J. Physiol. (1938) 93, 10–23 612.282

ON THE MECHANISM OF PRODUCTION, AND THE PHYSIOLOGICAL SIGNIFICANCE OF "APNEUSIS"

By G. STELLA

From the Department of Physiology, Pharmacology and Biochemistry, University College, London

(*Received* 8 *February* 1938)

According to Markwald [1887, 1890], when bilateral division of the vagi in a rabbit is preceded or followed by transection of the brain stem immediately behind the posterior colliculi, the respiration is profoundly altered, it ceases to be rhythmical, and consists of an irregular series of long powerful inspiratory "cramps", each of several seconds' or minutes' duration and only interrupted at irregular intervals by short expiratory pauses. If the nucleus of the trigeminus nerve is intact and functional, the inspiratory cramps begin gradually to become shorter, eventually being reduced to powerful, cramp-like respiratory acts of a few seconds' duration, following one after the other in a more or less regular, rhythmical series. If, however, the nucleus of the trigeminus nerve has been damaged, as shown by the disappearance of the corneal and nose reflexes, the inspiratory cramps remain long and irregular and lead to death from asphyxia. When division of the vagi, on the other hand, is performed in an animal decerebrated just in front of the posterior colliculi, or at any higher level, the effects on respiration are not different from those which are usually observed after vagotomy in an animal with the central nervous system intact: i.e. they simply consist in a deepening and slowing of the breathing. Markwald concluded that in the posterior colliculi there is a centre inhibitory to inspiration, whose action becomes clear when the inhibitory activity of the vagi is abolished by their section. He further suggested that the nucleus of the trigeminus nerve, when both the vagi and the posterior colliculi had been put out of action, would acquire a new tonus and exercise a rhythmical inhibitory influence, though a less efficient one, upon the inspiratory centre.

Markwald's findings were confirmed by Loewy [1888], Asher & Lüscher [1899], Langendorff [1888] and others. Lewandowsky [1896], while confirming the inhibitory influence on inspiration of the posterior colliculi, could not agree that the nucleus of the trigeminus nerve played any part in reducing the duration of the inspiratory cramps, since this modification of the cramps was not constant and in any case it occurred after sections in the lowest parts of the pons.

More recently, after an extensive study in the cat Lumsden [1923*a*, 1923*b*] arrived at results which differ in some important points from those described above. First, he found that section of the brain stem immediately behind the posterior colliculi did not modify the usual effects of division of the vagi, and saw therefore no necessity for postulating in the posterior colliculi the existence of a centre inhibitory to inspiration. According to Lumsden the inspiratory cramps described by Markwald appeared only when the section fell some millimetres below the upper limit of the pons, whether the vagi were intact or cut. When the brain stem was cut below the striæ acousticæ, inspiratory cramps ceased, and respiration consisted of a more or less regular series of gasps, i.e. of quick inspirations, beginning and ending abruptly and followed by passive expiration. Lumsden concluded that the centre causing the latter type of respiration represented a sort of primitive centre, the "gasping" centre which had been superseded in the course of evolution by two higher centres, (i) an inspiratory centre at the level of the striæ acousticæ, the "apneustic" centre, on whose unrestrained activity the long inspiratory cramps, or "apneuses" depended, and (ii) a centre in the upper region of the pons rhythmically inhibiting the activity of the "apneustic" centre and thus producing normal respiration or pneumotaxy. This he called the "pneumotaxic" centre.

Henderson & Sweet [1929] and Teregulow [1929] repeated the investigation in the cat and obtained results different from those described by Lumsden, but similar to those observed by Markwald in the rabbit, i.e. they found (i) that the inspiratory cramps, or "apneuses", appeared only after section of the vagi, (ii) that it was not necessary to cut the brain stem as low as some millimetres below the upper limit of the pons, as found by Lumsden, a section just behind the posterior colliculi being sufficient. Henderson & Sweet, however, differ from all previous investigators in the interpretation of apneusis, which they regard not as an exaggeration of the normal inspiratory activity, but as a manifestation of decerebrate rigidity, extended to the inspiratory muscles, and having no relation with respiratory activities proper. To prove their contention, they cut the rubrospinal tracts in a vagotomized cat by a median longitudinal incision of the mid-brain reaching down to the upper part of the pons, when full apneuses immediately developed. To account for the fact that when the vagi are intact apneuses are not seen, they admitted that these nerves have a specific inhibitory influence upon those foci of decerebrate rigidity which control the inspiratory muscles. Hess [1931] also takes the same view of apneusis as Henderson & Sweet, i.e. he regards it as a phenomenon of decerebrate rigidity.

A third hypothesis on apneusis was put forward by Barcroft [1934], who regarded the gasp as the fundamental phenomenon of respiration and apneusis as a modified, interrupted gasp. Barcroft suggested that under normal conditions the gasp is damped or smothered into normal respiration, and that this damping is brought about by afferent inhibitory impulses set up by the very act of inspiration, in the whole respiratory system, including the respiratory muscles. The reflex arcs concerned reach the brain stem at different levels, probably up to the level of the posterior colliculi, and the meaning of "apneusis" is that the afferent inhibitory influences still reaching the "gasping" centre after division of the vagi and section in the pons are not sufficiently powerful to break the gasp, but only to interrupt it half-way.

From the above account it is seen that although the phenomenon of "apneusis" has often engaged the attention of physiologists, there still prevails a great degree of uncertainty as to the mechanism of its production and as to its physiological significance. The object of the present research was to obtain more accurate information about the experimental conditions under which apneusis ensues, and to subject to experiment the various hypotheses put forward.

EXPERIMENTAL

The research was made on cats to the number of forty-six. At the beginning of each experiment the animals were decerebrated by the trephine method, under ether anæsthesia, by intercollicular transection of the mid-brain passing, ventrally, 2 or 3 mm. in front of the pons. The tentorium cerebelli was then removed. For all subsequent sections in the brain stem very sharp cutting instruments were used, and, when necessary, the animal was temporarily placed again under slight ether anæsthesia. Intracranial division of the cerebral nerves was effected by very gradual compression by forceps until they were completely crushed. This method had advantages over acute section, in that it did not seem to disturb the animal, and that it caused very little bleeding.

The same procedure, i.e. crushing by slow gradual compression, was employed for dividing the spinal cord, thus dispensing with the necessity, otherwise, of putting the animal again under anæsthesia during the operation. The respiration was recorded by means of tambours.

The site of the centre which inhibits apneusis

Section of the brain stem immediately behind the posterior colliculi. Section at this level, passing ventrally through the superior border of the pons, caused no appreciable alteration of quiet breathing. The present experiments, therefore, do not support the findings of Markwald [1890], Lewandowsky [1896], Pike & Coombs [1918], nor those of Trevan & Boock [1922], according to whom the operation leads to a deepening and slowing of respiration similar to that observed after vagotomy.

After subsequent bilateral vagotomy, respiration became deeper and slower, but continued regular, and the effect was in no way different from that observed after simple vagotomy with the central nervous system intact. This was particularly well seen in those experiments in which the vagi had been divided before making the brain section. It was then seen that, while, in the limbs, neck and back a full degree of decerebrate rigidity developed, the chest, on the contrary, did not undergo any change and remained quiescent in the expiratory position, while respiration continued to be purely diaphragmatic, as before. Also the rate and depth of breathing remained absolutely unmodified.

With the exception of Lumsden, with whose findings my own agree, all previous authors had found that respiration after vagotomy became apneustic, but attributed the result to removal of the posterior colliculi (Markwald and others) or to exclusion of the red nucleus (Henderson & Sweet). The complete removal of the posterior colliculi and red nucleus was confirmed in all my experiments by microscopic examination by Dr Una Fielding, of the Department of Anatomy, University College, London, to whom I have great pleasure in expressing my gratitude.

Intracranial division of the trigeminus nerve. That respiration continues to be regular after brain section at the level described, and bilateral vagotomy, might be due, according to the findings of Markwald, Asher & Lüsher and Loewy to rhythmical reflex inhibition from the trigeminus, similar to that of the vagus. This view of the trigeminus seems to have been accepted by Barcroft [1934], when he suggests that in order to smother the gasp into a normal respiratory act, a few afferent inhibitory influences out of all those normally reaching the centres are probably alone sufficient, especially those of the vagus and trigeminus.

In cats, under experimental conditions similar to those here described, Lumsden tried the effect of section of the trigeminus, but obtained results of doubtful meaning. In five out of six of his cases, typical apneustic respiration immediately developed, while in one case the respiration did not seem to suffer much, and continued regular.

In view of the importance of this point, it was desirable to repeat the experiments. Division of the nerve was carried out in twelve animals with the brain stem divided behind the posterior colliculi and the vagi cut. The nerves were crushed and divided close to their entrance into the pons. In three, after bilateral division of the nerves, respiration became rather irregular and eventually apneustic, but in all the other nine the double operation was well tolerated and did not cause any appreciable modification of the type of breathing, or of its rate and depth. It seems justifiable, therefore, to ascribe the alterations of respirations obtained after section of this nerve in some of my experiments and in those of previous observers simply to damage of the near parts of the brain stem, and not to the removal of a tonic reflex influence of the trigeminus on inspiration.

Section in the pons. While extirpation of all nerve tissue above the pons leaves the mechanism of quiet breathing to all appearances completely unaffected, a section falling in the pons, not higher than 2 or 3 mm. below its upper limit, leads, when combined with double vagotomy, to the classical development of apneustic respiration. A typical example is given in Fig. 1, where the brain stem had been cut by a section passing dorsally immediately behind the posterior colliculi, but reaching, ventrally, a level about 2·5 mm. lower than the superior border of the pons. It will be seen that, as long as the vagi were intact, respiration continued to be not appreciably different from normal, but immediately became apneustic when the vagi were blocked by a cold thermode. It was the same when the nerves were cut. It was immaterial whether the vagus was cut before or after the section in the pons.

So far, therefore, as the influence of the vagus is concerned, the present experiments do not agree with Lumsden [1923*b*], but do agree with all previous observers, in showing that as long as the vagi are intact, apneustic respiration does not occur even after section of the brain at this level, nor, indeed, at any level in the pons down to the superior limit of the striæ acousticæ.

It was seen above, that in animals breathing regularly after division of the vagi and section immediately above the pons, no tonic inhibitory influence of the trigeminus on inspiration could be detected. The same negative result was obtained after vagotomy and sections of the brain stem at the level described here. The development of apneusis, in fact, was entirely independent of whether the trigeminus was cut or remained intact, as could be judged from the preservation of good corneal and nose reflexes.

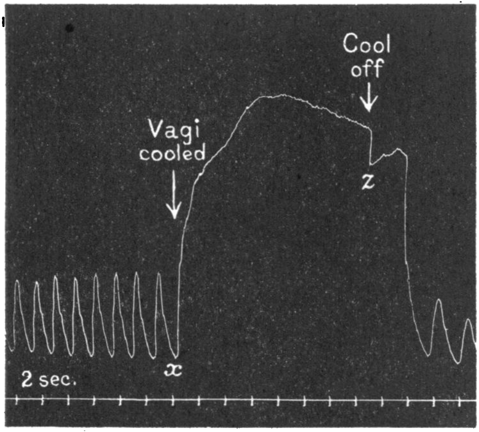

Fig. 1. Abdominal respiration. Section of the brain stem along a plane passing dorsally immediately behind the posterior colliculi; and ventrally 2·5 mm. below the upper border of the pons. Between *x* and *z* the vagi were blocked by cold thermodes. In this and all subsequent figures read from left to right. Time: 2 sec.

Apneusis and afferent nerve roots

Having established the level at which the brain stem has to be cut in order to observe the phenomenon of apneusis, and confirmed the inhibitory influence of the afferent pulmonary vagus upon it, I proceeded to investigate how apneusis itself depended on influences coming from the periphery. If, in fact, the mechanisms here at work were the same as those responsible for the decerebrate rigidity, one would expect to see apneusis, like decerebrate rigidity, disappear, when all afferent channels have been cut off [Sherrington, 1898; Magnus, 1924; Pollock & Davis, 1931; etc.]. On the other hand, if apneusis is a gasp interrupted half-way by afferent inhibitory impulses set up in the periphery by the very act of inspiration, then removal of such inhibitory influences would result in the transformation of apneusis into gasping. To test the latter hypothesis, I endeavoured to make the deafferentation of the respiratory centre as nearly complete as I could in view of the possibility, already hinted at by Barcroft [1934], that if a few afferent channels escape section, they might be sufficient to mask the effect of the abolition of all others.

Section of the dorsal (afferent) roots of the cervical and thoracic cord, and of the last four cranial nerves. After intercollicular decerebration, the spinal cord was exposed from the first cervical down to the 11th thoracic segment, below which it was divided. All dorsal roots above the section were then divided, from the first cervical pair to the 11th thoracic inclusive. This operation did not seem to alter respiration, which was diaphragmatic before, and remained the same after the operation, continuing regular and rhythmical and at a normal rate (Fig. 2). These findings, therefore, disagree with those of **Coombs** [1918], who obtained a marked slowing of the respiratory rate after section of the dorsal roots of the cervical and thoracic cord. Blocking of the vagus, in such a preparation, results in slowing and deepening of breathing of the usual type and degree (Fig. 2).

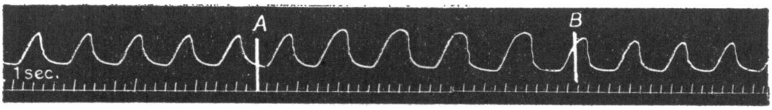

Fig. 2. Abdominal respiration. Intercollicular decerebration. Spinal cord cut below 11th dorsal segment. All dorsal roots of the cord cut above the level of section. Between *A* and *B* cooling of the vagi.

When, as well as the dorsal roots, the phrenic nerves were also cut, in the neck, the costal respiration which resulted showed some small irregularities of rhythm and depth, and the cycle did not seem to proceed so smoothly as the previous diaphragmatic respirations (Fig. 3). My findings, however, do not confirm **Foá**'s observations of the importance for costal respiration of the integrity of the dorsal (afferent) roots of the cervical cord. In dogs and rabbits, **Foá** [1911] divided the vago-

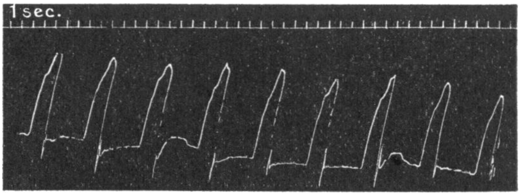

Fig. 3. Costal respiration. Same conditions as in Fig. 2; in addition the phrenic nerves were cut in the neck.

sympathetic, and the phrenics, in the neck, and proceeded to cut the dorsal roots of the cervical cord. He noticed that "the costal respiration which continued regular although modified, after section of the vagi and phrenics, began to alter after division of the first dorsal roots, and when the last two (cervical roots) were cut it became profoundly altered, taking up that irregular type which it maintained after the subsequent sections", namely, "division of the cervical from the thoracic cord, and, later on, division of the bulb from the encephalon"—"The breathing became slower and markedly irregular both in the number and amplitude of the respiratory acts. It was a respiration which could be called ataxic, like the movements of deafferented limbs. It was also an asthenic breathing since its depth was, on the whole, smaller than normal and because the respiratory muscles moved with difficulty, rising slowly, sometimes by jerks, often incompletely, falling back, exhausted, to the expiratory position." I always found that, whether the vagi and the phrenics had been cut or not, section of the dorsal roots, confined to the cervical cord alone, had no detectable influence on quiet respiration.

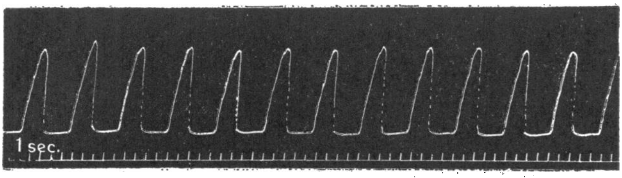

Fig. 4. Abdominal respiration. Intercollicular decerebration: dorsal cervica and thoracic roots and last four cranial nerves cut.

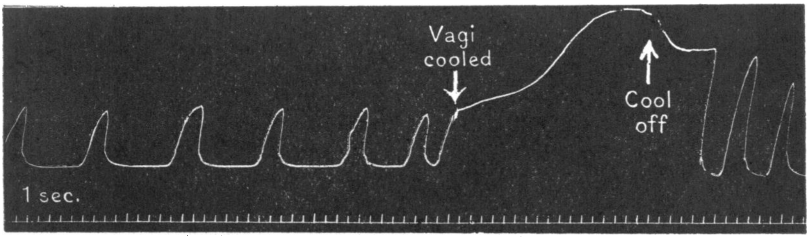

Fig. 5. Abdominal and costal respiration. Section of the brain stem across the pons. Dorsal roots of cervical and thoracic cord and IXth and XIIth cranial nerves cut. Between arrows the vagi were blocked by cold.

In animals with phrenics cut, Gesell & Moyer [1932] sometimes found no change in the type of costal respiration, after section of the last 5 cervical and the first 9 thoracic dorsal roots.

In Fig. 4 is given another experiment in which, in addition to section of the dorsal roots of the cervical and thoracic cord, as already described, the last four cranial nerves were cut at their exit from the skull. It will be seen that even this further interference with afferent impulses from the periphery had remarkably little influence on respiration. After a further section in the pons, a few millimetres below its superior border, combined with double vagotomy, apneuses ensued as usual. An example of this is seen in Fig. 5, in which after sectioning all dorsal roots of the cord, and the IXth and XIIth cerebral nerves, the pons was transected about 3 mm. below its upper border. When the vagus was cooled, apneusis immediately developed.

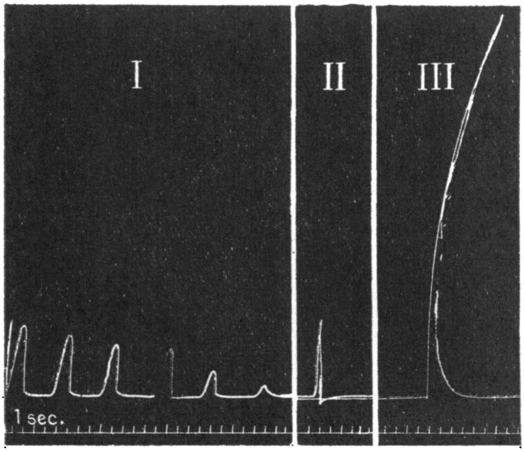

Fig. 6. Abdominal and costal respiration. Intercollicular decerebration. Dorsal roots of cervical and thoracic cord cut. It shows respiration dying down as a result of continued compression of the common carotids and vertebral arteries. Between I and II the arteries were released. II and III were taken 1 min., 30 sec. and 4 min. after I, showing the ability of the deafferented inspiratory muscles still to perform powerful gasps.

The same result was obtained if, in addition, cocaine was applied, beforehand, to the nasal mucosa so as to paralyse the trigeminal nerve endings.

If, after section of the dorsal roots of the spinal cord, acute anæmia was induced in the respiratory centres, by sudden and continued occlusion of the carotid and vertebral arteries, respiration became gasping, as usual (Fig. 6). Gasping was also obtained, as usual, after a low section, in the upper part of the medulla. The above experiments, therefore, show that deafferentation of the respiratory centres leaves the fundamental respiratory cycle practically unaltered—in particular it brings about, by itself, neither apneusis nor indeed gasping, these two modifications of respiration only occurring when in addition to deafferentation of the respiratory centres the usual experimental conditions for their production are obtained.

Intracranial section of the VIIth and VIIIth nerves

It was shown by Magnus [1924], Ranson [1929], Spiegel [1923], Pollock & Davis [1931] that the proprioceptive impulses from the muscles are not wholly responsible for rigidity after decerebration. According to Pollock & Davis [1931], for instance, the forelegs, after complete deafferentation, still show a marked rigidity, which is dependent on the position of the head and disappears only when the labyrinthine influences are abolished.

Very strong rigidity of the forelimbs after section of all the dorsal roots of the cervical and thoracic cord was also observed here, and its dependence upon the position of the head was fully confirmed. Therefore it was probable that in apneusis also the afferent influences still reaching the centres through the VIIth and VIIIth cranial nerves were responsible for its persistence under the experimental conditions described.

That the labyrinthine influences might play a part in the production of apneusis is suggested by the results of Lumsden [1923*a*] after intracranial section of the VIIth and VIIIth nerves, which "usually resulted in prolonged inspiratory spasms and convulsions, followed by gasping respirations alone, or gasping interspersed with a few incoordinated inspiratory spasms", in short, by such phenomena as he observed after section of the brain stem below the "apneustic" centre. Lumsden added that this did not happen when the VIIth and VIIIth nerves were destroyed from the external auditory meatus, and attributed the disturbances to compression of the brain stem by hæmorrhage and to the trauma by traction on the nerves. Since, however, he gives no details of the technique employed for the destruction of the nerve, from the external auditory meatus, it is not certain whether in this case also he destroyed the labyrinthine branch of the VIIIth, or left it intact instead, thus accounting for the negative results.

I repeated, therefore, the experiments of bilateral intracranial division of the VIIth and VIIIth nerves, after transection of the brain stem behind the colliculi. The cerebellum was retracted by smooth hooks, and the auditory nerves crushed in the manner already described. The operation was more difficult than that on the trigeminus, because of the special liability to hæmorrhage; but the latter was soon overcome by plugging the region of the section with small swabs of cotton wool which were left permanently in place. Between the operation of one side and the other 10–15 min. were usually allowed to elapse.

The results were entirely negative. In no case did the spasms and gasps observed by Lumsden follow, and respiration continued quite regular. Nor did the operation interfere at all with the development of typical apneusis when later the pons was cut and the vagi divided. This was true whether the dorsal thoracic roots I–XI were intact or cut.

Attempts to find a relation between the position of the head and the development of apneusis, or its degree, in cats after section of the pons and vagi, with the VIIth and VIIIth nerves intact, were also all negative.

DISCUSSION

A centre which is tonically inhibitory to inspiration, or, in accordance with the views of Henderson & Sweet, a centre which inhibits decerebrate rigidity of inspiratory muscles, is found to lie not in the posterior colliculi [Markwald, Asher & Lüscher, Lewandowsky, Trevan & Boock] or in the red nucleus [Henderson & Sweet], but in the upper region of the pons, as had been suggested by Lumsden [1923*a*]. Henderson & Sweet [1929], in order to cut the rubrospinal tracts, and thus prove that apneusis, like decerebrate rigidity, was dependent on the exclusion of the red nucleus, made a median longitudinal incision in the mid-brain, carrying it down as low as the upper region of the pons. It is, therefore, possible that the apneusis in their findings was due to the fact that with injury of the rubrospinal tracts they also damaged the centre inhibitory to inspiration in the superior region of the pons, or that they cut its connexions with the inspiratory centre, these connexions being probably crossed like the rubrospinal tracts.

Besides the difference between decerebrate rigidity and apneusis, namely, that the latter, unlike the former, does not arise after simple exclusion of the red nucleus, apneusis differs from decerebrate rigidity in its mechanism of production, because it is independent of proprioceptive impulses from the muscles involved, and from the other known sources of decerebrate rigidity, in particular, the labyrinthine influences.

In conclusion, there is between apneusis and decerebrate rigidity a superficial similarity, but the two phenomena are fundamentally different. In the light of our present knowledge there seems to be no reason for regarding apneusis as anything else but an exaggeration of the inspiratory activity. This activity, according to Lumsden, was kept in check by the "pneumotaxic" centre, in the higher region of the pons. The present experiments, however, show that this is not the only mechanism, another being the tonic reflex inhibitory influence of the pulmonary vagus.

Deafferentation of the diaphragm has very little influence indeed on the strength and form of its respiratory cycle; deafferentation of the costal inspiratory muscles, on the other hand, makes their inspiratory contractions somewhat weaker and less smooth than normally. But the respiratory cycle remains fundamentally the same as in the intact animal. This was also true when the last four cranial nerves were severed in addition, so that the only remaining afferent channels still available were reduced to the Vth nerve, or to those fibres of this nerve which had escaped the action of the cocaine. In spite of this nearly complete deafferentation of the respiratory centres, regular respiration continued, while section in the pons caused the usual development of apneusis. If now one accepts Barcroft's theory of respiration, this would mean that the few afferent nerves still reaching the centres were sufficient very effectively to mask the absence of all the others. This is improbable, since the trigeminus, as shown in this paper, has not a tonic inhibitory action on respiration. Moreover, all available evidence shows that the effect on inspiration of inhibitory influences is in proportion to the number of fibres active and the degree of activity in each fibre. Such relation is seen in the action of the vagus, which is relative to the degree of expansion of the lungs, and in the effect on respiration of cutting only one vagus, or by crushing part of each vagus, when the inhibitory effects diminish in proportion.

The interpretation of the normal respiratory cycle, and indeed of apneusis, as reflex modifications of a fundamental gasp, brought about by inhibitory, moderating influences from the periphery, is not supported by experiment.

The results obtained here show that the automatic activity of the respiratory centres is not represented by the gasp, but by the form of the respiratory cycle which is seen after vagotomy in an intact animal. This conclusion finds further support in the work of Adrian & Buytendijk [1931] on the action currents of the brain stem of goldfish, completely isolated and removed from the body, when waves of potential change, almost certainly due to the persisting respiratory activity, were seen to be represented by large and slow curves, very similar to the usual respiratory cycle of the animals, and not by sharp and short discharges, as would have been the case if their activity had now consisted of a series of gasps.

The results show in conclusion that apneusis represents the automatic activity of the respiratory centres, after excision of the upper part of the pons.

SUMMARY

1. Respiration does not undergo any alteration if the posterior colliculi, or the red nucleus, or both, are cut off. After such extirpations vagotomy causes only the usual slowing and deepening.

2. If the section in the brain stem damages or cuts off the upper region of the pons, respiration continues rhythmical as long as the vagi are intact, but immediately becomes apneustic when these are divided.

3. Therefore, in the upper region of the pons there exists a mechanism (Lumsden's "pneumotaxic centre") which, apart from the vagi, rhythmically inhibits inspiration, thus producing regular respiration. This rhythmical inhibitory activity of the pneumotaxic centre is not due to afferent influences from the trigeminus nerve.

4. Apneusis, unlike decerebrate rigidity, is not dependent for its production on proprioceptive reflexes from the muscles of respiration or from the labyrinthine nerve. This, together with the fact that it is not brought about by extirpation of the red nucleus, disagrees with Henderson & Sweet's and Hess's contention that apneusis and decerebrate rigidity are two manifestations of the same fundamental process.

5. Regular, smooth, respiration and apneusis are still observable after such thorough deafferentation of the respiratory centres as would leave them still connected only with a few territories of distribution of the trigeminus. It is more reasonable, therefore, to regard them as expressions of the automatic activities of the centres concerned, rather than as the results of peripheral inhibitory influences, checking or interrupting a fundamental gasp.

The expenses of this research were defrayed by a grant from the Dixon Fund of the University of London, and I have pleasure in expressing here my indebtedness and gratitude.

REFERENCES

Adrian, E. D. & Buytendijk, F. J. J. (1931). *J. Physiol.* **71**, 121.

Asher, L. & Lüscher, Fr. (1899). *Z. Biol.* **38**, 499.

Barcroft, J. (1934). *Features in the Architecture of Physiological Function.* Cambridge University Press.

Coombs, E. C. (1918). *Amer. J. Physiol.* **46**, 459.

Foà, C. (1911). *Arch. Fisiol.* **9**, 453.

Gesell, R. & Moyer, C. A. (1932). *Amer. J. Physiol.* **101**, 40.

Henderson, V. E. & Sweet, T. A. (1929). *Ibid.* **91**, 94.

Hess, W. R. (1931). *Die Regulierung der Atmung.* Leipzig: Georg Thieme.

Langendorff, O. (1888). *Arch. Anat. Physiol., Lpz.*, 282, 303.

Lewandowsky, M. (1896). *Ibid.* 483.

Loewy, A. (1888). *Pflüg. Arch.* **42**, 245.

Lumsden, T. (1923*a*). *J. Physiol.* **57**, 153, 354.

Lumsden, T. (1923*b*). *Ibid.* **58**, 81, 111.

Magnus, R. (1924). *Körperstellung.* Berlin: Julius Springer.

Markwald, M. (1887). *Z. Biol.* **23**, 149.

Markwald, M. (1890). *Ibid.* **26**, 259.

Pike, F. H. & Coombs, H. C. (1918). *Amer. J. Physiol.* **45**, 569.

Pollock, L. J. & Davis, L. (1931). *Ibid.* **98**, 47.

Ranson, S. W. (1929). *Arch. Neurol. Psychiat., Chicago*, **22**, 265.

Sherrington, C. S. (1898). *J. Physiol.* **22**, 319.

Spiegel, E. A. (1923). *Zur Physiologie und Pathologie des Skelettmuskeltonus.* Berlin: Gustav Fischer.

Teregulow, A. G. (1929). *Pflüg. Arch.* **221**, 486.

Trevan, J. & Boock, E. (1922). *J. Physiol.* **56**, 331.