Title : PREVENTION OF VENOUS AIR EMBOLISM IN NEUROSURGICAL SITTING POSITION BY COMBINATION OF LOWER BODY POSITIVE PRESSURE AND PEEP WITHOUT FLUID LOADING

Authors : P SCHURANDO MD, D PAYEN MD PhD, S BELOUCIF MD, B GEORGE* MD, JL RAGGUENEAU MD, E ECHTER MD.

Affiliation : Department of Anesthesiology and Intensive Care and Neurosurgical Unit* University LARIBOISIERE Hospital, 2 rue Ambroise Paré 75010 PARIS, FRANCE.

INTRODUCTION:
Venous air embolism (VAE) is a frequent complication during neurosurgery in sitting position (SP)[1]. Maintenance of a very high right atrial pressure (RAP) seems to be essential to prevent this risk[2]. We designed the present study to 1) determine the effectiveness of mild (40 mmHg) Lower Body Positive Pressure (MLBPP) application followed by PEEP to increase intraluminal RAP[3] and 2) test if this RAP increase is transmitted to the cerebral veins.

METHODS:
Written informed consent was obtained for each patient and the protocol was approved by the Ethical Committee. 14 patients (38 yrs ± 15 SD), free of lung or cardiac disease, undergoing neurosurgery in sitting position were selected for the study. Anesthesia procedure included sodium thiopental (5 mg/kg), fentanyl (5 mcg/kg) and pancuronium bromide (.1 mg/kg). Mechanical ventilation (N_2O in O_2: .5) was adapted to maintain $PaCO_2$ between 30 and 35 mmHg. Early detection of VAE was realized by continuous monitoring of end-tidal CO_2 tension ($ETCO_2$, Siemens 930 CO_2 analyzer). Following parameters were measured or calculated : heart rate (HR in bpm, ECG lead), systemic arterial pressure (SAP in mmHg, radial catheter), right atrial (RAP), pulmonary arterial (PAP), and pulmonary wedge (PWP) pressures in mmHg via a Swan-Ganz catheter. Pressure transducers (Hewlett Packard) were carefully positionned at the level of the right atria in each situation and connected to an amplifier (Hewlett Packard) and to a multi channel recorder. Cardiac output (in l/min) was measured in triplicate (thermodilution). Cardiac index (CI), stroke volume index (SI) and systemic vascular resistance index (SVRI) were calculated using the standard formula. After MLBPP application, arterial blood gases were collected before and after PEEP institution to determine O_2 availability (TaO_2= $CI.CaO_2.10$ in ml $O_2.min^{-1}.m^{-2}$).

PROTOCOL:
4 successive sets of measurements were performed :
(1) Supine, 30 min after induction;
(2) 10 min after sitting position (SP);
(3) 15 min after MLBPP inflation in SP;
(4) 15 min after 10 cmH_2O PEEP added to SP+ MLBPP.
In this last setting, if RAP was lower than 15 mmHg, fluid loading was given to reach this value. MLBPP was realized using Military Anti Shock Trousers (MAST), inflated at relatively low pressures[3] (40 mmHg on lower limbs and 20 mmHg on abdominal area) in order to maintain a positive gradient between peripheral and central compartments.
Statistical analysis was performed using 2 way analysis of variance and Newman-Keuls test.

RESULTS:

	1	2	3	4	ANOVA
RAP	4.4	3.4	11.6	17	<.001
PAP	12.4	12.3	19.8	25.1	<.001
PWP	8	6.8	14.7	19.2	<.001
SAP	85	85	93	101	<.01
CI	2.8	2.7	2.7	2.7	NS
HR	67	66	62	60	<.001
SI	43	42	45	46	NS
SVRI	31	33	33	33	NS
TaO2			508	483	NS

	1Vs2	1Vs3	1Vs4	2Vs3	2Vs4	3Vs4
RAP	NS	<.001	<.001	<.001	<.001	<.001
PAP	NS	<.001	<.001	<.001	<.001	<.001
PWP	NS	<.001	<.001	<.001	<.001	<.001
SAP	NS	NS	<.001	<.05	<.001	<.05
HR	NS	<.001	<.001	<.001	<.001	NS

Only 5 out of 14 patients needed a small quantity of fluid (3 ml/kg) to achieve a RAP of 15 mmHg. Finally, in 3 patients the transmission of the rise of RAP to the lateral venous sinus (VSP) was verified at different RAP values. A strong positive linear relationship was found between RAP and VSP : $VSP = .55 \times RAP - 11$; $r = .998$; $p < 0.001$; $n = 12$.

DISCUSSION: MAST inflation during SP increased RAP mainly because systemic venous compliance was reduced[4]. However, the major finding was that PEEP, added to MLBPP, amplified the increase in RAP without any deleterious systemic hemodynamic effects. This further intraluminal RAP increase after PEEP resulted from an increase in right heart surrounding pressure, whereas PEEP-induced peripheral venous blood pooling had previously been prevented[3]. In addition, the maintained SAP (cerebral "input pressure") during PEEP associated with an increased RAP ("backpressure" of the system) explains the increase in intracerebral venous pressure. This latter effect could be so efficient that VSP increased to the atmospheric level, thus preventing VAE, without any surgical discomfort. Accordingly, no VAE could be detected with the methods of detection used. However, if these two non pharmacological and reversible techniques are not sufficient regarding to the RAP goal (> 15 mmHg), only a limited amount of fluid loading has to be given. This is of particular interest for the management of potential postoperative cerebral edema.

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