Changes In Alveolar Dead Space During General Anaesthesia In Posterior Spinal Surgery. An Observational Study

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Abstract

Background and objective: during general anaesthesia the alveolar dead space can change with the patient position and type of procedure. The aim of our study was to asses these variations in the prone position, during general anaesthesia in spinal surgery.

Methods: we have studied twelve patients undergoing spinal posterior surgery that assumed kneeling prone position, in TIVA and ventilated with constant tidal volume and zero PEEP. The measurements of the respiratory parameters was done with the volumetric capnometry technology after 30 and 90 minutes from kneeling prone position.

Results: the alveolar dead space increased at 30 min and after it decresead to baseline at 90 min; the PaO2 increased and PaCO2 reduced over time; the static compliance and Plateau pressure did not change.

Conclusions: our study demonstrates that in the kneeling prone position the alveolar dead space rises at 30 minutes with costant tidal volume.

INTRODUCTION

Respiratory function changes during general anaesthesia with intermittent positive pressure ventilation (IPPV) in the supine position. Gas exchange is impaired by the development of atelectasis, resulting shunts, ventilation/perfusion mismatching (1) and lung overdistension can be characterized by an increased physiologic dead space (2-4). Dead space is the portion of ventilation that is not participating in gas exchange, because it does not come in contact with the pulmonary capillary blood flow (5-7).

In the supine position under general anaesthesia, tidal volume is directed primarily towards the lung regions not dependent, incomplete alveolar gas mixing with areas of increased alveolar ventilation/perfusion (V/Q) mismatching within terminal respiratory units and the preferential spread of distribution of ventilation to areas of less perfusion, increases alveolar dead space (VDalv) (8). The increase of dead space appears to be linked to the increase of the positive pressure within the alveoli (9). The resulting hyperinflation determines the collapse of the alveolar capillaries, with the formation of lung areas with high ventilation perfusion ratio, that are ventilated but not perfused (10).

Knowing pulmonary dead space in patients under general anaesthesia is clinically useful because it can aid in detecting disease processes characterized by low cardiac output states, with reduced pulmonary blood flow, or alveolar stretching, preventing ventilation to participate gas exchange (11).

Various studies demonstrated that recruitment maneuvers can remove atelectasis and reduce dead space fraction (VD/VT) (12-17).

Also the prone position used during spinal surgery increased oxygenation and the functional residual capacity (FRC), for more homogeneous lung perfusion (18) and reduction of ventilation/perfusion mismatching, with the decrease of atelectasis, provided that a correct positioning is implemented (19). Indeed, it was documented a reduction in the compliance of the respiratory system to inadequate positioning (20). During spinal surgery is adopted kneeling prone position, assuring free abdominal and supports

inserted under the sternum and the iliac crests with knees flexed.

Prone position during general anaesthesia, reducing atelectasis, should reduce the dead space.

This observational study was designed to evaluate the effects of ventilation in the prone position on the alveolar dead space during general anaesthesia.

METHODS

Ethical approval for this study (Ethical Committee N 153 del 15/5/2013) was provided by Ethical Committee Azienda Sanitaria Locale Salerno (Chairperson Prof G. Buda) on 21 May 2013

After approval of the local ethics committee and written informed consent, 12 patients, ASA I II, (age 18-70 years), undergoing for posterior discectomy surgery in the prone position, were studied prospectively between June 2013 and October 2013.

Exclusion criteria were age <18 years, pregnancy, obesity, cardio-respiratory patologies, interventions performed under the emergency, patients who refuse to sign the consent form.

On arrival in the Recovery Room a peripheral vein was cannulated and performed premedication with Midazolam (0,02 mg/Kg) and Fentanyl 0,1 mg. It was initially performed a calibration device with loss compensation circuit. The patient was transferred into the operating theatre, lying on a stretcher in supine position. Monitoring consisted of: electrocardiogram (ECG), Heart rate (RR), Arterial oxygen saturation (SpO2) measured by pulse oximeter, blood pressure measured by the non invasive automated method every 5 minutes, body temperature with esophageal catheter, (Infinity® Delta, Dräger Medical Italia).

Anaesthesia was induced with TCI TIVA (Target Controlled Infusion, Total IntraVenous Anaesthesia) method with Propofol 2% (Alaris® CC Plus Syringe Pump, CareFusion) according to the following scheme:

1 step: 1 mcg /ml

Step 2: 2mcg/ml

Step 3: 3mcg/ml

Step 4: 4mcg/ml

at intervals of 30 seconds, Remifentanil 0.12 mcg/Kg/min, the neuromuscular blockade was obtained with Cisatracurium 0.2 mg/kg.

After tracheal intubation, the patient was connected to the AVEA Ventilator (CareFusion®), in IPPV mode, tidal volume 8 ml/kg, respiratory rate 12-14, PEEP absent, FiO2 50%, an inspiration/expiration ratio of 1:2 with an end inspiratory pause of 0.5 seconds.

Ventilator settings were kept constant throughout the study.

All patients were ventilated without PEEP, as previous studies have shown that the addition of PEEP during mechanical ventilation causes redistribution of both lung blood flow and ventilation. In prone position the redistribution toward dependent regions was much greater for blood flow than for ventilation, causing increased V/Q mismatch (21).

The maintenance of anaesthesia was obtained with 2% Propofol in TCI, maintaining a context sensible concentration of 3-4 mcg/ml, variable to maintain stable hemodynamic parameters, Heart rate and arterial blood pressure within 20% limits of pre induction values, 50% oxygen, Remifentanil 0.20 mcg/kg/min, Cisatracurium 2 mg every 40 minutes, as needed to maintain the bispectral index (BIS) between 40% - 60% (BIS™ Complete 4-Channel Monitor, Covidien).

Intraoperative crystalloids were administered at a rate of 2–4 ml kg/h and blood loss was initially replaced with crystalloid or with red blood cell concentrates if haemoglobin levels decreased below 80 g litre.

All patients were extubated in the operating theatre and admitted to a postoperative care unit for at least 6 h before being transferred to the surgical ward.

After positioning bladder catheter, nasogastric tube, eye bandages, the patient was positioned prone on the operating table after 20 minutes, in kneeling prone position.

Anatomical silicon holders were placed under the sternum and the iliac crests, with free abdomen.

End tidal CO2 (EtCO2), CO2 elimination (VCO2), anatomic dead space (VDana), alveolar dead space (VDalv), dead space fraction (VD/VT) were calculated with integrated module volumetric capnometry in AVEA ventilator, Capnostat 5. The Capnostat measure the CO2 through the infrared absorption technique.

It is based knowing that the molecules of CO2 absorbing infrared rays (IR) of specific wavelengths and the amount of energy absorbed is proportional to the concentration of CO2.

When an IR beam is passed through a gas sample of CO2 it is possible to achieve an electrical signal emitted by a photodetector.

The measurements of volumetric capnometry required a mainstream CO2 sensor, inserted into an adapter and positioned in the ventilation circuit between the endotracheal tube and the Y junction, previously calibrated (figure 1).

The sensor was connected by a cable to the user interface module to the AVEA system. The CO2 signal is obtained by mainstream, non dispersive, infrared capnometer. Single breath curve is displayed by the monitor. Expired CO2 (y-axis) is plotted against expired volume (x-axis). Expired single breath CO2 waveform is divided into three phases, as first described by Fowler (22). Phase 1 represents CO2 free gas expired from large airway and endotracheal tube dead space, phase 2 represents a mixture of gas from both airway and alveolar deadspace, phase 3 represents alveolar ventilation.

The physiological, anatomical, alveolar dead spaces are calculated cycle by cycle from this curve by Capnostat 5, provided that an arterial PaO2 and PaCO2 from arterial gas analysis is introduced.

The VTCO2 is the amount of CO eliminated during one breath obtained by integration of expired airway flow and PaCO2. PECO2 is the partial pressure of CO2 at the end of expiration. Airway dead space (VDaw) was calculated as the inflection point of phase II of the capnogram or the limit between VDaw and the alveolar tidal volume (VTalv) (12).

Physiological dead space to tidal volume ratio (VD/VT) (13) was calculated with the Enghoff modification of the Bohr equation VDphys/VT = (PaCO2 - PECO2)/PaCO2 where PECO2 is the mixed PCO2 of an expiration. Physiological dead space (VDphys) was then calculated by multiplying VD/VT and tidal volume. Alveolar dead space was obtained by subtracting VDaw from VDphys and then normalized by the alveolar tidal volume (VDalv/VTalv).

Respiratory parameters, Plateau pressure (Pplat), Static Compliance, were calculated with the integrated module to the ventilator and detected by the monitor.

The Plateau Pressure was measured by pushing end-inspiratory hold button for 3-4 seconds. Occlusion was maintained until airway pressure (Paw) decreased from maximum value (Pmax) to a plateau.

The Plateau pressure represents the static end-inspiratory recoil pressure of respiratory sistem. Static compliance (Cstat) of the respiratory sistem was calculated dividing VT by plateau pressure, since the external PEEP was zero.

The static compliance and Plateau pressure were determined automatically by Avea ventilator. Intrinsic PEEP (PEEPi) was measured during a four-second end-expiratory occlusion period.

Arterial blood gases, volumetric capnography, and ventilatory data were recorded at the following study points. The respiratory data were obtained 20 minutes after induction of anaesthesia while patients were in supine position and 30, 90 minutes after turning to prone position.

For the measurements of arterial gas analysis, blood specimens were processed and corrected for body temperature within five minutes of extraction by a gas analyzer (Gem Premier® 3000, Instrumentation Laboratory).

Statistical analysis

Nominal qualitative variables were expressed as moda, more frequent distribution of cases, discrete and continuous quantitative variables were expressed as mean ± standard deviation.

Analysis of Variance (ANOVA) Test for repeated measurements was used for each parameter, following the Post Hoc analysis for the adjustment of significance for each comparison with the Bonferroni test.

Wilcoxon's signed rank sum test was used, when $P < 0,05$, being considered statistically significant. The analysis was performed using SPSS 18 software.

RESULTS

Twelve patients, nine male (75%) and three female (25%) were studied. They had a median age of 46 ± 11, weight of 86,5 ± 6,5 Kg.

The results of the respiratory changes are presented in table 1

Any episodes of respiratory depression occurred in the premedication.

The alveolar dead space was 165,3±50,4 ml in the supine position. After the kneeling prone position, the alveolar dead space increased at 30 minute (195,5±75,1) and reduced to 90 minutes 161,6±53,5.

The difference in mean values of the alveolar dead space at 30 and 90 minutes after the prone positioning has been significantly, P< 0,05 (Figure 2).

The Dead space VD/VT ratio followed the same trend as alveolar dead space, in supine position was 39,8±9,2 ml at 30 min in prone position 43,1±9,2 and 36,5±18,3P< 0.05 at 30 and 90 min.

The anatomical dead space increased after prone positioning, 92,3±37,2 ml in supine position, 102,7±43,1 - 109,2±44,3 at 30 and 90 min in prone position (Figure 2).

Cstat, Pplat did not change.

The End Tidal CO2, VCO2 and VtCO2 decreased from supine to prone position, EtCO2 36,9±2,3 mmHg in supine position, 32,8±4 - 32,9±3,6 at 30 and 90 min in prone position; VCO2 234,3±35,3 ml in supine position, 203,1±26,8 - 200,5±21,3 at 30 and 90 min in prone position; VtCO2 21,9±2,2 ml in supine position, 20,7±2,5 - 20,8±2,4 at 30 and 90 min in prone position.

In association with turning the patients into prone position there were rises in PaO2 and a decreased in PaCO2 (Figure 3).

The values of the systolic and media blood pressure have remained stable.

CONCLUSIONS

The present study showed that the alveolar dead space increased at 30 min in prone position, in patients undergoing elective posterior spinal surgery, under general anaesthesia and it decreased at 90 min, meantime PaCO2 decreases and the PaO2 increased over time.

Moreover even EtCO2, VCO2 and VtCO2 decreased from supine to prone position, without hemodynamic changes that could account for any reduction.

Capnography is the analysis and continuous record of the concentration of CO2 in respiratory gases, with the wave display exhalation of CO2, while the Capnometry measure only the amount of CO2 (23).

The device of volumetric capnography measures with each tidal volume the elimination of CO2, through the analysis of the area under the curve of capnography.

The capnogram can be divided into three phases. First phase represented by the tidal volume free from CO2, the second phase of the CO2 coming from progressive emptying of the alveoli, the third phase represented only by the alveolar gas (24).

Through Capnometry volumetric we can establish the proportion of dead space.

The physiologic dead space is the ventilation portion which does not participate in gas exchange because it is not in contact with the blood flow in the pulmonary capillaries and is formed from areas of the lung that are ventilated but not perfused, in which, therefore, there is not CO2 elimination (25).

The dead space is divided into anatomical dead space, represented by the airways, external ventilation device, circuit, endotracheal tube, and the alveolar dead space, expressed as the amount of respiratory volume for time unit or as a fraction of tidal volume.

The physiological dead space is calculated with the equation of Bohr modified by Enghoff.

It is assumed that the PaCO2 is similar to the alveolar CO2:

VDphys / VT = (PaCO2 - PECO2) / PaCO2 (26)

It is well now established that in during general anaesthesia atelectasis are formed, which cause a reduction in functional residual capacity and compliance (17, 27).

Atelectasis are created mainly in regions dependent, because during general anaesthesia in the supine position tidal volume is directed primarily toward the anterior regions not dependent, (for diaphragmatic muscle paralysis), leading to an increase of mismatching ventilation perfusion, and increasing the alveolar dead space (28). Alveolar dead space may be the consequence of non perfused or poorly perfused lung areas in ventilated anterior areas.

Previously during general anaesthesia it was seen the rising of the physiologic dead space, during abdominal surgery, especially when it was used a lithotomy position.

Unoki demonstrated that the fraction of dead space increase in patients undergoing abdominal surgery (29).

Other studies have shown instead that in patients undergoing non abdominal surgery the fraction of dead space was not modified.

Lumley et al. (30) in patients undergoing femoral popliteal bypass have seen that the fraction of dead space was not modified, Miyazaky has observed the same in patients undergoing minor surgery (31).

Downs has shown, in cardiopulmonary bypass, that the increase in physiologic dead space was linked over to the frequency of mechanical ventilation cycle and the increase of pressure in the airways with under perfusion of the alveoli. In fact, the increase of the pressure in the alveoli determines an overdistension with the collapse of the small pulmonary capillaries, resulting in a reduction of the perfusion in these areas with increase in the ratio ventilation perfusion (9).

Praetel and others have observed too that the increase in dead space during anaesthesia, was greater with inhalatorian anesthesia with isoflurane compared to TIVA (8).

Previously, in prospective study of 466 patients undergoing cardiac surgery Weiss had made an similar observation. This was speculated to be due to the action of inhalatorian agents direct bronchial muscular tone (32).

Soro and others, using the volumetric capnometry, have shown that the physiologic dead space and alveolar is not changed during the general anaesthesia in patients in the prone position after three hours, while improving oxygenation, moreover had detected no changes in PaCO2 (19).

This is in contrast with the results of Casati and Wahba (33, 34), who observed an increase of 10% of the fraction of dead space to 20 minutes during of general anaesthesia in the prone position. Our data, increased alveolar dead space to 30 minutes in the prone position, are similar to those Casati and Wahba, even if the anesthesia was maintained with Isoflurane, while we have used the TIVA TCI.

The main explanation is that we used a constant tidal volume during the study, compared to Soro et al., that modified the ventilatory parameters to maintain PaCO2 constant, but we can not exclude that the different measurement techniques of dead space can modify the results.

Tusman and collaborators have found that alveolar dead space and dead space fraction are closely related with the formation of atelectasis in the lung parenchyma, since the collapse of the lung areas induced alterations in the distribution of ventilation and gas exchange, moreover, in several studies it was established that the same changes of the tidal volume may alter the measurement of dead space (26).

The prone position improves oxygenation in acute respiratory failure by improving the ventilation perfusion ratio, provided that the positioning is done correctly.

In fact our results were moving in this direction with an increase in PaO2 and a decrease in PaCO2 during the study, while the static compliance remains constant.

Lynch in anesthetized patients in the prone position has found a reduction of 20% in the respiratory system compliance (35).

It was due to the use of rigid supports that did not leave free the chest and abdomen.

Lumb and Nunn however found that the prone position cause an increase in functional residual capacity (36), in awake patients, not paralyzed, and they did not observe changes in the Compliance and Plateau pressure, because we believe that the positioning has been corrected. Pelosi has demonstrated that the prone position, if correctly followed, does not affect the lung and chest wall compliance, but improve oxygenation and lung volumes (FRC), the same observation was also made in obese patients (20, 37).

The kneeling prone position has little effect on the consumption of oxygen, but immediately improves oxygenation and reduces the alveolar ventilation.

Radstroom has seen that after 10 minutes in the prone position VCO2 was reduced by the reduction in alveolar ventilation, because the legs, that were positioned below the heart caused blood accumulation and consequently reducing cardiac output (38).

This minimizes the elimination CO2 by reduction of alveolar ventilation.

In conclusion our study demonstrates that in patients undergoing surgery in prone position under general anaesthesia, by using a constant tidal volume, alveolar dead space rises after 30 minutes from the prone positioning, and then decrease to baseline values.

Figure 1

The airway adapter and sensor placed into the ventilator circuit between the wye and endotracheal tube as shown in the illustration.

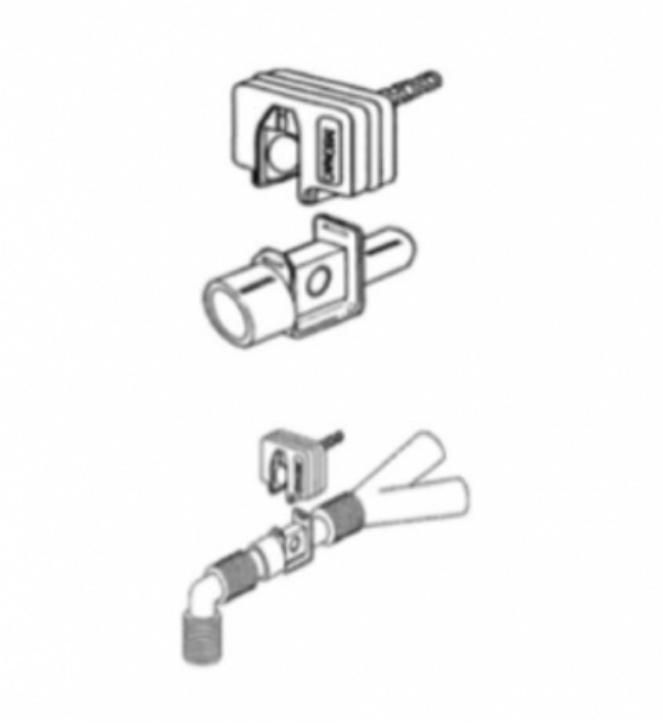

Figure 2

Alveolar dead space; anatomic dead space; P < 0,05 compared with baseline value. † < 0,05 compared with 30 min in prone position

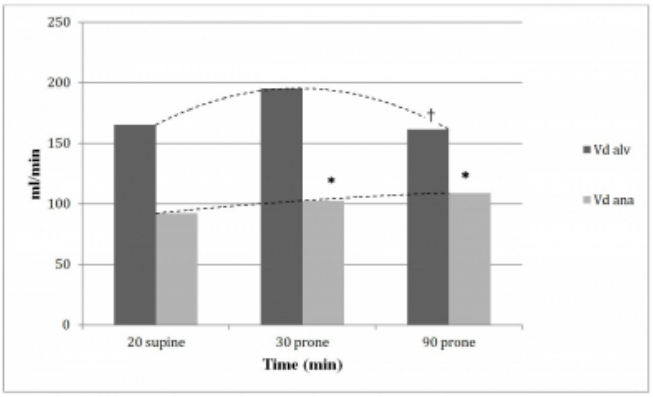

Figure 3

Arterial PO2 and PCO2. P < 0,05 compared with baseline value.

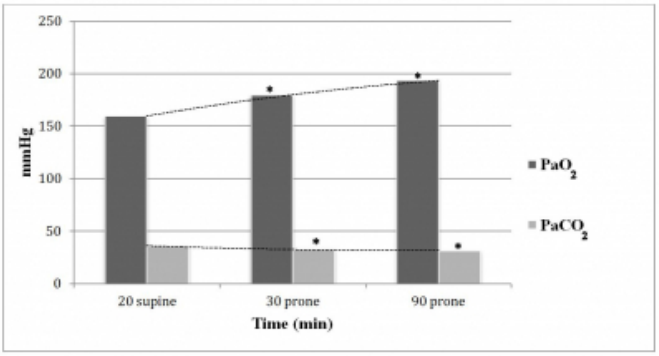

Table 1

VDalv: Alveolar dead space; VD/VT: physiological dead space/tidal volume ratio; Cstat: compliance; Pplat: plateau pressure; EtCO2: end tidal CO2; VCO2: CO2 elimination; VTCO2: CO2 elimination for tidal volume; VDana: anatomic dead space; SBP: systolic blood pressure; MBP: media blood pressure. Results are expressed as mean ± SD. P < 0,05 for prone vs. supine; † P < 0,05 for 30 vs. 90 min in prone position

	Supine	Prone	
	20 min	30 min	90 min
Vd alv (ml)	165,33±50,4	195,50±75,1	161,6±53,5†
Vd/Vt (ml)	39,8±9,2	43,1±9,2	36,5±18,3†
$C_{stat\ ml/cmH2O}$	41,5±6,5	40,9±8,4	39,1±4,1
$P_{plat\ cmH2O}$	17,1±1,9	16,5±1,9	17,3±1,4
$EtCO_{2\ mmHg}$	36,9±2,3	32,8±4 *	32,9±3,6 *
$VCO_{2\ ml/min}$	234,3±35,3	203,1±26,8 *	200,5±21,3 *
$VtCO_{2\ ml}$	21,9±2,2	20,7±2,5 *	20,8±2,4 *
$V_{ana\ ml}$	92,3±37,2	102,7±43,1 *	109,2±44,3 *
$PaO_{2\ mmHg}$	159,7±22	180±20,7 *	193,5±18,7 *
$PaCO_{2\ mmHg}$	36,1±3,3	32,5±3,6 *	31,5±2,9 *
Hb g/dl	14,3±0,4	13,9±0,4	14,5±0,3
SBP mmHg	120±20	110±23	118±19
MBP mmHg	75±10	70±12	69±11

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