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.

to saccular aneurysm. It may make its way in various directions, and bones, cartilages, and other hard structures may become eroded and absorbed under its pressure. According to its position, aneurysm of the aorta may be either very easy or very difficult to detect. If it involves the ascending aorta, near the *front* of the chest, it is soon revealed by definite *physical signs*. If the transverse or descending parts are involved, and the tumour extends backwards, there may be no physical signs, and even the *pressure symptoms* may be obscure. Thus the clinical manifestations belong to two categories—physical signs and pressure symptoms; and we have two varieties of aneurysm: (*a*) The *aneurysm of physical signs*, when the ascending aorta is involved; (*b*) The *aneurysm of pressure symptoms*, when the transverse and descending parts of the aorta are involved.

The *Symptoms Common* to aortic aneurysm in all positions will be considered first, because these are the symptoms which will probably first attract our notice. Then we will turn to certain others *special* to the ascending, transverse and descending parts of the aorta respectively.

Symptoms Common to All Positions:

1. Dyspnœa is often one of the earliest complaints which the patient makes. When it is due to pressure on the trachea, as in aneurysm affecting the transverse aorta, it is persistent and stridulous in character. When it is due to narrowing of the coronary arteries by the diseased aortic wall, it is often paroxysmal. Orthopnœa of a marked degree may be present.

2. Cough is often present and has a characteristic brassy sound (gander cough). Pressure upon the recurrent laryngeal nerve is common, with consequent paralysis of the left vocal cord, and there may be hoarseness or even aphonia from the same cause. *Paralysis of the left vocal cord* in the absence of central nerve lesions, suggests aortic aneurysm or a neoplasm. Laryngoscopic examination should be a matter of routine in all suspicious cases, because abductor paralysis occurs before complete paralysis, and the former may be unattended by any alteration of voice.

3. Pain is another common symptom; it is felt in the chest, or in the back. It is frequently worse at night, or on exertion. It may be in the form of angina of effort if the coronary orifices are involved, shooting down one or both arms, usually the left, especially in aneurysm of the ascending aorta. The pain may be neuralgic when there is pressure on nerves; or it may be of a dull boring character when due to erosion of bone, such as occurs in connection with aneurysm of the ascending or descending aorta. This variety of pain is often worse at night. Short of definite anginal attacks of this kind, patients with aortic aneurysm are liable to feelings of suffocation, constriction, or "spasm" in the chest, and nameless dreads come over them from time to time without cause. Such attacks may in many cases be brought on by bending the head backwards, or by any movement which stretches the neck.

4. Palpation of the chest provides two signs. Diastolic shock is felt over the aortic base and is synchronous with the second sound. Systolic pulsation may be locally palpable on the thoracic surface, or may be observed by sitting the patient upright and by placing the palm of one hand firmly and flat over the chest and the palm of the other over the corresponding area of the back.

5. A reinforcement of the aortic second sound is the most constant of the auscultatory signs of aortic aneurysm. Dilatation of the first part of the arch gives rise to a ringing aortic second sound, which is quite characteristic.

6. Inequality of the radial pulses is a fairly frequent sign. It is present whenever the aneurysm is so placed as to cause a difference in the arterial pressure in the great vessels which spring from the aorta. The typical aneurysmal pulse occurs in the vessel just beyond the sac, and its characteristic is a decrease of the pulse wave, the blood tending to flow in one continuous stream. Owing to the fact that the radial arteries are often of different sizes the blood pressures on the two sides should be carefully compared to confirm the impression obtained by feeling the pulses. The sign, however, is not diagnostic, for atheroma of the vessels may cause it.

7. Inequality of the pupils occurs from inequality of carotid blood pressure and corresponding inequality of blood-pressure in the vessels of the iris. In the early stages the irritation of the sympathetic nerve causes dilatation of the pupil on the same side. Later on there is paralysis, contraction of the pupil, enophthalmos, ptosis, sometimes vascular dilatation and unilateral sweating of the face and neck (Horner's syndrome).

8. The heart may be displaced when the aneurysm is large, usually to the left.

9. Hæmoptysis may occur; profuse, from rupture, and slight, from the associated pulmonary congestion.

10. Congestive heart failure may occur.

(*a*) Symptoms peculiar to aneurysm of the **ascending** or **first part of the arch.** Aneurysm of this part of the arch is usually easy of detection, and in marked cases the *Physical Signs* are unmistakable. (i.) On inspection, pulsation is often visible over the right upper thoracic spaces. (ii.) On palpation, a diastolic shock may be felt unless the aortic ring is stretched by the aneurysm. Supra-sternal pulsation and a tracheal tug may be felt. The pulsation is felt to be expansile. A systolic thrill may be palpable. (iii.) On percussion, dulness is present, continuous with that of the heart and expanding upwards and outwards from this organ to the right. (iv.) On auscultation, a systolic murmur may or may not be audible. Since the aortic ring is frequently involved, all of the signs and symptoms of aortic incompetence may be present in addition (see § 60). (v.) The right bronchus may be pressed upon, leading to diminished or absent respiratory murmur of the right lung. In severe cases there may be pressure on the superior vena cava, with œdema of the neck

and arms. (vi.) The dyspnœa is paroxysmal; and the right recurrent laryngeal nerve may be involved, with *right laryngeal paralysis*.

(*b*) The symptoms of aneurysm of the second or **transverse part of the arch** may be equally easy to detect when it makes its way forwards. But when the posterior part is affected it may present considerable difficulty in diagnosis, especially from other intrathoracic tumours. (i.) The dyspnœa may be either paroxysmal or continuous, with inspiratory stridor, owing to the pressure upon the trachea. (ii.) Pressure upon the left bronchus may lead to diminished breath sounds in the left lung, partial collapse or bronchiectasis, and symptoms (2) and (5) above are especially marked in aneurysm of the transverse arch. (iii.) Tracheal tugging is a very characteristic sign of aneurysm in this situation. Standing beside the patient, whose head is held level and straight, the examiner defines the cricoid with finger and thumb, and lifts it upwards without backward pressure, away from the thorax. If the aorta is in close contact with the bronchial tree, either by pressure of an aneurysm or by adhering to it by growth, a systolic downward tracheal tug will be felt. (iv.) The physical signs—which are in this situation less marked, or may be absent—consist of a thrill felt on palpating the suprasternal notch; dulness on percussion over the manubrium, continuous with that of the heart, and extending from the middle line to the left of the sternum.

(*c*) Aneurysm affecting the **descending aorta** may be very difficult to diagnose. (i.) Pain in the back and dysphagia are the most constant symptoms. The pain may pass to the side, following the course of an intercostal nerve. It is due to erosion of the vertebræ, which can be demonstrated radiologically. (ii.) Other pressure symptoms are dysphagia, from pressure upon the œsophagus; wasting, from pressure upon the thoracic duct, and signs in the left lung, from pressure upon its bronchi. (iii.) The most diagnostic sign when present is the "Lateral Thoracic Jerk" (Bourne).[1] The whole thorax is jerked to the left during systole. This can best be seen by inspection from the foot of the bed. The jerk is caused by the fact that the ventricles, the aneurysm, and the vertebral column are in direct propinquity, and a thrust is thus transmitted laterally to the left chest wall from the left side of the vertebral bodies when the first two structures become hardened during systole. (iv.) If the swelling enlarges, physical signs on auscultation and percussion may become apparent in the left (occasionally the right) scapular region; and in advanced cases there may even be a pulsating swelling without the knowledge of the patient. Osler found that in some cases there is absence of pulsation in the femoral arteries.

Etiology.—(1) Aortic aneurysm is far more frequent in men than in women, especially in those in the prime of life—namely, between the ages of thirty-five and fifty. (2) It is especially frequent among soldiers, blacksmiths and others who do laborious work, probably due to the fact that these classes are subjected to sudden and severe muscular exertion and

[1] The *Lancet*, 1932, II, 68.

heart-strain at certain times. (3) Syphilis accounts for the large proportion of cases of thoracic aortic aneurysm, and for many of those of the abdominal aorta. (4) Atheroma is the cause in a very small minority of cases. Atheromatous disease generally produces a fusiform aneurysm, and is responsible for a small minority of cases of aneurysm of the descending thoracic aorta and of a higher percentage of aneurysms of the abdominal aorta (§ 263). (5) Some cases of aneurysm date from a period of over-exertion, exposure, and destitution, or from an injury as an exciting or secondary cause.

Diagnosis.—The diagnosis of a deep-seated aneurysm is sometimes difficult in the early stages. The diagnosis from *cardiac valvular disease* is made by the pressure symptoms. Moreover, aneurysm does not cause cardiac enlargement unless there is secondary aortic incompetence. Many of the local signs of a saccular aneurysm may be produced by a *dilated and rigid aorta*, but here the pressure symptoms are wanting. The *throbbing aorta* of hypertension and of aortic regurgitation, as felt in the supra-sternal notch, is apt to be mistaken for aortic aneurysm, and it is sometimes difficult to differentiate these conditions. The throbbing aorta in Graves' disease and severe cases of anæmia may also give rise to difficulty. *Mediastinal growths* may have the same pressure symptoms as aneurysm. Pressure upon the veins is more common with growth than with aneurysm, and may only be diagnosed by the absence of the physical signs referable to the heart. There is no murmur on auscultation over the dull region, the area of dulness is usually not so limited or defined, there is usually no expansile pulsation over the tumour, and there are signs of collateral circulation. The course of mediastinal tumours rarely lasts longer than eighteen months. X-ray examination, together with a Wassermann or Kahn test, help to distinguish aortic syphilis and tumour.

Prognosis.—Treatment can do much to prolong life, and the patient may live a good many years if his occupation does not necessitate much exertion. Death may occur from rupture, exhaustion, congestive failure, or complications. Rupture usually leads to a sudden copious hæmorrhage, which terminates life ; but sometimes there is a slight leakage, which may recur each few days. With aneurysm of the *ascending* aorta rupture usually takes place into the pericardium, pulmonary artery, or superior vena cava ; with aneurysm of the *transverse* arch, into the trachea (a very frequent situation) or bronchi ; and, when the *descending* aorta is involved, the blood usually finds its way into the pleura or œsophagus. The process may be so gradual that there is no sudden onset of symptoms, such as dyspnœa, cyanosis, or bleeding, and death may not occur for some time. The severity of any case is measured by the amount of dyspnœa present and the rapidity of the evolution of symptoms. Other consequences or complications are usually due to the effects of pressure—such as collapse of the lung or a low form of pneumonia, hydrothorax, and œdema of the head and neck.

Treatment.—Give a concentrated, nutritious diet ; Tufnell's régime is

very severe.[1] Clear up any septic condition and remove carious teeth where necessary. In syphilitic cases, as there is danger of sudden death if treatment is begun with arseno-benzol, the course of N.A.B. or bismuth injections should be preceded by six weeks of full doses of potassium iodide and mercury. This therapeutic programme should be repeated at six monthly intervals until the Wassermann reaction becomes negative. If the myocardium or liver are defective, arsenic injections should never be given. Large doses of potassium iodide, starting with gr. xx. t.d.s., used to give good results before other antisyphilitic measures were known, *i.e.*, before it was realised that these aneurysms were commonly of syphilitic origin. Penicillin may prove valuable in syphilitic aortitis: as it is likely to produce Herxheimer reactions its use needs careful control. For the pain, morphia injections are used; if of anginal character, nitroglycerin. Even if the dyspnœa is very urgent, tracheotomy is not called for. If there be an external swelling, some elastic support is needed. Phenobarbitone is valuable for palpitation. For venous distension or severe dyspnœa, venesection may be performed. Surgical measures have been adopted from time to time in the treatment of superficial aneurysms, but they are not free from danger.

Other Mediastinal Tumours

§ 81. The *Symptoms and Signs* **of Mediastinal Tumour** belong to three categories—namely, (*a*) the signs of displacement of organs; (*b*) the physical signs of tumour; (*c*) the symptoms of pressure. There are also (*d*) certain symptoms special to the different kinds of tumour.

(*a*) The **displacement** of organs is sometimes the first intimation we receive. The liver is rarely displaced, but the lungs and heart are often moved to one side. The tumours may compress a main bronchus, collapse the lung, and thus draw the trachea and heart towards the same side. These organs will be displaced to the opposite side (i.) if the tumour is very large; (ii.) if it causes a secondary pleural effusion, when the signs of fluid will be present on the affected side.

(*b*) The **physical signs of tumour** may appear on the anterior or posterior aspects of the chest, and consist of: (1) Dulness on percussion, corresponding to the position of the tumour; (2) auscultatory signs, which differ somewhat with the position and nature of the tumour. If it be solid, the breath sounds will be tubular and perhaps differ on the two sides, and there may be an increased conduction of the heart sounds. If it contain fluid (such as aneurysm or, more rarely, hydatid) there will be a diminished respiratory murmur, and in the case of aneurysm a characteristic murmur (§§ 65, 80). (3) Ausculto-percussion will aid in defining the boundaries of the tumour. (4) Radiography is used for defining the nature and position of mediastinal growths, but is generally useless if a pleural effusion is also present. A lipiodol examination of the bronchial tree should be made.

(*c*) The symptoms of mediastinal tumour which are due to **pressure** on the various structures around are as follows:

(1) Dyspnœa always appears sooner or later, and may be of a type peculiar to mediastinal tumours when there is pressure upon the trachea and larger bronchi; it has a stridulous character, which resembles tubular breathing heard without the aid of the stethoscope. The breathlessness is often paroxysmal or asthmatic when there

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[1] The solids consist of well-cooked meat or fish and biscuit, and for the fluid 10 ounces of milk are permitted per day. From 12 ounces to 18 ounces of solid food may be permitted, but the fluid must not exceed 16 ounces.

is pressure upon the heart and cardiac plexuses; or it may be of a Cheyne-Stokes' nature. But the character of the dyspnœa depends upon whether it is the heart, the great vessels, the bronchi, or the nervous apparatus of the heart, lungs, or larynx, which is pressed upon by the growth of the tumour.

(2) Cough, sometimes of a laryngeal brassy character, is also present, and it is accompanied by expectoration if, as is usual, there is also bronchitis or congestion of the lungs. There may be laryngeal paralysis from pressure upon the recurrent branch of the vagus, and hoarseness, or even aphonia, may result. Hæmoptysis may occur.

(3) Cardiac and circulatory symptoms, such as palpitation, cyanosis, or a difference in the pulses of the two sides in the neck or radial arteries. There may be signs of collateral circulation, with enlarged superficial epigastric and mammary veins.

(4) Dysphagia, from pressure on the gullet, is present chiefly with posterior mediastinal growths, and when the œsophagus is the site of the primary growth.

(5) Inequality of the pupils may appear, owing to pressure on the sympathetic. Usually the pupil on the affected side is contracted from paralysis of the sympathetic, but it may be dilated during the stage of irritation. Other signs of sympathetic paralysis are malar flush, ptosis of the upper lid and enophthalmos.

(6) Pleural effusion occurs if there be pressure on the thoracic veins or if there be growth in the pleura.

(7) The inferior vena cava is rarely compressed, but cyanosis or œdema of the head, neck, and arms may occur from pressure on the superior vena cava.

(8) In suspected tumour of the superior mediastinum, it is well to remember that when the head is thrown back, the veins of the neck become distended, owing to the increased thoracic pressure producing venous obstruction. Dyspnœa is marked, and the sternum may bulge forward.

(9) Pain down the arms and in the back occurs when there is pressure on the spinal nerve trunks. A persistent dull pain is often one of the first symptoms.

(10) Pyrexia is fairly common.

(*d*) **Causes.**—There are certain symptoms which are **special to the nature and situation of the tumour.** There are seven clinical groups of tumours, in addition to aortic aneurysm.

I. Malignant Tumours, which may be primary or secondary. If, in addition to the above physical signs, the expectoration is coloured red or brown by blood, and if on paracentesis a bloody fluid is drawn off from the pleura, the presumption is strongly in favour of malignant tumour. The fluid may contain cells recognisable as malignant. Out of 520 cases of mediastinal tumour, Hare found 134 were cancerous. *Cancer* is a common mediastinal tumour, secondary to cancer of the bronchus or œsophagus. In the latter case it lies in the posterior mediastinum. Primary cancer, as of a bronchus, tends to affect secondarily the posterior mediastinal glands. *Sarcoma*, especially lymphosarcoma, may start in the mediastinal glands as a primary growth, or originate from the pleura and from the thymus remains. Primary sarcoma is most frequent in the superior mediastinum. If secondary in origin (as when the abdominal viscera or sex organs are the seat of the primary tumour), it occupies chiefly the posterior mediastinum. In primary mediastinal sarcoma enlargement of the glands above the clavicle and elsewhere may occur. When the diagnosis of the cause of pressure is obscure, glandular enlargements suggest malignant disease in the mediastinum.

II. Innocent Mediastinal Tumours, though more rare than the foregoing, are sometimes found, *e.g.*, fibroma, dermoid cyst, hydatid. Lipoma, gumma, and enchondroma, the latter growing from the sternum, are also occasionally met with.

III. Enlargement of the Mediastinal Glands.—With these there is often a dulness posteriorly in the upper half of the interscapular space, but occasionally there is dulness over the sternum. Paroxysms of coughing, "croupy" or like whooping-cough, may be present, especially at night, together with stridulous breathing from pressure upon the trachea. The *causes* of enlarged bronchial glands are:

(*a*) As described above, *malignant disease* of the glands is the most common cause.

(*b*) *Tubercle*, generally secondary to tubercle of the lung, is more common in children than in adults. The condition may be suspected when concurrent disease of the lungs is present, and symptoms such as the above arise. If the glands suppurate, sweatings and intermittent temperature become more pronounced than when the lung only is diseased. An abscess may form and open into a bronchus (compare IV below).

(*c*) *Lymphadenoma* (*Hodgkin's disease*) may start in the mediastinal glands, and is then difficult to diagnose from lymphosarcoma. See also § 572.

(*d*) *Bronchitis* and the *pneumonia* which complicates measles, influenza, and whooping-cough, are often attended by enlargement of the bronchial glands, which may occasionally be recognised in children.

(*e*) *Whooping-cough*, without bronchitis or other disease of the lungs, may give rise to swelling of the bronchial glands, although the condition may be hard to make out. Some observers consider that it is the pressure of these glands which causes the paroxysms of whooping-cough.

IV. SUPPURATIVE MEDIASTINITIS (abscess of mediastinum) is a rare condition which may affect the anterior or posterior mediastinum, or both, but more often the anterior. (i.) The most prominent symptom is pain, in the site of the inflammation, or passing down the nerves pressed upon. (ii.) Dulness, with œdema and redness, may be present over the upper part of the sternum if the disease be in the anterior mediastinum. Pulsation communicated from the aorta may be present, and lead to a diagnosis of aneurysm, but the pulsation is not expansile, and fluctuation may be felt. (iii.) Pyrexia is present, usually intermittent, with the rigors, sweats and weakness which attend all deep-seated inflammations. (iv.) The presence of leucocytosis is an important diagnostic feature. The causes of acute mediastinitis are trauma, carcinoma of the œsophagus or bronchus. The chronic form is usually due to tuberculous disease, rarely to actinomycosis. It may rupture in various directions.

V. Diffuse GUMMATOUS MEDIASTINITIS, especially affecting the mediastinum, may give all the symptoms and signs of a tumour. The Wassermann reaction and the response to antisyphilitic remedies are diagnostic.

VI. ENLARGEMENT OF THE THYMUS.—A certain degree of enlargement is normal to childhood, and may cause dulness over the manubrium. It begins to decrease after the second year of life, and should have disappeared by adult life. In status lymphaticus (§ 37) the thymus may be found enlarged even in adult life. An enlarged thymus is also frequently found in myasthenia gravis, Graves' disease, and rarely in Addison's disease, myxœdema, and rickets. Inflammation, œdema, and tubercle may affect the gland. Tumours may occur—cysts, sarcoma, rarely epithelioma and thymoma. In lymphatic leukæmia a large thymic tumour may be present.

VII. When an ENLARGED THYROID grows behind the sternum, it may give rise to symptoms of mediastinal tumour.

Diagnosis.—X-rays may give valuable help.

Prognosis.—In cases of intrathoracic tumours which are large enough to produce symptoms the prognosis is unfavourable. Moreover, all of these conditions entail much suffering to the patient. Malignant tumours are fatal in six to twelve months, depending upon the site and progress of the growth. Innocent tumours may last for a long time or may be removable surgically. Syphilitic, tuberculous, and simple inflammatory glandular enlargements may recover under treatment, but even in these no confident prognosis of recovery can be given in any case. Suppurative mediastinitis may open externally or into the pleura, and run a course of a few days or weeks only; other cases are chronic, and last for years, or lead to pulmonary gangrene and other serious complications when the pus burrows into adjoining organs. An enlarged thymus may lead to sudden death from pressure upon the trachea.

Treatment in intrathoracic tumour is almost wholly palliative. For aneurysm, see § 80. Abscesses, hydatids, dermoids, or growths connected with the sternum may be dealt with by the surgeon in some cases. X-ray and radium applications yield the best results in glandular and malignant tumours. Penicillin therapy is indicated for secondary infection.

CHAPTER V

THE PULSE AND ARTERIES

§ 82. The Meaning of " The Pulse ".—The pulse is the wave of increased pressure which passes along the arteries with each contraction of the heart. It is important to distinguish between the transmission of pressure within the arteries and the movement of the blood itself.

The clinical features to be studied in palpation of the pulse are its (1) frequency, (2) rhythm, (3) tension and character, and (4) the state of the arterial wall. These features depend on the frequency and rhythm of contraction of the left ventricle, on the strength of the contractions, and on the output at each beat. They also depend on the elasticity of the arteries and the peripheral resistance encountered by the flow of blood, especially in the arterioles and capillaries. On account of the peripheral resistance the pulse generally ceases at the arterioles, but when the arterioles are relaxed the pulse is often transmitted through the capillaries and may even appear in the veins. *Capillary pulsation* is thus to be seen in a healthy person who has taken exercise on a hot day, and it is a clinical feature of aortic regurgitation (§ 60). In the great veins near the heart a pulse is normally present. Visible *venous pulsation* is to be seen in the veins at the base of the neck in congestive heart failure, and is due to tricuspid regurgitation (§ 59). Venous pulsation is sometimes seen in the veins on the backs of the hands in Graves' disease.

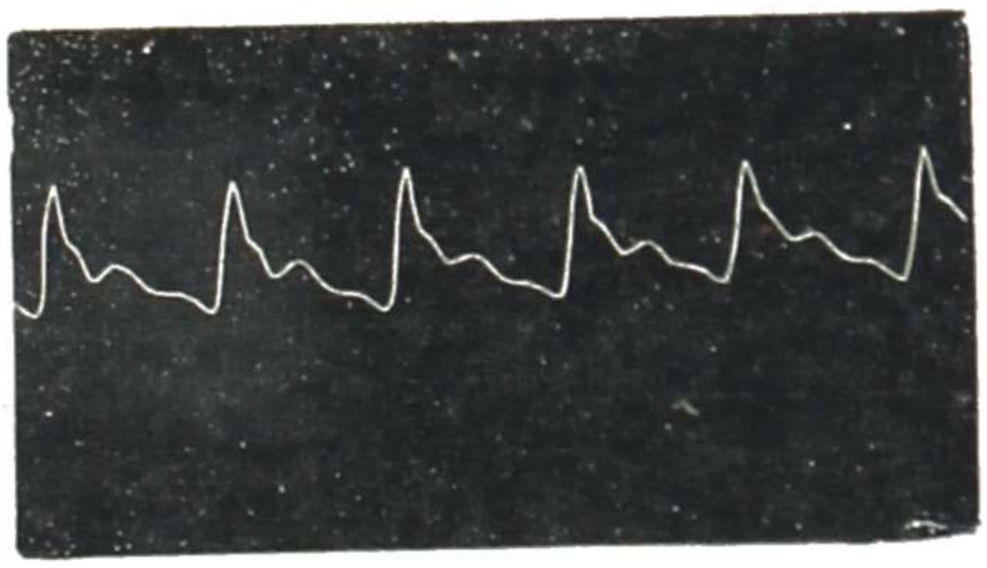
§ 83. Clinical Investigation.—Examination of the pulse provides evidence of great value both as to the state of the circulatory system and the general condition of the subject. Whatever examination is to be made, palpation of the pulse is the first observation to make. If the subject is nervous or emotionally disturbed, or has lately hurried, the observation is repeated later when the pulse has settled. For accurate record the pulse is always taken under similar conditions as to posture, time of day, relation to meals, etc. The radial pulse is generally chosen, since it is easily accessible and lies against bone (the radius). If it is aberrant, the opposite radial artery is palpated. Whenever disease of the cardiovascular system is suspected, both radial pulses should be felt simultaneously and carefully compared. The pulse can also be felt in other arteries near the surface, such as the temporal, facial, dorsalis pedis and posterior tibial arteries, and in the abdominal aorta. To feel the pulse three fingers are placed over the course of the radial artery, the index finger nearest the heart. Allowance is made for the thickness of the subcutaneous tissues.

The special features of the pulse may be brought out more clearly by holding the forearm up when palpating the pulse. The main points to note have been mentioned in § 41. After noting the frequency, rhythm

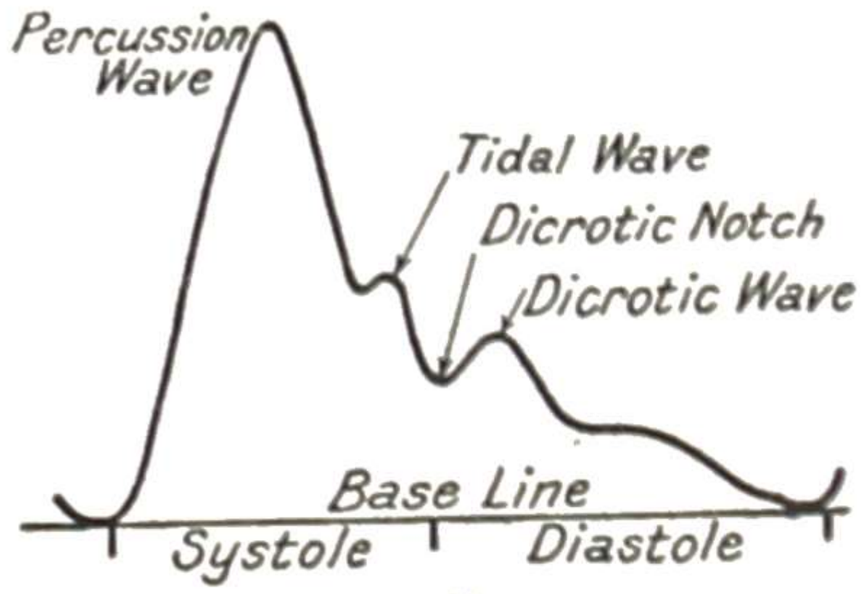
and character of the pulse (the term character refers to the nature of the pulse wave, its rise, summit and fall), the tension is estimated by the amount of pressure exercised by the forefinger in order to obliterate the pulse wave and prevent it reaching the middle finger. In case there is a return pulse wave through the palmar arches it may be necessary at the same time to obliterate the pulse with the third finger. Finally, after obliterating the pulse by pressure with all three fingers, the wall of the artery is felt by rolling the empty vessel under them.

The SPHYGMOGRAPH is an instrument employed to obtain a record on smoked paper of the characters of the pulse. With Dudgeon's instrument strapped to the wrist, a system of levers magnifies the pulse wave and records a tracing.

The SPHYGMOGRAM or sphygmographic tracing is useful as a graphic record of the pulse, but its readings can never be quite accurate. Fig. 40*a* is a normal pulse tracing. Fig. 40*b* shows the principal named parts of which it consists. The first or *percussion wave* is caused by the arrival of the pulse in the artery under the sphygmograph. Its form is determined by the output per beat of the ventricle, the rate at which the blood is ejected from the ventricle into the aorta, the peripheral



(*a*)



(*b*)

FIG. 40.

(*a*)—NORMAL PULSE TRACING, taken with Dudgeon's Sphygmograph. (*b*)—NORMAL PULSE TRACING magnified, with the names of the principal parts. The dicrotic (or aortic) notch indicates the closure of the aortic valves, and therefore the termination of the ventricular systole and the commencement of the ventricular diastole. The diastolic line is that part of the tracing from the dicrotic notch to the next percussion wave.

resistance and the extensibility of the arterial walls. The percussion wave is abrupt and the pulse is sudden when the diastolic pressure is low and the ventricle has little resistance to overcome in discharging its contents. The *tidal wave* represents, according to Crighton Bramwell, the summation of the outgoing percussion wave, and waves reflected back from the periphery of the arterial field. It is prominent in aortic stenosis and hypertension, for in both conditions ventricular pressure is well maintained throughout systole, and the summation of the percussion and the reflected waves may cause the tidal wave to be higher than the percussion wave. In aortic regurgitation, on the other hand, the ventricle ejects most of its contents during the early part of systole, because of the lower diastolic pressure. During the latter part of systole the ventricular output is much reduced, the falling pressure tends to neutralise the wave reflected from the periphery. Hence the tidal wave is inconspicuous. The *dicrotic wave* indicates the rebound of blood against the closed aortic valve. It is most marked with a forcibly beating heart, a low peripheral resistance, and an elastic arterial wall.

Many instruments have been devised for the **measurement of the blood pressure.** For practical purposes only the aneroid and the mercurial manometers need be considered. Of the former type the Tycos instrument is the best. It is portable and accurate, but it should be

checked against a mercurial manometer from time to time. The mercurial manometer is the more reliable, and of these the Riva-Rocci *sphygmomanometer* is the type on which all are modelled. The column of mercury should be open to direct atmospheric pressure, and unspillable. The armlet is at least 5 inches (12 cms.) wide, and tapers after the first 18 inches. The cuff is better made of fabric than leather, because fabric is more easily adjusted. The Baumanometer type of sphygmomanometer is very good. The armlet is wrapped round the patient's upper arm well above the bend of the elbow. The second turn fixes the upper limit of the armlet, the third turn fixes its lower limit so that, when the rubber bag contained in the armlet is inflated, an even pressure over the whole width of the armlet will be exerted. The sphygmomanometer is placed about the level of the heart. The patient's arm is placed extended on the bed or couch. The position of the brachial artery at the bend of the elbow is located by feeling the pulse in it, and the stethoscope is gently placed over it. The armlet is then inflated until the vessel is so compressed that no sound is heard. The pressure is then evenly released by turning the screw-valve attached to the pump. As the column of mercury falls, four different tones, or phases of sound, will be heard before there is complete silence. (1) The first phase consists of short sharp sounds, and the mercury level at which the first sound is heard indicates the SYSTOLIC PRESSURE. (2) As the column of mercury continues to fall the sound acquires the character of a murmur. This phase is often of short duration, and is on occasion absent. (3) It is succeeded in the third phase by loud and clear sounds; this is the longest and most distinct phase. (4) The clear sounds suddenly become dull and distant; the level of the mercury column at which this occurs is the DIASTOLIC PRESSURE.

The average reading in 150 young soldiers aged 23 to 27, in the fourteenth week of training, was—First phase, 135–125; second phase, 125–104; third phase, 104–80; fourth phase, beginning at 80. The average systolic pressure in this group was, therefore, 135 mm. Hg., and the diastolic 80 mm. Hg. In practice the blood pressure is always recorded in even numbers. Both systolic and diastolic readings in this group are somewhat above the average normal figure (say 10 mm. Hg.). The blood pressure in adolescence and early adult life is often somewhat raised. It returns to normal with maturity.

Having recorded the systolic and diastolic readings by the auditory method as described above, the systolic level is checked by taking the blood pressure again by the tactile method. With a finger on the radial pulse the pressure in the mercury column is quickly raised to 10 or 15 mm. Hg. above the systolic level determined by auscultation. At this level no pulsation is felt in the radial artery. The column of mercury is immediately allowed to fall gently. The level at which the first beat comes through to the radial artery is the systolic pressure. This reading must agree with the reading obtained by auscultation within 10 mm. Hg. The systolic reading as taken by auscultation is often raised above normal

in a nervous subject, and is then usually higher than the tactile reading: occasionally the auscultatory reading is lower than the tactile reading. When a succession of readings is taken the two usually approximate, but when there is still a discrepancy between them the final systolic level should be that obtained when a series of readings taken by the tactile method has given a constant figure.

The PULSE PRESSURE is the difference between the systolic and the diastolic pressure, *e.g.*, with 120 mm. Hg. systolic and 80 mm. Hg. diastolic, the pulse pressure = 40 mm. The DIASTOLIC PRESSURE may be regarded as representing the resistance to be overcome by the heart when the aortic valves are opened. The SYSTOLIC PRESSURE indicates the maximum work of the heart. The diastolic is the more constant, more significant and less liable to alter with nervous influence. The systolic reading normally is as three to two of the diastolic; *e.g.*, S. 120 to D. 80.

This is a rough rule only, the diastolic being more often about 74 mm. Hg. when the systolic pressure is 120. The systolic pressure tends to increase with advancing years. The saying that the systolic pressure should not exceed 100 plus the age is less true than the average generalisation. Old people often have nearly normal systolic pressures and many live to a good age with a relatively high systolic pressure. In general terms, a systolic pressure above 170 mm. Hg. or a diastolic above 90 mm. Hg. is pathological. Pressures below these levels may, however, be too high for the particular individual, and of clinical importance.

The following figures give the average found in several thousand actual readings. The range of the normal limit is not more than fifteen millimetres above or below. Age 21–30: 124 (*Systolic*), 82 (*Diastolic*); 31–40: 126 and 84; 41–50: 130 and 86; 51–60: 134 and 90.

§ 84. Rapid Pulse (Tachycardia).—The rapidity or frequency of the heart beat varies considerably within the range of normal health due to variations in the rate of impulse production in the normal pace-maker (the sino-auricular node). This is called sinus tachycardia. The normal pulse rate is about 70 per minute. A few people have pulse rates under 60 or over 80, but such must not be accepted as within normal limits without careful consideration. Rarely a pulse rate of 50 or just under, or of 90 or just over, is compatible with perfect *health*. The pulse tends to be faster in the female than in the male. It varies at different ages. In the fœtus and new-born infant its average rate is 140 per minute; under 1 year, 120; under 3 years, 100; from 7 to 14, 90; from 14 to 21, 80; from 21 to 65, 70; in old age, 80 per minute. The pulse is *normally* more rapid during the menstrual period and menopause, in the evenings and after meals. After a severe illness and in asthenic states the pulse more easily becomes rapid. When the tachycardia is due to *simple causes*, not the result of myocardial changes, the number of the beats falls ten to twenty per minute when the patient alters his position from standing to lying. Exercise, emotion, meals, fever and sleep modify the rate, and the electrocardiogram is normal. These features differ-

entiate simple tachycardia from Paroxysmal Tachycardia, in which the pulse-rate is unaffected by posture, exercise, etc.

The **pathological** causes of sinus tachycardia are numerous. (1) *Pyrexia* is the most common. (2) Early *tuberculosis* should always be borne in mind. Any other *bacterial* infection is a common cause, *e.g.*, streptococcal and pneumococcal infections, whether generalised or focal. Pulse frequency is increased in the acute specific fevers, especially in scarlet fever. (3) Of *endogenous toxæmias*; (i.) Graves' disease is the most common; close observation for larval forms of this disease should be made in any obscure case of tachycardia; (ii.) uræmia; (iii.) malignant disease, especially when undergoing degenerative changes; (iv.) all blood diseases, including moderate and severe anæmia. (4) *Exogenous toxæmia* includes a large variety of drugs and poisons, such as tobacco, alcohol, tea, coffee, thyroid extract, belladonna and atropine. (5) *Nervous states*, including ordinary emotional disturbance, often of trivial kind; neurasthenia, anxiety neurosis and neuro-circulatory asthenia are common causes in which the border-line between physiological and pathological disturbance is hard to define. (6) Most forms of *heart disease*, toxic, inflammatory or degenerative, and whether acute or chronic. Increased pulse frequency is an important sign of heart failure. Forms of tachycardia in which the stimulus for contraction arises from an abnormal focus are described in § 66.

§ 85. Slow Pulse (Bradycardia). A slow pulse should be verified by counting the frequency of heart beats on listening to the apex. A frequency of 60 per minute or under requires careful consideration, as it may be the first indication of serious organic disease such as heart block or cerebral tumour. Bradycardia may be a personal idiosyncrasy. It is compatible with perfect *health*. It is sometimes familial. A slow heart rate is an advantage because it allows of an increased cardiac output without increase of heart rate. In a group of 28 Marathon runners examined by Bramwell and Ellis the average heart rate was 58, and 4 of these had heart rates under 50, while only 9 had heart rates over 60. In healthy subjects bradycardia is due to a slow rate of impulse production in the sino-auricular node. It is known as *sinus bradycardia*.

Pathologically, sinus bradycardia may be (1) the result of *reflex nervous effects*, via the vagus nerve; *e.g.*, in gastric disorders. (2) Bradycardia is one of the cardinal features of myxœdema, and other states of *lowered metabolism*, such as exposure to cold, starvation, anorexia nervosa, cachexia and melancholia except in the terminal stages of these conditions. It is associated with low B.M.R. (3) *Toxic conditions*: (*a*) endogenous, such as jaundice, diabetes and uræmia, and (*b*) exogenous, such as may be due to digitalis, strophanthus and opium. At first tobacco may slow the heart. (4) Bradycardia is not uncommon in *convalescence* from acute infection, *e.g.*, influenza, and in exhaustion states. A pulse rate low in proportion to the fever is found with infections by the typhoid and salmonella groups, B. coli, and sometimes staphylococcal infections

and influenza. (5) *Increased intracranial pressure* of whatever etiology. In meningitis a slow and irregular pulse is of diagnostic importance. (6) In *heart disease* a slow pulse may be found in aortic stenosis, fatty heart and senile heart. It is the rule, however, that the heart rate increases gradually with advancing years. Bradycardia in heart disease is generally due to heart block (§ 69). *Temporary slowing* of the pulse rate occurs with pressure on the vagus in the neck, and characteristically in an ordinary fainting attack (vaso-vagal slowing).

§ 86. The **Irregular Pulse,** apart from *Sinus arrhythmia*, indicates an abnormal action of the heart. Pulse irregularities are dealt with in § 41, and the heart conditions responsible for them in § 63. A few additional points may be noted here. Sinus arrhythmia (§ 65), which is probably due to rhythmic alterations in vagal tone, is most common in young persons and is generally regarded as physiological. Bramwell has observed the association of pronounced sinus arrhythmia with a liability to simple fainting attacks, and he attributes both to an over-active vagal mechanism. *Premature beats* (§ 64), unless very frequent, can be recognised by the fact of a regular pulse interrupted by an occasional irregularity recurring rarely, or say once in every 5 to 10 beats. The diagnosis is made by auscultation of the heart, and confirmed by electrocardiogram, especially if intrinsic disease of the heart is suspected. *Auricular fibrillation* (§ 68) is recognised by the irregularity in which no two beats or intervals are alike: exercise considerably exaggerates this irregularity and also the rate of the heart, whereas premature beats usually disappear with increased frequency of heart beat.

In auricular fibrillation the apex and pulse rates must be counted at the same time. The pulse rate is generally less than the apex beat—and the difference between the two rates (the "pulse deficit") is represented thus, A/p. = 124/92. With recovery the pulse deficit becomes less, and it disappears when every ventricular beat reaches the radial artery.

Pulsus alternans (§ 71) denotes alternate weaker and stronger ventricular contractions. It is occasionally diagnosed by recognising an alternate weaker and stronger pulse, but it is readily diagnosed when taking the systolic pressure by the auditory method. At the top level at which the sounds are heard only alternate pulse waves are audible, for the weaker heart beats do not produce a pulse wave strong enough to overcome the resistance of the arm band. Pulsus alternans indicates exhaustion of the heart muscle and is of grave prognostic significance.

In *pulsus paradoxus* there is complete, or almost complete, disappearance of the pulse during inspiration. It is due to either (1) an increase of the "negative" intrathoracic pressure which normally takes place at the end of inspiration, or (2) to extreme weakness in the left ventricle, or to both. It can be produced in even healthy persons at the end of inspiration by so contriving that the negative intrathoracic pressure can be *suddenly* increased. It is met with in intrathoracic tumours, pleural effusion, mediastinitis, and adherent pericardium.

In the *anacrotic pulse* the tidal wave is higher than the percussion wave. It occurs in aortic stenosis and hypertension. It is due to the ventricle being forced to empty its contents more slowly, and so throughout the ejection phase of systole the rate of discharge of blood into the aorta is much more uniform. The pressure being thus maintained when the reflected wave returns from the periphery, it increases the height of the tidal wave. It is only recognisable by instrumental methods.

The *dicrotic pulse* is due to a marked dicrotic wave. It is said to simulate coupled beats, but once felt it is quite distinctive. It is common in asthenic states with a full soft pulse, as in typhoid fever.

§ 87. By **Blood Pressure** is meant the tension in the arterial system. In general terms, it refers to the pressure in the brachial artery. The pressure of blood in the veins is referred to as venous pressure. The blood pressure depends on two main factors—(1) the peripheral resistance; (2) the output of the heart: i.e. the force and frequency of the heart. Any gross variation in blood pressure is due to alteration in one or more of these factors. The determining factor is peripheral resistance.

High Blood Pressure (Syn., Hyperpiesis,[1] Hypertension) is due to many different causes. It is a symptom and not a disease *sui generis*. For the methods of measuring the blood pressure, see § 83. In a healthy adult the blood pressure is fairly constant, but the limits of normal variation are wide, namely—systolic, 100–146 mm., and diastolic, 64–84 mm. In older people the systolic pressure may be as high as 160 mm., but persistent pressures above 170 mm. systolic and 90 mm. diastolic indicate the presence of arterial disease and are certainly pathological.

Temporary hypertension (Symptomatic hypertension), may be due to emotional disturbance and to emotional or physical fatigue. It may occur during convalescence from acute infection and in certain cases of suprarenal tumour. The pressures are rarely higher than 170 mm. systolic and 90 mm. diastolic.

Persistent hypertension. (1) *With Renal Disease.* In any case of persistent hypertension the first object is to determine the presence or absence of kidney disease. Persistent hypertension is common in acute nephritis, in the several varieties of chronic nephritis, in polycystic disease of the kidneys and in toxæmia of pregnancy.

Pyelonephritis, hydronephrosis, renal calculus, renal tuberculosis, hypernephroma, fibrosis of the kidneys, and impaired blood supply from occlusion of the renal artery (*e.g.*, by severe arterio-sclerosis or aneurysm) also can cause renal hypertension. When unilateral, removal of the diseased kidney may allow return of the blood pressure to normal, provided the other kidney is healthy. Interference with emptying of the bladder (*e.g.*, by an enlarged prostate) may produce persistent hypertension.

(2) *Without Renal Disease.* Apart from the fact that heredity is an important factor, the etiology is ill-defined. (i) The patients may be obese, plethoric and jovial-spirited, or thin, pale, anxious and liable to depression. Persistent hypertension is uncommon under 35 years: particularly in younger patients, unilateral renal disease should be excluded by intravenous pyelography. (ii.) Associated with obesity, especially when due to over-eating or excessive drinking. (iii.) Modern city life, worry, anxiety and prolonged mental work, especially when combined with lack of regular exercise. (iv.) Endocrine disturbances, as at the climacteric or after removal of both ovaries. Hypertension may be associ-

[1] *Hyperpiesis* is a state of hypertension, whatever the cause, and may be temporary (as with emotion) or persistent. *Hyperpiesia* is a clinical condition associated with arterial disease (diffuse hyperplastic sclerosis, § 94).

ated with Graves' disease, or with myxœdema. It is part of the syndrome of pituitary basophilism and occurs with adenomata of the adrenal cortex. The relation of Graves' disease to hypertension is probably due to a constitutional predisposition rather than direct cause and effect. (v.) There is an ill-defined relationship with bacterial toxæmia. Chronic urinary infections may cause persistent hypertension : but syphilis, focal sepsis and other chronic infections are not causal. Diabetes mellitus and hypertension often occur together, and after the age of 40 may be due to arterio-sclerosis of the pancreatic vessels (Moschcowitz). Gout and osteo-arthritis are often associated with hypertension. (vi.) The only exogenous poisons are chronic alcoholism and lead. (vii.) For polycythæmia and its relation to hypertension, see § 31.

When a definite cause for hypertension exists, such as a chromaffin tumour of the adrenal or lead poisoning, the raised blood pressure is referred to as symptomatic hypertension, for removal may lead to a relatively normal blood pressure. When the etiological factor is concomitant or contributory, *e.g.*, obesity, or in all cases where no definite factor is found, other than heredity, the condition is called essential hypertension (hyperpiesia).

For the *symptoms* and *treatment* of essential hypertension see § 94.

§ **88. Low Blood Pressure** (Syn. Hypotension) in an adult is indicated by a systolic blood pressure persistently below 90–100 mm.. To the examining finger the pulse wave comes up rapidly, declines rapidly, and is easily obliterated. It may be suspected if the pulse, when counted with the patient erect, is rapid, and the rate falls 30 or 40 beats when the patient lies down. *Symptoms* may be absent. When present, headache, giddiness and sometimes syncope may be complained of, especially when rising from a recumbent posture : depression, lassitude and undue fatigue are usual. In cases of "postural hypotension," the systolic blood pressure is 20–30 mm. less when the patient is standing than when lying down.

Etiology.—In *health* a persistent state of low blood pressure is rarely a hereditary condition. It may be found also after meals, a warm bath or moist heat. In *disease*, the chief causes are (*a*) Cardiac disease, especially weakness of the left ventricle, such as occurs in coronary thrombosis and toxic myocarditis with diphtheria ; (*b*) General conditions : (i.) suprarenal atrophy or tuberculosis (Addison's disease) ; (ii) pulmonary tuberculosis ; (iii) cachexia, and deficient food ; (iv) shock, collapse, hæmorrhage or dehydration ; (v.) exhaustion due to mental or physical overstrain ; or following asthenic types of fever, especially typhoid and influenza ; (vi.) occasionally with certain types of advanced renal disease and with senile arterio-sclerosis.

The *treatment* of low blood pressure depends upon the cause, but special attention is given to the myocardium. The diet should be nourishing, easily digestible, and rich in vitamins, especially vitamin B. The avoidance of standing about for long, of free purgation, of *hot* baths, and of

mental and physical exhaustion, are all important. Give graduated exercise and an abdominal belt to support the splanchnic area. Nikethamide B.P. (coramine), cod-liver oil, and small doses of strychnine may be helpful. Vaso-constrictors (including ephedrine hydrochloride) may be used, provided the myocardium is not seriously damaged. Stimulants must be used with caution. See also Addison's disease (§ 560). Collapse is dealt with in § 35 and § 239. In severe cases rest in bed is advised.

§ 89. The Pulse in Relation to Prognosis and Treatment of Disease. Examination of the pulse affords valuable information both as to the general condition of the patient and the state of the cardio-vascular system. Indeed there is so much to be learnt from palpation of the pulse by the experienced finger that it should always be the first step in the general examination of a patient. The pulse frequency in *febrile diseases* should be charted four-hourly, so that it may be read in conjunction with the temperature and respiration rate. In an adult the pulse frequency increases 8 to 10 beats per minute for each degree rise of temperature. A pulse frequency increased out of proportion to the rise of temperature may be an indication of a *toxic myocarditis*, and a pulse rate over 130 per minute in *pneumonia* is evidence of severe toxæmia. In a *child*, the increase of pulse frequency with each degree rise of temperature is greater, namely, 12 to 15 beats per minute.

Slowing of the *pulse frequency* in relation to *fever* may be an indication of heart block. A sudden drop of temperature, pulse and respiration rates together takes place at the crisis in pneumonia; but a fall of temperature without a fall in pulse rate, or perhaps even a slight increase of pulse rate, is evidence of a complication. In *abdominal conditions* the pulse rate may decide a diagnosis between inflammation (rapid pulse) and colic (slow pulse), and fall in temperature with an increase of pulse frequency occurs with intestinal hæmorrhage, in perforation of the bowel, and with profuse diarrhœa complicating typhoid fever. The pulse rate may be of outstanding importance in the diagnosis and treatment of appendicitis; in a doubtful case, when the patient looks ill, has indefinite abdominal discomfort but no localised pain, and a soft abdomen, an increasing pulse rate observed half-hourly may be the deciding factor for immediate operation. The pulse-temperature ratio in abdominal disease is considered in § 239. Again, in a patient recovering from a severe *hæmatemesis* the temperature tends to oscillate about normal with an occasional rise to 99° or 99·4°; the pulse rate may be 100–110, gradually falling to 80. With recovery the temperature gradually becomes subnormal at a steady level, and the pulse drops to 70–80. A rise in pulse rate or an irregularity of the pulse curve may accompany further hæmorrhage, and thus provide an indication for more cautious treatment, or if the hæmoglobin is at the borderline of 30–40 per cent. it may determine treatment by blood transfusion. The pulse rate in *afebrile toxic states* is to some extent a measure of the degree of toxæmia, as in alcoholic poisoning, especially delirium tremens. In *Graves' disease* the pulse rate and

the height of the pulse pressure with the patient at rest in bed provide a fair index of the basal metabolic rate and the toxæmia. (See Tachycardia, § 84, for other toxæmias to which these observations also apply.) Pulse frequency to the extent that it is a measure of the *degree of toxæmia* thus provides important information as to prognosis and treatment. Rapidly rising pulse rate is a common terminal event in both febrile and afebrile diseases. A transient increased frequency is some measure of *emotional* reaction. The pulse rate in response to *exercise* and the time taken for its return to normal tells us something of cardiac efficiency. Variations in pulse volume are also of great importance. A full bounding pulse is characteristic of an acute febrile illness and asthenic state. In contrast is the small thready pulse which is felt in all states of shock, both medical and surgical. The pulse in relation to heart disease is discussed in §§ 41, 45.

ARTERIAL DISEASE

Part A.

§ 90. Symptomatology. The symptoms of arterial disease *per se* depend in the first place on changes in the function and structure of the blood vessels, and in the second place on the effects of these changes on the activity of the organs which the affected blood vessels supply. The symptoms vary according to whether the vascular affection is general or local, and necessarily according to the part which is chiefly affected. The CARDINAL LOCAL symptom of active arterial disease is **pain.** The outstanding example of this is the pain due to ischæmia of the myocardium; this may be due to structural disease of the coronary arteries, as in coronary thrombosis complicating arterio-sclerosis of the coronary vessels, or due to spasm of the coronary vessels, whether or not associated with structural disease. The pain of active arterial disease may be felt as a rheumatic pain in the limbs or trunk. Arterial disease in the cerebral vessels can cause neuralgic headache, vertigo or tinnitus. The pain of intermittent claudication is due to failure of the blood supply to meet the extra demands of muscular activity. It may occur as a result of healed vascular disease, and does not necessarily imply an active phase of it. Although often not possible of accurate diagnosis, arterio-sclerotic disease may on occasion be the cause of *dyspeptic symptoms*, especially in the form of flatulence or colic. The GENERAL symptoms depend upon the variety and location of the arterial disease. See Part C.

Part B.

§ 91. The Physical Examination of the arteries has been described in the preceding pages which deal with the pulse. The condition of the arteries can be gauged by inspection of the retinal vessels, by palpation of the superficial arteries, by blood pressure observation and by X-ray examination which will show the size of the aorta, and calcification, whether

present in the aorta or in vessels of smaller calibre such as the limb arteries. Finally, examination of the urine may provide an indication of changes in the capillaries of the glomeruli. In fact, in order to arrive at a diagnosis, in certain cases a complete examination of the cardio-vascular and renal systems is necessary.

Physical Signs. (i.) Hæmorrhage is the cardinal sign of the active phase of arterial disease. The common sites are from the nose (epistaxis), uterus (menorrhagia), in the eyes (retinal hæmorrhages or hæmorrhage into the vitreous: conjunctival hæmorrhage has not this significance), and kidneys (microscopic and sometimes macroscopic hæmaturia). Hæmorrhages from the lungs (hæmoptysis), from the stomach (hæmatemesis), and bowels (melæna), are occasionally seen. Such hæmorrhages may be the forerunners of more serious events. Thus epistaxis may precede a cerebral accident, such as hæmorrhage or thrombosis, and is sometimes the first sign of incipient psychosis due to cerebral arterio-sclerosis. (ii.) Visible or palpable thickening or tortuosity of visible (retinal) vessels or palpable vessels, such as the temporal, radial, brachial, and sometimes the carotid and other arteries, indicates arterio-sclerosis, as commonly understood. This thickening of a palpable artery may be due to hypertrophy of the media in response to hypertension. In such a case on post-mortem examination the thickened artery may be found healthy except for the hypertrophy of its walls, which may involve the intima as well as the media. In other cases, such as the senile form of arterio-sclerosis, the thickening and tortuosity of the vessel felt by palpation may be due to degenerative and hyperplastic changes. The thickening may feel irregular, as in the Mönckeberg type of arterio-sclerosis, where there is much calcification of the media, with or without minimal changes in the intima. (iii.) The loss of elasticity in the arterial system in arterio-sclerosis is recognised by an increase of pulse pressure. (iv.) A rise in the diastolic pressure, as occurs in persistent hypertension, also causes a loss of elasticity and increase in pulse pressure. (v.) Accentuation of the aortic second sound is caused by sclerosis of the ascending aorta. With considerable clinical experience this may be distinguished from the accentuation of the aortic second sound due to a persistent raised blood pressure (persistent hypertension), because in the former the second sound often has a quality which is scrunching or clanging, while in the latter the sound is sharp and clear. (vi.) Calcification in the vessels can be demonstrated by radiography. (vii.) Obstruction of the arteries occurs with (*a*) thrombosis due to arterial sclerosis, or syphilitic endarteritis (especially of the cerebral vessels), and (*b*) embolism. Thrombosis less often occurs in a healthy vessel. When blocking has occurred, the arterial pulse is reduced or abolished beyond the obstruction (see §§ 577, 579). Minor degrees of obstruction and complete obstruction may be demonstrated by arteriography.

The arteries are commonly held to be more prone to disease than are the veins, but it is not as yet known in what proportion of cases major

vascular accidents, such as hæmorrhage or thrombosis, are located in the arteries, capillaries or veins. In those areas that are more available for observation, such as the vessels of the nasal septum and retina, capillary and venous accidents are certainly of great importance.

Part C.

The DISEASES OF THE ARTERIES which admit of clinical recognition are as follows:

I. Arterio-sclerosis, of which the following pathological types are recognised: *a*. Nodular arterio-sclerosis, including its terminal phase atheroma; *b*. Senile arterio-sclerosis, including the Mönckeberg type of arterio-sclerosis; *c*. Arterio-capillary fibrosis (Gull and Sutton), otherwise called Diffuse hyperplastic sclerosis (Jores). II. Thrombo-angiitis obliterans (see § 580). III. Periarteritis nodosa. IV. Chronic and acute endarteritis. V. Aneurysmal dilatation. VI. Complications, such as embolism and thrombosis. VII. Functional disease of the arteries.

§ **92.** I. *a*. **Nodular Arterio-sclerosis,** including **Atheroma.** This word is derived from the Greek word *ἀθήρη*, which means porridge. It is the name applied to the terminal stage of nodular arterio-sclerosis when a patch of intimal thickening has undergone degeneration and necrosis (Andrewes).

Symptoms are generally absent. Atheroma and the senile type of arterio-sclerosis are commonly associated: the presence of atheroma *per se* cannot be clinically recognised and gives no symptoms (unless a dissecting aneurysm develops).

Atheromatous localised or patchy thickening of the tunica intima occurs for the most part in persons past middle age, and is unaccompanied, as a rule, by any obvious symptoms during life. It starts as a localised hyperplasia in the deeper (external) layer of the tunica intima, or secondary to localised medial degeneration (Virchow). The localised hyperplasia may remain as such, when it appears post-mortem as a raised circular or oval patch of pearly white or pale grey tissue. Or it may undergo fatty degeneration (when it appears yellowish in colour), or when it undergoes necrosis and caseation with or without calcareous degeneration, it becomes atheroma. When the necrotic process involves the superficial layers of the intima and the endothelial lining of the vessel wall an atheromatous ulcer is formed. In advanced cases the middle coat or media is always involved. The disease is more or less widespread, but generally commences and predominates in the larger vessels, *i.e.*, in the aorta and its branches. Although nodular arterio-sclerosis, when marked, tends to be widespread, when detected in the peripheral vessels, such as the radial or temporal, it cannot be concluded that it is also present in the visceral arteries, such as the arteries of the brain, heart and abdominal viscera. This statement also applies to the distribution of senile arterio-sclerosis.

§ **93.** I. *b*. **Senile Arterio-sclerosis.** This is a diffuse form of arterial disease chiefly affecting the larger arteries and distributing trunks. It is characterised by widespread medial degeneration, as shown by fibrosis, fatty degeneration and more or less calcification. It is complicated by localised or at least uneven changes of the intima. These changes include

thickening of the subendothelial tissues with intimal hyperplasia, increase of the internal elastic lamina, which may or may not undergo fatty degeneration. It is commonly complicated by nodular arterio-sclerosis. The extreme form of this type of arterio-sclerosis is Mönckeberg's sclerosis.

The *symptoms* form a wide and varied group: (i.) Arterio-sclerotic arteries do not respond so readily to the extra demands made on them by organs in a state of increased activity. As a result, in muscular activity, for instance, anoxæmia may develop. Hence the muscular cramps in the legs on exertion (intermittent claudication) and angina of effort. (ii.) Diseased arteries are more liable to functional disturbance; therefore transient paresis and other cerebral disturbances are common in cerebral arterio-sclerosis. (iii.) The narrowing of the lumen in arterio-sclerotic vessels may lead to impaired vitality and degeneration in the organs they supply; hence the various forms of senile psychosis due to cerebral arterio-sclerosis. (iv.) Other examples are seen in myocardial degeneration, due to disease of the coronary arteries; in diabetes mellitus and in the varied forms of dyspepsia (including flatulence, colic and constipation) due to arterio-sclerosis of the splanchnic vessels. (v.) Arterio-sclerosis contributes to the loss of weight, fatigueability, debility, and many other signs of old age. (vi.) Lastly, arterio-sclerosis may be the determining cause of thrombosis, and thus lead to gangrene, myocardial infarction, cerebral softening and other well-known clinical syndromes.

The *physical signs* due to vascular disease *per se* have already been referred to: (i.) the thickening of the palpable arteries, (ii.) the irregularity of calibre of the retinal vessels with deviation of the veins and obstruction to the blood flow in them at the arterio-venous crossings. (iii.) Increased pulse pressure, generally associated with a rise in systolic pressure, and (iv.) an alteration in the second aortic sound.

Etiology.—(i.) *Heredity.* As Osler said, certain families seem to "inherit bad tubing." A history of arterial degeneration causing "strokes," angina, high blood pressure or "sudden death" is common; and sometimes "anticipation" occurs, so that subsequent generations show the essential changes at earlier ages. (ii.) *Age.* Arterio-sclerosis is rare before 40, apart from kidney disease. Age is an important etiological factor, as indeed the term "senile arterio-sclerosis" indicates. At the same time the changes, described under the heading of senile arterio-sclerosis, are not due only to age, but to other factors, constitutional, dietetic, metabolic, endocrine, infective or toxic, not as yet clearly identified. Senility alone is responsible chiefly for a diffuse thickening of the intima, with increased connective tissue formation in both the intima and the media, and the deposition of calcium salts. There is little fatty or necrotic change. Age is chiefly of importance in providing a longer opportunity for the causes of arterio-sclerosis, whatever they may be, to have their effect. (iii.) *Constitution.* In general terms there is an association between cardio-vascular sclerosis and diabetes mellitus, osteo-arthritis and gout. (iv.) The Mönckeberg type of arterio-sclerosis is chiefly found

in patients suffering from a serious disease such as cancer, chronic phthisis, advanced morbus cordis, and diabetes mellitus. It may be a different lesion from other forms of arterio-sclerosis, and possibly due to an error in calcium metabolism (Moschcowitz). (v.) Hypertension of sufficient degree and duration is an established cause of arterio-sclerosis. Therefore the etiological factors concerned in the causation of persistent hypertension are potential causes of arterio-sclerosis (see § 87).

Diagnosis.—The signs and symptoms of vascular disease already described in detail provide the diagnosis of arterio-sclerosis, but whether the patient who has arterio-sclerosis is or is not suffering from it, will depend on the signs and symptoms of activity of the disease. This is to be judged in the first place by the presence or absence of hæmorrhages and pain, and in the second place on an evaluation of symptoms referable to organs other than blood vessels, and the opinion as to what extent such symptoms are determined by a disorder in structure or function of the vessels which supply them.

Prognosis.—The question of prognosis depends firstly on the activity or quiescence of the arterial disease ; and secondly on the degree of cardiac and renal involvement. If there are signs of heart failure the prognosis is necessarily guarded, and with the development of retinitis and severe kidney involvement it becomes grave. The thickening of the artery, and indeed many of the structural changes which characterise arterio-sclerosis, are largely the result of recovery and repair. The structural pathology of arterio-sclerosis is to some extent comparable with that of fibroid phthisis, in which type of tuberculosis the lesion may be active, but the process of repair keeps pace with it or dominates it. If it is realised that the process of repair may keep pace with the smouldering vascular lesion, and perhaps overtake it, it will be readily understood how often patients have arterio-sclerosis without at any time suffering from it.

§ **94.** I. *c.* **Diffuse Hyperplastic Sclerosis** (arteriolo-sclerosis : arterio-capillary fibrosis) is the structural equivalent of persistent hypertension.

Functional Pathology.—Increased peripheral resistance is the cause of persistent hypertension. It is determined by narrowing of the very small arteries and arterioles, and is accompanied by an increased force of the heart beat. This means increased work for the heart and leads to left ventricular hypertrophy. The blood flow to the periphery is thus maintained in spite of the contraction of the vascular bed. Neither increased output of the heart, nor an increased flow of blood, nor increased viscosity, contribute materially to persistent hypertension. Goldblatt, Page and others have caused persistent hypertension in dogs either by reducing the blood flow to one or both kidneys or by the production of an aseptic capsulitis, thus causing a reduction of arterial pulsation in the kidney, and renal ischæmia. According to Page, the secretion of a pressor substance from the kidney depends rather on reduction of intra-renal pulse pressure than on renal ischæmia. As a result of this change in renal circulation the kidney secretes a substance known as renin, which reacts on a protein-like substance (renin-activator) in the blood plasma to form a heat stable pressor substance called angiotonin (Page and Helmer). It seems probable that other pressor substances are produced. The action of angiotonin is directly on the arterioles and no the heart. As to the mechanism by which angiotonin and like substances cause arteriolar hypertonus, it has been shown that nervous mechanisms have no part

but although the pressor substance seems to be quite distinct from adrenalin, integrity of the adrenal cortex is necessary to allow of the production of hypertension.

Normal kidney tissue prevents renin from exerting its pressor effect, and it would seem that the presence, persistence and severity of hypertension, experimentally produced, is a function of the ratio of ischæmic to normal renal tissue present (Katz). This holds out the hope of isolating an anti-pressor substance from normal kidneys for therapeutic use.

In an animal in which persistent hypertension, comparable to benign hypertension in the human subject, has been produced by an alteration of the intra-renal circulation in one kidney, removal of the other kidney will determine the development of a condition comparable to malignant hypertension in man. The same transition from benign to malignant hypertension may be determined on occasion by the large addition of sodium chloride or meat to the animal's basic diet. This observation gives support to the prescription of a vegetarian diet and restricted salt intake in certain cases of human hypertension.

Structural Pathology.—In contrast to atheroma, which is *localised*, the lesion in diffuse hyperplastic sclerosis (hyperpiesia) is *diffuse*. In contrast to senile arterio-sclerosis, in which the lesion greatly affects the media as well as or more than the intima, in diffuse hyperplastic sclerosis the intimal thickening (or hyperplasia) is the distinctive pathological feature. The coincident thickening of the media is explained in terms of physiological response to the persistent hypertension always found in this form of arterial disease. Further, in senile arterio-sclerosis the main incidence of the lesion is in the conducting arteries, whereas in arteriolo-sclerosis it is in the arterioles. The lesion is characterised by the following pathological features. In the terminal arterioles there is intimal thickening due to endothelial or subendothelial proliferation of cells, followed by an increase of hyaline substance and fibrous connective tissue. The process may go on to complete closure of the lumen, and in the terminal stage there is fatty degeneration of the thickened intima, so that in cross section of an arteriole the lumen appears blocked by a plug of fat. In the parent vessel from which the terminal arteriole springs, there is the same intimal thickening accompanied by an increase in thickness and number of strands which form the internal elastic lamina, at the same time an increase of fibrous tissue. There is little or no fatty change in the arteries of this size. In serial sections the marked fatty degeneration of the terminal arterioles may be seen to stop short at their offshoot from the parent vessel (Jores). It is important to note that the lesion known as diffuse hyperplastic sclerosis is recognised by the coincident presence of these particular changes in the terminal arterioles and their parent vessels, because either the lesion as described in the arterioles, or that described in the parent vessels, is found in a number of other disease states, but the two together with a distribution to be described are only found in patients who have had persistent hypertension during life. The initial hyperplasia is accompanied by hypertrophy of the media, and this may be a prominent feature and widely distributed. Hence the term **Arterial Hypermyotrophy** which has been given to it (Savill). The distribution of the lesion is diffuse in the vessels affected. In the terminal arterioles it is often moniliform, and there are varying grades of fat staining with Sudan III. In the parent trunks the initial hyperplasia is very uniform, both in the circumference of the vessel and in its length. In any organ the distribution of the lesion is partial, some vessels being more affected than others, and in the arterioles the lesion may be complete in some, whereas others escape. The organ distribution is characteristic. The lesion is always found in the kidneys or spleen, and generally in both. It is commonly found in the brain, pancreas, and suprarenals, and rarely in the liver or digestive tract; it does not occur as a complete lesion in the heart or skeletal muscle.

Essential Hypertension.—Persistent hypertension without kidney disease was first recognised in this country by Sir Clifford Allbutt, who called it hyperpiesia. Allbutt maintained that hyperpiesia pursues its

course and ends in a cerebral catastrophe or cardiac defeat, or life is terminated by intercurrent disease, but that at no stage of the disease does uræmia develop. The clinical syndrome described by Allbutt as hyperpiesia is now known as Essential Hypertension.

Essential hypertension is diagnosed in a case of persistent hypertension in which primary kidney disease (renal hypertension) and a known cause of hypertension (symptomatic hypertension) have been excluded (§ 87.) It is subdivided into benign and malignant types according to whether the kidneys are normal or are secondarily involved. The term hyperpiesia is no longer used, because it is now known that benign hypertension may (in 10 per cent. of cases) develop malignant hypertension and die of uræmia.

Benign Hypertension. *Symptoms.*—There may be none, especially in the early stage of the disease and in its benign forms. (i.) When symptoms are present, they may be general, such as might be ascribed to neurasthenia, namely, loss of energy, fatigueability, insomnia and nervous exhaustion. (ii.) Cerebral symptoms are common and varied. Headache is often occipital, but may be vertical or frontal, and is sometimes paroxysmal like migraine. (iii.) Dizziness and vertigo, or a sensation of faintness, are common symptoms. (iv.) Actual fainting attacks are less common, but are of more serious import. They vary from slight interruption of the continuity of thought on the one hand to a prolonged faint or epileptiform seizure on the other. (v.) Cardiac symptoms are common: cardiac pain, anginal attacks, palpitation, shortness of breath on exertion, or attacks of nocturnal dyspnœa. (vi.) At a later stage heart failure develops. This may take the form of congestive heart failure with auricular fibrillation or with regular rhythm, cardiac asthma or syncopal attacks.

Signs.—(i.) In established cases the blood pressure is persistently 180 mm. systolic and 90 mm. diastolic, or over; it may be much higher, even reaching 260 mm. systolic and 120 mm. diastolic. (ii.) There is left ventricular hypertrophy, recognised by an increase in the force of the cardiac impulse, an increase in the area of cardiac dulness, and a lengthening and lowered tone of the first sound at the apex. Or it is recognised by an increase in the size of the heart in a radiogram and by left axis deviation in the electrocardiogram. (iii.) The second aortic sound is accentuated. (iv.) The radial pulse is hard and resists compression. The artery is generally felt to be thickened, and it may be tortuous. (v.) The retinal arteries may be pale and contracted while the veins are somewhat full, or there may be signs of retinal arterio-sclerosis, of which irregularity of calibre of the arteries is the most important. Papillœdema is a grave sign. (vi.) The urine is normal, apart from the presence in some cases of a trace of albumin, and slight excess of granular and hyaline casts. Renal function is normal. As was pointed out by Sir Clifford Allbutt, there is no anæmia or other effect of a chronic toxæmia. In fact, in benign essential hypertension the patient is often over-weight and plethoric. The complexion is a good colour in contrast to the pale and

muddy complexion of chronic renal disease accompanied by persistent hypertension. In women more often than men the complexion may be pale and sallow in spite of the absence of kidney disease. In these patients, however, the deep colour of the lips contrasts with the pallor of the face, and indicates a good hæmoglobin content of the blood..

The *differential diagnosis* of hyperpiesia is from the various forms of urinary disease on the one hand and symptomatic hypertension on the other. *Urinary disease* is excluded by the absence of a history of kidney disease or its symptoms, a normal or practically normal urine, normal renal function and pyelography. *Symptomatic hypertension*, such as may be due to heart failure, endocrine disturbance (as at the climacteric, also in certain tumours of the suprarenal gland), and such poisons as alcohol and lead, is diagnosed by recognising the etiological factors, and the diagnosis is confirmed by response to treatment and progress of the patient.

Prognosis. The disease is essentially progressive, but there are some cases which pursue a benign course, and in these progress is very slow. Indeed the blood pressure often remains constant at a level of, say, 200 mm. systolic and 110 mm. diastolic, while only occasional symptoms appearing from time to time show progress of the disease or a failure on the part of compensatory mechanisms to make adjustments necessary to well-being. Some maintain so good a level of health that the disease must be regarded as quiescent, and indeed it is often stationary for a year or more at a time. In the most favourable event the patient may live in good health, with little limitation of his or her activities, for a period of ten years or more, and die from intercurrent disease or natural causes. In other cases, particularly those with a diastolic pressure of 130 mm. or over (in which cases the systolic pressure may be 260 mm. or over), the disease tends to be progressive and ends fatally from heart failure or coronary disease in about 60 per cent. of cases ; from apoplexy in 19 per cent. ; from renal failure in 8 per cent. ; from intercurrent disease in 12 per cent. (Bell and Clawson). It is uncommon for a high blood pressure, once established over a length of time, to return to normal. Nevertheless this may happen on occasion following a severe illness, as after a severe attack of coronary thrombosis : and after treatment of obesity. There is another type in which after the blood pressure has become established at a moderately high level, such as 210 mm. systolic and 100–110 mm. diastolic, there is a period of good health for four to ten years, when without obvious cause the disease suddenly takes on a progressive form and the terminal picture is that of malignant hypertension. In the majority of cases the disease in its general course is slowly progressive, with periods of activity alternating with periods of quiescence, and ending in a cerebral hæmorrhage or thrombosis, in heart failure or intercurrent disease.

Treatment.—There are no means known of arresting these arterial diseases, nor is there any remedy available which will directly promote the healing and repair of diseased arteries. Nevertheless a good deal can be done to improve health and prolong life of these patients. Irksome

restriction of activities is to be avoided unless essential. It is generally better for the person to continue at work provided it is within the limits of the patient's strength. Extra rest, a longer night spent in bed, or an hour spent horizontal between lunch and dinner, may be the means of keeping a man at work without overtaxing his strength. Exposure to cold should be avoided. Patients with arterial disease are more liable than others to feel the effects of chill. Though the part played by infections in causing arterio-sclerosis is doubtful, nevertheless an infection may determine complications of the disease. Thus cerebral thrombosis may supervene a week or two after an attack of tonsillitis or bronchitis in an arterio-sclerotic subject. The habits of life should be regular. Large meals and over-eating are to be avoided, and alcohol should be taken in strict moderation. Constipation must be corrected to prevent straining at stool, and habitual loose stools are weakening. Mild aperients such as rhubarb, aloes, cascara and magnesia are indicated. An occasional colonic douche is good in some cases. A mercury pill followed by a small dose of salts in the morning and taken once or twice a week may be beneficial. The activity of the skin should be promoted by attention to clothing, a daily warm bath, and in some cases a Turkish bath once a week. Some patients seem to benefit by a reduction of animal protein in their *diet*. For these, fish, chicken or meat taken only once daily may be prescribed, or trial may be made of a vegetarian diet, and continued so long as it seems to benefit the patient. The empirical restriction of meat and salt has received support from recent observations on the adverse effect of giving meat and sodium chloride to dogs with hypertension experimentally produced. In other respects the diet should be plain, varied, and easily digestible, care being taken to see that it contains an optimum of protective food substances, especially vitamins B and C. In some cases, where there are signs or symptoms of active disease, or during a progressive phase of the disease, especially when there is reason to think that the complaint is of recent origin, trial may be made of a period of intensive treatment, namely, two to four weeks' rest in bed on a strictly vegetarian diet of low calorie value, together with sedatives, and an occasional small dose of mercury by mouth. *Venesection* is indicated when there is peripheral congestion; 15–20 fl. oz. of blood are withdrawn. This may be repeated after six months. It is not well tolerated after the age of 65, and is contra-indicated in the presence of kidney disease. *Drug* treatment is chiefly of value for symptomatic treatment. It is important to secure a peaceful sleep at night, and small doses of aspirin, bromide or phenobarbitone are given for this purpose. For heart weakness or failure digitalis should be given. Bicarbonate of soda prescribed with powdered rhubarb and compound infusion of gentian may improve gastric function. Phenobarbitone ¼ gr. is given for relief of restlessness and nervous tension. A change of environment from time to time, residence in a warmer climate during the winter months, and the provision of congenial occupation to take the place of work which has to be given up on account of failing strength, are helpful

measures. Benefit may result from the treatment of associated conditions, as for instance, by reducing a body that is over-weight, by the successful treatment of osteo-arthritis or gout, the control of carbohydrate metabolism when there is diabetes mellitus, and by the eradication of gross sepsis in infected teeth. Lastly, there is a variety of treatment which can be directed towards the control of structural and functional changes in the various organs and tissues of the body which are due to arterio-sclerotic disease. Gangrene in the extremities may be delayed or prevented by intermittent venous occlusion or by sympathectomy. The circulation may be improved by such drugs as digitalis, nikethamide (coramine), cardophylin and nicotinic acid. Symptomatic treatment helps to prolong both the patient's life and activities.

When conservative treatment on the above lines fails, and in severe cases, other medicinal or surgical treatment may be advised. Potassium thiocyanate 0·1 G. given in solution t.d.s. after meals relieves headache and giddiness when other measures have failed : in some it reduces blood pressure with beneficial results. The dose varies between the wide limits of 0·8 G. and 4·2 G. weekly : the object is to secure a serum concentration of 6–10 mgm. per cent. : in no case must it be more than 12 mgm. per cent. A serum thiocyanate estimation must be done at the end of the first week and again at the end of the second week : after this it is made at longer intervals ; after some months, when the maintenance dose is well established, it is made each 3 or 6 months. Some patients cannot tolerate thiocyanate. A dull red maculo-papular rash may appear in spite of well-regulated dosage with a low serum thiocyanate level. Other symptoms of intolerance or over-dosage are asthenia, extreme tiredness, malaise, pains in the limbs, loss of appetite and nausea : rarely there is hypothyroidism or even myxœdema. On the appearance of any symptom of poisoning the drug is stopped immediately and a serum thiocyanate estimation is done. Potassium thiocyanate is a symptomatic remedy and does not cure the disease, but it is on occasion so effective that patients take it for years. The value of *dorsi-lumbar sympathectomy* is well established. It is advised in severe cases of benign hypertension especially in those between the ages of 25 and 40. (It is not advised after 50 years of age.) The results are better in women than in men. It is generally contra-indicated when hypertension is complicated by heart disease, in those who have had cerebral vascular accidents and in malignant hypertension. Before advising this operation renal efficiency should be tested and intravenous pyelography carried out. When the operation fails in its objective or is only partly successful, treatment with potassium thiocyanate may be effective even when it failed before operation.

Malignant Hypertension.—This disease may suddenly declare itself after an insidious onset, as judged by symptoms, over a period of a few weeks or months.

Structural pathology.—The changes observed are those already described in diffuse hyperplastic sclerosis (§ 94). In malignant hypertension these changes are over-

shadowed and partly obliterated by fibrinoid degeneration of arterioles and acute arteriolar necrosis, and in the more rapid and severe cases hyperplasia may be negligible or absent.

Symptoms.—The disease is recognised by the presence of papillœdema with or without retinal exudates or hæmorrhages, or by a hæmorrhage elsewhere, such as hæmaturia, hæmoptysis, hæmatemesis, and so on. It is suspected when the diastolic pressure is 130 mm. Hg. or over. In addition to macroscopic or microscopic hæmaturia renal involvement is registered by albuminuria, cylindruria, and urea retention of mild degree (§ 401). At the same time, the patient's general condition deteriorates. There is malaise; loss of strength, energy and body-weight, often loss of appetite, and anæmia.

Diagnostic importance has been attached to papillœdema, with or without retinal changes, as described above. However, these changes in the fundi, which are known as hypertensive retinopathy, though generally indicative of malignant hypertension, may in some cases be due to local retinal arterial disease (angio-spasm). In such cases there may be little or no evidence of renal involvement and no general loss of health, as shown for instance by malaise, loss of energy, loss of appetite and anæmia. In such cases intensive treatment, with rest in bed, sedation, perhaps venesection, and the prescription of potassium thiocyanate, or dorsi-lumbar sympathectomy may arrest the disease for a period (within present experience) of five to ten years.

Prognosis. When malignant hypertension is clearly established with retinopathy, renal involvement and anæmia the disease generally ends fatally in six months to two years.

Treatment.—The treatment is on the same lines as that described for benign hypertension. Symptomatic treatment is generally without much effect, but potassium thiocyanate may relieve the severe headache and a course of careful treatment with rest in bed for three or four weeks should always be given a full trial.

Hypertensive Cerebral Attacks (hypertensive encephalitis) are a cerebral form of this. *Symptoms* are sudden headache, drowsiness, coma or convulsions, vomiting and albuminuria. The retinal arterioles are seen to be constricted, retinal hæmorrhages, exudates and papillœdema suddenly appear. The systolic pressure may rise rapidly during a period of a few hours by as much as 100 mm. Hg. The C.S.F. pressure is raised, and there is cerebral œdema. *Diagnosis* is from a cerebral vascular complication of benign hypertension.

Treatment.—In any case of hypertensive cerebral attack the rising blood pressure is an indication for venesection. A raised C.S.F. pressure is an indication for the removal of 10–20 c.c. of cerebro-spinal fluid. Restlessness is controlled by the injection of heroin hydrochloride gr. $\frac{1}{8}$ to gr. $\frac{1}{6}$. Sweating should be promoted and the secretion of urine stimulated.

III. **Periarteritis nodosa** (Syn. polyarteritis nodosa) is a low grade infection of the smaller arteries and arterioles which may be localised or generalised. The essential lesion is a whitish-grey nodule consisting of aggregations of polymorphonuclear cells, together with eosinophils and monocytes. The earliest change is in the adventitia. There is necrosis of the media and proliferation of the intima. Thromboses with infarction and even aneurysms are complications of this lesion.

Symptoms.—The onset may be acute or subacute. In the generalised variety, the patient feels progressively weak and ill, loses weight and later shows mental apathy. There is an irregular temperature with tachycardia. Headache and vague pains in the limbs, and sometimes polyneuritis, suggest a diagnosis of rheumatism. Chest symptoms comprise shortness of breath, cough, sputum and sometimes hæmoptysis, and these are often associated with rales or consolidation of the lungs. Abdominal pain may occur, and thromboses in the abdominal organs at times give rise to gastro-intestinal hæmorrhages or even perforation. Pericarditis or myocardial infarction may be found. The urine almost invariably contains albumen, casts and red cells, and uræmia with or without hypertension is common. A blood count shows a more or less severe degree of anæmia, with a polymorph leucocytosis and rarely an eosinophilia: blood cultures are invariably sterile. In a proportion of cases, one or more palpable skin nodules, the size of a millet seed or a pea, materially aid diagnosis: less often purpuric or vesicular skin lesions are found. The *diagnosis* may be confirmed by biopsy of a skin nodule or of voluntary muscle.

The *prognosis* used to be considered invariably fatal sooner or later, but after a period of many months, recovery can ensue. *Treatment* is symptomatic. Chemotherapy has no effect.

IV. **Chronic** and **Acute Endarteritis**, due to syphilis and other causes, is recognised by its pathological effects (cerebral softening, aneurysm and gangrene). Acute endarteritis has pathological rather than clinical significance. It is common in arteries at the base of chronic ulcers, such as the perforating ulcers of tabes, syringomyelia and diabetes, in new growths, both malignant and benign, in the terminal branches of the coronary arteries in patients dying of rheumatic carditis, in tuberculous, actinomycotic and lymphadenomatous lesions. **Syphilis** affects the arteries in two ways: (1) a proliferation of the intima of small vessels reduces their lumen and interferes with the nutrition of the parts supplied by these vessels (syphilitic endarteritis). This condition also predisposes to thrombosis in the affected vessel, and explains many cases of cerebral thrombosis. (2) A weakening of the muscular coats of the large vessels is seen typically in syphilitic mesaortitis and brought about probably by obliterative changes in the vasa vasorum. With this is commonly associated a proliferation of the intima, especially of the first part of the aorta: it may lead to extensive scarring, and often gives rise to anginal pain.

V. **Aneurysmal Dilatation of the Arteries** belongs to surgery, excepting aneurysm of the thoracic aorta (see § 80), the abdominal aorta (§ 263), and the cerebral arteries.

VI. Complications of vascular disease are:

(*a*) **Embolism,** *i.e.*, the blocking of an artery by an embolus, which may result from heart disease, especially infective endocarditis (§ 50) and mitral stenosis; or may be secondary to thrombosis.

(*b*) **Thrombosis,** the coagulation of blood in the living vessel, results either from local vascular disease or an altered blood state.

Embolism and Thrombosis are dealt with elsewhere. See, for example, §§ 570, 577, 580, Phlebitis and Localised Dropsy.

VII. **§ 95. Other forms of Disease of the Arteries.**—Of functional diseases or vasomotor derangements we know but little, although several important maladies are attributed to this cause—*e.g.*, Raynaud's disease and migraine. Functional derangement of the arteries is also manifested by a large number of symptoms, many of which are vague and evident only to the patient. On this account they are apt to be regarded by medical men as unimportant, and it is true that they are not serious in the sense of being lethal; but to the patient they are often extremely disagreeable, irksome, and often terrifying. Of such we may mention alternate flushing and pallor

("flush-storms"), dead hands, cold hands and feet, chilblains, various other erythematous conditions, blue nose, palpitation, tachycardia (§ 84), paroxysms of copious urination, acroparæsthesia, erythromelalgia, feelings of suffocation, pseudo- and true angina pectoris, feelings of tingling, itching, throbbing, and actual swelling of the limbs. (See § 575 *et seq.*)