EVALUATION OF POSITIVE END-EXPIRATORY PRESSURE THERAPY IN THE ADULT RESPIRATORY DISTRESS SYNDROME*

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HYPOXAEMIC RESPIRATORY FAILURE occurring in association with a wide variety of antecedent illnesses has been termed the Adult Respiratory Distress Syndrome (ARDS) and is being recognized with increasing frequency.¹⁻⁴ It is characterized by low partial pressure of arterial oxygen (PaO_2) and increased right-to-left shunt ($\dot{Q}_s/\dot{Q}_t$), dead space ventilation, and lung stiffness. Many of these patients require controlled mechanical ventilation with 100 per cent oxygen, thereby risking the added problems of oxygen-induced pulmonary dysplasia.⁵ Recently, it has been demonstrated that PaO_2 can be increased and that the inspired oxygen concentration required to produce a haemoglobin-saturating PaO_2 reduced in this syndrome by application of positive end-expiratory pressure (PEEP)⁶⁻¹¹; in fact, this ventilatory modality has been suggested to be the mainstay of treatment.⁴ Three commercially available ventilators (Bennett MA-1, Ohio 560, and Engstrom) have mechanisms for the provision of PEEP and increased use of this means of support in clinical practice is to be anticipated. It is recognized that elevation in mean intrathoracic pressure may seriously compromise cardiac output,¹⁰ and, although the haemodynamic effects of PEEP in ARDS patients are allegedly small,^{4,6,7} there is conflicting evidence on this point.^{8,9,11} With the non-linear relationship between PaO_2 and O_2 content, diminution in cardiac output can be predicted to reduce net oxygen delivery to tissues in patients operating on the flat upper part of their haemoglobin dissociation curve, thus negating, at least in part, some of the potential benefits of PEEP therapy.

This study was undertaken to ascertain the effects, in eleven ARDS patients, of 5, 10, 15, and 20 cm H_2O PEEP in contrast to conventional intermittent positive-pressure breathing (IPPB) support with constant volume ventilation on PaO_2 , right atrial blood partial pressure of O_2 (PvO_2), partial pressure of arterial carbon dioxide (PaCO_2), pH, cardiac output (CO), $\dot{Q}_s/\dot{Q}_t$, dynamic compliance (C_{dyn}), dead space ventilation (V_D/V_T), O_2 delivery, and mean peripheral capillary oxygen tension ($\bar{\text{PcO}}_2$).

METHODS

Patients: Table I outlines relevant clinical data. None of the patients had a history of previous respiratory failure. All were haemodynamically stable and

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TABLE I
CLINICAL DATA. FIO_2 REFERS TO THE FRACTION OF INSPIRED OXYGEN EXPRESSED AS PERCENT

Patient	Sex	Age	Associated or antecedent problems	Before Study		Outcome
				FiO_2 %	PaO_2 mm Hg	
R.G.	M	27	Staph. aureus septicaemia, shock	100	117	Died
C.M.	F	60	Pneumococcal pneumonia (associated with influenza A2 pneumonitis)	100	90	Died
O.O.	M	57	Cirrhosis, Wernickes' encephalopathy, cerebral contusion, aspiration pneumonia	100	50	Died
D.G.	M	35	Multiple fractures, hypovolaemic shock	100	97	Recovered
J.G.	M	57	Peritonitis, Gram negative septicaemia, shock	100	60	Recovered
H.W.	M	44	Urinary tract infection, Gram negative septicaemia, shock	100	42	Died
W.W.	M	25	Multiple fractures, hypovolaemic shock	100	63	Died
F.L.	M	55	Gram negative septicaemia, shock, cerebral abscess	60	75	Died
I.M.	M	69	Gram negative septicaemia, shock	100	52	Died
E.S.	M	55	Aorto-femoral bypass, hypovolaemic shock	100	135	Recovered
F.S.	F	57	Polymyositis, multiorgan vasculitis	100	64	Died

FIGURE 1. Graduated plexiglass PEEP chamber connected downstream from the expiratory valve of the ventilator.

euhydrate when studied, and had alveolar-arterial O_2 gradients of at least 450 mm Hg with a measured Q_s/Q_t of 20 per cent or greater. Patients in clinical shock, those with panlobar emphysema or blebs, and patchy necrotizing pneumonias were excluded from this study. Chest X-rays showed the presence of bilateral homogeneous alveolar infiltrates. All patients were studied with cuffed 7 mm Portex tracheostomy tubes, aortic catheters (green Kifa placed by femoral Seldinger technique), and right atrial catheters in place. None had chest tubes. They were supported with controlled constant volume mechanical ventilation (either Bennett MA-1 or Ohio 560) at tidal volumes of 12–18 cc/Kg, and, with the exception of patient F.L., all were maintained on 100 per cent oxygen.

PEEP: End expiratory pressure was provided at the desired level by immersing a wide bore tube from the ventilator's expiratory port under a measured level of water (Figure 1). Expired gas was collected above the water from the enclosed PEEP chamber for determination of partial pressure of expired CO_2 ($PeCO_2$).

Protocol: After baseline determination of PaO_2 , PvO_2 , PaCO_2 , pH, CO, $\dot{Q}_s/\dot{Q}_t$, C_{dyn} , PcO_2 and O_2 delivery on IPPB, patients were placed successively on 5, 10, 15, and 20 cm H_2O PEEP for periods of 20 minutes, with return to IPPB for 15 minutes between each PEEP application. All measurements except V_D/V_T were made in 11 patients: dead space to tidal volume ratio was measured in five of these. In one patient (F.L.), who was on 60 per cent oxygen, shunt was determined with and without PEEP, when the inspired oxygen concentration was increased for 15 minutes to 100 per cent. The order of PEEP application was changed from 5, 10, 15, and 20, to 15, 5, 20, and 10 in three patients. Measurements on 20 cm H_2O PEEP were not made in two patients as their PaO_2 response to 5, 10, and 15 cm H_2O PEEP was poor and it was felt that study on a higher PEEP level could not be justified.

Measurements and Calculations: PaO_2 , PvO_2 , PaCO_2 and pH were measured with an Instrumentation Laboratories 113 system and haemoglobin saturations (SO_2) were determined from the standard Severinghaus nomogram.

Cardiac output was measured by dye dilution technique (indocyanine green, Waters XC 302 cuvette, X 302 densitometer and CO-4 computer) with injection into the right atrial catheter and withdrawal through the aortic Kifa catheter.

Right-to-left shunt was estimated from the standard shunt equation with the patient on 100 per cent oxygen.

$$\dot{Q}_s/\dot{Q}_t = C\dot{c}\text{O}_2 - C\text{aO}_2 / C\dot{c}\text{O}_2 - C\text{vO}_2$$

where: $C\dot{c}\text{O}_2$ = theoretical O_2 content of pulmonary end-capillary blood (ml O_2 /100 ml blood), the partial pressure of which was assumed to be the same as alveolar PO_2 (PAO_2). $C\dot{c}\text{O}_2$ was thus calculated from: $(S\dot{c}\text{O}_2/100 \times 1.34 \times \text{haemoglobin concentration}) + (\text{PAO}_2 \times 0.0031)$; where PAO_2 was equal to the barometric pressure (P_B)* - (PaCO_2 + partial pressure of water vapour) and haemoglobin concentration was measured by standard ferricyanide technique.

$C\text{aO}_2$ and $C\text{vO}_2$ = O_2 contents of arterial and right atrial blood respectively (ml O_2 /100 ml blood). These values were calculated from: $(S\text{aO}_2 \text{ or } S\text{vO}_2/100 \times 1.34 \times \text{haemoglobin concentration}) + (\text{PaO}_2 \text{ or } \text{PvO}_2 \times 0.0031)$.

O_2 delivery was calculated from $\text{CO} \times C\text{aO}_2$. Mean peripheral capillary oxygen tensions were estimated by using the Bohr integration procedure as modified by Riley and Cournand,¹² and adopted for use in the peripheral capillary beds by Flenley.¹³

Systemic arterial pressure (SAP) and right atrial pressure (RAP) were continuously recorded by Statham P37 transducers and Hewlett-Packard 8800 series preamplifiers and multichannel recorder.

C_{dyn} was derived from the expired tidal volume (measured by a Wright spirometer, the mean of three readings being used) divided by the peak airway pressure (measured by the ventilator's pressure gauges with calibration checked against a Statham PM-5 differential transducer and standard pressure system) minus the level of PEEP being applied.

PeCO_2 was measured along with simultaneously drawn arterial samples for PaCO_2 in five patients. Expired gas was bag-collected and analyzed for CO_2 by Godart capnograph calibrated full scale from 1-5 per cent. PeCO_2 was calculated

*Mean P_B in Edmonton (altitude 2,200 feet) is 700 mm Hg.

TABLE II
CHANGES IN P_aO_2 (mm Hg) ASSOCIATED WITH 0, 5, 10, 15, AND 20 cm H_2O PEEP.

Patient	IPPB	5 cm	IPPB	10 cm	IPPB	15 cm	IPPB	20 cm	IPPB
R.G.	117	122	94	153	186	245	90	294	123
G.M.	90	86	86	77	74	76	73	164	73
O.O.	50	53	47	59	51	71	52	106	57
D.G.	97	132	100	131	114	118	114	—	—
J.G.	62	76	64	58	66	83	80	117	128
H.W.	42	40	45	38	37	40	36	54	39
W.W.	63	127	89	191	55	297	103	297	102
F.L.	75	79	96	96	85	91	98	—	—
J.M.	52	72	65	78	63	72	60	136	59
E.S.	135	125	132	163	109	199	139	382	133
F.S.	64	137	61	215	129	345	54	160	59
Mean	77.0	95.4	81.5	114.5	88.0	148.8	81.7	190.0	85.9
S.D.	29.5	34.4	26.9	59.3	43.3	104.8	30.7	108.7	35.8
P_1	—	N.S.	N.S.	0.04195	N.S.	0.0382	N.S.	0.4941	N.S.
P_2		(0.05298)	0.04703		0.04469		0.00478		

from $(P_B - P_{H_2O}) \times \text{capnograph readout}/100$. V_D/V_T was then derived from the Böhr equation: $PaCO_2 - P_ECO_2/PaCO_2$.

RESULTS

PaO_2 (Table II*): Mean PaO_2 rose with increments in PEEP, the response curve being best described by a parabolic regression (Figure 2). The degree of change in PaO_2 from one PEEP level to another between patients was variable. PaO_2 fell slightly in three patients (G.M., H.W., E.S.) on 5 cm H_2O PEEP and two of these patients as well as one other (J.G.) demonstrated a fall in PaO_2 when 10 cm H_2O PEEP values were compared to inter-PEEP IPPB measurements. PaO_2 rose significantly above baseline IPPB levels in all but two patients (G.M. and H.W.) on 15 and 20 cm H_2O PEEP. Some patients required PEEP levels of 15 or 20 cm H_2O to improve their PaO_2 (e.g. G.M., J.G.) and one patient (F.S.) demonstrated a decline in P_aO_2 on 20 cm H_2O PEEP when compared to the 15 cm H_2O PEEP value. Another patient (H.W.) failed to show significant improvement in PaO_2 regardless of the level of PEEP applied.

$\dot{Q}_s/\dot{Q}_t$ (Table III). Patients in this study were very ill as evidenced by a mean baseline shunt of 45 per cent and the fact that only three of the eleven survived. Mean calculated shunt decreased significantly with successive PEEP increments (Figure 3), although, again, variation between individuals was evident. The fall in shunt with PEEP was both real and CO-related as judged from PvO_2 values.

CO (Table IV): With the exception of 5 cm H_2O PEEP, mean CO significantly declined with increasing PEEP levels (Figure 4). Variation occurred be-

*For all data tables, multicentered comparisons were done using the hotelling t^2 test (B.J. Winter, Statistical Principles in Experimental Design, McGraw Hill Book Company, New York, 1971). The computer program was taken from the DERS program Documentation 360/67. P_1 refers to comparisons between baseline IPPB values and subsequent measurements on PEEP; P_2 refers to comparisons between inter-PEEP IPPB values and the subsequent measurements on PEEP. NS = not significant ($P > 0.05$).

FIGURE 2. Graph demonstrating relationship between 0, 5, 10, 15, and 20 cm H₂O PEEP and associated mean levels of PaO₂.

TABLE III
CHANGES IN RIGHT-TO-LEFT SHUNT ($\dot{Q}_s/\dot{Q}_t$) EXPRESSED AS PERCENT ASSOCIATED
WITH 0, 5, 10, 15, AND 20 cm H₂O PEEP.

Patient	IPPB	5 cm	IPPB	10 cm	IPPB	15 cm	IPPB	20 cm	IPPB
R.G.	48.5	48.1	43.6	32.5	39.5	32.4	47.8	36.9	50.1
G.M.	38.4	42.9	42.5	39.5	39.7	36.5	39.0	22.0	39.0
O.O.	67.0	61.0	53.0	49.0	55.0	47.0	44.0	45.0	52.0
D.G.	21.0	19.0	23.0	16.0	20.0	17.0	18.0	—	—
J.G.	41.8	36.2	—	35.2	36.4	29.3	49.3	19.9	35.7
H.W.	62.4	66.0	64.1	68.1	69.7	66.0	64.7	56.4	72.8
W.W.	40.0	27.2	29.3	22.1	35.7	16.3	29.7	16.3	32.3
F.L.	47.2	43.2	37.7	36.6	33.7	32.0	40.5	—	—
J.M.	61.5	35.3	40.3	36.1	42.8	33.5	39.5	32.7	40.4
E.S.	34.4	30.0	32.3	30.5	36.8	26.8	34.7	14.1	33.7
F.S.	34.1	22.3	33.2	14.9	21.3	11.6	34.7	15.7	48.2
Mean	45.2	39.2	40.0	35.0	39.1	31.7	40.2	28.8	45.0
S.D.	14.0	15.0	11.9	15.0	14.0	15.2	12.0	14.9	12.7
P ₁	—	0.04617	N.S.	0.00461	N.S.	0.00122	N.S.	0.00038	N.S.
P ₂	—	—	0.02235	—	—	0.00052	—	0.00318	—

tween individual patients in that on 5 cm H₂O PEEP, when compared to base-line IPPB values, CO increased slightly in three patients (R.G., J.G., E.S.) and in one of these on 10 cm H₂O PEEP. Other patients also showed slight increases in CO on 10, 15, and 20 cm H₂O PEEP when compared to the preceding inter-PEEP level. Patient O.O., in whom an increase in CO was demonstrated on 20 cm H₂O PEEP, showed little change in CO at all PEEP levels.

FIGURE 3. Graph demonstrating relationship between 0, 5, 10, 15, and 20 cm H₂O PEEP and associated levels of right-to-left shunt.

TABLE IV
CHANGES IN CARDIAC OUTPUT (LITRES/MINUTE) ASSOCIATED WITH 0, 5, 10, 15, AND 20 cm H₂O PEEP.

Patient	IPPB	5 cm	IPPB	10 cm	IPPB	15 cm	IPPB	20 cm	IPPB
R.G.	12.30	13.60	14.70	11.10	12.60	10.70	15.30	10.60	12.80
G.M.	8.40	7.70	7.40	7.00	7.20	9.30	6.70	4.50	6.00
O.O.	6.10	5.25	6.00	5.65	5.10	5.10	5.10	5.80	4.90
D.G.	6.55	6.00	5.90	5.40	5.85	5.30	4.65	—	—
J.G.	5.11	5.66	5.52	6.42	7.02	4.81	5.30	3.70	5.60
H.W.	6.08	3.16	5.94	3.10	5.38	4.50	5.88	3.95	—
W.W.	4.60	4.40	4.53	4.58	5.75	4.23	5.32	4.21	5.10
F.L.	9.25	8.35	8.30	6.40	7.70	5.40	6.95	—	—
J.M.	6.60	6.20	5.75	5.20	5.80	3.90	4.85	3.15	5.00
E.S.	10.10	11.60	11.20	10.80	10.00	7.60	8.50	6.90	10.00
F.S.	3.90	3.60	3.70	3.40	3.80	3.05	4.10	2.65	4.60
Mean	7.18	6.87	7.18	6.28	6.93	5.81	6.60	5.05	6.75
St. Dev.	2.55	3.26	3.20	2.60	2.48	2.38	3.14	2.45	3.00
P ₁	—	N.S.	N.S.	0.04622	N.S.	0.00582	N.S.	0.0036	N.S.
P ₂	—	—	0.04858	—	0.01922	—	0.00813	—	—

O₂ Delivery (Table V): Mean O₂ delivery closely followed the effects of PEEP on mean CO, progressively declining with each PEEP increment (Figure 5). Statistically significant changes in mean O₂ delivery were not seen up to and including 10 cm H₂O PEEP. Variation between patients was again prominent. When compared to baseline IPPB values, six patients increased their O₂ delivery on 5 cm H₂O PEEP. Two of these patients had a slightly increased O₂ delivery

FIGURE 4. Graph demonstrating relationship between 0, 5, 10, 15, and 20 cm H₂O PEEP and associated mean levels of cardiac output.

TABLE V
CHANGES IN OXYGEN DELIVERY (ml O₂/MINUTE) ASSOCIATED WITH 0, 5, 10,
15, AND 20 cm H₂O PEEP.

Patient	IPPB	5 cm	IPPB	10 cm	IPPB	15 cm	IPPB	20 cm	IPPB
R.G.	1684	—	1982	1547	1769	1522	2061	1524	1755
G.M.	1120	1015	986	921	947	1198	863	629	772
O.O.	740	644	702	694	611	649	619	776	588
D.G.	1129	1144	1097	1030	1101	998	875	—	—
J.G.	656	745	—	815	912	647	705	509	769
H.W.	653	322	630	305	522	465	558	468	—
W.W.	635	669	677	709	797	672	800	669	768
F.L.	1198	1088	1108	855	1010	713	930	—	—
J.M.	820	903	766	727	772	539	662	486	679
E.S.	1325	1535	1485	1447	1308	1032	1131	983	1329
F.S.	672	676	631	656	712	601	671	501	774
Mean	976	874	1006	882	951	821	898	727	929
St. Dev.	354	339	440	355	352	325	419	343	400
P ₁	—	N.S.	N.S.	N.S.	N.S.	0.0115	N.S.	0.012	N.S.
P ₂			0.04821		0.02616		0.02445		

when compared to the preceding inter-PEEP IPPB level on 10 cm H₂O PEEP. Two other patients (G.M., O.O.) had enhanced O₂ delivery on 15 cm H₂O PEEP. In one of these latter patients (O.O.), O₂ delivery was maintained up to and including 20 cm H₂O PEEP.

Despite observed increases in P_aO₂ with PEEP increments, mean calculated peripheral-capillary PO₂ increased only slightly with PEEP and these changes

FIGURE 5. Graph demonstrating best fit parabolic, linear and exponential relationships between 0, 5, 10, 15, and 20 cm H₂O PEEP and associated mean levels of oxygen delivery. The parabolic equation shown provides the best R² value.

were not statistically significant (Figure 6). The mean capillary to mitochondrial gradient was therefore not significantly improved.

PaCO₂, pH, C_{dyn}, SAP, V_D/V_T: Mean baseline values for PaCO₂ (28 mm Hg) and pH (7.48) did not change significantly throughout the studies. Mean baseline C_{dyn} (27.8 ml/cm H₂O) reflected marked lung stiffness, but likewise failed to show statistically significant changes with PEEP. Mean SAP (90.1 mm Hg) was not altered significantly until 20 cm H₂O PEEP was applied when it declined to 83.8 mm Hg. In the five patients in which V_D/V_T was measured, no PEEP-associated change from the baseline value (0.58) could be demonstrated.

Incidental Observations and Complications: The first expired breath after cessation of PEEP was higher than the PEEP tidal volumes in all cases. Randomization of the order of PEEP application did not appear materially to affect any of the measured variables. No complications such as pneumothoraces occurred.

DISCUSSION

This study demonstrates that although PaO₂ can be improved in most ARDS patients with application of PEEP, the increase in PaO₂ is generally purchased at the price of diminishing cardiac output. Depending on the increase in O₂ content relative to decline of cardiac output, O₂ delivery to peripheral tissues may be slightly enhanced, remain unchanged from IPPB levels, or fall.

FIGURE 6. Graph demonstrating levels of P_{aO_2} , P_{cO_2} , and P_{vO_2} for 0, 5, 10, 15, and 20 cm H_2O PEEP. Although small increases in P_{cO_2} occurred on application of 15 and 20 cm H_2O PEEP, the difference between these levels and IPPB was not significant. P_{mO_2} refers to a speculative value for mitochondrial O_2 tension.¹³

Considerable interpatient variation in the effect of PEEP on P_{aO_2} was evident. A wide range in critical opening pressures for shunting gas exchange units likely accounts for this observation. The progressive increase in mean P_{aO_2} with diminution in shunt is probably due to recruitment of previously closed gas exchange units through elevation in functional residual capacity (FRC).^{7,9} It may also be due in part to a more beneficial distribution of blood flow or an absolute reduction in pulmonary perfusion. We have shown that in Xenon-133 ventilation/perfusion studies in the ARDS oleic acid model, the major functional basis for shunt reduction appears to be enhancement of ventilation to previously shunting regions, with elevation of FRC.

The decline in mean CO noted with PEEP might have been predicted from earlier experiments that showed impairment of CO with increases in mean intrathoracic pressure.¹⁰ Although it has been stated that any tendency toward a

PEEP-induced fall in CO is minimized in patients with stiff lungs,⁴ the haemodynamic effects of PEEP are undoubtedly the end result of a variety of factors. These include: transmitted airway pressure acting on either great veins¹⁴ or alveolar vessels,¹⁵ the presence of a normal or supernormal effective circulating fluid volume¹⁶ and intact cardiovascular reflex activity (Jones and King, *In Press*, *J. Appl. Physiol.*).

Because most of the patients studied were on or near the flat upper part of their haemoglobin dissociation curves, mean O₂ delivery closely followed changes in cardiac output produced by PEEP, progressively falling with each PEEP increment. However, it is apparent that for any individual patient O₂ delivery may be enhanced, remain unchanged, or decline with PEEP application, depending upon the relationship between increased O₂ content and decreased CO. Despite fall in CO and O₂ delivery at higher PEEP levels, pH remained unaltered. With the exception of one patient (F.S.) CO even with 20 cm H₂O PEEP, did not fall to the point where anaerobic tissue metabolism would be anticipated to occur. PEEP application is associated with a decline in peripheral O₂ consumption^{8,17} and, although the mechanism for this is obscure, this phenomenon may also account in part for the absence of anaerobic metabolism. $\bar{PcO}_2$ remained essentially unchanged despite significant elevations in PaO₂. Although no attempt was made to assess the effect of PEEP on individual tissue oxygenation, the whole body mean peripheral-capillary to mitochondrial oxygen gradient was unaffected by PEEP. This of course assumes no change in mitochondrial PO₂ during PEEP application. However, in light of previous reports showing that O₂ consumption decreases during PEEP, the lack of any significant change in $\bar{PcO}_2$ would likely produce an O₂ supply in excess of tissue demands.

V_D/V_T was unchanged with PEEP application in this and other studies conducted with patients;¹⁸ however, in dogs with the oleic acid induced ARDS, we have demonstrated an increase in V_D/V_T with PEEP, and at 15 and 20 cm H₂O levels, PaCO₂ may rise. These observations in animals are probably based on the increase in both anatomical and physiological dead space that occurs with high ventilation volumes which in turn result in diminished effective alveolar ventilation.¹⁹

Although it has been postulated that PEEP may "squeeze" water out of the lung and thus produce an improvement in compliance,⁸ mean C_{dyn} did not change with the application of PEEP in the eleven patients studied. The short term nature of this study may have mitigated against being able to demonstrate any significant change in this parameter.

Systematic arterial pressure was unaltered by PEEP application until 20 cm H₂O PEEP was applied suggesting that vascular reflexes were intact and that effective circulating fluid volume was adequate in most patients. More importantly, it also demonstrates that SAP represents an unreliable index of the effects of PEEP on the circulation and, in particular, cardiac output and O₂ delivery. The duration of increased end-expiratory pressure in this study was only 20 minutes for each application, a time period that may have been insufficient for 5 and 10 cm H₂O PEEP to complete their redistribution of ventilation and allow for cardiovascular adjustments. If this were the case, then prolonged application

of low PEEP levels may be expected to increase O_2 delivery to higher levels than was observed in this study. Also, O_2 delivery would be expected to increase more at low PEEP levels if the pre-PEEP PaO_2 was low enough to produce O_2 saturations on the steep part of the O_2 dissociation curve. Small increases in PaO_2 would then produce larger increases in O_2 content.

The precise role of PEEP in support of hypoxaemic patients remains to be clarified: It is possible that the ultimate place for PEEP in the therapeutic armamentarium will be in the sustenance of high lung volumes with preservation of pulmonary architectural integrity. Despite improvement in PaO_2 and the welcome possibility of being able to reduce inspired oxygen concentration, O_2 delivery to peripheral tissues is usually reduced when levels greater than 10 cm H_2O PEEP are applied. PEEP levels of 10 cm H_2O or less were capable in this study of increasing PaO_2 to levels which nearly saturated haemoglobin with oxygen. This increase in O_2 content, coupled with only minor decreases in cardiac output, make low PEEP levels relatively safe, but interpatient variation in response to all levels of PEEP makes careful monitoring mandatory.

SUMMARY

The effects of 5, 10, 15, and 20 cm H_2O positive end-expiratory pressure were studied in eleven patients with the adult respiratory distress syndrome (ARDS). Arterial PO_2 increased but cardiac output and O_2 -delivery decreased during 10, 15 and 20 cm H_2O PEEP. With 5 cm H_2O PEEP there were no significant changes in these variables. Calculated mean peripheral-capillary PO_2 remained essentially unaltered even though arterial PO_2 increased progressively with PEEP.

RÉSUMÉ

Nous avons étudié les effets d'une pression positive de fin d'expiration de 5, 10, 15 et 20 cm H_2O chez onze malades souffrant du syndrome de détresse respiratoire de l'adulte (ARDS).

La PO_2 artérielle a augmenté mais le débit cardiaque et la quantité d'oxygène disponible ont diminué lors de l'usage de 10, 15 et 20 cm d' H_2O de PEEP. Lors de l'usage de 5 cm H_2O de PEEP, nous n'avons observé aucun changement de ces facteurs. La moyenne calculée de la PO_2 capillaire périphérique est demeurée inchangée même si la PO_2 a augmenté progressivement avec la PEEP.

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