THE CARDIOPNEUMATIC MOVEMENTS.

By JOHN BERRY HAYCRAFT, M.D., AND ROBERT EDIE, M.B.

(*From the Physiological Department of the University of Edinburgh.*)

IN addition to those great respiratory movements of the chest, which are due to the alternate expansions and contractions of its cavity, there are superadded movements, comparatively insignificant, which seem to depend for their production upon the circulatory mechanism.

Historical. C Voit[1], as long ago as 1865, found that on closing the nose, keeping the glottis open, and holding the breath, the air within the respiratory passages moves to and fro in tiny expiratory and inspiratory puffs alternating with one another. He timed these with the heart-beats, and observed that each inspiratory motion coincided in point of time with a cardiac systole, each expiratory puff with a cardiac diastole. He concluded that these movements are due to changes in the size of the heart, which organ diminishing in size during its systole, draws air into the chest-cavity, and, on the other hand increasing in size at diastole, forces air out of the chest.

Soon afterwards Ceradini[3] confirmed Voit's observation, and demonstrated the phenomena by means of a glass tube filled with tobacco-smoke and held between the lips. With the breath held and the glottis open he observed the smoke rising and falling in the tube synchronously with the circulatory movements. He also took tracings; but these were never published.

The condition in which the heart assumes its smaller dimension Ceradini termed Meiokardie (*μειοῦν*, to make small, and *καρδία*), and the condition in which it assumes its greater bulk Auxokardie (*αὐξάνειν* to increase). But Ceradini pointed out that the influence exerted upon the air within the chest cavity could not be accounted for by the difference between these two conditions. For, although the heart may diminish in size at each systole (condition of Meiokardie), yet not more than half the blood which then leaves it, will leave the chest cavity;

the remainder will merely be displaced into the pulmonary circulation. Further he pointed out, that during the cardiac systole, blood is entering the chest by the veins, compensating in some degree for the diminution in size of the heart and loss of arterial blood from the chest.

In a subsequent paper Ceradini(4) pointed out that the inspiratory movement of air cannot exactly coincide in point of time with the systole of the heart, because at the beginning of contraction, the blood from the heart is merely displaced from its cavity to the big arteries within the chest, any diminution in its volume being compensated for by an equal increase in their size. It is only after a short but appreciable interval that the blood is displaced into the extra-thoracic region; and at this moment, said Ceradini, the inspiratory movement occurs; while he goes on to say that immediately following is an expiratory movement due to the rebound of arterial blood to the chest, and to the fact that venous blood at this moment is rapidly pouring into the cavity and filling the expanding heart.

Landois(2) (whose observations were subsequently confirmed by Brunn) investigated the matter still more fully, experimenting both on human subjects and on dogs under the influence of curara. He demonstrated the cardiopneumatic movements by means of water manometers, and by the manometric flame; and at the same time by using a writing tambour he took careful tracings. His results were confirmatory of those of Ceradini, but his explanations of the phenomena rather more detailed.

At the time of the first sound—commencement of systole—he observed that the air was being driven from the chest, and he explains it on the assumption that blood is flowing into the chest while the accumulated arterial blood is still intra-thoracic; and further, he considers that the expiratory movement will be assisted by the dilating pulmonary vessels exerting a pressure on the bronchioles around which they anastomose. Rapidly following the commencement of the systole, a powerful inspiratory movement occurs, explained by him as being due to the fact that arterial blood is now leaving the chest much more rapidly than venous blood is entering.

At the moment of the second sound—when the semilunar valves are suddenly closed by the rebound of the arterial blood—an expiratory movement again takes place (vide Ceradini). Landois also enumerates several other factors which he considers influence the cardiopneumatic movements. For instance, the cardiac impulse, which

slightly pushes forwards the fifth costal interspace (so expanding the chest) will produce an inspiratory movement which will go to neutralise in some slight degree the faint expiratory movement which precedes the main inspiratory movement. Again, as the blood flows through the vessels lining the mouth and nose, these cavities are rendered smaller, just as he supposes the air-passages of the lungs are diminished by the dilating pulmonary vessels: and this will necessarily contribute towards an expiratory effect which will tend to neutralise the main inspiratory movement. He also takes into account the diminution in the bulk of the muscular substance of the heart during its contraction.

In the case of the pulmonary vessels which have been referred to as pressing on the air-passages when distended, we consider that it is doubtful whether the distension of these arteries immediately after the commencement of the systole may not expand the lungs, just as it was at one time supposed that the coronary vessels of the heart helped to distend that organ when they themselves were engorged with blood. And the same may possibly apply to the vessels of the mucous membranes of the mouth and nose; though it is more unlikely owing to the comparative rigidity of the walls of these cavities.

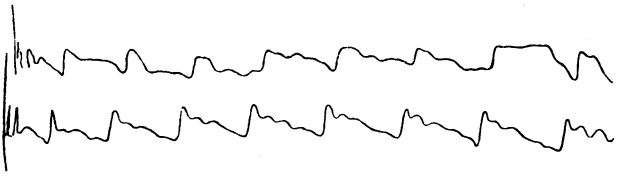

FIG. 1.

The lower curve is a tracing of the human carotid pulse. The upper is a tracing taken simultaneously with the lower one, and shews the pressure variations in the mouth, pharynx, and nose. A tube from the tambour which traced it was held by the lips while at the same time the nostrils and glottis were kept closed. Lines giving simultaneously formed points on the two curves occur at their commencement.

Fig. 1 represents two tracings taken simultaneously, the upper one of the air pressure within the nose and mouth, the lower one of the carotid pulse. The upper curve was taken by means of a Marey's tambour in communication with the mouth, while the glottis and the nostrils were closed. It indicates clearly enough a pressure variation and an expiratory puff synchronous with each carotid pulsation; and Landois is here perfectly correct. If however the lungs of an animal (the lungs we experimented with were those of the rabbit and sheep)

be removed from the body, the trachea brought into connection with a Marey's tambour and blood or normal saline solution forced in jerks through the pulmonary circulation, the lungs will be found to expand and draw the air in from the tambour in puffs.

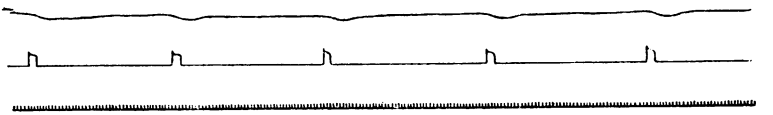

FIG. 2.

The upper curve is taken by a tambour connected with the trachea of a rabbit so as to record any variation in pressure within the respiratory passages. The curve falls on four occasions indicating that the respiratory passages are then expanded. These "falls" almost immediately follow peaks on the lower curve indicating when fluid was injected into the pulmonary artery of the animal.

Under the tracing (Fig. 2) the time is given when the syringe was discharged which is recorded by pressing upon a marking key at the moment of emptying the syringe. The tracing above, indicates the air pressure within the lungs.

It appears therefore in the case of the lungs, that during each cardiac contraction an *inspiratory* movement of air will take place. It is possible, however, that within the chest cavity this may not occur. If it does, the extent to which this takes place will depend upon the rigidity of the chest cavity. We cannot without experimental evidence decide the question, and that experimental evidence is not yet forthcoming.

Movements occur with the opened chest.

As already so fully pointed out, previous observers looked upon the cardiopneumatic movements as resulting from diminutions and expansions of the heart and blood-vessels within the closed cavity of the chest, these alterations of volume only taking place at the expense of the respiratory air within the cavity. But (as we were able to demonstrate on more than one occasion) they occur equally well with the chest cavity open and freely communicating with the outer air.

A rabbit was etherised and placed under the influence of curara, artificial respiration being kept up, and a side branch from the tracheal cannula was placed in communication with a Marey's tambour. On

stopping the respiration, opening the communication with the tambour and clamping the tube passing to the bellows, the cardiopneumatic movements were well seen, and tracings can thus readily be obtained on the revolving cylinder (Fig. 3, *A*).

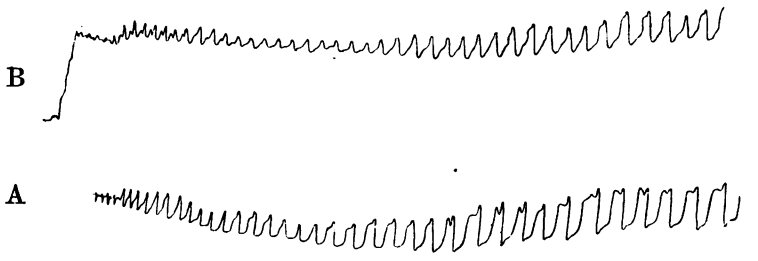

FIG. 3.

Two cardiopneumatic curves from the rabbit are here represented, the lower one *A* before, and the upper one *B* after opening the chest cavity. They are in every way similar. In both curves the amplitude of the curve increases from left to right, because the animal deprived of air was becoming asphyxiated, the heart becoming engorged with blood during each diastole.

If, after having taken a tracing, the artificial respiration be resumed, the abdominal cavity opened, the anterior attachments of the diaphragm cut away along with the anterior chest wall in its lower part, so that the cavity freely communicates with the external air, the cardiopneumatic movements may be seen as well as before as soon as the respiration is stopped (Fig. 3, *B*). It is obvious therefore that the hypotheses of Ceradini and Landois can hardly be accepted as explaining, at any rate, the chief movements to be observed.

Probable cause of the movements.

It occurred to us that probably the cardiopneumatic movements were for the most part simply due to the heart pressing against the lungs, and, that the lungs acted like an oncometer placed around it. The lungs which invest the heart will be subject to similar influences and the air within them will suffer variations in pressure similar to those within a cardiograph placed upon the chest wall.

As this action of the heart would still operate after opening the chest provided the heart remains clasped by the lungs, it appeared to us to be the only possible explanation of, at any rate, the chief movements. The proof of the soundness of our deductions was easily

obtained; for, on lifting the heart away from the lungs by means of a pair of forceps, the cardiopneumatic movements almost entirely ceased (Fig. 4), slight undulatory movements alone remaining, due no doubt to trifling alterations in the capacity of the respiratory passages owing to the periodic variations in the turgidity of the pulmonary vessels which occur with every heart-beat.

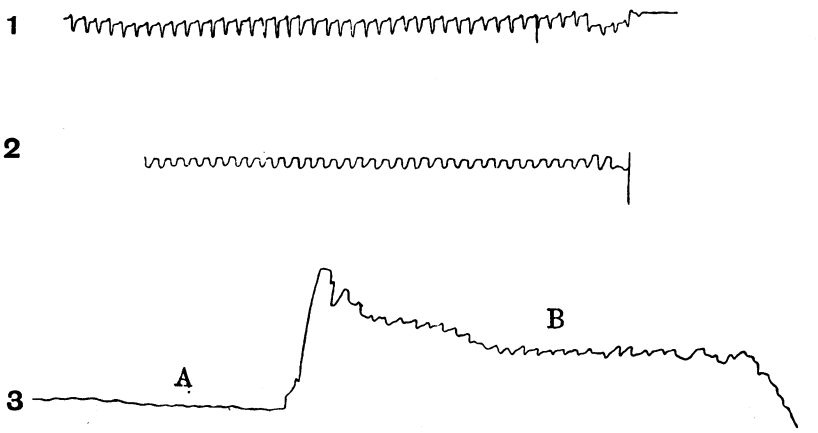

FIG. 4.

In figure 4 there are three tracings, the upper one (1) was taken from a rabbit with the chest cavity in its normal condition, and the tracing (2) after making a free opening into the cavity. Tracing (3) was obtained by lifting the heart (*A*) away from the lungs when the movements almost entirely ceased, and then allowing it to fall back upon the lungs (*B*) when they recommenced once more.

That the movements are still somewhat small in Fig. 4 after the heart has been allowed to fall back upon the lungs, is no doubt due to their displacement; for, whereas in the closed or partly opened chest they clasp the heart receiving the full effect of its every movement, when the whole chest wall has been removed, the pericardium opened and the heart pulled away from its position, the relation of parts has been disturbed and the lungs never come into their former intimate association with the heart.

These facts therefore clearly prove that the cardiopneumatic movements simply result from what we may term the "massage" of the lungs by the heart itself, and it becomes now a question of some interest to compare the cardiopneumatic with the cardiographic tracings.

Tracings compared with those of a Cardiograph.

If a cardiopneumatic tracing is compared with a cardiographic tracing taken simultaneously as in Fig. 5, it will be seen to be—in the

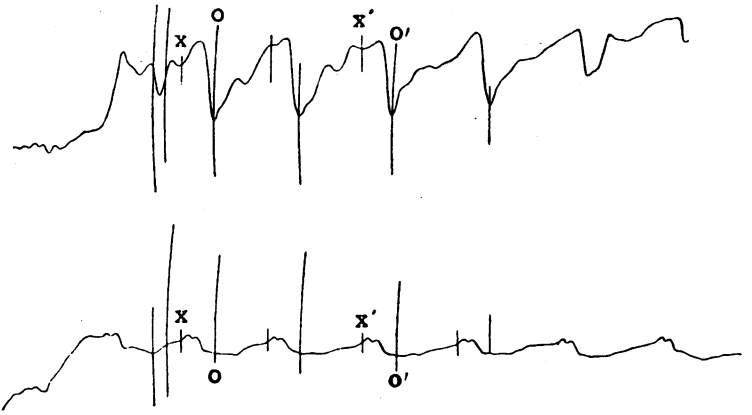

FIG. 5.

Two tracings taken simultaneously are here shewn, the upper one of the cardiopneumograph, the lower one of the cardiograph. The lines (x and x') in both curves run through points marking the commencement of the ventricular systole. In both curves the levers ascend and then fall to o and o' marking the end of systole, being the lowest point on the cardiopneumatic curve.

main—a simple cardiogram. In these tracings taken from the human subject the upper represents the cardiopneumatic movements, the lower a cardiogram taken by means of an ordinary Marey's cardiograph. The cardiogram is not a typical one, as neither of us possesses thin chest walls; but the commencement of the systole is sufficiently though only slightly marked (x'). It will be observed that as the heart changes in size it exerts pressure on the tissues surrounding it, causing nearly simultaneous changes both in the cardiogram and in the cardiopneumatic curve. If we begin our study of the curves at (x) we shall see that at the moment of the commencement of the ventricular systole both levers go up exactly together giving a slight but distinct elevation to both curves (Fig. 5, x and x'). Ceradini and Landois are right therefore in saying that a slight expiratory movement of air takes place at this time, though, obviously, their explanation is erroneous.

This expiratory movement occurring after x and x' corresponds then with the upstroke on the cardiogram, so evident in a more typical tracing taken from a person with thin chest walls. In a paper devoted to the study of the cardiac movements (7) the nature of this part

of the curve will be fully explained, we may here simply affirm that like the simultaneous upstroke of the cardiograph it is caused by the heart, which, yielding and flaccid during diastole, is distorted and pressed upon by both lungs and chest wall, and as soon as it passes into systole resents this distortion, pressing back upon either the lungs or the chest wall or both. After this slight elevation, not always present, the lever descends at once to its lowest point, during which the lever of the cardiogram descends as well. The explanation is very simple, for as the heart contracts and empties itself of blood, its walls recede from the chest wall in front and from the lungs behind. The cardiogram does not reach its lowest point quite as soon as the cardiopneumatic curve, the latter rising quickly after its main fall, and then more slowly to the point *x*. As however physiologists are by no means agreed as to the interpretation to be placed upon the cardiogram, it is superfluous at present to do more than indicate the general resemblance between these two tracings. The more detailed comparison, involving as it does a thorough analysis of the cardiogram itself, will be discussed in a future paper[1].

The cardiopneumatic curve represents the variations in the mean of the pressures exerted by the heart during a cardiac cycle, while the cardiographic curve represents the variations of the pressure at a definite spot. This is the interpretation that we put upon the curve, and, if it be correct, the cardiopneumatic curve will be the truer interpreter of the heart as a whole.

It will be noted that there are several tiny waves (two or three) on the cardiopneumatic curve during the latter part of diastole. These we

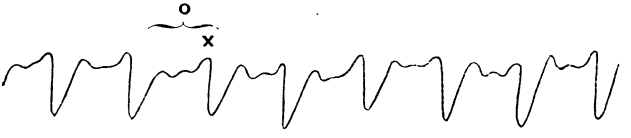

FIG. 6.

A cardiopneumatic curve. This curve shews the tiny movements of the lever below the letter *o*. These are evidently inertia oscillations following the previous displacement of the lever. The movement at *x* is however larger than those that precede it and is due to the commencing systole.

believe are simple oscillations, and this fact is especially well brought out in Fig. 6. After the displacement caused by a violent systole

[1] The nature of the Cardiogram is fully discussed in a paper by one of us in this vol. of the *Journal of Physiology*.

neither the heart nor lungs or registering apparatus will at once come to rest, and oscillations will occur similar to those seen when working with the sphygmograph etc.

Tracings compared with those of Carotid pulse.

In Fig. 7 are represented tracings, taken simultaneously, of both the cardiopneumatic movements and of the carotid pulse. The tidal

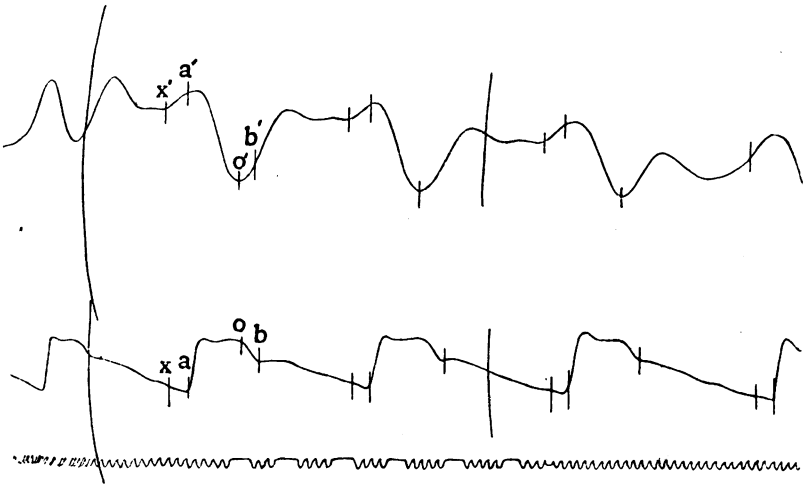

FIG. 7.

Simultaneous tracings of the cardiopneumograph (upper curve) and carotid (lower curve). Time marking = $\frac{1}{30}$th sec. At x in upper curve the ventricular systole commences, and the corresponding point on the carotid precedes the tidal wave by about $\frac{1}{15}$th sec. The lowest point on the upper curve marked indicates the end of systole, and precedes the commencement of the dicrotic wave also by about $\frac{1}{15}$th sec.

wave of the carotid follows the commencement of the systolic upstroke of the cardiopneumograph in from $\frac{1}{10}$ to $\frac{1}{15}$ of a second, that time elapsing before the pulse wave reaches the carotid.

By comparing these two tracings we can obtain at any rate approximately correct information as to the actual spot on the cardiopneumatic curve at which the second sound occurs (closing of the semilunar valves). The dicrotic wave leaving the heart after the tidal wave will not in so short a distance appreciably lag behind the latter. If it takes $\frac{1}{15}$ of a second for the tidal wave to reach the carotid, it will take the dicrotic wave practically the same time, and in this case if we find the point on the cardiopneumatic curve which corresponds in point

of time with the commencement of the dicrotic on the carotid, and if we put back that point $\frac{1}{15}$ of a second, then that will be the position on the former curve marking the time at which the dicrotic wave starts from the closed semilunar valves. This will at once be seen to correspond to the time at which the heart assumes its smallest dimensions, falling at the lowest part of the cardiopneumatic curve (Fig. 7 *x*).

Landois' tracings.

If we return to our historical view and ask how Voit, Ceradini and Landois could have overlooked the causes really at work to produce the cardiopneumatic curves, it will not be difficult to find an answer. Voit never took tracings of the movements at all; Ceradini apparently did but never published them; while Landois alone obtained satisfactory tracings, and attempted experimentally to investigate these. His curves are similar to ours but are very small; and the position of the first and second heart sounds roughly indicated by figures placed below the curves. It is a pity that he did not go a step or two further when their entire explanation must have dawned upon him. There can be little doubt that Voit, Ceradini and Landois thought the explanation of the cardiopneumatic movements so simple that they considered, not unreasonably, that more prolonged investigation would be an unnecessary waste of labour. Their position was this: that the chest is filled with both blood and air, and anything which empties it of blood will tend to fill it with air; and truly enough the inspiratory part of the cardiopneumograph corresponds with the greater part of the systole, and the expiratory part with the diastole. But as soon as we enter into details, these factors are seen to be complicated; and when we read the views of Landois detailed in the first part of the paper, we feel that with so many factors of undetermined value, and acting at periods of time not definitely known, it would be possible to explain the cardiopneumatic curve, even if it had quite another appearance from what it has. In fact, a priori reasoning in such a case is not only useless, but is almost certain to be misleading; and where the factors are so numerous, the method must be analytical rather than synthetical.

The truth of this assertion is amply shewn in this particular case; for by opening the chest, the movements go on as before; and none of the factors mentioned by Ceradini and Landois is measurably concerned in producing the movements they described: they are due to another cause which seems to be almost the sole determining factor in their production.

Utility of the Movements for investigation.

These cardiopneumatic movements will, we believe, be of great value in physiological work, inasmuch as they give one a very precise idea of the changes in volume of the whole heart within the opened or unopened chest, whereas the apex-beat simply gives one information concerning changes of pressure at one spot at a time.

For clinical purposes they will be of little utility. One of us has experimented for some time in the hope of making these tracings of assistance to the clinical physician, but he encountered great obstacles, the chief one being that only about half of the patients experimented upon could demonstrate them at all. The chest must be kept rigid, and the glottis opened, all movements being avoided, and this is very difficult except in the case of patients who are both intelligent and phlegmatic.

Conclusion.

(1) The cardiopneumatic movements do not result from changes in the quantity of blood within the chest expelling the air from it or causing it to enter the cavity as Voit, Ceradini and Landois affirm, inasmuch as they occur with the chest cavity open.

(2) They are due to the changes of pressure exerted by the heart upon the investing lungs; and these movements cease on lifting the heart away from the lungs. The lungs act as oncometers to the heart.

(3) They resemble cardiographic tracings in general outlines, but are really what one would obtain by getting the mean of a number of cardiograms taken from different parts of the heart.

(4) They shew the "apex-beat" followed by the inspiratory fall of the lever as the heart assumes its smallest volume, at the lowest point of the lever the semilunar valves close, after which the expiratory rise of the lever occurs—marked with oscillations due to "mass"—as the heart distends and presses upon the lungs.

(5) Distension of the pulmonary vessels causes expansion of the lung, not contraction as Landois affirms, although it may possibly do so when within the closed chest cavity.

PAPERS REFERRED TO IN THE TEXT.

(1) C Voit. *Zeitschrift f. Biologie* I. p. 207. 1865.

(2) L. Landois. *Graphische untersuchungen über den Herzschlag.* Berlin, 1876.

(3) Ceradini. *Heidelberger Jahrbücher der Literatur*, 1869, No. 58.

(4) Ceradini. *Annali universali di medicina fond. dal Dr A. Omodei* 1870. Vol CCXI. Milano.

(5) Landois. *Physiology*, trans. by Professor Stirling. Third Edition, pp. 86 and 87.

(6) *Text-book of Physiology*, by Prof. McKendrick, p. 218.

(7) Haycraft. "Movements of the heart" etc. *Journal of Physiology*, 1891.