Mechanical ventilation in ARDS

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TIDAL VOLUME REDUCTION IN PATIENTS WITH ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

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Mechanical ventilation induced pulmonary lesions is a subject of great concern in ARDS. Experimental animal studies have shown that alveolar damage resulted from mechanical ventilation with high tidal volumes above total lung capacity (TLC). It is not known however whether classical ventilation with PEEP and V_T of 10 ml/kg usually reaches TLC in patients with ARDS. Analysis of the total respiratory pressure volume (PV) curve in ARDS can reveal an upper inflection point (UIP) corresponding to the flat part of the curve and a decrease in compliance at high lung volume. It can be assumed that tidal ventilation nearing this point might be deleterious in term of barotrauma. During 6 months, we propectively studied 14 patients with ARDS (age 42 ± 17, PO_2/FiO_2 99.7 ± 49.5). They were ventilated with V_T = 10 ml/kg ; I/E = ½ ; ventilatory rate = 18 ; $FiO_2 \geq 0.7$. PV curves were performed using a 2 liter syring and with a Servo ventilator 900C by intermittently changing the inspiratory time. When an UIP was evidenced, the corresponding tidal volume above PEEP was calculated. If necessary, i.e., if end inspiration was above the UIP, V_T was lowered and changes in $PaCO_2$ studied (group I). When the preset V_T was below this point it was kept unchanged (groupe II).

Results : PEEP was chosen as the lower inflection point (10.8 ± 3.0 cmH_2O) ; 12/14 (86 %) patients had an UIP. 5 entered group II and 7 entered group I (50 %). Mean compliance was 39 ± 9 ml/cmH_2O for group I and 42 ± 5 ml/cmH_2O for group II (ns). The mean fall imposed in V_T in group I was 2.03 ± 0.6 ml/kg. The corresponding increase in PCO_2 was from 42.1 ± 3.3 to 74.2 ± 2.8 mmHg without any visible side effect. For group I patients, the peak static pressure with limited V_T was reduced from 31.4 ± 3.2 to 25.4 ± 2.6 cmH_2O ($p < 0.05$). Lastly, if a tidal volume of 15 ml/kg had been initially chosen, limitation of V_T would have been required in 67 % of the patients.
In conclusion, in half of patients with ARDS, usual ventilation takes place near total lung capacity. Reduction of tidal volume and permissive hypercapnia are therefore recommended.

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A NEW APPROACH TO MECHANICAL VENTILATION IN ARDS: PRELIMINARY RESULTS OF A PROSPECTIVE RANDOMIZED PROTOCOL

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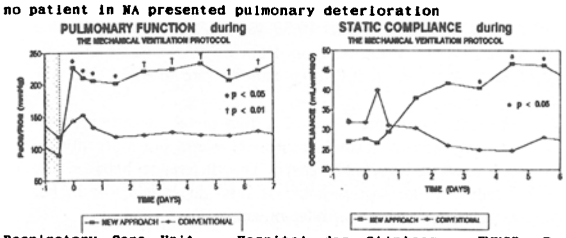
By using high PEEP levels, low tidal volumes (V_T), and limiting peak airway pressures, some authors have reported an improved prognosis in experimental models of ARDS.
This study reports the preliminary results of a prospective, randomized trial comparing two different approachs (new approach - NA vs. conventional - C) to mechanical ventilation in 19 consecutive patients with severe ARDS (Murray score > 3.0).
The new approach consists of a) Maintenance of PEEP levels above the inflection point of the P x V static inspiratory curve b) V_T < 6 mL/kg c) Tolerate PCO_2 up to 80 mmHg; Bicarbonate Sodium infused when pH < 7.20 d) Mantain Peak Airway Pressure < 40 cmH_2O e) Sequential use of Inverted-Ratio Pressure Controlled Ventilation (PC-IRV), Volume Assured Pressure Support Ventilation (VAPSV) and Pressure Support Ventilation (PSV), according to the FIO_2. The conventional approach consists of a) V_T = 12 mL/kg (fixed square wave flow) b) PEEP level as necessary to maintain PaO_2 > 60 mmHg and FIO_2 < 60% c) $PaCO_2$ between 25 and 38 mmHg.
All patients had a Swan-Ganz cateter and received a predetermined hemodynamic, infectious, nutritional and general support. Despite a worse APACHE II score in the NA group (mean score = 26 vs. 18 in the C group), the pulmonary function of the NA group had a better evolution, as showed below. Mortality rate was similar (6/10 in NA vs. 6/9 in C), but 4 patients died from progressive respiratory failure in C, whereas no patient in NA presented pulmonary deterioration



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LOW MORTALITY ASSOCIATED WITH ADVANCED TREATMENT INCLUDING V-V ECMO FOR SEVERE ARDS

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Today's therapy for severe ARDS is limited to procedures which predominantly support and maintain pulmonary function, i.e. pressure-limited controlled mechanical ventilation with PEEP and permissive hypercapnea, positional maneuvers, and reduction of pulmonary edema. Should these procedures fail to improve impaired gas exchange, veno-venous extracorporeal gas exchange (v-v ECMO) provides an alternative form of treatment. From April 1989 to May 1992 49 ARDS patients were transferred to our intensive care unit and treated according to an algorithm including the above mentioned therapeutic intervention strategies. The table shows our patients' survival rates related to their treatment groups.

	number of patients	number of survivors	survival rate (%)
ADVANCED TREATMENT (sine v-v ECMO)	26	24	92
v-v ECMO fast entry	8	2	25
v-v-ECMO slow entry	16	11	69
Total	49	37	76

The results of our ARDS treatment to date, including v-v ECMO with heparin-coated systems, suggest that the high death-rate among ARDS patients can be improved when all of the presently available therapeutic measures are applied.

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PRONE POSITION IN SEVERE ARDS INFLUENCES VENTILATION/PERFUSION RELATIONSHIP OF THE LUNG

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In patients with severe ARDS computed tomography studies revealed a predominantly posterobasal distribution of densities in the lung. Prone positioning in these patients has been reported to have a potentially beneficial influence on pulmonary gas exchange. However, the mechanisms causing this enhancement of oxygenation remain unclear. The present study was designed to test the hypothesis that alterations in ventilation-/perfusion relationship induced by prone positioning may be responsible for the improvement of oxygenation.
Nine patients with severe ARDS were included in our study (n=9). Classification of the patients using Murray's score revealed severe ARDS in all cases. All patients were ventilated in a pressure controlled mode and the ventilatory settings were not changed during the study Systemic and pulmonary hemodynamic parameters as well as arterial and mixed venous blood gas samples were obtained before, 30 min and 120 min after turning the patients into prone position. The patients then were turned back to supine position and data were collected after 30 min. Distribution of ventilation/perfusion ratios ($\dot{V}_A/\dot{Q}$) of the lung were determined by using the multiple inert gas elimination technique. Data are presented as means ± SD. According to the study performed by Langer[6], patients showing a minimum increase of PaO_2 by 10 mmHg were considered responders, those not fulfilling these criteria, non-responders. Five of nine patients revealed a significant increase in PaO_2 after 30 min and 120 min in prone position ($p \leq .05$) and a concomitant significant increase in arterial $PaCO_2$ and cardiac output. $\dot{V}_A/\dot{Q}$ distribution analysis showed a significant decrease in the shunt fraction ($\dot{V}_A/\dot{Q}=0$) and a significant increase in blood flow to lung regions with normal $\dot{V}_A/\dot{Q}$ ratios ($\dot{V}_A/\dot{Q}=1$) ($p \leq 0.05$). The improvement in PaO_2 was reversible within 30 min after turning the patient back to supine position ($p \leq .05$) without a significant decrease in $PaCO_2$ and shunt.

	supine	prone 30 min	prone 120 min	supine 30 min
PaO_2 (mmHg)	74.7±11.3	111.5±13.1	123.5±29.6	79.9±19.2
$PaCO_2$ (mmHg)	52.0±13.7	56.3±16.2	63.1±18.3	54.0±18.3
$\dot{V}_A/\dot{Q}=0$	0.43±0.09	0.35±0.09	0.37±0.11	0.39±0.16
$\dot{V}_A/\dot{Q}=1$	0.53±0.06	0.62±0.08	0.61±0.09	0.53±0.06
CO (ml/min)	8.53±5.66	8.37±5.87	8.87±6.17	9.31±5.13

Thus positioning patients with severe ARDS may result in an improvement of oxygenation. We conclude that these findings are induced by a redistribution of shunt to areas with a regular $\dot{V}_A/\dot{Q}$ ratio an a concomitant increase in CO.

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PRONE POSITION DEPENDENCY IN SEVERELY HYPOXIC PATIENTS. JM DUBOIS, Ph GAUSSORGUES, M SIRODOT, JM SAB, G CHATTE, B LANGEVIN, D ROBERT.

Prone position (PP) has been recommended in hypoxic ventilated patients because it may promote recruitment in the posterior gravity dependent areas with significant improvement in PaO2 and Qs/Qt. We report 4 patients in which arterial oxygenation improved under PP and deteriorated as soon as the patients returned to supine position (SP).

PP was introduced because of bilateral alveolar consolidation, severe hypoxemia despite adequate level of PEEP (12±2). PP and SP were alternatively used by 4 hours periods with constant ventilatory settings. All patients were hemodynamically monitored (Swan-Ganz catheter). Patients were mean aged 59±19 with a SAPS at 12±3 and under mechanical ventilation since 3,5±4,3 days for pneumonias, 2 evolving as ARDS (Murray score of 3,5 each).
All patients improved during the first period: PaO2/FiO2=92±37 before PP (min.=56, max.=128), PaO2/FiO2=153±77 at the end of PP (min.=82, max.=230). When the 4 patients were placed back on SP, there was a fall of PaO2/FiO2 resolutive when they returned to PP (208±65 to 77+±21, extreme variations 120 to 61 and 275 to 95). This PP dependency lasted 5,2±0,9 days including 67±14 h under PP before stabilisation of PaO2/FiO2 values whatever the posture was. No severe complication (haemodynamics, extubation) occurred during the procedure. The 2 patients with ARDS died.

These data suggest that repeated phases of PP may represent a usefull measure in severe hypoxic ICU patients. PP periods may be needed for several days.

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COMPARISON OF RESPIRATORY AND HEMODYNAMIC EFFECTS BETWEEN POSITIVE END EXPIRATORY PRESSURE (PEEP) VENTILATION AND "DECELERATED CONTROLLED EXPIRATION" (DCE) VENTILATION.
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Respiratory and hemodynamic effects of PEEP has been extensively studied. The purpose of this work was to compare PEEP effects with DCE effects. DCE was obtained on a CESAR ventilator (TAEMA-CFPO) by modulating expiratory pressure P from the end of inspiration (pressure Pi) to the time tf (pressure Pf) which is a part of the expiratory duration tE. Expiratory pressure P is a descending function of time t : $P = Pi - (Pi - Pf)\ (t/tf)^n$. n conditionned the "slowing-down" intensity and was equivalent to 2. At the time tf, expiration became passive. 7 sedated and curarized patients with severe hypoxemia were ventilated during 3 hours with PEEP = 10 cm H20 or with DCE (Pf = 25 % Pi and tf = 75 % tE were selected because they provided the best PaO2) after randomization. At the end of each period of ventilation, systemic arterial pressure, right heart pressures, cardiac output, airway and oesophageal pressures, were measured. Mean airway pressure (8.8 VS 14.8 cm H20) and oesophageal pressure (4.2 VS 11.3 cm H20) were significantly decreased in DCE group. Intrinsic PEEP (PEEPI) in DCE group is significantly lower than PEEP + PEEPI in PEEP group (4.5 VS 10 cm H2O). PaCO2 is significantly lower (41 VS 46 mmHg) with DCE ventilation ; in DCE group, PaO2 did not significantly decrease but right atrial pressure and pulmonary wedge pressure decreased, and cardiac output increased.
DCE should improve ventilation of hypoxemic patients by minimizing barotraumatic effects.
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Hemodynamic effects of PEEP

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THE HEMODYNAMIC EFFECTS OF PEEP IN PATIENTS WITH FAILING HEART
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There is a good experimental evidence that the increase in the intrathoracic pressure produced by positive pressure ventilation, and PEEP, may augment left ventricular ejection. However this proposition has not as yet extensively tested. In this clinical study we applied PEEP (0,5,10,15,20 cm H_2O) in 12 mechanically ventilated patients with normal cardiac index and pulmonary-capillary wedge pressure group A (CI>2.5 l/min/m^2, PCWP <15mmHg and in another 12 patients with reduced cardiac index and increased wedge pressure group B (CI<2.5l/min/m^2, PCWP >15mmHg). Hemodynamics were measure with a Swan-Ganz catheter and cardiac output by the thermodilution technique .The results are shown in the following table: A)Group with CI>2.5 l/min/m^2, PCWP<15mmHg. B)Group with CI<2. l/min/m^2, PCWP>15mm, *=P<0.05,**=P<0.01.

PEEP	CO A	CO B	PCWP A	PCWP B	MAP A	MAP B
0	5.9±1	4.4±0.7	10±4	17±2	83±17	76±14
5	5.5±2	4.5±0.8	11±4	17±2	83±17	74±16
10	5.3±1	4.4±0.7	12±5	18±3	82±20	73±17
15	4.8±2	4.3±0.7	14±4	19±3	81±19	75±18
20	4.3±1*	4.5±0.6	16±5**	20±3	78±21	75±18

PEEP	VO_2 A	VO_2 B	DO_2 A	DO_2 B	Cst A	Cst B
0	131±34	120±20	491±80	321±54	31±7	27±7
5	126±30	125±24	465±86	336±64	36±8	30±8
10	130±35	127±28	442±87	323±70	39±9*	32±9
15	126±35	133±28	407±98	330±69	40±9*	30±8
20	124±42	139±29	374±94	341±68	46±14**	28±6

Although with the application of increasing levels of PEEP cardiac output was decreased in the control group A it remained unchanged in the patients with impaired left ventricle group B.It is shown in the table that PEEP improved lung compliance in group A but not in group B although we found no difference in blood gasses,Shunt,(A-a) DO_2,PCO_2,between the two groups. Thus we conclude that PEEP has no significant effect,on the CO in patients with heart failure.This may be due to the reduction of the left venticular afterload and/or due to the inability of transmission pressure into the thoracic cage and right chambers as result of low lung compliance.
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HEMODYNAMIC EFFECTS OF APPLIED POSITIVE END-EXPIRATORY PRESSURE IN COPD PATIENTS MECHANICALLY VENTILATED FOR ACUTE EXACERBATION. A. Mercat, J.L. Teboul, R. Boujdaria, L. Graini, O. Pinamonti, F. Lenique, J. Depret, Ch. Richard, G. Conti.
Intrinsic positive end-expioratory pressure (PEEPi) is a common feature in COPD patients ventilated for acute exacerbation. Applied PEEP (PEEP) has been proposed to offset the increase in work of breathing induced by PEEPi during weaning. The aim of this study was to assess the hemodynamic consequences of PEEP in ventilated COPD patients with PEEPi. Thirteen mechanically ventilated patients (mean age 62 years, 48 to 78) sedated and paralysed were enrolled. PEEPi was measured by the end-expiratory occlusion method while patients were in a volume controlled mode with I/E = 1/3 and respiratory rate (18 ± 2) and tidal volume (9.4 ± 2.2 ml/kg) adjusted to achieve a normal pH. Hemodynamics (Swan-Ganz) and airway pressures were measured while patients received in a random order three levels of PEEP : 0, PEEP = PEEPi-5cmH2O, PEEP = PEEPi. Results are expressed as mean ± SD and compared by Anova.

	PEEP=0	PEEP=PEEPi-5	PEEP=PEEPi
pPaw cmH2O	34±7	35±8	38±8**
mPaw cmH2O	9±2	14±3*	18±4**
PEEP cmH2O	0±0	7±4*	12±4**
PEEPi cmH2O	12±4	5±2*	2±1**
Pplat cmH2O	22±6	23±7	27±8**
PaO2 mmHg	101±26	102±27	102±24
PaCO2 mmHg	47±5	46±6	46±5
MAP mmHg	89±10	90±7	83±8**
CI l/min/m2	3.5±0.4	3.4±0.5	3.1±0.6*
DO2 ml/min/m2	567±86	538±97	508±119*

pPaw : peak airway pressure, mPaw : mean airway pressure, Pplat : plateau pressure, MAP : mean arterial pressure, CI : cardiac index, DO2 : oxygen delivery. * : significantly different from PEEP=0 ($p < 0.05$). ** : significantly different from PEEP=0 and PEEP=PEEPi-5 ($p < 0.05$).

Application of a level of PEEP equal to PEEPi resulted in significant decrease in CI and DO2, linked to an increase in airway pressures; Despite a negative correlation between changes in CI and Pplat when PEEP was added, there was no threshold value of change in Pplat which could predict a PEEP induced fall in CI in a given patient. By contrast, application of PEEP equal to PEEPi-5 cmH2O has no deleterious effect on hemodynamics.

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