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Original Contribution

The relationship between end-expired carbon dioxide tension and severity of venous air embolism during sitting neurosurgical procedures – A contemporary analysis

Caitlin J. Kapurch, M.D.*, Arnoley S. Abcejo, M.D., Jeffrey J. Pasternak, M.D.

Department of Anesthesiology and Perioperative Medicine, Mayo Clinic College of Medicine, 200 First Street SW, Rochester, MN 55902, USA

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ABSTRACT

Study objective: Determine if changes in expired carbon dioxide tension correlate with the severity of venous air embolism (VAE) associated hemodynamic changes in humans.
Design: Retrospective case series.
Setting: A single academic medical center with high-volume neurosurgical practice.
Patients: One hundred forty seven adult patients having neurosurgical procedures performed with general anesthesia in the sitting position who experienced venous air embolism.
Interventions: Identification of documentation of venous air embolism by either precordial Doppler sonography or transesophageal echocardiography.
Measurement: Retrospective determination of changes in end-expired carbon dioxide ($EECO_2$) changes associated with venous air embolism.
Main results: Greater absolute and relative decreases in end-expired carbon dioxide tension were associated with greater hemodynamic manifestations of venous air embolism. However, based on receiver operating characteristic curve analysis, the absolute and relative changes in $EECO_2$ have moderate utility for predicting the severity of hemodynamic consequences of venous air embolism as area under the curve for absolute and relative carbon dioxide tensions were 0.7654 and 0.7263, respectively.
Conclusions: Greater magnitude of decreases in $EECO_2$ is associated with hemodynamically-significant VAE in mechanically-ventilated patients. However, the magnitude of changes may have limited utility to diagnose VAE or exclude the diagnosis of VAE in patients with unexplained intraoperative hypotension.

1. Introduction

Venous air embolism (VAE) is a known complication of procedures performed in the sitting position. The incidence of VAE in the sitting position has been reported as high as 25% [1–4]. The severity of VAE events can vary from clinically insignificant to life-threatening hemodynamic collapse, respiratory failure, permanent neurologic deficits, and death [1–4]. Many institutions have chosen to forego utilization of the sitting position for surgical procedures as a result of these documented risks [5,6]. However, the sitting position offers several physiologic, surgical, and anesthetic advantages for neurosurgical operations when compared to the supine position, including improved visualization of the surgical field, decreased brain bulk, and improved postoperative cranial nerve function [1]. As the sitting position continues to be utilized, prediction and early identification of VAE is critical.

Transesophageal echocardiography (TEE) and precordial Doppler sonography are most commonly utilized to monitor for perioperative VAE [1,2]. Though nonspecific, reductions in end-expired carbon dioxide tension ($EECO_2$) may also suggest the presence of VAE. Such changes occur during VAE events due to the development of dead space in the lungs.

VAE events are not limited to neurosurgical procedures performed in the sitting position. In these cases, clinicians may not monitor for VAE events with TEE or precordial Doppler sonography. Consequently, they may be limited to changes in $EECO_2$ and hemodynamic changes to warn of acute VAE. Thus, understanding the utility of changes in $EECO_2$ as a predictor of the severity of VAE in cases where routine monitors for VAE are not employed can serve significant clinical utility.

The correlation between the magnitude of changes in $EECO_2$ and the

* Corresponding author at: Mayo Clinic, 200 First Street SW, Rochester, MN 55902, USA.
E-mail addresses: Kapurch.Caitlin@mayo.edu (C.J. Kapurch), Abcejo.Arnoley@mayo.edu (A.S. Abcejo), Pasternak.Jeffrey@mayo.edu (J.J. Pasternak).

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severity of hemodynamic manifestations of VAE in humans has been sparsely reported in the literature [7]. The present study aims to determine the ability of observed changes in $EECO_2$ for predicting the severity of venous air embolism.

2. Materials and methods

Following approval from the Institutional Review Board at the Mayo Clinic in Rochester, MN, patients who underwent a neurosurgical procedure performed in the sitting position utilizing general anesthesia between January 1, 2000 and October 8, 2013 were retrospectively identified. Neurosurgical procedures performed in the sitting position were identified from the electronic listing form completed by the surgeon where only procedures performed by neurosurgeons were included and the surgical position recorded on the anesthesia record was "sitting" or "seated". Each electronic medical and anesthetic record was electronically queried for the following terms to screen for those who suffered a potential venous air embolism: "VAE," "air," "venous air embol," "air embolus," "air emb," "air he," "air de," "air se," and "bubbl," as well as the presence of the automated VAE documentation field "Air detected, surgeon and anesthesiologist notified." The medical record of each positive "hit" was manually reviewed to confirm evidence for VAE as indicated by specific documentation of VAE in the record, documentation of a change in the sound of heart tones obtained from precordial Doppler sonography, or the presence of intracardiac air on transesophageal echocardiography. The surgical operative reports were also reviewed for VAE. As it was important to know the time of VAE so that concomitant changes in $EECO_2$ could also be noted, any patients who had a documented episode of VAE but for whom the precise time of the event to within ± 16 min was unknown were excluded (i.e. in cases wherein the surgeon reported a VAE but no anesthesia documentation was performed). In patients having neurosurgical procedures in the sitting position but not entailing general anesthesia, such as patients having lead implantation for a deep brain stimulator system, $EECO_2$ is either not recorded or is likely unreliable when sampled from a closed face mask. Thus, we included only patients in whom general anesthesia was employed.

Each VAE event was stratified based on severity into three categories using the following criteria:

- Mild: Minimal or no physiologic changes: vasopressors not administered and mean arterial blood pressure (MAP) still within 20% of baseline.
- Moderate: Significant hemodynamic change such that vasopressor drugs were administered to maintain MAP within 20% of baseline.
- Severe: The hemodynamic and physiological consequences of VAE mandated emergent return from the seated position to the supine or lateral position.

The electronic medical record of each patient included in the study cohort was reviewed, and the following information was abstracted: patient demographics, mode of ventilation, vasopressor medications administered during the VAE event, and the need for emergent surgical closure and return to supine position during the VAE. Intraoperative data were recorded every 2 min. Several terms were defined:

- $EECO_2$ baseline: defined as the mean $EECO_2$ value obtained from data taken at 2 min intervals from 20 to 30 min prior to the documented VAE event. We defined the range of baseline $EECO_2$ as the difference between the minimum value and maximum value occurring during this 10 min period.
- $EECO_2$ minimum: defined as the lowest $EECO_2$ value within ± 16 min of the documented VAE event.

Twenty to 30 min prior to VAE was chosen as the baseline, as this time was a physiologic steadfast point in the operation, occurring after surgical incision. The absolute and relative changes in $EECO_2$ were calculated as follows:

- Absolute change = $EECO_2$ baseline − $EECO_2$ minimum
- Relative change = Absolute change ÷ $EECO_2$ baseline

Anesthetic techniques during the interval of 20–30 min prior to VAE were noted. Changes in fraction of oxygen inspired (FiO_2) and management of nitrous oxide during VAE were also noted. Lowest peripheral oxygen saturation (SpO_2) within ± 16 min of the documented VAE was recorded.

To characterize the temporal relationship between decrease in both $EECO_2$ and mean arterial blood pressure during VAE, we included only patients who suffered a moderate VAE and did not receive any drugs to alter blood pressure, as these drugs could alter the rate of decrease and nadir in blood pressure after VAE. For this analysis, we excluded patients with mild VAE, as hemodynamic changes in this group were minimal. We also excluded those with severe VAE, as administration of drugs to increase blood pressure and the position change may affect the rate of decrease in blood pressure and nadir. We report both mean arterial blood pressure and $EECO_2$ during the interval of ± 16 min of the reported VAE event.

Two one-way ANOVA tests were conducted for (1) absolute and (2) relative reduction in $ETCO_2$ based on VAE severity with post-hoc Paired Student's *t*-tests, if appropriate. A p-value $< .05$ was considered statistically significant with correction for multiple comparisons, if appropriate.

To further quantify the utility of either absolute or relative changes in $EECO_2$ to predict the severity of hemodynamic changes associated with VAE, receiver operating characteristic (ROC) curves were calculated. For this ROC analysis, absolute and relative changes in $EECO_2$ were stratified based on the presence of mild vs moderate or severe VAE. Sensitivity, specificity, and the predictive power of both positive and negative tests were calculated for various cut-off values for both absolute and relative changes in $EECO_2$ during VAE to distinguish between those with (i.e., moderate or severe) and without (i.e., mild) significant hemodynamic changes.

3. Results

During the time interval between January 1, 2000 and October 8, 2013, a total of 1886 patients underwent neurosurgical procedures in the sitting position. One hundred eighty eight patients were excluded due to inconsistent or missing data. As such, VAE events were identified from among 1698 sitting neurosurgical cases that met inclusion criteria. Of these, 168 were identified as having a VAE event that met criteria for inclusion into the study cohort yielding a VAE incidence of 9.9%. Four of these 168 cases were excluded from the study due to missing timing of the VAE event in the medical record. Fig. 1 illustrates the inclusion and exclusion of patients resulting in the final study cohort. Specific demographic data are displayed in Table 1 according to type of neurosurgical procedure.

During the period 20 to 30 min prior to VAE, general anesthesia was maintained with either a combination of isoflurane and nitrous oxide ($n = 93$; 63%), isoflurane without nitrous oxide ($n = 47$, 32%), desflurane without nitrous oxide ($n = 7$, 5%) or total intravenous anesthesia utilizing propofol and fentanyl infusions ($n = 1$, 1%). Management of fraction of inspired oxygen and nitrous oxide stratified by VAE severity can be found in Table 2. Overall, we found that the SpO_2 occurring in the setting of VAE was below 95% in only 4 cases occurring during 1 mild, 2 moderate, and 1 severe VAE episodes. In the single case of $SpO_2 < 95\%$ during mild VAE, SpO_2 decreased to 91% as a single data point while FiO_2 was 28%, however FiO_2 was then immediately increased to 100% with subsequent increase in SpO_2 to above 95%. In the first case of moderate VAE, a single SpO_2 of 94% was noted while FiO_2 was 33% and resolved without a change in FiO_2. In the