merely sedated. Overactive psychiatric patients showed some evidence of behaviour improvement.

The observations are taken as indicating that 10–20% bemegride with barbiturate reduces or even prevents the development of coma or of prolonged sleep.

The observations are tentatively presented for consideration in the treatment of patients in whom sleep treatment or prolonged sedation is desired but can be maintained only by the use of high barbiturate doses which, if given alone, involve the risk of barbiturate poisoning or other serious complications.

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PORTABLE OXYGEN EQUIPMENT AND WALKING-AID IN PULMONARY EMPHYSEMA

BY

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The patient whose case is described below suffers from severe emphysema and has been greatly helped by portable O₂ equipment and a portable walking-aid. Physiological studies showed how these measures improved his pulmonary function. The main aim of this paper is to show how an appreciation of simple physiological principles enables an assessment of the potential value of such therapeutic measures to any individual patient to be made on clinical evidence.

Clinical Details

The patient, a motor engineer aged 59, was 5 ft. 9 in. (175 cm.) tall and had a surface area of 1.8 sq. m. (19.4 sq. ft.). He had suffered from increasingly severe dyspnoea for 33 years. There was no history of long-standing cough or sputum preceding the onset of dyspnoea. He had been increasingly incapacitated for the last seven years, and before admission could walk only 25 yards (23 metres). He continued to work by driving his car from the door of his house to the desk in the garage where he was employed. To climb the 14 stairs in his house caused him intense distress lasting 10–15 minutes and often forcing him to kneel on the stairs. He had not walked out of the front gate of his house for 10 years nor had a pair of shoes repaired for seven years. He had great difficulty in dressing himself and in eating his food. In recent years he had had a variable amount of mucoid sputum which caused bouts of

coughing leading to intense dyspnoea, and interrupting his sleep every few hours. He slept more comfortably lying flat than when propped up. Clinically and radiologically he had the changes of severe emphysema. He was cyanosed at rest and became very cyanosed on slight exertion. There was no clinical evidence of heart failure, and the transverse diameter of the heart was within normal limits. On screening, however, the right ventricle was moderately enlarged and the main pulmonary arteries were prominent. E.C.G. showed an incomplete right bundle-branch block. Repeated bacteriological examination of the sputum had not shown any pathogenic organism to be persistently present. The haemoglobin concentration was 19 g. per 100 ml., and the P.C.V. 63%.

A selection of the measurements of pulmonary function is given in Table I. These show the following abnormalities: chronic inflation of the lungs (increased residual volume);

TABLE I.—Pulmonary Function Tests (August, 1956). Patient Seated With Arms Supported

	Patient	Expected Normal
Vital capacity (l. B.T.P.)	2.5	4.1
Residual volume (l. B.T.P.)	7.9	3.0
Forced expired volume in 1 sec. (l. B.T.P.)	0.7	3.2
Maximum voluntary ventilation (l./min. B.T.P.)	24	100
Arterial blood:		
O ₂ tension (mm. Hg)	52	100
O ₂ saturation (%)	82	>95
CO ₂ tension (mm. Hg)	58	40
Maximum diffusing capacity for O ₂ (ml./min./mm. Hg)	8	40

difficulty in expelling air during expiration (reduced vital capacity and reduced forced expired volume); reduced ability to ventilate the lungs (low maximum voluntary ventilation); inadequate alveolar ventilation (elevated arterial CO₂ tension); reduced alveolar-capillary surface (low diffusing capacity). The low O₂ tension and saturation are due to a combination of factors as discussed later.

In spite of the intensive use of all forms of conventional treatment he remained completely incapacitated after two months in hospital.

Methods

Blood gas tensions were determined in duplicate by the Riley method (Riley, Campbell, and Shepard, 1957); the pH was measured at 37.5° C. with a Cambridge pH meter and Stadie electrode; gas analyses were performed with the Scholander apparatus; gas volumes were measured either with a spirometer or with a dry gas meter; and oxygen saturation was calculated from the oxygen tension and pH.

Portable Oxygen Equipment

Apparatus.—The apparatus was essentially that described by Cotes and Gilson (1956a, 1956b) and made by the Walter Kidde Company (Fig. 1). The oro-nasal mask did not always fit well, so that too much air tended to be drawn in around the facepiece during inspiration. We therefore also used a mouthpiece, which ensured that the O₂ in the reservoir bag was all inspired early in inspiration. This mouthpiece is like those used in physiological studies (Fig. 2). It is less comfortable than a mask, but as the chief use of the apparatus is during short periods of physical exertion the disadvantage is slight.

Clinical and Physiological Effects

The patient's clinical condition was variable and it was not possible to study all the factors at once, so a number of short controlled trials were performed; these gave the following information. (1) Wearing the apparatus with no O₂ flowing (unknown to the subject) did not increase exercise tolerance. (2) O₂ improved exercise tolerance threefold to sixfold. Without it he could walk 30–70 yards (27–64 metres), and with it 150–300 yards (137–274 metres). He was much more distressed at the limit of tolerance when exercising without added O₂ and his pulse rate was much higher. (3) The use of the "low" flow rate of the apparatus (2 l. O₂/min.) was as effective as the "high" (4 l./min.).

(4) The oro-nasal mask was as good as the mouthpiece if care was taken to keep it fitting properly. (5) A reservoir bag that permitted partial rebreathing was not as satisfactory as one that did not.

In Table II the effects of exercise on the arterial blood gas tensions are shown. This experiment was carried out as follows. The patient breathed with the apparatus at the selected flow rate for 10 minutes. He then exercised by stepping on and off an 8-in. (20-cm.) step 15 times in one minute (the most severe exercise he could perform without added O₂). Arterial blood was drawn immediately afterwards.

The following conclusions may be drawn from the results shown in Table II. (1) When exercising without added O₂ the arterial CO₂ tension increased only slightly above the resting value, showing that ventilation was increased sufficiently to maintain the composition of the alveolar air.

TABLE II.—Effects of Portable Oxygen on Arterial Blood During Exercise

	Rest	Exercise		
	Arterial Blood	Air	Air + O ₂ 2 l./min.	
			Mask	Mouthpiece
CO ₂ tension (mm. Hg)	59	62	70	73.5
pH	7.32	7.30	7.25	7.27
O ₂ tension (mm. Hg)	52	27	53	49.5
O ₂ saturation (%)	83	45.5	80	78

FIG. 1.—170-litre DH type portable oxygen set. Approximate weight, 3½ lb. (1.6 kg.); length, 18 in. (46 cm.); diameter, 3 in. (7.5 cm.).

FIG. 2.—Mouthpiece and reservoir bag.

The fall in the arterial O₂ tension and saturation must therefore have been due to deficient O₂ transfer from the alveolar air to the pulmonary capillary blood. (2) When exercising with added O₂ the arterial CO₂ tension rose significantly, showing that the ventilation was less than when exercising without added O₂. The increase in the O₂ tension and saturation must therefore all be attributed to the higher O₂ concentration in the inspired air. (3) The differences between the findings with the mask and with the mouthpiece are probably not significant.

It is noteworthy that the improvement in the arterial O₂ tension was obtained with a flow rate of only 2 l. O₂/min. added to a pulmonary ventilation of about 15 l./min. This result can be expected only with well-fitting masks.

Portable Walking-Aid

Apparatus.—The patient noticed that he was able to walk about the ward with much less distress if he leaned on a dressing trolley. A walking-aid of the type described by Hendry (1956) and manufactured by Messrs. Fordham Pressings, Ltd., of Wolverhampton, was obtained and modified to the patient's design as follows. Both hand-grip assemblies were removed from the standard model. Two new T-shaped brackets were made with the top pieces offset, longest end forward and sloping upwards from rear to front (see Fig. 3). A platform approximately 16 by 12 in. (40 by 30 cm.) was made to fit on top of the T-shaped brackets. On one side of the under-surface of the platform were placed two metal spring clips which mate with the top arm of the T-bracket and lock the machine in the open position. At the other end of the under-surface of the platform two brackets were fitted carrying a spindle which fitted through the tubular arm of the T-bracket. This arrangement enabled the platform to be swivelled downwards when the machine was folded up. The hand brake was fitted to the front end of the platform with a finger-control lever. A piece of foam rubber was cut to fit on the platform.

FIG. 3.—Portable walking-aid.

Clinical and Physiological Effects

Table III shows some of the effects of using the walking-aid. The use of the support caused little change in the ventilation at rest, but there was a decrease in the O₂ uptake and an increase in the arterial O₂ tension. These changes could be due to a decrease in the O₂ cost of standing or to a decrease in the O₂ cost of breathing, or to both. Studies on a normal subject whose O₂ cost of breathing was known to be negligible showed that the use of the support caused no decrease in total O₂ consumption. This finding suggests, therefore, that the reduced O₂ consumption of the patient when using the support was due in considerable part to a decreased O₂ cost of breathing. With special techniques (Campbell, Westlake, and Cherniack, 1957) the O₂ consumption of the patient's respiratory muscles when seated with back and shoulders supported was found to be about 40 ml./min. at a ventilation of 12 l./min. and 80–100 ml./min. at a ventilation of 18 l./min. On inspection and palpa-

TABLE III.—*Effects of Portable Walking-Aid (January, 1957). Level of Exercise Was Not Standardized, But Was Always Greater When Walking With Support than Without It*

	Unsupported	Supported
Ventilation (l. min. B.T.P.):		
Standing still	12.6	13.2
Walking	14.8	18.7
Maximum voluntary	19.5	26.5
Vital capacity	2.7	2.7
O ₂ consumption (ml. min. S.T.P.):		
Standing still	335	298
Walking	472	502
Arterial blood:		
O ₂ tension (mm. Hg) and (saturation %):		
Standing still	32 (56)	40 (70)
Walking	26 (44)	33 (59)
CO ₂ tension (mm. Hg):		
Standing still	69	65.5
Walking	62	64.5

tion it was obvious that standing unsupported demanded much more use of the extensors of the back and the elevators of the shoulders during inspiration. This increased effort must have caused the O₂ cost of breathing to be even greater when standing than when sitting. Use of the walking-aid obviously reduced this extra effort and probably accounted for the smaller total O₂ intake when standing leaning on the trolley.

Table III also shows that during exercise the patient was able to maintain a greater ventilation and O₂ consumption when supported, and it is probable that a higher proportion of the total O₂ uptake was available to the rest of the body. The changes in the arterial blood gas tensions show that gaseous exchange was adequate to meet the increased requirements of exercise when the support was used.

Use of the support did not increase the vital capacity (or the inspiratory capacity). It did, however, enable him to take a much greater tidal volume with much less effort. It is noteworthy that the level of ventilation he was able to maintain when walking with the support was almost as great as his maximum voluntary ventilation when unsupported.

Simple trials showed that his exercise tolerance was increased sixfold to tenfold by the use of the walking-aid. Controlled trials showed that the combined use of the walking-aid and portable O₂ equipment further increased his exercise tolerance, and he was usually able to walk over half a mile (800 metres) without distress. These controlled trials also showed, in contrast to those performed without the walking-aid, that the "high" O₂ flow rate was more effective than the "low" flow rate, presumably because of the higher ventilation and O₂ consumption attained.

The main aims of physiotherapy in emphysema are increased diaphragmatic movement and relaxation of the muscles of the neck and shoulder-girdle. Despite intensive efforts under the guidance of a physiotherapist trained at the leading school of breathing exercises the patient was unable to increase diaphragmatic movement as judged radiologically, and unless he used his accessory muscles his ventilation in the erect posture was clearly inadequate. (He could not tolerate this type of breathing long enough for full measurements to be made, but his tidal volume fell from 530 to 480 ml.) The walking-aid supported his shoulders and caused relaxation of many muscles of the neck and back. The result is therefore not as greatly at variance with the aims of physiotherapy as might initially appear.

Discussion

The assessment of measures designed to help patients with emphysema is always difficult because the degree of breathlessness is very variable and may not correlate with measurable changes in respiratory function. This patient's case was exceptional in that the severity of his condition set physiological limits to his exercise tolerance which could be measured without having to rely on his subjective assessment of dyspnoea.

Although he can walk 30–70 yards (27–64 metres) unsupported and without O₂, this is only at the expense of an O₂ debt and severe desaturation of the blood. At such times his distress and cyanosis are extreme and his pulse rate usually exceeds 130 per minute. He is unable to main-

tain any steady level of exercise, and is unable to stand still unsupported for more than 10 minutes. Consideration of the work of breathing suggests that the O₂ cost of standing and breathing unsupported is greater than the O₂ uptake by the lungs. Any attempt to increase the O₂ uptake by increasing ventilation causes the respiratory muscles to use more O₂ than is taken in. This suggestion is difficult to prove but is supported by the fact that the arterial O₂ tension is lower when standing unsupported than when standing supported or sitting. In marked contrast to his distress when standing unsupported is his relative comfort when walking with the support of the trolley and using the portable O₂ equipment.

Modes of Action

When considering measures such as these it is convenient to regard the functional disorders of patients with emphysema as grouped in two categories: (1) difficulty in moving the air in and out of the lungs (impaired ventilatory function); and (2) difficulty in exchanging gases between the alveolar air and the blood (impaired alveolar-respiratory function). Impaired ventilatory function affects both O₂ and CO₂ exchange. If the impairment is severe the alveolar air and arterial blood have a lowered O₂ tension and a raised CO₂ tension. Impaired alveolar-respiratory function causes a low arterial O₂ tension but has a negligible effect on the arterial CO₂ tension. Detailed function studies of the patient showed that the low arterial O₂ tension and saturation at rest are almost equally due to both types of limitation. The further lowering of O₂ tension by exercise is, however, predominantly due to limitations of the second type. The walking-aid helps the patient by increasing the ventilation he can comfortably maintain and probably also decreases the O₂ cost of breathing, thereby making more O₂ available to the rest of the body. The portable O₂ equipment helps him by increasing the alveolar O₂ concentration, thereby facilitating the diffusion of O₂ into the blood and preventing the severe hypoxia brought on by exercise.

Selection of Patients

There are several reasons why it is not desirable to try these measures and simply to accept the patient's opinion about their benefit. Firstly, the sets of equipment described are moderately expensive; secondly, only a minority of patients are likely to be helped; thirdly, the patient's assessment of improvement in this condition is unreliable. There are few places where facilities exist for physiological studies such as those described in this paper. Fortunately, clinical guidance can be derived from the known functional disturbances in emphysema as described above. A useful clinical assessment of the relative importance of ventilatory and alveolar-respiratory insufficiency can be obtained by exercising a patient to the limit of his tolerance. He will, of course, be very breathless whichever type of disorder is dominant. However, if his arterial blood is not significantly desaturated—that is, he does not become very cyanosed—his limitation is probably ventilatory and he is having equal difficulty with CO₂ excretion and O₂ uptake. If he develops arterial desaturation he probably has an important degree of alveolar-respiratory insufficiency which limits O₂ uptake but not CO₂ output.

(1) *Portable O₂ Equipment.*—It follows, therefore, that portable O₂ is only likely to help patients who become cyanosed on exercise; that is to say, those with predominant alveolar-respiratory insufficiency.

(2) *Portable Walking-Aid.*—A portable walking-aid enables the accessory respiratory muscles to ventilate the lungs at less O₂ cost and with less discomfort. In few patients with emphysema, however, is the ventilatory inadequacy seriously aggravated by difficulty in using the accessory muscles. In most patients the respiratory muscles function adequately but the ventilation is limited by airway obstruction. Such patients do not require a walking-aid. Clinically the patients who are most likely to be helped by a walking-aid are those who have received full treatment to reduce airway resistance and who have had skilled instruction in breathing exercises but who still require to use the

back and shoulder muscles when walking. Orthopnoea is not by itself an indication, as it may be due to other factors such as congestive heart failure. Our patient sleeps lying flat. Clearly, therefore, only a minority of patients with emphysema will benefit from these measures. Moreover, there are obvious practical limitations to their usefulness. However, the condition is so common and the disability of those severely affected is so distressing that any measures that can help them should be welcomed.

Summary

A case is described in which the patient was incapacitated by diffuse pulmonary emphysema. He has been greatly helped by portable oxygen equipment and by a portable walking-aid.

Physiological studies are described which show how these measures have improved his pulmonary function. Simple clinical methods can help to indicate which patients are likely to be helped by these measures.

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TREATMENT OF CHILBLAINS BY PHENOXYBENZAMINE

A CLINICAL TRIAL IN GENERAL PRACTICE BY THE SOUTH-EAST SCOTLAND FACULTY OF THE COLLEGE OF GENERAL PRACTITIONERS*

In 1954 a member of the Scottish Research Advisory Panel of the College of General Practitioners suggested that an investigation should be made into the effect on chilblains of a new substance, phenoxybenzamine, which opposes the vasoconstrictor action of the sympathetic nervous system. The Faculty Research Committee therefore designed a questionnaire to elicit not only data about the effect of this new substance but also a considerable amount of information on the natural history of chilblains and some of the causative factors. That inquiry was a failure in spite of much hard work by many of the practitioners taking part. The main reason for failure seemed to be a too ambitious and complex questionnaire.

The following winter, 1955–6, it was decided to repeat the inquiry on simpler lines. The Committee therefore prepared a new questionnaire, but it failed to satisfy our statistical adviser, who demonstrated its shortcomings and eventually redesigned the experiment.

Two lessons have been learned as a result of these early efforts: first, the importance of a simple questionnaire, and, second, the need of preliminary advice from a statistical expert.

Method

Arrangements were made with a pharmacist that the test substance, phenoxybenzamine, and lactose, as a control,

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should be distributed to the doctors taking part in such a way that neither doctor nor patient would know which was being administered. Capsules of 10 mg. of phenoxybenzamine and capsules of lactose were put in similar bottles, each bottle having an individual code number, the code being known only to the pharmacist. The practitioners concerned received equal numbers of bottles of phenoxybenzamine and of lactose, but the sequence of each set differed.

It was agreed that the patients should be told the object of the trial, that they should be given an explanation of the "double-blind" method, and that neither doctor nor patient should be asked to take part in this trial if either knew of any other medical substance which was believed by them to be effective in the treatment of chilblains.

The action of phenoxybenzamine was explained to each doctor taking part and the possible undesirable effects were described. The patients were told that they were taking part in the trial of a new drug, and treatment was begun cautiously with one capsule a day. If no undesirable effects had occurred and if cure had not been achieved at the end of the first week the dose was increased to two capsules a day during the second week. Similarly, the dose was raised to three capsules a day during the third and fourth weeks. If cure had been achieved at the end of the fourth week, treatment was continued with the same substance—whether it was phenoxybenzamine or lactose—until the end of the chilblain season or until the patient defaulted. If, on the other hand, there had been no cure at the end of four weeks, capsules known to contain phenoxybenzamine were given, beginning cautiously and continuing until the doctor felt that enough treatment had been given or the patient wished it to be stopped. The doctor might, if he thought fit, decide to give another form of treatment at the end of the fourth week. All treatment after the first four weeks, whether by phenoxybenzamine or lactose, was regarded as outside the trial, and none of these later results was used in the assessment. The trial, for any patient, lasted therefore for four weeks or less, but never more.

During the period of trial each doctor was expected to state in the appropriate column of the card, at the conclusion of each period—first, second, third, and fourth weeks—whether the outcome of the treatment was "good," "fair," or "poor." Good meant complete remission of the chilblains, poor meant no apparent effect from treatment, and fair meant some clinical improvement. The doctor was also asked to give in the appropriate space a final assessment at the end of each individual's trial period of four weeks or less.

It was eventually decided, since the numbers were relatively small, to add those assessed fair and poor together for statistical purposes.

Results

Of the 128 patients who took part in the trial, only 62 completed the course; 41 fell by the wayside in one way or another, but had continued treatment long enough to make analysis of the results possible; 9—all of whom, as was shown later, were taking phenoxybenzamine—had to be withdrawn from the trial because of undesirable side-effects; 7 defaulted so early that no results could be recorded; in 3 cases the cards were spoiled because of misunderstanding; and in 6 others the information was inadequate.

Table I shows the age and sex distribution of the 121 patients involved. It confirms our clinical impressions that chilblains are less common among older people and that they are found much more often in females than in males. It also shows that 59 were treated with phenoxybenzamine and 62 with lactose—about equal numbers.

Table II shows the results of treatment. The two groups that are most strictly comparable are the 28 patients who received phenoxybenzamine continuously for four weeks, and the 34 who had lactose continuously over a similar period. The months when these patients received their treatment are shown in Table III.