ON THE
CAUSATION OF THE WATER^{ER} HAMMER
PULSE,

AND

ITS TRANSFORMATION IN DIFFERENT ARTERIES AS
ILLUSTRATED BY THE GRAPHIC METHOD.

BY

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THE characteristic pulse of aortic regurgitation is so familiar to the observation of every one that it may perhaps be thought that its mode of origin must be so well known that there can be little room for further discussion on the subject. With regard to the theory of its causation, little or nothing has, I believe, been added to the explanation given by Sir Dominic Corrigan in his first well-known paper. This paper, published in the 'Edinburgh Medical Journal' in 1832, gives indeed an account of most of the characters which are now recognised in this form of pulse. Sir Dominic Corrigan there describes, as a symptom of the permanent patency of the aortic orifice, a visible and forcible pulsation of the

arteries, most distinct in those of the head and neck, especially the carotids and temporals. He states that it is there most visible in the erect posture, and that it becomes more distinct in the radial artery when the arm is raised. He explains it as due to the fact that the aorta pours back blood into the left ventricle, and becomes flaccid during diastole, the result of which is that the arteries adjoining become also rapidly emptied. The fact that the character is most marked in the arteries of the head and neck he ascribes to the circumstance that these most readily empty themselves backward, being assisted by the gravity of the blood. The same reason he applies also to account for the effect produced by elevation of the arm. He considers that the strength of the heart has but little to with the result, since the pulsation is but little marked in the lower extremities. I believe that the explanation thus given by Sir Dominic Corrigan is that which is now generally accepted, except that most observers would now consider the dilatation and hypertrophy of the left ventricle to be a more important element in the matter. We may consider, then, that the received explanation of the pulse of aortic regurgitation is, that it is simply the effect of an ample pulse-wave being thrown into empty arteries, and that the reason why it is developed by elevating the arm is solely that the radial artery is thus rendered more empty during diastole.

In the present communication I propose to consider whether all the characters of the aortic pulse, as discovered by the finger and by the sphygmograph, are fully accounted for by this theory, which in itself is undoubtedly a true one, or whether any further mechanical explanation is required or can be discovered. I will first refer to a character on which Sir Dominic Corrigan does not lay stress in the article which I have quoted, but one which is well known to every one, and which has given to this form of pulse the title which I have taken in the heading of my paper, namely, that of water-hammer

pulse. The quality which is well expressed by this name is that of extreme suddenness in the commencement of the pulse, which in marked cases gives to the finger the impression of a sudden blow or jar. I think it is not too much to say that this is *the* diagnostic quality of the pulse of aortic regurgitation, for a merely ample wave entering a relaxed artery may be found, to a considerable extent, under other conditions, as for instance in persons who have habitually relaxed arteries, when under the influence of excitement, or violent muscular exercise. Moreover, skilled observers do not hesitate to found a diagnosis of aortic regurgitation on this sole quality of suddenness in some instances where the pulse-wave is not ample at all, as in cases where mitral is combined with aortic regurgitation. It may be added that it is the quality of suddenness which is especially developed by the elevation of the arm. Now, in the explanation which I have mentioned there is nothing to account for this suddenness, as apart from the mere height and fulness of the pulse-wave. For we know, as a general rule, that a hypertrophied muscle commences its contraction in a more slow and deliberate manner than one which is weak. This is confirmed in the case of the heart by the dull and prolonged first sound associated with hypertrophy, as opposed to the short and sharp first sound of dilatation. The point can also be tested by direct measurements. From cardiographic tracings I have found that the interval occupied by the main upstroke of the trace, which indicates the time which the ventricles take in becoming fully hardened, may be increased in cases of aortic disease from its normal value of about one twelfth of a second to as much as from one eighth to one sixth of a second, that is to say, that it may be nearly doubled. There is therefore no reason to think that the hypertrophied heart of aortic disease would throw a more sudden wave than usual into the aorta, and I need not say that the water-hammer pulse is not confined to those cases, if such there be, in which there is dilatation without hypertrophy.

There is another quality about the aortic pulse which I would mention from my own observation, namely, that it is less marked in the large arteries than in those which are somewhat smaller, as the temporal, facial, or radial, which are often seen pulsating much more violently in proportion. I believe, indeed, that the quality of suddenness is scarcely marked at all in the largest arteries, the carotid, subclavian, or abdominal aorta. I hardly know whether this is generally recognised as being the fact, but I will refer hereafter to the evidence on this point afforded by the sphygmograph.

Now, the quality of pulse which depends simply upon an ample wave being thrown into an empty artery would of course be more marked near to the heart than at a distance, since the aorta would more readily empty itself backward than the more distant arteries.

I would say, then, that *there are two qualities of the aortic pulse manifest to ordinary observation, one of the two being its most diagnostic quality, as to which the common theory of its causation offers no explanation whatever.*

I pass on to the evidence afforded by the sphygmograph as to the aortic pulse. As might be anticipated, one of its characteristics, in marked cases, is its extreme amplitude when taken at a low pressure. This, when it is present, is sufficient to be diagnostic, but it is absent in milder cases. An instance of it, although by no means so extreme as may often be found, is shown in Pl. XV, fig. 11. This was taken from a patient in whom only a systolic murmur at the base and no diastolic murmur could at the time be detected, although the pulse, both to the finger and the sphygmograph, had a marked quality of regurgitation. One of the most important and characteristic qualities of the pulse-curve of aortic regurgitation is that of a flatness in its diastolic portion, such as may be seen in Pl. XV, figs. 5, 16, and Pl. XVI, figs. 6, 10, 11, 12, 14, and 15. This at first sight seems to confirm the common view that the most essential point about the

aortic pulse is the emptiness of the artery during the diastolic period. In many cases, however, when the pulse has to the finger the aortic quality, this flatness does not appear in the trace, and probably many persons have on this account been disappointed at finding less assistance from the sphygmograph than they expected in the diagnosis of aortic regurgitation.

In Pl. XV, fig. 12, is shown the trace of a pulse which to the finger had a distinct, although by no means an extreme, aortic quality. In this there is no flatness whatever in the diastolic portion, and the only thing abnormal about it is its great amplitude and the suddenness of the upstroke. This latter quality is not obvious to the eye, and is only adequately appreciated when arcs of circles are drawn through the different points of the curve, and accurate measurements are taken. Thus, the general shape of the curve in Pl. XV, fig. 12, does not seem very different from that of the healthy pulse shown in Pl. XV, fig. 2, but on taking measurements it is found that the upstroke of the aortic pulse (fig. 12), although higher, occupies about one third less time than that of the healthy pulse in fig. 2. I believe that in all instances in which the flat diastolic portion is seen in traces taken at a somewhat high pressure it would be replaced by a descending line in the same situation if the trace were taken at a lower pressure, but this the dimensions of the recording plate will frequently not allow, so great is the amplitude of the pulse when pressure is very low.

Now, it is to be remembered that a similar flatness in the diastolic part may be produced in a healthy pulse by applying too strong a pressure. Nevertheless, the fact that such flatness is readily produced by a pressure much less than that which extinguishes the pulse is of great diagnostic importance, but it is to be regarded rather as an indication of freedom of regurgitation than as a delicate sign of its presence. It indicates, not a sudden emptying of the artery at the end of systole, for then it would be seen in

a trace taken at a lower pressure, but merely that the tension throughout the diastolic period is greatly less than that during systole.

In Pl. XV, figs. 4 and 5, taken from a case of extremely free regurgitation, is shown the flatness readily brought out by a moderately increased pressure, namely, one of $4\frac{1}{2}$ ounces (*vide* fig. 5), although the most perfect trace (fig. 4), taken at a pressure of three ounces, which is about the pressure applicable to a normal pulse, shows no flatness whatever.

Closely allied to the flatness in the diastolic portion is the absence, or relative smallness, of the dicrotic wave. I shall avoid entering here on the vexed question of the causation of dicrotism which I have elsewhere discussed. I will remark only that great expectations might be raised as to the diagnostic value of the trace from the common view that the dicrotic wave is due solely to the closing of the aortic valves, for then it should be absent or much diminished in cases of regurgitation, but that these expectations will be in great measure disappointed. Other things being equal, the dicrotic wave is less when the aortic valves are imperfect, but the degree of dicrotism is affected by many other conditions besides the state of the valves, and it may appear to be as great as, or sometimes even greater than, normal in a pulse which has to the finger a distinct aortic quality, as will be seen in Pl. XV, figs. 11 and 12, where the dicrotic wave is indicated by the letter c. A considerable diminution or absence of the dicrotic wave is, however, a valuable sign of freedom of regurgitation.

It is interesting to note the effect of fever on the pulse of free aortic regurgitation, an instance of which is given in Pl. XV, fig. 17, taken from a patient suffering from pneumonia, whose temperature was 103.1° . The pulse has become hyperdicrotic, but the actual magnitude of the dicrotic wave (c) in proportion to the primary is much less than it might be expected to be with this degree of fever. Moreover, probably in consequence of

the cardiac hypertrophy, there is still seen in the trace the tidal wave (B), which is generally lost at such a temperature, and before the pulse becomes hyperdicrotic. When the aortic quality of the pulse is more extreme, even a great elevation of temperature may fail to bring out any dicrotism, and if there is very great hypertrophy, there may be still a marked tidal wave, even when the pulse is very rapid, indicating a comparatively prolonged systole, in place of the short contractions of the ventricle, which generally occur when the pulse-rate is much increased. Both these qualities are strongly marked in the pulse shown in Pl. XVI, fig. 12, which was taken from a patient suffering from extreme aortic regurgitation, complicated by pneumonia. He had at the time a temperature of 104° and pulse of 126, and he died within twenty-four hours from the time when the tracing was taken. The tidal wave is marked in the figure by the letter B, and no dicrotic wave is visible.

The abnormal development of the tidal wave, which, in the instance just mentioned, persisted even at a temperature of 104° , is a very general characteristic of aortic pulses in the absence of fever. In these cases it has not quite the same interpretation as when the vascular system is healthy.

A pronounced tidal wave has become commonly known as a sign of increased arterial tension, understanding by this term not merely an increase of the tension reached at the summit of the wave, but an elevation of the minimum tension at the end of diastole, in consequence of a diminished freedom of outflow from the arteries. This interpretation is established by the experience which shows that such a tidal wave, the vascular system being healthy, is associated, as a rule, with an increase, not only of the pressure required to extinguish the pulse, but of that which it will bear without diminution, or much diminution of the amplitude of the trace. It is confirmed by the consideration that the systole of the heart will be prolonged if it meets with unusual resistance, owing to an obstructed

outflow from the arteries. The evidence of elevated tension derived from the tidal wave should, however, always be corroborated by that afforded by the recorded pressure, and I think that it has been used by some who have comparatively small experience in sphygmography, without a sufficient regard for the necessary qualifications. That it does not always hold good in cases of valvular disease is shown by the fact that the tidal wave attains its very greatest development in cases of great obstruction of the aortic orifice (Pl. XVI, fig. 8), a condition which certainly does not tend to increase arterial tension. Apart from any change which the pulse-wave has undergone in receding from the heart, a marked tidal wave directly indicates a prolongation of ventricular systole.

Now, in most cases of marked aortic regurgitation in which there is a large tidal wave, it is found that the pressure required to extinguish the pulse is much increased, but that the greatest amplitude of curve is obtained at a low pressure, and that even a moderate increase of pressure is often sufficient to flatten its diastolic portion. This confirms the conclusion, which is almost self evident, that in aortic regurgitation the minimum arterial tension is rather diminished than increased, the arteries being able to empty themselves backwards as well as forward. The prolonged systole in such cases is therefore simply due to the hypertrophy of the heart. Hence a distinction must be made between the increase of maximum and that of minimum arterial tension; and it should be remembered that the former only, and not the latter, is found in aortic regurgitation.

I have next to consider the evidence afforded by the sphygmograph as to the effect produced upon the radial pulse by the elevation of the arm. By this means the water-hammer quality of the pulse is often developed in an extraordinary degree, and the finger sometimes receives the impression that the force of the stroke against it is actually increased. In many cases, however, this is not confirmed by the sphygmograph, as may be seen by a

comparison of figures 3 and 13 in Pl. XVI, taken while the arm was elevated, with the corresponding traces, figures 2 and 12, taken when it was horizontal. The height of the upstroke is generally diminished, as well as the pressure which the pulse will bear, and it would seem that the wave loses somewhat of its force by ascending in opposition to gravity. The eye does not detect so readily as the finger what is the peculiar quality about these pulses. But if arcs be drawn through the several points, and measurements be taken, it is found that the suddenness of the upstroke is increased. In the two instances given the time occupied by the upstroke, with the arm elevated, is only about two thirds of the corresponding interval, already shorter than normal, with the arm horizontal. It is therefore, in many cases, solely the quality of suddenness, and not that of fulness in the aortic pulse, which is increased by elevation of the arm. Probably, indeed, the suddenness thus produced is not quite adequately represented in the sphygmographic trace, since suddenness of motion is of all qualities the most difficult to register by any recording instrument, the mechanism of which is apt to lag behind the motion to be followed, when it is of a very abrupt kind.

In some instances, on the other hand, the amplitude of the trace is actually increased by elevation of the arm. Usually the pressure applied to the artery must be diminished to obtain this result, but the fact remains that, in these cases, the most ample trace with the arm raised is loftier than the most ample curve which can be procured when it is horizontal. Occasionally the same result is found even when the pressure applied is the same in the two cases, an instance of which is shown in Pl. XVI, figs. 14 and 15, the former taken with the arm horizontal, the latter with the arm raised, and both at a pressure of three ounces. The case was one in which the water-hammer quality of the pulse only became manifest upon elevation of the arm. I think that a comparison of the curves will show that these require some further explanation than

that afforded by the common theory of the aortic pulse. For according to that theory the increased amplitude is explained on the ground that, when the arm is raised, the artery is more completely emptied during diastole, and thus the base-line of the curve is lowered, so that its summit appears more lofty. This, however, is not sufficient to account for the difference in these curves. For, in the first place, the flatness of the diastolic portion of the curve in figure 14, in which the aortic notch descends almost to the level of the base-line, shows that the artery was already, when the arm was horizontal, almost completely flattened under the spring of the sphygmograph even at the beginning of that portion of the wave which corresponds to diastole; and secondly, the systolic wave in figure 15 has not simply acquired an increased elevation above the rest of the curve, but its shape is transformed, the primary summit having become more lofty and pointed, and the tidal wave lower than that in figure 14. In other words, the wave has undergone an increased degree of that change, which, as will hereafter be shown, always takes place in the aortic pulse as it recedes from the heart.

It is not very easy to obtain reliable tracings for comparison when the arm is raised, and some precautions are necessary to avoid error. For, in general, it is better to dispense with the small secondary spring, used to keep the recording lever in apposition with the knife edge of the sphygmograph, but when the arm is raised this must be employed, since the lever is no longer sufficiently kept down by its own gravity. The secondary spring therefore should be so delicate as only just to prevent the lever from separating from the knife edge, and tracings, with the arm horizontal, should first be taken both without and with the small spring, in order to observe the change, if any, produced by its presence, and ensure that the difference so made is unimportant.

I think that a careful estimation of the quality developed in the aortic pulse by elevation of the arm, even as

ascertained by the finger only, will be in agreement with the conclusion that it belongs to the upstroke of the wave only, and that, in most cases, it consists chiefly of the character of suddenness. For since the finger scarcely detects the artery during the diastolic period, except when abnormally thickened, its greater or less emptiness at that time can have but little effect on the impression conveyed; and I believe that most observers, on analysing the process of judgment, will find that they draw their conclusion as to an aortic quality solely from the stroke against the finger, without waiting for the diastolic portion of the wave.

I pass on to the changes of the aortic pulse in the different arteries, as revealed by the sphygmograph; and I will first draw attention to a case in which these changes were unusually marked. In Pl. XVI, figs. 9, 10, and 11, are shown tracings from the carotid, radial, and dorsalis pedis arteries of a patient suffering from aortic regurgitation, which were taken while he was lying in bed. It will be seen that, in the carotid artery (fig. 9), the systolic portion is extremely broad and square, corresponding to the prolonged systole of the hypertrophied ventricle. In the radial artery (fig. 10) the systolic portion is still broad, but the diastolic portion has become more flattened, while in the dorsalis pedis (fig. 11) the pulse-wave has become completely transformed; the primary wave is exceedingly sudden, lofty, and narrow, and is followed by a long flat interval. The characters of the pulse to the finger corresponded exactly to that seen in the tracings, for in the carotid artery there was no perceptible suddenness, in the radial it was somewhat marked, but in the dorsalis pedis, an artery whose pulsation is not generally very distinct, the water-hammer quality was developed in the most extreme degree possible. In all other instances which I have examined by the sphygmograph the sudden, lofty, and pointed primary wave has been absent in the arteries nearest to the heart, and has only been developed at a certain distance from it.

A series of tracings corresponding to the last is shown in Pl. XVI, figs. 5, 6, and 7. These were taken from the subclavian, radial, and dorsalis pedis arteries of a patient who was suffering from aortic regurgitation of a very free character, as is indicated by the almost entire absence of the dicrotic wave in the traces. In the subclavian trace (fig. 5) the primary upstroke is even more gradual than is usual in the normal pulse, and there is no flatness in the diastolic portion. In the radial pulse (fig. 6) the systolic wave has become relatively higher and steeper, but it is still broad at the summit; in the dorsalis pedis (fig. 7) it has become much more pointed, while its right hand shoulder, formed by the tidal wave, has become slanted off. A corresponding change is manifest in the singular series of tracings shown in Pl. XVI, figs. 1, 2, and 4, also taken from a case of free aortic regurgitation. In the carotid artery (fig. 1) the second summit of the bifurcated systolic wave is higher than the first. In the radial pulse (fig. 2) the first summit has become the highest, and in the dorsalis pedis (fig. 4) the first summit has become very high and lofty in proportion to the rest of the trace, while the second summit (B), which is formed by the tidal or predicrotic wave, is very slightly marked in comparison, although it retains about the same relative position. Yet another series of a similar kind is shown in Pl. XV, figs. 6, 7, and 8, taken from the radial, femoral, and dorsalis pedis arteries of another patient suffering from aortic regurgitation. The femoral trace does not differ from that of the radial or dorsalis pedis so widely as does the curve taken from the carotid or subclavian; but these tracings also show exactly the same kind of change. The systolic wave is much more lofty, narrow, and sudden in the dorsalis pedis trace (fig. 6), while the dicrotic wave (c), which in the radial was well marked, has become almost entirely lost.

I think that the evidence afforded by these tracings, which in all cases were in accordance with the quality of the several pulses as manifest to the finger, is sufficient to

show that *the water-hammer quality of the aortic pulse does not exist in the pulse-wave as it is thrown into the aorta, but is a character which is gradually developed as the wave recedes from the heart, and reaches its maximum at a point which, in some cases, is as distant as the dorsalis pedis artery.*

It is true that the carotid arteries are often seen in these cases to pulsate strongly, but this is merely the effect of the amplitude of the wave, due to the dilatation and hypertrophy of the left ventricle, and not to its suddenness. It is perhaps unusual for the water-hammer quality to be so relatively prominent in the dorsalis pedis artery as it was in the cases of which I have shown tracings. I have observed that this happened when this quality was not very marked in the radial artery, and the maximum of suddenness seemed only to be acquired at a greater distance from the heart. When the radial pulse is extremely sudden, that of the dorsalis pedis is generally but little so, for the maximum of suddenness has then been reached at a shorter distance from the heart, and the wave is afterwards rendered progressively more gradual, a change which the elastic walls of any tube tend to produce in all waves which pass through it. I think, however, that it is important to remember, as a practical aid to diagnosis, that in a few cases in which the aortic quality is absent, or but slightly marked in the radial pulse, it may be distinct in the dorsalis pedis, when the leg is horizontal, or, still better, when it is elevated.

In order to arrive at a correct understanding of the cause of this acquired suddenness of the aortic pulse, it is necessary to compare the changes which it undergoes with those of various forms of pulse, when no valvular disease is present. This physiological change in the pulse-wave I have discussed in a paper published in the 'Journal of Anatomy and Physiology' for January, 1876, founded on a large number of tracings taken from the different arteries of the body, and from different points of

a schema representing the arterial system, made up of bifurcating elastic tubes, and adapted to an artificial heart of india rubber. I found that in all cases the systolic wave underwent a progressive change of such a kind that its right hand portion, generally made up of the tidal wave, became gradually shaved off, while the primary summit became narrower and comparatively loftier than before. At the same time the descending curve of the wave became more and more gradual, as all waves transmitted through elastic tubes eventually do, but this change did not occur in anything like a corresponding degree in its ascending curve, but on the contrary this often retained its steepness for a considerable distance. There was therefore evidence of the existence of some cause which, with regard to the ascending curve of the wave, counteracted, up to a certain point, the tendency which the elastic walls of tubes have to render gradual, and eventually obliterate, all waves. The change of shape of the systolic wave may be described as a progressive piling up of more and more of its volume in the front of the wave, that is to say, at its left hand side, as it appears in the trace, while its descending portion acquires a more gradual slope. This change as seen in the healthy pulse is shown in Pl. XV, figs. 1, 2, and 3, taken from the carotid, radial, and dorsalis pedis arteries of a healthy man, who had a somewhat unusually strong and full pulse. In Pl. XV, figs. 9 and 10, are shown tracings from the tubes of the schema which have a considerable resemblance to the human pulse, and show all the secondary waves which are seen in it. Fig. 9 was taken from the main trunk of the schema, not far from the aortic valves, and fig. 10 from a small tube at a distance of four and a half feet from the former. It will be seen that the systolic wave in the latter curve has undergone a similar change to that which occurs in arteries.

There are some cases, even when there is no valvular disease, in which the pulse-wave undergoes a far greater

transformation than that which normally occurs. In Pl. XV, figs. 13, 14, and 15, are shown the carotid, radial, and dorsalis pedis arteries of a patient suffering from phthisis. His arteries were exceedingly relaxed, and he had at the time a temperature of 102.8° . It was noticeable also that there was a marked interval between the pulses in the different arteries, indicating an unusually slow transmission of the pulse-wave. It will be seen that in the carotid pulse, fig. 13, there is a marked tidal wave (B), which forms the summit of the trace, and dicrotism is not great. In the radial artery, fig. 14, the systolic wave has undergone the usual kind of change, but in a very unusual degree. The primary summit has become narrow and pointed, and the tidal wave is almost lost. In this instance another curious change has also taken place: the pulse has become hyperdicrotic, and the dicrotic wave of considerable magnitude as compared with the primary. In the dorsalis pedis artery, on the other hand, the primary wave is still pointed, although less so than in the radial, but the dicrotic wave is entirely lost, and its former place is occupied by a flat interval. The unusual degree of transformation in the pulse-wave which took place in this patient is not usually found in febrile or dicrotic pulses, and the only thing which could be found in him to account for it was the extremely lax condition of his arterial system.

The result, then, of the comparison of the several series of pulses in aortic regurgitation with similar series taken from persons having healthy vascular systems is to show that *the transformation which the systolic portion of the pulse of aortic regurgitation undergoes, and by which the quality of suddenness is developed, is only an exaggeration to an extreme degree of a change which takes place to some extent in the normal pulse.*

In seeking the mechanical explanation of this change of shape we are naturally led to look for analogy to the modifications which other kinds of waves undergo. Now, a precisely similar change of form is undergone by waves

on the sea, or on the surface of any water, as they approach the shore. The wave becomes more lofty, and its front progressively steeper, while its descending curve grows more gradual, until at length the front becomes so steep that the wave curls over and breaks. The mechanical explanation of this is well known. It depends upon the principle that each individual portion of a wave may be regarded as itself moving as a separate wave, the effects of which are combined with those of all similar component waves. The whole wave-motion is then the aggregate formed by adding together the motions due to all the parts, or component waves, into which it is divided. If, then, the conditions of motion are different at different parts of the wave, the velocity of transmission may be also different at different parts, and, if this is the case, the shape of the wave will become altered as it proceeds. Now, in the case of a wave on the surface of water, we know from hydrodynamics that the wave-velocity is greater the greater the depth of the water. The depth of the water is of course greater at the summit of the wave than at its base, and hence the crest of the wave has a greater velocity than its front, and constantly tends to overtake it. So long as the water is deep this effect is extremely slight, and is overcome by the tendency which all waves have to become more gradual as they proceed. As soon, however, as the water becomes very shallow, the difference between the depth at the summit and that at the base of the wave becomes considerable in proportion to the whole depth. The crest of the wave then rapidly overtakes its commencement, so that the front of the wave grows steeper and steeper, until at length it breaks.

A precisely similar cause acts, under some circumstances, upon another sort of wave, extremely different in shape and dimensions, namely, the tidal wave. When concentrated by funnel-shaped estuaries, this sometimes acquires, in consequence of the shallowness of the water, the steep and broken front which is known in some rivers

by the name of the "bore." The bore on the Severn or the Ganges has thus a precisely analogous cause to the breaking of the smallest ripple upon the bank of a pond.

In order, then, to find an explanation in the case of pulse-waves, we have to examine the velocity of transmission of waves in elastic tubes. The mathematical solution of this question is one of great difficulty, for it requires as a starting-point a knowledge of the laws, to be ascertained by experiment, which connect the tension and the expansion of elastic tubes, and these have not been sufficiently ascertained. I have not therefore been able to arrive at an accurate calculation of the actual velocity, but I have succeeded in deducing the conclusion, that the velocity is greater the greater the pressure within the tube. Since, however, there is something doubtful in the data for calculation, it is fortunate that this result can be easily confirmed by experiment, and my observations have shown me that this law does hold good. More elaborate experiments on the point have been made by Dr. Marey, and are described in his recent work (*'Physiologie Expérimentale,'* Paris, 1875). He has estimated the wave-velocity by taking a very large number of simultaneous tracings from successive points of the tubes under observation, and he arrives at the same conclusion, namely, that the velocity is greater the greater the tension (op. cit., p. 111). Thus he finds that, if a succession of waves be thrown into the tube, the velocity of transmission progressively increases as the tension rises (op. cit., p. 115). The same explanation will apply to another result which he observes, namely, that the velocity of each wave progressively diminishes as it travels onwards; for as the wave becomes flattened as it proceeds, the tension at its summit will become less, and its velocity will be diminished in proportion.

Now, as the pulse-wave proceeds along an artery it is manifest that the tension at the summit of the wave is much greater than that at its base. The summit of the wave will therefore have a greater velocity than its front,

and will constantly tend to overtake it. The result will be that the highest point of the wave will tend to become nearer and nearer to its commencement, and more and more of its volume to be piled up at its front, so that the front itself becomes steeper. This is precisely the kind of change which the tracings show actually to take place in the pulse-waves. In the normal pulse, however, this tendency, as far as regards the steepness of the upstroke, seems to be pretty much counteracted by the opposing influence, namely, the tendency of the elastic arterial walls to render all waves more gradual, and eventually to obliterate them entirely. The influence tending to make the upstroke steeper is proportional to the difference of tension between the summit and the base of the wave. This will obviously be very much greater in cases of aortic regurgitation, in which a powerful wave is thrown into the vessels, and the arteries become very empty during diastole. We may expect then the result which we actually find, that in such cases the influence tending to make the front of the wave steeper preponderates, and the quality of suddenness in the pulse is developed as it recedes from the heart, so that the well-known water-hammer character is thus produced. This effect, however, must at some point reach a maximum, after which the obliterating influence alone is at work, and both upstroke and downstroke are gradually rendered slanting, somewhat as the wave approaching the shore loses all its steepness when once it has broken. We must expect, then, that, if the pulse of aortic regurgitation has become very sudden at a moderate distance from the heart, as in the radial artery, it will have lost its suddenness at a greater distance, as in the dorsalis pedis.

The same principle fully explains also why the quality of suddenness is increased by elevation of the arm or leg. For the arteries are then rendered more empty during diastole, the difference between the tension at the summit and that at the base of the wave is increased, and therefore the tendency to render the front of the wave steeper,

which is proportional to this difference, is increased likewise. It may perhaps be regarded as a confirmation for this explanation, that the case in which a great transformation in the pulse-wave was found apart from any valvular disease (Pl. XV, figs. 13, 14, and 15), was one in which the arteries were excessively lax, and therefore the tension very low during diastole.

It may perhaps be thought by some improbable that there should be any analogy between waves transmitted through an elastic tube and waves on the surface of water, which seem to be very different in their nature. It is well known, however, to mathematicians that there is a certain degree of analogy in all kinds of wave motion, and close correspondence sometimes exists between waves produced under very different circumstances. . Thus the same form of equation is found to represent the motion of waves on the surface of a straight closed trough or canal of water, and also the transverse vibrations of elastic strings, and the peculiarities of the oscillations have a close resemblance in the two cases, although they are very slow in the one and very rapid in the other. With regard to pulse-waves and waves on the surface of water, the similarity in the change of shape results from a resemblance in one point only, namely, that in both there is a cause which renders the velocity of transmission greater at the summit than at the base of the wave, that cause itself being quite different in the two cases. The only other resemblance in the nature of the two motions necessary to render the analogy valid is the undoubted fact that each is transmitted as a wave.

For the production of a change of the shape of the wave in the manner which I have described, it is of no consequence whether the motion of the particles of fluid is transverse to the direction of propagation of the wave, or longitudinal, or whether, as is the case in most waves, and in both of the forms of wave under consideration, the motion of each particle is partly longitudinal and partly transverse. The wave on the surface of water is convenient for the pur-

pose of illustration, because a considerable proportion of the motion of the particles is transverse, and the wave has therefore a visible shape. A similar transformation might, however, take place even in a wave in which the motion of the particles of fluid is entirely longitudinal, such as that produced by the propulsion of an elastic fluid through a non-distensible tube. The only difference is that in such a case the wave would not be visible, unless rendered so by some special contrivance, and therefore the change of its shape would not be manifest. In the case of the pulse there is some lateral as well as a longitudinal movement of particles, and a true wave of lateral expansion in the artery occurs. It is true that the sphygmographic trace indicates directly the wave of variation of pressure in the artery rather than the wave of expansion, for upon the pressure within the artery depends the degree of flattening which it undergoes under the spring of the sphygmograph. Since, however, the expansion of the artery when uncompressed varies with the pressure within it, although not according to a strict arithmetical proportion, one quantity may be taken as the representative of the other, and the sphygmographic curve will nearly express the shape of the magnified wave of expansion of the uncompressed artery. It is scarcely necessary to add that there can be nothing in the case of arteries which corresponds to the breaking of a wave on the surface of water, except the fact that in the former case also there may be a certain point at which the front of the wave reaches its maximum of steepness, after which it becomes progressively more gradual.

It is instructive to observe the contrast in the change of shape of the dicrotic as compared with that of the primary wave. In all cases the ascending curve of the dicrotic wave (c) begins from the first to become rapidly more gradual, instead of, like the primary wave, retaining its steepness for a considerable distance. This will be seen best by a comparison of the tracings taken from the carotid, radial, and dorsalis pedis arteries of a healthy person, shown in Pl. XV,

figs. 1, 2, and 3. The distance from the commencement to the summit of the dicrotic wave in the radial artery is about three times as great as in the carotid, and in the dorsalis pedis about four and a half times as great. The same change is apparent in numerous corresponding traces obtained by Wolff from the radial and dorsalis pedis arteries, and also in some published by Professor A. H. Garrod. The explanation is that the difference in tension between the base and summit of the dicrotic wave is but slight, the tendency to make the upstroke steeper is therefore slight likewise, and is far overpowered, even from the commencement, by the obliterating influence of the elastic arterial walls, which tends to make both upstroke and downstroke alike more gradual, and the base of the wave broader.

As seen in the tracing of the aorta, which may sometimes be obtained when that vessel has undergone aneurismal dilatation, without the formation of any sacculated aneurism, the ascent of the dicrotic wave is still more abrupt and steep than in the carotid. So different does it look in this form from the appearance which it has in the radial pulse that Marey, who obtained the abrupt pointed wave in his tracings representing the pressure within the aorta of the horse, considered the two to be quite different in their origin. The abrupt wave in the aortic trace he attributed to the closure of the aortic valves, but he believed that the dicrotic wave, as seen in the peripheral arteries, was produced in those arteries themselves. If, however, in a patient who has aneurismal dilatation of the aorta, a series of tracings be obtained from the aorta, subclavian, brachial, and radial arteries, the change in shape of the dicrotic wave is found to be quite a gradual and progressive one, and a still further transformation of the same kind is found to have taken place in the dorsalis pedis.

The latest view held by Marey as to the causation of the dicrotic wave, as explained in his recently published work ('*Physiologie Expérimentale*,' 1875), is that it is a

wave which proceeds from the centre towards the periphery, and that it is caused by an oscillation depending chiefly upon the inertia of the fluid and the velocity with which it is thrown into the arteries, but that this oscillation takes place not in the aorta, but at the commencement of each individual artery, as it arises from the aorta. Amongst other objections to this theory I would state that I have found the dicrotic wave, for instance, in the abdominal aorta to be as well marked, or nearly as well marked, as in the femoral artery of the same person. My own view, as far as regards the chief origin of the dicrotic wave, differs from Marey's only in this, that I maintain that the oscillation takes place, in great measure at any rate, at the commencement of the aorta itself, and that it is modified in an important degree by the action of the aortic valves. I think that this view receives a strong confirmation in the almost entire absence of the dicrotic wave, in traces taken near to the heart as well as in those from more distant points, in some cases of very free aortic regurgitation. Of this the most striking instances among the tracings appended to this paper are figures 9, 10, and 11 in Plate XVI, representing the carotid, radial, and dorsalis pedis pulses, figures 5 and 6 in Plate XVI, representing the subclavian and radial pulses, and figure 12 in Plate XVI, representing the radial pulse, the last being taken from a case in which acute fever was present, the temperature being 104° .

There is another circumstance about the dicrotic wave which it is important to note, namely, the change of its relative position. In all cases it is found that, while the interval between the commencement of the primary and that of the dicrotic wave does not greatly vary, there is a marked and progressive increase, as the pulse-wave recedes from the heart, in the interval measured either from the commencement or summit of the primary wave to the summit of the dicrotic wave (c). This is seen in the series of normal tracings from carotid, radial, and dorsalis pedis arteries shown in Pl. XV, figs. 1, 2, and 3,

and also in the corresponding tracings from a case of aortic regurgitation (Pl. XVI, figs. 1, 2, and 3). The results which I have obtained are in this respect also confirmed by a comparison with the tracings obtained from different arteries by Wolff and by Professor A. H. Garrod. Since, then, the velocity of a wave must be regarded as being the velocity of its summit, it is proved that the dicrotic wave is transmitted more slowly than the primary. This is readily explained by the same cause which has been already found to account for the change in shape of the primary wave. For the tension is much less at the summit of the dicrotic than at the summit of the primary wave, and the velocity of the former wave is therefore less. The difference of tension will of course be greater when the pressure is unusually low during diastole, and therefore we may expect that in such cases the dicrotic wave will fall behind more rapidly. Accordingly we find that in the radial pulse shown in Pl. XV, fig. 14, taken from a patient whose arteries were extremely lax, the interval between the commencement of each beat and the summit of the dicrotic wave is increased by more than one half, as compared with the same interval in the carotid pulse (fig. 13). So great is this increase that, supposing it to take place at the same rate in the pulse-wave which proceeds to the foot, we should expect to find that, in the *dorsalis pedis*, the dicrotic wave had been entirely lost upon the succeeding beat. This seems indeed to have been actually the case, for in the curve of the *dorsalis pedis* (fig. 15) no dicrotic wave is visible, and its former place is occupied by a flat interval. The difference of tension and therefore difference of relative velocity will be still greater in many cases of aortic regurgitation, and hence it is possible that the absence of the dicrotic wave in traces of such pulses, taken at a distance from the heart, as in the *dorsalis pedis* artery, may sometimes be due, not so much to its having never existed as to its having become lost upon the succeeding systolic wave.

There are some radial pulses, and still oftener pulses

taken from arteries nearer the heart, in which the tricrotic wave (see Pl. XV, fig. 2 d) is seen following the dicrotic wave. It appears in general to be chiefly formed by the second member of that series of oscillations of which the dicrotic wave is the first, and it has the same, or a still greater, relative slowness of transmission. Accordingly it is never seen in the dorsalis pedis pulse (see Pl. XV, fig. 3), but it has always become lost upon the succeeding beat, before the pulse-wave has arrived at that distance from the heart.

I have introduced one radial pulse-tracing, namely, that in Pl. XVI, fig. 8, as a contrast to the tracings of aortic regurgitation. It is one which indicates considerable aortic obstruction, without any perceptible regurgitation. Cases in which such a result is obtained by the sphygmograph are infinitely more rare than those in which only a systolic and no diastolic murmur is heard by the stethoscope. It will be seen that there is a very large tidal wave, which rises far higher than the summit of the primary wave, and indicates the prolonged ventricular contraction; the pulse is irregular and very slow; and there is a marked dicrotic wave, to be associated with the fact that there was no free reflux through the aortic orifice.

I think that an interesting inference may be drawn from the characters of the water-hammer pulse, as revealed by the sphygmograph, with regard to the causation of that wasting, pallor, and cachexia which so distinguish aortic regurgitation from other forms of heart disease, and which often make the subject of it somewhat resemble a patient suffering from phthisis. One of the most important of the natural functions of the elastic coats of arteries is to render equable the stream of blood, which when it starts from the heart is interrupted, and it is probable that this is accomplished not so much in the very smallest arteries—whose coats are chiefly muscular, and have the function of regulating the blood supply at different times—as in those of larger size. The uniformity

thus produced in the current through the capillaries is no doubt of essential importance for the due progress of that exosmosis and endosmosis between the vessels and the tissues upon which nutrition depends. Now, it has been shown that, when there is any considerable aortic regurgitation, so far from the larger arteries rendering the blood stream more uniform, it becomes for a considerable distance progressively more unequal, and the point of maximum inequality is often not far distant from the smallest arteries. In such cases it can hardly fail to be the result that there is perceptible inequality even in the current through the capillaries, and it is not therefore wonderful that there is more effect upon nutrition, as compared with other results of disease, in aortic regurgitation than in other valvular imperfections.

It may be well to add, to avoid any risk of misconception, that throughout this paper I have spoken of as the systolic portion of the pulse-curve that part which corresponds to the systole of the heart, not that during which the artery is contracting. Similarly, by the term diastolic portion I mean that part of the curve which corresponds to the diastole of the heart. The junction of the two portions is nearly, but not precisely, marked by the aortic notch, or commencement of the dicrotic wave, its precise position being somewhat anterior to that point.

DESCRIPTION OF PLATES XV AND XVI.

The tracings have been copied by photo-lithography, and are represented of the original size. The traces from large arteries, the subclavian, carotid, or femoral, were taken with an amplifying power less than is usual in the sphygmograph, and the extent of amplification in vertical height of each of such traces is indicated in the description of the figures. For the radial and dorsalis pedis arteries the ordinary sphygmograph was used, having an amplifying power of about 90. In many of the curves measuring lines are drawn to show the relative intervals of time occupied by the main upstroke in different cases. These are arcs of circles, having a radius equal to the length of the recording lever of the sphygmograph, and are so drawn that the centre of each arc corresponds exactly in relative position to the axis of motion of the recording lever, at the time when the tracing was taken. In this way all points on the curve may be reduced to a horizontal line for purposes of exact measurement. All the traces of aortic regurgitation were taken from patients in whom the valvular disease appeared to have been due to rheumatism, and not to extension to the valves of disease affecting the aorta, in order to exclude, as far as possible, from the curves any effects due to atheroma of the arteries. The letter *B* in the figures denotes the tidal (or predicrotic), *c* the dicrotic, and *D* the tricortic wave. In the comparative tracings of radial and dorsalis pedis arteries both curves were taken while the patient was recumbent, except where it is otherwise specified.

PLATE XV.

Fig. 1.—From the carotid artery of a healthy man, *æt.* 26. Amplified 30 times.

Fig. 2.—From the radial artery of the same person. Pressure 4 oz. The curve is not exactly typical of a normal radial pulse, for it is of unusual amplitude, approximating in this respect to the pulses of aortic disease, and it shows the secondary waves with unusual distinctness. It represents rather the pulse of an athlete. The trace was obtained while the person was in a sitting posture, and the pulse is therefore quicker than those in figs. 1 and 3, which were taken while he was recumbent.

Fig. 3.—From the dorsalis pedis artery of the same person. Pressure 3 oz.

Fig. 4.—Thomas G—, æt. 20. Aortic regurgitation. Radial pulse-curve. Pressure 3 oz.

Fig. 5.—From the same pulse at a pressure of $4\frac{1}{2}$ oz.

Fig. 6.—James B—, æt. 29. Aortic regurgitation. Radial pulse-curve. Pressure 2 oz.

Fig 7.—From the femoral artery of the same patient. Amplified 40 times.

Fig. 8.—From the dorsalis pedis artery of the same patient. Pressure 2 oz.

Fig. 9.—Tracing taken from a schema of bifurcating elastic tubes representing the arterial system, and adapted to an artificial heart of india rubber, which was worked by means of the hand. This curve was taken from the main trunk of the schema, not far from the valves. Amplified 20 times.

Fig. 10.—Tracing simultaneous with fig. 9, taken from a small tube of the schema at a distance of $4\frac{1}{2}$ feet from the former. Amplified 40 times. The clockwork movement was very slightly slower than in fig. 9.

Fig. 11.—James J—, æt. 60. Aortic regurgitation. Radial pulse-curve. Pressure 4 oz. Only a systolic murmur at the base was audible at the time, but the pulse had a marked aortic quality to the finger.

Fig. 12.—John S—, æt. 17. Aortic regurgitation. Radial pulse-curve. Pressure 2 oz. The pulse to the finger had an evident, although not a very marked, water-hammer quality.

Fig. 13.—Th. J—, æt. 31. Tracing from the carotid artery. Amplified 30 times. The patient was suffering from phthisis, and had a temperature of 101.8° .

Fig. 14.—From the radial artery of the same patient. Pressure 3 oz.

Fig. 15.—From the dorsalis pedis artery of the same patient. Pressure 3 oz.

Fig. 16.—James T—, æt. 20. Aortic regurgitation. Radial pulse-curve. Pressure 2 oz.

Fig. 17.—John P—, æt. 17. Aortic regurgitation. Radial pulse-curve. Pressure $\frac{1}{2}$ oz. The patient was suffering from pneumonia, and had a temperature of 103.1° .

PLATE XVI.

Fig. 1.—Mary T—, æt. 22. Aortic regurgitation. Tracing from the carotid artery. Amplified 40 times. The pulse was manifestly double to the finger, the second beat preceding the second sound of the heart, and therefore not being due to the dicrotic wave.

Fig. 2.—From the radial artery of the same patient, the arm being horizontal. Pressure $3\frac{1}{2}$ oz.

Fig. 3.—From the radial artery of the same patient, the arm being raised. The pressure was 2 oz., higher pressures not giving so ample a trace. The water-hammer quality was very marked to the finger when the arm was raised, although the pulse was not a full one.

Fig. 4.—From the dorsalis pedis artery of the same patient. Pressure 2 oz.

Fig. 5.—Thomas L—, æt. 30. Aortic regurgitation. Tracing from the subclavian artery. Amplified 20 times.

Fig. 6.—From the radial artery of the same patient. Pressure 5 oz.

Fig. 7.—From the dorsalis pedis artery of the same patient. Pressure 4 oz.

Fig. 8.—Henry G—, æt. 54. Aortic obstruction. Pressure 4 oz.

Fig. 9.—George G—, æt. 33. Tracing from the carotid artery. Amplified 20 times.

Fig. 10.—From the radial artery of the same patient. Pressure 4 oz.

Fig. 11.—From the dorsalis pedis artery of the same patient. Pressure 4 oz. In this patient, when recumbent, the water-hammer quality was to the finger far more marked in the dorsalis pedis than in the radial artery. There was also an unusually marked interval of time between the pulsation in the different arteries.

Fig. 12.—Charles P—, æt. 18. Aortic regurgitation. Radial pulse-curve. Pressure $4\frac{1}{2}$ oz. The patient was suffering from incipient pneumonia (which quickly proved fatal), and his temperature was 104° . This tracing was taken with the arm horizontal.

Fig. 13.—From the radial artery of the same patient, the arm being raised. The pressure was 3 oz., higher pressures not giving so ample a trace. The suddenness of the pulse was very marked to the finger when the arm was elevated.

Fig. 14.—William T—, æt. 24. Aortic regurgitation. Radial pulse-curve taken with the arm horizontal. Pressure 3 oz.

Fig. 15.—From the radial artery of the same patient, the arm being raised. Pressure 3 oz.