

## CORRESPONDENCE

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### Pulse Oximeter Overload

*To the Editor:*—We describe a case in which a patient's finger pulses were too strong for a Nellcor N-100 pulse oximeter with a Durasense probe.

The patient was a healthy 31-yr-old man undergoing a right carpal-tunnel release. Before induction of anesthesia, the hemoglobin oxygen saturation ( $SpO_2$ ) was 99% during administration of 100% oxygen by mask. Anesthesia was induced with propofol and maintained with isoflurane and nitrous oxide by mask. The pulse oximeter sensor was located on the patient's index finger, and plausible readings with a strong signal were obtained until 5 min after induction. At this point, the  $SpO_2$  decreased to zero over the course of eight to ten heart beats. Yet the signal remained strong, the pulse-rate reading was accurate, and cyanosis was not present. The patient's blood pressure was 110/70 mmHg, and he had an unusually strong and palpable digital artery pulse. The oximeter sensor worked correctly when applied on the finger of the anesthesiologist. Occlusion of the patient's radial artery returned the  $SpO_2$  to 95% over the course of five to six beats. On release of this occlusion, the  $SpO_2$  reverted to zero.

During the operation, the probe malfunctioned on the thumb, index, and middle fingers, but worked normally on the fifth finger. The zero  $SpO_2$  was improved to 95% repeatedly with occlusion of the radial artery. In the recovery room, the patient's finger pulse was no longer as palpable, and an oximeter functioned normally.

When contacted about this incident, the Nellcor company confirmed that when pulsatile flow is strong, the N-100 pulse oximeter is unable

to determine the  $SpO_2$  correctly. When the device detects this condition, it displays the pulse rate with an  $SpO_2$  of zero.

In contrast, the Nellcor model N-200 pulse oximeter tolerates a greater pulse signal before becoming overloaded. If overload does occur, the N-200 will go into "pulse search" mode instead of displaying a zero  $SpO_2$  and the pulse rate. This design should be a great improvement. However, to our knowledge, all of the N-100 pulse oximeters currently in use have the same fault that we discovered in ours. If surgical or other problems had occurred simultaneously, our confusion and inability to determine the  $SpO_2$  accurately could have contributed to a critical event.

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*In Reply:*—Armstrong and Perrin are correct in noting that the N-100 pulse oximeter can, in the very unusual condition of very large pulses, become overloaded and display a zero value for saturation. If the N-100 oximeter is presented with abnormally large optical pulses, the integer mathematical computation within it can "overflow" (i.e., exceed the allowed integer range of the microprocessor), resulting in a computed (and displayed) hemoglobin oxygen saturation of 0%. The size of the optical pulse required to overflow the computation approaches a value of 20% pulse modulation (i.e., 20 parts per 100). This is not just a large signal; it is a *gigantic signal*, rarely seen outside of the laboratory. By way of comparison, the normal upper limit of *very large* pulses is approximately 10–12%, whereas the usual pulse modulation is only 1–4%. The occurrence of the pulse overflow noted by the authors is a very rare event, indeed, but it can occur in extreme circumstances.

When the above overflow occurs, the N-100 actively indicates the error condition by the display of a zero value for saturation, an event

that brings the error condition to the attention of the operator immediately. No silent failure occurs. As the authors note, the heart rate is displayed accurately, even though the saturation display is "zeroed out."

In later models of the pulse oximeter, such as the N-200 pulse oximeter and the N-1000 Multifunction Monitor, we changed the design of the error notification to result in a pulse search alarm instead of a zeroed display because we believed that the use of the pulse search alarm for the overflow case was less ambiguous.

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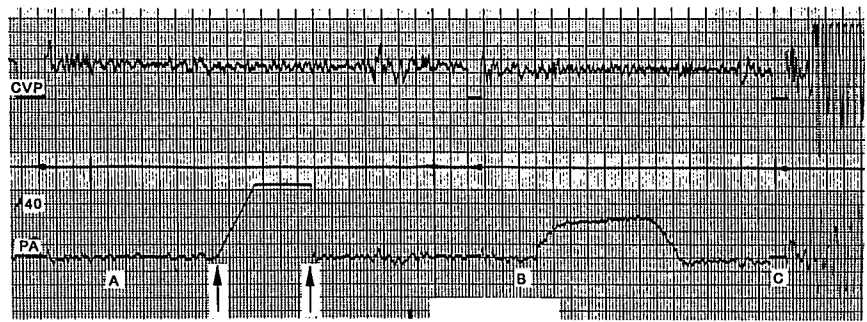
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### A Method for Ensuring Proper Function of Multiorifice Catheters

*To the Editor:*—Recently, Bergman and Jiminez<sup>1</sup> described difficult pulmonary artery catheterization attributable to mislabeling of the proximal and distal orifices. A modification of a previously described maneuver<sup>2</sup> would have prevented their problem.

Before the catheter is inserted and after patency of the various channels is checked, pressing a finger lightly over the distal port will cause a distinct rise in the tracing monitoring the distal lumen. This maneuver ensures correct labeling of the port as well as the function

FIG. 1. A: The fingertip occlusion test identifies the correct monitoring channel. B: The calibration test confirms the accuracy of the system, giving a reading of approximately 30 mmHg. C: Shaking the catheter causes movement in both the central venous pressure (CVP) and pulmonary arterial (PA) pressure tracings and is not helpful in confirming either proper labeling or function. Fingertip pressure was applied to the distal lumen between the arrowheads.



of the transducer and its cable. However, it does not check the accuracy of the calibration system.

This can be done by holding the catheter at the 30-cm mark and extending the tip vertically above the patient's chest. This column of water, now about 40 cm above the zero of the transducer system, should give a reading of approximately 30 mmHg ( $40 \text{ cmH}_2\text{O} \times 0.735 \text{ mmHg/cmH}_2\text{O}$ ) (fig. 1).

We have been performing these maneuvers for several years, and although we have never detected and mislabeled catheters, we have found several malfunctioning or incorrectly labeled transducers.

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## Prediction of Myocardial Oxygen Consumption

To the Editor:—Hoeft and colleagues are to be complimented on a fine study in which they determined the appropriate human coefficients for the pressure-work index (PWI).<sup>1</sup> Their study reconfirms the benefit of cardiac output in the estimation of myocardial oxygen consumption ( $\dot{M}\dot{V}\text{O}_2$ ). It is curious that stroke volume remains largely ignored despite numerous studies demonstrating its utility.<sup>2-4</sup>

Regression analysis on the pooled data of many subjects as used by Hoeft *et al.* may underestimate the relative value of the external work term in the PWI because the best-fit values for the pressure-rate term and the external work term are influenced by the intersubject variability. The coefficients for the PWI were originally defined as the average values obtained from regression analysis performed separately on each animal.<sup>2</sup> When a pooled data analysis was performed on the same data, the coefficient for the external work term decreased from  $3.25 \times 10^{-4}$  to  $2.40 \times 10^{-4}$ , and the coefficient for the systolic pressure-rate term increased from  $4.08 \times 10^{-4}$  to  $4.64 \times 10^{-4}$ . The overall correlation coefficient was 0.870, better than the published value of 0.867 for the PWI.<sup>2</sup> The choice between the two methods depends on whether one wishes to know the actual value of  $\dot{M}\dot{V}\text{O}_2$  or if one is more concerned with the percentage change of  $\dot{M}\dot{V}\text{O}_2$  within a subject. If one desires the actual value, then the pooled regression method used by Hoeft *et al.* may be used. If percent change in  $\dot{M}\dot{V}\text{O}_2$  is important, then average coefficients from separate regressions on individuals will probably be superior.

Lastly, if cardiac index is expressed in liters per squared meter, should not the coefficient for the external work term in PWI modified

for clinical use ( $\text{PWI}_{\text{mod}}$ ) be  $8.0 \times 10^{-3}$ , not  $8.0 \times 10^{-6}$ ? Otherwise, the  $\dot{M}\dot{V}\text{O}_2$  associated with the external work term would be negligibly small and make  $\text{PWI}_{\text{mod}}$  no different than the systolic pressure-rate product.

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