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Lung recruitment maneuver in patients with cerebral injury: effects on intracranial pressure and cerebral metabolism

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Abstract *Objective:* To investigate the effects of a lung recruitment maneuver on intracranial pressure (ICP) and cerebral metabolism in patients with acute cerebral injury and respiratory failure. *Design:* Prospective investigation. *Setting:* Ten-bed intensive care unit of a university hospital. *Patients:* Eleven patients with acute traumatic or non-traumatic cerebral lesions, who were on mechanical ventilation with acute lung injury. *Interventions:* Hemodynamics, ICP, cerebral perfusion pressure (CPP), jugular venous oxygen saturation (SJO_2), and arterial minus jugular venous lactate content difference (AJDL) were measured before, during and after a volume recruitment maneuver (VRM), which included a 30-s progressive increase in peak pressure up to 60 cmH_2O and a sustained pressure at the same level for the next 30 s. *Results:* At the end of VRM, ICP was elevated (16±5 mmHg vs 13±5 mmHg before VRM, $P<0.05$) and mean arterial pressure was reduced (75±10 vs 86±9 mmHg, $P<0.01$), which resulted in a decrease of CPP (60±10 vs 72±8 mmHg, $P<0.01$). SJO_2 deteriorated at the end of the procedure (59±7 vs 69±6%, $P<0.05$), AJDL was not altered. In the following period all parameters returned to normal values. An improvement in arterial oxygenation was observed at the end, but not in the period after the maneuver. *Conclusions:* Our VRM reduced cerebral hemodynamics and metabolism. We conclude that our VRM with high peak pressure effects only a marginal improvement in oxygenation but causes deterioration of cerebral hemodynamics. We therefore cannot recommend this technique for the ventilatory management of brain-injured patients.

Keywords Volume recruitment · Brain injury · Jugular venous oxygen saturation

Introduction

Respiratory failure due to chest injury, aspiration of oropharyngeal contents, nosocomial pneumonia or a "neurogenic" pulmonary edema is often presented in patients with acute traumatic or nontraumatic cerebral lesions [1]. Consequently, therapeutic strategies in intensive care units have to combine treatment and prevention of cerebral ischemia as well as management of acute lung injury. In patients with acute brain lesions at risk for cerebral ischemia, monitoring of intracranial pressure and of global cerebral hemodynamics and metabolism has become a feasible tool for clinical management [2]. Current methods of monitoring include continuous measurement of intracranial pressure (ICP) and the technique of jugular venous oxygen saturation (SJO_2) measurement, which yields information about brain oxygenation or the calculation of the arterial minus jugular venous lactate difference (AJDL [3]). In general, therapeutic management consists of deep sedation, adequate artificial ventilation, and maintenance of a high cerebral perfusion pressure (CPP [4]).

Treatment of acute lung injury (ALI) is managed by artificial ventilation along with a suggested "lung protective strategy" [5], and by other supportive measures, e.g., systematic changes of the patient's body position [6,7]. In patients with acute brain injury, monitoring of carbon dioxide partial pressure in the arterial blood ($PaCO_2$) is of special interest, since hypercarbia may result in further increase of ICP, while hypocarbia may affect cerebral oxygenation [8]. Furthermore, application of a high positive end-expiratory pressure (PEEP) is limited in neurosurgical intensive care patients, since high PEEP levels may induce an increase in ICP [9].

For therapeutic strategies in patients with ALI, ventilated at low PEEP-levels, a volume recruitment maneuver (VRM [10]) is advocated as an alternative measure for optimizing alveolar recruitment, but an explicit recommendation for this technique has not been given up to now. On the other hand, the application of VRM might be combined with a deterioration of the hemodynamic system by reducing intrathoracic blood volume as well as cardiac output and mean arterial pressure and – consecutively – cerebral hemodynamics. We therefore hypothesize that the application of VRM may improve oxygenation but may affect ICP, CPP, and, in consequence, cerebral oxygenation. The present prospective study was designed to investigate the possible effects of VRM on systemic as well as cerebral hemodynamics and cerebral metabolism in patients with acute cerebral and pulmonary injury.

Patients and methods

After approval by the local ethics committee and informed consent by next of kin, 11 patients were included in this prospective study within 3 days after admission at the intensive care unit. The patients presented with acute cerebral lesions (Glasgow Coma Scale 4–12) of traumatic or non-traumatic origin (Table 1) as well as ALI due to aspiration or nosocomial pneumonia and radiologic signs of atelectasis. ALI was defined following a recently published consensus [11]: an acute onset with a decrease in arterial oxygenation (PaO_2/FiO_2 <300 mmHg) and the presence of bilateral infiltrates on chest roentgenogram with a negative history for cardiac disease.

All patients received intracranial pressure measurement, central venous catheter, and arterial line and a retrograde cannulation of an internal jugular vein for placement of a jugular venous bulb catheter to monitor jugular bulb oxygen saturation (SJO_2), reflecting the balance between supply and consumption of oxygen in the brain [12]. The cannulation was performed on the side which anatomically representing the largest jugular foramen in the computed tomography [13]. Additionally, the calculation of AJDL was used to gain further information on the presence of cerebral ischemia [14].

All patients were deeply sedated by continuous infusion of opioids and midazolam and were not responsive to pain. Mean arterial pressure (MAP) was adjusted by volume replacement to maintain adequate cerebral perfusion pressure (CPP ≥65 mmHg) and a sufficient cerebral oxygenation (SJO_2 >55%). Vasopressors (norepinephrine) were only used after volume expansion (central venous pressure ≥6 mmHg).

Patients were mechanically ventilated in a pressure-controlled mode (inspiratory/expiratory ratio 1:1, 840 Ventilator System, Nellcor Puritan Bennett, Hennef, Germany). Moderate PEEP ranged from 6 cmH_2O to 12 cmH_2O and maximum inspiratory pressure (p_{max}) was set between 25 cmH_2O and 30 cmH_2O. Intrinsic PEEP was not measured. First of all, the patients were normoventilated ($PaCO_2$ between 36 mmHg and 42 mmHg), hyperventilation therapy ($PaCO_2$ <36 mmHg) was reserved for patients

Table 1 Characteristics of patients

Patients	Age	Diagnosis, type of ALI	Initial GCS	PaO_2/FiO_2 (mmHg)	CVP (mmHg)
1	60	Intracerebral hemorrhage (nontraumatic), pneumonia	8	177	7
2	58	Head injury, "neurogenic" edema	6	266	12
3	45	Infarction of anterior cerebral artery pneumonia	12	225	8
4	67	Intracerebral hemorrhage (nontraumatic) atelectasis	9	276	10
5	70	Thrombosis vena basilaris, atelectasis	8	216	11
6	42	Head injury, lung contusion	8	277	6
7	33	Head injury, "neurogenic" edema	8	261	6
8	22	Head injury, pneumonia	8	184	7
9	53	Subarachnoid hemorrhage (WFNS Grade IV), pneumonia	7	285	10
10	45	Subarachnoid hemorrhage (WFNS Grade IV), atelectasis	4	240	8
11	83	Head injury, pneumonia	12	280	11
mean±SD	54±16		8±2	244±39	8.7±2

Table 2 Respiratory parameters

Patients	PEEP (cmH_2O)	Tidal volume (ml)	Respiratory rate (bpm)
1	6	480	14
2	10	520	13
3	8	430	16
4	11	450	14
5	9	510	13
6	9	560	15
7	12	490	17
8	10	500	14
9	7	480	15
10	8	530	17
11	7	500	16

showing signs of cerebral hyperemia (SJO_2 >75%). Those patients received hyperventilation therapy.

Supportive therapy was managed according to our standard intensive care protocol [14], which is aimed at preventing cerebral ischemia. Antimicrobial therapy was initiated immediately after obtaining positive test results of the tracheal aspirate.

VRM was performed by periodic increases of p_{max} from the baseline level to a level of 60 cmH_2O. This technique modified the description of Lachmann et al. [10], who proposed a successive increment of peak pressure in steps of 3–5 cmH_20 until a p_{max} between 45 cmH_20 and 60 cmH_20 is reached. Our increment was performed within 30 s and the upper level of p_{max} was held for 30 s. The time span of the complete VRM was 1 min. After this procedure, the ventilatory regimen was set back to the previous condition. We decided to use the highest level of peak pressure of Lachmann's recommendations (60 cmH_2O) to reach a maximal open lung effect, since we had set a relatively low PEEP in our patients which we did not want to modify after the procedure. For safety reasons we decided that the maneuver had to be stopped if ICP levels >25 mmHg were reached or CPP decreased below a critical value of 50 mmHg. In one patient VRM had to be stopped after 50 s due to hemodynamic instability.

Arterial blood gases, heart rate (HR), MAP, ICP, CPP, SJO_2, and AJDL were measured as follows: 1) immediately before VRM; 2) at the end of VRM; 3) 10 min after VRM; and 4) 30 min after VRM.

Statistical analysis was performed using the SPSS software package. Data shown are mean values±standard deviation (SD). Data were analyzed by the Wilcoxon matched-pairs signed-ranks test and for linear regression analysis by Pearson's correlation coefficient. Significance was accepted at $P<0.05$.

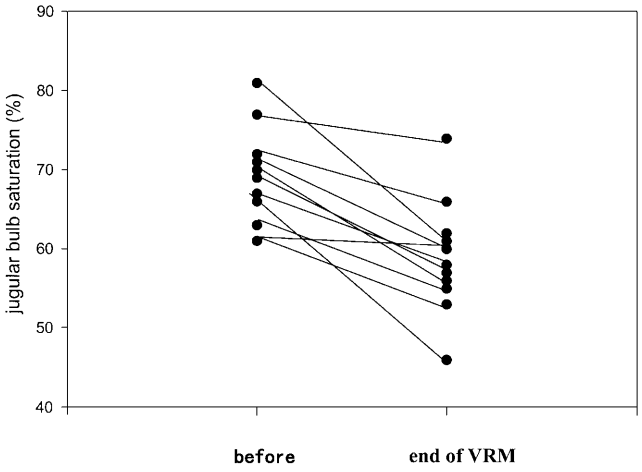

Fig. 1 Individual values of SJO_2 before and at the end of VRM

Results

Clinical characteristics of the patients are given in Table 1. All patients suffered from moderate to severe cerebral lesions [mean Glasgow Coma Scale (GCS) = 8±2] and reduced pulmonary gas exchange (PaO_2/FiO_2-ratio =244±39 mmHg). The respiratory parameters are shown in Table 2.

VRM caused a significant decrease in MAP and an increase in ICP, both resulting in a critical reduction of CPP <65 mmHg (Table 3). Ten minutes after the end of VRM, all hemodynamic parameters were normalized. At the end of VRM, SJO_2 declined significantly, indicating the beginning of cerebral ischemia (≤ 55%). We found no differences between trauma and nontrauma patients. The patients with cerebral hyperemia, who were already hyperventilated, did not respond differently. After the end of VRM, SJO_2-values returned to the previous condition before VRM. AJDL-values before VRM showed no signs of cerebral ischemia (critical value: >0,.7 mmol/l [15]), but AJDL tended to be increased 10 min after VRM. The individual SJO_2-values before and at the end of VRM are plotted in Fig. 1 and the values for AJDL are shown in Fig. 2. Furthermore, we found no significant relationship between the values of the Glasgow Coma Scale and changes in SJO_2.

Table 3 Changes in hemodynamic and cerebral parameters

	Before VRM	At the end of VRM	10 min after VRM	30 min after VRM
Mean arterial pressure (mmHg)	86±9	75±10**	84±10	83±7
Intracranial pressure (mmHg)	13±5	16±5*	13±4	13±4
Cerebral perfusion pressure (mmHg)	72±8	60±10**	70±11	70±8
Jugular bulb oxygen saturation (%)	69±6	59±7*	67±6	67±6
Arterial minus jugular venous lactate content difference (mmol/l)	0.06±0,07	0.07±0,07	0.09±0,07	0.06±0,05

* $P<0.05$; ** $P<0.01$ in comparison with "before VRM"

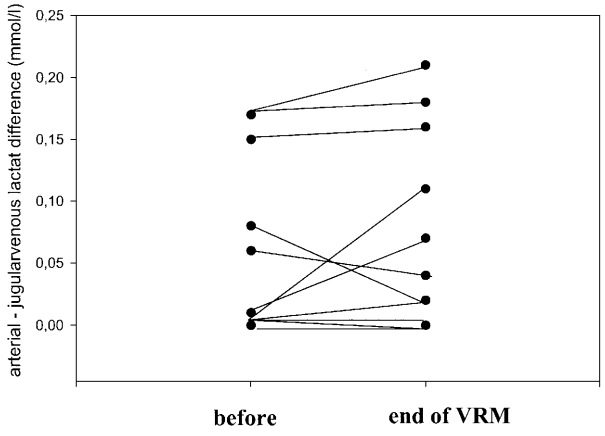

Fig. 2 Individual values of AJDL before and at the end of VRM

Arterial oxygenation improved significantly at the end of VRM (Table 4). Arterial carbon dioxide values were reduced markedly at the end of VRM and arterial pH increased significantly. In the following period, the beneficial effect of the maneuver disappeared and the pulmonary gas exchange remained unchanged.

Discussion

Patients with acute head injury or subarachnoid hemorrhage often present with ALI, with an incidence range from 20% (post-resuscitative GCS <8 [16]) to 50% ("neurogenic" pulmonary edema in fatal head injuries [17]). The main strategy of intensive care management in this particular population of patients is to avoid cranial hypertension or cerebral ischemia as well as to improve pulmonary function and gas exchange. The ventilatory management in these patients is limited, since established supportive strategies, such as "permissive hypercarbia" or systematic changes in the patient's body position have some limitations. Application of high PEEP-levels is matter for controversy regarding neurosurgical patients, since it has been demonstrated that high PEEP levels (>15 cmH_2O) may induce or aggravate intracranial hypertension [9].

The effect of other supportive strategies on cerebral injury, such as ventilation in the prone position [6] is still not exactly understood. Tillett et al. investigated the effect of continuous rotational therapy on intracranial pressure in severely brain-injured patients [18]. They found no deleterious effect of this treatment on intracranial pressure and recommended the use of a rotating bed.

The concept of an open-lung approach in patients with ALI was advocated by Lachmann et al. [10]. The beneficial effect of deep breaths or sighs on lung function has been known for more than 30 years. Recently, Lachmann proposed a variation of an open-lung approach by the procedure of a successive increase of p_{max} until a level between 45 cmH_2O and 60 cmH_2O is reached ("open up the lung"). In a second step, p_{max} should be reduced carefully before the critical level is reached at which the lung starts to collapse again ("keep the lung open"). In recent years, several studies tested some modifications of the open-lung approach in critically ill patients with lung failure. Lapinsky et al. found a significant improvement in oxygenation in adults with respiratory failure using a sustained inflation using a pressure of 30 cmH_2O to 45 cmH_2O for a short time-period [19]. Foti et al. investigated whether VRMs could improve alveolar recruitment in ARDS patients [20] and they found that VRMs (periodic increases of PEEP) improved oxygenation and alveolar recruitment in these patients ventilated at relatively low PEEP levels, but VRM was less effective than using continuous high PEEP ($\geq$ 15 cmH_2O). In the present scientific literature there are numerous variations on the way lung recruitment techniques are implemented in clinical settings and which produce different results. In other words, we are far from being able to give clear and precise recommendations or guidelines for a special volume recruitment procedure in specific conditions of respiratory failure.

Since high PEEP-levels may be harmful in patients with acute brain lesions, we wanted to find out whether a single VRM might improve lung function in head-injured patients ventilated with moderate PEEP levels. On the other hand, we were interested in discovering any possible negative effects of VRM on cerebral hemodynamics and brain metabolism.

In our study, using the technique of jugular bulb catheterization to estimate brain oxygenation in patients at risk for cerebral ischemia, we found a significant deterioration of jugular venous oxygen saturation during our VRM. Simultaneously we observed a depression of mean arterial pressure and an increase in intracranial pressure, both resulting in a critical decrease of the cere-

Table 4 Changes in blood gas analysis parameters

	Before VRM	At the end of VRM	10 min after VRM	30 min after VRM
Arterial pH	7.44±0.06	7.51±0.05**	7.47±0.05	7.46±0.04
PaO_2 (mmHg)	103±19	139±39**	104±11	110±14
SaO_2 (%)	98.1±2.2	99.2±0.3**	98.7±0.4	98.8±0.6
$PaCO_2$ (mmHg)	41±9	34±6**	38±7*	39±7

* P<0.05; ** P<0.01 in comparison with "before VRM"

bral perfusion pressure. All parameters returned to the previous values within a short period after VRM.

On the other hand, the beneficial effect of our single VRM on pulmonary gas exchange was poor: the improvement in oxygenation at the end of VRM disappeared immediately. This finding is in accordance to Lachmann's hypothesis; after the maneuver PEEP has to be adjusted to a (higher) level to prevent lung collapse. To keep the lung open, elevated levels of PEEP might be necessary, which we did not use in our patients, since they are contraindicated in patients with acute cerebral lesions.

We conclude that our single volume recruitment maneuver with a high peak pressure (60 cmH_2O) in patients with acute brain injury and lung failure effects only a marginal improvement in oxygenation but may lead to deterioration of cerebral hemodynamics and oxygenation. To our knowledge there are no data regarding the effects of lung recruitment maneuvers in brain-injured patients and – as far as we know – there exist no recommendations or guidelines on the use of this maneuver in these patients. In the light of our data, we do not recommend the technique that we used for the ventilatory management of this population of patients. However, these data cannot be considered so conclusive as to abandon this strategy. Caution is needed and further studies are necessary which should aim to elucidate the "critical" level of VRM, PEEP, and the effects of other strategies for therapy of lung failure in patients with brain injury.

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