different mechanisms by which these drugs increase the blood pressure. PH is a selective alpha$_1$-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 × 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$_1$-adrenergic agonist, a vasopressor acting as mixed beta- and alpha-adrenergic agonist, such as epinephrine or ephedrine, can increase both the CO and SVR. The resulting pressor response is due to vasoconstriction but mainly to cardiac stimulation,[7] 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.

ANIS BARAKA, M.D.
Professor and Chairman
Department of Anesthesiology
American University of Beirut
Beirut, Lebanon

REFERENCES

1. Massagee JT, McIntyre RW, Kates RA, Reves JG, Bai S: Effects of preoperative calcium entry blocker therapy on α-adrenergic responsiveness in patients undergoing coronary revascularization. ANESTHESIOLOGY 67:485–491, 1987
2. Schwinn DA, McIntyre RW, Hawkins ED, Kates RA, Reves JG: α_1-Adrenergic responsiveness during coronary artery bypass surgery: Effect of preoperative ejection fraction. ANESTHESIOLOGY 69:206–217, 1988
3. Baraka A, Haroun S, Baroody M, Nawfal M: Effects of ephedrine on resistance and capacitance vessels in humans. Anesth Analg 67:482–483, 1988
4. Robicsek F, Masters TN, Niesluchowski W, Yeager JC, Duncan GD: Vasomotor activity during cardiopulmonary bypass, Pathophysiology and Techniques of Cardiopulmonary Bypass, Vol II. Edited by Utley JR. Baltimore, Williams & Wilkins, 1983, pp 1–12
5. Reves JG, Karp RB, Buttner EE, Tosone S, Smith LR, Samuelson PN, Kreush GR, Oparil S: Neuronal and adrenomedullary catecholamine release in response to cardiopulmonary bypass in man. Circulation 66:49–55, 1982
6. Benazon D: Hypothermia, Scientific Foundation of Anaesthesia, 2nd edition. Edited by Scurr C, Feldman S. Chicago, Year Book Medical Publishers, Inc., 1974, pp 344–355
7. Innes JR, Nickerson M: Norepinephrine, epinephrine, and the sympathomimetic amines, The Pharmacological Basis of Therapeutics, 5th edition. Edited by Goodman LS, Gilman A. New York, MacMillan Publishing Co., Inc., 1975, pp 477–513

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

To the Editor:—Some authors have recommended[1]* or used [2,3] the agreement of the pulse oximeter pulse rate with the EKG heart rate as a criterion for arterial oxygen saturation (SpO_2) reliability. Since the pulse rate of patients with arrhythmias may not match their heart rate, we wondered if pulse deficits truly predict SpO_2 reliability.

Following approval from the institutional review board, we studied 163 consecutive surgical Intensive Care Unit patients with pre-existing arterial catheters. We measured SpO_2 with an Ohmeda 3700, software version M (Ohmeda, Boulder, CO), and followed the manufacturer's recommendations for ensuring a reliable SpO_2 (a stable SpO_2, a signal strength greater than 5 pixels, and at least three consecutive pulsatile waveforms). During SpO_2 and oximeter pulse rate recording, an arterial blood sample was withdrawn and immediately analyzed for $\%HbO_2$ 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 (SpO_2-$\%HbO_2$) 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 31 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 volunteers[5,6] and in hospital patients.[7] There was no relationship between the pulse deficit and (SpO_2-$\%HbO_2$).

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.

SpO_2 is as accurate in non-NSR patients and patients with pulse deficits as in NSR patients when SpO_2 is stable, the signal strength is greater than 5 pixels, and at least three consecutive pulsatile plethysmograms are noted. When evaluating SpO_2 reliability, a noise-free pulsatile plethysmogram is more important than the absence of a PD.

* Miyasaka K, Katayama M, Kusakawa I, Ohata J, Kawano T, Honma Y: Use of Pulse Oximetry in Neonatal Anesthesia. J Perinatology 7: 343–345, 1987.

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

	n	SpO_2-%HbO_2*	SpO_2-(%HbO_2 + %HbCO)†	P	Power‡
NSR group	134	0.4 ± 2.1	−2.7 ± 2.1	>.60	>.95
Non-NSR group	31	0.5 ± 2.0	−2.5 ± 2.4		
No pulse deficit group	139	0.8 ± 1.9	−2.8 ± 2.1	>.05	>.95
Pulse deficit > 3 group	24	0.3 ± 2.0	−2.2 ± 1.9		

* Mean ± SD.

† Two wavelength pulse oximeters recognize COHb mostly as %HbO_2.[8] 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.

DAVID H. WONG, PHARM.D, M.D.
Assistant Clinical Professor
Department of Anesthesiology
University of California, Irvine
Chief, SICU—VA Medical Center

KEVIN K. TREMPER, PH.D., M.D.
Associate Professor and Chairman
Department of Anesthesiology
University of California, Irvine

JONATHAN DAVIDSON, M.D.
Medical Student on Elective
Department of Anesthesiology
University of California, Irvine

JUNE ZACCARI, B.S.
Research Associate
Department of Anesthesiology
University of California, Irvine

PAUL WEIDOFF, PH.D., M.D.
Fellow
Department of Anesthesiology
University of California, Irvine

STEVE WILBUR, D.D.S., M.D.
Assistant Clinical Professor
Department of Anesthesiology
University of California, Irvine

EDWARD A. STEMMER, M.D.
Professor
Department of Surgery
University of California, Irvine
Chief
Department of Surgery
VA Medical Center

University of California, Irvine and
Long Beach Veterans Administration Medical Center
Long Beach, California 92668

REFERENCES

1. Wukitsch MW, Petterson MT, Tobler DR, Pologe JA: Pulse oximetry. An analysis of theory, technology, and practice. J Clin Monit 4:290–301, 1988
2. Durand M, Ramanathan R: Pulse oximetry for continuous oxygen monitoring in sick newborn infants. J Pediatrics 109:1052–1056, 1986
3. Boxer RA, Gottesfeld I, Singh S, LaCorte MA, Parnell VA, Walter P: Noninvasive pulse oximetry in children with cyanotic congenital heart disease. Crit Care Med 15:1062–1064, 1987
4. Bland JM, Altman DG: Statistical methods for assessing agreement between two methods of clinical measurement. Lancet I:307–310, 1986
5. Severinghaus JW, Naifeh KH: Accuracy of response of six pulse oximeters to profound hypoxia. ANESTHESIOLOGY 67:551–558, 1987
6. Nickerson BG, Sarkisian C, Tremper K: Bias and precision of pulse oximeters and arterial oximeters. Chest 93:515–517, 1988
7. Cecil WT, Thorpe KJ, Fibuch EE, Tuohy GF: A clinical evaluation of the accuracy of the Nellcor N-100 and Ohmeda 3700 pulse oximeters. J Clin Monit 4:31–36, 1988
8. Barker SJ, Tremper KT: The Effect of carbon monoxide inhalation on pulse oximetry and transcutaneous P_{O_2}. ANESTHESIOLOGY 67:677–679, 1987

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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,[1] 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.[2] 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 $l \cdot min^{-1}$ by the N_2O/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.