I. Acute fibrinous and sero-fibrinous pericarditis. Acute inflammation of the pericardium.

Acute inflammation and its results constitute by far the most frequent and important morbid conditions of the pericardium with which we have to deal in medical practice; and they often lead to serious consequences, both immediate and remote. As an acute affection pericarditis varies considerably in different cases, whether as regards its intensity and extent, the rapidity of its progress, the nature and amount of its pathological products, or its terminations and ultimate effects; but the complaint must always be looked upon with concern. In some instances it may be described as subacute rather than acute, but there is no line of demarcation between the two classes.

Etiology and Pathology.—In accordance with modern views of the relation of micro-organisms to acute inflammatory diseases, it may be assumed that particular agents of this nature are immediately concerned in originating acute pericarditis. It will be more convenient and practical, however, in the first instance, to point out the circumstances under which this affection occurs, and afterwards to try to ascertain what is really known of its bacteriology.

It has been customary, from an etiological point of view, to divide cases of acute pericarditis into primary or idiopathic, and secondary. The former include those which cannot be referred to any of the recognised causes of the disease, and in which it attacks an apparently healthy subject. Under such circumstances the complaint has been usually attributed to chill of some kind, but cases thus originating are probably of a rheumatic nature. In some cases called idiopathic the patients were drunkards, or were suffering from the effects of privation. In my own experience I have never met with an instance of acute pericarditis which, when carefully investigated, could not be included as a "secondary" event in one or other of the etiological groups now to be discussed.

(a) Rheumatic pericarditis.—This is by far the most important variety, and the number of cases coming under this head is very much larger than that of all other cases put together. There are several features to be noticed in this group, and in their discussion I propose to draw attention to some interesting and practical facts observed by the late Dr. Sibson, whose article on "Pericarditis," in Reynolds' System of Medicine, contains the outcome of extensive personal observations, with comments, many of which are noticed or quoted in the following pages. The definite connection between acute rheumatism and pericarditis has long been recognised. The pericardial inflammation is not to be looked upon as a mere complication, but is an essential part of the disease. The frequency of the association has been very differently stated by different writers, and doubtless it varies under different circumstances. In rheumatic cases pericarditis is not nearly so common as endocarditis; Sibson found the
latter fully three times as frequent in those which he observed. Further, he noted that, in the large majority of cases of pericarditis, endocarditis was present also. The late Dr. Sturges drew special attention to this association in children, and he applied the names peri-endocarditis or carditis to the combination, which he regarded as exclusively rheumatic. He also laid down the proposition that "the rheumatic heart inflammation of children when pericardial is always endocardial as well, and when endocardial is extremely likely, with the recurrence of rheumatism, to involve the pericardium also." My own experience is fully in accord with the observations of these eminent physicians, and the effects of a combination of pericarditis and endocarditis come before us in a considerable proportion of the cases of chronic heart disease which can be traced to one or more rheumatic attacks in early life.

It has been stated on high authority that pericarditis of any kind is a rare disease in children; and the complaint has been said to prevail most frequently in middle life. More recent and accurate observations, however, have shown that such statements are quite contrary to fact. Sturges pointed out that pericarditis is very common in children. He noted that "out of 100 fatal cases of heart disease occurring at the Children's Hospital, Great Ormond Street, between June 1881 and April 1892, of which 54 were of rheumatic origin, and 46 due to other causes, in 6 only was there no evidence of pericarditis." When introducing the discussion on Acute Rheumatism at the meeting of the British Medical Association in 1895, Dr. Cheadle also spoke of pericarditis as less and less frequent with the advent of puberty. Certainly, so far as my own experience goes, while prepared to meet with rheumatic pericarditis at any age, it is in children, growing boys and girls, and young adults, that I have found it necessary to be more particularly on the look-out for the disease, and I am convinced that this is a point of considerable practical importance. [Vide art. "The Acute Rheumatism of Childhood," vol. iii. p. 44.]

Rheumatic pericarditis is decidedly more common in males than females, but the exact difference cannot be stated. Sibson found the proportion to be 1 in 4 to 1 in 6; and he explained this difference in part by the influence of age and occupation on acute rheumatism and its complications. He observed that one-half of the males and more than one-half of the females were below the age of 21; while two-fifths of the male and only one-fifth of the female patients were above the age of 25. Servants formed fully two-thirds of the female patients affected with pericarditis; and three-fourths of the servants attacked with pericarditis and endocarditis were below the age of 21. These facts Sibson explained by the hard occupation of patients of this class, in view of their time of life and constitution, which exposes them to the causes of acute rheumatism and its attendant inflammation of the heart. Those of tender age who followed no occupation were not attacked with inflammation of the heart with anything like the same frequency as young female servants. Women who at mature age followed occupations as laborious as that of the young servants were affected with pericarditis in but
a moderate proportion, and in a comparatively mild form. He concludes—"We thus see, in brief, that in acute rheumatism affecting the female sex, youth with labour is nearly always attacked or threatened with endocarditis or pericarditis, or both; that youth without labour is thus attacked with comparative infrequency; and that mature age with labour is attacked much less frequently and much less severely with inflammation of the heart than youth with labour."

With regard to males Sibson observed the following facts:—Of laborious workers out of doors attacked with pericarditis only 1 in 10 was below the age of 21; while of indoor workers thus affected fully three-fourths were below that age. The scale was entirely reversed in those of older age. Of those labouring out of doors four-fifths were above 25; while of those working indoors only one-sixth were above that age. Sibson writes: "We here, I consider, find the explanation of the twofold fact, that the male cases of pericarditis usually combined with endocarditis outnumber the female cases by one-fifth, and that the number of the men so affected above the age of 25 is three times as large as that of the women. I think we may infer that excessive labour in men of mature age is a frequent cause of acute rheumatism having a strong tendency to pericarditis." He further concludes that "in both sexes the same causes produce, under like conditions, the same effects; and that a very large proportion of the young persons who work on foot indoors during many hours daily are attacked with inflammation of the heart when affected with acute rheumatism, while a very small proportion are thus attacked of the men and women of mature age who are engaged in the same manner."

The relation between the severity of the rheumatic condition, as seen in the joints, and acute pericarditis must next be considered. Sibson noted that in servants attacked with pericarditis the severity of the joint affection in the great majority of cases bore a strict relation to the severity of the heart affection. Taking all cases into account, however, this rule is by no means true in a considerable proportion of them, and it is highly important to remember that pericarditis may set in and become very pronounced while the articular complaint is comparatively or actually mild; and it may even occur alone, or come first of the rheumatic series. This statement applies particularly to children, who are liable even to carditis of rheumatic origin with little or no joint affection or pyrexia. Another observation made by Sibson is that in about half the cases, when the pericardial effusion is at its height, the joint affection tends to lessen in severity. The number of articulations involved, and the implication of particular joints, bear no relation to the frequency of pericarditis. The disputed question whether it is more prone to occur during first or subsequent attacks of acute rheumatism is not of much practical significance, though the general experience is in favour of first attacks. The appearance of the affection must be watched for during each attack, whether it has or has not previously occurred, unless indeed it has left behind universal adhesion.
Sibson stated that previous rheumatic attacks favour the occurrence of endocarditis much more than of pericarditis, and this is in accordance with my own experience. As to the time at which acute pericarditis supervenes in the course of a rheumatic attack, it seems to appear in a certain proportion of cases—according to Sibson, about one-eighth—at the very beginning, and to be coincident with the joint affection; or, as already stated, it may even precede such a manifestation. Not uncommonly it appears between the third and the sixth day; and, according to Dr. George Balfour, most cases occur within the first week of the rheumatic onset. In nearly one-half of Sibson's cases signs of pericarditis were observed on or before the eleventh day of the illness. On the other hand, the complaint may not be revealed for two or three weeks or more; and in seven out of sixty-three of Sibson's cases it showed itself between the twenty-fifth and sixty-third day. Moreover, it may follow a relapse of articular rheumatism, the pericardium having been quite free from any affection during the primary attack. In the case of children pericarditis may arise at any stage of the rheumatic series, but, according to Cheadle, most often it comes late, in association with recurrent endocarditis, when the heart is already hypertrophied and dilated (vide vol. iii. p. 38).

The opinion has been advanced that excessive action of the heart, set up by the rheumatic condition, may help in the production of acute pericarditis. This was evidently Sibson's opinion concerning the relative severity of the joint affection and that of pericarditis, for he writes: "When the joint affection is severe, it may call forth excessive labour or even tumultuous action of the heart. In acute rheumatism inflammation attacks the fibrous structures, especially if these structures are unduly strained, and the increased action of the heart may therefore, I consider, induce inflammation of the fibrous tissues of that organ, such inflammation being proportioned in severity to the augmented action of the heart."

Cold, damp, and changeable climate and season have necessarily a marked influence upon the frequency of cases of rheumatic pericarditis.

(b) Renal pericarditis. — The association of acute pericarditis with different varieties of Bright's disease cannot be doubted, but statistics seem to show that the frequency of this form depends upon certain circumstances. Sibson collected a large number of cases, from various sources, of renal disease thus classified, and found that pericarditis occurred in 8.1 per cent; but it differed materially in frequency in different countries, the proportion in Germany being 10.4, in France only 3, and in England 8.4 per cent. With regard to its relative frequency in the several varieties of Bright's disease, it appears from Sibson's statistics to be uncommon in connection with acute scarlatinal nephritis in young subjects, but frequent in adults who suffer from acute Bright's disease, as well as during the transitional stage to the large white kidney. When the latter has become established, however, the tendency to general pericarditis disappears almost entirely; yet it may occur in a partial or circumscribed form. In connection with the contracted granular kidney the complaint is far more common; and it is of average or moderate frequency in cases of
lardaceous disease. It has also been stated to be especially associated with fatty kidney. Dr. George Balfour regards acute pericarditis as usually a late phenomenon in renal disease, and states that it is often a precursor of fatal uremia. Sibson believed that over-action of the heart increases the tendency to pericarditis in Bright's disease, as well as the enlargement of the organ associated with the granular kidney. He also affirmed that great enlargement tends to develop partial into general pericarditis.

(c) Pericarditis from extension or irritation.—The occurrence of pericarditis as the result of extension of the inflammatory process from neighbouring structures is an important fact; and my observation of cases in which the disease has thus originated has led me to believe that they are more frequent than is generally known. In most instances it follows pneumonia or pleurisy, more particularly when the inflammation is on the left side. It spreads from the pleura to the outer surface of the pericardium, and thence penetrates to the interior of the sac, kindling a more or less pronounced pericarditis. Professor Shattuck has recently called attention to the frequency of the association of pericarditis with pneumonia. It must be noted, however, that in some cases in which these combinations of acute inflammatory diseases are met with in the chest, the pericardium was involved first, and from it the inflammation spread to other structures; or the whole of them may be implicated so rapidly that it is difficult or impossible to determine where the inflammation started. In exceptional instances the process may extend, through the diaphragm, from the peritoneum to the pericardium, without any direct communication between the two cavities. It will suffice to mention that cases are now and then met with in which pericardial inflammation is set up by some neighbouring cause of irritation, apart from the inflammatory diseases just considered; such as abscess, anurysem, enlarged glands or tumours, or bone disease.

(d) Traumatic and perforative pericarditis.—These two forms may be considered together. They are of very rare occurrence, and I cannot speak of them from personal knowledge. The chief injuries from without which may cause pericarditis are a blow or contusion over the precordial region; fractured ribs; penetrating wounds by sharp instruments or gunshot wounds, and lesions produced by way of the cesophagus, especially by foreign bodies, purposely or accidentally swallowed, which may actually perforate the pericardium, or even gain access into its cavity, or, remaining lodged in the gullet, may injure the adjacent pericardium; examples of such bodies are false teeth, needles, or fish-bones. Perforative pericarditis may result from the bursting of any neighbouring abscess into the sac, whether associated with the bronchial glands, bone disease, or other like conditions; or, in very exceptional instances, a communication may be established from an empyema, from a phthisical cavity, or from the cesophagus if it be the seat of ulceration or new growth. Still more rarely the contents of an abdominal abscess find their way through the diaphragm into the pericardium; and even a gastric ulcer has perforated
its walls. In all these cases definitely irritating or septic materials of different kinds gain access to the pericardial sac.

(c) Pericarditis secondary to cardiac or aortic disease.—A separate group may be recognised of cases in which acute pericarditis is secondary to some affection of the heart itself, or of the arch of the aorta. As already pointed out, this affection and endocarditis frequently occur together in rheumatic cases. It seems probable, moreover, that inflammation primarily involving the endocardium may traverse the myocardium, so as ultimately to implicate the pericardium as well; and this applies particularly to infectious endocarditis. Myocarditis itself, especially if ending in suppuration, may likewise originate pericarditis, quite apart from the bursting of an abscess of the walls of the heart into the sac. Among very rare causes may be mentioned cardiac aneurysm, or intra-pericardial aortic aneurysm. With regard to chronic diseases of the heart, pericarditis has now and then appeared in cases of valvular affection, chiefly aortic, especially when associated with cardiac hypertrophy; but the connection between these conditions is not very clear, and it seems to me that on careful investigation of such cases some more definite cause of the pericardial inflammation would probably be found.

(f) Pericarditis associated with new growths.—The chief kinds under this head are malignant and tuberculous pericarditis. The former class of growths may be situated in the heart, the pericardium, or both; and no practical distinction can be drawn between them. Tuberculous pericarditis is probably more frequent than is usually supposed, and should not be forgotten. When the inflammation is set up in chronic phthisis, apart from the bursting of a vomica into the sac, it is essentially a slow process; but in acute tuberculosis or very active phthisis it may certainly be acute. In very exceptional cases pericarditis seems to be the main tuberculous manifestation, and it is then rather subacute in its onset and mode of progress.

(g) Septic pericarditis.—This variety deserves separate recognition, as it may arise in all kinds of general septicæmia and pyæmia; though under such circumstances the pericardium is far less frequently affected than the pleura. Septicæmia associated with puerperal conditions and acute necrosis of bone have to be especially remembered in this connection.

(h) Pericarditis associated with miscellaneous general diseases and blood-states.—It will suffice under this heading to draw attention to the fact that in exceptional instances acute pericarditis occurs as a complication of some of the acute specific diseases, particularly scarlatina (most commonly during the period of desquamation, when it has been attributed to rheumatism or renal disease), measles, and small-pox; rarely of enteric fever, typhus, diphtheria, erysipelas, cholera, severe malarial fevers, scurvy, purpura, and hæmophilia; in these last conditions it is probably secondary, in some cases at any rate, to pericardial hæmorrhage: it may occur exceptionally in the gouty state; and in diabetes.
Bacteriology of acute pericarditis.—On a comprehensive survey of the etiological groups just considered, the relations of micro-organisms to the inflammatory process can in many instances be clearly recognised on general grounds. Such organisms have also been frequently demonstrated in the inflammatory products and in the pericardium itself. At the same time it cannot be said, so far as present researches go, that they are in any way specifically related to the disease. The notion seems to be gaining ground that the rheumatic process is due to a specific bacillus, to the direct action of which the pericarditis would in this case be attributable; but I am informed on the highest authority that up to the present time no such organism has been demonstrated. The organisms usually found are those ordinarily associated with the inflammatory process, namely, different kinds of streptococci and staphylococci. In cases following pneumonia the pneumococcus may be present, and it has also been found independently. Tubercle bacilli have been demonstrated in tuberculous pericarditis.

Morbid anatomy.—The changes which occur during the progress of acute pericarditis are similar in their general nature to those which characterise inflammation of other serous membranes. It is customary to describe the disease as following successively the stages of—(i.) Increased vascularity; (ii.) Fibrinous exudation; (iii.) Fluid effusion; (iv.) Absorption; and (v.) Adhesion. These stages, however, cannot always be definitely recognised, and in many instances they run more or less concurrently. Moreover, the pericardium itself is often involved in the inflammatory process. It will be expedient, in the first place, to describe individually the changes which take place during the progress of a pronounced case of acute pericarditis; and afterwards to point out the more important aspects under which they are presented in practice.

(i.) Hyperaemia, or increased vascularity, no doubt constitutes the earliest change in acute pericarditis. It involves the serous lining of the sac and the subserous tissue, and is accompanied with more or less parenchymatous swelling of the membrane. In its lesser degrees the hyperaemia is revealed by a fine network of vessels; but in its more pronounced form the surface is extensively and uniformly red, the redness being either bright or dark. Sibson described the appearance as follows:—

"When the surface of the heart becomes inflamed, a blush of fine vessels, consisting of a velvety network, appears on the surface of the organ, and especially over the larger coronary vessels at the base and septum of the ventricles. The inner surface of the pericardial sac, wherever it rests upon the inflamed heart, kindles also into a blush of fine vessels. The surfaces of the heart and sac, instead of being smooth and glistening, become dull and velvety." Sometimes minute hemorrhages are observed, especially around newly-formed vessels. The hyperaemic condition is of short duration, it may last but a few hours, and then either subsides or is concealed by exudation. As a matter of fact it is seldom seen at necropsies, and usually only in pericarditis connected with Bright's disease. It is said to be generally less marked in elderly and cachectic persons.
The deposit of fibrinous exudation or inflammatory lymph is an invariable accompaniment of acute pericarditis; though its quantity, extent, mode of arrangement, and exact characters vary much in different cases. As a rule, it is observed both over the surface of the heart and the interior of the pericardial sac. In some instances there are merely a few shreds about the roots of the great vessels; in others a thin film or coating forms at different spots, especially on the visceral surface; or a more or less thick and stratified layer covers both surfaces extensively or universally, and is often very abundant. Owing to the incessant movements of the heart the arrangement of the exudation is often peculiar. It very rarely presents a smooth surface; and in the large majority of cases it exhibits an alveolar, reticular, or honeycomb pattern. Sibson thus well describes the appearance:—"Where the two surfaces touch each other, the soft lymph is drawn into threads and little pointed ridges and prominences, and wrought into a network, so that when ridges and prominences are present on the heart, ridges or prominences are present on the inner surface of the pouch lying upon it, and when a network of lymph covers the heart, a network of lymph lines the corresponding sac. The constant play of expansion and contraction of the heart alternately stretches and relaxes its coating of lymph, so that its surface resembles a honeycomb in structure." Laennec's well-known and oft-quoted comparison likens the appearance to that presented on suddenly separating two smooth pieces of wood between which a small pat of butter has been forcibly compressed. It has also been called the "bread-and-butter like" appearance; or it has been likened to tripe. It must be noted, however, that the lymph does not always present this kind of arrangement; it may exhibit a shaggy or villous surface, or peculiar characters, to which such names as cor hiratun, cor tomentosum, etc., have been applied. When abundant, it is said to accumulate in large masses in the auriculo-ventricular groove and about the auricles. Should there be much lymph associated with fluid its surface is covered with floating shaggy processes, which sometimes have a mammillated aspect. Occasionally fibrinous papillæ or bands pass across between the opposing surfaces of the pericardium, and these may even form partitions.

The lymph exuded in pericarditis is usually of a whitish yellow, yellowish, or reddish colour; but it may be brownish. In a very short time a fine network of vessels is developed in its substance, and not uncommonly spots of hemorrhage are present, or the whole exudation may be deeply stained. In connection with purpura, scurvy, and allied blood diseases alternating layers of blood and lymph are now and then observed. In consistence the material is, as a rule, somewhat firm and elastic, but it may present different degrees of softness down to that of an almost liquid jelly. Not infrequently it is mixed with serous fluid. In exceptional instances of a low type it has been described as granular, crumbling, or boggy. At first the exudation can be readily separated and peeled off from the surface of the membrane, but after a while it becomes more
adherent and difficult to detach. In structure it consists of coagulated fibrin and cell elements, the latter chiefly occupying the deeper layers. When the material is very soft the cells are in great abundance, and at the same time molecular disintegration has taken place. Microorganisms of different kinds may be found in the exudation.

(iii.) There can be no doubt that in not a few cases of acute pericarditis there is little or no fluid effusion; a form of “dry pericarditis” being met with, which can be recognised clinically. In such cases very rapid adhesion may take place between the visceral and parietal surfaces, even over an extensive area; the lymph being thick, sticky, gelatinous, and specially agglutinative. Sturges drew particular attention to this course of events in children. Occasionally a kind of network of fibrinous strings passes between the adjacent surfaces, the meshes of which are filled with serum. As a rule, however, during the progress of an attack of acute pericarditis, where there are no adhesions, an effusion of fluid takes place into the pericardial sac, separating its parietal and visceral layers. Effusion may indeed supervene after the formation of early soft adhesions, sometimes limited to one side; or when the sac is partially filled with heavy gelatinous masses of lymph. The average quantity of fluid is from 8 to 12 ounces, but it may range from an ounce or two to two or three pints or more—Balfour says “several pints.” The amount of fluid effusion is by no means in proportion to that of the fibrinous exudation, and the result of the inflammatory process may chiefly be evidenced by either one or the other. It is in rheumatic pericarditis that large accumulations usually occur, and the effusion then generally collects and increases rapidly, often reaching its acme in two, three, or four days (Sibson). In opposition to this statement Dr. John Broadbent affirms that “it is the exception rather than the rule to find effusion of any extent in cases of pericarditis of rheumatic origin.” Dr. Church seems to be of the same opinion [vide “Acute Rheumatism,” vol. iii. p. 16]; and Dr. Cheadle states that in children the effusion, though fluctuating in amount, is never very large, and is usually reabsorbed quickly. In my experience, cases of rheumatic pericarditis have differed very much in the quantity of effusion. In
Bright's disease the quantity is often very small. Abundant effusion is likely to be met with in scorbutic cases, and, in these as much as five pints has been recorded.

The effusion in acute pericarditis is generally of a serous or serofibrinous character, and yellowish or greenish in colour; it is most commonly bright, clear, and transparent; but may present small fibrinous particles or flakes in suspension, or be opalescent, or even more or less cloudy and opaque. Occasionally it is brownish or reddish. The specific gravity averages about 1.018. Under certain circumstances, as when pericarditis is associated with purpura or scurvy, the effusion is obviously mixed with more or less blood or its colouring matter—"hemorrhagic pericarditis." The cases in which the inflammation leads to the formation of pus will be separately discussed; and it will suffice to state further that in very rare instances—of which there is an obvious explanation in the presence of some general septic condition, the effects of neighbouring gangrene or malignant disease, or the entrance of air containing septic micro-organisms into the sac—its contents undergo a putrefactive change, and become "ichorous," foul in appearance and odour, or actually stinking.

It will be convenient in the present connection to discuss briefly from a general point of view the immediate effects of pericardial effusion upon the sac itself and its contents, as well as upon neighbouring structures, effects which are met with by far most frequently in cases of acute pericarditis; and at the same time to point out the changes which the inflammatory process is apt to set up in the pericardium and heart, and which tend more or less to influence and modify these effects. Obviously they must vary considerably in nature and degree, according to the amount of the fluid accumulation, and the rapidity of its collection. The fact must be acknowledged that a certain quantity of effusion is sometimes found in the pericardium at the autopsy, it may be as much as 6 or 8 ounces, which had not given rise to any evident disturbance, and was not detected during life. In all such cases, however, which have come under my personal observation, there has been every reason to believe that the effusion had taken place shortly before death, from obvious causes, and usually under circumstances rendering adequate physical examination impracticable; and no doubt it is often increased by transudation of serum from the vessels after death.

Beginning with the pericardium itself, when a collection of fluid exceeds a certain quantity the sac necessarily becomes more and more distended, in proportion to its amount, and at the same time stretched and thinned, so far as the normally tough and firm parietal pericardium will permit. When acute pericarditis has lasted for some time, however, and the structures forming the walls of the sac itself are involved in the inflammatory process, they become more or less swollen, thickened, soft, and yielding; so that the pericardium becomes capable of far greater distension than in its natural state. As the fluid accumulates in increasing quantity the sac undergoes changes in form, which have been
well described and figured by Sibson. When artificially distended with 15 ounces of fluid, he noted that the pericardium became pyramidal or pear-shaped, and in more detailed description he writes: "It is formed, so to speak, of a larger and a smaller sphere, the smaller one resting on the top of the larger. The larger and lower sphere contains the heart, the ascending vena cava, and the pulmonary veins; and the smaller sphere holds the great vessels. The distended sac occupies the whole centre of the chest, filling up the space between the sternum in front and the spinal column behind, and extending across the chest from a little within the right nipple to a little beyond the left nipple. The whole sac is lengthened; its smaller end reaches upwards almost to the top of the sternum; and its floor, being formed by the central tendon of the diaphragm, presents a large spherical prominence that bulges downwards into the abdomen, occupies the epigastrium, and reaches as low as the tip of the ensiform cartilage and the lower edge of the sixth costal cartilage." This description will apply to the shape which the pericardium usually assumes when distended with fluid from pathological causes; but when, owing to inflammatory changes in the walls previously referred to, they give way further, the form alters considerably. "As the sac cannot expand to a material degree either upwards towards the neck, or downwards towards the abdomen, it yields sideways and backwards, and widens to the right and especially to the left" (Sibson). Under these circumstances, in short, its width becomes decidedly disproportionate to its height, and it loses its pyramidal outline, becoming in extreme cases almost globular.

The secondary changes of the heart and great vessels which may supervene in acute pericarditis, as well as the effects of a considerable effusion of any kind on these structures, must be discussed in some detail; and in individual cases they should always be borne in mind, and carefully studied from a clinical point of view. On these matters there are important differences of opinion, and they have given rise to much controversy.

There can be no doubt that inflammatory changes beginning in the pericardium are apt to extend to the muscular tissue of the heart itself, and this tissue may also undergo an acute degenerative change. These lesions are, speaking generally, proportionate to the intensity and duration of the pericarditis; but they are most frequently met with in the hemorrhagic and purulent varieties. They are naturally more pronounced in the superficial layers, but may gradually extend throughout the entire thickness of the walls. The degenerative changes sometimes attain a high degree, even in wholly acute cases. Whether the inflammatory process may extend from the pericardium through the cardiac walls to the endocardium is difficult to determine positively, but I think it is highly probable. The changes in the heart tissues have been attributed, not only to extension of the inflammation, but also to persistent high bodily temperature, and to the circulation of toxins in the blood. Degeneration has, moreover, been supposed to result from the inflammatory products pressing upon the coronary
arteries, and thus impeding the normal distribution of blood to the cardiac walls. Pericardial effusion, however abundant, has no direct influence upon the structure of the muscular tissue of the heart. It must be noted that the nerves distributed to the surface of the heart and great vessels may be involved in the inflammatory process.

Dr. John Broadbent maintains that considerable dilatation of the heart, especially of the right ventricle, occurs during pericarditis; and he has brought forward cases to prove that the physical signs usually attributed to pericardial effusion are really due to this condition. That such a dilatation does take place in some instances is indisputable, especially when extensive adhesions have rapidly formed; but my experience is certainly opposed to the supposition that it is usually an immediate result of pericarditis, or that it is apt under these circumstances to be mistaken for effusion.

What is the mode in which the fluid collects, and what position does the heart assume within the sac? These questions have been the subject of special controversy; and although to some writers they present no difficulty, and are unhesitatingly answered in a particular way, without reserve, I must confess that in my own clinical experience of individual instances I have not always found them easy of solution. It will be convenient at the outset to explain Sibson's later views on this subject, and some of his remarks deserve to be quoted at length. Describing the mode in which fluid collects in the pericardium, he writes: "At first it falls into the back part of the sac, but as it increases in quantity it makes a space for itself between the floor of the pericardium, which it depresses, and the lower surface of the heart, which it elevates; . . . and the result of this is to displace the apex and body of the organ and its great arteries upwards and forwards." He adds: "The heart, elevated by the fluid between the under surface of the ventricles and the base of the pericardium to a degree proportioned to the amount of the fluid, leaves the broader part of the chest below, and ascends into the narrower part of the chest above." In another place he writes: "The distension of the pericardium with fluid produces two other effects on the heart. (a) The heart is heavier than the fluid in which it plays, and its ventricles consequently tend to sink backwards so that the left ventricle rests upon the posterior wall of the pericardium. (b) The other effect of pericardial distension on the heart is the lifting or tilting upwards of the organ within the sac. The heart is attached by its great vessels to the posterior and upper part of the sac, and the whole organ therefore tends to shrink upwards and gravitate backwards towards its points of attachment." Sibson concluded that the natural effect of this gravitation, shrinking, and upward displacement of the heart, owing to great accumulation of fluid in the sac, if not modified by other agencies, would be to cause the interposition of a layer of fluid between the front of the heart and the anterior wall of the chest. He affirmed, however, that in practice this is not usually the case over the mass of the ventricles, though a layer of fluid covers the lower part of the right ventricle.
The displacement of the apex of the heart upwards and outwards in cases of pericardial effusion, was formerly taught as an indisputable fact. By most authors at the present day, however, though not by all, this doctrine is regarded as a mistake. The general opinion is that the fluid collects towards the front, and that the heart, being heavier than the fluid, falls or sinks backwards, away from the anterior thoracic wall; the ventricles, right auricle, and great vessels being successively covered from below upwards, and thus separated from the parietal pericardium. Some writers have maintained that an effusion first collects about the base, which is turned downwards, the heart lying rather more horizontal than normal, and the apex turned outwards; but this part is described as descending when the diaphragm is pushed down by the effusion.

Another opinion is that the position of the heart is not altered. This is the opinion of Dr. William Ewart (19), who affirms that the apex will be found in the usual situation at any necropsy on a case of uncomplicated pericardial effusion; and that whilst the heart has preserved its normal situation the floor and the sides of the pericardium have receded from it. Dr. Ewart regards the impossibility of any elevation of the apex as almost self-evident. He writes: “Slight mechanical displacement might conceivably be brought about by one circumstance only—the lifting by the distended pericardial sac of the tracheal bifurcation and of the bronchi, and with them of the pulmonary veins and of the heart. Practically this rise is very inconsiderable, and moreover it does not directly influence the ventricle. On the other hand, we must remember that the heart is tethered to the bottom of the pericardium by the attachment of the inferior vena cava to the foramen quadratum in the central tendon, and that the considerable descent of the diaphragm must depress the level of the right auricle and tend to depress the apex, far from allowing it to rise. I have in some cases detected a lowering of the heart’s apex in pericardial effusion, and with it a more median position of the heart, which then tends to hang more vertically from the aortic arch, the latter becoming slightly straightened.”

The late Dr. Sturges, in summing up the opinions just discussed, expressed his belief that “though apparently conflicting, they are in fact reconcilable.” They all express the truth in various circumstances. The heart may be moved either forwards, upwards, or backwards in effusion; or it may remain where it was; and of the factors that determine its conduct, pericardial adhesion, here or there, temporary or permanent, is the chief.” He further stated: “I have repeatedly in fatal cases of pericardial effusion inserted needles, just before the post-mortem examination, into the proper apex place, and above the fifth right costal cartilage, close to the sternum, without being able to detect upon opening the chest any dislocation of the heart. The validity of such experiments may be questioned; but there are clinical facts to show that the early pushing forward of the heart, . . . although it may be the rule, is not without exception. The fluid may cover the heart from the first.” It appears to me that Dr. Sturges’ observations are rational and practical; and in
dealing with particular cases it is well not to have too fixed or positive an opinion as to the position of the heart in pericardial effusion. Should the sac be quite free, there can be no doubt that in very abundant effusions the organ is covered by the fluid; and this is evident at necropsies under such circumstances, the body being in the usual recumbent position.

The next question is what effects, if any, are produced by pericardial effusion upon the walls of the heart and great vessels, when it becomes so considerable as to interfere directly with these structures? Sibson writes on this point: “The muscular walls of the ventricles are so thick, and their action is so powerful, that the direct effects of the fluid pressure upon them cannot be very great. But the pressure of the fluid tells inwards upon the weak and unresisting walls of the auricles, the vena cava descendens within the pericardium, and the pulmonary veins, so as to compress and lessen the cubic contents of those vessels and the auricles, and to resist and impede the currents of blood, on the one hand from the system along the cava, and on the other from the lungs along the pulmonary veins. This partial blocking of the double stream from the system and the lungs to the heart lessens the contents of the organ, and, tends to diminish the size of its cavities. At the same time the supply of blood to the aorta is lessened, and the ascending aorta is therefore also compressed by the fluid. The pulmonary artery, however, owing to the obstacle to the flow of blood through the lungs, tends to resist the pressure of the fluid in the swollen sac, and to remain distended.” This seems to be a correct description of the case of large effusions. Sibson, however, was further of opinion that in cases of pericarditis the compressing influence of pericardial effusion is counteracted by the protecting and sustaining covering of lymph, which to some extent shields the weaker parts of the heart, and strengthens the naturally feeble walls of the auricles and veins.

As regards the effects of pericardial effusion upon the action of the heart, it is believed that the systole of the auricles and ventricles is not restrained by such a collection; indeed, according to Traube, the systolic motion of the organ is greater than normal, the fluid being less resistant than the pericardium. The compression of the walls already referred to may, however, interfere with the diastolic distension, and thus diminish the flow of blood into the cavities, especially into the auricles. The direct interference with the entrance of the blood from the veins into the auricles, and impairment of the normal elastic traction of the lungs upon the walls of the heart, add to this difficulty.

It will be obvious that distension of the pericardium with fluid must interfere with neighbouring structures in proportion to its amount, and such consequences are chiefly seen in the respiratory apparatus. Some observers maintain that the portions of the lungs in front of the sac are pressed at first against the inner surface of the anterior wall of the chest. The ordinary effects of pericardial effusion upon these organs are complex. It necessarily embarrasses them more or
less, and large collections of fluid also press upon the bifurcation of the trachea and the bronchi, especially the left bronchus. Hence it is found in many cases that the upper lobes of the lungs, particularly the right, are abnormally distended with air, or in a state of inflation, and in time become the seat of catarrh also; while other portions are collapsed in various degrees. As the effusion increases, and attains an excessive amount, it pushes these structures to either side and backwards, at the same time compressing them more and more, the left lung especially, which in extreme cases may become almost or even completely collapsed and airless. In some instances rapid and repeated serous effusion has taken place into one or both pleurae in connection with great pericardial distension. Ewart states that pleuritic effusion is among the most common complications of severe pericardial effusion; that it frequently begins in the right pleura, but not uncommonly occurs ultimately on both sides; but that its occurrence belongs to the later rather than to the earlier stages. This condition is regarded as of mechanical origin, being attributed to pressure on the vessels in the roots of the lungs.

A very abundant pericardial effusion may press upon the oesophagus and descending aorta sufficiently to interfere with their channels. Whether the phrenic or other nerves within the thorax may be affected by the mere physical consequences of such an accumulation it is difficult to say; but some observers are of opinion that this may be the case, and it is highly probable, especially if the effusion be rapid.

A considerable pericardial effusion will tend to cause more or less protrusion of the corresponding portion of the thoracic walls, particularly in young subjects. When these walls have become rigid no such protrusion can take place. In a downward direction the diaphragm is not only embarrassed, but often considerably depressed, as well as the contiguous viscera, as chiefly evidenced by the liver.

(iv.) The course of events and the ultimate pathological results in acute pericarditis differ much in different cases. The natural tendency is for any serous or sero-fibrinous effusion to become absorbed sooner or later; sometimes very rapidly. According to Sibson's observations in rheumatic pericarditis the fluid after reaching its acme soon begins to lessen, and in from four to six days usually falls to the normal amount. There is every reason to believe, moreover, that even fibrinous exudation, up to a certain amount, can be absorbed completely, after undergoing a molecular fatty change; a little pericardial thickening or opacity at the most being left behind. The probability of such absorption is in inverse ratio to the extent and thickness of the lymph deposited, and to the duration of the inflammation. In respect of the "white spots" on the pericardium, it may be well to note again that fibrous patches resulting from pericarditis are usually distinguished by greater thickness and extent, irregular distribution, and special characters; and as a rule by the coexistence of adhesions. Very rarely irregular knob-like projections or pedunculated outgrowths are formed, and the latter may even become detached, and lie loose in the pericardial sac.
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In most cases, after absorption of the fluid, or where only lymph has been exuded, adhesions of various kinds and degrees are formed. At first these are soft and easily broken down, and on account of the movements of the heart firm and permanent adhesions are much less easily established than in other serous membranes. Loose adhesions of connective tissue are probably torn by the repeated pulling and stretching; and it is believed that the cardiac action considerably interferes with the circulation in the newly-formed vessels. On this question Sibson writes: "In most instances slight threads of adhesion form between the sac and portions of the right auricle, and often also between the sac and the apex and interventricular septum, that being the portion of the front of the heart that presents the least movement during the action of the ventricles. These soft threads of adhesion are generally drawn out by the oscillating movements of the heart, until they at length yield and break away, but sometimes permanent adhesions form which may be partial or universal."

I have a strong impression that there is a general tendency to make light of the conditions remaining after acute pericarditis, or at any rate not to regard them as of much consequence; and I feel it necessary, therefore, to call special attention to the fact that well-marked pericardial adhesions not uncommonly persist, particularly in young subjects, and subsequently often become of decided importance. A new growth of connective tissue takes place, originating mainly in the cells present in the exudation; the fibrinous portion taking no part in the process, but being absorbed after undergoing fatty degeneration. In severe cases the tissues of the pericardium itself contribute to the growth. As the subject of pericardial adhesions is separately discussed in this article no further reference need be made to it here. It must be noted that in exceptional cases an ordinary inflammatory effusion into the pericardium does not undergo absorption, but remains as a chronic collection, or may become hæmorrhagic or purulent. These conditions will also be referred to more fully hereafter.

According to the extent of the disease, cases of pericarditis have been divided into circumscribed or local, and diffuse, the latter being in many instances general or practically universal. Local pericarditis may be met with in any part, but is chiefly observed at the base, about the origin of the great vessels; and the inflammation may thence extend to the coats of the arteries, so far as they are covered by pericardium, and subsequently give rise to thickenings and callosities.

In the preceding discussion pericarditis has been dealt with only so far as it affects the sac internally. It must be mentioned, however, that in not a few instances the external surface of the pericardium is acutely involved at the same time, or alone; though it is more commonly involved in a chronic process. The condition has received the names of external pericarditis, mediastino-pericarditis, or pleuro-pericarditis when the contiguous surfaces of the pleura and pericardium are affected. This form of disease and its results will be more conveniently dealt with later, and in other connections.
Clinical history.—Acute pericarditis presents considerable differences in its clinical history, depending upon a variety of circumstances; and this fact must be clearly recognised at the outset, and always borne in mind in practice. At the same time the phenomena to be watched for and studied are definite, and when at all pronounced bear an obvious relation to the morbid changes which are associated with the disease. The signs observed by physical examination are of special clinical value, for the symptoms are not uncommonly anything but characteristic; while the more important of these signs can be investigated as a rule without much difficulty, and it is only by their aid that we can positively determine the pathological conditions of the pericardium. Indeed, it must never be forgotten that, when symptoms are practically absent or latent, they may reveal the presence of even serious acute pericarditis; and this statement applies still more to cases in which the inflammation is localised. Moreover, physical examination gives the only trustworthy information as to the progress of the morbid changes.

Taking a comprehensive survey of the circumstances under which acute pericarditis usually supervenes, it might be anticipated that an attack is not ushered in, as a rule, by any striking premonitory symptoms, such as rigors and the like; and experience confirms this conclusion. The fact must not be overlooked, however, that in certain classes of cases the illness may begin with phenomena of this nature; nor must it be forgotten that even rheumatic pericarditis may appear as a primary acute disease, before the joints or any other structures reveal the presence of the rheumatic condition.

Discussion of symptoms.—From what has been just stated, it may be gathered that it is useless to attempt to give a definite clinical picture of acute pericarditis, and it will be more practical in the first instance to consider individually the several symptoms which may be associated with this disease; remembering that they differ much in their exact nature, severity, and combinations in particular cases.

(i.) Subjective sensations.—Pain is a symptom to be looked for in the early stage of acute pericarditis; but it is by no means always present, nor does it bear any necessary proportion to the seriousness of the attack. I can corroborate, from personal observation, the statement that severe pain may be associated with a limited dry pericarditis of short duration; while, on the other hand, it is a familiar fact that in cases of large effusion no such sensation may have been complained of from first to last, or it may have been so slight and transient as not to have attracted any attention. In young children pain seems to be generally absent. Sibson made numerous careful and interesting observations on this symptom in rheumatic pericarditis, and some of his conclusions are incorporated in the following remarks. In the majority of cases where pain is present it is referred to the precordial region, extending usually from the right of the sternum at its lower two-thirds to the left nipple. This pain is more or less continuous, but varies in severity, being in exceptional instances very intense. In other
acter it is described in different cases as dull, aching, shooting, stabbing, burning, or tearing. Sibson noted that it came on, as a rule, at an early stage, afterwards diminishing; and usually relief, which was permanent, came when the effusion was at its height. Pain may, however, either precede or follow friction sound. Occasionally a return of the pain occurs with a relapse. The suffering is often increased by deep pressure or percussion; and now and then there is tenderness without spontaneous pain. Sibson observed in many cases that the skin over the region of the pericardium was tender and sensitive; so much so in some instances as to forbid the slightest manipulation of the chest, and to make a full examination of the heart impossible. Sometimes this superficial hyperesthesia is certainly very remarkable. In other cases the structures of the intercostal spaces seem to be tender.

Another not uncommon seat of pain or tenderness, or both, is the epigastric region, where, according to some writers, it is even more frequent than in the precordial. The tenderness is said to be most marked at one or other of the costal angles, and is particularly brought out when upward pressure is made. Epigastric pain comes on, as a rule, later than that over the heart, and in a considerable proportion of Sibson's cases it appeared when the effusion was at its height. Both varieties are likely to be increased by the act of respiration and by bodily movements. Sometimes painful sensations radiate in different directions from the central points. A deep pain in the chest, between the shoulder-blades, was noticed in a few cases by Sibson; it was increased by swallowing or eructation, and occasionally was only thus brought out. He thought that in these instances the pain was seated in the back of the inflamed pericardium; and he also believed that pain and fulness after food might result from pressure made by the distended stomach over the lower and posterior part of the sac. In exceptional instances pain of an anginal character, shooting up the left side of the neck, to the ear, to the shoulder, or down the arm, is associated with acute pericarditis; but endocarditis has almost always been present at the same time, and generally chronic valvular disease also. The sensations just discussed are believed to be located mainly in the sentient nerves distributed to the surface of the heart, the pericardial sac itself and the portion of diaphragm incorporated with it, or the pleura covering the pericardium. They are often associated together in different combinations. Moreover, there may be pain in one or other side, evidently of pleuritic origin, or referred indefinitely to the chest. Taking all his cases together, Sibson found that there was pain of some kind over the heart or pericardium in 70 per cent.

Other subjective sensations besides those actually painful are not uncommonly complained of in acute pericarditis, as the disease progresses; and especially if a large accumulation of fluid takes place. They are described in different cases as feelings of precordial uneasiness, oppression or pressure, a weight or load over the heart, tightness, or ill-defined distress and anxiety. The respiration may be distressed; and not only may the patient be conscious of the disturbed heart, but sometimes there
is a distinctly painful form of palpitation. Baumler has noticed painful sensibility of the left side of the larynx, increased by every movement of the heart.

(ii.) Disorders of the cardiac action and pulse.—It might naturally be expected that acute pericarditis would affect the action of the heart in various ways. In the early stage the heart is excited and irritable, as evidenced by increased rapidity and force of the beats, the movements in some instances being more or less tumultuous. Subsequently, not only as the result of large effusion, but also of the implication of the myocardium and its nerves, as well as of other influences, the cardiac action becomes more or less embarrassed and ineffectual, and this may culminate in marked feebleness or exhaustion, with irregularity and intermittence, or even faintness or actual syncope which, in exceptional instances, has come on suddenly or very rapidly, and proved fatal. With regard to the frequency of the pulse, according to Sibson, "it rises in number as the disease rises in intensity, is at its greatest rapidity when the disease is at its acme, and falls in number as the disease declines." "During the early stage the pulse usually mounts up to 90, 100, or even 120; but later on it tends to become more rapid, and in rare cases reached 160." It may, however, not be much changed from the normal, or from what it was before the pericarditis supervened; or after an initial acceleration it may soon subside. In exceptional instances the pulse is retarded in the course of the disease. A much quickened pulse-rate, 120 or 130, without adequate rise of temperature, is said by Dr. Cheadle to be very characteristic of the subacute pericarditis of early life. In the early stage the pulse is generally full and strong, and may be increased in tension; as the case progresses, it becomes small, weak, often dicrotic, and of very low tension. Dr. Ewart, however, draws special attention to the large and slapping pulse which he has frequently observed in pericardial effusion. He writes: "The peculiarity of the pulse is its great size and velocity of impact, and the sudden collapse of the wave. In fact it is Corrigan's pulse, almost of a typical kind, though never so extreme as in well-marked aortic regurgitation." Irregularity or intermittence may accompany a similar disturbance of the cardiac rhythm; occasionally this is an early phenomenon, but usually comes on later. It has been stated that in some cases of copious pericardial effusion the left carotid and radial arteries are smaller and pulsate less forcibly than the corresponding arteries on the right side (Traube). The sphygmograph has been much used to investigate the pulse in cases of acute pericarditis, but I venture to doubt whether it is of much practical value. Speaking from personal experience of this disease, I think it must be acknowledged that no definite description of the pulse can be given; but at the same time the study of it in individual instances affords most useful information, and it needs to be watched at frequent intervals. In grave cases it may become almost imperceptible. The pulsus paradoxus has been observed occasionally in large pericardial effusions.
(iii.) Respiratory system.—Some disturbance of breathing is noticed in the great majority of cases of acute pericarditis, varying much in its degree and exact characters, but often well marked or even decidedly grave. By pain in the early period respiration is rendered quick and hurried, but restrained and shallow; and this cause may also modify the movements later, when the physical effects of pericardial effusion, as well as other influences, especially the cardiac changes, come into play. If there be much fluid, actual dyspnœa supervenes, the respirations increasing in frequency, with marked activity of upper costal breathing, but more on the right side than the left. As it accumulates, the breathing becomes more and more difficult and laboured, the alæ nasi work, the extraordinary muscles are called into play, there is a corresponding sense of oppression, distress and air-hunger, and the patient may have to be propped up more or less. In extreme cases the dyspnœa is very urgent, the respiratory movements are greatly impeded, and there is persistent orthopnœa, or the patient instinctively bends forwards to seek relief. As a rule it is more comfortable to lie on the left than the right side, but dorsal decumbency is usually preferred. Occasionally the dyspnœa is intensified paroxysmally. As the fluid is absorbed the respirations fall, and the breathing improves; but a relapse may cause fresh disturbance. With regard to the pulse-respiration ratio, even at the early period it may be altered from the normal to 3:1; and later the proportion may come to be 2 or 2½:1. The difficulty of breathing interferes with the act of speaking; and changes in the voice have been described in exceptional instances by Sibson and others, and attributed mainly to pressure upon or implication of one or both recurrent nerves. A short, irritable, spasmodic cough is not uncommon with a large pericardial effusion, and there may be a little mucous frothy expectoration. Distressing and painful hiccup is an occasional symptom, attributed to implication of the phrenic nerve in the inflammatory process.

(iv.) Dysphagia.—Difficulty or pain in swallowing is occasionally noticed, mainly the result of the pressure of a large pericardial effusion upon the oesophagus; but sometimes it appears to be due to nerve-irritation. Deglutition is more difficult in the recumbent posture, and is made easier by raising the shoulders and bending forwards. In exceptional cases the difficulty is only associated with swallowing solids, or is brought on by oesophageal spasm induced by an attempt to drink. Rarely a feeling of spasmodic choking in the throat or along the gullet is complained of.

(v.) General symptoms and appearance.—More or less pyrexia may be expected in cases of acute pericarditis, but it does not present any special course or characters. Its manifestation in rheumatic cases may not be attended with any increase of temperature previously raised; it seldom rises above 102° or 103° at any time, and may soon subside. Sometimes it is practically normal throughout, or only slightly elevated, from 99° to 100° or 101°, especially in children. It is affirmed that rapid absorption
of inflammatory products may occasion some rise of temperature. As a rule, strength is fairly maintained; but in some instances, especially in children, there is marked prostration. In severe cases of acute pericarditis, especially when associated with endocarditis, the expression generally indicates anxiety, distress, or depression; and the face is flushed, dusky, or palid, or presents alternating hues. Rarely it has a muddy or glazed appearance. The eyes at the same time are dull, heavy and injected. Sibson noted a marked change in the appearance of the patient in four-fifths of his cases, to which he attached much importance: as the complaint subsided he found that the aspect quickly improved, the eyes becoming bright and clear, the cheeks rosy, and the expression often quite suddenly cheerful. This observer attributed the flushing and pallor of the face to the influence of the inflammation on the nerves at the surface of the heart, inducing reflex dilatation or contraction of the arteries of the head and face. He stated that the flushing seemed to tint the face all at once.

The most striking general symptoms in the grave forms of acute pericarditis are those indicative of interference with the aeration of the blood, and of general venous obstruction. The patient then presents a more or less livid or cyanotic appearance; sweating, often profuse; fulness of the veins of the neck, sometimes with pulsation; and in extreme cases coldness of the extremities. Possibly dropsy of the legs may occur. A large effusion in children is said to affect the action of the heart more rapidly than in adults, and to lead to an earlier interference with the circulation. In these subjects progressive anæmia and wasting are in some instances pronounced symptoms. Perspiration was observed by Sibson to be usually copious when there was flushing. The amount and characters of the urine will depend very much upon the condition with which the pericarditis is associated. It tends to be deficient in quantity, and to present the usual changes associated with the rheumatic and febrile states. Albuminuria may occur altogether independent of renal disease.

(vi.) Nervous symptoms.—Patients suffering from pronounced acute pericarditis are generally very restless, but movements may be checked by the rheumatism. Headache and sleeplessness are frequent symptoms, and slight delirium is not uncommon. Vomiting is sometimes a marked symptom in acute pericarditis, and is regarded as of nervous origin. In exceptional cases nervous disturbances become very prominent, and may be grave, such as delirium, either active and noisy, or even violent and maniacal, chiefly nocturnal; or low and muttering; sometimes a transition from one to the other variety takes place. The condition may resemble delirium tremens, the patient being strange in manner, excited, and incoherent; or there may be a tendency to stupor, semiconsciousness, temporary insensibility, or actual coma; or to motor disorders, such as subseultus tendinum and jactitation, “risus sardonicus,” clonic or tonic spasms, rolling of the head from side to side, choreiform movements, general convulsions ending in extreme exhaustion,
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Tetanic rigidity; or to curious emotional attacks in early life, in which the child is moved to tears or laughter by a word (Cheadle); or even to temporary insanity, usually with taciturn melancholy, and often with hallucinations; this derangement may last some time, but is ultimately recovered from. The particular symptoms of this class and their combinations differ much in different cases, and delirium may pass into coma. They cannot, as a rule, be referred directly to the pericarditis, but depend rather on the disease to which it is secondary, its associated complications—hyperpyrexia in some instances, the state of the nervous system, want of oxygenation of the blood, the previous habits of the patient, or other circumstances. Some authorities, however, attribute the phenomena to the influence of the pericarditis upon the nervous system; and Bright believed that such an influence can be communicated through the phrenic nerve to the spinal cord, and is the cause of choreic and tetaniform affections. Dr. George Balfour writes: "The occurrence of delirium in the course of rheumatic fever ought at once to direct attention to the heart; and the sudden occurrence of spasms or coma in chronic renal disease is only too frequently found to be associated with pericarditis, both of these phenomena being probably caused by the saturation of the blood with the products of retrograde metamorphosis, due to the sudden development of the inflammation." It is important to note, however, that even in the gravest cases of acute pericarditis, ending fatally, and accompanied with other intrathoracic inflammations, there may be no marked nervous symptoms throughout, the patient being perfectly clear to the last.

Sturges described a very fatal form of pericarditis in children, with little or no effusion, exudation being followed by rapid adhesion; and he referred the grave symptoms observed in these cases to the nervous system. Dr. Cheadle also calls attention to occasional cases in such subjects which run an acute course with dangerous symptoms; but he states that, as a rule, they arise when pericarditis occurs late, when the heart is already seriously damaged by previous attacks of endo- or pericarditis, and when the secondary changes of dilatation and hypertrophy, and perhaps adherent pericardium, have already advanced to a marked extent (vide vol. iii. p. 45).

Physical signs.—In discussing the physical signs of acute pericarditis, it is convenient to recognize certain stages corresponding to the progress of the morbid changes already described; although it must be clearly understood that there is no actual line of demarcation between them, the conditions which give rise to these signs being commonly present at the same time. It may be remarked that the excited or turbulent action of the heart which often occurs at the onset of the disease will be evident on examination, but there is nothing characteristic in this disturbance.

First stage.—During the early period the signs to be looked for are those indicative of abnormal states of the contiguous pericardial surfaces, which are pressed and rubbed against each other during the movements of the heart. They are, commonly known as pericardial friction—fremitum or
thrill, and friction murmurs or sounds; signs which must be considered in some detail. Many deny that any phenomenon of this kind can be produced by mere increased vascularity and dryness of the surfaces, but in my opinion a faint friction murmur may certainly be thus originated. It is, however, to the fibrinous exudation that the more pronounced and characteristic signs of the early stage of acute pericarditis are due. They can, in my opinion, be brought out only when the conditions producing them exist on the anterior aspect of the heart, although some writers have made a contrary statement; and it is highly probable that when the inflammatory lymph is of a very soft consistence, it may not give any definite sign perceptible on physical examination.

(i.) Pericardial friction-fremitus or thrill.—The tactile sensation thus named is practically only recognisable in a comparatively small proportion of cases of acute pericarditis, and when present it is always accompanied with a loud friction-sound. For the detection of this sign careful palpation with the finger-tips may be needed, and I believe that it can thus be made out more frequently than is generally supposed. It depends more immediately upon the amount and characters of the exudation, though it is also influenced materially by the force of the heart's action.

When any abnormal sensation is felt over the precordial region, the chief point to be determined is whether it is a pericardial fremitus or an endocardial thrill. It must suffice to summarise here the more characteristic features of a pericardial fremitus, and to any one practically acquainted with the usual endocardial THRILLS the points of difference between them will be at once apparent.

(a) A pericardial friction-fremitus has no definite "focus of intensity" (Sibson), and varies much in its seat and extent. As a rule its area is circumscribed, and it is felt more towards the base of the heart or over the middle of the precordium; sometimes it is limited to the apex. Now, and then, however, the sensation is perceptible over a considerable extent of surface, or in more than one spot. (b) It always gives the impression of being peculiarly superficial, as if the condition producing it were close under the finger. (c) The rhythm is practically systolic, the fremitus being associated with the cardiac impulse; it usually begins and ends rather abruptly, and there is no shock at the close: sometimes it is irregular in rhythm, differing in exact time in successive beats. (d) In quality a pericardial friction-fremitus gives more or less the impression of the rubbing together of rough surfaces, and in different cases it is described as harsh and grating, rasping, vibrating, or cracking. Sibson speaks of it as being in many instances a sensation of a thrill, but in my experience the feeling has certainly never been like that of an endocardial thrill. (e) As a rule this sign is short-lived and transient (Sibson), and, should it last any time, often changes from day to day in its situation, extent, and character. Pericardial friction-fremitus may be simulated by one of pleuritic or mediastinal origin, brought out by the movements of the heart; and this fact must not be forgotten.

(ii.) Pericardial murmur or friction-sound.—It is by the adventitious
sounds heard on auscultation that, in the large majority of cases, the early stage of acute pericarditis is recognised. Some writers distinguish between a pericardial murmur and friction-sound, and Sibson made a great point of this distinction; but there is no practical line of demarcation between them. In the following remarks, therefore, I shall employ the term "pericardial friction-sound" inclusively, merely remarking that the so-called murmur may be regarded as representing the minor degrees of this sign, and that now and then an adventitious sound of pericardial origin may no doubt closely resemble an endocardial murmur in quality.

It is requisite to have a comprehensive and intelligent conception of the more characteristic features of pericardial friction-sounds, so as to be able to contrast them with those of endocardial murmurs; but as a rule they are easily distinguished. Moreover, by careful attention to the special qualities of the sounds heard, it is practicable in many cases to arrive at a tolerably definite notion of the conditions of the pericardium upon which they depend. It may be observed here that pericardial friction-sound also may unquestionably be simulated by one of pleuritic origin, or by a sound originating in the mediastinal cellular tissue over the pericardium.

(a) While usually more or less circumscribed in extent, pericardial friction-sound does not correspond in any way, as regards its situation or its point of maximum intensity, to any of the recognised endocardial murmurs. In some cases it is audible extensively, though not of the same loudness throughout its area; but even then it is generally defined with remarkable abruptness, and is never conducted in the directions peculiar to the several intra-cardiac murmurs; nor, according to my experience, can it ever be heard over the back of the chest. During the early stage of acute pericarditis friction-sound never extends beyond the region of the heart, but in the later it may do so in exceptional instances. When associated with a fremitus it usually spreads, as from a focus, in all directions more or less beyond the area where this sensation can be felt.

(b) As a rule pericardial friction-sound has a double or to-and-fro rhythm, being both systolic and diastolic; but in some instances, or over certain parts of the heart, it may be confined to the systole. In pronounced cases the two parts are of about equal duration, each sound seeming to fill up its respective space, leaving a short interval of silence between the two (Sibson). They may, however, occupy the whole time of the cardiac movement, thus often giving at first a confused impression to the ear; but intermissions can be made out. As regards the cardiac sounds, the pericardial murmur seldom corresponds exactly in rhythm, with either, and is prolonged beyond them, while they are often distinctly audible through it; though, on the other hand, the friction-sound may be so loud as to drown them entirely. Moreover, its precise time is frequently irregular, varying with successive beats of the heart. This is more especially noticed in connection with the diastolic portion, which is usually not so loud as the systolic. A double "to-and-fro" adventitious sound heard in connection with the cardiac movements, of maximum intensity
at the same spot, is regarded as highly characteristic of pericardial origin. It has been stated that four murmurs may be audible; the two sides of the heart each producing a systolic and diastolic murmur of different duration; but that most frequently three are heard, one presystolic, belonging to the systole of the auricles, and two longer sounds, corresponding to the systole and diastole of the ventricles. Rarely pericardial friction is divided into several parts.

(c) While varying much in its intensity, pericardial friction-sound strikes the ear as being peculiarly superficial; and this character is more pronounced in proportion to its loudness. Sibson spoke of it as a "surface noise."

(d) The precise characters of a pericardial friction-sound vary considerably within well-recognised limits, according to the nature of the conditions upon which it depends. In the large majority of cases it conveys to the ear a distinct impression of the rubbing together of contiguous surfaces during the cardiac movements; in short, it is of the quality of a "friction-sound." In its lesser degrees it is soft or grazing, whiffing, brushing, or rustling; but its more pronounced varieties are described by such terms as harsh, rough, grating or vibrating, and cracking, like the bending of new leather. Bouillaud classified pericardial friction-sounds as grazing, new leather sound, and grating, which are the common varieties. Sometimes they resemble the rubbing of sand-paper. Under certain circumstances the sound is more of a crackling (as of paper or parchment), clicking, churning, or rumbling character, or it may be scraping, scratching, or sawing. It has also been described as "sticky." While thus various, pericardial sounds are as a rule entirely different in quality from endocardial murmurs. Moreover, the double pericardial friction-sounds never begin with an accent or shock, but begin, continue, and end, as a rule, with the same tone throughout (Sibson). When pericardial friction-sounds and endocardial murmurs exist together, the combinations may be very peculiar and difficult to define.

(e) Tests.—In certain cases in which a pericardial friction-sound is not distinctly audible, but its presence is suspected, or where it is doubtful whether an adventitious sound heard on auscultation be pericardial, endocardial, or pleuritic, the difficulty may be cleared up by the judicious application of certain recognised tests. These may also help in affording a more correct knowledge of the conditions of the pericardial surfaces upon which a friction-sound depends.

(a) Pressure test.—Firm but not too forcible pressure with the stethoscope over different parts of the region of the heart has long been known as an important and useful test of pericardial friction-sound. It may bring out this sign when not previously audible, especially over the lower two-thirds of the sternum (Sibson). Its effect upon the sound, when present, may be to intensify it and make it louder; to enlarge the area over which it is heard; to modify its duration and rhythm, rendering it more prolonged and continuous, or making it double—systolic and diastolic—when previously only systolic; to alter its character, tone, and
pitch, causing it to become more harsh and rough, and especially grating or creaking, or these qualities come out more prominently under pressure (Sibson described a peculiar double sound thus elicited, like the noise made by sharpening a scythe); or to silence the natural cardiac sounds previously heard, or even mask endocardial murmurs.

(8) Respiration test.—The act of respiration may unquestionably produce a definite influence upon pericardial friction-sound, especially as regards its extent, less frequently as to its intensity and quality; and possibly some help in diagnosis may thus be afforded in doubtful cases. It is generally stated that inspiration always increases pericardial friction-sound. Sibson observed that the area of the friction-sound increased below during inspiration in a large number of cases; while in a much smaller number it increased above during expiration. It became more loud or harsh sometimes during expiration, sometimes during inspiration; and in one instance it disappeared at the end of a deep breath. I may refer here to pleuritic friction simulating pericardial. As a rule it can be distinguished by its situation at the left border of the pericardium, and by its cessation when breathing is stopped, but certainly not always.

(γ) Effects of exertion and posture.—Should a pericardial friction-sound not be heard at all or but feebly, in consequence of weak action of the heart, it might possibly be brought out or made louder by exciting the organ by some kind of effort. Moreover, it certainly may be intensified or increased in area by bending the body forwards; while occasionally it is audible in the recumbent but not in the sitting posture. Change of position may affect the locality and extent of this sign in certain cases. Personally I doubt whether the tests mentioned under this head are of much practical value, and at any rate special discretion and caution are required in carrying them out.

(δ) Variability.—Marked changes in the site, rhythm, intensity, and characters of pericardial friction-sound from day to day, or within shorter periods, constitute a series of most important tests in a large number of instances.

Stage of effusion.—When fluid collects in the pericardial sac in any quantity, it may be expected to give rise to a very definite group of physical signs, varying in their degree according to its amount and other circumstances. It must not be forgotten, however, that rapid adhesion may take place without any effusion, so that the phenomena of this stage may be entirely wanting, especially in children. Conversely, it occasionally happens that a large quantity of fluid accumulates very rapidly and insidiously without forerunning friction signs, or at any rate without their detection. The possibility of considerable cardiac dilatation, as described by Dr. John Broadbent, must also be borne in mind, lest a wrong diagnosis of pericardial effusion be made.

We shall first consider how pericardial effusion may modify the friction phenomena. Sibson made numerous observations on this problem, and his conclusions were believed by him to support his own views of the position of the heart in these cases. According to his
observations the tendency of the effusion is to shift the whole region of actual friction, and with it the friction-sound, upwards; and steadily to increase its area in this direction and to the right and left. In the large majority of cases he found the area of friction-sound greater at the time of the acme of the effusion than before; in a few it remained the same; in two only was it less than before. In two instances the friction-sound disappeared during the acme, but Sibson attributed this mainly to lowered heart power. He observed that the tendency is for the sign to increase in intensity also, but in this respect the exceptions were more frequent. It may be stated with certainty that even large effusions do not necessarily obliterate the friction-phenomena; indeed there may be an abundance of fluid, at least as much as two pints, in the pericardium, while these signs are pronounced. Dr. George Balfour goes so far as to affirm that if a friction-sound be once heard over the base of the heart in front, no amount of subsequent effusion suffices to efface it. I do not think that this statement will hold good absolutely, and friction-sound over other parts of the heart is likely to be completely silenced as a rising tide of fluid separates the two pericardial surfaces.

I proceed now to discuss the more positive signs which are associated in various degrees with pericardial effusion.

1. The tendency of pericardial effusion, when in sufficient quantity, is to cause proportionate bulging or prominence of the corresponding portion of the front of the chest, and occasionally this is a very striking sign. Some writers have asserted that this condition leads to a uniform enlargement of the left side; but although there may be a certain degree of general distension the prominence is always greater in front. In the case of a large effusion the margin of the sternum and the left costal cartilages are pushed forwards, while the ribs are raised bodily upwards, and the intercostal spaces widened. In extreme instances the fullness may extend from the second to the sixth or seventh cartilages, but chiefly from the fourth to the sixth; the spaces are sometimes felt to be quite smooth, and an obscure sense of fluctuation may possibly be detected in them. Sibson stated that in very large effusion the dorsal portion of the spinal column deepens itself and is curved backwards. Bulging is naturally more easily produced in children and growing subjects, on account of the yielding condition of the chest walls; while it may be entirely prevented by rigidity of these walls, which thus adds seriously to internal embarrassments by the fluid. The enlargement has been partly attributed by some writers to inflammatory paralysis of the intercostal muscles.

Dr. William Ewart (19) regards what he calls the "first rib sign" as important in the diagnosis of considerable pericardial effusion. This is an altered relation between the left clavicle and the first rib, so that the upper edge of the latter can be felt as far as its sternal attachment. He writes: "This points to a raising of the clavicle not only in its outer but also in its inner portion, and to a relaxation of the ligament between
it and the first rib. The left clavicle is apparently lifted to a higher level than it is possible for the first rib to reach."

A prominence of the epigastric region may be noticed in cases of abundant pericardial effusion, due partly to the fluid itself pressing down the diaphragm, partly to the liver, which is also depressed and congested.

In his case Dr. Samuel West observed the rare phenomenon of a peculiar elastic semi-fluctuating depression in the epigastrium, which he regards as additional evidence of effusion having its seat in the pericardium. Dr. Clifford Allbutt has met with a similar phenomenon.

2. Certain signs of pericardial effusion, associated with the cardiac movements, as revealed by the impulse and apex-beat, demand careful study:—

(a) There can be no doubt that one of the obvious effects of a free and uncomplicated accumulation of fluid in the pericardium is a real or apparent elevation of the apex-beat, which seems at the same time to be carried somewhat towards the left. Moreover, the movement becomes unusually extensive in an upward direction, its diffusion being often easy to recognise by inspection and palpation. According to Sibson's observations there is, as a rule, a relation between the extent of the effusion and the height of the impulse. This he found raised so that its lower boundary corresponded to the fourth or even the third space or cartilage, being also felt at or to the left of the nipple line. The diffusion varied according to the position of the lower boundary; but in the large majority of cases, at the time of the acme, it extended above this boundary to the extent of one or more of the higher intercostal spaces. In exceptional cases the impulse was diffused from the fourth to the second spaces, but generally it was confined to the fourth and third, or the third and second spaces. Sibson attributed the raising and diffusion of the impulse to elevation of the heart by the fluid, and to enlargement of the right ventricle and pulmonary artery from obstruction of the flow of blood through the lungs. He believed, in common with other authors, that it is the actual apex-beat which is felt, displaced upwards and to the left. At the present time, however, most writers regard this opinion as erroneous, and consider that the impulse is communicated by a higher portion of the heart. Thus Dr. George Balfour believes that the true apex is pushed inwards by the effusion, and removed from the anterior wall; while the part of the heart actually in contact with the chest wall is nearer the base of the ventricles. Dr. William Ewart (19) also writes: "That an impulse can usually be felt there (at the third space) is not surprising, since the antero-posterior diameter of the chest at that level (between sternum and spine) is not much greater than that of the heart itself, whilst the left lung no longer intervenes between the latter and the chest wall. The impulse is not, however, that of the apex of the heart, but rather of its base." My personal observations lead me to agree with these views on the whole, but there may be conditions in certain cases to cause actual uplifting of the apex-beat.
Series of figures (Nos. 36 to 43), from cases described by Sibson, illustrating the morbid conditions in pericarditis and the physical signs associated therewith. The black spaces correspond to the pericardial dulness, the curved lines to the impulses, and the zigzags to the friction-sounds. In Fig. 41 there is complete adhesion of the pericardium to the heart.
Occasionally it has been noticed in acute pericarditis with effusion that the apex-beat is somewhat lower than normal. This may be due to enlargement of the heart; but it has also been attributed to the presence of a large quantity of fluid pressing down the diaphragm; or it may be associated with a more median and vertical position of the heart, the aortic arch becoming slightly straightened (Ewart).
Over the pulmonary artery at the base a double beat is sometimes felt, the second being the diastolic shock due to the closure of its valves.

(b) The next change to be noticed in pericardial effusion is a progressive weakening of the cardiac impulse from below upwards. This depends mainly upon the amount of the effusion, but partly upon feebleness of the heart’s action. When the fluid is in moderate quantity there is often, as just stated, a strong impulse over the upper spaces, its lower and outer boundaries being also well defined. As it accumulates, however, in increasing abundance, and separates the heart from the chest wall, the cardiac movements become more and more obscured, until finally they may be wholly lost, and not perceptible over any portion of the precordial region. This sign is occasionally very striking in a case of inflammatory pericardial effusion when it first comes under observation.

(c) It is a disputed question whether pericardial effusion can produce any definite change in the character of the cardiac movements, tactile or visible. Certainly the impulse observed over the upper part of the chest may be more or less undulatory; and a wave-like motion has been described, which can be seen but not felt, and is supposed to be communicated to the fluid by the action of the heart. I must say that I have never been able to recognise this phenomenon positively. Some authorities regard an undulatory impulse as a sign, not in favour of pericardial effusion, but against it.

(d) In some cases of pericardial effusion the rhythm of the impulse has been described as lagging behind the ventricular systole in a peculiar way. Irregularity, with or without inequality in the strength of the beats, may become very marked as the result of embarrassment of the heart by a large collection of fluid, and of changes in its walls.

3. One of the most frequent and characteristic signs of pericardial effusion of any extent is an increase in the area of the normal cardiac dulness, with change in its shape and outline; and not uncommonly these alterations are so pronounced as to attract immediate attention in cases of acute pericarditis. The exact quantity recognisable by percussion cannot be definitely stated, and no doubt it varies under different circumstances; but I believe that methodical and careful determination of the cardiac dulness may afford valuable information in cases where the fluid is present in comparatively small quantity. It is necessary to study systematically and thoroughly both the superficial or absolute and the deep or relative cardiac dulness. As the patient lies on his back the increase of dulness is first observed towards the base of the heart. The limits ultimately reached vary much in different cases. The extension takes place chiefly in a lateral and upward direction, the length and breadth of the dulness being thus increased; the former usually preponderating. In most instances it reaches the third cartilage or space, but may extend as high as the second cartilage or first space,
or even above the clavicle. Dr. Sansom maintains that whenever marked dulness extends above the third rib there is a strong probability of pericardial effusion. Over the sternum, which is absolutely dull, as the fluid increases the dulness reaches a higher level than over the costal cartilages, and in extreme cases it may reach its upper margin. From side to side at its greatest width the dulness may extend from an inch or more to the right of the lower part of the sternum, or the right mammary line, to an inch outside the left nipple, or even to the left axilla. In a downward direction it seldom passes below the sixth rib, but in extreme cases it may be made out as low as the seventh or eighth rib, and be indistinguishable from the hepatic dulness. A sign suggested by Dr. Rotch, as valuable in the early diagnosis of effusion into the pericardium, is the presence of dulness in the fifth right intercartilaginous space, due to the accumulation of the fluid in the right corner of the sac; but this sign is by no means invariably to be trusted.

A notable feature of the dulness in cases of considerable pericardial effusion is its shape, which corresponds with that of the sac itself. Thus it narrows from below upwards, assuming a more or less triangular, pyramidal, or, more strictly speaking, pyriform or pear-shaped outline, with its truncated or "peaked" apex above, and its base below, at the level of the lowermost limit of the fluid. The left border has been described as usually somewhat curved, or indented at its upper part, while the right is more nearly vertical. Dr. Ewart well describes the outline of a large effusion as "that of a bag of fluid spreading out at the base." In its diagnosis from cardiac dilatation he lays stress upon the projection of lower angle of the dulness to the right, as well as to the left; a prominent angular outline being made out by careful percussion instead of the normal outline of the heart. When the pericardium becomes extremely distended, the characteristic shape is more or less modified, and may ultimately be altogether lost. Shattuck renounces all faith in the doctrine of a pyriform or pyramidal area of dulness in pericardial effusion, but I differ from him entirely on this point.

In cases of pronounced pericardial effusion the extreme degree of the dulness is very striking. Dr. Sansom insists on the importance of the well-defined transition from the resonace of the lung to such dulness as a factor in the diagnosis of this condition, and in many cases the contrast is certainly very remarkable. In other instances, however, it must not be forgotten that the distended pericardium is overlapped by the margins of the lungs, which yield a superficial resonance; and that its full extent can then be made out only by very careful percussion beyond the limits of absolute dulness. A large effusion imparts an increased sense of resistance to the fingers.

Another important point is that the dulness of extensive pericardial effusion can be made out distinctly towards the left, considerably beyond the position of the apex-beat, which is then only to be recognised by auscultation.
Fig. 44.—Illustrating "Rotch's sign" (dulness in the right 5th space, δ to H); also contrasting the angles (on either side of H) of the dulness as due respectively to effusion and to dilatation. The heart's outline is normal in size and position. The outer lines are those of the dulness in moderate effusions. The "supra-hepatic line" (dotted) and the "hepatic line" limit the normal "modified" dulness of the liver; and H is placed on the absolute dulness. (After Ewart.)

Fig. 45.—Outline of a large effusion, which the pulmonary fringes overlap, and of its total area of dulness. The liver is depressed from its normal level H (infra-sternal notch) to the tip of the xiphoid. F shows the position of the finger for the "first rib sign." (After Ewart.)
Fig. 46.—Outlines of the total and of the absolute areas of pericardial dulness. A, position of the cardiac apex (6th space) in the effusion. The dulness is shown by the arrows to extend far beyond and below A. The right auricle (not shown) descends with the diaphragm. T, the infra-mammary patch of tubular breathing. (After Ewart.)

Fig. 47.—The "posterior pericardial patch of dulness" (shaded); and the "posterior pericardial patch of tubular breathing and egophony." (After Ewart.)
The rapid development of increased precordial dulness while a patient is under observation is strongly in favour of accumulation of fluid in the pericardium, and under circumstances where acute pericarditis might be anticipated this sign must be specially looked for. It may soon become quite pathognomonic, but the possibility of the occurrence of acute dilatation must not be overlooked.

4. The auscultatory signs which may directly result from effusion into the pericardium demand brief notice. The tendency of the fluid itself, as it increases in amount and rises higher and higher, is to weaken the heart-sounds in a progressive manner from apex to base; or they may seem deep and distant. These effects may be due both to imperfect transmission of the sounds through the intervening fluid, and to embarrassment with enfeeblement of the cardiac action. Most commonly in pronounced pericardial effusion the sounds are weak or perhaps inaudible over the region of the normal apex-beat, and for some distance upwards, but become gradually more perceptible towards the base of the heart, where they may be well heard; over the pulmonary artery the second sound may actually be intensified. In cases of extreme effusion the sounds may be practically absent over the whole precordial region.

Some observers have described a basic systolic murmur as a sign of pericardial effusion, the result of pressure by the fluid upon the great arteries. I have never met with such a murmur within my own experience, but it may possibly occur. On the other hand, pericardial effusion may certainly obscure or render inaudible endocardial murmurs previously heard.

5. Signs connected with neighbouring structures.—The effects produced on the lungs, especially the left, by a large pericardial effusion, are likely to be indicated by more or less pronounced signs, which, however, will vary in different cases according to their exact nature and degree. The respiratory movements over the upper part of the chest are often obviously excessive, but especially on the right side; and should the fluid be very abundant, a striking contrast will probably be observed between the activity of the two sides, the movements on the left being very deficient. Over the region of absolute cardiac dulness there will be entire absence of breath-sounds, as well as of vocal fremitus and resonance. Beyond its limits there may be hyper-resonance and puerile breathing; and towards the left side the percussion sound is occasionally somewhat tubular, and the breathing bronchial or tubular, with increased vocal fremitus and resonance. Ewart (19) calls attention to a sign which, although not constant, should, he says, be looked for in severe cases; namely, tubular breathing below the right mamma. He describes it as situated usually in the nipple line, a little above the hepatic line, and he states that it is sometimes restricted to expiration. Dry rhonchi of various kinds may be audible in severe and protracted cases, the result of catarrh of the bronchial tubes.

In considerable pericardial effusion the condition of the left lung may give rise to a definite group of signs at the back of the chest on that side; namely, a limited area of deficient resonance or actual dul-
ness, about the size of a crown piece, generally referred to the vicinity of the angle of the scapula, with increased vocal fremitus, bronchial or tubular breathing, and bronchophony or egophony. Sansom regards these as valuable signs in children and young subjects. Ewart attaches special diagnostic importance to the dulness, which he describes as follows:—"Whenever fluid is effused into the pericardium the normal resonance is modified at the left posterior base in a most definite way. A patch of marked dulness is found at the left inner base, extending from the spine for varying distances outwards, usually not quite so far as the scapular (angle) line, and ceasing abruptly with a vertical outer boundary. Above, its extension is also variable according to the size of the effusion; commonly it does not extend higher than the level of the ninth or tenth rib, and here again its horizontal boundary is abrupt. Its shape is that of a square, and it is quite unlike that of any dulness arising from pleuritic effusion." He attributes this patch of dulness to the altered dorsal relation of the liver, and states further that partial dulness also extends for a short distance to the right of the corresponding vertebrae, and that, when the effusion is considerable, the extension of the patch in the right chest may become almost absolutely dull. With regard to auscultation signs, this writer affirms that over the dull patch to the left of the spine respiratory sounds are found to be absent and the voice sounds feeble. He locates tubular breathing and egophony to a patch about two inches in diameter immediately below or slightly to the left of the tip of the left scapula. He concludes that this sign, although not so important as that of the patch of dulness, is very commonly, if not always, present in cases of considerable effusion, and gives valuable confirmation to other signs. The editor of this work tells me that he once found these signs very definitely in a case of a large collection of blood slowly effused into the pericardium from a ruptured coronary artery. The patient, a lady of some threescore, lived about sixteen hours from the onset of the symptoms.

As previously stated, pleural effusion on one or both sides is not uncommon as a consequence of a large collection of fluid in the pericardium; in which case the signs will be modified accordingly. When it begins on the right side the contrast may be helpful in diagnosis. Signs indicative of downward displacement of the liver are very pronounced in cases of extensive pericardial effusion, and there may also be some degree of enlargement due to venous congestion.

6. Effects of change of posture.—The study of the effects produced by changes of posture upon the chief signs just discussed has generally been regarded as important in the diagnosis of pericardial effusion. In a large proportion of cases these signs are so definite that it is quite unnecessary to test them in this way, and under such circumstances it is highly dangerous to place the patient in the sitting or erect posture; such disturbance may even prove immediately fatal.

The following are the chief modifications in the signs produced by changes of posture, which are regarded as of more or less diagnostic
value. It may happen that the impulse is not perceptible in the recumbent position, but becomes evident when the patient is made to sit up or bend forwards. Increased mobility of the apex-beat with change of posture has also been looked upon as important, but certainly this is very untrustworthy, to say the least. The effects of position upon the dulness have been more particularly insisted upon as evidence of pericardial effusion, and in doubtful cases may be worth studying. It is increased in extent, especially at its upper part, in the sitting posture, and still more if the body is bent forwards. It may also be modified in a lateral direction, as the patient turns to either side. The relative loudness of the cardiac sounds or of endocardial murmurs might also possibly be similarly influenced. Modifications of the signs observed in connection with the left lung posteriorly are also said to be produced by change of posture. Sansom writes: "If the patient bend well forward, or assume the knee-elbow position for a short time, the dulness disappears, and in its place a tympanitic sound is elicited; in like manner the former auscultatory signs of lung-consolidation vanish, or only slight crepitant rales are heard, which also very soon become inaudible. The former signs return when the vertical position of the body is resumed."

It must again be insisted upon that the physical signs of pericardial effusion vary much in different cases, and most of the special signs described by Ewart and others apply rather to cases in which it is so abundant as to raise the question of operative interference, when a positive diagnosis is obviously of extreme importance. With some of them I am not familiar, and I hardly think that any of them are absolutely trustworthy. Shattuck denies the existence of the dorsal signs of pericardial effusion. It may be mentioned that skiagrapy has been employed for the diagnosis of pericardial effusion and other conditions of this sac, but it is too early at present to attempt any definite statements as to its real and positive value.

Stage of absorption.—During the progress of absorption of inflammatory pericardial effusion the signs indicative of this condition progressively diminish, until the phenomena become practically normal, or point to the formation of adhesions. The friction signs, if they have been obscured by the effusion, return for a while; or they alter in their situation, intensity, extent, and characters. Friction-sound in most cases increases in a downward direction as the fluid declines (Sibson). It lasts a variable time. Friction-fremitus may at this period be noticed for the first time; and the sound is often rough and creaking or churning... The dulness diminishes more or less rapidly from above and laterally; while at the same time the sounds become more distinct. It must be remembered that one or more relapses may take place, with further increase of the fluid, the signs of which then return, again to subside as the fresh effusion becomes absorbed. What the ultimate position of the heart and the apex-beat will be depends on the course of events. As a rule, in simple and uncomplicated cases of pericarditis it returns to its normal situation, but this return may be prevented by adhesions, by the effects
of endocarditis, or by other causes. The signs indicative of adherent pericardium will be separately considered, but it may be remarked that in not a few instances, if carefully watched for, they can be traced in process of development during the period of convalescence.

Course and terminations.—As already stated, acute pericarditis presents much diversity in its clinical history, and it does not follow any uniform course. When, however, the symptoms and physical signs discussed in the preceding pages have been adequately and intelligently mastered, they can be studied with advantage in individual cases on the lines indicated. Among the chief circumstances which influence the nature, severity, and combinations of the symptoms, may be mentioned the causation of the pericardial inflammation, and the character of the general disease to which it is secondary; its intensity and rapidity of progress; the characters and amount of the inflammatory products, especially of the effusion; the presence of previous organic changes affecting the heart or pericardium, or of other chronic intrathoracic diseases; and the association of the pericarditis with endocarditis or myocarditis, or with pleurisy or pneumonia.

Attempts have been made by Stokes and others to classify cases of acute pericarditis into groups, according to the intensity of the symptoms, and the morbid changes affecting the pericardium and heart associated therewith; but distinctions of this kind are quite arbitrary, and have no practical foundation or value. It may be affirmed that as a rule the clinical phenomena are not so pronounced or so grave as is commonly supposed, or as the older writers used to describe. Not uncommonly the symptoms are not at any time prominent; they may be practically latent, or they may quickly attain some degree of severity, and as speedily subside. Shattuck, indeed, specifies “latency” as the most characteristic clinical phenomenon of pericarditis. In some instances one or more relapses occur, with corresponding increase of the symptoms after their subsidence. Acute pericarditis may run a favourable course in a few days, even when there is considerable effusion, which then undergoes rapid absorption. The entire duration of the majority of cases is from eight or ten days to a fortnight, but not uncommonly longer; convalescence may not be established for three to six weeks or more, or the disease, after beginning more or less acutely, may afterwards assume a subacute or chronic course. As a rule it terminates in recovery, so far as the immediate result is concerned, and no doubt in a considerable proportion of cases the restoration is practically complete; but in not a few instances definite organic changes are left behind, the effects of which are sooner or later revealed, it may be within a short period. Sometimes the patient can hardly be said to recover, a condition of obvious chronic pericarditis being established, with well-marked symptoms and physical signs which will be considered later. It is impossible to make any definite statement as to the direct fatality of acute pericarditis, and the more important points bearing upon this matter will be more conveniently referred to under prognosis. It may be affirmed, how-
ever, that death is seldom due solely to this affection, though evidences of pericardial inflammation may not uncommonly be found at post-mortem examinations, or it may partly contribute to the fatal result. Occasionally acute pericarditis assumes a very grave aspect from the first, advancing with great rapidity, exhibiting extremely severe symptoms, and ending in death within a short time, it may be even in less than twenty-four hours; but such a course of events only occurs under special circumstances, and mainly in haemorrhagic cases.

The course of rheumatic pericarditis in children is described by Dr. Cheadle as usually subacute, chronic, recurrent. It frequently merges into the condition of pericardial adhesion and its consequences, with their attendant phenomena. [For fuller details the reader is referred to the article, "The Acute Rheumatism of Childhood," vol. iii. p. 44 et seq.]

Diagnosis.—Several important matters bearing upon the diagnosis of acute pericarditis have been sufficiently dealt with under its clinical history, especially in the discussion of its physical signs; and in further consideration of this part of the subject, I propose merely to draw attention to its more prominent and important aspects.

An ordinary case of acute pericarditis arising in the course of definite rheumatic fever ought to present little or no difficulty in diagnosis, if due attention be paid to the symptoms and physical signs. Remembering, however, that the inflammation may supervene very insidiously in this complaint, and when the joint-symptoms are not pronounced, it is necessary, whenever any rheumatic condition is suspected, to be constantly on the watch for its appearance. Nor must we forget that pericarditis may be the first indication of such a condition. From these points of view it is a disease to be particularly watched for in children, though in such subjects its symptoms and signs, as well as its mode of progress, may be very anomalous, even where there is well-marked or perhaps a large pericardial effusion, a state of things, however, which ought not to occasion any difficulty to an intelligent and practised clinical observer. The occurrence of acute pericarditis in other than rheumatic cases may easily be overlooked by an incautious observer, but it should be thought of at any rate as a possible complication of Bright's disease, or of pneumonia or pleurisy.

Assuming that the diagnosis of pericarditis has been made, it is obviously very important to determine, within due limits, and without endangering or needlessly distressing the patient, the actual morbid conditions present, and more especially the amount and characters of the fluid effusion, as well as the changes which take place during the progress of the case. Most of these points can be positively made out by physical examination only, conducted on the lines already explained. It must not be forgotten that extensive friction-sound is not incompatible with a very abundant effusion. The rapid extension of general pericardial adhesion in some cases is also worthy of note, especially in children. The probability of the fluid being haemorrhagic, suppurative,
or ichorous is mainly founded on the conditions with which the pericarditis is associated, and on the general symptoms; yet these may be in no way characteristic.

What other conditions of the pericardium, or of the heart itself, are apt to be confounded with pericarditis? A dropsical accumulation—hydropericardium—may certainly be mistaken for an inflammatory effusion, especially if it be abundant. However, the circumstances under which it occurs, the fact that it usually follows hydrothorax, the absence of symptoms of pericarditis and of any friction phenomena, and, as a rule, the comparatively small amount of the effusion, will usually enable a diagnosis to be arrived at readily. A morbid growth involving the pericardium has more than once been mistaken for pericarditis with effusion. The distinction of pericarditis from endocarditis at an early stage is mainly founded on the differences between the tactile and auscultatory signs already discussed, but the symptoms may also help. When marked effusion occurs, any previous difficulty is cleared up. Of course the frequency with which the two diseases are associated together, especially in children and young subjects, must always be borne in mind. Implication of the heart substance is indicated by evidences of serious embarrassment and feebleness of its action, and when grave symptoms arise in the course of pericarditis, changes in the muscular tissue of the heart may be regarded as highly probable. Much has been written about the difficulties of distinguishing between pericardial effusion and cardiac enlargements, especially dilatation, but in my opinion they have been greatly exaggerated, when we remember that due consideration is to be given to all the facts of an individual case. It is possible, indeed, that a much dilated heart, especially if associated with extensive adhesions, might be mistaken for effusion; and such a mistake has actually happened several times, the heart having been punctured in an operation for the removal of a supposed pericardial collection of fluid. Difficulty might also arise when acute dilatation with rapid adhesion occurs in pericarditis, instead of effusion. Should inflammatory effusion supervene where the heart is enlarged, and the pericardial sac distended, the diagnosis might likewise be obscure; as well as when acute inflammation involves a narrow area of the pericardium, the rest of the sac being obliterated by previous adhesions.

The diagnosis of acute pericarditis from neighbouring conditions is, as a rule, quite easy. Occasionally the distinction between this complaint and pleurisy might be difficult, and certainly this applies to the friction-sound. A superficial exo-pericardial sound, or even a fremitus produced in the mediastinal cellular tissue, might also simulate pericardial phenomena. The only circumstance in which a pleural effusion is at all likely to resemble one in the pericardium is when it happens to be peculiarly limited by previous adhesions. It has been stated that such conditions as pneumonia, phthisis, aneurysm, accumulation of fat, or intrathoracic tumour might be mistaken for acute pericarditis, but I have certainly never met with any difficulty of this kind. It must not be
forgotten that this disease may be associated with other inflammatory affections within the chest, or be secondary to certain adjacent morbid conditions.

**Prognosis.**—Acute pericarditis must be regarded as a serious disease, though in uncomplicated cases the immediate prognosis is usually favourable. The mortality is comparatively small, but it is not practicable to give any definite percentage of deaths. Much depends upon the conditions with which the disease is associated, rheumatic cases being seldom immediately fatal. It is far more dangerous when it supervenes in connection with Bright's disease or other such grave chronic maladies, and is then likely to end fatally. Septic cases of all kinds are also very grave. Seeing that pericarditis and endocarditis so often go together, the prognosis under such circumstances must be guided by a due consideration of the effects of the combination in each particular case; but obviously it must always be more serious, especially if the myocardium is involved also. When there are other acute inflammatory affections within the chest, in addition to those implicating the heart and its covering, the danger is very imminent.

Among the factors influencing the immediate prognosis in individual cases the following are worthy of note:—Pericarditis is very serious in infants and young children; and the very fatal form described by Sturge, attended with grave nervous symptoms, and ending in rapid adhesion, must again be specially mentioned. In old people also the danger is decidedly greater. Previously impaired health, or a weak condition of the patient, and particularly the presence of old heart trouble or other chronic diseases, especially intrathoracic, may further complicate matters. The character and amount of the morbid products in acute pericarditis greatly affect the prognosis. The danger is obviously more serious in proportion to the quantity of fluid effusion; as well as if there be reason to believe this to be of a hemorrhagic, purulent, or ichorous nature. Due observation and study of the symptoms may afford important indications. Among those of more or less grave import are serious dyspnœa, especially if amounting to orthopneœa, with signs of cyanosis or asphyxia; greatly embarrassed or very feeble or irregular cardiac action, with corresponding pulse, and tendency to faintness or syncope; hyperpyrexia; dysphagia; severe vomiting; marked prostration; and pronounced cerebral or other nervous disturbances. The general appearance and the expression of the face and eyes are often useful guides to the immediate prognosis. It must never be forgotten that sudden death from syncope may happen in cases of large effusion into the pericardium, especially if the patient is made to sit up, or to change his posture for the purpose of physical examination. Finally, the mode of treatment materially influences the immediate prognosis in acute pericarditis. Undue activity may certainly do much mischief; but, on the other hand, a dread of energetic measures, when circumstances demand them, may as certainly lead to a fatal result.

*The remote prognosis in a case of acute pericarditis always demands
special attention, though it is often impossible to give a positive opinion on this point until the course of events has been watched for some time. I believe that the general tendency is to take too favourable a view of the ultimate prognosis, and not adequately to recognise the importance of the after-effects of the inflammatory changes. Such after-effects are met with in a considerable number of cases, and may be very serious, as will be pointed out in relation to pericardial adhesions. They are more likely to give trouble in proportion to the amount of lymph effused; to its presence over the exterior as well as the interior of the pericardium; to the slow or subacute progress of the disease; and to the formation of pus.

Treatment.—The treatment of each individual case of acute pericarditis demands careful and intelligent consideration, and it is decidedly a mistake to follow any regular routine plan, or to adopt needlessly active measures. When it occurs in connection with rheumatism it may not be requisite or desirable to change the previous treatment in any way, but much will depend upon the nature and degree of the morbid changes which the pericardial inflammation produces. The administration of salicylates is not contra-indicated, and many believe that they help in averting the complaint, but certainly their use requires caution. Dr. Gee has recently stated that large pericardial effusions are much less commonly met with now than formerly, and suggests that this may be due to the use of salicylates. Whether it be possible to prevent the development of pericarditis in rheumatic cases is a doubtful question, but at any rate complete rest, avoidance of chill, and due protection of the precordial region may help in this direction. Should there be a tendency to much cardiac excitement, I believe it is a good plan to administer opium or morphone as a preventive measure in suitable cases, the effects being of course duly watched.

When acute pericarditis has actually arisen, the treatment must be guided by circumstances. In every case the patient must be kept as much as possible at rest, and must not be unduly disturbed or moved for the purpose of physical examination. Posture must be intelligently studied in relation to the pericardial conditions, the symptoms, and the feelings of the patient. As fluid accumulates it is often necessary to have the head and shoulders raised; but, if so, the patient should be propped up comfortably and effectually supported; this arrangement requires special attention. The judicious administration of nourishment constitutes an important part of the treatment in many instances; and alcoholic stimulants, especially brandy and champagne, are often needed; the quantity must be determined by the requirements of each individual case, as judged chiefly by the degree of general weakness or depression, and the cardiac action and pulse. In bad cases a considerable amount may be required.

The treatment of acute pericarditis in the early stage has for its objects the relief of pain and restlessness, the calming of the heart's action, and the arrest or control of the inflammatory process. The practice of
bleeding and giving calomel, formerly adopted by many as a matter of routine, need only be mentioned to be absolutely condemned; nor in my opinion can anything favourable be said for the use of cardiac depressants, such as antimony, aconite, or green hellebore. In suitable cases advantage may certainly be derived sometimes from the application of a few leeches. As a rule, however, efficient poulticing over the front of the chest gives most relief at first, and answers best in the majority of cases, cotton-wool being afterwards applied. Fomentations or spongipiline are also convenient applications. I have thought that the application of a blister over this region at an early period has in a few instances checked the progress of the inflammation, but it is easy to be deceived in this matter. The application of cold, by means of ice-bags over the precordium, is strongly advocated by Dr. Lees and others, but this treatment certainly requires caution. Should the pain be severe, opium may be given, Dover's powder being a useful preparation; or morphine may be administered subcutaneously, and repeated as occasion demands. There is no harm in judiciously applying anodynes, such as belladonna, over the precordial region; but I doubt whether they are really beneficial.

The treatment of pericardial effusion must be guided by its quantity and mode of progress. If it is not abundant, and shows the natural tendency to become absorbed quickly, no special measures are needed. Otherwise it may be desirable to apply a blister, or even two or more in succession. Some prefer applications of tincture or liniment of iodine as counter-irritants; others advocate the injunction of mercurial ointment or oleate of mercury. The internal administration of iodide of potassium or sodium may be of service, combined with tincture of digitalis. Iron preparations may also be helpful, especially the tincture of perchloride; and a combination of tartrate of iron with the iodide has been recommended. Very active measures to promote absorption are certainly to be deprecated; and, when the effusion is large, special care must be taken not to make the patient sit up suddenly lest fatal syncope should occur.

In all cases of acute pericarditis it is necessary to watch carefully the action of the heart and the pulse from the point of view of treatment. I have already expressed my opinion that at no time, is it desirable to give cardiac depressants. Some authorities recommend the administration of tincture of digitalis from the outset, but I do not think that a routine use even of this drug is desirable. However, should there be any indication of cardiac weakness, or a marked want of tone in the arteries, with dicrotism of the pulse, the tincture should be given every three or four hours in ten-minim doses, its effects being duly watched. Strychnine affords valuable help in bad cases, and may be combined with digitalis; or it may even be thought desirable to employ subcutaneous injections of strychnine and digitalin. Of the use of strophanthus or other cardiac tonics in pericarditis I have no experience. As temporary stimulants, ammonia and ether might be of decided service in some cases; or possibly subcutaneous injection of ether. Of course alcoholic stimulants are often
of the greatest assistance, and large quantities of champagne or brandy may be demanded. The administration of the agents mentioned in the preceding remarks needs the most careful supervision, and they must not be employed indiscriminately or rashly, for it may be desirable at any time to diminish the dose, or to stop them. Special care must be taken in the treatment of children.

Pericarditis not of rheumatic origin must always be treated as a part of the general condition with which it may be associated, such as septicemia, tuberculosis, or renal disease; and in its association with endocarditis, or with other intrathoracic inflammatory affections, the knowledge, experience, and judgment of the practitioner will often be severely taxed, though not uncommonly but little can be done. Much difficulty may also be experienced in the treatment of symptoms, which must be conducted on ordinary principles, though considerable discretion and caution are demanded in carrying them out. Among the most important symptoms which may need attention are dyspnea, especially if accompanied with a tendency to cyanosis or apnea, dysphagia, severe vomiting, restlessness and sleeplessness, delirium or other cerebral disturbances, and high fever. Dr. George Balfour recommends chloral hydrate as a sedative and antiphlogistic along with digitalis; it is, however, a depressant of the heart, and must at any rate be cautiously used. Want of sleep is a very trying symptom, but such remedies as sulphonial, trional, or paraldehyde in suitable cases may help us better. Subcutaneous injection of morphine may be imperatively demanded, even if risky. Dr. Cheadle speaks highly of nepenthe for children. Inhalation of oxygen may help the breathing in some cases. The measures to be adopted to bring down temperature, especially hyperpyrexia, must be determined by circumstances. Difficulty in swallowing may, perhaps, be relieved by making the patient bend forwards, so as to relieve the esophagus from the pressure of the distended pericardium; but special care must be exercised in doing this. The bowels need due regulation; and in bad cases it is important to see that the bladder is properly emptied.

The quantity of a serous effusion, and the imminent danger to life resulting therefrom in exceptional cases, may raise the question of surgical interference, but I cannot agree with those who are too ready to resort to paracentesis for pericardial effusion. It is rarely required at any rate in rheumatic pericarditis. Dr. Clifford Allbutt (1) was the first to introduce as a practice the operation of paracentesis pericardii into this country in 1866, when it was successfully performed on a patient of his by Mr. Wheelhouse; the patient, who was moribund at the time of the operation, made a good recovery. In another case it was performed for him by Mr. Teale in 1869. For a full description of the operation reference must be made to surgical works (see especially Surgery of the Chest, by Mr. Stephen Paget), and it will only be necessary to refer here to two or three practical points. To determine that fluid is really present an exploratory puncture may be made, in the first instance, with a hypodermic syringe; and, as a dilated heart has even within a recent period
been actually perforated for a supposed pericardial effusion, this precau-
tion is certainly advisable in any obscure case. Some prefer even to
make an incision down to the pericardium. The fluid is best removed
by means of an aspirator with antiseptic precautions, but the instrument
must not be too powerful, as the effusion needs to be taken away very
gradually. Some operators prefer a small trochar and canula. Either
the fourth or fifth left interspace is usually selected, at a distance of an
inch (Dieulafoy) to 2 or 2½ inches from the margin of the sternum; but
the exact spot may vary with circumstances. The puncture has even been
made on the right of the sternum. Rotch recommended the fifth right
interspace. The late Marcus Beck recommended the use of a No. 2
needle, which he passed obliquely upwards and inwards, taking care to
turn on the vacuum as soon as the eye is covered. The moment the
fluid gets into the syringe the needle must be held steadily until the flow
ceases. The patient must be in the recumbent posture during the opera-
tion, and its effects carefully watched. When pericardial is associated
with pleural effusion, the removal of the latter may sufficiently relieve all
urgent symptoms, but if it tend to return it may then become necessary
to relieve the pericardium also. The subject of paracentesis pericardii
has been very ably dealt with in a paper by Dr. Samuel West, who gives
a tabular summary of eighty cases thus treated up to 1883. Subse-
quently it has been discussed by Sir T. Grainger Stewart and others,
and many scattered cases have been recorded.

The management of cases of pericarditis during convalescence is a
matter requiring due consideration, especially in relation to the formation
of adhesions. Personally I have been disposed as a rule to enforce pro-
longed rest, but some years ago Dr. Cantlie drew attention to the
desirability of encouraging exercise after an attack of acute pericarditis
in young subjects, with the view of exciting the cardiac action, and thus
helping to make the adhesions loose and filamentous. This question has
usually to be considered in relation to the presence or absence of endo-
carditis and its consequences, as well as the state of the cardiac walls; so
no general rule can be laid down, and every case must be studied on its
own merits.

II. SUPPURATIVE PERICARDITIS; PYOPERICARDIUM

The formation of pus within the pericardium has already been men-
tioned under acute pericarditis, but it will be expedient briefly to consider
this condition separately, including also those cases in which the fluid is
of an ichorous kind.

Etiology and Pathology.—Pyopericardium is occasionally acute in
its manifestation, but is much more commonly the result of a subacute or
chronic process. It is very rarely the outcome of an ordinary acute
pericarditis, either primary or rheumatic, being then a late or secondary
phenomenon, a serous or sero-fibrinous effusion gradually changing into a
more or less purulent collection. In the large majority of cases, however, the circumstances under which such a collection is met with are peculiar, and it may not only be formed within the pericardium, but in some instances is partly due to the bursting of a neighbouring accumulation of pus into the sac. Pathologically it is associated, of course, with pyogenetic organisms. It has been stated that the production of pus within the pericardium is favoured by abundant exudation, and the extensive formation of new blood-vessels in its substance. The longer a pericardial effusion remains unabsorbed the more likely it is to become purulent.

Pyopericardium occurs most frequently in cases of pyæmia or septiæmia of all kinds; thus it may appear as a complication of certain of the eruptive fevers. It has been said to be associated particularly with injuries and diseases of bones, such as osteomyelitis and acute necrosis. Purulent pericarditis is more likely to occur if an abscess has previously formed in the myocardium, but this is by no means necessary. Very rarely it appears to have been secondary to malignant endocarditis. In another class of cases pyopericardium is due to the rupture of a neighbouring collection of pus into the sac, especially of an empyema; or it may even be set up by contaminated air, which has entered through a perforation. Exceptionally it results from the extension of empyema, low forms of pleuro-pneumonia, neighbouring ulcerative or gangrenous diseases or abscesses, or possibly peritonitis. The pericarditis associated with Bright's disease is believed to have a special tendency to the formation of pus; and a similar tendency has been attributed to the tuberculous variety. Among the cases of operation collected by Dr. Samuel West (53), however, in no instance of tuberculous pericarditis was the effusion purulent. Such a condition may be associated with pulmonary phthisis, owing to the rupture of a cavity into the sac. Pyopericardium is far more common in young subjects, and in males.

Anatomical characters.—As the name indicates, the essential change in pyopericardium is the presence of pus in the sac. It may be in small amount, or the accumulation may be very large; in the latter case it will produce the same mechanical effects upon the heart and neighbouring structures as other forms of effusion. In Dr. Dickinson's most interesting case (17) as much as 19½ oz. were drawn off at one time; and in Dr. Samuel West's case 14 oz. and 16 oz. were successively removed. It may collect entirely in the posterior portion of the pericardium, the anterior surfaces being adherent as in a case of Sears. The pus is usually laudable and inodorous, but may often be shreddy, flocculent, curdy, or even membranous; and it may be mixed with lymph. Exceptionally and under particular circumstances it is offensive, and may be of an "ichorous" nature, very foul or even stinking. It may also become festid after operation. Occasionally there is an admixture of blood. In most cases the surface of the membrane becomes like that of the granulating surface of a wound. Rarely part of the parietal pericardium becomes destroyed, and perforation takes place, which has even ended in a superficial fistula;
but at the present day such a termination could hardly be permitted to occur. There seems to be good reason to believe that a purulent collection in the pericardium may in exceptional instances be absorbed, leaving dense and thick adhesions; or some of it may remain in an insipissated condition as a yellowish white paste, limited and encapsuled by adhesions, consisting of caseous material, in which calcareous particles may afterwards form; thus it may ultimately be converted into a chalky pulp, or even into a hard calcified mass.

**Clinical history, Diagnosis, and Prognosis.**—Speaking generally, the symptoms and physical signs of pyopericardium will be more or less like those of serous effusion, modified not only by the quantity of the pus, but also by the circumstances under which it has formed. It will only be necessary, therefore, to draw attention to certain special points in the clinical history of this condition. When it supervenes in an ordinary case of acute pericarditis, there are no trustworthy indications of a change from a serous or sero-fibrinous effusion to one of a purulent nature; but if the course of the case happens to be prolonged, such a deterioration would be suggested if fever, perhaps of a septic type, persist. Pyrexia may, however, be entirely absent. Considering the circumstances under which pyopericardium occurs, it is easy to understand how insidiously it may set in; its symptoms, if any, being entirely overshadowed by those of septicaemia: thus it often remains undiscovered until the necropsy, especially if the amount of pus be small. In cases of this kind symptoms of serious interference with the respiratory and circulatory functions may show themselves suddenly; and on examination be found to be due to a large but previously latent purulent collection in the pericardium. General symptoms are of little or no value in the diagnosis of pyopericardium. In some of the most pronounced cases neither rigors, pyrexia, nor sweating have been present. Edema of the legs seems not to be uncommon, but probably is not more frequent than in connection with other large pericardial effusions and their consequences. It may be noted here that oedema over the precordial region may suggest the purulent nature of such an effusion.

With regard to the **physical signs**, the absence of friction-sound throughout cases of purulent pericarditis has been noted by careful observers; or it may be very indefinite and transient. Whether this sign be usually absent, as has been affirmed, it is difficult to say; at any rate it cannot be relied upon in diagnosis. The ordinary signs indicative of pericardial effusion will be evident on examination, in proportion to the amount of the pus. Should gas be present at the same time, the phenomena associated with this combination will probably be noted, but these will be considered separately.

From the foregoing remarks it will be gathered that the diagnosis of pyopericardium is extremely uncertain, and often impossible. Should there be evidence of effusion into the sac, its purulent nature can only be determined positively by the aid of the exploring needle or other apparatus, by which a specimen can be obtained for examination. Some
such instrument should be used at once if there be any reason to suspect the presence of pus.

The prognosis of pyopericardium is necessarily grave, especially on account of the conditions with which it is associated. In suitable cases, however, efficient operative interference gives reasonable hope of recovery; and some remarkable results have been thus achieved by modern surgery.

Treatment.—The treatment of pyopericardium is entirely surgical, and it would be quite beyond the province of this article to attempt to discuss the important questions involved. Suffice it to say that mere paracentesis is of no use; the operative procedures adopted must be thorough and bold, and should be carried out as promptly as possible. Free incision, with drainage and due antiseptic precautions, is the method of treatment usually practised.

III. CHRONIC PERICARDITIS; CHRONIC EFFUSION; PERICARDIAL ADHESIONS AND THICKENING

The cases which come within the category of chronic pericarditis may be arranged for practical purposes under two groups; namely, those of—(1) Chronic effusion; (2) Pericardial adhesions and thickening. These conditions are in exceptional instances more or less combined, but it is needless to make an independent group of such complex cases. It will be sufficient to discuss separately the two main divisions just indicated.

1. Chronic pericardial effusion.—This morbid condition requires but brief comment. It occasionally happens that acute or subacute inflammatory effusion into the pericardium remains chronic, though fluctuating in amount; or it may return again and again after paracentesis. In rare instances even a simple pericarditis is chronic from the outset; but this course of events is observed chiefly in elderly persons, and there is reason to believe that in some of these cases the effusion is originally a mere hydropericardium. Chronic pericarditis is more likely to be of a hemorrhagic or purulent nature; or it may be associated with new growths, especially tubercle or malignant disease. Dr. Samuel West mentions a remarkable case of supposed mediastinal cyst, which was tapped several times during a period of four years; the fluid removed on the first occasion deposited a large amount of cholesterine; on post-mortem examination it proved to be a chronic pericardial effusion. In very exceptional instances an accumulation of this nature originates a diverticulum of the pericardium.

Clinically, chronic pericardial effusion does not, as a rule, give rise to any prominent symptoms; practically it is only recognisable by the physical signs already described. In prolonged cases, owing to the changes produced in the pericardium and the walls of the heart, the circulation becomes more or less seriously obstructed, with the usual symptoms, including dropsey. The treatment of this condition must be conducted on the general principles applicable to different kinds of peri-
cardial effusion, some operative procedure being generally required; but each case must be dealt with on its own merits.

2. Pericardial adhesions and thickening.—The conditions coming under this head are of much pathological and clinical importance, and are worthy of far more attention than they generally receive. It is a familiar fact that they are frequently met with at necropsies in various degrees, when they have not been diagnosed during life. It may be acknowledged at once that their diagnosis is often, for obvious reasons, impracticable, or may be a matter of great difficulty or mere surmise; not uncommonly, indeed, there is no reason whatever even to suspect their presence. On the other hand, to teach that the diagnosis of adherent pericardium is impossible is absolutely wrong and misleading. If pericardial changes of this nature were always borne in mind and systematically looked for, they would be recognised much more frequently than they have been hitherto; as a matter of fact they are seldom even suspected in the ordinary routine of practice, and are therefore necessarily overlooked. Not uncommonly they can be positively demonstrated by physical examination; while in other cases their presence may be reasonably inferred. Dr. John Broadbent, in his valuable monograph on "Adherent Pericardium," duly recognises this truth, and writes: "The comparative rarity with which the existence of adherent pericardium is diagnosed may be accounted for in many instances by the fact that it is not thought of. Especially is this the case when it is associated with valvular disease, for the valvular lesion is judged to be sufficient to account for the symptoms that arise."

Etiology.—The various conditions of the pericardium now under discussion are always of inflammatory origin, and in the large majority of cases they are the remains of one or more acute or subacute attacks of pericarditis, of which there is often, but not necessarily, a definite history. As was mentioned in relation to this disease, extensive adhesions may rapidly form in the stage of fibrinous exudation, especially in children; and if the termination be not fatal, they become organised and permanent. Most commonly, however, they are formed after the absorption or removal of fluid effusion. As might be anticipated, pericardial adhesions are likely to be more firm and extensive in proportion to the number of attacks of inflammation, and to their duration. After a first attack partial adhesions may form, which in subsequent attacks become extensive or general. When a pericarditis beginning acutely assumes a prolonged and chronic course, they are usually well marked, and again when the effusion becomes purulent. The occurrence of acute inflammation over the external surface of the pericardium leads to the formation of adhesions between this structure and the chest wall, the pleurae and, sometimes, the posterior mediastinal structures or the spinal column.

An important group of cases in which pericardial adhesions and thickening occur are those which are chronic from the outset, and in these cases they are particularly liable to be overlooked. They may naturally be expected when an inflammatory effusion runs a chronic course through-
Diseases of the Pericardium

out; but the cases which must be more especially borne in mind are those in which there has been no such effusion, but the morbid changes leading to the pericardial conditions have taken place slowly and imperceptibly. Some of the "white patches" are of this nature, but the most striking cases are those in which a chronic inflammatory process extends from neighbouring structures, particularly in connection with pleurisy or pulmonary phthisis. Adhesions are also usually associated with new growths involving the pericardium, which are practically either of a tuberculous or malignant nature. When the changes leading to these conditions have once started, it seems highly probable that they may extend and increase considerably, as the result of a continued chronic process, which may be regarded as inflammatory, and leads to a progressive hyperplasia of fibrous tissue. In this way it may possibly happen that an adhesion may, as it were, grow through the parietal portion of the pericardium from within outwards or from without inwards, and thus ultimately fix it more or less extensively on both aspects.

Pericardial adhesions may be met with at all ages. They have been observed in very young infants, and even in new-born children, when they are attributed to pericarditis occurring during foetal life.

Anatomical characters and effects.—It would not serve any useful purpose to describe in detail the numerous and varied aspects under which pericardial adhesions present themselves, but a comprehensive knowledge of the more important groups of cases in which changes of this kind are met with is of decided practical advantage. Before attempting any such classification it will be well to point out that the adhesions are either partial or general; internal or external, or both; that they differ much in length, toughness, and firmness, and are often accompanied by more or less pericardial thickening, which may reach an extreme degree. In exceptional instances there is much thickening, with little or no adhesion between the surfaces. Structurally the morbid formations now under consideration consist either of cellular or fibrous tissue—pericardial fibrosis. Sometimes they are associated with the encapsulated remains of fluid, thickened pus, soft caseous or chalky pulp, or dry brittle calcareous concretions, which may attain a considerable size. As already stated, an adherent pericardium may itself undergo calcification. The effects which the morbid changes may produce upon the heart and vessels must be considered separately.

The groups under which I propose to arrange the cases, as they have come under my personal observation, are as follows:

(a) In a large proportion of instances there are merely partial and small adhesions between the contiguous surfaces of the pericardium, it may be in different portions of the sac at the same time. Usually such adhesions assume the form of filaments or threads, or of bands, often of considerable length, stretching between the two surfaces. They may be delicate and cellular, or firm and fibrous, sometimes attaining the thickness of a finger or more. Occasionally adhesions occur in circumscribed closely adherent spots or patches. Ultimately the bands often give way
by stretching and attenuation, their remains hanging loosely within the sac, especially near the apex of the heart. The situation, extent, and characters of localised pericardial adhesions are affected by the degree and range of the movements of different parts of the heart and arteries; the relation of the heart to the pericardium; and the effects of gravitation of the organ within the sac in cases of effusion. According to Sibson, they are more frequent a little above and to the left of the apex, and along the line of the ventricular septum; at the outer border of the left ventricle, and the outer side of the right auricle; along the posterior surface of the left auricle and of the ventricles which rest upon the sac; and over the great arteries at their higher part. In several instances he noticed that a patch of the right ventricle, to the right of the septum and midway between the pulmonary artery and the lower border of the ventricle, was adherent, when the rest of the ventricle was free; this being the part of least extensive movement.

(b) A second group of cases may be made to include those in which an extensive or general internal adhesion exists between the pericardial surfaces, the external surface being quite free; and this group may be subdivided into cases without and with thickening. Here again many varieties are observed in individual instances, and in the same case the adhesions often differ in their characters over different parts of the pericardium. They may be in the form of fibrous threads or bands, more or less loose and long, and interfering but little with the free play of the heart; or of short, close, firm, and strong attachments. Again quoting Sibson's observations, the adhesions are generally longer at the apex than elsewhere; those over the left are longer than over the right ventricle; those over the auricular portion of the right ventricle are longer than those over its body and near the septum, and the same holds good in the case of the left ventricle. Over the right auricle they are much shorter than over the right ventricle. The attachments of the left auricle, the aorta, and the pulmonary artery are generally closer than those of the right auricle. In some cases the contiguous surfaces of the pericardium are agglutinated together, the sac being entirely obliterated; and when this condition is of old standing, separation of the two surfaces is impossible without tearing the heart substance. Occasionally, when comparatively recent, they may with care be drawn asunder; or firm adhesions of old standing may exist side by side with those of recent origin, the result of a fatal intercurrent acute pericarditis, which can be easily broken down. The degree of thickening differs a good deal, but it may be very remarkable, as much as a quarter to half an inch or more; it chiefly affects the visceral layer. The heart is then enclosed in a dense, strong, tight envelope or casing, which compresses and strangles the organ in its grip.

(c) There is a distinct class of cases in which the adhesions are entirely external or exo-pericardial, the outer surface of the pericardium being more or less extensively fixed to the front of the chest, and often to the pleurae, while the internal surfaces are quite free. They are usually chronic in their course, and secondary to neighbouring morbid con-
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...conditions; they are especially met with in association with very chronic phthisis. These exo-pericardial adhesions may, however, extend from similar pleuritic changes, or may possibly result from a mediastinitis occurring at the same time as the attack of pleurisy which led to the pleural lesions. I have comparatively recently had under my care a case in which pericardial and pleuritic adhesions were diagnosed, associated with extreme double mitral disease and much enlarged heart; and except that the pericardial adhesion was entirely external, the diagnosis proved to be correct. The condition now under consideration is really mediastinal, and has been named chronic mediastinitis (vide "Diseases of the Mediastinum," vol. vi.)

(d) The most serious group of cases of pericardial adhesion are those which are both internal and external, there being a general matting of the sac to the heart, as well as to the chest wall in front, to the adjacent pleura, especially the left, to the diaphragm more extensively than in health, and occasionally to the structures in the posterior mediastinum and the spinal column. As a rule these conditions are accompanied with much thickening. When there is little or no general mediastinitis the term pericarditis externa et interna is applied; when there is a considerable increase of fibrous tissue in the mediastinum the condition is known as indurative mediastino-pericarditis. [These changes are more fully dealt with under "Diseases of the Mediastinum," vol. vi.] The external adhesions vary considerably in area, but in extreme cases may extend from the second cartilage to the sixth; from the manubrium to the upper half of the ensiform cartilage; and from the right border of the sternum to the apex of the heart to the left of the nipple line (Sibson).

(e) Exceptional instances are met with in which the prominent change is marked thickening of the pericardium, especially of its visceral portion, with little or no adhesion of the surfaces; and there may even be more or less fluid incarcerated between them. It is important to bear this variety in mind, for it may produce very serious effects upon the heart, with the consequent symptoms, without giving rise to any of the physical signs of pericardial adhesion. A very striking illustrative example was under my care not long ago in University Hospital.

Effects upon the heart and great vessels.—There has been much controversy as to the effects of pericardial adhesions upon the heart; they may vary much, of course, under different circumstances. In a considerable proportion of cases the organ is unaffected, either functionally or structurally, and, provided it be free from valvular disease, remains of its normal size. The obvious tendency is to embarrass its action more or less; the embarrassment is greater in proportion to the extent and firmness of the adhesions, and greatest when they are both internal and external.

One of the most frequent and important structural changes affecting the heart which may result from adherent pericardium is enlargement of the organ. Hope maintained that this morbid condition always gave rise
to compensatory cardiac hypertrophy; but systematic and accurate observations have amply shown that such a statement is not correct: even complete obliteration of the sac is not necessarily followed by enlargement. No trustworthy statistics of the frequency of this change can be given; but it is certainly not uncommon. There is a distinct class of cases in which this lesion is the sole cause of considerable enlargement of the heart, which probably occurs in more than half of such cases (Sibson affirmed in about two-thirds); while in other instances the increase in size may be due mainly to associated valvular disease. Indeed it has been questioned whether in the latter group of cases the pericardial changes have anything to do with the enlargement. Sibson compared a double series of cases of valvular disease side by side, in the one series with, in the other without, adherent pericardium. He found that the cases with adhesions were on an average 5½ ounces heavier than those in which there were no adhesions; but, in many instances, the increase was to a considerable extent accounted for by the augmented thickness and weight of the pericardial sac. He concluded that in these cases the valvular disease is the essential cause of the enlargement of the heart, yet that the adhesions, by an additional demand upon the strength of the organ, add to the enlarging causes. From personal observations I am decidedly of opinion that a generally adherent pericardium, when associated with valvular disease, does often materially contribute to the cardiac increase; at any rate it promotes and hastens its development.

With regard to the mode in which adherent pericardium may promote cardiac enlargement, the explanation usually given and accepted is that it is mainly by the additional work imposed upon the heart, by the hampering of its movements and the increased resistance, aided by the changes in the myocardium which accompany the process. It has also been suggested that the eccentric contraction of cicatricial tissue may in some instances bring about dilatation of the ventricles, especially when the structures are fastened to the spinal column or anterior chest wall. It seems highly probable that inability on the part of these cavities to empty themselves, on account of the adhesions and muscular changes, may lead to dilatation, followed by compensating hypertrophy. Dr. John Broadbent gives the following explanation of the cardiac enlargement, when it occurs:—"When the heart is found to be dilated and hypertrophied as a result of adherent pericardium, there being no valvular disease to account for it, it is due to the fact that it has been left in a condition of dilatation after the original attack of pericarditis, and that while in this condition of dilatation the pericardium has become adherent; then the adhesions becoming organised, the heart is effectually prevented from again recovering its normal size. Subsequently it undergoes some hypertrophy." He further believes that, when the heart is of normal size, it either had not dilated during the original attack of pericarditis, or else had recovered from its dilatation before adhesions were formed. I have no doubt that this explanation is applicable to
As regards the nature, extent, and degree of the cardiac enlargement, considerable differences are observed in different cases of simple pericardium.

In some cases, but I cannot think that it represents the usual course of...
cardial adhesion. As a rule there is a combination of hypertrophy and dilatation, the latter commonly preponderating; and it may exist practically alone. Both sides of the organ are usually involved more or less; but I fully accept Dr. John Broadbent's statement that pericardial adhesions in themselves are much more likely to affect seriously the right ventricle than the left, for reasons which he has pointed out in his monograph. The auricles are much less affected; indeed it may happen that, while the right ventricle is much enlarged, the auricle is compressed and may even be practically obliterated. When the enlargement of the heart is associated with valvular disease, it will necessarily be influenced chiefly by the nature of such disease, but in particular instances it may certainly be modified by the adhesions. In some of these combined conditions, with firm adhesions, Sibson described the ventricles as undergoing a change in form, becoming flattened out, the right in front of the left, and the septum flattened instead of bulging forwards into the right cavity. As a result of dilatation produced by adherent pericardium, and involving the orifices, valvular incompetence is prone to follow, especially at the tricuspid opening, which may become greatly enlarged.

In a small proportion of cases the effects of pericardial adhesions upon the heart are quite the opposite to those just considered. In children the natural growth and development of the organ may be prevented; or it becomes small and atrophied, its walls being grasped and compressed, and its cavities forcibly contracted in size by the dense, thick, tight envelope surrounding them. This may happen also from mere thickening of the visceral pericardium, without any adhesion. Other cardiac changes apt to occur in these conditions are degenerations—either fatty, pigmentary, or fibroid. They may result from direct pressure, or pressure on the coronary vessels; or the last may be due to a chronic interstitial myocarditis spreading from the pericardium. In some instances, no doubt, these cardiac changes are the outcome of myocarditis associated with an acute attack of pericarditis. They are often of considerable importance, and contribute largely to the symptoms of pericardial adhesions and thickening.

When the pericardium is fixed externally, the great vessels at the base of the heart are often abnormally exposed. Sibson observed that with enlargement of the heart, "the great arteries are lifted up on the top of the ventricles into an unusually high position, and are crowded into the narrow space at the top of the chest, almost as high as the root of the neck." OccasionallY one or both are compressed or constricted by pericardial adhesions; or their walls undergo degenerative or fibroid changes. As the result of obstruction to the general venous circulation, produced indirectly by adherent or thickened pericardium, the large veins become more or less dilated, and such dilatation may ultimately be extreme.

Clinical history.—It is obviously impossible to give any definite clinical description that will apply even to the majority of cases of adherent pericardium; all I can do will be to point out the symptoms
and physical signs which may be associated with this condition, as well as the relations of these phenomena to each other, upon which a diagnosis may reasonably be founded. They vary considerably in individual instances, not only in respect of the actual nature and degree of the changes affecting the pericardium, but also of their effects upon the heart, and their association with endocardial lesions, with vascular diseases, or with neighbouring morbid conditions.

As was stated in the introduction to this subject, a large number of cases of pericardial adhesion do not exhibit any symptoms or physical signs whatever; and, unless there happen to be a well-known history of acute pericarditis, the condition cannot even be suspected during life. This applies not only to partial and loose adhesions, which often do not disturb the heart in any way, but even to cases in which there is general agglutination of the internal surfaces; provided the organ itself be not materially damaged. It is well to bear in mind the possibility of this condition, if with acute pulmonary inflammatory affections the heart should exhibit signs of embarrassment quite out of proportion to their severity. My observations have led me to the conclusion that it may add seriously to the danger under these circumstances, and even account for an unexpected death. The more pronounced the pericardial changes, the more prominent and definite are the clinical phenomena likely to be; and they are especially well marked when there is much thickening, and when the adhesions are both external and internal.

The symptoms and physical signs which may be met with will now be considered separately.

That pericardial adhesions may be the cause of pain—of painful, dragging, or other unpleasant sensations over the precordial region, I have not the slightest doubt, and when in cases of obvious chronic cardiac disease such sensations are much complained of, their existence may be reasonably suspected, and they should be carefully looked for. I have met with not a few instances in which they were associated with adhesions easily demonstrable on physical examination. Moreover, the pain occasionally comes on in attacks of an anginal character, when the case, if accompanied by other symptoms characteristic of such attacks, may present a perilous aspect. A feeling of precordial oppression, and inability to take a deep breath, are sometimes prominent symptoms, especially when the external adhesions are extensive. The patient is usually conscious of the disturbances of cardiac action associated with adherent pericardium, and is then likely to complain of palpitation, even at rest, but especially after exertion; and this symptom is sometimes very prominent.

Adherent pericardium ought always to be thought of as a possible cause of palpitation. The heart’s action is in some instances irregular or unequal, and it may be so embarrassed as to lead to faintness or actually to syncope. The persistence of rapid cardiac action, in spite of treatment, may be important evidence of the formation of pericardial adhesions in children and young persons.

Pericardial adhesions may themselves unquestionably cause dyspnea
on exertion, sometimes well marked; and thus also they often add to
the difficulties of other cardiac affections. No other respiratory symptoms
can be definitely attributed to these conditions alone; but when there is
much thickening, with compression of the heart and changes in its walls,
the pulmonary circulation is likely to be embarrassed, and cough,
epectoration, or even hæmoptysis to set in.

A very important and prominent group of symptoms in certain
cases of pericardial adhesion are those indicating serious hampering,
or actual failure of the right ventricle, and consequent interruption
with the general venous circulation. These either come on gradually,
becoming more and more pronounced; or, occasionally, they supervene
with great rapidity, the ventricle appearing to break down and give
way very speedily, or even suddenly. They occur not only in cases where
this cavity is obviously dilated, but also where the heart is strangled
and compressed by dense fibrous thickening; and in such cases they may
be extreme. No doubt they depend in great part upon the associated
changes in the cardiac structure. These symptoms are similar to those
which arise in other forms of heart disease affecting the right side; namely,
general dropsy, involving the serous cavities as well as the subcutaneous
tissue more or less extensively, congestion of the hepatic and portal
system and its consequences, and also of the kidneys, nervous system,
and other structures. The dropsy usually begins in the legs, but it may
ultimately involve the trunk, and even the arms. In exceptional instances
ascites is noticed before anasarca. Remarkable cases occasionally occur,
entirely due to pericardial adhesions and their consequences, in which the
peritoneal cavity and pleura become repeatedly full of fluid, and have to be
tapped again and again in order to afford temporary relief. Under these
circumstances the breathing is likely to be much distressed, even to
the degree of orthopnoea. The appearance of the patient differs in
different cases. Cyanosis with distended veins may be evident; or,
on the other hand, there is sometimes marked pallor, with puffiness of
the face. The liver becomes enlarged so that it can readily be felt
below the ribs, and may be painful and tender. Occasionally it reaches
even below the umbilicus, appearing to be very large; but then it is
usually displaced downwards as well: "after a time the organ yields an
abnormally firm sensation on palpation, and may become irregular; in
prolonged cases it may even pulsate. Symptoms connected with the alimentary canal are often prominent, and sickness may be troublesome. The spleen is sometimes perceptibly enlarged. The urine is more or less diminished in quantity, concentrated, and often albuminous. I have known the amount of albumin to be so large that the urine became almost solid on boiling, simulating serious renal disease. In bad cases the patient is very
restless and sleepless.

Dr. John Broadbent, speaking of the symptoms which have just been
discussed, deduces from his observations the following corollary:— "That
when symptoms of right ventricle failure supervene in cases in which
there is no evidence of left ventricle failure due to, valvular disease or
kidney mischief, constant high tension, or other obvious causes, or of lung disease such as chronic bronchitis, etc., to account for their appearance, the presence of adherent pericardium should be suspected as the cause, and other indications of it carefully sought for. So, too, in valvular disease of the left ventricle, in which the lesion is judged to be slight, and compensation breaks down unaccountably, adherent pericardium should be thought of.” Further, speaking of the difference in the symptoms of right ventricle failure when due to pericardial adhesions, and when secondary to valvular disease of the left ventricle, he writes: “In cases of right ventricle failure attributable to adherent pericardium, there is no cyanosis, though the respirations may be hurried, and there may be some dyspnoea; there may be an entire absence of dyspnoea, though the other symptoms are severe; there is usually no congestion or edema of the lungs. The dyspnoea, when present, is probably due to deficient supply of blood to the lungs and a feeble pulmonary circulation owing to the failing powers of the right ventricle.” While fully recognising the correctness of these conclusions in their application to a certain class of cases, I must point out that they by no means always hold good in relation to adherent pericardium; for the effects of the difficulties in the right side of the heart may themselves lead to cyanosis and dyspnoea, while the lungs may be also implicated when the entire heart is gripped by strong adhesions. Moreover, pericardial adhesions may help in producing this class of symptoms in cases where there is pronounced valvular disease on the left side.

Physical signs.—The existence of pericardial adhesion can often be recognised positively and demonstrated by careful and systematic physical examination; and it is most desirable to have a clear and definite knowledge of the signs which, in different combinations, have to be looked for and studied in respect of this condition. At the same time it must be understood that they are frequently absent, or at any rate not at all characteristic; and this may happen even when there are very pronounced symptoms directly due to an adherent pericardium; for example, if the heart is compressed and atrophied, though the symptoms may be extreme, the signs will be wholly indefinite. They are likely to be better marked as the adhesions are more extensive and dense, and especially when these are external as well as internal. They result not only from these lesions themselves, but also from their effects upon the heart and vessels, and upon the circulation. They may be considered in the following order:

(i.) Change in shape.—In exceptional instances a distinct and permanent depression of more or less of the precordial region, with narrowing of the intercostal spaces, is observed; the structures being drawn in by thick external adhesions. Far more commonly, however, there is abnormal fulness or bulging, due to enlargement of the heart; but as this usually depends mainly on other causes, it can hardly be regarded as an indication of adherent pericardium, except under particular circumstances.

(ii.) Signs associated with cardiac movements.—Certain visible and tactile
signs coming under this head are of the utmost importance, and demand somewhat detailed consideration. Sometimes there are peculiarities in the cardiac movements which cannot well be described, but which are very suggestive of these changes, when prominent cardiac symptoms are present, and are not obviously due to any other organic affection of the heart. The following are the more definite signs to be studied:—

(a) Apex-beat.—In cases of adherent pericardium, the ordinary apex-beat presents many differences as regards its position, force, and characters; but these depend mainly upon the effects of the particular valvular disease or diseases with which the condition happens to be associated. Thus it has been noticed far to the left, and presenting all the indications of a greatly hypertrophied left ventricle. One of the signs to be looked for is a displacement of the apex-beat, which is fixed in its abnormal position, and cannot be modified by any change of posture. As a rule it is carried somewhat outwards; but the most suggestive displacement is elevation, it may be to the fourth space or even higher, while perhaps at the same time there may be marked evidence of hypertrophy. In many instances the apex-beat is very feeble, or even imperceptible when other phenomena, to be presently described, are well marked; and even when it extends 1 to 1 ½ inch outside the left nipple. This is attributed to small size and weak action of the heart; to restraint of the organ by adhesions; or to much thickening of the pericardium. When it is wholly due to feeble cardiac action, the beat may at times be perceptible, at other times not. There may, however, be a distinct impulse over the ensiform cartilage or in the epigastrium.

(b) Impulse.—Taking into account the entire impulse, it must be admitted that in cases of adherent pericardium great variation of its situation, extent, force, and characters is observed; but there are certain points deserving of attention. A remarkable extension of its area is often noticed, especially upwards over the precordial region; and it may reach the second space or cartilage. This may be associated with obvious elevation and fixation of the apex; or it may be impossible to localise any definite apex-beat. At the same time the impulse is often strong and superficial, the heart pulsating in close contact with the chest walls. In some instances the movement presents to the eye a decidedly undulatory or wave-like character, from the base towards the apex. In others it is peculiarly jarring, or has an abrupt jogging quality. The rhythm of the cardiac action is sometimes markedly disturbed, and pericardial adhesions may undoubtedly give rise to irregularity. When the heart is at the same time enlarged, the extent of the impulse is correspondingly increased, often passing considerably beyond its normal limits, and probably tending more towards the right, in consequence of the greater enlargement of the right ventricle.

(c) Systolic recession or retraction.—A visible recession or retraction of certain parts of the chest wall, associated with the ventricular systole, has attracted much attention in respect of adherent pericardium. There can be no doubt that the signs coming under this head are of great im-
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Importance in the diagnosis of this condition, and they deserve particular study in any suspected cases. They come practically under three categories, namely:—

(a) Recession over the spot corresponding to the apex of the heart, occurring with or immediately after the systole. This phenomenon, when present, is usually associated with a definite apex-beat, but is sometimes noticed when there is no perceptible impulse at this point.

(b) Systolic depression of more or less of the precordial region, generally involving one or more of the intercostal spaces to the left of the sternum, especially the third, fourth, and fifth, along a variable extent of their length. The movement is sometimes distinctly wavy. In certain cases, where the adhesions are extensive and strong, and the heart is acting powerfully, the cartilages are also involved, or indeed even the lower half of the sternum, the ensiform cartilage, and the epigasstrum. When the recession occurs simultaneously with an obvious and strong apex-beat, the combination is very striking, but it may be indefinite or absent. Should the right ventricle be greatly enlarged, a similar movement may possibly be visible in the intercostal spaces to the right of and close to the sternum; of this I believe I have seen examples. According to Friedreich, the pitting is more marked at the height of inspiration.

(γ) Retraction of the posterior or lateral portions of the thoracic walls.—I cannot say that I am personally familiar with this sign, which, when present, is regarded by Dr. John Broadbent as a most important diagnostic sign of adherent pericardium; he describes it in the following words:—“In cases of adherent pericardium, marked systolic retraction of some of the lower ribs on the lateral or posterior aspect of the thorax may sometimes be seen. This phenomenon is best seen when the patient is sitting up in a good light, and the movements of the chest are carefully observed from a short distance off, first from the front and then from the lateral aspect. When a pulsatile movement is seen over the lowest part of the left side posteriorly, it may at first sight appear to be expansile. On a more careful scrutiny it will be found that there is a tug on the false ribs during the cardiac systole, and a sharp rebound during diastole, which can be felt as well as seen when the hand is laid flat upon the chest wall at the spot; it is more marked when a deep inspiration is made; it may be seen occasionally not only on the left side but also on the right, especially if the patient leans over to the left.”

Space will not permit of any long discussion of the associations of the phenomena just indicated with conditions other than pericardial adhesions, or of their precise significance in any individual case of such adhesions. A few general observations on these points must suffice. Apical recession very rarely occurs except as the result of adherent pericardium, but it was observed by Friedreich in a case of aortic stenosis where there were no adhesions; and has also been noticed under other circumstances. When it is associated with a definite beat it probably indicates that the apex of the heart is fixed to the
chest wall, and drags on it during the systole. The adhesion need not, however, be extensive, for a narrow band may cause the depresion, provided the pericardium be fixed externally. When there is no palpable apex-beat, it is supposed that the heart is prevented by adhesion to the diaphragm or vertebral column from performing its normal forward and rotatory movement during systole; or that the cardiac impulse is too feeble to be felt through the adhesion.

Skoda was of opinion that systolic recession of the intercostal spaces is pathognomonic of adherent pericardium, but numerous observations have shown that this is not the case, as the phenomenon may occur in cases of considerably enlarged heart, as the result of atmospheric pressure, especially when associated with aortic regurgitation. Still it is an important sign of adhesion, and its presence should always have due weight in diagnosis. As a rule it indicates that the contiguous surfaces of the pericardium are adherent, and also that the sac is fixed in front to the chest wall, and to some structures posteriorly, so that when the heart contracts, being firmly attached behind, it pulls in more or less of the yielding anterior thoracic wall. When there is no posterior adhesion, and yet systolic depression occurs, it is supposed that the firm attachment of the pericardium to the central tendon of the diaphragm forms the fixed point from which the heart acts in drawing in the front of the chest, or possibly that the effect may be produced by the contraction of the organ itself. Friedreich is of opinion that the lower surface of the heart must be firmly adherent to the diaphragm. I have met with this phenomenon in a pronounced form in cases of external pericardial adhesion with enlarged heart, where the internal surfaces of the sac were quite free. As a result of diminution in the force of the cardiac action, a marked systolic retraction may in course of time become less and less evident, and finally disappear.

"The systolic retraction of the posterior or lateral portions of the thoracic walls, which, as we have seen, is regarded as a positive sign of adherent pericardium by Dr. John Broadbent, who states that it is quite distinct from recession of the lower ribs in inspiration, is explained by him in the following way:—"The heart is, by means of the pericardium, adherent not only to the central tendon of the diaphragm, but probably also to a large area of the fleshy or muscular portion of the diaphragm, and, it may be, to the anterior thoracic wall as well; as it contracts it drags upwards and inwards the less resistant fleshy part of the diaphragm towards the central tendon or anterior chest wall; hence the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and downwards. It will always be found in such cases that the retracted portions of the chest wall correspond to the floating ribs or costal cartilages of the lower ribs at the points of attachment of the diaphragm."

(d) Diastolic shock or concussion.—This is a very exceptional sign, only occurring where the pericardium is firmly adherent to the anterior chest wall, and when the heart is acting powerfully. It follows imme-
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Immediately after the systolic recession, and is in proportion to its force. The diastolic shock is felt by the hand as a "back stroke." It may be perceptible only at the apex-beat, over one or more intercostal spaces, over a more extensive surface—possibly over the entire precordial area; or even round the left side to the back. The phenomenon is attributed to the elastic recoil or rebound of the chest wall, at the beginning of diastole as soon as the systolic dragging force has ceased. In well-marked cases it may be felt as a distinct jerk or blow, which is occasionally so strong as to be like the impulse of the heart. When present it is regarded as a pathognomonic sign of adherent pericardium.

Apart from the sign just considered, I feel sure that in some cases of adherent pericardium, with exposure of the heart and great vessels, a diastolic impulse is felt, due to the closure of the aortic and pulmonary valves. It is noticed over the base, and is quite independent of systolic retraction.

(c) Posterior systolic impulse.—I believe that this sign is sometimes of value in the diagnosis of adhesion of the pericardium to the structures posteriorly; especially when there are indications of probable agglutination of its two surfaces, and of anterior adhesions. It is best recognised, not by the hand, but by the head, when this is placed over the back of the left side of the chest in the practice of direct auscultation. The movement is directly due to the hypertrophied heart, and is often associated with more or less compression of the lungs, which therefore conducts the sensation more readily; but I think that it is likely to be more pronounced when the structures are matted together by adhesions.

(iii.) Cardiac dulness.—Pericardial adhesions or thickening do not in themselves appreciably affect the cardiac dulness, as a rule; but a mass of fibrous tissue about the vessels may certainly cause some increased dulness towards the base. When, as a consequence of adhesions to the chest wall, the heart and great vessels are abnormally exposed and superficial, the area of cardiac dulness will be proportionately enlarged, and may be of considerable extent, being often markedly increased in an upward direction, sometimes reaching the second rib. Part of this altered percussion sound may be due to adhesion and collapse of overlapping lung. When enlargement of the heart is associated with the pericardial condition the dulness will be modified accordingly, and is not uncommonly very extensive. Dr. John Broadbent writes: "When, during an attack of pericarditis, the area of cardiac dulness has been noted to increase considerably in extent, and after the subsidence of the attack remains permanently increased, it is extremely probable that adhesions have taken place, fixing the heart in a condition of dilatation." In well-marked cases the dulness resulting directly or indirectly from pericardial adhesions and thickening is very pronounced or even absolute. As already stated, when extensive calcification has taken place, the percussion sound in rare instances has been described as presenting a peculiar osteal quality.

(iv.) Auscultatory signs.—It cannot be said that there are any actually
pathognomonic or trustworthy auscultatory signs of adherent pericardium; but one or other of the following points may be worthy of attention in particular cases:—

(a) Should the pericardium be fixed to the chest wall the heart sounds are likely to be remarkably superficial. The first sound is certainly often abnormal in character. In some cases it is peculiarly sharp and valvular in quality; in others it is markedly dull or muffled at the apex or over the mid-cardiac region; or again it may be prolonged and reduplicated. The second sound is frequently reduplicated, but Friedreich maintains that this may be due to the rebound of the chest wall which causes the diastolic shock, and produces a dull sound heard after the second sound of the heart. Dr. John Broadbent regards a weak pulmonary second sound, when there is evidence of hypertrophy of the right ventricle, as a very important indication that the cause of the hypertrophy was probably not back pressure through the lungs due to left ventricle trouble, but some intrinsic cause, perhaps adherent pericardium. Marked conduction of the heart sounds towards the back of the left side of the chest, especially when associated with the feeling of pulsation already referred to, is suggestive of posterior pericardial adhesion.

(b) A rough pericardial friction-sound may remain over different points of the precordial region, especially towards the base, for some time after an attack of pericarditis; and, should it be associated with suspicious signs of adhesion, might be useful as corroborative evidence. Its eventual disappearance would probably indicate that adhesions had formed at the spots where it was previously audible, and have since become more or less general.

(c) With regard to endocardial murmurs, a kind of rumbling pre-systolic murmur is sometimes heard at the apex, which does not, however, indicate the presence of mitral stenosis; this kind of presystolic murmur is specially common in children (J. Broadbent). It is possible that a basic systolic murmur may result from the pressure of pericardial thickening upon one or both of the great arteries. The several valvular diseases, when present, will give rise to their corresponding murmurs, but I believe that these may be modified in their character by adherent pericardium, and a tricuspid regurgitant murmur, may ultimately result from enlargement of the right ventricle owing its origin to this condition.

(v.) Signs connected with respiratory movements.—When searching for pericardial adhesions, it is often highly advantageous to study the effects of deep inspiration and expiration. In the first place, the fact that the position of the apex-beat and the area of extended cardiac impulse are not thus affected may be of much importance; as well as that the area of precordial dulness is not altered. It implies the presence of adhesions between the external surface of the pericardium and the thoracic wall, and the want of any modification in the dulness is particularly marked when the sac is adherent to the margins of the lungs also. As a result of extensive external pericardial adhesions, inspiratory expansion may be
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Decidedly less on the left than on the right side. Another sign sometimes observed, coming under this head, is impeded descent of the left half of the diaphragm in inspiration, as indicated by diminished movement of the upper part of the abdominal wall on that side. This may occur either with or without adhesion to the anterior chest wall; in the latter case it has been attributed to abnormal attachment of the pericardium to the muscular portion of the diaphragm, which hinders its descent. Tracheal tugging might possibly result from adhesion of the pericardial sac to the bifurcation of the trachea, but I have never found this to be the case.

(vi.) Arterial signs.—In some cases in which pericardial adhesions were proved after death to exist, I have observed a peculiar visible movement, in connection with the large arteries at the root of the neck, which I believe may be of more or less significance. It gives the impression that the heart is making an effort to drive the blood into these vessels, but is prevented from doing so effectually on account of the embarrassed action due to the adhesions. The movement may be modified by the coexistence of aortic or mitral disease. Irregular pulse may be associated with adherent pericardium, but commonly this is not the case until cardiac failure sets in; and in diagnosis no positive reliance can be placed on this disturbance. I think that in cases of mitral disease the condition tends to increase the irregularity. The arterial sign to which Kussmaul attached special importance is the presence of a marked pulsum paradoxum, the pulse intermitting with inspiration, which has been chiefly noticed in cases of indurative mediastino-pericarditis, but occurs under other circumstances, and is by no means trustworthy. Kussmaul attributes it to the presence of fibrous cords encircling the aorta, which, by dragging on it during inspiration, constrict its lumen.

(vii.) Venous signs.—Sudden collapse of the veins of the neck during the ventricular diastole has been specially studied by Friedrich, who regards it, when associated with systolic retraction of the intercostal spaces, as a most valuable sign of adherent pericardium; it is never present in any striking degree without such retraction. The veins, often tensely filled during systole, disappear from view during diastole, the subsidence being synchronous with the diastolic shock felt in connection with the chest wall. Sometimes the supraclavicular fossae are deepened at the same time. The explanation of this phenomenon given by Friedrich is that, owing to the diminution of the thoracic space, the return of blood through the cervical veins is hindered during systole; and that the subsequent sudden diastolic enlargement has an aspiratory effect, drawing in the blood from the veins: it is supposed also that the diastole takes place with unusual force and rapidity, owing to traction by the adhesions from without, and the descent of the raised diaphragm. He further assumes that in consequence of the diastolic descent of the heart, especially as caused by the action of the diaphragm, the large vascular trunks, including the superior vena cava, become elongated, and thus the downward current of blood from the cervical veins is hastened.
Dr. John Broadbent quotes a case of adherent pericardium, observed by François Franck, in which systolic emptying of the veins of the neck occurred, and was ascribed to an aspiratory periventricular effect caused by the adhesions. He also describes another case—where the pericardium was universally adherent to a greatly hypertrophied heart, and also to the chest wall over a large area—in which systolic emptying of an enlarged vein on the front of the chest, to the right of the sternum, was followed by filling during diastole. "The explanation suggested was that the pericardium adherent to the heart and chest wall dragged apart the walls of the internal mammary vein during systole, causing a suction action, so that the blood was drawn into its lumen from the afferent veins during systole." I think I have recently met with a similar case.

Diagnosis.—It must be repeated that in a large proportion of cases where pericardial adhesions exist, there are no trustworthy data upon which a definite diagnosis can be based; though nevertheless the possibility of their existence may suggest itself in explanation of cardiac disturbance of obscure origin. The rule is not to forget these lesions in any case, and to take some trouble in their clinical investigation. In not a few instances the diagnosis of adherent pericardium is evident enough, and yet the condition is entirely overlooked. It is not enough to say that pericardial adhesions exist; an endeavour must be made to determine their extent and nature; whether they are external, internal, or both; and their effects upon the heart. Moreover, their association with valvular diseases of this organ must not be lost sight of, as they are often important factors in such combinations.

If the patient have had one or more attacks of acute or subacute pericarditis, or of rheumatic fever, we may suspect adhesions; especially if they have formed under the observation of the practitioner who has subsequent charge of the case, and who can give definite information at first hand. I have not uncommonly watched their formation during the period of convalescence, and had the opportunity of studying their after-effects. In other instances an indefinite history merely points to cardiac inflammation of some kind. The frequent association of pericarditis and endocarditis in childhood has an important bearing on diagnosis; and, when the origin of valvular disease can be traced to early life, pericardial adhesions should be particularly looked for. Unfortunately, in a large proportion of cases no history pointing to pericarditis can be obtained; and it must not be forgotten that the formation of adhesions may be a chronic process throughout.

The positive diagnosis of adherent pericardium is founded upon careful and systematic investigation and study of the symptoms and physical signs already discussed, not only in themselves, but also in relation to each other. Individual cases differ much in their exact characters. Sometimes the diagnosis has to be made on physical signs alone, there being no prominent symptoms. On the other hand, progressive signs of general venous obstruction following an attack of
pericarditis, inducing extreme dropsy of the subcutaneous and serous cavities, only to be relieved by repeated operations, may alone indicate the presence of a thick, dense, adherent pericardium, compressing the heart, there being no obvious physical signs of the condition. In other instances, again, enlargement of the heart, especially of the right ventricle, occurring without other adequate cause, or perhaps developing with unusual and inexplicable rapidity in connection with valvular disease, suggests adherent pericardium as a possible cause. With regard to the relation of symptoms to physical signs, Dr. John Broadbent writes: "When symptoms of cardiac failure, more especially of right ventricle failure, occur of greater severity than the physical signs present seem to warrant, or where compensation breaks down unaccountably, adherent pericardium must be suspected. When rest and suitable treatment fail to give relief, provided the patient is not of advanced age or thoroughly broken down, this affords further evidence in favour of adherent pericardium, and other confirmatory signs of it should be carefully looked for." With these remarks I cordially agree. Sir Samuel Wilks has expressed the opinion that severe heart symptoms in young persons, without valvular murmurs, point to pericardial adhesions; while in persons of mature age they indicate cardiac degeneration. There is a good deal of truth in this statement, though not a few exceptions will be met with in both directions.

Prognosis.—No general rules of practical value can be stated under this head; every case in which pericardial adhesions exist must be studied individually as regards prognosis. Often they are of no consequence whatever; in other instances they are merely a source of discomfort, and do not endanger life. Sometimes, however, they are extremely grave in themselves, and then the outlook is very serious, while they make life exceedingly miserable. It may be impossible to give any relief to the symptoms; or a dropsical condition may be kept at bay only by repeated operations. That pericardial adhesions add seriously to the effects and dangers of valvular diseases cannot be doubted, and they often hasten their progress and fatal termination.

Treatment.—Pericardial adhesions once formed cannot be got rid of. Rest, good nourishment, and other suitable measures are of value in preventing or delaying their ill-effects, and in maintaining the nutrition of the myocardium. Whether the various exercises now in vogue in the treatment of cardiac disease are likely to be of any service in this kind I do not know, but in cases where extensive and firm adhesions exist they undoubtedly may do much mischief, if carried out thoughtlessly. Cardiac tonics may be useful in some cases; but it must be remembered that pericardial adhesions may materially interfere with the action of digitalis and allied agents upon the heart, and then such agents may do much more harm than good. Symptoms must be dealt with on ordinary principles; and dropsy often requires repeated removal by operation.
IV. HYDROPERICARDIUM; DROPSY OF THE PERICARDIUM.

Pathology and Etiology.—Hydropericardium, or hydrops pericardii, signifies a serous effusion into the pericardial sac, occurring during life, of a dropsical nature, as distinguished from one of inflammatory origin. As has been previously stated, a certain quantity of fluid, varying under different circumstances, is found in this sac at most necropsies; this is merely due to transudation from the vessels and heart occurring during the act of dying, and for a time after death. It usually amounts to from half an ounce to an ounce, but under favourable conditions may reach three ounces or more. Definite hydropericardium may occur under the following circumstances:—(i.) As an acute or active effusion in connection with certain cases of Bright's disease, thus it may follow scarlatina. (ii.) As a part of chronic dropsy, more or less general, usually in cases of cardiac or renal disease; but occasionally associated with scurvy and allied states, grave forms of anemia, tuberculosis, cancer, and other cachexies affecting the blood. In this group the pericardial dropsy almost always follows effusion into the pleurae, and the pericardium is much less frequently involved than other serous membranes. (iii.) Exceptionally from some mechanical difficulty interfering with the local circulation. It may thus occur in connection with certain affections of the lungs, or even of the heart itself, impeding the return of blood from the cardiac and pericardial veins; and with disease or thrombosis of these veins, atheroma of the coronary arteries, aneurysm, chronic mediastinitis, or a mediastinal tumour causing pressure upon the veins. Hydropericardium has been known to follow sudden extreme pneumothorax.

Dr. W. Ewart (20) has drawn special attention to cases of latent and transient pericardial effusions, which may occur, independently of acute pericarditis, under the influence of rheumatism, of cardiac affections, of Bright's disease, and so forth. He considers that they may be dependent upon a subacute inflammatory process, but that probably they are more often passive or mechanically induced. No doubt such cases are met with, and if the fluid be rapidly reabsorbed they may run their course entirely unobserved.

Anatomical characters.—The essential morbid condition in hydropericardium is the presence of a quantity of serous fluid in the sac, which has collected during life, but which is not accompanied by any indications of inflammation. The amount varies considerably in different cases. In the large majority of instances it is moderate, from six or eight to twelve ounces; but it certainly may reach a pint to a pint and a half; as much as four pints have been reported, though it is very doubtful whether such large effusions are not really of inflammatory origin. The fluid is, as a rule, clear, and either colourless or of a yellowish or greenish tint. It is sometimes turbid from admixture of degenerated epithelium, or may be tinged with blood pigment or bile. Haemoglobin may, however, have escaped after death. The effusion is alkaline; and in composition re-
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...sembles more or less the serum of the blood, with differences in the relative proportion of the albumin and other constituents. Even a dropsical accumulation in the pericardium may be spontaneously coagulable. In renal cases it may contain urea. When the fluid is abundant it tends to produce, in proportion to its amount, the physical effects upon the sac itself, the heart, and neighbouring structures already discussed under inflammatory effusion. In prolonged cases the pericardium may become sodden, its epithelium being also changed; and it is said that the subserous tissue about the heart loses its fat and becomes oedematous. Possibly, moreover, the pressure of the fluid in course of time may impair the nutrition of the myocardium. In the majority of cases of hydropericardium, however, there is but little to be noticed beyond the presence of the fluid.

Clinical history.—The circumstances under which it occurs make it unlikely that there will be any definite symptoms of hydropericardium, especially if the fluid be but in small or moderate quantity. There is never any pain or other acute subjective sensation such as is met with in pericarditis. Should the effusion attain a large amount, it may certainly cause a feeling of weight and oppression across the chest, with precordial anxiety; and it will either induce or aggravate previous dyspnœa, obstruction of the venous circulation, and low arterial pressure, with the usual symptoms arising therefrom. In the large majority of cases it merely intensifies pre-existing symptoms, and it is often very difficult to determine the share of pericardial dropœy in their manifestation, though sometimes its effects are obvious enough, especially if it come on rapidly. It does not give rise to any febrile symptoms; and, as a rule, there is no particular disturbance of the heart’s action.

It will thus be evident that by physical examination only can hydropericardium be positively recognised. The absence of friction phenomena, such as are associated with acute pericarditis, is a most important point of distinction between the two conditions. The signs of the effusion are similar to those fully described under pericarditis, to which the reader is referred. As a rule they only indicate the presence of a moderate amount of fluid, and there may be so little that it cannot be detected at all. It is affirmed that the dulness is more readily altered by changes of posture than in cases of inflammatory effusion. Hydropericardium generally follows effusion into both pleœæ; and the physical signs of this latter condition will probably be well marked before those of pericardial dropœy are revealed. The combination may also cause a difficulty in diagnosis. I have never met with a case in which acute pericarditis and hydropericardium could not be distinguished by due attention to the circumstances under which they severally occur, and to the points of distinction already indicated. Possibly in connection with Bright’s disease an effusion might collect which it would be difficult to classify definitely as inflammatory or dropsical. As regards diagnosis, the chief danger is that hydropericardium is not thought of, and is consequently overlooked when physical examination would clearly have
revealed it. The cases of latent and transient pericardial effusion referred to by Ewart must also be borne in mind, for it is probable that even when considerable it is likely to be overlooked, unless accurate and searching physical examination is made. Should the condition be associated with, and secondary to certain local affections within the chest, the diagnosis may be very obscure and difficult. The prognosis in cases of pronounced dropsy of the pericardium is, for obvious reasons, usually very grave, and it generally indicates a speedily fatal termination. Temporary improvement or even recovery may, however, take place in some instances under favourable conditions.

Treatment.—As a rule treatment has to be directed to the cause of the hydropericardium, and the measures persisted in which have been previously carried out for the relief of the general dropsy which it usually complicates. It might be desirable in some instances to relieve the venous circulation by venediatheresection or local removal of blood. Cardiac tonics are to be used when required. The application of blisters has been found advantageous occasionally in promoting the absorption of pericardial dropsy. Whether tapping is permissible or desirable can only be determined by a careful consideration of the circumstances of each individual case.

V. Hæm- or Hæmato-pericardium; Blood in the Pericardium

Etiology.—It is not uncommon to find a certain amount of blood mixed with inflammatory products in the pericardium; but the circumstances under which pericardial hæmorrhage may occur as an independent condition are as follows:—(i.) As a consequence of traumatic injury from without, or by foreign bodies penetrating from the œsophagus. (ii.) Associated with scurvy, purpura, or, extremely rarely, leucocythaemia and allied conditions. (iii.) From rupture of the heart or of a cardiac aneurysm. (iv.) From lesions of the aorta. An aneurysm of the first part of the arch is very apt to open into the pericardium, not uncommonly by a pin-hole rupture. Rarely this event happens in the case of aneurysm of the descending aorta; and in one reported by Dr. Herbert Habershon the aneurysm was situated at the junction of the transverse and descending portions of the arch of the aorta. A case is reported by Dr. Charlewood Turner in which rupture of the inner coats of the aorta was followed by a dissecting aneurysm, which perforated into the pericardial sac. Dr. Rolleston has described a very interesting condition (39) where the inner and middle coats of the commencement of the aorta ruptured transversely, and the blood leaked into the pericardium through a small hole the size of a pin's head in the external coat; but there was no dissecting aneurysm. (v.) From rupture of smaller vessels, namely, one of the coronary arteries, especially if it be the seat of aneurysm; or of vessels in a new growth.

Anatomical characters.—The quantity of blood which collects in the pericardial sac varies under different circumstances. When there is a
large opening and rapid extravasation takes place, it is much less than when it escapes gradually through a small aperture. When an aneurysm bursts freely into the pericardium, the quantity usually found is said to be about 7 ounces, whereas in the case recorded by Dr. Rolleston already referred to it amounted to over 24 ounces. A traumatic case has recently been reported by Dr. Mansel Moulin in which over 6 pints of thin dark fluid blood were removed from the pericardium in the course of three hours. The patient recovered. The blood may appear as a soft red clot, jelly-like, or more or less decolorised; while a variable and sometimes considerable amount of serum will probably have separated from it. In Dr. Habershon's case the pericardium contained about a pint and a half of dark fluid blood. Hæmorrhage in the pericardium may set up pericarditis. The sac is distended in a proportionate degree when there is a large collection of blood in its interior.

Clinical history.—There may be previous symptoms or physical signs of the morbid condition which causes the pericardial hemorrhage, but not uncommonly such is not the case, and the lesion is quite unexpected and sudden. Immediate or very rapid death usually occurs, but the event may be preceded by grave cardiac symptoms or collapse. In those cases where the accumulation takes place gradually, the patient may live some time, and may complain of pain, associated with serious cardiac disturbance, faintness or syncope, dyspnoea, and signs of loss of blood. The physical signs, if noted, will be those of an accumulation of fluid in the pericardial sac. (Vide Dr. Allbutt's case, p. 767.) The prognosis is hopeless as a rule.

Treatment, as a rule, can only be symptomatic. Stimulants and cardiac remedies may be of temporary service in the more prolonged cases. No operative interference is practicable in the great majority of cases, but Mansel Moulin's case, above referred to, is very suggestive as to what may be possible in some instances.

VI. PNEUMOPERICARDIUM AND ITS EFFECTS; GAS IN THE PERICARDIUM

Pneumopericardium is extremely rare, and it needs but brief consideration in this article.

Etiology.—Gas in the pericardium has been referred to the decomposition of fluid in the sac, especially if the fluid be of an ichorous nature; and it has even been said that this is its most frequent source. The probability is that such decomposition, in the large majority of cases if not always, is a post-mortem change. Its presence has also been attributed to secretion by the membrane, but on no adequate grounds. The two classes of cases in which it is clinically important are—(i.) Traumatic, from penetrating wounds, including paracentesis for effusion; fractured ribs; contusion or crushing of the chest, or injury from the side of the œsophagus. (ii.) Perforative, in which a communication is formed externally, or between the pericardium and a cavity or tube containing air. This kind of lesion
has been already sufficiently described in relation to acute and suppurative pericarditis, and it will suffice to mention, as illustrations, perforation from the oesophagus, especially in connection with cancer; rupture into the pericardium of a phthisical cavity or pyopneumothorax; and perforation of a gastric ulcer. A remarkable case is on record in which a hepatic abscess communicated with the stomach and the pericardium, and thus air gained access to the latter. The entrance of gas into the sac may be aided by pressure, by the elastic traction of the lungs upon the pericardium, or by diminution of the size of the heart during systole.

Anatomical characters.—The gas in cases of pneumopericardium varies in its amount and composition, but it is generally offensive. It may so distend the sac, that when this is punctured the gas escapes with a hissing noise. Blood or other materials often gain an entrance at the same time as the gas; or at any rate inflammation is so speedily set up that pneumopericardium has never been clinically observed alone, fluid being always present, rarely serum—hydro pneumopericardium—usually pus—pyopneumopericardium; or the fluid may be ichorous. In a case described by the late Dr. Begbie (9), yellow lymph was present on the surface, and a quantity of dark brown foetid fluid in the sac. Whatever the position of the patient the gas will always be uppermost and the fluid below. The lungs will be pushed aside and compressed, and the diaphragm depressed, in proportion to the degree of distension of the pericardial sac.

Clinical history.—As might be anticipated, the symptoms of pneumopericardium and its consequences vary much in different cases, and are by no means characteristic. Sometimes there are none; or the patient is merely weak and apathetic. Should gas collect rapidly, there will probably be much precordial distress and sense of distension. The chief objective symptoms which have been observed in different cases are severe dyspnoea, cyanosis, fits of syncope, collapse, a feeble and irregular pulse, and rarely dysphagia. Sleep is necessarily disturbed, and delirium sometimes occurs. Occasionally pneumopericardium is accompanied with rigors, high fever, profuse sweats, and diarrhoea; but such symptoms are probably due to other and more general causes.

Physical signs.—It is upon the physical signs, that the diagnosis of pneumopericardium and its consequences is practically founded; these being due to the presence of gas and fluid within the sac: most of them are very striking and peculiar. They may be briefly described as follows:—

(i.) The precordial region is likely to present abnormal fulness or bulging, which may be very pronounced.

(ii.) The apex-beat is weak or absent, but is better felt when the patient bends forwards. Sometimes an impulse is observed over several intercostal spaces.

(iii.) The cardiac movements occasionally bring out a very peculiar crackling sensation, due to the bursting of air-bubbles. Possibly a suction-splash might be felt on shaking the patient.
(iv.) Percussion signs are usually very remarkable. Over the region corresponding to the distended pericardium there will be a tympanitic percussion sound, often with a pronounced metallic quality. It is said that a variation in its height, owing to alterations of the shape of the body of gas in the pericardium by the rhythm of the heart, may be detected by rapidly-repeated percussion. It has also been affirmed that the note differs in its degree of resonance during the systole and diastole respectively, the organ being situated farther forward and downward during the former period, and thus pressing back the air. A distinct cracked-pot sound has been described in several cases, but only when there was an opening in the pericardium. In the recumbent posture the extent of tympanitic resonance is greatest in front. When fluid is present, if the patient be slowly raised to the sitting posture and made to lean forwards, this area diminishes progressively, and the clear sound is replaced below by the dullness of fluid. Lateral changes of position will modify the relations of gas and fluid in a similar way, and thus very rapid and striking changes in the situation and relative limits of the respective percussion sounds are produced. Metallic instruments have been used to bring out the peculiar characters of the percussion sound.

(v.) Auscultation signs are also very peculiar, and often remarkable for their loudness. They vary according to the relative amount of gas and fluid in the sac, and the consistence of the latter; but as a rule different sounds are audible. If there be but little fluid the heart-sounds are abnormally loud, and are accompanied with a clear metallic ring, compared to a chime. Should there happen to be an endocardial murmur or friction-sound, it will probably assume a similar quality. The agitation of fluid and air within the pericardial sac by the action of the heart, and also by deep inspiration, produces adventitious sounds of the most extraordinary kind. They are all of metallic ringing quality, and have been described in different cases as splashing, spluttering, gurgling, gurgling, rattling, large crepitating, and churning. They have been likened to the sound of a water-wheel or mill-wheel (bruit de roue hydraulique, bruit de moulin); and in one case to the "shaking of shot in a shot-pouch." Occasionally metallic tinkling has been noticed due to the dropping of fluid in the pericardial sac. From a case observed by Dr. Flint, in which recovery took place, it would appear that sounds of the character just described might be produced by the presence of air and blood in this sac. In some instances the cardiac and adventitious sounds are so intense as to be heard, not only by the patient, interfering with sleep, but by those about him, or, it may be, even at a considerable distance off. Sometimes a splashing sound is brought out on succussion; or a bell-sound can be elicited by percussion with coins. It is affirmed that the signs of pneumopericardium have followed those of pericarditis, namely, friction-sound and evidences of effusion, when it is supposed to have resulted from decomposition of fluid.

Diagnosis.—If the physical signs just indicated were always pronounced, the diagnosis of pneumopericardium and its accompaniments
would be quite easy. Otherwise it would present much difficulty, or might be impossible. No reliance can be placed on symptoms. The only conditions with which it could possibly be confounded are a large cavity in the lung, in the vicinity of the pericardium; a localised pneumothorax; or a greatly distended stomach. Due consideration of the general circumstances of each case, and of the clinical history and phenomena, should obviate any such mistake.

Prognosis.—This is obviously very grave, and the termination is almost always fatal, especially as the pneumopericardium is usually a complication of some grave disease. A few cases of supposed recovery have been reported, but these have been chiefly of traumatic origin.

Treatment.—But little can be said under this head. The patient must be kept as quiet as possible, and in the position most comfortable to him. Stimulants, sedatives, or cardiac agents should be administered as circumstances require, but each case will dictate its own methods. The question of operation naturally presents itself, and in suitable cases it might be desirable to let out some of the gas by means of a fine trochar, the patient being in the recumbent posture; or to open up the pericardium freely, especially if it contain inflammatory or other products of a low type. This matter must be regarded and dealt with entirely from a surgical point of view.

VII. NEW GROWTHS AND PARASITES

In order to complete the account of diseases of the pericardium some reference must be made to the morbid growths which may affect it. At the same time it is difficult to say anything of clinical importance, and a few general remarks must suffice. The reader may also be referred to the article “Diseases of the Mediastinum,” in the sixth volume.

Tubercle is by far the most frequent morbid growth met with in the pericardium, and perhaps in its minor degrees it is more common than is usually supposed. It is only in exceptional cases, however, that the membrane presents gray granulations in general acute miliary tuberculosis. In the large majority of instances tubercle of the pericardium is chronic, and secondary to tuberculous disease elsewhere, especially of the lungs, from which it spreads directly. It may, however, follow disease of the bronchial or mesenteric glands. A simple pericarditis appears to be more common than tuberculous, even in cases of pronounced phthisis; and chronic inflammatory products in the pericardium may possibly become infected with tubercle. Dr. Habershon records an interesting case of general tuberculosis affecting unusual structures, where there was extensive tuberculous pericarditis. In a case of phthisis which came under my observation, changes due to chronic pericarditis were well marked, but careful examination failed to detect any tubercles or tubercle bacilli. In some instances gray and caseating tubercles are scattered over the serous coat, or in the midst of inflammatory products or bands of adhesion.
Carcinoma of the pericardium is extremely rare, and always secondary. The sac is nearly always involved by extension from neighbouring structures. A growth in the heart walls may project into the pericardium; but most frequently this structure is implicated during the progress of a mediastinal tumour, or one starting from the oesophagus. Exceptional cases are those in which cancerous nodules appear on the serous surface, associated with a similar condition of other serous membranes, these being secondary to cancer elsewhere. When the growth results from extension, the parietal portion of the pericardium usually presents a diffuse infiltration, but occasionally a nodular mass projects into the sac.

A case of malignant sarcoma of the pericardium, believed to be primary and independent, has been described by Sir W. Broadbent (14a).

Hydatids of the pericardium are so rare that out of 1897 cases collected by the late Dr. Davies Thomas of Adelaide, in only two was this structure affected. Moreover, in no instance had a hydatid cyst in the cardiac walls ruptured into the pericardial sac, probably because of adhesions between the two surfaces. This writer mentions one case, however, in which a cyst situated between the liver and the diaphragm ruptured into the pericardium.

The effect of any new growth in connection with the pericardium would probably be to set up inflammatory changes. These have already been fully discussed, and it will suffice to state here that they are very seldom acute; they may be subacute, but by far most commonly are chronic in their development and results, constituting the ordinary forms of tuberculous and carcinomatous pericarditis. The combinations in these chronic cases of adhesions, pericardial thickening, and localised collections of fluid, along with the morbid growths, may be very complicated. The effusion is commonly haemorrhagic; but in malignant cases it may be purulent or ichorous, and possibly also in those of a tuberculous nature.

Clinically new growths in the pericardium could only be suspected or recognised by the appearance of symptoms and physical signs of pericarditis, especially chronic, in such cases as tuberculosis or old phthisis, or an intrathoracic tumour. It certainly is desirable to watch the pericardium in cases of chronic phthisis, though, as already stated, the changes which may then arise are by no means always tuberculous. It is very likely that tubercle or cancer may produce a friction-sound, and this has been definitely asserted; but no definite diagnosis could be founded on this sign. The implication of the pericardium in these growths, in cases where the primary seat of mischief is away from the chest, could only be made out by the occurrence of pericarditis and its consequences, which would draw attention to this part.

Treatment is entirely symptomatic and constitutional, and no definite rules can be laid down. Operative interference might be indicated for the removal of fluid to give temporary relief, but nothing can
be done for the growths themselves. Obviously when the pericardium becomes involved in malignant disease the end cannot be far off.

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REFERENCES

FUNCTIONAL DISORDERS OF THE HEART

To the purist the vulgar distinction between functional and structural disease is a false one. We are assured that in every change of function a change of structure is implied; indeed, that structure and function are one, and to use them severally is to see the same thing in different aspects. It is not so much that the materialist and the idealist have lain down together, as that the idealist has swallowed the materialist. Yet, granting all this, we remember that as it is convenient to detach the study of physiology more or less from that of anatomy, so it is with nosology when we analyse symptoms apart from morbid anatomy; although we shall not forget that knowledge thus obtained must be integrated by bringing the two studies together from time to time.

Furthermore, we shall not be discouraged from using the term functional disease in a still narrower and more artificial sense,—in the sense of a perturbation of a more or less contingent kind, of a contingency sufficient to rock but not to upset the moving equilibrium (11). Every beat of the normal heart is a disturbance of equilibrium, and we do not forget that, in any system, cessation of all disturbance is the peace of death; on the other hand, disturbance beyond the resistance of the equilibrium is disease or death also. Between the death of apathy, as of the old man who falls asleep, and the death of defeat, as of the man who succumbs in his prime to a clot in the pulmonary artery, there may be two periods,—the period of health and the period of transitory discord. In health the disturbances are rhythmic, harmonious, controlled; in functional disease they are arrhythmic, uncontrolled. In functional disease the going system halts or staggers, but not beyond recovery; the humming-top swerves under a puff of wind, or reels as it travels over a grain of mustard seed; but the deflection is counteracted, and is presently resolved. Such temporary eccentricities are common to the heart with other organs, but are more conspicuous in the heart, because its workings are nearer to our consciousness, and lie, moreover, in the track of emotional gales and typhoons. Is there a man so stoutly knit, whose inhibitory nerves are so powerful and alert, that in passion or "twixt doubtful fear and feeble hope" he has never felt his heart climb into his throat? Thus it is that functional disorders of the heart are familiar to us all, and occupy our thoughts the more, as the heart tells us where
the centre of life is, and where we cannot afford to have things go wrong. But it may be objected, and in a very important sense truly objected, that these are but matters of degree—that persistent functional disease ends in structural disease. With this inquiry we shall deal at length; meanwhile I would say that this is not necessarily so. While, on the one hand, we warn the student not to overlook the stealthy inroads of structural disease, of “functional” disorders, which are the first signs of the invasion of structural disease—such as retardation of the heart, for example, on the other hand, we shall not put them in the same reckoning with the functional disorders which are not of this kind—such, for example, as acceleration of the heart. Whether a purely functional disorder by damnable iteration can hammer disease, as it were, into a harassed organ is hard to say; as yet we can only say that in many cases a lifetime of functional disorder of no little persistency is not long enough to bring this event about, and perhaps that such is the usual issue: on the other hand, it seems no less certain that perennial depressing causes, exile or bondage in an invisible Babylon, may induce degenerative changes in the heart and blood-vessels, or in the kidneys, as I alleged in 1877, and have had yet more reason since to believe. That tachycardia, usually perhaps when severe, may wear out the heart is true; yet I scarcely think we can regard this truth in the light of our present argument, as such gradual inroads are rather of the nature of dilapidation than of mere disorder: moreover, in a particular case it may be hard to distinguish between a perturbation, such as a variation in rate, which is an indication of incipient heart failure, and a perturbation of central or eccentric nervous origin. Anxiety long continued seems to pervert nutrition at its sources; perhaps to prevent healthy metabolism, and to favour auto-intoxication with its damaging effects on kidney and heart. Such influences, however, come rather under the head of the remoter causes of diseases of the myocardium than under that of functional disease of the heart; as, again, many of the conditions of functional heart disorders will be dealt with in the chapter on Neuroasthenia. For our present purpose functional disease may be taken to include temporary irregularities of rate, rhythm and tone, and even of force and volume; though these last rather pertain and are subordinate to other diseases—that is, to other symptom groups. While rate, rhythm, and tone make important parts of many maladies, yet their errors are often themselves the leading morbid features, and appear to the patient, and often indeed to his medical adviser likewise, to stand almost alone. For instance, if in the irritative stage of meningitis we mark a slow pulse, we do not group this phenomenon with functional disease of the heart, however logically we might do so; thus to class it would be to darken our conceptions, to introduce false connotations. So again the quick pulse of a later stage of meningitis, or that of pneumonia, will in like manner be classed, not with functional diseases of the heart, but with the phenomena of fever. For our present concern is with clinical medicine, not with the broader views of general pathology.
A more difficult problem of nosology is to decide where we are to place the quick pulse, say, of larval Graves' disease; if both goitre and exophthalmos be absent, as often they are, are we in the presence of an obstinate case of functional disease of the heart? Again, I think that to speak thus would be an abuse of terms; if we suppose that on due analysis this pulse has affinities with the symptom group which we call Graves' disease, we must not put the pulse characters in an independent category; we shall regard them as a part of that other group. Let it not be said that this discussion is otiose; for if the argument be well founded we shall no longer allow ourselves to call any quick pulse "tachycardia," nor any slow one "bradycardia." Tachycardia, for instance, appears to be a definite and primary functional disease of the heart; the affection has characters of its own: whether bradycardia is such a substantive malady is less certain; this question we shall discuss presently. If it is not, the specific name should be given up, as one without a consistent signification.

We cannot consider the heart apart from its nervous connections; like a well-handled pair of horses, its good going depends as much on the man on the box as on the muscles in action. Although the heart muscle has an independent and inherent rhythm of its own, this rhythm goes astray if the organ be severed from its nervous governance. The inherent rhythm may suffice for less complex organisations, but it will not do for a mammal. In the higher animals, for instance, the contraction of the left ventricle, although it is always a maximum effort, does not at every beat supply the whole arterial tree. That at a very low pressure, all the arteries being expanded, it might do so is possible; some of the strange perturbations of women attended with heat and flushing may thus come about; but probably even in them the distribution is more or less partial. In health, at any rate, the output is turned now here, now there, as—if I may be permitted so unsavoury a simile—in a sewage farm the fertilising streams are diverted by locks in this way or that. The lock-keepers belong to the nervous parts of the cardiac machinery. In study the active brain, after a meal the stomach, demand their alternative streams; by means of the nervous system an anemic area calls for more blood, satisfied areas for less; and by means of the vagus nerves the heart itself is protected from too great an importance. If in an anemic girl the heart beat too fast, we shall not call that a functional disease, which is an attempt on the part of the heart to respond to the cries from anemic areas all over the body; though in many such cases as this we do for the moment, and provisionally, apply such a name to mark a region of our own ignorance.

As we carry our explanations into such regions we gradually diminish our group of functional diseases of the heart. Let us consider the effect of certain poisons on the heart. In so far as these and their effects are known—as, for example, in the cases of coffee, tea, and tobacco—we shall scarcely call their ill effects on the heart functional disease of this organ; we shall turn rather to the chapters on these
drugs, and regard the cardiac perturbations subordinately as features of
the symptom group or series of groups associated with the agent concerned.¹
Now the heart is often set on edge by obscure causes which seem to us
to be of the nature of poisons, of poisons generated, perhaps, in the
body and circulating in the blood which irritate or depress the heart
directly; or, perhaps, disturb it indirectly by some obscure interference
with the blood-pressure: such a state of things is surmised to exist in
the malady popularly known as “suppressed gout.” But when we know
all about “suppressed gout” and wherein it consists, we shall remove the
cardiac phenomena from the chapter of functional diseases of the heart,
and put them in their own place as subordinate phenomena of the gouty
group. All we know about “suppressed gout” at present is that it is not
a mere dilution of articular gout; that, however related to the latter, it
is a different disease rarely occurring in the same persons; or, if in the
same, at different times of the life of the individual. Cardiac distur-
ances often appear; it is true, in articular gout also, and are described in
treatises on this disease as “gouty”; but of “suppressed gout” high arterial
pressure is characteristic, from articular gout high blood-pressure is
commonly absent. How terrific and how various may be the effect
of the poisons of certain of the infectious diseases upon the cardiac
mechanism is familiar to us all. In diphtheria the heart’s action may be
reduced “almost to extinction” (Powell), and of the effects of influenza in
the same direction an excellent account is given by Dr. Sansom (11).
Syphilis, again, is said to cause irregular heart, as a functional disorder
apart from arterio-sclerosis. Of this I have no personal knowledge.
Such considerations as these seem to threaten the very existence of
Functional Diseases of the Heart, save in the sense of a survey of the
general behaviour of this organ under all sorts of maladies, not excluding
its own structural diseases. Meanwhile, however, we must deal with the
unrelated cardiac disorders in a somewhat miscellaneous way; and certain
of them seem to have an individuality of their own. Before studying
functional diseases of the heart as groups of symptoms, we may profitably
consider the elements of the groups separately—such as tone, tension,
rate, rhythm, volume, and so forth.

Tone.—The old-fashioned word “tone” has fallen into disuse; the
more is the pity. When I was a student we were asked how the pulse
might be for tone; now if a student be asked such a question he talks
about “tension,” although he does not clearly know what he means. To
measure or even to estimate roughly the degrees of stretching of the
coats of an artery is a very complex and usually an insoluble problem;
yet to these coats only can the word “tension” apply. The blood itself
cannot be tense in any but an abstruse mathematical sense, which no
student of this subject has in his mind. If the radial artery contract
tightly on the blood within it, the pressure on each superficial unit of

¹ A pair of interesting tracings is published by Dr. Waller on page 33 of his Physiology
(ed. 1897); the first of irregular and low pressure pulse under tobacco, the second a correc-
tion of the same pulse under digitalis.
internal surface is increased no doubt; but this is not tension, or at any rate not in any simple sense. Tension is that stress which tends to split the artery either longitudinally or transversely; and such stress is at more advantage when the vessel is relaxed. Tension and tone have, indeed, something like an inverse relation one to the other, as we see more readily, perhaps, in the ventricles of the heart. We may say, indeed, that one of the chief functions of tone is to resist the tension which calls it forth. How tension acts upon an artery is best seen in aortic regurgitation, in which malady the effects of tension seem at their highest. We have but to look at any long artery in an advanced case of this kind to see what tension, in the longitudinal direction, really is; the artery is not actually split transversely, perhaps, but it is lengthened enormously and thrown into curves. No doubt, under all circumstances, whether the radial artery be tight or slack, there is more or less tension of its coats; but it is most difficult to ascertain the degree of this, even roughly: yet such is the love of obscure diction, that, instead of endeavouring to express the facts in terms as comprehensible as possible, that factor which is at once the least appreciable and the least immediately important is chosen for description.

Without saying that any factors in this application of hydrostatics are easily estimated, we may assert that tone and blood-pressure are easier to measure approximately than the tension of the arterial coats. The finger can tell with some approach to accuracy whether the pressure be low, moderate, or excessive, though it is only by such instruments as the sphygmometers of Roy, Leonard Hill, or Oliver that the degree of it can be recorded. Tone, again, is easy to make some guess at, or even to formulate with sufficient accuracy for clinical purposes. Tone in a vessel is that which preserves its mean diameter, which preserves a certain proportion between the extremes of dilatation and recoil, and which has furthermore the somewhat different virtue of keeping the vessel well home upon its contents. Therefore when we speak of a pulse of good or ill tone we are not talking altogether of what we do not understand. We mean that the difference of pressures between the base of the pulse-wave and the apex is somewhere about 35 mm. Hg. And again, when we speak of high arterial blood-pressure we are talking of that which we can estimate with some correctness—namely, a mean pressure of about one-eighth of an atmosphere. These two conditions the skilled finger is able approximately to ascertain. But when we speak of the tensile stress on the walls of a vessel we are talking in the dark; other things being equal, the higher the blood-pressure the more the tensile stress, but until we have allowed for tone the net tensile stress, however considerable it may be, is inappreciable. Now, in functional disorders of the heart and arteries tone is often signally deficient. The aorta, structurally healthy, may nevertheless be seen beating diffusely in the episternal notch and in the epigastrium; the wall of the chest may thrill as the hand is laid over the heart; the sounds of the heart are carried far along the vibrating walls of the carotid; the abdominal aorta
leaps like an aneurysm; nay, even the patient himself may complain of the bounding of slack arteries all over his body. In some such cases even a capillary pulse may be seen. To the finger the radial or other artery is ill-filled, and the sphygmographic curve shows that the due proportion between the expansion and the recoil of the vessel is no longer preserved; the lever falls almost to the abscissa before the dicrotic wave is formed. I have often seen a temporary extension of the area of cardiac dulness in such cases. This state of the circulation is perhaps never so primary and eminent as in amount to a functional disease of the arterial circulation, and as a derivative condition its importance is discussed under Chlorosis, Neurasthenia, and elsewhere. The mechanics of the subject will be dealt with hereafter by Dr. Leonard Hill.

Tone, Dr. Gaskell tells us, is innate in muscle, but it may be excited, raised, or reduced by nerves. Tone may vary under nervous governance, but it persists beyond all nerves. Some of Dr. Waller's experiments suggest that nerves like muscle may have their refractory periods, and the same character has been indicated by certain observers, for example by Richet at the Toronto meeting of the British Association in 1891.

Rate.—How widely the rate of the heart-beats may vary between its extremes is too familiar to need description. In one bed may lie a patient with a pulse of 30, in the next one whose pulse is 180; and even these are not the utmost extremes. Under bradycardia and tachycardia we shall discuss those phenomena more intimately. The most general factor in acceleration of the heart is loss of vagus control, for the vagus may be regarded as the escapement of the arterial train. Loss of vagus control may be relative or positive; the accelerator nerves may be abnormally stimulated, and thus may overbear even a normal vagus control; or the vagus may itself be more or less in abeyance, as after a dose of atropine which paralyzes its ends in the heart. Again, agents acting directly on the heart itself may either stimulate the vagus, and so slow the pulse, or may overbear its control and the pulse-rate may rise; variations in blood-pressure have these effects, an increase of pressure tending, as a rule, to the retardation of the heart, and a fall to acceleration of it. In functional heart disorders we are frequently met by problems of this kind, and sometimes they are very difficult to analyse; we may remember, however, that controls are a later development than the functions below them, and therefore tire sooner. Vaso-constrictor action never tires so long as the nutrition of these nerves goes on, and the vagi tire before the accelerators. Thus the accelerating nerves often fatigue the vagi and run away with the heart. This may be the explanation of rapid pulse in certain poisonings, infections, and the like; but we have also to remember that in fever blood-pressure often falls also, probably from some change in the viscosity of the fluid; and again that quasi-normal catabolic products may act directly on the heart, as we believe that fatigue products do. That states of the cardiac muscle itself
are often directly concerned in its rate seems also probable from the clinical phenomena of "irritable heart," which can scarcely be due to fatigue products only. Conversely fatty degeneration of the heart is often betrayed by retardation of the pulse.

Once more; we have to deal not only with the nerves, but also with the cardiac centre in the bulb, a nervous factor which may conveniently be considered apart, as through its efferent fibres it is chiefly concerned in regulating response to the demands of the system. Not in the case of circulating poisons only, but also under the fluctuations of ordinary blood changes, the cardiac centre is constantly in exercise. In haemorrhage or chlorosis, for instance, the call of extensive anaemic areas throughout the body,—the afflux, in this case, of impoverished blood to the cardiac centre,—excites the centre to quicken the heart. On the other hand, a rise of arterial blood-pressure stimulates the vagus roots in the bulb, and the pulse is slowed. The name tachycardia, as we shall see, is improperly applied in the sense of mere rate; it is the name of a particular disease. The name "embryocardia," which is creeping into clinical language, is pedantic if it means merely a very rapid heart, misleading if it suggests that the heart has undergone some reversion to a fetal quality, or even that the organ is primarily failing. The heart goes "tic-tac" whenever its rate reaches a certain degree, and I may repeat that a quick heart is not in itself a sign of enfeeblement, but of extreme reflex excitation of the accelerantes, due probably in typhoid and the like to a diminution of the total volume of the blood, or to alterations of its density, though, no doubt, the effects of morbid or catabolic poisons often intensify the state. It must be remembered that a rapid rate does not necessarily mean an increase of total work done: on the contrary, although dilatation is no uncommon result, hypertrophy, in the absence of valvular disease, is rare.

Abnormal rates of the heart depend then on many factors, and the variation of any one of these will modify the action of the organ under observation.

Rhythm is not synonymous with rate, as is too often assumed. A few weeks ago I read a valuable physiological essay in which rhythm was used almost throughout in the sense of rate; such abuse of language leads to confusion of thought. Rhythm is not the rate but the proportion of motion. Strictly, force and volume are contained in the conception of rhythm; but custom and convenience have ordered that by rhythm we shall mean the numerical proportion of motion; that is, a true cardiac rhythm shall consist of the same number of beats in every unit of time. Here again, although we find that the vagus is chiefly concerned in the variations of rhythm, such variations being due for the most part to vagus interference, yet, as in the case of rate, we learn that the rhythm at a given moment is due to a composition of causes which are not always easy to analyse. For instance, clinical experience suggests to me that intermittence of the heart is often due to a direct effect on the cardiac muscle itself, or is a compound effect of direct influence on the heart and
vagus together. Digitalis may be an instance of an agent acting in such a double fashion, and some morbid poisons, such as that of influenza, seem to have a like compound property. Intermittence, transient as it usually is, is no uncommon feature in the degenerate heart.

We divide disturbances of rhythm into "irregularity" and "intermittence," terms which speak for themselves. That these two abnormalities may be and often are present together is familiar to every student. Irregularity of rhythm is for the most part graver as a sign of disease than intermittence. Its signification in muscular and valvular disease of the organ in chorea, in cerebral disease, and so forth, will be discussed in the several parts of this work which deal with such subjects. I need scarcely say that there is an irregularity of the radial pulse and another of the heart, and herein we see that irregularity is not only an alteration of rate, but also of volume and force; the ventricle not only acts irregularly in time, but also delivers variable quantities of blood with variable impulse; the output is unequal. There may be, as in cerebral disease, for instance, an irregularity of time only, the volume and force remaining constant; but such a condition is rare, for if equal quantities of blood are not delivered from the several chambers in equal times, inequalities of distribution in the chambers and of systolic output must accumulate. Strictly speaking, no pulse is regular, as a time line at the foot of a sphygmographic tracing will prove; if not otherwise influenced, the respiration at any rate disturbs the order, as does muscular effort, even the slightest, especially in nervous or otherwise unstable systems. To ascertain how far the effort and position of the upright attitude, or a slight muscular exertion, quicken the rate is a good test of vascular resistance; for Dr. Waller's electrotonic work brings out into more prominence the truth that increased capacity is associated with diminished susceptibility to contingent impressions, such as relatively slight changes of blood-pressure. At the same time, it seems that in some persons the pulse is habitually irregular in the clinical sense. Sir Thomas Watson mentions such a case in a brother of his own; whether the brother was a tobacco-smoker his distinguished kinaman does not record. In my own experience I have often met with an irregular pulse in smokers—never, I think, in the normal state. In acute disease irregularity generally means irregularity of output, and warns us of evil: probably of dilatation of one or both ventricles.

Intermittence is often of grave augury, no doubt: in suspected cerebral disease it is an alarming sign; it is a grave sign in any acute disease, especially in the pulmonary attacks of the elderly; but in cardiac disease it is of less gravity than irregularity. It is common enough also in dyspepsia, in suppressed gout, in smokers, and even in persons in whom no flaw is to be found. I once found intermittence in two brothers who came together to me for life insurance; both of them were very angry with me for refusing them, or rather for stating the facts which led to their refusal by the company. Neither were smokers, or very moderately so, nor were they large tea or coffee drinkers. They were vigorous young men, their digestions were good and their teeth sound. The intermissions
were occasional, on an average about one in thirty or forty. Perhaps no one passes through life without an occasional sense of cardiac intermission; and therewith is often found, though at much longer and more uncertain intervals, a flutter, felt rather in the epigastrium than about the heart. This flutter seems not always to be cardiac; there may be some alternative machinery for its production: sometimes it is certainly due to a series of rapid and irregular beats, but the disturbance is so quickly over, so hard to catch, that its precise causation is undetermined. This flutter, like the intermittence which is often associated with it, is of dyspeptic origin; and the best remedy for these discomforts, for they are little more, is to insist on slow mastication. They are very apt to arise in persons who bolt their food. It is incorrect to say that if such intermittence arise in advanced life it necessarily signifies incipient cardiac degeneration, for even in cases when the symptom has endured for two or three years in persons of sixty years and upwards, careful attention to the diet and a vigilant supervision of the use of coffee, tobacco, and the like, will spare them to die at a riper age of some other symptoms; on the other hand, even in much younger persons, intermittence may accompany vascular deterioration, cardiac strain, or valvular disease. Sometimes the intermittence is radial only; the heart beats regularly, but not always effectually. Sometimes the intermittence is rhythmic; it will occur every two, three, or four beats for a while; such an intermittence is often found in persons under the use of digitalis. As a functional disorder the form is insignificant, or no more significant than ordinary sporadic intermittence. To say that the "pulsus bigeminus," the "pulsus trigeminus," or the "pulsus alternans" is a sign of cardio-arterial degeneration, to assert that it is necessarily significant of grave cardio-arterial involution, is to ignore daily experience. If indeed it be associated with an abiding or persistently recurrent retardation of the pulse the prognosis is less hopeful, as it may be also when such coupled intermittences obstinately return in spite of treatment. I had written these lines when a pamphlet by von Noordden came into my hands, giving descriptions and sphygmograms of such pulses in hysterical cases (9). It is said that an intermittting action which does not reach the consciousness of the patient is of worse omen than that which attracts his attention. Many persons are alarmed by a perceptible intermittence, especially by the bounce which often follows it; perhaps it is this bounce or thump, rather than the intermittence, which gives rise to the well-known sensation. Certainly that the comparatively harmless intermittence is perceptible enough common experience tells us; and I have noticed that intermittences occurring in failing hearts are less obtrusive or indeed unfelt; whether the absence of the sensible bounce indicates a feeble heart in all instances is more than I can say myself or find in the records of other observers. Certainly in the intermittences of acute disease, as of senile broncho-pneumonia, the missing beat is not perceived by the patient. The mechanism of intermittence is not quite understood; it is probable that a beat occurs, but is abortive, and
the bounce is a leap of the heart against the low pressure of the unfilled arteries. The sign is more ominous when associated with irregularity.

**Palpitation.**—This disorder is even more common than intermittence; in greater or less degree it lies within the experience of every one. It is more common in women than in men; and in the former is often a very distressing and persisting torment. Under the alarm of a severe attack of palpitation, with its no less painful sense of choking, even long and trying experience is scarcely enough to steel the patient against the dread of its return. Indeed, as the gale in which the heart is caught often arises from the quarter of the nervous system the apprehensions are disordered as soon as the heart itself, or even before it. A sensitive woman, physically courageous perhaps, yet one who starts at every sudden sound, may well be appalled by the fear of heart disease and of sudden death. Attacks of palpitation often pounce upon the sufferer in a moment—even in a quiet moment—and, it may be, without apparent cause. It is no unusual thing for an attack to set in with nightmare during sleep. Either thus, or more gradually, the heart begins to throb tumultuously, and its function is often beset in all the directions in which we have been regarding it; it becomes irregular, intermittent, variable in force, volume, and rate, though always rapid, until the vagus control is regained either by the lapse of time or by some reflex stimulant such as smelling-salts, or a cordial; or again by some pain or conflicting impression. The attack may subside gradually, or it may cease suddenly with a shock, as if rending the patient before quitting her body. Such a finish is usually seen also in tachycardia, and may be due to the same causes as the throb of an intermittence. The patient instinctively presses her hand upon the region of the heart during palpitation; a kindly pressure seems to soothe the tumult. Under the hand the heart's beating, like the arterial pulse, is vibrating, diffused, turbulent, and disorderly; now striving and violent, now tremulous and faint. The attack is followed by the calm of exhaustion. The history and circumstances of such seizures are generally enough to serve us for interpretation; indeed, such storms are unusual in organic cardiac disease. Still, the static conditions of the heart are not often to be appraised during the discordant and confused dynamics of such seizures. It is well, in the case of a new patient at any rate, to postpone a final diagnosis till the ship is in calmer waters.

*Murmurs* are often present in the palpitation of functional disease; they may be heard at apex or base, and at any part of the cardiac revolution. A systolic murmur at the apex is the most frequent of these. The causation of these transient murmurs is unknown; some may be "anaemic"; some may be due to inordinate action of the papillary muscles; some again may be "pulmonary" (Potain). Until the patient is tranquil, and the physician at liberty to map out the heart and to listen to its sounds without embarrassment, no final opinion should be given. In a functional case the murmur will probably then have ceased, and dilatation, if any, will be reduced; although resonance of the second
sound at the apex and the sharp knocking quality of the systole will probably mark the case as neurotic.

Of these murmurs Dr. Sansom says (13) that a systolic murmur, arising independently of structural disease, seldom attains its maximum audibility at the exact apex, but slightly to the right and left of it. It is usually soft, and does not replace the first named. Again, it does not occupy the whole, but the middle of the systole ("it is meso-systolic"). It is much influenced by respiration; it is intensified both during expiration and inspiration (especially the latter), but it often becomes inaudible at the end of an expiration. I may add that to me the quality is often that of the apex first sound in those cases of systolic murmur generated at the base in which the murmur is scarcely audible, as such, at the apex, yet where the first sound is blurred by it. For Potain's elaborate and almost too ingenious doctrines concerning the pulmonary origin of such murmurs—anaemic and the rest—the reader is referred to his well-known article in the Clinique de la Charité, 1894.

The immediate prognosis can rarely need much direction. Generally speaking, the diagnosis in such cases is too dark; a woman is told that she has got a "weak heart," and thus the confidence in herself which is essential to her cure is shaken. The palpitation of chlorosis I am accustomed to regard as the result of the combination of poverty of the blood in oxygen value with persistent mass, with no less a demand upon the heart, that is, in respect of output. The treatment during the attack consists in recumbency, warmth to the legs and feet, and such stimulants to the abated vagus nerves as ether, ammonia, valerian, smelling-salts, and hot applications to the cardiac region; remedies which are rather to be recommended than alcohol. Belladonna also is better avoided, and digitalis, if an occasional ally, is not to be trusted. In acute attacks these measures will suffice; but in some cases the palpitation does not take the form of isolated attacks, but, though less violent, is either persistent or chronically recurrent. In these cases treatment, if addressed still to the vagi, may well be addressed also to the accelerators, especially if the pupils be dilated and the face flushed, and thereby excitement subdued. As palpitation, if consisting partly in defect of central control, is nearly always set up by some eccentric cause, rules for general management, such as regulation of the bowels and other secretions, attention to piles, uterine disorders, overwork; temperance in food and avoidance of alcohol; moderate exercise, cold baths, and regular hours of sleep, will be found in the articles on hysteria and other neuroses. At times such sedatives as aconite and the bromide of soda, ammonia, or camphor may be needed. Aconite has served me well in many such cases, and its use, cautious as it must be, may yet be more than occasional. With palpitation run other symptoms, such as precordial pain, panting, globus, vertigo, and perhaps even syncope—though I have never seen it under ordinary circumstances. During the attack the urine is scanty, but it is generally profuse after it, as in megrim and other neuroses accompanied by fluctuations of blood-pressure. Such symptoms receive
full attention in other parts of this work. The causes of palpitation, also, are dealt with elsewhere. I will but remark that sudden vaso-motor changes, either in the direction of constriction or relaxation, are common incidents in palpitation, and perhaps common causes of it. When we remember that, in the bulb, the cardio-inhibitory, the vaso-motor, the respiratory and the gastric centres abut upon each other we shall feel no surprise that the functions related to all these centres should often influence each other or be influenced together. The expulsion of a worm has sometimes proved to be the cure of troublesome palpitation. Palpitation coming on for the first time in later life is a matter for anxiety, but may be gouty or dyspeptic (bad teeth).

False palpitation.—It is not uncommon for patients, especially for highly neurotic or neurasthenic patients, to complain of palpitation although on examination little or nothing of it is perceptible, or the heart may be accelerated by some five or ten beats at most; yet to judge by the bearing of the patient the distress is acute. Such patients will probably complain of other hyperaesthesia, and of pains in other regions, such as the head and back. In the cardiac region the patient complains of tightness and oppression—"precordial anxiety"—of urgent heaving, or bursting of the heart, or of cramp in the part, in which they fear to die. Or the pains may be boring or cutting: the husband of such a sufferer, in writing to me, tore out from his Bradshaw the advertisement of a corset-maker, and drawing a dagger with its point entering the left submammary region, enclosed the picture as a graphic representation of his wife's agony. As many of these patients suffer from air hunger and pains in the chest and arms the cases melt into the class of "pseudo-angina pectoris." The attacks may recur many times a day, and are not difficult to appraise in the broad sense as neurotic: the story of the case rarely leaves much doubt of this interpretation. The blood-pressure rises during the attacks and rapidly falls as it passes off. But in my opinion the vaso-motor phenomena are not causes, but consequences—are of reflex origin and secondary to the neuralgia or distress. As auscultation and other means of investigation reveal no change or but little, the intimate nature of these phenomena is not easy to ascertain. In ordinary palpitation, as the pulse rises, perhaps to 150, the pressure falls and the face flushes; or the patient turns pale and the pressure rises; but neither of these events is seen in the false palpitation. Until a better hypothesis is suggested, we may suppose that there is some morbid susceptibility to the impact of ordinary stroke of the heart. There would seem also to be a like hyperesthesia in the vessels, as rushings in the arteries, whizzings in the head, and other "determinations of blood" are complained of, sensations perhaps due to slackness of the arterial walls.

Weak heart is used in two senses; as a heart of lax or even failing fibre, and as a heart subject to certain kinds of transient disturbance. Of the former we have not here to speak; the second is as follows:—The patient usually, but not always, a neurotic woman, tells us the heart ceases to beat; in the severer cases the patient is convinced of this,
and fears that each attack in turn may be fatal. Sometimes as the attack comes on the face turns gray, and the lips blench; in other cases illness is betrayed rather by the expression of apprehension and distress in the face than by any signs of organic disease. The hand is pressed to the region of the heart where pain may be felt; but often it is not so much a pain or a throb as a sinking, and the sinking is not at the Heart only, but a general "lyphothymia." She may also complain of pins and needles, of turning cold, and other evidences of irregular blood distribution. After a time the distress passes off, and the patient recovers with that sense of extraordinary exhaustion which is so well marked in functional affections of the heart. The pulse during the attack is not very characteristic; it is certainly weaker, it falls more or less in pressure; and therewith it increases in rate a little—say 100 to 110; but it is not the pulse of syncope, nor indeed do these patients faint away; they gasp and return to life with a sigh or two of relief. Speaking generally, there is no danger in the attacks except that which lies in the habit of taking drams to cure or prevent them. Mrs. Gamp's prescription of two drops of brandy on a lump of sugar is too well known to these patients and their friends; the medicine is at hand and is assiduously administered, with the rubbings of the extremities, the hot bottles, and the like, which are grateful to these patients. And no doubt for the moment the alcohol is helpful; it pulls the heart together, or imparts something or other which may be mere Dutch courage, or something more mechanical; probably its chief effect is to dilate the arteries of the surface, and thus perhaps to divert the blood from the splanchnic areas into the arteries of the skin and limbs which were certainly for the time anaemic. It is not apparent, however, that constriction of the arteries is primarily at fault—a dilatation of the splanchnic areas may be. Almost as I write these lines I saw with Dr. Henry Head a very curious case of functional cardiac instability. A gentleman, aged thirty-three, apparently healthy in all other respects, but of nervous temperament, complained to us of breathlessness on ascents. No anginal or other pain. At nights he awakes with a sense of faintness or impending death. His pulse, while he is standing, is 130, and but little less on sitting down; but as he lies down flat the radial pulse instantly undergoes striking oscillations for two or three seconds, and then falls to a steady rate of 80. He has some reason to suspect that his nocturnal discomforts are due to a still slower rate of the circulation. Aspect healthy; no cyanosis. Does not smoke nor drink tea or coffee. The heart on examination proves to be free from any abnormal sign, unless it be that the apex beat is obscurely seen, and the impulse rather diffused. He has had attacks of the kind before, if not quite so severe, and has always been cured by going to sea. I thought that the effect of a well-adjusted abdominal pad might be tried. I have seen at least one other such case. As bearing on the conclusions of Dr. Leonard Hill and Dr. George Oliver, I may say that in one patient, who suffered much from heart sinking, as above described, to raise the arms was almost a certain means of producing an attack or a threatening
of it; hence she assured me that she dared not raise her arms to knock
at the outer door of a house. Until I read Hill's papers I thought this
was all moonshine; now I think it was not. Whatever be the underlying
conditions, the repeated taking of drams is very mischievous; it encour-
gages the very oscillations in blood distribution which we ought to control by
the wet sheet, douche, regular exercise, massage, and such means;
and after a few months or more of the dram-drinking the doctor is told
that an unaccountable nausea and retching in the morning, and loose
motions, either before breakfast or during the forenoon, are added to the
tale of her symptoms. The next stage is that of pains and palsy in the
legs and feet. Such is a common enough story. Some cordial these
patients will have, perhaps ought to have; they are frightened out of their
wits, and a stimulant seems their only help. Well, then, let us prescribe
ether, valerian, ammonia, or peppermint for the moment; and as the
immediate anxiety passes away, attention to the general therapeutic needs
of the case will, in a broader and more wholesome sense, ere long remove
the need for dramming at all. It cannot be too strongly urged upon these
patients that temperance, even to the point of total abstinence from
alcohol, is paramount in the treatment of neurotic cases: it is even a
more important condition in them than in the gouty. Cardiac neuroses
are nearly always part of general neurosis; in all its phases neurosis
means lack of inhibition, relative or positive; generally relative. Dr.
George Oliver's comparisons of the range of radial volume in the healthy
and in the unstable respectively are full of instruction in this respect.
It is possible that in some of such cases there may be a lack of suprarenal
incretion and a corresponding loss of arterio-vascular tone. But this is a
dark matter; I have even found the radial blood-pressure rise at the
outset of an attack and fall again as it passed off. In these phases of
high initial pressure the patient is flustered at first and sinks afterwards.
An increase of the muscular reflexes is often seen in these patients, as in
the following disorder:—

Passing by gradations into, or even confused with the above derange-
ment, is that of cardiac asthenia, which, in a recent pamphlet, Da Costa
has distinguished from irritable heart (4). The author says that for
long periods the action of the heart in these sufferers is feeble; a feeble-
ness to be distinguished from the weakness due to organic causes, and
again from that of lassitude, gout, tobacco, and the like.

The affection generally manifests itself in those persons whose nervous
system has been strained by worry or overwork; whatever the warning
signs, the full brunt of the disease is often sudden in its incidence. The
patient is prostrate in bed; all attempts at sitting up cause swooning and
vanishing pulse. The heart's action is feeble; the pulse is small and soft
and generally increased in frequency. Although without pain there is a
sense of uneasiness in the cardiac region: The bodily temperature, as
well as the warmth of the extremities, is lowered. The breathing is un-
affected—a point of distinction from organic disease. "I am out of heart
rather than out of breath," was the reply of one of Da Costa's patients.
FUNCTIONAL DISORDERS OF THE HEART

Insomnia is not infrequent. The patient rallies but slowly; two months in bed may be his portion, and months more of idleness before he recovers; for the issue is as tedious as the onset may be brusque. In some few cases the rhythm of the heart is irregular. The disorder may occur in either sex, and at any time of life between childhood and old age. There is no percussion dulness, the impulse is feeble. The first sound is short, lacking in volume; the second sound is not accentuated. Hysterical symptoms are conspicuously absent. In "irritable heart" the patient can get about, the heart's action is more obviously disordered, the impulse is jerky and diffuse, the second sound is sharp and distinct. Tobacco heart might resemble that under discussion, but in my experience the tobacco heart is more prominently irregular, and is often "irritable." In distinction from organic disease are the disproportionate prostration, the absence of dyspnea, and the freedom from any edema of the shins or feet. An apex murmur may appear in the functional disease as in almost any kind of cardiac functional diseases. The prognosis is good.

The treatment recommended by Da Costa is as follows: At first rest in bed; then, as some ground is gained, carefully regulated shower-baths are to be given. The next stage may be massage, but often some time elapses before this means can be borne. Then Swedish exercises and gentle riding on horseback can be arranged by degrees. Nutritious feeding is of course essential, and, in Da Costa's opinion, a generous allowance of alcoholic stimulants is necessary also. Among drugs strychnine "stands pre-eminent." The dose need not exceed 30 gr., but it must be given continuously. Arsenic is the next best drug; iron is not usually indicated, and the need of digitalis, if any, is but occasional. Nitroglycerine does no good. Bromides, valerian, or even opium may be required under special circumstances. If I may venture to guess at the pathology of these cases, it would seem that a dilatation of the vessels in the splanchnic area is the most probable explanation of them, and an artful compression of the abdomen might be found useful. The observed uselessness of the nitrites may support this view of the pathology. My patient, who could not knock at the door (p. 820), was perhaps one of this class, and I think I have seen many cases of the kind described. The state of the pupils might give us some useful indications in such cases; my impression is that they are either dilated, or at any rate contract slowly and imperfectly. Diminution of the mass of the blood, with correspondingly small output, may, as for example in the acute fevers, be a cause of such cardiac symptoms, however sound the organ itself. In cases such as these we have a truly "functional" disorder; the heart may be healthy, but its work is upset by circumstances.

Irritable heart.—Since the publication of Da Costa's and Myers' well-known papers this derangement has been too exclusively attributed to muscular over-exertion. It seems, however, that we must divide the subject of irritable heart into two classes: the irritable heart of young persons now to be described, a very curable disease, and the "Soldier's Heart," to be described under "Mechanical Strain," p. 851, which is too often
incurable. The irritable heart of young persons is a product of many conditions. The irritable heart of older persons—the irregular fretful heart which goes on too often to dilatation and static disease—is more definitely the result of over-exertion than that of young adults. In the irritable heart of young adults the upstroke in the sphygmogram is brisk and high; in that of dilating heart it is low and less brisk, and the rhythm is often irregular. The irritable heart of the former kind is much as follows:—A young man, for a man it is often than a woman, comes to tell us that he is bothered by his heart; he has a pain in it, always tiresome, often sharp; and the organ throbs and jumps; it never lies outside his consciousness. If he exert himself it beats violently; if he lie still in bed it also makes itself a nuisance, banging away when he ought to be asleep. When he is stripped he is generally a spare, long-chested fellow with wide intercostal spaces; and in the fifth space the apex is seen as it were kicking, rather than heaving, against the thin web of the interspace, although the blood-pressure is low and the dicrotic wave high. The heart may be a little out of place, displaced somewhat outward, but more downwards; still this is difficult to ascertain in lanky young men so built that the flat chest and the ill-developed lung leave more of the heart uncovered. A few years later such a man thickens, his lungs become more expanded, the heart relatively recedes. It may have been rather dilated before, possibly a little hypertrophied; but it was probably no more than too palpable and visible. Now in many of these men there is no doubt a story of considerable if not of excessive exertion. On the other hand there often is not; the youth, indeed, has been warned not to play football, not to row, and so forth—advice which has its good side, but which may be too rigidly enjoined. To the stethoscope the beat simulates hypertrophy: it is possible that in some cases there is a true hypertrophy (vide p. 916). In some cases of physical strain a little hypertrophy may exist, but even then dilatation is the main change. That in the intervals of rest the mean arterial blood-pressure is nearly always low is witness against persistent hypertrophy: when pressure falls, the heart cannot long remain above its strength. The peripheral arteries are lax, the pulse is dicrotic, and its slackness is in remarkable contrast with the excitement of the heart itself: the action seems laboured and perhaps heavy under the hand; the rhythm is often a little uneven, and the second sound at the apex too loud. The first sound is rarely muffled, however, as in unquestionable hypertrophy; it may even be shorter than normal, or at any rate smart enough. Sometimes there is a murmur, more often there is an "impurity" of the first sound, as if dimmed by some distant murmur overheard. These murmurs are often "pulmonary" in origin.

To account fully for this state of things in the circulation of such patients, is to know all the ins and outs of the habits of youth. This comes to us best by reflection on our own young days. Is it with laughter or with tears that one looks back on the reckless forenoon breakfasts washed down with those detestable compounds called "cups"; the
sherry and half a box of mixed biscuits at luncheon; the manly absorption of grown-up and, more than grown-up doses of tobacco; the black coffee and cognac of an evening after a large gobbled-up dinner; the hot arguments on the framework of the universe and the destiny of man protracted till two o’clock in the morning; the spasmodic bouts of study, the examination bogie; the conflict with untamed and rebellious passions, some wholesome, some not so wholesome; the violent games and the “bear-fights”; the ardent hopes and the bitter griefs—what elder is there who recalls all these things, and does not long to dash pell-mell into it all again and accept irritable heart into the bargain? There is but one step between the wise young man and the prig, and this a narrow one; still that is no priggish advice which would cut out of this gay, ardent and carefree life some of its idler and less lovely follies, and complete the cure of irritable heart by better-regulated exercises,—not violent stress one day, and idleness not unmixed with dissipation on the next, but regular training which shall promote a uniform development, not only of lungs and heart, but also of all the parts of body and mind. Muscular exertion, then, is a cause of irritable heart when it is pursued in an irrational and unsystematic manner by a more or less nervous and dyspeptic young person whose lungs are not big enough to carry off the blood as quickly from the right heart as it is delivered there; and whose ethical and intellectual life is lived after the same fitful fashion.

The irritable heart described in recruits, especially those suddenly removed from civil into military life—clerks turned into soldiers (Da Costa, Herz, etc.), is a different disease; and Mr. Simson Snell of Sheffield, in a private letter to me, says that colliers are very liable to an acute irritability of the heart, due probably to severe bodily efforts in awkward positions and in bad air. In these persons transient dilatation not infrequently becomes permanent (vide p. 851).

The treatment of the slighter and common forms of the malady is then one of regulated habits, and the avoidance of such poisons as alcohol, tobacco, tea, and coffee, except in doses which prove to be harmless to the individual. Muscular exertion must be systematic or indeed prohibited for a while or severely restricted. Of specific means, none is required; it is better to avoid digitalis and the like, unless the symptoms be unusually vexatious, when small doses with a little bromide may be used economically. These patients are often a little shy and sombre in spirit; change of scene, pleasant society of both sexes, and frank and kindly advice on sexual matters, are a part of the services which a sympathetic physician may render to young men; for while we may have a kindly smile for their heroics, we must remember, nevertheless, that they are often acutely miserable. Some excellent remarks on this subject by Sir William Broadbent are reported in the Lancet.

The neurotic element in organic disease of the heart.—We are too much disposed to think that death from organic disease of the heart is the direct result of its utter demolition; that the crippled organ stumbles along until it can do more, and staggering under an intolerable burden
sinks to its rest. We are too ready to assume that the diseased heart fails by means of its sheer mechanical inability. This may perhaps be the case here and there. Dr. Solomon Smith has on more than one occasion reminded us, however, that in many instances, at any rate, this is not the course of events. To put Dr. Smith's view of the matter summarily, he would have us see that the heart in advanced disease may fall, or stagger, under the intrusion of some neurotic accident, of some nervous perturbation, whether of reflex or inherent origin. The harmony between the reflex stimuli from the different segments of the heart may and frequently does become deranged; and it is not surprising that irregularity should result. Again, derangements of the stomach or bowels, torpor of the liver, pulmonary spasm, cerebral or bulbar interference, the absorption of toxic products, and so forth, are potent to depress or disturb the heart's action far beyond its mere mechanical disadvantage. Thus it is that in most cases great oscillations occur: at one time the patient is pretty well, at another he is at death's door; yet again he comes round, and this not necessarily as a result of treatment, or if of treatment, of such a remedy as an injection of morphine, which may readjust or permit the readjustment of the harmony of internal cardiac stimuli; or may block some reflex arc with its superadded neurosis. Again, the vomit of a little sour mucus or the discharge of an offensive stool may set matters right, even in a few minutes. It was with this conception in my mind that in 1869 I recommended the subcutaneous injection of morphia in heart disease; not only does it, in appropriate cases, cut short a neurosy of dyspnea or restlessness, or restore the order of rhythm, and thus pacify the organ rocking under the tumult of its unbalanced parts, but it may prevent the heart from being "tripped up by the intrusion of a neurosy," as Dr. Smith puts it. The complex rhythm of the several parts of the heart and its allied vessels is but too easily broken in upon at one or more points. The importance of these considerations in respect of treatment is obvious. "Our choice of remedies lies no longer only among cardiac stimulants or depressants, arteriole constrictors or dilators; a whole range of remedies is opened to us which, although without direct action on the heart, relieve heart trouble all the same by removing the starting points of nerve derangements." I may add that not only are new remedial means thus opened out, but in these words we have the explanation of the value of many remedies which, in a more or less empirical fashion, have long been familiar to us.

TACHYCARDIA.—The names tachycardia and bradycardia are often used merely to signify rapid heart and slow heart respectively; such uses have not even the accuracy of pedantry. Dr. Herringham, indeed, thinks that tachycardia is a "symptom rather than a disease," but in thus writing he scarcely does his own monograph justice. If any rapid pulse, ranging, let us say, over 130, is to be decorated with this fine name there is an end to clinical nomenclature. Dr. Watson Williams implies that tachycardia is a disease, but he prefixes the qualifying epithet "paroxysmal,"
which from his point of view is superfluous. Dr. Herringham, in refusing to go beyond the bare etymology—the "prairie value"—of the name, argues, truly enough, that "no real distinction can be drawn between the cases with and those without ... organic lesions." But is not this to deny also the validity of the names asthma, epilepsy, chorea? The author is right in warning us against the danger of "erecting a separate type" in such a case as this, a truth which I have endeavoured to emphasise in the introduction to this work; but, as I have there said, I do not think that the existence of mixed or transition cases forbids us the precise use of types. For what do we mean by "a disease"? Surely no more than the recurrence of symptoms in fairly uniform groups. A disease has no more "real" existence than has a constellation; stars, like symptoms, have a way of grouping themselves about centres of relative density; to such groups we give names, and no one should pretend that any disease has more than this relative or, if the reader please, this "subjective" existence. A type is an abstraction, an ideal pattern constructed from an infinite number of cases; and the moment we contemplate a particular case we leave type for embodiment: no two cases are identical, and no case corresponds in all respects with the type. Like Dr. Herringham, I am not fond of teaching by types, but they have their use in summarising and classifying our observations; and if we remember that they have no more claim to "reality" than this, we may use these conceptions without much harm. If, whenever we talk of "tachycardia," for example, the mind is to range over an indefinite scattering of cases in which the pulse is excessively quick, we shall waste a great deal of time in discussion and a great deal of space in books.

To what symptom group, then, do we apply the name tachycardia? Not to any case of quick heart, but to an enormous quickening of the pulses of a heart not necessarily the seat of static disease; a quickening which attacks the patient suddenly; which does not persist indefinitely, but for a variable space, rounded off by an equally sudden reversion to the normal state less certain phenomena of exhaustion. Heart disease, in the static sense, may coincide with tachycardia, it is true; mitral stenosis may coexist with chorea, may, may even favour the occurrence or intensify the peculiar symptoms of chorea; but that surely ought not to deprive us of the name chorea, nor justify us in including under this name, as too often we do, any twitchings or gestures whatever which look at all like chorea.

Careful clinical observation and no less careful verification after death (so far as this has gone) indicate, at present, that attacks of rapid heart coming on suddenly, departing suddenly, and attended with certain other symptoms, objective and subjective, are consistent if not always coincident with a heart apparently sound; that coarse heart lesion is therefore not a necessary antecedent, or, in other words, is not a cause of this malady. Hypertrophy is not usual in rapid pulse, of any origin, as such; for increase of rate generally means diminution of output per beat, and probably per second also. If output per beat and per second is increased the rise in rate can be but moderate (Stewart). The records of necropsy in tachy-
cardia are few, no doubt, but we can only go upon such evidence as we have; and the sudden subsidence of these attacks without leaving behind them any evidence of disease of the heart supports the interpretation of the scanty pathological material. The interpretation is that tachycardia is a fairly uniform symptom group; and, as one of its eminent characters is its paroxysmal occurrence, the addition of this qualification to the name is superfluous.

The attack is as follows:—As I describe it I have in my eye two cases now under my care. The first is in a woman, passing (at the time I now write) through the menopause without any peculiar derangement, who since her adolescence has been liable to seizures of tachycardia. She is a well-nourished person, and is now getting stout. Her anxious and fidgety temperament may indicate the neurotic bent, or may be the consequence of her distressing malady; but the family history is without apparent bearing on the case. Her own life, though broken into more than once by calamity, has, on the whole, been one of prosperity; moreover, her ailments dates from adolescence, years before these heavier trials had afflicted her. She is happily married, but has had no children. The attacks, which have preserved the same characters from her adolescence, are as follows:—She turns a little shivery and pale, at times even ashy; and a peculiar lassitude and restlessness possess her; the extremities are cold, and these and other parts are “numb.” She soon becomes aware of a tightness, tremor, and oppression rather than of a beating about the heart; the tightness may amount to actual pain, and may dart here or there. The pulse is now beating at the rate of 160 to 200 a minute (the reckonings of the pulse have not been systematic; and often the only record is that the pulse could only be counted at the heart). After the attack has continued for a dry or two I find that the area of cardiac dulness has extended towards mid-sternum, or even beyond it; the sounds are tick-tack, but no added sounds are to be detected. As the attack goes on she becomes very fretful and wretched, but the oppression and tightness and other signs suggestive of peripheral arterial contraction pass off. The urine in most cases is scanty; at first, perhaps, from contraction of the renal arterioles, later from low arterial pressure; but in her and in another of my cases nervous polyuria attends the attacks throughout. It seems certain, from the change in the volume of the heart, that the residual blood in the ventricle is large and the output correspondingly small. The relief of oppression does not signify that the tension of the ventricular walls and aorta is diminished, but that the sensibility of the heart is blunted. In severe attacks she is more or less aphasic, with the aphasia of exhaustion—a phenomenon not uncommon in megrim and in persons spent by fatigue. Such an interval of aphasia is described by Tyndall in his own person after a dangerous and exhausting scramble among the rocks above the Grimsel. The most complex of muscular co-ordinations give out early, as we might expect; but, as in many cases of nervous prostration, she has often a special sense of weakness or palsy in the left arm.

The duration of the attacks is very variable. In some patients an attack
may cease after a few hours or a few minutes; or again it may continue for three, four, or five days. It is said to have lasted in some cases as long as ten or eleven days; or indeed for weeks at a time, but suspicions of a wrong diagnosis present themselves on the consideration of such records. Perhaps the longest attacks of those carefully recorded were in a case recorded by Bouveret, in which they endured for thirteen days. In my second patient the attacks would return in groups, giving thus an impression of a longer paroxysm than was strictly the case. He might have a series of four or five attacks, and then none for a year or more. During the one or more nights of an attack the patient may be almost sleepless; but during sleep the tachycardia pursues the same course. Sometimes during these nights the female patient described above is a little delirious. The beating of the heart is regular in all cases unless the ventricle be dilated, when every pulse may not reach the wrist. In a case recorded by Dr. Bristowe the pulse number reached 308 a minute.

The cessation of the attack is always brusque, generally sudden; it may end in a few slow hard beats, or in one violent rebound, followed, says my second patient, by "a sort of swim." The trial is now over; exhausted as the sufferer may be, there is freedom—till next time. The urine in these two cases has never contained sugar, albumin, or any substantial excess of urates or phosphates. Attacks cannot be traced as a rule to any cause or to any season; they may come on at a moment of rest; often they begin or end during sleep. My second patient is an epileptic tailor, with a good family history. In him over-exertion often brings on an attack; but an attack thus produced can always be arrested by holding his breath in inspiration and then stooping tightly down with his belly on his thighs. Spontaneous attacks cannot be thus cut short, though once (the third of four attempts) he stopped one in my presence for a few seconds; the pulse fell suddenly from 166 to 80. As he rose up the rate as quickly returned.

Such is the ordinary course of a well-marked case of tachycardia, though cases of greater and of less severity occur. In one of my present patients there is no abnormality of the heart save the usual short, sharp action of neurosis; in the other there is a permanent apex systolic murmur, but no dilatation; in worse cases irremediable dilatation of the heart is brought on. During the severer attacks oedema of the lungs may accentuate the signs of dilatation, and later the feet may become oedematous, and albumin may appear in the urine. A repetition of such incidents renders the heart less and less able to recover its normal tone, and the symptoms of dilapidation set in which need no description in this place. Death may be by asystole, or by syncope; probably nearly always by syncope.

I may add to the story of my lady patient, that her first attack cut short a prolonged and severe skipping effort, when she had reached a high tale of skips. Ever since she has been subject to attacks, but they are not so severe as formerly. They seem to come on capriciously, she thinks more in spring and autumn than at other seasons. Sometimes they have been determined by a shock, physical or emotional, as once
when she made a false step in the street and “jarred” her foot; and once again when a drunken man seemed disposed to accost her. Dyspepsia may seem to call forth an attack, but, in both these cases, by far the majority “come on of themselves.” Her pulse generally runs about 290; the highest that has been noted accurately was 280. The attacks go off somewhat variably: either “hardly,” that is, more gradually with a peculiar sense of agony, when she used to think she must die; or suddenly with a thump or two. The attacks may last for a few seconds, a few minutes, or a few days; some attacks have lasted as much as ten days, but this duration has been unusual. She feels conscious enough of the beating; it is like a rapid tapping or vibration: when she was younger it would shake the bed and even the room. In latter years she can bear them better, no doubt they are milder; she can even read during the attack. Formerly she was prostrate throughout the course of them, and long after them; indeed, they are all most exhausting. Her family history is very good; her parents are hale octogenarians. No notable nervous disease has been heard of in the family. I described her as a nervous and fidgety person, but she assures me that the tachycardiac attacks have been the cause of this disposition by the injury they have wrought upon her nervous system. This is probably true, as she is of sturdy build and well nourished. She has no permanent signs of cardiac failure. In the epileptic case the fits came on at set. 42, the heart attack at 14. The two maladies move quite independently of each other.

Morbid anatomy.—I have said that the evidence of necrosies is as yet scanty, though the two or three careful examinations on record agree in indicating that, these evidences of cardiac decadence apart, no constant changes are found. Examination of the vagi, of the sympathetic nerves, and of the intra-cardiac ganglia have been negative, except for secondary changes such as the degeneration of muscle and ganglia in common. As then the evidence of the stethoscope is also negative, and as for many years the patients recover their ordinary health between the attacks, we must regard tachycardia for the present as a functional disease. If the ultimate prognosis be doubtful, if in a certain number of cases the event be death, the immediate prognosis, in the earlier years at any rate, is hopeful. Indeed, Dr. Watson Williams records a well-marked case in a patient aged—at the time of his writing—eighty years. The patient I have referred to is about forty-eight. In cases which, after the lapse of years, have proved fatal the necropsy may reveal, as in a case of Fraentzel’s, fibroid degeneration of the walls of the heart and dilatation of its cavities in all directions. Broadly speaking, then, in tachycardia no primary or constant morbid lesion has been discovered. Ultimately the disease often wears out the heart; but where or how it is engendered we know not.

Pathogenesis.—With the best will in the world I cannot follow the example of recent writers in discussing the “theory” or “theories” of tachycardia. No theory exists. Certain surmises, such as no competent physiologist would fail to suggest at first sight, are offered to us; but it would be an abuse of language to call them even hypotheses. I will take
them in order: (i.) That the vagi are spent, or thrown out of gear. The suddenness of the attacks, both in onset and issue, seems against the opinion that these nerves are spent; thrown out of gear they may be. We know of many cases in which the vagi are thrown out of gear; as for example in bulbar disease, or under the pressure of growths or glands (cf. Probsting's well-known case), or in experiments upon animals; but in such cases the rapidity of the heart has not been by any means so great. It does not seem probable that abeyance of the vagi in man gives the heart play beyond 120 beats in the minute, or thereabouts. (ii.) That the vagi may be in abeyance, and at the same time the accelerators may be excited or vaso-motor centre affected. This suggestion sins against the economy of causes, and, as we must assume a close synchronism of disorder in each, we should be thrown back upon some cause behind them both. Some temporary change in the bulb might at once throw out of gear both vagi and vaso-motor governance; nevertheless such may be the case. (iii.) That the accelerators may be so stimulated as to overbear the normal vagi. It must be admitted that the onset and issue of the attacks seem in favour of some such supposition. In no experiment, however, has such a rate been obtained by accelerator stimulation; and reflex irritations of eccentric origin do not push the heart beyond 150 as an extreme limit. (iv.) That the cardiac ganglia are the seat of the disorder. But we find no changes in them which are inconsistent with a secondary origin; moreover, the latest researches on these ganglia by Dr. Gaskell and others seem to prove them to be remnants of the innervation of the arteries, and thus to have but a secondary importance in the cardiac functions. (v.) Some sudden change in arterial blood-pressures; this will not serve us, as the arterial blood-pressures are by no means constant, they are always rising and falling; any constant change of pressure would soon be compensated in the normal way: furthermore, while no ordinary tides of blood-pressure, as Mosso's experiments (Jumot’s boot, etc.) show, are competent to bring about so extreme a change of rate, there is no evidence of extensive areas of anæmia, as on dilatation of splanchnic veins, which might be adequate to call forth such phenomena. Were the heart abandoned to its own inherent rhythm its action would be irregular, which in tachycardia it is not, unless considerable dilatation has taken place. I confess that I leave my suggestion of extensive areas of anæmia somewhat wistfully, as a sudden expansion in the areas of the abdominal venous system would produce such results, as we see by the results of experiment and in exhausting diseases; still on this supposition the heart should not be distended, unless simultaneously the peripheral arteries are constricted. I think, however, the pulse is small in tachycardia, because by virtue of their tone the arteries fit themselves to diminished contents. We cannot, then, do more than guess whether the immediate causes of tachycardia lie in the cerebral cortex, in the mesencephalon, in the bulb, in the vagi, in the accelerators, in the cardiac ganglia or muscle, in anæmic areas, or in eccentric irritation, such as floating kidney (Balfour). Neuritis has been alleged as a cause; but
there is no evidence of its presence, nor would it consist with the long intervals of health. As the phenomena are remarkably uniform, the causation is probably not complex.

Of the immediate causes there is little to say. Over-exertion, dyspepsia, mental shock or emotion, uterine disorders, auto-intoxication, loaded bowels, any or all of these have been alleged with more or less hesitation. All that we can say, then, with any approach to certainty is that the victims of this disease are of the neurotic habit, and that in a few cases it has seemed to be hereditary (Williams). It is alleged that Graves’ disease, in which a rapid heart is the chief feature, may be the instant result of an acute mental shock.

Sex.—The records of cases indicate that this factor has little or no influence in the causation of tachycardia, the disease falls almost impartially on the two sexes.

Age.—In forty cases of Dr. Herringham’s collection the age was recorded. In seven the malady dated from childhood; of these seven, five were women. In twelve the first attack appeared between the ages of twenty and thirty; of these, six were men and six were women. In thirteen cases the onset fell between the ages of forty and fifty; in three the patients were over fifty when it occurred. Dr. Watson-Williams reports a case in a man of eighty-one, in these attacks the pulse would leap suddenly from 60 to 130. H. C. Wood reports a case as still recurrent in a physician of eighty-seven years of age; the attacks began in his thirty-seventh year; the onset is abrupt, and the pulse rises quickly to 200.

Diagnosis.—Tachycardia is an intermittent disease; if we remember this we shall avoid confusion with other diseases in which a rapid action of the heart is a more persistent feature. The larval form of Graves’ disease—by no means uncommon in which the thyroid is not enlarged nor the eyes prominent—may be distinguished from tachycardia, in cases which have been watched for a sufficient time, by the long persistence of the rapidity. Moreover, in Graves’ disease the action of the heart is more thumping. Fine tremor may be seen in many cardio-neurotic cases, as may exalted reflexes also. Tachycardia is not a mere incident of neurasthenia. The pressure of a tumour on the vagi may be attended with a persistent rapidity of pulse. In cases of idiosyncrasy, cases in which the pulse runs in the individual at accelerated rates, the persistence of the peculiarity will again decide the judgment against tachycardia; and it may be added that in these cases, and in others of more or less persistently quick pulse, the patient suffers less instant distress. Cases are recorded on good authority in which the pulse of a person presumably healthy habitually ran at 150 a minute. Binawanger has recorded such a case in a woman; in her the peculiarity had endured all her life. I remember one day, when I was driving with a medical friend, a man passed us on horseback—a fine-looking country squire in whom there seemed no flaw; my friend told me to note him as he passed, because his pulse ran habitually at 120. The owner of the pulse, patient I cannot call him, enjoyed fair health, but in the doctor’s opinion would
be a "bad subject" for acute disease; this opinion he founded not only on a mistrust of the pulse, but also on a certain lack in him of resistance to fatigue and trivial ailments. Dr. G. Balfour, again (Senile Heart), refers to the case of a lady, then over seventy, who had had a large family and enjoyed good health, though of nervous temperament; her pulse had never been under 150. Of heart diseases the two to be excluded are dilatation and mitral stenosis. The tobacco pulse, if rapid (at first it is slow), is irregular. Alcohol, if it accelerate the heart's action, does so by inducing degeneration of the organ, and this lesion may be betrayed by its own phenomena. The accelerated pulse of cardiac dilatation is irregular. Old men who give themselves up to sexual indulgence have a pulse of increased rapidity, but tachycardia is not very likely to attack a man for the first time in old age. Fevers, diarrhoea, and other toxic or exhausting causes may be attended by a quick pulse, but such causes are not likely to be overlooked. In bulbar palsy the pulse is persistently changed; and if accelerated, is irregular and intermittent; in tachycardia the rhythm is even; moreover, bulbar disease has its own characters, such as faintness attended with a fear of death, a kind of "angina sine dolore." Finally, in none of these is the disease paroxysmal.

Prognosis.—Dr. Herringham thinks that after thirty years of age no patient of tachycardia is safe, and that few pass fifty. This, I think, is rather a darker forecast than I should be disposed to make. Much depends, as Herringham says, on the duration of the particular attacks and on the frequency of their return; if these last longer than five days the stress on the dilating heart leads to strain, especially in the elder patients. Two patients of mine are well past their climacteric, and to Dr. Watson Williams' patient of eighty-one I have already referred. In him, as in most patients as they advance in years, the return of the attacks is generally postponed; the intervals are longer, and there is more time for recovery.

Treatment.—Unfortunately this paragraph is but a short one—not because we have a prompt remedy, but because little or nothing seems to be of much service either in cutting short the attacks or in the prevention of them. As I have said, the attacks may get less both in number and severity with advancing years; and, perhaps, something can be done on general principles to make the system less susceptible to the causes of them, whatever these may be. That they lie in the nervous sphere the result of tonic treatment seems to indicate. During the attack tincture of digitalis in a little brandy is sometimes serviceable. The brandy I find is necessary, as in tachycardia the foxglove is especially apt to set up nausea. However, brandy or no brandy, it is often of little use, and patients soon give it up. If digitalis does not modify the rate of the heart it often causes diuresis; now in a heart quickened by the failure of intrinsic disease the drug often fails to produce diuresis, a result of bad prognostic meaning. One of my patients still clings with faith to a prescription of salicylate of soda and sodium bromide which I gave her ten years ago; she assures me that it is of much service to her in
mitigating and shortening the seizures. I gave it on a strong hint of goutiness in her family. This patient has had a fibroid tumour for many years, but the attacks are certainly of still older date; there is no evidence that the fibroid has affected her tachycardia in any way for good or evil. I recommend compression of the abdomen with a binder, but I think this method has not been well applied; a trained midwife should be engaged to instruct the patient in the proper use of the bandage. Wood's patient was relieved by drinking iced water and strong coffee, as if to arouse reflex inhibition by the vagi. The application of electric currents of this kind or that, to the vagi in the neck, however promising at first sight, has disappointed those who have well tried it. Finally, it is said that a compression of the chest by the patient himself sometimes succeeds in stopping an attack. I have not had a good opportunity of putting this method to trial. It is to be essayed as follows:—The patient will thrust his feet as hard as he can against the foot of the bed; then, pressing his arms closely into his sides, he will take a long inspiration; in the next place, closing the glottis, he will make a strong expiratory effort, thrusting hard the while against the walls of the chest with the upper arms, and clasping them with the forearms. In this way it is said that the rate of the heart may be directly controlled. After this fashion an old friend of mine used to cause his heart to intermit. During the intervals of quiescence persevering efforts must be made to nourish and invigorate the system. The digestion and the excretory organs are to be vigilantly watched and corrected, and all means are to be adopted to secure serenity of life and a wholesome and regular occupation. One of my tachycardiacs began to ride a bicycle two years ago, and with much advantage. Oertel's "heart massage" seems to me to be no more than ordinary massage plus suggestion; but massage is very useful in emaciated or podgy people, and, in the more vigorous, Swedish gymnastics may be cautiously used with advantage. It will be remembered that any over-exertion or stress may bring on an attack; the treatment must therefore be trimmed between the extremes of indolence and fatigue or sudden effort. A patient who rides the bicycle tells me that in this respect the bicycle is better than horse exercise; a horse may, and often does make a sudden demand on the rider's nerve. The use of the graduated douche or of the wet sheet proves very useful in some cases; but for further particulars of this kind the reader is referred to other chapters.

Bradyocardia.—The reasons which justify us in retaining the name "tachycardia" make for the banishment of "bradyocardia." Bradyocardia is a superfine name to denote slow pulse; it connotes nothing. In literature a little pedantry may be harmless, nay, as a protest against slovenliness may have occasionally its welcome side; in science it is a pest. The name "bradyocardia" is as pestilent as the rest because it hoodwinks the student, who does not rid himself of the false prepossession that in so large a word must lie a specific meaning; he does not realise its emptiness. By neurasthenia we do not mean mere nervous
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debility, but a particular and definite group of symptoms of which nervous debility is but one feature. With the word "tachycardia" we introduce a new conception—that which I have endeavoured to set forth; with bradycardia we introduce nothing; the word is but wind. We know of no symptom group to be thus designated; bradycardia is slow pulse and nothing more.

Relatively to the heart slow pulse is a "functional" disorder when it is found independently of intrinsic and static lesion of the heart; thus slow heart in meningitis, terrible as is the disease itself, is yet a functional disease in respect of the heart. Slow pulse in fatty degeneration of the heart is not, in clinical language, a functional but an organic change. But, slow pulse connected with organic disease, whether in the heart itself or elsewhere, will not be discussed here.

The proposition that in all cases slowing of the pulse is due to the control of the vagus is not perhaps invariably true, though it is of very general application. I have said already that in a few cases slowing seems to be attributable, at any rate in part, to the muscle itself; but even then it is hard to say how far pulse retardation may be due to the vigilant nursing of the vagi. But in the case of certain poisons the muscular contractions seem to be slowed down directly, though even in them; as in fatty heart, it is difficult to share between the vagus and the muscle the function of each; whatever be the inherent failure of the muscle the vagus may and generally does intervene to spare it. In such states as senile broncho-pneumonia, where the tendency is to dilatation, the action of the vagus, whatever its immediate protection may be, turns to evil ere long even in the heart itself; as vagus action not only reduces the rate of the heart, which in itself might not lessen its work, but reduces the work also; and the organ cannot overtake its a grء. Therein lies dilatation, excessive internal stress and imminent strain. However, to leave these questions we have to turn rather to the slow pulse which depends not upon organic disease of the heart, nor indeed upon organic disease of eccentric position acting by reflection on the heart through the vagus, but to those functions, all perhaps following some reflex paths, which slow down a relatively healthy heart. Of these the following classes may be made:—(i.) Rise of blood-pressure, as seen, for instance, in its simplest form in the "expiratory diminution of rate"; or, conversely, in the temporary suspension of vagus action by continual sipping of a fluid: thus the heart's frequency may be raised twenty or thirty beats a minute (Waller). (ii.) Blood containing intrinsic poisons, such as carbonic acid or that of uræmia; or extrinsic poisons such as lead, tobacco, digitalis; or bacterial products, as in diphtheria, most of which act directly on the vagus or its centre, but some of which seem to affect the heart itself. (iii.) Reflexes from the irritation of eccentric derangements, such as those arising in the gastro-intestinal canal (dyspepsia, etc.), in the pelvic organs, in the throat or ear, and so forth. (iv.) The slow pulse of children. (v.) The slow pulse of hysteria, melancholia, and other psychical disorders. (vi.) The slow pulse of exhaustion, as after fevers.
or great fatigue (probably not reflex). (vii.) The slow pulse of pain. The slow pulse of cerebral, bulbar, and cervico-spinal disease. (The slow pulse of heart disease, disease rather of its walls than of its valves, we have deliberately excluded from the section of functional disorders.) Epileptiform attacks (Stokes-Adams disease) seem, like syncope, rather to be an occasional consequence of slow pulse than a cause of it; in uremia the two events may spring from a common cause. Vertigo and syncope are more frequent consequences of the kind; they are in my experience grave symptoms when associated with slow pulse, and suggestive if not conclusive indications of cardiac degeneration.

Again, in some persons an infrequent pulse may pertain to their normal state. I have never had my finger on the pulse of an epileptic at the earliest moment of an attack, but scores of times, as for instance in the wards of lunatic asylums, I have felt the pulse at the instant of the seizure becoming manifest; I have never, however, found any characteristic change in the rate. I find that Sir R. Gowers makes the same remark. In the cases in which the association of slow pulse with epileptiform convulsion has been noticed it seems probable that the pulse retardation comes first, and that the intermediate factor is cerebral anemia; that, indeed, the phenomena are those of convulsion on extreme phlebotomy, the stage beyond delirium. Of "normal slow pulse" we see many examples; the most remarkable I have recently seen was in a vigorous, cheerful man who was in the Radcliffe Infirmary during the Michaelmas examination for the M.B. degree in 1897. In this man a pulse of 28 could be raised on excitement to 32 or 33. Being a weather-beaten person well over 60 years of age his arteries were not, of course, free from signs of degeneration; but it was difficult to say that they were older than his years. Of the rate of his pulse in former years he knew nothing; he was unaware of it until we told him. I suspect that it had gradually come on as he grew older. He felt quite well, and was vastly amused by our determination to find some grave mischief within him. He was admitted for some trivial ailment, in order that he might be hunted well over by the candidates, who, however, found nothing more to report; and Dr. S. West, Dr. Mallam, and myself, found him free from any other malady than that of "old. A pulse of 50 is no very uncommon rate in healthy persons, rather in men, perhaps, than in women; in a friend of my own a pulse of 58, sometimes slowing down on fatigue to 54 or 55, has proved consistent with great nervous and muscular activity up to years which are now more than mature. For him a pulse of 80 is fever; it never rises over 100 or thereabouts, except of course under severe muscular exertion. Corvisart's record of Napoleon's pulse as habitually 40 is well known; Sir William Broadbent has, I believe, recorded somewhere the case of an athlete with a pulse of 36. Osler, who within the limits of his Practice of Medicine rarely misses a point, tells us that physiological slow pulse is seen in parturition, whether premature or at term. The rate may decline from 60 to 44, and has sometimes fallen as low as 34. It is needless to say, that in all cases of alleged slow radial pulse the number of the cardiac
revolutions must be counted at the centre; as some of the waves may fail to reach the periphery. Some records of egregiously slow pulse can scarcely be credited with the existence of cardiac pulses. Roy used to say that a healthy heart might drop six beats and recover; but can a deteriorated organ cross such an abyss of time? We read of pulses of 20—nay, of 12 a minute; of stops of 15 seconds' duration—in one instance of an arrest of 30 seconds. An absolute stop of 15 or 20 seconds must surely mean fatal syncope, or epileptiform convulsion. Very feeble heart-beats may be insaudible even to the stethoscope. Fibrillary contraction is sometimes recovered from in animals, probably not in man.

All I know definitely about "hysterical slow pulse" I have found in von Noorden and Buchholz. If I have seen it I have made no note of it. For the variations of the pulse in mental diseases the reader is referred to the following chapters on these subjects. In respect of poisons we know that some of them, such as lead, may act indirectly by perverting the metabolism of the body, and thus generating intermediate poisons; uræmia and jaundice are often associated with a slow pulse. Most if not all these catabolic substances act, no doubt, directly on the vagi, centrally or peripherally. The poisons generated by bacteria—the infections—not infrequently begin by stimulating the vagi, so that the pulse is slowed; then the vagus is exhausted, the pulse quickens, and in the later stages is much accelerated—the mass of the blood being often much reduced in these diseases. In convalescence the cardiac centre seems unstable, and the pulse may be slowed or quickened by influences which in the normal state would prove indifferent. That muscarine slows the pulse is a familiar laboratory demonstration; and the accelerating effect of its antidote atropine is more familiar still. Tobacco, again, stimulates the vagi at first, and then paralyses them, or leaves them exhausted so that, in extreme cases, the heart is rapid and so irregular as to seem to be abandoned to its own rhythm. Rise of blood-pressure may retard the pulse remarkably; the fact is familiar to all clinical observers; but the rule that the rate of the pulse is inversely as the blood-pressure is open to many contingencies; it only holds when other things are equal: I think it better to put it that pressure is that part of the energy of the blood which is not turned into speed. The sum of the energy may be reduced. In the slow pulse of exhaustion the blood-pressure is often low; if vagus control be its cause the low pressure is due to the effect of this nerve in slackening as well as of slowing the heart; the residual blood in the left ventricle is more. I have seen this retardation fall to 45 in many cases of persons whose pulse in the normal state is of ordinary frequency. Some fifteen years ago, when very arduously engaged in practice, I was returning by night from a consultation in the west of England, when, on leaning my head on my hand I felt my temporal arteries beating too slowly; the rate, then about 48, fell gradually to 44. I got a glass of hot brandy and water at Bristol Station, and in a quarter of an hour my condition was better. It was a month before it recovered to some 50 beats, again to 45. On the basis of this observation I have
supposed that the slowing of exhaustion is a protective effort of the vagi, which, in my case, were further stimulated in their gastric area by the brandy. After the brandy I fell asleep, and on awaking my malady was gone. It was attended with a sensation of sinking or depression; and at times I have since recognised some abnormality of the kind by the same warning. During the last eight years, of a less harassing life, the derangement has altogether disappeared. Now here we had a bold breach of the rule that rate is inversely as pressure, for in my case the pressure was, as I have said, low; and it rose as the normal rate was regained. The heart’s output was probably increased.

Sexual exhaustion is efficient to reduce pulse-rate. But the other day a patient was sent to me by a distant medical friend who had found in him a slow pulse, about 40, attended with a sense of depression, almost melancholic, especially of a morning. It was a great effort for him to get up to breakfast; although after he had got to work or play the sensation wore off. At the times of slow pulse the temperature also would fall to 95°. He was in business, but in an easy one; he had no cares, his habits appeared to be correct, and he had had no troubles. He was fond of physical exertion, and could and did ride, shoot, and so forth even to the full, without being the worse. His age was forty. On examination of his heart nothing abnormal was to be found. His own medical man had cut down his tobacco (usually 2½ ounces a week) with advantage, but without much relief. I ascertained that he gave himself up to excessive marital intercourse, even to daily indulgence. My prescription was a separate bedroom, which will probably work a cure.

In some cases of temporary slow pulse with “nervous exhaustion” the voice becomes hollow or even feeble. In one case I remember the patient, partly in timidity perhaps, intimated that he was too much exhausted to do more than whisper a brief reply. It is possible that some of the cases of slow pulse in children are due to self-abuse; but by no means all. To find a pulse of 50 or 45 in a little boy or girl used to frighten me no little; I regarded them as the barbarians regarded St. Paul. But as, often enough, nothing happened I gained heart; and am now, if still on my guard, not prophetic of evil. In some cases worms may be the cause of the retardation; but antidotes for worms do not always prove the connection. Nevertheless, as some arrhythmia may be present, and perhaps some heaviness or drooping of manner may be exaggerated by anxious parents, these cases are not a little embarrassing for a few days. Gastric catarrh, again, is among the causes; and, probably in the child the heart centre, like the temperature centre, is more susceptible than in later years. The ages of such patients run from four or five to fourteen or fifteen. The child may be languid and out of spirits, or dyspeptic, when the state of the pulse is found out, as it were, accidentally. Irritation of the vagi is again the probable explanation; indeed, this seems to be the first factor to be thought of in all cases of slow or intermittent pulse, yet it may not be the invariable cause. Slow pulse children are usually of neurotic constitution.
The slow pulse of convalescents from fevers and other exhausting
diseases, is a common event, and is sometimes suggestive of cerebral
complications, especially in children; it is probably due to vagus irrita-
tion, set up, it may be, by carbonic acid or by some toxin. Or the
cardiac muscle may be poisoned. Thus I have seen it in severe bronchitis
with distended right ventricle, much residual blood, and greatly over-
charged veins. Intermittence is seen in these cases also, which may point
to vagus protection.

The slow pulse of pain is a phenomenon full of interest: it must
be due to reflex stimulation of the vagus; thus it can readily be
produced by experiment; and it is not unfamiliar, under the like condi-
tions, to the practising physician. Sir Richard Powell mentioned an
interesting case of this kind at the meeting of the British Medical As-
sociation in 1894. The patient was subject to neuralgia and to palpita-
tion, but not together. An attack of pain would stop the cardiac
disturbances. Sciatica is perhaps the pain most efficient in producing
this result; but almost any sudden paroxysm of pain of sufficient
severity may be reflected in the pulse. Its chief interest lies in its
bearing on the causation of angina pectoris, whether of the graver or of
the “functional” kind. Whether slow pulse may ever be due to a failure
of the accelerants we cannot tell; in the cases of “exhaustion” above
described such may be the case entirely or in part. Of the intimate
relations of the intra-cardiac ganglia to the functions of the heart we
know little, or indeed nothing; Dr. Gaskell regards them rather as
survivals of the nervi vasorum than as dominant factors in mammalian
cardiac evolution.

As bradycardia denotes a symptom and not a disease, or as, in
other words, it signifies no more than a phenomenon common to many
definite groups of symptoms, and as there is not, as with tachycardia,
any peculiar group of which it is itself the main or central feature, it can
have no diagnosis or prognosis. All that can be said is that it may
depend upon irritation of the vagus only, the heart being sound. In such
cases it will often, of course, be associated with arrhythmia and inter-
mittence. Such conditions are usually curable by removal of the causes,
and especially by careful mastication of the food. Momentary efforts
often aggravate the condition, but in a sound heart persistent exercise
removes it for the time. It is usually worse after meals, and is attended
with flatulence. The urine must, of course, be minutely and repeatedly
examined in all its qualities, and signs of cardio-arterial degeneration
duly appraised; remembering however that, if due to degeneration of
the coronary arteries, the most usual organic substratum of slow pulse,
signs of disease may be absent or very indefinite. But we cannot pursue
these parts of the subject; from what has been incidentally said the
reader will know where to turn for descriptions of the symptom groups
to which slow pulse is subordinate. Under these several heads will also
fall the means of treatment, if slow pulse can be said, any more than
cough or dyspnœa, to have any treatment of its own. Static disease of
the heart apart, slow pulse needs not even palliative treatment; it has no dangers of its own.

It is impossible to give any list of references in respect of a mere symptom such as slowness of the pulse. The reader will find two recent articles on the subject in the Lancet of 30th January 1897 by Dr. John Ogle, and one by Professor Osler in that of 27th February 1897. In these articles, however, the symptom is chiefly regarded as significant of intrinsic cardiac degeneration.

SYNCOPE.—Whether the heart stops altogether in syncope is yet unknown; it probably beats with a beat so feeble as to escape our senses. It may be arrested, but it seems impossible that the heart should be arrested during all the span of a long faint; I have said elsewhere that Roy, on the basis of large experimental observation, thought that the heart certainly may drop six beats, possibly more; but that beyond some such number as this there is great danger of death. Yet when we are discussing the ordinary fainting fit these calculations of more or less around the margin of the grave seems fanciful: "No one dies of a faint," one may say; or another may say with equal truth that sooner or later almost every one does. Yet the syncope which cuts the vital thread at the end of most fatal illnesses is evidently something so different in degree and contingency from the ordinary faint of the ladies who are carried out into the vestry, that here we must fix our attention exclusively upon the functional disorder. The church faint is not primarily a cardiac failure, but an expansion of cutaneous and splanchnic vessels with fall of arterial pressure.

Yet of this curious disorder no full explanation is forthcoming, surmise as we may. It is a very common malady; perhaps no woman passes through life without experience at least of its premonitory symptoms. To faint is not the exclusive privilege of woman; every physician has seen men fall like oxen—for instance, in the gallery of an operating theatre. A very sturdy and stout-hearted man once fell suddenly to the floor in my consulting-room, where a moment before he was complaining to me of some temporary disorder; partly dyspepsia, partly fag. I have known him for some quarter of a century since that day, and, so far as I am aware, he has never fainted since. Again, an old friend of mine, then a young man of some five-and-thirty years, then and since hardy and sound, on rising suddenly from bed in the middle of the night to empty his bladder, fell backwards, drenching himself with the contents of the chamber-pot. His wife told me that he lay unconscious for a "minute or two." The anxiety in such a case is whether the attack were a faint or an epilepsy: the circumstances of this attack, chiefly the person's sudden uprising, pointed rather to syncope, and time seems to have ratified this opinion, for no such attack has reappeared. On the other hand, syncope is not usually an isolated event in the life of the patient. People who faint are, as a rule, "given to fainting"; such persons dread hot rooms and congregations where the distribution of the arterial blood may widely
FUNCTIONAL DISORDERS OF THE HEART

oscillate. Or, again, they dread certain strong sense impressions—such as the sight of blood or strong odours, which may inhibit the heart; Italian women are said to be peculiarly liable to faint on the smell of flowers. On one occasion I was dining with a charming hostess who had decked her table with charms like her own: as we sat down, one of her guests, apologising for his weakness, said that he should faint if he sat with his back to the fire, and at some sacrifice of harmony he was conveyed to another seat; no sooner had he been dealt with than another guest thought he had better add that he himself was subject to faint in the midst of a strong scent of flowers, and that he also had better mention his unhappy susceptibility in time. There was nothing for it but to clear the table of the spoils of the Riviera; after which twofold commotion things fell a little flat. Both these men were literary men of more sensitiveness, perhaps, than virility, and had better have stayed at home. In such persons, of either sex, the pulse varies too widely on quickly rising, sitting, or lying down. The limits of such variations should be within five beats; if they are wider, and they are often as wide as twenty beats or more, the compensatory mechanism is defective.

Syncope without any organic disease may be fatal; such cases are not extremely rare; they are common enough to give a colour of caution to prognosis, and of care to the treatment. In my experience of such sad events I am disposed to think that the faints due to agonising pain are more likely to be fatal than those arising from sudden displacements of blood-pressure. The inhibitory effect of intense pain may, it would seem, arrest the heart through the vagus to a degree incompatible with life. Death in angina pectoris is due to this reflex effect of pain; the pain, in my opinion, having its seat in the aorta.

The premonitory symptoms of fainting are known to every one. He is a fortunate man who, in the weakness of some acute malady, influenza or the like, has not been conscious of the swimmings and exhaustions which may usher in a full attack. If some of us have never fainted, we have all of us felt faint. When the attack is fully established unconsciousness is complete, the respiration is only to be detected by the use of a feather or a mirror, or not even thus; and the pulse, cardiac and arterial, is likewise imperceptible. If the urine or faces are voided, it may be said with some certainty that the attack was worse than a faint.

Whatever the remoter causes, such as general anaemia and debility and the rest, the immediate cause of fainting is encephalic anaemia. The same is true, of course, in organic diseases, such as those of the heart. It is the first duty of the physician, as it is the care of nature herself, to place the patient in a position to favour the return of blood to the brain; the head must be dropped even lower than the trunk of the body. As on the one hand Junot's boot will produce syncope, so on the other to elevate the legs will aid in its dissipation. The blood-pressure must also be raised by causing contraction of the superficial blood-vessels; cool air, and the admission of it to the skin by unfastening the bodice, is one means of attaining this end; and it is, no doubt, of some use.
to loosen any bands which may be hampering the respiration; a deep
gasp, if it can be obtained, stimulates the heart to contract by unloading
the right ventricle. The respiration is called upon by reflex stimulants
also, such as smelling-salts, dashes of cold water, and so forth. In cases
of anemia compression of the abdominal veins may be useful, or the
application of an Esmarch's bandage to one leg or both legs, and in
extreme cases artificial respiration, or even transfusion of blood, might
be needed; but such difficult means are fortunately rarely if ever
necessary in the functional cases which alone are under our discussion
in this place. It is desirable, perhaps, to add that after the restoration
of consciousness the physician should not leave the patient without a
strict caution against the resumption of the vertical position until all
tendency to a return of the attack is averted. For a fuller discussion of
the physiology of events of this kind the reader is referred to an article
on the circulation of the brain, which will appear hereafter.

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to Dr. Harrington's article.

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MECHANICAL STRAIN OF THE HEART

Strain of the heart, it need scarcely be said, is not a malady, but the cause of maladies, both of this organ and of the aorta; possibly, also, of arterial disease beyond the aorta (Traube); in respect of this last suggestion, however, the evidence of an affirming kind is as yet scanty and uncertain. We shall see presently that to measure stress as a factor of heart and aortic disease, with any approximate accuracy, is beset with no small difficulty. That stress—mechanical stress—is an important factor in disease of the heart no experienced observer can doubt; moreover, as we shall find, in certain acute cases of strain this factor can be indicated with some precision: in chronic cases, however, stress is so intimately confused with other factors—such as the abuse of alcohol, the infections of rheumatism or syphilis and the like—that it is often exceedingly difficult to distribute its due weight to each one of such factors. For example, many most useful observations concerning strain of the heart have been made upon soldiers; yet there is perhaps no class of persons in whom the various factors of cardio-arterial disease, including improper dress, are more difficult to estimate severally. On the other hand, however, the part of stress in the causation of heart diseases comes out plainly when we consider such cases in numbers large enough to eliminate, or rather to reduce, the risk of error;—when, for instance, we contrast large numbers of persons engaged in laborious callings with large numbers of those whose
pursuits are mechanically less urgent,—when we compare forgemen, hodmen, navvies, wharfingers, Cornish miners or Tubingen wood-cutters, who have no monopoly of vice, with clerks, professional men, or even with persons whose callings are in the open air, but not to heavy muscular exertion. The part of stress, mixed as it still is with other factors, is made evident, again, in the comparison of the cardiac affections of men with those of women and children. In any case, while we remember that, relatively speaking, the function of every heart, healthy or diseased, is concerned in muscular exertion, yet when we enter upon a discussion of strain of the heart we are understood to refer to the effects of muscular exertion upon hearts which before the strain were either strictly or virtually sound. At the same time, we shall not forget that unusual exertion too often brings out a latent defect in a heart which under ordinary circumstances, and for some time at any rate, would have passed as sound. In men beyond middle life a breakdown of the heart is often thus acutely determined. In most cases of angina pectoris, suddenly appearing in persons previously regarded as healthy, some unusual bodily effort determines the first attack.

For clinical purposes strain in its effects upon the heart may conveniently be divided into functional disorders, injuries to the cardiac muscle, and injuries to the orifices and valves of the organ,—chiefly to the aortic. It is apparent at once that this distinction is a superficial one; mechanical disorders tend to become permanent, and aortic diseases, especially in the long run, are apt to be associated with muscular faults. Still, the distinction may be admitted for clinical purposes; and disorders of the first class have been considered in the chapter on “Functional Disorders of the Heart,” p. 821; those of the myocardium will be in the chapter devoted to this subject in the next volume; and those of the third class among the valvular defects. Without some such divisions the subject of heart diseases would be almost unmanageable.

When Harvey announced that the circulation of the blood belongs to the sphere of mechanics he wrought a revolution in physiology. Under his teaching vague and fanciful apprehensions gave place to more positive conceptions. From the time of Harvey, although physiologists have not asserted, that mechanical conceptions can cover the whole phenomena of the circulation, they have learned to see, nevertheless, that these conceptions cover so much of the ground that in mastering them they and their children may find reward enough. If this lesson be not thoroughly learned it has gained a good hold, and is proving its fruitfulness; yet it is not till the days of Marey, Ludwig, Roy, and Gaskell that we find a serious endeavour to ascertain the order of the phenomena of the cardio-vascular apparatus as a machine, and to indicate the limits of its physics in the direction of those nervous agencies which can only be called mechanical in a forced acceptance of the word. For a full discussion of cardiac physics, however, I have the advantage of referring the reader to the chapter on this subject from the hand of Professor Shervington (p. 464).
Cardiomotive force is equal to the output of the heart plus the resistance to the travel of the blood in the vascular system; a resistance chiefly due to friction, or, in other words, to the viscosity of the blood and the diameter of the channels through which it runs. The elasticity of the arteries adds nothing to the cardiomotive force; by it some considerable part of this energy is stored up in a potential form during certain moments of the revolution, to be given out at other moments. The elasticity of the arterial tree diminishes from youth to age, and as it is lost the work of the heart is increased; the work of the heart is thus increased at a time when the powers of the body are on the wane: but it is so difficult in later life to distinguish between lesions due to variations of stress and those due to intrinsic degeneration in the texture of the viscera, that when we speak clinically of strain of the heart,—that is, of a permanent "after-strain" or "set" towards other than the normal lines of its action, or of a permanent loss of capacity within these lines,—we are understood to contemplate young or comparatively young subjects, to contemplate premature tensile or shearing strains in the causation of which degeneration has had but a small initial share or none.

In what way or ways may stress in the heart produce strain? What are the conditions of abnormal pressure within or about the organ? How does it adapt itself to unusual stress? In case of failure where do the effects make themselves felt?

In the first place, we must realise that while, on the one hand, the arterial blood-pressure is incessantly oscillating, yet ordinary changes of stress do not raise blood-pressure permanently. If I lift a weight, say of ten kilos, my blood-pressure will rise promptly, even by some 20 per cent. After a few seconds or minutes, however (the interval depending on incidental circumstances), the blood-pressure will have returned to the initial level. Again, I may constrict a large artery, even the aorta, or by injection I may increase the whole mass of the blood in the body by as much as 20 per cent, yet in neither case will the blood-pressure (by "blood-pressure" the mean arterial pressure is usually signified) present more than a temporary rise. This is not the place to enter into the wonderful mechanism—none the less mechanical that the nervous system is largely concerned in the balance—by which these adaptations are made, the heart and aorta saved from strain, and the various areas of the body protected from irregular asflux of blood: suffice it to say that the re-adjustment is largely determined by reductions of resistance. But there is another factor, the factor of output; if the output of the left ventricle be increased, and this increase be not compensated, as is usual, by a fall of peripheral resistance, the chamber will be under increased stress, and may suffer strain.

Seeing then that, however transiently, blood-pressure is raised by muscular effort, and that output likewise may undergo considerable and even extreme variations, are the compensatory mechanisms always adequate to readjustments so rapid and so complete as to make the notion
of injury to the heart under ordinary circumstances improbable? If we
decide that the evidence points in this direction we may infer, neverthe-
less, that an unhealthy heart, or one subject to other adversity, will suffer
under great exertion if these compensatory mechanisms fail, or are in-
sufficient.

A series of experiments upon the blood-pressure of persons engaged
in muscular work was projected for the years 1895-96 by the late Professor
Roy and myself, but my colleague's unhappy illness prevented this and
other investigations. One rather curious fact, however, seems to come
out in the course of the more or less desultory observations which I and
others have made upon athletic men in Cambridge and elsewhere, namely,
that in them, as a rule, the habitual blood-pressure ranges low. A few
observations were made with Roy's sphygmometer upon men given to
arduous muscular work. These observations, taken at times of complete
or comparative rest, seemed to indicate that in them the arterial pressure
ranged habitually under the average. In my own person Alpine climb-
ing and, in later life, cycling have always been followed by a fall of
blood-pressure. It is hard to say what happens during spurs or at the
outset of an excursion, but very soon afterwards the pulse not only
quickens but softens; for the rest of the day and night, at any rate, the
pulse is soft and dicrotic. If on account of bad weather I cannot take
sufficient exercise, my pulse gives me the sense of higher pressure, and I
am conscious of a falling off in vigour and temper. I am well aware of
the difficulty of measuring the blood-pressure in man, and for the most
part we have as yet to be content with the impressions of experienced
clinical observers whose impressions must be taken for no more than they
are worth: still such impressions are not without value. If a number
of observers skilled in the pulse agree that the radial pressures of a set
of men seem to them to be low, this agreement is worth consideration;
at any rate nothing better is to be had except a few records with Roy's
sphygmometer, which corroborated those of the finger. An interesting
passage in Dr. George Oliver's treatise on Pulse-Gauging came under my
notice as I was correcting these pages for the press. He says (p. 126):
"Observations with the pulse pressure gauge have shown that, when
other indications are favourable, the lower ranges of pressure are not
only more salutary, but are very often compatible with the highest health."
Since these words were written, Dr. Tunnicliffe, in conjunction with Dr.
Brunton, from Mosso's laboratory has published like conclusions; and
so likewise have Tangl and Zuntz.

The converse of this preposition is seen in the rise in blood-pressure
in advancing years observed not by myself only (3), but by such experi-
enced physicians as Dr. George Balfour.

Habitual muscular exercise, then, tends in the main not to raise, but to
reduce mean arterial blood-pressure; or persons with relatively low
pressure may be well adapted to such exertions and naturally take to
them. During the first stages of muscular exertion, no doubt, the range
of blood-pressure is high; perhaps throughout severe exercise the mean
may be above normal. But during steady work it probably falls at least to the normal mean, and during rest and on quiet days may range below the average standard in sedentary men. If this be so, the hearts of athletes and of ordinary labourers should be, not at a disadvantage, but positively at an advantage.

How are we to reconcile these apparent contradictions? In one breath we say that excessive muscular exertion may damage the heart; and in the next, that on the whole the stress on the heart in muscular men is not more but perhaps less than in men who lead more sedentary lives. For while on the one hand I note that the blood-pressure of athletes runs a little lower than the average, on the other I note that the blood-pressure of men who lead sedentary lives, without denying themselves a like abundance of food, often runs high. I venture to think, from some little experience, that in members of a university or of the learned professions the blood-pressure tends to rise as athletic habits are laid aside. Perhaps by abstinence this disposition may be prevented; but I am always assured by brain-workers, and I share the prejudice, that for them also a somewhat liberal diet is required. For my own part I have found that I crave for food more when using my brains from day to day in my study than when taking vigorous exercise in the open air.

We have arrived, then, at the paradoxical result that muscular exertion tends on the whole to lower blood-pressure, and a sedentary life to raise it; yet that certain diseases of the heart are to be attributed to the mechanical effects of muscular labour. To reconcile these opposite positions we may make a twofold reply: although the mean result of muscular exertion may be to reduce arterial pressure, yet the initial effect of such exertions is to raise it, often enormously. If we may make the assumption of a man steadily working with his muscles at a uniform rate without rest the pressure in his arteries would probably be slightly under the mean of ordinary citizens; and although the rate of the heart would be increased, the total daily output might not be increased: if, on the other hand, we assume that the same man carries eight bushels of wheat up a flight of steps every ten minutes, although the mean of his blood-pressures for twenty-four hours may not be very excessive, the maximum pressures, that is, the initial rise at the outset of each effort, may be very high. Again, let us suppose that this man does not carry sacks hour by hour and day by day, but that he is engaged as a checkweighman and takes a sack up occasionally; it is likely in this case that his maximal arterial pressure, as he shoulders the sack, will be higher than under the same stress more regularly undertaken by a porter whose respiration, blood volume, and vascular distributions are better adapted to the recurrent stresses. Yet the weighman may be more or less accustomed to labour, and, if not used to such efforts as the porters are, he is nevertheless in something like training; if, however, a clerk from the office were fired, by exhaustion of the porters, to carry sacks, the absence of habitual adaptation to such exercises might cause so sudden and relatively so great an increase of arterial pressure as to rupture a limb of the aortic valve.
In what, then, does such adaptation consist? Partly in the behaviour of the skeletal muscles; partly in the function of the respiration. To take the muscular system first: we readily and rightly understand that the first effect of a general contraction of the muscular system must be to compress the vessels embedded therein, and thus at first to raise the blood-pressure to a degree answering to a partial closure of this vast area of the circulation. Marey demonstrated with the sphygmograph that even to throw the muscles of the legs into spasm (while breathing freely) raised the arterial pressure considerably. But in the next place, such is the exquisite provision of nature, the blood-vessels, under the reflex influence of the afferent nerves of the muscles, or, it may be, under the influence of an increasing acidity of their lymph when in action, dilate, and, reopening the vascular area which was momentarily constricted, they flood the muscles anew with arterial blood; thus at once the muscles are fed for the work and the peripheral resistance is lowered. This afflux is independent of the general arterial blood-pressure. Even under passive exercise also (massage), as Mitchell, Brunton, Tunnicliffe, and others have shown (vol. i. p. 378), the flow of blood through voluntary muscles becomes more abundant; and thus blood-pressure is reduced, if the kneading be unattended with irritation of the skin, which raises blood-pressure. To this compensatory mechanism we must add, in most cases, an increased circulation in the cutaneous area and sweating, as we see in the major epilepsy. If the blood-vessels, by the deterioration of advancing years, or of poisons such as lead, alcohol or syphilis, be less lively; if the blood be more viscous, or deficient in oxygen; or if after some disease the nervous machinery be less sensitive, less effective, or suffer any other disadvantageous change, the muscular reservoirs may open more slowly or less completely, and the arterial pressure will not fall so readily to the normal mean; the heart may not get the relief which is its due, and this organ and the larger arteries may suffer strain: or, again, the output of the left ventricle, increased probably in any case as the first acceleration of the rate subsides, may continue in a greater ratio than the fall of peripheral resistance, and the mean blood-pressure may be continuously higher than during rest. Dr. Weber, during an ascent at the beginning of his holiday, stated the initial rise of his pulse-rate to be from 74 to 122; but after a week's active walking the rise was from 74 to 105. He also noted that at first the systolic sound was shortened and less distinct (the systoles probably being "fractional," and the residual blood on each contraction large); but, as training advanced, the systole betrayed less interference, or even improved upon the quality of its tone before training began. Some cardiographic tracings taken on a man in severe exercise indicate at first great rise of blood-pressure; the upstroke is much higher, and systole encroaches more on diastole. As the impulse quickens, although the percussion is still powerful, the summit of the curve becomes sharper, the ascent more upright, and the duration of systole less extended.

Once more; an abundant supply of blood to the muscles, whatever
the remittant checks of the actual muscular contractions, brings about, after the initial moment, a large increase in the mean volume of the flow. What becomes of this abundance? Will it not try the heart at first in another way by flooding the right side of it, and thus throwing stress upon another part of the organ. This danger is, I think, as great as the rise of pressure on the arterial side; when the functions are duly adjusted it is counteracted by the capacity of the muscular and pulmonary systems, which may not only hold nearly all the blood of the body, but are less liable to be embarrassed by incidental adversity: moreover, Dr. Oliver has shown that as muscular exertion goes forward a considerable transference of juices from the blood-vessels takes place into the lymphatic areas. Nevertheless, engorgement of the right side of the heart is an evil to be counted with, and one which happens under exertion more often than we are disposed to think; and thus, unless the output be enlarged, the residual blood is excessive in one or both ventricles. As arterial blood-pressure falls venous pressure may rise, and the pulmonary artery and right heart may be fatally distended. In elderly people, whose lungs may be emphysematous, grave heart disorder may come about in this way; but in the young and vigorous the heart, though, as I have said, it often dilates until it beats in the epigastrium, soon recovers itself as the lungs expand and the blood is redistributed. But if the exertion be both hard and long continued, harm may be done even in the young, especially in boys; the more so as fatigue products are passed into the blood. Hence it is that prolonged efforts, such as paper chases and the like, are bad for boys, and murderous to the middle-aged. Such a case I have seen recorded in a man who, at the age of 46, took to a tricycle, and after a brief apprenticeship rode from Brighton to London (53 miles). The physician who was hurriedly summoned on his arrival in town, found him faint, with a pulse of 141, and cyanosed; the cardiac dulness was extended a quarter of an inch to the right of the sternum, the apex beat being in the 6th interspace in the mammary line.

That the respiration is an important factor in the blood-pressure, and in the run of the circulation, is apparent to every one who has watched the traces of the kymograph. Dr. Waller, Professor Tigerstedt, and others have carefully discussed the effects of the respiration on the functions of the heart; and Dr. Morison has recently drawn the attention of physicians to them again in the pages of a medical journal (58, p. 966).

That the respiration is quickened in exercise is a matter of constant experience. Stimulation of the peripheral end of a muscle-nerve produces considerable increase of respiratory movement, even when the muscles affected have been removed from the sensorium by cutting the sensory paths. The muscles may manufacture something which, reaching by way of the circulation some nervous element in the respiratory mechanism, stimulates it; the respiratory centre in the bulb may be thus stimulated. Professor Sherrington tells me that Zuntz and Goppert have proved that this something is not CO₂; nor again a deficiency of O.
Thus some waste product of the muscles seems to excite the respiration to greater activity, while at the same time larger quantities of blood are being injected into the vena cavae; how large this quantity is we may guess when we remember that on the contraction of a muscle its blood-vessels open out so widely that it can contain at least one-third more blood than when at rest; even when at rest, the skeletal muscles hold something like a quarter of the blood in the body. To this forcing of successive charges of blood by the muscles beyond the valves of the veins we have to add the suction of the respiratory movements.

The most important condition in the filling of the heart during diastole is of course its own previous contraction; but how far the heart itself exercises suction upon the blood as it enters is a problem which, as yet, is far from being solved. In the well-known experiments of Goltz and Gaule, negative pressures in both right and left ventricles were recorded in dogs, ranging from 100 mm. of water in the left, and from 10 mm. in the right, to numbers between 300 and 400 mm. It is not yet known, however, what the negative pressures in a strongly acting human heart may be. Prof. Tigerstedt, in his new work on physiology, says that the conditions of an effective suction are so many and complicated that at present no accurate opinion can be given on the matter.

As regards respiratory pressures, it is obvious that the pressure of the atmosphere on the extra-thoracic veins must be greater than that which, through the lungs, can be exercised on the veins within the chest; thus these veins and the heart must be distended in proportion to the difference. In inspiration this difference must be increased: the negative pressure within the chest must be increased, and in some proportion to the depth of the inspiration. Hence the aid of orthopnea in venous retardation. The intra-thoracic veins, the auricles, and the pulmonary artery must be distended, and the circulation would cease if a sufficiently deep inspiration were held on. In expiration, on the other hand, the negative pressure in the chest falls, and the access of the blood to the thoracic veins is slackened. Even the systole of the heart itself, by which movement much of the blood is driven out of the chest, must exercise some influence in the direction of suction towards itself, or, more accurately towards the great venous reservoirs. Now, whatever values we put on these several factors, we perceive that violent exertion must be attended by a considerable oscillation of pressures in the thoracic veins and right heart, oscillations due in part to the temporary rise in arterial pressure on the initial compression of the intra-muscular vessels, in part to the subsequent afflux of blood from the expanding muscular areas, and in part to the varying negative pressures of the respiration. The thick-walled ventricles and the aorta in which blood-pressure is high "will be least influenced, and the right auricle and the vena cava, which are thin-walled and almost at zero pressure, will be sensibly affected, the amount of blood-flow to the right side of the heart will be practically determined by it, and the left side will rapidly be affected in its turn. The left side will then drive a large quantity of blood forward soon after inspiration has begun, a smaller
quantity soon after expiration has begun. A violent and prolonged expiratory effort with closed mouth and nose may even cause a temporary arrest of the circulation, the intra-thoracic vessels being distended and the auricles unable to contract. Conversely, an expiratory effort made in the same way may arrest the circulation, as the venous blood cannot enter the compressed right auricle” (Waller). Now we know that on sudden and violent effort the chest is often fixed and the glottis closed. Again, in prolonged exertion, such as hard and long running, the advent of products of waste into the blood—sarcolactic acid, it may be, and others—must not be forgotten; for if they do not tend directly to increase the stress on the heart, they may do so secondarily and relatively in so far as they weaken the muscle of the organ. The rôle of each of these factors in the play of muscular exercise upon the heart cannot as yet be distinguished, still less calculated.

The clinical features of heart-strain, broadly speaking, are not so obscure as their causation. In my first paper on heart strain (1), I stated my opinion that dilatation of the right side of the heart is an early effect of prolonged exertion. The effect of sudden stress tells rather on the aortic area; that of more prolonged exertion, such as running, hill-climbing, or steady rowing, rather on the right heart. Time has strengthened me in this opinion; and Roy and Adami, Oertel, Dr. James Barr, and other authors have given their support to it. Not only are we strengthened in the opinion that dilatation of the right side of the heart is an occasional consequence of these prolonged exertions, but I now believe that dilatation of the right chambers is a frequent, I had almost said a normal incident of such exertion. I shall not be surprised to learn, from Röntgen rays or otherwise, that dilatation of the right heart and pulmonary artery is a common and transient feature in the adaptation of the heart to the variations of its work, especially in youths. The development of the muscles of respiration and of the lungs to capacities far beyond those of ordinary life, is a condition of training far too little understood or sought after, and takes a considerable place in the amendment of those selected cases which benefit under the Stokes, Oertel, or Nauheim methods. The safety-valve action of the tricuspid valve (Wilkinson King), and the apparent provision against this dilatation in ungulates by the moderator band, demonstrated by the late Professor Rolleston, are not to be forgotten in this connection. However this may be,¹ that dilatation of these chambers and secondarily of the left side also is a common result of prolonged exertion, and that it is often aggravated by the disabling effects of the circulation of waste products of a “curarising” kind, or by the nervous exhaustion of great fatigue, is tolerably well ascertained in a broad if not very accurate sense. The dilatation is, I think, concerned in “second wind”; the healthy heart increases its output, the lungs expand, resistance falls, the

¹ The dilatation of the left ventricle, although it certainly occurs, cannot, I think, be satisfactorily explained with our present knowledge. Probably it is due to loss of tone and large residual blood, or may be due in part to some nervous sympathy.
right ventricle pulls itself together, and second wind is established: This process, trying enough to an unsound or defective heart, to young boys, and to elderly men, is to the healthy heart of comparatively young adults perhaps never injurious; I have many times seen undergraduates, and others look ghastly at the end of a long spurt of hard exercise, but I never saw a sound young man the worse for a temporary stress of this kind: if, as in a few cases which I have seen again and again in growing youths, dilatation of the right heart occurs, leading to cyanosis, panting, and confusion or vertigo, this oppression is generally sufficient of itself to stop the exercise in time. Even in children, whose frames are immature, and who are apt to be overdone by prolonged stress, how rarely is the brief strife of hooping-cough attended with any ill consequences to the heart. In a few cases, however,—in untrained men hard driven by haste or peril,—prolonged effort, exhaustion, heart stress, and fatigue products come in to complicate the reckoning, and persistent harm may be done. I have already published one carefully observed instance of cardiac dilatation in my own person (1), I will now describe another. Some fifteen years ago I was called in the middle of the night, when no doubt more or less fatigued already, to take a mail train at a station about four and a half miles distant; when I had hastily dressed, I discovered that the foolish cabman who had brought the message had driven back to town. In forty minutes I had to catch that train; and, running all the way on a hilly road, I did catch it. Profusely perspiring, I stripped the instant I sprang into the carriage, and found the transverse dull area of the heart considerably extended, as it was on the Dom in 1869. The radial pulse was rapid, of small volume and low pressure; I felt a little sick, and my face was cold. After a good rub down and an hour’s rest in the train I was quite restored; the borders of the cardiac area had receded, and I felt no more of the stress. But it might well have been otherwise; it would have been otherwise if at that time I had been in bad condition. It is thus in persons at and after middle life that the physician has to patch up the heart thus strained; some of these patients recover after months of disability, others never recover, though life may continue for some years. An old friend of mine, when about fifty years of age, thus strained his heart by hard walking in hot weather on the Italian side of the Alps. He broke down and came home, when we found the dulness of the heart much extended transversely, and other signs of dilatation. The pulse was extremely irregular and intermittent, and these characters it never lost, though some fifteen years of a valetudinarian life remained to him before edema and albuminuria ushered in the closing scenes of his life. I have notes of many such cases of strained heart, especially in men who by years or by frailty were passing or past their prime.

Pain and constriction are felt in the acuter cases, but rarely (or never) shooting into the arms; though it is sometimes felt as far as the second left intercostal space. There may be a panting dyspnea, a cold dew on the forehead, yawning, and exhaustion. The pulse is proportionately irregular in force and rate, and intermittent (vagus protection). A man of letters,
whose constitution had been shaken by profuse hemorrhages in early life, took to the bicycle in middle age, and often rode hard and far. He complained to me that at times he felt some discomfort from it. On careful examination I found no sign of disorder; but I begged him to end his next hard ride at my house. I then found his heart irregular and intermittent, the arterial pressure low, and the right ventricle dilated. Fortunately on his next visit he was well again, but repentant. In the worst cases edema of the bases of the lungs is found on the following day. Such attacks pass off hardly and slowly; the pulse long remains irregular and feeble, and the breathing embarrassed by the least effort. There is probably a large quantity of residual blood in both ventricles for a longer or shorter time, the signs of dilatation appear on the left side also, arterial pressure falls, and the mitral orifice may yield. Such a patient may, indeed, fulfill the duties of a tranquil existence for some years; but he may remain languid and pallid, unfit for much physical exercise, and in all the work of life soon wearied into fretfulness and depression of spirits. In the next stage of the disease albumin appears in the urine, and edema about the legs and feet; yet even then the end may not be imminent. But on this part of the subject the reader is referred to the section on diseases of the myocardium (p. 885).

Soldier's heart.—I venture to give this name to a disease well known to physicians in the army, not by any means with the intention of confining the class of cases now to be considered to the soldier, but to indicate a state of heart which is peculiarly apt to occur in him, as its causes are of kinds to which soldiers are more exposed than civilians. Nevertheless if civilians, or men in other services, are exposed to like influences, they also will be liable to "soldier's heart." Our attention was first drawn to this condition by Brg.-Sur. Lieut.-Col. Myers more than thirty years ago; and his most recent views on the subject will be found in Quain's Dictionary under the head "Exercise." In the United States the subject was first studied by Dr. Da Costa. Many cases of the kind, occurring in civil life, come under the notice of the general physician, so that the condition is now well known. The degrees of the malady range between the transient disorder of the heart seen in any youth who in a somewhat too reckless pursuit of exercise may be disturbed with some palpitation and dyspnea for a few days or weeks only, and a persistent disease of an incurable severity. The former transient cases fall under the head of "Irritable Heart," in the chapter on "Functional Disorders of the Heart" (p. 807); the latter fall into the present section on "Strain of the Heart." The differences are indeed no more than of degree; but in comparing the extremes cases we find a difference of degree amounting to a difference in kind.

In Quain's Dictionary Myers says: "The young soldier of light frame, with irritable, palpitating heart, who has broken down in his preliminary training, is a marked and good example of the early injurious effect of overstrain of the heart, under the impediments, caused by tight
clothing and accoutrements, to the free expansion of the chest. When at
rest he feels perfectly well, and has little or no throbbing in the chest.
So soon, however, as he puts on his tunics and accoutrements, and begins
his drill, throbbing occurs with more or less violence, accompanied with
a feeling of oppression and with difficulty of breathing, and this being
followed by a sensation of faintness, sickness, or dizziness, he has to fall
out of the ranks. At first the condition is one purely of functional dis-
turbance which, though rendering him unfit for the duties of a soldier,
does not interfere with his gaining his livelihood as a civiliaip.” In dis-
cussing the late Dr. Morgan’s evidence of the safety of athletic pursuits
afforded by the experience of University oars during the years 1820-1869,
Myers properly warns us that these were men picked for their large frames,
full chests, and exceptional strength. On the other hand, from a large
experience of University men, I must say that considering their “violent
and unguided efforts to achieve success” and their “ill-regulated emula-
tion,” the ill effects are surprisingly small. Many are the “irritable
hearts” (p. 821), but permanently or gravely injured hearts are few
or none. On the other hand, I agree with Myers in his admonition to
“men who have settled down into the real business of life who, during
their nominal periods of rest from their daily labours, undertake violent
exercises without any preliminary training, and thus throw such an
unexpected strain on the heart and blood-vessels that instead of mere
functional disturbance, as in early life, they sow the seeds of organic
disease.” Although I am tempted to minimise the allegations of serious
harm to the emulous young men (for among them there is now a sort of
natural selection, the weaklier taking to girls’ games, such as lawn tennis,
hockey, cycling, and the like), yet I cannot enforce too strongly his
warning to older men who are not in the casual training in which all
healthy youths are constantly, if more or less unsystematically, engaged.
Bear-fighting among themselves, running and shouting with the games of
others when not themselves at work, bounding up and down long flights
of stairs, scampering, always a minute too late, to lecture or chapel, they
are always more or less in training, and, being well and plainly fed and
devoid of care, they bear what the elder brother cannot bear, who goes
to his work in a stuffy office by underground rail, loafs to his club in a
hansom, dawdles at dinner-parties and At Homes, takes his exercise
vicariously by watching the games of others, and spends the save of his
time with his feet on the chimney-piece with the eternal cigarette in his
mouth. This overfed and self-indulgent person, who is plucky enough
when needs must, is surprised that he goes to pieces when, on his month’s
holiday, he competes with mountaineers or sportsmen who are in fit con-
dition, and who live sparingly. If this be true of the eldest son, what of
the father, who will not be forgotten but, with his nervous system corroded
by drudgery and care, is determined to search on his bicycle, or to climb
the Alps with any of them. These forcing kinds of effort it is which tell
for evil far more than ordinary sports by field and stream, which never
lead to strain of the heart.
One of our younger graduates, Dr. M'Carthey, has recently taken up this matter of soldier's heart in an exercise for his degree. He obtained his materials at Netley. After stating that the modern valise equipment is less injurious to the young soldier than the old knapsack, which by its cross belts constricted the chest, he adds that the malady is still common enough nevertheless. He was able in a short time to collect twenty cases, and also to examine the first batch of twenty soldiers invalided from the campaign on the Indian frontier, and of these again five were found to be patients of this class, though not included in his series.

In dealing with his twenty cases, M'Carthey took out in each the age, total service, the trade of the recruit before enlistment, the habits as to tobacco and alcohol, the climates of foreign service, and the infectious and other diseases which he might have undergone. Fourteen of the men were in infantry regiments, three in the Royal Artillery, two in the cavalry, one in the Royal Engineers. At the date of examination two were under the age of twenty-one; fourteen were between twenty-one and twenty-five; four were of twenty-five years and over. "Taking the statements of the men as true," the average amount of beer consumed daily was from three to four pints. Other alcoholic drinks were taken but occasionally. The average amount of tobacco was three to four ounces a week, the tobacco being generally twist or plug. Twelve had suffered from syphilis; fifteen from malarial and other tropical fevers; two only from rheumatism of any kind. Some of the men figured, of course, in more than one of these categories.

"The patients state that while not exerting themselves they feel quite well and free from any shortness of breath; but as soon as they begin to march they are troubled at once with a throbbing sensation in the chest; and with this there is difficulty of breathing, followed in some cases by faintness or giddiness. Rest may relieve for a time, but in most cases all the trouble returns shortly after returning to duty."

On the other hand, many men (not in the above list) have the disease, yet state that it has never been of any inconvenience to them whatever. "In fact, many cases of disordered heart have been detected quite by accident while going through the usual routine examination, when soldiers come into hospital for other complaints, especially malarial fevers."

To take the symptoms in detail—Cardiac pain was present in seventeen cases, dyspnoea in seventeen, giddiness in six, sleeplessness in five, nervousness in seven cases. Three cases were noted in which the men were unaware that there was anything wrong with the heart.

Physical signs.—In fourteen cases the pulse was regular while the patient was at rest, but in some of these it became irregular after slight exertion; in the remaining six it was irregular even when the men were confined to their beds. In twelve cases the pulse during rest was below 100; in six it was between 100 and 115; in two between 115 and 120. The pulse rarely exceeded 120 when the man was at rest, but would always rise very rapidly to 140 or so on his swinging the arms three times round the head. In nearly all the cases the pulse was of abnormally low pressure.
The area of cardiac dulness was increased in fourteen cases; but in some of them the increase was so slight that it was recorded with hesitation. In all the cases the impulse was diffused, and in many the apex was displaced—in two cases between 1 and 1½ inches outside the nipple line. Abnormalities of the cardiac sounds were uncommon. In two cases the second sound was reduplicated at the base; in five the pulmonary second sound was accentuated; in six the first sound was sharp; in three, prolonged and booming; in four cases there was a systolic murmur at the apex.

Dr. M'Carthy lays stress on the history of malarial fevers in many of these men; he reminds us of the evil effect of fevers on the cardiac muscle, and urges that soldiers recovering from these fevers should be exempted from drills and other manual work for several weeks after discharge from hospital.

Alcohol is the next cause on which he lays stress; and, as to tobacco, he says that men smoke more in the tropics where they loaf more; and that the tobacco is bad and strong. On campaign the rations also are often necessarily short, while the labours are excessive. Finally, the author urges that tropical heat reduces the value of hemoglobin in the corpuscles of the blood, and leads to anemia. If in this condition the soldier is called upon to do hard muscular work, is badly fed, and mayhap attacked by some fever, the softened and flabby heart muscle yields, dilatation occurs, and the man is invalided.

The prognosis is not good; in the majority of cases the patients return to hospital till they are invalided out of the service. The author found the difficulty which might be expected in tracing the men thus invalided. However, he obtained records of thirty cases of men discharged from the Netley Hospital, and his impression from these returns is that in many cases the soldier's heart ends in valvular disease. It is said in Cambridge that influenza is very mischievous in lodging-house keepers, who cannot keep their beds and are frequently running upstairs.

As in various parts of this article I have more or less incidentally referred to cases of this kind as they occur in civil life, I have nothing to add to this careful inquiry of Dr. M'Carthy. I have already said that the obstinacy of these cases is remarkable. Those which pass the line between "irritable heart" and "soldier's heart" rarely end in recovery, but in permanent dilatation often resulting, sooner or later, in mitral insufficiency. I have only to add that these cases are not only unrelieved by cardiac gymnastics (Nauheim methods and the like), but are aggravated by such means. Although muscular exertion is the determining, and perhaps an indispensable, cause of "soldier's heart," yet it manifestly depends also upon many contingent conditions.

I cannot conclude this section without a formal opinion, founded on thirty years of close observation of heart stress, that the importance of muscular effort as a factor in cardiac disease has been much exaggerated. I have shown that in the sound adult organism the effects of physical
stress upon the heart are promptly counteracted by equilibrating machinery, and especially by large expansion of muscular and pulmonary areas. Such a statement as that made three years ago by the editor of a leading medical journal, namely, "that the violent strains of hard exercise bode in the end the certainty of premature decrepitude," and that "the heart can only perform a certain total measure of work," so that "whether this be done by a rapid or a slow process determines the length of days in which it is done," seems to me, both on clinical and physiological evidence, to be unjustifiable.

The clinical story of strain in the aortic area of the heart will find its place in a later section.

T. Clifford Allbutt.

N.B.—For references the reader is referred to the list on page 966.

INJURIES BY ELECTRIC CURRENTS OF HIGH PRESSURE

Since electricity has come to be so widely employed, and is being increasingly used as an illuminating agent and for motive power, accidents of varying severity have been frequent. It is desirable, therefore, that we should be cognisant of the effects of high electrical currents upon the human body. We know that there is considerable danger attendant upon the generation of electricity, and we look to the expert electrician to adopt measures to prevent accidents. During the four years ending 1896 twelve deaths occurred in this country from electric shock; and when we add to these the many lesser accidents that frequently occur, we recognise the need for careful precaution wherever electricity is being generated and distributed. Many of the accidents have been due to inadvertent contact with exposed parts of highly charged metal not properly insulated. The consequences of the current thus passed through the body vary with the amount of current entering, the insulated position of the individual at the time, and the kind of contact. Such conditions, for example, as standing on wet earth, the wearing of damp boots, and a moist skin tend to increase the effects of an electrical current. The danger, therefore, is not one simply of high potentiality of current, but of current plus the conditions under which it has been received. The word voltage used in this article is synonymous with "pressure" as used by the Board of Trade, and with the "electromotive force" of the text-books.

It is difficult to say what voltage is fatal to man. Speaking in terms of voltage Dr. W. S. Hedley says that 1000 to 2000 volts will kill. In America, where electricity was adopted as the official means of destroying criminals, a current of 1500 volts has been regarded as capable of
causing death; but there are many cases on record of persons having been exposed to higher voltages without fatal consequences, and, on the other hand, contact with lower pressures has caused death. Of the two kinds of electric current—the "continuous" and "alternating"—it is impossible to say which is the more dangerous to the human body. There is an opinion that the alternating is the more fatal; but a larger experience and further experimental data are wanted before any definite conclusion on this point can be arrived at. Under either the difference may be less (Tatum). On the relative danger to life of the continuous and alternating currents, the Report of the Board of Trade states that alternating currents are twice as dangerous as the continuous, but I know of no evidence upon which this statement is based. As electricity is too difficult a subject for a non-expert to handle, only those points are here discussed which bear upon the medical aspect of the subject, points with which medical practitioners should be familiar, as at any time they may be called to persons injured by high electric currents.

A person, for example, may be seriously injured either by direct personal contact with a highly charged piece of metal, through the medium of damp clothes or through an iron tool in his hand by which accidental contact is made with the live metal. As an illustration I may mention the fatal accident to a youth at St. Peter's, Newcastle-on-Tyne, in January 1897. Carrying an iron ladder through the factory he accidentally brought the top of the ladder into contact with the terminals of an arc lamp. He was killed instantaneously. In regard to arc lighting, it may be mentioned that while each arc light requires an electrical pressure of only from 40 to 50 volts, the lamps are usually arranged in a series and are supplied by the same current. A workman who is himself insulated may touch the terminals of an arc light without receiving any injury; but should his insulation be defective, if he stand on moist earth for example, he may receive, as did the youth at St. Peter's, a fatal shock, since the electrical pressure between the ends of the cable is the sum of the pressure of all the lamps in series in the circuit (2).

We have no positive proof that one individual is more susceptible to electric shock than another. It is, as already stated, rather a question of the amount of current and whether it wholly enters the body. Where contact with currents of high potentiality has not been followed by disastrous results, it is more than probable that at the time of contact the skin was dry, in which state it is a bad conductor, and offers considerable resistance to the penetration of the current. As might be expected, the electrical current produces very varying effects upon the human body. Where the voltage is low and the contact fairly good the muscles are thrown into a state of tetanic rigidity which makes it impossible for the individual to relax his grasp of any charged metal he may have seized, nor can he be released until the circuit is broken. The effects of electric currents are experienced when they enter and when they leave the body. It is sufficient for us to remember that
effects are produced at the moment of the entrance into and exit of currents from the body, and that these, therefore, are periods of danger. Hedley, in supporting the opinion that the quantity of electricity passed determines the amount of electrolytic action and physiological effect, considers that more pain is felt the higher the electromotive force, even when the current is the same. One element entering into the causation of pain is the local action of the accumulated products at the point of contact consequent upon electrolytic decompositions, and the relative resistances between the electrodes and the different layers of the skin. The individual through whose body there is passing an electric current of not too high potentiality generally experiences pain, but some of this must be due to the extreme contraction of his muscles quite apart from the influence of any products of electrolysis. If there be no immediate loss of consciousness, terror may cause him to faint. The memory of this plays no small part in the subsequent development of nervous symptoms. Once liberated, the patient, as a rule, is soon well again, but there are instances on record where for many months after exposure to the current there was complaint of ill-defined pains and headache which recurred with electrically disturbed conditions of the atmosphere, and of a form of persistent nervousness which was rather the result of the mental than of physical shock.

Another consequence of the exposure to high electric currents is burning. That portion of the surface of the body which has accidentally been brought into contact with the charged metal may become black and charred, the peculiarity of such a wound being that it is sometimes deep and apt to slough, and that while the burned part is insensitive to pain the surrounding tissues are extremely sensitive. If the skin at the time of contact was moist so much more severe is the burning if a current sufficient to produce this severe local burning pass through the body, fatal results are the more probable; but if the current merely passed locally, as from the hand to the wrist, for instance, the damage will probably be local only.

When, therefore, the pressure has been high, the contact good, and conditions of resistance slight, the patient may at once be rendered unconscious, or be suddenly killed. Thus stricken by a powerful current a man suddenly falls, or he is thrown a distance of several feet before falling. A peculiar cry is involuntarily uttered, especially when the contact is broken, which, in electrical generating stations, for example, at once attracts workmen to the spot where their comrade is lying, pale, or slightly cyanosed and pulseless, apparently dead, and with mucus escaping from his mouth and nose; now and then a feeble and gasping respiration is observed, but he lies helpless, his pupils keep dilating, and unless artificial respiration is at once resorted to, and sometimes even then, death is inevitable. There is something appalling in the extreme suddenness and severity of the shock in these cases, towards which the unexpectedness of the accident possibly contributes largely.

Cause of death.—In conjunction with Dr. R. A. Bolam I undertook
a series of experiments in the Physiological Laboratory of the Newcastle College of Medicine upon anaesthetized dogs, with the view of ascertaining the cause of death by electric shock, and of testing the means of resuscitation (7). Two opinions are held by the profession: (i) that death under such circumstances is due to respiratory arrest; (ii) that it is consequent upon sudden cessation of the heart’s beat. By placing dogs under the influence of ether we were able to take a tracing of the arterial pressure and respiratory movements, and thereby to record the effects of high electric currents passed into the body. Immediately on making contact the animal is thrown into an attitude of opisthotonos, its muscles become extremely rigid, and as a consequence the lever recording respiratory movement is suddenly and violently thrown up, whilst the other, which traces the arterial pressure and heart-beats, suddenly rises owing to general arterial constriction, and falling shortly afterwards oscillates rapidly, but within a narrower range. On breaking the current the respiration becomes deeper and quicker than before the shock, and in the course of a few seconds the breathing and the beat of the heart return to the normal. When the current proved fatal there were the same initial respiratory and general muscular spasm, and a sudden rise of arterial pressure followed by an immediate fall; one or two quivering oscillations of the lever mark the arterial tracing, and then all at once a further and complete fall of the lever follows, indicating that the heart has ceased to beat. Respiration deep and ‘spontaneous may continue for several seconds, or even for a few minutes after the heart has ceased to beat. The experiments invariably showed that in electric shock the death was cardiac and not respiratory. Other steps were taken to confirm this opinion, notably by listening to the heart of the animal with the stethoscope as the current entered. If the current were insufficient to kill the dog the heart’s beat was momentarily delayed and then quickened, the cardiac sounds being well maintained; but when, on the other hand, a current of higher potentiality was employed, the sounds of the heart would cease, immediately or very shortly after contact. Respiration deep and rhythmic might continue, but if no treatment were adopted the cardiac sounds would not return; increasing pallor would gradually steal over the whole surface of the body, the pupils meanwhile dilating, and mucus being forcibly driven from mouth and nares. By exposing the heart of other anaesthetized dogs, and inserting a canula into the trachea so as to carry on artificial respiration, Bolam and myself had ocular demonstration that it was the heart which was primarily arrested in death from electric shock, and not the breathing. Dr. A. M. Bleile (3), Professor of Physiology, Ohio State University, in a paper read before the American Institute of Electrical Engineers, Niagara Falls, N.Y., June 27th, 1895, states that “death in electric shock is really due to the fact that the current produces a contraction of the arteries through an influence on the nervous system, and that this constriction of the arteries throws in such a mechanical impediment to the flow of the blood as the heart is unable to overcome, and that where drugs are given to counteract this effect, much
larger doses of electricity can be borne." As to the constricted state of the arteries, we ourselves found, with Bleile, that if nitrite of amyl were inhaled by an animal before the electrical experiment, much stronger currents could be borne. My results, then, and those of Dr. Lewis Jones likewise, are opposed to those of D'Arsonval, who attributes death to asphyxia.

**Morbid anatomy.**—There is usually well-marked rigidity of muscles. The skin may or may not show any signs of burning or of eschars; it may be pale or livid. The abdominal viscera and large veins are usually deeply congested. The heart is usually flaccid: sometimes the right side is flaccid while the left is hard and tense. The right auricle and ventricle are considerably distended and are filled with dark fluid blood; the left auricle is generally in moderate distension and contains fluid blood, whilst the left ventricle is firm and almost empty. The lungs present nothing abnormal; they may be slightly congested or at places show ecchymoses, particularly if artificial respiration has been attempted. The brain and spinal cord are congested, but are otherwise normal. I have seen it stated at a coroner's inquest, a diagnosis was based upon the assertion, and the corresponding verdict of the jury returned, that in death from electricity the blood is fluid and not coagulated after death. This is too sweeping a statement, and not quite correct. In most cases, it is true, the blood is found fluid after death, but in some of our experiments we found coagula in the right side of the heart, and occasionally some of the large veins were blocked by dense dark clot—particularly when the autopsy was made twenty-four to thirty hours after death. It is maintained that on spectroscopic examination the oxyhæmoglobin of the blood is reduced. If a strong solution of blood is examined, only one broad band may be observed in the spectrum, and it appears at first sight as if this were due to reduced hæmoglobin; but where the spectrum is very carefully scrutinised, and particularly, too, when the solution of blood is further weakened by the addition of water, two distinct bands of oxyhæmoglobin can be clearly discerned. It would appear, therefore, that the blood contains both oxyhæmoglobin and reduced hæmoglobin. The blood on microscopical examination shows very marked creation of its coloured corpuscles. The pupils were invariably found widely dilated immediately after death.

**Treatment.**—Persons who have received only a slight shock and who have not been rendered unconscious require no special treatment. The effects almost immediately pass away, and should any nervous symptoms remain they must be treated on general principles. For any burns or wounds ordinary surgical remedies will avail. It is to the treatment of persons who have been exposed to high electrical currents, and who are apparently dead, that the following remarks apply. D'Arsonval (1), believing the mode of death to be akin to asphyxia, recommended artificial respiration, and of all modes of treatment, quite irrespective of whether the death has proceeded from failure of the respiratory centre or of the heart, I know of, no treatment more likely to be beneficial than artificial
respiration, systematically carried out by Sylvester's method, and continued for half an hour or longer. Balam and myself have twice succeeded in resuscitating a dog whose heart had ceased beating, once for thirteen minutes, and on the second occasion for eight. The heart, which was exposed to view, had become rapidly distended so as to bulge out the pericardium, and had become perfectly motionless after having passed through a stage of fibrillary tremor; but by persisting in artificial respiration, aided by the occasional spontaneous inspirations which from time to time occurred, and the rhythmic traction of the tongue, the contents of the right side of the heart were gradually aspirated into and through the lungs, auricular beats were re-established, at first irregularly and feebly; gradually, however, they became stronger and passed over into the ventricle, so that after thirteen minutes, during which the heart was apparently irresponsible, we had the satisfaction of seeing the normal beat of the organ restored, the pulmonary and systemic circulation re-established, and life return. Too often, however, the sufferer is killed outright. Rescuers on approaching the injured must beware lest the current be not broken.

THOMAS OLIVER.

REFERENCES


T. O.

ENDOCARDITIS

I. ACUTE ENDOCARDITIS

Definition and Classification.—By endocarditis we mean inflammation of the endocardium or lining membrane of the heart. The inflammation affects principally and often exclusively the valve segments of the endocardium (valvular endocarditis), but other parts of the endocardium may be affected also (mural endocarditis). Both clinically and pathologically we distinguish between acute and chronic endocarditis. The acute form is again divided into benign or simple and malignant or infective endocarditis. Of the chronic form, likewise, we distinguish two kinds,—one which is the result of acute endocarditis, and the other the retractile, fibroid, or sclerotic form, which results from arterio-sclerosis or atheroma.

In this article we shall consider acute endocarditis; chronic endo-
carditis, giving rise to the majority of so-called valvular affections of the heart, will be dealt with hereafter.

**Acute endocarditis.**—In the article on Infective Endocarditis I have already considered the difficulty of separating simple from infective endocarditis. In both forms micro-organisms have been found in the affected valves, though only in the infective form do they play an essential part, so far as symptoms are concerned. While the two kinds have many features in common, in others they differ; and as the difference is often essential we follow the custom and consider the two kinds separately.

**Acute simple endocarditis.**—(Syn.: Benign, Papillary, Verrucose, Rheumatic Endocarditis.)

**Causation.**—By far the largest number of cases occur with (a) acute rheumatic arthritis, hence by some authors the name acute rheumatic endocarditis is given to the disease. Its frequency in acute rheumatism is differently estimated by different authors; and this is readily to be understood, for in many cases the symptoms of endocarditis may be so slight as to escape detection; or again persons recovering from acute rheumatic arthritis may show signs which simulate those of endocarditis, and yet are due only to some functional derangement of the heart. The most trustworthy observations on this subject are those in which a large number of cases of rheumatic arthritis have been kept under observation, and their after-history watched for some time. Sibson analysed 325 cases of acute articular rheumatism which he observed during fifteen years at St. Mary's Hospital, and found that in 79 there was no endocarditis; in 63 endocarditis was threatened; in 13 endocarditis was probable; in 107 endocarditis was present without pericarditis; in 54 there was endo-pericarditis; in 6 there was pericarditis without endocarditis; in 3 there was pericarditis with doubtful endocarditis.

The proportion given by other observers is somewhat less: the mean of the numbers given by older and more recent writers amounts to 20-23 per cent.

Of other noteworthy facts which have been made out regarding the relation of rheumatism and endocarditis we may note:—

(i.) That, in connection with rheumatism, endocarditis occurs more frequently in children than in adults. Dr. C. West estimated its incidence at 61-3 per cent; Fuller about 66 per cent; and some authors, such as Cadet and Gassicourt, give as high a percentage as 80 per cent.

(ii.) The first attack of acute rheumatism is more often followed by endocarditis than the subsequent attacks.

(iii.) Endocarditis may accompany mild as well as severe attacks of acute rheumatism. Sibson (loc. cit. p. 199) states the more severe the rheumatic attack the greater the tendency to endocardial inflammation; but this is not the opinion of other observers, and in children especially we see mild attacks of rheumatism followed by endocarditis. [Fide art. "Acute Rheumatism in Childhood," vol. iii. p. 42.]
(iv.) The physical signs of endocarditis usually appear early in the attack of rheumatism. Sibson in about one-fourth of his cases noticed the presence of a systolic bruit, which he looked upon as characteristic of endocarditis, at the end of the first week of the rheumatic fever, and in two-thirds at the end of the second week. Sometimes, however, the signs of endocarditis appear much later, though probably in many of these cases the endocardial affection had existed some time before it gave rise to physical signs. The endocardial affection may precede the rheumatic attack by several days.

(v.) Rheumatic fever, or acute polyarthritis, is the disease above all others accompanied by endocarditis; but occasionally endocarditis may follow monarticular rheumatism and chronic rheumatism. Gonorrhreal rheumatism stands in close relation to infective endocarditis, though the benign form may follow definite attacks of gonorrhreal rheumatism.

(vi.) The endocarditis dependent on rheumatism most frequently affects the mitral valve; the aortic valve less frequently, and the right side of the heart in very exceptional cases only.

(vii.) The pathogenesis of what we may call the meta-arthritic endocarditis cannot be determined as long as our views on rheumatism are as indefinite as they are at present. The endocarditis cannot be looked upon either as a mere complication or as a sequel of rheumatism; it is an integral part of the disease. As most pathologists look upon rheumatic arthritis as an infective and most likely a microbiic disease, the poison of which chiefly attacks fibrous structures, the endocarditis may be regarded as a localisation of the rheumatic poison in the fibrous tissue of the valves of the heart. In some few cases the same micro-organisms have been found both in the effusion of the inflamed joint and in the inflammatory deposit of the cardiac valves. As in other microbiic affections, so probably here, the lesions are due to some toxic product of the microbe circulating in the blood, as is the case in other infectious diseases; to wit, diphtheria, cholera, and epidemic influenza; and if so, the absence of micro-organisms from the deposits is quite intelligible. As the opportunity of examining the valve often does not arrive until the endocarditis has become chronic, the absence of all micro-organisms, even if the disease be microbiic, is not astonishing; for this negative condition occurs in certain other diseases which are undoubtedly microbiic.

(b) Chorea.—Endocarditis is frequently met with in persons who have had chorea; and in fatal cases of chorea inflammatory deposits on the valves are almost invariably found. Thus Sturges (24) collected statistics of 80 fatal cases, and in only 5 of these were the heart valves normal. Reymond’s figures bear out the same rule. As regards the frequency of endocarditis in chorea authors differ considerably; and, as the endocarditis may not reveal itself till years after, the exact proportion is not easily made out. In many cases of chorea a murmur may be due to functional disturbance and not to endocarditis; or, on the other hand, as seen in some fatal cases, endocarditis may be present and give rise to no physical signs. Osler states that of 554 cases of
chorea, at the Infirmary for Diseases of the Nervous System, 170 presented heart murmurs; of these, in 149 the murmur was apical, in 21 basic. Of 449 cases reported to the Committee on Collective Investigation of the British Medical Association, 113 had heart murmurs; how many of these were functional and how many organic it is impossible to estimate: a basic murmur is heard much oftener in purely functional cases, yet an apex bruit may be present from various causes without the existence of endocarditis. More trustworthy results are obtained if the subsequent history of persons having had chorea is taken, an estimate which has been made by several observers. Dr. Stephen Mackenzie examined 33 patients at periods varying from one to five years after the attack of chorea, and noted signs of undoubted heart disease in 60·6 per cent; Donkin in 40 per cent; Osler out of 140 cases found the heart normal in 51; in 17 there was disturbance which might reasonably be looked upon as functional, and in 72 cases (51·4 per cent) there were signs of organic heart lesion: it may be noted that only in 25 of these 72 cases was there a history of acute arthritis.

Nothing more definite can be made out concerning the relation of chorea to endocarditis. So many cases of chorea show signs of acute rheumatism during the course of the attack, or are followed by an arthritic affection having all the characters of acute rheumatism, that the endocarditis has been regarded as a manifestation of the rheumatism only; yet, as will be seen from the figures given above, in many cases where heart murmurs were noted there was no history of acute arthritis; and this is also noticed in fatal cases of chorea, in which endocarditis is almost invariably found. The report of the Collective Investigation Committee of the British Medical Association gives of a total of 439 cases of chorea 97 with a rheumatic history (about 22 per cent). Statistics on this subject, however, are not of great use, as the joint pains occurring in chorea are not always due to rheumatism; and, especially in the severe cases, are probably due to a septic condition. We must note, however, that in children the joint affections in rheumatism may be very slight. I cannot therefore agree with Roger that chorea, rheumatism, and endocarditis are three terms of one and the same pathological series; though undoubtedly in a good many cases endocarditis and rheumatic arthritis, with other signs of a rheumatic diathesis (tonsillitis, subcutaneous nodules, erythema, profuse acid perspiration, etc.), complicate chorea. As yet there is no proof or evidence that chorea is due to minute cerebral symbolisms of microscopic nature; moreover, the form of endocarditis we notice in chorea is—in by far the largest number of cases—of the benign or verrucose and not of the malignant nature, in which latter the microbe plays the important part. We can but surmise that chorea, by weakening the system, and exercising some deleterious effect on the heart valve, acts only as a predisposing agency.

- (c) Acute endocarditis may be associated with the acute symptomatic fevers. Among these scarlet fever occupies the first place. Often we have, before the occurrence of the endocarditis, pains and slight swelling of
a joint, which are apparently rheumatic in nature; thus here again
the rheumatic poison is the cause of the endocarditis. Some authors,
however, look upon the arthritic symptoms as an outcome of the scarlet
fever toxin, and upon the endocarditis as the result of the action of the
game toxin on the endocardium. In the other acute fevers, such as
typhoid, measles, small-pox, diphtheria or malaria, endocarditis is a very
rare complication. Pneumonia is more often associated with infective
endocarditis; and the same is the case with erysipelas, with puerperal
and septic diseases generally, and with gonorrhoea.

(d) In cases of acute and chronic tuberculosis we meet with endocarditis
occasionally. Are we to look upon such cases as belonging to the infective
type of endocarditis, or do they belong to the benign form, the tubercle
bacillus acting as a remoter cause? It must be noted that in a few cases
the tubercle bacillus has been found (2) in the valve deposits; and in some
cases of acute miliary tuberculosis vegetations on the heart valves of
recent origin have been observed. I remember in a child, who died from
general acute tuberculosis, that the pericardium was found studded all
over with miliary tubercle; and the mitral valve showed deposits which
proved to be masses of fibrin and leucocytes, and contained tubercle
bacilli. In this, and probably in a good many other cases, the endocarditis
is of a specific nature, and therefore, as part of the general disease, belongs
to infective endocarditis; how far, however, this applies to all cases where
valve deposits are found in persons who have died of tuberculosis can
only be settled by a microscopic examination of these masses. Even if
the old view that phthisis and heart disease are antagonistic be not
strictly true, yet it is rare to meet with either acute rheumatic arthritis
or valvular affections of the heart in persons suffering from phthisis.

(e) Syphilis attacks the myocardium and the endocardium; in the
former it causes endo- and periarteritis with tracts of fibrous tissue in
the midst of the myocardium, or it may lead to granular deposits. In
the latter case valvular disease may result from arterio-sclerosis, of which
syphilis is one of the remoter causes; that acute endocarditis is ever due
to the syphilitic virus is very doubtful. Chronic endocarditis of syphilitic
nature does occur, but is a very rare occurrence (8).

(f) Of other causes of chronic endocarditis we may mention gout
and Bright's disease.

Gout.—Several cases are on record (6, 3, 9) in which endocarditic
processes showing the presence of urate of sodium crystals were found in
persons affected with gout.

In Bright's disease we often find chronic valvular heart affection from
arterio-sclerosis, yet occasionally it may be associated with acute endo-
carditis (15, 21, 7).

(g) Traumaism.—Several cases have been recorded, amongst others
by Clifford Allbutt and Litten, where all the signs of endocarditis
followed a blow or fall on the chest. Litten tabulated the recorded
cases and added two more. The endocarditis is most likely due to a
rupture or injury to the valve; how readily endocarditis is set up by
such an injury is proved by the experiments of Rosenbach and others, who, on injuring the valve in animals by introducing a fine wire through the carotid, noticed distinct endocarditis to follow after a few days. Roy and Adami, on lightly ligaturing the aorta, and thus increasing the blood-pressure, produced oedema of the valves and cell exudation.

That infective endocarditis may be occasioned by an injury I have already stated in my article in vol. i. p. 632. The cases cited by Allbutt and Litten were cases of benign endocarditis, some affecting the mitral valve and producing stenosis, and others the aortic valve; in some of the cases, however, the connection between the traumatism and the endocarditis was not satisfactorily established.

(b) Endocarditis without any apparent cause, and occurring as an idiopathic disease, has been described by some authors. Such an occurrence is quite possible, yet it must be extremely rare; and probably the endocarditis is in such cases the outcome of acute rheumatism, for, as often happens in children, the joint affection may be quite insignificant.

Other etiological factors relate chiefly to age. It appears that endocarditis occurs most frequently between the ages of 15 and 40; it is rare in old people, in whom valvular lesions are mostly due to an atheromatous process; it is not rare in children, as already observed by West, who noticed it 71 times in 122 cases of heart disease (see also on the subject Dr. Cheadle’s article in vol. ii. of this System, p. 42). In very young children, however, the affection is rare; in them pericarditis is more often found than endocarditis.

Fetal endocarditis is by no means a rare affection. It may occur with or without congenital anomalies of the heart. As is well known, the right side of the heart is generally affected in the fetus; but according to the observations of Rauchfuss stenosis of the aorta occurs as frequently as stenosis of the pulmonary artery; and he comes to the conclusion that when there is no congenital malformation of the heart, the left side is as frequently affected as the right. Apart, then, from the anomalies which predispose to right-sided endocarditis, other factors are in play which determine the frequency of right-sided endocarditis, as compared with its rarity in extra-uterine life. Such factors are the thickness of the right ventricle, the increased pressure to which it is exposed, and the absence of pulmonary respiration, which causes such a difference between the blood of the right and left sides of the heart after birth; especially as regards the amount of oxygen. Klebs, who was one of the first to attribute all forms of endocarditis to micro-organisms, gives another explanation; namely, the direct infection of the right side of the heart through the blood coming from the placenta. Recent observations have shown that micro-organisms do not readily pass through the placenta; but, if the micro-organisms do not pass, the toxic substances produced by them may do so, and thus give rise to inflammatory deposits. Fetal endocarditis cannot well be recognised before birth, and may be
undetected for years after birth. The fetal right-sided endocarditis affects principally the pulmonary valves—often when there is already obstruction or stricture; occasionally the tricuspid valve only: similarly, left-sided fetal endocarditis more frequently affects the aortic valve with or without contraction of the lumen of the aorta, and the mitral valve only occasionally.

Finally, endocarditis may be secondary, being an extension of an affection either of the myocardium or of the aorta.

Pathological anatomy.—Endocarditis affects principally the valves of the heart, hence the name valvulitis; and, except in the intra-uterine form, it is almost always confined to the valves of the left side: here again it affects the mitral more frequently than the aortic valve (the tricuspid valve, however, is occasionally also affected in combination with stenosis of the mitral valve). Of the mitral valve it affects the auricular surface, and here again principally the portions of the valve which are in close apposition when the valve closes; when it affects the aortic valve it is found on the ventricular surface round the corpora Arantii. That the left side is much more often affected than the right side is due to several causes, but principally to the higher blood-pressure and the difference in the oxygenation of the blood: the first factor leads more easily to abrasion of the endocardium, and other changes favouring the deposits of inflammatory material or thrombi, and by the latter the action of the microorganisms is greatly favoured. That the mitral valve is more frequently affected than the aortic may be due, as Sibson (loc. cit. p. 458) pointed out, to the fact that the mitral flaps press against each other when the valve is shut with much greater tension and force than the cusps of the aortic valve. To the combined agency of a finer margin of contact, greater pressure of blood, and the muscular force and tendinous traction proper to the valve, another fact may be added, namely, the absence of vessels in the aortic and pulmonary valves (Langer, Coen), a condition which protects them, at any rate, against the invasion of microbes by embolism. The endocarditis is localised chiefly at the part of the valves indicated, because at these spots we have the greatest pressure and stress.

It must be noted, however, that other parts of the endocardium, especially the chordae tendineae, are implicated in the process. An endocarditis affecting chiefly other portions of the endocardium, to the exclusion of the valve, has been described by Neuwerck; it is more or less chronic, and leads not only to superficial cicatrices, but also to sub-endocardial and myocardial inflammation (Rosenbach).

Appearance of the affected valves.—In the early stage, which we have but rarely the opportunity of seeing except perhaps in some case of fatal chorea, the endocardium at the affected parts is slightly swollen, and of the rosy tint of increased vascularity. At a more advanced stage we notice little pedunculated vegetations, forming a string or garland of small beads, on the auricular surface of the mitral and ventricular surfaces of the aortic valves; not on the free edges of the valves, but at some slight distance from the border, corresponding to the lines of the
maximum contact of the valve segments when the valve is closed. When the chordae tendineae are involved, the endocardium covering appears opaque, and slightly raised; and in rare cases may be the seat of small vegetations.

The further progress of these vegetations varies: in rare cases they may be completely absorbed; in other cases the vegetations increase in size and in extent, forming large fungating masses which may extend to the chordae tendineae, and cause a serious obstacle to the free circulation of the blood. In most cases the inflammatory deposits undergo fibrous change as in inflammations elsewhere; and these sometimes also affect the chordae tendineae, and even the papillary muscles, as seen in some of the chronic valvular affections; or some of the cusps may become adherent to each other or to the walls of the heart. The fibrous and contracted valve segments assume an almost cartilaginous appearance, and, being deficient in blood, may give rise to degeneration of the valves; notably to calcareous degeneration and the formation of necrotic ulcers.

Histologically the affection shows changes of an inflammatory nature in the endocardium, and deposit of fibrin in the form of thrombi, both white and mixed, from the blood. Sections of a small vegetation in a very early stage show proliferation of the endothelial layer, increase of the branched cells in the subendothelial tissue and infiltration of the layers of the endocardium with leucocytes, fibrin and serum between the trabeculae, and a deposit of fibrin on the free surface of the endothelium; this is deposited from the blood, and may be granular or sometimes fibrillar. When the process has lasted some time this embryonic tissue is changed into fully-formed fibrous tissue; and after a time calcareous particles may be deposited in the newly-formed structures, and the chordae tendineae and papillary muscles then show the changes to be described under chronic endocarditis. In the severe cases the myocardium shows indurative changes, which may be looked upon as due to an extension of the inflammatory process; the fibrous septa and the lymphatic spaces being chiefly involved: even the muscular fibres may show changes partly due to compression, and partly to mycarditis.

An examination for micro-organisms may reveal in some of the acute cases the presence of staphylococci, or streptococci, or diplococci (12); when the affection has become chronic microbes are rarely found. The organisms are found in larger numbers in the superficial layer of the deposits, and but sparingly in the deeper.

From acute endocarditis must be distinguished—

(a) Patches of red coloration of the valves, which are sometimes seen in persons who have died of an acute infectious disease. These patches are simply due to blood imbibition.

(b) Certain deposits on the auriculo-ventricular valves, which are the remains of an embryonic condition of the valves, as pointed out by Bernays; and in these Luschka has demonstrated pigment particles due to old hemorrhages (Rosenbach, loc. cit. p. 156).

(c) A form of endocarditis probably due to sudden disturbance of
intra-cardiac pressure, whereby the endocardium is injured. We have small, close, hard vegetations, firmly fixed and without adherent fibrin (Dickinson).

Symptoms.—Acute endocarditis is sometimes easy, at other times most difficult to recognise; and at times we find after death the characteristic lesions on the heart valves without there having been any symptoms of endocarditis during life.

The subjective symptoms vary considerably with the age of the patients, the primary diseases, and the presence of complications such as pericarditis, and the effects of any previous attack of endocarditis. The physical signs may sometimes be absent or appear only when the acute process has passed into a chronic state; and they are sometimes difficult to distinguish from those of a functional disturbance of the heart. In some cases the symptoms are absent, and it is only perhaps when the patient is brought under our notice in an attack of hemiplegia by embolism that endocarditis may be detected.

If we take the acute rheumatic as the most common form of endocarditis, we find in many cases no subjective symptoms to lead us to suspect an endocardial affection; the febrile symptoms, such as temperature, pulse, perspiration, do not differ from those in cases of rheumatic fever without endocarditis; and it is by the physical examination of the heart only that the existence of endocarditis is detected. In a second group of cases the patient, who has generally been affected with the rheumatic fever for a week or more, has suddenly a rise of temperature without any fresh pains; or he complains of oppression, uneasiness, or pain over the region of the heart and palpitation; the pulse becomes small and quick, and the heart’s action tumultuous: in other cases, of subacute course, dyspnœa on exertion is the only symptom complained of, yet physical examination of the chest reveals the existence of an endocardial murmur. In children, when pericarditis complicates endocarditis, which it frequently does (the carditis of Sturges), the symptoms are more pronounced and fairly characteristic; the breathing, with als nasi dilated, is hurried and laboured, and there is greater orthopnœa; the child has an anxious look and is somewhat cyanotic, sleep is very much disturbed, and there is generally marked delirium. The pulse in these cases is very quick, small and compressible, and there may be persistent vomiting. It must be noticed that in children the joint affection in rheumatic arthritis may be so slight as to be easily overlooked [see art. on “Acute Rheumatism in Children,” vol. iv. p. 40]. It may happen, as in a case I saw recently, that the only noteworthy feature is a rise of temperature with profuse sweating, which may go on for some time. The daily examination of the heart shows at first nothing abnormal, but in a short time the physical signs of endocarditis present themselves. In other cases in children, as in chorea, little general disturbance is noticed.

Physical signs.—The physical signs are sometimes very marked and admit of no other explanation; at other times they are indefinite.
On inspection of the thorax nothing abnormal is noticed, unless there be pericardial effusion; nor do we get any evidence of valvular disease on palpation unless the affection has already existed for some time.

On percussion it is only in exceptional cases that we notice the increase of the area of cardiac dulness due to dilatation of the left ventricle, the right, or of both. An increase in the area of dulness, however, more especially in the transverse diameter, is often noticed in children, and may be due to pericardial effusion; if so, the pulse is feeble, the apex beat is not so well felt as usual, and the area of dulness has the characteristic outline of pericardial effusion.

The most trustworthy and important physical signs of valvular endocarditis are noticed on auscultation. As the mitral valve is most frequently affected, and as the fibrinous deposit is apt to prevent the complete closure of the valve, we get the signs of mitral regurgitation; that is, a systolic murmur heard best at the apex, and conveyed towards the axilla and also towards the sternum. In a good many cases of acute rheumatic endocarditis, under my own observation, which afterwards lapsed into chronic valvular disease, a systolic murmur, soft and blowing in character, was noticed at the apex; but as a rule better heard over the lowest portion of the sternum close to its junction with the left costal cartilages. The appearance of a systolic murmur is preceded for days by an impurity and prolongation of the first heart sound which is in itself suggestive of endocarditis. Prolongation of the first sound is the first whisper of an "approaching murmur" (Sibson, loc. cit. p. 493). This is probably due to the soft gelatinous deposit, which alters the first sound while the valves are still smooth and elastic. According to Sibson, we may notice occasionally, besides the mitral bruit, a tricuspid systolic murmur also; but this is not heard at the very beginning of the endocarditis (Sibson, loc. cit. p. 242). As regards acute endocarditis in children, Sturges (25) gives as the earliest physical indication, "Tumultuous, quickened, and uneven heart's action and sounds, that are changeful from day to day, especially the first; sounds reduplicated, at and above the apex (not at the base); a temporary tricuspid murmur; marked accent commencing the first sound, whether mitral or tricuspid." Occasionally, however, even in children, a loud systolic murmur may rapidly appear; this is sometimes only heard when the child lies down; in the erect position it becomes fainter and may even disappear.

In acute febrile affections, such as the acute zymotic diseases, and in rheumatic arthritis, a systolic murmur may be heard under conditions other than that of endocarditis; therefore, when we hear such murmur we must not conclude at once that there is endocarditis. The murmur may be due to relaxation or other changes in the heart muscle, or to a change in the blood (hemic bruit).

Although it is not always easy or even possible to distinguish these conditions, certain signs will help us. The pulse in myocardial affections is often quick, small, and irregular; and there is marked dyspnea, and
vertigo. The heamic murmur is noticed when there is well-marked anæmia; it is heard not only over the mitral, but often also over the pulmonary and aortic areas, and is accompanied by venous murmur in the neck, while the pulse may be dicrotic. (According to Sansom, marked dicrotism occurs only in the severe cases of endocarditis.) Besides the mitral murmur, especially if the heart's muscle is weak and early dilatation of the right ventricle comes on, we may note reduplication of the second sound, accentuation of the second pulmonary sound, and sometimes also a systolic bruit over the tricuspid valve.

If the endocarditis affects the aortic valves we may find no special physical signs if the vegetations are very small; at other times we get evidence of aortic regurgitation, a diastolic bruit heard best at midsternum; and, if there be much regurgitation, we get other indications of aortic incompetence.

The physical signs denoting stenosis of either mitral or aortic valve are very rarely to be noticed; as the narrowing results from a contraction of the valves which generally takes place as the endocarditis becomes chronic. Dr. Sansom states that in some cases he has observed reduplication of one or other of the heart sounds as an early sign of endocarditis; and in these cases the endocarditis was followed by stenosis rather than by regurgitation (22).

In the rare cases of right-sided endocarditis we have the signs of tricuspid or pulmonary regurgitation.

Complications.—Leaving out of consideration the rare cases—which, however, mostly belong to infective endocarditis—where there is rupture of the inflamed valve or rupture of chordæ tendineæ, the most frequent complications of endocarditis are pericarditis and myocarditis. Sibson in 161 cases of acute endocarditis noticed that pericarditis was present in 34 cases, and in children the proportion is even larger. Changes in the myocardium varying very much in degree have, according to recent observations, been found so often that they too must be looked upon as common; though they do not often give rise to symptoms which lead to their recognition. Yet we may suspect their occurrence if the heart's action becomes weaker, or if there are physical signs of an acute dilatation of the heart; the pulse becomes quicker, weaker, and often irregular, the apex beat weaker, and the murmur less distinct; there is also marked dyspnoea, and the patient complains of tightness and oppression, and occasionally of pain and palpitation: these signs are often followed by vertigo, delirium, and cold, clammy perspiration. Cheyne-Stokes breathing has sometimes been noticed towards the end, and death takes place either from syncope or pulmonary congestion and oedema.

Pleurisy and pneumonia are occasional complications. The relation of pneumonia to infective endocarditis has already been noticed (vol. i. p. 633). Rheumatic endocarditis may, however, be associated with croupous pneumonia, or it may give rise to embolic pneumonia.

Embolie infarcts occur more frequently in infective endocarditis, and in chronic valvular diseases, than in the acute rheumatic endocarditis.
In some rare cases (11) the endocarditis propagated to the aorta may produce acute aortitis, a complication which is difficult to diagnose; it is said to produce severe paroxysmal pains behind the sternum with radiation towards the shoulder, dyspnoea, and perhaps diastolic aortic murmur.

Course and termination are most variable; in some cases the symptoms may disappear, and the patient completely recover; in others the patient apparently recovers, but for some time looks very anemic, and the physical signs never disappear. Or the patient may enjoy excellent health and be not aware that he has any valvular lesion till many years afterwards, when the first symptoms of want of compensation of the heart-defect make themselves felt; the length of time before these symptoms come on depends on many factors, such as the extent of the lesion, the condition of the heart muscle, the occupation of the patient, intercurrent diseases, and so on. In other but fortunately very rare cases, where the valvular lesion is very severe, or the myocardium very much enfeebled, the symptoms denoting failure of compensation (dyspnoea, quick, weak, or irregular pulse, oedema and dropical effusion) may come on early after the onset of the disease. When pericarditis complicates the endocarditis the patient may pass years without any serious troubles, till the compensation, whether of the valvular defect or of an adherent pericardium, begins to fail.

Death may take place during the acute stage from the presence of complications such as pericardial effusion, myocarditis, pneumonia, embolism, or in some rare cases from hyperpyrexia; or some infective agent may convert the rheumatic into infective endocarditis.

In children, in whom the physical signs are usually well pronounced, and pericarditis often present, all the signs may completely subside, and a restoration to complete health take place; in most cases, however, the child apparently recovers and may enjoy good health for many years in spite of the presence of signs of valvular disease; yet eventually, either without apparent cause or on the appearance of some incidental disease, he manifests the subjective and objective signs of valvular disease. In some few cases belonging to the group called active carditis by Sturges (loc. cit. p. 506), death takes place from the associated pericarditis or from pulmonary oedema, embolic pneumonia, or cerebral embolism. The cause of death in some cases of chorea with endocarditis is often very obscure, and not due directly to the endocarditis.

Diagnosis.—From what has been said of the symptoms of acute rheumatic endocarditis, it will be clear that the diagnosis, though easy in some cases, is occasionally impossible; in many cases, indeed, the endocarditis can only be suspected. When no murmur is heard over the precordial region we can only suspect endocarditis when, say in a case of acute rheumatism, the heart sounds become veiled and impure (such changes are best noticed when the heart has been daily examined), and the patient complains of palpitation or oppression, as pointed out above. When a murmur is heard over the region of the heart we have to distinguish
between an exocardial and endocardial murmur, and if endocardial whether it is due to endocarditis.

The exocardial murmur, which is occasionally difficult to discriminate, especially in children, is a pericardial friction sound; but the character, the rhythm, the situation, the variability of the murmur, the direction in which it is propagated, and some other points will help us to distinguish pericarditis from endocarditis. Thus in pericarditis a double murmur is heard which does not replace the heart sounds, but only obscures them; nor is the double murmur synchronous with them; the murmur may have the character of a hard or soft friction sound; it is heard usually over the right ventricle, though it may be audible with less intensity near the apex; it appears to be superficial, is localised over a small area, is not propagated either to the axilla or along the sternum, and it is variable within short periods of time. Occasionally the rub may be felt when the hand is placed over the precordial region. If there be much effusion the precordial region may bulge; the area of cardiac dulness increases, and has a peculiar pear-shaped outline; the apex beat is raised, displaced towards the left and indistinctly felt; and, on auscultation, tubular breathing may be audible over a small area of the back. In pericarditis pain and oppression are often noticed.

An exocardial murmur may be due to pleurisy. As a rule there is no difficulty in distinguishing the pleuritic rub from pericardial friction and from an endocardial murmur, since when the patient is asked to hold his breath it disappears; but occasionally the pleuritic rubs, though lessened, persist and are rhythmic with the heart's contraction: as a rule, however, the rub extends towards the left beyond the limits of the heart, and there is often pleuritic pain.

Another exocardial murmur may sometimes be heard above the apex beat towards the left; it is rough, varies in intensity, begins after the systole, and disappears when the patient sits up and bends forward. According to Potain, this murmur is due to the intrusion of a thin layer of the lung, close to the heart, into the space before occupied by the base of the heart, as with each ventricular contraction the apex is projected forwards and the base retreats slightly from the chest wall.

Having eliminated the exocardial murmurs, we have yet to determine whether the murmur be due to endocarditis (so-called organic murmur) or functional; and, if due to endocarditis, whether recent or old, benign, or infective. The chief points of distinction between the hæmical murmur and the murmur produced by the dilatation of the heart, and by endocarditis, have been given above. As a rule there is no difficulty in distinguishing recent from old endocarditis; we have to take into account the history of the case—whether there have been previous attacks of rheumatism or chorea, or of some of the other diseases followed sometimes by endocarditis; or whether the patient has suffered from dyspnoea on exertion or oedema of the feet. The presence of secondary changes in the heart due to chronic valvular disease, such as hypertrophy of the left ventricle
or dilatation of the right ventricle, indicated by accentuation of the second pulmonary sound with signs of venous stagnation (edema, enlarged liver, albuminuria), are of great help; but we have to bear in mind that dilatation of the right heart may come on occasionally in acute endocarditis, and that a previous attack of rheumatic endocarditis favours the recurrence of such attacks, should the patient suffer again from acute rheumatism; thus we may have an acute endocarditis implanted on an old one.

The discrimination of rheumatic or benign from malignant endocarditis will be considered when treating of the latter disease.

Prognosis in acute endocarditis is sufficiently evident from what has been stated concerning the course and termination of the disease. Death during the acute stage is generally due either to the severity of the primary disease—be this rheumatism, chorea, or an infective fever—or to some complication, such as myocarditis, pericarditis, or pneumonia; in some rare cases symptoms of dilatation of the right side, with venous stasis, shown by dyspnea, dropsy, irregularity of the heart’s action, may come on and lead to death. A very large majority of patients recover from the acute attack, remain well for years, but become the subjects of chronic valvular disease; and this may occur in cases in which the murmur had disappeared for a time; lastly, in some few instances complete and permanent recovery takes place. When an acute endocarditis occurs in persons already affected with valvular disease the prognosis is still more serious; for often the fresh endocarditis is of the infective or malignant kind; or, even without this, the fresh deposit may lead to embolism or, by increasing the weakness of the heart, hasten the downward course of disease.

Treatment.—Prophylactic treatment.—As acute endocarditis is associated most frequently with acute rheumatism, our attention must be directed to prevent the occurrence of this malady in persons with a family or personal proclivity to the disease; such persons should wear flannel next to the skin, avoid living in damp houses and in districts where clay forms the subsoil and rheumatism abounds, and avoid as much as possible those sudden changes of temperature which are so apt to produce chills.

If a person is taken with acute rheumatism, can we by speedy and proper treatment prevent the occurrence of endocarditis? This question has been the subject of many discussions, especially since the introduction of the salicylates, which have such a decidedly beneficial effect in acute rheumatism, often causing a speedy disappearance of the symptoms, and cutting short the duration of the disease. There is, however, now abundant evidence that the cardiac affections are not warded off by this treatment [see discussion in the Medical Society of London, 1881]; on the other hand, we cannot say that their frequency has been increased by this now universal treatment of rheumatism. Some maintain that the treatment of acute rheumatism with large doses of alkali, combined with absolute rest in bed, has a more protective effect against the cardiac
complications than the salicylates or salicin; and many now use this combined treatment. So far the prophylactic treatment has had but little success; yet it is most important that every case of acute rheumatism in adults, and still more the various modified and less pronounced forms in children [see Dr. Cheadle’s article on “Acute Rheumatism in Children,” vol. iii. p. 52], should be treated at once by rest in bed, with complete repose and appropriate medicine (alkalies and salicylates).

Local treatment.—Venesection, recommended by Bouillaud and his school, and extensively practised for years, need only be mentioned as of historical interest. The application of a few leeches to the precordial region, especially in young and plethoric subjects with a quick and full pulse, and when there is precordial pain and oppression, indicative of early pericarditis, may be safely recommended for the relief of these symptoms. The local application of ice, long since recommended by Friedreich, and extensively practised on the Continent, has been warmly advocated by Dr. D. B. Lees, especially when pericarditis complicates endocarditis; it may be tried also in simple endocarditis: it reduces the fever, diminishes the frequency of the pulse, calms the action of the heart, and relieves such subjective symptoms as pain and oppression. It is well to apply flannel next to the skin and the ice-bladder over the flannel; for dry cold is much better borne than wet cold. "As a rule it is quite tolerable, and indeed comforts the patient. It is contra-indicated when there is marked cardiac dilatation with a small and intermittent pulse; but even when these conditions obtain, it may be cautiously tried for a short time.

Other local remedies used are blisters, sinapisms, and tincture of iodine. Large blisters have often been recommended as derivatives, and recently Dr. Caton has spoken favourably of repeated small blisters. I have often applied blisters both in endocarditis and in endo-pericarditis, and with relief of some of the subjective symptoms; but I cannot say that they have influenced the disease very much. Painting the precordium with tincture of iodine, repeating this from time to time, and persisting with this for weeks or months, is asserted by some observers to be attended with good results.

General treatment.—With the appearance of the first symptoms of endocarditis some physicians recommend the administration of larger doses of alkalies and suspension of the salicylates, which have a depressing effect on the heart; others see no objection to a continuance of the salicylates, unless signs of failure of the heart or of myocarditis appear; others again prefer to give salicin, which has a much less depressing effect. As, however, with this treatment the endocarditis, when once it has shown itself, is rarely completely cured, I have tried from time to time both local and general means to check, if possible, the inflammation of the endocardium and to minimise the damage done by it.

Tartar emetic, as recommended by Jaccoud, is scarcely ever employed by English physicians; nor do many of us give mercurial preparations which were once so highly spoken of both by Graves and by Stokes,
except in obedience to special indications. Iodide of potassium has been given at a later stage of endocarditis to hasten the absorption of the deposits.

As essential as the medicinal treatment is the general management and diet of the patient. The patient must be confined to bed for weeks, kept quiet, and all excitement avoided; the diet should be light, but nutritious, and, unless the heart show signs of failure, stimulants had better be avoided altogether.

Other drugs than those given above may be indicated by certain symptoms and under certain conditions. If there be much pain and restlessness small doses of morphine may safely be given. Antipyretics are only indicated when the temperature is high and the pulse very quick. Quinine in fairly large doses (15-20 grains), or phenacetin (gr. 5 to gr. 10), are preferable to antipyrin or sodium salicylate. Digitalis is not required unless the pulse becomes quick and small, or irregular; the tincture of digitalis or digitalin may be given when signs of cardiac failure appear. Besides this drug we may give strychnine, ammonia, brandy, and ether under the above conditions. When there is much dyspnoea and cyanosis inhalations of oxygen will be found useful, especially in children. In cases in which the pulse is quick but full, and in which the heart’s action is good, digitalis had better be avoided, as an increase in the force of the heart might lead to a detachment of clots or parts of the vegetations, and thus to embolism.

For the anaemia, which often persists for weeks and months after the acute symptoms have passed off, preparations of iron are given with quinine and arsenic; the latter drug appears, indeed, to have a better effect than the iron preparations. Convalescents from acute endocarditis should be sent for some weeks into the country or to the seaside; a dry, bracing climate being preferred. If there be much subsequent dilatation the Nauheim treatment may be tried, and the other measures recommended for chronic valvular disease [see later articles].

Those cases of rheumatic endocarditis which assume a malignant type, which run a long and protracted course, and in which fever persists, rigors and hemorrhages appear, and further complications (septic pneumonia, embolic abscesses) arise, require the same treatment as cases of infective endocarditis, to which class indeed they belong.

REFERENCES

II. INFECTIVE ENDOCARDITIS

SYN.—Malignant Ulcerative Endocarditis

I have already (vol. i. p. 626) considered the causation and patho-
geny of this form of endocarditis; it remains now to discuss the patho-
logical anatomy, symptoms, prognosis, and treatment.

Pathological anatomy.—Lesions are found in the heart and in
various other organs of the body. Some are primary, and represent the
seat of inoculation; others are secondary, but the most important of
these are produced by the micro-organisms circulating in the blood. The
changes found in the heart vary considerably according to the microbes
which produce the disease, the extent of it, its duration, and especially
whether the infective endocarditis affect a healthy heart, or one already
the seat of old endocarditis or sclerosis of the valves. We may have simple
vegetations, or—and this is the most frequent occurrence—we find a more
or less extensive ulceration of necrotic character; or, occasionally again,
the formation of one or more abscesses. Malignant endocarditis, like
simple or rheumatic endocarditis, principally affects the valves; and much
more frequently the valves of the left side than those of the right,
though the latter are more liable to be affected than in rheumatic endoc-
arditis: thence it may extend to other portions of the endocardium, and
to the aorta or pulmonary artery. Mural infective endocarditis, in which
the valves remain free, is extremely rare.

When there are only vegetations these are generally small, and
grayish or yellowish in colour; they affect the base as well as the margins
of the valve. Such a condition we sometimes see in cases which run a very rapid course; histological examination of the valve reveals
numerous microbes, embryonic cells, and leucocytes; besides the layers of
fibrin. In most cases, however, the vegetations are larger, occasionally
pedunculated, and more or less extensively ulcerated; these may be
superficial, not extending deeply into the tissues; they are grayish,
and often partly covered with fine blood coagula; if the valve yield to
the blood-pressure, depressions (aneurysms) may result. Sometimes the
ulceration may penetrate deeply into the valve, and perforate it; often
the inflammation spreads to the chordae tendineae, and is followed by
further ulceration, so as to cause a detachment of the valve segment;
the valve is thus rendered incompetent, and with every cardiac contrac-
tion the loose segment, flapping against a part of the auricle, sets up fresh inflammation there, and gives rise to the formation of warty growths on its walls. The loss of substance caused by ulceration extending to parts of the endocardium (chordae tendineae, septum) may lead to an aneurysmal bulging of the wall, or even to rupture of the septum or of the heart.

If pyrogenetic organisms be the immediate cause of the ulcerative endocarditis small abscesses may form in the tissues of the valves; occasionally one or more larger abscesses are found in other parts of the heart, extending deep into the myocardium; and these again may lead to an aneurysm of the heart, or to rupture into the pericardium. In rare cases the pus may be reabsorbed, and leave scars or calcareous residues (1).

In the more chronic, but sometimes also in the acute cases, we may find deposits of lime salts on the vegetations; but, as a rule, we find these calcareous incrustations in cases in which ulcerative endocarditis has attacked a person already suffering from valvular disease. It is not always easy to distinguish these ulcerations from the atheromatous ulcers due to simple necrosis in a valve with calcareous deposits, the result of either chronic endocarditis or atheroma; in most cases a bacteriological examination will help us to distinguish the two, but not always.

The frequency with which the various valves are affected is shown by the following analysis from the post-mortem records of the Manchester Royal Infirmary (2) for 1891-1895; 20 cases are noted: 7 men and 13 women; average age, 34½; youngest 15, oldest 57. In 15 cases out of 20 previous cardiac disease was noticed: the right side was involved in 1 case only; herein there were vegetations on the tricuspid as well as on the mitral valve: in 7 the mitral alone was affected: in 8, the aortic valve alone. The spleen was found enlarged in 17 cases.

From 1895 to 1897 25 cases were noted: 14 men, 7 women, 3 boys, and one girl; average age, 32; oldest 72, youngest 9. In 20 out of the 25 previous cardiac disease was noticed: in one the right side (pulmonary artery) was affected alone; in 3 both right and left sides; in 6 the mitral valves only; in 3 the aortic only; in 6 both mitral and aortic valves were involved. Splenic enlargement was found in 19 cases (3).

Kanthack and Tickell analysed 84 cases occurring between 1890 and 1897; of these, 51 were males, 33 females; and in all but 16 cases old cardiac lesions were found.

The changes noted in other organs vary considerably, and may be grouped as follows:

(i.) Primary; such as crepusc pneumonia, pleurisy, empyema, meningitis, primary septic foci in the uterus or its adnexa, gonorrhea, a primary abscess in pyæmia, osteomyelitis, disease of the middle ear, tonsillitis, gastric ulcer, appendicitis, gallstone, etc. (see vol. i. of this work, p. 631).
It must be noted, however, that septic pneumonia may be secondary to infective endocarditis.

(ii.) Lesions due to embolism. These vary as the embolus acts simply mechanically or has infective properties: in the first case we meet with infarcts chiefly in the spleen and the kidney, and in the brain; in the brain the area of the blocked artery softens, in peripheral arteries the embolus may lead to gangrene. In the second case we meet with metastatic abscesses, which may occur either in small or in very large numbers; and are found in the liver, the spleen, the kidney, the lungs (especially if there be right-sided endocarditis). In the intestines, or even in the stomach, haemorrhagic infarcts are found, sometimes of a septic nature infested with numerous micro-organisms, and occasionally ulcerations of the mucous surface; at other times corresponding to simple infarcts, the intestines present intense congestion, haemorrhage, and even gangrene.

Small capillary emboli are no doubt the cause of the haemorrhages noted in the skin and subcutaneous tissue, the serous surfaces, the retina, and other parts.

3. Lesions which are common to most infectious fevers, and which may be due to micro-organisms, their toxins, or to the accompanying pyrexia. Enlargement of the spleen, so-called "cloudy degeneration" of the liver and kidney, and nephritis (in which the kidney is large, pale, and shows small haemorrhages), are included in this group.

Symptoms.—The symptoms of infective endocarditis vary considerably in individual cases: the heart symptoms may be quite insignificant or even absent; as, for example, when acute infective endocarditis complicates a septic disease, as pneumonia, empyema, or meningitis, in which often only the autopsy reveals the endocardial lesions. In other cases the heart symptoms are more pronounced; this is more particularly the case in the subacute or even chronic form which complicates rheumatic endocarditis.

Owing to the great diversity of the symptoms certain types of infective endocarditis have been formulated. We may distinguish in the first place between an acute form and a subacute or chronic form.

The acute form includes the septic type, the typhoid type, and the cerebral type; the chronic form is noticed in old valvular affections of the heart; by some it is called the cardiac type, or, owing to the peculiar fever curve which is noticed, it has been named the intermittent febrile or malarial type. We will briefly consider the principal features of these various types, and then note the symptoms in detail.

(a) The septic or pyemic type, which is noticed in puerperal cases and in other forms of septicemia and pyæmia, includes all the symptoms of severe septic infection. The onset is acute; with or without preceding general malaise the disease is ushered in by more or less severe rigors, followed by heat and sweating, which may be repeated after a shorter or longer interval; between the rigors the temperature generally remains high, it may, however, be remittent; the skin may show patches
of erythema, hemorrhage, or superficial collections of pus; the pulse is quick and feeble; the respiration is hurried and superficial; nervous symptoms, such as headache, delirium, somnolence, are usually present; at times symptoms of cerebral embolism may appear; the tongue is usually furred, and may become dry and brown; there may be great thirst, anorexia, and vomiting; there is often a good deal of tympanites and diarrhoea. Metastatic abscesses may form in various organs and tissues, but often do not give rise to definite symptoms, as, for example, in the lungs.

The examination of the heart may reveal either no abnormal signs, or audible murmurs; from their presence alone we may not conclude that we have to do with infective endocarditis, for such murmurs are not uncommon in simple cases of pyæmia and septicæmia, without any ulceration of the valves of the heart. Of other symptoms common in ordinary pyæmia I may mention albuminuria, jaundice, and pain and swelling of the joints with suppuration. Death generally takes place within one or two weeks.

(b) In the typhoid type infective endocarditis resembles enteric fever as regards the general aspect of the patient, the condition of the tongue, which is brown, dry, and furred, the presence of diarrhoea and cerebral symptoms; but we do not infrequently see rigors, petechiae, and optic neuritis—symptoms which are very rare in enteric fever: the heart symptoms in this form again may be absent, or indefinite. The temperature is generally very irregular; rigors may occur throughout the whole duration of the disease, followed by profuse sweating; and attacks of embolism in the brain, kidney, and spleen are not uncommon. The duration of the disease, when assuming this form, varies from two to three weeks; sometimes it lasts longer.

(c) Cerebral type.—This type is chiefly abstracted from cases of malignant endocarditis complicated with meningitis, either cerebral or cerebro-spinal. The affection begins in these cases with cerebral symptoms—headache, somnolence going on to unconsciousness and coma, or delirium and convulsions. The heart symptoms are less pronounced and often absent. Rigors are not often present, but attacks of embolism may occur and direct attention to the heart.

(d) Cardiac or Malarial type.—This represents by far the largest number of cases; it occurs in persons in whom the heart has already been damaged by previous disease. It runs, as a rule, a subacute and chronic course, and may last six months, or even more than a year. Though recovery is extremely rare, this variety is not always fatal.

The onset of the disease is generally insidious; the patient complains of general malaise, and has an anæmic appearance. Sometimes an increase of body temperature, with but few other symptoms, may be the first sign of it, as in a case under the care of my colleague Dr. Steell: a young man suffering from an old valvular affection of the heart, whilst in the hospital suddenly showed a rise in temperature, and after a few days manifested characteristic signs (rigors and so forth) of infective endo-
carditis. At other times the affection resembles rheumatic arthritis, pains in the joints and slight pyrexia being prominent features. After these symptoms have lasted a few days, rigors appear, followed by heat and sweating. During rigor the temperature may reach 104° F. or more, and a few hours later the temperature may come down to normal. The rigors occur at irregular intervals; two or three may occur in one day; at other times several days or weeks may elapse before a second rigor is observed. In a good many cases the rigor is replaced by a mere sense of chilliness followed by sweatings; in others, again, a remittent or intermittent pyrexia, going on for weeks or months without any rigors, is a prominent feature. Thus, in one case, which I saw with Dr. Renuad—a girl, aged 20, who at the age of 16 had had an attack of rheumatic fever from which she recovered, but which left her affected with mitral disease—the only noticeable feature was an intermittent pyrexia—the morning temperature being 98°, the evening temperature 99° to 100°; beyond this no other symptom was noticed, and the patient felt no further inconvenience. This state persisted for over six months, when she had an attack of cerebral embolism. From this she had partially recovered when a second and fatal attack of embolism supervened. At the necropsy new deposits were found upon an old affection of the mitral valve, and the vegetations showed the presence of numerous streptococci.

A remarkable feature in the cardiac type of infective endocarditis is the occurrence of embolism. This occasionally affects peripheral arteries (posterior tibial, brachial, popliteal, and even abdominal-aorta), but more often the left middle cerebral artery, or one of its branches, especially the Sylvian artery. The blocking of the cerebral vessels may only produce temporary paralysis or aphasia; but often these attacks are followed by others which leave a permanent lesion, and most frequently lead to complete hemiplegia.

Some of the viscera also may be the seat of emboli; thus splenic infarcts are not uncommon, which may give rise to no symptoms: but occasionally certain symptoms enable us to diagnose the infarction; namely, sudden pain in the region of the spleen, with enlargement of the organ, and occasionally a friction sound over the spleen. It must not be forgotten, however, that, without the presence of an infarct, the spleen is often considerably enlarged in infective endocarditis.

Quite as common are renal infarcts, which only give rise to symptoms when the infarct is large; in such a case sudden pain is felt in the region of the kidney, and hematuria and remittent pyrexia appear.

Infarcts of the lungs can be inferred if the patient have a sudden pain in the chest, with dyspnoea, followed by the expectoration of sanguinolent sputum. On physical examination, if the infarct be large, we notice over a small area dulness on percussion, increased vocal fremitus, bronchial or tubular breathing, and fine crepitations; the temperature also rises and assumes a remittent character. Pulmonary infarcts frequently lead to embolic pneumonia, and often also set up localised pleurisy. If the
endocarditis be situated on the right side of the heart we occasionally meet with multiple metastatic abscesses in the lungs, which give rise to no definite symptoms.

*Emboli sm of the mesenteric artery* — a rare occurrence — may give rise to severe abdominal pain, with haemorrhage from the bowels and grave general disturbance leading to collapse.

Other symptoms often noticed are pronounced anaemia, which may be present from the beginning: examination of blood shows the red blood corpuscles to be diminished; the leucocytes are often increased, and a few eosinophile cells may be detected. Bacteriological examination of the blood reveals the presence of micro-organisms, notably streptococci.

*Petechiae and haemorrhage* from the mucous membranes are occasionally noted, the latter more particularly when the aortic valves are affected.

*Pains* in the joints are often complained of; in many cases the joint is neither swollen nor reddened, and the affection is probably of a toxic nature; at other times we meet with a definite arthritis, or again, with suppuration of the joint.

*Haemorrhages in the retina* and *optic neuritis*, according to some observers, are of common occurrence. I have seen haemorrhage more frequently than optic neuritis.

*Enlargement of the spleen* is very often noticed, and may reach a considerable degree, so that the spleen can readily be felt; it is not a constant symptom, however, and in some cases the spleen, as shown by the necropsy, is even smaller than in the normal state.

The *liver* is sometimes found enlarged, and jaundice may be present. In rare cases the liver appears diminished, and the case may simulate acute yellow atrophy. The occurrence of infective endocarditis in persons suffering from gall-stones has already been alluded to when speaking of the pathology of the disease (vol. i. p. 631); this may occur in persons who have not had rheumatic endocarditis.

The *urine* often shows traces of albumin and blood, and the presence of casts, both epithelial and granular.

The *bowels* are often constipated; occasionally we meet with profuse diarrhoea, and sometimes (see above) with haemorrhage from the bowel.

The ordinary *complications* are pneumonia, pleurisy, peritarditis, aneurysm, cerebral haemorrhage; this last was noticed in two cases which occurred in the Manchester Infirmary; an embolus was carefully searched for, but with negative results.

The symptoms which relate to the heart are well pronounced in the cardiac form, and we meet with the signs of mitral or aortic disease, or of both; in rare cases we have evidence of an affection of the valves of the right side of the heart. There is nothing in the character of the bruits or, in the size of the heart to enable us to diagnose infective rather than benign endocarditis; during the course of the disease the murmur may undergo some change, but this may also occur in rheumatic endocarditis. The presence of right-sided valvulitis is of greater diagnostic value, as it is of
very rare occurrence in the rheumatic or benign endocarditis. Some authors lay stress on the loudness of the murmurs, on their peculiar (metallic) character, and on the propagation of the mitral murmur to the axilla and angle of scapula; but these signs are also noticed in the benign form of endocarditis. Subjective symptoms, such as palpitation, pain over the region of the heart, excessive dyspnoea, have no diagnostic value.

As already stated, the cardiac form of infective endocarditis almost always runs a chronic course; occasionally it may occur in an acute form. When treating of the pathology, I mentioned one instance in which, previous to the occurrence of infective endocarditis, there probably had been a ruptured aortic valve. Recently I saw, with Mr. Coutts of Blackley, a case of infective endocarditis in a compositor, aged 50, who had always enjoyed good health, and who had never been troubled with rheumatism; he was suddenly seized with a rigor while at his work; he was brought home, and his wife, who had been a nurse, took his temperature and found it 103°; in the course of a few hours the temperature was again normal, and the patient felt quite well. The morning after, he had another rigor and rise of temperature; and in the evening he had still another rigor. When I examined the patient soon after, I found the temperature normal, and the patient complaining only of some oppression; the heart's action was somewhat tumultuous, and the arteries beating rather forcibly; over the aorta a faint systolic bruit was audible. The spleen was enlarged. The patient had been taking quinine, and now some arsenic was added to this; the rigors, however, continued for two days, when the patient suddenly died. I looked upon this case as one of idiopathic acute infective endocarditis.

The above types by no means represent all the clinical forms of infective endocarditis. Thus it is found in association with pneumonia, in which case there is very often no special symptom to lead one to suspect its presence. It may occur with gonorrhoea, in which cases the heart symptoms are often pronounced, whilst septic symptoms are less obvious; and, lastly, we meet with cases in which the distinction between rheumatic and infective endocarditis is impossible.

Diagnosis.—In spite of our improved clinical methods, and the application of bacteriology to clinical medicine, the diagnosis of infective endocarditis is still often a matter of difficulty.

Enteric fever may be distinguished from infective endocarditis by the mode of onset, the temperature curve, the roseolar spots, tympanites, and so forth. [See “Enteric Fever,” vol. i. p. 836.] Repeated rigors are rare in enteric fever, and cardiac murmurs seldom appear at the beginning of it. In doubtful cases Widal’s serum test may be a useful help; if, after the sixth day of illness, this test give negative results, enteric fever may with great probability be excluded; on the other hand, repeated rigors, and especially the occurrence of attacks of embolism, speak most strongly for infective endocarditis.

From septic and pyæmic infection, unless heart symptoms are pro-
nounced and signs of embolism are present, the disease is not easily distinguished. This will be easily understood, for infective endocarditis is indeed nothing more or less than a septic disease with the special localisation of the micro-organism in the heart valves. Bacteriological examination of the blood (see below) commonly shows us the presence of septic micro-organisms; and the same observation applies to the meningeal or cerebral form. It is only in cases of tuberculous cerebro-spinal meningitis that the withdrawal of fluid by means of puncture of the spinal membrane in the lumbar region—which would show the presence of tubercle bacilli—can be of any diagnostic value; as the same organism that is found in non-tuberculous meningitis, be it suppurative or cerebro-spinal, has been found in infective endocarditis.

In the cardiac form, when the heart symptoms are well pronounced, several signs help us to distinguish between the rheumatic (or benign) and the malignant endocarditis. These are:—

(i.) The presence of pyrexia.—This is often one of the first symptoms, and may show the remittent or intermittent type; should the pyrexia be accompanied by rigors occurring at irregular intervals and not affected by either quinine or arsenic, the diagnosis may be looked upon as almost certain.

(ii.) The anaemic appearance of the patient.—Anaemia often follows the first attack of rheumatic endocarditis; but the persistence of anaemia for a long time, or the occurrence of anaemia long after the attack, should certainly make us suspect malignant endocarditis.

(iii.) Enlargement of the spleen has already been discussed on p. 881.

(iv.) Changes in the retina, whether in the form of optic neuritis or of small hæmorrhages, when occurring in persons suffering from endocarditis, are indicative of the infective form; and it is well to examine the eye in all cases of endocarditis.

(v) Hæmorrhages in the skin and from the mucous membranes.—Epiptaxis is a common symptom in rheumatic endocarditis when the aortic valves are affected; and hæmoptysis is frequently noticed early in mitral disease, and at a later stage in other heart affections. Hæmorrhages into the skin and subcutaneous tissue, on the other hand, due probably to numerous small capillary emboli, are indicative of infective endocarditis. Of hæmaturia from renal infarcts and of melena from infarcts of the mesenteric arteries I have already spoken; but in themselves, and without other signs of infective endocarditis, these hæmorrhages are of no diagnostic value, as they may be the result of the chronic venous congestion secondary to chronic endocarditis.

(vi.) Bacteriological examination of the blood.—Many are the observations on this subject, and various the methods which have been devised to obtain sufficient blood for the culture of micro-organisms. Rettruschky uses the blood obtained by cupping. Lithmann withdraws about 5 c.c. of blood directly from a vein of the arm by means of a sterilised syringe. A portion of this is mixed with agar-agar which has been previously liquefied in a water-bath at a temperature of 40° C., and
the mixture is poured out into Petri's capsules to secure cultivations of the micro-organisms present. In the acute septic cases numerous cultures of streptococci and other cocci are found; in the chronic cases, though the case may be one of infective endocarditis, this method does not always show the presence of micro-organisms. Of three chronic cases of infective endocarditis H. Cohn found a few colonies of staphylococci in one only. In several cases of chronic infective endocarditis under my own care, in which the diagnosis was verified by the autopsy, some venous blood was aspirated after the method of Lithmann, and examined bacteriologically by my colleague Dr. Delépine, but with negative results.

Prognosis.—The prognosis of this disease is in all cases very grave. The acute form, be it of the pyemic, typhoid, or meningeal type, is almost invariably fatal, death taking place sometimes within a few days. Eberth gives the case of a man who began with typhoid symptoms, soon followed by coma and hyperpyrexia; the case ended fatally the next day. The aortic valves showed ulcerations, and a metastatic abscess was found in the brain. In other cases the symptoms may go on for several weeks. The chronic or cardiac form may last for months and occasionally over a year; yet a fatal termination either by exhaustion, embolism, or complications is the rule: several recoveries of undoubted cases have, however, been recorded. When speaking of the pathogeny (vol. i. p. 632), I mentioned a case in which malignant endocarditis occurred after an injury, and in which the patient recovered with a damaged aortic valve, and is at the present time in a satisfactory state of health. Another patient, whom I saw with Dr. Hassall of Northwich, with all the signs of infective endocarditis implanted on a diseased aortic valve, recovered.

Treatment.—Many are the drugs that have been recommended in infective endocarditis. Apart from the general treatment with tonics, stimulants, and rest, the same drugs as are given in rheumatic endocarditis—such as the alkalies and salicylates, antipyrin, phenacetin, and so forth—have been recommended, but the results have not been encouraging. Large doses of quinine appear more useful, though the quinine does not prevent the occurrence of the rigors, even in large doses. Fraenkel recommends large doses of quinine with arsenic, and I have, for some years given this combination; yet, except in the two cases quoted above, and in a third case in which the symptoms of endocarditis occurred after an attack of gonorrhœæ, and in which there was also a peri-urethral abscess, the fatal termination was not averted.

Benzoate of sodium, recommended by Kleber and others, has not given any good results in my hands.

Sulpho-carbulate of soda (half-drachm doses) is recommended by Dr. Sansom, and records one case in which, when death took place at a later period, distinct cicatrical tissue was found at the site of the old ulcerations.

The subcutaneous administration of antistreptococcus serum has been recently recommended; judging from the successful cases published by
Sainsbury and by Pearse, this treatment deserves a trial. Sir Douglas Powell has tried in five cases subcutaneous injection of yeast, but without any marked result; and in one case nuclein was used, which caused a temporary fall of the temperature.

J. DRESCHFELD.

REFERENCES


For further references the reader is referred to the article on this disease in vol. i.

J. D.

DISEASES OF THE MYOCARDIUM

As with other muscular organs, the heart is liable to fatigue, to overstrain, to disturbed innervation, to impaired nutrition; either, in the first place, from defect in the nutritive qualities of the blood with which it is supplied, or, in the second place, from temporary or permanent restriction in that supply through temporary or permanent alteration of the vessels. Further, the heart muscle may undergo degenerative changes, or may atrophy and be replaced by fibrous tissue; and this degeneration or atrophy and fibrous replacement may be general or localised. Yet, again, the heart muscle may undergo physiological hypertrophy in obedience to the demands of excessive labour, and this condition, although not one of disease, has to be reckoned with, since it leads to textural changes; finally, the heart may be invaded or occupied by growths, parasitic or other, of various kinds. With the various diseases of the endocardium, pericardium, and valves of the heart I have here no immediate concern, although I shall have to refer to them incidentally in an endeavour to give a clear account of myocardial lesions.

The several lesions of the myocardium above mentioned will be found to group themselves naturally under the pathological headings of—

I. Impairment secondary to general blood conditions.—(A) Anæmia;
(B) Toxic changes.

II. Impairment secondary to altered blood-supply.—(A) From paroxysmal affections of coronary arteries; (B) from permanent changes in coronary arteries; (i.) Atheroma; (a) fatty degeneration; (b) fatty infiltration; (c) fibroid infiltration; (d) aneurysm of the heart; (ii.) Thrombosis or embolism; (iii.) Aneurysm.
III. Impairment due to senile changes.—(a) Pigmentary degeneration; (b) atrophy.

IV. Impairment arising from functional strain.—(a) Hypertrophy; (b) acute dilatation; (c) textural damage.

V. Impairment of inflammatory origin—Myocarditis.—(a) Interstitial; (b) parenchymatous; (c) purulent; (d) syphilitic.

VI. Growths.—(a) Sarcoma; (b) myxoma; (c) fibroma; (d) gumma; (e) carcinoma; (f) lipoma; (g) cyst; (h) myoma; (i) tuberculosis.

VII. Parasites.—(a) Hydatid; (b) cysticercus cellulose; (c) actinomycosis; (d) trichina spiralis.

I. Impairment secondary to general blood conditions.—A. Anæmia.—Pathology.—In cases of marked anæmia, as in chlorosis, the nutrition of the heart muscle suffers; the organ is paler than natural, somewhat glistening and wet-looking on section, and gives less than the normal resistance to the pressure of the finger. On microscopic examination in persons who have died from some intercurrent malady no change may be noticed; but most commonly the fibres have undergone a certain degree of fatty change, and present a few refracting granules. In some cases of extreme anæmia, however, a very notable degree of fatty change may be found in the muscular fibres; the internal surface of the organ, especially over the left ventricle and papillary muscles, presents a streaked or flecked appearance, due to groups of small opacities seen through the transparent intima, the degeneration affecting the muscular fibres having a patchy distribution.

Although the fatty heart is always somewhat increased in size, it may not be increased in weight; the specific gravity of muscles being reduced by fatty change. The pericardium and endocardium usually escape change, but the cavities of the heart are enlarged, especially the left ventricle; and slight incompetence of the mitral valve is often revealed when the valve is properly tested by a fluid pressure equal to that of the blood. I have often seen a heart inadequately tested in this respect. A degree of regurgitation, clinically observable, may be overlooked if the ventricle and valve are not subjected to sufficient fluid pressure.

On microscopic examination, groups of fibres are found in which the fibrillae are more or less replaced by rows of refracting fatty granules, the change appearing first in the neighbourhood of the nuclei of the fibres. Besides the groups of more intensely fattyly changed fibres, the other fibres are more or less dotted with fatty granules.

Clinically, in all cases of extreme simple anæmia of any considerable duration, one may observe a certain degree of enlargement of the heart; the apex beat is little to the left of the normal, and the area of percussion dulness extends slightly upwards; frequently a soft murmur is to be heard over the apex beat, which is not merely conducted from the pulmonary area, but has the characteristics of mitral regurgitation, and is no doubt due to a dilatation of the left ventricle, so that the base of attachment of the papillary muscles becomes displaced, and the mitral
valve slightly incompetent at the moment of greatest intra-ventricular pressure. The heart's action is quickened, and is peculiarly irritable to the calls of slight effort or to reflex or emotional stimuli. These symptoms, which constitute the cardiac features of anæmia, are of course only in part directly due to the state of the heart muscle, they depend rather upon the condition of the blood and the debilitated state of the nervous system; and to both these latter causes, as well as to the cardiac enfeeblement, is also attributable that degree of edema of the extremities which is so common in marked anæmia.

B. Toxæmia.—Hyaline degeneration.—A peculiar hyaline swelling of the muscle fibres of the heart in diphtheria has been described by Bouchut, Labadie, Lagrave, and Rosenbach. The last-named author looks upon it as an inflammation. Similar changes are met with in the voluntary muscles in typhoid fever. Boyce speaks of it as a hyaline degeneration of connective tissue, consisting of hyaline material similar to amyloid, but without the chemical reaction of the latter. Hyaline degeneration identical with that in the myocardium is more commonly observed around the arteries, sometimes permeating, and causing extensive atrophy of the muscle fibres of their middle coat.

Cloudy swelling is a condition in which the fibres of the heart lose their striation and become finely granular; it is met with especially in diphtheria, typhoid and typhus, and is indeed common to all febrile states of sufficient duration.

Fatty degeneration.—In certain poisoned conditions of blood, as from lead, arsenic, and, in a most notable degree, from phosphorus, fatty degeneration of the muscular fibres of the heart may be very extensive; and, in cases of phosphorus poisoning in which the patient has survived the more immediate gastro-intestinal symptoms, it is the principal source of danger. The mildest form of blood contamination—although very important from its being so common—is the absorption of ptomaines from the colon in neglected torpidity of the bowels, a source no doubt operative in the production of the fatty heart of anæmia. The most intense of the poisons of organic origin affecting the heart is that modification of the toxine of diphtheria which is formed in the later stages of this disease, and which appears to be responsible for that profound fatty degeneration of the heart (in common with other organs) which is only equalled in cases produced by phosphorus.

Repair in fatty degeneration.—Clinical observations would lead us to suppose that repair of fatty degenerated hearts is possible, and even of frequent occurrence; Dr. Coats is of opinion that it takes place by absorption of the fat and an actual new formation of muscular tissue. That such new formation is abundantly possible is evident from the readiness with which healthy hypertrophy is established to compensate valvular defects, or in response to other unusual calls upon the muscular activity of the heart.

Under the heading of changes of the myocardium of toxic origin we should certainly include those consequent upon chronic gouty conditions.
and chronic uræmic poisoning; although, as in the less defined changes induced by alcoholism, nicotinism, and the like, the lesions have features in common with those induced by other causes, and will be described later.

There can be little doubt that the high-pressure pulse and consequent increased call upon the heart which are associated with chronic affections of the kidney are combined effects of central nervous induction, having for their purpose such an increase of blood-pressure as shall promote compensatory kidney function. In chronic gouty conditions the cardiac vascular function is similarly modified; and in other cases of habitual high arterial blood-pressure from mental strain or other causes the same effects, although less in degree, are observed in the myocardium. These effects are, first, hypertrophy; and, secondly, fibro-fatty degeneration.

II. IMPAIRMENT OF THE MYOCARDIUM SECONDARY TO ALTERED BLOOD-SUPPLY.—A. Paroxysmal conditions of coronary arteries.—Many authors have pointed out the occasional occurrence of angina pectoris in young people attributable to excess in tobacco-smoking; and have observed the anginal paroxysm of like causation in older persons. Besides its other effects tending to disturbance of the cardiac innervation, Dr. Huchard holds the view that nicotine has a more direct action, by causing spasmodic contraction of the smaller vessels, and, in these cases, especially of the coronary vessels. It is difficult to bring evidence sufficiently demonstrative to prove this opinion or to refute it. Dr. Huchard relies chiefly upon the spasm of voluntary muscles and upon the pallor and arterial contraction observed in nicotine intoxication, upon the high arterial tension often to be observed in smokers, and upon the experiments of Claude Bernard in 1857, and by himself and others since, showing the local effect of nicotine in causing contraction of the vessels in the frog's foot. There is every reason to believe that the coronary arteries, like other vessels of equal size and equally richly endowed with muscular tissue, are liable to spasmodic contraction; and it is quite possible, as maintained by Huchard, that in some cases the abuse of nicotine may directly cause such constriction and produce temporary anæmia and disturbed function of the heart muscle. It has not been shown, however, that any textural damage to the heart's substance has been caused by the vaso-motor effects of nicotine upon its circulation. Of course, the remoter effects of nicotine in causing arterial and muscular degeneration, if such there be, are not included in the present subject.

B. Permanent changes in the coronary arteries.—(i) Atheroma of the coronaries.—This may arise: (a) From the natural effects of age leading to degeneration of the intima, with secondary thickening and softening, or calcareous deposition.

(b) These senile changes may be anticipated by constitutional conditions, especially syphilis, alcoholism, and gout; the sequence of events being much the same, namely, degenerative impairment of elasticity, patchy thickening, fatty change, or calcareous deposition.
(c) Hereditary disposition plays an important part in determining premature decay of the arterial system.

The above conditions are general to the whole arterial system, but are most manifest at those portions of it at which the stress of normal arterial pressure is most heavy. The origin and arch of the aorta and the coronary arteries are the portions thus affected which concern us at the present moment; and it may be noted that atheromatous narrowing of the coronaries is generally most marked at their aortic origins, and is often limited to these parts.

(d) The chronic arterial strain of laborious occupations has a very important influence in producing chronic patchy endarteritis of the aorta and the coronary arteries; and it operates very commonly in conjunction with the causes of arterial degeneration spoken of under headings \( b \) and \( c \).

There can be little doubt that the peculiar patchy distribution of endarteritic thickening is due to small rifts at points of least resistance of an intima rendered more brittle by degenerative changes, and to the secondary nuclear overgrowth and subsequent degenerative changes ensuing thereupon.

(e) Apart from the general effect of syphilis in disposing to arterial atheroma, syphilitic granulomata may form in and about the arteries, especially thickening their inner coats, and thus often leading to narrowing or obliteration. [Vide art. "Disease of Arteries," vol. vi.]

(f) Vessels of small calibre, such as the coronary arteries when narrowed, and with their intima changed by atheromatous or specific arteritis, are very apt to become abruptly and completely closed by thrombosis.

(g) The coronary arteries, like other vessels, are liable to embolic closure, although they are much less prone to this obstruction than are other vessels more directly in the current of the circulation. Such embolisms when they arise may be simple or septic.

Having now enumerated the possible causes of narrowing or obliteration of the coronary arteries, let us look to the consequences of such narrowing, which we shall find to embrace the most important lesions of the cardiac muscle.

(a) Fatty degeneration of the heart.—A gradually increasing impairment in the blood-supply of the heart, and a correspondingly diminished thoroughness of the irrigation of its tissues with blood, are the most frequent causes of fatty degeneration of the muscular fibres. I have already spoken of fatty degeneration of the heart as a consequence of general anaemia, and in certain states of toxemia; the degeneration arising from local anæmia, due to constriction of the supplying vessels, is of the same kind, but is much less acute, and is more patchy in its distribution. In hearts in which the coronary narrowing affects both vessels at their origins, the distribution of fatty change would be more uniform; but these cases are rare. Often only one coronary vessel is thus affected, and sometimes only certain branches within the substance of the heart are much contracted by atheroma. Thus the change, at least
in any serious degree, may be limited to one side of the heart, or to one or more portions of one or both ventricles or auricles.

The change, for reasons to be mentioned immediately, besides being much more insidious and slow in its progress, is mingled with other changes and, in particular, with false or fibroid hypertrophy of the organ. In very old people, in whom the whole process is one of senile decay, the fatty degeneration may be simple and unattended with fibroid changes.

The process of fatty degeneration of the cardiac muscle consists, as already stated, in the gradual replacement of the sarcolemmum elements by fatty granules, the deposition of granules beginning about the nuclei and extending linearly towards the fibre-ends. The affected tissue is thus rendered more opaque in streaks of a tawny yellow colour, is softer and more friable under the finger, and in well-marked patches gives a greasy section. In some cases, in which the degeneration is extreme over a restricted area corresponding with an occluded vessel, the fatty softening may be so great as to resemble abscess. It is said (11a) that in many cases the fatty change proper is preceded by a “cloudy swelling,” in which the fibres become finely granular from the deposition in them of fine protein granules, which are to be distinguished from fatty granules not only by their more dim and cloudy outline, but also by their reaction to strong acetic acid or caustic soda or potash, either of which obliterates them, whilst the same reagents bring out the granules of true fatty degeneration in stronger relief. Both the protein and fat granules are derived from the sarcolemmum elements of the muscle which they replace. As the disease advances the striation of the fibres becomes gradually lost; at first at the extremities of the fibres, finally towards their nuclear centres.

In combination with the fatty degeneration there is more or less atrophy of the muscular fibres, and in substitution for them an overgrowth of connective tissue elements resulting in the formation of fibroid tissue (fibroid or false hypertrophy). In this respect fatty degeneration of the heart, induced by restricted blood-supply from narrowed vessels, differs from the same degeneration due to general anaemia or toxic causes. I have already indicated an exception in the case of old people, in whom the degenerative changes are a part of general senile decay.

Although the internal surface of the ventricles may be specked and streaked with opacities—much more irregularly disposed, however, than in the case with degenerations of general blood origin—the endocardium itself is rarely affected. The size and weight of the heart, and the thickness or thinness of its walls, depend chiefly upon the amount of fibroid substitution which is associated with the fatty change. The pericardium is not necessarily involved, although it may be more opaque and thicker than normal.

It was found, in speaking of the more acute fatty degeneration of the heart due to general blood conditions, that partial or complete repair was possible by a renewal of the muscular fibres in the same way
as an extra growth of such fibres can take place in healthy hypertrophy, whilst at the same time the fatty degenerated fibres became absorbed. In degeneration due to perniciously narrowed blood-supply, however, no such repair can take place to any appreciable extent; for the anastomosis of the two coronary arteries, supposing only one to be affected, is not free enough to provide a sufficient circulation for the purpose. Nevertheless, we may see in the overgrowth of fibrous tissue, of a somewhat deprived sort it is true, an attempt to maintain the due resistance of the heart walls to blood-pressure, without however any corresponding preservation of contractile power.

Symptoms and signs.—The fatty heart is a weak heart, weak in its muscular power, and weak in its resistance to blood-pressure. It is either more or less arrhythmic in action, or readily becomes so under any extra demand upon it from excitement or effort. It is also (except in cases in which the degeneration of the heart goes hand in hand with general atrophy of blood and tissues in old age) an enlarged heart, increased in size by the dilatation of the ventricles, and especially of the left ventricle, under the normal blood-pressure; and often increased in size also by false (fibroid) hypertrophy. Hence, in a person usually beyond middle life, with a feeble circulation and a tendency to blueness of the extremities, if we find the superficial dimensions of the heart increased, the apex more to the left than natural, the dulness extending an interspace higher, and perhaps a finger’s-breadth more to the right than is proper, and if on auscultation we find a marked indistinctness of the first sound and an irregularity of beat both as regards time and force, we may be sure of degeneration of the heart, and that the degeneration is more or less fatty.

A very common symptom is the occasional occurrence of attacks of syncopal or anginal failure [see "Angina Pectoris," vol. vi.], but as these attacks are often simulated by those of a much less serious nature, they are always to be taken into account in conjunction with the signs of a cardiac enlargement. In cases of anginal attack attendant upon organic change in the cardiac muscle, and especially when such change is associated with coronary stenosis, the immediate cause of the earlier attacks is generally some increased call upon the heart from excessive exertion, such as walking quickly or uphill; and the result is to bring the patient to a stop at once. Later attacks may occur when the patient is at rest or asleep. In anginal seizures, when the heart is sound, the patient often tends to move about, and if the attack be not very severe it may not prevent the continuance of walking or other exercise.

In advanced cases of fatty heart, cases in which more distinct anginal symptoms may not have occurred, an altered respiratory rhythm is not infrequently to be observed, which is especially apt to occur during sleep; namely, an increasing shallowness of breathing down to absolute cessation for 20, 30, 40 seconds, then several profound and heaving respirations take place which again gradually subside to complete pause (Cheyne-Stokes breathing). During the pause the patient generally wakes up with a start, and his sleep is thus much interfered with and
becomes reduced to a succession of short dozes. The peculiar breathing
is to be observed during the waking hours also. The pulse continues
with its usually irregular action practically unaltered during the
arrhythmic breathing and pause; it is to be noted, however, that in such
cases during ordinary or deep breathing the pulse is distinctly weaker
during the inspiratory wave. It must, lastly, be confessed that rare cases
are met with in which, even with a marked degree of fatty heart, no signs
are discovered up to the moment of fatal syncope or angina. I must state
my belief, however, that if the opportunity presents itself for a careful
examination of such cases, and the possible presence of emphysema be
taken into account as masking an increase of the cardiac area, the clinical
evidence of fatty degeneration is rarely to be missed.

The disease is most common at or beyond 50 years of age. Men more
frequently suffer than women in the proportion of nearly two to one (Quain).
All the functions of a person with fatty heart are performed in a languid
manner. He is the subject of atonic dyspepsia, with a great tendency to
flatulent distension of the stomach; his bowel and liver functions are
torpid; the urinary secretion, very sensitive to external surface tempera-
ture, is of low range of specific gravity, and often contains a trace of
albumin. The brain is easily fatigued, the temper irritable. Only
gentle level exercise can be taken with comfort.

Treatment.—The treatment of fatty degeneration of the heart due to
altered blood-supply is a matter of great importance, hence the necessity
of recognising the lesions at the earliest possible stage.

In the earlier stages regular exercise short of fatigue, and adapted to
a person in whom a weakness of the central organ of the circulation is
recognised, is of importance; quiet walking on the level, riding (not
hunting), cycling (avoiding hills), driving, sailing, quiet rowing, may all
be allowed; and gentle incline walking, adapted to the case, may be
taken as prescribed exercise. Covert shooting may be followed, but not
rough walking or hill shooting. Golf and croquet are games well adapted
to such people. For these early cases, a course of Nauheim baths and
exercises may be taken with advantage from time to time, the exercises
being especially valuable in aiding by tonic muscular contractions the return
of blood to the heart, disgorging the venous circulation, and thus aiding
the forward movement. Cold bathing should be forbidden, and a warning
given against walking against cold winds.

A nutritious diet, rather nitrogenous than fatty or starchy, may be
allowed, distributed in three regular meals daily, eaten slowly, and adapted
in quantity to the lessened requirements of a less active life. A moderate
amount of wine is usually required.

Arsenic, iron, and strychnia are the tonics especially valuable; but
they should not be given in more than two, or at most three doses daily
for short courses—the most careful regard being given to avoid digestive
disturbances. In all cases of lowered blood-pressure there is a tendency
to passive congestion of the organs and especially of the liver, so that
a mild dinner pill and an occasional mercurial alterative are desirable.
In advanced cases of fatty degeneration the same general plan must be followed still more carefully, and with narrower restrictions as regards exercise, which should only be allowed on smooth level ground, all stair-climbing being strictly forbidden. The diet must be closely watched, special care being taken to avoid overloading of stomach and acute dyspepsia, as many fatal seizures are attributable to gastro-intestinal disturbance. Persons with fatty heart are extremely sensitive to external cold, and should be clothed very warmly. A thorough rest, lying down, once or twice a day should be enjoined; the best times to select are before meals; a short rest being taken before luncheon, and a more prolonged rest, of one and a half to two hours, before the late dinner. Such patients should only use warm water for bathing, and for them the Nauheim baths and exercises are not to be recommended.

To a strychnia and arsenic or iron tonic some digitalia, strophanthus, or convallaria may be added; and an aromatic stimulant and carminative draught should always be at hand in case of syncopal attacks, and may often usefully contain a little nitro-glycerine.

Finally, in cases of fatty heart which have advanced to the production of any decided symptoms, the employment of oxygen inhalations twice or three times a day is valuable as a cardiac restorative; it operates principally, no doubt, in stimulating cardiac nutrition and in facilitating the removal of waste tissues from the organ by flushing it with more highly oxygenated blood. For the Cheyne-Stokes respiration, in advanced stages, there is no more powerful means of affording relief than oxygen inhalations in combination with strychnia. In such cases, however, it should not be employed with the nose-oral inhaler, but a current of oxygen should simply be played over the mouth and nostrils of the patient for five or ten minutes without any extra respiratory effort on his part.

(β) Fatty infiltration of the heart.—Fatty infiltration of the heart is a condition in which, owing to the deposition of fat in the interstices of the muscular fibres, these fibres themselves are compressed, impeded in action, and become atrophied.

A certain amount of adipose tissue is naturally present on the heart, especially along the superficial course of the coronary vessels and in the sulci at the base; under certain conditions this tissue develops in inordinate quantity and spreads over the cardiac surface, penetrating, chiefly with the arterial branches, into its muscular substance. This increase and extension of the adipose tissue is most marked over the right ventricle, and may constitute a layer of considerable thickness which by its encroachment upon and between the muscular fasciculi may cause their atrophy and replacement, and thus considerably weaken and embarrass the heart. The extension is always from the subpericardial surface and chiefly along the arterial lines. The atrophy of the muscular tissue which attends upon fatty infiltration is for the most part consequent and secondary; but it is probable that in some cases a primary atrophy of the muscle leads to the secondary development of fat in the connective tissue—which is everywhere present and potentially fat-bearing.
Thus, clinically, we have two forms of fatty infiltration of the heart: the one in which the fat is rapidly stored and extends into and encroaches upon a higher tissue, the function of which it embarrasses, and the nutrition of which it mechanically interferes with; the other in which the fatty tissue merely, as it were, fills up the interstices left by an atrophying muscular tissue.

Of these varieties the first is by far the more common and important. It is met with in persons of inactive and often indolent and self-indulgent lives, in men at middle age, in women towards the climacteric period, or soon after it. It is the people who have good appetites and good primary digestion with faulty assimilation and inadequate eliminative power that are especially liable to this disease. Indulgence in alcohol, and especially in malt liquors and the sweeter wines, certainly favours its occurrence; and there are certain maladies upon which it is peculiarly liable to ensue, especially those affections which involve a deprivation of respiratory surface, such as chronic emphysema, or fibroid disease of the lung in old-standing quiescent phthisis, or secondary to pleuritic effusion or unresolved pneumonia, etc. Defective elimination by the bowels and kidneys is similarly efficacious. It must be carefully remembered, however, that no organic disease of any kind is necessary as the forerunner of this affection, which may arise solely from an excess of alimentary supply over demand, however this may be brought about.

Symptoms and signs.—Persons thus affected are stout, increasing in weight, with a thickening layer of adipose tissue, full abdomens, and often tender livers. Their circulation is feeble and usually slightly quicker than was normal in them. There is some excess of venosity in their colouring, they are short-breathed on exertion, and sweat easily. Later they manifest functional disturbances of the heart’s action, rapidly induced on exertion or coming on without it. The cardiac dulness is increased by an interspace upwards, but the apex beat is difficult to feel, and the cardiac impulse tends to be more felt towards the epigastrium than beyond the normal position to the left. The sounds are less clear than natural, otherwise unchanged. There is no change to be felt in the arteries; the pulse is usually soft, of low pressure, and, if full, is compressible. Of course this condition of pulse may be varied by other intervening states, such as gout, to which, however, these people are not peculiarly liable. The urine varies, but is habitually pale and copious, and of rather low than high range of specific gravity.

Whilst in a far less dangerous condition than that attendant upon a truly fattily degenerated heart, these patients are nevertheless very liable to succumb to acute disease of any kind, and particularly to bronchitis, pneumonia, enteric fever, or surgical injury.

The treatment is simple, rational, and, if loyally followed, very successful. The dietary must be mainly nitrogenous, all superfluous starches, sugars, and fats being discarded. Only claret, moselle, or equivalent quantities of spirit well diluted must be allowed, and in sparing quantity. The meals should be at regular times, slowly eaten and strictly moderate in quantity.
But little fluid should be taken with the meal, but tissue change duly ensured and thirst satisfied by a moderate quantity of hot or cold fluid slowly sipped about a quarter to half an hour after the meals, or, sometimes better still, half-way between the meals. A tumbler of hot water with a little fresh lemon juice may be taken at bedtime or in the early morning. Raw fruits, root vegetables, and bread must be avoided, or only very sparingly taken. Daily walking, riding, or cycling exercise must be imperatively enjoined; for the advantage of regulated exercise is not merely to quicken muscular nutritive changes, and so to convert the food taken into proper force-yielding material, but to deepen respiration and to promote the respiratory and other eliminative functions. Hence dumb-bell, fencing, or other home exercises carried on indoors, although they may be useful supplementary aids, are not adequate to replace open-air exercise. Medicinal treatment is of quite minor importance, and may be limited to promoting due elimination, and giving a heart tonic if needed. Turkish baths, or a course at Homburg, Carlsbad, Marienbad, Harrogate, or Nauheim, may be suggested in appropriate cases.

The other form of fatty infiltration attendant upon atrophy of the heart is met with in an altogether different type of individual, one who commonly is already the subject of some grave organic disease, such as tuberculosis or cancer; and its importance and treatment are both merged in the graver malady.

(γ) Fibroid infiltration of the heart (Fibrous transformation, Coats; Fibroid degeneration of the myocardium, Orth.; Myocarditis productiva or interstitial myocarditis).—This condition essentially consists in the separation and replacement of the muscular fibres of the heart by an imperfect fibrous tissue generated by overgrowth of the connective tissue of the organ. It is very closely analogous to fatty infiltration, and it cannot rightly be described as a degeneration of the myocardium. It would seem, therefore, that the term "fibroid infiltration" most fitly describes the morbid state present; interstitial myocarditis is also a fairly accurate term, although it conveys a false impression of the disease being an inflammatory one, which it rarely, if ever, is.

Fibroid infiltration of the heart may be described as general and local, although even in general infiltration the disease is not uniformly distributed.

Causes.—Besides the coronary obstruction general fibroid infiltration has another principal cause; namely, chronic congestion of the heart from mechanical impediment to the return of blood from the cardiac veins. This cause is chiefly met with in cases of old-standing emphysema, and in cases in which the whole or a large portion of one lung is the seat of cirrhotic change from old pleurisy, unresolved pneumonia, or fibroid phthisis. Extensive narrowing and destruction of pulmonary vessels and impairment of that inspiratory aid to the cardiac circulation which obtains in healthy respiration, results in a difficulty in the pulmonary circulation, at first overcome by greater diligence of the right heart, but gradually
increasing until the venous return to the right auricle is seriously impeded. A chronic congestion of the walls of the heart ensues, most marked on the right side, but involving the left also; and, as a result of this chronic congestion, overgrowth of connective tissue and atrophy and degeneration of the cardiac muscle proper. In the more advanced stages of mitral stenosis and regurgitation the same conditions are to be observed, having similarly a mechanical origin.

Nevertheless the most important cause of general fibroid infiltration of the heart is the obstructive disease of the coronary arteries at or near their origin from the aorta, under which head we now consider it. A more marked degree of fatty degeneration of the muscular fibres is met with in association with fibroid infiltration arising from this cause, for a degree of blood irrigation which will suffice for the overgrowth of an inferior tissue, such as connective tissue, will not suffice for the nutritive maintenance of a tissue of such activity, and requiring such frequent renewal and restoration as the muscular tissue.

It is thus to be remarked that hearts which are the seat of general fatty degeneration from coronary obstruction (except quite as a senile change) are always large hearts, the seat of so-called false hypertrophy; and this it is which furnishes us with an important clue to their clinical diagnosis. The increase in size is partly due to increased thickness of the cardiac walls, in part to dilatation of the cavities of the heart; for fibroid infiltration, although it increases the toughness of the cardiac wall, diminishes its resilience and contractile power; hence a gradual yielding to the blood-pressure, each stage of which is permanent.

Fibroid infiltration as a local affection of the heart arises from—1. Local obstruction to the circulation, due to local plaques of thickening, and degenerative constriction of the coronary branches. The heart's substance may be the seat of innumerable patches of grayish white fibroid infiltration from this cause, or there may be one or two such patches of larger dimensions corresponding with the territory of a larger branch.

2. Corresponding with well-marked patches or "scars" in the heart's substance there will often be found a complete occlusion of a coronary branchlet from thrombosis or embolism, and in an earlier stage the more distinct signs of a hemorrhagic infarct may be seen.

3. It is very possible that some of the heart scars which are found may be due to a fibrous repair of partially ruptured fibres.

4. An extensive, although usually superficial fibrous infiltration of the heart may ensue upon pericarditis and adherent pericardium, the change beginning in the subpericardial tissue and extending more or less deeply into the muscular interstices of the heart. Such changes are started by direct inflammatory irritation, and are often accompanied with a certain degree of fatty infiltration.

Pathology.—The minute pathology of fibroid infiltration of the heart is the same, other things being equal, as that of the same process taking place in any other organ; that is, it begins with a proliferation of the
nuclei of the connective tissue, so that in the earliest stage, rarely observed
except at the margins of extension, areas or groups of crowded nuclei
are to be seen which are gradually transformed into fibres; these again
in their turn, losing their characters, form dense areas of wavy, glue-like,
interlacing processes, entangling a few nuclei. In the denser portions the
muscular fibres of the heart are completely replaced or destroyed, or
only appear as small islets of a few isolated fibres; and towards the cir-
cumference of any local patch the muscular fibres are observed to present
broken or atrophied terminations, and to be more or less widely separated
by the intruding tissue. Here and there streaks of pigment granules
may mark the site of destroyed muscular tissue.

The process of fibroid infiltration must by no means be regarded as
in all cases a destructive lesion; on the contrary, it is in most instances
the result of an effort at repair. This is most distinctly the case in
heart "scars," where the necrosed muscle, infiltrated with blood elements
which constitute an infarct, is gradually removed by absorption and
replaced in the only possible way by the growth of a living but inferior
tissue, which serves the purpose at least of healing the breach and giving
mechanical support to the heart wall. And, rightly regarded, the fibroid
infiltration more generally dispersed through the heart substance in cases
of retarded or restricted circulation is the means of maintaining the
resistance of the ventricle walls to the blood-pressure, a conservative
effort, although attended with but poor and temporary success.

In cases of local fibroid infiltration reparative of necrosing infarcts,
the scars sometimes become infiltrated with lime salts, and grate under the
knife on section.

Symptoms and signs.—The symptoms of general or extensive fibroid
infiltration of the heart are those of chronic heart failure, and difficult to
distinguish from those of fatty heart, with which, as already observed,
the disease is often associated. The patient, usually fifty or upwards,
and more commonly a man, has for some months been aware of scantness
of breath, and of oppressed feelings about the heart on exertion; but he
has become accustomed to this, and the first symptoms compelling his
attention, and leading him to seek advice, generally supervene quite
suddenly. During some accustomed or slightly increased effort—the
walk home from business or an extra round at golf, or a tramp with the
gun over a turnip field or up a sharper hill than usual—he is seized
with severe breathlessness and oppression at the heart, which compel him
to stop and rest for a time and to get home very quietly for fear of a
further attack, of which he has some dread. The first attack may amount
to a distinct anginal seizure (see "Angina Pectoris," case 3). After this
experience his cardiac power is never on the same level as before, and often
deteriorates rapidly. His breathing fails him on slight exertion, he
becomes liable to dyspnoea on slight distension of the stomach, his face
becomes somewhat puffy and dusky in colour, he is apt to be awakened
at night with more or less urgent dyspnoea and wheezing, which he
regards as asthmatic. The ankles and legs become puffy and oedemaous,

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and finally he is confined to his room and chair on account of the con-
stant and readily increased dyspnoea.

On physical investigation the fibroid heart is always found to be
associated with other conditions in the same plane of degeneration, and
which therefore help to point to the diagnosis. Thus in extreme
emphysema, in the later stages of Bright's disease, as well as in the
early manifestations of cardio-vascular degenerations associated with
gout, intemperance, and syphilis, we often find fibroid infiltration of the
heart as a factor of importance in the illness of the patient; indeed,
it is more than doubtful if there be such an independent disease as
fibroid infiltration of the heart.

Diagnosis.—Having indicated sufficiently, therefore, the general
symptoms which may be attributed to this state of the heart, I may briefly
add the salient points of physical diagnosis. In the majority of cases
there is evidence of degenerative thickening of the vessels generally.
The systemic vessels are wanting in elasticity, and more or less
thickened; the radial artery is more thick and palpable than natural;
the pulse is not as a rule quick, it may be regular; but often it is irregular
in force and rhythm; the pressure varies, but is not high unless it be
raised by some other disturbing condition. In cases in which the cardiac
state is secondary to emphysema, mitral stenosis, or adherent pericardium,
there may be no arterial thickening; and the pulse is feeble, vacillating,
or compressible. Indeed, it will often interest the clinical observer to
note the big labouring heart, with no important valve lesion to waste its
force, and to contrast the work apparently done with the feeble result at
the wrist. The dimensions of the heart are increased in all directions,
the apex beat is extended beyond the line of the left nipple, the upper
margin of dulness is raised to the third space or cartilage, the right
margin of dulness extended to the median line or a finger's-breadth beyond
it. The size of the organ varies, however, of course, with the stage of
the disease, but it is always increased considerably by the time the patient
complains of symptoms. Again, in cases having their origin in cardiac
congestion from emphysema or mitral disease, the evidences of enlargement
of the right side of the heart are most considerable, the extended impulse
is most apparent towards the ensiform cartilage, and the dulness to the
right of the sternum. The presence of emphysema tends to mask the
percussion and palpation signs very considerably, and must therefore be
taken into careful account. The cardiac impulse, although somewhat
heaving, has notably less of the thrusting quality than would obtain over
a heart of anything approaching to similar dimensions from pure muscular
hypertrophy; it is also more generally diffused over the cardiac area.
In cases of difficulty in defining the limits of the cardiac outline by
palpation and percussion, a stethoscope with a small chest-piece may be
usefully employed. There is not necessarily any marked alteration in the
sounds of the heart, but the first sound at the apex is always longer,
duller, and less defined than normal, and it is often attended by a soft
murmur; whilst the first sound at the base is barely audible, and the
second sound there is dull, muffled, and prolonged. In mitral cases, however, the second sound over the pulmonary area may be strongly accentuated, although duller and less acute than in the earlier stages of the valve disease.

There is frequently some albumin in the urine, especially in the later stages; and other evidence of visceral congestion from retarded circulation, such as occasional congestion at the base of the lungs, fulness of the liver, and the dyspeptic phenomena of slow digestion with flatulence and loaded urine. With increasing failure of cardiac force the urine falls in amount, and dropsical phenomena set in.

Treatment.—The treatment of fibroid degeneration of the heart is best considered under the diseases—emphysema, angina pectoris, and failing compensations in cardiac lesions, into the symptomatology of which it enters as an important factor.

(8) Aneurysm of the heart.—Aneurysm of the heart is a rare condition, and one still more rarely clinically recognisable. It is questionable whether all the cases recorded by Heschl and Willigk are cases of true aneurysm. The left ventricle is almost exclusively affected and most commonly (in 59 per cent) at the apex; occasionally the septum between the ventricles is the seat of aneurysmal bulging. The pouch varies in size from that of a filbert to that of a large cocoa-nut; it is lined by stretched endocardium, and contains laminated clot and more recent coagulum.

Local destruction of the muscular fibre from any cause may lead to aneurysm. Local softening, consequent on disease or occlusion of a branch of a coronary artery, is commonly responsible for acute aneurysm. Circumscribed suppurative myocarditis is another cause of it. Chronic fibrous myocarditis disposes to aneurysm when the heart wall is thin, not when it thickens.

Dr. Wickham Legg attributes such aneurysms to fibrous degeneration of the heart muscle, and points out that while there is abundant evidence that this degeneration is commonly due to impaired coronary circulation, there are yet many cases of aneurysm of the heart which occur in people under forty years of age, in whom the coronary arteries show no change, so that he doubts whether coronary obstruction is responsible for the myocarditis in all cases. Hilton Pagge regarded fibrous myocarditis as the cause of chronic aneurysm in almost all cases.

The tendency is for the sac to rupture into the pericardium, causing death. In other cases death occurs from mechanical interference by the sac with the movements of the heart. Spontaneous calcification and partial obliteration of the sac may result.

(ii.) Thrombosis, Embolism, and (iii.) Aneurysm of the coronary arteries require brief notice, although the symptomatology and diagnosis of thrombosis and aneurysm are for the most part included in the phenomena arising from atheroma of the vessels, whilst embolism is a very rare affection, and difficult, if not impossible, to recognise during life.

Embolism of the coronary arteries may occur under any of the con-
ditions which occasion embolism of other systemic vessels; but the situation of the vessels at the commencement of the aorta, the wide angle at which they leave the vessel, and the bulk and impetuosity of the blood-current at this portion, are all conditions unfavourable to the passage of clot into these small side arteries.

The embolii may be of the ordinary fibrinous character, or, as in cases of ulcerative endocarditis, may be septic. It is quite possible for débris from a softening atheroma of the main coronary trunks to be conveyed onwards to occlude some of their terminal branches.

Thrombosis of the coronary arteries is a frequent result of previous atheromatous change and is also occasioned by specific arteritis. Any portion of the vessel already thickened and narrowed by atheromatous change may thus become more or less suddenly and completely occluded by coagulation. Thrombosis may occur at any portion of the coronary arteries, but is most frequently met with near their origins from the aorta for the reason that these portions are the most common seats of extensive atheroma. When it occurs deeper in the heart it is often associated with gummatous arteritis.

It is to be borne in mind that although it has been shown by Wickham Legg and West, contrary to the opinion previously current amongst pathologists, that there is at least some intercommunication between the peripheral distribution of the two coronary vessels, yet this communication is very restricted, and the effect of a complete closure of one of the coronary arteries in any part of its course is to produce anæmia of the territory beyond. Fringing the anæmic area and encroaching upon it is a line of congestion or partial capillary stasis; but there is no filling up of the area with blood so as to form the damson cheese-like appearance of recent infarcts in more vascular tissues. The yellowish tinge of the area is that natural to anæmic muscle. A softening from fatty degeneration and molecular necrosis of the area follows and hemorrhages may occur into the softened area. Microscopically the muscular fibres are found broken up, their transverse striae are lost, and the remains of the fibres have assumed a hyaline or waxy appearance (Coats). The area of congestion surrounding the infarct becomes the seat of more or less inflammatory reaction, attended with the usual proliferation of connective tissue, and infiltration with leucocytes. The softened area wastes (falling below the surface on section), and gradually undergoes contraction by encroachment of fibroid growth extending from its periphery, the semi-liquefied tissues becoming slowly absorbed; the final result being a heart scar of dimensions varying with the size of the original infarct. In cases, however, where the softened territory is of considerable dimensions, the branch occluded being large, the softened area of the heart wall yields before the blood-pressure, and an acute aneurysm of the heart is formed which may terminate in rupture.

The result of a partial occlusion of the coronary artery by thrombosis or atheroma has already been described, namely, a fibrous transformation of the corresponding territory; and, in cases in which the
complete occlusion of the vessel is slowly effected, the same effect is
produced.

Symptoms and signs.—The symptoms of sudden occlusion of a con-
siderable branch of the coronary artery generally begin with an anginal
paroxysm which may be fatal at once. In cases in which the first
seizure is survived, the subsequent phenomena are those of rapid
heart failure, dyspnoea with acute anginal paroxysms, rapid and more or less
irregular heart’s action, dilatation of the organ to the right or left
according to the ventricle affected; systemic and pulmonary edema are also
correspondingly predominant. These acute phenomena almost invariably
supervene upon chronic heart difficulties already ascribed to degenerative
changes, and more or less quickly close the scene. Even the rare cases
of embolism of the coronaries have generally been preceded by the signs
of acute or chronic endocarditis, usually of the aortic valves.

Aneurysm of the coronary arteries is a disease the secondary effects of
which upon the cardiac muscle are of less importance; the disease itself
will be treated of in the sixth volume of this work (art. “Aneurysm”).

III. Impairment due to senile changes: Pigmentary degenera-
tion; Atrophy.—(a) Pigmentary degeneration.—This is a condition
seen in nearly all people above the middle period of life, but the change
is not met with in the voluntary muscles (Wilks and Moxon). The
heart weighs less than normal; it is hard and tough, and the muscle
fibres are a dark chocolate colour. The pigment itself consists of hemato-
toidin granules of a reddish yellow colour collected about the nuclei
of the muscle fibres. Atrophic changes usually accompany the pigmenta-
tion, though the striation of the fibres is not much altered. Besides
senile states it is met with in any general emaciation (Wilks and Moxon);
its does not seem to impair the functions of the organ.

(b) Atrophy of the heart.—Atrophy of the heart may be part of
general wasting, as in old age or chronic disease. It may become reduced
in weight—from 9 oz. in woman, or 10 or 11 in man, to 6 or 5 oz.,
drier in texture from loss of fat and fluid, and darker in colour from the
accumulation of pigment granules about the nuclei of the muscular
fibres. Local or general atrophy may result from impaired circulation
in tortuous and diseased vessels; but under these conditions, except
perhaps in old people, the muscular atrophy is attended with the over-
growth of another tissue—the connective. Fatty and fibroid infiltration
are both attended with more or less separation and atrophy of the
muscle proper.

IV. Impairment of the heart from functional strain requires
little more than a reference here, since the forms it assumes are discussed
elsewhere.

Functional strain, resulting in hypertrophy, may be due to the pro-
longed endeavour of the heart to overcome some increased resistance to
the circulation, or to compensate some defect in its valve mechanism.
Undue rigidity of the vessels, generally from atheromatous changes, chronic heightening of the arterial blood-pressure, as in Bright's disease, obstructed circulation through the lungs, atheroma of the main vessel, disease of the different valves of the heart, or congenital alteration of one of the orifices, are amongst the chief causes leading to hypertrophy. The hypertrophy thus occasioned cannot be strictly spoken of as disease of the heart; it is rather an attempt—for a time successful—to compensate a pre-existing defect; and the portion of the heart affected is dependent upon the seat of obstruction in the circulation.

Hypertrophy of the heart, secondary to obstructed coronary circulation or pericardial adhesions, is not real hypertrophy at all, but a thickening of the organ due to changes (mostly fibroid) secondary to chronic interstitial myocarditis.

Idiopathic or simple hypertrophy is a condition of muscular over-development from excessive cardiac exercise. Allbutt has described changes in the heart ensuing upon prolonged muscular exertion, such as hill-climbing, athletic exercise, and the like. Perhaps the best-known example of alleged simple hypertrophy was that recorded by Professor Haughton in the case of the celebrated greyhound, "Master Magrath"; but veterinary surgeons are not yet agreed whether simple cardiac hypertrophy is found in horses and other labouring animals (vide p. 916).

Bollinger recorded forty-two cases of simple hypertrophy without valvular disease—thirty-eight men and four women—in which the hearts were one-third heavier than in health. The observations were made at Munich, and Bollinger considers the great consumption of beer in that city as the chief cause of the hypertrophy, producing its effects (a) through the toxic effects of the alcohol; (b) by the quantity of liquid taken into circulation; (c) by increased nutrition. The view that the heart (left ventricle) hypertrophies towards the end of pregnancy was first put forward by French accoucheurs. German obstetricians denied this. Macdonald upheld the view in this country, and Hamilton's observations confirm the French view. The probable cause is the increased work the heart has to do in driving blood through the enlarged uterus (Hamilton); it has also been attributed to a toxic state of the blood.

Acute strain of the heart may mean either acute over-distension or acute over-function.

In the first case, under sudden accession of the blood-pressure chiefly arising during great effort, especially when associated with some obstructive valve defect, such as aortic or mitral stenosis, the portions of the heart most concerned may become over-distended to the suppression of their function. Sudden death may ensue from complete cessation of the heart's action, or a grave embarrassment, threatening death, may only be averted by a timely bleeding. Again, an obstructed function, less in degree, may be to a certain point recovered from, but leaves the heart temporarily or permanently strained. What precisely does this mechanical strain of heart mean? With what changes in the myocardium is it associated?
In the St. George's Hospital Reports, 1870, and in a previous paper read before the British Medical Association in 1869, Professor Clifford Allbutt describes the effects of overwork and strain on the heart and great blood-vessels, especially to be observed amongst such hard labourers as forgemen, colliers, wharfingers, etc. He also relates some cases illustrative of the earlier stages in which, after excessive exercise in mountain-climbing, hard gymnastics, and rowing respectively, signs of dilatation from acute overstrain are followed by those of hypertrophy of the heart. Professor Allbutt considers the sequence of events to be as follows:—(i.) Dilatation of right heart; (ii.) dilatation of left heart; (iii.) hypertrophy of one or both ventricles; (iv.) chronic inflammatory endarteritis of the aorta; (v.) dilatation of the aorta; (vi.) incompetency of the aortic valves; (vii.) further left ventricle hypertrophy compensating aortic defect; (viii.) degenerative changes ensuing upon hypertrophy. (Vide art. "Mechanical Strain," p. 841.)

Mr. Myers in 1870, in a paper on "Diseases of the Heart among Soldiers," drew attention to the effects of prolonged exertion in tight-fitting uniforms, and especially whilst wearing the tight breast-strap, in producing cardiac and aortic diseases from overstrain.

Da Costa has described a condition of "irritable heart" as of very common occurrence in soldiers during the fatigues of a campaign, and observed by him especially amongst the soldiers in the American Civil War. A persistently quick action of heart, with precordial and left shoulder pains, and bouts of severe palpitation under slight exertion, or digestive disorder, are the principal symptoms. At first these are unattended with any notable physical signs, and they may subside without such signs; but in the cases of greater severity or longer duration there is obvious enlargement of the heart. The pathology of these cases would no doubt be for the most part the same as that described by Allbutt, namely, a chronic myocarditis ensuing upon dilatation and mingled with muscular hypertrophy; but probably there is also some direct damage to the cardiac nerves, originating at the terminals of the vagi and sympathetic. (Vide Soldier's Heart, p. 851.)

V. IMPAIRMENT OF INFLAMMATORY ORIGIN.—(a) Interstitial myocarditis. —Myocarditis most generally consists of an irritative overgrowth of the interstitial connective tissues of the heart, which may extend from a pericarditis or an endocarditis. In its first stages an increased nuclear proliferation, permeating the muscular fibres, causes a "cloudy swelling" of the tissue, and a certain degree of increased softness to the touch, but the later result is more or less fibrous toughness of the part involved.

Disturbed circulation, general or local, will occasion cardiac fibrosis; such as chronic congestion of the cardiac veins, or restricted or obstructed circulation through the coronary vessels. The cicatricial or tendinous patches of the heart are produced by interstitial myocarditis. An impairment even to destruction of the true muscular fibres of the
(b) Parenchymatous myocarditis, which is met with in certain cases of septic poisoning, such as pyæmia and diphtheria, is probably but a very active form of the preceding process in its earliest stages. Professor Leyden has studied this lesion most carefully, and regards it as an acute myocarditis characterised by intermuscular nuclear proliferation and by secondary atrophic changes towards necrosis and deposition of pigment; fatty degeneration of the muscular fibres accompanies it, possibly in consequence of the inflammatory changes. This form of myocarditis is always secondary to infective fevers, such as diphtheria, scarlet fever, and the like; and has been met with in greater or less degree in all acute febrile diseases, rheumatism, cerebro-spinal meningitis, variola, erysipelas, malaria, septicaemia, influenza, and so forth. In enteric fever and in gonorrhoea the respective specific bacilli and cocci have been found in the heart.

(c) Purulent myocarditis is in most cases secondary to infective embolism of the coronary vessels; as in pyæmia, and ulcerative endocarditis. In all cases infective microbes are conveyed to the cardiac muscle through the coronary arteries, and set up foci of virulent myocarditis resulting in minute or larger suppurations.

(d) Syphilitic myocarditis.—Syphilitic myocarditis almost invariably occurs either in the immediate neighbourhood of a gumma or secondary to and in the territory commanded by a specific arteritis. Attention was first drawn to the occurrence of syphilitic lesions of the myocardium by Sir Samuel Wilks in 1856; and many isolated cases have been reported since at the Pathological Society of London and in various English and foreign medical journals. ‘Our knowledge of the disease, however,’ is mainly derived from the post-mortem observation of cases in patients, by no means all of whom died with heart symptoms.

Syphilitic disease affects the myocardium in one of three ways, and either singly or combined:—

(a) There may be syphilitic arteritis and secondary or combined chronic myocarditis.

(b) There may be gummatous formation in the heart wall, around, and extending from which chronic myocarditis takes place.

(c) There may be a diffused chronic myocarditis of specific nature affecting a considerable portion of the heart. It is doubtful, however, whether this latter form of diffused syphilitic myocarditis does not originate in scattered gummatous depositions.
Syphilitic arteritis of the coronary vessels does not differ from similar arteritis elsewhere. The endocardium is only affected in cases in which there is subjacent gumma of the muscle, and the pericardium as a rule also only in connection with such gummata or gummatus affections of the vessels. Pericardial adhesions in syphilitic subjects are, however, not uncommon. Syphilitic disease, limited to the valves of the heart, is almost unknown, but the aortic valves frequently partake with the aorta in an endarteritic thickening having its origin in the syphilitic cachexia.

With the undue rigidity, actual narrowing and occasional thrombosis, with which coronary arterial diseases of syphilitic source are attended, we find fibrous degeneration, dilatation, angina pectoris, and so forth, as later consequences.

Gummatus formations may occur in any part of the heart, most commonly in the ventricles or septum. They have the usual features and ill-defined microscopic characters of gummata elsewhere; they may soften, or undergo fibroid change, and they are always surrounded by more or less spreading fibroid condensation of the heart wall from associated chronic myocarditis.

It cannot be said that any symptoms have yet been formulated which in their grouping or individual significance are characteristic of syphilitic disease; and for the obvious reason that very different portions of the heart may be affected in different cases, and that the upshot of the morbid condition in each case is a spoiling of the cardiac muscle at the part affected, and more or less interruption or spreading disturbance of the cardiac mechanism therefrom.

Functional irregularity, anginal seizures, syncopal attacks, any of which may prove fatal, are amongst the most common symptoms. It is remarkable that sudden death has terminated a large proportion of the recorded cases of gumma of the heart in most instances without any previous recognition of the disease. The first case recorded by Sir Samuel Wilks ended in death in this way, as did fourteen out of twenty-five cases recently collected by Dr. S. Phillips. Enlargement of the heart, or displacement of the apex beat to the left, or more marked evidence of enlargement to the right, are amongst the later signs; especially in cases of the more diffused form of syphilitic myocarditis.

The absence from the history of the case of rheumatism, of gout, of alcoholism, or strain; and evidence—whether in the form of a distinct history or of collateral lesions of a specific kind—pointing to a syphilitic cachexia, are circumstances which, in the presence of such signs and symptoms of cardiac disease, may lead us to suspect its syphilitic nature, and to direct our treatment to that probability. When the signs and reservations with regard to such heart diseases as are above spoken of are found in men, and before middle life (nicotine poisoning being also excluded), an additional argument in favour of syphilis will be found. The success of antisypilotic measures of treatment, which, however, would be combined with cardiac tonics, strychnia, digitalis,
iron, or arsenic appropriate to the case, would further help the diagnosis.

VI. TUMOURS OF THE MYOCARDIUM.—The heart is one of the organs least commonly affected by new growths. Primary growths are exceedingly rare, but sarcoma, myxoma, fibroma, carcinoma, and fatty interstitial tumours have been met with. Tubercle, common in the pericardium, is rare in the heart substance, and then occurs almost exclusively as an accompaniment of general tuberculosis. Moreover, the heart is but rarely invaded by secondary growths. Round-celled sarcoma is occasionally to be met with, extending apron-like over the pericardium, greatly thickening it, and embedding the great vessels, yet not invading the heart itself. Carcinoma of the lung and mediastina frequently invades the parietal pericardium in cauliflower-like excrescences, and yet spares the heart. Sarcoma sometimes invades the heart from the mediastina along the sheaths of the coronary vessels and their ramifications, penetrating into the intermuscular tissue and separating the cardiac fibres, causing them to atrophy (Boyce). Secondary cancerous deposits, both epithelial and medullary, have been met with in the substance of the heart at post-mortem examinations.

VII. PARASITES.—Hydatid is rarely met with in the heart of the human subject, but it does occur, as does also cysticercus cellulosae. The cysticercus of tenia solium is common in the heart of swine, and that of tenia mediocanellata in cattle; but they are rare in man.

Actinomycosis may extend to the heart from the mediastina and lungs.

Trichina spiralis, according to Wilks and Moxon, is never found in the heart, or extremely rarely.

R. DOUGLAS POWELL.

REFERENCES

DISEASE OF THE AORTIC AREA OF THE HEART

In formal presentation of this subject it is customary to divide it into two parts—into aortic stenosis and aortic regurgitation. To carry out this division, however, leads to some embarrassment: on the one hand, by far the larger number of cases of aortic regurgitation are attended with signs of interference with the issue of blood from the left ventricle; on the other hand, cases of stenosis of the aortic orifice may be attended with regurgitation. Again, the causation of the two evils is similar if not identical, and the determination of the one or the other event may be accidental; so that, although the clinical features of the two events, taken singly, are very different, as the two are often coincident it seems more convenient to take them together except in those sections in which their distinction becomes imperative.

Subject.—By aortic regurgitation we mean that in diastole some of the blood driven into the aorta returns to the left ventricle; when we hear the sound characteristic of this disorder the inference that the aortic valve is out of order is almost irresistible. A definite diastolic mmmur is heard in the areas of the murmur of aortic regurgitation is perhaps the surest diagnostic indication of its kind. Not so with aortic systolic murmurs: of such signs these are perhaps the least definite. I need no say that an "aortic systolic mmmur" may not be significant of organic disease at all; or if significant of disease about this orifice the alleged stenosis may be more apparent than real, the murmur may signify no more than a roughness or other deformity of the part implying no constriction of the orifice; nay, it may be consistent with dilatation of the orifice. Clinical clerks are far too ready to assume aortic stenosis in all cases of organic disease of this orifice revealed by a direct mmmur; aortic obstruction, though open to some objection, is a better name.
Causation.—The causes of the diseases of the aortic area of the heart (omitting congenital malformation, which is dealt with in another article, p. 697) are chiefly three; namely, infective diseases, mechanical strain, and atheroma.

Infectious diseases.—Of these, rheumatism, if of such it be, is the chief; syphilis perhaps comes second, for the poison of the other infections, such as diphtheria and influenza, fall rather upon the muscular structure of the heart than upon its valves or orifices. Syphilis will be considered presently. Acute endocarditis has been dealt with already, and Dr. Dreschfeld describes a case in which infective endocarditis fastened upon a ruptured aortic valve (p. 882). In its liability to disease, and in the nature of it, the aortic area of the heart is so bound up with the aorta itself that for the consideration of some part of the present subject the reader is referred to the chapters in the sixth volume on "Diseases of the Arteries" and on "Aneurysm" respectively. This community of suffering is seen especially in the cases of atheroma and of syphilis. Indeed, whether the aortic orifice is ever attacked by syphilis primarily and more or less exclusively is still a matter of some doubt. Pathological histology has not yet enabled us by inspection to recognise the differential characters of syphilitic disease. Gout and its associates, such as plumbism, seem to produce lesions not distinguishable from "atheroma," under which easy fitting name their agency may be included.

Acute rheumatism is by far the chief cause of aortic disease in persons under middle age; as is atheroma in those over this time of life. It is admitted, however, that acute rheumatism falls first, and as it were by preference, upon the mitral valve; when the aortic valve is implicated it usually suffers with the mitral, or after it. That acute rheumatism should attack the aortic valve primarily and exclusively is not unknown in our experience; we see it occur thus even in women, but it is unusual. With the mitral valve I need not say the reverse is the case: it may almost be called a rule that acute rheumatism of ordinary severity maims this part of the heart without implicating any other part; though the proximity of the mitral valves to the aortic, and the bond of fibroid tissue between them may gradually lead to an extension of chronic inflammatory or sclerotic change from the one area to the other—from the mitral to the aortic—without direct cooperation of the specific rheumatic factor. In other cases the simultaneous implication of both areas, or the rapid succession of inflammation in the two, together with the severity of the heart symptoms, indicates that the cause of the aortic inflammation is directly rheumatic. Yet even thus the invasion of the aortic valve in women is so much rarer than in men—that is, the coexistence of both mitral and aortic rheumatism is so much commoner in men than in women—that we are led to infer the not infrequent presence of some other factor in the double valvular disease, besides the acute rheumatism. This factor may well be mechanical strain. Some cases indeed of coexistent mitral and aortic disease after rheumatism we may regard as confirmatory exceptions; such, for
instance, as the concurrence of aortic disease in women engaged in labours harder than those usual in the sex—in women who have worked in the fields, in washerwomen, in women employed in brick-making, or on the bank-tops of mines. Making every allowance in such cases for the greater exposure to weather, there seems to be a greater prevalence of aortic mischief in such women after acute rheumatism than among women who have led lives of less muscular stress. I have not found that either in alcohol or syphilis we have factors to invalidate these propositions; but to pursue them much farther would be to trench upon the subject of endocarditis entrusted to abler hands than mine: suffice it to say that I conceive that although in an unusually severe attack of acute rheumatism both sets of valves may be attacked, whether directly in each area or by extension from the mitral to the aortic, yet in ordinary attacks, if the aortic valve be involved as well as the mitral, it will often appear that the patient, either in work or play, has been wont to put out considerable muscular exertion. Disease of the aortic valve alone is a most unusual event in the young subjects of chorea (of 250 cases Gowers found aortic regurgitation in two, and obstruction in one), whose muscular efforts are fitful, not exacting; indeed its association with mitral disease in this disease is rare enough.

The predominance of rheumatic inflammation on the left side of the heart is often explained likewise by the fact that mechanical stress falls more hardly on these valves than on those of the right side, and so it may be; yet it is not easy thus to explain this preference: are we to assume that muscular labour in these persons had already produced some cardiac strain, and that before the rheumatic attack these structures were more or less impaired? This would seem to be a grave charge against the physical uses of the body; a charge which on the face of it seems unreasonable, if so be that without the rheumatism no harm would have come of them. A remote suspicion of such a deterioration can scarcely justify us in discouraging all exercises beyond nursery games. Short of lesion one would anticipate that increased work would enhance nutrition, and thus fend off rather than invite the approach of disease. Roy's article on the elastic properties of the arterial wall may, however, be usefully consulted on this problem.

Syphilis is probably concerned in the causation of many cases of aortic disease, though, except when it exists in the form of a definite gumma, we have no certain test of the syphilitic process, whether in the living or in the dead body (p. 905). How large a factor, however, syphilis may be in arterial disease will be shown by Dr. Mott in the article on this subject in the next volume. Dr. Parkes Weber (95) finds that syphilis is apt to be the starting-point of atheroma. For many years I have been wont to infer from the state of the radial artery the effects of syphilis on the vessels of almost every man who had been saturated with this poison; and such surmises have been reinforced by the more direct observations of Dr. George Oliver. We can scarcely suppose that a destructive agency, so active as we know it to be in all other arterial regions, should be without effect in
the aortic area of the heart; yet in deciding in a particular case that an aortic lesion is syphilitic, we are confined to the inferences which may be drawn from the story of the case or from associated changes elsewhere—which indications may, indeed, bring us to a moral certainty. We know that a comparatively young man of otherwise healthy habit does not suffer from local disease of the aortic region of the heart unless it be in consequence of extraordinary muscular stress, of rheumatism, or of syphilis; so that although there may be no direct means of detecting the syphilis, yet if muscular stress and rheumatism be both denied, we fall back upon syphilis as we do with some assurance in the case of aortic aneurysm in such a person; the inference, pathologically speaking, may not be positive, but it is usually justified in practice. The following case illustrates these remarks (vide also art. "Tabes Dorsalis," in a following volume):

Dr. Pye-Smith reported a case of a man, aged 32, who died with heart disease, the physical signs being those of aortic obstruction and regurgitation. Rheumatism and chorea were excluded. Atheroma was improbable owing to the comparatively young age of the patient, who was, moreover, not subject to laborious work. After death there were no signs of rheumatic or infective endocarditis, but a patch of recent aortitis and deformity of the valve. The lesion was soft, injected, with a swollen, crescentic margin suggesting the advancing edge of a secondary syphilitic eruption of the skin; there was no atheroma. The only other evidence of syphilis was a fibroid condition of the testicles, though this was not very marked. He suggested that the syphilitic aortitis had spread to the valve and so produced the disease in question (70).

Atheroma.—A full discussion of the nature and fashion of this disease or chapter of diseases of the arteries is deferred to the next volume. Here we may ask whether the disease of the aortic orifice sheds any light on the origin of this insidious and rather peculiarly human disease. Does it appear that muscular labour plays any important part in the origin or determination of the change? For my own part I cannot say that, likely as it may seem at first sight, there is much evidence in favour of this hypothesis. It is true that this disease also is found more or less exclusively on the left side of the heart—the side of stress; it is also true that atheroma may be the ultimate form of arteritis of whatsoever origin—rheumatic, syphilitic, or mechanical—mechanical, as in the pulmonary artery in mitral stenosis; still we must admit that atheroma is as likely to occur in the elderly lady who has spent her life in trotting amiably about the parish, as in her husband who has ridden, for his falls, felled his own trees, and stumped about after his birds from his boyhood.

Again, atheroma is by no means constant or approximately uniform in its position: although well marked, no doubt, on the greater curvature of the arch where tensile strain is highest; and at bifurcations and reflexions, yet it does not by any means confine itself to the parts which receive the main stress of muscular exercise, or to parts where, elastic tissues being most abundant, tone is least and tension most. On the con-
DISEASE OF THE AORTIC AREA OF THE HEART

It is one of the surprises of practice to find it in all sorts of odd areas; and within such areas it is patchy. If in one necropsy the cerebral vessels are like branched corals, in another, with atheroma elsewhere, the cerebral vessels seem clean enough. In one body atheroma is abundant about the region of the heart; in another, the heart and its orifices are fairly normal, but extensive patches of atheroma are discovered in the abdominal aorta or in peripheral areas of the arterial tree. Such contrasts are too well known to need the support of recorded cases in this place.

Again, is it that the main cause of atheroma of the heart is mechanical stress, yet stress due not to muscular exercise, but to that more persistent high arterial pressure of constitutional origin which may be established as well in the squire’s wife, with her indolent habits and gouty inheritance, as in the sportsman himself who works off his meat and drink day by day in the fresh air? Is the comparative freedom from atheroma enjoyed by animals to be attributed to the fact that they do not suffer from chronic high arterial pressure; that they have exercise enough—muscular stress enough, many of them—but are fed by their owners, and fed therefore economically? Certainly we see daily that hard exercise keeps the ill effects of a too vigorous appetite at bay. This is clinical gossip, I fear, rather than science; but we cannot at present get much nearer to the facts. Frequent high blood-pressure, then, as in excessive muscular stress, and more persistent high pressure due to luxus-consumption relative or positive, to gout (especially in its non-articular forms, for the frank articular form of gout leads less surely to high arterial pressure), to lead poisoning (by way of gout), and possibly to certain products of metabolism engendered in old and defective organs or tissues, may produce atheromatous changes which often involve the aortic region of the heart, directly, by friction and local irritation as in central and distal arteries, or indirectly by more immediate mechanical strain as in more central arteries. To quote Dr. Balfour, “there is a consensus of opinion that the arterial system is that upon which the finger of decay is first laid.” We see daily in the post-mortem room, yet still with some surprise, how readily the heart even of an old man may take upon itself no puerile hypertrophy. It is no unusual thing to find a big heart, and one big with no bad stuff, in old persons subjected in later life to increased blood-pressure, even when the coronary arteries have undergone some measure of deterioration; in such cases the aortic valves, even if competent, are practically always thickened. Still, with all this, can we say that aortic regurgitation, common as the disease is, is frequently found in the decay of elderly persons? I think not; on the other hand, it seems much less common in them than aortic obstruction—by which I mean no more than the presence of an organic direct murmur; now an aortic systolic murmur may continue as long as life holds together, and afford one of the many evidences of the long story of cardio-arterial degeneration. This form of aortic disease is rarely of itself the immediate or proximate cause of death; we may call it but an accident in the course of a general cardio-vascular involution,
which was described almost as well by our grandfathers, before auscultation was a popular accomplishment, as by ourselves.

Muscular strain.—The effect of bodily exertion in producing disease of the heart, which was apprehended by Morgagni, had again been overlooked in the study of the effects of rheumatism until attention was recalled to the subject by Peacock (64). Myers, Da Costa, Seitz, James Barr, and others, including myself, followed in the investigation. That muscular exertion is among the causes of aortic disease, and especially of aortic regurgitation, is now admitted on all hands. If, indeed, a man under forty-five years of age presents symptoms of aortic regurgitation without mitral disease, and without indication of syphilis, we may well suspect that muscular effort in one way or other initiated the disease. [Vide also art. "Mechanical Strain of the Heart," p. 841.]

That sudden muscular stress may damage the healthy aortic valve, even to the point of rupturing a limb of it, is now well known; the accident is not uncommon, and the cases on record are so many as to make it unnecessary to cite examples of it. Peacock in his Croonian Lectures adduced seventeen such cases. It is more difficult to estimate or to apprehend the part taken by muscular strain in the production of aortic regurgitation of insidious origin. When a vigorous and fresh-complexioned man of some thirty-five years of age, carrying a heavy patient on a sudden emergency up a flight of stairs, feels a sense of something having given way inside his chest, and becomes suddenly breathless and oppressed; when thereafter a murmur of aortic regurgitation is heard, which murmur continues to the end of a life prematurely cut short by this disease; when, moreover, no trace of syphilis can be even suspected by himself or his medical friends, we can scarcely err in deciding, in accordance with many other cases of the kind, that by the sudden stress he strained and ruptured a previously healthy valve. Again, when a young, slightly-built housemaid of very healthy stock presents the ordinary signs of aortic stenosis without any other lesion, and no rheumatism, chorea, or other sign of infectious disorder is to be heard of; and when, again, she tells a clear tale of a sudden sensation of pain and distress in her chest, while she was lifting a heavy bed, from which moment she became incapable of exertion, can we avoid the conclusion that during this effort an acute valvulitis was set up with subsequent constriction? 1 Peacock also stated that aortic disease is to be found in young women servants subjected to straining efforts before they are fully grown. Such cases scarcely admit of more than one interpretation. Interpretation is less easy when, in a person the subject of aortic regurgitation, we learn first that there is no definite story of a sense of injury on any one occasion; that the oppression came on more or less insensibly; that the patient has been in the way of syphilis, of alcoholic excess, or

1 This patient has been in Addenbrooke's Hospital twice at least, and during the University examinations her case has been investigated by many physicians; and the view here given of the causation of the mischief generally accepted. The signs are those of stenosis of the aortic orifice, and the symptoms those of increasing "stenocardia."
of some other cause of arterial degeneration, while, at the same time, he has been, following a laborious employment: yet we may fairly presume, perhaps, that in many such cases muscular stress and decay together have gradually impaired the valve to the point of insufficiency. As I have said, senile decay alone does not usually cause aortic regurgitation; more commonly it produces disease of the aorta with implication of the orifice, which is betrayed by a direct murmur. When, therefore, we find that regurgitation occurs rather in persons under fifty years of age, of the laborious sex, and especially in men who have been engaged in heavy toil, we cannot escape the suspicion that muscular stress, if not the sole or always the chief agent in these cases, is at any rate a potent determining cause. Acquired aortic disease in children is one of the rarest of clinical cases; even in the acute rheumatism of women and children we have noted that the mitral valve is affected first, and that if the aortic be involved it is as it were by overflow (p. 908).

Once more, if not infrequently, yet almost exclusively in men, we discover that aortic insufficiency establishes itself in patients under the age of senile degenerations, free from evidence of syphilis or other infection, including rheumatism, and telling no story of sudden rupture, shall we not be justified, at any rate in the large majority of instances, if we suppose that the disease may be attributed to the accumulated effects of muscular strains recurring at longer or shorter intervals over a number of years? Finally, if a man of irregular habits, and deteriorated tissues, describes to us the symptoms of sudden rupture of the aortic valve, we may reasonably infer that an effort, inadequate to rupture a healthy aortic valve, had sufficed to rupture a valve already impaired.

Thus, in the causation of aortic insufficiency due to muscular strain, we are led to recognise three classes, namely, acute rupture; chronic forcing of a valve previously impaired by some constitutional poison, such as syphilis; and chronic forcing of the valve by the impurity of repeated strains none of which alone was sufficient to break down a healthy valve, but all of which, by molecular rather than massive strain, contributed gradually to increase valvulitis and to break down the resistance of the part. The condition of the tricuspid valve in protracted cases of mitral stenosis is an excellent example of chronic valvulitis due to stress. As in the case of the housemaid mentioned in the last paragraph but one, this strain of the tricuspid tends to stenosis. We may note in passing that, in respect of progno stis, it is important to know whether and for how long the ruptured valvular limb is supported by tissues otherwise healthy. Although I have said (p. 910) that atheroma as a general disease of the arterial tree is not due, in the main, to muscular stress, yet local atheroma very often has this origin; it is indeed the common result of more than one kind of chronic arteritis.

It has been alleged that prolonged acceleration of the heart, as in Graves' disease, may produce the valvulitis of strain; but unless the sum of work done be considerably increased, which is not usually the case, such a result is not to be anticipated.

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External violence.—Finally, one or more of the aortic crescents may be ruptured by a blow on the outside of the chest. Within my own experience blows or crushes resulting in the split of a vessel after this manner have produced aortic aneurysm, not rupture of an aortic valve; still there are many cases of this kind on record. I distinctly remember, indeed, in the Leeds Infirmary a case of mitral stenosis which, after the closest inquiry, we confidently attributed to the kick of a horse in the cardiac area; the patient was a young man, and the symptoms were long in declaring themselves; yet the connection between antecedent and consequence seemed inevitable (p. 864). Dr. L. Heidenhain of Greifswald has studied these cases; his conclusions are that, with or without injury to the ribs or obvious external bruising, an external blow may (a) rupture a valve in the heart, may (b) damage or rupture the cardiac muscle, or rarely (c) set up a stenosis by insidious endocarditis. Sir Samuel Wilks has recorded a case of this kind which occurred in a youth aged nineteen; a blow on the chest ruptured the posterior cusp of the aortic valve from its free margin to its base. A small deposit of fibrin had begun to form on the raw edge. An analogous case, in which the heart itself was ruptured in a lad of sixteen, by a blow on the chest which caused no external bruise, has been reported by Dr. William Groom of Wisbech, and the preparation is now at Cambridge. Potain argues that if a blow, such as a jockey received who was heavily thrown so that his chest smote the ground, rupture an aortic cusp, the heart at the moment of the blow was in systole and the aorta distended. In cases of rupture of the mitral valve in like manner, of which he records two, he conceives the heart to have been in diastole when the blow fell, and the ventricle full.

Nervous shock.—In the earlier medical writers, not in the poets only, we often meet with the assumption that intense emotion may be attended with injury to the heart. In any careful consideration of this point we should divide the question: we should first consider injury due to interference with the circulation itself more or less directly; as, for example, by such an effort of the inspiration as to force the intra-thoracic negative pressures to an extreme; and, secondly, indirect interference through the nervous system. Of the first kind of case I remember a strange example in the West Riding Asylum at Wakefield. A woman, afflicted with violent mania, one day in a fury held her breath preparatory to a outburst; she became livid, fell to the ground, and died (93, p. 146). At the necropsy it appeared that death was due to extreme fulness and dilatation of the right heart and ventricle cave, though it is possible that it was due to a fulminating shock, by way of the vagus nerve, arresting the auricles. Of death through the heart, clearly dependent upon nervous shock alone, I have no experience. All that we know, as yet, respecting nervous influence on the mechanics of the heart, is that vagus shock by diminishing auricular contractions lessens the output of the heart, which is also slowed, and its diastole enlarged. This for the heart is a conservative function, but it is conceivable that, even in a healthy adult, it may be carried too far. The depressor effect, produced through dilatation
of the splanchnic veins, could scarcely harm the heart. The accelerator nerves are probably stimulated during emotion, which, as we all know, quickens the rate of the heart; but if, as we may presume, the output is proportionally less, and the resistance less rather than more (if the depressor be influenced also), no excessive mechanical strain thus falls on the organ. Augmentor action is too little understood to allow us to argue conclusively about it. Intense emotion might be attended with a universal or very widespread constriction of the peripheral vessels by which blood-pressure might perhaps be dangerously raised; if such a constriction occurs, however, it is transient, and relaxation of these vessels and of the sphincters seems to be the ordinary effect. Moreover, we see no great cardiac distress during a rigor, or after many returns of quotidian agony. So far as experiment goes, it would seem indeed that nervous shock tends to lower the blood-pressure. Finally, it is a reasonable surmise that some deterioration of the nerves or their centres, due to prolonged mental distress, might be followed by fatty degeneration of the cardiac muscle; such a process scarcely comes under the head of aortic strain, and it is at least as likely that such impairments of its nutrition take place by way of the blood. What may be the truth concerning these problems, however true it may be that prolonged grief may invalidate the chambers of the heart, such evidence as we have, physiological and clinical, seems to indicate that the aortic machinery at any rate is subjected to no especial stress, but, perhaps, rather the reverse.

Pathogeny and Morbid anatomy.—Whether the heart be liable to undergo primary hypertrophy under normal or relatively normal conditions is one of the most important problems which meet us at the outset of this section. The answer to the question is not yet given; but the opinion that it is so liable is not without strong support (p. 903): so far, indeed, as my reading goes, I think that the affirmative opinion is gaining ground. For my own part, I find that to be assured of the lesser degrees of hypertrophy of the left ventricle is a much more difficult clinical task than I used to suppose; unless, of course, the hypertrophy be attended with notable dilatation, and a clinical dilatation would surely forbid us to describe the conditions even as quasi-normal. Unless the person under observation be an inordinate drinker of fluids, alcoholic or other (p. 902), there seems no reason to anticipate increase of the mean ventricular output; if, however, the sum of the conditions of resistance is higher in amount than under ordinary circumstances, the supervision of hypertrophy may be explicable; and in this case distension might no more take place than under the fluctuations of output in persons leading a life in which muscular stress is not an important condition. Prolonged exertions in untrained men make themselves felt, as we have seen (p. 849), by more or less uncompensated dilatation; but perhaps in men such as sprint runners, putters of weights, wrestlers, and the like, in whom sudden and repeated efforts, under which initial rises of pressure are frequent, bear a large proportion to more regular exercises, the mean blood-
pressure may rise, as the maxima are high and of very frequent recurrence. If this be so, simple hypertrophy may follow, though I find such a result very difficult to verify. However, healthy men do not come to the doctor, and in unhealthy men the conditions no longer apply. In slim, long-chested youths with wide costal interspaces a thumping or uncovered heart may well be mistaken for a hypertrophy; and a great many young men have rather thudding hearts. Whatever books may repeat, it is no easy task to appreciate a moderate hypertrophy of the left ventricle, as many are the sources of error, as for instance in the relation of lung to the cardiac area, in chests of different build. Violence of impulse is by no means directly related to the volume of the heart or to the blood-pressure; the "heave" in the impulse, a quality not insignificant when the hypertrophy is considerable, may be hard to appreciate in the degrees of hypertrophy we are now contemplating; and a slight vertical displacement of the apex is no less difficult to ascertain, seeing that the form of the chest and its landmarks are far from constant. The researches of Myers, Da Costa, Thurn, Fraentzel (29), and others, on hypertrophy of the heart found in men submitted to physical stress, were made chiefly upon soldiers (vide p. 851). In these men, however, contingent conditions have to be considered: omitting drink and syphilis, many ill-fed, untrained, half-developed recruits are (or then were) clad in ill-fitting clothes, girted with belts and breast-straps, loaded with 20 lbs. and more of weapon and kit, and unavoidably sent on long harassing marches, for which they are untrained. In civil life we see the muscular or nervous muscular evils which flow from like causes, and we see how tedious may be the recovery from them. Now, if we turn to sailors, to whom drink and syphilis are not unknown, but who are clad in easy dress and not "trash'd about," we hear nothing of cardiac hypertrophy.

It is said that in hard-worked animals, such as greyhounds and race-horses, simple hypertrophy of the heart unassociated with cardio-vascular disease is met with. I have referred this question to Professor M'Cadyean of the Royal Veterinary College, who replies, "I have not formed the opinion that an amount of muscular tissue notably above the average is ever found in the heart of the horse or dog as the result of great muscular stress, but that hypertrophy of the left heart is always the result of some morbid condition of the valves or of the arteries. . . . If muscular effort were a cause of simple cardiac hypertrophy it should be almost the rule in bus horses, and such is certainly not the case." Arguments from analogy must not have much weight until verified; and we must regard hypertrophy of a hollow viscus in a different light from that of a solid muscle such as the biceps; moreover, we have in the heart not only a hollow organ, but a hollow organ in incessant activity; finally we do not know that the heart of a sound man engaged in active pursuits is over-worked on the whole, seeing that the machinery for the equilibration of arterial blood-pressure is of miraculous efficiency (vide p. 472 and p. 846).

The load factor of the heart, the ratio between its average and its
maximum work, is ample; as Cohnheim impressed upon us, the heart has a large "reserve capacity." If, say, by partial ligation of the pulmonary artery the resistance be increased to three or even four times the normal mean, the arterial blood-pressure will remain constant, although the left ventricle may be doing three or four times its ordinary work. I must not delay any longer on matter which is dealt with in other sections, but these inquiries are germane to my subject, as we are inquiring first whether hypertrophy of the left ventricle is a quasi-normal consequence of muscular exertion, and, if so, whether it can of itself provoke aortic disease, in the adjoining section of the aorta or at the orifice itself. If excessive pressure is thus induced we may see how muscular stress may lead to aortic disease. Roy and Adami noted (vide vol. i. p. 123) that "when the aorta of a dog is suddenly and greatly constricted, and consequently the pressure in the proximal portion of the vessel greatly increased, the plasma of the blood is forced into the cusps of the aortic valves, and vesicles of lymph make their appearance on the under surface in that region where fibroid thickening is most frequent in cases of chronic high arterial pressure." This is probably the way in which the chronic inflammation of the tricuspid valve is established which not infrequently ensues upon mitral stenosis. Our study of muscular exertion, however, as we have seen, suggested to us that such work does not create a state of abiding high pressure, but of intermittent high pressures more or less compensated by a mean pressure habitually rather low.

Are we not led, then, to suspect that muscular exertion, unless very sudden and excessive, and attended perhaps by fixation of the chest walls, arrest of breathing, and by some phase of differential pressures in ventricle and aorta of which we know little—in which case we know that a valve may be directly ruptured—needs some other factor to bring about aortic disease? This factor may be one of the causes of constitutional high blood-pressure; or, on the other hand, it may be some debilitating cause, such as syphilis, anæmia, or "miserer," which so reduces the normal strength of the cardio-vascular textures that ordinary blood-pressures are high relatively to their feeble durability. A patient of mine, who had certainly done all that he could to strain his heart, if by physical stress it could be done, died of dilatation ("true aneurysm") of the aorta, a result put down unhesitatingly by his friends to over-exertion; yet I knew well, what no one else knew, that there had been an old syphilitic infection, and that not a few evidences of the infection, among which had been symptoms of encephalic arteritis, had from time to time betrayed its persistency. To my mind it was far from clear that muscular stress had the first place in dilating the great vessel, although, no doubt, the vessel once disintegrated by arteritis, muscular stress accelerated the evil. If we are to form a definite opinion of the part played by muscular stress in the causation of aortic disease with or without the intermediation of hypertrophy of the left ventricle, we must weigh with it in the particular case all other factors which may have conspired to the same end. If we except active destruction such as that of infective or severe
rheumatic endocarditis, it may be true that all aortic disease is due more or less to the effects of arterial blood-pressure.

Peter, Traube, and others have insisted upon a distinction between aortic disease originating in the heart itself, such as that of rheumatic valvulitis in young and otherwise healthy persons,—cases in which the cardiac affection is in its initiation a local disease,—and implication of the heart in a more general constitutional change such as syphilis or arterio-sclerosis—wherein the heart disease is but an expression of a general disease. These divisions have been distinguished by such names as "cardiopathy" and "arteriopathy." The distinction is worth making, though it has been made far too literary; in it too little heed has been taken of the tendency of cases in practice to defy these logical devices; and much harm is done, especially by French writers, in decorating the several phases of a continuous involution by imposing names which suggest differences in kind, or at any rate in quality, which do not exist. While primary cardiac disease tends to generalise itself, constitutional disease derives much of its peril from the cardiac factor; the series, starting, it is true, from opposite points, meet and overlap; thus, unless it be in well-marked extremes in the interpretation of which we are not likely to go astray, the distinction is often too artificial to be of much service. Still, on paper at any rate, the contrast is worth making as a study of origins, for reasons which have already appeared; and occasionally it may influence the prognosis and treatment of a particular case. The observations of Roy and Adami, already quoted, throw some light on the process by which blood-pressures, relatively excessive, set up that opacity and condensation of the valves which we set well marked in the aortic valve, and clearly enough at times in other valves also, under high blood-pressures; as the heart suffers so may the aorta (74), and atheroma may invade no small part of the intra-thoracic arterial structures. Whether, then, the initial injury be such as this, or it be a rheumatic valvulitis or a syphilitic, the valvular lesions may blend into a common form which we conveniently call atheroma; and, the line between the aortic area of the heart and the aorta itself being no line at all, we find this atheromatous change not only in the valve, but spread, and often widely spread, in the neighbouring great vessel also: thus the aorta dilates, its elasticity is impaired, its walls are diseased, and the heart, caught in a vicious circle, has to meet an increased resistance. For a full account of atheroma the reader is referred to the article on "Diseases of the Arteries" in the next volume; I may briefly say of the valves that the milky opacity of the acuter stages of valvulitis is followed by an increase of fibrous tissue, both in the fibrous ring itself, where it becomes excessive, and in the valves themselves, chiefly about their points of mutual contact and the corpora Arantii. With the deformities secondary to the contraction of these cicatrised tissues we are but too familiar: induration of the fibrous ring, or of the infundibulum below it, leads to stenosis; and of the limbs of the valve to their contraction, puckering, or cohesion.
Thus the valve may become incompetent, or the orifice contracted; or these results may be concurrent.

The following remarkable case of sub-valvular constriction is published by Dr. Langwill. The patient was a poorly-developed lad of nineteen. He complained of pains in the chest on exertion, though he worked as a foundry labourer till four weeks before admission. A strong systolic thrill was felt at the base, and a loud systolic murmur was audible five inches from the chest. The chief cardiac disease found at the necropsy was as follows:

Pathological Report on Heart, by Dr. Shennan, Pathologist to Leith Hospital.

Right auricle.—Normal; tricuspid orifice, 1·2 in. Right ventricle.—Nothing particular to note. Left auricle.—Endocardium somewhat thickened. Mitral valve.—Cusps slightly thickened, particularly inner cusp. Musculi papillares small, and fibrous at apex where they join the chordic tendines. Left ventricle.—Walls hypertrophied; cavity 3 in. long; thickness of walls varies from 1 in. to 0·5 in. There are a few narrow fibrous bands stretching across the cavity, at whose points of attachment to the wall there is marked thickening of the endocardium from old endocarditis. On passing the finger up towards aorta, it passes through a fibrous ring—0·7 in. diameter—about 1 in. below the aortic opening. This is continued on to the ventricular surface of the inner mitral cusp. In this position, and extending upwards from the ring on to the lower surface of the postero-external aortic cusp, is a narrow band of comparatively recent vegetations. These cover the lower surface of all the aortic cusps, which also show fibrous thickening and contraction—cone diameter of the opening being 0·7 in. Above the valves the aorta dilates slightly—1·2 in.—but in the second half of the transverse part of the arch begins to contract, so that at the upper part of the descending aorta the cone diameter is 0·6 in.

Thickening of a limb of the valve, says Prof. Hamilton, may lead to the formation of a relatively large spur which, by intruding into the area of the two other shrivelled cusps, may accidentally prevent regurgitation.

From such rough edges and points "vegetations" may sprout, and form fringes on the free or ventricular edges of the valve, rarely on its aortic aspect, whereby friction is increased and extended; and chronic inflammatory changes operate on the endocardium, where the diseased valve brushes it, on the valvular structures themselves, and on the corresponding aortic surfaces. Below the valve "ripple-marked" thickening of the endocardium, due to the strain of aortic regurgitation, has been demonstrated by Dr. Glynn and other observers. Hamilton reminds us that, in disease of the aortic valve, it is rather the base of it which is the seat of the mischief, while the cusps may even be free; in the mitral it is the edge of the cusps and their substance which suffer first. Conversely the valve may be very incompetent, while the orifice is as wide or wider than its normal section.

Stenosis depends often, but not always, on contraction of the fibrous ring; not always, for adhesion and condensation of the limbs of
the valve may narrow the orifice, converting it into a slit or funnel, as is so well known in the case of the mitral valve. In a case which recently occurred in Addenbrooke's Hospital, under the care of Professor Bradbury, the adherent margins were united along their surfaces of apposition, and the blood seems to have been forced through a casual chink so small as almost to evade discovery even on inspection. There was no regurgitant murmur during life, nor was any regurgitation detected by Professor Kanthack. In this case calcification was far advanced in the ring, no doubt, as well as in the valve; indeed, in stenosis attributed to the valves the ring is usually concerned in the mischief also. Similar cases have been published by other physicians.

I need scarcely say that the presence of "vegetations" and of other detachable fringes on these dog-eared cusps is a matter of far more than local importance, as by their means embolism may come about.

Ulceration of one or more of the limbs of the valve is always a perilous process. When dependent upon micro-organisms, and we cannot say how frequently they enter in, the process may be terribly destructive, as the records of infective endocarditis give us too much reason to know; on the other hand, decay or perforation may be very gradual, and not always due to infection: probably in chronically diseased valves it may be a mere mechanical disintegration. Perforation of a segment is said to betray itself by a piping quality of the regurgitant murmur. Other rasping or "musical" qualities of these murmurs are attributed to the projection of spurs or shreds of segments which, fluttering or vibrating as reeds, give peculiar qualities to the sounds. It is commonly said that murmurs may be generated by a rough surface, as a brook murmurs over pebbles; this assertion must be taken with considerable reserve, for to produce a murmur the column of the blood must be broken: this a merely mammillated or corrugated surface will not do unless the eminences be such as to set up vortices around or behind them. The common notion that murmurs may be generated in a rough aorta without any contribution from the valves or orifice, is improbable and not supported by experience. If, in the absence of any cause in the valvular area, such murmurs arise, which is rather doubtful, they are to be attributed to dilatation of the aorta, wherein vortices may form between the slower external and the swifter internal layers of the issuing blood. However, we meet with cases every day of advanced disease of the aorta in which no systolic murmur is heard. Again, that there is more than the satisfaction of an anatomical curiosity in the endeavour to fix the incompetence or the obstruction upon this limb of the valve or that I am indisposed to believe; nor can a leaf or stump of a diseased segment hamper the access of blood to a coronary artery, unless, of course, it so adhere to the wall of the aorta, or the mischief so extend from it, as to choke the mouth of the vessel. That the propagation of a regurgitant murmur in this direction or that can indicate the limb of the valve affected, or chiefly affected, is not very probable in itself, nor is it borne out by experience, and that the deformation of one particular limb of the valve should affect
the coronary circulation more than the same incompetence in another
is impossible; there cannot be differential pressures within this area.
Congenital peculiarities of the aortic valve and coarctations of a con-
genital origin are dealt with in the article devoted to this subject
(p. 706). In simple rupture of a healthy valve one limb only is torn in the
large majority of cases; but a few cases of the rupture of two limbs are
on record. Generally the limb is rent on the free edge, but sometimes
it is torn from its attachment to the vessel.

Whether these chronic changes in and about the aortic orifice lead
to regurgitation or to stenosis without incompetency, crucial as the
distinction is in clinical medicine, is pathologically a matter of accident;
the result depends, that is, on contingent causes. At the same time
it is well to remind the pathologist that to test the competency of an
aortic valve by means of a column of water, a test which is more useful
in the post-mortem room than one might expect, is insufficient in a
doubtful case, unless the height of the column of water be equal to the
maximum aortic pressure—to the pressure, say, at the moment of aortic
diastole of 180 mm. Hg. Moreover, the water may even then escape from
the coronary arteries. Practically, as the water brings the valves into
apposition we have to judge of their competency by the eye.

Of "gouty valvulitis," of a primary kind, after the manner of
rheumatic valvulitis, and apart from the chronic sub-inflammatory and
degenerative changes in the aorta resulting from abnormally high arterial
pressure, I have no knowledge, either pathological or clinical.

It is very important to remember that these degenerative changes
involve the area and, sooner or later, the orifices of the coronary
arteries; so that the heart, instead of enjoying that increase of nutrition
which its greater work demands, and which at first the hypertrophied
ventricle supplies, may receive, after the first stage of the malady, con-
siderably less than its normal nourishment.

It is commonly asserted that insufficiency of the aortic valve may
come about, not from any defect in its own structure, but from dilatation
of the aorta, whereby the sectional area of the orifice is enlarged.
Intermittent or temporary aortic regurgitant murmurs are thus ex-
plained. Barić, a careful and experienced observer, reported thirteen
cases of aortic regurgitation from widening of the orifice without
disease of the valves; but a persistent slapping second sound cannot be
taken as definitely indicative of a normal aortic valve. Vierordt assumes
that in weak dilated hearts dilatation of the aortic ostium may cause
"relative Klappeninsuffizienz."

Cases of alleged temporary aortic regurgitant murmur are few and need
very careful interpretation. My own conviction is that if such cases be
followed up, the regurgitant murmur will be found permanently estab-
lished at no distant date; as in Dr. Hermann Weber's very interesting
case (p. 946). That dilatation of the aortic orifice does often occur is
well known to every pathologist; but I have never happened to meet with
such an increase in the sectional area of this orifice as to permit of
regurgitation without disease of the valve; however, a few specimens of
the kind are to be found in museums. Professor Osler (63), on Bénéke’s
authority, tells us that “the aortic orifice, which at birth is 20 mm.,
increases gradually with the growth of the heart until at one-and-twenty
it is about 60 mm. Of this size it remains until the age of forty, beyond
which date there is a gradual increase up to the age of eighty, when it
may reach from 68 to 70 mm. Thus at the very period of life in which
sclerosis of the valve is most common, there is a physiological tendency
toward the production of a state of relative insufficiency.” But when I turn
to Osler’s opinion on the point before us, I find that “relative insufficiency
of the sigmoid valves, due to dilatation of the aortic ring, is a rare condition”;
he adds, “Indeed I have myself never met with a pure instance of the kind,
for in such cases I have always found the valve segments involved with the
arterial coats.” I may repeat once more that aortic insufficiency is not
eminently a disease of old persons, but rather of persons about or under
middle age; there is no difficulty, of course, in collecting cases of aortic
regurgitation due to senile arterial disease,—I have such a case under
my occasional observation at present,—yet the prevalent effect of aortic
disease in the old is obstruction. Again, although in elderly persons,
and in younger men the subjects of syphilis, we meet with considerable
and even enormous dilatation of the aorta, yet even in these cases aortic
regurgitation does not generally appear unless there be disease of the
valve itself also, for the orifice is prone rather to stiffen than to yield.
Whether then regurgitation, permanent or temporary, may arise directly
out of a mere dilatation of the aorta, if no longer an open question, is a
rare event; and, as the accompanying tracing shows, the incompetence is
slight. I gather from Prof. Tigerstedt’s new volume
that in his opinion the semilunar valves are efficient
under conditions of considerable relaxation, whether
of heart muscle or of the supporting structures; and
Professor Sherrington (p. 466) states on experimental
evidence that the limbs of the valves may aid each
other by mutual readjustments. Dr. Newton Pitt
has recently investigated this matter (67).

Sometimes, as Corrigan showed, on examination
of the aortic valve after death from whatsoever
disease, its segments are found atrophied; the flaps
are thin, and not infrequently “fenestrated,” especially on a line parallel
to the free edge. It is alleged that these conditions are not necessarily
morbid or mischievous; if, on overlapping margins they do not give
rise to regurgitation. Aneurysms of the parts about the valves need no
discussion here, as their pathology is dealt with in the article on Aneurysm.
Nor will I stay to discuss such pathological curiosities as morbid growths,
polypi, and the like.

The effect of aortic disease on the other valves and orifices has been
carefully studied by Professor Hamilton. Aortic regurgitation, as he
observes, is “anticipated in its injurious results on the other orifices
by its own peculiar sources of mortality." From his measurements, however, the following results appear; namely, that, unless in addition to the incompetence of the valve the aortic orifice be dilated, "the effect upon the size of the other orifices is nil"; if, however, the aortic orifice be dilated, a general distension of all the other orifices is apt to follow. "Constriction of an incompetent orifice, then, exerts a salutary effect"; so far, that is, as stress on the other orifices of the organ is concerned.

Simple stenosis of the aortic orifice, in the strict sense of the word, seems to be a rare disease. I find that both Osler and Hamilton speak of it as beyond their experience. Fagge and Pye-Smith speak of it as "most rare"; Frøenfelt as "ein seltener Herzfehler." As Frenchmen will not put indexes to their books, I cannot say what their experience may be. For my own part I should say with Frøenfelt that the condition is rare, but not excessively so. It has happened to me to see many cases of mere aortic stenosis without any trace of regurgitation, and to have verified not a few after death, the last case being that of Professor Bradbury’s, to which I have already referred. Aortic stenosis is a long disease, for life may continue under favourable circumstances until the aperture is reduced to the size of a crow quill or less. The chink by which the blood found access to the aorta in Professor Bradbury’s case was only discovered on the closest search after death. It is commonly said that aortic contraction in this simple form is the result of chronic endocarditis; that aortic stenosis is connected with arterial disease which spreads down from the aorta. But in St. George’s Museum are a number of cases of well-marked aortic stenosis, and in many of them the aorta seems healthy. The stenosis seems to protect the aorta in spite of the high velocity of the "choke bore." Most of these cases occur in persons of fifty years of age and upwards, in whom the incident is usually due to "atheroma." In younger or sounder persons it is often fibrotic. In these cases the effects of aortic disease upon the left ventricle are most clearly seen; it is in them that hypertrophy takes its simplest form. In so far as the aortic orifice is narrow, the inner surface of the left ventricle is protected from the pressure of "recoil."

In diseases of the aortic valve, as of other parts of the heart, our attention may be too much given to murmurs; the working calculation which we have to make is the effect of the lesion on the chambers, for by their efficiency the organ stands or falls, at any rate for a time. In stenosis the left ventricle may approach that mythical type "concentric hypertrophy." In regurgitation, especially if attended, as it is wont to be, by dilatation of the aorta, the ventricle is at least as much dilated as hypertrophied. The pathogeny of this event has been much discussed, and the outcome of the discussion is that this dilatation is due to the recoil of blood from the aorta upon the wall of the ventricle in diastole. Besides this resistance head in the arteries, that large fraction of the force of the systole which is stored up in the aorta in its diastole is expended not only upon the forwarding of the blood, but in large part also upon
the inner surface of the ventricle. It is incorrect, then, to describe this force so released as "wasted." It is often stated that the dilatation is due to the filling of the ventricle from two sources; but it cannot matter whether the cavity be filled from two sources or from twenty; the matter is not one of the accessibility of blood, but of the resultant intra-ventricular pressure. The aortic pressure is no doubt so much greater than the auricular that the latter may count for comparatively little; yet the resultant pressure is not the sum of the two; it will lie between them—the amount depending upon the relative pressures at the respective orifices. If, for example, the pressure of the current returning from the aorta = 100 mm. Hg., and that from the auricle = 20 mm. Hg., the resulting pressure on each square unit of ventricular surface will not be 120 mm., but a quantity somewhere between the two numbers; and the resistance of the aortic stream, being greater than that of the auricular, will head back the latter more or less, according to the degree of its excess. Furthermore, this heading back will partially close the mitral valve, and fill the ventricle still more with refrent aortic blood during its diastole: otherwise we should find the auricle still more distended from the aortic head. From experiment upon animals it seems that, on suddenly produced insufficiency of the aortic valve, the aortic pressure may be so great as to cause rupture of an unprepared ventricle. The distress felt on rupture of the valve in a straining man is probably due to this distension of an unprepared ventricle. Usually, however, "reserve capacity"—the factor of safety—sustains the arterial pressure till the ventricle can grow up to the new call upon its strength; if it rupture it will give way at its weakest point, but to speak of the regurgitating stream "impinging on the inner surface of the apex of the left ventricle," and "of the repeated blows of a jet of blood disabling the ventricle," is to regard the cavity as if it were the pan of a water-closet. Writers on heart diseases are apt to lose the conception of the heart and arteries as a plenum. If the heart be regarded as a screw with reserve driving power at command, both dynamic and static, why should not the work still be done? We shall see, under the head of symptoms, that the work is well done for an indefinite interval during which the patient is usually unaware of any defect in his circulation. The failure comes about, partly because "compensation" is never complete, partly because of the excess of pressure of aortic blood over auricular upon the inner surface of the ventricle: were the auricular blood impelled under a pressure at least equal to that in the aorta the valve might be dispensable. The dilatation is due to the abnormal pressure of aortic blood, — abnormal, that is, in respect of the pressure which the parts have ordinarily to bear. If the ventricle of a frog beat in a tonometer under a supply of blood from a pressure bottle at varying heights, curves may be taken to measure the volume of the ventricle; and as long as the pressure from the bottle remains constant so long the line of the volume at diastole is remarkably level. Increase the pressure slightly and the diastolic line immediately sinks, showing
greater capacity, even though the height of each systole may be as before (Gaskell). The distensible force being greater, the corresponding increase follows a well-known physiological law. In like manner the increase of the muscular coat of the smaller arteries described by Sir George Johnson and Dr. Savill comes about in the course of resistance to distensible forces; and I may add that, as in the heart, this overgrowth is intimately associated with degenerative implications.

Dilatation gains on hypertrophy, as Starling clearly puts it, because, although a loaded does more work than an unloaded muscle, the amount of contraction (that is, the height of the lever) is less. The cardiac muscle may be more tense, and the contraction therefore more powerful, but it is not equal to the increased length of the muscular fibres; thus some dilatation remains, the residual blood is more, and the output less. On the next diastole the heart is overfull, but even under this increased stimulation only the normal inflow is sent out: arterial pressure is thus kept up, but work is increased and hypertrophy should follow. Not only has some of the output to be lifted again, but the backward motion of the reffluent blood has to be converted into a forward motion. It is often said that the heart attains a larger bulk in aortic regurgitation than in any other disease; this is, generally speaking, true; but in chronic Bright's disease the "cor bovinum"—the "heart of a pantophile," as Voltaire called that removed from Diderot's body—may attain to no less a bulk; that is, say, 2-3 cm. at greatest thickness and 1-2 cm. at apex. It is usual to speak of this enlargement as a compensation of the defect it counteracts. There is no objection to this expression if it be remembered that it is a figurative one; all we know is that increase of function within certain limits is followed by hypertrophy; it is not in physiology only that function creates structure: yet if in respect of one factor the difficulty is postponed, the readjustment, as we shall see, brings other evils in its train. We do better, then, to get rid of these teleological connotations, and to regard the hypertrophy of the ventricle simply as the result of increased function, whatever the consequences. How does this alleged compensation break down?. The late Mr. George Busk once reminded me that all muscular overgrowth may be transitory, owing perhaps to the transitoriness of all conditions less stable than the normal; he then adduced for the first time the example of the hypertrophied biceps of the file-cutter, which is said to fail after a certain number of years. But perhaps the failure is a particular instance of the general truth that a disproportionate increase of one part of a system disturbs the relations of all associated parts, and it begins to rock: hypertrophied engines in swift passenger ships mean a shorter life for the ships. Not to look beyond the immediate neighbourhood of the heart, the aorta is strained under the immoderate stress; it dilates; atheroma, the effect of strain, being usually found just above the valve, implicates in time the mouths of the coronary arteries; from the first these arteries, like the rest, are injured by the excessive percussion, and perhaps from the first the sudden and early fall of pressure
in the aorta may be greater than the higher pressure with which they are filled on systole. Thus areas of the cardiac muscle come short of blood; fatigue is cumulative, and fibroid tissue, which is more economical to feed, supplants the active muscular fibres. If the aorta be unhealthy to begin with, this disintegration takes place so much the sooner. [See also the postscript to this article.]

The only other point to which I must refer in this place is the effect of a persistently large residuum of blood in the left ventricle on each contraction. The most recent observations seem to indicate that the healthy ventricles never quite empty themselves — that there is always some residual blood, some difference between contraction volume and output; if this be so, how much more must this be the case as the work of the ventricle, distended under higher pressure, increases as the cube of the radius of curvature. This consideration alone, when we recollect the cumulative effects of fatigue and the many incidental causes of atony of the heart, may go far to account for the wane of compensation. On the other hand, in aortic insufficiency the ventricle contracts against low resistance, in stenosis against high resistance, so that the output must be far more in the former, as indeed the upstroke of the sphygmograph shows us that it is. In the normal state the blood-pressure falls suddenly in the ventricle, gradually in the aorta; in regurgitation it falls suddenly in the aorta also; moreover, in insufficiency the pulse-rate is usually more rapid. That dilatation is nevertheless the feature of insufficiency rather than of stenosis would indicate that mere residual blood is not the predominant factor in dilatation which is usually supposed: excessive contraction volume is probably far before it in this effect. It is as cardiomotive energy begins to fail that residual blood becomes so grave a condition in dilatation. Finally, in regurgitation dilating stresses tell on the ventricle when this muscle is relaxing, in stenosis when it is contracting. When regurgitation and stenosis occur together the results will be compounded of the characters indicated for each alone, stenosis probably having some protective effect.

The consequences of aortic disease are so often confined to its own sphere that disease of this part has a character of its own; the consecutive pathological changes which, if the patient survive, make themselves felt sooner or later in other parts of the heart, need not detain us. It is far from uncommon in aortic regurgitation, after long persistence even of its extremer symptoms, not to detect any implication of other parts beyond thickening of the mitral cusps under the effects of the hypertrophy of the left ventricle: under increasing dilatation, however, 'the mitral valve may give way, and the patient may die of mitral rather than of aortic disease, although death with dropsy may occur without any audible mitral regurgitation.' It has been said that forcing of the mitral orifice with moderate regurgitation gives relief to the overstrained arterial circulation; such a temporary effect it may have for a while, but it is the opening of one more of the gates of death.

The pathological changes in the arteries, due to their high tension
under the heavy beating of the heart, do not differ in kind from those to be described in the chapter on "Diseases of the Arteries."

In senile aortic disease, emphysema and other evidences of decay too frequently increase the burden of the patient's latter days. The pathology of these associated degenerations is described elsewhere.

STENOSIS.—Symptoms and signs.—The invasion of stenosis, as of regurgitation, is often long latent. While speaking of the causation of aortic stenosis I said that this disease, standing alone, is a rare one; aortic systolic murmurs are, indeed, among the most frequent of clinical signs, but in many cases, even if organic, they signify no more than a deformation of the orifice, whether sectional area be diminished or not; again, in many cases in which this area is diminished the valve is also incompetent, and the case is no longer a simple one. To understand stenosis we must study it in its unmixed form. It has been my fortune to see not a few of these cases, and I cannot begin better than by a brief record of one of them. Mr. X., a patient of Mr. William Hall of Leeds, became aware of an increasing oppression in the chest. Mr. Hall found a direct aortic murmur and hypertrophy of the left ventricle, and was good enough to ask me to see the case. Mr. X. was a man of about fifty years of age; he had never suffered from rheumatism, his life had been anything but laborious; there was no history of syphilis nor any evidence of this infection. He had always been a man of correct and domestic habits. Nor was there any sign of kidney disease or of general arterial disease; his arterial system, apart from the aortic valve, seemed to be no older than his years. As no great improvement came about, nor was likely to come about, Sir William Gull came down to meet us, the case being even for him a rare one, and he took the greatest interest in it; so interested was he that he took occasion to call on me on a later day to talk over the symptoms again and to impress the facts on my memory. Often I recall him as he sat in my room describing with his hand an imaginary cardiographic curve in the air—the portentously long upstroke, percussion it could not be called, while the heart was heaving under the hand as the back of some imprisoned monster; the curt diastole with faint second sound; the irregularly protracted pause as if the heart were slowly gathering itself together for another effort; the deliberate rhythm, some forty in the minute, in which each reluctant beat, stout as it was, seemed as if it might be the last effort; the small hard pulse; the subternal oppression, all these features combined to make a striking clinical picture. The slowness of rate necessary to compass as much output as possible was well illustrated in a case of this kind reported by Dr. S. West, in which the pulse was 30, and in another by Dr. Parkea Weber (96) in which the heart's beats became so slow as to give rise to syncopic attacks.

For the most part aortic stenosis appears in persons beyond middle life in whom this deformity is but part of a general decay: in such
persons the compensatory reactions may be less obvious; for, to take one point alone, the mass of the blood to be lifted—the cardiac output—is less in old persons than in such a subject as Mr. X.; nevertheless, as we have seen, even the hearts of old people can attain to no inconsiderable amount of hypertrophy; the old woman referred to on page 920 had a heart of 24 ounces, and apparently of good muscle.

Although it is true that the left ventricle, spared the recoil of regurgitation, does not dilate in stenosis as it does in insufficiency, yet it is untrue, on the other hand, to say that it does not dilate at all; the residual blood on each contraction may be large, and as the auricle gains a little in strength to meet the increased pressure in the ventricle, the contraction volume of this chamber is excessive, and some dilatation is inevitable. The enlargement, however, is more in the downward and outer than in the transverse direction; the dulness does not cross the sternum, or at any rate not until the later phases of the disease. Gradual and restricted as the output may be, the mean arterial pressure is fairly high—the heart being usually slow, the systole is not only strong but absolutely, though not relatively, protracted, as shown in the curve here reproduced; the pulse is thus "sustained." It may be that the arteries contract upon their smaller content. The aorta, on the other hand, is not dilated, at any rate not as a direct consequence of the stenosis; if there be no arterial disease to weaken it, the vessel being less distended is, theoretically at any rate, not increased in diameter, and may be diminished. The second sound will vary with the state of the valvular segments; if these be hardened the sound may have the "parchment" character; but it will always be short as the blood-pressure above them, even if of normal mean owing to the length of systole, has not a high maximum; and unless the vessel be drawn nearer the sternum it will not be loud because the sectional area of the aorta at its orifice is diminished. It may indeed be quite inaudible as in J. D. (Fig. 51). The contrast between the big heart-beat and the small pulse may be startling, in which respects stenosis

![Fig. 51.—James D., age 46, acute rh. wt. 7. Loud syst. m. in aortic area; no diastolic m., no second sound. P.M. No incompetence. (Graham Steell.)](image)

differs from regurgitation, wherein the pulse, although of brief duration—"collapsing," has a very high maximum. In regurgitation the "arterial tension" is enormous, as we see by the damage done to the structures. In stenosis, then, the protraction of the phases of the cardiac revolution makes up for the smaller delivery of blood into the aorta per unit of time. In Dr. West's case, as I have said, the pulse-
rate was only 30. Sir S. Wilks again describes the pulse in stenosis as "small and slow." Yet many cases of positive stenosis with a quicker pulse are recorded, wherein the rate may be due to cardiac failure or to a call from the tissues for more blood, a call transmitted through the accelerators. In strict stenosis, then, we ordinarily have a long slow pulse with a low maximum, unaffected by raising the arm; in regurgitation, on the contrary, a short pulse not slowed, of extreme maximum pressure, and far more injurious to the arterial tree.

The murmur of stenosis may be heard widely over more than the cardiac area; it is often heard at the apex and over the aorta in the interscapular region. The character of the murmur varies to some extent both in quality and in order. Sometimes its sound vibrations are attended with others less numerous, not rapid enough to cause a sound, but perceptible to touch as a thrill. I need scarcely say that these coarse vibrations need not indicate extreme stenosis. A thrill is often to be felt in stenosis of the mitral valve, but its position is then at or about the apex, whereas in aortic stenosis it is chiefly about the base. A thrill perceptible over a large part of the cardiac area but rather more towards the apex, is occasionally present in other labouring hearts—especially when, as in the arterio-sclerosis of old folk, the vessels become rigid while the heart itself remains vigorous and the blood-pressure high; but a thrill at the base is almost pathognomonic of aortic stenosis, whether in combination with other mischief or not. Hence we expect that the murmur also will, in part at least, be compounded of slow sound vibrations, and, whether "musical" also or not, will be noisy; we call such murmurs sawing, rough, or harsh. As the ventricle begins to give way under its toil the murmur will grow softer, possibly even to extinction; under digitalis also it may alter in quality, and the pulse may quicken in rate; then again the harshness of the murmur may return as the pulse slows down. The thrill likewise depends on the vigour of the heart; when strong, it may be felt in the vessels of the neck. As regards the order of the murmur, I have heard it sometimes in a post-systolic rhythm occupying the shorter pause; I have noted two very definite cases of this kind in private patients within the last few weeks; the first moment of systole was free from murmur, then followed a very brief murmur, and instantly thereafter a clear second sound—clear of murmur, that is, though in such cases rarely normal in tone. This is no "pulmonary" murmur.

I am interested to find that Vulpian also reports such a murmur so placed:—

A woman, 40, suffered severely from acute rheumatism; two years later she presented herself with mitral regurgitation revealed by the ordinary signs. At the base a roughish bruit was also heard; this basic bruit was placed between the two normal sounds ("entre les deux bruits normaux"). The murmur was heard also at the mid-precordial region, and upon the localisation of a rough short systolic murmur. The pulse was regular, small (rate not given) "et un peu concentré." The ascending sphygmographic line was ill-marked (très peu..."
Vulpian was bold enough to diagnose a contraction "soussortique" "une lésion de canalisation... à une certaine distance audessous des valvules aortiques."

Vulpian does not give his reason, it may lie in the increase of velocity as the ventricle contracts. Constriction in this place might be revealed by a "presystolic" murmur as recorded by Lemoine (quoted by Sansom); that is, by a murmur coincident with the earliest ventricular effort: such is Dr. Sansom's supposition.

In most cases, when the murmur of aortic stenosis is said to be in part "presystolic," this apparent origin of the murmur is suggested by the great protraction of the "prophygmic interval." This interval may indeed become perceptible to the finger. Ordinarily the murmur is a long one occupying the whole of the first phase up to diastole.

The propagation of the murmur from the second right costal cartilage depends much on the stage of the disease. If the murmur be loud—it is often loud enough to be heard at a distance from the chest—its area of diffusion will be considerable, both about the basic region and towards the periphery in the arteries. Thus it tends to gain an ascendency over other murmurs, and quite possibly by interference vibrations to alter or resolve them. When stenosis is extreme, however, it is said that the murmur may fail to reach the carotids.

**The anacrotic and the biaferiens pulse.**—It has often been said that the anacrotic pulse (Tracings Nos. 52, 53) is so marked a peculiarity of aortic stenosis as to be pathognomonic of this condition. This is certainly not the case; for my own part, I have found the pulse anacrotic in so many different cases of cardio-arterial disease that I would not go farther than to say that it suggests disease of the aorta or aortic stenosis; moreover, it is doubly inconstant, it is not persistent from day to day in the same case.

Our attention has been drawn more particularly to this matter of late by Dr. Graham Steell, who is good enough to allow me to reproduce some tracings of the anacrotic and of the biaferiens pulse respectively. Dr. Steell reported the behaviour of the pulse in four cases in which the observations on this formation were interpreted by autopsy. The author concludes as follows: "Three of the four cases bore out the belief that the anacrotic pulse is a valuable sign of aortic stenosis, provided the physical signs correspond. The fourth case taught, however, that pathognomonic value must not be attributed to this pulse, inasmuch as other conditions besides aortic stenosis may produce it (Fig. 54). Moreover, in cases i. and iv. the pulse was not constant in this character; in case iii., however, unalterableness of the pulse was a striking feature of the most definite case of all,
inasmuch as it was the least complicated. Such unalterableness of the anacrotic pulse is probably of great diagnostic value, although it may be rare.

Of the pulsus bisferiens (Fig. 55) Dr. Steell says that although cases of stenosis are so often associated with regurgitation it is not easy to find material on which to make conclusions regarding pure stenosis, yet we may

Fig. 55.—Margaret G., st. 23; rh. st. 14. Exemplary P. bisferiens. R. radial, double beat plainly felt; L. radial, ordinary tracing of aortic incompetence. Loud syst. m. and thrill in aortic area; diastolic v. P.M. Stenosis of aortic orifice with incompetence of valves; no explanation of difference between radial pulses. (Graham Steell.)

assert, on Mahomed's authority, that the pulsus bisferiens is consistent with pure stenosis. In his own two cases of pulsus bisferiens there was some regurgitation also. On the other hand, in some cases of regurgitation associated with the bisferiens pulsation stenosis was scarcely present, if at all (Figs. 56, 57); and again, for our yet greater uncertainty, we read that in Steell's cases the phenomenon was unequal on the two sides, and in one of them chiefly unilateral, the other radial assuming the character very occasionally
and imperfectly; a careful examination of the arteries concerned afforded no explanation of this peculiarity (Figs. 58, 59). In two cases, moreover, the phenomenon was manifested in the one on the right side, in the other on the left. Dr. Steell concludes thus:—"We are unable to explain the mode of production of these pulses; and I do not think we are warranted in affirming either that the anaerotic or the biseriens pulse is the direct result of aortic stenosis; both pulses are found, however, so often in association with aortic stenosis that we cannot deny them diagnostic value; of the two the anaerotic pulse probably possesses the greater diagnostic value."

As regards the anaerotic pulse Dr. Sansom virtually comes to the same conclusion: he emphasises the deduction that a persistently anaerotic pulse means organic disease, whether aortic or chronic renal; and that in case of doubt an anaerotic pulse might signify that a systolic murmur at the base of the heart is not anaemic. In aortic stenosis, as it seems to me, anaerotism is easy to explain: the pressure in the aorta is lower than normal, that within the ventricle is much higher; at the first moment, then, of the opening of the valve the blood issues readily, but as the stenosis throttles the wave the increased velocity of the blood is counteracted by the rising pressure in the aorta, and the farther delivery becomes slower and more laborious; though so long as the heart is strong the pulse is regular. Perhaps the most general expression under which we can bring the anaerotic pulse is that during systole the flow from the aorta to the periphery is at a slower rate than that from the ventricle to the aorta: in aortic stenosis the current issuing from the choke bore is of a high velocity; that from the aorta to the periphery, however, is slackened as the blood occupies a relatively large channel, an adverse condition which may be exaggerated by increased peripheral resistance; though on the other hand it is diminished by arterial rigidity, in which case, although the wave is accelerated, the blood-current is not.
Pain is not a constant feature of aortic stenosis; as in regurgitation it probably depends on aortitis, and is much aggravated by cardiac stress. In my experience neither anginal pain nor the sense of substernal oppression are so great, if as frequent, as in regurgitation. Still it is often pain, either acute or oppressive, which sends the patient at first to the physician. It is rarely severe, but may run down the left arm and serve as a warning to the patient when he transgresses the limits within which he must conduct his life. In some cases, however, the angina, whether an obstruction or regurgitation, may harass the patient even when at rest in bed (Douglas Powell, vol. vi.). Here I may digress so far as to state my opinion that the seat of the distress in angina pectoris is in the aorta, and not in the heart.

Dyspnoea.—In aortic disease dyspnoea is not so frequent nor so prominent a feature as in mitral disease; and this for obvious reasons. The substernal oppression of stenosis and of regurgitation is often falsely called dyspnoea; it is rather the complaint of the aorta and of an overworked left ventricle in distress.

There is not the same eccentricity of symptoms in stenosis that there is in regurgitation; there is less tendency to gastric perturbations, to headaches, to pains having the degrees of angina, to cough (cough depends rather on a dilated aorta), or to faltering of the mind and memory.

Diagnosis.—Latent as stenosis of the aortic orifice in its early phases may be, on the other hand I need not repeat that to infer this deformity in every case of basic systolic murmur would lead us into error. Not even in persons of advanced years, in whom in all probability such a murmur does indeed signify disease of this area, is it to be assumed, as too often it is, that the aortic orifice is positively contracted. We have already seen that aortic contraction without regurgitation is a rare condition; yet systolic murmurs in the aortic area are of the commonest of clinical events: in young persons they are usually due to perversions of the blood, in the elderly to atheromatous disease. We have then in the first place to decide whether in a given case an “aortic systolic murmur” is of the kind known as hæmio, or due to atheroma or other chronic arterial disease, such as the syphilitic. Of murmurs due to the atheroma degrees of endocarditis I do not speak; they are discussed in the chapter of “Acute Endocarditis” (p. 869).

In the first place, then, is a given aortic systolic murmur, hæmio or organic, dynamical or statistical? Neither age nor sex is conclusive: a young woman may suffer from aortic stenosis of a fixed organic kind, without regurgitation, and without any history of rheumatism, chorea, or other constitutional disease; how are we to decide that in her the murmur is one of stenosis of the aortic orifice? Well, in the first place, there may be no definite signs of anæmia, no venous hums, no characteristic blood changes, no change of intensity on varying her position; the first cardiac sound is inaudible. In anæmia a murmur may be loud, occasionally even harsh, and in stenosis the murmur may be soft; but a sawing sound, especially if associated with a thrill, would strongly suggest organic disease.
In stenosis the apex of the heart is perhaps a little displaced in the vertical direction, and the cardiac impulse not merely forcible, but merely violent, but steady, long, and heaving. The over-action of the left ventricle may be manifest, yet the cardiac dulness scarcely increased transversely. A substernal oppression may make itself felt on exertion, or even during rest, which differs altogether from the painless and more panting dyspnoea of anaemia. This oppression often amounts to pain, and may then run into their left arm. For a broader discussion of this part of the diagnosis the reader is referred to the article on “Chlorosis,” p. 503. Again, although Bright’s disease be not present, nor general arterial disease, the pulse may be anacrotic; this feature of the pulse and its long plateau would set aside that extremely rare affection pulmonary stenosis. Again we shall endeavour to ascertain from the history of the case whether the disease is congenital. Can such a murmur be one of mitral regurgitation, or even of mitral contraction? As regards the latter, the propagation into the carotids, perhaps down the aorta behind, and the position and time of a thrill, are important distinctions, even if we suppose a mitral disease insufficient to cause symptoms of venous retardation; the murmur, again, may be comparatively feeble in the axillary line, and is usually so at the apex. But why stenosis? May it not be that a spur of a diseased valve in the blood-current is the cause of a murmur within an orifice of at least normal dimensions? May not the cardiac hypertrophy be accounted for by arterial resistance, whether in the aorta or elsewhere; or again may there not be an aneurysm of one of the sinuses of Valsalva? This last chance cannot be eliminated; but against it is the hypertrophy of the left ventricle, which is not a feature of these aneurysms; and (hereafter under regurgitation) I shall have to say something about retardation of the radial pulse in aneurysms which may have some importance in this respect. Moreover, a harsh murmur is not, in our general experience of aneurysms, characteristic of them. If, however, the systolic murmur be less harsh, this point loses its force, and exclusion of aneurysm rests only on the rather uncertain basis of a moderate ventricular hypertrophy, the absence of retardation and the small volume of the radial pulse. Again, may the murmur be generated in the aorta without stenosis? I have watched so many cases of large aortic dilatation to their close in death in which neither a systolic murmur nor any other murmur ever appeared, that I hesitate to say that murmurs arise in the absence of implication of the orifice. That vortices should form as the blood spreads into the larger channel seems likely, and that they should thus set up murmurs seems also likely; yet in all cases in which I have followed organic systolic aortic murmurs to the post-mortem table the orifice has presented disease amply sufficient to have caused the murmur. Aortic systolic murmur with valve and orifice virtually normal is outside my experience; dilated aorta, even in extreme degrees, without murmur is abundant within it. Finally, is the orifice actually constricted, or is it merely deformed without constriction? In stenosis I think that there are three tests of the condition: the degree
of hypertrophy of the ventricle, the volume of the radial pulse, and the
diameter of the aorta.

Rosenstein, following no less an authority than that of Traube, asserts
that in aortic stenosis the ventricular impulse is weak or even imper-
ceptible; on the whole this opinion is contrary not only to my own experi-
ence, but to that of others; moreover, we must admit that it may not be
possible always to distinguish between cases in which a murmur is gener-
ated at the orifice without constriction in the positive sense and those of
stenosis proper. If there be any power of response in the heart at all
it seems inconceivable that an increase of resistance such as we con-
template should fail to produce hypertrophy, and that it does so is a
matter of certain observation; moreover, in the cases in which signs of
hypertrophy are absent the pulse is of normal or excessive rapidity.
If an organic systolic murmur be heard at the aortic orifice, if the pulse is
70 or over, and there is no hypertrophy of the left ventricle, I should
say that the disease of or about the orifice has not the effect of stenosis,
or, if it has, that the nutrition of the heart is failing.

Prognosis.—It is said that the forecast of aortic stenosis is of all
heart diseases the least unfavourable. No doubt this is true if we
bulk together all organic murmurs heard at the aortic orifice; but this
is a pell-mell classification. We see, it is true, well-to-do old ladies
leading tranquil lives up to fourscore years or more with systolic aortic
murmurs of a quarter of a century’s standing; as we see such persons
with arteries reduced to trees of coral, yet living the length of the
human span. I suppose that such survivors persist in virtue of fair
cardiac hypertrophy, and of the absence of aneurysms on their cerebral
arteries. In my eye as I write is an old lady whose carotids were
jumping to the eye, and whose radials were as tobacco-pipes fifteen
years ago, who still pursues the unbroken tenor of her existence with
no more to trouble her than a slight dry gangrene of the toes which left
her lame half-a-dozen years ago. In these patients, however, the de-
mands of life are of the narrowest and the lightest; the expenditure is
almost nothing. In Professor Bradbury’s case of stenosis—that in which
the issue of the blood was by an almost imperceptible channel—the
heart of 24 oz. was evidently able still to drive an attenuated stream
of blood through this tiny hole at a velocity, perhaps, of some four metres
per second; so that the blood column in the aorta was sustained for a
long time at a pressure compatible with life. When we regard such
cases, and those again in which the aortic mischief sets up corrugation
rather than strict stenosis of the orifice, the prognosis seems better
than in that next best disease, mitral regurgitation. But a broad
division may be made which will show aortic stenosis in a less
favourable light. If we take patients under fifty-five years of age
we shall find the prognosis much worse. In most of these cases the
stenosis is stenosis proper and of a kind to lead to further constriction; it
probably consists in fibrous inflammation about the ring and the limbs of
the valve of a progressive kind. In them the prognosis would seem to be
rather worse than better for the absence of atheroma: moreover, in the young the system is more exacting in its demands, and the patient is not becalmed in the senile torpor of body and mind. Much depends, of course, on the rate of a subinflammatory process, but my impression is that a person who in young or middle life begins to suffer overtly from the symptoms of aortic stenosis has but a few years to live. Of the duration of its latent period it is, of course, hard to judge; the mischief may be detected by chance, but such discoveries are too irregular to provide materials for prognosis. Old folks apart, then, my estimate of the duration of a case of stenosis is not a sanguine one. The final phase may be by dilatation and backward pressure; but the usual mode of death is exhaustion of the left ventricle and syncope, or degeneration of it and asystole: the former being the predominant and sometimes the only condition, as in the case of a boy (mentioned above by Sir R. D. Powell) who succumbed and died suddenly while running. On the necropsy extreme aortic stenosis was discovered for the first time.

The treatment of aortic stenosis is considered at the end of this article.

Regurgitation.—Symptoms and signs.—Like stenosis regurgitation is often covert in its invasion; moreover, the cases are many in which the signs and symptoms of this disease are found without apparent cause. I have said that aortic regurgitation is not the ordinary course of events in elderly and atheromatous persons; a systolic murmur in this area is the ordinary result of atheroma. Regurgitation is practically always accompanied by a systolic murmur, but I repeat that such systolic murmurs do not always or even usually indicate positive stenosis; as indeed we may readily infer from the volume of the pulse.

Whether the mischief be due to past rheumatism (acute endocarditis is dealt with in another chapter), to strain, to syphilis, or to atheroma, the symptoms and signs are much alike. But in the two latter cases we expect to find, and we generally do find, by other signs and symptoms, that the cardiac disease, pressing as it may be, is but a part of a widespread arterial disease [vide "Diseases of Arteries," vol. vi.]. Moreover, in the atheromatous and syphilitic cases incidental disasters, such as apoplexy or embolism, are more likely to befall the sufferer.

Pulse.—Aortic regurgitation is sometimes revealed, either to the patient himself, or to an observant friend, by the characters of the pulse, a very prominent and peculiar feature of the malady; but it usually betrays itself in the first place by substernal oppression, a symptom which we have already considered under stenosis.

The incomprehensible statement is commonly repeated that in aortic regurgitation the arterial tension is low, and this in face of evidences of tensile strain witnessed in like degree in no other disease. This tensile strain is due to the stress of the hypertrophied left ventricle upon the arteries, a stress often not mitigated by the bar of stenosis or by the protection of tone: the arteries are large and slack. Under conditions of
high pressure, as in Bright's disease, if the arterial charge vary within narrow limits, high as the mean pressure may be, the maxima and minima are not widely apart. Under these circumstances, especially if the muscular coat of the arteries be hypertrophied, tone secures something like a uniform adaptation of the vessels to the blood. If tone be deficient in an arterial system otherwise normal, we find a wide divarication of the maxima and minima; but this is temporary and harmless.

It is otherwise when, as in aortic regurgitation, the condition is both exaggerated and persistent. In this case tensile strain, acting both in the longitudinal and transverse directions, widens and lengthens the vessels, tends to split them across or along; and arterial tone, weakened by strain or anemia, or put in abeyance by some reflex mechanism, is unable in any conservative degree to adapt the continent to the content; the extremes of volume are too far asunder. The circulation changes into the form of a series of discontinuous discharges, as if from a catapult. The well-known tracing of the radial pulse in regurgitation shows a high and violent percussion, usually with an inertia "crochet" at its summit, and as sudden a descent without plateau (Fig. 60). Now it is not necessary to make a long series of observations to ascertain whether in a number of cases the mean pressure is higher under these extremes than in an equal number of cases of hypertrophy of the left ventricle without regurgitation; nature has given us the information in the state of the arterial tree, in the lengthened and dilated vessels; strains which eventuate in general arterial disease, especially in the parts most exposed to the intermittent pulses of the blood. These results justify us in supposing that in aortic regurgitation the mean arterial tension is higher than in any other disease; though as, for some obscure reason, it seems to be less modified by tone, a deduction may have to be made in this respect. The effect of elongation of the arteries is to throw them into curves; and as these are straightened at each diastole, the vessel is then thrown out of its bed with a visible and palpable jerk. The wife of such a patient told Sir Thomas Watson that for some time, on taking her husband's arm, she had felt this uncomfortable jarring.

Whether in a normal peripheral artery, such as the radial, the pulse should be visible is a matter of doubt. In some thin people, in whom a fine skin allows the radial artery to be seen, the pulse is perceptible to the eye, especially if its tone be slack. It is alleged that this beat is made visible by the tension of the skin over the vessel, and that were such a vessel without dimple or dint the pulse would not be visible. In arteries such as the temporal, which are without much cushion, elongation takes place more readily; and in men still young the temporal is often thus thrown into curves which reveal the pulses clearly enough.
though in all other respects the vascular system may be free from any hint of disease. After the tension of aortic regurgitation has been continued for a longer or shorter time, all the arteries exhibit the jarring impulse of which I have already spoken—"danse des artères," as the French call it; they start out of their beds with each pulsation. "Sometimes the whole of the patient's body," says Watson, "nay, his very bed, is shaken by the strong shock of the heart during its systole." In many cases this jerking is well seen in the tonsils. On raising a limb—the arm, for instance—to a vertical position, the refurent character of the pulse becomes still more apparent, for obvious reasons; and if in any case of the kind the mischief or the incidents of it be not such as to produce this character in the horizontal limb it will surely appear in the limb when raised. On raising a limb the pressure in the peripheral vessels may fall to such an extent that the pulse may actually disappear; though entire disappearance is perhaps always due to some constriction of the vessel at a higher point, as at the flexure of a joint or by the fold of a garment: raise the arm of the patient while he has an overcoat on and the radial pulse may vanish; remove the dress and the pulse may persist. Dicrotism does not disappear so regularly as one might expect; in the tracing, if not to the finger, it is often perceptible. We might have expected that with the loss of the support of the aortic valve this recoil would wane or disappear; accordingly in the degree of its persistence a prognostic test has often been sought, but sought in vain: we find a dicrotic pulse sometimes in the least promising cases; the subject needs further investigation. [For other sphygmographic tracings the reader is referred to the paragraphs on the sphyferiens pulse, etc., pp. 930-932.]

It is the function of a healthy heart and healthy vessels to promote at each beat the maximum of blood displacement with the minimum alteration of pressures. At each beat the heart leaves a portion of its energy in the arterial tree which, given out again between the pulsations, converts or tends to convert the intermittent pulses into a continuous flow: it is plain that in aortic regurgitation we have the very converse—the maximum of pressure disturbance with the minimum of blood translation. Thus, in a well-marked case, in no part of the arterial tree is the flow made continuous, not even in the capillaries; and Quincke's "capillary pulse," although not peculiar to aortic regurgitation, is very characteristic of it. If in any malady, even in health, the arterial tone be so low that storage of cardiomotive force in the elastic coats is defective, the capillary pulse may be seen. One of my pupils once demonstrated to me the capillary pulse in his own person, while in health; he told me that it was habitual in him. Dr. Waller says it may be detected in many normal persons, but that in its extreme degrees it is characteristic of aortic regurgitation. On the other hand, in many cases of this disease the capillary pulse is not to be seen. That its presence or absence is of prognostic value is not yet known; we have not hitherto connected its phases with the course of the mischief, nor do we clearly know why tone in this disease is so low. The readiest way
of obtaining the reaction at the bedside is to press highly upon one of
the patient's finger-nails with the point of a pencil, or to depress
its edge with one's own nail; with a little management the pulsation
becomes visible. It may be visible also in the vessels of the retina, or
in the areolas about such an eruption as urticaria. The same evidence
may be obtained again by pressing a glass slide upon the mucous lining
of the everted lower lip; or the skin on the forehead, or elsewhere, may
be rubbed until the cutaneous vessels dilate, when their visible pulsing
will prove how great is the factor of vascular tone in integrating the
circulation. In aortic regurgitation tone yields to tension, or in the per-
ipheral vessels atony may be due to lack of due nutrition.

Retardation of the pulse.—It has been currently reported that in
aortic regurgitation the arterial pulse is retarded. Fagge held this opinion
and many other physicians likewise, among whom is an observer so dis-
tinguished as Sir William Broadbent. As this proposition has always
seemed to me to be contrary to observation, both physiological and
pathological, it must receive some closer attention.

In the normal heart there is an interval of about 0·1 of a second be-
tween the beginning of the ventricular contraction and the carotid diastole.
This interval (the Anspannungszeit or prosphygmic interval) has been care-
fully studied by Chauveau and Marey, Hürthle, von Frey, Keyt, Tigerstedt,
Chapman, and others; and its interpretation is that in this interval the
intra-ventricular pressure is rising to that in the aorta; that not until the
contraction pressure of the left ventricle equals the resistance of the column
of blood in the aorta does the aortic valve open and the aortic diastole
occur. Generally speaking, in the normal course of ventricular systole
the relative pressures in ventricle and aorta preserve the same proportions,
and the prosphygmic interval is invariable; but in aortic regurgitation,
the support of the valve being removed, the pressure within the aorta is
not sustained, and the relation of the pressures is altered: virtually the
aorta and the left ventricle become one chamber. Where there are no
differential pressures there can be no differential times, and the diastole
of the aorta must be coincident with the first contractile effort of the
cardiac muscle. On the other hand, the moment the heart relaxes the
pressure in the aorta falls to that in the ventricle. How then can there
be retardation of the radial pulse? How can the radial pulse be delayed
beyond the time of the velocity of the blood-wave? Lest I should be
wrong somewhere in these suppositions, I have referred the matter to Dr.
Chapman of Hereford. Dr. Chapman, in supporting the arguments
used above, points out that Keyt independently foresaw and demon-
strated this order of the phenomena in 1879. In 1887 he repeated the
same conclusion, that "immediately upon the contraction of the ventricle
the blood-pressure in the aorta begins to rise." Dr. Chapman renders me
the service of tracing the history of the alleged retardation in aortic
insufficiency. Dr. Henderson, he says, first started the notion in 1832.
He was followed by authors of no less ascendency than Flint and
Walshe. Keyt explains the discordance in this way:—"The enlarged
ventricle suddenly filling from both the aorta by reverse and the auricle by direct flow, communicates a shock so marked as to be mistaken for systole. This impulse occurring in the first part of diastole, and preceding the arterial pulse at such a distance, gives the impression of enormous delay of the pulse." In his Goulstonian Lectures I find that Dr. Chapman dealt with the whole question, and gave, moreover, in one such case, the actual measurements, when the interval between the systole and the radial pulse was 0·039" to 0·031", instead of the normal of 0·080". Now if I am asked why I trespass so long upon the reader's time and attention with details so minute as these, I reply that in nature there is no large and small; and that for some few years I have anticipated that by such measurements as these a diagnosis might possibly be made between simple aortic regurgitation and aneurysm of a sinus of Valsalva: unfortunately no test case has yet come under my notice. Perhaps ere long some skilled observer, entering into this controversy, may light on a case of aortic regurgitation in which the radial pulse is considerably retarded; and following it up to the post-mortem table, may decide the presence or absence of sinus aneurysm therewith. Again, in double aortic murmur, in murmurs direct and regurgitant, can we tell by these means whether the direct murmur be due to stenosis proper or merely to a broken blood-stream large or small? Without returning to what I have said under the head of stenosis, I will guess that, whereas in stenosis proper without regurgitation the summit of the radial wave is retarded by conditions the opposite of those in regurgitation, a persistence of the normal interval between cardiac systole and full radial diastole may indicate a combination of stenosis and insufficiency, the pressure in the aorta which the valve is no longer able to sustain being kept up more or less by stenosis of the orifice. [The reader is here referred to the postscript.]

The characters of the pulse are well known; the gifted physician to whom we owe most of our knowledge of this subject has given a memorable description of them. Corrigan compared the pulse of aortic regurgitation to the "water hammer," a toy in which water, imprisoned in an exhausted tube, falls from end to end, on every turn of the tube, with a thud. With some such thud the charge of blood is shot along the arteries. How this effect is intensified by raising the limb, and the effect it has on the vessels themselves, we have seen already; I have still to describe some other characters which are not without interest.

In cases of extensive arterial sclerosis, or at any rate of sclerosis of the radial and brachial arteries, the stiff walls of the vessel do not collapse with the sudden ebb of the pulse wave as a comparatively normal artery does. Nor, indeed, can the arterial diastole be so well marked. Yet, unless stenosis be present, the stiffened arteries will vibrate or jar, and the jarring in the carotids and contorted brachials will be plain enough.

The pulse during the more stable phases of aortic insufficiency is regular. This is the rule, and a very important rule it is. Trivial as an intermittence of the pulse may be in a healthy
heart, in the disease before us it is of grave significance; as grave as it is in pneumonia or in enterica. An occasional intermittence may be of no ill omen; but recurrent slips unmistakably indicate dilapidation of the heart. A irresolute or dropped beat is a far more serious event in aortic regurgitation than in stenosis, or in mitral disease: in the first case, as in "fatty heart," it is more likely to indicate a failing than a merely faltering heart, or a passing inequality in blood delivery. As in other disorders, the heart may fully intermit or contract so feebly that the pulse either fails to reach the wrist, or is but a flicker there. Irregularity of the pulse is a warning of like omen. In the aortic insufficiency of cardio-arterial disease, intermittence occurs earlier than in that of disease more strictly cardiac, at least such is my experience.

The sudden distension of the collapsed and inanimate arteries gives rise to signs which are perhaps something more than curiosities; the chief of these is the sign of Duroziez. We remember that for the most part murmurs are produced in the arterial system by the passage of the blood into a wider channel, when fluid veins are generated. If then pressure be made on an artery in health, say with an edge of the stethoscope, these conditions are fulfilled and a murmur is set up. This phenomenon is intensified in aortic regurgitation, because in this state the walls of the arteries being slack vibrate more readily, as may be conveniently observed on the femoral artery. But in aortic insufficiency, as Duroziez pointed out, there is something more than this: the artery gives out not only this single murmur, a murmur of its diastole, but a murmur on its systole also; there is a double murmur, and this double murmur cannot be obtained in the normal state. As French writers have a confusing habit of taking the word "bruit" to mean either tone or murmur, it is well to say that a tone is produced by the diastole of a normal artery near the heart, such as the carotid—a tone to be heard on light pressure of the stethoscope; but here we are discussing not the tone, but a murmur artificially produced by stronger pressure; and in aortic regurgitation this murmur is followed by a second murmur generated on the arterial systole or collapse. The causes of this latter murmur are unknown; it is easy to show that it is not diastolic, but whether it be a "recoil" murmur as surmised by François Franck, we cannot decide. To get it clearly, Potain directs us to press on the artery with that edge of the stethoscope which is farthest from the heart, so that the whole wave, if it be a recoil, passes under the base of the instrument. He adds that for its production there is a "most favourable point of pressure," a degree between too light a pressure and obliteration of the vessel, which is, of course, to be discovered in each case at the moment of examination. Now it is said that the second murmur—Duroziez's murmur—dies out as compensation fails; if so, it is not a mere curiosity: in any case attention to such incidents as these encourage that painful research in clinics which is the only way to the increase of knowledge. Duroziez's phenomenon is not, I think, peculiar to aortic insufficiency, though
Vierordt says it is; I think I have found it under other atomic conditions when the arteries are unduly vibratile: if so, all it tells us is that the diastole is brusque and the systole "collapsing."

If again the stethoscope be lightly laid on an artery of the size of the carotid, a "tone" may be heard on its diastole, and not infrequently on its systole likewise—"the double tone"; this, though characteristic of aortic insufficiency, is certainly not peculiar to it. If pressure be made a murmur occurs, as we have seen; or if an aortic direct murmur be present, the tone is replaced by murmur without the use of pressure. Again, under normal conditions, on systole of the carotid the second sound of the heart is audible; now, in aortic insufficiency, and in stenosis, this second sound is usually lost. In the smaller arteries, under normal conditions, the tone of their diastole is inaudible; but in aortic insufficiency an arterial diastolic tone may often be heard down to the smaller and distant arteries—in the dorsalis pedis, for instance; so that I have been in the habit of guessing the amount of the regurgitation from the intensity of this tone in the femoral artery. Arterio-sclerosis, however, tends to reduce it. The same phenomenon occurs in anaemia, fevers, and other states in which the artery is slack and the diastole sudden. The murmur of aortic insufficiency is often heard in the carotid, but by no means always; the conditions of its propagation thither are of some clinical moment, in so far as they may help in the discrimination of aortic from other diastolic murmurs.

The Heart.—Of the dilatation and hypertrophy and their signs so much has been said already in this and other chapters that I will not dwell upon the subject. I may repeat that the enlargement may be greater in aortic insufficiency than in any other disease; and usually, at least, is unmistakable. Perhaps its size is only rivalled in certain cases of chronic Bright's disease. In young persons with soft ribs the cardiac area may become prominent. In cases of doubt it is better to lay the ear direct upon the wall of the chest, whereby the heaving impulse is more readily appreciated. It is, I suppose, conceivable that in slight insufficiency hypertrophy may coexist with but a nominal degree of dilatation. Dr. Sansom reminds us that the less the element of dilatation the more "triangular" is the apex area; the superficial area of dulness is extended downwards and outwards, and does not extend far in a transverse direction. Although a dull area corresponding to dilatation of the aorta may occupy the region of the manubrium sterni, and may transgress it to the right, the ventricular dulness may not be enlarged to the right; or not at any rate until in some grievously protracted case the chambers of the heart are involved in a common defeat. François Franck points out that the apex itself may be "dicrotic"; the first shock being due to the reflux, the second to the propulsion of the blood (vide p. 940). Sir W. Broadbent gives us the useful warning not to mistake the systolic recession of intercostal spaces, due to atmospheric pressure acting upon the space left by the diminution of volume of a large heart, for a sign of adherent pericardium. In cases of arterial disease the observer will not forget that
causes of hypertrophy may have been in operation for an indefinite time before the establishment of regurgitation. In such cases the hypertrophy cannot be taken as a direct measure of the insufficiency, which accident may be recent and inconsiderable, the chief part of the changes being attributable to the common causes of both; however, whether recent or of long standing, cardiac enlargement, considerable as it may be, is not so large as in young and sounder persons. It is in the aortic insufficiency of the young, due almost always to rheumatism, that the huge hearts are found which lift life along for many years.

Sounds of the heart.—In a very large proportion of cases of aortic regurgitation the first sound is impure if not actually replaced by a murmur; whether there be positive stenosis or not, insufficiency is generally accompanied by such changes in the structure of the ostium as to give rise to a direct murmur also. In arterial disease the occurrence of a diastolic without a systolic murmur is very rare, as the regurgitation arises incidentally in the course of the atheroma. In strain or rheumatic injury the murmur of regurgitation may exist alone, at any rate for a time; yet even in these cases the first sound is seldom pure. At the apex the first sound is usually prolonged, especially if there be coincident stenosis; and it takes a more "booming" quality as the hypertrophy increases. The direct murmur is usually but not always carried well up into the carotids, so that the carotid diastolic tone is replaced by a murmur, or even by a thrill; yet sometimes neither sound nor murmur is heard with their systole. The second sound at the pulmonary cartilage is unchanged, unless the whole heart be thrust still nearer to the wall of the chest, when it will seem accentuated. The second aortic sound in aortic regurgitation has not been studied very precisely. If it persist with a regurgitant murmur, it is said, a little too readily, to be the sound of the pulmonary valves only. That this is not the case seems to be proved by its frequent propagation into the carotids. Again, it is argued that if the aortic second sound coexist with a regurgitant murmur there is still a substantial area of valve closure, either by fractional parts of the valve or by the establishment of a measure of stenosis which in aortic insufficiency may be conservative. On the other hand, though the disappearance of it may be of ill omen, it is certainly incorrect to say that persistence of the second sound always means a moderate degree of regurgitation. These are points which need verification, but after all sounds and murmurs make but a part of diagnosis; in quickly beating hearts indeed such points are inappreciable. I am disposed to regard persistence of the second sound, in some cases at any rate, as due to the sudden systole of a slack and vibratile aorta, such a tone as that of the systole of the femoral in this disease. It may be audible in cases where the valve is quite disorganised.

Murmur of regurgitation.—If, on the one hand, it be true that this murmur is usually very definite in its characters and its meaning inevitable, it is none the less true that, with the exception of mitral stenosis, it is the murmur most frequently overlooked; and not by
pupils only, but by experienced practitioners. Sometimes the murmur lurks in unexpected places; sometimes its quality is so soft and evanescent that a quick ear is required for its detection, especially if a rasping systolic murmur precede it. If it be both soft and aberrant in its site even a skilful observer may be deceived, at any rate at first. Not only may the murmur of regurgitation be soft and distant, and may lurk in strange places, but it may be aberrant in time also. It may occupy fractional parts of the diastolic period, and not always the initial part. Like the murmur of mitral stenosis, it may be perceptible only in the middle or final third of the long pause; and if discovered accidentally in this rhythm, before the advance of secondary changes, it might deceive even the elect. One such case I remember which divided three hospital physicians in opinion. One inferred mitral stenosis; two held to aortic regurgitation. Whether either opinion was afterwards verified I cannot say. Dr. Douglas Stanley described an interesting case of the kind at a branch meeting of the British Medical Association. In this case a diastolic murmur arose immediately on diastole, a murmur of aortic regurgitation well marked at the base; at the apex was heard a mid-diastolic murmur, rougher than that of the base, and not heard outside the mitral area, which ceased before the first sound. After death the aortic valve was seen to have but two cusps, and these involved in a mass of vegetation. The mitral valve was healthy. Both murmurs were clearly aortic.

Loudness of murmur is no indication of severity of lesion; the reverse is rather to be anticipated. A loud murmur generally signifies a vigorous heart; and a refruent stream returning at a high velocity may set up more active veins in the ventricular content than a large return falling back through a large opening at a low velocity. If a murmur previously loud fall in intensity we may be apprehensive of evil. Sudden ruptures of the valve often give rise to very loud murmurs audible over a large area of the chest; in such cases the murmur has been audible to bystanders, and even to the patient himself. In the presence of stenosis a regurgitant murmur is louder, other things being equal; as the velocity of the refruent current is greater. In these cases a small and fairly sustained pulse is associated with a sawing double murmur. A jet returning through a perforation of a limb of the valve is said to be attended with a piping or mewing sound; or it has been compared to the chirping of chickens. A murmur direct or regurgitant, audible without contact with the wall of the chest, is always aortic—an inference sometimes of diagnostic value.

Prof. Sewall of Denver has investigated the behaviour of all cardiac murmurs under increasing pressures of the stethoscope. He says that murmurs of aortic stenosis audible at the apex disappear under pressure there, and are herein distinct from mitral regurgitation. Also that, great dilatation of the aorta or aneurysm apart, the murmurs of aortic regurgitation may be annulled by pressure at the base but not at the apex. "Inorganic murmurs" at the base, he says, can all be obliterated by pressure; and by the same means used in the second right inter-
DISEASE OF THE AORTIC AREA OF THE HEART

space close to the sternum the normal second sound can be stopped, unless the aorta be so dilated as to be in contact with the wall of the chest.

The student is often directed to track a murmur to its origin by shifting his stethoscope along the surface of the chest from one area to another, in order to note where one murmur dies and another is born. This is a misleading device, only to be used by skilled observers. A murmur, like a river, may run underground in part of its course; the conditions of conduction differ from place to place, and one and the same murmur, as the stethoscope travels, may so wax and wane, as the structures about it vary in conductive capacity, as to appear twofold. Many a misapprehension thus arises as the observer slips the instrument diagonally upwards; a murmur heard at the apex disappears to reappear at the aortic cartilage; and thus a murmur generated at the aortic orifice only may be regarded as indicative of two lesions. Another error is to assume that the murmur certainly follows the direction of the blood-current; the blood does not run in the air as water over gravel; the murmur we hear is due to the vibrations of surrounding structures—chiefly the walls of the heart—set up by the vortices within them; the heart is the fiddle, the blood is but the bow. We must rid our minds of these conceptions of blood running here and there in the chambers, as if it were from a water-cock into a pipkin, and realize that the walls are thrown into vibration by molecular collisions in a plenum.

Of aortic insufficiency with regurgitation, but without a murmur, I know nothing; but murmurs which can be extremely soft may be very rare in instances be evanescent. Weismayer, in a paper which has been much quoted, accepts such statements, a little uncritically I think, and proceeds to explain them. Dr. Hermann Weber's case (vide infra) is an example of the manner in which in incipient cases the murmur of aortic insufficiency may cease for a time with the insufficiency which gave rise to it; again, like any other murmur, that of insufficiency may wane with the heart in which it is generated; but that with a persistent insufficiency regurgitant murmurs come and go in a comparatively vigorous heart is contrary to experience, or at any rate to mine. As testing exceptions I may refer to a case reported by Dr. Saundby, and to another reported by Dr. Musser. In Dr. Musser's curious case the corpora Arantii had been transformed into calcareous buttons (4 mm. by 2 mm.). During the formation of these excrescences regurgitation took place and a murmur was generated; but as they wore down and the free surfaces became faceted, after the manner of gall-stones, the incompetent valve became again competent.

Whether regurgitation is prone to occur in dilatation of the aorta with an unimpaired valve, and without aneurysm of a sinus, has been considered already (p. 922). Relative insufficiency at the mitral orifice is well understood and by no means rare; but this is known to depend upon the conditions of the muscular and tendinous attachments of the
limbs of this valve, a kind of attachment which does not exist in the aortic valve.

*With this problem is bound up that of intermittent aortic regurgitation* considered above; for these cases are said to depend also on conditions of the aorta rather than of the valve. I believe that in cases of intermittent aortic regurgitation the valve is nearly always diseased; but that in the earlier stages, say in disease of one of its limbs, the valve becomes able, by mutual accommodation of its parts (p. 466), to close the orifice until the blood-pressure becomes excessive, or some other physical change supervenes. Such a patient may indeed be examined at a time when the valvular disease is latent, or is not revealed by a murmur at any rate; and in such a case a grave error of diagnosis might be committed. The following case, resting on the authority of Dr. Hermann Weber, is most instructive:—

A very active young man, aged 32, of weak muscular development, was examined by Dr. Weber on arriving at a height of 8000 feet. The second aortic sound was replaced by a musical murmur at mid-sternum and a little to the right. The first sound was rather indistinct. The pulse was 105-112, feeble, but not characteristic of aortic regurgitation. On the following day the murmur had disappeared; the heart sounds were normal, and the pulse 88. Two days later, at 9000 feet, the same murmur became audible; and in like manner disappeared on the day following. Further climbing was forbidden, and he returned to work in good health. Seven years later the patient died of *Herzschlag*.

Regurgitation may occur at times of high blood-pressure—as for instance in exertion or in senile arterial plethora, and may disappear—the valve becoming again competent—as, under treatment or otherwise, arterial pressure falls.

Murmurs occurring during diastole may be heard in pericarditis and aneurysm; the former murmurs are not difficult to interpret (*vide* p. 953).

Dilatation of the aorta is said to be the rule in cardio-arterial degeneration, the exception (in any considerable degree) in primary aortic regurgitation. If so, the exceptions are many; I recently lectured in Cambridge on a case of mere rheumatic aortic disease in which there was considerable dilatation of the aorta with "fireman's helmet" dulness.

The association of aortic disease with murmurs simulating more or less those of mitral disease remains to be discussed. The murmurs may be divided into two classes: those suggestive of mitral regurgitation, and those suggestive of mitral stenosis. First, of aortic murmur simulating that of mitral regurgitation four cases were brought forward by Dr. Dickinson at the meeting of the Royal Medical and Chirurgical Society on the 8th of June 1897. In them, although after death the aortic orifice in each was found to be advanced in stenosis, a systolic murmur was heard at the apex, so that mitral regurgitation was either assumed or
could not be excluded. In many such cases, either my own or
shown to me from time to time in hospital wards, I have perhaps too
promptly and confidently declared my opinion—quantum valuiisset—in
favour of aortic obstruction; I admit, however, that in some of them
mitral disease can only be excluded on the principle of “ne enti
multipliicanda.” Still this principle is a sound one if we do not lean too
much upon it. In two cases in which I was led to hazard such an opinion
it was borne out by necropsy; there was no mitral insufficiency. In one
of these examples the murmur at the apex was musical, and I guessed it
to be four to one that a distinctly musical murmur is aortic. In another
the murmur was audible an inch away from the patient’s chest; it is
twenty and more to one that a murmur so audible is aortic. In another
again a thrill was perceptible at the base. That such murmurs may be
audible in the back I admit—aortic systolic murmurs often are; but in
the cases I have seen such murmurs were not confined to the axillary and
infraclavicular regions, but were audible anywhere—passim, not ordinatum.
Again, in such cases—and this is true, I think, of those collected by Dr.
Dickinson—the arterial pressures were in themselves almost conclusive;
the pulse in each was not “mitral,” but “aortic,” regular and of fair
mean pressure. In mitral regurgitation the arterial system is ill-filled,
while signs of a rise in venous pressure, cardiac and systemic, are soon
manifested. Dr. Norman Moore has suggested that the sphygmograph
might be useful in the diagnosis of such cases; for once in a way it
might: an anacrotic tracing would settle the question in favour of aortic
stenosis, though it might not exclude coincident mitral incompetency
of slight degree.

Secondly, an aortic regurgitant murmur may simulate that of mitral
stenosis. The murmur of aortic insufficiency generally begins on diastole,
is then loudest, and falls as the aortic pressure falls; that of mitral
stenosis generally rises up to the systole. Aortic diastolic murmurs in
the later part of the pause are very soft, because the pressures in aorta
and ventricle are then nearing equality, or have attained it, the vibrations
persisting for a sensible moment longer in the walls of the heart. If the
murmur be heard at upper and mid sternum, if, it begin with the diastole
of the heart and taper off during the pause, it is an easy sign to interpret.
But if the murmur, not as a rule so harsh or vibrating as that of mitral
stenosis, be so soft that it may escape an unpractised ear; if, instead of
tapering off from the beginning of the pause, it occupy the middle, or
even the latter part of it; if, again, it be barely audible or inaudible at
the upper sternum, distinct at the lower sternum, and loudest about the
fourth left interspace, the student of the aortic cartilage may be misled by
whispers so stealthy and devious. He may attribute the murmur to mitral
stenosis; or he may add the case to the list of vanishing aortic regurgi-
tant murmurs, or again he may add himself to the cloud of witnesses
to “pulmonary regurgitation.” However distinct the murmur may be
in the fourth left space, it dies off rather abruptly as the apex is
approached.
Finally, the murmur of mitral stenosis may be simulated in aortic regurgitation. There are many cases on record in which a "presystolic murmur" was present without mitral stenosis; in some of them the only perceptible lesion was aortic. From what we have seen already (p. 944), the student is prepared to understand that murmurs occupying the long pause, or parts of it, not necessarily the initial part, are consistent with, and under certain circumstances significant not of mitral but of aortic disease. Against this source of error the observer will be on his guard. But this explanation does not cover all the ground; observers of the highest authority assure us that a presystolic murmur, heard in the mitral area, such a sound as to be characteristic of mitral stenosis, is to be heard in cases which otherwise would be regarded, even on the post-mortem table, as uncomplicated aortic regurgitation. To these cases the late Dr. Austin Flint first drew attention, and his lead was followed by many other observers whose records have been well summed up by Dr. Lees: Dr. Sansom, who recorded cases of this kind in 1881, has carefully discussed the difficulty again in the new edition of his work; to this discussion I refer the reader for further detail, as no explanation of the phenomenon is as yet established on anything like a certain basis. Sansom and Potain lean to the belief that the presystolic murmur (if it be generated in the mitral area, and not in the aortic) may be due to impingement of the refluent aortic current on the anterior mitral curtain before it is made taut, whereby either vibrations are set up in the valve itself or, by bulging the valve, the orifice is practically narrowed. Dr. Fisher has published two cases of this kind (one of Dr. Hale White's), in both of which thickened endocardium upon the ventricular septum showed the formation of the eddy was not in the region of the mitral valve. "The presystolic thrill and bruit were well marked and mitral stenosis was diagnosed; but at the necropsy the mitral valve was found quite normal. The aortic valves were healthy also it is interesting to add, and the aortic regurgitation heard during life was due to pouching of the sinuses of Valsalva with dilatation of the first part of the aorta." A third case, of Dr. Goodhart's, is adduced to prove that this presystolic murmur may be heard in disease of the aortic valve without regurgitation. Other authors suggest that a meeting of the aortic and auricular currents may produce a murmur; if so, surely Flint's murmur should be far more common than it is. One case shown to me in a hospital three years ago by two physicians as one of this kind, was in my opinion a case of broken aortic diastolic murmur, not generated in the mitral area at all. There was no rumble; the murmur was audible to left of the sternum, but not in the scapular region. Still rumbling presystolic murmurs, with thrill, do no doubt occur in aortic disease unaccompanied by mitral disease. All I can do then is to warn the reader of this source of error; and that murmurs form but a part of cardiac diagnosis. It has been good for us that these invaluable aids to diagnosis have received even a disproportionate share of attention, but it has been at some loss of perception of other aspects of cardiac disorders, some of which are of no less value.
Pain.—Distressful sensations of the nature of pain are more common in disease of the aortic area than in other diseases of the heart. The distress may range from a slight oppression to breast pang; while a fair compensation is maintained, and there is no active aortitis, no discomfort may be felt, otherwise the distress may become agonising and almost constant. Its form is that of angina pectoris. When the insufficiency of the aortic valve is of acute onset, as in sudden rupture, the pain and oppression may be very great; but unless the mischief be of extraordinary severity—bad indeed almost beyond hope—the pain will pass off as the inflammation in the area subsides, the reserve capacity of the heart comes into play, and pressures are re-adjusted: thenceforth, until the organ begins to fail, discomfort may be absent—at any rate in patients under middle age. If it be in elderly persons, the subjects of general arterial disease, that angina pectoris in its major or minor forms most frequently occurs, it is by no means confined to them. I recently witnessed a very distressing and persistent angina in an undergraduate, the subject of recent rheumatic disease of the valve, and probably of aortitis. A sense of substernal oppression is the first hint of it; it is felt on distension of the stomach and bowels, and on ascents, even the gentlest. In extreme cases, or in persons of the peculiar temperament which favours the phenomena of angina, assaults of this kind may come on during complete rest, probably in obedience to unseen tides of blood-pressure. That angina pectoris comes on during effort only is a false aphorism based upon too smart a description of such cases; not infrequently it comes on even during sleep, adding a new torment to the bitterness of death. Of muscular movements those of the arms seem to be the most efficient in producing anginose pains; it has been stated that movements of the arms are the most instantaneous in their effects upon blood-pressure. Another surmise is that persons of a gouty habit are peculiarly liable to anginose attacks and complications, an opinion based upon no little clinical experience; it is but a part of the truth, however, as angina is even more tyrannous, if less lethal, in persons in whom the neurotic habit is conspicuous. Such sensations are not so common or conspicuous in aortic stenosis as in regurgitation. As the aorta is probably the seat of them this distinction is intelligible. Another seat of pain in aortic regurgitation, and this too rather if the later phases of it, is gastralgia, or a suffering so described. This pain is to be discriminated from the aches, severe and trying as they often are, which seem to have no deeper source than the intercostal and neighbouring spinal nerves. With the gastralgia is often associated the persecuting flatulence which besets all cardiac affections, even the functional. To belch up wind is attended with relief, but it is another thing to say that the wind is the sole cause of the distress, and it cannot explain the recurrence of the “gastralgia,” which I suspect is allied to angina.

It is alleged that there is some connection between tabes and aortic disease. Ruge and Hütter found aortic disease in nine cases out of 138
of tabes (6.5 per cent). In only one of these was there no probability of syphilis, and in five this antecedent was definitely ascertained. Articular rheumatism counted for very little. Sir W. Gowers accepts the association as a causal one, and Grasset and Rauzier are of the same opinion. The probable explanation is that both diseases belong to the syphilitic series, and may be associated in young persons before the approach of senile atheroma. Other authors regard the connection as one of simple coincidence. No confident opinion can be expressed at present; but it may be that in many cases of aortic regurgitation the gastralgic phenomena are directly of tabetic origin. How often do we wish our cases back again for better investigation! It is but the other day, after I had completed an examination and discussion of a case of thoracic aneurysm, that my colleague in consultation was wicked enough to tell me I had not found out that the patient was tabetic. Though the gait was scarcely affected, I had to admit, when told, that such was the case. Here again syphilis was no doubt the nexus, and an insidious tabes may be the origin of some symptoms not directly attributable to the cardio-arterial disease.

The nervous system.—Besides pain, which strictly speaking should come under this head, there are other nervous disorders which are better marked in aortic regurgitation than in other forms of cardiac disease. In an article on cardiac delirium, published many years ago, I said that the sufferers from aortic disease show an occasional liability to cerebral derangements. Even in the latent or stealthier phases of aortic insufficiency we may note more especially certain mental perturbations which are not unknown in other heart diseases. We note a restlessness, a fretfulness, a change in temper amounting sometimes, as the mischief advances, to violence; in rare cases the restlessness sometimes goes so far as to urge the patient to spring from bed, to perambulate the house, or even to jump out of the window. We may compare the delirium of such cases of aortic regurgitation to that of alcoholic pneumonia; and, as in these extreme degrees it occurs chiefly in men, it may be so troublesome as to make a male attendant necessary. That it is not alcoholic is proved by its outbreak or persistence in patients who are and have been under continuous observation and restriction. Much of the restlessness of the delirium is due to the fact that it is usually a delirium of place: the patient is under the delusion that he is in a strange house, or far away from home; pacified for a few minutes, or for a few hours, the delusion seizes him again and again with an agitation which is fraught with the worst consequences to the cardiac disease. Prof. Osler (62) makes a like observation. The association of insanity with cardiac disease has been studied by Mickle, Ball, Fauconneau, and others. Apart from mental disorder, headache is frequent in aortic insufficiency; and 'buzzings, dizzy sensations, momentary obscurations of consciousness, twitchings, or even convulsions, may indicate the perturbed conditions of the cerebral functions by way perhaps of the circulation. The vascular inconstancy is perceptible to the patient whenever he stoops. Sleeplessness, not by
any means always due to cardiac uneasiness, is often very troublesome, and is especially noticeable in aortic insufficiency.

**Nutrition.**—Although the arterioles cannot be contracted, as sometimes alleged,—or we should not see the capillary pulse,—yet pallor and some falling off in flesh mark another distinction between aortic insufficiency and mitral disease, in which the face is congested; and emaciation, if present, may be concealed by venous turgescence or arterial oedema. So long as dilatation of the left ventricle is compensated by hypertrophy, so long as the cardiomotive force keeps up, there is practically no anasarca or ascites. Filling of the pleural cavities, swollen legs, albuminuria indicate a slackening ventricle and increasing residual blood; the heart is entering upon that final phase of demolition which has been described under the diseases of the myocardium, and must not detain us here.

**Respiratory system.**—While the mitral orifice and the myocardium are sound the pulmonary circulation is protected. It is in the final stage of a shattered heart that the bases of the lungs begin to fill. These changes often appear before there is definite evidence of mitral insufficiency—at any rate before a murmur is generated, and even before the extension of dulness over the right ventricle. As the ventricle is distended the papillary muscles may fall relatively short; or in some other of many ways the mitral machinery may be deranged: yet even with a competent mitral valve, as the residual blood in the left ventricle becomes more and more, and the regurgitations perhaps larger and larger, the arterial head will dam back the venous. When a murmur of mitral regurgitation appears the end is not far off; and therewith the case travels out of my sphere.

Dyspnoea is scarcely to be called a prominent symptom till this last stage is reached. The dyspnoea of the earlier stages is rather an inexplicable perturbation which the patient himself can hardly describe, and which, if an exact person, he usually declines to call shortness of breath: he speaks of it rather as a sense of oppression which impels him to sit up; it partakes of the nature of angina. Nay, often, as in angina, he may evade a strong inspiratory effort. At times, however, and in later phases of the disease, the patient may be seized with "cardiac asthma," when the gasping and shortness of breath are distressing. Still, this is not quite the panting of mitral disease: the excursions of the chest are less conspicuous, and have more of a nervous or spasmodic character. It may be a call of the bodily tissues upon the heart for more blood, a call not so much for the "respiratory pump" as for more driving power; or, again, it may have a toxic origin. If expiration becomes audible a little distance away, and both inspiration and expiration assume a tubular quality, such as horsemen call "roaring" or "whistling," then, however slight this may be, the trachea is so far constricted by a dilating aorta.

Cough is often present—generally indeed—and may be an intolerable evil. This cough, when it does not spring from incidental causes, is due to pressure of the diluted aorta, either directly upon the trachea, or upon the laryngeal nerves. Unless there be some contingent catarrh
there is no expectoration, or no more than is hawked up by any cough. In cases of considerable dilatation of the aorta the cough may be of frightful severity. One patient of mine, when he felt an attack coming upon him, used to throw himself on his hands and knees; or such sufferers will anchor themselves to bed or table to mitigate the racking of it.

Sphygmographic signs.—The ordinary tracings which adorn our books and essays are of little worth. The more valuable ones, such as those of Mahomed, Galabin, Riegel, Lorain, and others, present some points of interest. Hundreds of tracings are published which prove no more than the inadequacy of the sphygmograph to analyse the finer components of the aberrant pulse. It is characteristic of the tracings in aortic insufficiency to show a hook or "crochet" at the summit of the percussion wave which, in aortic regurgitation with a strong ventricle and little or no aortic obstruction, is of course very high. The sharp return of this "hook" is said to exhibit the rapid arterial recoil; but to my eye, like many other such notches, it exhibits nothing more than the inertia of the lever. Notches and waves due to this cause are too often interpreted as records of this or that secondary vascular wave. After a sharp fall of the lever in aortic regurgitation, or in other states in which arterial resistance is low, a second wave of inertia may also be seen, and even a third, as in a tracing recently published and elaborately explained. Such waves mean nothing more than the bouncings of the long and light lever after strong percussion. It is remarkable that the dicrotic wave often persists (Fig. 60, p. 937). Now if the dicrotic wave be due to recoil of the aorta, we might expect that when the bottom of this vessel is knocked out this recoil would be prevented; but this is by no means always the case. Dr. Samways urges that the dicrotic wave is due to the longitudinal recoil (shortening) of the first part of the aorta; this may possibly explain the persistence of the wave under the circumstances we are considering in any case stenosis would promote it. It seems probable that as regurgitation increases the dicrotic wave would be obliterated; but it does not appear that this indication has any important prognostic value. The presence of more or less stenosis might be indicated by an aortic wave in the tracing. It is difficult to draw any precise conclusions from the sphygmograph as to degrees of atheroma; the tendency in such cases is, of course, to a broader-topped wave. The sphygmometers of Hill and Barnard and of Dr. George Oliver seem likely to take a practical shape, and, if so, mechanical aids of great value will be placed at our service. By such means many difficult problems, now obscure, will be made clearer to us.

Diagnosis.—Much has been already said indirectly in this respect. In cases of uncertain diastolic murmur the absence of thrill or its distribution about the base, the absence, in the earlier stages, of the short first sound of mitral stenosis, of reduplicated sounds, of evidence of rise of pressure in the pulmonary circulation, and constancy of murmur on
changes of position, will indicate that if there be a murmur in the mitral area also, it is but the flapping of the upper limb of this valve (Potain and Sansom). I repeat that in following a murmur from apex to base, it may not only go underground for a space, but also may emerge with a change of quality; and that murmurs of aortic regurgitation may be exceedingly distant or faint, may frequent strange quarters of the cardiac area, and may be inaudible at the aortic cartilage. In the last stage the failing systolic sound is as short as in mitral stenosis, and the liver enlarges and hardens. The jerking of the arteries too may then subside, and the case becomes virtually mitral. Duroziez's sign may be useful, but is hard to make out in an edematous thigh.

In a patient, whom I saw but once, I had some hesitation at first in deciding whether a chafing diastolic sound at the base were due to aortic regurgitation or to the pericarditis of chronic renal disease. A study of the whole case, however, left no doubt of the latter interpretation.

Prognosis.—The course of aortic regurgitation is towards death. As in all heart diseases the main factors in prognosis are four: the age of the patient, his calling and habit of body, the kind of lesion, and the degree of lesion. An accurate knowledge of the history of the patient and of his symptoms is very important, but it is not always to be had. I have a difficulty in recalling cases of mere aortic regurgitation in children; such cases, if rheumatic, have no doubt a long average survival. A deformed valve segment must in all cases be a strained segment, and meet for chronic inflammatory and atheromatous degenerations. A clean rent in a healthy valve segment should be a less destructive process than a lesion of equal degree due to atheroma; it is said that a clean rent in an aortic cusp has been known to heal. As age advances the prospects of the duration of life grow less and less; the lesions may be worse in kind, certainly adaptation is less ready. In atheroma aortic regurgitation signifies not only progressive disintegration, but also an accelerating rate of it, and prognosis is graver with regurgitation than with obstruction: aortic direct murmurs being, as I have already said, the ordinary feature of atheroma, regurgitant murmurs, the extraordinary. Death may suddenly intervene in the period of latency, primary or secondary, but the period is one of comparative safety; when the attention of the physician is drawn to the disease by complaints of retrosternal oppression or of uneasiness on ascents, the stage of dissolution has begun; whether the origin of the mischief be in old or young, in strain, rheumatism, or atheroma; though in this last kind dissolution may be more rapid. In strain such sensations may be felt at first before readaptations of cardiomotive functions have become established; but if the patient's life is to be a comparatively good one they should pass off for some years, as the reserve capacity of the heart comes into play (secondary latency). The patient may go about his work again in ignorance of the fatal rift; yet, when he is brought up, sooner or later, by some uneasiness about the heart, he does not forget to tell the physician how that on a certain occasion of effort he felt a strange and distressing sensation in
the heart. This event may have been five years before; but usually it is not more than two or three, and may be much less.

The duration of the latent period—primary or secondary—depends more on the degree of insufficiency than on the soundness of the cardio-arterial system; for, unless it be in the case of syphilis, patients undermined by atheroma are withdrawing on account of virtual age from heavy work; and if in older men the conditions of nutrition may be less favourable, those of labour are less exacting. If, however, rupture occur in a man whose arteries are degenerate, the latent period is very brief. In such a case, recently under my care, the consequent symptoms of disease never receded at all. When we turn from rupture to insufficiency gradually established, we find, as I have already said, that too literary a view of the matter is taken by many writers, especially in the division of chronic aortic disease into the cardiac and the cardio-arterial. A long survival is not unusual in cases of general cardio-arterial disease in elderly persons, while on the other hand "young cases" often do poorly, and last for a briefer span than we had anticipated. That the duration of a heart maimed by aortic insufficiency may be at least as short in young persons as in the old and atheromatous, will be granted in respect of younger subjects in whom the invasion of syphilis is unchecked; it is not usually admitted of rheumatic disease, though this process consists in a proliferative fibrosis which, as opposed to mere "replacement fibrosis," too often has ruthless cicatricial consequences. Healthy as, apart from the local disease, the heart and arteries may otherwise be, the progress of such cases is often inexorable. It were paradoxical to say that the outlook may occasionally be better in cardio-arterial atheroma, but the part may not be far from the whole truth. I have said that the capacity in elderly persons for a fairly sound hypertrophy of the left ventricle is usually much underrated; even in the presence of dilatation of the aorta, and of stiff vessels, the crazy machine with a fair muscle at its centre may last many a year, unless one or other coronary artery be blocked; or miliary aneurysms form on the cerebral arteries. Let sanguine prophets say what they may, ten years is a long time in any case of aortic insufficiency; and, given equal degrees of insufficiency, I would not despair of such a respite in temperate and tranquil eldiers, until they "be with ease gathered, not harshly plucked, for death mature." Every physician's experience must remind him that to be "harshly plucked" is not the fate of the older of these patients only; of young men who die suddenly, no small tale die of aortic insufficiency; and to die of syncope with a sound or fairly sound heart muscle happens to old as well as to young patients. To say that the disease in the cardio-arterial cases is "progressive," and in the rheumatic or strain cases not necessarily so, is too academic a distinction, and untrue even as that: aortic insufficiency is always "progressive," even if the local disease is not. If the contrary be asserted it is because observers are in a state of reaction against the black prognosis of all and any heart disease which prevailed among
our fathers; now we are in the opposite extreme, and are buoying
our patients with too crude a hope. Some young patients die un-
expectedly soon, some old ones live beyond expectation. Mitral in-
sufficiency is the only heart disease which, under favourable circumstances,
can be cured to an indefinite duration. I have now under my occasional
observation persons still leading useful and active lives who have
lived a quarter of a century and more with mitral regurgitation; but
I cannot remember the survival of any patient with aortic regurgitation
for fifteen. If the patient, whether in a palace or in a workhouse,
be a man of easy circumstances and tranquil occupations, he has the
greater chance of survival. Care or worry, bustle or toil will kill him.
There are men of such a temperament that they cannot form sedate
habits: recklessly, as it seems to the doctor, they skip up stairs two at a
time; they puff after trains; they climb over five-barred gates; they
bounce up from deep sleep to pass water, and so forth: they do not mean
to run these risks, but such is their incorrigible temperament. With such
persons discipline must be attained by spending day after day in
drill, in gaining self-control, in repressing volatility. In this precaution
there is nothing false to a man's best self; it is the way to get the most
work out of himself before he dies. Persons in toilsome callings must
change them; and spend the perhaps no less useful remnant of their
days in some easier duties. Due vigilance may be exercised without the
encouragement of hypochondria; as some one well put the rule: find out
what you can do, and do it; find out what you cannot do, and never do it.
The conditions of survival are more favourable in women than in men.

The big ventricle, efficient as it is, racks the machine from the begin-
ning; the aorta, being of elastic tissue and not of muscle, suffers under
the thrust, and the means of the heart's nutrition, instead of increasing
as demand requires, are gradually sapped. The watchful physician may
then note that muscular effort no longer raises, but even reduces the
blood-pressure—a bad sign indeed.

Anginose pains are always menacing in regurgitation, yet even they
may be kept at bay by the nitrates, it may be for a year or two; but
the respite is a life of troubous days—a life of pain, of slavery to
drugs, of bitter physical and mental adversity. Anginose pains seem
to signify less imminent danger in women than in men, although in
men they are more common; this, if true, may depend on the greater
docility of women under treatment. Sometimes angina appears only for
a time, with a push of aortitis.

Insufficiency death is not always sudden; some patients drink
the cup to the dregs; life is protracted from phase to phase of cardiac
disorganisation. Usually, however, the thread of life is snapped before
involution is complete, before these later stages of cardiac dilatation and
rise of venous pressure are accomplished. Such patients sometimes
die of asystole, far more frequently they die of syncope: the heart, not
yet quite played out, comes to a sudden stop, probably under some reflex
interference. Although then the signs of cardiac dilapidation will be
noted with apprehension, gradual dissolution is often avoided: with seeming caprice death cuts the thread after rather too good a dinner, a quick step into a railway carriage, or a start up from bed; or again, the bolt may be mercifully drawn during sleep, and the last years of such a life may be happy even in the ending of it; for as Bacon says: "Many times death passeth with less pain than the torture of a limb; for the most vital parts are not the quickest of sense."

To enter into a discussion of combined lesions of the heart would lead to repetition of the work of other contributors; but it is almost needless to say that in every estimate of the duration of life in aortic insufficiency the values of the other component parts of the heart must be estimated: such estimates are to be found in the chapters on other diseases of the organ. Again, it is of the first importance to decide whether a coincident lesion elsewhere, valvular or muscular, be independent or dependent on the aortic. It is contrary to my experience to assert, as many have done, that coexisting mitral regurgitation is helpful in any stage of aortic insufficiency, except as a relief to the aorta in the case of angina; that moderate mitral contraction may be so is conceivable. In rheumatic cases, aortic disease usually means a more extensive cardiac damage, and in this respect again the prognosis is worse in aortic than in mitral insufficiency.

That "apex murmurs" are often mere aortic direct murmurs I have said already. Loudness of murmur, other things being equal, speaks in favour of sustained cardiomatic force, and, although a murmur soft to the point of indistinctness may be consistent with slight or incipient injury, on the other hand a murmur may wane with the heart which generates it. A quickening pulse is of ill omen; if not due to temporary causes, it means a larger residuum at each contraction and ill-filled arteries, as tested by raising the arm. We are told that a fall of the specific gravity of the blood is likewise of ill augury. If stenosis coexist with insufficiency the peripheral arteries will be the less in diameter; moreover, in stenosis they contract upon their contents, in regurgitation they are slack. Increase of the area of cardiac dulness vertically may be a good sign; its increase transversely is a bad one; and, speaking generally, changes in the chambers are of far more importance than changes in the murmurs; as we have seen there is an element of caprice in murmurs, which may rise, fall, split, or perhaps vanish for a time, without definite prognostic meaning.

Of intercurrent diseases the infections are the most injurious in their effects upon the lame heart; and of these influenza and diphtheria are the most malignant.

If possible "functional" perturbations of a transient kind must be distinguished from changes in the myocardium; but to estimate the value of the myocardium in fairly stable cases of heart disease is very difficult. The results of treatment, especially in the use of digitalis, perhaps may give us some hints of this kind. Arrhythmia, alteration of other sounds, diminution of urine, the appearance of albumin or hyaline casts, failure
of remedies previously effective, are of sinister meaning. Neglect of treatment until late in the disease is against the patient's prospects; the command of skilled treatment and the means of carrying it out are in his favour.

Mitral insufficiency is not infrequently cured; aortic never. As in Hermann Weber's case, though the murmur may cease, the mischief stealthily advances, and may bring down the stricken man when he least expects it.

No error is worse than false precision; none more gratuitous than prophecy: still in human affairs we cannot get beyond moral certainties, and patients or their friends often demand of us a fallible prediction. Given a moderate lesion and good conditions within and without, I should say that in a patient under five-and-thirty years suffering from rheumatic, syphilitic, or traumatic aortic regurgitation, the prospect of life is about ten years; rarely more than twelve, save in cases where the lesion is nominal in degree. In persons over fifty, in whom the arteries are atheromatous, and the aortic insufficiency a later stage in the work of decay, three or four years may be expected. On the other hand, if the aortic insufficiency be an early sign of atheroma about the base of the aorta, and the patient in easy circumstances, death may be kept at bay for six or eight years. The previous rate of change in the individual is of course a most important element in our judgment in each case. In obstruction alone the expectation is much longer. If in aortic disease, even at this later age, the lesion be syphilitic, as in a case now under my occasional observation, the prospects are much better; by careful treatment even ten years may be added to the sum of days.

Cases of alleged recovery from aortic regurgitation are recorded from time to time. I have said what I think of such stories: the patient was not watched long enough; murmurs may be evanescent, not so the lesions they signify. No less an authority than Leyden, however, has recorded such a case of recovery, but after a traumatic lesion (51).

Cerebral embolism is prone to occur in aortic disease of whatever kind; pulmonary apoplexy occurs, but does not take the place it does in mitral disease, for obvious reasons. For an account of these events the reader is referred to the chapters which deal with them.

Treatment.—Give your prognosis on the best suppositions, treat your patient on the worst. The treatment of aortic disease, and I now imply both kinds of it, falls into the natural divisions of diet, management, and drugs. In diet we have to look to three points: to the sympathy between the heart and the stomach, to good nutrition of the heart, and to moderation of its work. We must avert indigestion, and administer nutritious food without either raising the arterial resistance or increasing the heart's output. Indistinctly we are aware that there are diets which promote arterial resistance, and so far as our lights go we must elude this danger. Many of the elderly sufferers from aortic disease are gouty. In such persons we should avoid all that encourages this habit (vol. iii. p. 187). On the other hand, to reduce the diet below the
needs even of a person who can take little bodily exercise may carry us into the peril of pining the diligent heart; and to exclude nitrogenous food in order to avoid goutiness may throw the patient upon a diet of carbohydrates, a diet both bulky and provocative of flatulence and gastric acidity. As indeed in gout itself, a careful mixed diet will answer best; and on two points we must especially insist—on restriction of liquids during meals, and on thorough mastication of the food, whether it be soft or hard. In more than one case I have seen great relief to follow fine chewing and the restriction of liquid at meals. Even between meals it is not well to allow the patient to drink largely; the blood-pressure can hardly thus be raised, as Huchard asserts, enormous quantities would be required for such a result, but the output of the ventricle may be increased, and therewith its work. It is scarcely needful to insist upon the use of food which is at once easy to digest and worth digesting; at the same time some foods are indirectly worth eating if they are grateful to the eater, and thus stimulate the secretions.

Alcohol is overdone in all heart diseases. The immediate relief to the sufferer is often considerable, and as a cardiac stimulant in time of danger it is indispensable. As an ordinary article of the patient's consumption its use is not without some drawbacks; it disturbs blood-pressure, its effects accumulate more rapidly for harm in persons who cannot take much exercise, and the perpetual nips, in which too often they are led to indulge themselves, directly induce those very conditions of venous stagnation and degeneration of the cardiac muscle which we are on our guard to avert. On the other hand, such patients are often cheered by a little claret and water, a light hock or some well-diluted spirit with meals, drams being strictly reserved for critical occasions. If on every access of palpitation or faintness the nurse is to run for the brandy bottle, the patient's state will grow worse rather than better.¹

In respect of management it is difficult to give general directions. In no cases are tact and experience more valuable. The young practitioner must remember that if, on the one hand, there be a danger of injury from the effects of a careless life, on the other the harmful effects of "valetudinarianism" are no less; and the patient in gaining his life may lose it. We must trim our treatment according to the phases and peculiarities of the individual. Fraenkel well says that to know that one has heart disease may be more mischievous than the disease itself. Let your patient understand that he has a weak heart, and that he must rigidly observe your rules of life, but not otherwise fash himself; and to some sensible and trustworthy friend of his tell the whole truth and the risk of sudden death if such there be; that like other wise men the patient may have his affairs in order.

In the matter of exercise often lies the decision whether the patient be allowed to follow his calling. If the occupation be one of muscular

¹ In his work on "Seule Heart" the veteran physician Dr. Balfour gives admirable directions for treatment of heart disease, and at greater length than it is possible for me to give in this place.
labour the patient cannot but leave it; a working-man must seek some quieter means of subsistence, as a caretaker or the like. A sportsman must contract the field of his pastimes: the salmon rod must give way to the lighter engine of the trout-fisher; cricket to golf: the moors must be forsaken for the stubble and the covert, and the hunter exchanged for the nag. Cycling is by no means an unfit recreation for the subject of heart disease, in its earlier stages; if so be that he is already a good rider, and will ride circumspectly. Whatever pursuit be admitted, and much will depend on the degree of incapacity, one caution must be remembered on all occasions, namely, that although the sense of oppression which checks exertion can be "worked off," unless very severe, by perseverance, it is a grievous error thus to persevere. It seems then that the heart does not so much pull itself together, as become blunted to the persistent strain. The cry of the burdened heart must never be disregarded. And yet again while we offer this necessary caution we shall not forget that perpetual timidity is even worse for the patient than occasional indiscretion. So long as he lives let him live, so far as may be, the life of a good citizen. Above all do not let him mope, or become entirely possessed by the blind and ignoble desire of the mere prolongation of days. We who have to minister too often to these unprofitable uses of the world, can proudly point to men, great examples in our own profession, who showed us how to live most nobly when death was treading in their footsteps. The physician who inspires moral health into his patient brings comfort also to his body.

Drugs.—During the latent period of aortic regurgitation those drugs only will be required which are of service in common ailments; specific remedies are rarely necessary. In case of acute onset, such as rupture of an aortic cusp, the measures described already under the heads of management and diet may be all that is required. Hearken, let us say again, to the cry of the burdened heart; no hypertrophy can go forward while the organ is embarrassed. Under the unwonted stress it may be necessary, while the heart is pulling itself together, to put the patient to bed until the heart has begun to turn its reserve capacity into the statical condition of hypertrophy. As this is attained the patient will return gradually to the ordinary habits of life. I have not found digitalis of great service in this stage. On the contrary, gentle mercurials, gentle salines, a little potassium iodide—means which reduce blood-pressure—are more helpful. In this stage too much care cannot be given to save the work of the heart in all directions, whether of muscular work, of the digestive and other organic functions, of cerebral and emotional activity. When this stage is passed, and something like compensation established, the patient will betake himself to moderated exercise and a more bracing moral life. If during the early period there be intercurrent times of strain, due either to indiscretion or to some fluctuations of inner health, intervals of more or less seclusion will again be enjoined, and the above indications repeated. The best all-round medicine for heart disease in these phases is blue pill.
In preparing this section I saw before me the duty of reading over the multitudinous arguments which have been written upon the use of digitalis in aortic insufficiency, a grievous prospect, in this intention I have given up. After all that is written, the subject lies in a good deal of physiological obscurity, and it is best for the present that each observer should give the results of his own impressions as simply as possible. Against its use in aortic insufficiency we have the eminent authority of Corrigan; in favour of its use that of Balfour.

Let me repeat that if the excised heart of a small animal be so attached to a pressure bottle, that pressure can be increased gradually, it will be seen that with each increment of pressure the base line of the cardiographic curve will fall; the ventricle dilates. Why does not the ventricle in all cases, in health or in disease, dilate to its extreme limits at once? Because of its "tone"; probably also because of the well-known reaction of the "loaded muscle." A loaded muscle, although prevented from lifting the lever so high as before loading, contracts more strongly. For what we know of tone, a property of the highest importance in cardiac functions, we are largely indebted to Dr. Gaskell. My own view is that if the property of tone be fundamentally one with that of contractility, it has become so far differentiated from it that the two virtues may be discussed separately. Tone we may define as that property in heart, artery, or other hollow viscus which preserves the mean diameter of the part; contraction as that which enables the organ, nevertheless, to obey stimulus and to perform particular acts. The vermicular movements of the bowel and of an arteriole are due to the quality of contractility; their tone preserves their mean diameters in spite of distension or contraction. Were it not for tone a hollow organ, often subject to extravagant demands, would be strained and perhaps ruptured. In the heart it is tone which does much, if not all, to prevent loss of form under the great variations of internal pressure.

In the year 1868, when Dr. Milner Fothergill was the resident medical officer to the Leeds Dispensary, I placed a large collection of cases of heart strain under his superintendence, and in order to test our remedies for these patients, we carried out together a series of experiments on digitalis, which Fothergill afterwards published in his Jacksonian Essay. We demonstrated the effects of digitalis on the hearts of frogs and small mammals, effects which are now too well known to need narration here. Suffice it to say that the chief effect is an increase of tone, which may be pushed to a degree inconsistent with normal function. When a solution of digitalis is dropped on a frog's heart we see an increment not of contraction but of tone. The heart goes on contracting with a smaller and smaller volume till for lack of blood the animal is moribund; when other variations, such as fibrillar contraction, may supervene. In aortic insufficiency, the regurgitant stream does not exactly "impinge upon the inner wall of the ventricle at a moment of relaxation," for it can scarcely be said that the ventricle is "relaxed"; the mischief is that the pressure is abnormally increased at a moment
when the muscle is at the disadvantage of greater cubic capacity, and when the direction of motion is with the regurgitant stream. The "loading" indeed, if not excessive, stimulates the organ to stronger contraction (reserve capacity), and this dynamical reinforcement becomes statical as hypertrophy. The same process being repeated again and again, the heart attains the huge dimensions with which we are familiar; and in the muscle itself there may be no limit to such increase, the limit being imposed by the scale of the associated structures. Were tone absolute, there would be no dilatation; hypertrophy alone would take place, and the output would be too small; on the other hand, if, as in chronic strain, the tone is overborne little by little, dilatation ultimately soon surpasses hypertrophy.

Tone, then, is the quality to be watched and supported; and in digitalis we have a means of intensifying tone, of moderating distensibility. Now tone, like any other quality in excess, may be injurious, and the output of the constricted ventricle may fall short of the demands of the system. That the residual blood should become less and less after each contraction, and possibly vanish, is good; but if the shrinkage of volume goes on farther, the output may fall farther below the needs of the system than it did in the case of excessive residuum. On the body the result is practically the same. Hence one chief reason why digitalis should be used with especial precautions is lest diminished capacity come to the same thing as asystole. Again, when the muscle falls into degeneration digitalis seems to have other injurious actions, the nature of which is obscure. We cannot get fatty hearts of frogs for experiment. It would appear that digitalis acts not only on the tone of the cardio-arterial muscular coat, but also on the vagus. In tachycardia, and other conditions of rapid pulse, digitalis by giving tone to the arterial system often causes diuresis without reducing pulse-rate—without, that is, being able to get a hold on the vagus. Conversely in a degenerated heart, digitalis often seems to stimulate vagus action while tone is failing; then we get slowing, coupled beats or intermittence of the heart without diuresis (Fig. 61). Indeed the vagus interference by slackening such a heart may conspire to its further dilatation with increase of residual blood. This perilous result of digitalis is but too well known to us not only in aortic regurgitation, but also in other kinds of failing left ventricle. If, then, diuresis do not soon appear, the drug must be stopped and a little alcohol substituted for it. So long as the cardiac muscle is in fair condition, the working of digitalis counteracts the distension of the left ventricle and lessens the volume of residual blood, an aid too valuable to neglect if contingent dangers, such as an undue reduction of output, vagus meddling, or oppression of degenerate muscle, can be avoided.
If we can use the drug so far as to pull the heart together without constricting its cavity or arousing the vagus too much, we shall gain ground so long as the muscle is sound. Now we find, prolonged diastole or not, that in practice digitalis, used with discretion so as to brace the heart and not to string it up too tight, is indeed the most valuable weapon in our armoury while the cardiac muscle is sound. The advent of degeneration of the muscle cannot well be detected save by administering the drug experimentally in single doses, say in one dose of 10 minims of the tincture once every second day, taking the flow of urine as our guide. As to the "prolongation of diastole," in so far as propulsion is better, refluence is less; in so far as the ventricular cavity is less, residual blood is less; moreover, the pause is not all active diastole; during it the pressures in aorta and ventricle approximate, and during the later part of it are indifferent, or even reversed. Again, acceleration of the blood is almost entirely an abbreviation of the diastole, yet acceleration is not a help to the heart, but a sign of its undoing. The organ is then dependent for its integrity on its tone, and if, as we have seen, by digitalis the residual blood may be reducible by moderate constriction of the ventricle, the abnormal pressure at the first part of the diastole, when it is highest, tells upon the walls at a moment of less cubic capacity, and at a moment of greater resistance. In a word, as the ventricle dilates, the output, other things being equal, remains constant, and the mass of residual blood increases; if by digitalis tone can be enhanced, output and contraction volume will approximate again.

What are, then, the rules for the administration of digitalis in aortic insufficiency? No one would give digitalis when a big heart is thundering along its course and the arteries bounding under its pulse. But if the left ventricle be relatively too capacious, and the apex beat becomes diffused, put the patient to rest with his feet up, so as to diminish blood-pressure; and put him on tender meats, avoiding much carbohydrate and much liquid. Gentle deobstruents will probably be required also. Now if under these means the symptoms and signs of dilatation continue, administer one dose of digitalis, and if it is at least harmless, administer another twenty-four hours later, noting the rate and rhythm of the pulse and the volume of the urine; thus watchfully a safe judgment may be made as to the further use of the drug. Although a pulse over 80 may suggest, it does not dictate the use of digitalis; some evidence of dilatation is required; on the other hand, it can rarely or never be well to give digitalis if the pulse be at or below 75. I have a prepossession against digitalis in any case in which the heart intermits: it may be more than justifiable to give it in cases in which the intermission is but a subordinate element in a rhythm otherwise quick and irregular; but if intermission be the sole or a leading feature the drug is better avoided. If in later stages the right side of the heart seem disturbed, digitalis can rarely be otherwise than helpful. In such cases, indeed, we do not look too curiously to murmurs or even to valves; we watch the apex beat, the area of cardiac dulness and the volume of the urine.
DISEASE OF THE AORTIC AREA OF THE HEART

Perhaps digitalis is least needed in aortic stenosis; yet even in this malady, if the stenosis be constant or increasing, and the heart yielding, it may be necessary to introduce it occasionally, remembering, however, that, if the obstacle in front be very great, to spur on the heart is to ride for a fall.

The preparations of digitalis are so many, and the advantages and drawbacks of this and that are so many, that I must refer the reader to works on pharmacology for full discussion of these very practical points. In a case to which I was called in consultation three or four years ago, Nativelle's granules were used by the medical man in charge of the case, one of dilated heart; these proved so helpful that I have prescribed them occasionally since that time, and certainly can confirm my friend's good opinion of the preparation. I see Dr. Balfour also uses these granules, or a syrup made by Nativelle. I believe in the “cumulative action” of digitalis, but have no notion in what it consists; whatever preparation be used, it is well to use it intermittently. Death is so often sudden in aortic insufficiency, that its occurrence during the use of digitalis, or of any other means, must not be attributed too readily to medicine. If the stomach be disordered, digitalis, if given at all, must be given subcutaneously.

• Strophanthus is sometimes of great service; more frequently it disappoints us altogether. I have little experience of it in aortic disease; generally speaking, I should say that it is much more useful in young than in old people; it is in patients under thirty years of age that I can recall many cases of heart disease, chiefly of mitral regurgitation, in which the drug acted with celerity and efficiency.

Arsenic and strychnine come to our assistance at times when drugs which should be more directly potent fail or are inadmissible. If strychnine be prescribed at a critical moment and rapid effects be desired, doses much larger than those regularly given are required. For an adult fifteen drops of the liquor are not too much thus to prescribe as a single dose. If the patient complain of some slight rigidity the dose is intermitted, and no harm comes of the reaction. Arsenic is more adapted, of course, to chronic medication, and, whether as a nerve or muscular tonic, is very useful. Sir William Broadbent, I see, regards the virtue of phosphorus as even superior to that of arsenic. Caffein—the pure caffein of Merck, not the citrate—is an old ally of mine; it stimulates the heart when it flags, and it promotes diuresis. It is also useful in “cardiac asthma.” From 1 to 3 grains may be given for a dose; and in some persons it is better to push the drug early in the day, pretermittin it of an evening lest it disturb sleep. Caffein is useful as a cardiac stimulant in cases of slow pulse in which digitalis is out of the question. Good and strong coffee taken black may be substituted for the caffein if no great precision of dosage be necessary.

The nitrites are perhaps never required until symptoms of an anginose kind arise; then they are invaluable palliatives. The researches of
Professor Bradbury and Dr. Marshall indicate that of these agents the erythrol tetra-nitrate is the most useful, as its effects are easily calculated and more persistent. The amyl nitrite, being the most rapid in action, is to be preferred at critical moments; but its effect is fleeting. In cases of severe angina these agents are very precious to us, probably by relieving the stress upon the aorta wherein the pains originate; an end attained by slackening the heart as well as by expanding the peripheral vessels. I cannot but suppose, however, that these agents have some anodyne virtue besides the mechanical, for I have seen angina relieved by a nitrite, while my finger was unable to detect any change in the blood-pressure. In extreme cases of aortic disease the assaults of angina may be so frequent that the life of incessant suffering and apprehension is almost more than can be borne; in these cases the use of the nitrite of amyl may become almost a slavery. A craving seems to spring up which is not easy to discriminate from the sinking of the angina itself. Bradbury and Marshall have made researches into a method of combining the use of vaso-dilators with digitalis which seems to be of considerable promise (48).

I suppose that chloral is a dangerous remedy in heart diseases, especially in degenerate heart. Sir W. Broadbent proscribes it altogether; Dr. Balfour, on the other hand, speaks of the drug with appreciation. When chloral first came out, being less troubled with modern speculations about blood-pressures than we are now, and undisturbed by Gaskell and Shore on chloroform, I used chloral freely in the restlessness of heart diseases, not excluding those of old people. Indeed, to many old people with degenerate hearts I gave the drug year after year, and certainly with the greatest comfort. The anxious, perturbed nights of these sufferers are full of trouble and peril, and sedatives cannot be forbidden. I now use chloralamide, which, I am told, is safer than chloral, and certainly acts well, though scarcely so well perhaps as the latter. Balfour, while clinging a little to chloral, suggests the use of chloralose or paraldehyde instead. Trional is perhaps better than sulphonal; but neither is so useful as the drugs just named.

It is now thirty years since, in the third volume of The Practitioner, I recommended the hypodermic injection of morphine in heart disease; and testimony of the best kind, such as that of Dr. Balfour, has supported my advice. Dr. Leonard Hill says "morphine is one of the best vaso-constrictors and cardiac tonics we possess." By the mouth opium is behind other sedatives in value, its use being attended by grave drawbacks; but hypodermically, in doses beginning at one-tenth of a grain and gradually ascending to a quarter of a grain if necessary, it is a precious means of relief. The physicians who still protest against its use are unfamiliar with the practice. There is no remedy which calls forth so warm a tribute from the patient himself, who, after nights of watching and agony, sleeps a peaceful and natural sleep, and awakes almost forgetful of his plight. Of the drawbacks to the continuous use of morphine I may refer to the article on the subject (vol. ii.)
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Like any other potent remedy, it must be used seasonably and discreetly.

Ammonium bromide is sometimes of service in the minor degrees of restlessness, but, if long continued, is apt to be depressing. All the salts of potash are to be avoided, even the nitrate. Convallaria, sparteine, cactus, and the like, are only known to me in the blind uses of despair.

In this section I have spoken almost entirely of the treatment of insufficiency; of stenosis, I need not say more than will be gleaned incidentally, here and under the head of Diseases of the Arteries. Indeed, if there be no means of dealing with the local process, as by the use of potassium iodide, the management of stenosis is an eminent example of "expectant treatment."

P.S.—As these pages are being printed off, I hastily intervene to qualify or indeed to contradict my statements in the text concerning pulse delay. It seems probable that Sir William Broadbent and other authors are right after all in asserting that there may be cardiac-radial delay in aortic regurgitation. The misunderstanding is one more instance of the drawbacks of making tracings without time lines. The error and its correction may be reconciled by the elevation of the whole problem into a wider generalization. Throughout this article I have assumed that, except in advanced decay and toxic states, the systole of the heart is relatively constant in duration; this, on the researches of Cohnheim, Roy, and others, has been generally accepted. But it appears that the proposition is open to grave doubt in respect of more than one kind of heart disease. Dr. Chapman, who is good enough to keep me informed of his researches on the physiology of the circulation, sends me (1st April 1898) tracings taken from a case of pure compensated aortic regurgitation, which prove that in this case, at any rate, the systole was relatively prolonged. This observation, if well founded, will throw a new light on the failure of compensation in aortic insufficiency; it means, of course, exhaustion in a proportionately shorter period of years. The prolongation of the heart's contraction accounts for the slower transmission of the wave. The details of Dr. Chapman's case are as follows:—The pulse-rate in the tracings was (about) 75. Two tracings were taken (among others). In the first the systole occupied 0.40" (the normal systole for this pulse-rate being 0.32" to 0.33"), the diastole was 0.36" to 0.39". In the second tracing the systole occupied 0.50" to 0.53"; the diastole 0.53". To apply this observation, in Dr. Chapman's language, "the duration of the heart's work (on this basis) is about fifteen hours of the twenty-four, instead of ten or eleven hours." On the first tracing the cardio-radial delay was as great as 0.4". The time measurements were made with a reed vibrating at 84 per second. If I am naturally disconcerted to find, when it is too late, that much of my text in respect of these points ought to be modified, I trust I need make no apology for our common fallibility. It would seem, from Dr. Chapman's records, that cases of aortic regurgitation differ widely among themselves.

T. Clifford Allbutt.

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T. C. A.