Pulsus Alternans during General Anesthesia with Halothane

Effects of Permissive Hypercapnia

Mahmood Saghaei, M.D.*, Mojtaba Mortazavian, M.D.†

Background: Pulsus alternans is a classic type of abnormal pulse. It can be defined as a regular alternation of pulse amplitude in which runs of weak and strong beats follow each other alternatively without any change in cycle length. It may be a sign of severe decompensated congestive heart failure. The authors infrequently encountered some cases of pulsus alternans during halothane anesthesia with spontaneous respiration in otherwise normal subjects in association with high levels of end-tidal carbon dioxide. This study was conducted to determine if there is any relation between this phenomena and hypercapnia.

Methods: One hundred twenty patients undergoing elective lower extremity surgery were selected. Halothane was used for maintenance of anesthesia, and the patients were allowed to breathe spontaneously. The occurrence of pulsus alternans was determined by plethysmographic display of pulse wave and then confirmed by palpation of the radial artery.

Results: Ten patients (8.3%) developed pulsus alternans together with elevated levels of end-tidal carbon dioxide (57 ± 4 mmHg vs. 41 ± 4 mmHg in patients without pulsus alternans [mean ± SD]). The pulsus alternans disappeared after switching to controlled ventilation and 15–20% reduction in end-tidal carbon dioxide. During the period of pulsus alternans, vital signs and electrocardiography remained within normal limits.

Conclusions: There may be some relation between occurrence of pulsus alternans and hypercapnia during halothane anesthesia. Pulsus alternans occurs in a small fraction of spontaneously breathing, halothane-anesthetized patients. Although hypercapnia is clearly a factor, the mechanism of this phenomenon is unknown. (Key words: Carbon dioxide; plethysmography; pulse oximetry; respiratory acidosis; spontaneous respiration.)

* Assistant Professor.
† Resident of Anesthesiology.

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Address reprint requests to Dr. Saghaei: PO Box 941, Al-Zahra Medical Center, Esfahan, Iran. Address electronic mail to: msaqaei@noornet.net

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pulse oxymetric hemoglobin saturation (SpO₂) lower than 94% on air were also excluded. No premedication was given. On arrival in the operating room, the patient’s electrocardiogram (ECG) was monitored continuously with central subclavicular lead (CS₃) attachment using lead I and II alternatively. Arterial blood pressure was measured with an automated noninvasive device (Cardiocap; Datex/Division of Instrument Corp., Helsinki, Finland). Pulse oxymetric monitoring of SpO₂ was performed using a device capable of plethysmographic display of pulse volume curve (Cardiocap). Side stream ET₇CO₂ monitoring with sampling port near the proximal end of endotracheal tube was performed throughout the procedure (Cardiocap). Anesthesia was induced with thiopentone 5 mg/kg, morphine 0.1 mg/kg, succinylcholine 1.5 mg/kg followed by tracheal intubation, and controlled and/or assisted ventilation until restoration of spontaneous respiration. The patients were then allowed to breathe spontaneously.

A mixture of halothane 1–2% and N₂O 50% in oxygen through a Bain circuit with a total fresh gas flow of 7 l/min was used for maintenance of anesthesia, so that end-tidal halothane and N₂O concentrations of 1.4–1.6% and 48–52%, respectively, were obtained (Cardiocap). The plethysmographic pulse display was examined visually throughout the procedure to detect any sign of PA that persisted for at least 30 s.

The presence of PA was verified by finger palpation of radial artery. Patients who developed dysrhythmias associated with PA (other than sinus tachycardia) as evidenced by ECG monitoring were excluded from the study. The highest ET₇CO₂ in every 15-min interval was recorded throughout the procedure together with its corresponding vital signs and SpO₂. After termination of the procedure, the maximum recorded ET₇CO₂ and its associated vital signs were selected as representative measurements of the patient. If the patient developed PA, then the ET₇CO₂ at that moment was selected. A fresh vital sign and SpO₂ record was taken after documenting the presence of PA (at 30 s after appearance of PA), and then controlled ventilation was started after administering 0.3 mg/kg atracurium. The level of ET₇CO₂ at which alternation disappeared was recorded.

### Statistical Analysis

Frequency distribution of patients with and without PA was calculated according to their ET₇CO₂ level. Mean ET₇CO₂, age, systolic and diastolic blood pressure, heart and respiratory rate, and SpO₂ for the two groups were determined and compared using the Student t test. The relation between age and ET₇CO₂ in the two groups was examined with linear regression analysis. P < 0.05 was considered significant.

### Results

None of the patients developed significant rebreathing as demonstrated by inspiratory concentrations of carbon dioxide near zero. The end-tidal halothane concentration was 1.4–1.6% in all patients. Onset of PA occurred 15–35 min after induction of anesthesia (four cases before and six cases after surgical stimulation). No significant differences in respiratory and heart rate, blood pressure, and SpO₂ were found between the two groups (table 1). Patients in the PA group were significantly more hypercapnic than those in the non-PA group. The mean age of patients in the PA group was also significantly higher than that of those in the non-PA group (table 1). Thirty-five patients (29%) developed moderate to severe hypercapnia (ET₇CO₂ > 44 mmHg), and 10

<table>
<thead>
<tr>
<th>Table 1. Age and Cardiorespiratory Parameters in Two Groups</th>
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<tbody>
<tr>
<td>Alternans</td>
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<td>-----------</td>
</tr>
<tr>
<td>Mean ± SD</td>
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<tr>
<td>Age (yr)*</td>
</tr>
<tr>
<td>ET₇CO₂ (mmHg)*</td>
</tr>
<tr>
<td>Heart rate (/min)</td>
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<tr>
<td>Respiratory rate (/min)</td>
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<tr>
<td>Systolic blood pressure (mmHg)</td>
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<tr>
<td>Diastolic blood pressure (mmHg)</td>
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<td>Pulse oxymetric saturation (%)</td>
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*P < 0.001.

ET₇CO₂ = end-tidal carbon dioxide.
(28.5%) of these hypercapnic patients (8.3% of total) showed alternating pulse (fig. 1 and table 2). Finger plethysmography showed an abrupt occurrence of PA in all patients in the PA group. The degree of alternation in pulse wave amplitude estimated visually was approximately 50%, i.e., the amplitude ratio of two adjacent pulse waves was approximately 0.5. Finger palpation of the radial artery confirmed alternation in pulse. No evidence of electrical alternans was detected in ECG monitoring. Careful visual examination of ECG monitoring did not show any pathologic changes in P wave morphology or any kind of dysrhythmia. ST segment and T wave pattern were normal before and after the event. Auscultation of the lungs and heart was normal (no rales, S₃, and/or S₄). No abnormal respiratory movement (e.g., inspiratory retractions, active expiration) was found before or after the onset of PA. There was no relationship between respiratory phases and alternans pattern in plethysmographic display (heart rate to respiratory rate ratio ≥ 2.5). The alternation in pulse disappeared completely after establishment of controlled ventilation, when a 15–20% reduction in ETCO₂ occurred, which took approximately 51 ± 2 s (mean ± SD; range, 46–55 s, not correlated with ETCO₂). The total duration of PA was 91 ± 3 s (range, 84–98 s). The ETCO₂ at the time of PA disappearance was 47 ± 1.9 mmHg (range, 44–51 mmHg) and significantly correlated with ETCO₂ at PA appearance (r = 0.95, P < 0.001). There was also some hysteresis in the ETCO₂ at onset and offset of PA. Unlike a significant positive correlation between ETCO₂ and age in the non-PA group, there was a significant negative correlation between threshold ETCO₂ for development of PA and the age in the PA group; older patients had a lower threshold ETCO₂ (fig. 2). The lowest ETCO₂ at appearance of PA was 52 mmHg in a 71-yr-old man, and the highest ETCO₂ in the non-PA group was 50 mmHg in two women 74 and 85 yr of age.

**Discussion**

Respiratory acidosis has a series of well-known cardiovascular consequences, especially during halothane anesthesia. Hypercapnia can cause direct depression of cardiac muscle, but at the same time it causes reflex sympathetic stimulation. In addition, increases in cardiac output in response to hypercapnia tend to be minimal during halothane anesthesia. In this study, a possible effect of hypercapnia on myocardial contractility was examined, which is the alternation in pulse amplitude. Although a very strong association between hypercapnia and development of PA has been shown in this study, it cannot be said that this relationship is a cause-and-effect one. Both may be the result of a coincidental factor such as certain patterns of autonomic nervous system activities that may be inherent in anesthesia with spontaneous respiration. This relationship has not been investigated and reported in vivo. McCall and Orchard have shown that hypercapnia caused mechanical alternans in isolated

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**Table 2. Frequency Distribution of the Patients between Two Groups**

<table>
<thead>
<tr>
<th>ETCO₂ &gt; 44 (mmHg)</th>
<th>ETCO₂ ≤ 44 (mmHg)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alternans</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Nonalternans</td>
<td>25</td>
<td>85</td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>85</td>
</tr>
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</table>

* P < 0.001 (Fisher exact test).

ETCO₂ = end-tidal carbon dioxide.
A special and possibly new use of pulse oximetry has been demonstrated in this study, i.e., monitoring of consecutive plethysmographic pulse waves with respect to the equality of neighboring waves' amplitude. It is evident that this device can be used to diagnose PA, although its sensitivity and specificity have not yet been determined.

In conclusion, in addition to the classic use of pulse oximetry for monitoring arterial oxygenation, there may be a role for it to detect high levels of hypercapnia during general anesthesia, especially with spontaneous respiration.

References