OXYGEN CONSUMPTION DURING SPONTANEOUS VENTILATION WITH CONTINUOUS POSITIVE AIRWAY PRESSURE: ASSESSMENT IN NORMAL VOLUNTEERS AND PATIENTS WITH ACUTE RESPIRATORY FAILURE

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ABSTRACT

Continuous positive airway pressures (CPAP) of 0.49 kPa and 0.98 kPa were applied to ten healthy volunteers and nine critically ill patients with acute respiratory failure. A modified Godart-Statham NV 16003 spirometer was used to measure respiratory frequency (f), tidal volume (V_T), oxygen consumption ($\dot{V}O_2$), and changes in functional residual capacity (ΔFRC). During CPAP of 0.49 kPa, volunteers had a decrease in f , and increased V_T and minute volume (MV). At 0.98 kPa CPAP, f did not change but V_T and MV significantly increased. $\dot{V}O_2$ did not change at either pressure. The volume of ΔFRC increased with an increased level of CPAP. The entire volunteer group was comfortable throughout the whole study.

When CPAP was applied to acutely ill patients, f decreased. V_T and MV increased at both CPAP pressures. ΔFRC was similar to the volunteers. $\dot{V}O_2$ in the patient group rose significantly at 0.49 and 0.98 kPa CPAP. Some of the patients were uncomfortable with 0.49 kPa pressure, while all the patients were distressed at 0.98 kPa CPAP.

The effects of increased oxygen consumption and patient discomfort should be considered in critically ill patients receiving CPAP therapy.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) has been employed to improve pulmonary gas exchange in spontaneously breathing patients. CPAP has been utilized in the treatment of the idiopathic respiratory distress syndrome of infants,¹ and has been applied to adults with acute respiratory failure.^{2,3} Adult respiratory distress syndrome is associated with a decrease in functional residual capacity (FRC). CPAP increases FRC to prevent or reverse closure of small airways and consequently improves oxygenation. In addition, the possible advantages of CPAP over mechanical ventilation with end-expiratory pressure include lower peak airway pressures and a lesser effect on cardiac output.² We applied CPAP to groups of healthy volunteers and to patients with acute respiratory failure in order to compare the oxygen cost of breathing and tolerance in subjects with normal and severely compromised respiratory function. Respiratory frequency (f), tidal volume (V_T), and oxygen consumption ($\dot{V}O_2$) were measured during CPAP. Oxygen consumption was used as a measure of the work of breathing.⁴

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METHODS

Subject selection

Ten normal volunteers without pre-existing lung disease were studied. Their ages varied from 20 to 58 years. Nine critically ill patients with acute respiratory failure were also studied. In two of the patients the studies were repeated on separate occasions. Their age span was 23 to 80 years. Details of the patient's history, surgical procedures and postoperative complications are presented (Table I).

Materials and technique

A Godart-Statham NV 16003 spirometer with a carbon dioxide absorber in the circuit was used to measure respiratory frequency, tidal volume, oxygen consumption and changes in functional residual capacity.

CPAP was applied by placing weights, manometrically equivalent to 0.49 kPa (5 cm H_2O) and 0.98 kPa (10 cm H_2O) on the spirometer bell. The spirometer was modified by increasing the length of the water container to prevent overflow. Silicone bars were used inside the water container to maintain the bell in a vertical position (Figure 1). This modification did not interfere with the free movement of the spirometer bell.

All volunteers were studied in the supine position. A mouthpiece and nose clips, reinforced with adhesive tape, were used to prevent gas

TABLE I
PATIENT DATA

Patient No.	Sex	Age (yrs)	History	CPAP
1	M	52	Ruptured aortic aneurysm, resection, severe G.I. hemorrhage	1
2	F	75	MVA, multiple injuries	2
3	M	23	MVA, quadraplegia, septicemia	1
4	M	74	MVA, multiple trauma, gas gangrene, leg amputation	2
5	M	72	Craniotomy	1
6	F	80	Bowel resection	1
7	M	63	Laparotomy, perforated gall bladder, peritonitis	1
8	M	57	MVA, ruptured diaphragm, splenectomy, bowel resection, fractured ribs, tracheostomy	1
9	M	20	MVA, fractured maxilla, mandible, ribs, pelvis, femur, ruptured diaphragm	1

MVA = motor vehicle accident.

CPAP = number of times observations made during CPAP.

leakage. The patients were also studied in the supine position. They had either a tracheal tube or a tracheostomy tube in place and the cuffs on these tubes were inflated during the study period to prevent airway leakage.

After allowing adjustment to the apparatus for seven minutes while breathing room air, the circuit was closed at end of expiration and respirations were recorded for seven minutes each at 0, 0.49 and 0.98 kPa of water pressure. During this period the subjects breathed 100 per cent oxygen. Between each pressure setting the subject breathed room air for a seven-minute period at zero end-expiratory pressure.

Respiratory parameters were calculated from the spiograph tracing. $\dot{V}O_2$ was determined from the slope of the spirometer recording. The vertical distance from the initial base line to the one established at the end of seven minutes was measured as the litres of oxygen consumed (Figure 2).

Correction for the compression of the oxygen in the total circuit (beneath the spirometer bell and in the spirometer circuit) due to the applied weight was made by calculating the actual

$$\dot{V}O_2 = \frac{\text{volume of oxygen in the total circuit before CPAP} \times \dot{V}O_2 \text{ measured}}{\text{volume of oxygen in the total circuit after CPAP}}$$

Mean f and mean V_T were measured from the

tracings. Mean minute volumes (MV) were calculated as their product. Changes in FRC (ΔFRC) were calculated from the change of the base line after several respiratory cycles with CPAP. No corrective factor was applied to the measured values of ΔFRC .⁵ The compliance of the total respiratory system (C_{rs}) was determined by dividing ΔFRC by the applied pressure for CPAP.

All measurements for each group were performed within the same room. V_T , MV, and ΔFRC were changed to BTPS and $\dot{V}O_2$ was corrected to STPD. The Wilcoxon matched-pairs signed-rank test was used for statistical analysis.

RESULTS

The values measured and calculated from the spirometer tracings are presented for the normal volunteers (Table II) and the patients with acute respiratory failure (Table III).

With a CPAP pressure of 0.49 kPa, the volunteers demonstrated a significant decrease in f , an increase in V_T and MV. When the airway pressure was raised from 0 to 0.98 kPa, f did not change but V_T and MV significantly increased. $\dot{V}O_2$ did not change at the 0.49 and 0.98 CPAP levels. The volume of ΔFRC increased significantly for each level of applied airway pressure. C_{rs} remained unchanged throughout CPAP.

FIGURE 1 Diagrammatic representation of the total circuit for applying CPAP with the modified spiograph. Total circuit equals the volume of oxygen beneath the bell in the apparatus tubes.

Respiratory frequency decreased in the patient group at both levels of CPAP. Tidal volume rose significantly at both levels of CPAP and this was reflected in a significant increase in MV at 0.49 and 0.98 kPa CPAP. $\dot{V}_{O_2}$ increased significantly on changing from zero end-expiratory pressure to 0.49 and 0.98 kPa CPAP. FRC results followed the same pattern as that observed in the normal volunteers and increased with changes to positive pressure breathing.

Further comparison between the two groups shows that respiratory frequency both initially and throughout the study period was twice as fast in the patient group compared to healthy individuals. Tidal volume in the patients with acute respiratory failure was smaller than in normal subjects prior to and during CPAP. A portion of this difference is due to the presence of tracheal

FIGURE 2 $\dot{V}_{O_2}$ (l/min) is calculated by dividing total oxygen consumption by the time. New base line occurs after the application of CPAP. Δ FRC is measured from the new base line to end expiration.

or tracheostomy tubes in the patients. This decreased their dead space and consequently their tidal volume. The volunteer group tolerated CPAP without discomfort, to the point where subjects five and six slept. Some of the ill subjects became uncomfortable at 0.49 kPa CPAP while all patients were obviously distressed at the 0.98 kPa level.

DISCUSSION

The work of breathing consists of three components: elastic work, flow resistance, and inertial work.^{6,7} Mead⁸ and Dubois⁹ found the latter to be negligibly small and recoverable at the end of a respiratory cycle. By neglecting this inertial component, Otis determined that the mechanical work of inspiration could be approximated by the formula

$$W_{\text{insp}} = \frac{1}{2C_{\text{rs}}} V^2_T + \frac{K_1}{4} \pi^2 f V^2_T + \frac{2K_2}{3} \pi^2 f^2 V^3_T$$

where K_1 and K_2 are constants related to the flow-resistive properties of the respiratory system. Expiratory work can also be ignored as it is usually passive except at very high values of ventilation.¹⁰

While not synonymous, the work of breathing has been estimated as the oxygen cost of respiration.¹⁰ During quiet ventilation the work of breathing represents only a fraction of the total oxygen consumption, and as such is difficult to measure.¹¹ Increasing f , particularly above 30 breaths/minute, disproportionately increases this value.¹⁰ In contrast, the supine position enhances the efficiency of ventilation.¹²

The spirometer circuit and techniques of measurement may influence the recorded values. During the application of CPAP with 100 per cent oxygen equilibration of nitrogen occurs throughout the circuit. However, this should be approximately of the same magnitude and in the

TABLE II
RESPIRATORY DATA DURING CPAP: VOLUNTEERS

Volunteer No.	Airway pressure kPa	f (breaths/minute)	V _T (ml BTPS)	MV (l BTPS)	V̇O ₂ (ml/minute) STPD	Δ FRC (l BTPS)	C _{rs} (l/kPa)
1	0	20	573	11.56	289	—	—
	0.49	18	672	12.1	330	0.661	1.3
	0.98	16	760	12.2	339	1.21	1.2
2	0	10	992	9.9	339	—	—
	0.49	13	1378	17.9	395	0.551	1.12
	0.98	16	1686	26.9	411	1.102	1.12
3	0	16	694	11.1	353	—	—
	0.49	12	981	11.8	298	0.386	0.79
	0.98	11	1135	12.5	338	0.529	0.54
4	0	8	716	5.7	173	—	—
	0.49	6	1069	6.4	212	0.331	0.68
	0.98	6	1124	6.7	173	0.441	0.45
5	0	11	838	9.2	266	—	—
	0.49	11	882	9.7	258	0.661	1.3
	0.98	10	992	9.9	249	1.21	1.2
6	0	13	463	6.0	232	—	—
	0.49	12	496	6.0	215	0.386	0.79
	0.98	11	573	6.3	232	0.606	0.62
7	0	15	661	9.9	372	—	—
	0.49	13	827	10.7	380	0.441	0.90
	0.98	11	1080	11.9	388	0.716	0.73
8	0	14	353	4.9	248	—	—
	0.49	10	529	5.3	250	0.419	0.86
	0.98	8	639	5.1	273	0.551	0.56
9	0	21	551	11.6	274	—	—
	0.49	16	716	11.5	265	0.551	1.12
	0.98	14	827	11.6	242	0.771	0.79
10	0	18	463	8.3	430	—	—
	0.49	15	551	8.3	397	0.441	0.90
	0.98	12	716	8.6	372	0.827	0.84
Mean	0	14.6 (4.3)	631 (191)	8.8 (2.5)	298 (76)	—	—
± SD	0.49	12.6 (3.3)	810 (279)	10.0 (3.8)	301 (71)	0.48 (1.2)	0.98 (0.22)
	0.98	11.5 (3.2)	953 (328)	11.2 (6.1)	302 (79)	0.80 (0.29)	0.81 (0.28)
	0-0.49	*	*	*	NS	*	NS
	0-0.98	NS	*	*	NS	*	

f = respiratory frequency.

V_T = tidal volume.

MV = minute volume.

V̇O₂ = oxygen consumption.

Δ FRC = change in functional residual capacity.

C_{rs} = total respiratory compliance.

SD = standard deviation.

* = p < 0.05.

NS = not significant.

same direction at each level of CPAP in individual subjects and, therefore, should not invalidate recorded observations. Increased alveolar pressure after the application of CPAP caused the measured ΔFRC to be larger than the actual ΔFRC.⁵ However, this error is very small at 0.49 and 0.98 kPa and therefore no corrective calculations were

done. The change in FRC on the application of CPAP in our volunteer group is almost identical to that noted by Abboud.¹³ In his study FRC increased 0.4 litres and 0.8 litres after 0.49 kPa and 0.98 kPa respectively.

Within the volunteer population, application of 0.49 and 0.98 kPa caused f and V_T to change in

TABLE III
RESPIRATORY DATA DURING CPAP: PATIENTS

Patient No.	Airway pressure kPa	f (breaths/minute)	V _T (ml BTPS)	MV (l BTPS)	ṀV _{O₂} (ml/minute) STPD	FRC (l BTPS)	C _{rs} (l/kPa)
1	0	25	331	8.3	306	—	—
	0.49	28	419	11.7	363	0.330	0.67
	0.98	28	452	12.7	379	0.551	0.56
2-A	0	44	220	9.7	228	—	—
	0.49	40	264	10.6	259	0.441	0.90
	0.98	38	287	10.9	314	0.661	0.67
2-B	0	24	441	10.6	232	—	—
	0.49	20	496	9.9	248	0.331	0.68
	0.98	20	496	9.9	248	0.551	0.56
3	0	28	551	15.4	494	—	—
	0.49	26	617	16.0	594	0.441	0.90
	0.98	25	749	18.7	594	0.771	0.79
4-A	0	31	276	8.5	218	—	—
	0.49	29	309	8.9	226	0.441	0.90
	0.98	28	353	9.9	242	0.771	0.79
4-B	0	20	386	7.7	223	—	—
	0.49	18	441	7.9	240	0.331	0.68
	0.98	15	551	8.3	265	0.551	0.56
5	0	18	386	6.9	190	—	—
	0.49	18	551	9.9	289	0.220	0.45
	0.98	16	716	11.5	331	0.441	0.45
6	0	38	220	8.4	207	—	—
	0.49	25	386	9.6	248	0.22	0.45
	0.98	25	441	11.0	273	0.551	0.56
7	0	40	231	9.3	223	—	—
	0.49	30	386	11.6	248	0.331	0.90
	0.98	25	496	12.4	273	0.771	0.79
8	0	20	386	7.7	331	0.33	0.67
	0.49	20	551	11.0	397	0.33	0.67
	0.98	15	661	9.9	455	0.55	0.56
9	0	21	441	9.3	314	—	—
	0.49	20	496	9.9	347	0.22	0.45
	0.98	20	551	11.0	372	0.44	0.45
Mean	0	28.1 (9.0)	351 (107)	9.3 (2.3)	270 (88)	—	—
± SD	0.49	24.9 (6.7)	447 (107)	10.6 (2.1)	314 (109)	0.34 (0.9)	0.70 (0.19)
	0.98	23.2 (6.9)	523 (144)	11.5 (2.7)	341 (106)	0.60 (0.12)	0.61 (0.13)
	0-0.49	*	*	*	*	*	NS
	0-0.98	*	*	*	*	*	NS

f = respiratory frequency.

V_T = tidal volume.

MV = minute volume.

ṀV_{O₂} = oxygen consumption.

Δ FRC = change in functional residual capacity.

C_{rs} = total respiratory compliance.

SD = standard deviation.

* = *p* < 0.05.

NS = not significant.

opposite directions. The increase in V_T was sufficiently large to increase MV. Expiratory work can be ignored as *f* was low. However, flow-resistive work might decrease secondary to

increased laryngeal diameter,¹⁴ increased airway conductance^{15,16} and decreased flow-resistance of the chest wall¹⁷ associated with the increased lung volumes. The net result is no appreciable

change in $\dot{V}O_2$ and the work of breathing at a CPAP level of 0.98 kPa in normal volunteers. This was substantiated by subjective statements of the volunteers and obvious comfort during CPAP.

In acutely ill patients, 0.49 and 0.98 kPa CPAP produced similar results. Respiratory frequency decreased, V_T and MV increased at both levels of CPAP. If the values for f , V_T , and C_{rs} are substituted in the formula for respiratory work, an increase in work would be expected. Measurement of $\dot{V}O_2$ supports this theory. Clinically, patient distress became obvious at 0.98 kPa CPAP.

Previous investigation by Barach^{18,19} disclosed an increase in $\dot{V}O_2$ with CPAP. The present study refined these observations and demonstrated that this may become measurable in a patient with respiratory impairment at 0.49 kPa CPAP and distress the adult patient at 0.98 kPa CPAP. This might explain the excellent clinical results obtained in the studies of Civetta² and Glasser³ who utilized levels of 0.49 kPa in two of the three patients they reported.

Increased $\dot{V}O_2$ during CPAP may be limited to adults with acute respiratory failure. Following open-heart surgery, Askitopoulou²⁰ showed no difference in $\dot{V}O_2$ during CPAP and spontaneous respiration in eleven patients. However, these patients had relatively normal pulmonary function following surgery.

Shah²¹ noted differences in $\dot{V}O_2$ in adult patients with respiratory distress syndrome during CPAP and intermittent positive pressure ventilation with and without positive end expiratory pressure (PEEP). On changing from PEEP ventilation to CPAP with the same end expiratory pressure, he found no differences in mean $\dot{V}O_2$ between the two modes of breathing. However, seven of eleven measurements of $\dot{V}O_2$ showed an increase on changing from PEEP to CPAP. In two of his patients he measured $\dot{V}O_2$ at 0.49 and 0.98 kPa CPAP and in both situations $\dot{V}O_2$ was greater with 0.98 kPa CPAP. Therefore, in any individual patient selected for CPAP, the benefits of this mode of therapy should be weighed against the possible deleterious effects of increased oxygen consumption and objective patient distress.

SUMMARY

Continuous positive airway pressure (CPAP) was utilized in spontaneously breathing adult volunteers and in adult patients with acute respiratory failure. Respiratory frequency (f), tidal volume (V_T), change in functional residual

capacity (ΔFRC) and oxygen consumption ($\dot{V}O_2$) were measured. Minute volume (MV) and total lung compliance (C_{rs}) were calculated. These values were determined for CPAP pressures of 0.49 and 0.98 kPa. Oxygen consumption in patients increased with increasing levels of CPAP, but remained unchanged in the healthy volunteers. The increase in $\dot{V}O_2$ coincided with the appearance of clinical discomfort and distress in the patient population. These observations suggest that in addition to measurements of cardiorespiratory performance, levels of CPAP administered to spontaneously breathing patients with acute respiratory failure might be titrated against patient discomfort.

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RÉSUMÉ

On soumis dix volontaires en respiration spontanée à des pressions positives continues (CPAP) de 0.49 et de 0.98 kPa (5 et 10 cm H₂O), ainsi que neuf grands malades en insuffisance respiratoire aiguë. Un spiromètre modifié de Godart-Statham NV 16003 a été utilisé pour la mesure de la fréquence respiratoire, du volume courant, de la consommation d'oxygène et des modifications de la capacité fonctionnelle résiduelle.

Une diminution de la fréquence respiratoire avec une augmentation du volume courant et du volume-minute a été observée chez les volontaires soumis à une CPAP de 0.49 kPa. Si on soumettait ces sujets à une CPAP de 0.98 kPa, la fréquence respiratoire demeurait inchangée alors que le volume courant et le volume-minute s'élevaient de façon significative. La consommation d'oxygène ne changeait pas avec l'une ou l'autre des pressions utilisées chez les volontaires. La capacité fonctionnelle résiduelle augmentait en relation avec l'importance de la CPAP appliquée. Ces sujets sont demeurés confortables durant toute l'épreuve.

La fréquence respiratoire diminuait avec augmentation du volume courant et du volume-minute chez les patients en insuffisance respiratoire aiguë, et ceci aux deux régimes de pression positive. On observait les mêmes modifications de la capacité résiduelle fonctionnelle déjà mentionnées chez les volontaires. Par ailleurs, la consommation d'oxygène s'élevait avec l'utilisation des deux régimes de pression. Avec une CPAP de 0.49 kPa, quelques uns des malades étaient inconfortables, alors que tous l'étaient de façon évidente à 0.98 kPa.

On devrait tenir compte du fait que la consommation d'oxygène est augmentée, ainsi que de l'inconfort des patients lorsque l'on utilise une CPAP chez les grands malades en insuffisance respiratoire aiguë.