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# *Transcutaneous $O_2$ and $CO_2$ Monitoring of Neurosurgical Patients: Detection of Air Embolism*

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**Transcutaneous oxygen tension ($Ptc_{O_2}$) and transcutaneous carbon dioxide tension ($Ptc_{CO_2}$) were monitored in 60 patients undergoing neurosurgical procedures. Twenty-six patients were in the sitting position and underwent routine monitoring for air embolism. Seventeen episodes of air embolism were diagnosed by precordial Doppler ultrasound or transesophageal echocardiography, and the $Ptc_{O_2}$ decreased early during the course of each episode. The mean $Ptc_{O_2}$ decrease was 48 ± 35 mmHg. During ten episodes the end-tidal carbon dioxide tension ($PET_{CO_2}$) decreased but only after the $Ptc_{O_2}$ had already begun to decrease. $Ptc_{CO_2}$ increased during air embolism but $PET_{CO_2}$ changes preceded the change in $Ptc_{CO_2}$ by 1–2 min. Transcutaneous values during air embolism were verified with simultaneous arterial blood gas values during six air embolism episodes. A strong positive correlation was found between transcutaneous and arterial oxygen and carbon dioxide tensions. Correcting the $Ptc_{CO_2}$ by the patient's baseline $Ptc_{CO_2}/Pa_{CO_2}$ ratio, $Ptc_{CO_2}$ monitoring correctly reflected hypocarbia, normocarbia, and hypercarbia in 92% of the cases. $Ptc_{O_2}$ monitoring was useful in detecting venous air embolism and may respond sooner than $PET_{CO_2}$. $Ptc_{CO_2}$ monitoring was not useful as an early detector of air embolism. (Key words: Carbon dioxide: transcutaneous. Embolism: air. Monitoring: carbon dioxide; oxygen. Oxygen: transcutaneous. Position: sitting.)**

THE VALIDITY OF transcutaneous oxygen ($Ptc_{O_2}$) and transcutaneous carbon dioxide ($Ptc_{CO_2}$) monitoring in anesthetized adults has been established.[1–5] The transcutaneous method has potential value for measuring and following change in $Pa_{O_2}$ and $Pa_{CO_2}$ during neuroanesthesia. The risk of venous air embolism is significant in neurosurgical cases performed in the sitting position.[6] Monitoring for air embolism is an important component of the anesthetic care of these patients. A moderate increase in physiologic dead space occurs during pulmonary air embolism leading to an increased $Pa_{CO_2}$.[7,8] Hypoxemia also occurs secondary to an increase in lung units with low ventilation–perfusion ratios.[9,10] Both $Ptc_{O_2}$ and $Ptc_{CO_2}$ may be useful in detecting the changes in $Pa_{O_2}$ and $Pa_{CO_2}$ that occur during venous air embolism.

The purposes of this study were to assess the value of $Ptc_{O_2}$ and $Ptc_{CO_2}$ monitoring in detecting both venous air embolism and the abnormalities of gas exchange that accompany venous air embolism.

## Methods

Transcutaneous $O_2$ and $CO_2$ were monitored‡ in 60 patients (aged 22–88 yr; 42 men, 18 women) undergoing elective neurosurgical procedures as approved by our Institutional Review Board. Patient ASA class included I and II (34), III (24), and IV (2). Surgical procedures included 40 craniotomies, 15 cervical laminectomies, and five carotid endarterectomies. Twenty-six patients were in the sitting position. Direct arterial pressure, electrocardiogram, and esophageal temperature were continuously monitored. End-tidal carbon dioxide tension ($PET_{CO_2}$) and isoflurane concentration ($FET_{ISO}$) were measured at least every 2 min by mass spectrometry (Perkin-Elmer, Pomona, CA). In addition, all patients in the sitting position had precordial Doppler ultrasound, right atrial pressure monitoring, and continuous $PET_{CO_2}$ monitoring (Hewlett Packard, Palo Alto, CA). $Ptc_{O_2}$, $Ptc_{CO_2}$, and $PET_{CO_2}$ were continuously recorded in all patients in the sitting position. In selected sitting-position cases, transesophageal echocardiography (TEE) (Diasonic, Inc., Milpites, CA) was used. Anesthesia was maintained with isoflurane/fentanyl/$N_2O$/$O_2$. No vasodilators or vasopressors were continuously required in any patient to maintain cardiovascular stability. In keeping with the routine practice at our institution, when air embolism was diagnosed, any $N_2O$ being administered was discontinued and the patient was ventilated with 100% oxygen.

After two-point calibration, the transcutaneous electrodes were placed on the anterior chest or forearm. The electrode temperature was 44.5° C for $Ptc_{O_2}$ and 44 °C for $Ptc_{CO_2}$. The site of electrode application was changed every 4 to 6 h, and the monitors were recalibrated with each change.

Baseline arterial blood gas measurements and transcutaneous $O_2$ and $CO_2$ readings were obtained 20 min after the electrodes were applied. Arterial $O_2$ and $CO_2$ were measured at 37° C (Instrumentation Laboratory Model 1301®) and were not corrected to the patient's temperature. Additional arterial samples were obtained at least every two hours or as clinically indicated. Simul-

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‡ Loaned by manufacturer, Novametrix Medical Systems, Inc., Wallingford, CT.

taneous arterial pressure, $Ptc_{O_2}$, $Ptc_{CO_2}$, $PET_{CO_2}$, $FET_{ISO}$ and temperature were tabulated with each arterial blood gas. When the Doppler ultrasound or TEE indicated the presence of intravascular air, the $PET_{CO_2}$, $Ptc_{O_2}$, $Ptc_{CO_2}$, and arterial pressure were closely monitored and the temporal relationship of any changes in these variables were noted. The hard-copy recordings of $PET_{CO_2}$, $Ptc_{O_2}$, and $Ptc_{CO_2}$ were reviewed postoperatively to verify the temporal relationship. The $O_2$ index ($Ptc_{O_2}/Pa_{O_2}$) and the $CO_2$ index ($Ptc_{CO_2}/Pa_{CO_2}$) were calculated.

The accuracy of $Ptc_{CO_2}$ and $PET_{CO_2}$ in reflecting $Pa_{CO_2}$ was evaluated by correcting $Ptc_{CO_2}$ and $PET_{CO_2}$ values by the baseline $CO_2$ index and $Pa_{CO_2}$–$PET_{CO_2}$ difference, respectively. Each patient's baseline $CO_2$ index was calculated from the first arterial blood gas sample, and subsequent $Ptc_{CO_2}$ values were corrected by dividing by the baseline $CO_2$ index. Likewise, the baseline $Pa_{CO_2}$–$PET_{CO_2}$ difference was calculated from the first arterial blood gas sample, and the difference was added to subsequent $PET_{CO_2}$ values. Six arterial samples obtained during an air embolism episode were excluded. There were 35 samples with $Pa_{CO_2}$ less than 35 mmHg (hypocarbia), 21 samples with $Pa_{CO_2}$ 35–45 mmHg (normocarbia), and four samples with $Pa_{CO_2}$ greater than 44 mmHg (hypercarbia).

The relationship between transcutaneous and arterial $O_2$ and $CO_2$ were analyzed by simple linear regression and correlation analysis.[11] The relationship between a change in arterial $O_2$ or $CO_2$ and the accompanying change in transcutaneous $O_2$ or $CO_2$ was also analyzed with simple linear regression and correlation analysis. The effects of age, sex, electrode location, and $FET_{ISO}$ were analyzed by one-way analysis of variance. The difference between $Ptc_{CO_2}$ and $PET_{CO_2}$ in correctly identifying hypocarbia, normocarbia, or hypercarbia was tested with chi-square analysis. A $P$ value of $\leq 0.05$ was considered significant. Means ± SD are given.

## Results

There was a significant correlation between $Ptc_{O_2}$ and $Pa_{O_2}$ (fig. 1). The $O_2$ index ($Ptc_{O_2}/Pa_{O_2}$) was 0.80 ± 0.15. The $O_2$ index was significantly higher in females (table 1), while electrode location and $FET_{ISO}$ did not significantly affect the $O_2$ index. There was a significant correlation between changes in $Pa_{O_2}$ and the accompanying change in $Ptc_{O_2}$ (fig. 2).

The first 24 patients were monitored with a $Ptc_{CO_2}$ unit that was markedly affected by electrocautery. Each electrocautery use distorted the $Ptc_{CO_2}$ reading for 90–120 s, and over a period of 2–3 h marked drifting of the $Ptc_{CO_2}$ calibration occurred. The next 34 patients (98 arterial blood gas samples) were monitored with a $Ptc_{CO_2}$ unit adapted to shield against the effect of electrocautery.

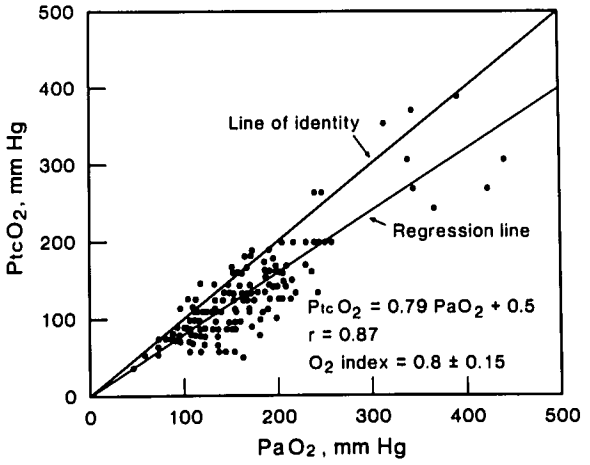


FIG. 1. Regression analysis of transcutaneous oxygen tension ($Ptc_{O_2}$) *versus* arterial oxygen tension ($Pa_{O_2}$). $O_2$ index is the $Ptc_{O_2}/Pa_{O_2}$ ratio.

The $Ptc_{CO_2}$ data from these 34 patients are presented. There was a significant positive correlation between $Ptc_{CO_2}$ and $Pa_{CO_2}$ (fig. 3). The mean $CO_2$ index ($Ptc_{CO_2}/Pa_{CO_2}$) was 1.58 ± 0.2. The $CO_2$ index was not significantly affected by patient's sex, electrode application site, or $FET_{ISO}$ (table 1). There was no significant difference between baseline $CO_2$ index and the $CO_2$ index after 3 to 4 h of use. There was a significant correlation between a change in $Pa_{CO_2}$ and the accompanying change in $Ptc_{CO_2}$ (figure 4).

There were 17 episodes of intravascular air embolism in eight patients in the sitting position. The diagnosis of air embolism was made by either an audible change in frequency on the Doppler ultrasound or the visualization of air in the cardiac chambers on TEE. $Ptc_{O_2}$ decreased in each episode. In 13 episodes, when the fractional inspired oxygen concentration ($FI_{O_2}$) remained constant, the

TABLE 1. Effect of Electrode Location, Patient's Sex, and Isoflurane Concentration on $O_2$ and $CO_2$ Index

| | $O_2$ Index* | $CO_2$ Index* |
|---|---|---|
| Electrode location | | |
| chest | 0.80 ± 0.15 (133) | 1.59 ± 0.21 (75) |
| forearm | 0.80 ± 0.19 (8) | 1.48 ± 0.08 (5) |
| costophrenic angle | 0.77 ± 0.16 (38) | 1.56 ± 0.15 (18) |
| Sex | | |
| male | 0.77 ± 0.15 (130) | 1.60 ± 0.18 (72) |
| female | 0.86 ± 0.14† (49) | 1.53 ± 0.22 (26) |
| End-tidal isoflurane (%) | | |
| <1 | 0.81 ± 0.18 (91) | 1.46 ± 0.28 (11) |
| 1–2 | 0.76 ± 0.12 (32) | 1.55 ± 0.18 (23) |
| 2–4 | 0.77 ± 0.14 (57) | 1.59 ± 0.21 (23) |
| >4 | 0.82 ± 0.17 (69) | 1.63 ± 0.13 (30) |

* Number of observations in parentheses.
† $P < 0.05$ by one-way analysis of variance.

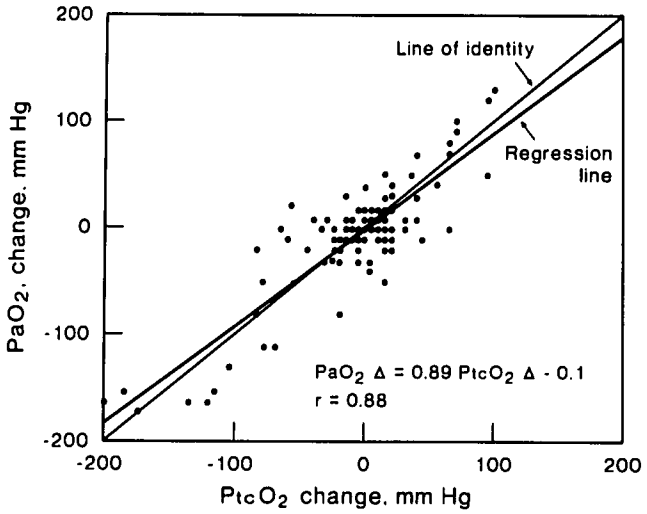


FIG. 2. Regression analysis of a change in arterial oxygen tension ($Pa_{O_2}$) *versus* the simultaneous change in transcutaneous oxygen tension ($Ptc_{O_2}$).

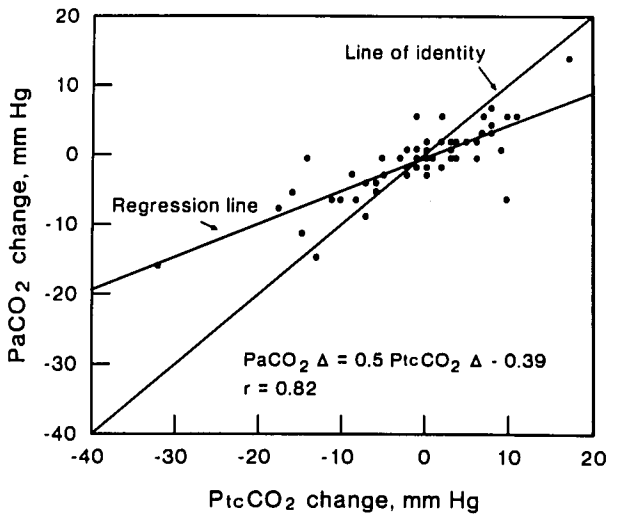


FIG. 4. Regression analysis of a change in arterial carbon dioxide ($Pa_{CO_2}$) *versus* the simultaneous change in transcutaneous carbon dioxide tension ($Ptc_{CO_2}$).

mean decrease in $Ptc_{O_2}$ was 48 ± 35 mmHg (range: 12–123 mmHg). $FI_{O_2}$ in these 13 episodes was 1.0 in ten and 0.5 in three. Maintenance of the nitrous oxide at 50 per cent in these three patients was considered clinically appropriate based on vital signs and the clinical judgement of those responsible for clinical care. The $FI_{O_2}$ was increased at the time of diagnosis of air embolism in four episodes and the decrease in $Ptc_{O_2}$ was 17 ± 14 mmHg (range: 4–37 mmHg). In only ten episodes was there a change in $PET_{CO_2}$ with a mean decrease of 6 ± 3 mmHg (range: 2–11 mmHg). In nine of the ten episodes the decrease in $Ptc_{O_2}$ preceded the change in $PET_{CO_2}$ by 12 to 355 s. There was an increase in $Ptc_{CO_2}$ of 10 ± 6 mmHg (range: 2–18 mmHg) in ten episodes. The change in $PET_{CO_2}$ consistently preceded the increase in $Ptc_{CO_2}$.

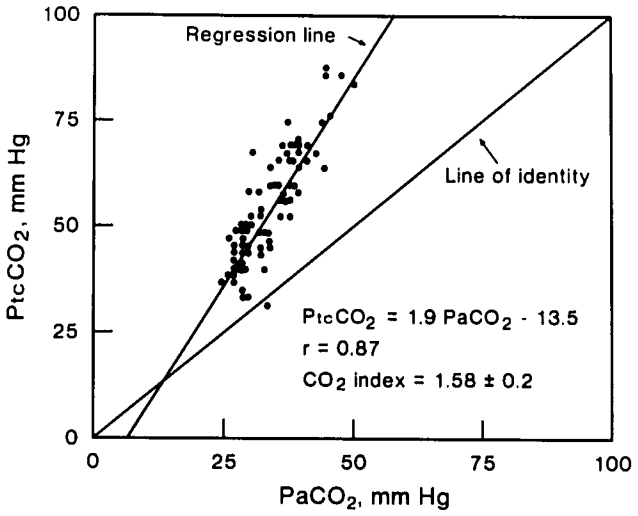


FIG. 3. Regression analysis of transcutaneous carbon dioxide ($Ptc_{CO_2}$). *versus* arterial carbon dioxide tension ($Pa_{CO_2}$).

The $Ptc_{O_2}$ decreased below 75 mmHg during five air embolism episodes (two patients). The low $Ptc_{O_2}$ was confirmed by arterial blood gas samples in four of these episodes. A total of six arterial blood gas samples were obtained during air embolism (table 2). The mean-$O_2$ index was 0.94 ± 0.12 and mean-$CO_2$ index was 1.63 ± 0.04. Correlation between transcutaneous and arterial $O_2$ and $CO_2$ was excellent (r = 0.93 for $O_2$, r = 0.99 for $CO_2$). The mean $Pa_{CO_2}$–$PET_{CO_2}$ difference of these six samples was 27 ± 4 mmHg.

$Ptc_{CO_2}$ correctly identified hypocarbia, normocarbia, or hypercarbia in 92% of the observations. The mass spectrometer $PET_{CO_2}$ correctly identified $Pa_{CO_2}$ in 82% of the observations. This difference was not statistically significant.

## Discussion

Venous air embolism commonly results in altered pulmonary function characterized by an increase in physiologic dead space.[7] During constant volume ventilation $PET_{CO_2}$ decreases concomitantly with the increased physiologic dead space, venous admixture, and continuous $PET_{CO_2}$ monitoring is a sensitive monitor for detection of air embolism.[8] Several studies in sheep and dogs have shown that a decrease in $Pa_{O_2}$ accompanies pulmonary embolism.[8,9,12–16] As little as 0.03 $ml \cdot kg^{-1} \cdot min^{-1}$ of air produced a decrease in arterial oxygenation in sheep.[14] A 0.25 $ml \cdot kg^{-1}$ bolus of air in dogs produced a 10 mmHg decrease in $Pa_{O_2}$ and a 2 $ml \cdot kg^{-1}$ bolus of air produced an 84-mmHg decrease.[8]

Our data indicate $Pa_{O_2}$ decreases early in the course of air embolism preceding the decrease in measured $PET_{CO_2}$ in the majority of patients. The $Ptc_{O_2}$ decrease accurately reflects the decrease in $Pa_{O_2}$. The alterations in pulmonary gas exchange during air embolism can be explained on the basis of a partial pulmonary vascular obstruction. Wagner *et al.* demonstrated the development of areas with high ventilation perfusion ($\dot{V}_A/\dot{Q}$) ratios during pulmonary embolism.[10] Simultaneously, the areas not obstructed receive increased blood flow but less ventilation. As a consequence their $\dot{V}_A/\dot{Q}$ ratios decrease, contributing to the decrease in $Pa_{O_2}$. In more severe episodes of air embolism the opening of intrapulmonary arteriovenous shunts have been demonstrated in dogs and sheep, contributing to hypoxemia.[14,17] Similar shunts have been postulated to occur in humans.[15,18]

The high inspired oxygen tension used during most neurosurgical procedures often prevents extremely low $Pa_{O_2}$. However, severe hypoxemia from air embolization during a neurosurgical procedure with the patient in the sitting position has been reported.[19] Marked hypoxemia occurred in two of our eight patients during air embolization in spite of 100% inspired oxygen concentration.

Defining the temporal relationship between air embolism and $Pa_{O_2}$ has been hampered by the inability to monitor $Pa_{O_2}$ continuously. Changes in $Pa_{O_2}$ have been detected within 1–2 min after a bolus injection of air.[8] To our knowledge, the temporal relationship between $Pa_{O_2}$ and $PET_{CO_2}$ during air embolism has not been reported.

$Ptc_{O_2}$ has previously been shown to correlate well with simultaneously measured $Pa_{O_2}$ during anesthesia[1,3] and the data presented here again demonstrate this strong correlation. A positive correlation between $Ptc_{O_2}$ and $Pa_{O_2}$ suggests $Ptc_{O_2}$ is a valid trend monitor of $Pa_{O_2}$, while the excellent correlation between a given change in $Pa_{O_2}$ with the simultaneous change in $Ptc_{O_2}$ does demonstrate that $Ptc_{O_2}$ reliably follows changes in $Pa_{O_2}$.

Evaluation of $Ptc_{CO_2}$ monitoring in adults has lead to varying results. Rafferty *et al.*[4] found $Ptc_{CO_2}$ to be a reliable trend monitor during anesthesia. Schachter *et al.*[20] found $Ptc_{CO_2}$ not to be useful clinically to monitor $Pa_{CO_2}$ in adults in an intensive care unit setting. The contradictory findings may be explained by the temperature of the $Ptc_{CO_2}$ electrode. Rafferty *et al.* used a heated electrode while Schachter *et al.* used a nonheated electrode. Our data indicate that $Ptc_{CO_2}$ (electrode temperature of 44° C) is a reliable and accurate monitor of $Pa_{CO_2}$ trends in hemodynamically stable anesthetized adult patients. $Ptc_{CO_2}$ should not be used to predict absolute values of $Pa_{CO_2}$. However, in our study, $Ptc_{CO_2}$ was reliable in identifying hypocarbia, normocarbia, and hypercarbia. Raemer *et al.*[21] have shown that estimation of $Pa_{CO_2}$ from $PET_{CO_2}$ during anesthesia is not always reliable. A redistribution of $\dot{V}_A/\dot{Q}$ can occur from temperature changes, hypotension, anesthetic agents, changes in pulmonary blood flow, surgical positions, and mechanical ventilation. This redistribution of $\dot{V}_A/\dot{Q}$ will produce changes in physiologic dead space with accompanying changes in $Pa_{CO_2}$–$PET_{CO_2}$ difference. In contrast, the relationship between $Pa_{CO_2}$ and $Ptc_{CO_2}$ ($CO_2$ index) remains reasonably stable in a hemodynamically stable patient. For these reasons $Ptc_{CO_2}$ is more reliable than $PET_{CO_2}$ in estimating $Pa_{CO_2}$ under certain clinical conditions. $Ptc_{CO_2}$ accurately reflected the increasing $Pa_{CO_2}$ during air embolism. The changes in $Ptc_{CO_2}$ lag behind the changes in $PET_{CO_2}$ by 1–2 min, therefore limiting $Ptc_{CO_2}$ use as an early detector of air embolism.

TABLE 2. Simultaneous Transcutaneous and Arterial $O_2$ and $CO_2$ (mmHg) during Air Embolism Episode

| $Ptc_{O_2}$ | | $Pa_{O_2}$ | $Ptc_{CO_2}$ | | $Pa_{CO_2}$ |
|---|---|---|---|---|---|
| Prior | During | During | Prior | During | During |
| 236 | 140 | 199 | 40 | 53 | 34 |
| 260 | 154 | 152 | 54 | 69 | 42 |
| 94 | 57 | 59 | 57 | 65 | 40 |
| 144 | 68 | 67 | 58 | 66 | 40 |
| 114 | 63 | 65 | 68 | 76 | 46 |
| 132 | 74 | 75 | 72 | 83 | 50 |

Transcutaneous monitoring was not difficult to use in the clinical setting. Calibration of the units could be performed while preparing the anesthesia machine and cart for the case and could be completed in 5–8 min. The electrode membranes required changing every 5–7 days. The electrodes were easily applied to the patient's skin. The Novametrix® equipment provided stable values for up to 6 hours and were not significantly affected by electrocautery. Other $Ptc_{CO_2}$ units which we have clinically tested, were significantly affected by electrocautery and were not acceptable for use in the operating room. Anesthetic gases ($N_2O$ and isoflurane) did not interfere with the electrode function.

$Ptc_{O_2}$ and $Ptc_{CO_2}$ are tissue-perfusion-dependent. A decrease in tissue perfusion (decreased cardiac output, occlusion to blood flow, or vasoconstriction) will decrease $Ptc_{O_2}$ and increase $Ptc_{CO_2}$, simulating the changes that occur during air embolism. Changes occurring in transcutaneous values must be interpreted in light of the entire clinical picture to prevent false positive diagnosis of air embolism. Increasing the $FI_{O_2}$ while discontinuing $N_2O$ when air embolism is suspected will likely lessen the maximal decrease in $Ptc_{O_2}$ but did not prevent an initial decrease in $Ptc_{O_2}$.

The ideal monitor to detect air embolism does not exist. The most sensitive detectors of air embolization are TEE or Doppler ultrasound. Less sensitive but still providing a back-up means of diagnosing air embolism when TEE

or Doppler ultrasound is equivocal include $Ptc_{O_2}$, pulmonary artery pressure (PAP), and $PET_{CO_2}$. This latter group will quantitate the size of the air embolism, identify significant physiologic changes, and provide information regarding the disappearance of air. A combination of TEE or Doppler ultrasound with PAP, $PET_{CO_2}$, or $Ptc_{O_2}$ will provide near-optimal monitoring for the detection of air embolism.

In conclusion, $Ptc_{O_2}$ decreases early during venous air embolism, reflecting a decrease in $Pa_{O_2}$. $Ptc_{O_2}$ monitoring is useful in detecting venous air embolism and may change earlier than $PET_{CO_2}$. In addition, $Ptc_{O_2}$ provides accurate and reliable assessment of a patient's oxygenation during anesthesia. $Ptc_{CO_2}$ is an accurate and reliable monitor of $Pa_{CO_2}$ trends in hemodynamically stable, anesthetized adult patients and can reliably reflect hypocarbia, normocarbia, and hypercarbia, making it particularly useful in neurosurgical procedures.

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