

ART. XI.—*Ascending and Descending Respiration: an Inquiry into its Cause and its Diagnostic Value.* By J. HAWTREY BENSON, M.B., T.C.D.; L.K. & Q.C.P.I.; Physician to the City of Dublin Hospital.

THE phenomenon of that peculiar form of respiration known as the *ascending and descending* is one of considerable interest, both practically and physiologically. It was first described by Dr. Cheyne in 1818, who observed it in a case of fatty degeneration of the heart, with steatomatous and earthy concretions in the aorta. Other examples of the same phenomenon were subsequently observed by Dr. Stokes, and it was considered by him to be pathognomonic, when present, of fatty degeneration of the heart.

Cases, however, have been lately recorded by Dr. Reid, of Belfast, Dr. Head, and Dr. J. Little, in which the phenomenon was present, yet in which (though either valvular lesions, or hypertrophy, or atheromatous disease of the aorta, or fatty degeneration of the diaphragm was present in all), no fatty degeneration of the muscular tissue of the heart existed.

Last year my friend Dr. Little read a paper before the Medical Society of the College of Physicians, in which he brought forward arguments to prove that the phenomenon in question is produced by a loss of balance between the efficiency of the two ventricles; whether that be due to fatty degeneration of the heart proceeding further in the left ventricle than in the right; or else to valvular disease, with or without aortic degeneration supervening so rapidly that the right ventricle has not had time to accommodate itself so as to work in harmony with the left. That, owing to the consequent insufficiency of the left ventricle for the work assigned to it by nature, arterial blood accumulates in the left auricle, in the pulmonary veins, and, finally, in the capillaries of the lungs, thus preventing the ultimate filaments of the pulmonary branches of the pneumogastric from receiving their customary impression from the black blood; which impression, being conveyed to the nervous centres, and converted there into motor force, is the chief cause of respiration. This impression, therefore, being absent, respiration ceases. But presently the gradual removal of the collected arterial blood leaves room for the advance of the black blood, and the consequent renewal of the impression on the pneumogastric, which is then followed by an act of respiration.

Such is Dr. Little's theory; and, though I believe it to embrace the true account of the essential mechanism of the phenomenon, yet, for certain reasons which I will presently mention, it seems to me, that in addition to the diseased conditions of the heart to which I have referred, a certain nervous complication is necessary to determine the accession of this peculiar form of respiration, and without which it would not occur. As an illustration of this complication I give the following outline of a case:—

E. M., a female, aged nineteen, was admitted last year under my care into the City of Dublin Hospital.

About seventeen months previous to her admission she suffered from a cold, and very soon after palpitations and cardiac distress began. When admitted into hospital there was considerable lividity of the features, especially of the lips and the extremity of the nose; and the veins of the forehead, chest, and arms were prominent and blue. There was some dyspnea, and the limbs were thin and wasted; no œdema anywhere. Corresponding to the base of the heart there was a slight systolic bruit, which was heard louder at the second *left* cartilage than at the second *right*, as if the bruit were situated in the pulmonary artery. The second sound was well pronounced and clear. At the apex a loud, slightly musical, systolic murmur was heard, and this was preceded by a slight presystolic murmur. This latter, however, was absent for several days before death. Tracing with the stethoscope to the right, towards the zypoid cartilage, the murmur lost its musical character, and became harsh, but not diminished in intensity. *Fremissement* was very well marked at the apex. The pulse was exceedingly feeble, rapid, and intermitting.

The diagnosis was, therefore, constriction of the mitral orifice, tricuspid regurgitation, and, owing to the fact that the murmur at the base was more distinctly traceable towards the left than towards the right, I was inclined to believe there was disease of the pulmonary sigmoid valves also, though it is known to be such a very rare phenomenon.

The patient did well for exactly a fortnight, but at the end of that time, while endeavouring to get out of bed at night, she fell on the floor, and was with difficulty returned to bed again. Next morning I found her lying in a semi-comatose state. "Perfect" hemiplegia had taken place, sensation and the power of motion having been entirely lost on the left side. The pulse was almost

imperceptible, and very rapid, about 120. She was capable of being roused from this semi-comatose state, and then she exhibited some delirium and childish imbecility, and had considerable difficulty of articulation. The pupils were slightly contracted. The surface of the body and the extremities were cold, especially on the paralysed side.

But the most interesting feature of the case was, that now, for the first time, the respiration afforded an example, the best I have ever seen, of the ascending and descending form. There were intervals of perfect apnea, and these were almost exactly equal in duration to the periods of respiration, and each lasted for an almost constant period of fifteen seconds. Thus each minute was divided into four periods—two of perfect apnea, and two of puerile respiration. And, furthermore, an important and interesting fact is, that it was *only* while the patient was allowed to remain in the semi-comatose state in which I found her that this peculiar form of breathing was present. When she was roused up the respiration became almost normal, but again it assumed the ascending and descending character when she was allowed to lapse into the semi-comatose state. During the apneal period the pulse was more frequent, but more steady and more easily counted, than during the respiratory period.

This semi-comatose state, along with the peculiarity of breathing, continued very well marked for about twenty-four hours, and then together gradually became less distinct, and by the end of another twenty-four hours they had both entirely disappeared, and never again returned.

The patient lived for six days longer, the hemiplegia remaining "perfect." She then died.

Assisted by my colleague, Dr. Hewitt, I made the *post mortem* examination. There was a great deal of venous congestion about the face, neck, upper part of chest, and shoulders. On opening the thorax the lungs were found very much congested. At the apex of the left lung, and also about the middle of its lower lobe, we found a circumscribed apoplectic extravasation, in each case oval, and about one inch and a half in diameter; and the entire remaining portion of the apex presented a good example of diffused apoplexy. The veins on the outer surface of the pericardium were highly congested. When this bag was opened about an ounce and half of healthy serum was found contained, and the heart presented an unusually large appearance. In the left auricle a yellow

fibrinous clot was found, and the auricle itself was considerably dilated and hypertrophied. The left auriculo-ventricular opening was found to be contracted to a very small size. When the left ventricle was opened the mitral valve presented a well-marked funnel-shaped appearance, a condition so well described by Mr. Adams in the fourth volume of the "Dublin Hospital Reports." The flaps of the valve forming the funnel were greatly thickened, and hard, and as if welded together, and the mitral orifice at the apex of the funnel appeared as an ellipse in shape, measuring about four lines in its major axis, and in its minor axis about two lines. The cordæ tendiniæ were absent. The musculi papillares, therefore, appeared to be inserted directly into the funnel. The walls of the ventricle were not hypertrophied, but its cavity was considerably diminished. The aortic valves were found to be slightly thickened, and about a line below the corpus arantii, on two of the flaps, was found adhering a small vegetation, about the size of a pin's head, sufficient to cause the murmur heard there. The caliber of the aorta was greatly reduced, and about an inch above the semi-lunar valves, the aorta, instead of preserving its natural curve towards the right sterno-clavicular articulation, became suddenly bent towards the left, at almost a right angle, thus accounting for the direction in which the systolic murmur at the base was most distinctly traceable during life.

On the right side of the heart the ventricle was found greatly dilated, and the tricuspid flaps considerably thickened and stiffened. The pulmonary sigmoid valves were quite healthy. The muscular tissues of the heart and diaphragm were perfectly free from fatty degeneration.

On examining the brain to ascertain the cause of the left hemiplegia, we found the whole brain rather flabby, but especially the right hemisphere. The lateral ventricles contained a few drops of slightly reddish fluid. The right corpus striatum was uniformly softened to a pap-like consistence, and embedded in its substance were three small extravasations of blood, extending from the size of a millet seed to that of a pea, and having the appearance as if several minute capillaries, in the immediate neighbourhood of each other, had simultaneously given way without producing any distinct clot.

This case, I think, goes far to prove that ascending and descending respiration is in great part a nervous phenomenon, though, for its production, it seems essential that there should be either fatty

degeneration of the heart, or constricted mitral orifice, or some cause tending to produce an accumulation of red blood at the left side of the pulmonary circulation, in fulfilment of the conditions considered necessary by Dr. Little.

That the ascending and descending respiration in this case was in great part a nervous phenomenon is borne out also by the fact, already mentioned, that it was *only* when the patient lay in the semi-comatose condition that it was present, whereas, when the patient was roused up, and the centres stimulated thereby to increased action, and also when the semi-coma had passed away permanently, respiration became almost normal.

In this case the extremely contracted mitral orifice, the diminished capacity of the left ventricle, the diminished caliber of the aorta and its great branches, and the very small pulse, indicate what a very scanty supply of nutrient arterial blood must have been sent to the brain in common with other organs. This was, doubtless, the cause of the softening, which had probably been proceeding gradually and insidiously for a considerable time, but which had not deprived the centres of their power of receiving and reflecting certain impressions. Consequently, as in health, the impressions produced by the black blood on the ultimate filaments of the pneumogastric were still received at the centres, and reflected back in the form of a motor impulse, which caused the natural respiration. When, however, the sudden disruption and disabling of a large proportion of the nervous fibres took place (which both caused the hemiplegia and permitted the fall out of bed), the centres were suddenly incapacitated for reflecting impressions with their previous energy. Respiration, therefore, ceased, producing the phenomenon of apnea. However, after a certain interval, during which the nervous centres were at rest, their excitability was so far restored that they became capable again of reflecting a motor impulse, and respiration was re-established. But owing to their weakened state the centres could not long sustain the effort, and another period of apnea succeeded; and so on.

Now, when we consider the number of cases of suddenly weakened nervous centres, unaccompanied with this peculiar form of respiration, on the one hand; and on the other, when we consider the number of cases of heart disease in which all the conditions conducive to a plethora of the pulmonary veins are present, and yet which are not accompanied with ascending and descending respiration; and when, again, we consider that, in the case before

us, this form of respiration was not produced by the diseased heart alone, and was only developed simultaneously with the manifestation of the nervous lesion; when, I say, we review these considerations in the light of the foregoing remarks, we cannot but believe that for its production a double pathological condition is necessary, viz. :—

1. A certain diseased state of the heart, by reason of which, indirectly, the excito-motor impulse upon the nervous centres, conveyed through the pulmonary branches of the pneumogastric is diminished.

2. A certain weakened state of those nervous centres, by reason of which the reflecto-motor impulse is diminished.

For were the heart alone diseased, and the centres perfectly sound, then, in obedience to the law of demand and supply in the animal economy, the excitability of the centres would doubtless become sufficiently increased (which it cannot when they are diseased) to compensate for the weakened impressions conveyed to them, and thus, the reflecto-motor impulse being undiminished, respiration would therefore remain continuous.

And were the centres alone weakened, and the heart sound, in this case, no accumulation of red blood at the left side of the pulmonary circulation could take place; so that in neither case, it would seem, could the phenomenon exist, since the conditions at present considered necessary for its production would be absent in each case singly, though present when both were combined. So far as I am aware no other record than mine has been made of the state of the brain in the cases of heart disease, in which this form of respiration was observed. Probably in every such case some imperfection of the nervous centres was present, though not apparent. In some of the cases there may have been no indication of its existence during life, as in my case there was none up to within a week of the patient's death.

I submit these considerations, hoping that they may induce others to observe narrowly cases presenting the form of respiration we have been dealing with, that, by an accumulation of accurately-recorded observations, we may at length arrive at a certain solution of this interesting and important question—important, because if my view be correct it involves two portions of the tripod of life.