possible, simultaneous observations were made by means of two armlets connected to the one manometer. In this way differences were observed in all the cases varying from 6 to 25 mm.; the differences in the limbs of the opposite sides did not correspond. In some instances the tibial reading was higher, and in others lower, than the brachial, but I have generally found both brachials to correspond. In a few cases also, when using the Riva-Rocci and Hill's instruments together, I have found a constant difference of 30 mm. or less between brachial and radial arteries, the latter being the lower. I assume that in all these cases the difference may be regarded as an expression of the variation in the resistance of the vessel walls under observation, but it of course gives no idea of the total resistance offered to compression. Then, on the other hand, one not infrequently obtains low readings in marked arterio-sclerosis.

CONCLUSIONS.

1. That maniacal and melancholic states are not associated with any constant modification of the arterial blood-pressure, nor any marked departure in it from the normal.
2. That the same applies to other types of mental disorder with the exception of congenital states in which the pressure tends to be low.
3. That the pressure bears some relation to the bodily tone, and increases as age advances; and that as melancholia is on the whole a disorder of more advanced years than mania it is only associated with higher manometer readings on this account.
4. That sedative drugs have a slight influence in lowering blood pressure.
5. That the manometer readings bear a fairly close relation to the sclerosis of the vessel wall.

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THE INTERPRETATION AND SIGNIFICANCE OF SOME IRREGULARITIES OF THE PULSE.*

BY

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In the textbooks of medicine the scanty accounts which are given of pulse irregularities do not elucidate their sources of origin. Of late years, however, an immense amount of work has been done upon this subject, both experimentally and clinically, and we now possess some real information as to the mechanism by which irregular pulses are produced. In clinical medicine methods of recording and interpreting irregularities of the pulse at the bedside have been elaborated.

Of this work Dr. James Mackenzie has been the pioneer. In 1902 he published his book on *The Study of the Pulse*, in which he gave descriptions of apparatus which he had invented for recording the movements of the various chambers of the heart, and he classified and described the various pulse irregularities. Since the publication of his book numerous workers have followed along his lines, and an immense amount of literature on the subject has arisen. The result has been to confirm Mackenzie's work in nearly all points, and to afford an insight into the mechanism of the heart's action which was previously unknown.

In most cases of irregular pulses it is impossible to know what the various chambers of the heart are doing without some graphic method, and without such a method it may be impossible even to detect an irregular pulse. Previous to the knowledge which graphic methods afford, I think that the main tendency has been to regard most pulse irregularities as having some unfavourable significance, though in reality sometimes an irregularity of the pulse has an important bearing, and sometimes appears as a harmless accident.

On the one hand, an irregular pulse may have an important bearing in disease. For instance, an occasional irregularity of the pulse during the course of pneumonia

may be the first sign of a fatal termination. On the other hand, I know a family in which all the sons possess an irregularity of the pulse in the form of ventricular extrasystoles. They are all perfectly strong, middle-aged men, and their father, who had a similar pulse irregularity, lived to the age of 82. In such cases a just opinion can only be formed by a comprehensive consideration of all factors. Again, it sometimes happens that a pulse may appear quite regular to the palpating finger, but when the movements of the heart are analysed, it is found to possess a gross irregularity. For example, in conditions of heart-block the radial pulse may appear to be quite regular, as in an instance which I shall show you, but by a graphic method it can be shown that the auricles and ventricles are absolutely dissociated. The factors which govern the normal beat of the heart are—first, stimulus formation; secondly, excitability; thirdly, contractility; fourthly, conductivity; and fifthly, tonicity.

The bias of opinion at present very strongly favours the view that the heart initiates and conducts by its own muscle fibres the proper orderly contraction of auricles and ventricles. This is known as the myogenic theory. Even if it is ultimately proved that the nervous system plays a considerable part in the normal beat of the heart, we have obtained from the myogenic theory a very good working explanation of many irregularities of the pulse. This theory assumes the muscle fibre to contain some physico-chemical material, which gradually accumulates until it is present in sufficient quantity to stimulate the muscle fibre to contract. Cardiac muscle has the peculiar characteristic that during contraction the stimulus matter is used up, and immediately following the contraction the property of excitability is temporarily lost. In other words, no further contraction can take place until the stimulus matter has been regenerated, and until the property of excitability has been restored. This period of enforced rest is known as the refractory period of the heart. It will be apparent that the heart, unlike ordinary muscle fibre, cannot be continuously stimulated to a tetanic condition, or even made to contract immediately after a previous contraction.

I wish to emphasize very strongly the fourth property of the heart—namely, that of conductivity. Any particular cardiac muscle fibre during its contraction has the power of exciting a contraction of the contiguous fibres, so that by causing the contraction of a single cardiac fibre a wave of contraction is conducted along the neighbouring fibres.

Can this principle of fibre to fibre stimulation be applied to the human heart? You will remember that the auricles are separated from the ventricles by distinct rings of fibrous tissue—that is to say, there is no direct continuity of ordinary cardiac muscle fibre between auricle and ventricle. A wave of contraction, beginning in the ordinary auricular fibres, will be arrested at this fibrous ring, and would not be conducted to the ordinary ventricular fibres. To obviate this difficulty a specialized band of muscular fibres, however, exists, which extends across the fibrous ring from auricle to ventricle, and is capable of conducting stimuli from auricle to ventricle. This specialized band, which is composed of cardiac muscle fibres of an embryonic type, is known as the "bundle of His." By this band of specialized muscle tissue the stimulus is conducted in an orderly manner from the auricle to the ventricle.

Lastly, the fifth property which the heart muscle possesses is that of tonicity. In other words, the heart muscle fibres, like the muscle fibres of the body generally, are never completely relaxed, so that the heart wall always maintains a certain degree of rigidity.

In a normal heart these five properties of heart muscle fibres are interdependent. In pathological conditions, however, they may be affected independently of one another. In some diseases of the heart they all suffer. In some cases we find a depression of the property of conductivity, and in others an increase of excitability. Certain poisons seem to have a discriminative effect upon the properties of the heart muscle. For example, digitalis depresses conductivity and increases the tonicity of the heart. Other poisons, like alcohol and muscarin, diminish the tonicity of the heart. As it is the orderly exercise of these five properties which determines the normal rhythm of the heart, it is only by the analysis of these five properties of the heart separately and collectively that we

* A paper read before the Sunderland Division of the British Medical Association on November 22nd, 1910.

can really understand to what extent the heart is affected in diseased conditions. I believe that, with a deeper insight into the detailed working of each diseased heart, the time is not far distant when we will be able to point to and treat the erring function—whether of excitability, contractility, conductivity, or tonicity.

From the ordinary examination of the radial artery at the wrist we can obtain information regarding the action of the left ventricle, but by all the ordinary methods of examination no information can be obtained of the movement of the auricles. By investigation of the venous pulse in the neck important facts regarding the action of the auricles can, however, be demonstrated. Dr. James Mackenzie has invented an ink polygraph by which simultaneous records can be taken from the venous pulse in the neck and the radial artery at the wrist. By so doing we are able by measurement to obtain exact information of the movements of the chambers of the heart. By placing the tambour over the jugular bulb, which is situated between the two heads of the sterno-mastoid muscle, a record of the variations of pressure within the jugular vein is obtained. It is known that these variations in pressure are similar to the variations of pressure which take place in the auricles, allowing for the delay in time required for the transmission of the pulse from the auricle to the jugular bulb.

Fig. A shows the similarity between the auricular and jugular curves. There are certain rises and falls of

Fig. A.—Diagrammatic representation of the events of the cardiac cycle and of the carotid and jugular pulses in relation to standard movements. The scale of abscissae is 1 mm. to 1/5 second. S.C. = Semi-lunar valve closure. A.O. = Auriculo-ventricular valves open. (After Lewis, *Further Advances in Physiology*.)

pressure in both, and without detailing the proof I may state that these variations in pressure are due to similar events in the cardiac cycle. We must now compare the time relationships of these rises and falls in auricular and jugular pressure with the time relationships of ventricular events. In the two uppermost curves, one of auricular and the other of ventricular pressure placed one above the other, it will be noted that the first positive wave of the auricular curve occurs before the systole and upstroke of the ventricle, and that the second positive wave in the auricular curve is synchronous with the sudden rise of

ventricular pressure. It will further be noted that during the contraction of the ventricle there is a depression and fall of pressure in the auricular curve broken towards its termination by a third positive wave in the auricular curve. It is generally recognized that the first positive wave in the auricular curve is due to the contraction of the auricle. The second positive wave is due to some factor synchronous with the ventricular contraction. The time of the third rise is variable, but the fall which occurs after its apex is very constant. This fall occurs as soon as the tricuspid valves open. In this way we are able to trace the events occurring in the auricle during a complete cardiac cycle. First the auricle filled with blood contracts, and causes a sudden rise of pressure. It then relaxes, causing a fall in pressure. This fall would continue, but it is interrupted by a rise of pressure which occurs at the moment of ventricular systole, and gives rise to the second positive wave. You see that the fall is continued after the second positive wave. During the systole of the ventricle the tricuspid valves are closed, and the blood gradually collecting in the auricle steadily raises the pressure. This gradual rise of pressure may contribute to the third positive wave. This rise continues until the tricuspid valves open, which event occasions a fall, because the blood then flows easily through the auricle into the ventricle. The blood continues to flow from the auricle into the ventricle until the pressures are the same. Then the auricle contracts, and we begin a new cardiac cycle.

Exactly similar events are shown in the curve from the jugular bulb, but are slightly later. During each cardiac cycle there is a rise (a) which is due to the contraction of the auricle; (b) a fall of pressure due to the relaxation of the auricle, interrupted by a second rise (c) synchronous with the pulse in the carotid artery; a second fall of pressure occurring in the early part of the ventricular systole, followed by a third rise of pressure towards the end of the systole, while the tricuspid valves are still closed. From these diagrammatic pressure curves it will be evident that the events in the jugular vein and carotid artery bear to each other time-relationships identical with those which exist between auricular and ventricular events. The variations in pressure in the jugular vein and carotid artery are merely later in time than the variations in the auricle and ventricle.

In this way a record of the venous pulse in the neck will afford direct evidence of the events taking place in the auricle and the ventricle. In tracing No. J, taken from the venous pulse in the neck by means of the ink polygraph, variations of pressure similar to those which I have described in the auricular and jugular curves will be seen.

Tracing No. J.—Auricular form of venous pulse. a = Auricular contraction. c = "Carotid impulse." Vertical line = Ordinate. Upper curve = R. jugular. Lower curve = R. radial artery.

At the top of the tracing is the record of the time-marker indicating fifths of seconds. Immediately below this is the tracing from the jugular bulb, and at the bottom is a record of the radial artery. The records from the jugular vein and the radial artery have been obtained by the simultaneous working of the two pens of the polygraph. At the beginning and end of the tracing, after the machinery has been stopped, the rubber tubes are squeezed so that the pens make straight lines up and down the paper. These are the ordinates and represent simultaneous events, and are used as standards from which to make measurements. We employ the radial artery as the standard of time. I have marked a point (4) on the radial tracing which indicates the commencement of a radial upstroke. By measuring the distance between (4) and the radial ordinate and transcribing this measurement into the jugular tracing you will see that it falls immediately in front of a sudden upstroke which is due to the pulse of the carotid artery. Now we know that the carotid pulse occurs about one-tenth of a second before the radial pulse. We have learnt, then, where the ventricular systole begins.

in the jugular tracing. We must now see where the ventricular systole ends. We proceed as follows: We know that as soon as the aortic valves shut there is a wave of pressure transmitted to the radial artery which gives rise to the *dicrotic notch*. Therefore the line which I have marked (5) just previous to the dicrotic notch indicates the period of time synchronous with the closure of the aortic valves—that is, the end of systole. The period, then, between the beginning of the radial upstroke (4) and this line (5) indicates the time during which the aortic valves are open—that is, the period occupied by the ventricular systole. We now transcribe this period into the jugular tracing, measuring from the beginning of the upstroke of the carotid (3) so that we can now divide the jugular tracing into two parts. All between the line (3) and the line (5) occurs during ventricular systole. The portion of the tracing between the line (5) and the commencement of the next (3) indicates the events happening during diastole. Let us take the two periods separately. During systole we have here represented a sharp upstroke (*c*) due to the carotid impulse, immediately followed by a second upstroke (*v*) which only attains its maximum at the end of systole, and of whose significance I have already told you. In the diastolic period a sharp upstroke (*a*) is followed by a corresponding downstroke, and these represent auricular contraction and relaxation respectively. It will be observed that in this tracing a wave due to the contraction of the auricle is always a very obvious feature. This variety of venous pulse is known as the auricular form.

We may roughly classify tracings obtained from the jugular pulse into those which possess a single wave due to the contraction of the auricle and those in which it

Tracing No. II.—Ventricular form of venous pulse. The figure 3 marks the time of the carotid impulse ($\frac{1}{10}$ second before the radial upstroke 4).

is absent. The latter is known as the ventricular form of venous pulse.

As will be seen, there is in the jugular tracing a sudden upstroke immediately preceding the point of time of the carotid artery; the pressure is maintained during the whole period of ventricular systole, and suddenly drops at the end of systole when the tricuspid valve opens. There is no sign of auricular contraction. This is a record of ventricular pressure rather than of auricular pressure, and for this reason, that there is merely interposed between the ventricle and the jugular bulb an auricle which is dilated and inefficient. There is evidence—both clinical and *post mortem*—that in cases in which this form of venous pulse has been obtained the auricle is dilated, and that it never gives rise to a single forcible contraction. There are times when the auricle gives rise to numerous incomplete contractions, of which I show you an example (auricular fibrillation).

Tracing No. III.—Auricular fibrillation. The small waves due to auricular fibrillation are marked *f*; *c* marks the carotid impulse.

I believe also that there are conditions when the auricle lies as an inactive reservoir between the great veins and the ventricle. All tracings from the jugular bulb in the neck broadly follow one of these two forms of tracing. That is, there is either evidence of auricular contraction,

the auricular form of venous pulse, or there is no sign of a definite auricular contraction—the ventricular form of venous pulse. With this knowledge of the types of pulse which can be obtained from the jugular bulb we may now proceed to consider some characteristic irregularities. I will show you examples of abnormally slow pulses, abnormally fast pulses, and some other irregularities, all of which I have met with in my own work.

Slow pulses may be due to one of the following causes:

(1) A slow rate, in which both auricles and ventricles participate. (2) The slow rate may be due to the failure of the ventricular contraction to make any impression upon the radial artery, because the ventricle has contracted too soon and too incompletely to transmit a wave to the radial pulse. (3) There are some cases where the slowing is due to the influence of digitalis upon a heart in which the auricle is completely or partially paralysed—cases, that is, of the ventricular form of venous pulse. (4) The slow pulse is due to the fact that impulses from the auricle to the ventricle are blocked.

1. In the group of cases in which the slow rate is common to both auricles and ventricles are included abnormally slow pulses, which are present in normal persons, and occasionally also in conditions where the heart is labouring against a high peripheral resistance.

2. Tracing IV shows how a premature contraction of the ventricle fails to make any appreciable impression on the radial artery. It will be seen that the large wave (*a*) in the jugular pulse is immediately followed by a second large wave (*b*), but that this second wave (*b*) has only the smallest representative in the radial pulse tracing. This tracing is one of the ventricular form of the venous pulse.

Tracing No. IV.—Ventricular form of venous pulse. The rises of pressure (corresponding to heart beats) against which "crosses" are placed have small or no corresponding radial impulses.

The next tracing came from a patient whom I saw about a year ago with Dr. Andrew Smith of Whickham. His pulse was then beating from 18 to 25 to the minute, and was occasionally beating even more slowly. In fact, sometimes the ventricle seemed to cease altogether, and at such times he would pass into an epileptiform attack. He sometimes had forty to fifty fits in the day. He presented the Stokes-Adams syndrome.

Tracing No. V.—Heart block. *a* = Auricular impulse; *c* = carotid impulse. In interposed diagram *As* = auricular impulses, *Vs* = ventricular impulses.

It will be seen that there are only four radial beats, that there are twelve auricular beats, and that the ventricular beats bear no proper time relationship to the auricular beats. Between the jugular pulse tracing and the radial pulse tracing I have placed a diagram which shows in a more graphic manner the relationships between auricles and ventricles. In the upper compartment are black upstrokes which correspond to the beginnings of auricular contractions, and you will see from this diagram that the auricles are beating perfectly regularly at a rate of sixty to the minute. In the lower compartment are upstrokes corresponding to the beginnings of ventricular contractions (*c*). The ventricles, as you will see, never receive normal impulses from the auricles, and have assumed a rhythm of their own, which is at the rate of twenty-three to the minute. Dr. Smith told me that after the patient had been under treatment for some time his pulse was almost normal, and that he was able to work in his garden. At this time I obtained tracing No. VI.

It will be seen that the pulse-rate is very much quicker—about 60 to the minute. When I felt his pulse with my fingers and found it quite regular and beating at such a rapid rate, I concluded that the block between auricles and ventricles was removed. In fact, I surmised that he

would have a normal jugular tracing, by which I mean that the ventricles would be contracting in response to stimuli from the auricles, but it will be seen that the auricles and ventricles are still dissociated. At the beginning of the tracing the contraction of the ventricle precedes that of the auricle; at the seventh beat the

Tracing No. VI.—Heart-block (same case). *a* = Auricular impulse; *c* = carotid impulse.

ventricular beat has caught up the auricular, and later again appears behind it. In a case such as this, examined at a period when the pulse-rate was regular and fast, it would be impossible to know that the patient possessed a very serious heart lesion without such a graphic record.

Fast pulses are met with most frequently (1) as the result of fever, (2) in cases of exophthalmic goitre, and (3) in cases of paroxysmal tachycardia. In Groups (1) and (2) the relationships between auricles and ventricles are perfectly normal, and both beat at the abnormally high rate. In paroxysmal tachycardia the condition of the heart changes, when the pulse-rate passes from the slow to the fast rate. I can indicate what these two changes are by briefly describing the case from which I obtained Tracing VII.

Tracing No. VII.—Paroxysmal tachycardia. In upper tracing the transition from a slow to a fast rate is shown. In lower tracing vice versa.

In this case the attack of quick heart-beat used only to last about ten minutes. During the attacks of tachycardia the heart could be demonstrated to have dilated—that is, the right margin of heart dullness extended 1 in. to the right of the sternum, having previously only reached the mid-sternum. The veins in the neck were engorged and pulsated with every beat of the heart. The patient became cyanosed and the breathing became hurried. The pulse was small and quick. After the attack had lasted for ten or fifteen minutes, the pulse would almost suddenly become slow, the veins of the neck would subside, and the area of heart dullness would diminish.

In the left-hand part of the upper tracing the jugular tracing is of the ventricular venous type and the pulse-rate is rapid and small. This period corresponds to the condition of acute dilatation with turgid pulsating veins. As we follow the tracing from left to right we see that the pulse slows and that the jugular tracing takes the auricular form. In the lower part of the tracing I have caught the transmission from a slow rate to the fast rate of an attack of tachycardia. The patient from whom this tracing was obtained had mitral incompetence as the result of rheumatic fever. He has a brother and a sister, both of whom have had rheumatic fever; they have rheumatic heart lesions, and are subject to similar attacks of tachycardia.

The last group of irregularities to which I wish to draw your attention is very common. With the fingers upon the radial artery it is noticed that every now and then there seems to be a long pause, followed probably by an excessively big impulse in the radial artery. This would be called an intermittent pulse. With the stethoscope on the heart and the finger on the radial artery it is found that two normal sounds of the heart are quickly followed by one or two short sharp sounds, and the first of the short sharp sounds corresponds to a very faint impulse, or no impulse at all, in the radial artery. Fig. B is a graphic representation of this.

In these conditions the excitability of the ventricle is so increased that a contraction takes place before the end of its proper resting period. If this extra systole occurs very early in the diastole of the ventricle the pressure in the

aorta will be so great that this additional weak contraction will be unable to open the aortic valves, and the extra-systole will have no representative in the radial artery. The later in diastole that the extra-systole occurs the

Fig. B.

stronger it will be, and the greater will be the fall in aortic pressure. Under these conditions the extra-systole will have a representative in the radial artery. Tracing VIII is an example of the ventricular extra-systole. In

Tracing No. VIII.—Ventricular extra-systole. Extra-systole at *x*. In the jugular tracing a carotid impulse has preceded an auricular impulse.

the lower tracing of the radial artery there is a small beat in the radial artery following the sixth upstroke of the radial tracing, measuring from the right hand end. If we measure from the right ordinate to the beginning of the upstroke of the small beat and transcribe this measurement into the jugular tracing it will be found to correspond to a carotid impulse. The interposed diagram shows what has occurred. The early appearance of the extra-systole in the lower compartment of the diagram will be noticed. In the upper part of the diagram it is evident that the auricles maintain their normal rhythm throughout. The auricular contraction which follows the extra-systole has no effect upon the ventricle because the ventricle is in the resting stage following the extra-systole. After the extra-systole there is a long pause before the next heart contraction. This is known as the compensatory pause. The interval occupied by the contraction of the extra-systole and the previous full beat is greater than the interval occupied by any two normal beats. Tracing IX shows an extra-systole

Tracing No. IX.—Ventricular extra-systoles. Ventricular extra-systoles occur alternately with normal beats.

which occurred later in the diastolic period. The extra-systole is still, however, of ventricular origin. Following the tracing from left to right, it will be seen that the first beat is represented in the jugular tracing by an auricular contraction followed by a normal ventricular contraction, but at the second beat it will be observed that there is one single very large rise in the jugular tracing. If we measure from (*s*) to the ordinate on the left of the radial tracing and make the same measurement from the ordinate in the jugular tracing it is plain that the ventricle contracted immediately after the beginning of the contraction of the auricle. This almost simultaneous contraction of the auricle and ventricle gives rise to a very large (*a* and *c*) wave in the jugular tracing because the auricle would find closed auriculo-ventricular valves during the greater part of its contraction. The condition of affairs is, perhaps, more easily seen from the interposed diagram. There it will be seen that the normal auriculo-carotid intervals of one-fifth of a second alternate with the almost

simultaneous contraction of auricles and ventricles. This type of pulse is one variety of the so-called bigeminal pulse. Though the abnormal extra-systoles reach the pulse they are considerably smaller than normal, and are followed by a considerable pause.

In the last two tracings the extra-systole originated in the ventricle; in Tracing X a too early contraction of the auricle gave rise to a premature contraction of the ventricle. This tracing came from a case of mitral stenosis.

Tracing No. X.—Auricular extra-systole. The carotid extra-systole x' , which corresponds in the jugular tracing with the mark c , is preceded by a large a wave, due to an auricular extra-systole.

At x' there is a premature and smaller beat in the carotid artery. This might be due to the fact that either the ventricle itself has generated a premature impulse or that the ventricle has the stimulus to contract conducted to it from a premature auricular beat. We find that the distance from the ordinate of the carotid to x' falls exactly at the beginning of the small c . This gives us the time of ventricular systole. Just previous to that there is a very big wave which can only be due to the contraction of the auricle. The jugular pulse shows that the a waves rise with perfect regularity, except at this particular spot, where the contraction of the auricle has occurred too soon. The sequence of events can be more easily understood from the interposed diagram. I have indicated the extra-auricular systole at x . This is followed at a considerable interval by the small contraction of the carotid at c . The interval of time occupied by the passage of the stimulus from the auricle to the ventricle is in the case of the extra-systole considerably longer than normal, owing to the fact that the too early auricular systole finds the conducting fibres of the auriculo-ventricular bundle in a resting or refractory condition, and the stimulus conduction is delayed. Further, the interval of time occupied by the extra-systole and the previous normal contraction is less than the interval of time occupied by two normal contractions, in contradistinction to the long compensatory pause which I pointed out in the case of a ventricular extra-systole.

Whenever an extra-systole occurs, it means that the muscle is in an unduly excitable condition. It means, further, as in the examples I have shown you, that the auricle or ventricle contracts prematurely in response to impulses other than the normal. It has been suggested that these abnormal impulses have their origin in the bundle of His, and therefore that they may arise in the auricle, in the node of tissue between the auricle and the ventricle, and lastly in the ventricle itself. One frequently meets with extra-systoles in elderly people, and it has been suggested that in these cases the heart muscle is made more excitable from lack of proper blood supply, due to thickening of the coronary arteries. In old people extra-systoles, apart from signs of heart failure, have no particular significance. They have no sinister meaning unless they are discovered at a time when some particular call is being made upon the heart. If a patient with extra-systoles is attacked with a disease which causes fever and quickening of the heart-rate it is a favourable sign if, with the quickening of the heart, the extra-systoles disappear. I presume that in such a case the quickened heart-beat exhausts the irritability of the cardiac muscle, or at any rate that part of the muscle which gives rise to the extra-systole. The extra-systole occurs sometimes in dyspeptic and neurotic people, and is simply an evidence of the general lack of nervous control. Further, one meets with extra-systoles in young people in whom there is no rheumatic history and no cardiac sclerosis. I would sum up the significance of extra-systoles by saying that their presence has no serious import except when they occur in such diseases as pneumonia and rheumatic fever. In

these conditions the heart muscle is involved in the disease process. In other cases the most serious result lies in the knowledge of the patient that he possesses an irregular pulse. He keeps feeling his pulse, and the irregularity fills him with unnecessary alarm.

I have endeavoured to show how by a graphic method we are able to unravel pathological conditions of the heart and to obtain a detailed knowledge of its mechanism which we previously did not possess. In fact, our ideas of heart disease are being revolutionized. Up to now too much attention has been paid to the valves and too little to the nervous and muscular components of the heart. As I said earlier, I believe that this recent knowledge of the heart's action will place our treatment of heart disease on a more exact basis. We must beware lest we are carried too far into the intricacies of a complicated mechanism like that of the heart, and place too much importance upon it; for, after all, the main question in any heart lesion amounts to this, What is the heart capable of doing? It is, however, always better to possess exact information rather than to work in the dark, and I have no doubt that with our more exact knowledge a more rational therapeutics will arise.

I take this opportunity of expressing my great indebtedness to Dr. Mackenzie for his help and encouragement, and to Dr. William Ritchie and Dr. John Cowan for their assistance in early difficulties. I thank also my colleagues at the Royal Victoria Infirmary for so readily placing their material at my disposal.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SALVARSAN.

I HAVE recently had the opportunity of using this preparation upon a badly syphilitized pregnant woman, and the notes of the case may be of interest.

A woman aged 26 was admitted to the union infirmary on February 27th, 1911, seven and a half months pregnant. The mouth and throat were extensively ulcerated, the voice husky, there were gummata on the chin and cheeks, a typical rash all over the body, numerous condylomata on the labiae and around the anus, and a foul vaginal discharge. There was severe dyspepsia and continual vomiting. She was placed on liquid diet, put to bed, and treated with mercury perchloride internally and black wash to the condylomata. She improved generally, vomiting ceased, and all the local conditions were modified. On March 21st an intramuscular injection of salvarsan was given. Beyond a slight soreness in the injection area no discomfort was felt. On March 23rd the patient said she felt much better. On March 24th the vaginal discharge had quite ceased, and the ulcers in the mouth and pharynx were looking much healthier, as also were the condylomata. On March 30th the ulcers were quite healed, the condylomata nearly gone, and all soreness had ceased in the vagina and vulvae, the patient stating that she felt quite well. On April 7th she was delivered of a male child of fully average weight and in good condition. The skin on its face, hands, and soles of feet had a puckered, dry appearance; otherwise there was none of the usual conditions found in the newly-born syphilitized child. From its birth there was desquamation, especially on the hands and feet, until the fifth day. On the seventh day the child's appearance was that of a perfectly normal and healthy infant, and there had been no sign of any tendency to ophthalmia. The placenta was dark, soft, and "rotten looking."

Although the mother showed the excellent effect of salvarsan, the interest of the case centres in the condition of the child, which but for the new treatment would no doubt have been born with the usual syphilitic lesions.

On the same day a dose of salvarsan was given to another badly syphilitized woman. She had been under mercurial treatment at intervals for several years, and had got steadily worse. On admission she was very emaciated. The soft palate was in a foul sloughing condition. She could barely swallow liquids and the voice was practically gone. Three days after the injection she could swallow. On the fourth day she could speak. Ten days after the pharynx and soft palate were healed, and on the twenty-fourth after the injection she appeared and felt quite well and had quite lost all appearance of emaciation.

Personally, I am disposed to think that the intramuscular method is preferable to the intravenous, at any rate in general practice.

Bishop Auckland.

MARK WARDLE.