On the recurrent brief apnœa, or ascending and descending respiration, observed in cases of cardiac disease / by Thomas Laycock, M.D.

ON THE

RECURRENT BRIEF APNŒA,

OR

ASCENDING AND DESCENDING RESPIRATION,

OBSERVED IN

CASES OF CARDIAC DISEASE.

BY

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Reprinted from the Dublin Journal of Medical Science—July, 1873.

PRINTED FOR THE AUTHOR,

BY JOHN FALCONER, 53, UPPER SACKVILLE-STREET, DUBLIN.

1873.

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THE profession is chiefly indebted to members of the Irish school of medicine for information as to a particular form of distressing breathing commonly associated with cardiac diseases, and which was named by Dr. Stokes "Ascending and Descending Respiration." The whole subject of the relations of morbid conditions of respiration to those of the heart is equally important and interesting. I therefore propose examining the phenomena described by Dr. Stokes, from some general points of view. In his valuable treatise on Diseases of the Heart and Aorta, p. 324, Dr. Stokes thus describes them and their ætiology:—"But there is a symptom which appears to belong to a weakened state of the heart, and which, therefore, may be looked for in many cases of the fatty degeneration. I have never seen it except in examples of that disease. It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnœa is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations. . . . The decline in the

length and force of the respirations is as regular and remarkable as their progressive increase. The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent apnœa occurs. This is at last broken by the faintest possible inspiration; the next effort is a little stronger, until, so to speak, the paroxysm of breathing is at its height, again to subside by a descending scale." Although this description is quite accurate as to some cases, there are others in which there is not the orderly regularity described; for not unfrequently the breathing is irregular in rhythm and depth, with little or no apnœa; and in these respects may, I think, be held as strictly analogous to irregular and intermittent pulse.

Dr. Stokes associates the phenomena specially with fatty degeneration of the heart as its cause; but Dr. Little, partly taking up Dr. Stokes's view that at least the interrupted breathing is dependent on structural cardiac disease, has framed another theory of its causation. According to his reported cases, it may occur without any fatty degeneration of the heart whatever, but that it accompanies other affections of the heart and large vessels, as atheroma of the aorta, aortic and mitral incompetence, and dilatation and hypertrophy of the left ventricle. Any theory of causation to be complete must include the origin of both the structural changes and the associated abnormal breathing and pulse, which three things are however distinct. Dr. Little's theory is[a] that there is unequal action of the two ventricles owing to some one of the lesions above-mentioned. Consequently, the left ventricle—as shown according to his idea, by the irregular and intermittent pulse—is unable to propel the aerated blood, and stops now and then. This blood, therefore, remains in the lungs, pulmonary veins, and left auricle, and as it has already been fully oxygenated it no longer stimulates the respiratory centre through the vagus. Thus the venous blood which is requisite to excite the vagus branches is not supplied, consequently respiration ceases, and the breathing takes on this irregular action By degrees the contractions of the ventricle partially free the auricle and pulmonary veins; venous blood is again sent to the lungs, which stimulates the filaments of the pneumogastric and causes respiration to begin again, faintly at first, but becoming gradually stronger as the act of breathing assists the circulation.

The fitness of this ingenious theory to explain the phenomena,

[a] See Dr. Little's paper on Ascending and Descending Respiration. Dub. Quart. Jour. of Med. Science, August, 1868, p. 46.

depends wholly upon the fact whether there be this prior cessation of the action of the left ventricle. If the heart continue to act during the morbid respiration then the theory is not sufficient. Now, as a fact this cessation does not occur in a great proportion of cases; on the contrary, in some to which I shall shortly refer, the heart's action was comparatively little disturbed. On the other hand, many cases of cardiac disease occur with irregular action in which there is no ascending and descending respiration. Nevertheless, although we may affirm as a practical fact, that structural diseases of the heart do not constitute a necessary condition for the manifestation of this irregular kind of breathing, the two are commonly associated, and I cannot but concur with Dr. Little in the opinion that the vagus system is involved.

A crucial example of the phenomena, as a neurosis of the vagus, occurred in the Royal Edinburgh Infirmary, in November, 1863, upon which I lectured at the time.[a] My friend, Dr. Herbert Taylor (now of Freshwater, Isle of Wight), was then one of my clinical clerks, and took copious notes of the case. The patient was an Irishman, aged fifty-four, admitted into Ward 1, suffering from hemiplegia; as he was repeatedly found to be sound asleep at the time of visit of the class, and as he was not awakened either by the apnœa or by feeling his pulse, his case afforded valuable opportunity of leisurely observing the entire series of phenomena. On some occasions his breathing stopped for twenty to forty seconds, and yet with hardly any arrest of the pulse. This case is subjoined:

J. O'H., aged fifty-six, labourer, admitted November 18th, 1863, No family history obtainable. Previously to admission was employed in sinking a well, which kept him constantly wet; has not been a temperate man, but enjoyed good health up to four days before admission. On that day he complained of pain in chest, and

[a] I also called attention to it in another lecture reported by the late Dr. Francis Ropes, of Boston, U.S., in The Medical Circular, 17th February, 1864, p. 116. "A complication, or coincidence, as it may perhaps be called [of fatty heart], and referred to chiefly by Dr. Stokes, consists in a temporary suspension of the respiratory act. In a case of hemiplegia, now under observation, this peculiar symptom has been well marked. This patient suffers from occasional fits of apnœa, which are preceded by some feeble inspirations, while, for the space of twenty or forty seconds, there is absence of respiration. There is no *besoin de respirer* during this interval, and the pulse becomes hurried and irregular. Feeble inspirations then commence, which soon pass into those of an almost stertorous character. The most probable explanation of these phenomena is that a sentient palsy of the respiratory centre occurs, or a paresis of reflex sensibility of the mucous membrance of the lung. Hence the absence of suffocative sensation."

some shortness of breath. He attributed this to over-work, but it was so severe as to oblige him to leave off work, and he consequently went to bed. Next day felt better ; but the day after he felt very giddy, and was forced to return to bed. On 17th, about seven a.m., his wife found him lying half out of bed, and passing urine on the floor; when spoken to said his left arm felt very heavy, and could not move it, and that left leg was the same; he also complained of headache, frontal and temporal, but not severe.

Present state.—Decubitus dorsal; eyelids and mouth half closed; body and limbs ill-nourished; vascularity over malar bones more marked on left side than right; right eyebrow wrinkled; left quite flat; left eyelid perhaps slightly more patent than right; incipient arcus senilis; pupils equal and acting; nostrils widely dilated, equally so, do not move with respiration; aural lobes thin and pendulous, equal; right cheek much wrinkled, not so left; upper-lip, as far as sulcus, in middle line, drawn over to right side, and a little upwards; lower-lip does not appear to deviate.

Right arm.—Temperature normal to hand; sensibility and movement unimpaired.

Left arm.—As well nourished as right; veins on forearm full and prominent, and knotty; temperature feels less, and thermometer proves a difference of four degrees; sensibility impaired, but not annulled; slight resistance to force of extension and flexion; arm semiflexed; when told to raise arm takes hold of it with other one for the purpose; fails to raise it by himself; when raised for him, and let go, it drops heavily and helplessly; does not feel numbed.

Left leg.—Temperature feels lower than on right leg; cannot raise leg when told; says it feels numbed when questioned; sensibility not perfect in left leg; reflex action perfectly excited on pricking sole of foot.

Speech somewhat affected, still what he says is pretty easily understood; tongue, furred thickly, is easily protruded, but turns to left side somewhat; urine and fœces not passed involuntarily.

Breathing appears calm for the most part, with frequent intervals of accelerated and laborious breathing. When the act of respiration is suspended the fact of coughing once or twice does not seem to bring it back, but if the patient is roused and made to speak, respiration is resumed. The cessation appears to be quite regular in point of time, occupying generally about thirty seconds, and respiration then continues for about twenty-five respirations. The heart's action at such times was accelerated and tumultuous.

Heart.—Percussion area normal; apex beat ill defined, but apparently where it should be; no thrill to be felt, but a fluttering is seen over the whole of the præcordial region when the heart's action is excited; action very quick and irregular, at times more forcible than at others, hesitating and undecided. A dry and harsh murmur takes the place of the first sound at left apex. Was ordered at visit an enema of an ounce of spirits of turpentine, common salt, and gruel; patient passed a good motion after its administration. Next day at noon temperature 2° less in left arm than right. On the 19th sensibility of left arm and leg very much diminished; sense of taste is also impaired; the rigidity noticed on admission is gone; deviation of face to right side less marked; when asked if he has pain, complains a little of forehead, and much of small of back; deglutition is not impaired; urine is ammoniacal, without albumen. He yawned much on day of admission, and also ever since; was often found sleeping at time of visit, when the same respiratory condition was present, but no alteration in cardiac action was detected; ordered sinapisms from neck to between shoulders for fifteen minutes, to be renewed every hour.

21st.—Complains of pain down affected side; speech somewhat more distinct; is certainly more thirsty and drowsiness increasing; moans constantly in his sleep, and is fidgetty and restless. When asked if he has pain, says "no, only a weakness in chest." When told to point out spot, puts his hand at a little above ensiform cartilage; denies being out of breath, and says "Oh, I get my breath all right;" no difference, however, exists in respiratory phenomena.

23rd.—Passed a restless night; some delirium. When asked how he feels, replies, "Oh! I am very well, only the left arm is not quite well yet." Face more drawn to right side; left nostril much dilated; speech less distinct; is emaciating; tongue still deviates to left side, dry and brown; has some difficulty is protruding it, and seems conscious of the fact; some blebs are seen on and about left ankle; yawning increased; deglutition is a little impaired.

24th.—Appears weaker; emaciation is progressing; still restless; says he feel a good deal better; another bleb on calf of left leg; no change in respiratory condition; ordered

℞ Strychniæ gr. ½.
Acid. acet. dil. ʒj.
Aquæ ad ʒx.

one tablespoonful three times a day.

26th.—Over left leg, and a little over right a plentiful eruption of urticaria is seen.

28th.—Commencing bed-sores on left nates.

Progress of the Case.—On December 1st, complained of pain in right side; friction sounds detected; coughs slightly; no expectoration; delirium occurred during the succeeding night; the following morning said he was better, and the ascending and descending respiration was not observed; friction heard in right infra-axillary region; the right side moved but slightly in respiration, which was chiefly diaphragmatic; dulness was perceived over right base posteriorly, where also friction and fine crepitation were heard. Dr. Laycock attributed this defective movement of the right side, and the lesion of the right lung and pleura, to motor and trophic paralysis. On the 3rd December the respiratory phenomena were less marked, but on the 4th weeping was observed in the afternoon, although the patient was cheerful about himself, and the peculiar breathing was again manifested. On the 5th heart's action was calmer. Œdema of the left hand commenced; on the 9th, œdema of the lower part of the left thigh; foot and lower part of leg very cold. On the 12th, a fresh pulmonary attack commenced—the left pleura now being the seat. During the three following days there was considerable delirium; he was very troublesome at night, crying out and groaning. On the evening of the 16th he had more frequent paroxysms of apnœa; the cardiac action was hurried—about 130 per minute, but regular. On the 18th, symptoms were found to have subsided somewhat, but on the evening of that day dyspnœa came on with greatly increased feebleness; pulse about 60, and irregular; respirations hurried and forcible, from 45 to 50 per minute. The pulmonary symptoms had become greatly aggravated, and the patient died about one o'clock on the morning of the 20th. *Post-mortem* was not permitted.

Such are the details of the case, as recorded by my friend Dr. Taylor.

In the absence of a *post-mortem* examination it would be useless to speculate on the lesions which affected the heart. It is not, I think, doubtful, however, that the *pulmonary* lesions were consecutive to the hemiplegia and dependent on centric change in the respiratory trophic system. It is obvious too that the lesion was spinal, for changes of nutrition took place such as occur in paraplegia and other spinal neuroses. These trophic lesions were—the lower temperature observed, the urticaria, the effusive inflammation, as

indicated by the blebs, and similar to that which occurs in herpes zoster and anæsthetic leprosy, the œdema, and the sloughing—all limited to the limbs which were deprived of motion and sensory power. The increased vascularity more especially observed on the left cheek may be attributed also to vaso-motor palsy.

A peculiarity of the case is to be noted in the fact that the pulmonary trophic lesion in this left hemiplegia commenced first in the right thorax, and it was not until towards the close of the case that the left became affected. To this, however, I will again refer. Further, when commenting on the respiration at the bedside, I pointed out that the slower breathing was due to the diminishing sensibility, and the accelerated breathing following the interval of apnœa, was due to the stimulus of unaerated blood—being hurried proportionately to the need of oxygen.

This connexion of the irregular breathing with special morbid states of the nerve-centres was manifest also in Dr. Cheyne's case, which is fully detailed by Dr. Stokes. That patient also had hemiplegia, with coincident symptoms of cardiac degeneration. Further, Dr. Hawtrey Benson details a case[a] of mitral disease in a woman aged nineteen, who manifested the breathing *only* while in a semi-comatose condition; when the patient was roused up, and the nerve centres thereby stimulated to increased action, and also when the semi-coma had passed away permanently, respiration became almost normal. Dr. Benson deduces from his observations two causes—1. A certain diseased state of the heart, causing a defective excito-motor impulse through the pulmonary branches of the vagus; and 2. An enfeebled state of the vagus centres leading to diminished reflex action.

There is one fact, however, of fundamental importance in considering the causation of this breathing not referred to in any of the essays to which I have referred, and which was exemplified in the case of the Irishman O'H., namely, its common occurrence during sleep. In none of the cases quoted is mention made of the sleeplessness which this neurosis of the vagus induces, nor of its occurrence during sleep. During the last session at the Royal Infirmary, Edinburgh, when I arrived one day at the bedside of a brewer's cellarman, he told me that he had had no sleep all night, because his heart always stopped just as he was dropping off. I described his feelings to my class, remarking that it was not his heart that

[a] Ascending and Descending Respiration: An Inquiry into its Cause and Diagnostic Value. Dub. Quart. Journ. of Med. Science, Aug., 1869, p. 127.

stopped, but his breathing, and he eagerly exclaimed, as he listened to my description, "That's it." The clinical resident physician, Dr. James, in accordance with my wish, watched this patient at night, and found that what he suffered from was the ascending and descending respiration. I subjoin an abstract of the notes of this case, well taken by one of my clinical clerks, Mr. Andrew Balfour.

William S., aged forty-three, brewer, admitted 6th February, 1873, complaining of cough, shortness of breath, swelling of the scrotum, legs, and thighs, and pain in both inguinal regions. He had a like illness four or five years ago, and again about two years since, being laid up on each occasion five or six weeks. This illness began about three months ago with cough and shortness of breath, but the swelling of the scrotum and legs only six weeks since. His father died of scarlatina, and a sister died dropsical. Was formerly of intemperate habits, but not for the last two years. On examination face was seen to be pale and swollen, cheeks vascular, a considerable vascular nævus at inferior angle of right scapula, and several smaller nævi over thorax and abdomen. Thighs and legs equally dropsical on admeasurement; scrotum very œdematous, prepuce unaffected. Patient always feels cold, even when near a good fire. Temperature 98°·5, skin moist, respiration 28; a tickling cough, with a tough muco-purulent spit, occasionally streaked slightly with blood; cough much worse at night; percussion slightly impaired, at both bases posteriorly; otherwise good. Sibilant and sonorous râles abundant.

Left external jugular vein a little distended; pulse 72, very weak, and slightly irregular; impulse felt over præcordial region; apex beat between fourth and fifth ribs, an inch internal to nipple line; a diffused impulse in epigastric region; percussion area increased; heart sounds very irregular; rough presystolic murmur at apex, and regurgitant murmur (with second sound) at base. These murmurs were fugitive and neurotic. They wholly disappeared subsequently. Urine 40 oz., sp. gr. 1025; albumen to $\frac{1}{4}$.

Does not sleep well; has occasional giddiness; deaf in right ear; position that of semi-orthopnœa.

February 8.—Cough reported much worse, and sleepless, for when he is dropping off to sleep his respiration gradually becomes slower and slower, and finally stops altogether for several seconds, and then patient starts up suddenly "in a fright," the respiration becoming very hurried. Pulse 100, irregular; temperature 97°·75.

Progress of the Case.—After vainly trying hydrocyanic acid, and opium, combined with digitalis and quinine, the patient, on 15th February, was ordered gr. v. of the iodide and gr. x. of the bromide of potassium thrice daily, the third dose to be taken at bedtime.

On 17th February the patient had sleep, and the urine less albuminous.

On 19th pulse 96, much more regular, but becomes irregular when slightly agitated in any way; heart sounds more regular, and the horizontal position taken; sleep much better, and breathing more regular; cough and expectoration less.

On February 22nd it was observed that the patient did not awake with the breathing, which was observed to become gradually slower and slower, but not to stop altogether. Then, when at its lowest point, occasional twitchings of the mouth and limbs were observed to take place, and the hurried breathing followed. On 26th the patient vomited, and on 1st March he again was awakened by the apnœa, and on 4th took chlorodyne m. xx. at bedtime with much relief. Next day pulse more regular. On 6th again took chlorodyne, but did not sleep at all, and at eight o'clock the following morning had a severe attack of dyspnœa, and was pulseless for some time. On the 8th took morphia, with benefit. From this date he rapidly improved, and was able to be up every day until the 23rd, when he was attacked by erysipelas of the left thigh; by the 25th gangrene set in, and the patient died on 27th March.

On *post-mortem* examination the heart was found to be hypertrophied and dilated, weighing 1 lb. 14 ozs. There was slight atheroma of the valves, but they were quite competent. Left lung consolidated at apex, either from pneumonia or pulmonary apoplexy, six pints of serum in abdomen. Cortical substance of kidneys increased; capsule somewhat adherent, and surface slightly granular; apparent increase of fibrous tissue. A few small cysts observed.

This case presents a good illustration of the kind of sleeplessness which occurs, and of the conditions under which the apnœa which causes it arises. It is not only exceedingly distressing, but the want of sleep induced increases the gravity of all the worst symptoms, and hastens on a fatal termination. It is less frequently observed than it occurs, because comparatively so few patients are sleeping, or just falling asleep at the time of visit. It must, however, have been often observed, but not differentiated, and probably included under the vague term cardiac asthma. Dr. Hope,

in his essay on disease of the valves of the heart, to be found in the *Cyclopœdia of Practical Medicine*, evidently includes it under this head, for he gives a highly graphic description of the sufferings of this class of cardiac cases. It is not always, however, that the distress is due to the feeling of suffocation which coincides with the period of apnœa. In the case of O'H. it was plain no such feeling was experienced or disturbed the patient. In the case of a cab-driver (J.), with hemiplegia, in my ward, in whom neurosis of the vagus was seen as irregular and intermittent breathing during waking, and ascending and descending respiration during sleeping—the patient complained that whenever he dropped off to sleep he was immediately roused up by a feeling of giddiness, making no complaint as to his breathing. This was a case of *right* hemiplegia, with more especially impaired sensory function. This patient had irregular pulse, without murmurs, and albuminuria and œdema of the right leg and arm. After death no cerebral lesions were found, further than extensive atheroma of the blood vessels, but there was a small aneurismal excavation in the wall of the enlarged left ventricle filled with a clot the size of a walnut. This case also manifested the trophic and vaso-motor changes in the lungs, only surmised in O'H.'s case. He had great bronchial congestion before death, but after death it was found that the lungs presented quite opposite conditions. The left lung was singularly pale and blood-less, while the right was purple with congestion, probably from crossed defect of the nervous system, as in O'H.'s case; the lesion being in the left hemisphere and corpus striatum, but above the decussation. In the case of this patient it was very interesting to observe also that the *right* kidney, like the *right* lung, was highly congested and large, the left being small, without congestion.

If then this condition of the breathing be a neurosis of the vagus, not necessarily dependent on structural or other diseases of the heart, it may occur under varying conditions of the nervous system in those who have such disease. A very distressing illustration of this occurred under my observation in the case of a lady who had disease of the heart and albuminuria, and who took the syrup of the phosphate of iron and strychnine. In her the first affection of the breathing was manifested while taking the medicine by sudden attacks, resembling angina pectoris of a most distressing character, occurring when she was sound asleep. These disappeared, and were replaced as dropsy came on by the ascending and descending respiration when falling asleep. That the state of falling asleep

modifies the vagus, so as to induce the phenomena, proves conclusively how pure a neurosis it is in its origin. It cannot be doubted, I think, that merely emotional states will modify the vagus centre in like manner. Experience in this respect as to attacks of paroxysms of whooping cough and spasmodic asthma indicate this conclusion; and it seems to me well worthy consideration whether surgical deaths from chloroform may not be due to complex causes acting on the cardiac and vagus centres, for in surgical operations, such as on the penis, there is the three-fold causation of—painful emotion—shock to the nervous system by the operation,—and depression to the nervous system by the anæsthetic drug administered. The study of the whole process of ascending and descending respiration in connexion with sleep shows, I think, that the gradually diminished motor activity is coincident with gradually diminished sensory activity until the carbonized blood rouses up the sensory centre.

I have just remarked that both the cardiac and vagus centres may be involved in cases of death from chloroform, and, I think, clinical facts show that this conjunction occurs also as a neurosis, either as foreshadowing or as accompanying cardiac disease, and that such conjunction probably occurs in such cases as have been observed by Dr. Stokes, Dr. Little, and others. The result might be manifested, either by the momentary stop to the action of the left ventricle, characteristic of intermittent pulse, or by one of a more prolonged character, as in angina pectoris. I shall not here enter upon the proof that intermittent pulse is not only a neurosis in a variety of cases of functional diseases of the heart, as is generally acknowledged, but is such also in numerous structural diseases; that this is the case has been fully established, to my own satisfaction, by long and careful observation, but the details would be too numerous to give here, so as to satisfy the judgment of others on this point.[a] It occurred to me, however, long ago, when studying the pathological relations of sleep, to note the combination of this kind of apnœa with cessation of cardiac action, and I quoted a case in illustration, as follows, taken from *Bell's Nervous System of the Human Body*, 3rd edit., p. 426.[b]

[a] Since writing the above I have read with much interest Dr. Nixon's cases of functional mitral murmur in the last (June) number of this Journal. My experience coincides with his—and I may add with that of Dr. Stokes as to functional aortic murmurs.

[b] See my paper on the Reflex Function of the Brain, Brit. & For. Med. Rev., Jan., 1845, p. 306.

A West Indian surgeon consulted Sir Charles Bell with these symptoms:—"On falling asleep, just at the time when volition and sensibility cease, the involuntary motions also stop, with a sensation of death, under which he awakes, generally convulsed. His medical friends have found that when sleep is overpowering him the breathing becomes slower and weaker, the heart and pulse also fall low, and cease to beat as sleep comes on, and after a very short time he awakes in terror."

This case is interesting, as showing that there are cases in which both the heart and lungs cease to act as sleep comes on; we may, therefore, infer that this condition may also arise during the waking state, and this is probably the condition in paroxysms of angina pectoris. Bell, in the work from which I have just quoted, and on the same page, records some most interesting particulars as to John Hunter's case, detailed by the great physiologist himself. On one occasion, when he had missed "his usual attack of gout," but had "a peculiar hard pain about the pylorus," he found that "this was attended with great weakness, and having accidentally cast his eyes to a looking-glass, he fancied that his countenance was like that of a dead man. He could feel no pulse in either of the wrists. Finding the involuntary respiration ceasing, and fearing that he should die in consequence, he imitated the involuntary by a voluntary action of the muscles, and breathed altogether by force as well as he could."

The condition which Hunter experienced was probably premonitory of that diathetic structural degeneration of the coronary arteries, which was found to have occurred in him, he having died in a paroxysm of angina pectoris, induced by emotion. My own experience has led me to the conclusion that intermittent pulse, heart-ache, and other sensory conditions referred to the heart in first attacks of gout, and in cases of fatty degeneration preceded by depressing emotions, as grief and anxiety, are premonitory of structural change, and indicate the beginning of a trophic neurosis of the heart, the coronary arteries, and aorta. Such results follow after the depressing emotions and other causes have occurred, at indefinite periods of from a few days to two or three years. Hence, to have a good history of the relations of the apnœa I have discussed to intermittent pulse and structural diseases of the heart, a more careful inquiry into the conditions of the nerve centres, which influence the nutrition or functional activity of the heart is needed. Of this, the case of O'H. is an illustration. I would

also further observe, that according to my experience, undue irritability of the heart, such as is manifested sometimes by persons who are being examined for the purpose of life assurance, so that the heart beats rapidly and nervously under the procedure, is premonitory of future cardiac disease, and sometimes at no long interval of time.

I have not entered upon the mechanism of this apnœa, the subject being too complex for discussion now. I would, however, remark here, that it is probable cases will be found to differ, according as the right or left vagus is affected. Recent experimental researches tend to show that the right vagus exercises a much more powerful inhibitory influence over the heart than the left, and is exerted equally over every part.[a] It is probable that in the case of O'H., the palsy being at the left side, the lesion was in the right half of the nerve-centres. So also in the case of the cab-driver J. Physiologists, however, have not unravelled the problem of crossed action, and the vagus may be affected, so as to seriously influence nutrition of the lungs and the co-ordinate action of the heart, yet with no general symptoms. This, I think, is frequently the case in unilateral pneumonias.

Another question arises on considering the mechanism, which I must, at least, glance at, and remark, therefore, here, that it is not improbable the phrenic nerve and diaphragm will be found involved in a certain class of cases, in which there is total cessation of breathing and pulse, such as occurs in paroxysms of angina pectoris and other cardiac diseases of a neurotic character. I notice that in one case referred to by Dr. Little (recorded by Dr. Head) fatty degeneration of the diaphragm was observed. It would, perhaps, be found that cases of the kind are more common than is suspected, if the diaphragm was carefully examined in *post-mortems*.[b]

A few words as to the prognostic significance of this apnœa. My experience leads me to the conclusion that conjoined with anasarca it is of evil omen, and this is not unfrequently the state of the patient. I observe that in Dr. Cheyne's (the first recorded case), there was anasarca.

But, whether or no, this peculiar apnœa is so distressing and tends so to abbreviate life, that I am induced to add a few words as to treatment. The iodide and bromide of potassium in doses of five

[a] See paragraph in Journ. of Anatomy and Physiology, Nov., 1872, p. 180.

[b] To this point I referred in the lecture published in the Medical Circular of February 17th, 1864.

grains of the former and fifteen of the latter taken at bedtime have proved in some of my cases an useful hypnotic; but the means which are available to give tone to the nervous system in general should be diligently adopted, and amongst these nothing is more valuable than a cool bracing atmosphere. Strychnine with iron, when there is fatty degeneration of the heart is beneficial, but is of doubtful, if not dangerous use, in other cases. The iodide of potassium is a valuable alterative and tonic with a bitter infusion. In cases in which hypnotics must be used the readiest is a few minims of the liquor of bimeconate of morphia given hypodermically. All we have to aim at in the use of hypnotics is, so to diminish the sensibility of the pneumogastric centres, that the urgent need for breathing, which comes on consecutively to the apnœa be not felt, for it is the feeling of suffocation which wakens. I have repeatedly watched a patient thus treated by morphia hypodermically, and observed that so soon as the patient was under the influence of the drug, which occurred in about fifteen minutes, the apnœa occurred and the hurried breathing took place without the patient being awakened. Ten or fifteen minims of the liquor of the bimeconate will suffice for this purpose. Upon the whole, however, the iodide and bromide must be preferred as they are both palliative and curative, whereas morphia is not curative *per se*, but, on the contrary, is injurious. I am satisfied it tends to increase the albuminuria and dropsy which so often accompany the cardiac and pulmonary lesions. Not unfrequently the gastric vagus is implicated, in which cases hydrocyanic acid is the most efficacious. The influence of the stomach on the heart and lungs should, indeed, always have careful consideration in cardiac and pulmonary neuroses.