causes signs of pneumonic consolidation.	Bronchial breathing, and tympanitic resonance are absent.

(See also page 13.)

MURMURS

Definition: A murmur is an adventitious cardiac sound which can be produced in one of the following ways:—

1. Obstruction to the onward flow of blood.
2. Changes in the direction of the blood current.
3. Changes in the composition of the blood.

The first two groups comprise the so-called **"organic murmurs"** due to either a narrowing (stenosis) or a widening of the valvular orifices. In the latter case the regurgitant stream produces the murmur. The third group comprises the so-called **"functional"** or **"hemic" murmurs.**

The characteristics of organic murmurs are:—

1. A lesion of some valve is always demonstrable.
2. Each murmur is heard best over a certain area. These areas are called "areas of maximum intensity."
3. Endocardial murmurs are transmitted in definite directions beyond the borders of the heart.
4. Evidences of hypertrophy or dilatation are very apt to be present.
5. The murmurs occur at definite periods with relation to the cardiac cycle. Murmurs therefore are presystolic, systolic or diastolic in time.

It must be remembered that a diminution in the intensity of a murmur does not necessarily mean that the patient is getting better. On the contrary, it may be due to a weakening of the heart muscle. While this is true only for organic murmurs, functional murmurs disappear completely when the anemia is cured and the blood is bettered.

The other **characteristic features** of **"hemic" murmurs** are:—

1. They are usually systolic.
2. Usually soft and blowing.
3. Usually located over pulmonic area (see drawing, page 8).
4. Accompanied by venous hum in veins of the neck.
5. They are due to changes in the blood itself and hence when the chlorosis or other form of anemia is cured the murmur disappears.
6. The signs and symptoms of hypertrophy and dilatation are absent unless it be a severe case of anemia, when dyspnea, palpitation and edema may occur.

THE HEART SOUNDS

First sound: Is divisible into two elements:—

(a) valvular: closure of the auriculo-ventricular valves.
(b) muscular: systolic contraction of the muscular walls of the ventricles.

Its characteristics are, as compared to the second, longer in duration, louder in intensity, and lower in pitch. It is the sound with the booming quality.

Second sound: Is purely valvular, due to the closing of the aortic and pulmonary valves.

An accentuated second aortic sound is characteristic of hypertrophy of the left ventricle, arteriosclerosis, etc.

An accentuated pulmonic second sound is characteristic of hypertrophy of the right ventricle, pneumonia in favorable cases, mitral lesions, emphysema, etc.

AORTIC REGURGITATION INSUFFICIENCY INCOMPETENCY

Etiology:

The aortic valve may be rendered incompetent by:—

1. Arteriosclerotic changes which cause a curling of the cusps.
2. Septic endocarditis which causes ulceration and necrosis of the valves.
3. Rheumatism which causes nodular excrescences on the valves.
4. It may be secondary to an aortitis. Syphilis first causes an aortitis and then involves the valve.
5. A "relative" aortic insufficiency may occur as a result of dilatation of the aortic ring by an aneurism located there.
6. It is most common in the middle period of life and attacks able-bodied men, especially those who have worshiped Bacchus and Venus with fervency and zeal.
7. It is a fact worthy to be remembered that sclerosis of the coronaries frequently accompanies lesions of this valve.

Symptoms:

1. Owing to a double stream flowing into the left ventricle this chamber must enlarge and hypertrophy (see page 17).
2. When compensation breaks dilatation must ensue (see page 18). A peculiar characteristic of lesions of this valve in the stage of dilatation is the infrequency with which generalized anasarca occurs. Dyspnea is less urgent and hemoptysis is relatively infrequent.
3. The concomitant sclerosis of the coronary arteries is held directly responsible for the attacks of angina pectoris. The anginal attacks occur more often in connection with lesions of this valve than any other.

4. Another noticeable feature is the frequency of sudden death.
5. If the valve is involved in consequence of a septic endocarditis embolic symptoms may occur, due to breaking off of a portion of the necrotic valve which enters the blood stream and finally into an endartery. The brain and the kidney are very apt to be involved in embolism, hence paralysis and nephritis are possibilities.
6. Inability to sleep and troubled dreams occur towards the close of the disease.

Physical Signs:

1. As a rule the signs of **hypertrophy** predominate (see page 17).
2. The adventitious sounds heard on auscultation are:—
   (a) **Diastolic murmur,** heard at the third or fourth left intercostal space at the left border of the sternum, transmitted downward to the ensiform cartilage and towards the axilla, but not by way of the apex.
   (b) **Presystolic Flint murmur** at the apex. It is not accompanied by a thrill. It is not always present. It may appear and then disappear.
   (c) In a minority of the cases (when dilatation supervenes, and this is not common) a systolic murmur of a relative mitral insufficiency may also be audible. It replaces the first sound.
   (d) By listening over the carotid artery (not the apex or the aorta) to the aortic second sound it may or may not be accentuated. If accentuated it means that the leakage is small and therefore a good prognostic sign (Broadbent). A weak sound diagnosis marked regurgitation.
3. The **pulse** of aortic regurgitation is very characteristic: The term **"Corrigan Water-hammer Pulse"** signifies the collapsing, shotty and throbbing characters of the pulse. The collapsing of the arteries is both visible and palpable. In palpating the radial artery be sure that the patient is not standing as gravity may cause a fullness of the artery (Broadbent). He states that the hand must be above the level of the heart, at least up to the shoulders. The peripheral vessels are also seen to pulsate.
4. The **pulse** is **usually retarded** or **delayed:** i. e., there is an appreciable interval between the beat of the heart and the radial pulse.

5. **Maximum systolic blood**-pressure is very high. Minimum diastolic blood-pressure is very low.

Note: The longer the murmur persists during diastole the smaller is the quantity of the regurgitant fluid and the effect on blood-pressure is not so marked. Murmurs that are heard during the early part of the diastolic phase indicate free regurgitation. The greater the regurgitation the greater the collapsible character of the pulse.

6. **Syphgmogram.** Shows a sharp high perpendicular upstroke, pointed apex and slight dicrotic wave (due to absence recoil of the aortic column of blood against the valve). The sharp high perpendicular upstroke is followed by a rapid decline.

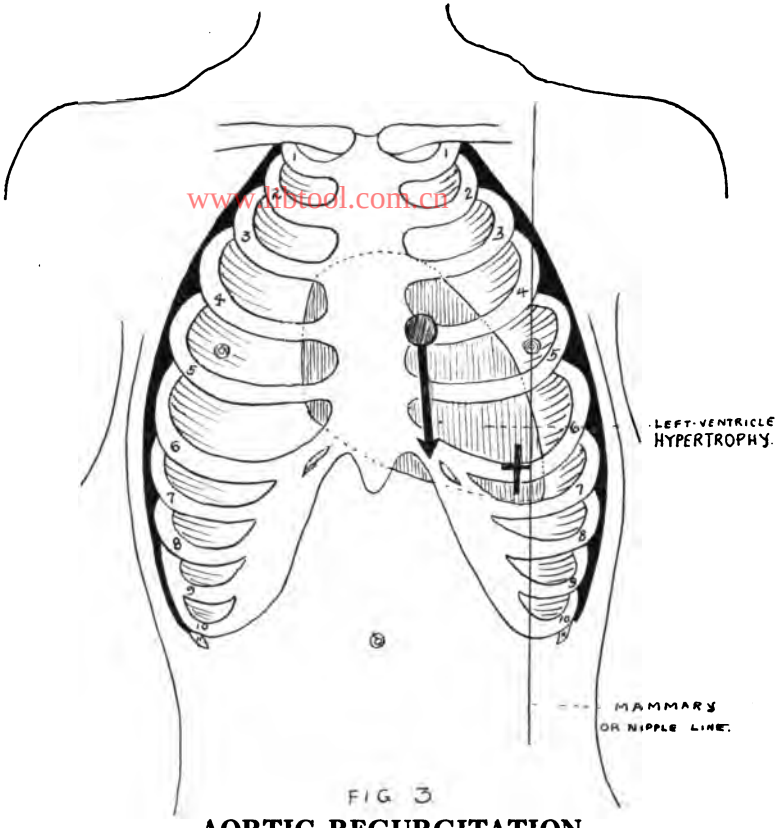

FIG 3

AORTIC REGURGITATION

SHOWING (SCHEMATIC)

Murmur—Maximum intensity at 4th rib at sternal junction and transmitted downward to ensiform as shown by the arrow. It is also transmitted to the left axilla (not shown).

Cor Bovinum = Enormous hypertrophy of the left ventricle.

Note: Apex lies beyond nipple line.

† Shows region of Flint's Murmur at apex.

AORTIC OBSTRUCTION STENOSIS

Etiology:

Narrowing of the aortic orifice occurs later in life than aortic incompetency. Peculiarly enough, it is always associated with some incompetency. It is the least common of the left heart valve lesions.

The gross appearances of the valves show either (a) firm contraction, (b) adherent cusps, or (c) rigid masses of vegetations approximating the valves.

In consequence of the narrowing of the orifice the left ventricle must hypertrophy in order to propel the blood into the aorta.

Symptoms:

There is no tendency to dyspnea or other signs of the "venous cycle" as long as hypertrophy predominates. This valve lesion can only be diagnosed by the physical signs, as there are relatively few symptoms even with an enormously enlarged heart. On the other hand, in mitral regurgitation, there may be only a slight degree of hypertrophy and still there is marked dyspnea, cough, etc., due to venous congestion. Sudden death has occurred at times.

Physical Signs:

1. Signs of **hypertrophy** of **left ventricle** (see page 18). The apex beat is not as strong as in other cases of hypertrophy of the left ventricle.
2. **Forcible thrill** at the same place where the systolic murmur is heard with maximum intensity.
3. **Systolic murmur,** rough or musical, at the second right intercostal space close to the sternal border transmitted to the great vessels of the neck. At this point the aorta comes from under the pulmonary artery.
4. The **first sound** is **prolonged.**
5. The murmur of a relative mitral insufficiency may also occur.
6. The **pulse is small,** its tension good and its rate somewhat slower than normal.
7. **Syphgmogram.** Shows a gradual ascent and a gradual descent of the pulse wave. The top is rounded and the secondary waves indistinct.

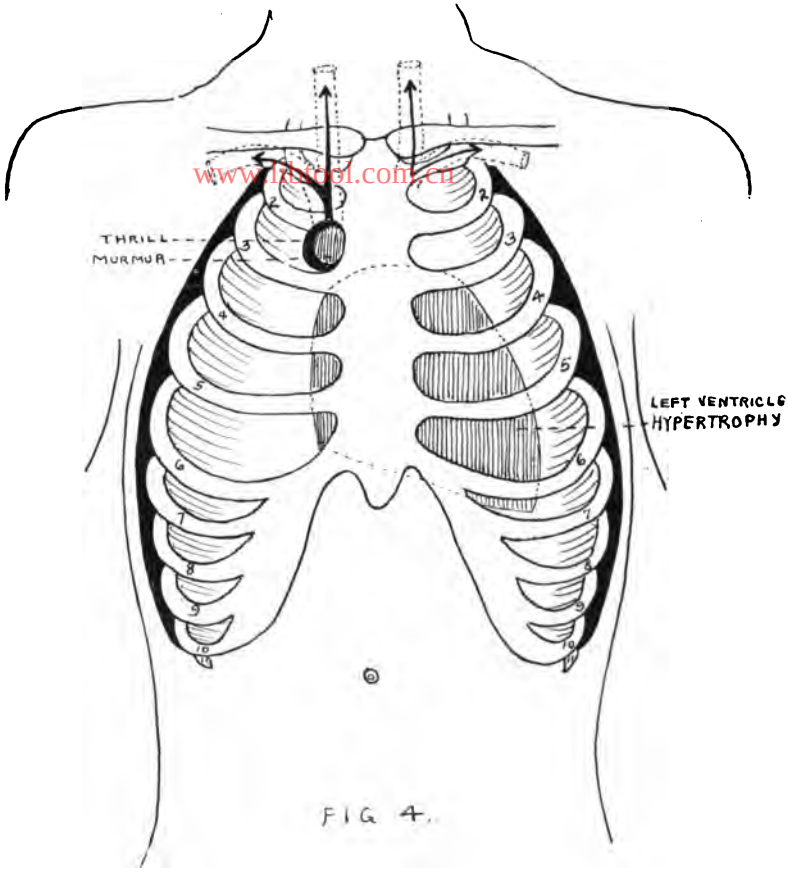

FIG 4.

AORTIC STENOSIS

SHOWING (SCHEMATIC)

Murmur—At 2nd right intercostal space transmitted to great vessels of neck.
Thrill—In same region. Left ventricle hypertrophy.

MITRAL REGURGITATION INSUFFICIENCY INCOMPETENCY

Etiology:

The usual causative factor is **endocarditis.** The lesions of this valve constitute the largest percentage of all the valvular lesions. A true case of mitral incompetency must be distinguished from a "relative" insufficiency as occurs in consequence of a dilatation of the left ventricle.

The blood regurgitating into the left auricle causes this chamber to hypertrophy and dilate. The hypertrophy is not strong enough to oversome the congestion of the pulmonic tissues, hence the right ventricle hypertrophies to endeavor to overcome this venous stagnation. Hence, we find an accentuated second pulmonic sound. The left ventricle also must hypertrophy, because with each systole of the left auricle more blood is discharged into the left ventricle. Therefore, there is a double ventricular hypertrophy. In spite of this combined hypertrophy, dilatation is a frequent occurrence and the signs of visceral engorgement are very conspicuous.

Symptoms: This valve lesion is conspicuous by its symptoms:—

1. At all stages of the disease one will be able to detect **failure in the circulatory apparatus,** regardless of whether compensation is intact or broken. The cyanotic discoloration of the face and fingers is early evidence.
2. **Dyspnea** and **cough** become more and more aggravated as the heart fails, and is especially marked if there is concomitant bronchitis or serous effusions into the pericardium, pleura or peritoneum.
3. The ultimate picture is that of a water-logged patient and is due to venous stasis.

Physical Signs:

1. Evidences of **both left and right ventricle hypertrophy.** The accentuation of the second pulmonic sound is heard best at the second left intercostal space at the sternum.
2. There is an enormous enlargement in the transverse diameter of the heart, as evidenced by an increase in dulness in the width.
3. **Systolic murmur** at the apex transmitted to the axilla. It is also transmitted to the scapulae and the back but not by the axillary route. It is conducted from the base of the ventricle back to the spinal column. The murmur is somewhat musical or blowing in nature.
4. The **pulse is always rapid and small** and is very apt to be **irregular** even from the start. There is less filling in of the arteries.
5. **Syphgmogram:** will show irregularity in force as well as in frequency. The tracing is small, the wave is short and small and ill-sustained.

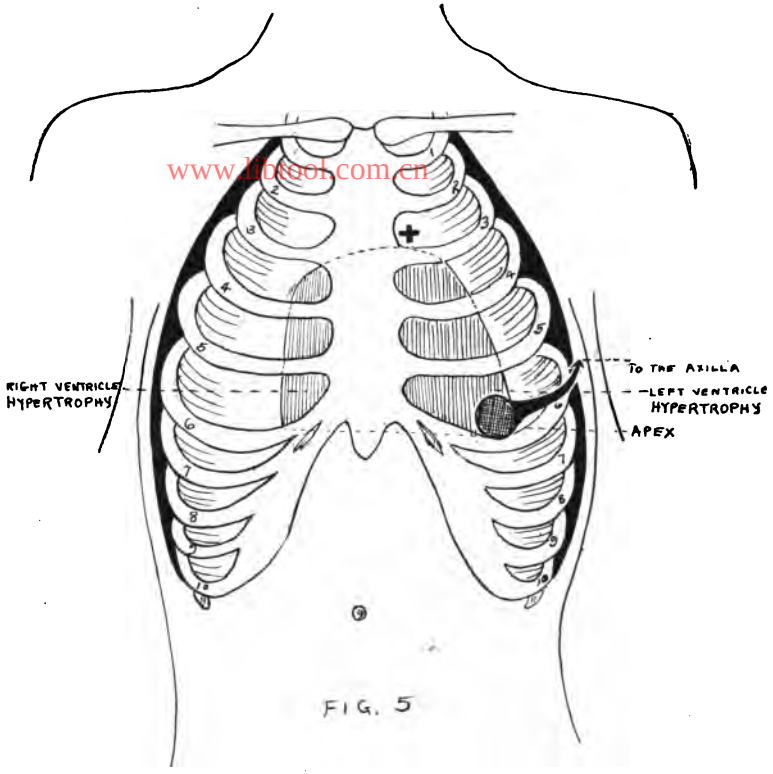

FIG. 5

MITRAL REGURGITATION

SHOWING (SCHEMATIC)

Murmur: Maximum intensity at apex and transmitted to axilla.

Hypertrophy: of both ventricles.

† Shows accentuated pulmonic 2nd sound at 2nd left intercostal space.

Note: The transmission to the scapula and back is not around the thorax but is conducted from the base of the ventricle back to the spinal column.

MITRAL OBSTRUCTION STENOSIS

Etiology:

Occurs mostly in the young, especially girls. It is due to endocarditis and the prevalence in children is explained by the frequent occurrence of rheumatism and chorea.

The left auricle hypertrophies and dilates. The regular course of events is dilatation and hypertrophy of the right ventricle. There is no occasion for the left ventricle to hypertrophy, as the mitral orifice is obstructed and only a small volume of blood enters this chamber with the auricular systole.

Symptoms:

Are dependent upon cardiac compensation, as in nearly all valvular lesions. Venous visceral engorgement and its associated symptoms occur sooner or later. Cyanosis is an early manifestation.

Physical Signs:

1. **Hypertrophy** of the **right ventricle** without hypertrophy of the left. The apex is even displaced upward and to the left while in hypertrophy of the left ventricle it is displaced downward. As long as hypertrophy predominates the pulmonic second sound remains accentuated.

2. A **presystolic purring thrill** can be heard in the fourth or fifth left intercostal space, within the nipple line.

3. The vibrations of the mitral curtains in the blood current give rise to a **presystolic blubbering murmur.** When the valves are bound by adhesions it assumes a blowing character. The murmur is heard for one inch in every direction from the apex. In the last stage, signifying approaching dilatation, and with occurrence of a tricuspid incompetence, the presystolic murmur disappears.

4. **Syphgmogram:** Shows short upstroke, long wave which is slowly extinguished. Irregularity is very apt to ensue.

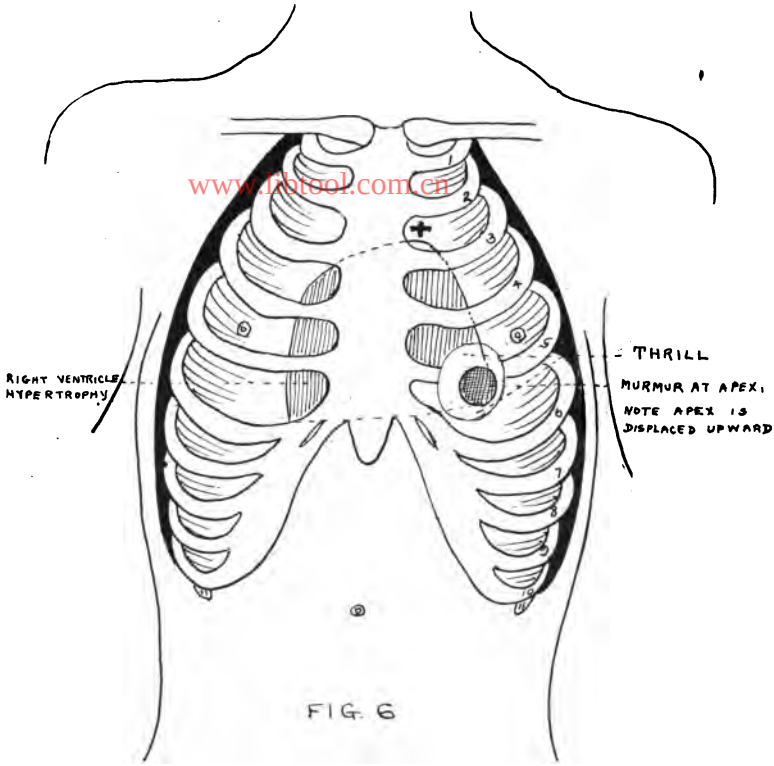

FIG. 6

MITRAL STENOSIS

SHOWING (SCHEMATIC)

Murmur: At the apex within a 2 inch diameter.
Thrill: In 4th or 5th interspace just within nipple line.
Hypertrophy: Only of right ventricle.
† Accentuated pulmonic 2nd sound.

DIFFERENTIAL DIAGNOSIS

AORTIC DISEASE	MITRAL DISEASE
Symptoms mainly due to anemia, viz.:	Symptoms chiefly due to venous congestion, viz.:
1. Pallor.	1. Cyanosis.
2. Throbbing of the carotids.	2. May get pulsations in the veins of the neck.
3. Attacks of angina.	3. Sudden attacks of severe dyspnea. Actual pain is rare.
4. Breathlessness on slight exertion.	4. Breathlessness, but often present without exertion.
5. Prominent nervous symptoms due to cerebral anemia. Syncopal attacks also common.	5. Pulmonary symptoms most prominent: i. e., cough, hemoptysis.
6. Left ventricle usually much hypertrophied.	6. Right ventricle alone or combined with left ventricular hypertrophy.
7. Pulse as a rule is slow.	7. Pulse as a rule rapid and very apt to become irregular.

(Modified after Wheeler and Jack.)

TRICUSPID AND PULMONARY VALVE LESIONS

As before stated, since the combined lesions of the pulmonary and tricuspid valves constitute an insignificant percentage of all the valvular lesions it behooves us to familiarize ourselves more with the so-called "left heart" lesions than with "right heart" lesions.

The murmurs of both **tricuspid** regurgitation and stenosis are heard at the base of the ensiform cartilage extending a little past the right sternal border. The regurgitant murmur is systolic in time and transmitted up to the third costal cartilage. The stenosis murmur is presystolic and heard only over the tricuspid area at the ensiform base. The only other important sign of tricuspid regurgitation is that the regurgitant stream back into the right auricle causes systolic pulsations in the cervical veins.

The murmurs of both **pulmonary** regurgitation and stenosis are heard in the pulmonic area in the second left intercostal at the sternal border. However, the pulmonary area is a favorite place for other murmurs; the cardio-inspiratory murmur, hemic murmur, etc., from which they must be distinguished. The pulmonary regurgitant murmur is diastolic in time.

DIFFERENTIAL DIAGNOSTIC CHART ON ORGANIC MURMURS

ORGANIC VALVE LESION	TIME	POINT OF MAXIMUM-INTENSITY	TRANSMISSION	
AORTIC REGURGITATION.....	Diastolic. (After second sound.)	Left fourth costal cartilage, near sternum.	Downward to ensiform cartilage. Also to the axilla.	Most common.
AORTIC STENOSIS.....	Systolic. (After first sound.)	Right second intercostal space, near sternum.	Upward to the great vessels of the neck.	
MITRAL REGURITATION.....	Systolic. (After first sound.)	Apex.	To the left, in the direction of the ribs. Often heard at inferior angle of the scapula.	
MITRAL STENOSIS.....	Preysystolic. (Before first sound.)	Apex.	Circumscribed area around apex.	
TRICUSPID REGURGITATION.....	Systolic.	Right inferior margin of heart.	To the right.	Very infrequent.
TRICUSPID STENOSIS.....	Presystolic.	Right border of heart, as well as right inferior margin of heart.	Circumscribed.	
PULMONARY REGURGITATION.....	Diastolic.	Second left intercostal space.	Downward and to the right.	
PULMONARY STENOSIS.....	Systolic.	Same as above.	Circumscribed.	

MYOCARDITIS

Definition: When valvular diseases of the heart are excluded the remaining diseases of the musculature of the heart are classified under myocardial disease. The process may be either acute or chronic. Acute myocarditis is, as a rule, a purely inflammatory process and is accompanied by febrile disturbances, while myocardial degenerations are not purely inflammatory and fever may be wanting.

(A) ACUTE MYOCARDITIS

Etiology:

The myocardium may be involved by:

1. An extension inward of a septic endocarditis. The necrotic process in the valves extends into the muscle.
2. Septic embolism in the heart.
3. Extension of an aortic endarteritis into the coronary artery, obliterating its lumen.
4. Extension by contiguity from a pericarditis, as well as from an endocarditis.
5. Mechanical injury from overaction of the heart: e. g., Exopthalmic Goiter, acute infectious fevers, etc.

Symptoms:

The symptoms are those of cardiac weakness. It must be remembered that the heart muscle cells are highly specialized, possessing only a limited power to repair cellular damage. Necrosis must therefore ensue and followed by replacement by hyperplastic connective tissue. The signs are ultimately those of dilatation.

(B) CHRONIC MYOCARDITIS

Definition:. A chronic inflammation of the interstitial tissue of the heart.

Etiology:

1. While any acute inflammation of the myocardium (such as may occur after acute infectious fevers as scarlet fever and diphtheria) may become chronic, the most frequent cause is arteriosclerosis (endarteritis).
2. All the causes that operate in the production of arteriosclerosis may cause this affection, e. g., gout, lead, alcohol, syphilis, etc.
3. It may occur in chronic diseases.
4. It is normal in old age. It always attacks people above

the age of 40 years. High living and an overactive life contribute.

5. The coronary arteries are also involved by the sclerotic process. The narrowing of the lumen leads to impaired nutrition of the heart muscle, necrosis and fibrous connective tissue hyperplasia.

Symptoms:

1. It is essentially a chronic process. The coronary circulation is only gradually narrowed. The extent of the cicatrization is in proportion to interference in the coronary circulation.
2. Tendency to dilatation.
3. Extensive replacement of muscle tissue by scar tissue weakens the heart so much as to favor the development of cardiac aneurisms, which may rupture and cause death.
4. Because of the concomitant arteriosclerosis we may have:—
   (a) attacks of angina pectoris (page 40).
   (b) attacks of cardiac asthma in which pallor and constriction are absent but dyspnea is a characteristic feature.
   (c) transient attacks of syncope and apoplexy at times, the first being due to cerebral anemia.
5. There are no murmurs. Arrhythmia and bradycardia are apt to be present.
6. Parietal thrombi may occur in the heart and this in turn may cause distal embolic processes.

DEGENERATIONS OF THE HEART
(A) PARENCHYMATOUS DEGENERATION

Etiology:

Occurs only as an acute lesion:—

1. Acute infectious fevers, especially those accompanied by a "toxemia;" e. g., scarlet fever, pneumonia.
2. Organic and metallic poisons: alcohol, mercury, arsenic, tyrotoxicon.
3. Sun-stroke due to the high temperature. It also accompanies prolonged fevers.

Symptoms:

Progressive cardiac asthenia. The great danger is the development of dilatation (see page 18). The heart muscle becomes the seat of a cloudy swelling hence greatly interfering with its functionation.

(B) FATTY DEGENERATION

Note: Fat replaces the muscular structure of the heart, whereas in fatty infiltration fat is deposited between the muscle fibers, hence the latter is not a substitution process.

Etiology:

1. A sequel to unhealed parenchymatous degeneration.
2. Protracted toxic conditions and in long standing cases of sepsis, osteomyelitis, tuberculosis and syphilis.
3. Of all acute infectious fevers, diphtheria ranks first.
4. Acute arsenical and phosphorous poisoning.
5. Deficient nourishment: scurvy and cachexias.
6. Severe cases of anemia and after severe hemorrhages.
7. It is also recognized as the beer drinker's heart.

Symptoms:

This affection cannot be diagnosed per se as its symptoms are those of myocardial disease in general. It is characterized by the liability to sudden death, anginal attacks (as the coronaries are affected), apoplectic and epileptiform seizures. While there is a great tendency to dilatation, we do not very often meet with cases of marked dropsical effusions.

(C) FATTY INFILTRATION

Note: Fat is deposited in the connective tissue around the heart cell. At times a thick capsule of fat surrounds the heart and also between the muscle fibers.

Etiology:

1. It occurs as a part of a general obesity.
2. Overeating and sedentary habits predispose.
3. Pernicious anemia, chlorosis and tuberculosis.
4. Arteriosclerosis of the coronary arteries.
5. The beer drinker's heart may show both fatty processes.

Symptoms:

There are no diagnostic symptoms until dilatation manifests itself. The heart muscle being compressed by the fat causes it to soften and this results in relaxation of its chambers, especially the left ventricle, and this is followed by dilatation. Sometimes the fat may cause pressure atrophy and this invariably results in an unexpected death. The symptoms are therefore those of a "weak heart."

SYMPTOMS OF MYOCARDIAL DISEASE OR THE SO-CALLED "WEAK HEART"

For clinical purposes, Osler divides all cases of myocardial diseases into three large groups:—

1. Those in which sudden death occurs with or without previous history of heart trouble. Sclerosis of the coronaries exists—in some cases with recent thrombus and white infarcts; in others, extensive fibroid disease; in others again, fatty degeneration. Many patients never complain of cardiac distress, but enjoy unusual vigor of mind and body.
2. Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of cardiac asthma, sometimes anginal attacks, collapse symptoms with sweats and extremely slow pulse, and occasionally marked mental symptoms.
3. Cases with general arteriosclerosis and hypertrophy and dilatation of the heart. They are robust men of middle age, who have worked hard and lived carelessly. Dyspnea, cough, and swelling of the feet are early symptoms, and the patient comes under observation either with a gallop rhythm, embrocardia, or an irregular heart with an apex systolic murmur of mitral insufficiency. Recovery from the first or second attack is the rule. It is one of the most common forms of heart disease.

FUNCTIONAL AFFECTIONS OF THE HEART
(A) PALPITATION

Definition: An irregular, forcible and usually rapid action of the heart perceptible to the patient. Many complain of precordial distress and a majority show an increased excitability of the nervous system. The attacks are usually paroxysmal. To be strictly called a neurosis, the element of consciousness of the cardiac disturbance must be present. The palpitation associated with valvular and myocardial disease does not always have the subjective consciousness of the throbbing heart and is therefore not a neurosis. It is very common in the following conditions:—

1. Hysteria and neurasthenia.
2. Puberty and the climacteric.

3. Over excitation of the heart by stimulants as tea, coffee, tobacco and alcohol.
4. Dyspepsia.

Symptoms:

As before stated the attacks are paroxysmal, lasting from a few minutes to an hour or more. In addition to the subjective element of the palpitation there is also precordial pain, anxiety, fullness in the throat and some dyspnea. Examination of the heart reveals the absence of all murmurs. The sounds of the heart are clear, ringing and metallic in nature.

(B) ARRHYTHMIA

Definition: An intermittent, irregular pulse in which one or more beats are omitted. It may be present without one being able to detect any signs of disease of the heart whatsoever. There may not even be the slightest discomfort. To be of diagnostic value other evidences of organic disease must be present. It is common in children and in the aged and in these cases it is not of much significance.

Varieties:

Bigeminal pulse: one in which the pulse beats occur in groups of twos.

Trigeminal pulse: one in which the pulse beats occur in groups of threes.

These two varieties are most often met with in mitral disease and signify failing compensation.

Delirium Cordis: is one in which the pulse is irregular in both strength and time.

Pulsus Alterans: strong and feeble beats alternating.

Pulsus Paradoxus: pulse becomes weak and small or imperceptible during inspiration. (See its diagnostic value under pericarditis with effusion, adherent pericarditis and in mediastinal tumor.)

Etiology:

1. Drugs: digitalis, coffee, tea, tobacco, belladonna.
2. Metabolic poisons.
3. Auto-intoxication and dyspepsia.
4. Neurasthenia.
5. Valvular disease of the heart, especially mitral disease.
6. Diseases of the intracranial structures: abscess, meningitis, concussion, apoplexy, etc.

(C) TACHYCARDIA

Definition: A rapidly acting heart as evidenced by an increased frequency in the pulse rate; 160-200 per minute. It is apt to be paroxysmal in character, appearing and disappearing abruptly. In true paroxysmal tachycardia the heart shows no evidence of organic disease.

Etiology:

True paroxysmal tachycardia occurs in:—

1. Neurasthenic and in anemic persons.
2. Drinkers and persons who smoke very much.
3. Diseases involving the vagus, the medulla or the cardiac plexus of nerves.

Continuous tachycardia occurs in:—

1. Exopthalmic Goiter.
2. A post diphtheritic symptom and in other acute infectious diseases.

(D) BRADYCARDIA

Definition: a marked slowing of the pulse; 40-20 or lower. It is physiological after delivery and in old age.

Pathological bradycardia occurs in:

1. Diseases of the nervous system: apoplexy, epilepsy, cerebral tumor, affections of the medulla, diseases of the cervical cord.
2. Convalescence from acute infectious diseases, especially pneumonia and diphtheria.
3. Disease of the digestive system; chronic dyspepsia, ulcer, jaundice, etc.
4. Diseases of the respiratory system: during the attack of emphysema or asthma.
5. Diseases of the circulatory system: arteriosclerosis, fatty and fibroid heart. It is not constant in the latter two.
6. Diseases of the urinary organs: uremia.
7. Toxic agents: lead, alcohol, digitalis, tea, tobacco, etc.
8. Anemia, chlorosis, diabetes, myxœdema.

HEART-BLOCK OR STOKES-ADAMS DISEASE

Definition: A **syndrome-complex** resulting from the independent action of the auricle and the ventricle in consequence of some pathological change in the "Bundle of His" placed between

the auricles and the ventricles and through which bundle the cardiac wave (impulse) must pass to excite a systole.

The **clinical features** are as follows:—

1. **Bradycardia:** either permanent or paroxysmal. It may be as low as 40-30-20.
2. **Visible auricular pulsations in** the **cervical veins,** especially the internal jugulars, viz., 2-1 or 3-1 rhythm on comparing the auricular impulses as shown in the cervical veins with the ventricular systoles (apex beats). Normally the relation between auricular and ventricular contractions is 1:1.
3. **Cerebral symptoms** as a result of anemia of the brain:—
   (a) transitory vertigo.
   (b) syncope and dizziness.
   (c) epileptoid seizures.
   (d) pseudo apoplectiform attacks.
   (e) dyspnea.
4. **Syphgmogram:** shows an intermittent pulse with a low arterial tension and lacks constancy in outline.
5. On palpation of the venous pulsations we find it rises slowly and falls rapidly. On palpation of the arteries it is just the reverse.
6. Besides the changes in the bundle of His (fatty degeneration, calcareous deposits, vascular changes, etc.), we find frequently arteriosclerosis of the coronary, vertebral and basilar arteries.

ANGINA PECTORIS (STENOCARDIA)

Definition: A symptom-complex characterized by the paroxysmal occurrence of excruciating pains in the precordial area and the pains having a tendency to radiate to the neck and down the left upper extremity. It is accompanied by the sensation of impending death and the heart seems as if it were compressed in a vise.

Etiology:

The majority claim that it is due to a spasm of the coronaries or to arteriosclerotic changes in the arteries, which results in an anemia or ischemia of the heart. Arteriosclerotic changes at the root of the aorta may readily extend into the lumen of the coronary arteries. It is usually an associated symptom in the following diseases:—

(a) aortic insufficiency.
(b) adhesive pericarditis.
(c) syphilis at all ages in life. Angina as a rule occurs between the ages of 40 and 60.
(d) some forms of myocardial disease.

Symptoms:

1. Onset: the attacks come on suddenly after exertion, mental excitement or gastric flatulence.
2. Breast pang: pain radiates in a definite direction (*vide supra*).
3. Heart feels compressed as if by a vise.
4. Facial pallor during the attack. Extremities are cold.
5. Profuse diaphoresis at the end of the attack.
6. In true cases of angina, dyspnea is not characteristic and this therefore distinguishes it from cardiac asthma.
7. Pulse: in many cases unaffected but the tension is usually increased during the attack.
8. It lasts a variable time: 5 to 30 minutes or longer. It may repeat itself any number of times.

DIFFERENTIAL DIAGNOSIS

TRUE ANGINA	NEUROTIC (PSEUDO) ANGINA
Age: 40 to 60 years.	Any age.
Sex: males exclusively.	Women.
Time of occurrence: follows exertion, excitement of flatulent distension of the stomach.	Any time. Often periodical and at night.
Attitude: silence and immobility.	Agitation and activity.
Pain: excruciating and radiates to shoulder and down left arm. Does not last very long.	Less severe pain. Does not radiate, but lasts longer.
Heart: sense of compression, as if by a vise.	Sense of expansion.
Sweating: profuse.	.....
Pulse: tension increased.	Tension unaffected.
Arterial medication.	Antineuralgic medication.

ARTERIOSCLEROSIS

Definition: A palpable thickening and hardening of the arterial walls. It is a natural occurrence in old age.

Etiology:

1. **Overeating** is a great predisposing factor. It may explain an early occurrence of what should be a senile change.

2. Males are more often affected than females, owing to their manner of living: strenuous and exciting life, helped along by **alcohol** and **syphilis.**
3. Race: common among the French owing to the prevalence of **syphilis.**
4. Occupation: the laboring classes: The increased strain and bad hygienic conditions under which they work predisposes. Lead, mercury and potassium ferrocyanide workers. Those professions requiring heavy mental work.
5. **Renal Disease:** The factors that are here concerned are:—
(a) High blood pressure which causes a rupture of the inner elastic membrane of the arteries and this is followed by connective tissue hyperplasia, which tissue contracts, shuts off its blood supply, and ultimately followed by necrosis, fatty degeneration and calcareous infiltration.
(b) The presence of poisonous matter in the blood which should have been excreted by the kidneys. The poisons may excite the intima to proliferate (endarteritis). A special variety of endarteritis is "Endarteritis Obliterans," in which the lumen of the artery is completely occluded. The mineral poisons, as lead and mercury, as well as the poisons of gout and rheumatism, act in a similar manner on the inner coat of the artery.

   Note: Renal disease is said to cause arteriosclerosis and vice-versa.
6. **Cardiac Hypertrophy** and **Hypertension:** The increased blood pressure causes a rupture of the intima which is the starting point of the disease, but sooner or later the media and the adventitia are involved.
7. **Toxemias** of all varieties, more especially the chronic intoxications:
   (a) metabolic poisons: gout, rheumatism.
   (b) metallic poisons: lead, mercury, copper.
   (c) alcohol and syphilis.

Results of Arteriosclerosis:

1. **On the arteries:** It causes a narrowing or obstruction of the lumen, hence the blood current is slowed. The roughening of the intima predisposes to thrombosis and embolism. There is a marked reduction in the elasticity of the arteries. The blood pressure being high, it favors aneurismal dilatations (saccular, spindle, etc.).
2. **On the heart:** The loss of the elasticity of the arteries causes a spasmodic and jerky flow with each systole. As the blood current is slowed the heart hypertrophies and it beats faster.

3. **On the general tissues:** They suffer as a result of lack of proper nutrition and deficient oxidation. It therefore predisposes to adipose formation.
4. **On the vital viscera:** A functional deficiency. It predisposes to degeneration and necrosis. The heart, brain, kidney, pancreas and liver suffer mostly.

Termination in Arteriosclerosis:

1. By rupture of an aneurism or a blood vessel.
2. By visceral insufficiency:
   (a) myocardial degeneration with dilatation.
   (b) loss of digestive power if liver and pancreas become affected.
   (c) thrombosis and embolism.
   (d) angina pectoris.
   (e) gangrene of the extremities.

Clinical Symptoms:

The **general** accompaniments of arteriosclerosis are:—
   (a) hardening of the arterial walls.
   (b) increased blood pressure.
   (c) signs of cardiac hypertrophy (see page 17).

The **focal** symptoms may be classfied thus (note that the structures are enumerated according to the relative frequency with which they are affected):—

1. **Heart:**
   Sclerosis of the coronary artery may cause angina pectoris. Aneurism of the aorta. Dilatation of the heart follows the hypertrophy.
2. **Brain:**
   Transient attacks of hemiplegia or aphasia which are very apt to repeat themselves. Attacks of simple vertigo or else as a part of Stokes-Adams Syndrome (see page 39).
3. **Kidney:** Chronic interstitial nephritis.
4. Pancreas and liver.
5. Miscellaneous:
   Gangrene: due to endarteritis, thrombosis or embolism in the extremities. Intermittent lameness or claudication.

THORACIC ANEURISM

An aneurism defined: A pulsating tumor containing blood and communicating with an artery, the result of a circumscribed dilatation of one or more of the arterial coats.

Etiology of Thoracic Aneurism:

All of the causes that operate in the production of arteriosclerosis may operate in the production of aneurism of the aorta,

but more especially syphilis and overwork, the former first producing an aortitis and then on overexertion a "saccular" aneurism results.

Diagnosis: In order to understand the symptomatology and physical signs, we must know the course of the aorta. The ascending portion and the transverse arch are situated directly under the middle piece of the sternum, while the descending portion goes backward and then down along the left side of the spinal column.

It is therefore evident owing to the superficial situation of the first two portions (anatomical division) the physical signs can be readily recognized, especially if the aneurism has eroded the bony superstructure of sternum and adjacent ribs.

The **physical signs** are:—

1. Aneurismal **pulsations:** expansile in character, can be both seen and felt. Located in about the same place as the murmur of aortic stenosis is heard and extending either to the right or the left.
2. A **diastolic shock** on palpation.
3. **Systolic thrills** may also be felt.
4. Percussion over the aneurismal tumor, especially after it has come close to the chest wall, will give abnormal **dulness.** The ascending portion will show dulness to the right of the sternum in the second and third intercostal spaces, while the transverse arch will show dulness under the upper part of the sternum and extending to the left.
5. On auscultation we have an **intense second aortic sound** and is especially characteristic in the increased area of dulness.
6. The **retardation of** the **pulse** in the arteries beyond the aneurism is a characteristic feature of all aneurisms. Both radial pulses may therefore differ in time and in volume.
7. **Tracheal tugging** is not always present. But if present it will exclude thoracic tumor.
8. Signs of **hypertrophy** must ensue.
9. A relative aortic insufficiency murmur may develop, due to aneurismal dilatation.

Pressure Symptoms: on

1. Œsophagus; dysphagia (painful deglutition).
2. Trachea or bronchus; brassy cough, bronchitis, dyspnea.
3. Recurrent laryngeal nerve; hoarseness, aphonia and characteristic "goose cough."
4. Superior vena cava or subclavian vein; enlargement and edema of arm (especially right).
5. Sympathetic nerve; pupil either dilated or contracted; at any rate inequality of the pupils.

Miscellaneous Symptoms:

1. Local paroxysmal lancinating pains especially in deep-seated tumors.

2. Angina pectoris very characteristic.
3. Hemorrhages from the lung: most severe in aneurismal rupture.
4. According to Broadbent:—
"The aneurism of 'physical signs' springs from the ascending aorta, while the aneurism of 'symptoms' springs from the transverse arch."

Note: In all doubtful cases the X-ray should be used.

DIFFERENTIAL DIAGNOSIS

THORACIC ANEURISM	THORACIC TUMOR (MEDIASTINAL)
Age: Older subjects. If young we get a syphilitic history.	Younger subjects.
Duration: If rupture does not occur life may be prolonged. If sac is obliterated, may live 5 years.	Never more than 1½ years.
Health: Slightly affected.	Marked cachexia.
Pain: Variable. Angina at times.	Always intense.
Metastasis: Absent.	It being a malignant tumor (usually sarcoma) metastasis is frequent. Glands in neck enlarged.
Physical Signs:	
Expansile pulsation.	Absent.
Diastolic shock.	Absent.
Murmurs absent.	Present over tumor.
Accentuated second aortic sound.	Absent.
Tracheal tug may be present.	Absent.
Pleural effusion absent.	Usually present.
Area of dulness is regular.	Irregular outline of dull area.
Heart slightly displaced.	Marked displacement, especially backward.

DIAGNOSTIC VALUE OF THE SYPHGMOGRAPH AND THE SYPHGMOMANOMETER

Definition: The **syphgmograph** is an instrument used to record pulse tracings. The tracing shows a majority of elements of the pulse. A thorough knowledge and interpretation of the normal pulse tracing will enable one to recognize the abnormal and draw conclusions as to the existing pathological condition.

The component parts of the normal pulse tracing are:

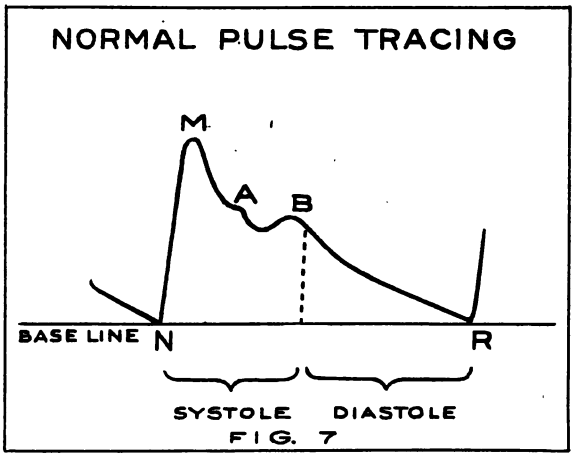

FIG. 7

MN: The sharp, almost perpendicular upstroke: due to ventricular systole forcing blood into the elastic aorta.

A: Predicrotic or tidal wave: its cause is probably the elastic oscillations of the arterial walls.

B: Dicrotic wave: due to closure of the semilunar valves. Note that this wave is on a lower level than the summit of the systolic upstroke.

R: End of the diastolic period.

The systole lasts 0.3 of a second.
The diastole lasts 0.5 of a second.

Total.....0.8 of a second.

The upstroke signifies a rise in blood-pressure, while the down-stroke means a fall in blood-pressure.

In examining a pulse tracing, first note whether there are any signs of intermittency or irregularity. A **base line** is therefore necessary as a guide in all pulse tracings. See its diagnostic value under "arrhythmia," page 38.

We next examine the **systolic upstroke.** A perpendicular upstroke terminating in a sharp apex and followed by a quick decline and inconspicuous dicrotic wave is diagnostic of aortic regurgitation. On the other hand a pulse tracing showing an oblique ascent with a rounded summit and secondary waves not very marked is diagnostic of aortic stenosis. Owing to the obstruction at the aortic valve the left ventricle discharges its contents slowly and the aorta therefore fills slowly, hence the gradual ascent.

As to the **dicrotic wave,** we may find it either increased, diminished or absent. Owing to the absence of the recoil this wave is inconspicuous in aortic incompetency. In obstruction of the peripheral circulation, as in arteriosclerosis, it is also only slightly marked. In cases of low tension (vaso-dilatation) this wave is marked.

As to the **descending limb** of the tracing, we may find it to be either sudden or gradual. A gradual descent occurs in aortic stenosis. A sudden decline is characteristic of incompetence of the aortic valve and is clinically corroborated by the so-called Corrigan water-hammer pulse which is felt to collapse suddenly. When collapse occurs, low tension is great. For this reason we find a low diastolic pressure and a high systolic pressure.

The **amplitude** of the tracings of aortic disease is greater than the amplitude of mitral disease. Irregularity in force and frequency and in the descent is very common in mitral disease. The lessened amplitudes of mitral tracings are corroborated by the small pulse felt in the arteries.

The **Syphgmomanometer** is an instrument used to determine arterial blood-pressure. Life insurance companies to-day recognize the importance of blood-pressure determinations of their applicants and this simple procedure has benefited them immensely because this instrument will recognize high blood-pressure (hypertension) that accompanies or antedates cardio-vascular-renal diseases. A survey of the mortality statistics of the Mutual Life Statistics of Death reveals the fact that 30 per cent of all deaths are due to cardio-vascular and renal diseases.

The diseases caused directly or indirectly by increased tension or associated with a high blood-pressure are arteriosclerosis, apoplexy, myocardial disease, interstitial nephritis, etc.

According to Emerson, a low blood-pressure (hypotension) is a characteristic of pulmonary tuberculosis, especially if advanced, or if a toxemia is marked,—but if nephritis or arteriosclerosis

co-exists there will be hypertension. A hypotension has been found at times even before physical signs of the incipient stage were detected.

The normal blood-pressures at the different ages are (according to Janeway):—

Infants..... 75- 90 m. m. Hg. (maximum systolic).
Children over two years.... 90-110 m. m. Hg. (maximum systolic).
Young adults.....100-130 m. m. Hg. (maximum systolic).
Older adults.....110-145 m. m. Hg. (maximum systolic).

Francis Ashley Faught of Philadelphia states that the average normal systolic blood-pressure at 20 years is 120, and for each year above 20, add ½ m. m. Hg. At 40, therefore, it should be 130 m. m. Hg.

A blood-pressure of over 145 before middle life, or of more than 160 after, must be considered as abnormal (Janeway).

Hypertension is also characteristic of puerperal eclampsia and lead colic.

According to Dr. Vaquez, hypertension may directly cause visual disturbances;—there may be amaurosis, homonymous hemianopsia, or a progressive loss of vision.

Hypertension may usher in convulsions;—e. g., puerperal eclampsia and in nephritis.

According to Dr. Leroy, every patient whose arterial tension exceeds 26 m. m. Hg. is liable to die suddenly.

To obtain the **maximum systolic** blood-pressure a broad arm piece should be used. When the pulse at the wrist is obliterated the height of the mercury column is equivalent to the maximum systolic pressure. Many observers claim that it is better to accept the reappearance of the radial pulse after obliteration than the initial disappearance of the radial pulse. The Janeway apparatus is best recommended. The **minimum diastolic** blood-pressure is obtained by letting the pressure fall 5 mm. Hg. at a time and at the same time watching the amplitude of the pulsations of the mercury column—and at that point where the greatest amplitude occurs, that point registers the diastolic blood-pressure.

DIFFERENTIAL DIAGNOSIS OF APICAL DISPLACEMENTS

Displacement Downward:

1. Hypertrophy.
2. Dilatation:

In these two cases the apex is usually within the nipple line. The biggest displacement occurs in aortic regurgitation, where we get a "Cor Bovinum" and the apex extends beyond the mammary line. For differential diagnosis between hypertrophy and dilatation, see page 20.

3. Hypertrophic Emphysema: due to:—
   (a) increased bulk of lung compressing the heart.
   (b) permanent inspiratory state of the lung.
   (c) hypertrophied right ventricle.
4. Mediastinal tumor: } For differential diagnosis between
5. Thoracic aneurism: } No. 4 and 5, see page 45.
6. Left side pleurisy with effusion.

Displacement to the Left (It is also usually **upward**):

1. Pericardial effusion: } For differential diagnosis between
2. Dilatation of the heart: } Nos. 1 and 2, see pages 13 and 20.
3. Right side pleurisy with effusion.
4. Chronic hyperplastic connective tissue processes in the left lung: chronic phthisis, interstitial pneumonia, adherent pleura.
5. Encroachment upon the thoracic cavity by abdominal distensions, be it gas, fluid or solid growths.

CACTINA PILLETS

FROM

CEREUS GRANDIFLORUS

IN CARDIAC THERAPY

It has been said that Cactus (Cereus Grandiflorus) belongs to the so-called digitalis group. While clinical evidence indicates that its ultimate action is that of a cardiac stimulant, it is certain that this stimulation differs somewhat from that effected by the foxglove preparations.

Dr. La Franca, the physiological chemist, reports his experimental and clinical observations ("Zeitung für Exper. Path. und Therap.," also referred to in the American Journ. of Med. Sciences), on the action of Heart Remedies. He has formulated the therapeutic action of various heart remedies in relation to their effects upon the irritability, conductivity and contractility of the heart muscle. He says that contractility of the cardiac muscle is increased by digitalis, spartein, convallaria, and cactus grandiflorus. On the other hand, strychnine, caffeine, sodium bromide and sodium iodide have no influence upon this property of the cardiac muscle. The irritability of the heart muscle, he says, is lessened by both digitalis and cactus, and that the irritability and conductivity are both increased by convallaria, strychnine, caffeine, sodium bromide and iodide.

The tonic stimulant action of cactus is explained by some authorities as an action upon the intra-cardiac ganglia and accelerator nerves through the cardiac plexus of the sympathetic; and secondly, by a direct stimulation of the cardiac muscle.

Thus Cactus, which is most conveniently administered in the form of Cactina (Pillets), the active therapeutic principles of the drug, has a sphere of action entirely its own, not replacing the digitalis group, but useful in many cases where the latter drugs are not only dangerous but contra-indicated. Of this there is a certainty, that after the administrations of cactus or its active preparation Cactina Pillets, the casual observer will notice a slight acceleration of the pulse, and not infrequently an increase in blood-pressure is perceptible.

An advantage of Cactina Pillets is that cumulative action never becomes manifest, even when administered for prolonged periods.

We are all familiar with the synergistic action of drugs. Cactus and digitalis are both cardiac tonics and may be used as such in selective cases. In the treatment of organic disease digitalis must be given first choice, but after once having established fairly good compensation we may then place the patient on Cactina Pillets.

It will be found that this will sustain the good effect brought by digitalis. Cactina Pillets are especially indicated in those cases where cardiac stimulation is to be a prolonged practice, e. g., in chronic cardiac derangements.

However, it is obvious that cactus preparations or Cactina Pillets cannot be employed in emergency cases where immediate heart stimulation is required, and it is always well to bear in mind that Cactina Pillets is a persuasive heart tonic rather than a therapeutic lash.

It often happens that one desires the strong action of digitalis on the heart without its marked vaso-motor action on the muscular coats of the arteries, vaso-dilators must then be employed to offset the arterial contraction. Nitroglycerine is the usual remedy employed. If, in these cases, the heart is not in immediate danger, Cactina Pillets may be tried as a substitute.

In, or convalescing from acute diseases which have weakened the heart and the circulation or in any case of protracted illness where a heart tonic is desired, Cactina Pillets are much safer to use than any of the digitalis preparations. A very noticeable change when employed as a heart tonic in the cases just mentioned is that the pulse becomes strong and regular and that the heart becomes more steadied.

In the so-called "weak heart" that accompanies myocardial degenerations, which results in weakening the heart muscle, Cactina Pillets are indicated. If the diagnosis of a weak heart can be made early, and the remedy is immediately prescribed and used judiciously, we may be able, in many cases, to retard the development of dilatation of the heart and its concomitant symptoms.

The existence of a properly compensating heart is a positive contra-indication to the employment of strong cardiac stimulants. However, the judicious administration of Cactina Pillets to a fairly compensating heart may control the subjective symptoms and contribute to the maintenance of cardiac balance.

Just as digitalis is our first choice in many of the organic diseases of the heart so does cactus as excellently presented in Cactina Pillets occupy the first place in the list of drugs that can satisfactorily control the so-called functional diseases.

The "tobacco heart" or "irritable heart" or the so-called "toxic-angina" are fortunately very amenable to the influence of Cactina Pillets. Tobacco acts upon the heart and the blood by virtue of the inhaled carbon dioxide, ammonia gases and nicotine, all of which are produced when smoking. The action of the aforementioned products upon the neurogenic apparatus of the heart is such as to cause palpitation, intermittent and irregular heart action, precordial pain and sometimes faintness and vertigo.

In all forms of tachycardia and intermittency from the abuse of tea, coffee, or alcohol, Cactina Pillets will give prompt relief.

In fact in all cases of cardiac neuroses, especially those in females, this remedy has "clinically" demonstrated itself as a dependable means of overcoming excitability and irregularity of the heart's action.

Cactina Pillets completely represent the drug, as each pillet contains one one-hundredth of a grain of Cactina, the active principles of Cactus, separated by special methods from the Mexican drug. The dose is from one to three pillets three or four times a day, according to the results desired.