ON THE NATURE OF THE CARDIOPNEUMATIC MOVEMENTS.

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For more than thirty years it has been known that the air in the respiratory tract shows slight periodical oscillations, synchronous in rhythm and time with the cardiac beats, — the cardiopneumatic movements, as they have been called since Landois first gave them the name.

C. Voit observed these movements while breathing through a Müller's valve. During a respiratory intermission in which the glottis remained open and the nose closed, regular oscillations were noticed consisting of tiny inspirations and expirations. Voit found the inspiratory motion synchronous with the systole, and the expiratory with the diastole of the heart. He explained the phenomenon by the assumption that each systole — diminishing the volume of the heart — causes a lowering of pressure within the thoracic cavity, and thereby an aspiration of air by the lungs, — an inspiration; while the diastole increases the pressure, and causes an expiration.

Two years later Terné van der Heul, under the direction of Donders, attempted to get tracings of these movements as they are obtainable from the nose, and was surprised to find that, in his case, the inspiration coincided with the diastole and the expiration with the systole, the tracing presenting a regular sphygogram. It is probable, however, that in van der Heul's case the glottis was closed while the tracings were taken, these therefore being in fact nothing but sphygmograms, i. e., the variation in the volume of blood within the closed cavity of the nose. Many persons, as Mosso has shown, are indeed unable to keep the glottis open while they stop breathing. The writer of this article can witness to that fact. In the few attempts which he has made to obtain cardiopneumograms from the nose the result has been invariably a sphygogram.

1 VOIT: Zeitschrift für Biologie, 1865, i, p. 390. The phenomenon had already been described before Voit by Buisson, Gaz. med. de Paris, 1861, p. 320.

2 TERNÉ VAN DER HEUL: Nederlandsch Archief voor Genees- en Natuurkunde, 1867.
Ceradini studied the movements at first by means of a manometer connected with the nose or mouth, and containing smoke or colored liquid; he confirmed the statement of Voit, and also adopted his explanation of the phenomenon. In a later study, however, Ceradini modified his first interpretation. He came to the conclusion that the systolic condition of the heart could not change the pressure within the thoracic cavity as long as the blood put out from the left ventricle is still within the cavity. Ceradini, therefore, now assumed that the oscillations did not originate from the change in the volume of the heart, but from the change in the volume of blood present in the thoracic cavity; the inspiration being due to the outflow from the cavity of the chest, and the expiration due to the inflow of blood into the cavity. Ceradini also obtained tracings of these movements by means of an instrument which he called “Hæmothorakographion,” but never published them.

In 1876 Landois published an elaborate monograph on these oscillations, which he termed, as stated above, cardiopneumatic movements. He studied the movements with the aid of the manometric flame, and, also, by means of his cardiopneumograph, obtained tracings of them from the nose and mouth of men and from the trachea of curarized dogs. The skeleton of his tracings shows a sudden steep inspiratory, and a gradual, slanting expiratory undulation, which Landois, adopting Ceradini’s later view, explains by the sudden arterial output of blood from, and the gradual venous flow of blood into, the thorax. Besides these main movements, Landois noticed on his tracings little secondary notches and peaks which he attempted to explain by certain subsidiary but constant causes. The rebound of the arterial blood causes a tiny peak on the expiratory line. Landois insists that each inspiratory movement is preceded by a small sharp expiratory motion, which he explains by an assumption that I shall not discuss here. Mosso, on the other hand, who accepts the views of Ceradini and Landois with regard to the chief cause of these movements, claims that no short respiratory upstroke precedes the inspiratory movement, and that, in his opinion, the beginning of this latter

1 Ceradini: Heidelberger Jahrbücher der Literatur, 1869, p. 912.
2 Ibid.: Annali universali di medicina, 1870, p. 587.
3 This instrument, as well as the cardiopneumograph of Landois, is practically nothing more than a Marey’s tambour.
5 Mosso: Die Diagnostik des Pulses, 1879.
movement is caused by the widening of the chest due to the heart impulse.

Finally, Martius, who wrote a very elaborate article on the "cardiopneumatic movements in the cesophagus," should also be mentioned, but I shall not discuss his article here. I wish only to say that Martius, in his very elaborate analysis of his tracings, also adopts the view that the flow of blood into and out of the thorax is the chief cause of the cardiopneumatic undulations.

Thus, the authors mentioned, and many other writers on the cardiopneumatic movements, all agree, so far as I can see, that the movements are expressions of the variations of pressure within the air-tight thoracic cavity caused by changes either in the volume of the heart or in the volume of the blood contained in the thoracic cavity.

A recent article by Haycraft and Edie opposes this view. These authors state that the cardiopneumatic movements "occur equally well with the chest cavity open and freely communicating with the outer air" (p. 429). It is obvious, therefore, they say, that the hypothesis of Ceradini and Landois can hardly be accepted as explaining the cardiopneumatic movements. They offer another hypothesis instead, namely, that the movements are "simply due to the heart pressing against the lungs, and that the lungs acted like an oncometer placed around it" (p. 430). The proof for this hypothesis they see in the fact that the cardiopneumatic movements almost entirely cease on lifting the heart away from the lungs. The writers are so thoroughly convinced of the correctness and simplicity of their position that they can hardly understand why it was overlooked by all the other writers on the subject, especially by Landois, who was the first one to obtain tracings of these movements. "It is a pity that he did not go a step or two further," they say, "their entire explanation must have dawned upon him" (p. 435).

The purpose of this Paper is to test the validity of the views advanced by Haycraft and Edie. Before entering into a further discussion, I wish, however, to remark that identical views were


2 HAYCRAFT and EDIE: Journal of physiology, 1891, xii, p. 426.
advanced by Klemensiewicz about fourteen years before Haycraft and Edie, just after the appearance of the monograph of Landois. The original article is not accessible to me, but there is a short report of it in the Jahresbericht of Hofmann und Schwalbe which I will quote here in part. "Klemensiewicz reports experiments on the cardiopneumatic movements, which can also be seen in the frog. He rejects the explanation of Landois because the phenomenon is also present when the thorax is open. The movements are due rather to the intimate relation between the heart and the lungs, the latter, like a large Marey's tambour, registering all the movements and vibrations of the heart." We see here the same hypothesis as that of Haycraft and Edie, and also the same argument for rejection of the hypothesis of Landois and others. In discussing the new hypothesis and the arguments against the older one, I shall use the statements of Haycraft and Edie, as I do not know the experiments upon which Klemensiewicz has based his opinion.

According to Haycraft and Edie, the cardiopneumatic curve represents the variations in the pressure exerted by the heart upon the lungs. As the heart slowly changes its size during diastole, it presses upon the lungs and causes the gradual (expiratory) upstroke. In the beginning of the systole the heart at first "resents" its distortion by the lungs, — it "asserts" itself, which again causes a small but steep upstroke; then as it becomes smaller in all dimensions, it recedes from the lungs, which now expand, thus causing a sharp downstroke, — an inspiration. In a certain sense the theory of Haycraft and Edie coincides with the original hypothesis of Voit and Ceradini, inasmuch as both agree that the movements are due to the change of pressure caused by the change in the size of the heart; but while Voit and Ceradini meant the pressure exerted upon all the contents of the thoracic cavity, Haycraft and Edie mean only the direct pressure upon the lungs.

The interpretation of these authors, however, seems to me to be open to some criticism. We can understand why the pressure of the heart upon the lungs during diastole should produce an expiratory movement; we can admit the possibility that the "self-assertion" of

1 Klemensiewicz, R.: Beiträge zur Demonstration des Pulses und Herzstosses mittels der manometrischen Flamme, nebst Versuchen über die sog. cardiopneumatischen Bewegungen. Mitteilungen des Vereins der Aerzte im Steiermark, 1875-76.

2 Jahresbericht über die Fortschritte der Anatomie und Physiologie, 1877, p. 56.
Nature of the Cardiopneumatic Movements.

the heart will also cause a slight expiratory puff; but we cannot comprehend why the recession of the heart during systole should be followed by an inspiratory movement, — by an expansion of the lungs. 

Even in a state of collapse, the lungs are not perfectly atelectatic, but contain some air, and their elastic tissues are still stretched. When, now, air is driven out by the alleged pressure of the heart, expanding in diastole, what force is then present in the open chest that could distend the lungs anew against their own elastic tension, after they are released from the heart’s pressure? In the tracings obtained from the lungs while the thorax is open, the apparent expansion of the lungs after the alleged diastolic pressure of the heart upon them has been withdrawn may be due to the elastic reaction of the rubber cover of Marey’s tambour, which strives to return to its position of equilibrium after being stretched by the preceding expiration caused by the hypothetical pressure of the heart in diastole. But in the normal individual, in whom the elastic lungs are always considerably stretched, what could cause the lungs to expand again during systole against the strong pull of their elastic fibres; especially in such experiments as Landols and Ceradini made, in which the cardiopneumatic movements were demonstrated by the gas flame and by smoke in a manometer? Smoke is lighter even than air, but, nevertheless, an inspiratory movement is seen in the smoke-column during each systole. What force except the negative pressure could compel the lung to follow the receding heart?

Again, Haycraft and Edie explain the entire cardiopneumatic curve also by the pressure of the heart upon the lungs, and do not permit the change in the pressure within the thorax to exert any influence upon it. But it is, nevertheless, an indisputable fact that during each cardiac cycle there is a considerable variation in the volume of blood present in the thorax. Now, why should this change of volume not cause a change of pressure in the normal, air-tight thorax, and why should this change of pressure not cause an expiration and inspiration, and leave a marked impression upon the cardiopneumatic curve? In this connection, it may be observed that Haycraft and Edie were not very fortunate in comparing the effect of the heart upon the lungs with the working of an oncometer. Here, too, the contractions of the organ enclosed within the oncometer are transmitted only by the changes in the negative pressure which they cause in the air-tight capsule of the instrument.
Furthermore, Haycraft and Edie in support of their statement that the open thorax has no influence upon the cardiopneumatic movements have published two figures, each containing two tracings, — one taken before and the other after the opening of the thorax. In both figures, however, the tracings taken when the thorax was open differ distinctly from those taken from the normal animal. I reproduce here one of these figures.

![Figure 1](Fig. 4 in Haycraft and Edie's article; Journal of Physiology, 1891, xii, p. 431). “Tracing (1) was taken from a rabbit with its chest cavity in normal condition; tracing (2) after making a free opening into the cavity.”

In order to gain a personal insight into this problem I have repeated the experiments of Haycraft and Edie. These authors do not tell us directly from what animals they derived their results. There is a remark in their article that they have experimented with the lungs of rabbits and sheep, but this has reference to another kind of experiments. It is said, however, that the tracings which they present were obtained from rabbits. I have therefore first experimented on rabbits. The method was in general the same as that employed by the authors mentioned and by Landois. The animals were given chloral and then curare, and were kept alive by artificial respiration. The tracheal cannula was connected with a Y tube, each limb being provided with a stop-cock. One limb was connected with the apparatus for artificial respiration, and the other with a Marey’s tambour registering on a revolving cylinder. When the stop-cock leading to the apparatus for artificial respiration was closed and that leading to the Marey’s tambour opened, the cardiopneumatic movements soon appeared. After taking a few tracings from the normal animal, the chest was opened and tracings were again taken. I repeated these experiments on eight rabbits, from every one of which I uniformly obtained the following result: when both pleural cavities were freely opened there was no cardiopneumatic oscillation on the tracing from the trachea.
Nature of the Cardiopneumatic Movements.

Fig. 2 shows a part of the tracings taken just before and after the opening of the thorax. The tracing taken soon after both sides of the thorax were opened is simply a straight line, drawn from a lower level on account of the collapsed condition of the lungs.

My results then were in direct contradiction to those described by Haycraft and Edie. I soon discovered, however, that there was a difference between the method of opening the thorax employed by Haycraft and Edie and that employed by myself. I usually opened each pleural cavity separately on their respective sides of the sternum. In one experiment in which I removed a part of the sternum some slight undulations could indeed be noticed on the tracing obtained from the trachea immediately after opening the chest (see Fig. 3).

Haycraft and Edie opened the abdominal cavity and cut away the anterior attachments of the diaphragm along with the anterior chest wall in its lower part. Of this method, which Haycraft calls the best,

he himself speaks as follows: "As soon as this is done, the heart will fall down in its natural bed formed posteriorly by the lungs and will be carried up and down by them with the movements of respiration; it will be loosened from its more solid moorings to the chest wall and will be seen floating as it were upon the lungs. It is in this position that physiologists from Harvey's time onwards have chiefly
studied its movements, making, I think, too little allowance for the fact that the organ is in an entirely unnatural condition in respect to its attachment." 1 I think that Haycraft is correct in this, but that he and Edie themselves make too little allowance for the fact that in their experiments the heart was in an entirely unnatural condition with respect to its attachments. I have repeated their experiment also on a number of dogs, and have convinced myself more than once that when the pleural cavities are opened without disturbing the natural relations of the heart to the front wall and diaphragm, the cardiopneumatic movements disappear either entirely or are hardly perceptible. When, however, as in Fig. 3, the attachments to the sternum and the diaphragm are removed and the heart allowed to fall back upon the lungs, some distinct movements appear again.

The experiment illustrated by Fig. 4 shows us (1) that the opening of the thorax, without disturbing the natural attachments of the heart, nearly destroys the movements (see tracings Nos. 2 and 4); (2) that the closure of the opening in the chest, though surely not absolutely air-tight, brings the movements out again to a certain degree (tracings Nos. 3 and 6); (3) that the removal of the normal attachments of the heart causes the heart to fall back upon the lungs and brings out movements that seem to be different in size and character from those which occur when the chest is air-tight and intact (tracing No. 5).

There is still another point which deserves special comment. If the communication between the trachea and the Marey's tambour were established immediately after the artificial respiration is discontinued, the rubber cover of the tambour would be exposed to an abnormally high pressure by the air escaping from the inflated lungs. To avoid this I have regularly permitted the thorough escape of the air from the lungs before a tracing was taken. To attain this I placed a T-tube between the stop-cock on the limb leading to the apparatus for artificial respiration and the tracheal cannula; this tube had a stop-cock on its vertical limb which was kept slightly open during the artificial respiration, and which I might call "the relief stop-cock." Before taking a tracing the following order was observed. First the relief stop-cock was opened fully, then the stop-cock of the artificial respiration was closed and that leading to the tambour was opened; and, finally, after the lever of the tambour reached its nor-

1 Haycraft, J. B.: The movements of the heart within the chest-cavity and the cardiogram. Journal of physiology, 1891, xii, p. 447.
mal position again, the relief stop-cock was closed and a tracing taken. With these precautions the tambour was under an equal atmospheric pressure inside and outside while registering the movements. If the relief stop-cock was closed before all the surplus air had escaped, the lungs did not thoroughly collapse and the tambour registered some movements, although the thorax was open and the attachments of the heart thoroughly removed; the movements disappeared as

**Fig. 4.** (1) Shows the cardiopneumatic movements in a small dog with chest unopened. (2) Taken after the left pleural cavity was opened, the undulations becoming very small. (3) The continuation of tracing 2; closing the opening with a towel caused the movements to increase again. (4) Taken after both pleural cavities were opened; there is only a trace of the movements to be noticed. (5) Taken under the same conditions, but after the attachments of the heart to the front wall and the diaphragm had been thoroughly removed. The movements are again considerably increased, but they differ in character from the normal pneumocardiograms of the same dog, and resemble tracing 2 in Fig. 1 (from Haycraft and Edie, see above). (6) Taken under the conditions of tracing 5, but both openings were closed with towels. The size and especially the character is changed. (7) The towels were removed and the tracings are once more the same as in 5.

soon as the air was permitted to escape thoroughly. These movements were quite large at times, especially when the attachments of the heart were thoroughly removed. The tracing in Fig. 5 illustrates this case.
While this tracing was being taken, both pleural cavities of the dog were wide open and the attachments of the heart removed, but the lungs were still considerably inflated and the heart was buried between them. The artificial respiration was interrupted for a long period; the dyspnea caused a slowing of the beats of the heart, which, becoming quite large during each diastole, pressed the lungs to the sides of the thorax, and hereby caused large but not characteristic cardiopneumograms. Haycraft and Edie, it should be remarked, do not tell us what precautions they have taken to avoid this artificial condition of affairs. We read there: "On stopping the respiration, opening the communication with the tambour, and clamping the tube passing to the bellows, the cardiopneumatic movements were well seen" (p. 430). I know by experience that even with an opening widely communicating with the air, it takes some time for an artificially ventilated lung to reach the limit of collapse. Haycraft and Edie do not tell us explicitly that they have taken the necessary precautions; perhaps the lungs of their animals still contained surplus air when the tracings were taken, giving them artificial cardiograms not to be obtained under normal conditions.

My experiments justify the following conclusions:

(1) The cardiopneumatic movements disappear when both pleural cavities are open, provided the heart retains its normal attachments and the lungs are thoroughly collapsed before the tracings from the trachea are taken;

(2) The results of Haycraft and Edie might have been caused by their method of opening the thorax, which permits the falling back of the heart upon the lungs, and also might have been influenced by an incomplete collapse of the lungs;

(3) Without the assistance of the elasticity of the rubber cover of the tambour, the lungs in the open chest cannot follow the receding heart in the systole and no inspiratory movement can occur;
(4) The hypothesis offered by Haycraft and Edie that the lungs act like an "oncometer" placed around the heart cannot be accepted as an explanation of the normal cardiopneumatic movements.

For these reasons I agree with the majority of the writers that the cardiopneumatic movements have their origin in variations of the intrathoracic pressure caused by certain changes in the circulation.