amining what has been called the first stage of phthisis, or that which is characterised by a morbid product in a portion of the lung. If we could define the pathology of this stage, we should have accomplished the most difficult part of our task.

One symptom to note in phthisis is, that not unfrequently an hæmoptysis ushers it in; and it is true that some of the more rapid forms of phthisis are so commenced. A patient, overworked, it may be, or enfeebled by anxiety or other depressing agents, will bring up a mouthful of blood, and soon present all the physical signs and the fever and waste of phthisis. In such an event you must watch the temperature and pulse quite as much as the physical signs, and if much fever, with an evening temperature of 102° or 103° prevail, and morning sweats, the case is likely to be rapid in its changes for the worse, even without pause, till a cavity is formed; or progressive softening of the lung may occur, and the case become one of galloping consumption. This event is to be looked for, but is not an invariable sequence of a rather profuse hæmoptysis occurring as an early symptom. Its meaning is undoubtedly great congestion of the lung, and we must remember that rapid softening occurs often. Either inflammatory products block the alveoli, or, as has been said, retained clots of blood form the nuclei of degeneration, and in their changes involve the lung-tissue itself.

Not all these cases so initiated do badly, but many pass into the chronic stage, exhibiting little tendency to degenerate or ulcerate the lung, and the symptoms, although primarily severe, may subside, and leave only the signs of a quiescent block of a portion of lung, while the patient recovers a fair condition of health. An hæmoptysis depletes the lung and relieves the congestion, as I shall have occasion to point out to you again, and its occurrence is often followed by a long period of quiescence or latency of disease.

ON GELSEMINUM SEMPERVIRENS.

(*Continued from p.* 417.)

PRELIMINARY ACCOUNT OF EXPERIMENTS RELATING TO THE ACTION OF EXTRACT OF GELSEMINUM ON THE RESPIRATORY MOVEMENTS.

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IN previous papers it has been shown that gelseminum exercises a very remarkable influence on the function of respiration, and that when the drug is given in fatal doses death takes place, as in poisoning by curare, by gradual arrest of the respiratory movements. Facts have also been referred to which plainly indicate that this result is not, as in the case of curare, to be attributed to suspension of the functions either of the motor nerves or of the muscles to which they are distributed, so that the conclusion can hardly be avoided that this agent exercises its influence directly on the cerebro-spinal centres, and particularly on those parts which preside over the rhythmical respiratory movements.

Before proceeding to relate the results of experiments made for the purpose of investigating the nature of this influence, it may be useful to place before the reader some statements as to the mechanism of what may be called the respiratory nervous system.

It is one of the most important results of the investigations of the past thirty years in relation to the physiology of the nervous system, that each of the combinations of muscular movements by which the great mechanical functions—viz., respiration, circulation, &c.—of the organism are kept going independently of the will or contrivance of the individual, is under the control or direction of a so-called "motor centre," *from* which emanate those outgoing channels of influence by which the muscles concerned are brought into harmonious action for the effectual carrying out of the function, and *towards* which those afferent channels converge, by which the centre is itself brought into relation either with the outside world or with other parts of the nervous system.

Of these centres some act automatically, others excito-motorially: by these expressions it is meant that in some instances the centre issues its commands from time to time to the muscles over which it presides, independently of any impression or influence received from outside through afferent nerves; whereas in others, although the centre acts independently of the mandates of the will, it exerts itself only when it is awakened to action by some stimulus of external origin.

Of the automatic centres, that which presides over respiration is one of the best examples, not only because we have very precise knowledge as to its anatomical situation in the floor of the fourth ventricle, but because the experimental evidence that its action is purely automatic, and not reflex, is clearer and more conclusive than that which can be given in respect of any other similar structure. For it has been shown by the most conclusive experiments that so long as the respiratory centre is supplied with blood which is sufficiently arterialised, and is in communication by motor nerves with the muscles over which it presides, it continues to discharge itself rhythmically towards these muscles, so as to determine that orderly combination of movements of which respiration consists, notwithstanding that all possible channels of extrinsic influence have been cut off.

In addition to this fundamental fact of the pure automatism of the respiratory centre, there are others which must be borne in mind in order to a proper understanding of its function. The first of these is the remarkable relation between its functional activity and the quantity of oxygen (in the form of oxyhæmoglobin—i.e., in combination with the crystalline colouring matter of the blood) which is supplied to it, which is of such a nature that respiration diminishes or (as is the case in most animals) ceases as the blood approaches the condition of complete saturation with oxygen; while, on the other hand, when the quantity of this gas held in combination by the blood-corpuscles becomes defective, the activity of the respiratory movements increases more and more until, as in asphyxia, the functions, not only of the respiratory centre, but of other essential parts of the nervous system, become so disordered and impaired that the continuance of systemic life is no longer possible.

A second fact relates to the channels by which the centre acts on the individual muscles which take part in the respiratory process. In man and many other animals, the only muscle concerned in ordinary tranquil breathing is the diaphragm. As respiration becomes more active other muscles—e.g., the intercostals, the scaleni, the external laryngeal muscles, &c.—come successively into auxiliary action. All of these act in respiration under the direct control of the centre, with which all must therefore be in direct communication by motor nerves; but the *facility* with which the centre discharges its influence towards these several muscles is evidently very different. Under normal conditions its area of influence is limited to the diaphragm, or at most to that muscle and to the intercostals; but as its supply of oxygen diminishes, this area, step by step, extends to an increasing number of muscles, as is familiar to everyone who has had the opportunity of watching a human being or an animal in the successive stages of dyspnœa. We can explain this in one way only—namely, by supposing that the channels of discharge leading to different muscles offer very different degrees of resistance to its passage.

There is, however, another fact which must not be overlooked. Although, as has been already stated, the respiratory centre possesses in itself the power of absolutely independent action, provision is made in its structure of a most remarkable kind for subjecting it to the modifying influence of external conditions, and particularly of conditions existing in the organs over which it presides. By means of a special nerve—the respiratory vagus (by which term may be designated those afferent fibres of the pneumogastric which are distributed to the air-passages)—the centre is constantly affected by the varying conditions of the respiratory organs in such a way that the relation which is thus maintained between the two parts (centre and organ) can only be spoken of as regulatory or compensatory, and cannot be compared with that which exists between a sensory surface and a reflex centre in the ordinary case of so-called reflex action. For here the influence which passes

from lung or bronchial tube to centre has no tendency to excite motion, but the contrary; its nature being such that —being comparable to that of a bridle, not to that of a spur—it can only be called inhibitory or regulatory. As to the nature, and still more the final cause, of this singular action of the respiratory vagus, although a great deal more is known than has here been pointed out, we have unquestionably much to learn.

In the power which the respiratory centre possesses of automatic rhythmical action; in the peculiar relation which we have seen to exist between its activity and the varying proportion of oxygen held in combination by the hæmoglobin of the blood; in that fine graduation of its channels of discharge, whereby it is enabled to widen its range of influence according to the requirements of the organism; and, finally, in the regulatory mechanism by which it is kept constantly *en rapport* with the organs of which it directs the movements,—we have a series of endowments which completely fit it for its function, and afford us a satisfactory explanation of all the phenomena of tranquil or exaggerated breathing so long as the normal rhythm is maintained.

There are, however, many well-known instances, both in health and in disease, in which muscular acts which are in effect respiratory are performed, to the understanding of which the mechanism of automatic respiration gives us no key. Of these acts many are unquestionably reflex—such, for example, as the inspiratory gasps which are induced by the sudden application of cold to the surface, or the sudden contraction of the diaphragm, which is the initial act of the complicated reflexes of vomiting, coughing, and sneezing. Other instances come under our knowledge experimentally in which sudden contraction of the diaphragm can be evoked by excitation of afferent nerves and particularly of certain afferent fibres contained in the vagus. All of them are, as I have said, in effect respiratory, for they result in inhalation, followed by expulsion, of air, but differ from normal respirations quite as much in their *mode* as in their origin. It is, I believe, characteristic of all reflex inspiratory acts that they are of extremely short duration, and that they are so immediately followed by expiration, that the latter seems, as it were, to cut short the former before its completion. For this reason, the English word gasp, in its ordinary and well-understood acceptation, fully comprehends the conception of reflex inspiration. The act of vomiting may be truly said to begin with a gasp, immediately followed, and as it were cut short, by contraction of the muscles of the abdominal wall; and so of the other instances. Another characteristic difference between the gasp and the rhythmical respiration is that in the former the auxiliary muscles of respiration do not take the same part as in the latter. In its simplest form the gasp consists in a spasmodic contraction of the diaphragm, differing in the most striking way from the deliberate manner in which that muscle draws its central tendon downwards in ordinary breathing, its fibres remaining in the shortened state during the whole of the respiratory pause. In other instances (whether from greater intensity of excitation or greater excitability of centre) the purely diaphragmatic gasp extends itself over a wide range of muscular actions, but the muscles thus brought into play are not those of inspiration. We have the best example of this in the well-known gasping inspirations which characterise the second stage of asphyxia, in which we see that each contraction of the diaphragm is but the starting-point of a general convulsion affecting the whole body of the asphyxiated animal, of which the most obvious character is expressed by the term usually given to it, "stretching spasm," for the muscles chiefly affected are the extensors of the trunk and limbs.

The space at my disposal will not permit me to enter so fully into this subject as I should like. It will be sufficient for my present purpose if it is understood that the automatic is distinguished from the reflex respiratory act—the sigh from the gasp—by well-marked characters. The two muscular actions have this in common. In both instances the combination of muscular movements is "led off" by the diaphragm, but the combination itself is so entirely different, that there can be no doubt that the co-ordinating centre from which those impulses originate which manifest themselves in spasmodic respiratory acts, whether accompanied with "stretching spasms" or not, is entirely different from that which regulates the rhythmical motions of ordinary respiration.

The preliminary experiments on which the following statements are founded were made for the purpose of investigating the influence of gelseminum on the movements of the diaphragm. This was accomplished as follows:—The animal (rabbit) employed having been narcotised by chloral, a small spatula (shaped like the handle of a teaspoon) was introduced into the peritoneal cavity through a small opening in the linea alba, and passed upwards in front of the liver until its convex surface rested against the under side of the centrum tendineum. The stem of this spatula was brought into connexion with a lever, by means of which its to and fro movement, and consequently that of the diaphragm, was inscribed on the cylinder. By this means graphical records were obtained of the diaphragmatic contraction before, and at successive intervals of three minutes after, the injection into the venous system of a fatal dose of gelseminum extract (six minims), or rather of such a dose as would have been fatal had not the result been warded off by the employment at the proper moment of artificial respiration.

The first effect of the injection is to augment the depth, but apparently not the frequency, of the respiratory movements. This condition (hyperpnœa) begins in from five to ten minutes, and is soon over. It is followed by a second stage, in which, on the whole, the diaphragmatic movement is diminished both in extent and frequency. This happens in accordance with the general principle applicable to most cases of toxical action, that paresis of a central organ is preceded by over-action. In this instance, at all events, it is certain that the diminished action is not due to exhaustion, for the previous hyperpnœa is far too inconsiderable.

On the whole, the diminution of movement is progressive; for, as has been already stated in previous papers, the final result is that the respiratory movements gradually fade away, but all our records show that the decline is invariably interrupted by intermediate stages, in which, to use old-fashioned language, the organism makes a struggle for the maintenance of life.

To understand how this happens we must call to mind that during the action of the drug the blood gradually becomes more and more venous. We should therefore expect that along with effects due to the purely paralysing influence of the drug on the automatic centre, others should mix themselves produced by the defect of oxygen and excess of carbonic acid.

The interruptions above referred to are precisely of this nature; they are due, as I believe, to the mixture of the asphyxial with the toxical element in the process, these being distinguished from each other, not only by their order of succession, but by their character; the first is but a weakened normal contraction of the diaphragm, the second a supplementary spasmodic effort of the muscle which ends as suddenly as it begins, and is immediately followed by an equally sudden recoil.

In other words, the same type of respiratory movement which presents itself in the beginning of the second stage of that form of asphyxia which is produced by the inhalation of a limited quantity of air without mechanical interference with the respiratory passages, and which has been described in the first part of this paper as spasmodic, mixes itself with normal breathing. The way in which this mixture takes place could scarcely be understood if we were to attempt to investigate it without the graphical method. With its aid there is little difficulty. The record shows that as the blood becomes venous the diaphragmatic descent is not, as normally, a continuous movement, but is broken into two stages or acts, separated by a period of inaction.

That this is the right interpretation of this remarkable phenomenon (of which some readers have no doubt seen the counterpart at the bedside) we can satisfy ourselves by very carefully observing the behaviour of the auxiliary muscles of respiration, particularly those of the larynx, at the same time that the diaphragmatic motions are indicated by the lever. By this mode of study we learn that those respiratory movements which are dependent on the automatic centres (e.g., the rhythmical movements of the arytenoid cartilages) keep time, not with the spasmodic ending, but with the automatic beginning of the diaphragmatic descent.

In most of the animals it was observed that the spasmodic element disappeared from the record as the toxical

process proceeded. In one, however, it attained so great a predominance that the tracing could not have been distinguished from one of the second stage of asphyxia. The record (Fig. 7) affords evidence of complete abolition of the normal element, but even here it could be seen, by watching the laryngeal movements, that the automatic centre was not entirely paralysed; for its inspiratory mandate, though it was neglected by the diaphragm, was punctually obeyed by the posterior crico-arytenoid muscles, which, at the proper moment, opened the glottis, fruitlessly of course, with the same precision as ever. As the expiratory closure of the glottis was immediately preceded by the spasmodic inspiration, it is obvious that this happened at a moment which, if respiration had been normal, would have coincided with the respiratory pause.

FIG. 1.

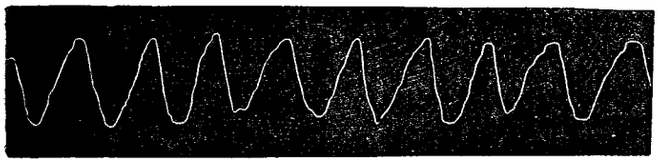

FIG. 2.

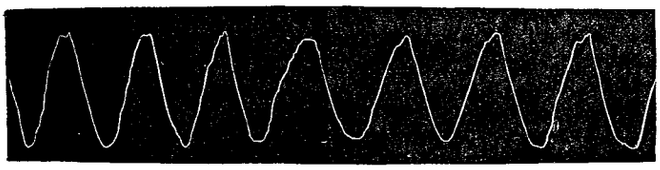

FIG. 3.

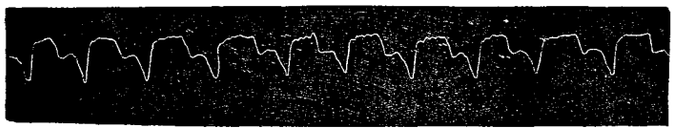

FIG. 4.

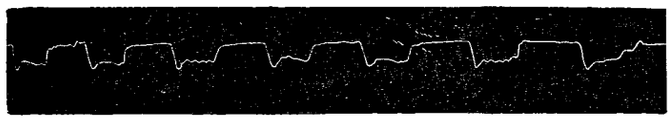

FIG. 5.

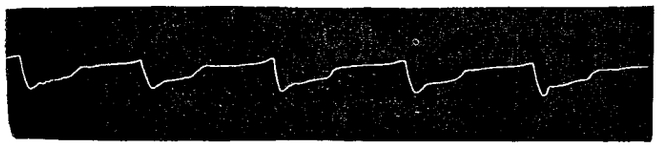

FIG. 6.

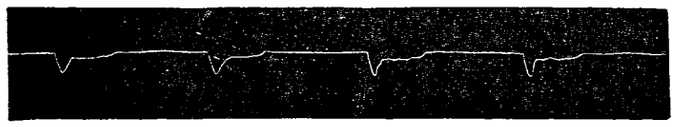

FIG. 7.

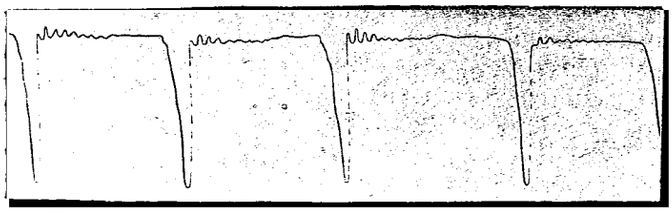

Fig. 1. Normal movement of centrum tendineum. The descending limb of each curve represents inspiration.
Fig. 2. The same six minutes after injection of six minims of extract (diluted) into the jugular vein.
Figs. 3 to 6 exhibit successive modifications of the record obtained in the same animal. In Fig. 3 the first descent expresses the normal, the second the abnormal (spasmodic) contraction of the diaphragm.
Fig. 7. Tracing obtained when the diaphragmatic contractions had assumed a wholly spasmodic character.

The further discussion of these facts must be reserved for a future paper. For the moment it will be sufficient to state that the drug acts on the respiratory function by paralysing the automatic respiratory centre; but that the process of extinction of the respiratory movements, which might otherwise be expected to be gradual and progressive, is prevented from being so by the intervention of disturbances of which the explanation is to be found in the imperfect arterialisation of the circulating blood.

ON

OBLIQUE FRACTURE OF THE HEAD OF THE HUMERUS.

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(*Concluded from p.* 420.)

THAT such an injury occurs there is no reason to doubt. The description in Guthrie's cases is sufficiently clear to warrant this conclusion. I am, besides, fortunate in being able to contribute four additional cases which have come under my notice, in some of which the advantages we possess in the present day of minutely examining such injuries under anæsthesia rendered it a matter of certainty that the injury described by Guthrie existed. They are as follows:—

6. (Reported by Mr. Wm. Fergusson.) Margaret S——, aged fifty, was admitted into Martha's ward, Aberdeen Royal Infirmary, on Dec. 30th, 1874, suffering from the effects of an accident sustained the previous evening. While walking on the pavement her foot slipped, causing her to fall against an adjoining building on the external posterior aspect of the left shoulder. She suffered a good deal during the night from pain in her shoulder. Her left forearm and hand were quite numb and devoid of sensation, and there was also considerable swelling of these parts.

On admission, the elbow was held a little away from the side, but could readily be brought into contact with it by gentle pressure. There was a good deal of swelling over the anterior aspect of the left shoulder, the swelling corresponding pretty much to the pectoral region of the deltoid muscle. The clavicle was entire, as were also the spinous, acromial, and coracoid processes of the scapula. The fibres of the deltoid were very tense. The deltoidal prominence on the outer side of the shoulder was obliterated. The acromion was prominent, but there was no vacuity beneath it such as is present in dislocation. The greater tuberosity did not rotate with the arm. On tracing the humerus from below upwards, it presented the appearance of passing upwards, forwards, and a little inwards from the insertion of the deltoid, to terminate in a small eminence below the coracoid process. The antero-posterior measurement of the shoulder was much greater than on the opposite side, and a groove existed in the region of the bicipital groove between the greater tuberosity and the prominence below the coracoid process. The independent mobility of these two parts showed that this groove indicated the line of fracture, and it was felt to pass obliquely downwards and a little backwards. There was neither elongation nor shortening of the affected limb. The coracoid process of the scapula was less distinct than on the right side, but was not unnaturally movable on raising and depressing the arm. The deformity could not be removed. The arm was immovable to the voluntary efforts of the patient, but movable to the examiner. The patient was able to flex and extend the forearm to a limited extent; on doing so she complained of pain in the region of the long head of the biceps; and on grasping the belly of the biceps while she attempted to flex the forearm the contraction of the muscle was scarcely if at all perceptible. The hand inside the axilla felt the head of the humerus in the glenoid cavity in connexion with the greater tuberosity, and rotation of the elbow communicated itself to the subcoracoid prominence. While rotating the arm hard crepitus was distinctly perceptible. The patient preferred to keep the forearm at about a right angle to the arm. Tenderness on pressure was complained of over the front of the shoulder and on the groove between the fragments. She complained of pain in front of the elbow on