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BRIEF REPORT

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Comparative evaluation of the haemodynamic effects of continuous negative external pressure (CNEP) and positive end-expiratory pressure (PEEP) in mechanically ventilated trauma patients

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Abstract *Objective:* To compare the haemodynamic effects of identical values of continuous negative external pressure (CNEP) and positive end-expiratory pressure (PEEP) in a group of mechanically ventilated patients.
Setting: General ICU, Vicenza Hospital, Italy.
Patients: 15 consecutive patients, admitted after road accident trauma.
Methods: We compared the haemodynamic effects of ZEEP, 10 cmH_2O of PEEP, and 10 cmH_2O CNEP, applied in random order, in 15 head trauma patients under going controlled mechanical ventilation; 9 had associated thoracic trauma, while 6 did not have lung involvement. CNEP was obtained with a "poncho".
Results: We observed a significant increase in CI during CNEP, compared with both ZEEP and PEEP 10 cmH_2O. Accordingly the oxygen delivery index significantly increased during CNEP, compared with PEEP 10 cmH_2O. Conversely, $\dot{Q}_s/\dot{Q}_t$ decreased with CNEP, if compared with PEEP, both in patients with and without lung damage.
Conclusion: CNEP can significantly increase CI in mechanically ventilated patients in patients with and without associated lung damage.

Key words Mechanical ventilation · PEEP · CNEP · Poncho · Trauma

Introduction

A reduction in lung volume represents the main anatomical and functional characteristic during acute respiratory failure of parenchymal origin. In this condition, arterial oxygen saturation can be increased by augmenting mean airway pressure (by PEEP or CPAP administration) due to a consequent increase of functional residual capacity (FRC) [1]. Conversely, venous return to the heart and cardiac output cannot withstand an increase of airways pressure particularly in hypovolemic patients [2].

In order to increase arterial oxygen pressure (PaO_2) with less detrimental haemodynamic effects, many authors have proposed ventilatory approaches, such as "best" PEEP, moderating the negative consequences on the circulation of continuous positive airways pressure [3]. Considering that FRC is a function of a transpulmonary gradient that can be increased either by higher airways pressure or by decreasing pleural pressure, it is consequent that a negative perithoracic (and abdominal) pressure can increase FRC by decreasing pleural pressure [4, 5]. For many years it has been known that continuous negative external pressure (CNEP) affecting only the thoracoabdominal area and not the whole body (as in tank-type respirators) causes a better venous return to the heart by increasing the peripheral-pleural pressure gradient [5–7]. The introduction into clinical use of simple devices called "ponchos", to provide CNEP, induced us to compare the effects of identical absolute values of PEEP and CNEP on haemodynamic and oxygenation variables in patients mechanically ventilated in control mode.

Patients and methods

A group of 15 consecutive patients admitted to ICU after a road accident were included in the study. They had all suffered mild head trauma that caused a confusional state with psychomotor agitation requiring pharmacological sedation and mechanical ventilation. The Glasgow coma score was always >6 and there was no sign of intracranial hypertension. All patients were mechanically ventilated [Servo 900C, Siemens, Elena, Sweden] in control mode with V_T 10 ml/kg, respiratory rate to maintain $PaCO_2$ between 28 and 32 mmHg, I:E ratio 1:2 and FIO_2 ranging between 0.3 and 0.5. Approval from our institutional ethical committee and informed consent by the patient's legal guardians were obtained. The patients were divided into two groups of 9 and 6, according to the presence of pulmonary parenchymal damage due to lung contusion.

Pulmonary damage was determined by the presence of compatible X-rays, a pulmonary shunt >20% and a static compliance for baseline inflation volume at ZEEP below 0.04 l/cmH_2O (mean of the group 0.035±0.004 l/cmH_2O). In no cases were rib fractures or important damage involving other organs associated.

All patients were sedated with fentanyl (0.1 mg/h) and flunitrazepam (0.5 mg/h).

Controlled mechanical ventilation with the 3 modalities (ZEEP, PEEP 10 cmH_2O, and CNEP 10 cmH_2O) was used in all patients in random sequence; in more detail, CNEP was applied to the thoracic wall by a "poncho" inside which a constant negative pressure was obtained using a calibrated aspirator (Empty Flow System – mod RAP 027, Bologna, Italy) working with the continuous negative mode.

PEEP and CNEP values were monitored by means of a pressure transducer connected by air filled lines to the airways of the patient (at the Y piece) and inside the poncho, at 20 cmH_2O from the aspiration port respectively (Abbott pressure transducers). When using the poncho air leaks were absent or negligible. Pleural pressure was measured by means of an esophageal balloon catheter; the correct positioning was checked with an occlusion test.

A Swan-Ganz catheter was inserted into the pulmonary artery via the internal jugular vein to evaluate hemodynamics; blood pressure was monitored with radial arterial catheterization (19 g, Abbott). All haemodynamic measurements were recorded with a Sirecust Multitrace Polygraph. ZEEP, PEEP and CNEP were applied for 20 min before collecting the data; cardiac output measurements were performed in triplicate. During the whole study no therapeutic modification was performed: in more detail no vaso-active drug or vascular fluid loading was administered and ventilator settings, including FIO_2, were not modified. All data, reported as mean±SD, were compared for statistical significance with the ANOVA two way test; if significant, the values obtained with CNEP and PEEP were compared with ZEEP and those obtained with CNEP were compared with PEEP, using the paired *t* test. A *p* value <0.05 was considered significant.

Results

All the results of our study are displayed in Table 1. The application of PEEP (10 cmH_2O) and CNEP (10 cmH_2O) to patients ventilated in control mode with FIO_2, V_T and I:E ratio constant produced the following functional modifications: the administration of PEEP induced a significant PaO_2 increase in all patients ($p<0.01$ PEEP versus ZEEP) and accordingly, right to left venous admixture ($\dot{Q}_s/\dot{Q}_t$) significantly decreased during PEEP application ($p<0.01$) in patients with lung damage

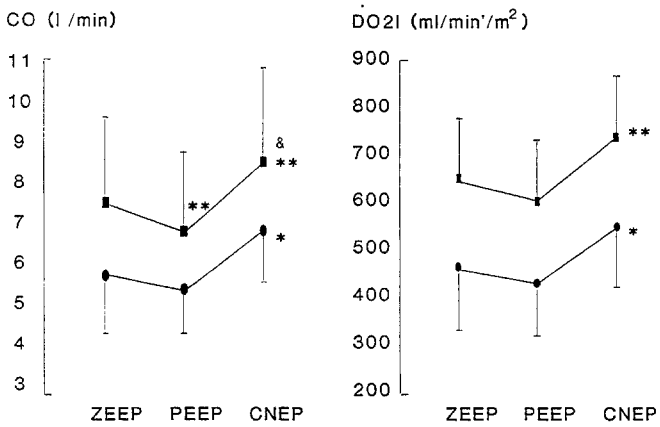

Fig. 1 Mean values and standard deviation. *ZEEP*, mechanical ventilation with PEEP of 0; *PEEP*, mechanical ventilation with PEEP of 10 cmH_2O; *CNEP*, continuous negative extrathoracic pressure of 10 cmH_2O; *Squares* = patients with pulmonary injury; *circles* = patients without pulmonary injury. *$p<0.05$; **$p<0.01$; & $p<0.001$ versus PEEP

(group A); cardiac output (CO) increased significantly during CNEP application, both in patients with and without lung damage ($p<0.01$ in group A and $p<0.05$ in group B for CNEP versus ZEEP, $p<0.001$ in group A and $p<0.05$ in group B for CNEP versus PEEP) and the oxygen delivery index (DO_2I) increased significantly during CNEP application both in group A ($p<0.01$) and group B patients ($p<0.05$), comparing CNEP with PEEP; a significant decrease in stroke volume (SV) was observed with PEEP in group A patients ($p<0.01$ PEEP versus ZEEP) while SV significantly increased both versus ZEEP and PEEP in group B and versus PEEP in group A, during CNEP application.

Esophageal pressure was not modified during the study in group A or group B patients; however it showed dramatic differences both at ZEEP and during PEEP and CNEP application when comparing patients with and without lung injury. No clinical or technical problems were observed during the study.

Discussion

CNEP application caused in our study, a smaller PaO_2 increase, in comparison with PEEP; this may be explained by a reduced increase in FRC due to a lower increase in transpulmonary gradient when the same absolute values of CNEP and PEEP are compared. This difference is probably related to an asymmetric effect of CNEP on thoracic and abdominal areas, and possibly, to the variable impedence of the chest wall particularly in patients with associated thoracic trauma. As a consequence of the different pressure transmission to the pleural space of patients with and without lung injury,

Table 1 Haemodynamic and oxygen transport patterns of patients with and without lung injury during ZEEP, PEEP and CNEP ventilation (mean ± SD). (Definitions and abbreviations: *SV* = stroke volume, *HR* = heart rate, *CI* = cardiac index, DO_2I = oxygen delivery index, SVO_2 = venous saturation of oxygen, Q_s/Q_t = right-to-left venous admixture, VO_2I = oxygen consumption index, *CVP* = central venous pressure, *MPAP* = mean pulmonary artery pressure, *PAOP* = pulmonary artery occlusion pressure, *SVRI* = systemic vascular resistance index, *PVRI* = pulmonary vascular resistance index, *Pes* = esophageal pressure)

	ZEEP	PEEP	CNEP
HR (mmHg)			
without lung injury	94.5 sd 21.8	96.5 sd 22.5	92.3 sd 23.9
with lung injury	86.3 sd 22	87.8 sd 24.1	92.5 sd 19.5
MAP (mmHg)			
without lung injury	92 sd 9.7	95.9 sd 13.9	94.6 sd 10.3
with lung injury	90 sd 13.9	88 sd 15.2	103 sd 21.5
Pes (mmHg)			
without lung injury	2.8 sd 0.4	4 sd 1.9	0.9 sd 1.9
with lung injury	10 sd 2.1	11 sd 1.8	9.1 sd 2.3
CVP (mmHg)			
without lung injury	8.5 sd 3.3	10.7 sd 1.9	9.2 sd 4.9
with lung injury	11 sd 4.3	12 sd 3.8	11 sd 3.6
MPAP (mmHg)			
without lung injury	20 sd 8.7	25 sd 6.9	22 sd 8
with lung injury	25 sd 5.9	26 sd 5.6	27 sd 6
PAOP (mmHg)			
without lung injury	11 sd 5.1	15 sd 4.4**	11 sd 5.6
with lung injury	14 sd 4.5	15 sd 4.2	14 sd 5.1
CO (l/min)			
without lung injury	5.7 sd 1.4	5.3 sd 1.1	6.8 sd 1.2*[a]
with lung injury	7.5 sd 2.2	6.8 sd 1.9**	8.5 sd 2.4**[a]
SV (ml)			
without lung injury	63 sd 19	58 sd 16	76 sd 26*[a]
with lung injury	91 sd 29	81 sd 26**	95 sd 31[b]
SVRI (dyne·s/cm^5·m^2)			
without lung injury	888 sd 354	1008 sd 565	775 sd 400
with lung injury	1057 sd 519	1132 sd 597	1028 sd 254
PVRI (dyne·s/cm^5·m^2)			
without lung injury	96 sd 54	92 sd 43	92 sd 43
with lung injury	137 sd 80	152 sd 79	141 sd 81
DO_2I (ml/min/m^2)			
without lung injury	456 sd 133	426 sd 107	546 sd 125*[a]
with lung injury	642 sd 137	602 sd 128	739 sd 131**
VO_2I (ml/min/m^2)			
without lung injury	129 sd 34	121 sd 39	137 sd 31
with lung injury	144 sd 34	147 sd 38	160 sd 37
SvO_2 (%)			
without lung injury	69 sd 7.3	68 sd 13	72 sd 8.9
with lung injury	72 sd 5.8	72 sd 4.7	74 sd 4.7
PaO_2 (mmHg)			
without lung injury	121 sd 16	153 sd 27**	132 sd 23
with lung injury	75 sd 12	97 sd 28**	88 sd 27
Q_s/Q_t (%)			
without lung injury	10.5 sd 2.1	8.6 sd 2.2	11.7 sd 2.8[a]
with lung injury	30.5 sd 9.9	21.2 sd 10.3**	27.3 sd 11.7[b]

** $p<0.01$; * $p<0.05$; [a] $p<0.01$ versus PEEP; [b] $p<0.05$ versus PEEP

transmural CVP values were dramatically different, and this can be a major explanation for the observed haemodynamic effects, for instance through the Starling mechanism. The main result of our study was that during CNEP cardiac output and DO_2I increased compared with mechanical ventilation with PEEP levels of the same absolute value. This was due to an increase in the stroke volume, observed both in patients without pulmonary damage and in patients with lung damage and a $\dot{Q}_s/\dot{Q}_t$ increase consequent to lung contusion.

The results are consistent with the better haemodynamic tolerance reported by Skaburskis et al. [9] in an experimental study comparing the haemodynamic effects of CNEP (obtained with a "poncho") and CPPV, and could probably be explained as the result of an increased peripheral-pleural pressure gradient; in other words, CNEP

increases the venous return from the periphery, increasing the preload and producing (in patients free from primary heart damage and in a normal volemic equilibrium) an increase in cardiac output. The increase of DO_2I observed during CNEP is an obvious direct consequence of the increase in cardiac output that can also explain the $\dot{Q}_s/\dot{Q}_t$ modifications observed during the study in patients with lung damage and anatomic shunt increase. It is therefore probable that CNEP behaves, in terms of cardiorespiratory consequences, in a very different way from PEEP; the latter decreases venous return and cardiac filling both if applied during controlled mechanical ventilation [10] and during spontaneous breathing [11].

The central role of preload modification is underlined by the fact that volemic expansion can reverse the negative effects of PEEP, restoring normal values of cardiac output [12]; however volemic expansion increases the amount of extravascular lung water in patients with parenchymal damage and increased capillary permeability, with potential further alterations in PaO_2 values. In this situation CNEP can be proposed as a different approach to minimize the haemodynamic consequences of alveolar recruitment, although a smaller PaO_2 increase must be expected in comparison with the use of the same absolute values of PEEP.

In this sense CNEP during mechanical ventilation can be considered as an alternative to PEEP in patients that require an increase in functional residual capacity and lung volume and an optimal or supranormal value of DO_2. However, more studies are needed to confirm this view in terms of long term clinical feasibility. Finally, it should be considered that although the negative effects of PEEP can be minimized by vasoactive administration, without alterations of fluid balance, a technique able to obtain the same results without the side effects of drug administration seems to be an interesting and more physiologic alternative.

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