indefinitely. Newcomers on Mt. Makalu, after four to six weeks' acclimatization, were, if anything, fitter and more active than men who had wintered at 19,000 ft. (5,790 m.).

Medical aspects of the expedition are described. On Mt. Makalu cases occurred of cerebral thrombosis, pulmonary infarction, acute pulmonary oedema, pneumonia, and frostbite. The ascent was made without oxygen equipment, but oxygen was available for medical treatment.

The expedition was sponsored by the publishers of World Book Encyclopedia, of Chicago, Ill., U.S.A. The physiological work was supported by the Medical Research Council and the Wellcome Trust. The map was drawn by Mr. G. S. Holland, of the Royal Geographical Society.

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"As a result of representations made by several members of the public to the Committee for Research on Apparatus for the Disabled a Working Party on Car Modifications was set up. This working party proceeded to examine the problems of the disabled, in the first instance, as far as private cars were concerned. It seemed clear that there was a considerable number of disabled people who were virtually home-bound because they could not, by themselves, get into or out of a car, or who, if they could do this, could not bring their wheelchair in with them, thus rendering themselves immobile at the end of their journey. There were men and women unable to obtain gainful employment because of this as well as housewives unable to shop, to fetch their children to and from school and to lead normal social lives. The Working Party is therefore developing an adapted chassis and body for a motor vehicle which, it is hoped, will enable a wheelchair-user to enter the car and to drive it in his wheelchair. Work on this is proceeding very satisfactorily, although it is still in its early stages." (*Ninth Annual Report*, National Fund for Research into Poliomyelitis and Other Crippling Diseases.)

EFFECT OF VARIOUS MODES OF OXYGEN ADMINISTRATION ON THE ARTERIAL GAS VALUES IN PATIENTS WITH RESPIRATORY ACIDOSIS*

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In subjects with obstructive emphysema and hypercapnia the ventilatory response to carbon dioxide is diminished. This is also true of normal patients made hypercapneic by placing them in an environment rich in carbon dioxide. In both groups hypoxia and a low *p*H remain as the main ventilatory stimuli. The removal of the stimulatory effect of hypoxia by administering oxygen is well known to worsen alveolar ventilation and increase carbon-dioxide retention. In spite of this, the need for oxygen in the treatment of acute respiratory acidosis is accepted by most physicians. However, there is disagreement on the mode of oxygen administration. Thus some (Barach *et al.*, 1952; Wilson *et al.*, 1954; Barach, 1961) feel that continuous administration of oxygen using low flow rates with graded increases will help avoid serious worsening of hypercapnia while maintaining acceptable oxygen tensions. Others (Cohn *et al.*, 1954; Harbord and Woolmer, 1958) suggest that giving oxygen intermittently each hour will lessen the hazards of further alveolar hypoventilation and yet increase the oxygen tension of the blood.

It is the purpose of this paper to report the effects of oxygen administration by various means in patients with obstructive emphysema and respiratory acidosis.

Methods and Materials

A total of 15 adult male patients were studied. Requirements for inclusion in the study were clinical and pulmonary function evidence of obstructive emphysema with carbon-dioxide retention at the time of the study. All patients were in a fasting state, and all procedures were begun in the early part of the day. A Cournand needle was placed in the brachial artery, and two baseline samples were drawn 15 minutes apart while the patient was breathing room air.

The patients were separated into three groups in a random fashion. The groups were comparable by degree of respiratory acidosis. Group A consisted of four patients; these received pure oxygen by mask for 10 minutes. Group B comprised six subjects receiving oxygen by nasal catheter for 10 minutes at flow rates

*Aided and supported in part by United States Public Health Service Grant HTS 5454.
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of 1 to 2 litres per minute. Five patients, group C, were given continuous nasal oxygen at the same flow rate as group B for two and a half to four hours. In all groups bloods were drawn at varying intervals during the procedure.

Blood samples were analysed for oxygen tension (Po_2), carbon-dioxide tension (Pco_2), and *p*H. For oxygen tension measurements the Clark electrode in the adaptation by Severinghaus was used. Carbon-dioxide tension was determined by means of the Severinghaus Pco_2 electrode, and read on an instrumentation laboratory meter. The *p*H was measured in a capillary glass electrode and read on a Radiometer *p*H meter, model 4-C PHM. All measurements were carried out at 37° C.

Results

Group A.—The arterial carbon-dioxide tension rose in all patients (Fig. 1), while the *p*H fell in three. When the oxygen was discontinued the oxygen tension fell to the baseline level within 30 minutes in all cases (Fig. 1). In two instances in which early tensions were measured, the oxygen tension had reached the baseline within 20 minutes. In three subjects the oxygen tension fell below the baseline level after oxygen administration was discontinued. The oxygen tension in one of these patients remained below the baseline for at least 40 minutes. The carbon-dioxide tension remained elevated even after the oxygen tension approached the control level. In three patients, by the time the carbon-dioxide tension reached the control level the oxygen tension was still below the baseline level. In none of the cases was the oxygen tension above the baseline level after the carbon-dioxide tension had returned to the control level.

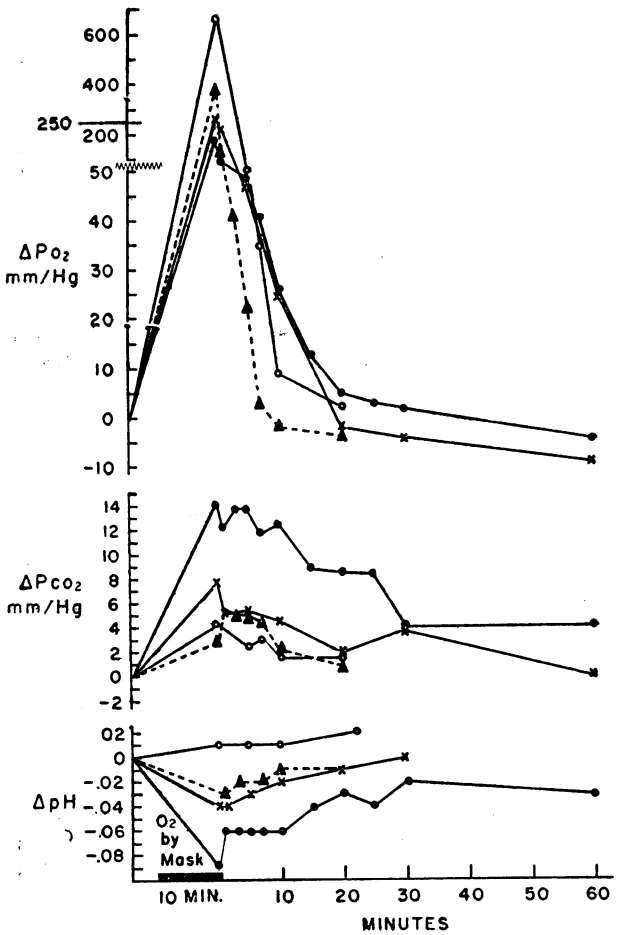

FIG. 1.—Changes in Po_2, Pco_2, and *p*H during and for the time indicated after administration of oxygen by mask. Thick black line on abscissa indicates duration of oxygen administration. Each patient designated by the same symbol on all three scales.

Group B.—The carbon-dioxide tension rose in all six patients (Fig. 2). The pH (Fig. 2) fell in five of the six cases. In five patients the oxygen tension (Fig. 2) had fallen to baseline levels within 20 minutes after oxygen was discontinued. In one subject it took 40 minutes for the oxygen tension to fall to resting levels. In four subjects the oxygen tension fell below the baseline and remained below the baseline for the greater part of the hour. One patient had no change in carbon-dioxide tension, but with the fall in oxygen tension the *p*H exhibited a moderate rise. In four cases the *p*H rose above the baseline level after the oxygen was discontinued and while the oxygen tension was at or below baseline levels.

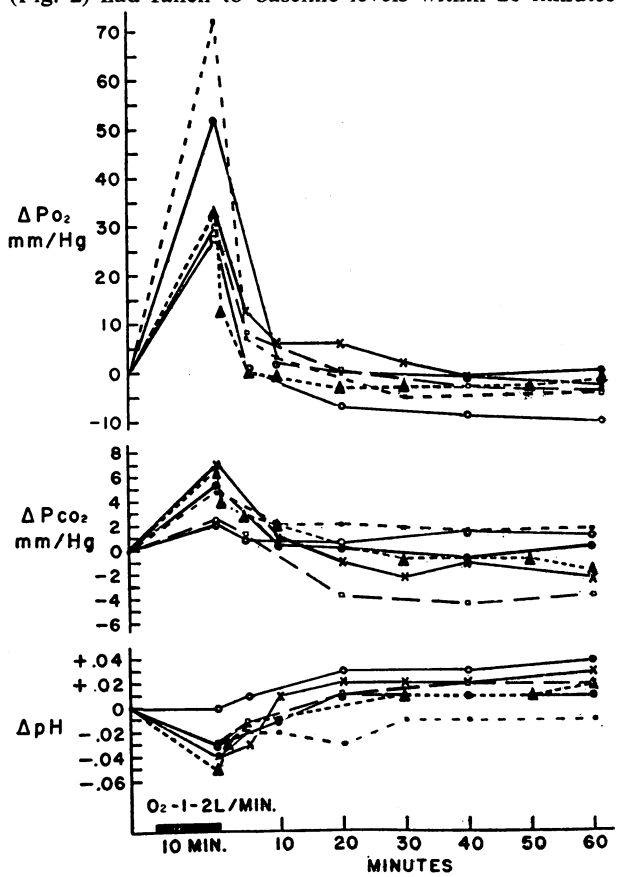

FIG. 2.—Changes in Po_2, Pco_2, and *p*H during and for the time indicated after administration of oxygen at 1 to 2 litres per minute. Thick black line on abscissa indicates duration of oxygen administration. Each patient designated by the same symbol on all three scales.

Group C.—Continuous administration of nasal oxygen at 1 to 2 litres per minute resulted in a rise of carbon-dioxide tension in four, and a fall in *p*H in three of the five patients (Fig. 3). In one case the *p*H dropped from 7.26 to 7.20. There is no evidence of adaptation to oxygen administration with subsequent lowering of the carbon-dioxide tension. The variation in oxygen tension while receiving nasal oxygen is probably related to minute ventilation and the degree of fixed shunt.

Discussion

The use of intermittent oxygen therapy has been recommended as a means of improving anoxaemia

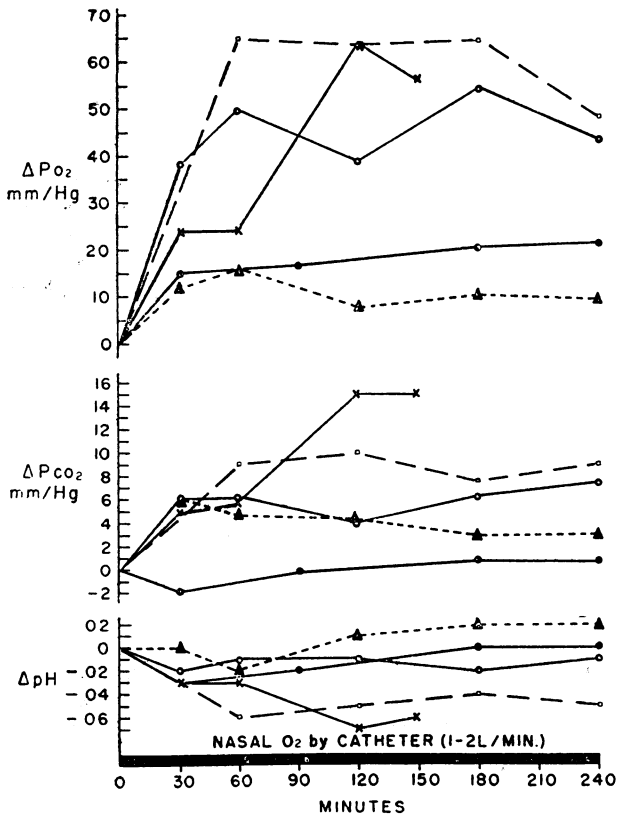

FIG. 3.—Changes in Po_2, Pco_2, and *p*H during continuous administration of oxygen at 1 to 2 litres per minute. Thick black line on abscissa indicates duration of oxygen administration for four patients. One study stopped at 150 minutes because of excessive fall in *p*H. Each patient designated by the same symbol on all three scales.

while preventing progressive respiratory acidosis (Cohn *et al.*, 1954; Harbord and Woolmer, 1958). Some feel that on withdrawal of oxygen the stimulus of anoxia to respiration is restored and the retained carbon dioxide eliminated. However, as shown by Farhi and Rahn (1955) and as our data indicate, the body compartments of oxygen and carbon dioxide are different. With a change in ventilation or in the composition of inspired air, the concentration of oxygen changes within a few minutes, whereas the change in carbon-dioxide tension takes considerably longer. In addition, the persistence of high blood-carbon-dioxide tension in arterial as well as venous blood causes the alveolar air to have an increased carbon-dioxide tension, and hence a lowered oxygen tension. This accounts for the fall in oxygen tension below the baseline after the withdrawal of oxygen. Campbell (1960) has noted the oxygen tension to fall as low as 15 mm. Hg five to six minutes after stopping oxygen in patients with respiratory acidosis. Likewise, some of our patients were more anoxaemic during the major portion of the hour following the administration of oxygen than prior to receiving oxygen.

Our data on intermittent oxygen therapy at high or low flows indicates little advantage to the patient in terms of correcting anoxaemia for any significant period. Oxygen given intermittently is often prescribed in the long-term treatment of chronic pulmonary emphysema. This study indicates how little advantage this has in maintaining a satisfactory blood oxygen tension. Indeed, ultimate worsening of the anoxaemic state often takes place.

The use of continuous low-flow oxygen without assisting ventilation by other means is hazardous. Thus one patient sustained a fall in *p*H from 7.26 to 7.20 in two hours, while the *p*H in another fell from 7.35 to 7.29 in one hour. This statement and these results are contrary to the opinion frequently stated by Barach *et al.* (1952) and Barach (1961).

During the administration of oxygen we were unable to detect clinical indications of a rising carbon-dioxide tension in any of our patients. Furthermore, the rise in carbon-dioxide tension could not be accurately predicted from the resting carbon-dioxide tension. In view of these and other studies (Comroe *et al.*, 1950; Hickam *et al.*, 1952; Campbell, 1960), it seems that the only safe way to administer oxygen to patients with acute respiratory acidosis is in conjunction with a mechanical respirator. Another theoretical possibility is the combination of oxygen therapy with the administration of pharmacological respiratory stimulants. We have used the former means and have found it satisfactory. Some workers, especially in Britain (Harbord and Woolmer, 1958), use nikethamide in high doses (2, 5, or 10 ml. every 30 minutes as needed) and claim good results.

Summary and Conclusions

The need to correct hypoxia and hypoxaemia in patients with respiratory acidosis is well accepted. However, the proper mode of oxygen administration is debated. We have evaluated various modes of oxygen administration by studying arterial gas values in 15 patients with respiratory acidosis before, during, and after oxygen administration. The subjects were divided into three groups. Group A received pure oxygen by mask for 10 minutes; group B received oxygen by nasal catheter at one to two litres per minute for 10 minutes; group C received oxygen as did group B but for between 150 and 240 minutes.

Intermittent oxygen administration does not correct hypoxaemia for any appreciable length of time. Furthermore, it is often followed by hypoxaemia more pronounced than before therapy. Increased carbon-dioxide tension occurred in all patients and frequently remained present after the oxygen tension had fallen to baseline levels.

Continuous oxygen administration by nasal catheter resulted in a sustained improvement of the oxygen tension. However, serious worsening of acidosis took place in two subjects.

We feel that intermittent oxygen therapy has little place in the treatment of respiratory acidosis. Irrespective of the mode of administration, when oxygen is administered to patients with respiratory acidosis it should be given with a mechanical respirator or pharmacological stimulant to ensure continued alveolar ventilation.

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