# THE EFFECTS OF SHALLOW BREATHING.

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The question as to the means by which the respiration is regulated so as to harmonise with the metabolism of the body has been the subject of many investigations. These have in the main led to two theories as to the nature of this regulation, namely (*a*) that it is a matter of reflex nervous control, and (*b*) that it is a chemical regulation.

Following on the classical research of Hering and Breuer(1) in 1868 the first theory became predominant, but a paper published by Haldane and Priestley(2) in 1905 led to a recognition of the fundamental predominance of the chemical regulation. A period then followed in which the nervous mechanism of the regulation of respiration did not receive so much attention and was indeed largely neglected until Haldane and Mavrogordato(3) investigated the relation between the chemical regulation and the Hering-Breuer reflex. Their results clearly demonstrated for the first time the connection between the nervous control and the chemical control.

Recent investigations carried out in this laboratory into the cause of the symptoms met with in men suffering chronically from the effects of gas poisoning, and from a group of symptoms known as D.A.H., "Soldier's Heart," "Irritable Heart," "Effort Syndrome," etc., have turned our attention to the importance of the regulation of the depth of respiration and have led to results which are of much physiological importance. A summary of these investigations in their more practical aspects was given in a report circulated for official purposes[1]. The present paper is a full account of one part of that work, namely the experimental investigation of the effects of shallow breathing.

[1] *Reports of the Chemical Warfare Medical Committee.* No. 5. "The Reflex restriction of respiration after gas poisoning."

The reflex first described by Hering and Breuer has been very widely misunderstood, and indeed as described in the physiological text-books of recent years is made to appear as a reflex singularly perfectly adapted to prevent the maintenance of efficient respiration. Reference to Hering and Breuer's original paper and the tracings therein contained shows clearly that the inspiratory and expiratory impulses from the centre are sent out in response to afferent impulses passing up the vagus and initiated by the state of collapse or inflation of the lung. Hering and Breuer therefore correctly speak of an automatic regulation of the respiratory movements, but their experiments afford no explanation of the means by which the breathing is kept slow and deep at one time and fast and shallow at another. There was indeed no clear understanding of how the regulation of the breathing by the Hering-Breuer reflex can be reconciled with the obvious fact that the breathing is also normally so regulated as to be deep or shallow according to the needs of the body until the question was investigated by Haldane and Mavrogordato(3). Soon after Hering and Breuer's discovery the question of this reflex was taken up by Head(4) who worked in Hering's laboratory and who employed for the first time a new method of obtaining respiratory records, namely, recording the contractions of a slip of a rabbit's diaphragm. Unfortunately this method resulted in focussing attention exclusively on the inspiratory part of the respiratory acts, so that Head's interpretation of the nervous control of breathing leaves out of account the expiratory side of the reflex and thus gives an entirely erroneous impression.

Owing to this paper and to the misleading accounts of the work of Hering and Breuer which became current, understanding of the meaning and importance of this reflex became obscured and when Haldane and Priestley showed the importance of the part played by $CO_2$ in regulating the ventilation of the lungs, interest in the nervous control of the depth of respiration became lessened. It also happened that the experiments of Haldane and Priestley seemed to indicate that it was a matter of no great importance whether the lung ventilation was effected—within limits—by slow and deep or by rapid and shallow respirations.

Later, when Haldane and Mavrogordato again investigated the Hering-Breuer reflex and showed that it is controlled by the $CO_2$ tension of the blood they still regarded the depth of respiration as a matter of secondary importance, and indeed some experiments performed by Haldane seemed to show that the ventilation of the lungs could be

equally well maintained at respiratory rates varying from 2 to 60. Haldane(5) found that if the rate of respiration was voluntarily maintained at either of these figures—allowing the depth to adjust itself—the alveolar $CO_2$ remained constant; but it must be noted that these experiments were of very short duration—about 2 minutes—and that it is practically certain that periodicity in the depth of breathing occurred but escaped notice.

Haldane and Priestley(2) had also found that alveolar $CO_2$ remained constant while the rate of respiration was voluntarily varied from 9 to 30 per minute, the depth being allowed to adjust itself. When, however, we came to investigate the respiratory phenomena of soldiers suffering from chronic effects of gas poisoning and "Irritable heart" we were forced to reconsider the interpretation of these experiments.

As is well known the condition known as "Irritable heart" is characterised by breathlessness on exertion, rapid pulse, fainting attacks, giddiness, exhaustion, lassitude, headache, irritability, etc. We found that the patients subject to this condition show invariably a remarkable type of breathing. The respiration is rapid—from 20 to 60 or more per minute—and shallow—250 to 350 c.c. or thereabouts—and on exertion the rate increases very abnormally while the increase in depth is abnormally small. It was also found that their alveolar $CO_2$ is abnormally low[1]. Hunt and Price Jones[2] drew attention to the occurrence of polycythæmia in chronic cases and Barcroft[3] described a condition of "acidosis" in these patients. All these conditions are such as would arise if the patient were short of oxygen either intermittently or continuously, and this conclusion is confirmed by the very definite result obtained when they breathe air enriched with oxygen. In fact the patients are in the same state as a normal individual at high altitudes where the diminished oxygen tension of the inspired air produces the same series of effects. We found that when they are breathing oxygen the patients are able to do an amount of muscular work which is quite beyond their powers when they breathe air only, and they state emphatically that the oxygen relieves their subjective symptoms. In many of the patients exhibiting this condition there is no sign of any serious structural change in heart and lungs. All these patients however show the shallow rapid type of breathing.

[1] C. G. Douglas' report forwarded to G.H.Q. France. Drury. Medical Research Committee Report upon Soldiers returned as cases of "Disordered action of the heart," p. 19. 1917.

[2] Report forwarded to G.H.Q. France.

[3] Barcroft. C.W.M.C. report.

Since, then, some of their symptoms were clearly due to want of oxygen and since a constant abnormality found was the peculiar type of breathing, the question arose as to whether this type of respiration could be the cause of the oxygen-want, although the experiments of Haldane mentioned above had apparently indicated that this could not be the case.

In order to investigate this matter we determined to imitate the condition of shallow breathing by restricting the volume of air per breath inhaled by normal individuals.

The apparatus used consisted of a collapsible bellows of india-rubber of suitable size. Two such "concertinas" were used in different experiments—one had a capacity when distended of $3\frac{1}{2}$ litres; the other of $\frac{1}{2}$ litre. The top and bottom of the concertina were formed by rigid pieces of wood, the top being suspended in a fixed position on a suitable stand. To the middle of the bottom plate was fixed a metal rod which passed through a metal tube and acted as a guide, thus ensuring that the only movement of the bottom of the concertina was a vertical up and down movement. Surrounding the tube through which the guide rod passed was another tube which could be clamped at any height desired by means of a screw, so that the downward excursion of the bottom of the concertina was limited. Fixed to the bottom plate of the concertina was a writing pen which recorded its movements. The whole was mounted on a stand provided with levelling screws; and the three uprights by means of which the concertina was suspended were threaded at the top and provided with nuts so that the large and small concertinas could be readily exchanged and the top in each case levelled as required. Through the top plate of the concertina passed a short metal tube, of 1 inch internal diameter and bent to a right angle. This tube connected the concertina to the remainder of the apparatus which is now to be described. (See Fig. 1.)

To a mouthpiece was attached a tube of 1 inch diameter provided with inspiratory and expiratory valves; the whole being so proportioned as to reduce the dead space as much as possible. From the part of this tube opposite the mouthpiece and between the inspiratory and expiratory valves a small tube $\frac{1}{4}$ inch in diameter led to a delicate tambour which thus became distended on expiration and collapsed on inspiration. This tambour was made to control a make and break mercury contact in an electrical circuit which included a powerful electro-magnet the use of which is described below. This arrangement was described by Yandell Henderson(6). The lower end of the tube carrying the mouthpiece

below the inspiratory valve was connected by means of a piece of rubber tubing as short as conveniently possible to another short piece of 1 inch metal tubing bent at a right angle and supported on a wooden stand. The upright part of this tube contained a mica valve which opened on inspiration, and on the upper side of the horizontal part was the seating of another mica valve which closed on inspiration. To this valve was firmly attached an iron armature situated just below the electro-magnet mentioned above. The open end of the horizontal part of the tube carrying the valves was attached to the tube leading through the top of the concertina.

The effect of the arrangement described above is as follows: during inspiration air is drawn from the concertina through the inspiratory valves to the lungs. At the same time the slight negative pressure is enough to cause the tambour to collapse and so break the circuit of the electro-magnet. Consequently the armature attached to the valve on the horizontal tube is not attracted and this valve remains closed. The only air therefore which can be inspired is that contained in the concertina and its amount is determined by the height at which the stop supporting the bottom of the concertina is set. On expiration the expired air escapes through the expiratory valve of the mouthpiece, and at the same time the inspiratory valves close. Simultaneously the slight expiratory pressure distends the tambour, which thus closes the circuit of the electro-magnet. The valve below the magnet is thus drawn up and thus a clear way is made from the outside air to the interior of the concertina. The bottom of the concertina falls by its own weight to the stop and thus a definite quantity of fresh air is drawn in ready for the next inspiration. The same sequence of events can then be repeated indefinitely. It should be clear therefore that the effect of the apparatus is to provide a constant and limited quantity of fresh air for each inspiration and at the same time give a quantitative record of the respiration, for the concertina may easily be calibrated.

A further arrangement consists of a wide glass tube which is fixed round the electro-magnet and the valve controlled by it. The ends of this glass tube are closed by rubber bungs to make airtight joints. Through the upper of these rubber bungs a smaller glass tube about $\frac{3}{4}$ inch in diameter passes. This arrangement makes it easy to supply fresh air, oxygen or any desired gaseous mixture of known composition to the concertina. The annexed Fig. 1 shows the apparatus. The apparatus can of course also be used to obtain a record of the normal breathing. All that is necessary is to use a concertina of greater capacity than the

volume of the respirations. The record is of course of the inspirations only. The expirations are not recorded.

Miller has published several very interesting papers in which he has worked out the structure of the lungs (7). He has shown that each terminal bronchiole ends in a cavity which he terms an atrium. Out of the atrium open several air sacs. The walls of both atrium and air cells are provided with alveoli. The atria act in effect as distributing chambers and ensure an even division of the inspired air to alveoli (containing by far the greater portion of the air in the lungs) which lie beyond the atria. Were it not for this arrangement the air cells near the termination of the

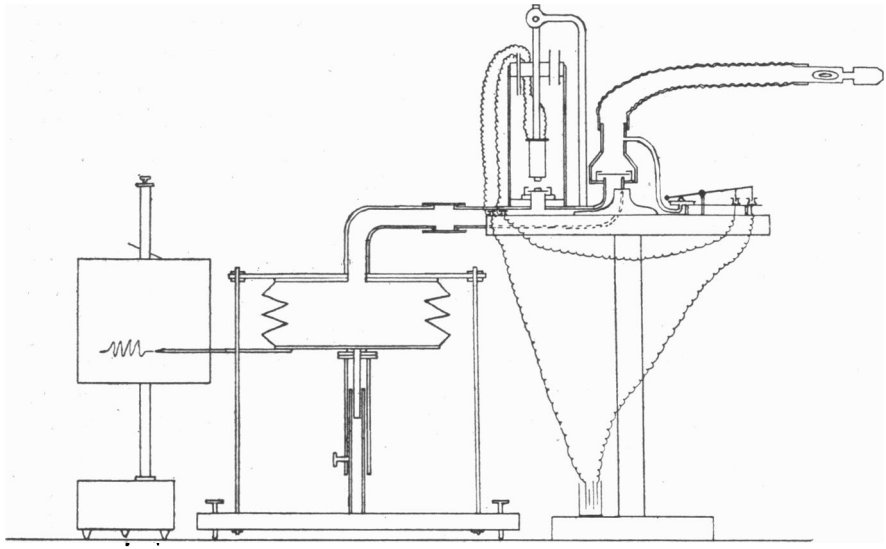


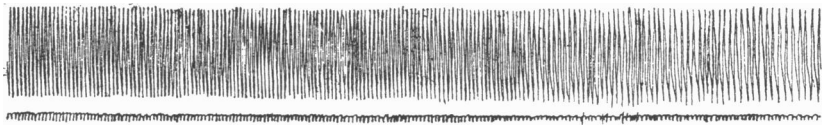
Fig. 1. "Concertina" apparatus for continuous record of respiration.

bronchiole would receive a greater amount of the entering air in proportion to their nearness, and consequently the ventilation of the air sacs would be very uneven, and the fact that the deeper alveolar air is uniform in composition, as Haldane and Priestley found, would be unintelligible.

At first we expected that by suitably adjusting the concertina so as to limit the volume of air inspired we might be able to so restrict the air entry as to cause the ventilation not to extend appreciably beyond the atria, leaving the air sacs full of stagnant or nearly stagnant air. If this were the case we might be able to detect a considerable difference between the respiratory quotients of the expired air and the deep alveolar

air: for it is clear from the shapes of the dissociation curves of blood for $CO_2$ and $O_2$ that there is practically no limit to the extent to which over-ventilation can remove $CO_2$ from the blood circulating through the well ventilated atria on the one hand, while on the other the amount of oxygen given up to this blood cannot be increased to any appreciable extent by increasing the ventilation beyond the normal. The respiratory quotient in the atria should therefore be high if the hypothesis as to relatively excessive ventilation of the atria were correct. In the air-sacs, however, where the air is supposed to be stagnant, oxygen will continue to be taken up by the blood after equilibrium is established between blood and air as regards $CO_2$. The respiratory quotient of the air-sac air or deep alveolar air should thus fall.

When we came to perform the experiment however this expectation



All curves are read from left to right. Inspiration being the upstroke.

Fig. 2. Subject J. C. M. Time marker = seconds.

TABLE I. Subject J. G. P.

| Vol. per resp. (c.c.) | Resp. per min. | Vol. per min. (ltrs) | Expired air | | | Deep alveolar air | | | Bar. |
|---|---|---|---|---|---|---|---|---|---|
| | | | CO % | O % | R. Q. | CO % | O % | R. Q. | |
| 161 | 106 | 17·1 | 2·01 | 18·44 | ·76 | 5·71 | 14·06 | ·79 | 758 |
| 155 | 106 | 16·4 | 2·10 | 18·35 | ·76 | 6·22 | 12·85 | ·72 | 758 |
| 147 | 69 | 10·1 | 2·97 | 17·38 | ·78 | 5·63 | 13·55 | ·71 | 758 |
| 105 | 118 | 12·3 | 2·36 | 17·90 | ·71 | 5·42 | 14·45 | ·79 | 760 |

was not borne out. With the concertina so adjusted that the volume of air taken in at each inspiration was only slightly below the normal the rate of respiration quickened correspondingly but there was no other apparent change. The lung ventilation could it seemed, be carried on indefinitely with the new rate and volume of respiration. There was no definite and constant difference between the respiratory quotients of the expired air and the deep alveolar air. Fig. 2 gives a record obtained under these conditions and Table I gives the respiratory quotients of expired air and deep alveolar air at various rates and depths of inspiration.

The concertina was then closed down further but no particular difference in the result was noticed till the inspired air was so much limited in amount that the rate of respiration went up to about 100 to

150 per minute. Under these circumstances the experiment was at first rather difficult to perform because the apparatus only fixed the amount of air that could be inspired, but constant attention and exercise of the will was required to overcome the natural tendency of the Hering-Breuer reflex and make the volume of expiration correspond to the volume of inspiration. However this became comparatively easy with a little practice, and one could prevent the lungs becoming more and more inflated or deflated during an experiment. The result was continually increasing discomfort and sense of suffocation until the experiment had to be stopped. The respiratory quotient of the deep alveolar air fell to 0·5 and that of the expired air also fell.

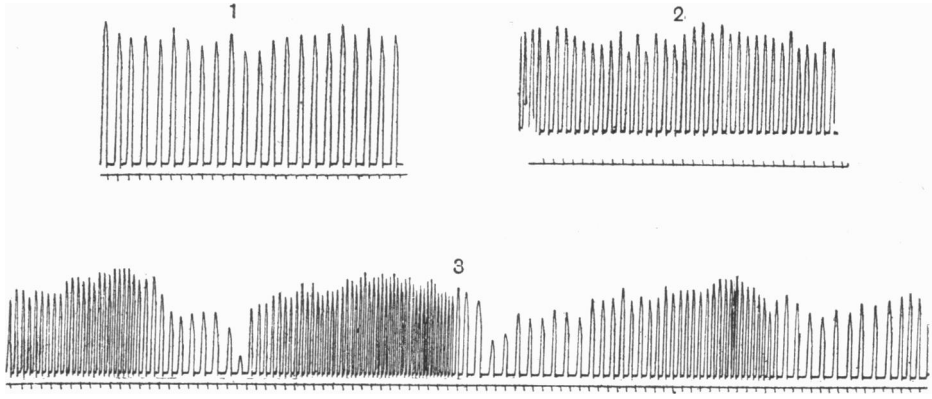



Fig. 3. Subject J. G. P. Time marker = 2 seconds.

It appeared then that when the concertina was closed down to this extent the result was not to prove any difference between the ventilation of the atria and the air-sacs but simply to cause asphyxia. The volume per respiration in these experiments was about 120 to 150 c.c. The results are set out in Table II.

TABLE II.

| Subject | Vol. per resp. (c.c.) | Resp. per min. | Vol. per min. (ltrs) | Expired air $CO_2$ % | Expired air $O_2$ % | Expired air R. Q. | Alveolar air $CO_2$ % | Alveolar air $O_2$ % | Alveolar air R. Q. | Bar. |
|---|---|---|---|---|---|---|---|---|---|---|
| J. G. P. | — | 108 | — | — | — | — | 7·23 | 9·02 | ·54 | — |
| | 64 | 116 | 7·47 | 1·03 | 19·21 | ·50 | 6·83 | 8·03 | ·47 | — |
| | 141 | 102 | 14·47 | 2·03 | 18·29 | ·71 | 6·38 | 7·77 | ·64 | 758 |
| | 159† | 156 | 24·9 | 2·32 | 17·73 | ·67 | 7·40 | 10·76 | ·67 | 755 |
| J. C. M. | 142 | 116 | 16·44 | 1·72 | 18·66 | ·69 | 6·61 | 12·62 | ·75 | 764 |
| | 115 | 80 | 9·2 | 1·02 | 19·45 | ·64 | 6·79 | 8·56 | ·48 | — |

† Doing light work. Cyanosis and periodic breathing on stopping.

On stopping the concertina breathing, however, it was noticed that there was a slight tendency to periodic breathing. This was an indication that the effect of the restricted breathing was something more than simple asphyxia.

We therefore began a new series of experiments in which the concertina was first adjusted so as to allow a full normal inspiration and was later closed down very gradually step by step. The breathing from the concertina at each stage was continued for a considerable time. As before there was at first no change beyond increase in rate of respiration corresponding to the restricted volume. But when the restriction in volume was carried to a very slight degree beyond a certain point a new phenomenon appeared. After breathing for some time with the concertina thus adjusted periodicity in the respiration began to appear. At first slight variations in the rate of respiration became noticeable, but with proper adjustment of the concertina the periodicity became accentuated and periods of complete apnœa alternated with periods of extremely rapid and urgent respiration. Owing of course to the restriction in volume caused by the concertina the periodicity manifested itself rather by changes in rate than by changes in volume. Fig. 3 gives a record showing this effect.

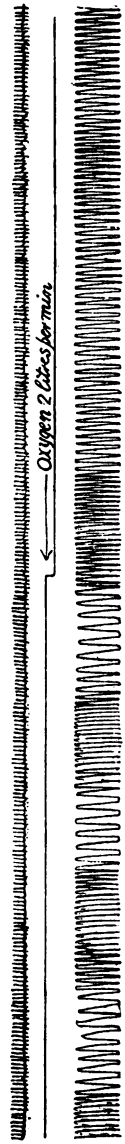



Fig. 4. Subject J. G. P. Time marker = seconds.

The clinical type of periodic respiration, *i.e.* Cheyne-Stokes breathing, was investigated by Pembrey and Allen (8), who showed that the periodicity was abolished and regular breathing restored by adding oxygen or $CO_2$ to the air inspired or by diminishing the oxygen of the inspired air. They attributed the periodicity to diminished excitability of the nervous system. Later the subject was further investigated by Douglas and Haldane (9) who proved that periodic breathing is unmistakeable evidence of anoxæmia. Confirmation of the conclusion that the periodic breathing produced by our concertina experiments was due to anoxæmia was at once obtained on adding a small proportion of oxygen to the air entering the concertina. The effect of this was to abolish completely the periodicity and restore regularbut rapid breathing. Fig. 4 reproduces a record showing this result.

The periodic breathing produced by restriction of the volume of air entering the lungs could be continued more or less indefinitely without discomfort, the periods gradually lengthening as $CO_2$ was washed out of the blood.

TABLE III.

| Subject | Vol. per resp. (c.c.) | Resp. per min. | Vol. per min. (ltrs) | Expired air CO % | Expired air O % | Expired air R. Q. | Alveolar air CO % | Alveolar air O % | Alveolar air R. Q. | Bar. | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| J. G. P. | 192 | 40 | 7·67 | 2·82 | 17·9 | ·9 | — | — | — | — | Mixed hypopnœa and hyperpnœa |
| | 190 | 26 | 4·90 | 2·98 | 17·53 | ·86 | 5·05 | 14·66 | 0·76 | — | Hypopnœic period |
| | 218 | 60 | 13·15 | 2·67 | 18·38 | 1·04 | 4·63 | 16·36 | 1·00 | — | Hyperpnœic period |
| | 242 | 25 | 6·30 | 2·35 | 17·82 | ·7 | — | — | — | 766 | Hypopnœa |
| | 225 | 60 | 13·50 | 2·53 | 18·10 | ·86 | 4·4 | 16·20 | ·92 | 766 | Hyperpnœa |
| | 250 | 64 | 16·99 | 2·71 | 17·82 | ·83 | 4·55 | 16·62 | 1·06 | 766 | ,, |
| | 220 | 20 | 4·38 | 2·56 | 17·28 | ·64 | 5·95 | 11·54 | ·57 | 766 | Hypopnœa |
| J. C. M. | — | — | — | — | — | — | 6·6 | 9·82 | ·53 | 759 | ,, |
| | — | — | — | — | — | — | 5·29 | 15·50 | ·96 | 759 | Hyperpnœa |
| | — | — | — | — | — | — | 6·38 | 11·40 | ·61 | 759 | Hypopnœa |
| | — | — | — | — | — | — | 6·00 | 13·08 | ·71 | 759 | ,, |
| | — | — | — | — | — | — | 4·88 | 16·11 | 1·00 | 759 | Hyperpnœa |

TABLE IV. (Subject J. G. P.)

| Duration in mins. | Vol. per resp. (c.c.) | Resp. per min. | Vol. per min. (ltrs) | Expired air $CO_2$ % | Expired air $O_2$ % | Expired air R. Q. | Alveolar air $CO_2$ % | Alveolar air $O_2$ % | Alveolar air R. Q. | Bar. | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ¼ | — | 60 | — | 2·0 | 18·26 | ·68 | — | — | — | 756 | — |
| 5 | 173 | 68 | 11·78 | 2·27 | 18·75 | ·93 | — | — | — | 756 | — |
| 27 | 212 | 54 | 11·43 | 2·12 | 18·54 | ·84 | — | — | — | 756 | — |
| 38 | — | — | — | — | — | — | 4·83 | 16·23 | 1·02 | 756 | — |
| 60 | 192 | 40 | 7·67 | 2·82 | 17·90 | ·9 | — | — | — | 756 | Breathing periodic |
| 103 | 190 | 26 | 4·9 | 2·98 | 17·53 | ·86 | — | — | — | 756 | Hypopnœic period |
| 108 | 218 | 60 | 13·15 | 2·67 | 18·38 | 1·04 | — | — | — | 756 | Hyperpnœic period |
| 144 | — | — | — | — | — | — | 5·05 | 14·66 | ·76 | 756 | Hypopnœic period |
| 148 | — | — | — | — | — | — | 4·63 | 16·36 | 1·00 | 756 | Hyperpnœic period |

The expired air and alveolar air were analysed both in hypopnœic and hyperpnœic periods and the results are given in Table III.

In Table IV are given the figures from an experiment in which the

restricted breathing was continued for a longer time. The table shows the gradual onset of periodic breathing and also shows that the oxygen percentage of the alveolar air was abnormally high just prior to the beginning of the periodicity, and also, at a later stage, during both hypopnœic and hyperpnœic periods.

How then are we to explain the undoubted fact that the subject of the experiment was suffering from anoxæmia at a time when his alveolar air contained a higher percentage of oxygen than normal?

Haldane and Priestley in the paper referred to above showed that in any individual at rest the alveolar $CO_2$ and $O_2$ are remarkably constant and that even a slight change in the alveolar $CO_2$ has a very great effect on the breathing, a rise of 0·2 p.c. in the alveolar $CO_2$ doubling the ventilation of the lungs. They assumed that this constancy extends to the alveolar air of all parts of the lung. Their experiments however did not exclude the possibility that the "alveolar air" as obtained and analysed by them was an average sample of alveolar air of different composition in the different parts of the lungs.

Now in a very interesting paper on the mechanism of respiration in man Prof. A. Keith(10) states that:

(1) The lung is composed of elements of varying degrees of extensibility. Hence the expansion of its parts is unequal during inspiration. The lung may be divided into three zones of different extensibility—(*a*) the root zone, (*b*) the intermediate zone, and (*c*) the subpleural zone.

(2) The lungs do not expand equally in all directions but execute a certain definite movement during inspiration. The expansion of the lung is a regulated act resembling the opening of a Japanese fan rather than the distension of a bladder when the external pressure is reduced. It does not take place instantaneously and equally throughout and consequently part of the lung may be in a state of partial or almost complete disuse.

If then the circulation of the blood is fairly uniform both through the well ventilated and badly ventilated parts of the lungs the result must be that the arterial blood going to the heart must be a mixture of blood one part of which has been exposed to fresh air in greater degree than the other part. Now Christiansen, Douglas and Haldane(11) have worked out the dissociation curve of blood for $CO_2$ and the effect thereon of varying degrees of saturation of the hæmoglobin with oxygen. Their results show that for the conditions existing in the lungs the curve of dissociation of blood for $CO_2$ approximates for a long distance to a straight line rising somewhat steeply. Fig. 5 gives the dissociation curves of blood for $O_2$ and $CO_2$. Reference to Fig. 5 shows that for the

tensions of $O_2$ and $CO_2$ which govern the gaseous exchange between blood and air in the lungs the $O_2$ curve and the $CO_2$ curve are entirely different and that diminished respiratory removal of $CO_2$ from one part of the blood in one region of the lung may be compensated for by increased removal of $CO_2$ from another part of the blood in another region of the lung. Thus it follows that despite uneven ventilation of the lungs the average $CO_2$ tension of the arterial blood is maintained at its normal level. Also just as the $CO_2$ tension of the arterial blood reaching the respiratory centre is the average of the $CO_2$ tensions of blood from well and badly ventilated parts of the lungs, so also is the $CO_2$ in the "alveolar air" an average from alveolar air of higher and lower $CO_2$ content in

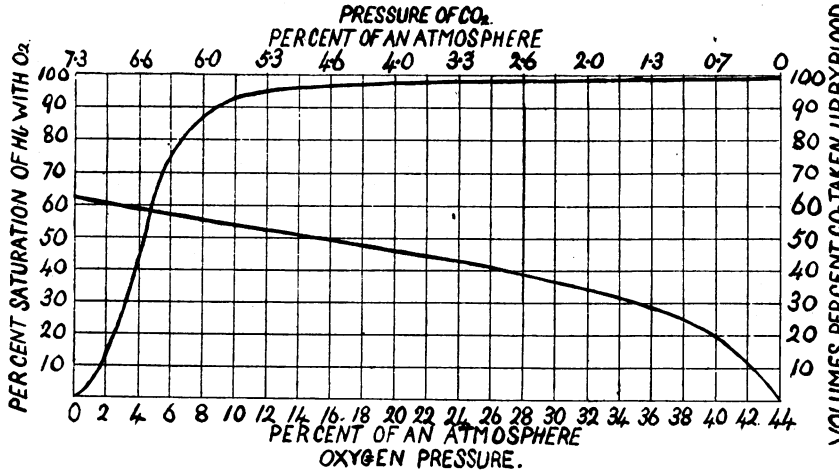



Fig. 5. Dissociation curves of blood for $CO_2$ and oxygen.

different parts of the lungs. The one average is correlated with the other and thus it is that so long as the average alveolar $CO_2$ remains at its normal level the ventilation of the lungs, as governed by the activity of the respiratory centre, also remains, *ceteris paribus*, at its normal amount.

Turning now to consideration of the oxygen: in the badly ventilated parts of the lung the volume of blood flowing per minute will be greater in relation to the amount of fresh air entering these parts than in the well ventilated regions. Hence the blood will take up more of the oxygen and the percentage of oxygen in the relatively stagnant alveolar air of the badly ventilated parts will fall below the average. But the figure shows that the dissociation curve of blood for $O_2$ is totally different from the $CO_2$ curve, and that as the oxygen in the stagnant air falls the per-

centage saturation of the blood passing through these alveoli must also fall greatly. But for oxygen pressures above about 88 mm. Hg. the curve is very flat. Hence any increase of ventilation beyond the amount sufficient to keep the alveolar $CO_2$ at this level is quite unable to add appreciably to the amount of oxygen taken up by the blood. Thus it follows that in the well ventilated parts of the lungs firstly the increased ventilation is incapable of yielding an excess of oxygen to the blood sufficient to compensate for the lack of oxygen in the blood from the badly ventilated parts and secondly, since more than the average amount of fresh air is passing through these alveoli while no more than the normal amount of oxygen is being absorbed by the blood, the air expired from these well ventilated alveoli must be abnormally rich in oxygen. Thus the average composition of the whole alveolar air may remain constant as regards percentage of oxygen, as is normally the case, or, when the breathing is abnormally shallow, the oxygen content of the average alveolar air may rise above the normal level even though at the same time the arterial blood is inadequately supplied with oxygen.

This, then, is the explanation of the figures obtained on analysing the alveolar air during restricted breathing from the concertina as described above; and similar effects must ensue in the case of "Irritable heart" patients who suffer from shallow breathing. If the shallow breathing from the concertina is prolonged a complicating factor comes into play. As shown above the restricted breathing produces some degree of anoxæmia and it is well known that one result of anoxæmia is lowering of the threshold for $CO_2$ of the respiratory centre. Hence, when the restricted breathing is prolonged, the alveolar $CO_2$ gradually falls on the average in the same manner as when the anoxæmia is consequent on lowered barometric pressure. Also if the total ventilation per minute is measured it is found that it is increased by the shallow breathing. This is an obvious consequence of both the relative increase of the dead space and the lowered threshold for $CO_2$.

The discovery that the composition of the air in individual alveoli may differ considerably from that of the mixed alveolar air throws an important new light on various phenomena connected with respiration and the blood gases. Thus it has been found by Fredericq and by Krogh that in experiments with the aerotonometer the oxygen tension of the arterial blood during rest is always lower than in alveolar air, whereas Douglas and Haldane, working with the carbon monoxide method, found the oxygen tension in the arterial blood the same as in the alveolar air, as ought to be the case if the oxygen is passing inwards

by simple diffusion. The difference between the results by the two methods was attributed by Douglas and Haldane to the arterial blood having, between the lungs and the carotid artery, lost a slight amount of free oxygen owing to the presence of "reducing substances." This explanation is now quite unnecessary. Evidently the carbon monoxide method gives the average of the oxygen tension in the blood leaving individual alveoli, while the aerotonometer method gives the oxygen tension of mixed arterial blood; and the two values are, as explained above, not the same. From the want of agreement between the two values it also seems to follow that even during normal breathing the ventilation of different parts of the lungs is not even. The slightly increased proportion of oxygen found in the arterial blood of animals during muscular exertion or forced artificial respiration is probably due to more even ventilation of the lungs.

It is evident also that at diminished atmospheric pressures the effects of uneven ventilation will, other things being equal, be exaggerated. In this way we can understand the fact that, as was very clearly shown by Miss Fitzgerald, even a comparatively small diminution of atmospheric pressure has a marked influence on the hæmoglobin percentage and alveolar $CO_2$ pressure in persons living under the diminished pressure.

The observation that the shallow breathing patients referred to above suffer from intensified symptoms at night led us to consider the question of the effect of posture on the respiration.

In order to obtain quantitative records of the respiration in the recumbent as contrasted with the upright posture we arranged an apparatus in which the breathing circuit comprised a mouthpiece with inspiratory and expiratory valves which were connected to the two ends of a galvanised iron cylinder of about 50 litres capacity. The end of this cylinder remote from the mouthpiece was also connected by a T-piece and rubber tube to the concertina or other recording apparatus. If the concertina was used it was connected direct to the cylinder, the valves and electromagnet, etc., not being used. Thus true records of expiration were obtained as well as records of inspiration. A soda-lime purifier could also be introduced into the circuit if desired, or the cylinder could be filled with oxygen or other gaseous mixture in place of air. The size of the cylinder (50 litres) allowed of a fair length of record of the respiration being obtained before the effect of accumulating $CO_2$ or diminishing oxygen became noticeable. The whole apparatus, except the recording part, was slung from the ceiling so that the mouthpiece could be comfortably adjusted either in the lying or sitting position. Of course if it

was not desired to obtain records of expiration as well as inspiration the complete concertina apparatus including valves and magnet could be used in place of the cylinder and a continuous record obtained.

The following tracings show the records both sitting and lying.

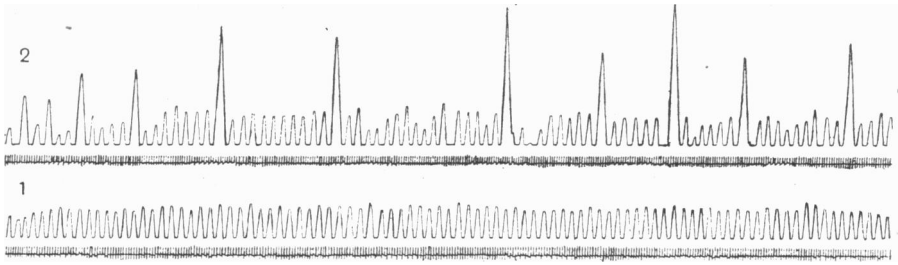



Fig. 6. Subject J. C. M. Unlimited concertina. Time in seconds.
1. Sitting. 2. Lying.

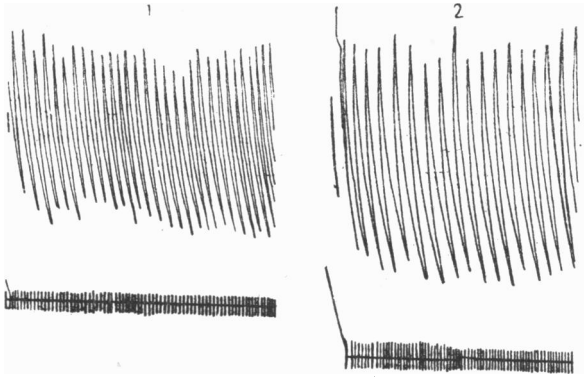



Fig. 7. Subject J. S. H. Rebreathing in and out of 50 litre cylinder. Time marker = 2 seconds. 1. Sitting. 2. Lying.

The tracings show, in accord with many other experiments, not quoted, that on lying down the respiration becomes slower and deeper, but they also show that a remarkable type of periodic breathing becomes evident in the case of some subjects when the recumbent position is assumed. In other subjects the breathing becomes slower and deeper but remains perfectly regular.

Thus it seems that the effect of lying down is to accentuate the irregularity in the expansion of the lungs which was referred to above and thus cause a definite though slight anoxæmia even in normal

individuals. The probability that this is the case is increased when we consider the annexed tracings from X-ray photographs.

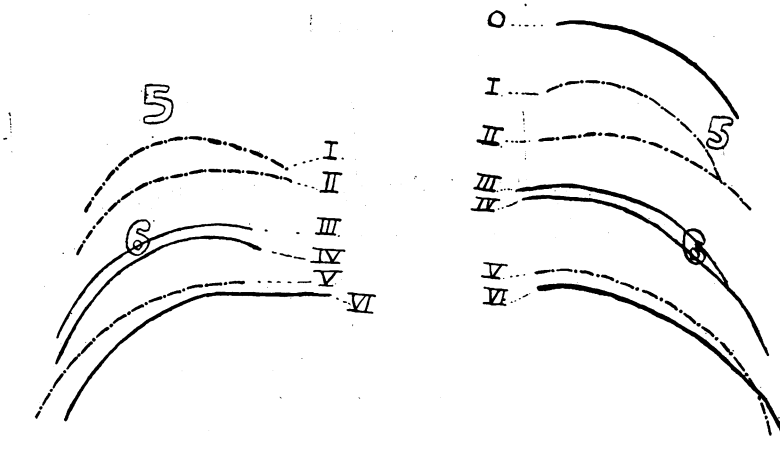



Fig. 8. 0, Deep expiration—standing. I, Deep expiration—lying. II, Moderate inspiration and moderate expiration—lying. III, Moderate expiration—standing. IV, Moderate inspiration—standing. V, Deep inspiration—lying. VI, Deep inspiration—standing. 5 and 6 are marks fixed on 5th and 6th costal cartilages.

These tracings show the position of the diaphragm in full inspiration, normal expiration and deep expiration both standing up and lying down. The photographs were taken from a normal individual.

In connection with the effect of the recumbent position in producing a tendency to anoxæmia it may be pointed out that this tendency would be still more pronounced and might even be definitely deleterious if another effect of the recumbent posture were not to cause deepening of the breathing as described above. This deepening of the respiration will obviously tend to diminish the inequality of the ventilation of different parts of the lungs and will also minimise the anoxæmia which results therefrom. Further evidence that the recumbent position tends to increase the irregularity of the lung expansion was obtained by means of the concertina. This was adjusted so as to produce periodic breathing when the subject was sitting. It was then gradually opened out until the periodicity definitely disappeared. The subject of the experiment then lay down and again breathed from the concertina adjusted at that depth which was found just not to cause periodic breathing in the sitting position. The result always was that the breathing became periodic again as shown by the tracings in Fig. 9.

We have here then further proof that there is a tendency towards

the production of anoxæmia when the recumbent position is assumed. In normal individuals this is of no great moment, for the development of a harmful degree of anoxæmia is prevented by the protective effect of the deepening of the respiration which also occurs as a consequence of lying down, and in addition the periodical deep breaths which occur in some persons tend to correct the lack of oxygen should there be any slight degree of anoxæmia.

In those cases however where there is some other cause at work which interferes with the proper oxygenation of the blood the matter is quite otherwise. For instance we may consider the conditions existing in a case of heart disease with impairment of the circulation. Here we have

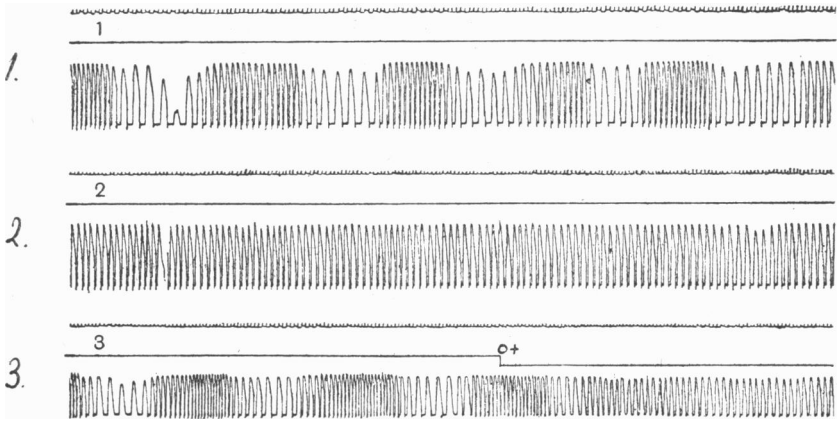



Fig. 9. Subject J. G. P. 1, Breathing restricted by concertina—lying. 2, Breathing restricted to same extent—sitting. 3, Breathing further restricted—sitting. Oxygen given. Curves read left to right. Inspiration upstroke. Time marker = seconds.

a state of affairs in which there is an abnormally great drain on the oxygen carried by the arterial blood, and consequently there is a condition of anoxæmia in the systemic capillaries. Now experiments, which were published in a previous paper, have shown that in the majority of individuals lack of oxygen acts upon the respiratory centre in a way which is strongly contrasted with the effect of excess of $CO_2$. Excess of $CO_2$ causes in the main deepening of the respiration; lack of oxygen causes in the main shallow and quick breathing. Hence in such a case as that under consideration the deficiency of oxygen due to the impairment of the circulation will tend to cause shallowness of the breathing; and this as we have seen prevents the proper oxygenation of the blood in the lungs. It is true that the ineffectiveness of the circulation will also tend

towards accumulation of $CO_2$ in the blood; but any excess, as we have seen, can be disposed of by the lungs whether the breathing be shallow or deep. We may suppose that there are patients in whom the circulation is so slightly impaired that as long as the blood is efficiently oxygenated in the lungs there is no great harm done. Equally it is to be expected that there are other patients in whom the ineffectiveness of the circula-

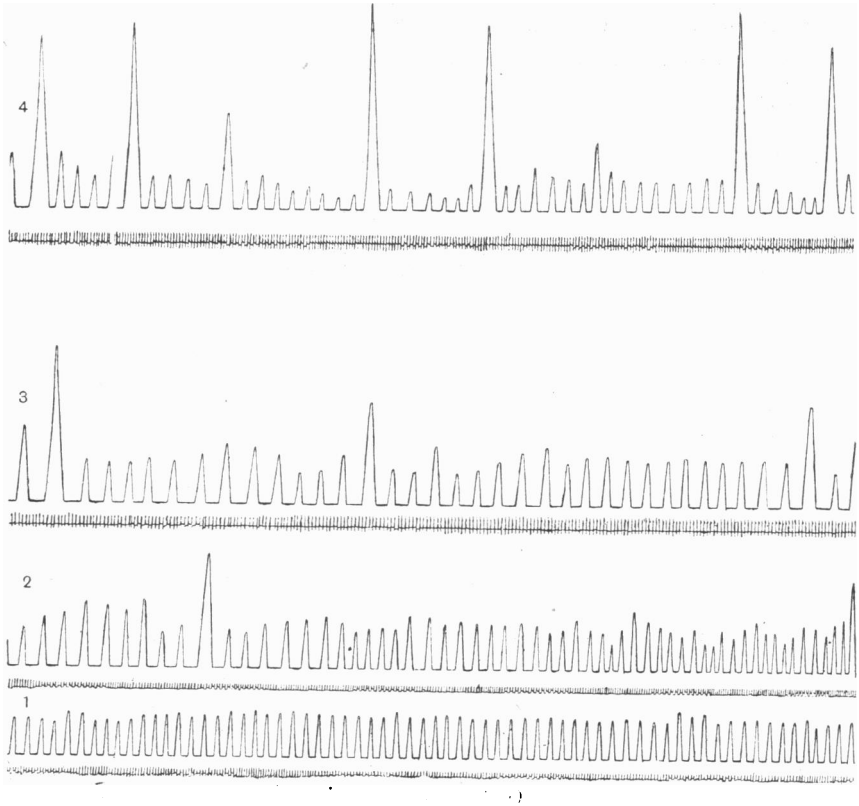



Fig. 10. Subject J. G. P. Concertina unlimited. Time marker = seconds.
1. Sitting. 2. Lying. 3. Sitting, with corsets. 4. Lying, with corsets.

tion reaches such a degree as just to make the respiratory centre begin to respond to anoxæmia, *i.e.* in whom the breathing tends to become shallow. In both cases the patient may be able to carry on fairly comfortably as long as he remains at rest in an upright position. But in the latter case as soon as he lies down his condition alters for the worse. He still has the abnormally great drain of oxygen in the systemic blood and in addition the oxygenation of the blood in the lungs is no longer so

efficiently carried out and consequently the arterial blood carries a less quantity of oxygen than before. There is therefore a condition in which an increased call on the oxygen carried by the blood is coincident with a diminished saturation of the hæmoglobin in the arterial blood with oxygen. Naturally therefore it is to be expected that there will be an exacerbation of symptoms when the patient lies down.

We have now a rational explanation of the phenomenon of orthopnœa, which has hitherto been a complete mystery; and, further the facts detailed above afford a clear indication as to the means to be adopted for the symptomatic treatment of this condition, which is a matter of considerable importance to the patient. Since the orthopnœa is due to

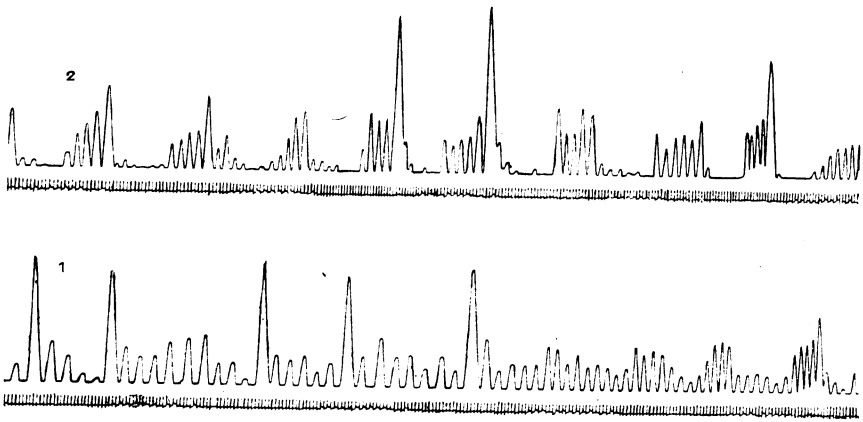



Fig. 11. Subject J. C. M. Unlimited concertina. Time marker = seconds.
1. Sitting, with corsets. 2. Lying, with corsets.

shallow breathing caused by and intensifying, anoxæmia, it is clear that the condition can be treated by adding oxygen to the air inspired.

Having found that the uneven ventilation of the lungs had such striking consequences we were led to investigate the effects of abdominal and thoracic constriction. First we adjusted a pair of ordinary corsets so as to limit as much as possible any abdominal movement while leaving the thorax as free as possible.

We then took records of the respiration with the apparatus described above both in the sitting and lying positions. The records obtained are shown in Figs. 9, 10, 11.

These tracings show that the interference with the abdominal movements caused by the corsets hampers the even expansion of the lungs and produces a remarkable type of periodic breathing. This is especially

noticeable when the subject is lying down. We have tried the converse experiment of leaving the abdomen free and constricting the upper part of the chest with a bandage. We did not obtain any very satisfactory results by this method because the bandage always slipped or stretched and so did not maintain the constriction. We found however that if the bandage did remain firm for a few minutes that the effect was to cause excessive periodicity of the respiration, periods of apnœa alternating with periods of very violent hyperpnœa.

In any condition such as bronchitis, asthma, or emphysema, in which the even distribution of air in the lungs is hindered by local narrowing of bronchi or local impairment of the lung elasticity, imperfect oxygenation of the mixed arterial blood must tend to be produced from the same cause as in shallow breathing. When shallow breathing resulting as a secondary nervous effect of the anoxæmia is superposed on the mechanical hindrance to even distribution of air the anoxæmia must become much more serious. An entirely new light is thus thrown on the symptoms. It is also evident that there is a very wide field for the application of continuous addition of oxygen to the inspired air as a means of relieving the symptoms and preventing the very serious secondary effects of anoxæmia. We hope, however, to discuss these matters in more detail in other papers.

## SUMMARY.

1. An apparatus is described which permits of regulated limitation of the volume of air inspired at each breath. This apparatus may also be used to obtain a continuous quantitative record of the rate and depth of respiration, normal or otherwise.

2. The effects of abnormal shallowness of the breathing were investigated and it was found that the shallow breathing causes uneven ventilation of the lungs and this in turn produces anoxæmia and consequently periodic respiration and other symptoms.

3. Observations were made on the effect of posture on the type and rate of respiration, and showed that the recumbent position is normally associated with slowing and deepening of the respiration and that if the deepening is prevented symptoms of anoxæmia are produced.

4. The effects on the respiration of abdominal and thoracic constriction were observed and recorded and were similar to those produced by the recumbent position.

5. These observations in conjunction with others recorded in this and a previous paper afford an explanation of orthopnœa.

The apparatus used in the work here described was partly provided by the Medical Research Committee and partly lent by Lt-Col. C. G. Douglas, M.C., R.A.M.C. and by Messrs Siebe Gorman & Co., Westminster Bridge Road, S.E. It was at the request of the Medical Research Committee to the British and Canadian Army Medical Authorities, and in consequence of facilities afforded to us by the latter, that the work was made possible. To all of these we wish to express our thanks.

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