A Lecture UPON THE NORMAL VENOUS PULSE.

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(a) A BRIEF HISTORY OF OUR KNOWLEDGE OF THE VENOUS PULSE IN ANIMALS AND MAN.

PULSATION of the veins includes many and diverse phenomena. Normally there is pulsation of the venous sinuses of the brain, a pulsation which is in part, if not in whole, transmitted from the arteries. In another closed chamber, the eyeball, venous pulsation is by no means infrequent. With such pulsations we are not directly concerned, and we may further limit ourselves by a passing reference to Quincke's "penetrating pulse," a pulsation transmitted through the capillaries of the periphery and appearing in the veins. Such a pulsation was recorded, as you may remember, by Claude Bernard as a result of stimulation of the chorda tympani. It is occasionally met with in pathological conditions; thus, in aortic disease it may be perceptible in the dorsal veins of the hands and is accompanied by pulsating capillaries.

The pulsation with which we must deal more fully is that of centrifugal origin, and it is due directly to the influence of the contracting cardiac chambers on the blood flowing into the heart. In man, pulsation of the veins is most readily observed in the neck, and in this situation it is such an obvious phenomenon in many pathological conditions that it must have been recognized for centuries. References to it can, indeed, be found in the writings of Lancisi and Morgagni during the early and middle years of the eighteenth century.

As a normal event the pulsation which we are discussing appears to have been first recognised in the dog. It was in this animal that John Hunter described it in 1793. Following upon Hunter's observation, others noticed its existence in the main tributaries of the heart in animals, and it is now well known as a normal event in dogs, cats, rabbits, and other animals, and may be seen in some extending to the smaller veins of the neck and limbs (Gottwalt).

Potain recorded the venous pulse graphically in his own neck and in that of his sister in 1867; and Gottwalt stated in 1881 that it is probably a constant event in all persons. This statement is receiving continual confirmation; recently Wenckebach writes that it may be recorded in at least 90 per cent. of cases, normal and pathological, an estimate in which many others would agree.

Since Potain made his contribution the writers on the subject have been numerous, and we need not wait at the present time to determine in detail the relative merits of the observations of these authors. Suffice it to say that there was little outcome of practical value until Dr. James Mackenzie elaborated the method of graphic representation and introduced to clinicians a reliable and practical instrument by which records could be won. The stimulus imparted by the new pulse work has created a new epoch in cardio-vascular work, which is little short of revolutionary, and the significance of which, it is hoped, will soon be apparent to you.

(b) THE METHOD OF OBTAINING TRACINGS.

Although a record may be obtained from the liver when the latter is pulsating, and occasionally, it is said, from the femoral vein, yet the majority of the work is done, and always will be done, upon the veins of the neck; for here the pulsation is most constantly present, and is most readily accessible. The anatomy of these veins has recently received special attention from Keith. A little above the points where the internal jugular and subclavian veins meet and join, the blood stream passes through a pair of valves on each side before it enters the innominate. That portion of the jugular vein which lies below the valves is known as the "jugular bulb," a name derived from the swelling in the vessel produced at this

point when the entrance of blood to the auricle is no longer free. The bulb, viewed from the front, with the patient supine, lies between the heads of the sterno-mastoid muscle, and directly anterior to the subclavian artery. These relationships are of importance, for it is from the bulb that most of the tracings are taken, and, as will be subsequently seen, the arterial pulsation may be, and often is, transmitted to a receiver placed over the vein.

The right vein has a shorter and more direct course from neck to auricle than has the left vessel, and consequently presents as a rule a more suitable field for observation. Further, the venous pulse is generally, and naturally, more prominent when the vein in which it is observed is distended with blood, and therefore tracings are best obtained with the patient supine and the head low. Frequently it may be necessary to request the patient to hold the breath, for then a tracing is obtained which is free from the complication of respiratory effects. Respiration should be suspended in the expiratory position, for during this act the filling of the veins is as a rule increased and the pulsations are maximal. The only further precaution, which it is necessary to take, lies in the position of the head. It should be inclined slightly towards the right shoulder, and the chin should be moved passively to the left. By this procedure the sterno-mastoid is relaxed and access to the bulb is easier. Under these circumstances, and with suitable apparatus, difficulty in obtaining venous records is rarely experienced.

The Apparatus.

The apparatus now in use is simple and effective. That which is most suitable for clinical purposes is Mackenzie's polygraph fitted to a modified Dudgeon sphygmograph. It consists in the main of a small receiving capsule, which is connected to a delicate tambour carrying a writing style. With the combined instrument double records are easily obtained; one of the radial pulse, the other from the jugular bulb, carotid artery, or apex beat. At present the apparatus is deficient in two respects: it allows of no time record, and the tracings are limited to a length of 1 ft. or $1\frac{1}{2}$ ft. These defects do not exist in the ink polygraph¹; but this instrument gives tracings which are less delicate and it is more cumbersome.

(c) GENERAL CONSTITUTION OF THE VENOUS PULSE, AND THE RELATION OF VENOUS RECORDS TO OTHER METHODS OF EXAMINING THE HEART.

It is important at the outset to recognize the general constitution of the venous pulse. It is in the main a curve of change of volume in the great veins and is dependent upon the pressure changes in the right auricle. Consequently in its general outline the venous curve is a duplicate of right auricular pressures. That it should be identical with the curve of auricular pressures is not to be expected, for the record is taken in a different manner and in a different situation. Thus there is every reason to believe that its separate events occur later than do the corresponding events in the auricle, and that during transmission they undergo minor alterations. Further, the tracing in the neck is complicated by the proximity of pulsating arteries. But, as we shall see, such minor differences do not appreciably affect the value of the record, nor do they, as a rule, hinder us from believing that the venous pulse gives in man data for estimating approximately the onset of auricular contraction in relation to other events of the cardiac cycle.

Now although this information is, for practical purposes, the most important obtained, yet it is by no means the sum total of the knowledge which it is possible to derive from this source. For the time being, however, it may be well to fix our attention upon the single fact that we have at our disposal the means of estimating the commencement of auricular systole. From the pulsation in the carotid or from that at the apex beat information is at hand as to the cycle of the left ventricle. As it can be shown in the majority of instances that the two sides of the heart—both auricular and ventricular portions—are so synchronous in their movements that they may be considered absolutely synchronous for all practical purposes, the two sources of information—arterial and venous pulse—give us all that we require for a full analysis of the separate movements of the cardiac

¹ Mackenzie, BRITISH MEDICAL JOURNAL, i, 1908, p. 1411.

chambers. Such an analysis may be amplified and checked in several ways. Pulsation in the epigastrium is a by no means infrequent occurrence, and it consists essentially of an inverted cardiogram of the right ventricle. Movements of the left auricle may be observed as may those of the right chamber by means of a fluorescent screen; they may be recorded also by passing an oesophageal sound, a rubber catheter over which a small balloon is tied. For the left auricle lies in apposition to the gullet. As a routine practice such a method is necessarily impossible, but by its occasional use it may serve as a useful check, and the information obtained from records of this character are not without value. Further it frequently happens that cardiograms taken from apex or epigastrium give distinct indications of the increase in size of the corresponding ventricle at each auricular systole; and again, in the case of defective chest wall, thin chest wall, retracted lung, or dilated auricle, in all and each of which conditions the auricle is rendered more accessible, direct tracings may be obtained from this chamber. Such are the main sources of information, obtained by graphic records, of pressure or volume changes in the separate cardiac chambers, and taken as a whole they are of sufficient accuracy to afford valuable material in the elucidation of the condition of the heart's action in any given instance. They have been supplemented, and will no doubt be further supplemented, in two directions. First, by a study of electrical changes occurring in the body as a result of systole of the cardiac chambers, records of which indicate separate changes of potential in the case of auricle and ventricle, and allow us to deduce the onset of contraction of either chamber (for the recent work, see Einthoven); and, secondly, by careful attention to the cardiac sounds, normal or pathological. The cardiac sounds may be recorded by photo-electric methods, and it is to be hoped that the further development of this branch of cardiovascular study will yield a fruitful store of information. (For recent work, see Weiss and Joachim.)

It is now possible to appreciate in some measure the value of the combined and simultaneous use of two or more of the methods which have been briefly scanned, and the whole foundation of this subject resolves itself into the interpretation of records obtained by one or other method and the comparison of simultaneous events in each. The value of the methods—I shall deal fully only with those consisting of graphic records of pulsating areas—lies in their application to disordered movements of the cardiac chambers.

Until recent years pulse irregularities have received little or no rational analysis, but with the introduction of the new methods we know that they are due in the majority of instances to disorder of the rotation of contraction of the cardiac chambers.

Of all the methods employed in investigating the cardiac cycle, that which yields a record of the venous pulse in association with the arterial has, up to the present, added most to our knowledge; and, before proceeding further, it is necessary that this venous pulse should be thoroughly understood.

(d) CONSTITUTION AND ANALYSIS OF THE NORMAL VENOUS PULSE.

The constitution of the venous pulse is best appreciated if the events of the auricular pressure curve are first considered. In animals the auricular pressure cycle may be obtained by introducing a sound, fitted at its end with a small rubber balloon, by way of the jugular vein into the right auricle, and connecting it to a recording tambour. The pressure reading so obtained has received careful investigation at the hands of Chauveau and Marey, Fredericq, Frey and Krehl, Porter, and others. The curves given by Porter are perhaps as much in accord with the general evidence as any, and it will be found convenient to take his curves as typifying the changes in auricular pressures. In common with many others, Porter found that the pressure curve shows, in the main, three elevations and three depressions, which we may term first, second, and third positive waves, and first, second, and third negative waves respectively.

It will suffice at present to examine the relationships of these waves to those of ventricular pressures. In the accompanying diagram you will observe two curves, one of auricular and one of ventricular pressures. Simultaneous

records show that the first positive wave of the auricular curve is presystolic in time, and that the rise of ventricular pressures is synchronous with the second positive wave of the auricular curve. The large depression (the second negative wave) occurs during the plateau of ventricular

systole and terminates somewhere near the end of the same. The third positive wave begins at the same point and ends in a sharp depression corresponding to a point near the end of the ventricular downstroke, where the tricuspid valves open.

We may now pass to certain differences of opinion which have an important bearing on the interpretation of the venous pulse. The tracings of Frey and Krehl do not show the second positive wave on the auricular curve, and it has been thought by some to be an artifact upon the tracings in which it does occur. It has been recorded, however, in many ways and in both auricles. Thus, Porter used an open cannula tied into the auricular appendix, and also registered the wave in the pulmonary veins. It has been registered in the lower end of the superior vena cava (Rautenberg), and in curves taken in animals and man by means of an oesophageal sound. We are probably justified in assuming that it is usually a real event, but that occasionally it is too insignificant to be recorded. The second point is the time relation of the third positive wave. There are those who hold that it commences during the plateau, as here figured, and others who consider that the rise begins at the end of the plateau at the point at which the semilunar valves close. It is probable that its relationships are not entirely constant, and that both series of observations are correct.

We may now examine the curve of the venous pulse and see how its relationships stand to the two curves which have been considered. Like the auricular curve it consists of, in the main, three elevations and three depressions, and for the time being it will be well to recognise the three main elevations of the venous curve (the *a*, *c*, and *v* waves of Mackenzie) as corresponding to the three positive; and the three depressions (the *x*, *x'*, and *y* dips of Mackenzie) as corresponding to the three negative waves of the auricle. These events may be tabulated as follows:

Vein.	Auricle.	Vein.	Auricle.
<i>a</i> ...	First positive wave.	<i>x'</i> ...	Second negative wave.
<i>x</i> ...	First negative wave.	<i>v</i> ...	Third positive wave.
<i>c</i> ...	Second positive wave.	<i>y</i> ...	Third negative wave.

The majority of writers are agreed in timing the commencement of the *c* wave as synchronous or almost synchronous with the primary wave of the carotid in the neck. That they are absolutely synchronous is open to question, but it may be safely stated that for practical purposes the time difference in the instants of onset is negligible. Now the instant of onset of the primary wave of the carotid is later by about $\frac{1}{100}$ second than the instant of onset of ventricular systole. Consequently the onset of the *c* wave in the neck occurs later than the second positive wave of the auricular pressure curve by about the same interval. In the neck the *a* wave starts normally $\frac{1}{2}$ second earlier

than the *c* wave, and this interval is surprisingly constant. In the auricle the first positive wave precedes the second positive wave by probably not much more than $\frac{1}{10}$ second. There is thus reason to believe that the *a* wave in the neck is of somewhat longer duration than is the first positive wave in the auricle, though the differences in the time relations can only be stated approximately. In regard to the *v* wave, which corresponds to the third positive wave, there is greater divergence of opinion, and this is due in part to its variation in form and onset from subject to subject. There are writers who hold that it commences during ventricular systole, others believe that the rise occurs as a purely diastolic event—namely, subsequent to the closure of the semilunar valves (Gerhardt, Wenckebach). The detailed evidence is too intricate for us to consider, and it will be wise for us to regard it at present as subject to variation. It is probable that it occurs later by about $\frac{1}{10}$ second than the corresponding wave in the auricle (Young and Hewlett), and that, like the latter, it is sometimes early and sometimes late in onset.

We are now in possession of the main facts, and may proceed to the detailed analysis of the several waves.

The "a" and First Positive Wave.

The wave in the auricle is synchronous with auricular contraction (Porter). It is presystolic in time in both jugular and oesophageal tracings. It is universally attributed, and with good reason, to auricular systole. The evidence is incontestable. The time relations of the wave alone are sufficient to indicate its origin. If the auricle be tetanized it ceases to contract in the ordinary way, and the wave is no longer seen. On the other hand, if the ventricle is subjected to faradism, the wave persists.

In the auricle it is a pressure wave; in the veins it is probably a wave of increased pressure and volume. There is no conclusive evidence of regurgitation into the neck veins during auricular systole in the normal subject. For, though there is probably a limited retrograde movement of blood in the veins immediately adjoining the heart (François-Franck, Chauveau), yet such a centrifugal current has failed of demonstration in the neck (François-Franck). It is probable that the backward movement of blood is largely controlled by the contraction of the taenia terminalis, which has anatomical relations suggesting its power of completely closing the mouth of the superior vena cava (Keith). The wave in the neck is consequently attributable to a retrograde pressure wave or to stasis, and the extent to which the two factors participate in its production is at present uncertain.

The "c" and Second Positive Wave.

The discussion which centres around this wave is intricate and involves some very nice questions. The position is briefly as follows. That the wave is ultimately attributable to ventricular systole is certain, for it vanishes when the ventricle ceases to beat. Whether it occurs in the auricle, superior vena cava, or jugular, its onset is almost, if not in most cases quite, synchronous with a rise of pressure in an adjoining structure. In the case of the auricle the synchronous rise of pressure is in the ventricle. In the case of the cava and jugular the wave is synchronous with the rise of pressure in aorta, innominate, subclavian, or carotid, as the case may be. The questions which arise for discussion are consequently numerous. First, we have to inquire whether there is an actual rise of pressure in the auricle. We have seen that the evidence is in favour of it. Secondly, we have to ask as to what such a wave in the auricle may be due. Upon this there is little or no discussion, for those who acknowledge its presence have little hesitation in attributing it to the outward bulging of the auriculo-ventricular valves, the hardened dome of which has been felt in the horse during systole by introducing the little finger into the auricle (Chauveau and Fredericq). Thirdly, we have to consider the possibility of transmission direct from aorta, innominate, subclavian, or carotid to cava, innominate, or jugular. The venous and arterial paths are in close apposition throughout, yet this in itself should not be allowed to have any great weight. For the actual expansion of an artery in systole is very slight; its increase in diameter has even been denied. The change in the capacity of the vessel is probably chiefly induced by change of shape. It is stated, that though the main vessels of the abdomen, arterial and venous, are

in close proximity throughout, yet the wave as a rule is not transmitted so as to permit of its being recorded in the femoral vein. Let the possibility that the arteries may transmit a pulsation to surrounding structures be acknowledged. It cannot at present be denied; yet it cannot be stated as a factor of importance in the connexion in which we are considering it. If such conduction does occur it must be slight. The pulsation or *c* wave, which has been recorded in both cava and jugular direct, cannot be attributed in its entirety to arterial transmission. If artery and vein be separated in animals, in a certain proportion of them at least the venous wave persists, and the same happens after clamping the artery as low down as possible (Fredericq and Morrow). Again, observations have been made, of a more exact nature than can usually be carried out at the bedside, with a view to determining whether the arterial pulsation in the neck is precisely synchronous with that in the vein at a corresponding level. It would appear from Fredericq's experiments on the dog and Bard's on the human subject that they are not necessarily so, observations which, if fully confirmed, would have an obvious bearing on the question under discussion. There can be no question that if the second positive wave actually indicates a rise of auricular pressure, such a rise of pressure might, and probably would, be communicated to the veins, and might be carried along these veins to a considerable distance. The *c* wave has been recorded by Rautenberg as part of the curve yielded by the liver pulsation; but the complex nature of these curves would incline us to assign little importance to the fact, more especially as we know that, as a rule, the *c* wave is absent from such tracings.* So far as we have been able to examine the evidence, it would appear reasonable to assume that the *c* wave, which occurs in the veins of the neck, upon which no pressure is exerted, is in the main the result of conduction from the right auricle, and that it may be due in some slight measure, perhaps, to pulsation transmitted from aorta, innominate, or carotid artery to adjacent venous paths. But when a receiver is applied closely over a relatively large area of skin the position is different. A record is taken not from the vein but from the neck, and there is little difficulty in obtaining by this means an arterial curve when the receiver lies over the carotid. Moreover, within certain limits, the more the receiver is pressed into the neck the more powerfully is the carotid felt to pulsate, and the more readily is a carotid tracing obtainable.

Thus, while we are probably justified in stating that the main part of the wave in the jugular of the undisturbed neck is of auricular origin, yet, with the application of the instrument for recording, the conditions are altered and must be very variable. In certain instances the difficulty—nay, impossibility—of avoiding the arterial pulsation, in attempting to obtain pure venous records, is well recognized by all who have even a slight acquaintance with venous pulse work, and this applies particularly to necks in which arterial pulsation is easily visible.

We may conclude that, in the record obtained from the human neck, the *c* wave is in part a wave transmitted from the auricle and in part a wave collected by the receiver from carotid or subclavian. To what extent the two factors participate in the full result it is not possible to say, but we can be quite sure that from case to case the participation of each will be of unequal relative amounts. Further, it is probable that the artery will tend to contribute most when pressure on the neck is greatest, and that to obtain the purest venous records, the lightest possible pressure compatible with closure of the mouth of the receiver should be employed.

Before leaving the consideration of this wave, it may be well to repeat, that whatever its direct derivation, it is eventually due to systole of the ventricle. And that when the neck curve is regarded as an expression of auricular pressures, the *c* wave represents the onset of ventricular systole. It consequently forms a valuable landmark in the curve, and the wave which directly precedes it must depend for its origin upon a presystolic event.

*The usual absence of the *c* wave from the liver pulse is the strongest argument for its causation by transmission from arterial structures; there is a possibility that Rautenberg's single tracing was taken too near a pulsating aorta. Its absence upon the liver tracing is so inexplicable as to necessitate caution in attributing it, as it occurs in the vein, mainly to transmission from the auricle.

The "v" and Third Positive Wave.

The chief difficulty in interpreting this wave lies in the conflicting evidence as to the moment of its onset. The explanation rendered by any given writer depends upon the time relations which he accepts for its various phases. Thus those who believe in its onset during systole consider that its chief cause lies in the obstruction to the entry of blood from auricle to ventricle. In short, that the blood in the auricle accumulates and produces a stasis wave in the veins. Those, on the other hand, who regard it as arising at the commencement of ventricular diastole, recognise it as caused by the rebound of the auriculo-ventricular ring. There is a good deal of evidence that such rebound occurs during diastole. The movements of the ventricle in systole and diastole have long been the subject of interest and research. The subject is a peculiarly difficult one, for there is little question that alterations of pressure on the superficies of the heart may alter the direction of movement of any point in the musculature very considerably. It consequently becomes of great importance that the chest wall should remain intact during the performance of experiments which have a bearing upon the subject. For many years it has been recognized that with the chest open the junction of auricles and ventricles moves downwards during systole, and that a corresponding upward movement occurs with the relaxation of the muscle walls. The same appears to be the case with the chest walls intact. Brücke and Haycraft, experimenting separately, used an ingenious method. They passed needles, carrying straws at their free ends, through the chest wall into the musculature of the heart, and by observing the movements of the straws were able to determine that of all points in the musculature that which constitutes the apex is most stationary, and that all other points move towards it and towards the middle line during systole. Keith has recently given close attention to the arrangement of the muscle fibres and to those structures with which they are connected, which may be considered as fixed points or origins. His conclusions, based in the main on anatomical arguments, are in agreement with previous work. Like Chauveau, he recognizes that the movement of the auriculo-ventricular septum in systole is considerable, and that, as it is drawn down towards the apex or fixed point, the auricle is expanded as is a bellows. With diastole of the ventricle the movement is reversed, and the ring is supposed to return to its original position. It may consequently be stated that the view that the third positive wave is due to the upward movement is a feasible one, so long as it is recognized that this wave is diastolic. The explanation attributes the wave to a movement of the ventricle. The direct evidence bearing upon this aspect of the question is conflicting; for, while Fredericq finds that fibrillation of the ventricle abolishes the wave, Morrow gives a tracing in which some portion of the wave remains, and therefore is the more inclined to attribute it to stasis. It is probable that both factors play a certain part in the production of the wave under certain circumstances, and if this view is accepted it will explain not only the disagreement of the observations just quoted, but will also account for the variation in the time of onset of the wave; it is further supported by the occasional division of the wave into two. Where ventricular systole is prolonged and slow, stasis will tend to play the greater part, while where it is sharp and quick the rebound of the ring may be supposed to be the dominating factor.*

The "x" and "x'" Depressions and First and Second Negative Waves.

These may be considered together, for the line of descent is continuous in direction, but is interrupted by the c or second positive wave. The depression commences with relaxation of the auricle and continues for a variable period. Either it ceases during the systolic plateau or at the commencement of diastole. The fall is a very conspicuous one, and is in fact the chief fall during the cycle. It is accompanied by the notable collapse of the veins, which received close attention by the older writers. Hunter, Brücke, Mosso and others were inclined to attribute it in part, if not in whole, to ventricular systole, supposing it to be caused by the increased vacuum in the chest at the

* It is also possible that a certain degree of regurgitation from the ventricle aids in the production of the wave (Mackenzie).

expulsion of the left ventricular contents. This explanation can no longer be regarded as of importance. Unquestionably the factor may be regarded as a supplementary cause, but that it is not the main cause is distinctly shown by the fact that opening the chest does not abolish the wave (Gottwalt, Riegel, François-Franck, and Fredericq). Active relaxation of the auricle has never been demonstrated, and, as François-Franck stated, relaxation must be regarded as due to a return of the auricle to its original state, as a result of the influence of intrathoracic pressure. That the depression is due in some measure to an auricular event is shown by its remaining to some extent on clamping off auricle from ventricle, and by its presence in part when the ventricle is no longer beating. At the same time the extent of the depression is so great that relaxation of the auricle is insufficient to account for it. A new factor must consequently be sought. It is necessary for you to recall the evidence and conclusions respecting the movements of the auriculo-ventricular line, and the view which has been revived by Keith, that the filling of the auricle is in the main dependent on ventricular systole. It is during the latter part of the depression which we are considering that the diminution in length of the ventricle is occurring; and Fredericq observed that during this ventricular systole the auricular appendix diminishes in volume, and is, as it were, aspirated towards the cavity of the auricle. According to this view, the main depression of auricular pressure is brought about by an expansion of those portions of the auricle which lie between the descending ring and the fixed mouths of the great veins, and is dependent almost entirely on ventricular contraction. The explanation balances our views of the causation of the whole auricular pressure curve. If any event produces a wave of a particular sign, when that event ceases or is reversed a wave of opposite sign should be produced. We attribute the rise of the first wave to auricular systole, its descent to auricular diastole, and the continuation of the descent to the downward movement of the auriculo-ventricular line. Lastly, the upward movement of the same line accounts for the ascent in the third positive wave, in part at least.

In conclusion, it may be stated that so far as present evidence goes, the large depression is due to a combination of three factors. The increased negative pressure in the chest consequent on the expulsion of the ventricular blood, the auricular relaxation due to the original intrapleural pressure, and the stretching of the auricular walls by virtue of contraction of the ventricle. Of these factors the first is of little consequence; the second is most active during the earlier phases; and the third most active during the later phases of the depression.

The "y" Depression and Third Negative Wave.

We have seen that during the systole of the ventricle, auricular pressure falls, and that towards or at the end of it, once more commences to rise. This rise of pressure must necessarily terminate early, for as the ventricle passes into diastole the blood flows into it so soon as the tricuspid valves open. Now the curve of intra-auricular pressure shows a sharp decline at or about the instant when the auriculo-ventricular valves open, and this is the depression which we are considering. The exact moment of its onset is probably variable within at least $\frac{1}{10}$ second, a time interval which is negligible in pulse work. Porter found that the fall commences earlier than the opening of the tricuspid valves, at a point more nearly approaching the closure of the semi-lunar segments. It must be borne in mind that the interval between the movements of the two sets of valves is a very short one, probably shorter than the error in human pulse work. The time relation given by Porter is perhaps more in conformity with the views previously expressed than that stated by many other observers—namely, the commencement of the fall with the opening of the tricuspid valves. For if the upward bulging of the tricuspid valve segments produces the second positive wave, when the pressure from below is relieved the segments must relax, and before they actually open the pressure in the auricle should fall. The main fall is due, however, to the filling of the ventricle as it rapidly enlarges, thereby relieving the auricle of the blood which has collected in it.

We may be certain that the fall of auricular pressure is due to ventricular diastole, for it is abolished by hindering

the ventricular movements. Finally, there is reason to believe that in the neck the event is delayed, as are the other waves and depressions of the curve.

BIBLIOGRAPHY.

- Bard: *Journ. d. Physiol. e. d. Pathol. gén.*, 1906, p. 454; *Archiv. d. Maladies d. Cœur*, 1908, No. 6.
 Brücke: *Vorlesungen über Physiologie*, Wien, 1885, Bd. 1, Aufl. 4, S. 178.
 Chauveau and Faivre: *Gaz. Méd. de Paris*, T. 8, 1856, p. 410.
 Chauveau and Marey: *Ibid.*, 1861, p. 673.
 Einthoven: *Archiv f. d. ges. Physiol.*, Bd. 122, 1908, S. 517.
 François-Franck: *Gaz. heb. de Méd. e. d. Chir.*, February, March, April, 1882.
 Fredericq: *Archiv. d. Biol. (Paris)*, T. 8, 1888, p. 497; *Archiv. Internat. d. Physiol.*, v. 1907, p. 1.
 Frey and Krehl: *Archiv f. Anat. u. Physiol.*, 1890, S. 31.
 Gerhardt: *Archiv f. exper. Path. u. Pharmak.*, Bd. 34 and 47.
 Gottwalt: *Archiv f. d. ges. Physiol.*, Bd. 25, 1881, S. 1.
 Haycraft: *Journ. of Physiol.*, vol. xii, 1891, p. 452.
 Hering: *Archiv f. d. ges. Physiol.*, Bd. 106, 1904, S. 1.
 Hunter: *A Treatise on the Blood, Inflammation, etc.*, London, 1794, pp. 185 and 187.
 Keith: *Journ. Anat. and Physiol.*, vol. xlii, 1907, p. 1.
 Lancisi: *De Motu Cordis*, etc., 1740.
 Mackenzie: *Journ. Path. and Bact.*, 1892, p. 53; *ibid.*, 1894, p. 84; *Amer. Journ. Med. Sci.*, July, 1907.
 Morgagni: *De Sedibus et Caus. Morbor.*, Napoli, 1762.
 Morrow: *BRITISH MEDICAL JOURNAL*, December 22nd, 1906, p. 1807.
 Mosso: *Die Diagnostik des Pulses*, 1879, S. 60.
 Porter: *Journ. of Physiol.*, 1892, p. 513.
 Potain: *Mém. d. l. Soc. Méd.*, Paris, 1867, p. 3.
 Rautenberg: *Zeitschr. f. klin. Med.*, Bd. 65, 1908; *Berl. klin. Woch.*, 1907, No. 46.
 Riegel: *Deutsch. Archiv f. klin. Med.*, Bd. 31, 1882, S. 1.
 Weiss and Joachim: *Archiv f. d. ges. Physiol.*, Bd. 123, 1908, S. 341.
 Wenckebach: *Archiv f. Anat. u. Physiol.*, 1906, S. 297.
 Young and Hewlett: *Journ. Med. Research*, vol. xvi, 1907, p. 427.

A CASE OF ADAMS-STOKES SYNDROME OBSERVED FOR MORE THAN EIGHT YEARS.

BY

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GENERAL HISTORY.

T. A., male, aged 70, was formerly a postmaster; on his retirement from the service about ten years ago he devoted himself on a rather large scale to gardening. Formerly he was accustomed daily to walk to his work at the General Post Office and to return up a stiff hill in the evening, a distance each way of about three miles. The sudden change from his former active life and from his daily walk has had in our opinion considerable influence on his present condition. Until his retirement he enjoyed good health, with the exception of some moderately severe attacks of bronchitis, and occasional attacks of dyspepsia, induced, probably, by eating too much and too quickly.

HISTORY OF THE ILLNESS.

The patient was first seen in 1899, when he consulted one of us for some digestive troubles. No notes of the case at that time were taken, but the general lack of tone of his system, the flabby, distended state of his abdomen, and the weak action of his heart were obvious. It was necessary to make him walk up and down the room several times before it was possible clearly to hear his heart sounds. Nothing abnormal in the rate of the pulse attracted attention. More exercise was recommended, and he was given directions in regard to his diet.

He was next seen on September 18th, 1899, when he had had a sudden fainting attack whilst showing a friend round his garden. This was merely a fainting attack, unattended, so far as could be ascertained, by any convulsive movements. No notes of the case were taken at this period, but so far as can be recollected there was nothing remarkable about the pulse-rate.

Nothing worthy of note then occurred until July 11th, 1900, when the first of a series of the convulsions took place. These were of an epileptiform character, lasting from a few seconds to three or four minutes; for two or three days they followed one another so frequently that no record was kept of their frequency, and the patient was practically in a status epilepticus. On account of the frequency of the attacks no nourishment could be administered by the mouth, nutrient enemata and suppositories were con-

sequently administered. During this attack hope of the patient's recovery was practically given up. After this date he had no attacks of similar frequency or severity; the seizures were for the most part fainting attacks or giddiness.

Attacks twice occurred when the patient was allowed to go to the w.c. by himself; during one of these he fell and severely cut his face and lip. Later mild attacks of short duration were induced by taking food; they supervened when he took his first few mouthfuls of food, and seemed to be due to a dilatation of the portal veins and a draining thither of the blood from the cerebral vessels. After the first few mouthfuls of food he could finish his meal free from attacks. Micturition, which was performed in the recumbent position, also frequently brought on a convulsion. Another point of interest was that the patient found that, by grasping tightly the head of the bed, he could frequently ward off an attack. His hands have often been noticed discoloured by the paint from the iron work of his bedstead from his adopting this precautionary treatment.

THE PULSE.

The accurate records of the pulse-rate kept by the patient himself form one of the interesting features of the case. For just over eight years the pulse-rate was charted thrice daily, oftentimes more. The records begin on July 25th, 1900, some few days after the series of convulsions; the rate was then 38 to 40. On July 26th, 1900, it became quicker, about 53 to 55. From that date on it ranged about 60, never being below 50 and never above 72 till September 21st of the same year, when a rate of 40 was recorded. On October 1st a pulse-rate of 32 was recorded, and from that date the rate was for the most part between 30 and 40, except for intervals of two or three weeks, when it was 50 to 60. From October 31st, 1901, it was below 40, except for an isolated observation on July 22nd, 1904, when it was 68 after an attack of convulsions. During this period up to his death the rate was never observed below 16—that is, apart from convulsive attacks—and seldom above 37. The variations in the pulse-rate is graphically recorded over a period of one month during November, 1900, in Fig. 1.

During all the period of observation the pulse had the habit of dropping beats. It was more marked when the pulse-rate was quicker, but with the slow rate, though not perhaps so frequent, it was of more consequence to the patient, for when the pulse dropped beats at the slow rate the patient was always more liable to an attack. In fact the patient himself informed one of us that if his pulse missed one beat, an occurrence of which he himself was conscious, it made very little difference to him, but if it missed two beats in succession, it was often very hard work to remain conscious, and was often followed by an attack. The patient did best with a pulse-rate of about 25 a minute; if his pulse-rate rose above 30 he anticipated trouble; below 25 the pulse was steady and regular; when it reached or exceeded 30, it became intermittent or irregular.

A record of the radial pulse, the jugular pulse, and the respirations is given in Fig. 2. The analysis below shows that the auricle is beating at 72 per minute, the ventricle at 24. It is impossible to say from this record, as indeed also from the others that have been obtained at different times, whether complete heart-block is present or not, for the auricular rate is exactly thrice that of the ventricular.

TREATMENT.

Many drugs were tried, but all failed to give any marked relief, with the exception of large hypodermic doses of strychnine (m̄viii to m̄xv of the liq. strych., B.P.), and small doses (3 j to 3 ij) of brandy frequently administered when the attacks were more than usually severe.

Raising the foot of the bed also proved beneficial at these times. A flannel belt or binder tightly applied to the extremely flabby and pendulous abdomen gave no relief. Extreme flatulency was a frequent precursor of an attack, consequently various measures were taken by means of drugs and strict dieting to check its occurrence. Drugs, however, proved useless in this respect.

MODE OF LIFE.

The patient for the most part stayed in bed till an hour after his midday meal, he was then wheeled round his