other hand, it may have been due to the herpes zoster virus. Unfortunately, the brain and spinal cord were not examined at autopsy, and consequently its interpretation must remain a conjecture. An indication of the virus's presence in the blood-stream is afforded by a study of the B.S.R. in case No. 2. This showed a normal reading during the earlier part of the herpetic attack, and a rise to 25 mm. per hour at the onset of the varicelliform eruption. In case No. 3 the similar incubation periods and the three different clinical relationships, namely, herpes zoster to varicella, varicella to herpes zoster, and the simultaneous appearance of the two conditions, are strongly suggestive of the common identity of the viruses.

It would appear, from a study of these cases and the current literature, that the virus of herpes zoster and varicella is one and the same, and that this virus possibly assumes a dual role. A neurogenic rôle when it is confined to the posterior root ganglia, its fibres, its peripheral nerves, the perivascular spaces, and the cerebro-spinal fluid. A hematogenous rôle when it finds its way into the general blood-stream. This view will explain the three clinical manifestations of this virus infection, namely, herpes zoster, varicella, and the joint diseases.

The frequency of varicella outbreaks originating from herpes zoster can be explained by the fact that the neurotrophic variant can more easily, given the right host, alter its nidus and thus become a hematotropic one. The early unilateral herpetic lesions, and the constant interval before the varicella attack, is brought about by the neurotrophic virus taking a time to force a break-through from the perivascular spaces into the blood-stream, to assume the role of a hematotropic virus. The frequency of varicella in children and the non-immune, and the relative frequency of herpes zoster in the elderly and those who had contracted varicella in early childhood, can be explained by the selective activity of the biphasic virus.

Finally, the modification of the varicella eruption, when present with herpes zoster, may be due to the presence of some anti-bodies, already produced during the latent interval by the herpetic attack.

Summary

Three cases illustrating the presence of the concurrent diseases herpes zoster and varicella are described. Case No. 3 caused an outbreak in the home, following the recognised incubation period, and also the co-existing diseases of herpes and varicella in case No. 1. A case for the common identity of the causal viruses of herpes zoster and varicella is made, and its dual role that is neurotrophic and hematotropic is explained.

I am indebted to Dr. Ivor Lewis, Medical Superintendent of the North Middlesex County Hospital, and Dr. V. L. Collins, Physician, for their permission to report the autopsy findings and some of the details in case No. 1.

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Practicalities

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THE CLINICAL STUDY OF THE CARDIO-VASCULAR SYSTEM

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PART II

In the previous article we discussed the clinical differential diagnosis of those diseases grouped under the general heading of cardiac pain, and showed how such diagnosis could often be made by carefully questioning the patient. The physical signs associated with these diseases often, in themselves, mean little, though they sometimes point to certain complications, such as auricular fibrillation, which we will not discuss in relation to these histories, as they in no way differ from the conditions which we are about to describe. Some auscultatory signs, such as the tone of the heart sounds or their reduplication, may help the diagnosis. For example, in a

patient whose heart is the seat of either acute or chronic myocardial change, the heart sounds tend to become of equal intensity and to have no tone—being, in fact, as dull and lustreless as is, physiologically, the cardiac muscle. This is quite different from the question of “distant” heart sounds. These depend upon the size of the chest, the subcutaneous adipose tissue, the amount of lung tissue covering the heart, and so on; but in this case, although distant, the heart sounds retain their characteristic cadence.

Reduplication of either the first or second sound leading to a gallop rhythm may be of importance in the diagnosis of myocardial disease. Reduplication of the second sound is a common condition, and is usually due to an asynchronous closure of the semilunar valves, following asynchronous contraction of the two ventricular muscles, which happens when the passage of the impulse down one or other of the main bundle branches is impaired. Reduplication of the second sound is usually evidence of myocardial failure, and as such is of ill-omen.

There are two conditions in which reduplication of the first sound is of no pathological significance. The first is the presence of an audible auricular sound, which is usually heard in the very slow-acting heart, especially in children. The second is a reduplication of the first sound in an otherwise healthy heart. This condition is often diagnosed as mitral stenosis, as the first sound is mistaken for a presystolic murmur. In many cases, radioscopy is necessary to confirm the diagnosis by confirming the normal shape of the heart.

Except for these conditions, the presence of gallop rhythm is a sign of myocardial failure, and should be given a bad prognosis. Sure diagnosis of myocardial change is, however, reserved for the electrocardiographic findings, which we will discuss at a later date.

Let us, now, turn our attention first to the abnormalities which can be detected by examination of the pulse.

The pulse rate may be abnormally fast—that is, over 84 per minute. Such an increased rate should lead one to consider these four conditions—simple tachycardia, paroxysmal tachycardia, auricular flutter and auricular fibrillation. Except in fibrillation, the rhythm is usually regular; though in cases of flutter it is sometimes otherwise, due to inconstancy of the prevailing degree of heart block. Each of these conditions should be diagnosed by clinical methods alone; but proof of such must be left to the electrocardiograph.

By the term ‘simple tachycardia,’ we mean that the normal action of the heart becomes more rapid, due to an increased output of stimuli from the sino-auricular node; and we cannot, therefore, say that it is primarily of cardiac origin. Thus it may occur as a physiological variant in a healthy subject, and such tachycardia is increased during exercise, mental excitement, or when there is increased heat produced in the body, as in fevers and acute febrile illnesses. It may also occur when there is a lessened blood volume such as occurs after haemorrhage; or in shock, due to the lowered blood pressure removing the normal vagal control through the sino-aortic nerves. In exophthalmic goitre, the increased secretion of thyroxin has a direct effect upon the sino-auricular node, causing an increase of the stimulus output, which leads to a simple tachycardia. In cases of chronic toxæmia due to pulmonary tuberculosis, chronic tonsillitis or other focal infections, a tachycardia is produced, due probably to metabolic disturbances within the body. In acute and chronic alcoholism, and in the effort of syndrome of DaCosta, likewise, a tachycardia occurs, caused probably by the mental instability present in these conditions.

Of the symptoms of simple tachycardia, the most frequent complaint is that of palpitation, often accompanied by an unpleasant throbbing in the head and neck, restlessness, agitation and nervous prostration.

Of the signs which occur in this condition, the cardiac impulse is often more diffuse than normal, and a soft, blowing systolic murmur tends to appear at the apex or base. Both these signs are a direct result of over-action of the heart.

Positive diagnosis of a simple tachycardia will only be made on electrocardiographic examination, but its presence may be presumed if one finds a tachycardia not above 140, and the signs already referred to, especially if this rate falls with rest. The possible aetiological factors should be reviewed then, and in most cases a cause will be found and the diagnosis made.

Paroxysmal tachycardia is due to the heart acquiring, temporarily, at some distant site on the myocardium, a new rhythm centre which is quite uncontrolled by the cardiac nerves. The pathogenesis of this condition is quite unknown, but the paroxysmal nature of the attack should be suspected—and indeed diagnosis is rarely open to question if the following characteristics are present, viz. the patient complains that at one moment he is perfectly normal, and that, suddenly, he has a throbbing or severe palpitation in the chest, which is often accompanied by

pulsation of the head and neck, lasting for a varying period of time and ending as suddenly as it began. It should be stressed that unless both beginning and end of the attack are sudden the diagnosis of paroxysmal tachycardia should not be made. Sometimes, however, the ending is not so characteristically abrupt because the attack may die away in a series of short paroxysms or by a declining number of extrasystolic interruptions. In these cases the patient is aware that the attack passes off gradually. Diagnosis is therefore made only by an examination during the attack or its period of regression.

The ventricular rhythm manifested by the actual state of the pulse is regular, and the rates vary from 110 to 220 beats per minute, usually in the region of 160 to 200 per minute. This condition may last for a few seconds or for days, but usually it is over in an hour or so. It occurs most often between the ages of 20 and 30, but it can arise at any age, often with no sign of organic disease between attacks, nor any history of previous cardiac disease. Of the associated diseases, rheumatic fever is that which is most frequently present.

The paroxysm may be provoked by exercise, or emotional disturbance, but in many cases no exciting cause can be found. Usually the length of the attack in any one patient is fairly constant. If the patient is of a nervous temperament, he will experience alarm, showing pallor, sweating, tremulousness and weakness. The end of the attack is often marked by a sharp pain in the chest or one or two violent heart beats. If this condition persists it may—and it should be noted that it is one of the few conditions which does—cause cardiac dilatation, passing on to acute heart failure, with congestion and all its attendant symptoms and signs. Death rarely occurs if the heart failure is treated rapidly; but if not the end may well be fatal. Even in severe cases of cardiac failure, the return of the patient's condition towards normality follows rapidly when the heart regains its normal rhythm.

Some patients with this condition find that certain things will cause the return of the normal rhythm, such as drinking a glass of very cold water, assuming some particular posture, or pressing with the thumb upon either one or other of the carotid sinuses in the neck. If these factors are present it gives weight to the clinical diagnosis. The auscultatory signs are those of a tachycardia only.

The next type of tachycardia which occurs is that due to auricular flutter, in which a continuous circus movement occurs around the auricle. It should be suspected in all cases on finding a pulse rate of about 150.

Actually the auricle is beating at a rate of 300 or more to the minute, but the A.V. bundle is incapable of transmitting this number of impulses, and therefore only every alternate beat is transmitted (2:1 block). Sometimes, however, a 4:1 block may develop, and in this case, instead of having a rate of 150, the rate will be 75, but in this latter case there will nearly always be a period during which the patient is conscious of a rapidly-beating heart, especially after exercise, and on examination during this period a rate of 150 will be found, due to halving of the heart block which has been present. Thus the patient may complain of attacks of palpitation or of syncope, and because of the mechanical disadvantage of the heart, there will be symptoms of cardiac insufficiency, with dyspnoea and pain on exertion, and the clinical features mentioned above will be found. Flutter differs from simple tachycardia in that rest makes no difference to the pulse rate. To differentiate between flutter and paroxysmal tachycardia may be more difficult, but the pulse rate in the latter is higher; and further, if such a tachycardia has lasted for over ten days, especially in the middle-aged, it is almost certainly flutter. Occasionally, in flutter, after exercise, the ventricle will respond to the actual auricular rate, thus leading to syncope because of an inefficient circulation; whereas in paroxysmal tachycardia acute heart dilatation with failure is more likely to occur.

In some cases of flutter there is marked pulsus alternans in which strong and weak beats follow each other in a rapid and regular sequence. Its appearance does not imply a graver prognosis, as was once thought, and the normal pulse waves will re-appear on the assumption of normal sinus rhythm. On auscultation a regular tachycardia only will be heard.

Auricular fibrillation is believed to be caused, like flutter, by a wave of contraction circulating in an irregular course around the auricle. Like flutter, this is, therefore, an intrinsic cardiac fault. The normal quick and sudden systolic contraction of the auricular wall is lost, and in its place fine tremulous movements occur which are insufficient to expel the blood in the auricle; therefore the auricle remains in the position of diastole. These fine, tremulous movements are caused by the irregular circus movement producing small, irregular contractions in a varying number of muscle fibre bundles. These contractions occur at any rate from 120 to 240

a minute. This rhythm, once established, is essentially persistent, although there are a few cases which are paroxysmal in type. The impulses which are therefore gathered together at the auriculo-ventricular node pass down the A.V. bundle in an irregular fashion, but providing the ventricular muscle is not in a refractory state, contraction of the ventricle as a whole will occur in an irregular fashion. This is revealed by the pulse, and it is typically an irregular irregularity which is increased by exercise. If the pulse rate is over 120 to the minute and irregular, auricular fibrillation is almost certainly present. The other irregularities of the pulse wave can usually be differentiated by exercising the patient; if the irregularity is due to sinus arrhythmia it will disappear when the rate is over 100 per minute; if due to extrasystoles when the rate is increased to 120 or more; in those rare cases of irregularity due to auricular flutter, if the rate rises above 130 to 140 the rapid regular rhythm of flutter will appear. If in doubt as to the diagnosis of fibrillation, raise the pulse rate by exercise or by the use of inhalations of amyl nitrite, and the gross irregularity of the pulse will become more evident.

Thus it will be seen that auricular fibrillation can be diagnosed from an examination of the pulse alone. Auscultation will confirm the diagnosis, since heart sounds having the same gross irregularity will be heard, only in a more intensive form, because the number of ventricular contractions which occur are greater than the actual pulse waves which they produce, since many of these contractions are insufficient to raise the semi-lunar valves. One hears, therefore, a mixture of irregularly spaced heart sounds, in many cases the second sound of the cycle being absent. This finding of a ventricular rate greater than pulse rate in an irregular tachycardia is itself almost diagnostic of fibrillation.

Auricular fibrillation occurs most frequently as a sequel to an attack of rheumatic fever or chorea, and it is very often associated with mitral stenosis. In this type the usual age-incidence of the onset is between 20 and 30 years. If fibrillation occurs for the first time during the age group 50-60 years, the pathogenesis is that of the degenerative changes which have occurred in the myocardium due to atheroma of the coronary arteries or to hypertension. If neither of these two great groups can be implicated in the aetiology of the condition, the next most common cause of fibrillation, especially in women, is hyperthyroidism.

Paroxysmal fibrillation, in which attacks of fibrillation are present, likewise, is most often due to a rheumatic infection, especially with an associated mitral stenosis, but there are other causes which should be remembered; of these, hyperthyroidism is the most frequent, but it may also occur in acute infections such as pneumonia or in acute coronary thrombosis. Occasionally, too, it may occur in chronic occlusion of the coronary arteries, by atheromatous change.

Of the other causes which should be remembered, paroxysmal fibrillation may be caused, in women, by taking thyroid tablets for slimming purposes, and in men it may occur in chronic alcoholism, especially whisky drinking. Finally, it may be due to the exhibition of digitalis for simple tachycardia, especially that associated with exophthalmic goitre. The diagnosis of paroxysmal fibrillation is often more difficult to make unless an actual attack is seen, but once this type of arrhythmia has occurred it tends to become permanent.

The next group of abnormalities of the pulse are those associated with slowing of the pulse;—bradycardia, but one must be certain that the slow pulse is due to a true bradycardia, and not to extrasystolic interruption which fails to give a pulse wave. This can easily be confirmed by simultaneous auscultation of the heart sounds and palpation of the pulse wave. For instance, if the heart be beating at a rate of 84 to the minute but each alternate beat is an extrasystole which fails to give rise to a pulse wave, the pulse at the wrist will be 42 beats to the minute; on auscultation one will find that there are three sounds to each pulse wave, the first two being the normal sounds and the third being caused by the ventricular contraction of the extrasystole; the second sound from the extrasystole being absent owing to the "failure-to-open" of the semilunar valves. Conversely, if on auscultation one hears as many heart sound cycles as there are waves, the cause of the bradycardia cannot be due to extrasystolic halving. This point is stressed because of its importance in making the clinical diagnosis of a bradycardia.

The causes of a true bradycardia are, firstly, a sinus bradycardia, secondly, heart block and thirdly, the very rare condition of A.V. nodal rhythm. Sinus bradycardia is due to increased vagal tone and is considered to be present when the heart rate is under 60. The heart itself is usually healthy, and it may occur as a simple physiological condition in old age or in trained athletes; but there are many normal people who have a persistent bradycardia all their lives.

Sinus bradycardia may also manifest itself during convalescence from acute illnesses, or after exposure to cold. Increased intracranial pressure (e.g. in meningitis, haemorrhage or

tumour) will cause a slowing of the heart rate from direct central control. In myxoedema or melancholia, sinus bradycardia is often present, and also, for some unexplained reason, it is often very marked in cases of jaundice. A similar bradycardia often accompanies the ordinary fainting attack, and may occur during vomiting, and in the vertigo of middle ear origin. All these are caused by inhibition of impulse output by increased vagal tone.

Heart block, on the other hand, is due to abnormal conduction in the heart, in which the ventricle fails to respond to the stimuli which originate in the auricle, because of the lessened power of conduction of the A.V. node or bundle. It may occur at any age; it may be congenital in origin (especially when such a defect as a perforated intraventricular septum involves the bundle), or it may be due to involvement of the A.V. bundle in inflammatory lesions such as rheumatism and syphilis. In later life, degeneration of the myocardium with or without calcareous deposits, may involve the A.V. bundle. Very rarely, tumours (benign or malignant) may cause heart block by involving the bundle of His.

In minor degree it may lead to occasional intermittence of the ventricle, notably in acute infections such as rheumatism, diphtheria, pneumonia, etc., and occasionally these minor disturbances are caused by a direct vagal influence. This, however, is usually temporary, and may be relieved by atropine. The only symptoms associated with heart block are due to a profound slowing of the ventricle or to sudden cessation of the circulation, leading to syncopal attacks.

A.V. nodal rhythm is a rare condition of no prognostic importance, in which the pacemaker of the heart shifts from the S.A. to the A.V. node. It is difficult to diagnose without an electrocardiogram, and the only clinical sign is the systolic filling of the jugular veins. This is due to the fact that, because the impulse stimulates both auricle and ventricle simultaneously, the blood is unable to pass from auricle to ventricle, and therefore, the pressure is transmitted to the venae cavae. The differentiation of bradycardia can usually be performed by bedside signs, although again, as with all these arrhythmias, the absolute diagnosis is obtained by electrocardiographic studies.

In simple bradycardia the ventricular rate is rarely less than 50, whereas in a 2:1 heart block, rates from 40 to 50 prevail. In simple bradycardia the ventricular rate can be increased with exercise, excitement, atropine or amyl nitrite, whereas in 2:1 heart block the rate will often abruptly double itself, due to the resumption of a 1:1 ventricular response. Complete heart block, on the other hand, is suggested when the ventricular rate is regular, below 35. A constant rate of 30 always means a complete heart block, especially if there is a history of syncope, and it is usually unaffected by exercise, etc. Diagnosis is made absolute in this case by the characteristic variation of the heart sounds, which is independent of respiration. If the breath is held, the first and second heart sounds are heard with each ventricular systole, but they vary in intensity, and they may show in constant reduplication from cycle to cycle. Occasionally, especially if the rate is very slow, the third auricular heart sound will be plainly audible, particularly over the right border of the heart or in the epigastrium.

Instead of noting a regular pulse, it may be found that there is marked irregularity. In this case, the following conditions, most of which can be diagnosed by clinical methods, must enter into the differential diagnosis. The chief causes of cardiac irregularity are (1) sinus arrhythmia, (2) premature or extrasystoles, (3) heart block, (4) auricular flutter, (5) auricular fibrillation, (6) paroxysmal tachycardia, (7) pulsus alternans;—much more rarely (8) A.V. nodal rhythm and (9) ventricular fibrillation. Some of these conditions have already been discussed, and so it will be necessary only to describe the others.

Sinus arrhythmia is a condition in which the heart rate accelerates during inspiration and decelerates during expiration. It occurs most commonly in children, and is of no prognostic significance. It is due to alteration in vagal tone from the impulses arising in the lung and pleural sacs. It will always disappear with exercise.

An extrasystole or premature beat is a condition in which the normal sinus rhythm is interrupted by the interposition of an abnormal beat; this beat is followed by a compensatory pause after which the normal sinus rhythm is resumed; therefore the interval between the beats which precede and follow the extrasystolic beat is the same as the interval between any three normal beats. Such an extrasystole may occur frequently or infrequently, regularly or irregularly, after each 1, 2, 3 or 4 beats, or in a group. The extrasystole, is due to a stimulus which arises on some part of the myocardium away from the sino-auricular node, and it can therefore be of three varieties—auricular, auriculo-ventricular nodal, and ventricular in origin, the latter

being the most common. In the ventricular variety there are usually four heart sounds for the normal beat and the succeeding extrasystole, but in those cases in which the extrasystole occurs early in diastole, before the cardiac muscle has fully recovered from the preceding contraction, there may not be enough power in the contraction to raise the aortic valves. In this case, only three heart sounds will be heard. The amplitude of the pulse wave so produced is not so great as normal, and the earlier in diastole that the extrasystole occurs, the less is the pulse wave. If it occurs early enough, no wave at all will be felt and, therefore, one will find an actual intermission of the pulse, or dropped beat. Should the extrasystole occur regularly after each normal beat, giving rise to a pulse wave, a coupling effect (pulsus bigeminus) is found; but if no pulse wave is produced there will be halving of the pulse rate. If after two normal beats a premature wave is produced the pulsus trigeminus will be present. Conversely, two normal beats followed by a dropped beat will give pulsus bigeminus again.

In the auricular variety of extrasystoles, the compensatory pause is often not as long, but otherwise it has the same clinical characteristics as the ventricular variety. The auriculo-ventricular variety is rare.

The exact diagnosis of extrasystoles must, of course, be reserved for the electrocardiographic study, but palpation and auscultation should lead to a correct diagnosis. When on palpation of an otherwise regular pulse a sudden pause breaks into the rhythm, it is either an extrasystole too weak to give a pulsation, or it is partial heart block, in which case there is no stimulus reaching the ventricular muscle. If it is the former, a premature beat can be detected at the cardiac apex, and the first heart sound will be heard in the early part of the pause; whereas in partial heart block, there is neither cardiac thrust nor heart sound present.

In some cases in which extrasystoles are occurring at very frequent, irregular intervals, it may be very difficult to distinguish the conditions from those attendant upon auricular fibrillation, but usually this can be done by exercising the patient. This leads to a decrease in the extrasystoles and therefore a more regular rhythm; in the fibrillation, however, the irregularity is increased.

Heart block, auricular flutter, auricular fibrillation and paroxysmal tachycardia have already been discussed.

The presence of pulsus alternans, i.e. the alternation of strong and weak beats, as detected at the radial pulse, used to be considered of serious import, but it may occur in conditions other than in left ventricular failure from hypertensive disease, this being, of course, the most common condition in which it is seen. In such a case it is of serious prognostic importance. Pulsus alternans is more frequently transient than continuous, and it may be due to partial systole, in which some of the muscle fibres fail to contract or only contract feebly with each alternate beat, as they do when the excitation process is not reaching them, due to defects in the conductivity of a branch of the A.V. bundle, or in those cases in which there is a prolonged refractory period of the ventricular muscle. Therefore alternation is fairly common after a premature contraction, in simple tachycardia, in auricular flutter and in paroxysmal tachycardia; it may also occur during convalescence from acute diseases.

A.V. nodal rhythm is a rare condition which we have already mentioned. Ventricular fibrillation is usually a terminal event commonly following an acute coronary thrombosis. It is rarely diagnosed clinically, although its occurrence is to be presumed in those cases of sudden death due to coronary infarction or thrombosis. It may, very rarely, occur in complete heart-block, and it is one of the three conditions which cause Stokes-Adams attacks. In this condition the circulation to the brain is not maintained, leading to sudden black-outs or periods of unconsciousness. Usually the cause of Stokes-Adams attack is ventricular standstill, during which time the ventricle fails to beat for a period of 5 to 15 seconds. More rarely, the ventricle may suddenly develop a tachycardia of its own, or else the ventricular muscle begins to fibrillate. That these two conditions can be compatible with life is shown by the fact that electrocardiographs have been taken during these attacks, and followed by the resumption of the idio-ventricular rhythm, but the patient rarely survives very long because, should these conditions continue longer than 20 seconds, death will almost certainly occur.

We must mention the causes of pulsus bigeminus and pulsus trigeminus. Pulsus bigeminus, or the coupling of beats, is a condition in which the pulse beats occur in pairs for long or short periods, either at regular or irregular intervals. This may happen in cases when, with a normal sinus rhythm, an extrasystole of sufficient force to produce a pulse wave occurs regularly in place of each alternate beat; or when an extrasystole insufficient to produce a pulse wave, occurs.

regularly after two normal beats; and when in partial heart block the ventricle does not respond to every third impulse sent out by the sino-auricular node. It may also occur in the rare condition of sino-auricular block, in which the sino-auricular node sends out two normal impulses followed by a period in which the normal third impulse is lost.

The most common cause of an irregular pulsus bigeminus is a ventricular extrasystole occurring regularly after each ventricular contraction in fibrillation; this is seen especially well in cases of fibrillation which have been fully digitalised.

The same causes hold for pulsus trigeminus, in which the pulse beats are grouped in threes. Clinical recognition of these conditions should be possible if the differential signs already mentioned are remembered.

We will now consider heart failure. Lately, the conception that there may be failure of either ventricle has been recognised. Left ventricular failure occurs in the late stages of hypertensive disease, in conditions of aortic incompetence and in atheroma of the coronary arteries; the symptoms and signs which are associated with such a failure show themselves directly upon the pulmonary system; in other words, it is a back-pressure effect. When either hypertension, or aortic incompetence, is present, the left ventricle is capable of responding, for some time, to the extra load, maintaining an efficient circulation. Later the ventricle fails, begins therefore to dilate, leaving a certain volume of blood within, at the end of systole. This in its turn leads to interference with the emptying of the left auricle, causing a rise of pressure within it. This rise of pressure is then transmitted to the pulmonary veins, leading to pulmonary congestion. The congestion in its turn diminishes the vital capacity of the lungs, lowering the dyspnoea point. Further, the congested pulmonary circuit produces an exaggerated vagal respiratory reflex, leading to rapid but shallow breathing. As the congestion becomes more marked, the increased pressure in the pulmonary circuit is transmitted to the right ventricle, adding greatly to its work. The vicious circle continues, the right ventricle dilates and fails, and this produces increased pressure in the right auricle. Since, for all practical purposes, the venae cavae and the right auricle are contiguous, this pressure is transmitted directly to the systemic circulation, which in its turn shows a marked rise in venous pressure. Such increased venous pressure reflexly stimulates breathing through the Bainbridge reflex. Hence it will be seen that a patient having a failing circulation, tends to have a higher resting pulmonary ventilation owing to these reflex stimuli, from not only the congested pulmonary veins but also from the congested systemic veins. Therefore we find that cases of failure are accompanied by an increased respiratory exchange. During exertion the muscular movements reflexly stimulate breathing further, because the venous return to the heart is increased; but since the heart, even at rest, cannot cope with the increased load, the pulmonary and systemic venous congestion must be intensified. Therefore, respiration is stimulated further, not only by increased reflex stimuli from these effects, but also by the normal chemical changes which are occurring during exercise. Because of all these factors, in failure, exertion causes a much more marked increase of the respiratory exchange than it would in the normal person, and because of the 'debt' which has been incurred, the respiration rate takes longer to return to its resting state. The insufficient blood-flow through the lungs also leads to excessive carbon dioxide retention, and an inadequate oxygen intake; the carbon dioxide accumulates in the blood, causing a further stimulation of breathing, and the anoxia will intensify this effect. Because of the retention of carbon dioxide, the buffering power of the blood is lost, resulting in acidæmia, which again increases the respiratory rate. Thus it will be seen that a vicious circle is set up which becomes very difficult to break. It will also be appreciated that a case of left ventricular failure will eventually progress until there is a superadded right ventricular failure.

Failure of either ventricle will produce different signs. Thus in left ventricular failure, the back-pressure makes itself felt upon the pulmonary circuit, and this leads to a condition of paroxysmal dyspnoea (cardiac asthma). The attack of dyspnoea usually sets in at night, and the patient wakes up with a feeling of anxiety and oppression in the chest. He sits upright, or even seeks an open window. His heart is pounding, he begins to cough, and is soon fighting for breath. The dyspnoea is extreme and distressing, the breathing is noisy, and often there is an associated bronchial spasm. If examined at this time, evidence of the raised pressure in the pulmonary circuit will be found producing a systolic pulsation over the second and third left intercostal spaces which may be both seen and felt. At first there is little sputum, but later a little frothy and often blood-stained sputum is coughed up. On auscultation the whole chest shows signs of bubbling secretion, and the patient may pass into an attack of acute pulmonary

oedema, with its characteristic profuse, watery, blood-stained sputum. This is the picture of a pure left ventricular failure. As already explained, it is due to the pulmonary congestion and to the diminished capacity of the respiratory reserve.

Such an attack may often occur during the day, following an attempt at coughing, but the alarm experienced is never as great. The fact of its occurrence at night is almost certainly due to the sudden stimulation of the depressed cough reflex by an accumulated secretion into the bronchial tree.

In right ventricular failure, on the other hand, the symptoms and signs are due to the increased back pressure in the systemic circulation. When the patient is ambulatory the first sign of this increased pressure is seen in the legs and ankles, where the patient may notice himself that on taking off his shoe at night an impression remains. If he is in bed, this oedema will be noticed first over the sacrum (but not by the patient!). Normally, when a patient is lying flat, the jugular veins stand out as cords which are easily compressible, and which may show some transmitted systolic pulsation from the auricle, but on sitting up, the veins collapse and are not seen, except, perhaps, in the root of the neck. When the systemic venous blood pressure is increased, the jugular veins stand out even in the sitting-up position, showing marked congestion, and are no longer so easily compressed. The height to which the jugular veins rise when the patient is sitting up can be taken as an indication of the degree of venous congestion. This venous congestion shows itself in other places. The small venules on the face may become distended, as will the veins coursing over the forearm. The liver takes a good deal of the initial shock of this increased pressure, becoming enlarged. This increased pressure may interfere with the normal functioning of that organ, and so lead to jaundice. If the pressure is great enough, or maintained for long enough, there will be a rise in pressure in the hepatic vein, which is then transmitted to the venous system of the abdomen. This will lead to a transudation of fluid within the abdomen, and ascites will result. In the kidney the back pressure manifests itself by the presence of albumin in greater or lesser degree. Casts containing red blood corpuscles may be found on microscopy of the urine, and in some cases the circulation is so slowed that infarction occurs, a frank haematuria may result. (The urine is usually brick red in colour, from the presence of urates.) An examination of the fundus oculi will reveal how general is this venous obstruction, for besides showing distended veins there may be small haemorrhages where those small veins have ruptured.

In left ventricular failure, because of the pulmonary congestion and the shallowness of respiration, cyanosis is often present, although never as marked as in right ventricular failure. In the latter, the cyanosis is due, at first, not to anoxia, but to the stagnation of blood flow which occurs on the venous side of the circulation. This is proved by the fact that the administration of oxygen does not improve the patient's colour, whereas venesection will do so immediately. Later, when pulmonary congestion becomes marked, oxygen administration will be beneficial.

(To be concluded.)

PART III.—Hypertensive disease; the diagnosis of valvular disease and a discussion on the significance of the systolic murmur.

Clinical Page

By STELLA INSTONE, M.D., M.R.C.P.

CASE I

A CASE OF POLYRADICULONEURITIS

Case Note

Mrs. C., aged 32, had never had any serious illness until July 1942, when she developed gastro-enteritis. No other member of her household was affected, nor was there any local epidemic of food-poisoning at this time. Full investigations proved bacteriologically negative, and her serum failed to agglutinate any organism of the Coli-Typhoid-Dysentery-Abortus group. Her blood-count showed a leucopenia with diminution of the granular cells: