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ON THE CAUSES OF THE SECONDARY WAVES SEEN IN THE SPHYGMOGRAPHIC TRACING OF THE PULSE.

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It is a fortunate circumstance for the application of the Sphygmograph, as a means of clinical research, that a knowledge of the cause of the several secondary waves of the pulse is not necessary for their practical interpretation. This may be learnt empirically by watching the association of the different forms of pulse-curve with the known conditions of the patients from whom they are obtained. Thus we find that authors who differ totally from each other as to the causation of any particular form of pulse, and even as to the state of circulation which it implies, yet are quite in agreement as to its clinical import, and the prognosis to be derived from it. But it requires much experience of tracings to be able to draw these inferences, and those who have not this are apt to interpret what they see from theory, and thereby easily fall into error. For instance, it has often been supposed that a high sharp primary summit, followed by a sudden fall, is a sign of aortic regurgitation, but this may occur just as much in the pulse of simple excitement. Hence, from the practical point of view, the study of causes is useful, and it is essential for the arriving at general physiological conclusions, such as the determining what is the true state of the circulation in fever, or in Bright's disease, or what is the effect on the vascular system of various drugs.

It needs but little study of the literature of the subject to discover that scarcely any two authorities agree together

as to the cause of the several waves, and of their variation. The question is one which must be settled by having regard both to the principles of mechanics, and to the results of observation. And it is impossible to avoid suspecting that some of those who have treated the subject experimentally, would have been assisted in the interpretation of their results, if they had possessed some theoretical knowledge of Hydrodynamics.

I have adopted an experimental mode of investigation by the use of a combination of bifurcating elastic tubes to represent the arterial system. I have not attempted to imitate the individual arteries of the body, for although such an apparatus looks well in a lecture-room, it does not any the more resemble the actual complexity of the human circulation. To these tubes I have adapted, in some cases, the heart of a sheep, in others, an artificial heart of india-rubber, and the contraction of the heart has been imitated by manual compression. It will be seen that counterparts have thus been obtained of the most important types of pulse found in the human body. Tracings 1 to 4 were procured with the real heart, those from 5 to 9 with the artificial. In the case of the real heart, I have found it more convenient to use the right side, for the thick walls of the left ventricle form an obstacle to manual compression. The aortic, or pulmonary valves act efficiently after death, but it is not so with the mitral or tricuspid. It follows from this, that the real heart will only work against a low pressure, and the highest which can be obtained with it (fig. 3) corresponds to nearly the lowest ever found in the living body, while the tracings at lower pressures, such as fig. 1, represent a state of things which never occurs in arteries. Tracings from the real heart, at the higher pressure, resemble closely those obtained at a similar pressure from the artifical heart, thus showing that the action of the latter is sufficiently like that of the real heart for the purpose of these experiments (compare tracings 2 and 5). It has been considered by Dr Burdon Sanderson impossible to make the contraction of the hand sudden enough to imitate that of the living heart, but I have not found this difficulty at all insuperable.

I had, in the first place, made use of a single elastic tube, corresponding to the schema constructed by Dr Burdon Sanderson. In this case I found the tracings different at different parts of the tube, and the variation was such as to show the presence of retrograde waves due to reflection from the end of the tube. This reflection took place as much from an open as from a constricted orifice. In my final combination, I made the several tubes of very unequal lengths, and in using compression in order to vary the tension, I applied it to each of the smallest tubes, not at one, but at several points, so that no reflected waves might be called forth of a kind which could not occur in the body. Applying then the same test of taking tracings at different distances, their resemblance to each other showed, as it does in the case of the arteries of the body, that all the waves were direct and not retrograde. The effect of friction in the capillary circulation was thus imitated by that of compression at several points of each small tube, and if it may be said that reflection might still possibly take place in the schema from the latter, so it has been held by many that it occurs in the body from the former. I shall refer to the several tracings obtained under different conditions as the points arise which they illustrate.

It was shown so long ago as in 1833 by Weber, that the motion of the pulse is that of a wave. By this term is meant the transmission with a definite velocity, not of matter, but of a state of motion and of pressure. It follows from the theory of waves, that on the large wave other waves may be superposed, which run each their own course almost exactly as if they existed alone, and which may be added together to form compound waves.

Among those who have not given special attention to the Sphygmograph, almost exclusive regard has been paid to one of the secondary waves of the pulse, namely, the dicrotic wave, and no doubt this is, to a great extent, justified by its importance in the theory of the pulse, its constant occurrence, and its great development in fever. But there is another wave preceding it, which sphygmographers find to be of quite as great clinical significance, and which varies as widely, for, while often absent, it may attain considerable dimensions. Being but

slightly marked in a healthy pulse (fig. 10), and often disappearing in that of a student (fig. 19), it forms a stronger feature in the pulse of an athlete, and shews itself also in that of ladies under the influence of excitement. Under another form again, with advancing years, it acquires a magnitude which is of ominous significance, but gains perhaps its greatest development in cases of chronic Bright's disease (fig. 12). I believe this wave would be better known if it had a convenient name. "First secondary wave" is too long, so is "systolic pressure-wave," and, moreover, that asserts a theory. The only short name applied to it is that of "tidal-wave," used by Mr Mahomed in his papers in the *Medical Times*. Although this also is intended to imply a theory, which I believe to be erroneous, yet, as it does not assert it so directly, I shall adopt it for its convenience. I propose, therefore, to discuss first the tidal, and afterwards the dicrotic wave, and in connection with the former it is necessary to consider the primary upstroke.

I will notice first existing theories. Dr Burdon Sanderson, writing in 1866, says that the contraction of the heart produces two waves, one of accelerated movement, and one of increased tension; that these, starting together, become separated in the distant arteries, because the former travels with a velocity of about 90 feet per second, and so is practically instantaneous. He compares the former to the communication of a blow along a line of ivory balls, but afterwards, accepting a correction on the ground that water is not elastic, admits that, but for the effect of the elastic receptacle, the resemblance would be rather to balls of clay. Now in the popular sense of the word elastic, that of compressible, water is almost inelastic, but in the more exact sense, in which elasticity is measured by the perfection of recoil to any force impressed, water is very elastic, as is shown by the fact that sound is conveyed much better in water than even in air. So far it is more like ivory than clay, but in truth the behaviour of neither has the slightest analogy to that of a fluid. It is the first principle of the mechanics of fluids, that at every point of a fluid, whether at rest or in motion, pressure is the same in all directions. It follows from this that there can be no wave of forward pressure (and without that there can be no accelera-

tion), which is not at the same time a wave of lateral pressure, expanding the walls of the tube.

The view of Dr Burdon Sanderson is therefore mechanically impossible: it is also contrary to observation. *In the first place*, the distinction of the two waves should, according to him, be most when tension is lowest. But, on the contrary, the tidal wave is most separated in pulses of high pressure, like that of Bright's disease, altogether absent in the febrile pulse of low tension, and precisely the same relation was found in my experiments with tubes. *Secondly*, the tidal wave should be farther apart from the primary wave in the more distant arteries; but a comparison of tracings 15, 17, and 18 from the brachial, radial, and dorsalis pedis, shows that this is not the case. And *thirdly*, it is not the fact that the wave producing the first upstroke is practically instantaneous. In the case of elastic tubing the interval is very perceptible, if a length of 6 or 8 feet be taken. The velocity varies altogether according to the diameter of the tube, its material, and tension, but in one case I found it to be less than 20 feet per second. The same thing has been shown as regards the body, in this *Journal*, by Mr Garrod, who by his Cardio-Sphygmograph measured the interval between the heart's contraction and the first upstroke of the pulse tracing. Weber, again, reckons the average velocity of the pulse wave to be about 28 feet per second.

Dr Anstie, writing in conjunction with Dr Burdon Sanderson, yet differs somewhat, at least in expression, for he speaks of the tidal wave as an expansive wave, which is a movement in the arterial wall, and so slower in propagation. This would seem to be the same as the theory adopted by Volkman to account, not for the tidal, but for the dicrotic wave, that one wave is transmitted in the fluid, another in the arterial wall.

Dr Balthazar Foster, as represented by the article in the last edition of Dr Aitken's *Medicine*, holds that the primary wave is a vibration of the blood column, travelling instantaneously, and that the next is a wave of distension by blood. It is obvious that a wave of vibration is quite a different thing from a wave of forward motion, yet Dr Burdon Sanderson would seem to have combined this view with his own, for he says in 1871, "The bursting open of the aortic valves produces a vibra-

tory movement of the blood transmitted instantaneously (that is, in about $\frac{1}{50}$ second)." Now one vibratory wave would indeed be transmitted, namely, that of sound, with a velocity due to the compressibility of water, not of 90, but of about 5000 feet per second, but this produces no motion in the lever. Tracing 22 was obtained by giving blows to a rigid part of the tubing. It shows that even a coarse and violent vibration produces hardly any upstroke. Dr Burdon Sanderson has published some tracings of waves produced by percussion, but in that case the blows were applied to the elastic tube, which would of course give rise, not only to vibration, but to waves of forward motion and expansion. Most of the other objections to the views of Dr Burdon Sanderson will apply also to those of Dr Foster.

Yet another theory of the tidal wave is maintained by Mr Garrod. Discarding the notion of an instantaneous wave due to the closure of the mitral valve, or the first impulse of the heart, he yet holds the tidal wave to be an instantaneous wave due to the closure of the aortic valves. I think this theory will commend itself to no one who has watched the variation of the tidal wave in many tracings; but, if any refutation be necessary, I would refer to tracing 8, showing its prolongation by a protracted contraction of the heart.

The view of Mr Mahomed, as far as I understand it, is the same as that of Dr Foster, except that he does not consider the first wave to be instantaneous.

Proceeding next to my own explanation, I have to remark first, that, since the sphygmograph is possessed of inertia, and is itself subject to the laws of motion, its construction must necessarily have some influence, however small, on the tracing produced. In sphygmographs having a secondary spring to depress the long lever, the tidal wave is often broken into two waves (fig. 21), and if this spring has a short time of vibration, a jagged line may appear. In the instrument I have used, of the form devised by Mr Mahomed, this spring is, with advantage, omitted, and no such waves are then ever seen. Thus from many tracings published, an experienced person may draw inferences, not only about the patient, but about the form of sphygmograph used. In order, therefore, to determine how

much is due to the instrument, since inertia cannot be got rid of, I have adopted, what is called by Mill, the "Method of Concomitant Variations," altering the moment of inertia of the lever about its axis of motion by a small sliding weight. The results are tracings 15 and 16. It will be seen that the relation of the primary and tidal waves is altogether altered, while the position of the dicrotic wave remains unchanged, although its amplitude is diminished. The method of measuring the position of these waves is first to draw a horizontal line of reference, then to place the tracing again on the moving plate of the sphygmograph, and draw curved lines with the writing point of the lever. It is obvious that if the tidal wave were a wave passing in the artery, its relative position could not be altered by weighting the lever of the sphygmograph; or, at any rate, if any effect at all were so produced, it could only be an apparent retardation, and not an acceleration of the wave. The separation of the primary and tidal waves is therefore due to an oscillation in the sphygmograph, caused by the inertia of the instrument, and the relation of the tracing to the true pulse wave is something like what I have drawn in fig. 23. In some cases the lever may be separated slightly from the knife-edge on which it rests, but generally the oscillation takes place in the instrument as a whole, and it may be followed by others in a descending series. Thus if the lever be moved, not by a knife edge, but by a rack and pinion adjustment, the tidal wave still occurs. Such an arrangement probably makes the tracing resemble the true pulse wave a little more closely, but for clinical purposes it is not so good.

I may mention that in the pulse shewn in fig. 15, in which the tidal and dicrotic waves are both so marked, one secondary beat could be felt by the finger, but only one. Such pulses do indeed give at first to the finger the impression of several secondary waves on account of their thrilling quality. But the sphygmograph gives no record of a thrill, as is shown by its application to the heart in the case of mitral stenosis. If the stenosis be moderate, the prolonged auricular contraction is shown in the tracing; but if it be so close that a thrill is continued throughout the whole period of rest, no record of it any longer appears.

My general conclusion is confirmed by the application of the little instrument which has been called a sphygmoscope, whereby the motion of the pulse is displayed by the variation of a jet of gas. By this means the dicrotic wave may readily be seen, but not a single tidal wave. Its place, however, is supplied by a slight quivering motion, which is due to the vibration of the elastic diaphragm, upon which the pressure of the pulse is received. This vibration varies with the size and tension of the diaphragm, and it might be possible so to adjust these that a wave should appear like the tidal wave of the sphygmographic tracing.

Another argument may be drawn from the fact that the development of the tidal wave varies in some degree according to the pressure which is applied to the artery. Thus in the pulses in which, at ordinary pressures, no tidal wave can be seen, it may sometimes be made to appear by using an excessively low pressure.

The explanation which I have applied to the whole tidal wave is adopted by Dr Burdon Sanderson, to account for the first of the two waves into which it is broken by the use of the secondary spring. But this must be entirely due to that spring, since it never occurs in its absence.

The view of M. Marey is, up to a certain point, the same which I have taken, for he says that the first pointed summit is due to the acquired velocity of the long lever. But he regards the tidal wave in some of its forms as an instantaneous wave due to the closure of the aortic valves: the dicrotic wave he believes to have no connection with those valves. We have seen that Mr Garrod has adopted the same theory as far as concerns the tidal wave.

Of all the diagrams in the work of M. Marey, one of the most interesting is that in which three simultaneous tracings are shown, of which the first represents the pressure within the right auricle, the second the pressure within the right ventricle, while the third is the tracing of the apex beat, obtained by means of an ampulla inserted within the thoracic wall. In all of these the primary summit is followed by two or more secondary eminences, much resembling the small waves which may be seen in the place of the tidal wave in the pulse tracing

as drawn by M. Marey's sphygmograph (Vid. fig. 21). I believe that their origin is similar, and that they illustrate the mode in which eminences may be produced in consequence of the inertia of the instrument. By M. Marey himself, however, they are attributed, in the case of the first two tracings, to an oscillation in the tension of the auriculo-ventricular valves, occurring after their closure, and causing a corresponding rise and fall of pressure within the two cavities. Now it is evident that if the oscillation of the valves causes a rise of pressure in the ventricle, it must, at the same moment, cause a fall of pressure in the auricle, and conversely. Hence the elevations in the tracing of the one cavity, if due to this cause, ought to be synchronous with the depressions in the tracing of the other. But on referring to the diagram of M. Marey, it will be found that the elevations in the auricular curve correspond, not to depressions, but to elevations in the ventricular curve, and therefore the explanation given by him would seem to be inapplicable.

While I thus believe that waves occur in the tracing, which have no separate existence in the pulse, I am yet of opinion that the instrument is more clinically useful than if it followed the artery more closely, for I think that slight differences in the form of the pulse wave, and in the suddenness of its commencement, are thus translated into a form much more manifest to the eye. The constancy of the form of the pulse tracing in the same person under similar circumstances, proves that it contains no casual oscillations, but that its form has, at least, a fixed and definite relation to that of the true pulse wave. My experiments with elastic tubes showed that the tidal wave could not be produced unless the pressure exceeded a certain point, and also the length of elastic tube were limited, or rigid tubing substituted for a part of it (fig. 7), and it could then be prolonged to a great extent by increasing the length of the heart's contraction (fig. 8). Its development thus indicates three things, high tension, diminished elasticity, and long laborious action of the heart. This conclusion agrees entirely with clinical experience of the kinds of pulse in which it is most manifested.

Passing on next to the dicrotic wave, it may be thought almost superfluous to consider its cause, since the common view,

ascribing it to a recoil produced by the closure of the aortic valves, appears so probable and intelligible. Yet the case is not so simple as it seems. Thus we find that Dr Burdon Sanderson, who, in 1866, appeared to adopt the common view, says in 1871, "The dicrotic wave has nothing whatever to do either with the closure of the aortic valves, or the cessation of the heart's contraction." His present theory is a little difficult of comprehension. He says, "In the largest arteries the expansion is ebbing, while in the smallest it is still culminating: so that for an instant the pressure is greater in the latter than in the former. The restoration of the equilibrium must take place by increase of pressure towards the heart, and diminution towards the periphery. This restoration of equilibrium constitutes the second beat." In another place he says that owing to the cessation of the heart's contraction the capillary arteries become relaxed, the capillary circulation retarded, and the aorta simultaneously distended in consequence of the increased resistance in front: and that this distension is in its turn propagated towards the periphery. These two accounts seem to me different, nor can I clearly gather whether or not Dr Burdon Sanderson considers the dicrotic wave to be retrograde. But such a transmission of influence from the periphery to the centre could only take place as a retrograde wave. To determine therefore whether this occurs I have compared two tracings from the same dicrotic pulse, one from the femoral artery just below Poupart's ligament, the other from the posterior tibial below the ankle. These are 19 and 20. The corresponding position of the dicrotic wave in the two shows that it is not retrograde but direct, and there is no retrograde wave at all present, for such a wave would be close to the primary wave in the posterior tibial, and farther from it in the femoral. The same tracings refute the theory that the dicrotic wave is due to reflection at the bifurcation of the aorta, for then it would be absent below that point. While I have thus opposed most of the views of Dr Burdon Sanderson, I should be the last to undervalue the service which he has done for the practical application of the sphygmograph, for I agree with his clinical inferences as completely as I differ from his mechanical explanations.

Perhaps the oldest theory of the dicrotic wave is that of

Dr Barlow, who observed it before the invention of the sphygmograph, and held it to be a reflection from the periphery. He believed dicrotism to indicate an obstructed capillary circulation, and therefore high tension, and to imply a stage of irritation and contraction, which, in disease, preceded the stage of fever and relaxation. The sphygmograph, however, has shown that great dicrotism belongs especially to the state of fever itself, and is found neither in the preceding stage, nor in that of exhaustion which follows. M. Marey and Dr Carpenter likewise consider the dicrotic wave to be a reflection from the periphery. Now this reflection is exactly what occurs in a single elastic tube, but not in the body, as already shown by the comparison of tracings 19 and 20. Duchek believes the dicrotic wave to be an oscillation, not in the aorta, but in the peripheral arteries. Vivenot regards it as an oscillation, but does not explain how it arises. The view of Volkman has been already mentioned.

Now, looking at tracing 1, from the elastic tube, and 14 from the radial pulse, it would seem that a dicrotic wave, equal in magnitude to the primary, could hardly be due to the simple closing of a valve. Experiments with tubes show that the dicrotic wave is only the first of a series, and when pressure is very low the aortic valves, being in glass tubes, can be seen to open and close a second time, after their first closure. In that case therefore the closing of the valves is not so much the cause as the effect of the secondary waves. I have tried the effect, in the case of the real heart, of dividing one of the semilunar valves, in that of the artificial heart of removing them altogether. The results are shown in fig. 4 and fig. 9. In both the dicrotic wave is less than before the alteration, but still very considerable. This fact of the occurrence of the dicrotic wave without any aortic valves has also been noted by Duchek, by Vivenot, and by Dr Burdon Sanderson. It agrees entirely with experience of tracings in the case of aortic regurgitation, for although the dicrotic wave is diminished when regurgitation is free, it is yet never absent, and even in a splashing pulse often retains considerable size. The use of the sphygmograph is thus disappointing for the diagnosis of aortic disease, although, the fact of regurgitation once known, it is of service in determining its extent.

My own view is, that, as the tidal wave is due to the inertia of the sphygmograph, so a wave occurs which is due to the inertia of the arterial walls. If it were not for this inertia, their distension would always be such as to be in equilibrium with the pressure of the fluid within at every moment. As it is, it takes a little time to reach this point of equilibrium, then by acquired velocity is carried a little beyond it, and so again passes within it as it recoils, and thus makes a series of oscillations about the equilibrium point. Thus there occur oscillations of expansion and contraction of the largest arteries, due to the effect of the inertia of the arterial walls on their lateral motion, but modified also by the inertia of the fluid. The first of these, the only one which commonly occurs, forms a part of the dicrotic wave. Be it especially observed that I attribute the oscillation to the inertia of the arterial wall and not to its elasticity, although that elasticity is of course necessary for this, as for every other part of the motion of the pulse wave, and its degree affects the period and extent of the oscillation. Nor, again, would any oscillation occur from the inertia of the fluid alone, so far as that affects its forward or backward motion; but such an oscillation being once set up, it is more ample the greater the momentum of the fluid, because the motion of the tube and of the contained fluid can only take place as a whole. It is thus that dicrotism is increased, as shown by M. Marey, if a denser fluid, as mercury, be taken instead of water. There is however another way also in which the inertia of the fluid may come into play, and that is by its effect on that slight lateral motion of the particles which must take place in consequence of the expansion of the tube. The effect of the acquired lateral velocity of such particles would be to expand the tube a little beyond the point which it would otherwise have reached, and by that means set up an oscillation. The effect of such acquired lateral velocity is generally disregarded in mathematical investigations of similar waves, as being too minute to have any appreciable effect. It would be difficult to ascertain whether in this case the part it plays in the general result ought to be taken into account. The aortic valves would produce a wave of their own without any oscillatory wave, and they also reinforce that wave by reflection. The second oscil-

latory wave I have found in but few pulses, of which one is shown in tracing 14. That pulse was in the highest degree compressible, and was taken only a few hours before death. The condition was just the opposite of that of the common tricrotic pulse (vid. figs. 11 and 15), in which the second wave is the tidal wave.

I think an argument for my view may be drawn from the possibility of the occurrence of a monocrotic pulse. According to the common theory of the dicrotic wave, this would imply that the aortic valves never close at all, in which case we can hardly suppose that the circulation could continue; but upon the other view, it only means that the rate of the pulse is equal to the rate of oscillation in the aorta. The dicrotic wave seen in a tracing may thus be made up of three waves superposed, the recoil from the aortic valves, the first oscillatory wave of the large arteries, and the second (or sometimes the first) oscillatory wave of the sphygmograph.

An opinion has been expressed by one of the ablest mathematicians of the day, especially in relation to physical problems, I mean Professor Maxwell, that no mathematical solution could be usefully applied to the theory of the pulse, and that for two reasons,—because the blood is not truly fluid, and because the motions of the arteries would be so much affected by their external attachments. These difficulties however would not apply to experimental elastic tubes, and since the principal forms of pulse can be imitated in them, I am led to the conclusion that blood is really fluid while contained in the arteries, and that their external connections are too loose greatly to modify their motion.

The introduction of the inertia of the arterial wall makes the question very complex for mathematical treatment. Disregarding that, a differential equation may be obtained of a form similar to that occurring in other kinds of wave motion. Its solution gives a velocity for the wave, which involves the material and diameter of the tube, and the pressure of the fluid, being greater when pressure is greater. This last result agrees with observation as to the retardation of the pulse, and is a much likelier explanation of that phenomenon than to suppose that what is felt by the finger is in some cases not the primary

upstroke, but the tidal wave, which is only a convexity in the descending curve. I think it will be found that the retardation is most in dicrotic pulses, where tension is low, and therefore the wave velocity less, but in which the tidal wave is entirely absent. Calculation gives no indication of the existence of any other wave travelling with a different velocity, except the wave of sound, whose velocity is due to the compressibility of water, and is nearly 5000 feet per second.

As a rule my experiments showed the dicrotic wave to be increased by diminution of pressure. This is the general view, and has been denied only by Mr Mahomed, on experimental grounds. But his schema differed from the arterial system by the introduction of a spherical elastic bag, which could hardly fail to introduce a set of oscillations of its own. It need hardly be said that it would be mechanically most unlikely, as well as for the sake of inference most unfortunate, that the same dicrotism should at different times employ opposite conditions. There is however one limitation to be made. If tension be increased by compressing a tube at a single point, dicrotism is often not diminished but increased, because the oscillatory wave is then kept in and reflected. This explains why dicrotism is increased by placing a tourniquet upon the abdominal aorta.

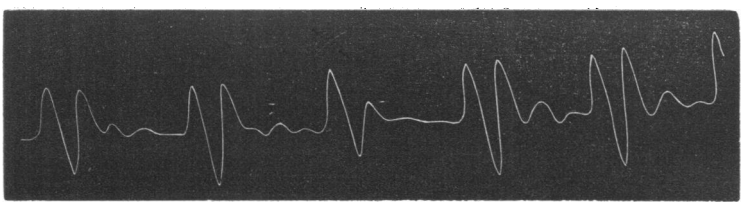
Theoretically both components of the dicrotic wave should be increased as pressure is lowered. The recoil from the aortic valves is not indeed greater, and could never produce such a dicrotic wave as that in fig. 1 and fig. 14, but it becomes more marked because preceded by a greater reflux, and consequent fall of pressure, when the valves close slowly. Oscillatory waves again are always more ample when tension is low.

In my experiments I have found that besides the variation of pressure, one other condition increases dicrotism, provided pressure be also low, namely to make the action of the heart short and sudden. In that case, however, the tracing has a rather different aspect, for sharper points are seen in the curve. This agrees with observations on the human pulse, for in that the rounding off of points is well known to be of bad prognosis. If the action of the heart be jerky, but at the same time pressure not low, the result is the common tricrotic pulse, in which the second wave is the tidal wave, and the first sum-

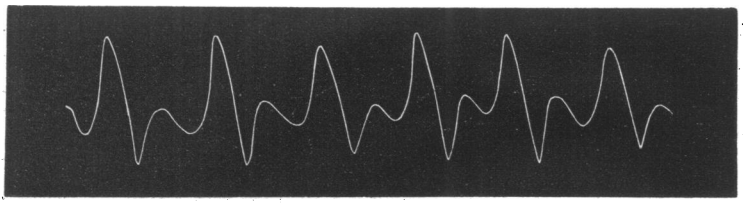
mit high and sharp. This state of things occurs in the body in the case of muscular exertion, mental excitement, from the smoking of tobacco, and, with a greater proportionate development of the tidal wave, in acute nephritis.

As an application of the foregoing principles, certain conclusions may be drawn as to the state of the circulation in fever. The degree of dicrotism together with the rounding off of sharp points indicates that arterial pressure is very low, and at the same time the action of the heart short and sudden, but that the former of these conditions preponderates. This would be explained by supposing a paralysis to occur of the nerves which cause contraction of the arterial walls. The rapidity of the heart would then be in part the direct consequence of low arterial pressure, according to the relation demonstrated by M. Marey, but its short and sudden action appears to indicate a disturbance of its own innervation in addition. The increased rate of circulation would however depend less on the action of the heart, than upon the arterial relaxation, which is therefore the most important element in the state of the vascular system existing in fever.

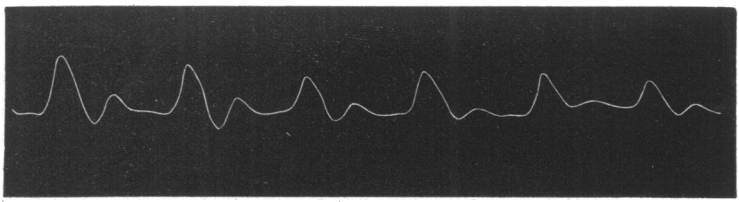
Experimental tracings from schema of elastic tubing, combined with sheep's heart.



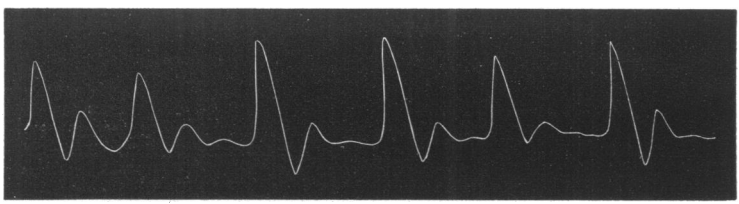
1. Lowest pressure. The dicrotic wave is about as high as the primary, and is followed by other oscillatory waves.



2. Low pressure. The dicrotic wave is still large, and the tracing resembles the hyperdicrotic pulse of fever.

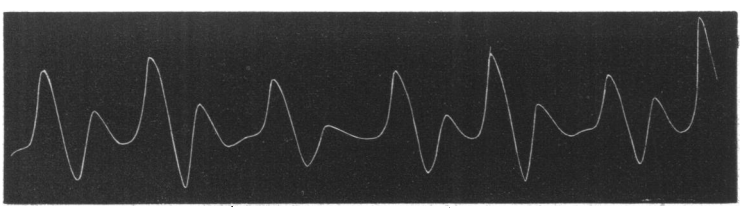


3. Higher pressure. This tracing is less dicrotic, and resembles the pulse of slight fever, or of feeble health.

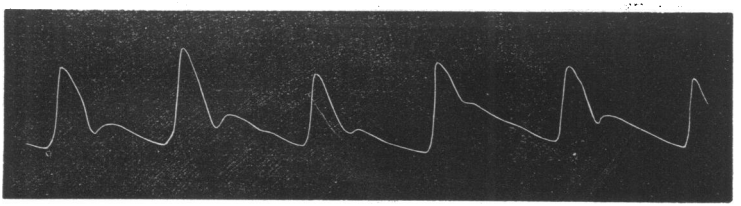


4. One semi-lunar valve divided. The dicrotic wave is less than before the alteration (compare figs. 1 and 2), but still considerable. It is followed by a second oscillatory wave.

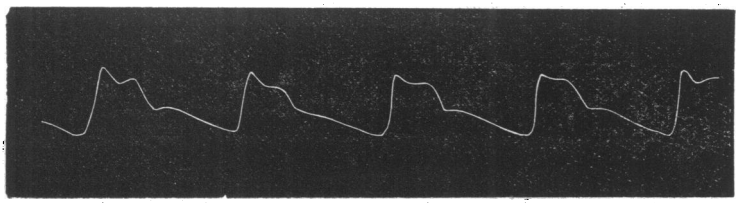
Tracings from the same schema, combined with artificial heart.



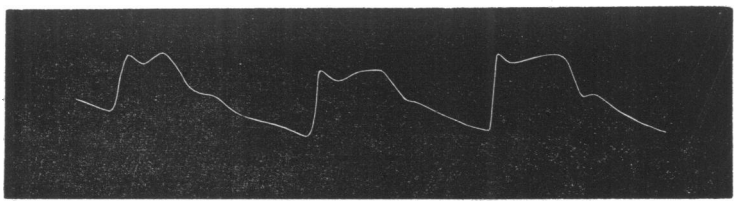
5. Low pressure. This tracing resembles 2, obtained with the sheep's heart, and 13 the pulse of fever, but the rate being slower, a trace is seen of the second oscillatory wave.



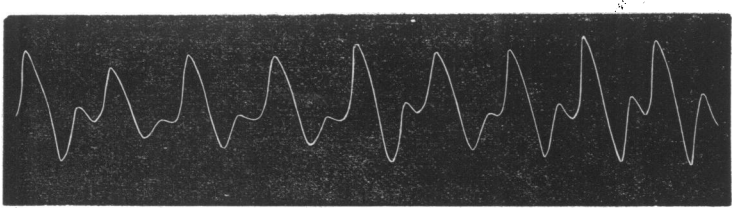
6. High pressure. This tracing resembles one of the forms of healthy pulse.



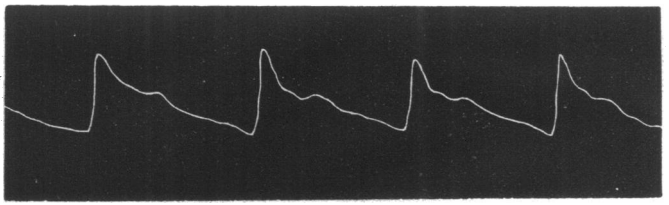
7. High pressure: rigid tubing. The tidal wave here for the first time makes its appearance, and the dicrotic wave becomes less in proportion. This tracing resembles the pulse of atheroma.



8. High pressure: rigid tubing: long contraction. The tidal wave is here much prolonged.

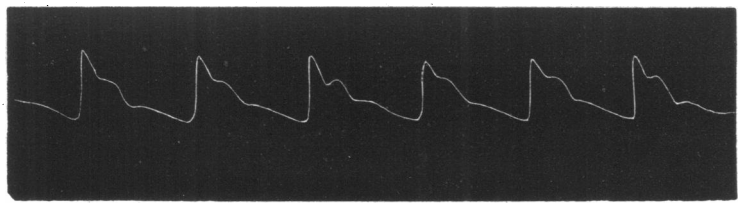


9. Aortic valves entirely removed: pressure low. The dicrotic wave is still considerable, the tidal wave absent.



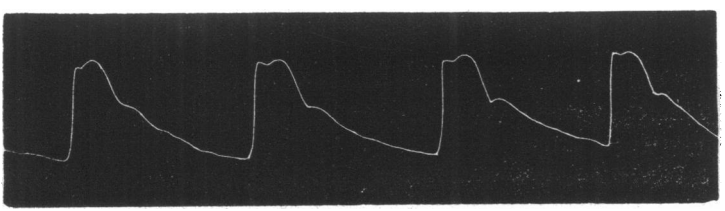
Pressure $2\frac{1}{2}$ ounces.

10. Healthy pulse. The tidal wave is seen preceding the dicrotic wave, but only slightly marked.



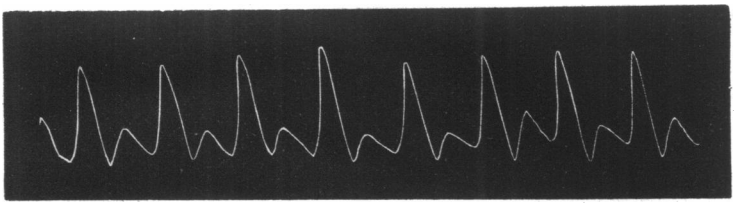
Pressure $3\frac{1}{2}$ ounces.

11. Atheroma without kidney disease. The pressure is somewhat greater than normal, the tidal wave large, and the dicrotic wave small in proportion.



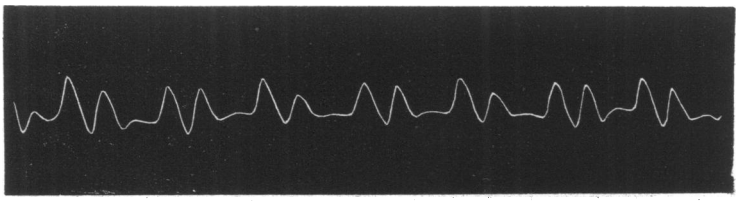
Pressure 5 ounces.

12. Atheroma with granular kidney. The pressure and height of upstroke are both increased, and the tidal wave is very large.



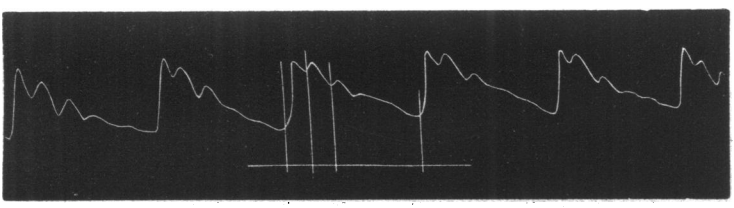
Pressure 1 ounce.

13. Dicrotic pulse of fever. Tidal wave absent; dicrotic wave large: pressure low.



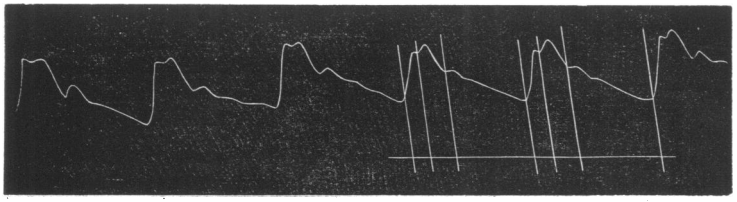
Pressure ½ ounce.

14. The pulse of a case of Bronchitis with chronic Alcoholism taken a few hours before death. The pressure is excessively low: the tidal wave absent: the dicrotic wave almost equal to the primary, and followed by a second oscillatory wave, which is hardly ever seen in the human pulse.



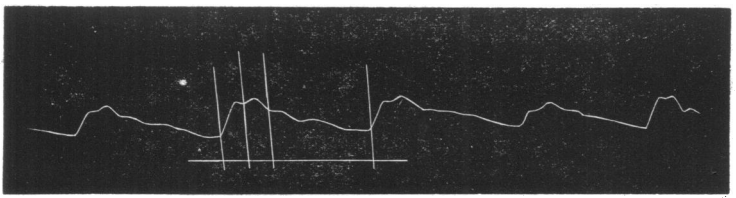
Pressure 5 ounces.

15. G. L. aged 18. Granular kidney. Tracing of radial pulse. The pressure is very high, the tidal wave large and distinctly separated: after it is seen the dicrotic wave, and after the dicrotic wave a third secondary wave, which is the third oscillatory wave of the sphygmograph, the second such wave being superposed on the dicrotic wave.



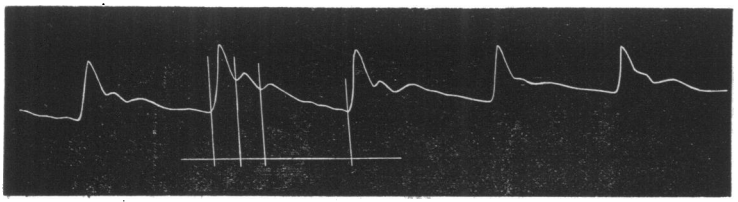
Pressure 5 ounces.

16. G. L. Radial pulse. Lever of sphygmograph weighted. The tidal wave is thus brought nearer to the primary wave, while the dicrotic wave retains about the same relative position.



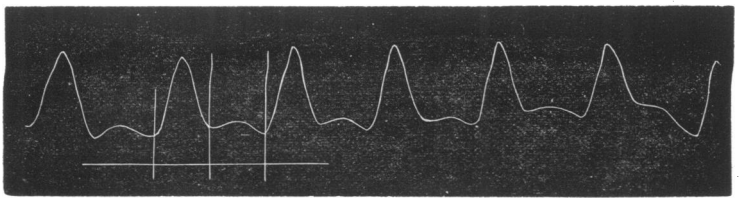
Pressure 4 ounces.

17. G. L. Tracing from brachial artery.



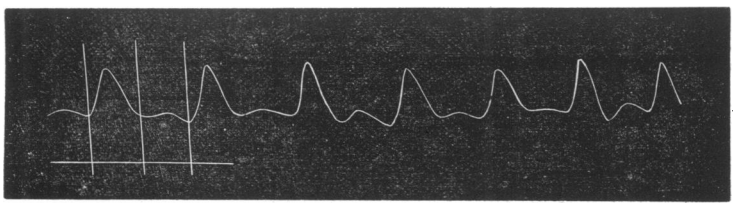
Pressure 4 ounces.

18. G. L. Tracing from dorsalis pedis. The tidal wave is not more widely separated from the primary wave than it is in the brachial or radial arteries, but, if anything, rather the reverse.



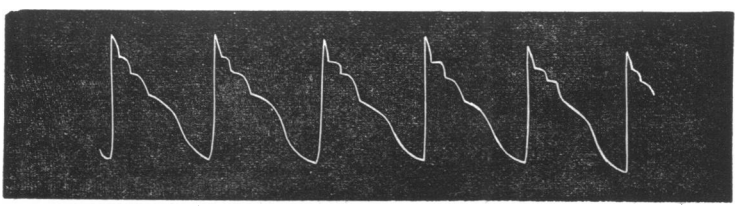
Pressure 3 ounces.

19. Tracing of dicrotic pulse from the femoral artery just below Poupart's ligament.

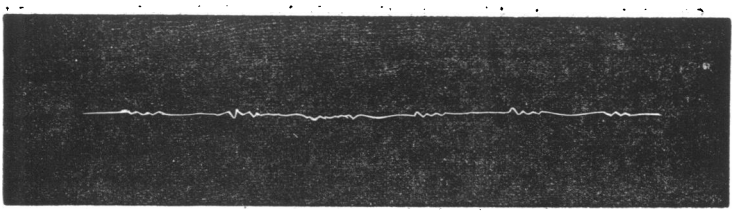


Pressure 3 ounces.

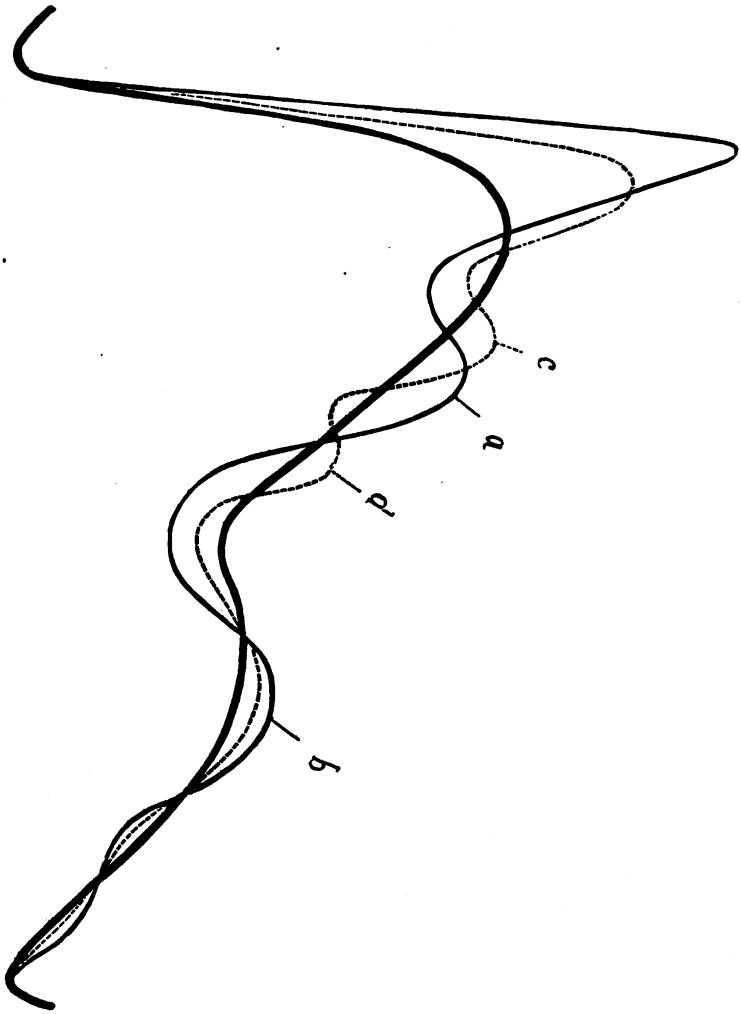
20. Tracing from the posterior tibial artery below the ankle of the same person. The dicrotic wave is not nearer in proportion to the primary wave, as it would be if it were a reflected or retrograde wave.



21. Tracing copied from M. Marey, shewing how the tidal wave is broken into two small waves by the use of a secondary spring in the sphygmograph.



22. Tracing shewing the effect of percussion on the exterior of a rigid part of the experimental tubing.



23. Diagram to illustrate the relation of the sphygmographic tracing to the true pulse-wave. The thick line is intended to represent the true pulse-wave, the thin line the sphygmographic tracing, the dotted line the tracing drawn by a sphygmograph having a secondary spring to keep down the lever.

a. Tidal or "first secondary" wave.

b. Dicrotic or "principal secondary" wave.

c. d. Two small waves into which the tidal wave may be broken by the action of the secondary spring, as shewn in fig. 21.

the circulation in mammalia, sensation is not remarkably affected; muscular contractility is very much diminished. More exact knowledge of the degree and order in which these various functions are affected, can only be obtained by carefully performed experiments made in Europe, where the more delicate electrical instruments can be had.

(*e*) It is exceedingly fatal to man, in doses of thirty grains of the kernel, if not promptly ejected.

(*f*) It causes a numb, tingling sensation in the part with which it comes into contact, and also throughout the body.

(*g*) It is powerfully emetic and purgative, produces great nausea and debility, paralysis of motion, occasionally delirium, narcotism, and perhaps vertigo.

(*h*) It may be inferred to cause death in man, as in all other animals, by tetanizing the heart.

NOTE by DR GALABIN, *in continuation of his Paper* (p. 22).

Yet another has been added to the already numerous theories of the dicrotic wave by Mr Mahomed, in a paper lately published in the *Medical Times*. It is the more deserving of notice since it is to the able researches of its author that we owe the most recent contributions to our knowledge as to the clinical use of the sphygmograph. Mr Mahomed then finds a sufficient cause for the dicrotic wave in the mere fact that the coat of the aorta is elastic, and considers that it originates from the contraction of this elastic coat during diastole. It appears to me that this theory involves a misconception of the nature of elasticity. If a surface is said to be elastic, nothing more is meant than that it is extensible in such a way that the degree of its extension has a definite relation to the forces extending it. Its contraction is not an active proceeding, but is merely the effect of the diminution of tension. Thus the contraction of the aorta is the consequence of the diminished pressure within, and therefore the mere fact that it contracts cannot at the same time be a cause of an increase of that pressure originating a second wave.