THE EFFECT OF MECHANICAL EXSUFFLATION ON RESPIRATORY GAS EXCHANGE IN CHRONIC PULMONARY EMPHYSEMA.

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by

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Introduction

An elevated arterial carbon dioxide tension is not infrequently found in patients with pulmonary emphysema. Donald and Christie (1) and Wilson et al (2) have shown that such patients are unable to lower their arterial pCO_2 significantly by voluntary hyperventilation. This mechanical defect in the control of blood carbon dioxide tension makes these patients particularly susceptible to the development of respiratory acidosis. Associated with these events is a depression of the response to increases of carbon dioxide tension so that anoxic stimulation of the carotid and aortic bodies becomes the prime stimulus for breathing (3). When oxygen therapy is instituted in an attempt to alleviate the anoxia the obviously distressed cyanotic patient may become almost apneic, drowsy and even comatose. This poses a difficult therapeutic problem.

The immediate object in the therapy of such patients is to provide an adequate lung ventilation which will overcome the anoxia and increase the elimination of carbon dioxide. Various measures aimed at counteracting respiratory acidosis have recently been advocated. These include the use of intravenous sodium lactate (4) pneumoperitoneum (5) electrophrenic respiration (6,7) and mechanical respirators using intermittent positive pressure breathing applied to the upper airway (8) or as applied in the conventional tank respirator (9, 10).

The purpose of this paper is to present the effect of the mechanical exsufflator on respiratory gas exchange in chronic pulmonary emphysema under various conditions, to compare its effect to that of the conventional tank respirator, and to describe their combined use in the treatment of a severely ill emphysematous patient who developed respiratory acidosis.

Methods

Mechanical exsufflation, devised as a means of eliminating bronchial secretions in patients with an ineffective cough (11, 12) is accomplished in a conventional tank respirator by producing a negative intratank pressure of 40 mm. Hg. for full inflation of the lungs, and, by means of a swiftly opening butterfly valve, returning the intratank pressure to atmospheric in 0.06 seconds. Expiration therefore, in contrast to that in other forms of intermittent pressure breathing, is not impeded except for the initial 0.06 seconds. Expiratory volume flow rates measuring 60% of those obtained during maximally vigorous coughs in normal subjects have been attained by this procedure (13).

In this study the exsufflator was cycled at 9 times a minute. The inspiratory pressure was built up over a 2 second period, thus allowing 4.6 seconds for expiration. The observations on the effect of the conventional tank respirator were made using an Emerson respirator cycled 17 times a minute with a pressure range of -20 to - 8 cm. water, inspiration and expiration being approximately equal.

Studies were made on eight cases of chronic pulmonary emphysema in whom retention of carbon dioxide due to impaired pulmonary ventilation was suspected. All reclined in a conventional tank respirator for 30 minutes before any experiment was begun in order to obtain a resting level. The exsufflator was attached to the respirator so that the use of one could be followed immediately by the other.

To determine the effect of these procedures and of the administration of oxygen, pulmonary ventilation was measured on a Benedict-Roth respirometer. Arterial blood samples were drawn by means of an indwelling Cournand needle and the Van Slyke-Neill technique

was used for the determination of oxygen content and capacity and carbon dioxide content (14). The arterial pH was determined on a Beckman pH analyzer and the carbon dioxide tension derived from the Henderson-Hasselbach equation.

Results

In four cases the effect of the exsufflator was compared to that of the conventional tank respirator. In case 1, 30 minutes of exsufflation was followed immediately by 60 minutes of respirator treatment. This procedure was reversed in case 2 and broken into two parts in cases 3 and 4, the patients receiving exsufflation for 30 minutes one day and respirator treatment for 60 minutes the next.

The effect of these two procedures on minute volume, arterial pH, carbon dioxide tension, and oxygen saturation is shown in Fig. I. The exsufflator resulted in a marked rise in pH, a rise in oxygen saturation averaging 4.6% and a fall in carbon dioxide tension averaging 10 mm.Hg. while the respirator produced no significant change in any of these measurements. It is seen that the minute ventilation was practically unchanged from the resting control level during respirator therapy while the exsufflator increased the average minute volume by 4 liters per minute or by 55%. The tidal volume, unchanged by the respirator, was increased to three times that of the control level when exsufflation was applied (Table I).

Respiratory acidosis was produced in three patients by the administration of 100% oxygen by mask. Cases 5 and 6 received oxygen for 60 and 30 minutes respectively and were treated with the exsufflator

immediately following cessation of oxygen. The exsufflator was applied for 30 minutes in case 5 and 15 minutes in case 6. Case 7 received oxygen for 90 minutes and exsufflation for the last 30 minutes of administration.

In all three cases oxygen caused a rise in carbon dioxide tension and fall in arterial pH. Table II shows that in cases 5 and 6 exsufflation promptly reversed these changes, the values becoming even more normal than in the control period. The complete reversal of acid-base disturbance when exsufflation was applied in Case 7, despite the continued administration of 100% oxygen is illustrated in Table III.

The usefulness of the exsufflator in the treatment of a patient with severe emphysema who developed a marked respiratory acidosis was demonstrated in the following case:

> Case 8 (A.M.)- a 53 year old white male, was admitted to hospital on July 5, 1952 complaining of severe dyspnea of seven months duration. He had been in good health until January, 1952 when he developed tightness in his chest, dyspnea and cough with expectoration of green sputum. Despite antibiotic therapy these increased in severity and he required admission to hospital for a short time in February and again in March. He was found to have pulmonary emphysema with bronchopneumonia and possible early cor pulmonale. Vital capacity was 2.0 liters. He improved markedly when antibiotics and digitalis were administered and phlebotomy performed and was discharged from hospital. On June 15 his dyspnea increased and by July 4, 1952 his symptoms had become very severe. He was then given continuous oxygen by mask at 6-7 liters per minute. Drowsiness was noted and he was taken to hospital.

On admission he was drowsy, cyanotic and extremely dyspneic. The jugular veins were distended. Chest was barrel-shaped and movement was limited bilaterally. Hyperresonance, distant breath sounds and bilateral scattered ronchi were present. Blood pressure was 140/85. The liver was palpable one and one-half finger-breadths. There was pitting edema of both ankles and slight clubbing of the fingers. Hemoglobin was 15.5 gm.%, red blood cells 7.8 million, hematocrit 55% and white blood cells 11,600. Vital capacity was 1.0 liter. The chest film revealed bullous emphysema with some right ventricular enlargement. EKG showed right ventricular strain.

On July 7 further oxygen was administered and he became almost apneic. The arterial pH was 7.31 and the carbon dioxide tension 84 mm.Hg. He was placed in a tank respirator and also received exsufflation for 1/2 hour every 2 hours. A comparison of the effect of the respirator and exsufflator on tidal volume and minute ventilation is shown in Table IV.

He was treated by the combined use of the respirator and exsufflator and oxygen by nasal catheter at a low rate of flow until July 10, 1952. The improvement in arterial pH and carbon dioxide tension are shown in Table V. By July 20th he was up and about, though limited in activity, and discharged from hospital.

Discussion

The results reported above indicate that the exsufflator is superior to the conventional respirator in managing the acid-base disturbance associated with respiratory acidosis in pulmonary emphysema. This might be explained by its ability both to overcome obstruction to breathing (15) and to more effectively ventilate the alveoli.

The presence of bronchiolar obstruction due to either spasm or thick viscid secretions in the bronchi is no doubt a factor in the development of anoxia and carbon dioxide retention in severe emphysema. The exsufflator has been shown to result in a marked elimination of secretions in many cases of emphysema (12). This effect could thus play a large role in the reversal towards normal of altered arterial blood gases and pH.

In the cases treated in the conventional respirator the tidal volume and minute ventilation did not change appreciably from that present during unassisted respiration. However a consistently marked increase in tidal volume occurred during the period of exsufflation. The increase in arterial oxygen saturation and fall in carbon dioxide tension despite a respiratory rate of only nine times a minute is due to the threefold increase in tidal volume resulting in a more effective alveolar ventilation. Case 8 demonstrates that despite only a slight increase in minute ventilation, the marked increase in tidal air produced beneficial results.

No post-exsufflator period of apnea was observed despite the fact that the stimulus of anoxia was removed. As is demonstrated by Case 8 new levels of arterial gases are maintained even after removal from the modified respirator. It seems possible that the sensitivity of the respiratory mechanisms was at least partially restored by the period of normal gas tensions induced by the exsufflator.

Respiratory acidosis developed very quickly in the cases under study when oxygen was administered in high concentration. It is significant that changes in pH and carbon dioxide tension are brought

about so quickly with the exsufflator because acidosis may rapidly prove fatal unless adequate measures are immediately taken. The application of exsufflation has been shown to deal effectively with the harmful accumulation of carbon dioxide, so that oxygen may be administered to patients with emphysema and therefore relieve the consequences of severe anoxia without inducing respiratory acidosis.

Summary

In four cases of pulmonary emphysema 30 minutes of therapy with the exsufflator resulted in an increased minute ventilation and beneficial effect on arterial pH, carbon dioxide tension and oxygen saturation. Respirator therapy for 60 minutes resulted in no appreciable change.

Uncompensated respiratory acidosis was produced in three cases of emphysema by the administration of oxygen. The acid-base disturbance was effectively treated by a short period of exsufflation.

The beneficial effect of the use of the exsufflator in the treatment of a patient with emphysema who developed respiratory acidosis was demonstrated.

The decisive increase in tidal air and minute ventilation with the rapid effect on pulmonary gas exchange during exsufflation appears to answer the problem of therapy in respiratory acidosis more effectively than do other measures previously employed.

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Table 1.

The Effect of the Respirator and Exsufflator on Tidal Volume

Case No.	Control ml.	Respirator ml.	Exsufflator ml.
1	360	312	1133
2	473	467	1400
3	312	369	1075
4	588	569	1700

Table II.

The Effect of Exsufflation on Acid-Base Disturbance Produced by Oxygen Therapy in Pulmonary Emphysema.

Case No.	State	Minute Volume (L/min.)	Arterial	
			pH	pCO_2 (mm.Hg.)
5	Rest	7.65	7.38	46
	O_2 60 min.	6.90	7.33	52
	Exsuff. 30 min.	9.90	7.42	39
6	Rest	9.46	7.35	64
	O_2 30 min.	6.85	7.30	77
	Exsuff. 15 min.	12.74	7.36	57

Table III.

The Effect of Exsufflation During Continuous Oxygen Therapy in a Patient with Emphysema.

State	Minute Volume (L./min.)	Oxygen Saturation (%)	Arterial	
			pH	pCO_2 (mm.Hg.)
Resting	6.8	92.8	7.44	44
Oxygen 60 min.	5.9	98.1	7.36	55
Oxygen and Exsufflation	9.2	99.0	7.42	43

Table IV.

The Effect of the Respirator and Exsufflator on Tidal Volume and Minute Ventilation. (Case 8)

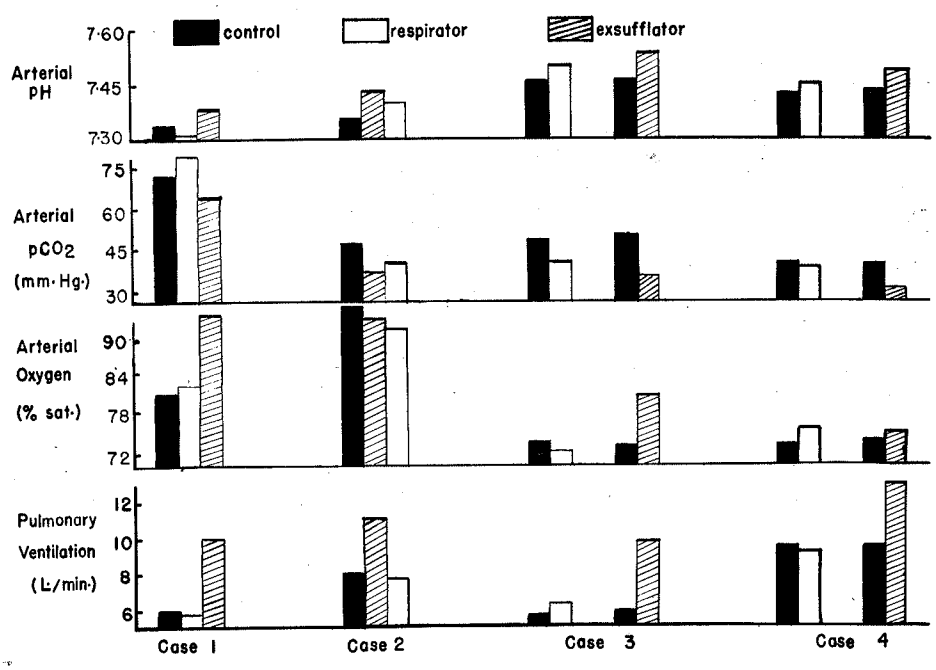
	Tidal Volume (ml.)	Minute Volume (L./min)
Control	286	5.2
Respirator	278	4.7
Exsufflator	645	5.8

Table V.

The Effect of Therapy on Arterial Blood Estimations
(Case 8)

Date	Arterial pH	Arterial pCO_2 (mm.Hg.)	Comment
7/7/52	7.31	84	prior to therapy
7/8/52	7.43	67)	during therapy
7/9/52	7.39	66)	
7/11/52	7.38	63	day after cessation of therapy
7/15/52	7.40	64	out of bed limited activity.

Figure 1.



A comparison of the physiological effects of the respirator and exsufflator in emphysema.