CHRONIC RESPIRATORY FAILURE AND PHYSICAL RECONDITIONING: CASE STUDY OF AN ELDERLY OBESE WOMAN

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ABSTRACT

A case is described of a 67 year old obese white woman who had a history of multiple medical problems and who was in chronic respiratory failure but responded poorly to intermittent positive pressure breathing, chest physiotherapy, and supplementary oxygen. She was treated successfully with a 600 k.cal diet and a 26-day physical reconditioning programme. Reconditioning techniques included free and treadmill walking, stair climbing, bench stepping, light calisthenics, and breathing retraining. Improvements were noticed in blood gases, spirometry, electrocardiogram, motor coordination, and physical working capacity.

KEY WORDS

Chronic obstructive pulmonary disease, dyspnoea, oxygen uptake, peak expiratory flow rate, maximal breathing capacity, diffusing capacity, pH, PCO₂, PO₂.

INTRODUCTION

Methods commonly employed in treating chronic obstructive pulmonary disease (COPD) include supplemental oxygen, intermittent positive pressure breathing (IPPB), assisted ventilation, endotracheal intubation, and tracheostomy (Conference on the Scientific Basis of Respiratory Therapy, 1974). Other methods which have been found useful are increasing arterial oxygen tension by decreasing lung water with diuretics and improving cardiac output with cardiotonic and vasopressor agents (Smith, 1976).

These are considered merely palliative measures in the inexorable course of disability and death commonly associated with COPD. Significant improvements in the clinical status of COPD patients can be achieved, however, by two additional modalities, physical reconditioning and breathing retraining (Degré, Sergysels, et al., 1977). These techniques have been applied both to mildly disabled patients and those who have progressed to congestive heart failure (Shephard, 1977).

Although it is not uncommon for COPD patients to manifest medical problems other than respiratory disease, the authors are not aware of a study of the effects of physical reconditioning on patients in chronic respiratory failure combined with as many additional problems as the case under reference.

THE PATIENT

Her history including atherosclerotic coronary vascular disease with congestive heart failure, myxoedema, systemic and pulmonary hypertension, diabetes mellitus, and obesity. Her weight had escalated to 159 kg. in the past. Redundant fat pads requiring surgery were present on both arms and several skin grafts to alleviate cellulitis of the lower extremities had been performed. She had endured these conditions remarkably well until a recent admission to the Plastic Surgery Service where she had previously been followed as an outpatient for leg ulceration.

HOSPITAL COURSE

An obese 67-year old white woman (weight 123 kg, height 160 cm) was admitted for treatment of cellulitis and lymphangitis of the right leg accompanied by fever and persistent pain. On initial examination, her temperature was 38°C; pulse, 72 beats/min, irregular and full; blood pressure, 136/80 mm Hg (controlled from 180/100 by Aldomet (methyl dopa) and a low salt diet).

The patient was in no acute distress. The skin was remarkable for erythema and pitting lymphoedema of the right leg. Head, eyes, ears, nose, and throat were essentially normal. Examination of the chest showed bibasilar rales. A Grade III/VI systolic ejection murmur and an S 3 gallop were present. Examination of the abdomen disclosed a marked panniculus and hepatomegaly.

The initial clinical impression was that of chronic myxoedema, cellulitis, lymphangitis of the right leg, congestive heart failure, and obesity, with the possibility of Pickwickian Syndrome accompanied by recurrent pulmonary emboli. Treatment, which included intravenous antibiotics, was initiated.

Shortly thereafter, the patient was observed to be in respiratory distress and subsequently was transferred to the Medical Intensive Care Unit. She was intubated and
placed on a respirator, beginning a protracted hospital course.

Initial blood gas measurements demonstrated a pH of 7.24, PCO₂ of 62.4 mm Hg, and PO₂ of 58.0 mm Hg, compatible with acute respiratory failure associated with hypoventilation and compounded by pulmonary congestion due to organic heart disease which was treated with massive doses of Lasix (frusemide) and Digoxin. The patient was observed to be in atrial fibrillation which persisted despite the discontinuation of Digoxin.

**EXERCISE PROCEDURE**

Spirometric and arterial blood gas measurements were obtained before and after training. A Godart Expirograph was used for obtaining the spirometry data with appropriate corrections being made to BTPS. Residual volume measurements were not made because exposure of the patient to pure oxygen for 7 minutes, as required by the nitrogen washout procedure, was considered hazardous. Arterial blood gas samples were collected in heparinized syringes from the right radial artery following injection of 0.3 cc of 1% Xylocaine. Samples were analyzed with an Instrumentation Laboratory Model 13 Blood Gas Analyzer for pH, PCO₂, and PO₂.

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>Arterial Blood Gas Measurements</th>
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<tbody>
<tr>
<td></td>
<td>pH</td>
</tr>
<tr>
<td>Predicted</td>
<td>7.40</td>
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<tr>
<td>Initial evaluation (63 days pretraining)</td>
<td>7.33</td>
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<tr>
<td>Second evaluation (61 days pretraining)</td>
<td>7.24</td>
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<tr>
<td>Pretraining evaluation (7 days pretraining)</td>
<td>7.43</td>
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<tr>
<td>Post-training evaluation</td>
<td>7.41</td>
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</tbody>
</table>

These data are representative serial blood gas determinations made during the course of the study.

Electrocardiograms were monitored during training by means of radio-telemetry using a bipolar CM₅ lead. Stride length and respiratory rate were also recorded.

For metabolic measurements, an Otis-McKerrow breathing valve was attached to a mixing chamber from which periodic samples of expired air were drawn into 50 cc hypodermic syringes. Ventilatory volume was assessed by means of a CD4 Parkinson Cowan dry gas meter and expired air samples analyzed with the micro-Scholander technique.

A vigorous therapeutic programme, including a 600 k.cal. diet, was instituted with the onset of acute respiratory failure. The patient failed to respond satisfactorily to intermittent positive pressure breathing (IPPB) with a bronchodilator as well as to chest physiotherapy and nasal oxygen.

<table>
<thead>
<tr>
<th>TABLE II</th>
<th>Spirometric Measurements</th>
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<tbody>
<tr>
<td></td>
<td>Predicted</td>
</tr>
<tr>
<td>Forced Vital Capacity, Litres (FVC)</td>
<td>2.97</td>
</tr>
<tr>
<td>Forced Expiratory Volume₁ (FEV₁/FVC,%)</td>
<td>81.2</td>
</tr>
<tr>
<td>Peak Expiratory Flow Rate, Litres/sec (PEFR)</td>
<td>4.61</td>
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<tr>
<td>Maximal mid-expiratory Flow Rate, Litres/sec (MMEFR)</td>
<td>2.56</td>
</tr>
<tr>
<td>Maximal Breathing Capacity, Litres/min (MBC)</td>
<td>94.26</td>
</tr>
</tbody>
</table>

*Not Measurable

When her condition had been sufficiently stabilized (Table 1, pretraining evaluations), including a reduction in weight to 101 kg., a progressive physical reconditioning programme was initiated in the Cardiovascular Research Unit (CRU). The patient had been bedfast up to this time.

Treadmill walking, light calisthenics, stair climbing, bench stepping, free walking, and abdominal-diaphragmatic breathing (ABD) with pursed lips (PLB) were the physical reconditioning techniques used. Training sessions were scheduled for 26 days. They were conducted in the mid-afternoon under controlled environmental conditions. A tracheostomy was performed during the second week of training. Two sessions were not completed because the patient was unduly tired. A standard warm-up of free walking and stair climbing, which was progressively increased up to one flight of nine stairs, preceded the primary mode of exercise, treadmill walking at 0.7 mph and zero percent grade.
Light calisthenics, bench-stepping at a self-determined pace, and ABD were practiced initially in the CRU and subsequently performed by the patient in her room. She was requested to perform these three times daily, progressing from 8 to 16 repetitions, and to record them in a diary. A small stool, 23 cm in height, served as the bench-stepping device. It was placed by the window, thus permitting the patient to maintain her balance by holding onto the windowsill. Calisthenics included half squats, heel raises, arm circling, and seated trunk twisting.

RESULTS AND DISCUSSION

As training proceeded the patient became less apprehensive of the dyspnoea experienced in earlier sessions and progressed from difficulty with ambulation to assisted walking and finally to unassisted walking with little or no dyspnoea. Her increased confidence and improved motor coordination were reflected during treadmill exercise by an 11 per cent increase in stride length (average increase of 2.5 cm per step in the final session).

Treadmill walking time improved from 4 to 7.5 min. during the training programme. Respiratory rate declined from 34 to 30 per min. and heart rate from 150 to 140 during the fourth minute of treadmill exercise.

Bates, Macklem, and Christie (1971) consider patients with impaired pulmonary function to be in respiratory failure when their arterial PO₂ is below 60 mm Hg or the arterial PCO₂ above 49 mm Hg at rest. Campbell (1967) has proposed two general categorizations of respiratory failure in this respect: A Type I category in which hypoxaemia is accompanied by a normal or lowered PCO₂; a Type II group in which the arterial PCO₂ is elevated and the PO₂ lowered.

As shown in Table I, this patient could be classified as a Type II case of respiratory failure at the time of the initial, second, and third evaluations. The pH of 7.24 suggests the onset of respiratory acidosis (Petty, 1975), p. 32). These blood gas data were associated with Pickwickian hypoventilation and were compounded by pulmonary congestion due to organic heart disease. Post-training measurements show that the patient no longer exhibited the clinical criteria for respiratory failure. Further, during the course of training, oxygen therapy was halted, the tracheotomy tube was removed and IPPB treatments taken only three times daily as opposed to every two hours around the clock.

Spirometry measurements (Table II) clearly show improved ventilatory (i.e., lung bellows) function. Noteworthy is the improvement in PEFR and almost a twofold increase in MBC. This was accompanied by an improvement in diffusing capacity (CO, ml/min. mm Hg) in the pre- to post-training period from 6.9 to 9.9 (Predicted: 10.5).

An oxygen uptake value of 4.11 ml/kg/min was the peak value observed under the symptom-limited conditions of work performed during the final training session. By comparison, the range of maximal oxygen uptake for normal women aged 60 to 69 is 27 to 35 ml/kg/min (Ryhming, 1960). Figure 1 shows this patient’s post-training exercise ECG response to be somewhat less severe than in the pretraining period for

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**Figure 1:** Pre- and Post-training electrocardiograms, lead CM₅, with the treadmill set at .7 mph and zero % grade. These tracings were made in the fourth minute of exercise.
the same workload. The posttraining trace reveals fewer premature ventricular contractions and less S-T segment depression. Atrial fibrillation persists.

The mechanisms underlying increased physical working capacity in COPD patients are unclear. Some investigators discount improved ventilation (Agle, Baum, et al, 1973) and there is little reason to assume that physical training can reverse alveolar tissue destruction, as Cumming (1973) has pointed out. Nevertheless, the demands on the respiratory system may be decreased during submaximal work by improved motor coordination and enhanced peripheral oxygen extraction (Holloszy, 1976), and from the patient's point of view, a major criterion is performance by whatever means are available.

The subject in the present study was an inpatient and was trained in the Cardiovascular Research Unit laboratory, possibly providing distinct advantages: environmental temperature and humidity were controlled and performance closely supervised. The technique of gradually increasing exercise intensity for COPD patients training in the presence of medical personnel appears to lessen apprehension and eventually permit the continuance of training without such supervision. This is especially important in assisting the COPD patients to overcome the fear of dyspnoea that leads some to avoid physical activity completely.

Although the gains reported here are modest, they represent, for this woman, the difference between being bedfast and ambulatory. Before being discharged for follow-up in the Medical Clinic, dietary and exercise recommendations were made, and an oxygen tank provided. She was also to be followed by the Plastic Surgery Clinic for her chronic lymphoedema and venous stasis changes in the lower extremities.

ACKNOWLEDGEMENTS

The writers thank Dr. George J. Horner, Medical Director, Wyeth International Laboratories, Philadelphia, Pennsylvania, for his generous collaboration in the preparation of this manuscript.

This study was supported in part by Grant No. 13-P-57759 from the Rehabilitation Services Administration Department of Health, Education and Welfare, Washington, D.C., and contract No. 494606 from the department of Health of the Commonwealth of Pennsylvania.

REFERENCES


