THE THERAPEUTIC ADMINISTRATION OF OXYGEN.

By J. S. Haldane, M.D., F.R.S.,

It is well known that the administration of oxygen often produces a least temporary benefit in cases of serious interference with the respiratory or circulatory functions; but sufficient attention has not hitherto been paid either to the amount of oxygen that may be obtained from the administration of oxygen, or to how it can best be administered. In the present paper I propose to discuss both these questions in the light of existing physiological knowledge, and to describe an apparatus designed for the clinical administration of oxygen.

Our knowledge of the physiology of respiration has advanced very rapidly within the last few years, and certain points which have been elucidated in this advance must first be referred to. Under normal conditions the breathing is so regulated as to keep the mean percentage of the amount of air breathed practically constant at a level which varies slightly for different individuals, but is about 5.6 per cent. The marvellous accuracy of this regulation was a revelation to physiology. A rise of about 0.2 per cent. in the mean alveolar CO₂ percentage doubles the amount of air taken into the lungs, while a fall of 0.2 per cent. produces apnoea. The rise in CO₂ acts through the blood on the respiratory centre in virtue of the respiratory mechanism, which consists, and the regulation of the blood alkalinity by the respiratory centre, kidneys, liver, and probably other organs, is so exact that no existing chemical or physical method of measuring variations in respiration (alveolar CO₂ or other ion concentration) can detect the more minute changes to which these organs are constantly reacting. For the present purpose it is, however, sufficient to remark that changes in the composition of the blood, and the balance of the blood, almost entirely dependent on the amount, not of oxygen, but of CO₂ in the blood leaving the lungs, and consequently passing to the respiratory centre.

By reducing the CO₂ in the arterial blood apnoea can easily be produced in a person blue in the face from want of oxygen; and an animal from the blood of which sufficient CO₂ has been removed by forcible artificial respiration is not only apnoeic for a time, but will die from want of oxygen without drawing a single breath.

Want of oxygen does help to excite the breathing, but can not act directly on the brain, and cannot therefore be produced by a sufficiently low percentage of oxygen to cause slight cyanosis is suddenly breathed, there is an immediate great increase in the breathing; but this soon moderates, while the blood becomes cyanotic, and consciousness begins to fail. What happens is that the want of oxygen lowers at once the alveolar percentage of CO₂ which is required to excite the respiratory centre. In consequence of the presence of the blood, etc., of a considerable store of preformed CO₂ the breathing is greatly increased at once, and remains increased till the proformed CO₂ is reduced so as to correspond to the new level. The breathing then quietens down, and in consequence the alveolar oxygen percentage falls still lower, so that cyanosis and the other direct symptoms of oxygen want increase. If the lowering of the oxygen percentage in the air is gradual, or if oxygen want is gradually produced by gradual CO₂ poisoning, or by a gradual ascent in a balloon or aeroplane, there is no evident hyperpnoea. The formidible symptoms (paralysis of movement and loss of consciousness; coma; on the other hand, warning from increase in the breathing. It is a mistake, constantly made, but which ought now never to be made, to regard the breathing as a sufficient index of the existing condition of the respiratory centre. In the presence of oxygen want, the breathing is greatly and persistently increased, some other cause is present than oxygen want.

The physiological effects, immediate and remote, of oxygen want may be considered. Having no oxygen produced, oxygen want acts almost instantly in increasing the sensibility of the respiratory centre to CO₂, and so producing an urge desire to breathe. The rapidity of this action is in the existence of oxygen want, the breathing is greatly and persistently increased, some other cause is present than oxygen want.

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exposure are usually needed. Those who have experienced the extreme depression of mountain sickness or CO poisoning will readily realize that such a condition may mean the difference between life and death to a patient batt ful with illness.

From the foregoing remarks it seems clear that a physician ought to make every effort to avert the effects of want of oxygen or cut them short. It may be argued that such measures as the administration of oxygen are at the best only palliative and are of no real use, since they do not remove the cause of the pathological condition. As a physician ought to be careful in every case to agree with this reasoning. The living body is no machine, but an organism constantly tending to maintain or revert to the normal, and the respite afforded by such measures as the temporary administration of oxygen is not wasted, but utilized for recuperation.

Let us now consider in more detail how oxygen want is produced, what seems to be possible in directly combating it by oxygen administration, and what risks have to be avoided. We may begin with a simple and easily intelligible case—that of poisoning by CO, or by a nitrite, chlorate, or other poison which causes death by disabling the haemoglobin, so that it is unable for the time to carry sufficient oxygen to support life. Normal human arterial blood carries about 18.1 c.c.m. of available oxygen in 100 c.c.m. of blood. Of this, about 17.75 c.c.m. are combined with the haemoglobin and 0.35 c.c.m. are in simple solution round the circulation. During this time this blood loses only about 4.5 c.c.m. of oxygen. In poisoning by CO and similar respiratory poisons, death occurs when about 80 per cent. of the haemoglobin is disabled. If the patient is still alive there will, therefore, still be 20 per cent. of his haemoglobin available. But by administering pure oxygen we can at once increase the amount of oxygen in simple solution to about 2.5 c.c.m. This promptly avails of the danger from want of oxygen, and as poisoning the oxygen rapidly drives out CO from the haemoglobin, so that after fifteen or twenty minutes of continuous administration the oxygen may be discontinued. In poisoning by nitrites, etc., there is also a fairly rapid return of the blood towards the normal, consequent on the gradual elimination or destruction of the poison. Experiments on animals have shown quite clearly that oxygen actually does avert death in the cases just considered.

In acute inflammatory conditions of the lungs there is sometimes also want of oxygen, as shown by cyanosis; and where the inflammatory condition is accompanied by the production of emphysema by extension throughout the lungs, the cyanosis is often very great. This condition is seen typically in the acute stages of poisoning by nitrites or nitrates of amyl, and is recognized as the preliminary to death. When a portion of the lungs, including even a greater part of the lungs, is entirely blocked by consolidation, as in emphysema, there is commonly no cyanosis. This indicates that little blood is passing through the consolidated parts. What passes through the healthy portion is amply sufficient for respiratory requirements during rest. It must be borne in mind that the normal lungs and circulatory organs are adapted for working about ten times the respiratory requirements during rest, since the respiratory exchange is often about ten times as great during work as during rest. Hence during rest in bed a very small proportion of the normal lung will suffice for meeting respiratory and circulatory requirements, provided there is but little circulation through parts which are useless. But when cyanosis due to a lung affection exists, in spite of the fact that air is entering the whole or a great part of the lungs freely, we seem driven to the conclusion that the entry of oxygen into the blood through the alveolar walls is impeded by exudation and increase in thickness of the alveolar walls. It is very important to realize that this may occur without any serious impediment to the passage of CO2 outwards. CO2 is about twenty-five times as soluble in water as oxygen, and hence it passes through the alveolar walls far more easily, with a given difference of partial pressure, than does oxygen. Moreover, a comparatively small increase in the breathing will enormously increase the small difference of partial pressure, and thus passage of CO2 outwards depends; but the same increase in breathing produces only a slight proportional increase in the diffusion pressure which drives oxygen inwards. Hence we may have cyanosis, and consequently very formidable effects from oxygen want, without marked hyperpnoea. The grey look of the patient's face will be the blood index of this. They will have a certain amount of venous blood pressure with its accompanying blue tinge.

In great hyperpnoea accompanies the cyanosis there must be some other complicating condition. This accompaniment is seen typically in the acute stage of poisoning by chloroform or nitrous fumes; and the results of post-mortem examination, together with clinical observation during the recent years, seem to go to show that the systemic arterial blood which enters the alveolar capillaries becomes extremely tawdry in effort to breathe despite the blocking of many of the alveoli by exudation or consolidation. In consequence of this there is widespread emphysema, which is very evident on post-mortem examination. The areas around the emphysematous cavities are partially collapsed and very imperfectly ventilated, so that CO2 accumulates in them to an abnormal extent. The emphysematous cavities are excessively ventilated, but this ventilation is of little use since the proportion of sound alveolar wall to air is far too small. In consequence of this the arterial blood is raised, and hyperpnoea, along with increased arterial and venous blood pressure, results. As the cyanosis clears up the cyanosis disappears, but the hyperpnoea may continue, and this takes a considerable time for the cyanosis to heal up.

When there is cyanosis (whether of the deep purple or grey type) due to hindered passage of oxygen through the alveolar walls, this can be corrected by raising the percentage of oxygen in the alveolar air and so increasing the diffusion pressure. The normal alveolar oxygen percentage is about 14, or 7 per cent. less than in the external air. By raising the percentage of oxygen in the inspired air to 35 we raise the alveolar oxygen percentage to 28, and thus much more than double the effective diffusion pressure, since the oxygen pressure in the venous blood passing through the lungs will probably be at least 40 mm. Hg. It is probable, therefore, require only a moderate increase in the oxygen percentage of the inspired air to remove the cyanosis.

Even in ordinary cases cyanosis accompanies the alveolar oxygen pressure may be a matter of decisive importance. This is clearly shown by the fact that these pneumonias do very badly at high altitudes. At Cripple Creek (altitude about 10,000 ft.) in the Rocky Mountains I found that this was so well recognized that all cases of pneumonia we put in the train and sent down to the prairie level.

Here, in lung affections, an addition of oxygen to the inspired air is needful in order to combat want of oxygen, it is evidently desirable to continue the administration over long periods. It was shown by Paul Bert that hypoxia at a pressure of 5 atmospheres is capable of producing convulsions and rapid death; but Lorrain Smith found that, apart altogether from this action on the nervous system, pure oxygen at high pressures produces pneumonias pretty rapidly, and even at ordinary atmospheric pressure acts slowly on the lungs, ultimately producing fatal pneumonia after several days in animals. This effect was soon occasionally produced in about four days by a mixture containing only 80 per cent. of oxygen. It is evidently desirable, therefore, to keep the oxygen percentage as low as possible during long administrations, and to know roughly what percentage is being breathed.

In cases where the source of danger is failure of the circulation, the inhalation of oxygen may also be of use, and I have seen the cyanosis in a case of varicella disappear if kept up at once on the administration of oxygen. The effect was so striking that it could hardly be attributed to the increased amount of oxygen going into simple physical solution in the arterial blood. It showed that cyanosis of the kind owing to back pressure in the lungs and consequent exudation, etc., there was hindrance to the diffusion of oxygen.
MINOR INJURIES TO JOINTS.

BY FRANK ROMER, LIEUT.-COLONEL R.A.M.C.(TEMP).

At the Croydon War Hospital, where we receive every class of crippled limb, I frequently come across cases of ordinary sprains and dislocations where recovery has been delayed from want of suitable treatment in the initial stages. The conditions of active service render men particularly liable to such injuries, but when the medical officers have many serious cases requiring urgent surgical attention, those who are incapacitated by minor injuries are often unable to receive the attention they would otherwise get. It is in no earing spirit of criticism, therefore, that I refer to this point, but with the view of drawing attention to the importance of immediate treatment when dealing with men sustaining these injuries in England, particularly so long as they can be returned to duty quickly than by waiting for treatment at a later date. Delay not only renders recovery more difficult, but prolonged acquaintance with hospital life is apt to sap the morale of the men.

A sprain has been described as an incomplete dislocation, and is said to occur when a joint has been wrenched by a force sufficient to overstretched or rupture the controlling ligaments and tendons. It may vary from a condition where the joint is completely incapacitated to a mere deviation from the normal, where pain is only elicited by some particular movement. Such conditions have been described as sprains of the knee, ankle, and wrist, and the rule is that all joints may be affected.

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