different mechanisms by which these drugs increase the blood pressure. PH is a selective alpha-adrenergic agonist that increases the blood pressure by increasing the systemic vascular resistance (SVR). Prior to CPB, the vasopressor effect of phenylephrine is usually accompanied by an increased afterload and by reflex bradycardia, which can decrease the cardiac output (CO). Since the arterial blood pressure is the product of CO X SVR, the decreased CO can partially counteract the effect of phenylephrine on the SVR. On the other hand, during CPB + AXC, the heart is excluded from the circulation and the pump flow can be maintained at a constant level, and hence the action of phenylephrine on SVR is not counteracted. In contrast to phenylephrine, which is a selective alpha-adrenergic agonist, a vasopressor acting as mixed beta- and alpha-adrenergic agonist, such as ephedrine or epinephrine, can increase both the CO and SVR. The resulting pressor response is due to vasoconstriction but mainly to cardiac stimulation, and hence a greater response may result during the prebypass period than during bypass.

It may be concluded that vasopressors which increase the blood pressure by a predominant increase of SVR will be more effective during CPB, while those acting by a predominant increase of CO will be more effective during the prebypass period.

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Pulse Oximetry is Accurate in Patients with Dysrhythmias and a Pulse Deficit

To the Editor—Some authors have recommended1 or used2-4 the agreement of the pulse oximeter pulse rate with the EKG heart rate as a criterion for arterial oxygen saturation (SpO2) reliability. Since the pulse rate of patients with arrhythmias may not match their heart rate, we wondered if pulse deficits truly predict SpO2 reliability.

Following approval from the institutional review board, we studied 163 consecutive surgical Intensive Care Unit patients with pre-existing arterial catheters. We measured SpO2 with an Ohmeda 3700, software version M (Ohmeda, Boulder, CO), and followed the manufacturer's recommendations for ensuring a reliable SpO2 (a stable SpO2, a signal strength greater than 5 pixels, and at least three consecutive pulsatile waveforms). During SpO2 and oximeter pulse rate recording, an arterial blood sample was withdrawn and immediately analyzed for 5%HbO2 and carboxyhemoglobin (COHb) with an IL 282 Co-Oximeter (Instrumentation Labs, Lexington, MA). EKG heart rate and rhythm were obtained from a simultaneous 1-min EKG rhythm strip recording.

We defined normal sinus rhythm (NSR) as an EKG rhythm with regular p-waves, QRS complexes, and <5 ectopic beats per minute. We defined non-NSR as an irregularly irregular rhythm or >10 ectopic beats per minute. We defined the pulse deficit (PD) as the EKG heart rate less the oximeter pulse rate. The mean (SpO2-%HbO2) difference (bias) was calculated for the NSR, non-NSR, PD, and no PD groups.4 The different groups were compared by unpaired t test.

Of 163 patients, 24 had PD > 3 (nine were NSR and 15 were non-NSR) and 139 did not. In ten patients, the EKG rhythm changed during their ICU stay and they entered both NSR and non-NSR groups. Paired t test of these patients showed no difference between the two groups (P > 0.45; power > 0.30). Eight patients had 5–10 ectopic beats per minute and were excluded from EKG rhythm comparison, leaving 134 NSR patients and 51 non-NSR patients. Other statistics are summarized in table 1. Our bias results for all groups are similar to those previously reported with the Ohmeda 3700 in normal volunteers5 and in hospital patients.6 There was no relationship between the pulse deficit and (SpO2-%HbO2).

A PD > 3 does not necessarily mean the pulse oximeter is unreliable. True pulse deficits may exist because irregular or premature myocardial electrical depolarizations do not always produce a peripheral pulse. Since the PD is the EKG heart rate less the oximeter pulse rate, different oximeter pulse rate and EKG heart rate machine algorithms may cause artifactual pulse deficits.

SpO2 is as accurate in non-NSR patients and patients with pulse deficits as in NSR patients when SpO2 is stable, the signal strength is greater than 5 pixels, and at least three consecutive pulsatile plethysmograms are noted. When evaluating SpO2 reliability, a noise-free pulsatile plethysmogram is more important than the absence of a PD.

REFERENCES


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### Table 1. Effect of Cardiac Rhythm and Pulse Deficit on Pulse Oximetry Accuracy

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>$\text{SpO}_2$</th>
<th>$\text{SpO}_2 (% \text{HbO}_2 + % \text{HbCO})$</th>
<th>$P$</th>
<th>Power$^\dagger$</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSR group</td>
<td>134</td>
<td>0.4 ± 2.1</td>
<td>-2.7 ± 2.1</td>
<td>&gt;.60</td>
<td>&gt;.95</td>
</tr>
<tr>
<td>Non-NSR group</td>
<td>31</td>
<td>0.5 ± 2.0</td>
<td>-2.5 ± 2.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No pulse deficit group</td>
<td>139</td>
<td>0.8 ± 1.9</td>
<td>-2.8 ± 2.1</td>
<td>&gt;.05</td>
<td>&gt;.95</td>
</tr>
<tr>
<td>Pulse deficit &gt; 3 group</td>
<td>24</td>
<td>0.3 ± 2.0</td>
<td>-2.2 ± 1.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Mean ± SD.
† Two wavelength pulse oximeters recognize COHb mostly as %HbCO. Adjusting for COHb did not alter the statistical significance. The mean COHb was 3.0 ± 0.8%.
‡ 1 − power = probability of a type II statistical error.

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**References**


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**A Circle System is Best when Anesthetizing Malignant Hyperthermia Susceptible Patients**

To the Editor—I read with interest the correspondence from Donahue and Schulz, recommending a partial rebreathing system for malignant hyperthermia-susceptible (MHS) patients. They used this system to anesthetize a child, “rather than cancel the case or purge a machine,” as was recently described by Beebe and Sessler. They also recommend it as a standby system for pregnant MHS patients. While this system may perform adequately when anesthesia is uneventful, if an MH crisis occurs, this system might be unable to maintain normocarbia, at least in adult patients. There is no CO$_2$ absorber, and the fresh gas flow rate of the system is limited to 15–18 l min$^{-1}$ by the N$_2$O/O$_2$ blender (personal communication, Bird Corp., Palm Springs, CA).

Rogers et al.$^3$ have described a case of a 45-kg female patient who developed intraoperative MH while being anesthetized using a Bain circuit. The end-tidal CO$_2$ concentration could not be controlled until the Bain circuit was replaced with a circle system with a fresh CO$_2$ absorber. The authors recommended the circle system as the circuit of choice for MHS patients.

Although it is important to avoid volatile anesthetic agents in MHS patients, MH can occur during so-called "nontriggering" general anesthesia.$^{4,5}$ Therefore, it is prudent to begin a case completely prepared to treat an MH crisis, including a "clean" machine with a CO$_2$ absorber, than to hurriedly try to obtain one once a reaction has begun.

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