Possible Value of Inhalation of Carbon Dioxide in Climbing Great Altitudes

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MOUNTAIN sickness is a form of asphyxia due to the diminished partial pressure of oxygen at great altitudes. The functional disturbances in this disorder are, however, not merely anoxial, but are largely the expression of a secondary and almost equally important deficiency of carbon dioxide in the blood and tissues. Deficiency of oxygen induces hyperpnea and acapnia: that is, overbreathing and the resulting deficiency of carbon dioxide. Acapnia in turn induces subnormal respiration and a continued or even increased deficiency of oxygen. Haldane, Priestley and Douglas[1] demonstrated the correctness of Miescher's somewhat poetical formulation: "Over the oxygen supply of the body carbon dioxide spreads its protecting wings."[2] Henderson[3] confirmed the importance of the relation between the two gases in respiration when he found that it was possible to produce so great a deficiency of carbon dioxide by over-ventilation of the lungs that thereafter an animal may die of lack of oxygen with no effort to breathe.

On Pike's Peak in 1911, Douglas, Haldane, Henderson and Schneider[4] made a number of observations on muscular work in which these relations were strikingly illustrated. One of them was as follows: a member of the party whose respiratory centre was peculiarly sensitive (as indicated by the fact that he acclimatised more rapidly than any of the others) exercised by walking as rapidly as possible for a quarter of a mile up the cog railway, grade 1 in 5. While walking, he experienced an almost intolerable panting; but when he stopped to get his breath, he very quickly stopped breathing entirely for a few seconds, and then developed alternating hyperpneas and apneas. Insufficiency of oxygen was clearly the condition inducing the excessive breathing and pumping out of carbon dioxide, and the consequent apnea. The apnea in turn intensified the anoxia and induced another period of overbreathing.

As the work of climbing would not have been an excessive exercise for this individual at sea-level, it is probable that the amount of carbon dioxide produced was insufficient to maintain a continuance of vigorous breathing, except during the time that it was reinforced by acute oxygen deficiency.

The cause of the overbreathing was indicated by another observation. Merely squeezing a rather stiff rubber bulb with one hand as vigorously as possible, without other exercise, until the muscles of the forearm were tired, induced such excessive breathing that cessation of the manual activity was followed by apnea and then by alternating periods of overbreathing and apnea. This observation accords with the classic investigations of Geppert and Zuntz[5] on animals. It indicates, as they concluded, that some respiratory stimulant, other than carbon dioxide, is formed in vigorously active muscles, or under a deficiency of oxygen, and is carried by the blood to the respiratory centre, or the closely connected sinus caroticus. For reasons that we will not now stop to discuss, we do not believe that this substance, 'respiratory X'[6], is lactic acid, but rather that it is some specific hormone that increases the sensitivity of the respiratory centre to carbon dioxide.

Recently, Winterstein[7] has suggested that inhalation of a small amount of carbon dioxide might be helpful in maintaining respiration and promoting oxygen absorption at great altitudes. In order to test this idea, two of the writers of this article, both vigorous young men, spent a few days on Pike's Peak during the summer of 1934.

As the time on the Peak available for this investigation was short, the observations were confined to the subject of the acapnia which is the result of oxygen deficiency, and which in turn aggravates that deficiency. For this purpose small cylinders were provided, charged with enough liquid carbon dioxide to form 400 litres of gas. The flow of the gas was controlled and adjusted by a small, but accurate and light, reducing valve and flow gauge. The whole apparatus* weighed only 4 kgm. and was carried at one side suspended by a strap across the opposite shoulder. The flow was usually set at 2 litres per minute, and was conducted through a rubber tube to an open mask with wide holes to the outside through which the wearer breathed the outside air without the slightest impediment. During expiration the gas was thus blown away and wasted; but during inspiration it mixed with the inspired air. The amount of carbon dioxide inspired was therefore only about 1 litre per minute. If the wearer of the apparatus breathed 50 litres of air per minute, the carbon dioxide was thus diluted to 2 per cent. With a respiration of 20 litres per minute, the dilution would come to one in twenty or 5 per cent.

* We are much indebted to the Ohio Chemical and Manufacturing Company for placing this very efficient apparatus at our service. It proved ideally convenient.

The two members of our party had been on the Peak for only a couple of days, but were sufficiently acclimatised to be comfortable. Three young men, who were employed in the hotel on the summit of the Peak (14,100 ft.) and were fully acclimatised, also performed the tests. Each of these five men made four experiments, wearing the apparatus and walking a distance of 250 yards up the cog railway, grade 1 in 5, in two minutes. The work amounted to lifting the man's body and the 8 lb. apparatus 150 ft. vertically. On one day the tests with inhalation of carbon dioxide were made first, and an hour later exactly the same exertions were made while wearing the apparatus with the gas shut off. On the next day the control exertions were made first, and those with inhalation of carbon dioxide were made an hour later.

The results on all five of the men were closely similar. None felt either the exertion of the respiratory strain to be at all increased by the inhalation; but rather the contrary. All noted the greater regularity of their breathing with the gas on than with it turned off. This subjective evidence was also in accord with objective findings from pulse counts and measurements of arterial pressure made immediately after the climbs. The average pulse rate was the same after the ten climbs in which carbon dioxide was inhaled than it was after the ten climbs without the inhalation: 126 heart beats per minute. Without the inhalation the average rise of systolic arterial pressure was 67 mm., namely 108–175. With the inhalation it was only 50 mm., namely, 110–160. Diastolic arterial pressure, instead of rising, was slightly lower after the exertion than before—lower by an average of only 1 mm. without inhalation and 3 mm. with the inhalation. The alveolar carbon dioxide was lowered 0·5 per cent in the control test without inhalation; but was 0·5 per cent higher after the exertion than before when the inhalation was used.

These effects are not large, but on the whole the evidence indicates that the exertion was made with somewhat less strain on the heart and respiration with inhalation of carbon dioxide than without. The reason appears in the fact that excessive loss of carbon dioxide was prevented. On the contrary, the alveolar carbon dioxide was raised. The supply of oxygen was thus protected and its utilisation aided by the influence of the carbon dioxide upon the Bohr-Haldane[1] relations of the blood gases.

In addition to these experiments, observations were made upon thirty tourists who were more or less affected with mountain sickness and who, out of several hundred visitors to the Peak, submitted to be treated and examined. All had made the ascent either by the cog railway or by automobile. In most of these cases cyanosis or pallor, dizziness, headache and nausea had developed when they walked about on the summit or came into the warmer air of the lunch room. In extreme cases, periodic breathing, confusion, muscular cramps and twitchings, and low blood pressure or even fainting occurred before the inhalation was administered.

The inhalation was usually continued for only 2–3 minutes at a time. The effects were in most cases subjectively beneficial. None felt the worse for it. The colour of their lips was distinctly improved. Respiration was in all cases changed from irregular, or even intermittent, to a regular and deep rhythm, but was seldom increased in rate. Systolic arterial pressure was not altered significantly, that is, only from an average of 105 mm. to 103 mm. The average diastolic pressure, on the contrary, was raised from 62 mm. to 69 mm., while the pulse-rate was decreased from an average of 92 to 82 per minute. Considering that these figures were the averages on thirty subjects, they are, we think, significant of distinct benefit from the inhalation.

The only persons whose conditions changed disadvantageously were two men who had ascended the peak on foot, arriving in a state of exhaustion, who developed conditions verging on collapse just as the inhalation was started. Their condition appeared so serious that instead of continuance of inhalation, arrangements were made for them to be immediately conveyed down the mountain. It is probable that their collapse was due to their exhaustion and that it would have developed anyway, as is often the case, as soon as they stopped climbing. It seemed safer, however, not to continue the inhalation, lest it should be blamed for their subsequent condition.

Our conclusions are that, at least up to an altitude of 14,000 ft. (barometric pressure 450 mm.) inhalation of a small amount of carbon dioxide with the inspired air is distinctly beneficial in protecting against both acapnia and anoxia during and after vigorous physical exertion. As the effects were even better on those who had made no great exertion, we suggest that such an inhalation might be of considerable value also for passengers travelling by air at altitudes up to at least 14,000 ft. Tests at greater altitudes on mountaineers and aviators are deserving of trial. For this purpose, carbon dioxide has one advantage over oxygen: much less is required.

[1] Haldane, J. S., "Respiration", Yale University Press, 1922.
[2] Miescher-Rusch, *Arch. Physiol.*, 355; 1885.
[3] Henderson, Y., *Amer. J. Physiol.*, **21**, 142; 1908.
[4] Douglas, Haldane, Henderson and Schneider, *Phil. Trans. Roy. Soc.*, B, **203**, 207; 1913.
[5] Geppert and Zuntz, *Pflüger's Arch.*, **42**, 189; 1888.
[6] Henderson, Y., *Physiol. Rev.*, **5**, 131; 1925; and Henderson, Y., and Greenberg, *Amer. J. Physiol.*, **107**, 37; 1934.
[7] Winterstein, H., *Acta Aerophysiologica*, **1**, Fasc. 2, 3; 1934.