CHAPTER III

DISEASES OF THE HEART AND PERICARDIUM

§ 25. Physiological Anatomy of the Heart.—Before considering systematically cardio-vascular disease, it is advisable to review the more important points regarding the physiological anatomy of the heart. The heart is developed as a tube of muscle which becomes bent on itself and develops diverticula, forming the chambers, auricular and ventricular, of the adult heart. This tube in the course of development becomes modified, but remains can be distinguished in the fully formed heart, and are of importance as they form the specialised tissues whose functions are the initiation and conduction of the normal cardiac impulse. This specialised system is made up of: (1) Sino-auricular node (Pacemaker), which is situated between the superior and inferior venæ cavæ, and with which the extrinsic nerves are closely associated; (2) a series of intra-auricular paths which connect the sino-auricular node with a similar structure;—(3) auriculo-ventricular node. Passing down from the auriculo-ventricular node is a neuro-muscular strand—(4) The Bundle of His, which has a special nerve and blood supply, and forms the main connecting muscular link between auricles and ventricles. It lies just under the endocardium, under cover of the septal cusp of the tricuspid valve. The Bundle of His divides into—(5) A main right and left branch. The right branch, passing along the right side of the septum under the endocardium, runs along the moderator band and curves backwards to terminate chiefly in the base of the right ventricle and papillary muscles. The left branch passes along the left side of the septum, finally terminating in the wall of the left ventricle in close association with the cells of Purkinje. The different parts of this system are shown in the annexed diagram, Fig. 9.

The heart has an enormous reserve capacity; the maximum output during exertion is about ten times that produced by the resting heart. The increase in the work done is frequently so sudden that the organ is subjected to great strains as the result of sudden mechanical efforts or violent emotions. Injury is prevented by a series of protective mechanisms: the more important of which may be summarised as follows: (1) Nervous Mechanisms.—*The Vagus* has two main sets of fibres in relation to the heart: (*a*) efferent fibres with the power of slowing, weakening or even stopping the beat; (*b*) afferent fibres (depressor nerve), which run from the arch of the aorta to the vaso-motor centre in the medulla and convey stimuli which cause peripheral relaxation, lower the systemic blood pressure and so relieve the left heart. Overaction of the vagal mechanism may produce fainting attacks. The focal point of vagal cardiac inhibition is the *carotid sinus*. Direct pressure on it, over the right internal carotid, in some individuals slows the heart and lowers blood pressure to such an extent that it causes syncope. Such pressure may be digital, or from a stiff collar or other external object. More usually the stimulus is psychological, or the result of physical pain. Vaso-vagal attacks may be abrupt, or of more gradual onset. In the former case they must be distinguished from epilepsy, and in the latter from syncope caused by cerebral anæmia associated with vasomotor failure. Bradycardia and very low blood pressure are the signs of a vaso-vagal attack. *Sympathetic* stimulation produces tachycardia.

(2) Pain Mechanism.—In most organs of the body a pain mechanism exists, the primary object of which is protection. In the case of the normal heart, the pain mechanism is one of the last called into play. *Afferent impulses* conduct painful sensations from the heart through sympathetic fibres. These leave the heart, and pass to the cervical and upper dorsal sympathetic ganglia of the left side, whence the grey *rami communicantes* of the five upper dorsal segments pass the stimuli on to the spinal cord.

Heart sensations rarely become painful unless there is damage to the heart muscle or to the pericardium. Exercise cannot produce it in a healthy adult. This mechanism will be referred to again in connection with angina (§ 51).

(3) Myocardial Mechanisms.—(*a*) In the absence of anoxæmia or toxæmia the myocardium maintains its tone. Excessive exertion produces no dilatation when such exertion has finished. In the diseased heart, however, dilatation occurs. It is probable that under certain conditions the tricuspid and mitral rings may relax so that the valves may be rendered for the time being incompetent and the pressure in the ventricles relieved. (*b*) The Moderator Band is a special band

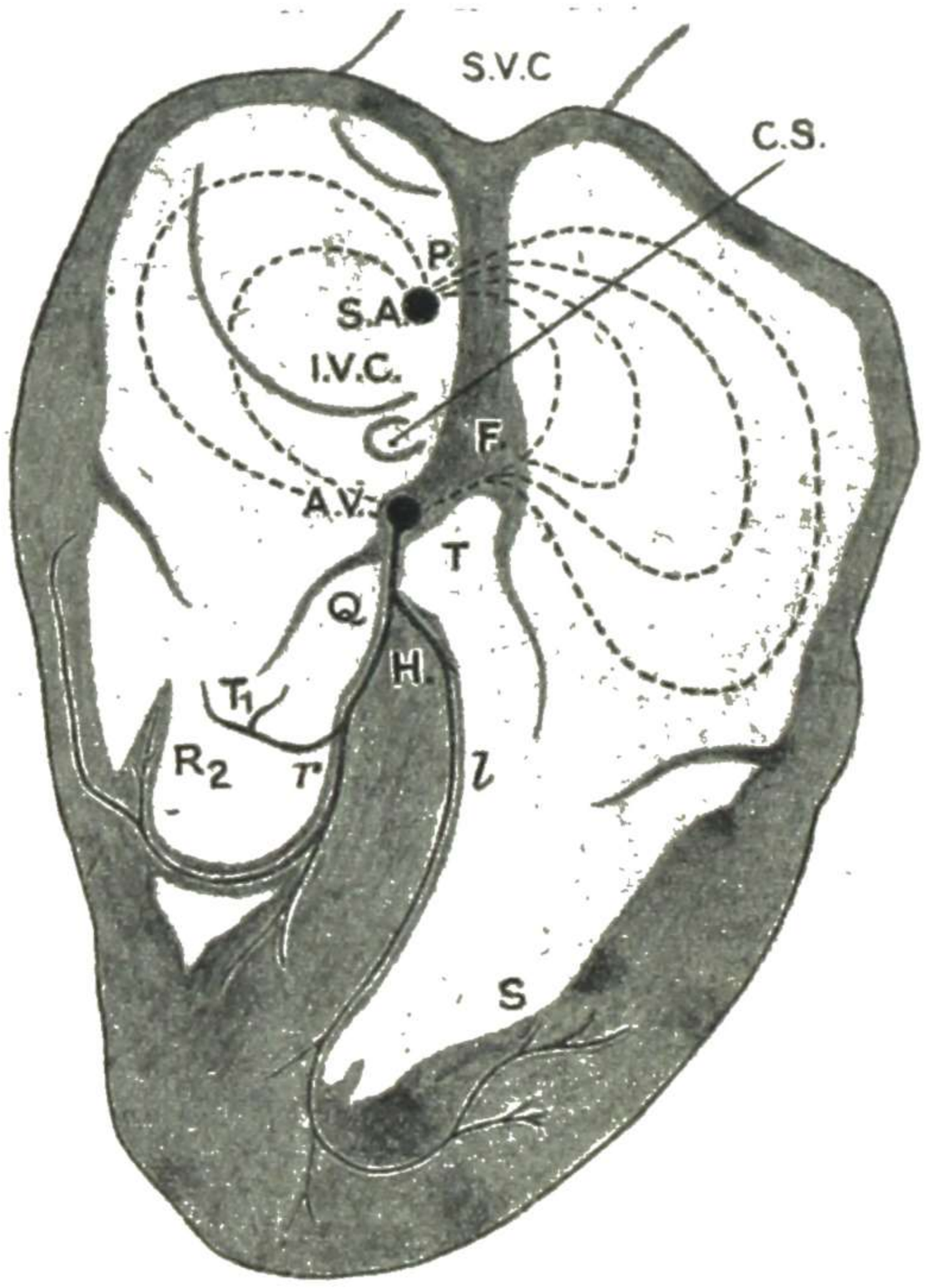


Fig. 9.—Diagrammatic section through the heart. S.A. = the sino-auricular node. A.V. = auriculo-ventricular node. The dotted lines joining S.A. and A.V.. = intra-auricular paths, which convey the stimulus to the ventricles. Running down from the A.V. node is the main Bundle of His [Q] which divides into a main right branch [*r*] and a main left branch [*l*], the former terminates chiefly in the right papillary muscle and base of right ventricle, the latter in the wall of the left ventricle.

of muscle which crosses the cavity of the right ventricle and is reputed to prevent over-dilatation of this cavity; it contains the right branch of the Bundle of His.

(4) Pericardial Mechanisms.—The pericardium is a tough fibrous bag, the main function of which is to prevent over-distension of the auricles.

PART A. SYMPTOMATOLOGY.

The general symptoms of cardiac disease, as distinct from the local signs referable to the heart, should be studied very carefully, inasmuch as the gravity of any given case depends not so much on the local signs present as on the general condition of the patient.

In the investigation of a case of cardiac disease the various methods have roughly the following relative values: history 50%, physical examination 25%, electrocardiography 10%, radiography 10%, and pathological

and other special methods of diagnosis 5%. It is thus clear that the importance of an accurate evaluation of symptoms is very great.

The CARDINAL SYMPTOM of cardiac dysfunction is **Breathlessness.** When there is marked failure, **Dropsy, Venous Engorgement** and **Cyanosis** are also present. **Pain, Palpitation** and **Cough** are found in certain cases. **Fainting Attacks,** although in the lay mind invariably attributed to the heart, have in practice only rarely a cardiac cause. **Sleeplessness** and **delirium** occur in cases of failure. **Fever** and its concomitant symptoms occur in acute affections. **Unexpected** or **Sudden Death** may terminate cardiac disease.

§ 26. Breathlessness, or Dyspnœa. Breathlessness may be present without cardiac disease; but it may be affirmed that no serious affection of the CARDIAC MUSCLE can exist without breathlessness. Dyspnœa is a physiological result of muscular exertion. It becomes pathological when evoked in excessive degree by an amount of exercise which previously had no such result. Two points to be elucidated are: (1) the amount of exercise now noticed to cause dyspnœa, and (2) the rapidity of development of the symptom. The severity of the myocardial failure and the progress of myocardial damage can in this way be estimated. The slightest degree of dyspnœa may be detected by observing that the scaleni and lower edges of the sterno-mastoids are brought into play at the end of inspiration.

Severe disease of the VALVES of the heart may exist for many years —provided the disability so caused is adequately compensated by muscular hypertrophy—without the patient having any noteworthy symptoms, or even being aware of its existence. When the heart muscle fails to compensate for the valvular defect, breathlessness appears. When the patient is unable to breathe on lying down, and the night is passed sitting upright in a chair, or propped up with pillows in bed, *orthopnœa* is present. It indicates pulmonary congestion and left-sided failure. It is absent in pure right-sided failure. This upright position relieves the embarrassed lung from the weight of the engorged liver, lowers the pressure in the venæ cavæ, assists the accessory muscles of respiration, and so reduces pulmonary congestion as far as possible. Towards the end in many cases of heart failure *Cheyne-Stokes' respiration* may be observed.

Sighing is frequently regarded as being due to organic heart disease, but this is not the case. It is suggestive of a cardio-vascular neurosis. It is frequently associated with vasomotor instability, with a labile heart rate and blood pressure, or may result from great nervous or bodily fatigue. The sighing is often long-drawn and occurs at frequent intervals.

OTHER CAUSES OF BREATHLESSNESS (DYSPNŒA).—Difficult breathing may arise in five different groups of disorders.

1. **Cardiac Disease.**—The dyspnœa of heart disease has no intrinsic features which distinguish it from that due to other causes, but it is often associated with cyanosis. There is also usually a history, or evidence, of some of the other symptoms of cardiac disorder. In cardiac

disease the amount of breathlessness present or the amount of exertion which can be taken without producing breathlessness are, of all symptoms, the most valuable indications as to the amount of inadequacy of the cardiac muscle (cardiac failure) present in any particular case (cf. § 43). If, in a dyspnœic patient, there is no cardiac enlargement, some cause other than heart disease must be sought.

2. **Embarrassment of the Heart by Neighbouring Structures,** such as mediastinal tumours, a large pleural effusion, ascites, a dilated stomach. Obesity may be a subsidiary cause of dyspnœa, but there is generally some associated myocardial change present.

3. **Laryngeal or Tracheal Obstruction.**

4. **Pulmonary Disease,** of which emphysema is the most common. In ACUTE PNEUMOTHORAX, dyspnœa of sudden onset is characteristic; pain in the side is usually present (§ 126).

5. **Blood Conditions.** Patients with a severe degree of ANÆMIA are often markedly dyspnœic; in addition they often have palpitation, hæmic murmurs, and œdema of the feet. Anæmic patients with dyspnœa prefer to lie flat; cardiac patients with dyspnœa prefer to be propped up. ACIDOSIS, due to diabetic ketosis or to uræmia, may cause dyspnœa, but this is present both at rest and on exercise.

Causes of Breathlessness which are apt to be overlooked.—The differentiation of the various forms of cardiac disease is given in the following pages; but, supposing *a patient over thirty-five or forty,* who complains of breathlessness, presents no definite signs of cardiac or pulmonary disease, nor any evidences of dyspepsia or anæmia, then there are certain conditions which should be *suspected:*

1. **Myocardial Degeneration,** often secondary to disease of the coronary arteries. The sounds and impulse may be feeble, and the other signs mentioned in § 57 may be present.

2. **Syphilitic Aortitis** with or without disease of the aortic valves.

3. Deep-seated **Aneurysm of the Aorta** and other **Intrathoracic Tumours** may give rise to the breathlessness and general symptoms of heart disease without the physical signs. In such cases the dyspnœa may be paroxysmal, and in neoplasm is often very severe.

In *a patient under thirty-five or forty* the three following causes of UNEXPLAINED BREATHLESSNESS may be suspected:

4. **Latent Pulmonary Disease,** and especially latent pulmonary tuberculosis, should always be suspected in cases of breathlessness without obvious cause.

5. **Pericardial Effusion,** also, is often attended by relatively few physical signs (§ 56).

6. When severe dyspnœa sets in suddenly in the course of cardiac or **Acute Renal Disease,** the chest should always be carefully examined, because hydrothorax may set in rapidly without any general dropsy or other warning symptom.

§ 27. Paroxysmal Dyspnœa is that form of dyspnœa which occurs in attacks from time to time. It is apt, as above mentioned, to occur in some cases of cardiac disease, especially in the last stages of myocardial degeneration, and in any given case attention should first be directed to the heart. But there are several other conditions which one would suspect in a patient in whom the chief or only symptom consists of paroxysms of breathlessness. If the paroxysms are cardiac in origin the heart is always enlarged; in the other conditions there is no cardiac enlargement, provided that there has been no hypertension.

1. Paroxysms of dyspnœa causing the patient to wake up at night are often one of the first symptoms of CHRONIC NEPHRITIS, and are spoken of by the patient as asthma; they are typical of cardio-renal failure (§ 372).

2. In ASTHMA, laryngismus stridulus, and whooping-cough, the attacks of breathlessness are typically paroxysmal.

3. NEUROTIC DYSPNŒA.—Some neurotic patients are liable to attacks of rapid respiration. These usually cease when the patient converses or thinks that he is not being observed, or during sleep. If prolonged, tingling in the fingers and other evidences of tetany are present (§ 778). The heart rate may be rapid in these attacks.

4. ANEURYSM and other INTRATHORACIC TUMOURS may give rise to paroxysmal dyspnœa before other signs can be made out.

5. ACUTE PULMONARY ŒDEMA (§ 118).

6. Enlargement of the THYMUS GLAND, whether due to neoplasm or to the condition known as LYMPHATISM, or status lymphaticus, in which there is general hyperplasia of lymphatic structure, associated with a persistent thymus, may cause paroxysmal dyspnœa, to which the name "thymic asthma" has been given (§ 37).

7. FOREIGN BODIES in the trachea and retropharyngeal abscess in children, and polypi or papillomata of the larynx, give rise to paroxysms of dyspnœa.

8. Sudden dyspnœa, coming on during vomiting, is the main indication of that rare accident, RUPTURE OF THE ŒSOPHAGUS. This dyspnœa is due to pneumothorax.

9. The LARYNGEAL CRISES of tabes dorsalis may take the form of paroxysmal dyspnœa.

§ 28. Cheyne-Stokes' Respiration (so called after its first observers) consists, in its typical form, of a series of eight or ten rapid inspirations gradually increasing in depth and rapidity, and then dying gradually away, each series being separated by a pause of some seconds (the stage of apnœa), in which there is little or no respiratory movement. It is due to lack of CO_2 in the blood and can be abolished by giving the patient inhalations of O_2 with 5 per cent. of CO_2. The hyperpnœic stage may produce such exaggerated movements as to wake the patient and cause a sensation of acute discomfort.

In a modified form, without the apnœic pause, Cheyne-Stokes' breathing is not infrequent. It is usually a serious symptom, and appears in cardiac patients *towards the end of life*. It has less significance at the extremes of life, for it may be observed during sleep in normal infants, and is occasionally compatible with a hale old age.

Its principal *causes* are as follows:

1. CEREBRAL ARTERIO-SCLEROSIS; 2. CARDIAC DISEASE due to coronary atheroma; 3. URÆMIA; 4. Rapidly increased INTRACRANIAL PRESSURE such as occurs with apoplexy, tuberculous meningitis, and in some cases of cerebral tumour; 5. SUNSTROKE.

Prognosis.—In the presence of organic heart disease, especially if there is also hyperpiesis, the expectation of life is reduced to two or three years.

Treatment.—The best methods of treatment are continuous oxygen administration, or in some cases the use of morphia, gr. $\frac{1}{4}$ by injection. The writer has found a course of daily intravenous injections of cardophylin (euphyllin) useful, in doses of 0·48 G. in 20 c.c., for a period of 7–10 days.

§ 29. Dropsy is a chronic effusion of fluid into the skin and subcutaneous tissues (when it is known as anasarca or œdema) or into a serous cavity (as in hydrothorax, hydropericardium, ascites). The former, **Anasarca,** is the variety of dropsy we are now concerned with; for it is a very constant feature of some forms of cardiac disease. General anasarca has to be differentiated from myxœdema, in which the swelling is harder, and does not pit on pressure. It is best to apply the pressure over a bone, such as the lower end of the tibia, on its inner aspect.

Causes.—The causes of localised dropsies are given in Diseases of the Extremities (§ 570). There are *three varieties of general anasarca*, which differ from each other both pathologically in their origin, and clinically in the course they pursue.

1. **Cardiac Dropsy** is partly due to the raised intracapillary pressure resulting from the venous engorgement of right-sided heart failure, and partly to the malnutrition of the capillary endothelium resulting from the slowed peripheral circulation. (1) It *starts*, and throughout the case predominates, in the *most dependent parts*, that is to say, in the legs if the patient has been walking about, or in the lower part of the back if he has been lying in bed. On inquiry, the patient may complain that the ankles swell towards evening around the top of the boot. (2) Other signs and symptoms of cardiac enfeeblement or dilatation are present. (3) In the history of the case dyspnœa will have *preceded* the dropsy. Dropsy does not occur with equal frequency in all forms of cardiac disease. The œdema which complicates pulmonary disease has the same features as cardiac œdema, because it is the resulting right ventricular failure which produces it: but here pulmonary congestion and orthopnœa are absent. Dropsy, in the absence of dyspnœa or of cardiac enlargement, is not due to heart disease; some other cause, such as phlebitis or obstruction to the vena cava, must be sought. Dropsy is often present in renal disease without dyspnœa; it is then usually a general œdema.

2. **Hepatic Dropsy** (1) usually begins and predominates *in the abdomen* (ascites), although the legs may swell subsequently by reason of the pressure of the fluid on the veins within the abdominal cavity. (2) There may be also enlargement or other signs of the liver affection which has given rise to the condition; and if these be absent some other cause of obstruction to the portal vein should be sought (§ 260). (3) The dyspnœa will have *followed* the abdominal enlargement.

3. **Renal Dropsy** is (1) *general in its distribution* from the beginning, occurring in the legs and eyelids at the same time; though it is probable that the œdema around the eyes on rising in the morning first attracts the attention of the patient or his friends. (2) Examination of the urine reveals the features of renal disease, but it should be remembered that some degree of albuminuria is common in heart failure. The presence of many casts is strong evidence of a renal origin. (3) The patient presents a characteristic pale or waxy appearance. In some cases of general anasarca associated with albuminuria the question arises whether the dropsy is renal or cardiac. This may sometimes be answered by finding the liver enlarged, for this is a natural sequence of right-sided heart failure, though not of renal disease. The frequent association of chronic renal disease and cirrhosis of the liver must not be forgotten.

Prognosis.—Dropsy usually indicates a severe degree and a late stage of heart disease. The outlook varies greatly, according to the cause.

Treatment.—The principles are as follows: Absolute rest in bed; raise the limbs and keep the patient warm. Reduce the fluid intake to

30 to 40 oz. daily; give small, dry, palatable, non-fermenting diet with a reduced sodium chloride content; also give digitalis and some diuretic—such as mersalyl ½ to 2 c.c. intramuscularly or intravenously, preceded for one day and accompanied by the administration of ammonium chloride gr. 15 t.d.s., p.c. (in capsules). Other diuretics to be tried are theobromine and sodium salicylate (diuretin) gr. 15 t.d.s., p.c.; theophyllin and sodium acetate gr. 2 to 5 t.d.s., p.c., and urea gr. 120 t.d.s., in lemon juice and water. If the dropsy in the limbs is extensive, wrap them in cyanide gauze or some other dressing, as they are liable to eczema, erythema, cellulitis or exfoliative dermatitis. Should the above methods fail, multiple small punctures with needles or with small incisions through penicillin cream, or the insertion of Southey's tubes under strictly aseptic methods may be practised. The patient's legs should have been dependent for several days to allow the fluid to accumulate. The abdomen or pleural cavity may require tapping. It is never wise in cardiac œdema to allow fluid to accumulate in the pleural cavities.

Obscure Causes of General Anasarca.—If, in a patient who complains of dropsy, no marked evidences of cardiac, renal, or hepatic disease are discoverable, the following causes may be *suspected*:

1. In women with **poor muscular tone,** but otherwise normal, œdema of the legs and feet is found. It is common in multiparæ and the left leg is often the more severely affected. It is especially marked after a preceding phlebitis. These patients generally have the symptom for many years, especially in hot weather; no marked dyspnœa and no cardiac enlargement are found.

2. **Anæmia** is not infrequently attended by some swelling of the ankles at the end of the day. Swelling of the feet and ankles may be present in the last stages of many exhausting diseases, such as phthisis, in septic and anæmic states, and in cases of insufficient nutrition and old age. 3. Among the causes of dropsy rare in this country are **Beri-Beri** (§ 795) and **Epidemic Dropsy.** Epidemic dropsy occurs in sporadic outbreaks amongst rice-eaters and those suffering from Vitamin B deficiency. This "nutritional" œdema has several causative factors, all secondary to the avitaminosis; they are myocardial failure, lowered serum albumen, and possibly an excessive (for the patient) intake of fluids and sodium. 4. Milroy first described a hereditary œdema in which a solid œdema of the legs existed in many members of a family (§ 570). 5. **Congenital general œdema** (hydrops fœtalis, § 551 V) is usually fatal.

Venous Engorgement. In an advanced stage of heart failure the veins are continuously distended. This is visible chiefly in the veins of the neck and is increased during systole. The level below which the neck veins remain distended in a normal individual is the lower border of the manubrium sterni. In heart failure the veins remain swollen to a higher level according to the amount of increase in the pressure. If the neck veins are in a state of distension and there is no dyspnœa nor cardiac enlargement, the cause is to be sought in some lesion producing intrathoracic venous obstruction, such as mediastinal tumour, aneurysm or constrictive pericarditis.

§ 30. Cyanosis, or bluish discoloration of the body surface, is due to an abnormal amount of reduced hæmoglobin in the peripheral capillary blood. This may be the result of (1) slowing of the peripheral circulation,

(2) slowing of the general circulation, (3) insufficient aeration of the blood in the lungs, (4) admixture of venous with arterial blood in congenital heart disease, (5) abnormal blood conditions. When the blood stream is slowed, more oxygen is taken from the blood by the tissues. *General* cyanosis affects also the mucous surfaces. *Local* cyanosis, as in Raynaud's disease, may be differentiated from general cyanosis, as in heart failure, by immersing the patient's hand in hot water for 10 minutes; in the former case the skin colour becomes pink, in the latter it remains blue.

Cardiac cyanosis is most pronounced in heart failure secondary to chronic pulmonary disease, or to mitral stenosis. It is found in congenital heart disease with pulmonary stenosis, or with gross auricular or ventricular septal defect. **Respiratory** causes of cyanosis are anatomically diffuse, such as emphysema, bronchitis, asthma, pulmonary œdema, pneumonia, miliary tuberculosis and pleurisy with effusion.

When there is polycythæmia, as in Vaquez' disease, the increased viscosity of the blood produces a slowing of the peripheral circulation and therefore some cyanosis.

Local cyanosis may be unilateral, as for example when an intrathoracic tumour is pressing upon the venous return of one arm. A venous thrombosis produces a similar effect. And see § 576.

Treatment of cyanosis.—The treatment of cyanosis of pulmonary origin depends on the cause. Oxygen, given by a B.L.B. or other type of mask, intra-nasal catheter, or oxygen tent or chamber, is most effective in emphysema, bronchitis, asthma, pulmonary œdema and pneumonia. Five per cent. CO_2 should be used with the oxygen, either when the breathing is shallow or when there is pulmonary collapse. Oxygen given by funnel is useless in every type of disease. Paracentesis of a pleural effusion always helps cardiac anoxæmia and cyanosis. In the absence of pulmonary œdema or bronchitis, oxygen is not of much use in heart failure, for the slowed circulation rate allows more, not less, time for the pulmonary capillary blood to be fully oxygenated. Heart failure with cyanosis is often benefited by rapid venesection and removal of 10 to 20 oz. of blood, the particular indication for this being distension of the veins in the neck.

§ 31. **Polycythæmia Vera** (Synonyms: Vaquez' disease, Erythræmia, Splenomegalic polycythæmia).—This is a disease in which there is an overgrowth of the red cell forming tissue in the bone marrow. (i.) The patients are usually middle-aged and complain of headache, vertigo and other nervous symptoms, pains in the limbs and dyspnœa. (ii.) They are easily recognised by the redness of their complexion, which often deepens to cyanosis, especially in cold weather. All the superficial vessels are dilated. (iii.) The spleen is enlarged to a variable extent. (iv.) The blood shows a marked increase in the red cells up to 13,000,000 per c.mm. with 120–160% hæmoglobin and a colour index of 0·7–0·9. Polychromasia and a few normoblasts are usually present and the platelets and white cells are increased: myelocytes may be present. Owing to the relative increase in the number of red cells, the viscosity of the blood is raised and also the blood volume. (v.) Hæmorrhage may occur from the distended vessels at any site. A variety, **Gaisbock's disease** or *polycythæmia hypertonica*, is described without enlarged spleen, but with high blood pressure and arterio-sclerosis. It follows a chronic course, with death, sometimes after many years, from heart failure, cerebral hæmorrhage or thrombosis. In **Ayerza's**

disease the polycythæmia is secondary to an obliterative endarteritis of the pulmonary artery. It is associated with extreme dyspnœa, a normal or low blood pressure, no splenic enlargement, but a dilated conus of the pulmonary artery seen radiographically.

Treatment may be (*a*) by removing excess blood with repeated venesection—this is the safest method; (*b*) blood may be destroyed by a hæmolytic poison, *e.g.*, acetylphenylhydrazine gr. ½ t.d.s. for 7–10 days, followed by a rest, as it is a cumulative poison; (*c*) erythropoiesis may be depressed by radiation to the bone marrow or by internal radiation using intravenous radiophosphorus—give 3–8 millicuries initially, followed by 1–5 millicuries each three to six months so long as the red cell count remains above six million. Treatment must be controlled by blood counts. It is essential to establish a correct diagnosis, for if the polycythæmia is secondary to pulmonary or cardiac disease, treatment with drugs is harmful.

§ **32.** Rare causes of cyanosis are: **Sulph-hæmoglobinæmia** and **Methæmoglobinæmia.** The most prominent symptoms are (1) cyanosis of a peculiar greyish leaden colour; (2) marked weakness, vague pains and collapse; (3) constipation, sometimes alternating with offensive diarrhœa and most marked in sulph-hæmoglobinæmia; (4) periods of relative freedom followed by exacerbations. Two factors appear to be necessary for the formation of these compounds: (i.) some activating substance in the blood, and (ii.) absorption of sulphur or reducing substances from the bowel. It has been shown that sulphonamide compounds, metadinitrobenzene, trional, sulphonal, pamaquin, potassium chlorate, acetanilide, phenacetin and related compounds and certain aniline dyes, can act as such sensitising agents, and a history of taking these drugs can usually be obtained. Certain nitroso-bacilli, which have the power of reducing nitrogen compounds, have been isolated from the saliva and bowel, and are probably causal in those cases without a drug history (*enterogenous cyanosis*). Magnesium sulphate and other saline cathartics predispose by increasing the fluid content of the bowel, and hence bacterial fermentation. The *diagnosis* is based on the history and cyanosis without a cardiac or respiratory cause; it can be verified by spectroscopic examination of the blood (Plate IV). *Prognosis.*—This condition is not fatal, but may prove very resistant. In *treatment*, any possible sensitising drug must be excluded, and it is advisable to restrict sulphur in the diet; the constipation must be relieved by liquid paraffin or enemata. In severe cases, inhalations 2–3 times a day of 5% carbon dioxide in oxygen may be given. In cases due to nitroso-bacilli a vaccine of these organisms may be of value. The cyanosis of methæmoglobin usually disappears within 48 hours of ceasing to take the drug, whilst that of sulph-hæmoglobin may persist for very much longer. Methylene blue helps to relieve cyanosis: it is given in doses of gr. 1–2 t.d.s. by mouth, or 1–2 mgm. per kilo intravenously. Ascorbic acid is effective in cases of familial methæmoglobinæmia.

A **Sallow Hue** of the skin is common in infective endocarditis. This sallowness is distinguished from jaundice by the absence of the yellow colour from the eyeballs and the absence of bile in the urine. True jaundice, however, does arise in cardiac disease, as a result of the hepatic congestion of severe cardiac failure.

Clubbing of the fingers is found in heart disease in the following conditions. (1) Congenital pulmonary stenosis is nearly always accompanied by clubbing. (2) Clubbing is found in malignant endocarditis. (3) Rarely, clubbing is found in cases of chronic rheumatic carditis. (4) An aneurysm pressing on or affecting the flow in one subclavian artery may produce clubbing of the affected side (and see § 568).

§ **33. Pain in the Chest** is absent in most forms of heart disease. It is present in coronary disease, less frequently in aortic insufficiency, and

occasionally in mitral stenosis. Pain due to heart disease is usually proportional to the amount of exercise the patient is taking: it is generally substernal, sometimes præcordial in position and affects the upper rather than the lower part of the chest. It may radiate to either arm or to both, or to the neck or jaw (§ 51). Many cases of neuro-circulatory asthenia (effort syndrome) suffer from pain which is often submammary; hyperæsthesia of the præcordium suggests a functional rather than an organic lesion.

The causes of præcordial or cardiac pain are:

(*a*) Arising from **Organic affections outside the heart and pericardium.** Intercostal fibrositis or "rheumatism," often known as intercostal neuralgia, especially that which precedes or follows herpes zoster; pleurodynia; neoplasms; pleurisy and pneumothorax (§ 103); spinal caries and carcinoma of the vertebræ, and tumours eroding the bones; the crises of tabes dorsalis; aneurysm. Muscular thoracic pain is increased by coughing or other muscular effort, and abolished by injection of a local anæsthetic; pleural pain is worse on breathing; herpetic pain is constant, and the pain of neoplasm is often worse at night.

(*b*) The pain may be of **Cardiac origin,** in which case it is usually called Angina (§ 51). This is a loose term and refers to three separate clinical conditions: (1) CORONARY THROMBOSIS (§ 52) produces an anginal pain, often of great severity. The onset is rapid, and the pain persists, with or without remission, for a period of three to six days, during which time its intensity slowly subsides. (2) ANGINA OF EFFORT is characterised by an anginal pain which comes on during exertion, disappearing again when exertion ceases; its intensity is directly proportional to the amount of exercise taken. (3) SPASMODIC ANGINA leads to paroxysmal attacks of very severe anginal pain, induced by exercise, emotion or cold, and relieved by nitrites; the pain is not proportional to exercise, and does not begin to subside directly exercise is stopped; angina of effort nearly always co-exists. These forms of pain are probably due to an interference of blood flow through the coronary circulation. The interference in coronary thrombosis is permanent; that in angina of effort is partial, but is relatively increased when the heart activity is increased by exercise. The mechanism of an attack of spasmodic angina is not yet understood (see § 51).

(*c*) Pain of a cardiac type is also found in **neuro-circulatory asthenia,** effort syndrome, or disordered action of the heart (§§ 34, 53). The pain may be little more than a dull ache, when the term left submammary pain is often used, but it is sometimes acute and radiates to the left arm, thus simulating severe angina of effort or spasmodic angina. In such cases a careful history will reveal that it is not quantitative to exertion, is left-sided rather than central, and is closely related to fatigue or to emotion. The term angina innocens (§ 53) is a useful stimulus to correct diagnosis. In some cases intercostal or subscapular fibrositis seems to be a causative factor, but heart consciousness or fear are often the reasons for localisation of the pain to the præcordium. Sharp sudden stabs of

pain sometimes occur in these cases, and accentuate the dull left-sided ache: syncopal attacks often follow these sharp stabs of pain. Organic heart disease is never indicated by pain of this type.

In cases of unexplained pain in the chest, and in the absence of cardiac signs, *mediastinal tumour* or *aneurysm of the aorta*, either of the arch or of the descending aorta, should always be suspected, and an X-ray examination made (§§ 80, 81).

Disease of the heart may also be an indirect cause of pain elsewhere than in the præcordium. For instance, with the engorged tender liver which is commonly associated with failure of the right auricle and ventricle, the muscles and skin of the abdominal wall are often also tender. In coronary thrombosis and in acute pericarditis the pain may be referred entirely to the upper abdomen. A simple cause of epigastric tenderness which must never be forgotten in cases of heart and lung disease is muscular strain of the upper rectus muscle and diaphragm from the exertion of constant cough. Pain in the shoulder, arm, neck and jaw is common in disease of the first part of the aorta; it is usually accompanied by superficial tenderness.

In the *treatment* of præcordial pain an endeavour should be made to ascertain and relieve the cause.

§ 34. Palpitation is consciousness of the heart's action. It arises under two sets of conditions, non-cardiac and cardiac. The essential symptomatic difference between the two types of palpitation is that in the *cardiac* group the onset is felt to be absolutely abrupt, as also in many cases is the termination of the paroxysm. The *non-cardiac* group is the larger and less serious; it includes:—

1. In **Anæmia** the palpitation is a frequent and often distressing feature.

2. **Dyspepsia** is a more common cause of palpitation than is cardiac disease. In such cases it often comes on at night, especially after a heavy meal. It may, in these circumstances, be accompanied by morbid dreads —*e.g.*, of impending death—by breathlessness, "night starts," cardiac pain, and other cardiac symptoms.

3. Certain **Local Conditions,** such as thoracic or abdominal tumour, or dilated stomach, which hamper the heart's action, may produce palpitation, although the heart be healthy.

4. In **Graves' Disease** (exophthalmic goitre) violent palpitation and increased rate of the heart are prominent features. In quite a number of cases this and the other nervous symptoms of the disorder exist for months before the two diagnostic features—thyroid enlargement and exophthalmos—become obvious. Graves' disease should always be suspected in cases of persistent palpitation (§ 186). In the type of thyrotoxicosis known as toxic adenoma, cardiac disease with congestive failure and auricular fibrillation may exist without exophthalmos, and the true cause may thus escape notice (§ 190).

5. Early stages of **pulmonary tuberculosis** (§ 131).

6. **Nervous** conditions, such as fright, fear, or other emotion, especially after an exhausting illness. It also occurs in hysteria and anxiety neurosis.

7. **Effort syndrome,** neurocirculatory asthenia, Da Costa's syndrome, cardiac neurosis, are all terms descriptive of a condition seen frequently in civilian life, but rising into prominence in times of war. The symptoms

are largely cardio-vascular—extreme lassitude and fatigue, dyspnœa on slight exertion, palpitation, præcordial discomfort, submammary ache and angina innocens, are the commonest cardiac manifestations. In addition, vasomotor and psychological abnormalities are often present. Undue sweating, especially of axillæ and hands, tachycardia and lowered blood-pressure on standing upright, fainting attacks and postural dizziness, are common. Psychologically there may be anxiety or hysteria, but far more frequently there is an idiosyncrasy of character rather than a neurosis, the individual being of the shy, hypersensitive, introspective type, who has avoided as far as possible situations in childhood or in adult life involving friction and physical or mental exposure and stress.

Treatment consists first in the thorough exclusion of all physical abnormalities. This is followed by explanation to the patient of the causes of his condition, and then by progressive exercises and interesting occupations, often while resident in a special treatment centre.

8. The excessive use of certain **Drugs** or **Articles of Diet,** notably **tobacco, tea, coffee, and alcohol.**

Cardiac causes include : (1) gross cardiac lesions ; (2) auricular flutter ; (3) auricular fibrillation ; and (4) paroxysmal tachycardia. These conditions are dealt with in Section C (§§ 63 *et seq.*).

Cough is a symptom which belongs chiefly to diseases of the lungs (§ 101), but it is met with in diseases of the cardio-vascular system in two circumstances. (*a*) Firstly, the lungs are very often involved in left-sided failure ; mitral stenosis produces a chronic pulmonary congestion, and coronary disease with left ventricular failure causes an acute or a chronic pulmonary congestion or œdema. The acute form is known as acute pulmonary œdema ; this produces a sudden attack of severe cough, with copious, frothy, albuminous sputum, which may be pink (§ 118). (*b*) Secondly, from pressure. When an aortic aneurysm presses on th recurrent laryngeal nerve, a peculiar dry, brassy cough is present. Peri carditis or an enlarged left auricle in mitral stenosis may produce cough Cough after effort may indicate heart failure.

§ 35. In **Syncope** there is transient loss of consciousness, due to anæmi of the brain. It is often preceded by giddiness, nausea, and a feeling o faintness. The face is ashy pale and the pulse and respiration feeble Its advent is usually sudden, but recovery, after the attack has laste some minutes, is gradual. Syncope is rarely caused by organic heart dis ease. With rare exceptions *patients with heart disease do not faint.*

Diagnosis.—Syncope has to be distinguished from *epilepsy* (§ 718*a* (1) Epilepsy is sometimes preceded by an aura, though this is eviden to the patient only. Its advent is more sudden than syncope, th duration of the attack is shorter, and the return to consciousness equall sudden and complete. (2) Syncope is rare without some definite dete mining cause, although it may be of a trivial nature—such as a heate room, or the sight of blood. *Aural vertigo* may be mistaken for syncope for differential features, see § 692.

Causes.—(1) Deficiency of blood, *e.g.*, hæmorrhage. (2) Vasomotor instability is seen in the common form of faint in which the abdominal vessels suddenly lose their "tone," dilate, and retain blood which is needed elsewhere. (3) Fainting is often due to vasovagal attacks (Lewis), in which as a result of disturbance of the carotid sinus or of the depressor nerve, vagal slowing of the heart is produced, which must not be confused with the type described by Gowers (§ 720). The onset is usually gradual, sometimes sudden. The patient sweats, loses consciousness, possibly as a result of some fright or other emotional disturbance; the heart rate is slowed and the blood pressure falls. The heart is not diseased, and the prognosis is good. (4) Senile syncope gives rise to attacks, preceded by giddiness, in old people who are the subjects of arterial degeneration (see § 719).

The **Vasomotor** group is the largest. The "faints" occur chiefly in the upright position and in young, anæmic, and nervous females and in boys at puberty; who, when exposed to grief, bereavement, or any sudden emotion, or too hot rooms full of vitiated air, develop the familiar "fainting attack." (See also postural hypotension, § 88.)

Predisposing causes are:—(1) Anæmia, debility, hunger, or starvation; (2) diminished resistance in the peripheral and splanchnic arteries, such as occurs with excessive heat, as in hot rooms or Turkish baths; (3) sudden assumption of the erect posture, as in jumping from bed, may produce syncope; (4) sometimes, in addition to the preceding, the splanchnic veins are suddenly dilated when the intra-abdominal pressure is rapidly lowered, as by emptying the bladder or by rapid paracentesis, and this leads to anæmia of the brain and syncope.

Cardiac causes.—Certain cases of aortic incompetence or stenosis, Stokes Adams' attacks in heart block, rare cases of auricular flutter, or paroxysmal auricular fibrillation. In aortic stenosis there may be a direct relationship between exercise and fainting.

Prognosis.—Syncope in the young is usually not organic in origin, whereas in the aged it is generally a proof of cardio-vascular degeneration. In the former, therefore, it is usually as trivial as in the latter it is serious —the gravity depending upon the nature of the lesion.

Treatment.—Place the patient immediately in a horizontal position with the head low. This may be most readily done on the floor, but if there is little space, instruct the patient to bend forward and lower the head between the knees. Apply ammonia to the nostrils, throw cold water on the face. If recovery does not promptly take place, and the pulse be feeble, a hypodermic injection of leptazol or niketham ide B.P. (coramine) may be resorted to. For further treatment, see Collapse (§ 239). The underlying cause must be carefully sought and treated when the patient has recovered from the urgent syncopal condition.

Sleeplessness is a distressing symptom of severe heart failure, and is due to slowing of the cerebral circulation. Morphia has no deleterious effect upon the heart, but it should only be used for short periods of time owing to the danger of habit formation; it is contra-indicated where there is much bronchitis. Paraldehyde in full doses, soluble barbitone

B.P. (medinal), hexobarbitone B.P. (evipan) and phenobarbitone are useful. Oxygen intranasally is helpful when pulmonary œdema co-exists.

Delirium, generally worse at night, is a more severe result of the same cerebral anoxæmia. Should the remedies above mentioned fail, hyoscine hydrobromide gr. 1/100 by subcutaneous injection may be necessary, and can be repeated after 4 hours.

Pyrexia and its concomitant symptoms (see Chapter XV) are present in many *acute disorders* of the heart and pericardium. The temperature in malignant endocarditis is usually of an intermittent or remittent type, with an irregular range, as in other forms of septicæmia.

§ 36. **Sudden** or **Unexpected Death** is not a common occurrence in patients with heart disease.

1. In *aortic valve disease*, stenosis more often than regurgitation, death may suddenly supervene during apparently good health.

2. In *acute coronary infarction* or after it has healed, in *myocardial degeneration*, in *syphilitic aortitis* involving the coronary orifices, unexpected death may happen, the mechanism here being the onset of ventricular fibrillation, which condition is incompatible with life.

3. *Pulmonary embolism* from previous phlebitis, *fat embolism* from skeletal trauma and *air embolism* from thoracic paracentesis, may cause sudden death.

4. Nerve diseases which in their progress involve the *medulla* terminate suddenly; and thus, among the rarer causes, atlanto-axoid disease and syringomyelia may be mentioned.

5. Emotional shock, injuries to the head, and other conditions acting on the *nervous system* by shock (§ 239).

6. *Hæmorrhage* into a previously silent *cerebral* or *pontine tumour* may also cause this.

7. *Poisons.*—Prussic acid acts very rapidly; others acting less quickly are cocaine, carbolic, volatile and non-volatile narcotics and anæsthetics.

8. Sudden rupture of a large cyst, an internal organ, acute disease of the suprarenals, or other cause of *Surgical shock* (§ 239).

9. Foreign bodies in the trachea, or other causes suddenly stopping the respiration (*asphyxia*)—*e.g.*, reflex apnœa from irritation of the pleura (pleural shock).

10. Status lymphaticus (§ 37).

§ 37. **Status Lymphaticus** (*Lymphatism*) is a rare condition frequently unrecognised during life, but it is a cause of sudden death in children and young adults. There is overgrowth of the thymus gland and of the lymphatic tissues throughout the body. There may be no symptoms, the first evidence of the existence of the condition being death after a trivial shock, such as a plunge into a cold bath, a hypodermic injection, or the first touch of the knife in a minor surgical operation. The patient is flabby and pale and the physical signs are indefinite, consisting only of hypertrophied tonsils and adenoids. In other cases the enlarged thymus causes dulness beneath the upper part of the sternum, the spleen is palpable, and there may be overgrowth of adenoid tissue at the base of the tongue. Subjects of this diathesis must be guarded against anæsthetics, sudden shocks, or exertion. X-ray application to the thymus is the treatment of choice and appears to be successful.

PART B. PHYSICAL EXAMINATION.

§ 38. Landmarks of the Chest.—There is a *ridge* on the sternum between the manubrium and the gladiolus; it can always be felt opposite the second costal cartilage; and the other ribs can be counted from the second one. The *nipple* is usually situated just external to the fourth costal cartilage, near its junction with the rib. At the back, the *lower angle* of the scapula just covers the seventh rib; and the *scapular line* is a vertical line drawn through the inferior angle of the scapula. The position and relations of the heart can be studied in Fig. 11, which is a sketch taken from the cadaver.

Inspection.—First inspect the patient from the foot of the bed. In a cardiac case the bed-ridden patient is almost invariably propped up. The appearance is often characteristic: The throbbing neck vessels of aortic regurgitation; the malar flush of mitral stenosis; the undergrown body and reddish-blue appearance of congenital pulmonary stenosis; the pinched patchy face, with the tortuous temporal arteries, and the often wasted body typical of cardio-vascular degeneration; the sallow toxic, anxious face of infective endocarditis; the large white face of renal disease; the blue face seen in congenital heart or failing mitral disease; the apprehensive look of the patient with angina, or the pale, puffy face of pericarditis, are all characteristic.

Other points to look for are the respiratory rate, depth and rhythm: cyanosis: engorgement, pulsation or otherwise of the jugulars: presence or absence of carotid pulsation: epigastric pulsation: thyroid enlargement: clubbing of the fingers and, if present, whether the fingers are blue (congenital heart disease) or white (infective endocarditis). Œdema around the ankles should be noted.

The abdomen should be examined for distension, and the importance of this sign, with its serious cardiac embarrassments, should not be underestimated. The presence of engorged veins, diminished respiratory movement, and the presence of ascites should all be observed.

Should the patient be confined to bed, attention should be directed to the position in which he lies or which he assumes. When the chest has been exposed, its shape and movements, any bulging of the præcordium (cardiac disease in early life before the chest has ceased growing) should be noted; also the cardiac impulse, its position and character, special attention being directed towards whether it is heaving (the true sign of cardiac hypertrophy) or slapping and diffuse in character. Systolic recession, indicative of adherent pericardium, should be looked for, not only in the region round the apex, but in the region of the epigastrium and also in the back (Broadbent's sign).

§ 39. Palpation and the Localisation of the Apex (see Figs. 11 and 12).—The apex beat is the point farthest downwards and to the left at which the cardiac impulse is distinctly felt. After inspection it should be first palpated by the flat of the hand, and then localised with the finger tips.

In an adult male it is normally situated in the fifth interspace ½ inch to the inner side of the mid-clavicular line, at a distance of about 3 inches from the mid-sternal line. *These and other cardiac measurements vary with the age* [1] *and proportions of the patient*—a fact which is apt to be forgotten.. The *most external* portion of the apex beat should be marked by a dot with an aniline pencil. At the level of the apex, measure and note the distance from the mid-line to the apex; measure also the distance from the middle of the neck to the middle of the left clavicle. In health these measurements should be the same, or at least the apex should not lie to the left of the mid-clavicular line. Thus at the first examination the position of the apex can be accurately defined, in terms of the mid-clavicular line. Further measurements need only be made from the mid-line, provided the patient does not grow. The principal features to observe about the apex are—its POSITION, CHARACTER and FREQUENCY. The beat of the left ventricle is felt as a forward thrust, if the myocardium is healthy; that of the right is less well defined. It is important to bear in mind that the apex beat is considerably modified if the apex happens (as is not infrequent) to pulsate precisely behind a rib. Only when the apex beats in an intercostal space can the three above features be satisfactorily noted. The apex can sometimes be felt more distinctly when the patient leans forward. In dextrocardia the apex is on the right side of the chest.

Roughly speaking, two abnormal types of apex beat can be recognised: (1) heaving; (2) slapping. A *heaving* apex beat can be recognised by the forcible lift which the fingers experience at each systole when pressed over the apex. It is *the* sign of cardiac hypertrophy and is typically met in cases of aortic regurgitation, hypertension, and, with modifications, in adherent pericardium. The *slapping* apex beat means a poorly contracting left ventricle, and this occurs in three conditions: (1) When the ventricle is badly filled, badly stretched and therefore badly stimulated, and consequently contracts badly; *e.g.*, mitral stenosis. (2) When the muscle has degenerated from coronary arteriosclerosis. (3) When the muscle is poisoned as in diphtheria.

In *hypertrophy* of the left ventricle the apex beat is displaced chiefly downwards, and the cardiac impulse is forcible and heaving. In hypertrophy of the right ventricle there is pulsation in the epigastrium and in the lower interspaces, but the apex is in its normal site. With *dilatation* the impulse is diffuse and weak and the apex beat is moved to the left. The apex is *displaced* in cases of empyema or pleurisy with effusion; if the latter be on the left side, the apex may even be displaced beyond the

[1] In the child the heart normally differs a good deal from that of the adult. The apex is outside the nipple line until 6 years of age; it is, moreover, often in the fourth space. The right cardiac dulness extends slightly beyond the right margin of the sternum, while on auscultation the first sound at the apex is short (not long, dull and booming); at the base the pulmonary second sound is louder than the aortic second; finally, the rhythm is irregular owing to the heart speeding up during inspiration—sinus arrhythmia (§ 65).

right border of the sternum (see Fig. 49). The apex is displaced *upwards* in pericardial effusion, collapsed lung, abdominal tympanites, or with any abdominal tumour pushing up the diaphragm. The apex beat is *obscured* by very muscular or adipose chest walls or by emphysema. It is *feeble* with myocardial and pericardial disease, and with ventricular dilatation. With pericardial adhesions there is a *systolic retraction* of one or more interspaces; with hypertrophy of the heart a similar condition may be seen near the apex.

The apex rate should be counted and compared with the pulse. Where the beats are regular, apex and pulse rates coincide, but where the rhythm is irregular, as in auricular fibrillation or premature beats, apex and pulse rates are different. The difference between the two is known as the pulse deficit.

THRILLS.—A thrill is a palpable "purring" sensation corresponding to the murmur of an organic lesion. If present, they should be timed with the carotid and their exact position noted; observe also whether they are constant or intermittent.

Two types of thrill are found in mitral stenosis, both being due to the flow of blood from left auricle to left ventricle. (*a*) The first, the *presystolic* or as it is sometimes called the auriculo-systolic thrill, is due to the blood flow produced by auricular systole. When the auricles fibrillate and no longer contract effectively, this thrill disappears. (*b*) The second is the *diastolic* thrill; it is due to the flow of blood from the left auricle to the ventricle through the stenosed valve during diastole, and is best felt early in diastole when the difference in the pressure in the two chambers is greatest. It may be found together with the presystolic thrill when the auricles are contracting, or alone when they are fibrillating; it generally indicates a severe degree of stenosis. A *systolic* thrill may be present at the apex in mitral regurgitation; at the pulmonary base in pulmonary stenosis; at the aortic base in aortic stenosis and aneurysm. Pericardial friction may produce a thrill. Systolic thrills are also common in congenital heart disease, especially in pulmonary stenosis and interventricular patency.

Any abnormal pulsation should be noted and investigated. Special attention should be directed towards the liver, and the spleen should also be palpated. The condition of the brachial arteries is of importance. Note whether they are visible, tortuous, thickened; if the latter, note whether the thickening is uniform or otherwise, bearing in mind that if patchy in character, this usually indicates involvement of the muscle coat as well as of the intima. The locomotor artery generally signifies two conditions, viz., a rigid vessel and a hypertrophied heart.[1]

§ 40. Percussion.—By percussion one is able to make out the position and the approximate size of the heart. Cardiac dulness is elicited by

[1] In any routine examination of the cardio-vascular system, ophthalmoscopic inspection of the retinæ and of the retinal arteries furnishes valuable information as to the condition of the smaller arteries (§§ 91, 848).

percussion and gives more or less accurately the actual size of the heart. In a normal heart the right margin extends slightly beyond the right margin of the sternum; the left margin is slightly external to the apex, and just internal to the nipple line, while the upper border is approximately level with the third intercostal space. The upper limit of the cardiac dulness is extended when there is dilatation of the pulmonary conus.

Method.—The student should lose no opportunity of PERCUSSING THE NORMAL HEART and of attending to the following points: (i.) *Having first localised the apex-beat*, begin outside the cardiac area in a perfectly resonant area. The middle finger of the left hand should be held vertically and placed flat and *firmly* upon the chest wall in an interspace; then moved ¼ inch at a time towards the centre of the heart. (ii.) Use only one finger—the second of the right hand—as a hammer, making a short sharp tap with the finger *tip*. The percussing finger should rebound immediately—"staccato," as pianists say. The movement should be made from the *wrist*, or from the knuckle (metacarpo-phalangeal joint), as in playing the piano, and the tap should be a light one. (iii.) By listening attentively to the sound elicited, it will be noticed that it is dull and flat over the heart, like that produced by striking any solid object; but louder and more resonant outside the area, like the sound produced by striking an empty barrel. It is only possible to define in this way the right, the upper, and the left limits of the dull area, because at the lower limit the cardiac dulness is continuous with that of the liver. Mark with a blue aniline pencil the right or sternal border in two places. The curved upper and left border of the dulness should also be marked by a pencil in two positions—viz., close to the left side of the sternum, and in another place near the nipple; these can then be joined and continued to the apex beat.

FALLACIES.—Cardiac enlargement may be *obscured* by the hyper-resonance of emphysematous lungs, and in these circumstances enlargement of the heart or pericardium is very difficult to make out. We have then to rely upon other means than percussion. On the other hand, cardiac enlargement may be *simulated* by a fibrous retraction of the left lung, the heart, nevertheless, remaining of normal size; or, thirdly, the heart may be *displaced* by an aneurysm or other mediastinal tumour pushing forward, and making the præcordial area appear larger. One or other border of the area of dulness may be *obscured* by pleural effusion. Ascites, pleural effusion, or abdominal distension may actually *displace* the heart.

§ 41. The Pulse.—At this stage, one may well investigate the arterial pulse. The radial is the one commonly selected. The usual method of palpating the pulse is to place three fingers of the right hand on it, when the following points can be systematically investigated: (*a*) *Rate*. Whether abnormally fast or abnormally slow. (*b*) *Rhythm*. Whether regular or irregular; if the latter, the nature of the irregularity should be investigated. Irregular pulses can be classified in two groups: (1) regularly irregular or (2) irregularly irregular. (*c*) The *Force* (estimated by the impact against the finger) depends upon the rapidity of the filling and emptying of the artery, *e.g.*, in aortic regurgitation, hyperthyroidism, anæmia and in certain febrile conditions, the force is considerable. (*d*) The *Volume*, estimated by the lift and duration of the wave, gives one the output of the heart, *e.g.*, in athletes and hyperpiesis. (*e*) The

Tension is estimated by the obliterative force and indicates systolic blood pressure. (*f*) The condition of the *vessel wall*.

The most common regular irregularities are: (1) Sinus arrhythmia (where the pulse speeds up during inspiration, and slows down during expiration), the slowing is vagal, and the irregularity is physiological (§ 65); (2) pulsus bigeminus or pulsus trigeminus (where the pulse goes in twos or threes followed by a pause), the result of regularly occurring premature beats; and (3) pulsus alternans (where big beats and little beats alternate at regular intervals), indicative of left ventricular failure (§ 71): it is of grave prognostic significance. It is difficult to determine by palpating the pulse, but can invariably be detected by the sphygmomanometer, the alternate beats coming through at a slightly lower systolic pressure.

The commonest irregular irregularities are: (1) The perpetually irregular pulse due to auricular fibrillation. Here the beats not only follow one another at irregular intervals, but are of unequal strength and volume. In addition the pulse rate may differ from the apex rate. The great clinical test of the presence of auricular fibrillation is that the irregularity is increased by exercise (§ 68). (2) The irregularity due to irregularly occurring premature beats or extrasystoles (§ 64). This indicates myocardial hyper-irritability, resulting from fatigue, inflammation or degeneration. This irregularity is in most cases abolished when the rate is increased by exercise.

§ 42. Auscultation.—For auscultation much practice is required, and the student should never miss an opportunity of listening to the sounds of the heart, *particularly the normal heart*.

The normal heart sounds are three in number—the *First*, or systolic sound, is long, dull and booming in character, and of lower pitch than the second sound. It is best heard over the region of the apex beat, *i.e.*, left fifth intercostal space just internal to the mid-clavicular line. It is due to two factors: (1) the contraction of the ventricular muscle, (2) the vibrations caused by the closure of the auriculo-ventricular valves. The *Second* or diastolic sound is short, sharp, slapping, and higher pitched, and is heard at the apex and at the base on a level with the second costal cartilage. It has two components, being produced by the closure of the aortic and pulmonary semilunar valves. The *Third* sound is also diastolic in time, is occasionally audible by the ordinary stethoscope, and can be easily detected by means of the cardio-phonograph. Its origin is doubtful. In diseased or damaged conditions of the heart not only are the normal heart sounds modified in various ways, to be described below, but adventitious sounds, murmurs or bruits, are liable to be produced either at the valve orifices or on the surface of the heart. When ausculating the heart, therefore, one should pay attention to (*a*) the characters of the normal heart sounds, and (*b*) the presence and character of any abnormal sounds (murmurs).

Alterations of the Heart Sounds and their Significance.—At the Apex. From what has been said about the origin of the First sound,

it is clear that its character will be modified by any condition which interferes with the contractility of the muscle or the closure of the auriculo-ventricular valves. This modification may be: (1) change in pitch, when the First sound becomes somewhat similar to the Second sound, *i.e.*, short and sharp in nature. This indicates a poorly contracting ventricle, due to inflammation, degeneration, toxæmia or non-stretching, *e.g.*, mitral stenosis. It is also found in shock, or after severe hæmorrhage

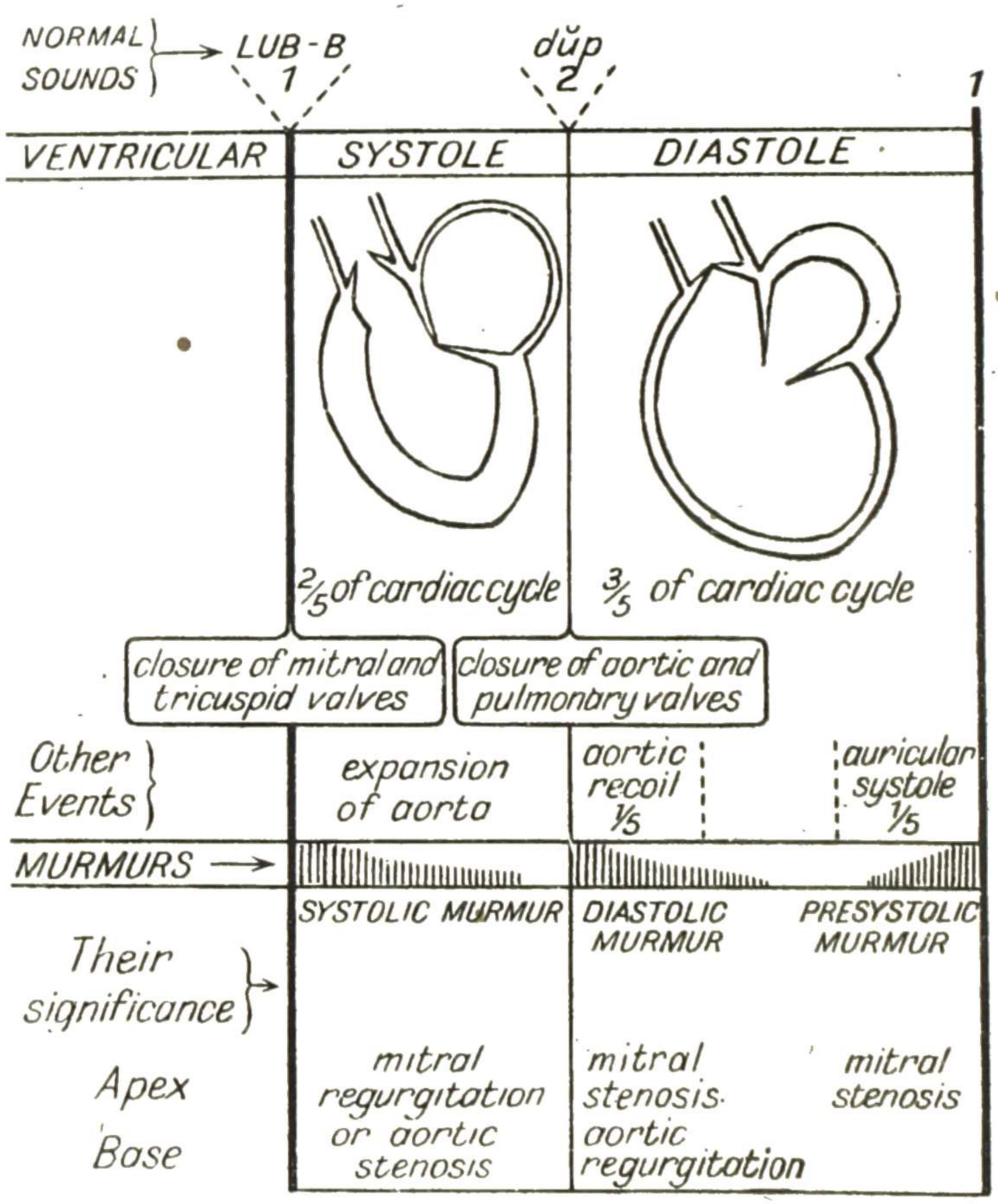


FIG. 10.—Diagram of a **Cardiac Cycle,** showing various events and their duration, how the different murmurs are produced, and their clinical significance. The student should study this and Fig. 11 very closely.

when the blood-pressure is low. (2) Reduplication due to a non-synchronising closure of the auriculo-ventricular valves. (3) Weakening or suppression due to pericardial effusion, emphysema, myocardial degeneration, myocardial infarction, etc.; or (4) partial or complete replacement by a murmur or adventitious sound.

The second sound at the apex, due to the closure of the aortic and pulmonary semilunar valves, may be (1) distinct; (2) modified by the presence of a murmur as in mitral stenosis, or (3) accentuated when the systemic or pulmonary tension is abnormally high.

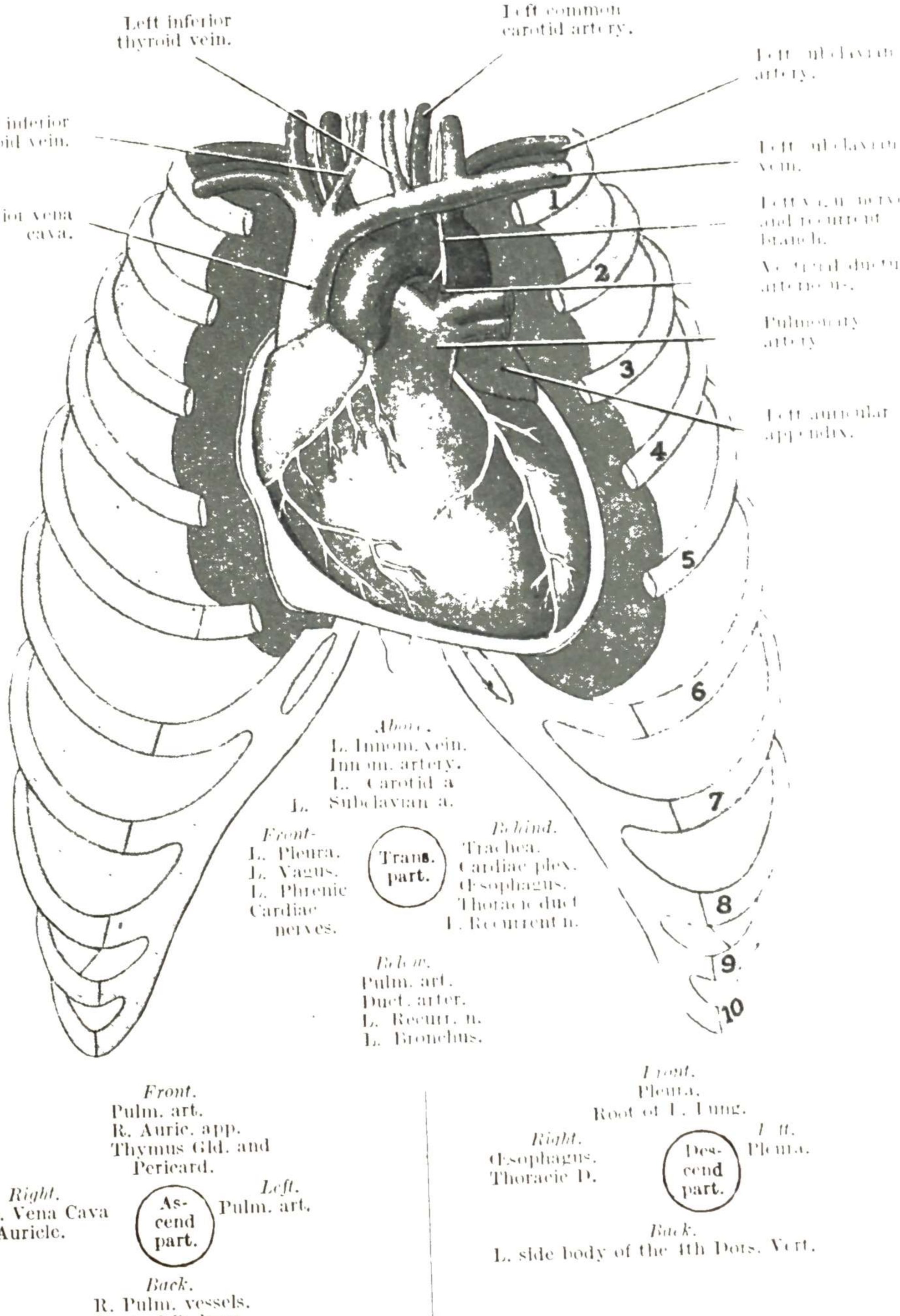


FIG. 11.—**The Heart and Great Vessels in Situ,** with lungs turned back, sketched from the cadaver. Right ventricle forms the greater part of the anterior surface of the heart. Above and to right of this is the right auricle, into which the superior vena cava opens, which collects the blood from the two innominate veins. Passing out from and above the right ventricle is the pulmonary artery, above which again is the remains of the ductus arteriosus, connecting it with the arch of the aorta. Just to the left of the pulmonary artery the left auricular appendix peeps round the corner. The arch of the aorta is seen coming forward from the left ventricle (which is at the back, and therefore only seen at the left margin of the heart) and from its upper convexity arise in order the innominate, left carotid, and left subclavian arteries. The trachea is seen behind the vessels, and the phrenic and vagi nerves are seen at the sides, those on the left passing down in front of the aorta behind the root of the left lung. The relations of the ascending, descending, and transverse portions of the aorta are given diagrammatically above.

A *canter, gallop*, or *triple rhythm* is a condition in which there are three distinct sounds at or internal to the apex. The canter is slower than the gallop rhythm. These sounds are due to reduplication of one of the heart sounds and are indicative of ventricular failure. A canter or gallop rhythm is common when conduction is defective in one or other

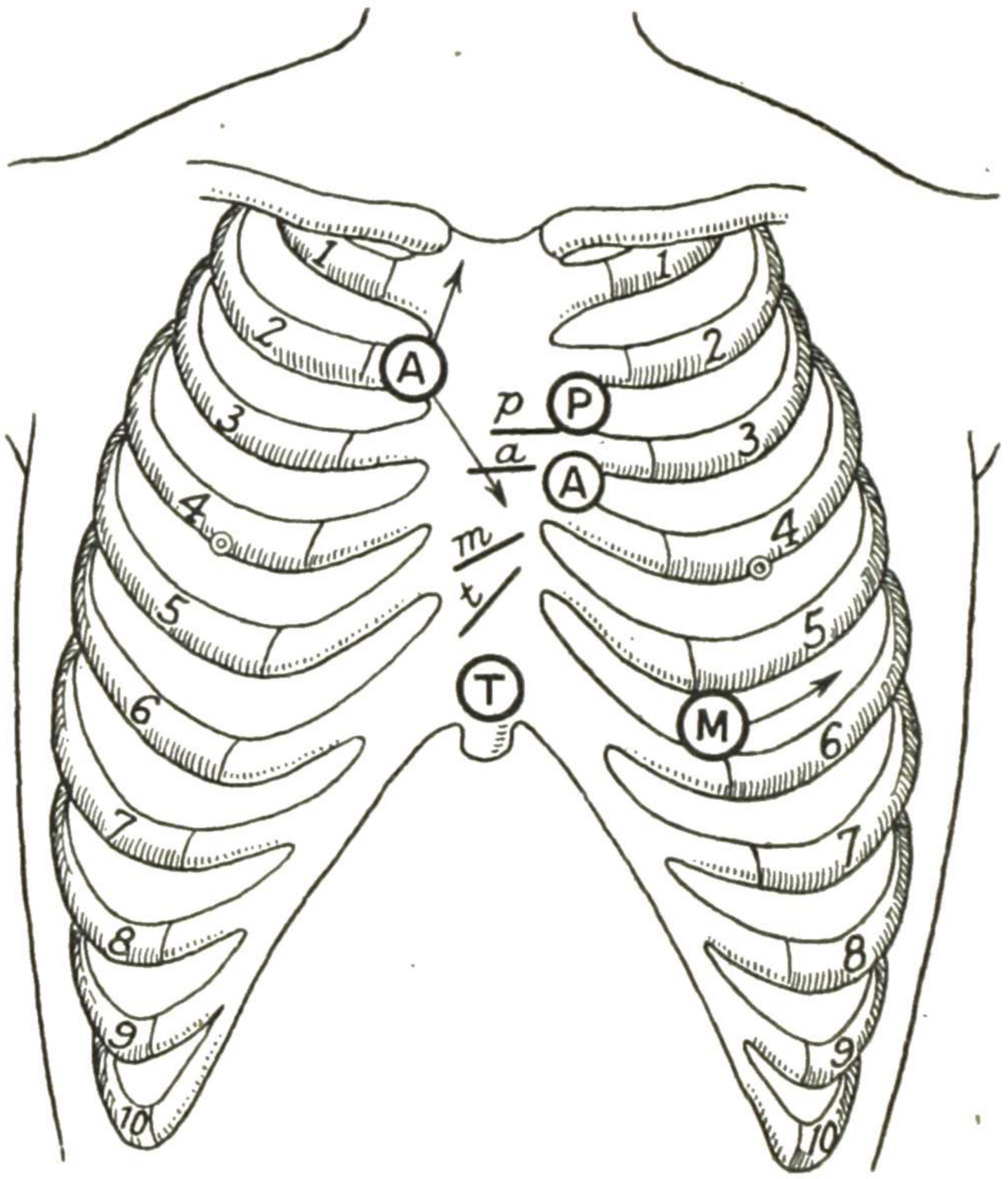


FIG. 12.—DIAGRAM SHOWING THE **situation** OF THE **Cardiac Valves** AND THE **position** IN WHICH THE SEVERAL **murmurs** ARE HEARD LOUDEST.

p = Pulmonary orifice, at level of upper border of third left costal cartilage.
a = Aortic orifice at level of lower border of third left costal cartilage.
m = Mitral orifice at level of lower border of fourth left costal cartilage.
t = Tricuspid orifice at level of fourth interspace, lying obliquely behind the sternum.

The positions where the sounds produced at the various orifices are best heard are indicated by the letters enclosed in circles. The arrows mark the direction in which murmurs produced at the corresponding orifices are conducted.

M, Mitral murmurs are best heard at the mitral area—*i.e.*, the apex.

A, Aortic murmurs are best heard at the aortic area—*i.e.*, second right costal cartilage; or along the left sternal border.

P, Pulmonary murmurs are best heard at the pulmonary area—*i.e.*, second left intercostal space.

T, Tricuspid murmurs are best heard at the tricuspid area—*i.e.*, at lower end of sternum.

of the branches of the Bundle of His (and see § 55). It is also frequent in large hearts with hypertension, when coronary disease has caused failure with much dilatation.

AT THE BASE, the *aortic second* sound, normally short, sharp and slapping, due to the closure of the aortic semilunar valves, may be (1) accentuated—indicative of a high peripheral resistance and a high blood pressure; (2) ringing, indicative of atheroma and often dilatation of the aorta and rigidity of the valves. This condition, most characteristic

to those who are familiar with it, differs from an accentuation and has another significance. It may, or may not be, associated with a high blood pressure. The aortic second sound is often very accentuated in cases of aortic aneurysm. (3) Absence of the aortic second sound means either that the aortic valves do not close owing to injury, destruction or absence, or that they close so quietly that they do not produce an audible sound. (4) The aortic second sound may be modified by the presence of a murmur which replaces it partially or entirely.

The Pulmonary second sound, due to closure of the pulmonary valves, is also short, sharp and sudden, and in adults less distinct than the aortic second sound. In young children the reverse is the case—the pulmonary second sound being louder than the aortic. It, in turn, may be accentuated (high pulmonary tension), as occurs in mitral stenosis and acute lung conditions; reduplication may be physiological, especially in children, and also occurs in mitral stenosis; or it may be modified by a murmur.

MURMURS.—Murmurs may be either systolic, presystolic or diastolic in time. The latter, moreover, are frequently divided into early, mid, and late diastolic according to the time at which they occur in the diastole. Further, murmurs may be produced either at the valve orifices when they are spoken of as endocardial, or outside the heart, *e.g.*, of pericardial origin. Endocardial murmurs are of two kinds: (*a*) those indicative of structural damage to the valves (organic), and (*b*) those indicative of softening or loss of tone in the auriculo-ventricular rings (atonic or functional murmurs). Endocardial murmurs may generally be differentiated from exocardial murmurs by the following points: (1) *Endocardial* murmurs are best heard in defined areas corresponding to the normal valve sounds; (2) are conducted or propagated in a definite direction; (3) are harsh or blowing in character. *Pericardial* murmurs are: (1) superficial and appear to be heard just under the stethoscope, (2) are usually not heard only over the valve area, (3) are not propagated in the same definite directions, (4) are not necessarily truly systolic or diastolic in time, (5) are often modified by pressure by the stethoscope, and are accentuated, diminished or removed by full inspiration or expiration. A single murmur of presystolic or diastolic time is usually an indication of organic disease at one of the cardiac orifices, but may be exocardial—*e.g.*, pericardial.

Functional Murmurs.—These murmurs, which may be heard over either apex or base, are usually soft and blowing in character. When present at the apex, a functional murmur may be local or conducted to the axilla. They are characterised by their variability under different conditions. Thus they are often present when the patient is lying and disappear when he stands; they may be heard during inspiration and not during expiration; they may appear only after exercise and disappear during rest—or they may only be audible when these conditions are reversed.

Hæmic Murmurs are frequently heard in anæmia and in some other

blood conditions (see Chapter XVI).[1] They are also common in thin-chested adolescents, and in patients with Graves' disease. They are usually systolic in time, are rarely double, are usually heard loudest in the pulmonary area, and are heard best when the patient is lying down.

Atonicity murmurs are found only when the heart has lost its tone from myocarditis or anæmia and are due to stretching of a valvular ring.

§ 43. Estimation of Myocardial Efficiency.—The measure of a heart's efficiency is its capacity for work; this is true of all hearts, whether healthy or diseased. Furthermore, it must be clearly borne in mind that many hearts work perfectly, exhibiting no defects at all, when the patients are at rest, but show serious derangements and definite evidence of myocardial impairment when called upon to do extra work. The great symptom of myocardial insufficiency is dyspnœa. If undue dyspnœa is absent, the heart muscle is not failing. The amount of dyspnœa is proportional to the degree of myocardial failure. The dyspnœa is complained of by the patient; it can also be observed objectively by the physician. When taking the history an exact idea should be formed as to how far and how fast a patient can walk, and whether hills or stairs cause shortness of breath. A definite idea as to the amount of work or exercise a patient can take in the course of daily life is the best test of cardiac function. A patient who is made short of breath by the exertion of undressing is unsafe for an exercise test and has a severe degree of cardiac failure.

Effects of Exercise upon the Heart.—(*a*) *The Rate.*—The normal heart responds to exercise by a gradual increase in rate. The increase is more or less uniform, the rate climbing up as exercise is increased. The normal heart rarely speeds up to over 150 for any length of time. It rapidly returns to normal on ceasing the exercise. A soft atonic or poisoned heart responds to exercise by undue acceleration, and very slowly settles down to its normal rate; while in certain diseased conditions of the heart one gets impaired acceleration, the rate scarcely altering at all. This may occur in very fast hearts (*e.g.*, auricular flutter), or in very slow hearts (*e.g.*, heart-block). Lastly, in a well-trained physiological heart, such as one meets with in young highly-trained athletes, the rate does not climb on exercise, but suddenly doubles (*e.g.*, at the commencement of the exercise, the rate may be 42, and on exercise suddenly becomes 84)—the so-called athletes' reaction.

(*b*) *Rhythm.*—The rhythm of the heart may be profoundly modified by exercise. (1) An irregularity may be produced, and any heart that becomes irregular on exercise is likely to be diseased. The most common irregularities revealed by exercise are auricular fibrillation, alternation (indicative of left ventricular failure), a sign of grave significance, and, in some cases, premature beats. (2) An existing irregularity may be abolished. Practically speaking, the only irregularities abolished by exercise are those caused by sinus arrhythmia and premature beats. (3) An existing irregularity may be increased. This is true of auricular fibrillation or extrasystoles due to disease.

(*c*) *Sounds.*—Under the influence of exercise, normal heart sounds may be reduplicated or modified by the production of adventitious sounds or murmurs. In early mitral stenosis, exercise brings out the signs of the lesion.

(*d*) *Thrills.*—Thrills may be actually made evident. This occurs in early mitral stenosis, when increased filling of the auricle results in increased stretching and increased contraction, and so produces a thrill, presystolic in time. An existing thrill is increased by exercise or abolished by exhaustion or by tachycardia.

[1] These so-called hæmic murmurs are not due to anæmia *per se*.

§ 44. Special Methods of Investigation.—1. **The Polygraph** is an elaboration of the older sphygmograph. Historically, it is of great interest, for by its invention and use Sir James Mackenzie was the first to analyse and classify the cardiac irregularities. His findings were confirmed by the electrocardiograph. Clinically, its chief value is to record auricular movement in cases of obscure irregularity. When the auricular and ventricular complexes overlap in the electrocardiographic record, simultaneous polygraphic and electrocardiographic tracings will often clear up the difficulty by defining the position of the hidden "P" wave. In the normal polygraph tracing the "A" wave coincides with auricular, and the "C" wave with ventricular systole. The "V" wave coincides with the opening of the tricuspid and mitral valves at the start of diastole.

2. **The Electrocardiograph** is an instrument for recording the minute electrical currents which are formed by the contraction of heart muscle. The two principles usually employed in the instrument are those of the string galvanometer, and the cathode-ray oscillograph. The electrical changes are photographed on a moving photographic film or plate, and the record is called an *Electrocardiogram*. The patient is placed in circuit with the machine by the following four sets of leads: With lead 1 the two electrodes are applied respectively to the right arm and the left arm; lead 2 to the right arm and the left leg; lead 3 to the left arm and the left leg. In the standard lead 4 the left arm electrode is placed at the apex and the right arm electrode either remains *in situ* (lead CR4) or is connected with the left leg (lead CF4): these give normally an upright "T" wave, and leads CR4 and CF4 give almost identical curves. Other leads may be used in certain conditions: the right pectoral lead—right lower sternum to right arm—is valuable in the diagnosis of pulmonary embolism. Fig. 13 represents a normal set of curves: 1, 2, 3, CR4 refer to the corresponding leads.

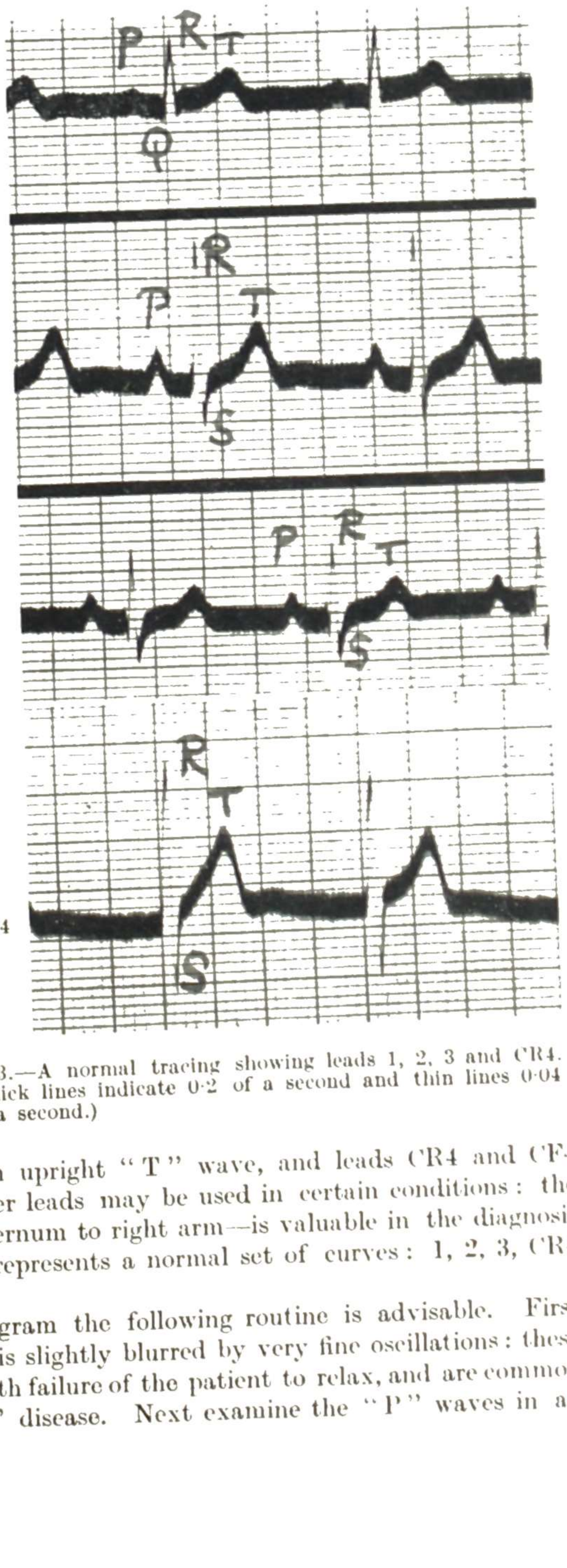


FIG. 13.—A normal tracing showing leads 1, 2, 3 and CR4. (Thick lines indicate 0·2 of a second and thin lines 0·04 of a second.)

When reading an electrocardiogram the following routine is advisable. First observe if the whole of the tracing is slightly blurred by very fine oscillations: these are due to fine muscular tremors with failure of the patient to relax, and are common in nervous subjects and in Graves' disease. Next examine the "P" waves in all

leads. These are produced by the auricles and for each lead should be uniform in shape and upright: occasionally in lead 3 they are diphasic or inverted in adipose patients with an abnormally elevated diaphragm. The amplitude of the "P" wave is increased if the auricles are hypertrophied, as in early mitral stenosis: while if the two auricles do not quite synchronise the wave is notched (*e.g.*, mitral stenosis). The "P" wave may be absent, and in its place there may or may not be irregularly occurring fine fibrillary waves (the ventricular waves being totally irregular): then auricular fibrillation is present (Fig. 14). Isolated "P" waves may be inverted, indicating that the auricles are here contracting from some ectopic pacemaker and not from the sino-auricular stimulus: these auricular premature beats may arise in any part of the auricular muscle, but the nearer they are to the sino-auricular node, the more closely does their shape approximate to normal (Fig. 15). If a series of these inverted or abnormal "P" waves occurs regularly at a rate higher than normal (between 120–200 per minute) and the ventricle contracts with each auricular beat, paroxysmal tachycardia (Fig. 34) is present. If no normal "P" waves are present, but instead there is visible a series of regular coarse undulations at a rate of between 200–300 per minute and best seen in leads 2 and 3, auricular flutter (Fig. 35) is present. Here the "QRST" follows either each second, third or fourth auricular undulation, giving an auriculo-ventricular ratio of 2 : 1, 3 : 1, or 4 : 1.

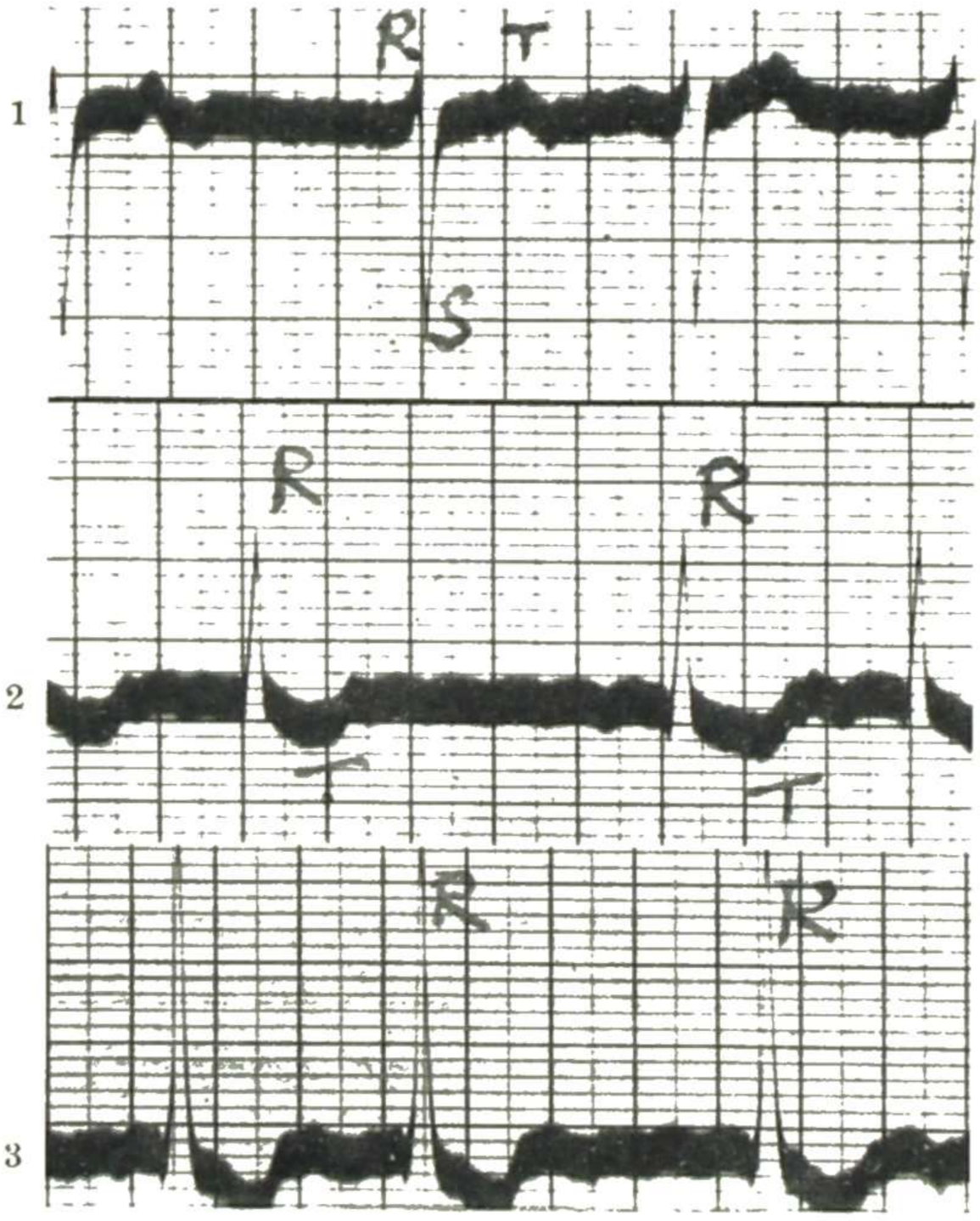


FIG. 14.—Auricular fibrillation. There is no "P" wave. Between the ventricular beats are seen fine fibrillary movements of the auricle. The ventricular rhythm is completely irregular. Note also the right axis deviation due to the fact that the tracing is from a case of advanced mitral stenosis.

Next measure the P–R interval. This represents the interval between the commencement of auricular and ventricular contractions, and is chiefly occupied by the time taken for the impulse to traverse the bundle of His. It should measure ·12–·20 sec. Heart-block is present in a minor degree if the P–R interval is prolonged beyond 0·20 sec. (Fig. 16), and in a greater degree if a ventricular beat drops out (Fig. 17): if a "P" wave is not followed by a QRS complex, but two, three or four "P" waves intervene between each QRS complex, then 2 : 1, 3 : 1 or 4 : 1 heart-block is present. If the interval between QRS and the nearest "P" wave varies continually, complete heart-block is present (Fig. 18). Occasionally the P–R interval is shortened. This is due to nodal rhythm with an abnormal pacemaker situated between the sino-auricular and auriculo-ventricular nodes (Fig. 19).

Next study the QRST portions representing ventricular action. The QRS portion should not exceed ·10 sec.; this is increased when conduction is impaired in one branch of the bundle of His. Normally, the "R" wave is tallest in lead 2 : left axis deviation (Fig. 20) is shown when the "R" wave is tallest in lead 1, and the "S" wave deepest in lead 3. Conversely, with right axis deviation (Fig. 14) the "S" wave is deepest in lead 1 and the "R" wave tallest in lead 3. A ventricular complex of abnormal shape and size, placed between others which are normal, indicates a premature ventricular beat

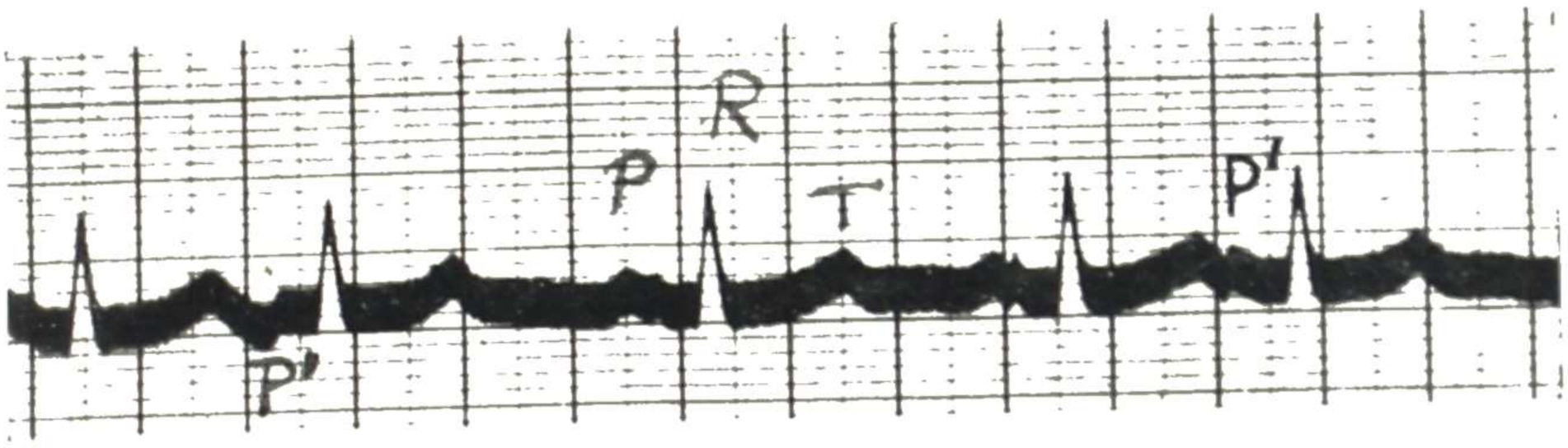


FIG. 15. —Two auricular premature beats (P') are shown, each arising from a different focus. The first ectopic " P " wave is inverted, showing its abnormal position of origin, and is also premature.

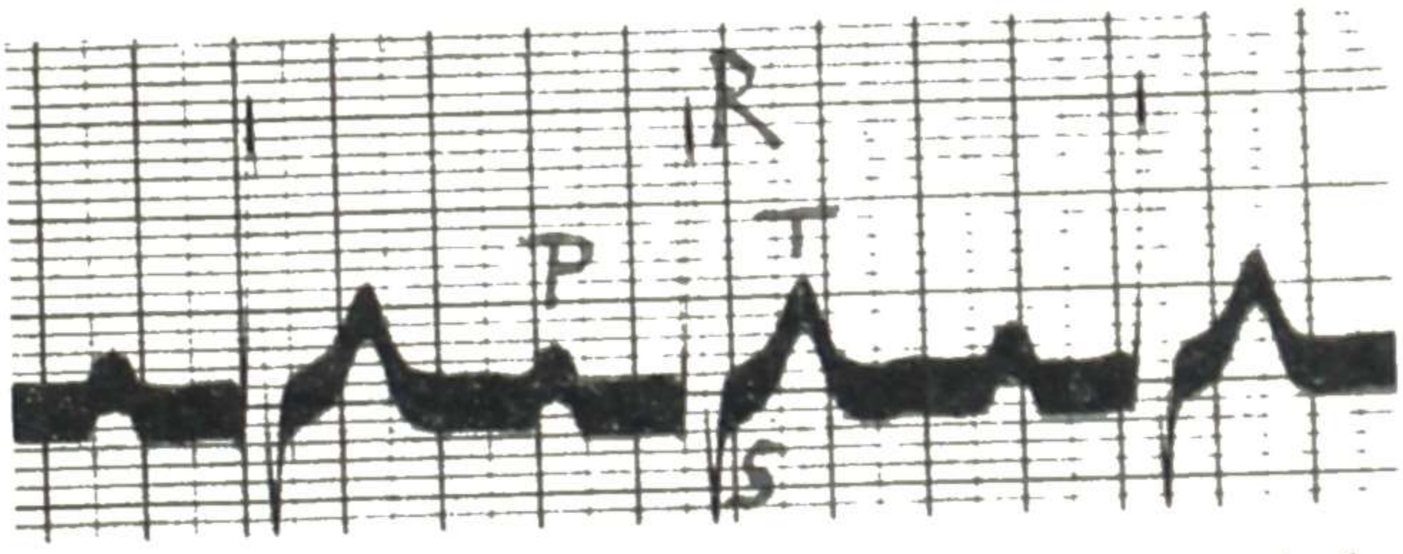


FIG. 16.—First stage of heart-block. " P–R " interval measures 0·3 of a second.

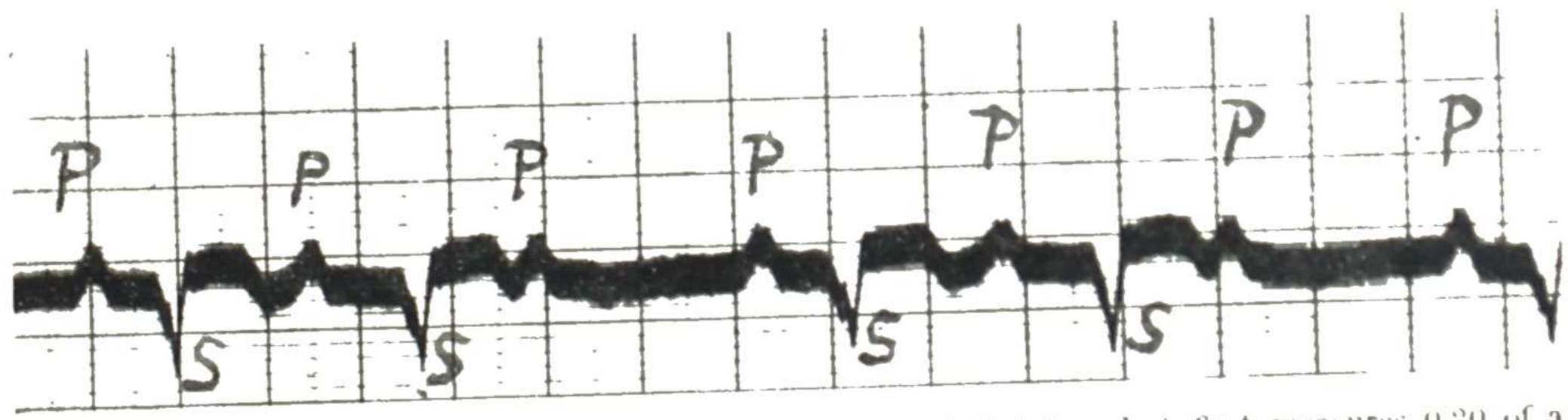


FIG. 17.—Heart-block. Stage of dropped beats. The " P–R " interval at first measures 0·20 of a second, then 0·25 of a second, and the third " P " wave fails to excite a ventricular contraction.

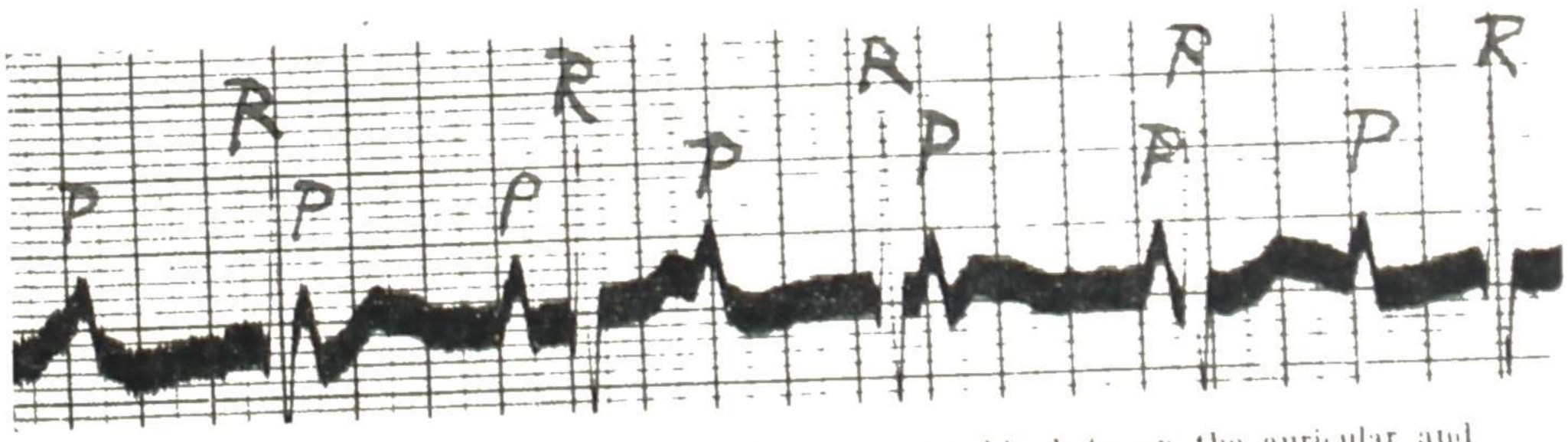


FIG. 18.—Complete heart-block. There is no relationship between the auricular and ventricular contractions.

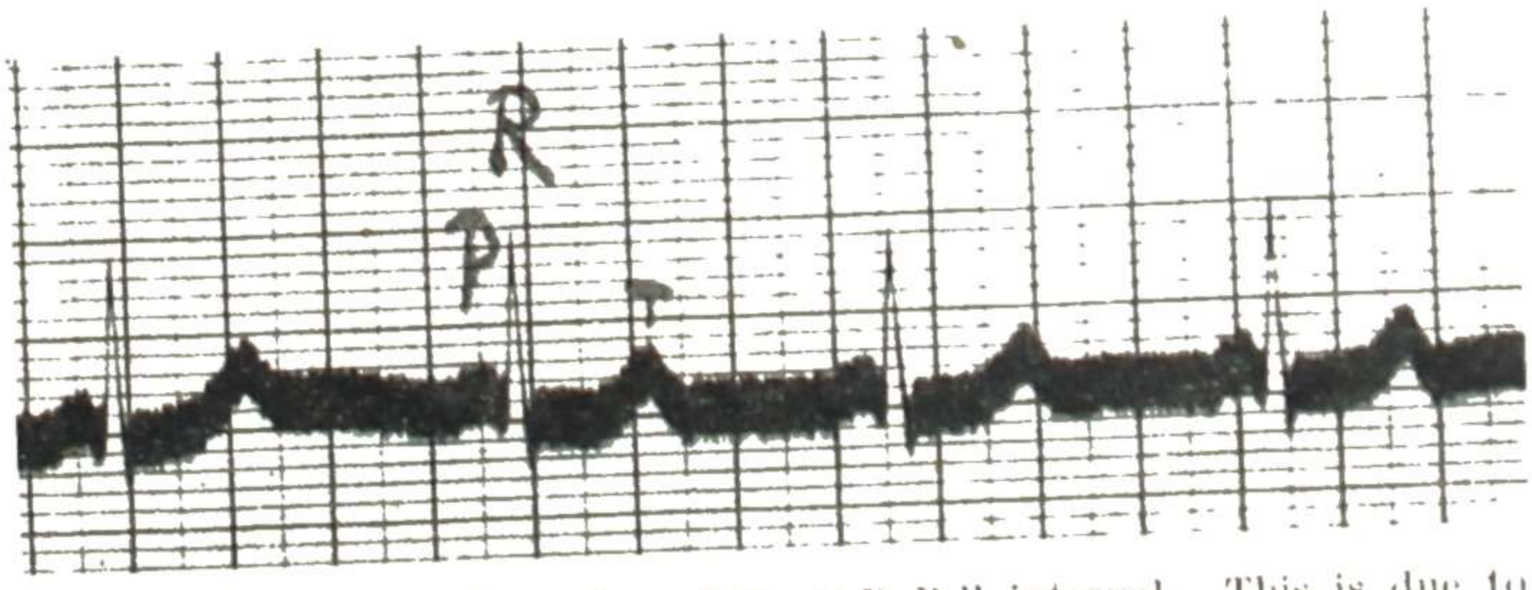


FIG. 19.—Tracing showing shortening of the " P R " interval. This is due to nodal rhythm, in which the pacemaker is situated between the sino-auricular and auriculo-ventricular nodes. The impulse reaches the auricles slightly before it reaches the ventricles.

(Fig. 32). The start of the ST interval should be isoelectric and the "T" wave well formed and upright in all three leads, although if the "T" wave in lead 3 (T_3) is inverted this has no special significance. This wave, especially in lead 2, is well developed in proportion to the physiological state of the ventricular muscle. It is inverted in leads 1 and 2 by full doses of digitalis, in myocardial disease, in acute and chronic pericarditis, and in some cases of aortic regurgitation, and is decreased in amplitude in myocardial toxæmia or degeneration. Suddenly occurring inversion of the "T" wave is a feature of coronary thrombosis (Figs. 25, 27), in which condition almost any variation may occur in the shape of the "QRST" waves, such variation occurring rapidly and often disappearing within a few weeks. In coronary infarction that part of the curve immediately following R or S fails to return to the resting base-line before continuing on to the "T" wave. This is

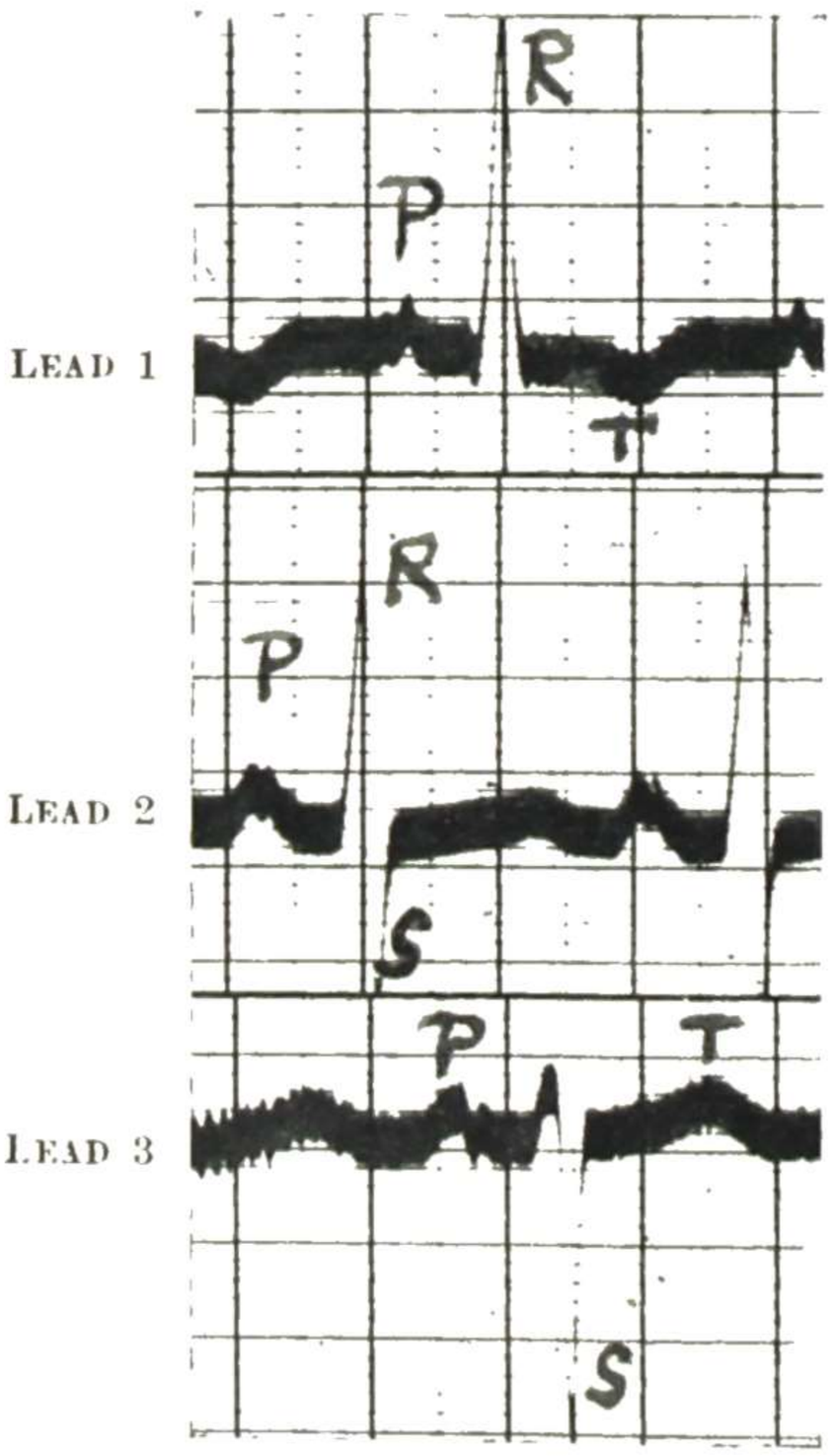


FIG. 20.—Left axis deviation, from a case of aortic regurgitation: "R" is tallest in lead 1, and "S" is deepest in lead 3.

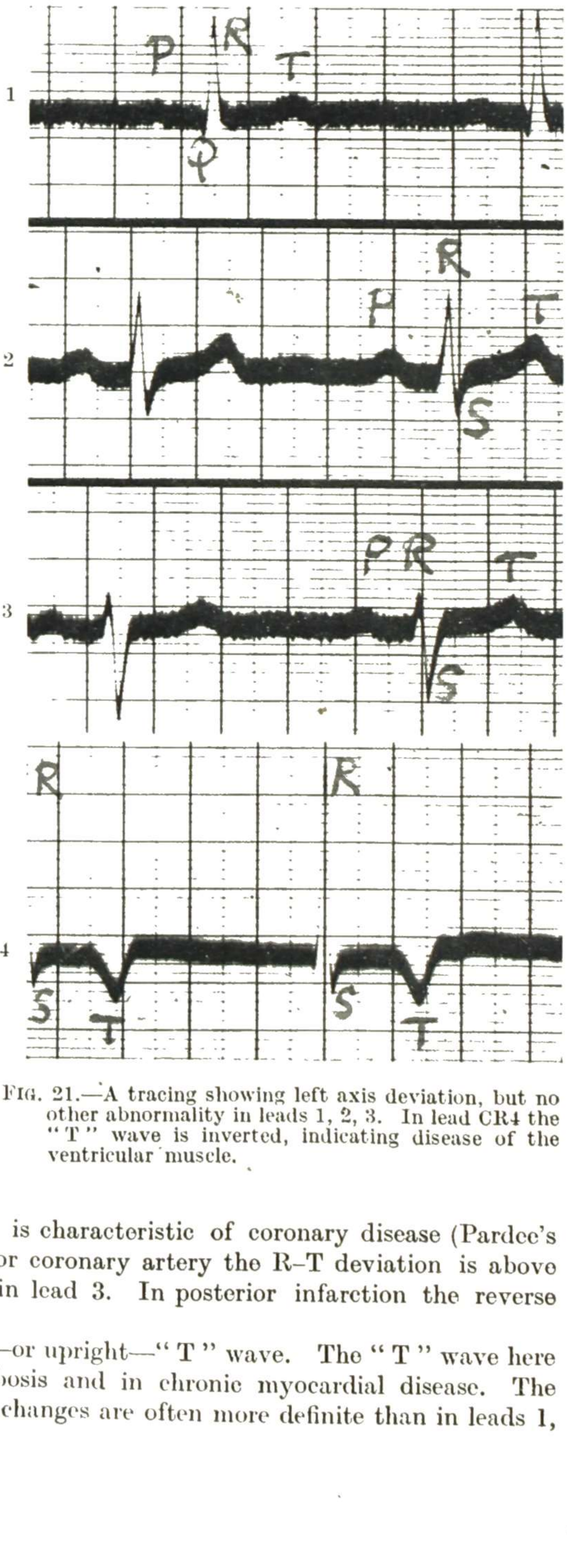


FIG. 21.—A tracing showing left axis deviation, but no other abnormality in leads 1, 2, 3. In lead CR4 the "T" wave is inverted, indicating disease of the ventricular muscle.

known as R–T or S–T deviation and is characteristic of coronary disease (Pardee's sign). With infarction of the anterior coronary artery the R–T deviation is above the base-line in lead 1 and below it in lead 3. In posterior infarction the reverse is the case in both leads.

Lead CR4 should show a vertical—or upright—"T" wave. The "T" wave here is also inverted in coronary thrombosis and in chronic myocardial disease. The advantages of lead CR4 are that the changes are often more definite than in leads 1,

2 or 3, and in some cases may be present when they are absent in leads 1, 2 and 3 (Fig. 21).

3. No examination of the cardio-vascular system can be regarded as complete unless it includes an *X-Ray* investigation (Figs. 22, 23A and 23B). The technical

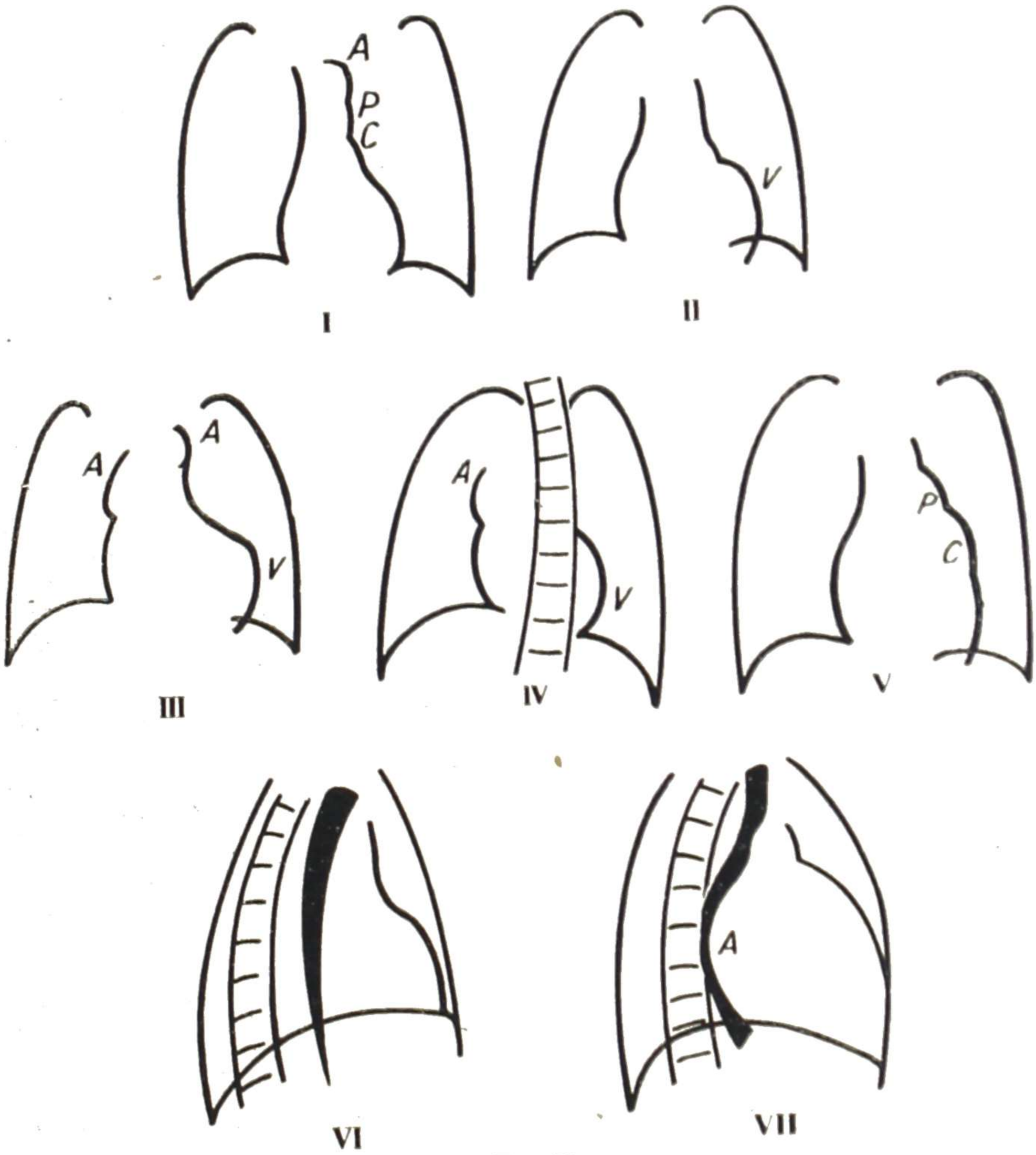


FIG. 22.

I. The normal anteroposterior outline of the heart.
II. Left ventricular hypertrophy (V), in aortic stenosis, without enlargement of the aorta.
III. Left ventricular hypertrophy and dilatation (V), with enlargement of the ascending and transverse aorta (A, A).
IV. Left anterior oblique view, showing left ventricular enlargement (V), and enlargement of the aorta (A).
V. Anteroposterior view in mitral stenosis, showing enlargement of pulmonary conus (C), and prominent pulmonary artery (P).
VI. Right oblique view, showing normal œsophageal shadow.
VII. Right oblique view in mitral stenosis showing displacement backwards of œsophagus filled with barium, outline of left auricle shown (A).

details must be obtained from a larger textbook; the main points to which attention should be directed may be thus summarised:—

A. THE HEART:—

(1) *Its position* in the mediastinum—its relation to the lungs and diaphragm.
(2) *Outline and form*: This is often characteristic, *e.g.*, in young healthy hearts

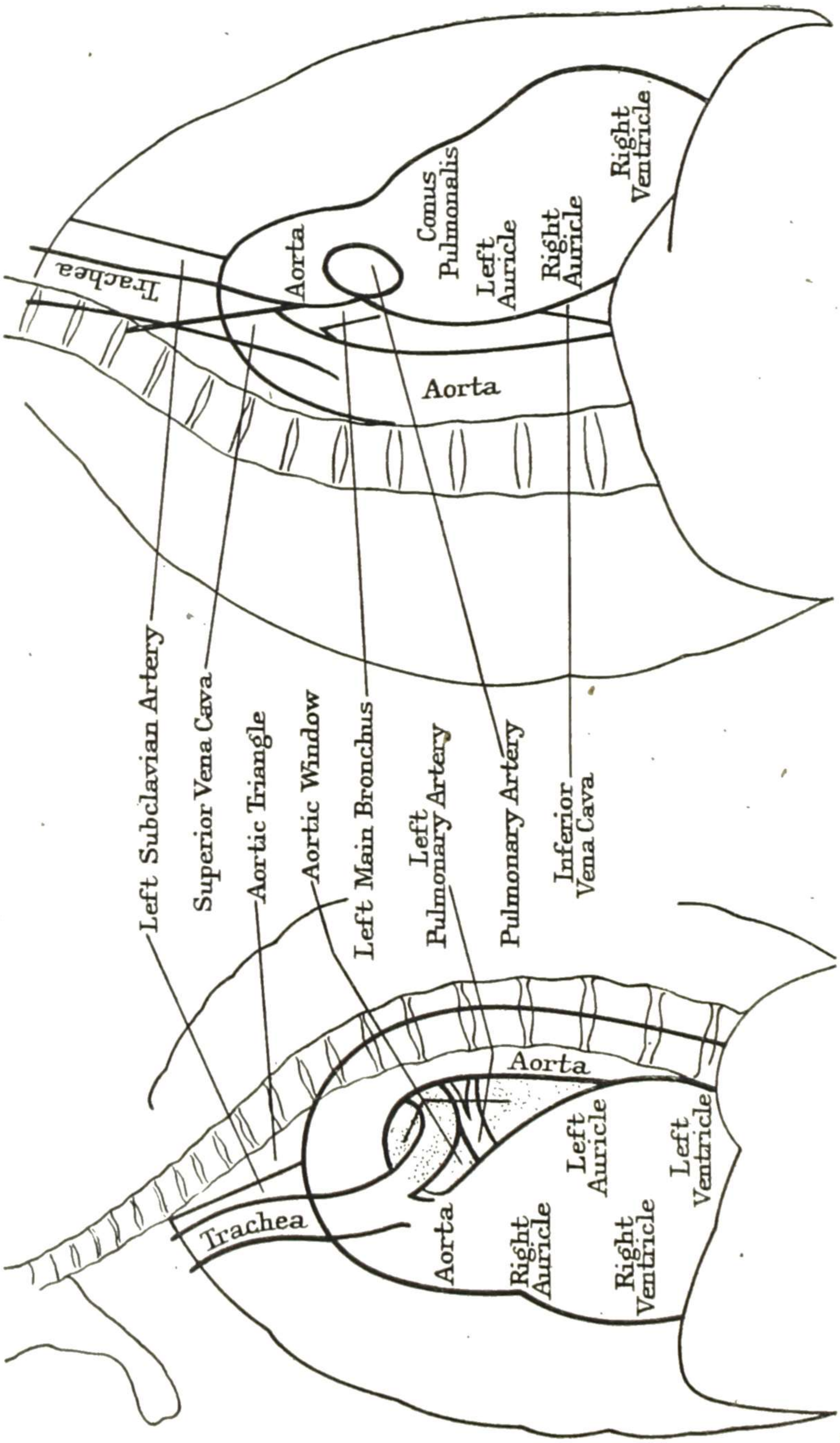


FIG. 23.—Outline of Normal Cardio-vascular Shadow.
A—Left oblique view (left shoulder forward).
B—Right oblique view (right shoulder forward).

it is vertical, in congenital heart disease round, in old valvular disease globular, but varies with the lesion. In enlargement of the left ventricle it is boot-shaped with the long axis horizontal, while in pulmonary tuberculosis it is elongated and tubular. Hypertrophy or dilatation of the left and right ventricles, enlargement of the left auricle, as in mitral stenosis, are some of the chief points to be demonstrated by radiography.

(3) *Size:* The maximum transverse diameter of the heart is less than half the greatest diameter of the thorax. Thus a cardio-thoracic ratio of more than 0·5 is evidence of enlargement.

B. THE GREAT VESSELS: Examine especially in oblique positions; note cardio-phrenic angle; ? dilatation or aneurysm; ? mediastinitis or growth or foreign body.

C. THE PERICARDIUM: ? adhesions; ? fluid.

4. *The Orthodiagraph* is an instrument whereby outline diagrams can be made of the actual size of the heart itself, so that accurate measurements can be obtained not only of the heart but also of the great vessels, etc. An outline of the cardiac shadow, resulting from parallel rays, is traced on an X-ray screen. A six foot X-ray film (*Teleradiogram*) is often substituted for this.

Circulation Times.—It may be helpful to determine local or general slowing of the speed of the circulating blood. The blood may travel more slowly as a whole, or it may be held back in some local area such as the venæ cavæ or the pulmonary veins and capillaries. This slowing can be measured. If decholin, which tastes bitter (10 c.c. of 20 per cent. solution), or calcium gluconate, which causes a hot sensation in the mouth (4 c.c. of 20 per cent. solution), be injected rapidly into a vein, the interval between the time of injection and the sensation of bitterness or sweetness in the patient's mouth, gives the time taken for the blood to traverse most of the venous and the whole pulmonary system, together with a second or so of arterial transit. In normal resting adults this time averages 12 sec., the upper limit being 16 sec. If ether is injected (5 ℳ with 5 ℳ of normal saline) the time interval taken is from the injection to the first notice by the patient of ether in the breath. This measures the arm to lung time or the amount of delay in the systemic venous system. (Average normal figure, 5 sec.; upper normal limit, 9 sec.)

PART C. DISEASES OF THE HEART AND PERICARDIUM: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 45. Classification.—For practical purposes diseases of the heart and pericardium may be classified under five prominent differential features: Disorders WITH PYREXIA; Disorders in which PAIN is a characteristic symptom; Disorders which are attended by an ENLARGEMENT of the AREA of CARDIAC DULNESS; Disorders in which an ALTERATION of the CARDIAC SOUNDS, or a MURMUR forms the diagnostic feature; and Cardiac conditions which are recognised by an ALTERATION of the RHYTHM or RATE of the PULSE.

A. PYREXIA	I. Pericarditis. II. Acute Endocarditis.
B. PAIN	I. Angina of Effort. II. Spasmodic Angina. III. Coronary Thrombosis. IV. Angina Innocens (Pseudo-Angina). V. Pericarditis.

C. Enlargement of the Area of Cardiac Dulness .	I. Cardiac Hypertrophy. II. Cardiac Dilatation. III. Chronic Pericardial Effusion IV. Adherent Pericardium.
D. Altered Heart Sounds and Murmurs . .	I. Myocardial Degeneration. II. Endocarditis. III. Congenital Heart Disease. IV. Pericarditis.
E. Alteration of Rhythm or Rate of Pulse .	I. Sinus Arrhythmia. II. Premature Beats (Extrasystoles). III. Tachycardia. IV. Auricular Flutter. V. Auricular Fibrillation. VI. Bradycardia. VII. Heart-Block.

The **Routine procedure** in the investigation of a cardio-vascular problem may be considered under the following headings—(1) The origin of the present symptoms, *e.g.*, whether they supervened on any definite illness, acute or chronic, or followed on some definite action, emotion, etc.

(2) The personal history, especially as regards (*a*) previous diseases such as rheumatic fever, growing pains, chorea, scarlet fever, tonsillitis, influenza, diphtheria, syphilis, etc. ; (*b*) habits of life, especially as regards exercise, alcohol and tobacco.

(3) Family history. Certain diseases, *e.g.*, rheumatic fever, arterio-sclerosis, etc., tend to run in families, and predispose to heart disease.

(4) Symptoms. The commonest symptoms associated with heart disease are dyspnœa, palpitation, pain, vertigo, faintness, and a sense of exhaustion. These are dealt with in Part A.

(5) Physical examination of the patient (Part B). Inasmuch as many hearts, when only slightly damaged, function normally when the patient is at rest and their "load" is light, but develop obvious defects of action under "load," it is essential to examine the patient three times—standing, lying, and after exercise. Further, it is convenient to divide the examination into: (*a*) Ordinary routine clinical examination, under which heading one would include the results of inspection (§ 38), palpation (§ 39), percussion (§ 40), the pulse (§ 41) and auscultation (§ 42). (*b*) Cardiac efficiency tests (§ 43) used for ascertaining the reserve energy of the heart. (*c*) Special instrumental methods of examination (§ 44).

Group A. If the symptoms of which the patient complains are unattended by Pyrexia, turn to § 51. If the disease is **attended by Pyrexia,** it is probably Acute Pericarditis or Acute Endocarditis, either rheumatic or infective in origin, or Coronary Thrombosis. It must be remembered that cardiac patients are often subject to other febrile illnesses.

I. The temperature is elevated, *the patient is in evident distress, and the præcordial area of* dulness is increased, *the shape of the dulness*

being PYRAMIDAL, *with the point upwards.* A PERICARDIAL FRICTION SOUND *is audible. The disease is probably* ACUTE PERICARDITIS.

§ 46. Acute Pericarditis is an acute inflammation of the pericardial sac. It is not infrequently met as a primary affection. It supervenes during the course of many different diseases, and the symptoms of these may mask its onset. Rheumatic fever is certainly its most common cause, and it should be remembered that it may be the first manifestation of this affection. We should always examine the heart daily in patients with rheumatic fever or nephritis, because in these acute pericarditis may come on insidiously, without pain or tenderness. Its advent in rheumatic fever is marked by high fever, tachycardia, pallor and vomiting (especially in children) or by the occurrence of delirium.

Symptoms.—(1) The patient wears an anxious, troubled look, and the cheeks are generally pallid; a distinct puffiness of the face, not amounting to obvious œdema, is often present; there are fever and a rapid pulse; the breathing is rapid, and he may complain of severe pain in the left chest (occasionally referred to the abdomen), increased by pressure, movement, or respiration; a short irritative unproductive cough is common. Abdominal rigidity may occur. (2) *Physical Signs.*—The præcordial dulness is increased in all directions, and the cardio-hepatic angle of resonance, to the right of the heart, becomes obtuse instead of acute. A friction sound is heard on auscultation. It is harsh, somewhat creaking in character, generally double, frequently triple, and occasionally systolic only. This may be distinguished from a murmur produced *within* the heart by (i.) usually being double, *i.e.*, accompanying the movements of the heart, and rarely exactly synchronous with the first and second sounds; (ii.) the second part of the rub is occasionally continuous with the first, without any diastolic pause; (iii.) it is often loudest at the root of the great vessels, over the third left costal cartilage; (iv.) it varies in its character from time to time, and is increased by firm pressure with the stethoscope; (v.) pressure will also elicit another character—viz., that the disease is usually accompanied by tenderness, as well as pain. The differentiation between peri- and endocardial murmurs is so important that it is also given in a tabular form below (Table I, p. 67). To distinguish pericardial from pleural friction is very easy, because the latter ceases if the patient holds his breath. Note that as the effusion occurs the murmur may become less distinct, but it rarely disappears entirely. It is again intensified as the effusion clears up. In most cases of acute pericarditis, with or without effusion, physical signs are to be found over the left lower lobe behind. These signs are those of collapse of the lung, and first occur, and are last to disappear, at the apex of the lobe; they may involve the whole lower lobe.

Second Stage, or stage of pericardial effusion. The inflammation may subside, but occasionally, in the course of a day or two, effusion of fluid occurs, and the pain and tenderness diminish. Effusion of a quantity of fluid or more than half a pint is very rare in rheumatic pericarditis.

When a large effusion does occur, the rub becomes less audible, though it may still be heard at the base of the heart. The breathlessness and other symptoms continue; the cough becomes more troublesome; dysphagia and vomiting rarely occur. Pulsus paradoxus may be present. *The increased area of dulness*, due to pericardial effusion, may be greater than the enlargement from any other cause. (i.) It is of *triangular shape*, with apex upwards, reaching to the third, or even second, costal cartilage. (ii.) There is often actual or apparent *raising of the position of the apex beat.* (iii.) The *dulness extends to the left* of the apex beat. There is progressive weakening of the heart sounds at this time, from the associated myocarditis.

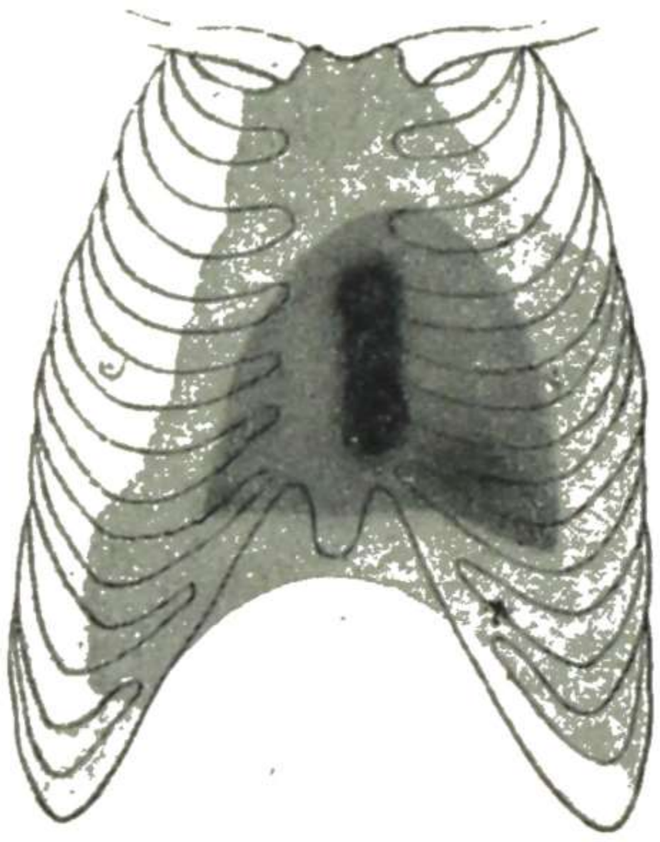


FIG. 24.—DIAGRAM FROM A CASE OF RHEUMATIC PERICARDITIS WITH EFFUSION.—Eliza P., aged twenty-seven. Heavy shading corresponds to position of maximum intensity of friction. Medium shading corresponds to the area of deep cardiac dulness. Light shading corresponds to the area over which the pericardial friction is audible. It is often taught that one of the features distinguishing peri- from endocardial murmurs is the limitation of the former to the præcordial region; but I have many times satisfied myself that this is not so, and this case is one of several examples I have met with verified by autopsy.

Etiology.—The causes may be classified under five headings: (1) acute infections, *e.g.*, rheumatism, scarlet fever, pneumonia, pyæmia, etc.; (2) extension from adjacent structures, *e.g.*, malignant disease and tuberculosis; (3) chronic nephritis; (4) coronary disease or infarction; (5) injury.

Course and Prognosis.—The duration of acute pericarditis varies widely, according to the cause, but it averages about fifteen to twenty-five days. It may undergo resolution with or without the formation of adhesions (Adherent Pericardium, § 56); or result in chronic pericardial effusion (§ 56); or become purulent (Pyopericardium, § 47 below). Pericarditis with effusion is always a serious malady, but the prognosis depends much on the underlying cause, the amount of distension of the pericardial sac, and the evidences of interference with the cardiac action—dyspnœa and cyanosis with feebleness, rapidity, and irregularity of the pulse. Pericarditis complicating rheumatism, like the other complications of that disease, tends to recover, but it frequently leaves a weakened heart (damaged myocardium), and leads to cardiac dilatation (enlargement). In renal disease it is a serious though often latent affection; and in pyæmia, when it is generally purulent, is nearly always fatal. In infancy and in debilitated patients it is also grave.

Diagnosis.—The diagnosis from acute endocarditis has been considered above, and in Table I, § 49. It is distinguished from enlargement of the heart by the following points: the left border of the dulness in pericardial effusion extends beyond the apex beat, and the apex beat may be displaced upwards; the right border of dulness has a convex outline and the cardio-hepatic angle at the right fifth intercostal space is obtuse; lack of movement of the epigastrium with respiration is another valuable

sign. Both conditions may be present at the same time. X-Ray examination will usually enable one to verify the diagnosis. It should be remembered that inflammatory conditions of the *left lung* and *pleura* not infrequently give rise to a to-and-fro friction sound along the left border of the heart; this is produced by pleural and not by pericardial inflammation (pleuro-pericardial friction). The intensity of this friction often varies with respiration. The signs of consolidation of the left lower lobe described above may occasionally suggest a diagnosis of *lobar pneumonia*.

Treatment.—In the inflammatory stage the patient must be kept lying comfortably in bed *and absolutely forbidden to move*. A fluid diet is advisable. A poultice, or warm fomentation applied to the præcordium usually gives more relief than the ice-bag, though this undoubtedly relieves the symptoms, controls the restlessness of a young patient, and possibly also reduces the heart rate. If the pain is great, relief is often obtained from the application of four or five leeches over the præcordium. Blisters are occasionally used. If cyanosis, orthopnœa, and venous distension are present, indicating considerable cardiac embarrassment, bleeding (15 to 20 ounces) is a prompt and efficacious measure. Opium by mouth, or morphia hypodermically, is of great value for the pain and distress, much smaller doses being given to children. The bowels should be regulated. Stimulants, such as nikethamide B.P. (coramine), can be given when the blood pressure falls. Mersalyl is often useful in the more chronic forms of pericardial effusion. For hyperpyrexia and delirium tepid or cool sponging is a useful means of lowering the temperature, and will often induce sleep.

Treatment of the cause of the pericarditis should be combined with the foregoing—*e.g.*, sodium salicylate combined with alkalies for acute rheumatism; diuretics and hot-air baths for renal disease. In the stage of effusion free blistering promotes absorption, but it must be remembered that renal disease is a contra-indication to blistering. If the effusion becomes chronic, give diuretics; mersalyl is very useful but must be used with great care in nephritis. Iodine paint and other local counter-irritants can be tried.

Paracentesis Pericardii.—When the amount of effusion becomes considerable, as shown by marked dyspnœa, cyanosis, distention of the neck veins, tachycardia and lowered blood pressure, paracentesis pericardii should be performed. The site chosen for this procedure may be in the fifth left intercostal space just inside the mid-clavicular line, or one inch from the margin of the sternum in the same space; or in the angle between the ensiform cartilage and the left costal margin, near the lower end of the body of the sternum and passing upwards and inwards behind it into the pericardial sac. A trocar and cannula or an aspirating needle or a Potain aspirator may be employed. Eight or twelve or even forty ounces (in a chronic case) of fluid may be slowly removed. This operation is rarely required; it is never necessary in the rheumatic pericarditis of childhood.

§ 47. Pyopericarditis.—Sometimes in debilitated children and in the course of pyæmia, in phthisis and empyema, and in some other conditions, the fluid in the pericardium takes on a purulent or sero-purulent character. This is sometimes revealed (as is a collection of pus in other parts of the body) by (1) shivering attacks,

(2) profuse perspirations, and (3) a temperature with wide variations in the course of a few hours, in addition to the clinical features of acute pericarditis above described. But it is difficult to diagnose, because the *friction sound is usually transient.* It is often fatal.

Pyopericarditis is the form which pericarditis frequently assumes in infancy, and is often not diagnosed. Progressive weakness, fever, anæmia, leucocytosis and X-ray examination may suggest the presence of pus.

Treatment.—A large sterilised (No. 10) needle should be very carefully introduced whenever the existence of pyopericardium is suspected. If the fluid withdrawn be of a purulent nature, paracentesis, or free drainage, should be effected. Penicillin is injected locally once a day, and is administered systemically by three-hourly injection if the infecting organism is sensitive.

Pneumopericardium is a very rare condition in which air reaches the pericardial sac from the lungs or stomach.

Hæmopericardium is rare. Aneurysm of the first part of the aorta or of the cardiac wall, rupture or wounds of the heart, scurvy and other blood diseases, may lead to sudden death owing to the sudden influx of blood into the pericardium. A small amount of bleeding may be seen in the pericarditis due to nephritis, malignant growths, acute rheumatism and tubercle.

§ **48. Latent Pericarditis**—*i.e.*, pericarditis without *symptoms* (though not necessarily without physical signs). In most patients in whom we find a pericardial effusion a history of acute pericarditis is obtainable; but it is not sufficiently recognised that pericarditis may come on insidiously, without acute symptoms. The effusion may be discovered during routine examination of the heart, or perhaps not until autopsy. Moreover, at the post-mortem a totally adherent pericardium is sometimes found in a patient in whom careful inquiry has failed to reveal any symptoms pointing to the heart during life. In ACUTE RHEUMATISM *its advent may be indicated only by delirium or vomiting.*

Pericarditis frequently results in Adherent Pericardium (§ 56).

We now pass to the other acute disorder with pyrexia.—II. ACUTE ENDOCARDITIS.

II. THE TEMPERATURE IS ELEVATED. *The præcordial area of dulness is not necessarily increased, and on auscultating the chest there is a* MURMUR *added to the heart sounds—the disease is probably* ACUTE ENDOCARDITIS. *It is not always easy to distinguish an endocardial from a pericardial murmur.*

§ **49. Acute Endocarditis** is acute inflammation of the valves or of the mural endocardium of the heart. It is usually attended by enlargement of the præcordial dulness, because some degree of myocarditis and dilatation is associated with it. In a large proportion of cases it complicates some other disease; and, like pericarditis, it is most frequently associated with acute rheumatism; it may even be the first evidence of that disease.

There are two varieties of endocarditis with fever: RHEUMATIC Endocarditis, and INFECTIVE or MALIGNANT Endocarditis.

In RHEUMATIC ENDOCARDITIS: (1) *a murmur develops* at the apex or base of the heart, corresponding to the mitral or aortic valves (see Fig. 12). The mitral valve is most frequently involved in acute rheumatism, but the mitral and aortic valves may be affected together, or rarely the aortic valve alone. The murmur is usually soft and heard over a limited area, and only occasionally is it harsh soon after its appearance. (2) There is often some myocarditis sometimes causing cardiac

TABLE I.—DIAGNOSIS OF ENDOCARDIAL FROM PERICARDIAL MURMURS.

Endocardial Murmurs.	Pericardial Murmurs.
1. May accompany first or second sound only, or both.	Usually double—always superficial and are as loud in diastole as in systole; not quite synchronous with the heart sounds.
2. Often loudest at one of the valvular areas.	Usually loudest over third left costal cartilage (root of big vessels).
3. May be conducted into the axilla, or along the aorta and carotids.	Mostly confined to the præcordium.[1]
4. Usually no pain or tenderness.	Often accompanied by pain.

dilatation, with a weakened diffuse apical impulse and weak cardiac sounds. (3) *Constitutional symptoms* may be marked when there are the general symptoms and signs of rheumatic fever (§ 582); but in children these may be slight or even absent. The onset of endocarditis may then be suspected when there is sudden pallor, with loss of physical vigour, accompanied by an evening rise in temperature and increase in pulse rate. Præcordial pain and distress are rarely found—a point worth remembering. The erythrocyte sedimentation rate is always raised when active endocarditis is present. The presence of rheumatic nodules (§ 573) around the elbows, wrists, knees and ankles usually indicates a severe form of active carditis.

Causes of Rheumatic Endocarditis.—It must be remembered that acute rheumatic endocarditis may complicate, not only rheumatic fever proper, but rheumatic tonsillitis, chorea and scarlet fever. The patient is generally young, usually a child: it tends to run in families.

The *Diagnosis* of acute rheumatic endocarditis is sometimes difficult. The murmur will be found over the mitral or aortic valve, will immediately follow a valve sound, will be localised and if conducted will follow the direction of the murmur of cardiac valvular disease (Fig. 12); it will not be materially affected by change of posture or by deep breathing. Care must be taken to avoid confusion with exocardial murmurs (§ 59). The distinction from pericardial murmurs is set forth in Table I. In view of the great tendency to recurrence of acute rheumatism and of rheumatic endocarditis, a careful history of previous attacks should be taken: a mitral stenotic murmur is evidence of an attack of acute rheumatism at least six months previously. Therefore, if signs of an acute endocarditis are also present, the acute attack now suffered from cannot be the first. Pyrexia, tachycardia, an increase in the size of the heart, and the intensity of the murmur and a raised erythrocyte sedimentation rate indicate active inflammation of the valve or valves. *Acute infective endocarditis* differs clinically (1) in the greater severity of the constitutional symptoms, with often a wide range of the diurnal temperature, and even rigors;

[1] For an exception to this, see Fig. 24, p. 64.

(2) in the occurrence of systemic emboli ; (3) the presence of splenomegaly, clubbing of the fingers and a positive blood culture. Acute infective endocarditis may supervene on a valve previously damaged by rheumatism, and the diagnosis may become extremely difficult : persistent absence of embolism, a persistently negative blood culture and the presence of auricular fibrillation suggest an active rheumatic lesion. Malignant endocarditis rarely supervenes upon a recently active rheumatic lesion. The endocarditis resulting from syphilis or arteriosclerosis is an afebrile condition.

The *Prognosis* of rheumatic endocarditis, though the malady may last for many weeks, or even months, is favourable as regards life, but the damage to the cardiac valves is generally permanent, and then the prognosis turns on many important considerations (§ 61). A tendency to recur is one of the most striking features of the disease.

Treatment should be directed primarily to the rheumatic fever : sodium salicylate is usually thought to have no control over the cardiac lesion. Digitalis is occasionally of use in reducing the heart rate in cases of persistent tachycardia. Iron is useful as ferri et ammon. cit. 15 gr. t.d.s. for the anæmia which is so often present. Penicillin is of no value in this condition. *Perfect rest*—hardly allowing the patient to turn in bed—is absolutely essential. The patient should be confined to bed until fever has been absent for at least 6 weeks, the sedimentation rate has been normal for 4 weeks, the pulse rate has returned to normal and has remained normal for 3 weeks, anæmia has vanished, and the patient has begun to put on weight. Careful treatment of the condition may require prolonged rest in bed for 6 to 9 months (and see § 582). The erythrocyte sedimentation rate is of great value in assessing the arrest of the active process (§ 927).

§ 50. Infective, Bacterial, Malignant or **Septic Endocarditis.** This is an acute infection which attacks the heart valves or the endocardium already damaged by previous disease. It is characterised by the presence of the large vegetations, by local destruction of the valves, fever, bacteræmia and multiple embolism. The disease probably never attacks a healthy endocardium. The commonest predisposing cardiac lesions are rheumatic or syphilitic endocarditis, and such congenital lesions as a bicuspid aortic valve, a patent intraventricular septum, or a patent ductus arteriosus.

There are two chief clinical types, Acute and Subacute.

Acute Infective Endocarditis is generally a terminal event following pneumonia, osteomyelitis, erysipelas, typhoid fever, bacillus coli infection, septic wounds, gonorrhœa and meningitis. The illness is usually continuous with the infection which precedes it. The organisms most commonly concerned are : streptococcus pyogenes, pneumococcus, staphylococcus, meningococcus, gonococcus, B. typhosus and B. coli.

Symptoms.—There is an exacerbation of the previously existing fever. The patient is more severely ill, perspires profusely and may have rigors. Typhoid fever may be simulated by the onset of a somnolent state or a muttering delirium. If the infection is affecting the mitral or aortic valves, signs of embolism may be present. These may be gross or minute. Gross embolism affects most commonly the spleen, producing pain in the left hypochondrium ; the kidney, producing hæmaturia and sometimes lumbar pain ; the retinal artery, producing blindness ; the cerebral vessels, producing hemiplegia or meningeal symptoms ; and the arteries of the extremities,

producing sudden pain in an arm, leg, finger or toe, pulsation then being absent distal to the block. More rarely, coronary embolism, producing sudden death, and mesenteric embolism, producing acute intestinal pain, bleeding and meteorism, may occur. Minute embolism produces a petechial rash, subconjunctival and retinal hæmorrhages, and hæmorrhages into the muscle of the hands and feet, producing localised red tender swellings known as Osler's nodes. Rarely the lesion is on the right side of the heart producing periodic attacks of cough, pleurisy and sometimes hæmoptysis, due to small pulmonary emboli.

Signs.—The fever is generally of the hectic variety and the pulse rate is raised. The heart is more or less enlarged, and the valvular murmur of the attacked valve is present. The enlarged spleen may be palpable. Blood cultures are generally positive, but several may have to be taken before a positive culture is obtained.

Diagnosis.—Septicæmia without endocarditis, typhoid fever, tuberculosis, meningitis, acute leukemia and pernicious anæmia, often have to be considered in the differential diagnosis.

Prognosis.—Acute malignant endocarditis may be curable by penicillin (see p. 70).

Subacute Infective Endocarditis (Synonym: Subacute Bacterial Endocarditis).—In this form of the disease also a previously existing cardiac lesion is attacked by a secondary infection. The cardiac lesion is as described above, but the infecting organism is in the great majority of cases a normal inhabitant of the mouth or intestine, the streptococcus viridans. Rarely the influenza bacillus is present.

Symptoms.—The four cardinal findings are fever, a cardiac lesion, signs of embolism and a positive blood culture. The fever may continue for months (Fig. 125), the only complaint of the patient being undue lassitude, marked sweating at night, and transient pains in the joints or fingers. But sooner or later a doctor is called in, and in addition to the fever a definite mitral or aortic valvular lesion is found. Embolism may produce a more dramatic picture, producing the symptoms described under the acute form of the disease. An earthy pallor with moderate anæmia is characteristic. Particular search should be made for the petechial emboli into the conjunctiva, retina and skin. Clubbing of the fingers is often found. Rigors are less common than in the acute form. The patient is at first not incapacitated to any severe extent, and may have continued his usual occupation.

Diagnosis.—The diagnosis is generally comparatively easy, but cases occur where owing to the absence at first of signs of embolism, or owing to difficulty in obtaining a positive blood culture, it may remain in doubt for a period. Fever and a cardiac lesion occur in *acute* or *subacute rheumatism*, and pains in the joints are common, but a positive blood culture is never obtained. The joint lesions of acute rheumatism are brought to an end by full doses of salicylate, those of subacute bacterial endocarditis are unaffected. A cardiac lesion and systemic embolism occur in advanced mitral stenosis, but here the patient is afebrile, and auricular fibrillation is generally present; this irregularity is very rare in malignant endocarditis. Continued fever, possibly with a systolic cardiac murmur, may be present in other causes of severe *anæmia*, sometimes with splenic enlargement and petechiæ, but the blood count and

response to treatment are generally diagnostic. *Lymphadenoma, acute tuberculosis, B. coli pyelitis,* and other forms of *local sepsis* must in some cases be excluded.

Prognosis.—These patients almost invariably die unless treated with penicillin, when there is a recovery rate of about 75 per cent. The remainder die of heart failure, renal failure, or embolism.

Treatment.—The invading organism should be isolated by blood culture, and its sensitivity to penicillin determined. When penicillin sensitive, 500,000 to 2,000,000 units per day, by three-hourly intramuscular injections, are given for 42 days. This treatment is of value in both acute and subacute infective endocarditis. Transfusion is valuable when severe anæmia is present; attention should be paid to the general health. Rest in bed is necessary until the patient is clearly convalescent. Massage and movements to the legs should be continued throughout the illness.

GROUP B. We now turn to those cardiac disorders in which **Pain** is the leading feature. The other cardiac condition giving rise to pain is PERICARDITIS.

In PERICARDITIS the degree of pain is very variable; and it is recognised by the other symptoms and signs fully described in § 46.

The patient, probably a male, at or past middle life, is suddenly seized with "constricting" PAIN IN THE CHEST, *accompanied by a sense of suffocation—the condition is* ANGINA PECTORIS.

§ 51. Angina Pectoris. There are four types of angina—I, Angina of Effort; II, Spasmodic Angina; III, Coronary Thrombosis (Status Anginosus); IV, Angina Innocens (Pseudo-angina). More than one of the varieties may be present in the same patient.

I. **Angina of Effort.** *Symptoms.*—The pain is of dull constricting type, substernal and often radiating to the arms. It is not present at rest, and is proportional to the amount of exercise taken. It may or may not be accompanied by dyspnœa. It is often more marked if exercise is taken after a meal, and is thus sometimes mistaken for dyspepsia. Hyperæsthesia is absent. The pain is relieved by amyl nitrite.

Etiology.—The pain is due to an inadequate coronary flow during exertion. It is caused by atheroma or by syphilitic aortitis. Even in cases with no sign of cardio-vascular disease a Wassermann reaction should still be done. After death one of three conditions may be found: (1) The heart itself may be unhealthy, as the result usually of changes in the coronary vessels; (2) the heart may be apparently healthy, but disease of the aorta may be present; (3) the heart and the vessels may both be diseased. The *immediate cause of the attack* is anoxæmia (lack of oxygen) of the heart muscle, due to increasing work thrown on the heart by physical effort, mental stress or mechanical embarrassment, such as is produced by cold (vaso-constriction of skin vessels), or distension of the stomach by food or flatulence.

Prognosis.—The two points to be clear about are (1) the sensitivity of the patient's nervous system; (2) other evidence of myocardial disease.

The more nervous the patient the slighter will be the lesion which will cause pain, and the better is the outlook. A bad prognosis is suggested by considerable cardiac enlargement, high blood pressure, pulsus alternans, cardiac asthma, and marked dyspnœa. In the syphilitic cases, if treatment is sufficiently thorough, recovery may occur or the lesion may be arrested; the presence of aortic regurgitation makes the outlook more serious. Relatives should be told that the condition is serious, and that an exact prognosis is not possible. The condition of the cardiac muscle is the best guide to the probable course of a case (§§ 57 and 61).

Treatment of angina of effort. The patient should be calmed and reassured. Undue apprehension and nervousness are apt to accentuate the symptoms. The term angina should not be mentioned to the patient. For the pain a tablet of glyceryl trinitrate should be crushed in the mouth or held under the tongue for a few minutes before being swallowed; the dose is $\frac{1}{130}$ to $\frac{1}{65}$ gr. Prophylactically, attention should be directed to lightening the cardiac burden, by adjustment of the activities, treatment of adiposity and of dyspepsia. A glyceryl trinitrate tablet should be taken just before undertaking exertion which is likely to produce an attack of pain. Hurry and nervous tension must be avoided. Rest should be insisted on after every meal. Diet the patient strictly according to these principles: four meals daily (the object of four meals being that comparatively little food is introduced into the stomach at once); no fluid to be taken until after a meal, so as to avoid diluting the gastric juices and delaying digestion. Avoid bulky food which takes a long time to digest. If there is marked flatulence, in addition to the above regime, the following mixture can be given immediately after food: Spir. ammon. aromat., spir. ætheris nitrosi, spir. cajuputi, in equal parts, 60 min. to be taken in a little water. Useful drugs are phenobarbitone gr. $\frac{1}{2}$ t.d.s., p.c., with or without theobromine and sodium salicylate gr. 10 t.d.s., p.c., or theophyllin and sodium acetate gr. 2 t.d.s., p.c. Thyroidectomy, sympathectomy and alcohol injection (p. 72) are useful measures for the prevention of pain in carefully selected and severe cases in which other means have failed.

II. **Spasmodic Angina.** The attack of pain is sudden, not proportional to exertion, is very severe, and often associated with a feeling of impending death. It is probably due to coronary spasm, accentuated by cold or emotion.

These symptoms may be superimposed on those of angina of effort; rarely they are present alone. The attack of spasmodic angina comes on quite suddenly, often after exertion, especially after a meal or in the cold. It consists of (1) acute pain in the chest, which radiates down the arms, especially the left: the site of the pain, as Mackenzie pointed out, is over the distribution of the four upper dorsal nerves, across the chest; the skin may be hyperalgesic over this area. The face is expressive of the pain which the patient suffers. Pallor and sweating are present. The patient usually keeps quite still, being afraid to move for fear of

increasing the agony. The sense of suffocation, of bodily discomfort, constriction of the chest, and of impending dissolution is extreme. The attack lasts for a few seconds to a few minutes, and is liable to be aggravated if the patient ventures to move from the position which he may have assumed. (2) The heart's action, when examined during an attack, is sometimes found to be unaltered, though palpitation may be complained of. Electrocardiographic examination often shows some irregularity, usually due to premature beats. The blood pressure during the attack is raised. The pulse rate is in some cases increased. The heart usually shows some enlargement, and often some form of aortic valvular mischief is present (see Etiology below). (3) The mind remains clear throughout. Many attacks are accompanied or succeeded by a profuse flow of urine; others by profuse perspiration. The limbs and other parts which were the seat of pain may afterwards feel "numbed." (4) Patients are usually of the male sex, and over 50 years of age. The disease also appears to affect by preference hypersensitive individuals.

Etiology.—There is often a familial tendency. Coronary atheroma, aortic regurgitation, and aortic syphilis are the most common causes.

Prognosis.—The same considerations enter into the prognosis in spasmodic angina as in angina of effort, and death from coronary thrombosis is always a possibility.

Treatment of spasmodic angina.—The pain is relieved by amyl nitrite or nitroglycerin. Hot whisky and water is sometimes useful, especially at night. Morphia may be necessary. Great attention should be paid to keeping the rooms warm, and the bed well warmed, as many attacks are produced by passing from a warm room to a cold passage, and by getting into a cold bed at night. Good nights must be ensured by the use of bromidia, chloral, or even morphia when necessary. If the cause of the condition is an inadequate oxygen supply, the object of treatment is twofold, namely: (1) to reduce the work of the heart, and (2) to increase the oxygen supply, *i.e.*, the blood supply to the muscle. Between attacks, the treatment is identical with that described for angina of effort.

There are two methods in use for blocking the stimuli in anginal pain, sympathectomy of the middle and lower cervical ganglia, and alcohol injection of the five upper dorsal sympathetic *rami communicantes*. The choice of appropriate cases is a matter for expert opinion, but it can be stated briefly that the chief indications for mechanical interference are: (1) The pain has a coronary origin; (2) syphilis has been excluded; (3) the cardiac function of the patient is otherwise good; (4) gross disease is absent.

§ 52. III. **Coronary Thrombosis.**—In disease of the coronary arteries, as in other damaged vessels, thrombosis is very liable to occur. Moreover, as with thrombosis in other positions, it often takes place when the circulation is slowed, and hence frequently comes on in the night. When it occurs the patient gets a sudden violent attack of pain, usually in the lower part of the chest behind the sternum. It does not radiate so definitely as in angina, lasts on and off for a period of hours or days and is associated with marked restlessness. The main differences between

an attack of angina and coronary thrombosis are indicated and summarised as follows :

TABLE II.

	ANGINA OF EFFORT.	CORONARY THROMBOSIS.
I.	Attack comes on during exercise, cold or emotion. Especially after meals.	Attack comes on when circulation is slowed, especially therefore in the night.
II.	Patient is brought to a standstill by pain, which then soon goes.	Patient is restless, collapsed, sweating and often slightly cyanosed or flushed.
III.	Attacks last a few minutes.	Attacks may last some hours or days.
IV.	Pain referred definitely to left or both arms, throat or epigastrium.	Pain not so diffuse, is substernal, and usually lower down—even epigastric ; is often more agonising.
V.	Pain relieved by vaso-dilators.	Pain unaffected by vaso-dilators.
VI.	Arterial B.P. usually rises.	Arterial B.P. falls markedly. Venous pressure very much raised.
VII.	No fever.	Slight fever within 24 hours.
VIII.	No leucocytosis.	Leucocytosis with the fever.
IX.	Erythrocyte sedimentation rate normal.	Erythrocyte S.R. raised.
X.	Friction sounds absent.	Pericardial friction often present after about the fourth day.
XI.	Electrocardiogram. Left-sided preponderance usually present. The tracing is unchanged in shape.	Electrocardiogram always shows a changing QRST (Fig. 27 and § 44).
XII.	Heart sounds clearly audible.	Heart sounds weak.

Diagnosis.—Conditions simulating coronary infarction are pulmonary embolism, perforated peptic ulcer, pneumothorax and gall-stone colic. Pulmonary embolism, if not fatal, may cause sudden pain, dyspnœa, collapse, and cardiac embarrassment. Here the neck veins are greatly distended, the electrocardiogram is like that of posterior coronary infarction except that in pulmonary embolism the "T" wave in the chest lead placed near the right sternal border (CR1) is inverted. The associated cardiac condition is often called "acute cor pulmonale."

Prognosis.—After coronary thrombosis it is improbable that the heart will be as efficient as before, but in certain cases where the myocardium is otherwise healthy, apparent recovery occurs. Points suggesting an unfavourable prognosis are : marked enlargement of the heart, signs of congestive failure and the presence of diabetes. A second thrombosis occurs in 25% of cases. Complications are cardiac irregularities, heart failure, occasionally hemiplegia from intra-ventricular clot, and rupture of the heart.

Treatment of Coronary Thrombosis.—Keep the patient in bed for six weeks at least ; give oxygen for cyanosis ; give full doses of morphia to relieve the pain and procure rest, and repeat it if necessary. After the second week given diuretin and phenobarbitone. Slow resumption of normal activities is encouraged up to the level of comfort : and mental and physical overstrain must at all times be avoided.

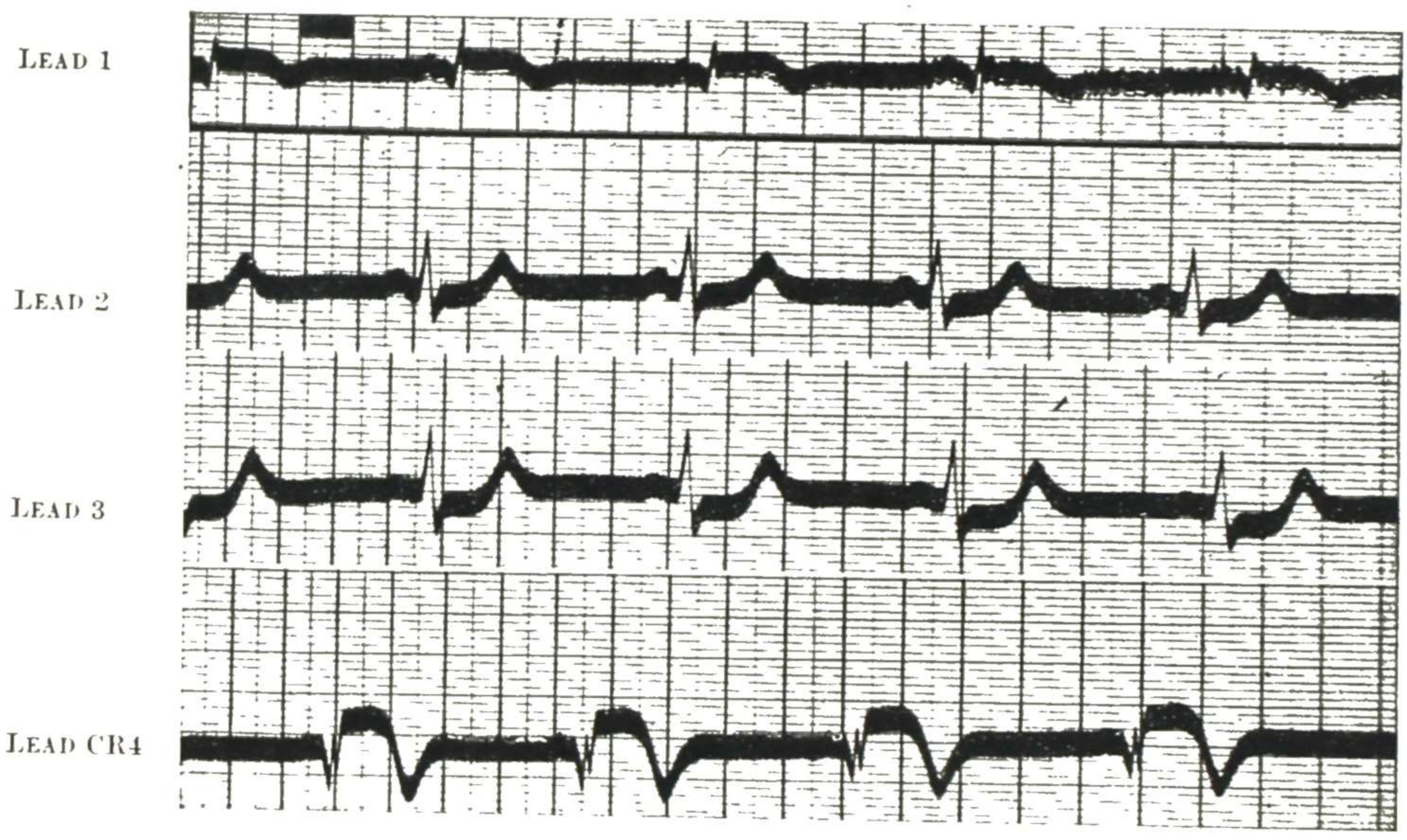


FIG. 25.—ELECTROCARDIOGRAM three days after an acute ANTERIOR CORONARY THROMBOSIS. Note S–T elevation in leads 1 and CR4, and S–T depression in leads 2 and 3.

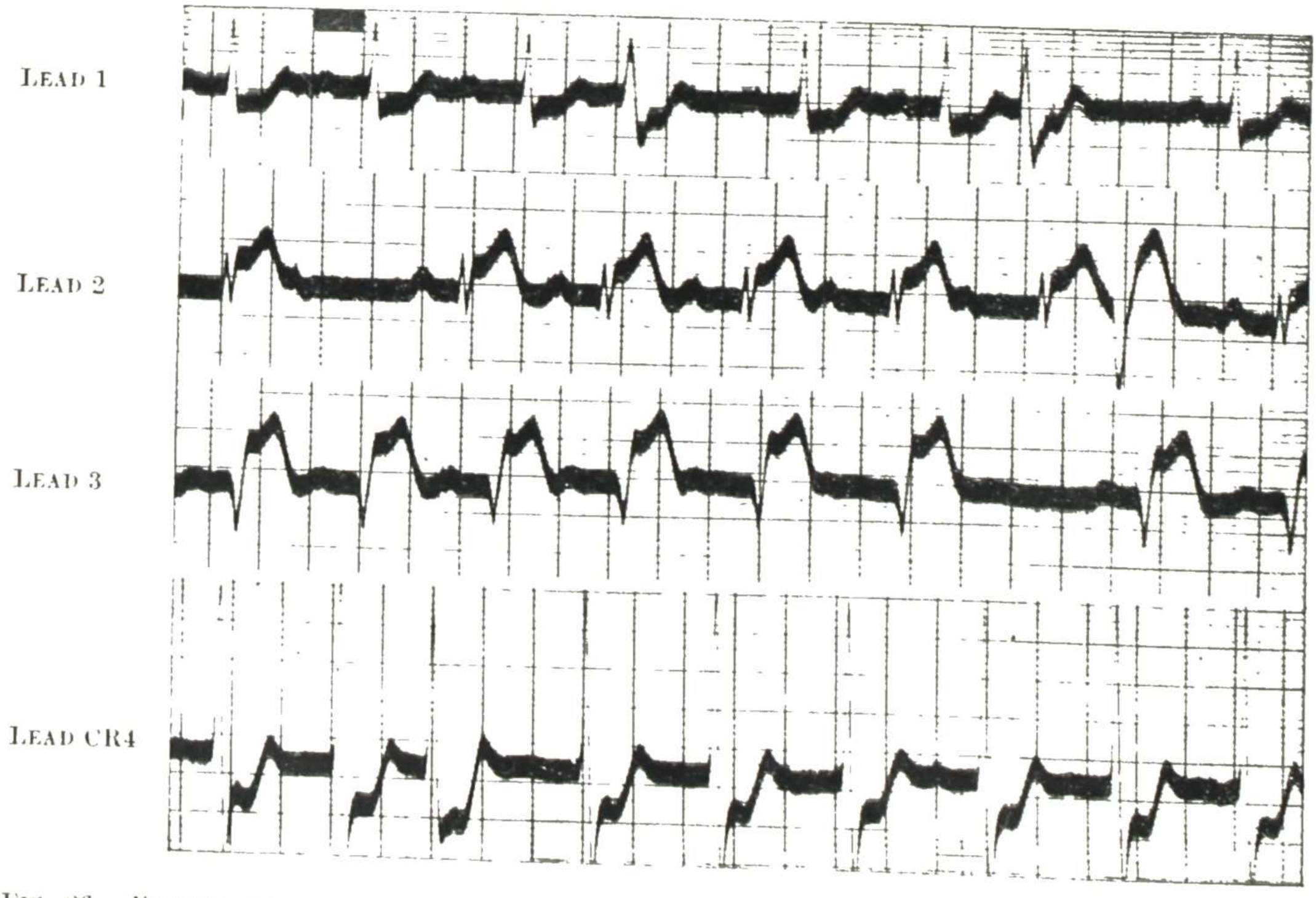


FIG. 26.—ELECTROCARDIOGRAM two days after an acute POSTERIOR CORONARY THROMBOSIS. S–T elevation is well shown in leads 2 and 3, and S–T depression in leads 1 and CR4.

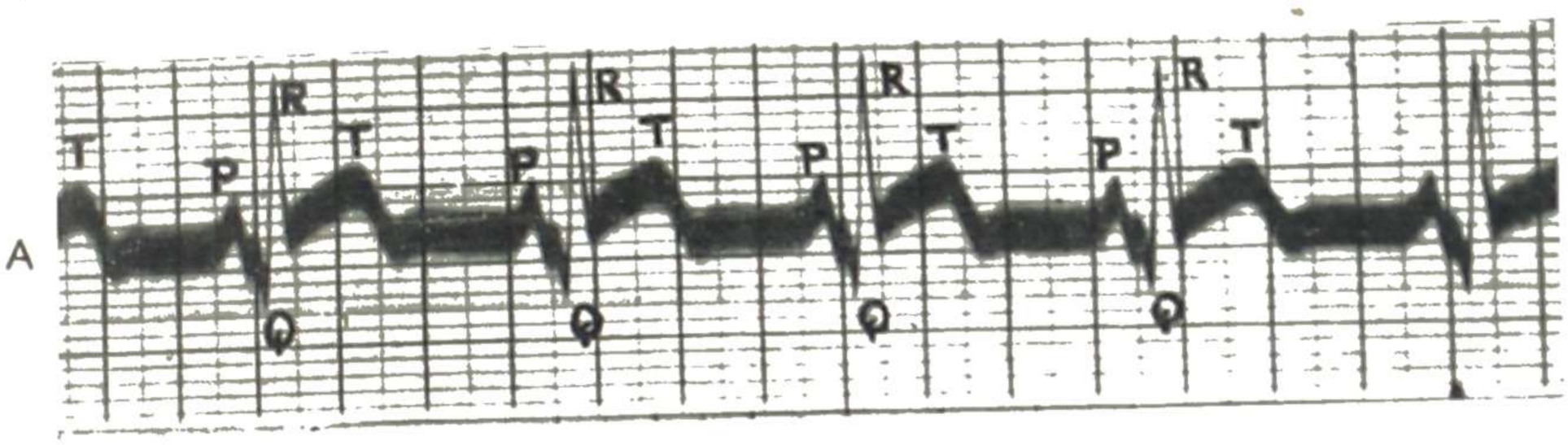


FIG. 27.—CORONARY THROMBOSIS (anterior branch).

All tracings are of lead 1.

A. Three days after coronary thrombosis; note the high "take-off" of the "T" from the "R" wave.

B. Ten days later. Note inversion and spiky shape of "T."

C. Three weeks after thrombosis. "T" is beginning to return to its normal upright shape; it is diphasic. Later, the RST curve may become quite normal again.

§ 53. IV. Angina Innocens. Cardiac pain of a functional type is common and is labelled angina innocens or *pseudo-angina*. It must be distinguished from pain due to an organic lesion. The safest criterion is the relation to exercise. If there is never pain at rest, if it is strictly proportional to exercise and only occurs during exertion, it is of organic origin. Other evidences in favour of true angina are: the presence of dyspnœa, of cardiac enlargement, hypertension or aortic disease, and the absence of præcordial tenderness. Evidences in favour of angina innocens are: an increase of pain after exertion—not during it, the presence of palpitation, giddiness and fainting attacks, the presence of præcordial hyperæsthesia, evidence of an unstable nervous system or a history of nervous breakdown. In angina innocens there is no cardiac enlargement, and no evidence of cardiac disease clinically, electrocardiographically, or by X-ray. However, there is an exception to this rule in cases of mitral stenosis with pain; these patients have pain of the angina innocens type.

There is a second type of pain occasionally found in patients with angina innocens. It is identical with that described by Gowers (Vaso-vagal

attacks of Gowers, see § 720). These attacks have a sudden onset, there may be a sense of impending death ; severe præcordial pain may radiate to the arms, or tingling in the arms may be complained of. Marked pallor and bradycardia are the most striking signs. The patient generally faints.

Prognosis. In angina innocens, though the symptoms may persist for years, there is no danger to life and recovery always occurs in time. The vaso-vagal attacks have no bad prognostic significance.

Treatment of angina innocens consists in reassuring the patient, in removing obvious physical abnormalities such as marked dental or tonsillar sepsis, in attempting to adjust any psychological stresses, and in allowing a gradual increase of exercise taken by the patient. Treatment in a special psycho-therapeutic centre is often advantageous. The best drugs are phenobarbitone and bromides.

GROUP C. We now consider those conditions in which examination reveals **Enlargement** of the **Area of Præcordial Dulness.**

a. If there is a history of acute onset, and there is PYREXIA, the condition is due to ACUTE PERICARDITIS, which is fully described in Group I, § 46, or to DILATATION secondary to a febrile process.

b. If there is no Pyrexia, the enlargement may be due to

I. Cardiac Hypertrophy.
II. Cardiac Dilatation.
III. Chronic Pericardial Effusion.
IV. Adherent Pericardium.
V. Congenital heart disease (rare).

VI. Aortic Aneurysm and Mediastinal Tumours must be remembered, because their existence is often revealed by finding enlargement of the præcordial dulness, or dulness above, merging into that of the heart.

Chronic conditions which may be, but are NOT NECESSARILY, attended by ENLARGEMENT of the area of præcordial dulness should be borne in mind ; their diagnosis may depend mainly on auscultation, and hence they are described under Groups D and E.

Method of Procedure.—It will be remembered that the routine examination of the heart consisted of (1) inspection ; (2) palpation ; (3) percussion of the præcordial dulness ; (4) auscultation ; and (5) in any patient in whom cardio-vascular disease is suspected, radiography and electrocardiography can give valuable help. The student should bear in mind the various *fallacies* which may give a false impression of cardiac enlargement, and also those conditions, such as emphysema, which obscure an enlarged heart (§ 40).

I. *The* APEX BEAT *is* BELOW *its normal position ; the impulse is* FORCIBLE *and heaving ; on auscultation, the first sound is* DULL *and prolonged. There is* HYPERTROPHY OF THE HEART.

§ **54. Hypertrophy of the Heart,** and the dilatation which not infrequently accompanies or follows it, are certainly the commonest conditions which produce an increased area of præcordial dulness.

Cardiac Hypertrophy is an increase of the muscular substance of the heart, and its weight, which is normally about 8½ ounces in women and 9½ ounces in men, may be increased to 10 or 12 ounces, and on rare occasions to 15 or 20 ounces. Its *signs* are as follows: (1) The increase in the præcordial dulness is downwards and outwards if the left ventricle be hypertrophied, outwards only if the right ventricle; (2) the apex beats below and outside its normal position; (3) the impulse is unduly forcible, heaving, or thrusting, the thrust of the hypertrophied right ventricle being generally felt in the epigastric notch; (4) on auscultation, the first sound is loud and prolonged. The pulse is firm, strong and bounding.

Symptoms may be altogether absent if the hypertrophy accurately compensates for the obstruction in the circulation which has caused the hypertrophy. The patient may, indeed, be unaware of any cardiac disorder.

Etiology.—Hypertrophy is caused by an increase in the work to be performed. The part of the heart which undergoes hypertrophy is that immediately behind the lesion: the signs of such a lesion will be additional to those caused by hypertrophy. Thus, there will be three sets of signs: (*a*) Signs of hypertrophy of the heart as a whole; (*b*) signs of enlargement of the chamber specially involved; and (*c*) signs and symptoms of the cause. The following causes will be more readily understood by consulting Fig. 28 (p. 86), and it must be remembered that the enlargement is rarely in actual practice strictly limited to one chamber of the heart.

(*a*) HYPERTROPHY OF THE LEFT VENTRICLE is indicated by displacement of the apex beat *below* and to the left of its normal position. The apical impulse is strong, sustained and heaving in character. There is also enlargement of the area of cardiac dulness to the left. The pulse is strong unless modified by the presence of a valvular lesion, and the carotids may be seen to pulsate.

Etiology.—Hypertrophy is always secondary, and is proportional to the extent of the causative lesion, provided that compensation has occurred. It is an illustration of the physiological law that increased use leads to increased growth. Hypertrophy of the left ventricle is due to one of the following causes:—hypertension (§ 94), aortic regurgitation or stenosis, mitral regurgitation, healed chronic myocarditis and adherent pericardium. The largest of all hearts are those in which chronic pericarditis with adhesions is present (§ 56, IV). The writer has seen one such case where the heart weighed more than the liver. The commonest cause of marked enlargement is aortic regurgitation. Aortic stenosis produces only a slight degree of enlargement, the whole of which is due to hypertrophy. *Aneurysm of the aortic arch*, if unattended by valvular disease or renal mischief or arterial disease, does not *per se* cause cardiac hypertrophy; if enlargement is present, it is due to an associated aortic regurgitation or hypertension. *Excessive muscular exercise*, whether athletic or laborious, may produce hypertrophy, and in support of this statement it may be mentioned that the normal increase with age is more noticeable in men than in women.

(*b*) HYPERTROPHY OF THE RIGHT VENTRICLE is indicated by enlargement of the area of dulness to the right; and a heaving impulse in the epigastrium. It is the result of resistance to the emptying of the ventricle into the pulmonary vessels. This may occur in:

(i.) *Pulmonary diseases* attended by obstruction in the pulmonary circulation, of which *bronchitis with emphysema* is certainly the most frequent. This condition, a very common one, is identified by a history or evidence of lung mischief (§ 142).

(ii.). *Mitral stenosis* is the next most common cause, and should be borne in mind even in the absence of a presystolic murmur (§ 60).

(iii.) *Mitral Regurgitation* (§ 58).

(iv.) Raised pulmonary pressure from pulmonary arterio-sclerosis, Ayerza's disease (see § 31).

(v.) Congenital pulmonary stenosis.

(*c*) HYPERTROPHY OF THE LEFT AURICLE is always attended by dilatation. It is a difficult condition to recognise with certainty except with X-ray examination (Figs. 22 and 23).

It may arise in *mitral regurgitation*, but its chief cause is *mitral stenosis*. In the latter condition, palpation generally reveals a thrill over the apex, and careful auscultation may detect the presystolic or mid-diastolic murmur (§ 60).

(*d*) HYPERTROPHY OF THE RIGHT AURICLE gives rise to the following physical signs: (i.) Increase of dulness to the right of the sternum; (ii.) powerful jugular pulsation, which polygraphic records prove to be due to forcible auricular contractions.

(*e*) Extreme HYPERTROPHY OF BOTH AURICLES AND VENTRICLES arises in congenital heart disease, but may be confined to the right side. It also occurs with Adherent Pericardium of the external type (§ 56).

All these conditions can easily be verified by X-ray examination (Figs. 22 and 23).

Prognosis and Treatment.—Cardiac Hypertrophy is essentially a compensatory process for some condition which causes obstruction in the circulation. It is Nature's method of compensating for the increased work.

1. *If the cause be removable*, the prognosis is favourable. Treatment in such cases should therefore be directed to the removal of the cause —*e.g.*, high blood pressure.

2. *If the cause be not removable*, the prognosis of the case depends on the avoidance of myocardial failure, which will show itself symptomatically by dyspnœa and physically by dilatation. To accomplish the first, the general health should be improved by general hygienic measures. In order to relieve the heart of part of its work, and to aid the systemic circulation, baths, massage, passive and active movements are of the greatest use (see § 62).

3. The *existence of cardiac hypertrophy* indicates an element of risk to a person's life from one of two factors. In the first place, hypertrophy nearly always indicates that there is obstruction somewhere in the circulation, and this, whatever it be, is in itself an injury to health, and may shorten life. Secondly, it is an indication in many cases of associated arterial disease and hypertension, with the risks of coronary and cerebral disease and of renal failure (§§ 93 and 94).

II. *The* AREA OF DULNESS IS INCREASED; *the position of the* APEX BEAT IS INDEFINITE; *the impulse is diffuse, wavy and slapping; on auscultation, the first sound is short and sharp. The condition is* CARDIAC DILATATION.

§ 55. Cardiac Dilatation (an important indication of "Myocardial failure") suggests that the heart is "failing" to keep pace with the demand made upon it, that the reserve power of the muscle wall is becoming spent. Dilatation is the immediate physiological response of the heart to increased work. If increased work continues, hypertrophy normally follows. Physiological dilatation is limited by the pericardium. Strictly speaking, both the dilatation and the symptoms and signs of failure are due to a common cause, the underlying myocardial failure—which is usually due to toxæmia or to anoxæmia.

Myocardial failure, nearly always accompanied by dilatation, generally involves both left and right ventricles. Usually the left side is first affected and its failure later causes the right ventricle to fail also. In rare cases the right ventricle fails alone: orthopnœa is absent in pure right ventricular failure. Heart failure with clinical œdema is called congestive heart failure.

(I) Dilatation of the Left Ventricle indicates some degree of **left ventricular failure.**

Physical Signs.—In the early stages we find: (1) the cardiac impulse by palpation is wavy and diffuse, and is displaced outwards rather than downwards: it may be so feeble as to be hardly perceptible. (2) There is increase in the area of cardiac dulness in a transverse direction to the left. (3) On auscultation the first sound is sharp and clear due to the closure of the mitral valve and the usual muscular element in the sound is largely inaudible. Both first and second sounds are often faint and the period of systolic output shortened. (4) Murmurs may be present from co-existing valvular disease, but a systolic murmur—the "murmur of dilatation"—may be heard apart from actual valvular disease, because the dilated auriculo-ventricular orifice allows a reflux of blood (§ 58, I*a*). (5) The pulse may be feeble and rapid. *In the later stages*, when left ventricular failure is more severe, there may appear (1) pulmonary congestion and œdema (*cardiac asthma*). This is most noticeable in the night: it is revealed by orthopnœa, dyspnœa, cough, expectoration of mucus sometimes tinged with blood, or actual hæmoptysis. The physical signs are abundant râles and, sometimes, scattered patches of dulness at one or both bases. (2) *Fœtal rhythm.* In this the systolic and diastolic intervals are almost identical, making it difficult to identify the first and second cardiac sounds. (3) In *delirium cordis* or auricular fibrillation the heart is so rapid and irregular that it becomes difficult to make out the relations of sounds and murmurs. (4) In *gallop rhythm* there is rapidity of action, together with a distinctly reduplicated first or second sound.

Gallop rhythm is a condition in which three heart sounds, instead of two, are heard in each cardiac cycle. *Pathological* causes are (i.) a Bundle-branch lesion (Fig. 38). Owing to the fact that conduction is unequal down each branch of the Bundle of His, the contraction of the two ventricles is slightly asynchronous. The first heart sound at the apex is thus reduplicated. (ii.) A reduplicated first

sound may be present with left ventricular dilatation, generally in association with a failing heart in a patient with hypertension. (iii.) A third heart sound may be produced in the early stage of mitral stenosis by the discharge of blood from the left auricle through the mitral valve in mid-diastole. *Apart from disease*, three heart sounds may occur in two conditions: (i.) A physiological third heart sound heard as a very faint sound in the middle of diastole. (ii.) When the ventricular systole, by mechanical means, produces a sound outside the heart. This exocardial sound generally disappears either during full inspiration or during full expiration.

(II) Dilatation of the Right Ventricle follows left-sided heart failure or is due to obstruction of the blood flow through the lungs. It causes enlargement of the area of cardiac dulness to the right, and often marked epigastric pulsation. Sooner or later the symptoms and signs of **right-sided heart failure occur :**

(i.) A *bruit* over the tricuspid orifice is sometimes heard (see p. 89).

(ii.) *Fulness* and *pulsation* in the neck veins, due to tricuspid regurgitation.

(iii.) *Dropsy*, which indicates congestion of the whole venous system. Cardiac dropsy *starts and predominates in the legs* or *the back*, whichever may happen to have been in the most dependent position. The skin is tense, and is very liable to be attacked by erythematous, erysipelatous, and inflammatory conditions (cellulitis, ulcer, etc.). *Ascites* in varying amount is generally present. It is often an early and prominent sign in mitral stenosis. *Cyanosis* and a general lividity of the surface are consequences of the same venous stasis. A case of mitral disease, therefore, presents a marked contrast to one of aortic disease, where the countenance is often pale.

(iv.) *Engorgement of the liver* is evidenced by pain and tenderness in that region, and later jaundice of the skin and sclerotics. The organ is enlarged, and it may extend to the umbilicus. Sometimes pulsation of the liver may be made out by placing one hand on the epigastrium, and pressing the other beneath the back in the dorsal region. In cases of *dropsy with albuminuria*, when in doubt whether the dropsy is of renal or cardiac origin, hepatic enlargement is a valuable diagnostic aid; its presence is usual in cardiac cases.

(v.) *Indigestion*, want of appetite, a sense of discomfort in the stomach after meals, nausea or actual vomiting, with streaks of blood, indicate congestion of that organ.

(vi.) *Albuminuria*, with high-coloured scanty urine of high specific gravity (and possibly casts and some blood in long-standing cases), point to congestion of the kidney.

The *Causes of Cardiac Dilatation* are of extreme importance as bearing on the prognosis and treatment of cardiac valvular disease and other circulatory disorders. The *clinical conditions* which produce dilatation are practically identical with those which produce cardiac hypertrophy (§ 54), when they are persistent and *are associated with some condition*

which impairs the nutrition of the heart (see Etiology). Undoubtedly the commonest causes of cardiac hypertrophy with dilatation are CARDIAC VALVULAR DISEASE, HYPERTENSION WITH MYOCARDIAL DEGENERATION (producing left-sided dilatation), and CHRONIC BRONCHITIS WITH EMPHYSEMA (producing right-sided dilatation). These are the possibilities which should first suggest themselves to the mind in a case where dilatation is evident.

Etiology.—Pathologically speaking, Dilatation falls under two heads:—Compensatory Dilatation and Dilatation due to failure.

Examples of *compensatory dilatation* are found in aortic and mitral regurgitation. In each case the left ventricle has to accommodate first the normal amount required for each normal systole, and secondly the quantity which will regurgitate through the damaged valves. *Dilatation due to failure* occurs whenever the heart muscle is insufficiently poisoned, or becomes anoxæmic. The commonest causes of this myocardial failure are: (*a*) Infections such as acute rheumatism, diphtheria, typhoid fever, septicæmia, pneumonia, influenza, tuberculosis, malaria and possibly syphilis. (*b*) Poisons such as arsenic and alcohol. (*c*) Metabolic states such as hyper- and hypo-thyroidism, beri-beri, scurvy.

The commonest causes of cardiac *anoxæmia* are: (*a*) Severe anæmia, of the primary or secondary type; (*b*) Coronary artery disease.

Any *sudden strain on an apparently normal heart* may produce acute dilatation. But no really normal heart will dilate under strain. In the cases where this occurs, some mild infection or other lesion is present. Thus severe muscular exertion in athletes or soldiers who have not had any previous training may seem to cause the heart to fail. Instances are met with in hill-climbers who are "out of form," and others who take sudden and unaccustomed exercise. Breathlessness may date from incidents of this kind, from which the patient may never, or only with difficulty, recover. Rest and gentle exercise are indicated. Prolonged fatigue may similarly overtax the heart muscle if it is already diseased.

The *Prognosis* and *Treatment* of Cardiac Dilatation are fully dealt with under Cardiac Valvular Disease (§§ 61 and 62).

III. *The area of dulness is* INCREASED UPWARDS, *and its shape is pyramidal, with the point upwards; the apex beat is raised, and the impulse is weak and undulatory; on auscultation, the sounds are feeble. The signs are of long standing. The disease is* CHRONIC PERICARDIAL EFFUSION.

§ 56. In **Chronic pericardial effusion** (Synonym: Hydropericardium), the *Symptoms* are due to increasing intrapericardial pressure: there is increasing respiratory distress. *Signs:* (1) There is congestion of the neck veins. (2) The shape of the dulness is very characteristic, being pyramidal, with the narrow end upwards. (3) The apex of the heart is *raised*, and *to the right* of its normal position, because the roof of the pericardium is raised by the fluid, and takes the heart with it. (4) For the same reason, the left margin of præcordial dulness extends *beyond* the apex beat. (5) On auscultation, the heart sounds are distant and muffled. There may be irregularity and rapidity of the pulse, and difficulty of breathing from the impeded action of the heart and lungs. (6) Pulsus paradoxus may be present; this is an inspiratory diminution in pulse volume. (7) The physical signs at the base of the left lung are identical with those described with acute pericarditis (§ 46).

Etiology.—Chronic effusion into the pericardium may originate in one of three ways. (1) As the result of Acute Pericarditis (§ 46), of which a history is generally obtainable, but by no means always (see Latent Pericarditis, § 48). (2) True hydropericardium seldom occurs excepting as part of a general dropsy due to renal or cardiac disease, and therefore the urine should be carefully examined. In these circumstances

dyspnœa is the most obvious symptom complained of and an X-ray examination should always be made if there is any doubt as to the existence of fluid. (3) If hydropericardium be not preceded by pericarditis, or be not part of a general dropsy, new growth or tubercle should always be suspected.

The *Diagnosis* from Cardiac Dilatation should be readily accomplished by the shape of the dulness, which is square instead of pyramidal in dilatation; and by the heart sounds, which are clear and sharp in dilatation, muffled in effusion. X-ray examination is of assistance. Pleural effusion is attended by pulmonary symptoms.

The *Prognosis* of hydropericardium depends on its causation, being favourable in Cause 1, adding only a little to the gravity of the primary malady in 2, and being necessarily fatal in malignancy: tuberculous cases often recover.

Treatment.—The treatment of inflammatory effusion is dealt with in § 46. If part of a general dropsy, our efforts must be directed to this. Counter-irritants are sometimes useful. Paracentesis should not be considered unless the cardiac embarrassment is urgent.

IV. **Adherent Pericardium** may exist in three forms: (i.) *Constrictive*, in which the visceral and parietal layers are adherent in varying degree. This condition may be latent, or may produce the signs of venous obstruction as a result of the presence of fibrous or calcareous interference with the venous filling of the heart. The heart may be constricted rather than enlarged. Ascites and peripheral œdema are the commonest signs. Pericardectomy may produce a striking cure. If the pericardial fibrosis or calcification does not obstruct the auricular filling there may be no signs or symptoms. (ii.) *External* adhesions may bind the pericardium to all the surrounding structures. The condition is a sequel to pericardial effusion, with softening and dilatation of the sac. In many cases some degree of internal adhesion is also present. The condition may cause congestive failure; the heart is dilated, and is so tethered that it cannot return to its normal size. The signs are many, but not very reliable. They are (1) a systolic tug at the apex; (2) fixity of the cardiac apex during respiration and with change of position; (3) systolic recession around the apex and along the attachments of the diaphragm, either in front along the lower costal border, or behind in the ninth and tenth spaces (Broadbent's sign); (4) signs of hypertrophy, greater than can be accounted for by the severity of any valve disease which may be present; (5) pulsus paradoxus, or diminution or disappearance of the radial pulse during inspiration of normal depth; (6) signs of incompetence of auriculo-ventricular valves; (7) a diastolic shock in the veins of the neck. Rheumatism is the commonest cause of the condition, and mitral stenosis is often present. The subjects of this condition seem unable to acquire more than a slight degree of "compensation"; slight improvement is soon followed by more complete exhaustion of their cardiac reserve. No permanent improvement is to be expected. Cardiolysis or removal of ribs has been successfully performed for this condition.

(iii.) Pick's Disease or chronic adhesive mediastino-pericarditis. In this condition a diffuse mediastinitis is associated with effusion into one or more of the serous cavities of the body. The *etiology* is obscure, but in some cases tuberculosis is present. The *prognosis* in Pick's disease is not good. Although remissions often occur, and may last for some months, inflammation and effusion recur in one serous cavity or another. *Treatment* consists in removing the fluid by paracentesis, whenever it becomes physically embarrassing, and in nursing the patient in conditions as pleasant and as airy as possible. Diet should be full and well balanced, with an adequate ration of milk, cream and butter.

V. In Congenital Heart Disease the enlarged area of præcordial dulness varies with the lesion. There are usually characteristic murmurs. For the differential signs of this condition, see § 59.

VI. In Aneurysm of the first part of the aortic arch, the upper part

of the dull area is increased transversely, and there is dulness over the sternum. Auscultation may reveal a systolic or diastolic murmur and a loud, sharp, ringing aortic second sound (see § 80).

GROUP D. We now turn to those *conditions in which there is found an* **alteration of the heart sounds, or a murmur.** It is well to bear in mind several fallacies referred to on pp. 48, 53, 87, 88, 89 and 95.

In the absence of these, if *the heart sounds are* FAINT, the disease is MYOCARDIAL DEGENERATION, unless a thick chest wall or marked emphysema is present.

The second sound is ACCENTUATED *or* DOUBLE. This may be due to (i.) High blood pressure; (ii.) aortic aneurysm; (iii.) pulmonary stasis in mitral disease.

The first sound at the apex is unduly LOUD. This may be due to: (i.) Nervousness of the patient; (ii.) mitral stenosis; (iii.) a thin chest wall; (iv.) hypertrophy of the ventricle; (v.) adjacent air-containing cavity acting as a resonator.

The first sound is unduly SHORT. This may be due to: (i.) Dilatation of the ventricles due to myocardial degeneration or toxæmia; (ii.) rapidity of the pulse; (iii.) incomplete filling of the left ventricle (mitral stenosis), resulting in an unduly hurried emptying.

I. *On auscultation the* HEART SOUNDS ARE VERY FEEBLE; *the impulse is weak and slapping. No murmur is heard.* MYOCARDIAL DEGENERATION *may be suspected.*

§ 57. Myocardial Degeneration.—Previously sometimes called Fatty Heart and Fibroid Heart.

The changes in the heart muscle may cause: (1) failure of tonicity, causing dilatation; (2) failure of contractility, causing circulatory inadequacy; (3) changes in the primitive musculature, causing irregularities of rhythm (see § 63). The disease should be suspected when cardiac symptoms arise in a patient with cardiac enlargement, and when other diseases (such as of the valves, or hypertension) can be excluded.

Symptoms and Signs.—The disease will be suspected when the heart is enlarged and there is evidence of arterial disease elsewhere. On exertion there is: (i.) undue dyspnœa; (ii.) marked lassitude; (iii.) palpitation; or (iv.) a tight feeling across the chest which goes on rest; (v.) cardiac irregularities (§ 63) such as premature beats may be present at rest, and are often exaggerated by exercise. Auricular fibrillation, and occasionally auricular flutter occur. When the disease is more advanced (vi.) dyspnœa may be present at night ("cardiac asthma"); (vii.) the cardiac impulse is feeble, the heart sounds poor and the radial pulse weak; (viii.) congestive failure may occur. Myocardial degeneration usually affects both left and right ventricles in fairly equal degree. If the left ventricle is the more diseased, pulmonary congestion and œdema are prominent. When emphysema is a causative factor, the failure chiefly involves the right ventricle.

The degree of degeneration is difficult to estimate clinically. Guidance

on this point may be obtained from. (1) Estimation of the amount of exercise which can be carried out without distress, such as pain, dyspnœa, palpitation. (2) The cardiac enlargement is always detectable radiologically, or clinically. (3) The character of the heart sounds: especially shortening of the first sound. (4) The systolic, and to a much less extent the diastolic, blood pressures fall, from ventricular weakness; this produces a diminution in pulse pressure. (5) Electrocardiographic changes, such as flattening or inversion of the "T" waves in leads 1 and 2, or the presence of a bundle-branch lesion, or R–T deviation due to associated coronary disease, are common.

Causes.—Myocardial degeneration is a consequence of interference with the nutrition of the heart wall. This in the majority of cases is the result of coronary disease associated with a progressive interstitial fibrosis. The fibrosis and the fatty degeneration are part and parcel of the same process.

The Prognosis is uncertain. The earlier stages of the malady are insidious, so that by the time pronounced symptoms appear the mischief may be irreparable. Some patients survive for years: those with marked Cheyne-Stokes' respiration, or pulsus alternans die sooner. In the early stages of cardiac degeneration plenty of fresh air, exercise and good sleep are essential for increasing the reserve power of the unaffected muscle fibres, and if the patient responds to this treatment he may live for many years (Mackenzie). Prognosis and treatment are discussed more fully in §§ 61, 62.

While auscultating the heart three questions should be in the physician's mind: (1) What is the character of the first sound? (2) What is the character of the second sound? (3) Is a murmur present?

GROUP D. II. *A* **Murmur** *is present.* Its source may be:

(1) Endocardial—Endocarditis of valve or wall; narrowing or dilatation of orifice of valves; congenital abnormalities.

(2) Atonicity murmur due to muscular stretching of a valve ring.

(3) Cardio-pulmonary, Cardio-respiratory (§ 59).

(4) Pericardial—friction of Pericarditis; and see §§ 46, 49.

(5) Functional and Hæmic murmurs (see §§ 42 and 535).

The chief points to be considered in diagnosing the *source of a murmur* are given in § 42 and § 49 (Table I).

§ 58. Chronic Endocarditis—Cardiac Valvular Disease—Cardiac murmurs.—Disease of the valves of the heart is revealed on auscultation by the presence of a bruit or murmur, which is added to, or replaces, a normal heart sound.

Method of Procedure. Five features must be carefully investigated in any murmur: TIME OF OCCURRENCE. Whether it REPLACES or merely ACCOMPANIES the first or second sound; POSITION of maximum intensity; direction in which it is CONDUCTED; and CHARACTER. The last named is relatively least important. In order to be quite sure of the time of a bruit, it is wise to place the thumb on the carotid artery while auscultating the chest.

The characters of PERICARDIAL MURMURS have already been given (§ 46); and their diagnosis from endocardial murmurs (Table I, p. 67).

Valvular disease is most commonly due in early life to endocarditis (acute or chronic), and in older persons, to chronic degenerative change. The effect is a thickening or puckering of the valves and ring, which results in one or both of two conditions: (*a*) *Stenosis—i.e.*, a narrowing (στενοω, to contract) of the orifice, which prevents the blood flowing freely through it; or (*b*) *Regurgitation*, in which the valves are incompetent and allow a reflux of the blood to take place from imperfect meeting and closure of the cusps. The remote effect of these two conditions is practically the same—viz., a retardation or obstruction to the circulation of blood through that orifice.

It simplifies diagnosis very much that cardio-valvular disease arising *after* birth is practically *confined to the left side of the heart—i.e.*, to the mitral and aortic orifices. Thus it happens that there are four principal valvular lesions—MITRAL REGURGITATION, MITRAL STENOSIS, AORTIC REGURGITATION, and AORTIC STENOSIS.

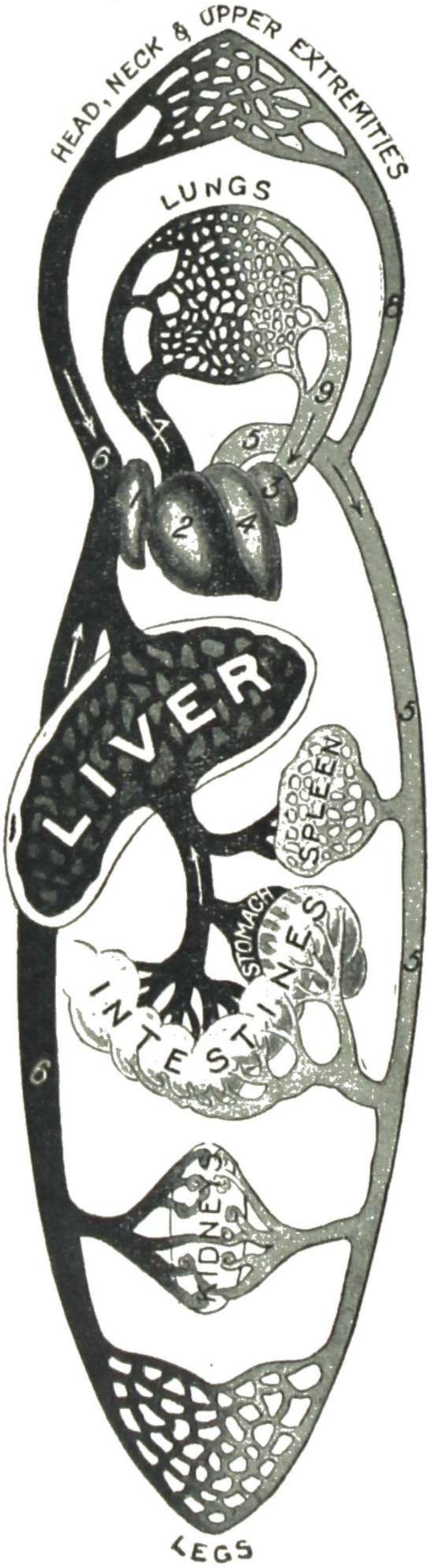
TABLE III.—DIFFERENTIATION OF CARDIAC VALVULAR DISEASES.

		Auscultation.	Pulse.	Other Symptoms special to the Disease.	
C.V.D.	**Mitral** (apical murmurs). *Regurgitation.*	*Systolic* murmur conducted into axilla.	Usually regular.		
	Mitral (apical murmurs). *Stenosis.*	*Presystolic* murmur localised to apex beat area.	Small, moderately firm; very irregular with onset of auricular fibrillation.	Dropsy, enlarged liver and ascites, etc., Hæmoptysis; emboli,	with signs of congestion of organs.
	Aortic (basal murmurs). *Regurgitation.*	*Diastolic* murmur conducted down sternum.	"Water-hammer," rapid and compressible.	Throbbing of arteries of neck,	with symptoms of cerebral anæmia and anginal attacks.
	Aortic (basal murmurs). *Stenosis.*[1]	*Systolic* murmur conducted into vessels of the neck.	Slow, regular, small and hard.	No special symptoms.	

The student should study Fig. 10, p. 50, so as thoroughly to comprehend the various events which occur during one complete cardiac cycle. He should also bear in mind that the left side of the heart is behind the right, and that the left ventricle comes nearest to the surface only at the apex, immediately behind or just below the fifth rib (Figs. 11 and 12, pp. 51 and 52). He should also remember that a cardiac murmur is not produced *in* a diseased orifice, but by the eddies in the blood-stream beyond, and is conducted in the direction of the stream of blood which is causing the murmur. For these reasons a murmur is not usually heard

[1] Real aortic stenosis is somewhat rare, but atheromatous roughening is common.

loudest directly over the orifice diseased. The student should also consult the diagram of the circulation (Fig. 28).



Diagnosis of Cardiac Murmurs.—The first thing to determine is whether a given murmur is related to the first or second sound of the heart—*i.e.*, whether its time is systolic or diastolic—and this will form a convenient basis of classification of cardiac murmurs.

A. **Systolic Murmurs** [1]—*i.e.*, bruits added to or replacing the first sound—may be produced by the following causes, which are mentioned more or less in order of frequency: mitral regurgitation, hæmic and functional conditions (see § 42, and Anæmia, § 535), aortic stenosis, dilatation of the aortic ring, congenital lesions, and tricuspid regurgitation: pulmonary stenosis, patent interventricular septum and patent ductus arteriosus.

I. In **Mitral Regurgitation** the systolic murmur is characterised by (i.) being loudest at the apex; (ii.) being conducted to the axilla, and often audible behind, at the angle of the scapula. When regurgitation is marked, the apex is displaced downwards and outwards owing to the hypertrophy and dilatation of the left ventricle. There is accentuation of the second sound in the pulmonary area, due to congestion in the pulmonary circulation. The pulse is soft, there is a characteristic florid physiognomy, failure occurs late.

The ultimate mechanical effect of Mitral Regurgitation upon the heart and lungs is as follows: (1) owing to the reflux of blood from the ventricle during ventricular systole the auricle becomes dilated.

[1] The term systole is used by clinicians to designate ventricular, not auricular, contraction.

FIG. 28.—Scheme of the Circulation of the Blood.—The superior and inferior venæ cavæ (6) bring the blood back from the organs and tissues into the right auricle (1). Thence it passes into the right ventricle (2), through the pulmonary artery (7) into the lungs. Returning from the lungs by the pulmonary veins (9), it passes through the left auricle (3) and left ventricle (4), and is distributed by means of the aorta (5, 5) and the carotids (8) to the organs and tissues of the body. Notice that the blood from the stomach and intestines passes through the liver before joining the general circulation. (From Huxley's "Physiology," modified.)

(2) In order to drive on the increased volume of its contents the auricle hypertrophies. (3) The effect of a hypertrophied auricle driving an increased volume of blood into a flaccid ventricle at each auricular systole is to produce dilatation of the ventricle. (4) When the power of the auricle begins to fail it is unable to empty itself properly, and there is difficulty in the free passage of blood from the pulmonary veins. Thus pulmonary blood stasis tends to occur. (5) To overcome this stasis it is necessary for the right ventricle to hypertrophy. In cases of failure, right-sided dilatation supervenes, often with the onset of tricuspid incompetence. (6) So that in cases of Mitral Regurgitation there may occur: (*a*) dilatation and hypertrophy of left auricle and ventricle; (*b*) pulmonary congestion; and (*c*) dilatation and hypertrophy of the right auricle and ventricle. These changes are less marked and develop more slowly in

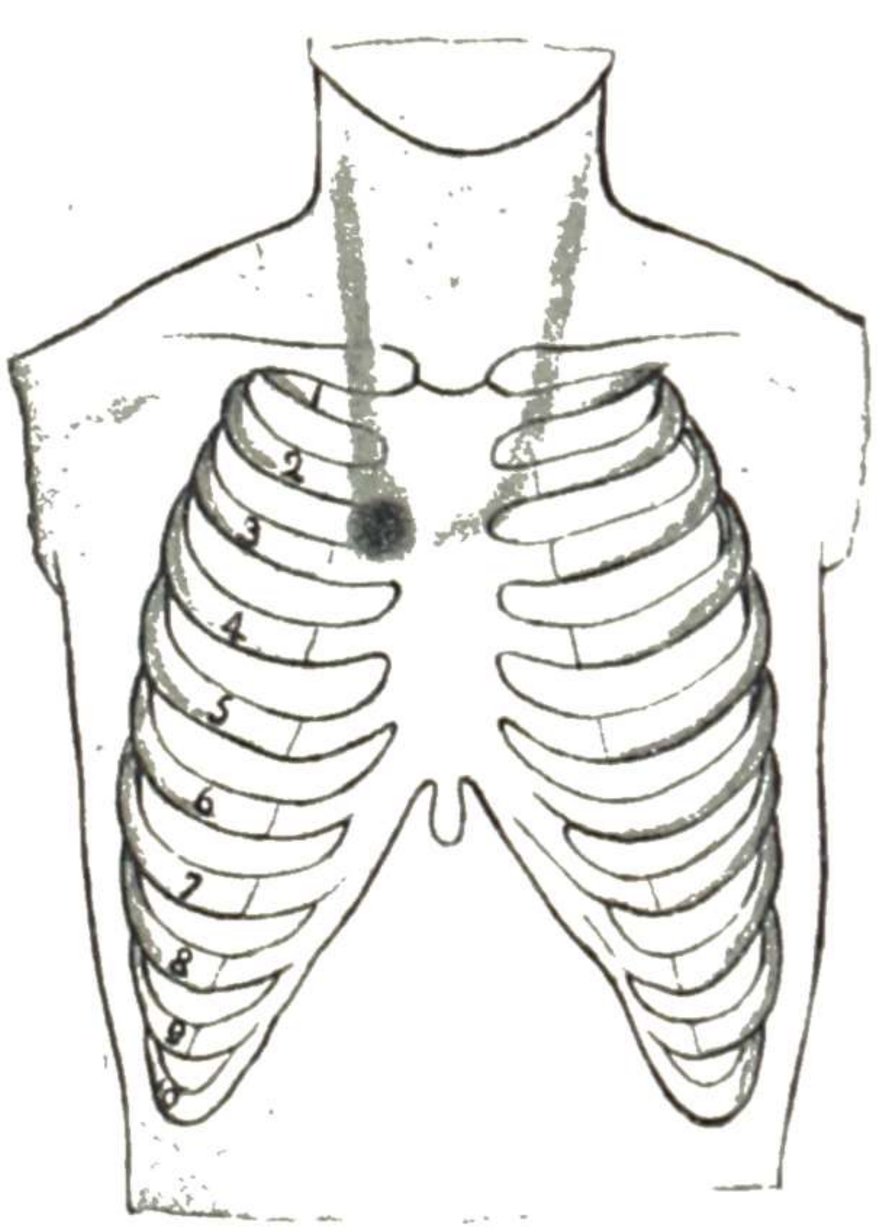


FIG. 29.—The systolic murmur of **aortic stenosis.** Depth of shading indicates intensity of murmur.

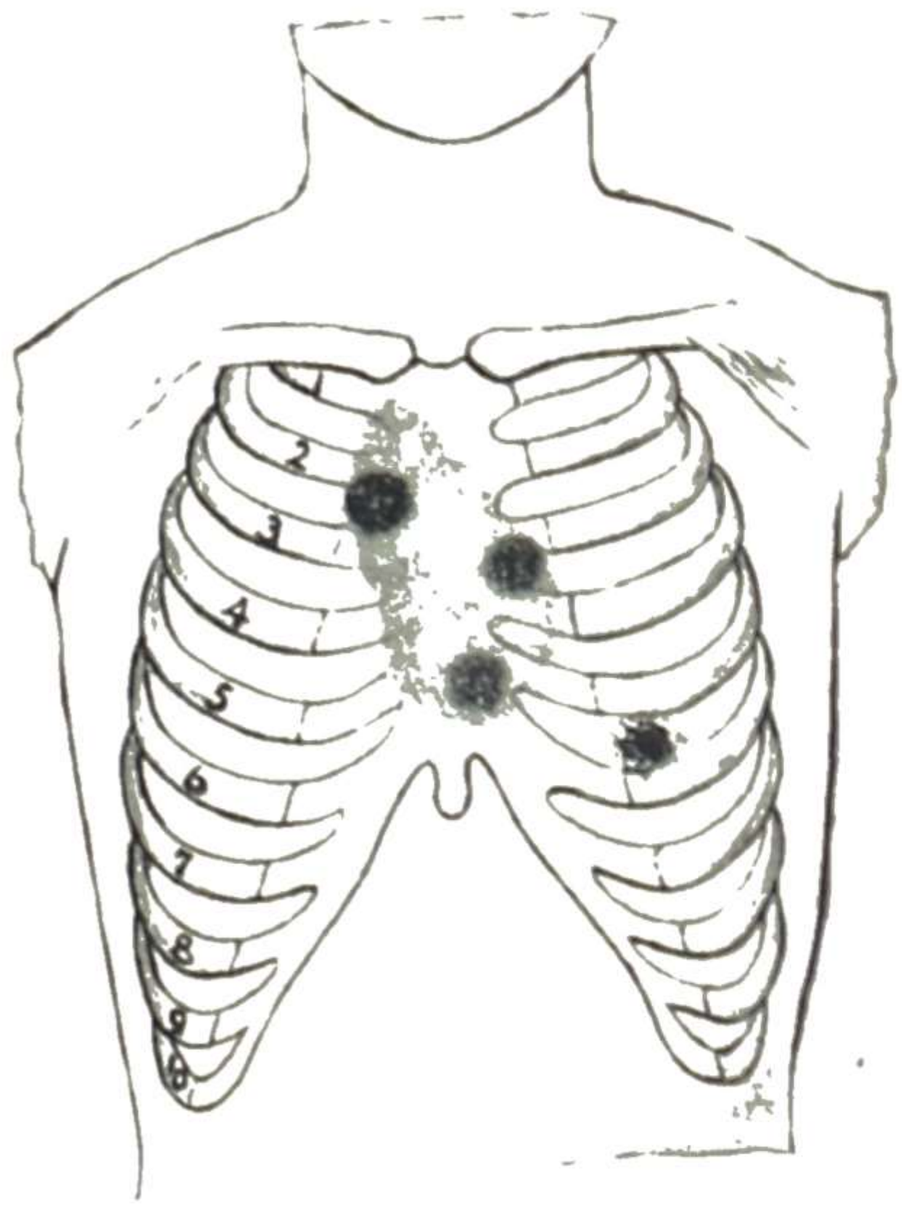


FIG. 30.—The diastolic murmur of **aortic regurgitation.** Depth of shading indicates intensity of murmur.

mitral regurgitation than they do in mitral stenosis, for in the latter condition the left auricle is less able to empty, and is kept in a state of more persistent tension. In mitral regurgitation the outlook is relatively good.

I*a*. A MURMUR OF DILATATION (**Atonicity M.**), systolic in time, having all the above characters, and, like it, due to mitral regurgitation, may occur without definite disease of the valve, when the *left ventricle becomes dilated*, and the muscular ring around the valve *fails to complete* the closure of the mitral valve. This condition is especially apt to occur (i.) in the stage when dilatation of the left ventricle supervenes on hypertrophy, (ii.) in anæmia, and (iii.) in acute myocardial disease, *e.g.*, diphtheria and acute rheumatism.

II. **Aortic Stenosis** is another lesion producing a systolic bruit. The same bruit is produced by endocarditis or atheroma of the aortic orifice.

True narrowing of the aortic ring should not be diagnosed unless five

signs are present: (i.) a systolic bruit in the aortic area and conducted into the carotids. It is harsh, sometimes musical, and may be audible at the apex. (ii.) A systolic thrill in the aortic area, second R. interspace, often best felt when the patient sits forward; (iii.) hypertrophy of the left ventricle; (iv.) a slow-rising, well-sustained, small pulse, often anacrotic in character; and (v.) a weak second sound.

General Symptoms are almost wanting in aortic stenosis—other than anginal pain, due to coronary atheroma, pallor or sallowness of the face, and faintness or giddiness.

The detection of aortic stenosis is sometimes as difficult as that of mitral stenosis and the characteristic murmur may be absent. It may then be suspected when the patient, generally an elderly man, presents persistent dyspnœa, bradycardia, nervousness, and occasionally anginal attacks, which are not otherwise accounted for. In true stenosis the second sound is short and not very loud; whereas in cases of high arterial pressure with a systolic murmur the second sound is loud.

III. In DILATATION and in ANEURYSM OF THE COMMENCEMENT OF THE AORTA a systolic murmur is the most common one heard. The condition is a "relative stenosis," *i.e.*, though the aortic ring is normal in diameter it is small as compared with the diameter of the enlarged aorta. A peculiar ringing character of the aortic second sound is the most constant cardiac physical sign (§ 80).

§ 59. IV. **Congenital Heart Disease** is comparatively rare.

There are two groups—the cases without cyanosis and those with cyanosis. Those *without cyanosis* include bicuspid aortic valve defect, imperfect ventricular septum, and patent ductus arteriosus. Those *with cyanosis* that survive generally have multiple defects which usually include pulmonary stenosis, or other abnormality in the size or position of the pulmonary artery. Patency of the inter-auricular septum (atrial septum defect) does not usually cause cyanosis, but does so when the left ventricle fails later in life.

(1) Bicuspid aortic valve is not possible to diagnose during life, but should be suspected when aortic infective endocarditis develops in a patient whose heart previously was free from murmurs.

(2) Imperfect ventricular septum is characterised by a loud systolic murmur close to the sternum in the third and fourth left intercostal spaces. The murmur is usually accompanied by a thrill. There is usually no marked limitation of the cardiac function.

(3) Patent ductus arteriosus is characterised by slight ventricular hypertrophy, a loud continuous murmur throughout systole and diastole, loudest in systole, and present in the second left interspace near the sternal border. A thrill usually coexists. The diastolic pressure is often lowered. The X-ray shows a typical enlarged pulmonary conus. If infective endocarditis supervenes, cure is possible by ligature of the ductus (§ 50).

(4) Congenital pulmonary stenosis is characterised by dyspnœa, cyanosis, clubbed fingers, and a loud systolic murmur and thrill over the pulmonary base. Polycythæmia is usually present. It is usually found in combination with three other lesions, when the condition is known as Fallot's tetralogy—pulmonary stenosis, interventricular defect, dextroposition of the aorta, and hypertrophy of the right ventricle. Surgical anastomosis of the subclavian to the pulmonary artery (Blaylock's operation) greatly benefits some cases.

(5) In atrial septum defect the whole heart is enlarged, especially the pulmonary conus, and the pulmonary arteries are enormous and are readily visible in the X-ray picture. A systolic murmur and sometimes a thrill are present over the pulmonary area. Cyanosis is absent at rest, but may appear during exertion: it may become permanent when failure sets in.

Prognosis.—A congenital lesion may remain latent for years, though few cases survive to middle age. The acyanotic cases often die of a superadded septic endocarditis; the cyanotic group develop pneumonia or tuberculosis. The prognosis is best if there is no sign of pulmonary stenosis, no clubbing of the fingers and only polycythæmia. In childhood fatal bronchitis and broncho-pneumonia are common. The prognosis is serious in proportion to the degree of dyspnœa and cyanosis, pointing to deficient aëration of the blood, and in proportion to the other symptoms of "cardiac failure" (§ 61).

The *Treatment* is the same as that of Cardiac Dilatation (§§ 55, 62).

V. Tricuspid Regurgitation takes place when that orifice is diseased or dilated. Some maintain that if the valve be healthy, though dilated, no bruit can be heard, but it is certain that in cases of confirmed bronchitis a murmur is often present which comes and goes under treatment, and which is not found to be attended with any marked changes in the tricuspid valve after death. The murmur is characterised by (i.) being heard best at the tricuspid area—*i.e.*, on the left side of the lower part of the sternum; (ii.) it may be heard as far out as the right nipple; (iii.) the pulse is of low tension, often irregular; (iv.) owing to the accompanying hypertrophy or dilatation of the right auricle, the area of cardiac dulness extends to the right of the sternum.

General Symptoms, as above indicated (p. 80), result from tricuspid regurgitation. By far the commonest cause is Chronic Bronchitis, which thus presents a clinical picture readily recognised.

VI. Cardio-pulmonary or Cardio-respiratory Murmurs are fairly common, and are probably produced by the expulsion of air from the adjacent lung tissue by the movements of the heart. They do not indicate any cardiac lesion, and the lung is usually healthy. They are heard in various parts of the antero-lateral region of the chest. They have a blowing, whiffing, or "sipping" character, are usually systolic in time, and in rare cases double, though the systolic element is always loudest. Often they are not loudest at the apex, and come rather between the two sounds than with the first sound. A common variety is audible at the apex, getting louder as it is conducted into the axilla, and is only heard during inspiration. Sometimes they disappear when the patient alters his position or stands up. When he stops breathing, they may be weakened, abolished, or unaltered. These murmurs have no pathological significance.

VII. The Exocardial Murmur is possibly due to a localised thickening of the visceral pericardium. Usually it is unattended by symptoms, but it may be of importance clinically, for it is apt to be mistaken for valvular disease. The exocardial murmur (based on twenty-three observations, verified by autopsy) is generally a prolonged rough bruit, systolic in time, though occasionally double; it is *strictly localised* to a circle of 1 or 1½ inches radius, whose centre is situated in the third left interspace, close to the sternum, which is also its position of maximum intensity. Another important feature is that *at one time it is very rough and loud*, and a day or so later it may have completely disappeared. These features, and the absence of the concomitant symptoms of cardiac valvular disease, or of anæmia, enable us to differentiate the exocardial murmur from other conditions. It was found more often in hypertrophied hearts than in those of normal size. The condition is more frequently met with in adult or advanced life. A history of pericarditis was obtainable in only one of the twenty-three cases.

VIII. A rare cause is *coarctation of the aorta*; this produces systolic murmurs in the enlarged superficial arteries; a systolic murmur produced over the left internal mammary artery may be confused with a cardiac murmur. Similar murmurs may be heard over enlarged arteries on both sides of the chest and over the back in these cases.

§ **60.** B. **Murmurs** heard in the **diastolic interval** may occupy either (*a*) the first half of that interval, replacing, accompanying, or following the second heart sound (*Diastolic* murmurs); or (*b*) they may occupy the

second half of the interval, preceding and leading up to the first heart sound (*Presystolic* murmurs; see Fig. 31). The latter can be accurately defined and described as auriculo-systolic.

TABLE IV.

Early Diastolic Murmurs	*Late Diastolic Murmurs (Auriculo-systolic)*
Aortic regurgitation	Mitral Stenosis
Mitral Stenosis	Austin Flint Murmur.
Dilatation of the Aortic Ring (Aneurysm)	
Pulmonary regurgitation (rare)	Tricuspid Stenosis (very rare)

I. In **Aortic Regurgitation** the murmur is *diastolic* (Ventricular Diastolic).[1] (i.) The diastolic murmur at the aortic valve (Fig. 30) must be listened for: (*a*) over the lower part and to the left of the sternum. It may be audible as far as the apex and indeed over the whole heart. This murmur may or may not be accompanied by a systolic murmur and is found typically in those cases of aortic regurgitation where the valve is the site of endocarditis or of aortic valve dilatation. (*b*) Over the junction of the second right costal cartilage with the sternum. Here the murmur may be loud, and just beneath the stethoscope. (*c*) Over the third left costal junction. Here the murmur is soft, blowing but distant, never harsh in character. This murmur should be carefully listened for in any case of mitral stenosis where the left ventricle is large. The diastolic aortic murmur of rheumatic valvulitis is best heard along the left sternal margin, that of the syphilitic lesion at the aortic base. (ii.) Owing to the amount of dilatation and hypertrophy of the left ventricle, the apex is displaced downwards and outwards more than in any other form of valvular disease. The increase in the size of the left ventricle is proportional to the amount of blood regurgitating. (iii.) The carotids visibly pulsate. Capillary pulsation is generally present, and is detected by drawing a line across the forehead, or by lightly pressing on the fingernail or on the lips with a glass slide; the alternate blush and pallor due to the pulsation in the capillaries is thus well brought out. The retinal arteries may also show visible pulsation. (iv.) In aortic regurgitation the pulse and the blood-pressure changes are of great diagnostic importance. The pulse wave is very forcible but is ill sustained, and this gives a marked pulsation in the carotids and the vessels of the limbs: it may be sufficiently marked to cause the head to nod to and fro with each heart-beat. At the wrist it is best felt by placing the flat of the hand and fingers just above the patient's wrist: pulsation is then felt in both radial and ulnar arteries and is increased by raising the forearm above the patient's head. Owing to the leakage back through the aortic valves during diastole, the

[1] *Diastolic* murmurs are sometimes spoken of as V.D. murmurs, being produced during the ventricular diastole. Similarly, *presystolic* murmurs are spoken of as A.S. murmurs, being produced during the auricular systole.

volume of blood suddenly expelled into the aorta at the commencement of systole is markedly increased, with a forcible systolic wave in the whole arterial system, and a high systolic pressure : this is ill sustained, as during diastole blood leaves the arteries not only distally, but also by leaking back through the aortic valves, with a correspondingly low diastolic pressure. The pulse therefore becomes " collapsing " or " water-hammer " (" Corrigan pulse ") : the pulse pressure is raised much above normal, and the diastolic pressure is lowered in accurate proportion to the diastolic leak. The systolic pressure in the leg is higher than in the arm, due to vasomotor hypertonus ; the difference between arm and leg pressure is usually taken as a measure of vasomotor reserve. A falling systolic pressure in aortic regurgitation usually means a failing myocardium. There is (v.) a pistol-shot sound over the arteries, and (vi.) a diastolic murmur (Durozier's murmur) over the large arteries.

The *appearance* of a patient with aortic regurgitation is often characteristic. (1) The rheumatic type occurs in children and young adults. There is marked pulsation in the neck, while the whole chest may be seen to pulsate with the heart-beat. The brachial arteries stand out prominently and seem to be definitely hypertrophied. (2) The syphilitic aortic case is usually middle-aged, and often presents signs of premature old age ; the apex beat is slapping, less heaving in character, and more diffuse ; dyspnœa is frequently present, and often signs of arterial degeneration. (3) In the arterio-sclerotic type the lesion is due to the associated atheroma.

As regards the *general symptoms*, pallor is generally stated to be characteristic but in fact this is not true (Lewis). Faintness and giddiness may occur, usually brought on by change of position ; frontal headache and consciousness of the heart's action may be complained of, especially on first lying down at night. Pain may be present in the chest on exertion, but until failure sets in, beyond the consciousness of a forcibly acting heart, the patient is usually fit and able to do a large amount of physical work.

II. **Mitral Stenosis** is characterised by narrowing of the mitral orifice with obstruction to the free passage of blood from the left auricle to the left ventricle. It is always caused by a rheumatic infection.

The *appearance* of the mitral stenotic patient is often characteristic. It is most frequently met in women. The face is more or less pinched, there is a marked malar flush, whilst the tip of the nose, ears and the extremities are cold and blue. Respirations are frequently rapid, and the jugulars are often prominent. The patient has a typically thin face, pale or yellowish, and the jugular veins may be engorged. The " mitral face " is typically a red face, but not infrequently Mitral Stenosis appears with a pale face. The pale Mitral Stenotic should always be regarded with suspicion as, generally speaking, one of the following conditions is present : (*a*) recrudescence of the rheumatic infection ; (*b*) supervening malignant endocarditis ; (*c*) an associated aortic leak, or (*d*) some independent condition such as associated renal disease or anæmia.

Physical Examination.—(1) The *pulse* in Mitral Stenosis may be characteristic, and gives a guide to the condition of the systemic circulation and left side of the heart. The *volume* is small (estimated by "lift" plus duration of wave), due to the diminished output. The *force* is small (estimated by impact against the finger), due to small output from the left ventricle. The *tension* is low (estimated by obliteration force), owing to diminished output and diminished force. The *rhythm* may be regular or irregular; in the latter case the irregularity is due either to ectopic beats or to auricular fibrillation. The *rate* is often rapid (round about 90). (2) The blood pressure is usually low, owing to diminished output and force of the left ventricle. It is sometimes reduced in volume after auricular fibrillation has set in. In older patients the blood pressure may be raised in proportion to the amount of associated arterio-sclerosis. Should mitral stenosis become complicated by aortic regurgitation, the blood pressure tends to rise and the pulse volume and force increase. (3) *Cardiac Signs.—Inspection:* Epigastric pulsation is often visible owing to the dilatation and hypertrophy of the right ventricle; the apex beat itself can often be seen inside the nipple line somewhat diffuse in character. *Palpation:* The apex beat is slapping in character and well inside the nipple line. Typically, a presystolic thrill, and in advanced cases a diastolic thrill, is felt at the apex (§ 39). The presystolic thrill may be intermittent and only brought out by exercise, deep breathing, rest or lying on the left side. The pulmonary valve closes so forcibly that it can usually be felt. *Percussion:* The cardiac dulness is increased slightly, if at all. The right cardiac dulness measured from the mid-sternal line is increased. On the left, the cardiac dulness does not extend out to the nipple line unless there is some other associated condition such as mitral or aortic regurgitation, pericarditis, etc. *Auscultation:* In the early stages a faint mid-diastolic murmur is audible, which after exercise, or in the left lateral position, may develop into a typical crescendo presystolic murmur.[1] Later, the typical presystolic murmur appears at rest: then the first sound at the apex is sharp, reduplicated or markedly accentuated. This presystolic murmur, typical of mitral stenosis, is heard only over a limited area, and is not conducted outwards. A long rumbling diastolic murmur is also audible in most cases, especially if the patient lies on the left side (Fig. 31). The second sound at the apex is inaudible in a well-established mitral stenosis, while at the base the pulmonary second sound is markedly accentuated and reduplicated.

General Symptoms.—The commonest symptoms associated with mitral stenosis are: (1) Dyspnœa, at first only on exertion after meals, later after ordinary exertion (*e.g.*, stairs). It then becomes continuous

[1] It may be difficult to differentiate early mitral stenosis from the overacting heart common in excited or neurotic young people. At this stage the X-ray or electrocardiogram may give no help, and the best guide is the character of the first sound. In the excitable heart, although loud and roughened, the first sound is low-pitched; in mitral stenosis the pitch is higher and approximates to that of the second heart sound.

and progressive, so that the patient is unable to lie down at nights (orthopnœa). (2) Palpitation is at first intermittent, occurring after exertion, and is simply of the nature of a physiological tachycardia. Later on it becomes more or less continuous, occurring independently of effort. (3) Cough is a common symptom, induced by exercise or change of position. It may be associated with marked cyanosis or the spitting of blood. The

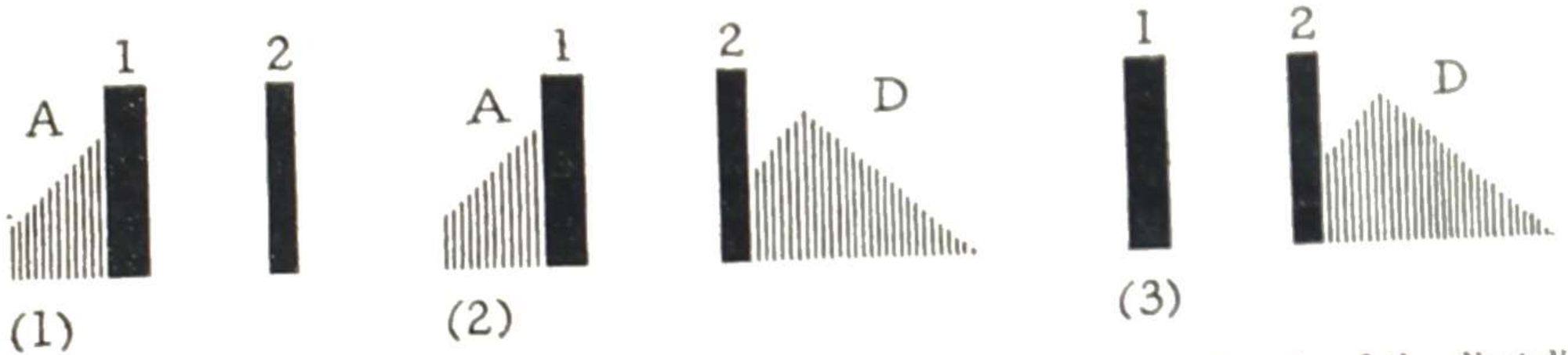


FIG. 31.—In mitral stenosis there are two murmurs, which occupy different parts of the diastolic interval—the presystolic or auriculo-systolic (A), and the diastolic (D) murmurs. The presystolic is present when the auricle is contracting; the diastolic is due to passive blood flow. The presystolic disappears when the auricles fibrillate. The figure shows:—(1) the presystolic murmur; (2) both murmurs; (3) the early diastolic alone as in fibrillation, the presystolic having disappeared. The reduplication of the 2nd sound is omitted for clearness.

underlying cause is congestion of the lungs. A severe type of cough is sometimes met with in embolism or infarction. Symptoms developing later are: (4) auricular fibrillation, flutter, or premature beats; (5) hæmoptysis, embolism, right-sided failure with liver engorgement and ascites are common (§ 55). Embolism, generally cerebral producing hemiplegia, sometimes renal producing hæmaturia, may occur, from the liberation of fibrinous clots formed in the appendix of the dilated left auricle.

In order to understand the progressive variations of the physical signs and symptoms met with in Mitral Stenosis, it is necessary to say a few words about the anatomical changes which develop in this lesion. Broadly speaking, narrowing of the mitral orifice from any cause results in: (*a*) a tendency to dam up the blood in the left auricle, pulmonary and venous circulation in this order, so that the pressure tends to rise; (*b*) there is a reduction in the left ventricular filling, with a reduced size of the left ventricle, a low tension pulse and a slapping first sound. The output and force are thus reduced and the systemic blood pressure falls. The damming of the blood in the pulmonary circulation is responsible for the physical signs met with in the chest, *e.g.*, accentuated pulmonary second sound, basal crepitations, etc., and subsequently it produces engorgement of the systemic veins, enlargement of the liver, peripheral œdema and other manifestations of congestive failure.

The progressive changes which occur in the myocardium in Mitral Stenosis and its actual condition at any phase of the disease can be followed by observing the changes in the electrocardiogram. The first result of Mitral Stenosis is increased auricular work and contraction, and this is reflected in the electrocardiogram by increase in the amplitude of the P or auricular waves, which still however retain their normal form. As the condition progresses, the two auricles become slightly divorced in their action, so that the P waves become flattened, widened and partly divided. Sometimes by the time this has occurred rheumatic fibrosis has invoved the Bundle of His, with impairment of its conductivity, as is shown by an increase in the P–R interval (Fig. 16). Usually also, by now right ventricular "compensation" has occurred, so that the electrocardiogram shows a distinct right axis deviation. The narrowed mitral orifice results in imperfect filling and therefore incomplete stretching of the left ventricle, which consequently contracts poorly (hence the slapping character of the apex beat). The condition continues to progress, the

muscle fails, the rhythm becomes irregular—the first irregularity noticed usually being due to premature beats of auricular origin (Fig. 15). This indicates myocardial hyper-irritability of the auricle, and is often succeeded by a complete irregularity due to auricular fibrillation (Fig. 14) which may rarely be preceded by a state of flutter (Fig. 35). The fibrillation at first is of the coarse variety, but with time it becomes finer and ultimately flat as the muscle gradually degenerates and its co-ordination and contractibility become more impaired. Definite heart block is not uncommon.

III. In AORTIC ANEURYSM a *diastolic* murmur is sometimes heard if the aortic ring shares in the dilatation of the aorta.

IV. An AUSTIN FLINT murmur is a presystolic apical murmur occasionally heard with aortic regurgitation. It is diagnosed from that due to mitral stenosis by its not being followed by an accentuated first sound, by the position of the cardiac impulse, and by the absence of the other signs of mitral stenosis.

V. REGURGITATION through the PULMONARY ARTERY is met with very rarely; it may be produced by congenital malformation of the heart.

VI. TRICUSPID STENOSIS is a very rare condition, but it is occasionally met with in young women, and is recognised by (i.) a presystolic murmur, heard loudest over the fifth right costal cartilage, close to the sternum. (ii.) Dropsy is an early effect, but in other respects the consequences are the same as those of regurgitation through this orifice. Dyspnœa is less than the degree of venous and hepatic distension would seem to warrant. Orthopnœa is absent.

FALLACIES IN THE DIAGNOSIS OF DIASTOLIC MURMURS.—1. A diastolic murmur due to *aortic regurgitation* may be heard at the *apex*. It must not be mistaken for that of mitral stenosis. In addition to the fact that the aortic murmur is heard louder at the base than at the apex, it has a rushing character, whereas a mitral diastolic murmur is low-pitched and rumbling, and the character of the pulse and the blood pressure is different.

2. *Mitral stenosis* is sometimes hard to detect in the stage of auricular fibrillation, when the characteristic murmur may be *altogether absent*. It may, then, be strongly suspected when there is—(i.) a loud, clear, sharp first sound at the apex, with marked accentuation of the pulmonary second sound; or (ii.) hypertrophy of the right ventricle, chronic pulmonary catarrh, and hæmoptysis, especially if the second sound is reduplicated.

C. **Double Murmurs** may be produced by a combination of any of the above systolic and diastolic murmurs.

(*a*) Double murmurs most audible at the **base** (other than hæmic):

I. COMBINED AORTIC OBSTRUCTION AND REGURGITATION is the most common condition, and causes a loud double to and fro murmur, heard best in the second right interspace.

II. ANEURYSM OF THE AORTA may be attended by a double murmur having the same characters as in disease of the aortic valves. This is heard loudest in the second right interspace, but it may also be heard at the back, to the left of the fourth dorsal vertebra.

III. A double murmur occasionally occurs in the DILATED AORTA of the aged, but with less marked features.

IV. A double murmur, loudest in the pulmonary area, usually indicates CONGENITAL HEART DISEASE, especially with patent ductus arteriosus.

(*b*) A double murmur most audible at the **apex** may be heard when both MITRAL STENOSIS and REGURGITATION are present. It consists of a typical presystolic murmur running up to the first sound, immediately

followed by a systolic mitral murmur which is conducted outwards to the axilla. A third murmur, a diastolic mitral stenotic murmur, often coexists.

FALLACIES IN THE DIAGNOSIS OF DOUBLE MURMURS.—1. When a double murmur can be heard both *at the base and apex*, do not take for granted that mitral regurgitation exists, as well as aortic disease. Remember that a systolic mitral and a systolic aortic may be alike in character, and that aortic murmurs can often be heard at the apex, as well as the base. To arrive at a conclusion is often difficult, but one must rely on the position in which the murmur is loudest, and on the other features which distinguish mitral and aortic lesions.

2. When a *double aortic* murmur is present, the lesion may be regurgitation, or stenosis, or both together. A diagnosis is made by examining the pulse (§ 83), the time of the thrill, if one is present, and the position of the apex beat. In regurgitation the apex is displaced farther downwards and outwards than in any other form of valve disease. In aortic stenosis the left ventricular wall is hypertrophied, with but little enlargement of the cavity, but as emphysema is so often associated with it, the apex may be hard to find.

3. Murmurs of *pericardial friction* may easily be mistaken for a double aortic murmur; but whereas endocardial murmurs begin synchronously with the first or second sounds, the rub of pericardial friction often lags slightly behind them.

4. *Hæmic, cardio-pulmonary* and exocardial murmurs are occasionally double.

§ 61. SYMPTOMS RESULTING FROM CARDIAC VALVULAR DISEASE.—The first effect of valvular disease is *hypertrophy* of the heart, as already mentioned, and so long as there is adequate compensatory hypertrophy there may be no concomitant symptoms at all.

But, sooner or later, in most cases hypertrophy gives way to *dilatation*, and then a series of characteristic symptoms ensue. Those special to each form of valvular lesion have been referred to in the preceding section. Certain *general symptoms are common to all forms of chronic valvular disease* when this has produced myocardial failure.

1. *Breathlessness* on walking uphill, or even on very slight exertion, is a constant feature. No serious enfeeblement of the heart wall or disturbance of its function can exist without this symptom; and it cannot be too much insisted on that breathlessness is not only a symptom, but, in general terms, is the most accurate measure of the extent of the cardiac failure.

2. *Dropsy* occurs early in mitral, late in aortic, disease.

3. *Palpitation* is of less diagnostic import, for it generally occurs without organic heart change.

4. *Pain* is by no means always present in cardiac dilatation, but few cases run their entire course without some præcordial discomfort. Pain is a fairly common feature of aortic disease, and sometimes is anginal in type; it is then due to interference with the coronary blood flow.

5. *Insomnia*, in advanced cases, is frequently a troublesome symptom, and in aortic regurgitation may be the first symptom of failure. Sometimes the patient, when dropping off to sleep, suddenly starts with the terror of suffocation, and gasps for breath. *Headache* and *delirium* are also met in advanced cardiac disease. The former is often due to variations

in the blood pressure. Delirium in heart disease is usually due to cerebral anoxæmia, resulting from slowing of the circulation rate (see § 35).

6. *Embolism* may occur, as described under Acute Endocarditis (§ 49), but without evidence of general infection. It is most frequent in mitral stenosis and in auricular fibrillation, and can occur in aortic disease. Emboli commonly occur in one of the middle cerebral arteries.

The chief ETIOLOGY OF CARDIAC VALVULAR DISEASE in *youth* is rheumatic endocarditis, which has a special tendency to attack the mitral valve. In *advancing years*, the commonest cause is an atheromatous degeneration. Rarer causes are: infective endocarditis (§ 50) and syphilis. The latter attacks only the aortic valve.

1. *Acute Endocarditis* of rheumatic origin is by far the most frequent cause, and a large majority of "heart cases" date their symptoms from an attack of that disease in youth or early adult life.

2. *Chronic Endocarditis* may come on insidiously. Syphilis causes aortic regurgitation between the ages of 40 and 60, often in association with arteriosclerosis. Chronic rheumatic endocarditis more often supervenes upon acute endocarditis—attacks of which may have been overlooked. Rheumatic heart infection may begin during rheumatic fever, chorea, rheumatic tonsillitis, or scarlet fever, and may be of insidious onset.

3. *Degenerative* changes (*e.g.*, atheroma) are the lesions chiefly met with after middle life. They affect especially the aortic orifice, either by injuring the valves or by causing dilatation of the aorta, which, extending to the situation of the valves, prevents them from meeting during diastole.

4. Any prolonged *high blood pressure*—*e.g.*, that which accompanies arteriosclerosis—may lead to valvular strain, usually aortic. Persistent obstruction in the lungs (*e.g.*, chronic bronchitis), or in the general systemic circulation, may have the same effect as persistent high tension on the right or left side of the heart respectively.

5. *Congenital* conditions are referred to in § 59.

Prognosis. In a case of chronic heart disease the fundamental factor is the heart muscle. Is the heart muscle handicapped? If so, is the handicap removable? If not, is a handicap likely to arise in the future? These questions must all be answered in any one case. In actual practice the prognosis is good in proportion to *the amount of exercise a patient can take without producing breathlessness*. The factors which influence the function of the heart muscle are toxins, metabolic factors, oxygen supply, and certain mechanical considerations, which include occupation or exertion, valvular defects and cardiac irregularities.

Toxins. Diphtheria, influenza, pneumonia, typhoid fever and certain septic foci produce a myocarditis which is completely recovered from if the patient survives the disease. Acute rheumatism not only produces permanent myocardial changes; it is apt to recur repeatedly during a patient's life, thus injuring the heart muscle ever more severely. If, however, the rheumatic process ceases, and if mitral stenosis has not been produced, a complete functional recovery is possible. If toxæmia of any kind is present, the myocardium will fail as a result of increased work, and the severity of the failure will be in proportion to the amount

of toxæmia and the amount of work. A healthy, non-toxæmic heart muscle will never fail, whatever the severity of the exertion undertaken. The factors which cause an athlete to be " rowed out " are not myocardial, but vasomotor and nervous.

Metabolic factors producing myocardial failure are hyper- and hypothyroidism and beri-beri. Complete cure is here possible.

Oxygen supply to the myocardium is deficient either in anæmia or in coronary disease. In the former case the prognosis depends upon the removability of the cause. The prognosis in coronary disease depends upon the following factors: (i.) with advancing years, as Gross has shewn, the collateral coronary anastomoses become freer; (ii.) coronary disease is often very localised, but it may or may not be part of a generalised arteriosclerosis; and (iii.) any individual coronary lesion tends to be progressive. Thus a coronary thrombosis in a man of 50, who has no hypertension, may heal and leave him with no dyspnœa; then a complete recovery is possible and the prognosis is good. On the other hand, a man with cardiac pain on exertion, due to coronary sclerosis and associated with hypertension, will be likely to lose ground progressively. Pulsus alternans, gallop rhythm and Cheyne-Stokes' breathing are bad signs.

Mechanical factors. The *valvular* lesions which seriously hamper cardiac efficiency are mitral stenosis and aortic incompetence. In mitral stenosis an increasing check is placed upon the amount of blood allowed to enter the left ventricle, and since this chamber can only expel what it receives the circulation rate is progressively reduced. Aortic incompetence is a mechanical handicap, for the left ventricle has extra work to do per beat, the diastolic coronary flow to the myocardium is less efficient, and the propulsive effect on the circulation of the aortic recoil is lost; also the factors which cause injury to the aortic cusps are apt to obstruct the mouths of the coronary vessels. The prognosis in aortic stenosis is much better. Cardiac irregularities chiefly cut down the circulation rate by the associated tachycardia, which reduces the diastolic time and thus interferes with ventricular filling.

The prognosis in *valvular lesions* depends upon the myocardial condition, and the size of the valvular defect. A case of arrested aortic valvulitis, from rheumatism or syphilis, may live a healthy life for many years. As a brief generalisation the least dangerous valvular defect is mitral regurgitation; next in order comes aortic stenosis, then mitral stenosis; and most dangerous of all is aortic regurgitation. But in every case the etiological factor must be taken into account. The extent of the lesion can be best gauged from the size of the heart chamber affected by it. The enlargement of the left ventricle is proportional to the extent of the lesion in aortic stenosis or regurgitation and in mitral regurgitation. Hypertrophy of the right ventricle from valvulitis is only found clinically in advanced cases of mitral stenosis. Hypertrophy is a compensatory function; dilatation is of definitely bad omen.

As regards individual examples : in *aortic regurgitation*, a good prognosis may be given in young rheumatic individuals with a good exercise tolerance, a not unduly large heart, a slow resting pulse rate and a normal diastolic blood pressure with a comparatively small pulse pressure. Combined with the above there must be no sleeplessness and an occupation in which there is a healthy amount of muscular exercise, which is essential for an efficient venous return. A relatively bad prognosis should be given in aortic regurgitation when the case is of syphilitic origin, where the heart is considerably enlarged, the apex beat slapping (not heaving in character), where the pulse is rapid, where the systolic pressure is high but tending to fall, and the diastolic pressure low ; when the patient is sleepless, gets vertigo on changing his position or after exercise, when the heart is irregular, whether the irregularity be due to extra systoles (left ventricular fatigue), to alternation (failing contractility), or to auricular fibrillation. Associated conditions, such as pregnancy, of course, add to the gravity of the prognosis.

In *mitral stenosis*, the prognosis is relatively good when the patient is capable of leading a sheltered life ; when the pulse is regular and of good volume ; when there is no marked dyspnœa or cyanosis, no raised pulse rate, and a relatively good exercise tolerance. The prognosis is bad when the pulse is small and irregular ; when there is marked cyanosis and dyspnœa on mild exertion, or orthopnœa ; when the blood pressure is very low, the pulse is rapid, the liver is palpable, and when there are crepitations at the bases of the lungs and œdema of the feet.

The occupation, sex, and temperament of the individual are of importance. In heart cases it is essentially the pace that kills. If the patient is peacefully occupied with work which he can carry out in his own way, in his own time, the prognosis may be relatively good, but if he has to work against time, especially at an occupation that he is not used to, the prognosis becomes relatively bad. The placid individual who takes things as they come and does not worry, usually lives longer than the worrying person who meets trouble half-way. Generally speaking, the prognosis is better in women than in men, but here again it is largely a question of the lives they lead and the amount of rest they are able to take.

The prognosis of *Heart Disease as affected by Pregnancy* demands special notice. In pregnancy the maternal heart is loaded (1) owing to the increased nutritive demands of the fœtus or embryo, and (2) by the mechanical embarrassment caused by the growing uterus. The former of these operates from the commencement of pregnancy ; the latter becomes of increasing importance as pregnancy proceeds. In conditions such as mitral stenosis, where the right heart is in a continual state of strain, special care must be taken. In the case of aortic regurgitation, where the left heart is under a strain, the early months of pregnancy are attended with no particular risk, but towards the end of pregnancy (the 8th and 9th months) the risk of failure is gradually increased. Furthermore, after delivery it is essential to keep the patient still and to support the splanchnic area, as sudden vasomotor collapse is liable to occur. With proper precautions heart cases stand pregnancy well. Decision as to the safety of pregnancy is a matter of experienced judgment and varies with individual cases. Briefly, it may be said that if there are no symptoms

or signs of failure, pregnancy can be considered. If such symptoms have previously existed, pregnancy is possible but is a definite risk. If signs of failure are present, pregnancy must not occur, or must be terminated.

§ 62. The **Treatment** OF CHRONIC HEART DISEASE (including Myocardial Degeneration and Valvular Disease) may be considered under three heads: (*a*) When compensation is fully established; (*b*) when compensation begins to fail; (*c*) when compensation has broken down.

(*a*) When there is efficient compensation, no symptoms are present and no active treatment is needed, but much may be done to prolong the patient's life, and to avoid the supervention of cardiac failure. Subjects of chronic valvular disease should be enjoined to lead quiet, regular, and orderly lives. With regard to exercise, it may be said, in general terms, that the patient himself is the best judge, provided always that he does not exert himself sufficiently to cause palpitation, undue dyspnœa, or præcordial pain. Some sports are more permissible than others; thus cricket, tennis, and golf may often be enjoyed, whilst football, racing, and rowing must generally be forbidden. Climbing, especially to high altitudes, must be disallowed. Alcohol, tobacco, and tea are all myocardial poisons if taken to excess, and should be used only in strict moderation. The skin should be kept active by the daily bath, and the bowels regular by means of purgatives if necessary. Whenever possible, a means of livelihood should be chosen in which the heart is subjected to but little strain. A sedentary occupation with moderate exercise in the intervals, is more suitable than one which entails earning a living literally by the sweat of the brow. Lifting or carrying heavy weights, climbing ladders, wielding heavy hammers, and physical labour in constrained positions, are liable to overtax the powers for compensation of the cardiac muscle. Meals should be regular, and heavy meals should be avoided. The diet should be easily assimilable, and contain only a moderate amount of fluid. A small quantity of stimulant with meals may be called for, but should not be used unnecessarily, because of the reaction afterwards, and of the tendency to excess, which exists in cardiac cases.

(*b*) When compensation is beginning to fail, the condition of the heart should be noted frequently; rest, drugs, and exercises being prescribed in accordance with the variations in the circulation and the capability of response to treatment by the cardiac muscle.

Drugs.—In cardiac failure, especially in auricular fibrillation, when the pulse becomes feeble, rapid, and irregular, *digitalis is par excellence* the remedy. It is especially indicated in failure of the right heart in mitral stenosis or regurgitation, whether primary or secondary to aortic lesions. It is contra-indicated when there is full compensatory hypertrophy, and the pulse is fairly strong, regular, and slow, or if vomiting is present. By its action on the vagal nerve endings in the Bundle of His, it reduces the number of auricular impulses reaching the ventricle. Its action is not quite so efficacious if fever is present. There are two methods of administering it—massive or intensive dosage, or maintenance dosage.

If a patient is very seriously ill with heart failure and auricular fibrillation, digoxin 1 mgm. injected intravenously is often used, and can be repeated in 6 hours. In a patient less ill tincture of digitalis can be given 6-hourly in three doses of ℥ 90, ℥ 60, and ℥ 30. After these emergency measures, a maintenance dose is given, 10, 15 or 20 minims t.d.s., p.c. according to circumstances. The two things to watch are the heart rate and the signs of digitalis overdose. The apex rate, not the pulse rate, is the only safe guide to the correct dose of digitalis in auricular fibrillation, owing to the apex-pulse deficiency which occurs. No nurse should be allowed to record the pulse figures in these cases who cannot count the heart rate by stethoscope; pulse figures alone are valueless. The result to be obtained is a reduction of the heart rate to 70–80 per minute. The signs of overdose to watch for, in order of severity, are: reduced urinary output, coupled beats, nausea, vomiting, headache, and visual disturbance. In less severe cases of failure, it is sufficient to give ℥ 30 t.d.s., p.c. for 2 to 4 days, subsequently giving a maintenance dose of about ℥ 10 t.d.s., p.c.

The most useful preparations of digitalis are, in institutions, the tincture; for outpatients, digitalis pulverata, one grain being equal to 10 minims of the tincture, or digitoxinum (*B.P.C.*), which is similar to Nativelle's digitaline; and for cases where rapid absorption is desired, digoxin B.P. (0·25 mgm.), one tablet being equal to 15 minims of the tincture. The latter drug has removed one of the chief deficiencies in digitalis medication, namely, the slowness of its action. It may be given in doses up to 1 mgm. intravenously. Strophanthin has an action similar to that of digitalis, and can also be used intravenously. It must never be so given to a patient who recently has been taking digitalis, as sudden death has been known to occur. Digitalis can be continued indefinitely, as tolerance is not acquired. In regularly beating hearts it probably has a definite but slight effect by improving contractility. The action of digitalis and many other cardiac remedies is expedited by an occasional dose of calomel. Formulæ 54, 57, 67, and 84 are useful. The absorption and action of digitalis are slow, and in very acute cases of heart failure recourse is often had to rapidly acting stimulants, such as brandy, caffein citrate gr. 2–3, or leptazol B.P. (cardiazol) subcutaneously, but it is questionable whether any direct cardiac effect is produced by them. These preparations commonly used act on the vasoconstrictor and respiratory centres and not on the heart. Digitalin injections have little action unless given intravenously. Strychnine is useless as a cardiac stimulant, but stimulates the respiratory centre and increases tone generally. In *aortic valvular disease* and in the early stages of *mitral stenosis*, digitalis is not so valuable a drug; but in the later stages of these affections, when compensation begins to fail, and especially when auricular fibrillation is present, digitalis gives relief. In aortic cases, where the blood pressure is high, or where angina is present, the vaso-dilators are often of use, such as nitroglycerin in the form of liquor trinitrini ℥ i. t.i.d., erythrol tetranitrate or sodium nitrite. Theophylline ethylene diamine

(cardophylin) intravenously once a day is a useful drug for cases of an arteriosclerotic nature. Sugar in doses of 1 to 5 ounces two or three times daily is useful for a failing myocardium from whatever cause.

The various symptoms may be met by appropriate remedies. For the *pulmonary congestion*, venesection is by far the most efficacious form of treatment. Pulmonary œdema is benefited also by injections of mersalyl. Nitroglycerin is useful for the headache and sleeplessness due to *hypertension*. Phenobarbitone is valuable for anxiety and restlessness, but in cases with cerebral arteriosclerosis it is apt to cause mental changes and even delirium. For the paroxysms of *dyspnœa*, morphia, cardophylin (0·48G. in 20 c.c. of sterile water, intravenously, daily for 7 days), or oxygen inhalations by mask or nasal catheter are useful. *Cough* is relieved by drinks of hot milk, and drugs such as codein, small doses of opium, and chloroform or ether. For *palpitation*, alcohol is a valuable sedative. The quantity should always be moderate. Other causes of palpitation which may be present should be treated (§ 34). For *sleeplessness*, in more acute cases, opium or morphia hypodermically is most useful, and should be given without hesitation. In children or in cases where the insomnia is not obstinate, other drugs may be employed, such as potassium bromide, phenobarbitone, sulphonal, trional, and paraldehyde. Chloral is harmless. The *hæmoptysis* of heart disease is best left alone, as it relieves the congestion. The *gastric* symptoms may be relieved by acting on the congested liver with calomel, ½ to 1 grain every night, with sodium sulphate and sodium bicarbonate (30 grains in 2 ounces of hot water) in the mornings. Digitalis must be stopped if it causes sickness. For the treatment of *pain* and *syncopal attacks*, *vide* §§ 33 and 35. Formula 56 is useful.

Massage and Systematised Exercises.—At one time rest was regarded as imperative for all forms of cardiac disease. But the advance of physiological knowledge has shown what an important part the skeletal muscles play in the circulation of the blood, by squeezing the fluids out of the soft-walled veins and lymphatics, while they cannot compress the lumen of the firm-walled arteries. There are three varieties of this treatment, which are invaluable for different degrees of cardiac failure. *First*, for the worst cases, *gentle massage*, combined perhaps with *passive movements*. These are available where any kind of voluntary movement on the part of the patient is attended with breathlessness. *Secondly*, slow *voluntary movements* of flexion and extension on the part of the patient while standing or sitting. In the Nauheim system these voluntary movements are gently resisted by the operator—"resistance gymnastics." These movements, combined with *baths* (see below), constitute the essence of the system. *Thirdly*, Oertel's method, which consists of three parts: first, reducing the amount of fluid taken to 31 ounces per diem (to include the amount contained in the solid food) and promoting perspiration; secondly, a diet largely consisting of proteins [1]; and thirdly, graduated exercise in

[1] Oertel's dietary is as follows:—*Morning:* 6 ounces of coffee, 3 ounces of bread. *Noon:* 3 to 4 ounces of soup, 7 to 8 ounces of roast meat or poultry, salad or green

the form of walking uphill, each day a little farther. Cases with plethora and obesity are the most suitable. In cases of early failure who are still ambulatory, much good can be done by reducing the weight when this is excessive. A diet poor in fat and low in carbohydrate provides the best method. Breathing exercises, by helping the venous return, are valuable in chronic heart disease.

Saline and effervescent *baths* may be usefully added. They act by relaxing the arterioles of the skin directly, and of other parts reflexly. By these means blood is transferred from the venous to the arterial system, and its flow accelerated (see Nauheim baths.)

(*c*) When compensation has broken down and marked cardiac failure is present, absolute rest is necessary. The patient is usually unable to lie down, but has to be propped up with pillows, and in severe cases sleep can be obtained only when the legs are hanging down. A special "heart" bed is valuable in such cases, as the degree of dorsal support and dependence of the legs can be adjusted to each case. In severe failure of the right heart, as indicated by distended jugular veins, cyanosis, the liver dulness extending well below the costal margin and the cardiac dulness extending far to the right, *venesection* is called for, and brings prompt relief. The rapid removal of from 10 to 20 ounces of blood is usually sufficient; this may be repeated. Three to six leeches may be applied to the right lower ribs in children, in whom venesection is more difficult to perform. In the treatment of cardiac œdema, mersalyl, 1 or 2 c.c. injected intramuscularly or intravenously, is one of the most useful remedies known for removal of fluid and usually renders tapping unnecessary. Ammonium chloride (20 gr. t.d.s., p.c.) should be given for one day before mersalyl is begun, and should be continued during the treatment. The mersalyl injections can be repeated every 4 or 5 days. Signs of mercurial poisoning such as salivation and stomatitis must be watched for. Mersalyl may be dangerous if renal disease is present. Similar compounds can be used rectally; in common use are suppositories of mersalyl. Theophyllin and sodium salicylate (diuretin), 10 gr., and theocin sodium acetate, 3 gr., may be useful. The diet should contain a minimum of salt. Dropsy may be treated by draining the legs by Southey's tubes (§ 29) or multiple superficial incisions, asepsis being maintained by penicillin cream and sterile dressings. Aspiration of a pleural effusion or paracentesis abdominis may be necessary. Pleural effusions are common in congestive heart failure: treatment by removal of the fluid will increase the expanding area of the lung, decrease the anoxæmia and benefit the myocardium. They should be looked for, and removed by aspiration even though comparatively small. Diaphoretics are of little use in cardiac dropsy. Digitalis and caffein should be employed in conjunction with

vegetable, a little fish, 1 ounce of bread or farinaceous pudding, 3 to 6 ounces of fruit; no liquid (excepting in hot weather, 6 ounces of light wine). *Afternoon:* 6 ounces of tea or coffee (1 ounce of bread occasionally). *Evening:* One or two lightly boiled eggs, 1 ounce of bread, salad, fruit, sometimes a small piece of cheese, 6 to 8 ounces of light wine, with 4 to 5 ounces of water.

diuretics, calomel, and hydragogue cathartics, such as pulv. jalapæ co. and cream of tartar. The digitalis, squill and mercury pill is useful at this stage; so also Formula 55.

Complete thyroidectomy has been successfully used in order to reduce the basal metabolic rate of cardiac patients, and thus to lighten the burden of the heart. It has been used for repeated attacks of congestive failure in myocardial degeneration; but it is most useful in carefully chosen cases of anginal pain of organic origin; for this sympathectomy has also been performed (§ 51).

§ 63. GROUP E. We now turn to the consideration of those cardiac disorders the recognition of which depends upon **Alterations in the Rate or Rhythm of the Pulse.** In all cases *it is essential to compare the radial pulse with the heart sounds*, and to observe the pulsation in the veins of the neck.

The Electrocardiograph and, rarely, the Polygraph (§ 44) may be required to make an exact study of a case presenting pulse alterations; but it is often possible to make a correct diagnosis without their aid.

I. With an occasional PAUSE in the radial pulse	Premature Beat (Extrasystole) (§ 64). Early Heart Block (§ 69). Sino-auricular Block (§ 64).
II. With RHYTHMIC alteration of rate DEPENDENT ON RESPIRATION	Sinus Arrhythmia (§ 65).
III. With INCREASED rate	Tachycardia, Physiological (§ 84) or Paroxysmal (§ 66). Auricular Flutter (§ 67). Auricular Fibrillation (§ 68).
IV. With DISORDERLY RHYTHM	Auricular Fibrillation (§ 68) or Multiple Premature Beats (§ 64).
V. With DECREASED rate	Bradycardia (§ 85). Complete Heart Block (§ 69).
VI. COUPLING of the Pulse Beats	Premature Beats (§ 64). Pulsus Alternans (§ 71). Extreme Dicrotism (§ 70).

The various causes of altered rate and rhythm of the pulse, other than cardiac disease, are considered in § 84 *et seq*. Here we consider only the cardiac conditions to which attention may first be called, and in which the diagnosis may be largely made, by alterations in the pulse rate and rhythm.

A PAUSE IN THE PULSE, which is OTHERWISE REGULAR, is due to three conditions: PREMATURE BEATS, HEART BLOCK, and SINO-AURICULAR BLOCK. The first of these is very common, the second somewhat rare, and the third very rare. The pause in the pulse caused by premature beats is due to the fact that the ventricle is prematurely stimulated early in diastole, and the output from it is therefore generally insufficient to

produce a pulse wave. Following the premature beat there is a compensatory pause. During the pause in the pulse, as felt at the wrist, the stethoscope, if placed over the heart, can detect the sound of the premature beat. This differentiates premature beats from heart block and sino-auricular block.

I. *There is an occasional pause in the radial pulse, during which the heart gives a short premature beat which can be heard over the præcordium.* The condition is PREMATURE BEAT.

§ 64. Premature beats (Extrasystoles) are due to hyper-irritability of the myocardium, causing early contraction in some part of the heart.

The normal beat of the heart always starts at the most irritable point, normally the sino-auricular node. Should any other point of the heart become more irritable it initiates the contraction; such a beat is said to be ectopic in origin, and inasmuch as the contraction occurs earlier in the cardiac cycle than the normal beat, it is called *premature*. Ectopic beats may arise either in the auricular, nodal or junctional tissue (*i.e.*, the tissue between auricles and ventricles) or in the right or left ventricles. Ectopic auricular beats give rise to a ventricular contraction of normal

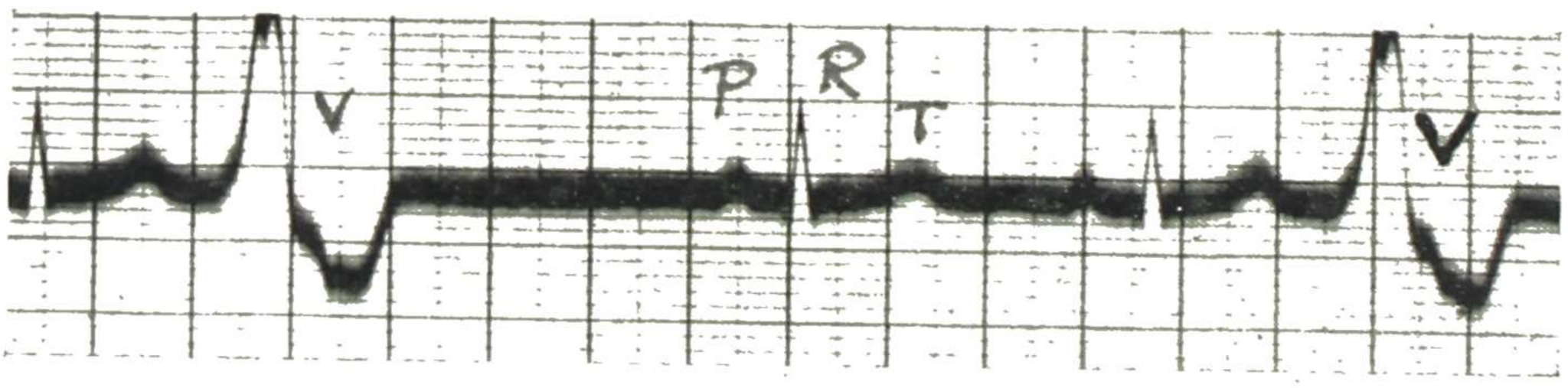


FIG. 32.—Premature ventricular beats (V). Note the prematurity and the large amplitude of the complexes.

type. Should the ectopic beat start in the nodal tissue, it travels upwards towards the auricles and downwards towards the ventricles, so that the auricles and ventricles contract more or less simultaneously.

Symptoms. The patient may or may not be conscious of the altered heart beat. The symptoms are palpitation, a catch in the breath, a thump, a sinking sensation, or even a sudden momentary stab of præcordial pain.

Broadly speaking, the *causes* of hyper-irritability which give rise to premature beats or extrasystoles are of (1) extrinsic, or extra-cardiac; or of (2) intrinsic, or cardiac origin. Extrinsic, *e.g.*, a distended stomach causing premature beats starting in either the right ventricle or auricle: such beats disappear on relief of the distension. In the eighth month of pregnancy the heart is often irregular, due to the distended abdomen.

The commonest intrinsic causes of hyper-irritability are: (1) Toxæmia; (2) fatigue; (3) inflammation; (4) degeneration.

(1) The connection between premature beats and *toxæmia* is seen with such conditions as malaria, typhoid, influenza, excessive tobacco, etc. Thyrotoxicosis, an abnormal susceptibility to coffee or tea, and digitalis poisoning are also common

causes. Septic foci may be causal. Generally speaking, all parts of the heart are involved, so that the irregularity is considerable, sometimes the auricles, sometimes the right and at other times the left ventricle showing premature beats. The abuse of tobacco as producing cardiac irregularities is well known and is a recognised method of evading military service.

(2) The relationship between premature beats and *fatigue* is especially interesting. The premature contractions may be associated either with a general fatigue and only occur after actual physical effort, or they may be associated with myocardial disease. Premature beats which appear or increase only after physical effort, are suggestive of myocardial damage. Any irregularity of the heart which develops with or after exercise, whether due to premature beats, auricular fibrillation, auricular flutter or any other cause, must always be looked upon as due to some definite myocardial disability. Even when premature beats are more frequent *after* exertion, they will generally be found to disappear during the period of exertion: this distinguishes them clinically from auricular fibrillation, where the rhythm is more irregular *during* exercise.

(3) *Inflammation.*—In acute myocarditis premature beats are common. In chronic forms of myocardial disease they frequently occur independently of any endocardial or pericardial involvement. Persistent premature beats increased by exercise always indicate myocardial damage. If auricular, they often precede fibrillation; if ventricular, they are often associated with acute local or diffuse myocardial disease. The association between premature beats and inflammation is often well seen in the course of an attack of rheumatic fever. The patient, perhaps, has well-marked signs of endocarditis or pericarditis, but as far as one can tell the myocardium has escaped damage. The heart then becomes irregular, and owing to the development of premature beats, one knows that the inflammation has involved the myocardium.

(4) In *myocardial degeneration* premature beats most commonly occur after fifty years of age, when the cardio-vascular system is degenerated; they are frequent antecedents to such conditions as auricular fibrillation or heart block, associated with generalised myocardial change.

Prognosis.—The essential point to remember first is that the majority of individuals who have ventricular premature beats do not suffer at the time or subsequently from heart failure or gross disease. The prognosis in all cases with premature beats must be decided independently of the presence of this sign. The prognosis has been briefly mentioned under each cause. Ectopic beats are important according to (1) the underlying condition on which they depend, and (2) the amount of circulatory disturbance they produce. Premature beats are occasionally so distressing to the patients as to require treatment. If so, a useful mixture contains quinidine sulphate gr. 2 or 3; atropine sulphate gr. $\frac{1}{150}$; acid. sulph. dil. ℥ 10; aq. chlorof. ad $\frac{1}{2}$ fl. oz., t.d.s., p.c.

The first stage of **heart block** (§ 69), in which the only change is lengthening of the "P–R" interval, is not diagnosable clinically (Fig. 16). The second change is that of an occasionally dropped beat, the "P–R" interval lengthening from normal progressively, until eventually an auricular stimulus fails to reach the ventricle. A ventricular beat then drops out. In this case the pause is completely silent (Fig. 17). The common causes of this condition are acute rheumatism, diphtheria, pneumonia, and occasionally other febrile states.

Sino-auricular block is a rare condition of no especial clinical significance, in which there is a complete obliteration of the whole of a cardiac cycle, both auricular and ventricular beats being equally affected. It is sometimes accompanied by other evidences of excessive vagal tone.

II. *The patient is* YOUNG, *and presents a* REGULARLY RECURRING *alteration of the pulse rate, usually dependent upon* RESPIRATION. *The condition is* SINUS ARRHYTHMIA.

§ **65. Sinus Arrhythmia** is a condition in which the discharge of perfectly normal impulses from the sino-auricular node occurs periodically, producing a rhythmic irregularity of the heart.

Symptoms.—The pulse rate increases with inspiration, but there is no great difference between the strength of any two successive beats. The regular waxing and waning of the pulse is often accentuated when the patient breathes deeply. Auscultation reveals no alteration in the heart sounds. This irregularity is without symptoms.

Causes.—The condition is common in the young and during convalescence from diseases in which the heart rate has been rapid. It is of vagal origin in individuals in whom there is exaggeration of the normal inspiratory increase and expiratory slowing of pulse rate.

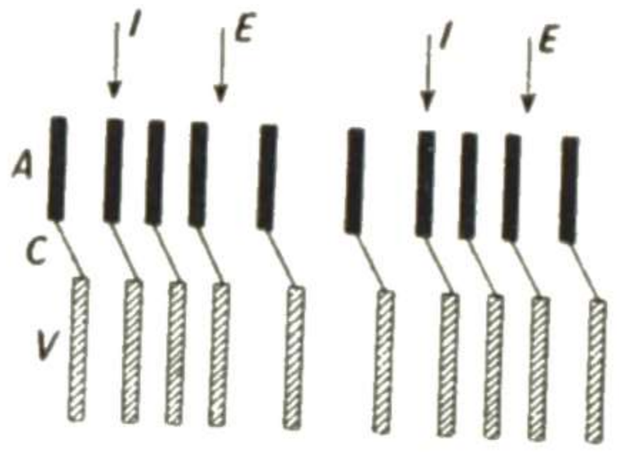


FIG. 33.—Sinus arrhythmia. The heart mechanism, auricular contraction (A), conduction (C) and ventricular contraction (V), is normal. The heart rate during inspiration (I) is increased, and during expiration (E) is slowed.

Prognosis.—The condition is of little importance; it ceases when the pulse rate quickens from any cause, *e.g.*, after exercise. When found after fevers, it is a good sign, inasmuch as it suggests the absence of extensive damage to the heart wall. No treatment is indicated.

§ **66.** III. *The Cardiac conditions in which an Increased Rate forms the most striking feature, are:* PAROXYSMAL TACHYCARDIA, AURICULAR FLUTTER, AURICULAR FIBRILLATION.

In the majority of cases of regular TACHYCARDIA the increase is physiological in character. In two conditions, paroxysmal tachycardia and auricular flutter, the heart beat starts from a new focus.

These two forms of tachycardia may be differentiated by the following features:—In the physiological type the pulse rate is (i.) affected by posture, falling 10 to 30 beats when the patient passes from a standing to a recumbent position; (ii.) the pulse rate increases with exercise, and is affected by emotion, meals, fever, and sleep; (iii.) the onset and termination are gradual; (iv.) electrocardiograms are normal; (v.) jugular tracings show no exaggeration of the force of the auricle. The causes of this form of tachycardia are dealt with in § 84.

The pulse rate is REGULAR, 130 to 200; *the rate is unaffected by exercise or posture. The condition is* PAROXYSMAL TACHYCARDIA OR AURICULAR FLUTTER.

Paroxysmal Tachycardia is a term reserved for cases of rapid action of the heart presenting the following characters: (1) the onset of the tachycardia is abrupt; (2) the duration varies from seconds to days; (3) the relief is sudden, and the pulse returns to its normal rate in the course of a few beats, which are often irregular in force and rhythm. During the paroxysm violent jugular pulsation may be visible.

The *symptoms* complained of by the patient depend upon the duration of the paroxysm. Many of the short paroxysms, lasting a few hours, are accompanied only by a fluttering or throbbing sensation in the chest or at the root of the neck, and a feeling of lassitude. Some attacks cause pain of an anginal nature. When the attack is prolonged over several days, grave cardiac embarrassment with dilatation, cyanosis, œdema of lungs, and engorgement and enlargement of the liver occur. Occasionally there is great distress and discomfort. The general disturbance of the patient, the rapidity of the pulse, and the severity of the abdominal pain dependent upon the engorgement of the liver may be so extreme as to simulate an acute abdominal condition calling for surgical interference. Cases are on record of exploratory laparotomy having been performed owing to such an error of diagnosis. The rapidity of the disappearance of the abdominal symptoms on cessation of the tachycardia is a very striking feature. The immediate *prognosis* depends chiefly upon the presence or absence of dilatation of the heart. The most severe symptoms may disappear in less than an hour if the heart muscle is healthy.

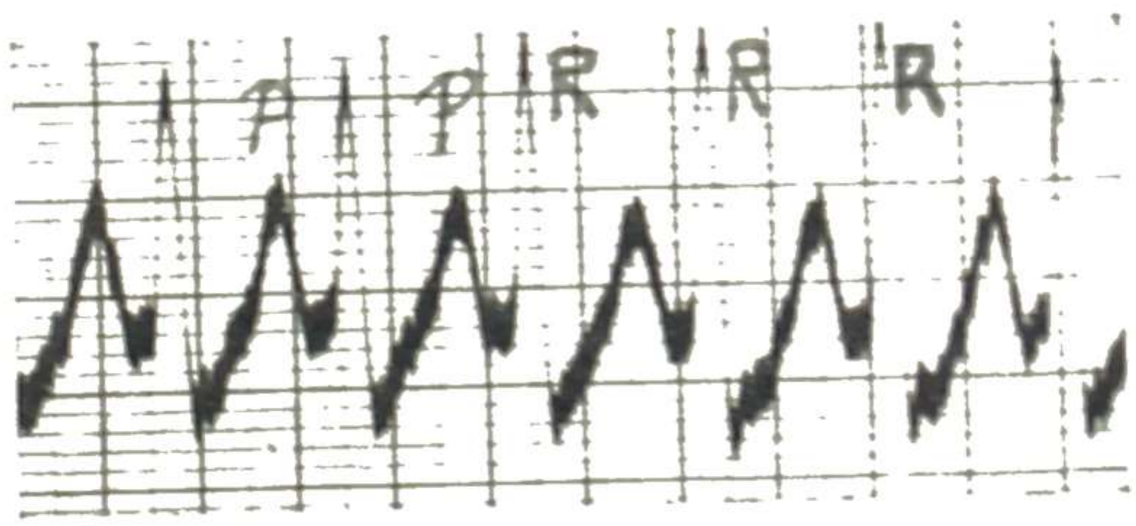


FIG. 34.—Auricular paroxysmal tachycardia. The "P" waves are abnormal in shape showing an ectopic origin, and at a rapid rate. The "QRS" waves are normal.

Etiology.—The condition is due to sudden rhythmic activity of some ectopic focus (usually in the auricle), which for a time overcomes and replaces the normal activity of the sino-auricular node. It is most common in young adults, but may occur in early childhood or old age. The attacks may be excited by exertion, emotion, flatulence, or change of posture. The disturbance is often not ascribable to physical disease, but is sometimes a consequence of rheumatism, scarlet fever, syphilis or coronary disease (see § 68, Etiology). Often no valvular lesion is present; if valvular murmurs are present they become unrecognisable during the paroxysm. Post-mortem examination has shown fibrosis, pallor, friability of the heart-muscle and sometimes coronary disease.

The *diagnosis* from tachycardia of purely *nervous* origin depends upon: (i) the abrupt onset and relief; (ii.) the presence of violent jugular pulsation; (iii.) occasional presence of a few premature beats in the intervals between the paroxysms. Many attacks of so-called "Paroxysmal Tachycardia" are really paroxysms of Auricular Fibrillation or Auricular Flutter (see below).

Treatment.—The brief paroxysms which produce no subjective symptoms call for no treatment. For the prolonged attacks the patients often discover for themselves some simple procedure which cuts them short, such as holding the breath, compressing the abdomen with a tight binder, or the assumption of some special posture. If these are inadequate, pressure may be made upon the vagus in the neck. Morphia may be called for, and should embarrassment of the right heart become extreme, venesection and the removal of fl. oz. 10–20 of blood may give relief. Drug treatment is useful during the attack and between attacks as a prophylactic measure. For the former

purpose digitalis in full doses (see § 62) can be tried. In a case of emergency quinidine sulphate can be given in increasing 2-hourly doses of gr. ii., iii., iv., v., vi., and vii.; but it is definitely dangerous in this condition, and should only be given if the danger of the attack itself seems the greater. Acetyl choline in the form of carbachol B.P. (doryl), intravenously, will often cut short an attack: 1 c.c., diluted in 10 c.c. of saline, is given slowly into a vein. Prophylactically, quinidine sulphate in doses of gr. 2 or 3 t.d.s., p.c. is a safe and useful remedy.

§ 67. Auricular Flutter.[1]—This name has been given to a condition in which "the normal beats of the auricle are submerged by contractions of this chamber in response to a series of new, rhythmic, and pathological impulses varying in rate from 200–350 per minute" (Lewis). The distinction from Paroxysmal Tachycardia is arbitrary as regards auricular rate, and is drawn at 200 beats per minute. Flutter differs, however, from Paroxysmal Tachycardia in that it is due to a circus movement and is almost invariably associated with some degree of heart block (§ 69); the auricle may, for instance, be beating at the rate of 300 per minute, while the ventricle responds with only 150 beats per minute. In other cases a higher grade of heart block (4–1) may be present, and the

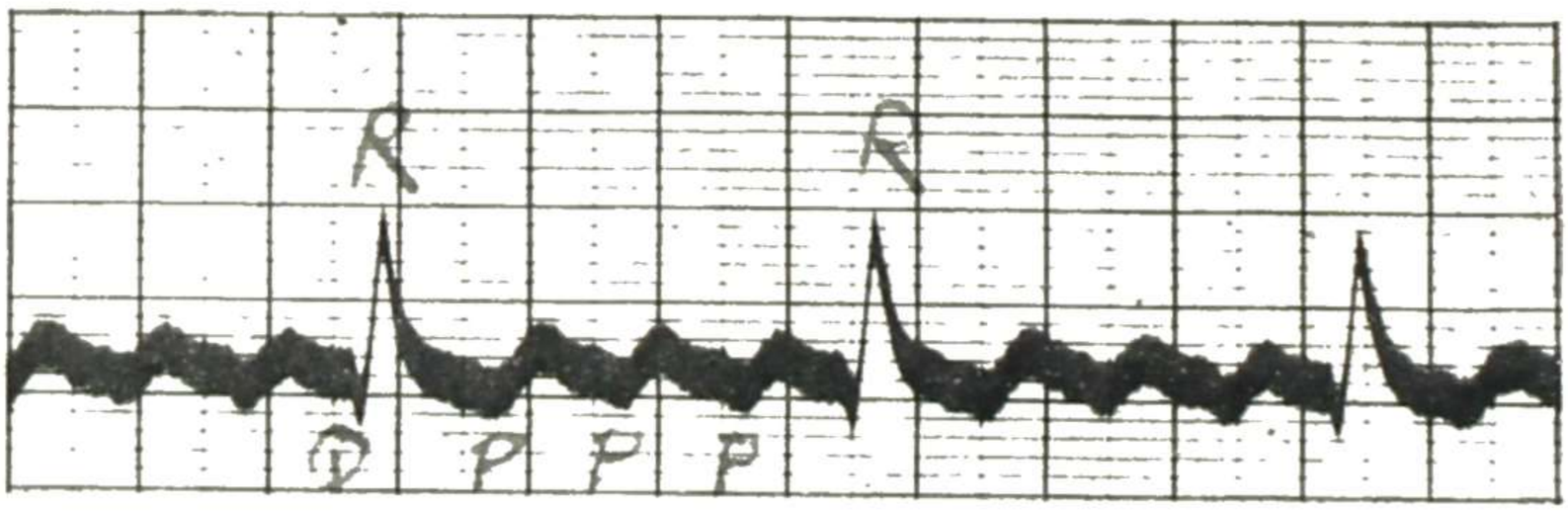


FIG. 35.—Auricular flutter. The auricular waves are undulatory at the rate of 300, and the ventricle responds only to every 4th auricular beat. One of the "P" waves is periodically obscured by the "QRS" waves.

pulse rate be about 75 and regular. The rate of the auricle is absolutely regular; that of the ventricle may be regular or irregular, depending upon the constancy of the degree of heart block present (and see p. 105). It will be readily seen what difficulty there may be in making a clinical diagnosis of this serious heart condition.

Symptoms and Signs.—The symptoms of auricular flutter are generally those of heart failure; palpitation is sometimes complained of and the pulse rate is found to be unexpectedly feeble and rapid (see Fig. 35). For the certain recognition of the disorder polygraphic or electrocardiographic records are necessary, but its existence may be suspected when (i.) the pulse rate is 130–160, regular, and maintained for long periods, especially if associated with syncopal attacks; (ii) the ventricular rate shows no quickening with exercise or slowing on lying down. This constancy of the rate on alteration of posture and with exercise is an important

[1] Flutter has been placed under this heading because it is usually associated with a regular ventricular pulse. When varying grades of heart block are present this regularity in rate disappears, but in contrast to the pulse of auricular fibrillation regularity in force and volume persists.

diagnostic feature. (iii.) A visible jugular pulse of greater rate than the apex should lead one to suspect auricular flutter.

Etiology.—The condition is usually associated with chronic myocardial degeneration, but occasionally accompanies mitral stenosis. The patients are often past middle life, and are subjects of arteriosclerosis. The irregularity is occasionally produced by quinidine when given in auricular fibrillation (and see p. 110).

Prognosis.—The duration of the condition varies. It may occur only shortly before death; more frequently it persists for long periods, even for years. This long duration is a further diagnostic point of great importance. There is a danger of the ventricle suddenly assuming a rate equal to that of the auricle—a condition which can lead to syncopal attacks which may be fatal.

Treatment.—Digitalis in 20 minim doses four times a day will often effect a cure; it acts by producing auricular fibrillation (see below). If the drug is then withdrawn, fibrillation may suddenly cease and the heart resume its normal rhythm. Even if digitalis fails to produce fibrillation, it may be relied upon to reduce the ventricular rate. If large doses of the drug are given, the period of fibrillation may be cut short. A daily dosage of 90 minims for two or three days is often necessary. Quinidine sulphate can be used as an alternative method of treatment, as described in § 68.

IV. *The heart rate is irregular, with* COMPLETELY IRREGULAR *rhythm, and in failure may beat at a rate as high as* 180 *per minute; the condition is probably* AURICULAR FIBRILLATION.

§ 68. Auricular Fibrillation is recognisable by (i.) Complete irregularity of the pulse; (ii.) the difference between the apex and the pulse rates; (iii.) increase in the irregularity by exercise; (iv.) absence of the "A" wave on the venous curve or absence of the "P" wave on the electrocardiogram (Figs. 14, 36). (v.) Auricular fibrillation is usually associated with a low blood pressure, due to the small output, but in cases where the peripheral resistance is raised (*e.g.*, arterial sclerosis, renal disease) the blood pressure may be high: it always varies from beat to beat. Furthermore, auricular fibrillation is almost invariably associated with (vi.) increase in

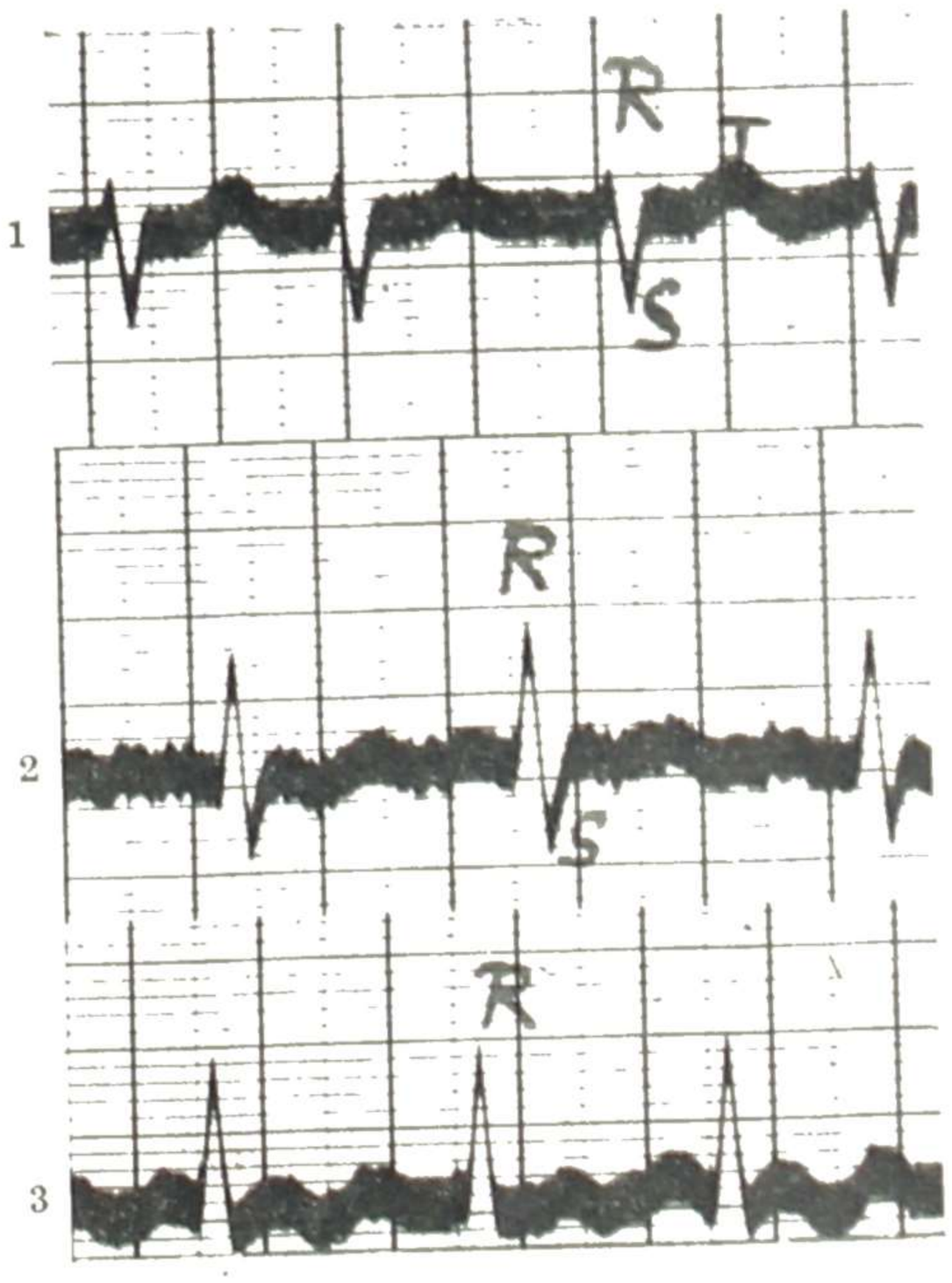


FIG. 36.—Right-sided preponderance. "S" is deepest in lead 1, and "R" is tallest in lead 3. (Auricular fibrillation is present.)

the cardiac dulness. (vii.) Auricular fibrillation is sometimes paroxysmal in character, the attacks lasting from a few minutes to hours, days, or even weeks: permanent fibrillation usually supervenes later.

Etiology.—The auricle is composed of a number of intimately connected muscle fibres, so much so that some people regard them as forming a syncytium; and a normal systole of the auricle is initiated at the sino-auricular node, and the stimulus travels from one muscle fibre to another, and gives a systematic and co-ordinate contraction of the auricle from above downwards, *i.e.*, from the sino-auricular to the auriculo-ventricular node. In auricular fibrillation the underlying process is a "circus movement" in which the sino-auricular node ceases to function and is replaced by a stimulus which makes a circuit of the muscle surrounding one of the large venous orifices 400–500 times a minute. The auricular muscle derives its stimulation from this circus movement in an entirely irregular fashion, for much of the auricular muscle is refractory to such a rate of stimulation. In consequence co-ordinate auricular contraction no longer occurs, but individual bundles of fibres, or even individual fibres, contract inco-ordinately, so that the whole auricular wall appears to be trembling or quivering in a state of diastole. The ventricle therefore receives impulses at very irregular intervals, and so the force and the rhythm of the ventricle varies from beat to beat. The stronger of these stimuli, at irregular intervals, traverse the Bundle of His and excite a ventricular contraction. According to the number of auricular stimuli which are transmitted to the ventricles, the rate of these will be fast or slow; the apex rate may vary between 180 and 40 per minute. Auricular fibrillation usually occurs: (1) secondary to valvular disease, especially mitral stenosis; (2) in myocardial degeneration, *e.g.*, cardio-vascular sclerosis; (3) in toxic conditions, *e.g.*, Graves' disease; (4) in the course of acute inflammatory conditions, *e.g.*, acute carditis; (5) very rarely as a result of syphilis.

The rate of circus movement in auricular fibrillation at 400–500 times per minute, is faster than in auricular flutter (200–350 per minute) because the path of the circus movement in fibrillation is shorter than in flutter.

The *prognosis* depends chiefly upon (1) whether the underlying cause is removable or not. In myocardial degeneration the cause is not removable but progressive; the prognosis is consequently bad. In Graves' disease, provided the muscle is only in the toxic and not in the degenerative stage, removal of part of the thyroid will remove the hyperthyroidism and the auricular fibrillation will cease. In mitral stenosis the immediately exciting cause of the fibrillation is a rise of pressure in the auricles. (2) The condition of the ventricular muscle. If the auricular fibrillation is due to a more or less local condition of the auricle, the outlook is comparatively good. But if the cause of the fibrillation has seriously affected the ventricular muscle, the prognosis is bad. (3) The extent to which the ventricle is overstimulated by the erratically acting auricle. Obviously the greater the ventricular rate and the larger the number of ineffective

beats, the greater the over-work of the ventricle and the worse the outlook. (4) Where the cause cannot be removed, how far it is possible to control the fibrillation. In some cases, *e.g.*, with mitral stenosis, it may not be possible to stop the fibrillation, but it may be possible to control it by treatment so that the ventricular rate is slowed and the number of ineffective beats few. In such cases the prognosis is good, the fibrillation making little difference to the conditions of life.

Treatment.—It is thus obvious that the treatment of auricular fibrillation first lies in attempting to remove the underlying cause. In some cases this is comparatively easy, in others it may be impossible. In hyperthyroidism it can be stopped by removing part of the over-active thyroid. In mitral stenosis, on the other hand, the removal of the cause is impossible. If it is impossible to stop the actual fibrillation, it is usually possible to control its effect on the ventricle. This is done by giving the patient that dose of digitalis which will keep the heart rate, at rest, between 70 and 80. If the heart rate is already as slow as this, digitalis is not needed. If there are signs of failure the patient must be treated in bed according to the scheme described above (see § 62). Digitalis must be continued, if needed, so long as the fibrillation remains, *i.e.*, usually for the rest of the patient's life.

Quinidine, an isomer of quinine, is sometimes useful in stopping auricular fibrillation. Contra-indications for its use are established mitral stenosis, signs of congestive failure, and marked cardiac enlargement. Where fibrillation is known to have persisted for many months, quinidine is generally not used. Thyrotoxic and early arteriosclerotic cases are the most suitable. Before quinidine treatment digitalis must be stopped; during quinidine treatment patients must be nursed flat, strictly confined to bed, and must not be allowed to sit up, for the drug is a myocardial poison. Dosage is as follows: gr. ii. t.d.s., p.c., for one day, to exclude the possibility of hypersensitivity to the drug, and on successive days eight two-hourly doses of gr. 2, 3, and 4, the final total daily dose being gr. 32. This final dose can be maintained for 2 or 3 more days, and is then stopped. If the auricular fibrillation ceases during treatment the course can be stopped, and a maintenance dose of gr. ii. or iii. t.d.s., p.c., continued for 2–3 months. In cases where auricular fibrillation is paroxysmal, a dose of 3–5 gr. a day will usually prevent attacks.

Quinidine sulphate acts as a myocardial depressant, and slows the rate of conduction as well as increasing the refractory period of the heart muscle undergoing circus movement. When the latter effect predominates, circus movement ceases.

V. *Conditions of the heart which are associated with a* SLOW PULSE.

§ **69. Slow pulse**—between 40 or 50 or below—occurs in four more or less common conditions. (1) Personal idiosyncrasy; (2) The heart of the well-trained athlete; (3) Debilitating and exhausting diseases; (4) Conditions of complete or partial heart block.

(1) In the bradycardia of the athlete, the subject usually looks and seems physically very fit; on exercise he shows no symptoms of distress;

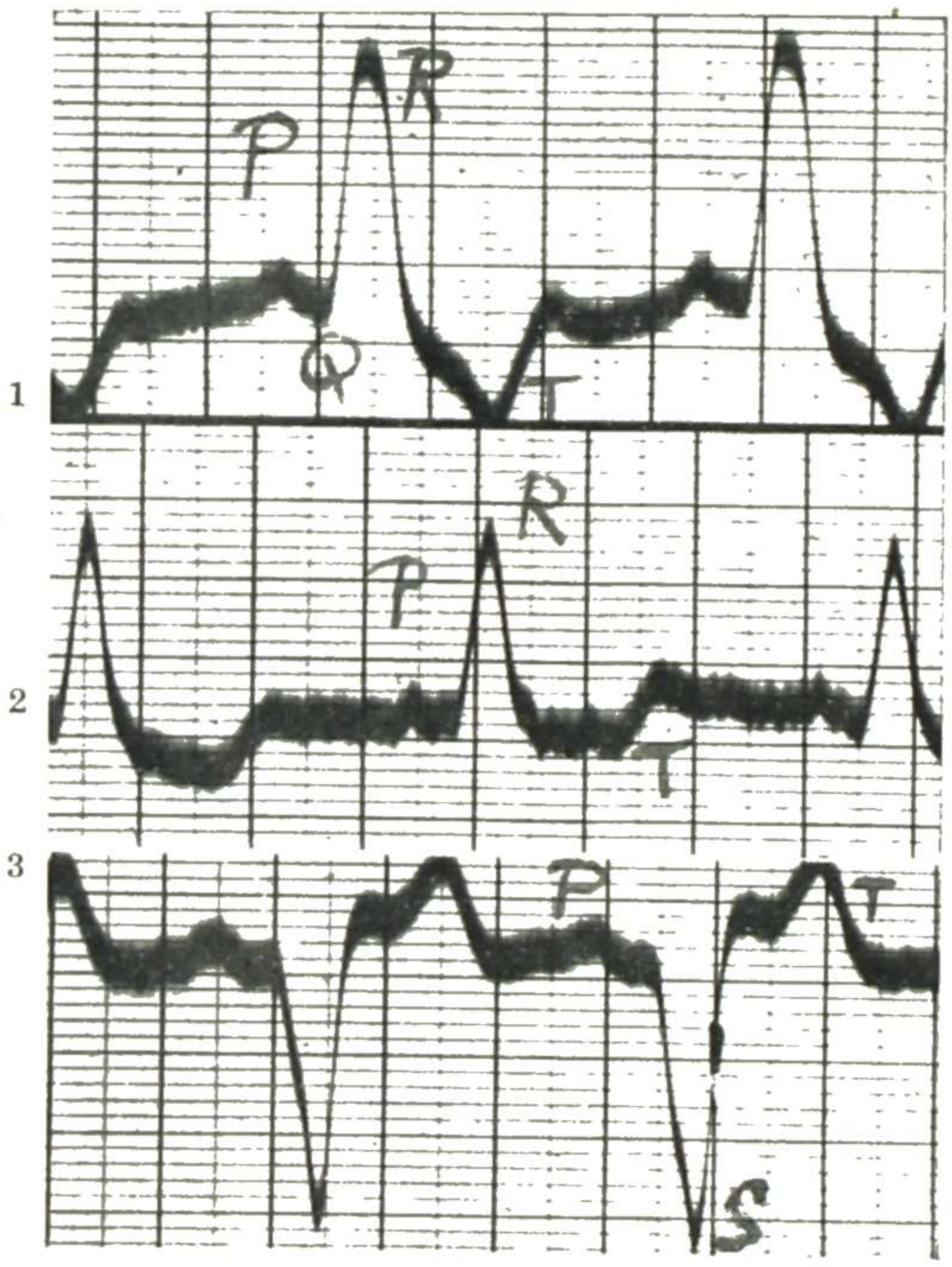


FIG. 37.—Lesion of the left bundle branch. Note (1) the widening of QRS to 0·12 sec. (normal less than 0·10 sec.), (2) the slurring of QRS, (3) the direction of T_1 and T_3, *i.e.* opposite to R and to S.

and his pulse rate, instead of climbing as the exercise is increased, suddenly doubles.

(2) Bradycardia may occur in conditions of lowered metabolism, such as inanition, exposure to cold, and myxœdema. It is also seen in some toxic states, as in jaundice.

(3) Digitalis may cause it in three ways—by producing heart block, by producing premature beats which fail to reach the arterial pulse, and by central vagal stimulation. In vagotonic conditions (*e.g.*, in increased intracranial pressure) and in shock bradycardia is often seen.

(4) In complete or partial **heart block** the rate is unaffected by exercise, and signs of cardio-vascular degeneration are frequently present. Periods of unconsciousness (Stokes-Adams' fits) may occur.

Etiology.—Conductivity is specialised in the Bundle system described in the introduction to this chapter. Any part of this conducting tract may be damaged, a whole series of conditions arising therefrom (Fig. 38). Sometimes the stimulus appears to be blocked in the sino-auricular node, when the whole heart is silent and misses a beat—a condition spoken of as sino-auricular heart block (§ 64). If the Bundle of His is damaged, then either partial or complete auriculo-ventricular block occurs. Furthermore, either the right or left main branch may be damaged, right or left bundle block resulting (Fig. 37). Bundle block not infrequently occurs in cardiac atheroma, with aortic regurgitation, in coronary thrombosis, in chronic lung affections and in certain cases immediately prior to death. All these conditions can be easily made out by means of the electrocardiograph.

The varieties clearly depicted in Fig. 38 are here summarised :—

1. *Supra-auricular* (Sino-auricular) (§ 64).
2. *Auriculo-ventricular :*
   - (*a*) Temporary { Complete. / Incomplete.
   - (*b*) Permanent { Complete. / Incomplete.
3. *Ventricular :*

   Main Branch Block :
   - (1) Right
   - (2) Left

   } Temporary or permanent.

The conditions of the Bundle which can produce heart block fall into three groups : (1) infective processes, (2) degenerative processes, and (3) the influence of

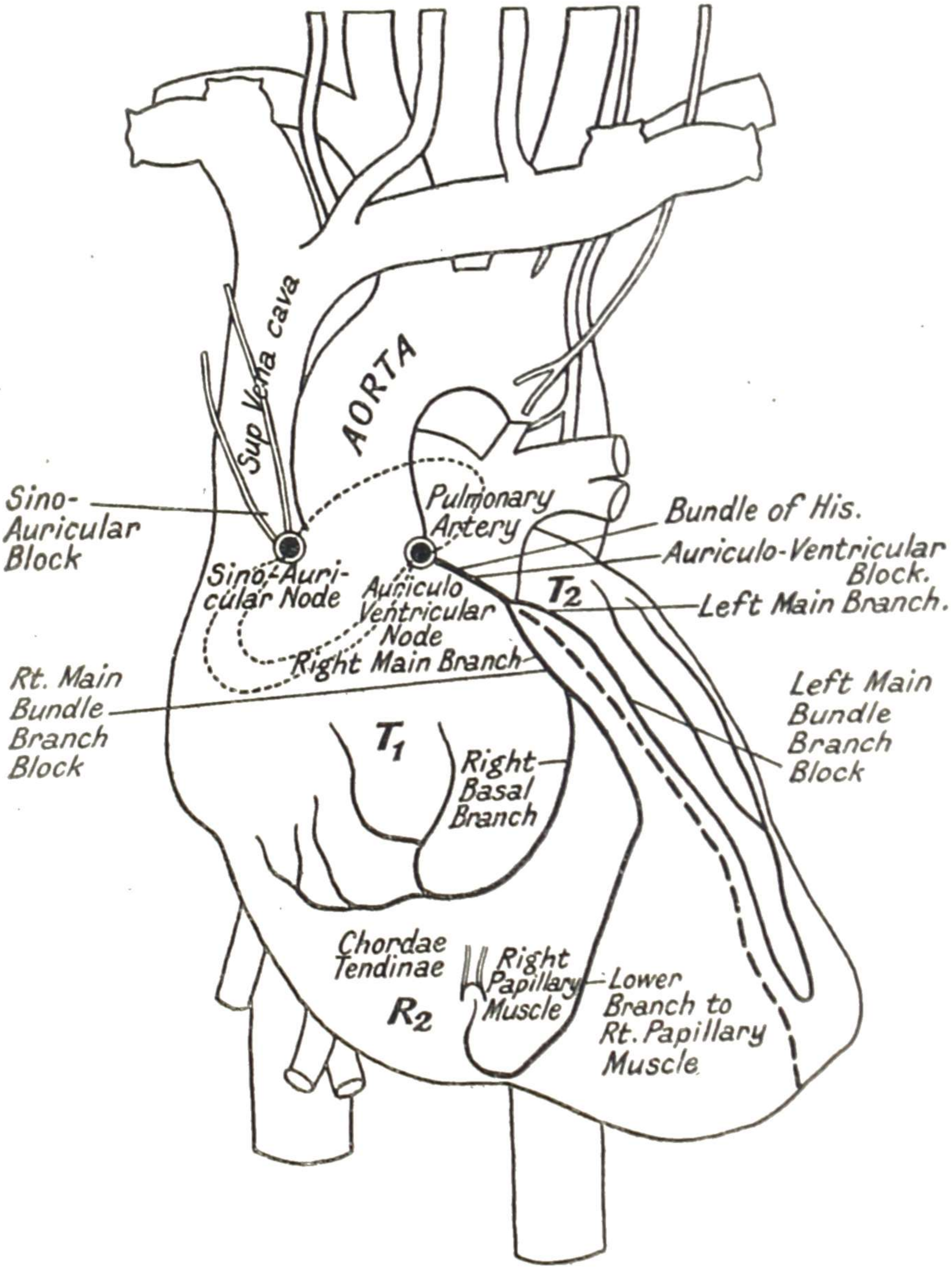


FIG. 38.—Diagram illustrating the positions of the lesions in the different varieties of Heart Block.

drugs. The infective processes are numerous: rheumatism is the chief; diphtheria, pneumonia, influenza, scarlet fever and syphilis are all common. Degenerative processes include fibrosis, the result of old rheumatic fibrotic processes, interference with nutrition through disease of the coronary vessels, and tertiary syphilitic lesions of the heart muscle itself. Digitalis has a pronounced action in lowering conductivity of the Bundle, an effect which is most marked when the Bundle is already diseased, and which is usefully employed in the treatment of auricular fibrillation.

In the milder forms the *prognosis* and the *treatment* are those of the associated disease. Digitalis is usually contra-indicated, owing to its action in lowering conductivity, but in severe cases, where signs of gross heart failure are also present, and rest in bed proves insufficient, digitalis may be serviceable by virtue of its beneficial

action upon the ventricular muscle. In cases in which the possibility of syphilitic changes can be entertained active anti-syphilitic remedies should be employed.

Complete Heart Block implies total interruption of impulse between auricles and ventricles (Fig. 18). When this occurs the ventricles initiate their own rhythm. This is regular, and at a rate which varies from 24–36 (usually 28–30) in different patients, but remains remarkably constant in any individual case. Complete heart block is not infrequently associated with syncopal attacks—**Stokes-Adams' Disease**, first described by R. Adams in 1827. The patients are usually advanced in years, complain of dyspnœa, and have marked bradycardia, the pulse rate ranging from 20 to 40. Any mental excitement is liable to bring on an attack. The attack is due to cerebral anæmia resulting from ventricular standstill, and lasts from four to thirty seconds. The shorter attacks are characterised by transient dizziness, longer ones by brief syncopal periods; in the more severe seizures the breathing becomes stertorous, the face cyanosed, there is dilatation of both pupils, rigidity of the body, accompanied by clonic movements of the limbs. The pulse occasionally ceases for a few seconds, the jaw drops, and for a brief period the patient is to all appearances dead. No pulse is felt in either wrist, and on auscultation the cardiac sounds are inaudible. Then a feeble sound is heard, followed by a stronger, and a second later the pulse begins beating at about 30 per minute (one can feel the artery fill), the cyanosis lessens, the pupils contract, and consciousness returns. Many such fits may occur in succession, from six to ten in a single night.

The *prognosis* in complete heart block depends upon the presence of dyspnœa and other signs and symptoms of failure. Life is often prolonged for years, for the slow heart rate saves the heart muscle.

Treatment.—The attacks may be prevented by the subcutaneous injection of adrenalin which in all probability acts directly on the Bundle of His viâ the sympathetic. Adrenalin in oil (1 in 100) 1 c.c., 3 or 4 times daily, is best for this purpose. The underlying cause must, however, be treated; for this purpose potassium iodide is often very useful. Ephedrine in doses of gr. ½ or gr. 1 three or four times a day by mouth is also useful.

§ **70.** *The Heart-beats occur in couples, with a pause after every alternate beat.*

This condition may be apparent or real, and may be due to:

(1) Regularly recurring PREMATURE BEATS; a common cause of which is DIGITALIS overdose. (Fig. 39.)

(2) PULSUS ALTERNANS. (Fig. 39.)

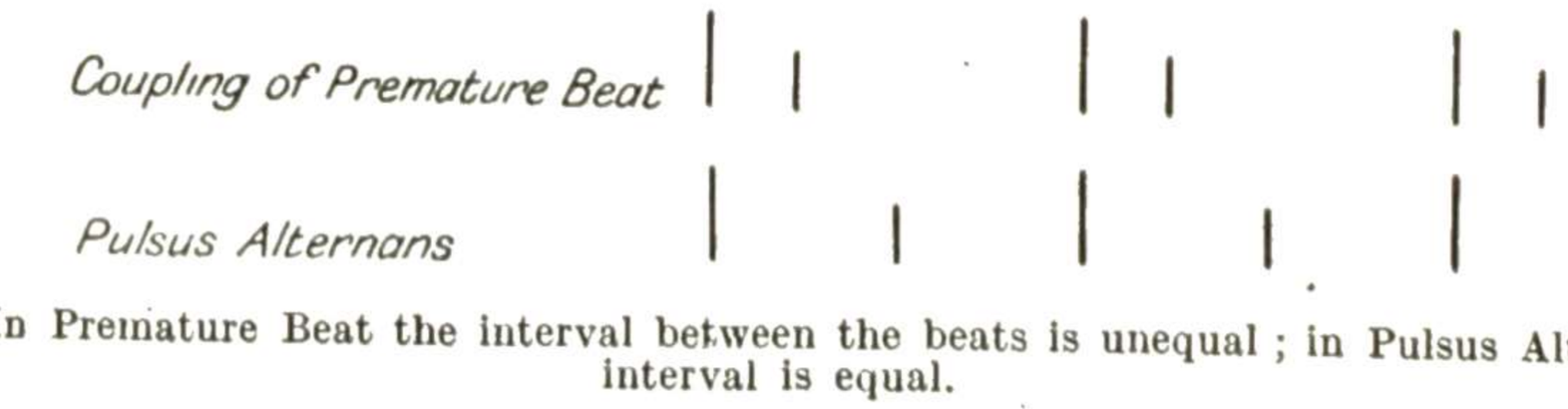


FIG. 39.—In Premature Beat the interval between the beats is unequal; in Pulsus Alternans the interval is equal.

(3) Extreme DICROTISM produces an appearance of coupling, but is distinguished from that due to premature contractions by the fact that the apparent second beat occurs synchronously with the closure of the aortic valves, and is unaccompanied by a systolic heart sound. It occurs where the diastolic pressure is low but the aortic valve competent (§ 88).

§ **71. Pulsus Alternans** is a condition in which every second ventricular beat is feebler than its predecessor and the rhythm remains regular. This sign is an evidence of exhaustion of contractility: it may be constant, or only appear occasionally

after premature beats. To distinguish from a coupled pulse see Fig. 39. The condition is of very grave significance when it occurs with a slowly acting heart. With a quick pulse rate it need not be regarded with such grave apprehension, but its appearance is always a warning sign of cardiac exhaustion. Pulsus alternans is most readily shown in a pulse tracing, but can be easily diagnosed by the sphygmomanometer. If the pressure is raised above the systolic figure and allowed slowly to fall, only the alternate stronger beats will at first be heard by the stethoscope. At a lower pressure the weaker beats, evenly spaced between the stronger ones, then appear.

CHAPTER IV

ANEURYSM OF THE AORTA AND OTHER INTRATHORACIC TUMOURS

Anatomy.—The mediastinum is the irregular space in the chest which lies between the two pleural sacs. For descriptive purposes it is divided into four parts—viz., the *middle mediastinum*, which is occupied by the heart and pericardial sac; the *anterior*, which is the space in front; the *posterior*, the space behind; and the *superior*, the space above the pericardial sac. The most important structures contained in these spaces are: The thymus or its remains; the arch of the aorta with its branches (innominate, left subclavian, and left carotid); the superior and inferior venæ cavæ, with the innominate and azygos veins; the pulmonary vessels, the trachea and bronchi; the vagus, recurrent laryngeal, phrenic, and splanchnic nerves; the cardiac and pulmonary plexuses; the roots of the lungs; the œsophagus, thoracic duct, lymphatic glands and vessels, and loose cellular tissue (Fig. 11). The lymphatic glands are important on account of the occurrence of lympho-sarcoma and other glandular enlargements which may form mediastinal tumours.

If, on percussing over the sternum,[1] or just beside it, the præcordial dulness is found to be **increased irregularly upwards**—the morbid condition may be PERICARDIAL EFFUSION, ENLARGEMENT OF THE PULMONARY CONUS, RETRACTION OF THE LUNG, AN ABDOMINAL SWELLING PUSHING UP THE HEART AS A WHOLE, OR AORTIC ANEURYSM OR SOME OTHER MEDIASTINAL TUMOUR. The two last named are generally to be distinguished sooner or later by the presence of pressure symptoms (p. 121). With the aid of X-rays the diagnosis of all mediastinal conditions is usually made clear.

If there is **abnormal dulness** *near the base of the heart, which is accompanied by* PULSATION, *and on auscultation, there is a* REINFORCED OR RINGING SECOND HEART SOUND—*perhaps a systolic or diastolic murmur—the disease is probably* ANEURYSM OF THE AORTA.

§ 80. Intrathoracic Aneurysm.—In regard to the anatomy of this serious and important malady, the student should study Fig. 11 (p. 51). Aneurysm of the aorta used to be the commonest of intrathoracic tumours. Since the introduction of organic arsenical compounds in the treatment of syphilis, this form of cardio-vascular syphilis has become comparatively rare.

The arch of the aorta is the favourite seat for aneurysmal dilatation. Any part of it may be affected—the ascending, transverse, or descending part of the arch. The dilatation may assume either a fusiform or saccular shape, the former being the more frequent. Fusiform dilatation arises as a rule in the first part of the aorta, and may lead to stretching of the valves and aortic incompetence. The fusiform aneurysm gives rise to practically no physical signs, andthe ensuing description refers, unless otherwise stated,

[1] Remember, in percussing over the sternum, the note elicited is of a much higher pitch than that just beside the sternum.