



# MONITOREO HEMODINÁMICO

## The Oximeter Boon or Bane?

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The patient needed to undergo a closed reduction of a dislocated shoulder in the emergency department where I was training. And, because the patient had been "fall-down drunk" at the time, the orthopedic surgeon decided to forego analgesia. This vignette took place fully 40 years ago, near the beginning of my training, but I still recall quite vividly how I reacted. I was horrified, initially believing that the decision to withhold anesthesia was designed as a punitive measure. Perhaps you, dear reader, can remember the first time you were involved in a situation such as this. Eventually, however, I came to appreciate the wisdom of this approach. Although analgesia would have undoubtedly secured the short-term advantage of pain relief, it would have done so at the expense of altering the patient's sensorium. To be sure, a patient's comfort is important, but his/her safety must necessarily take priority. Skilled practitioners administer drugs sparingly, or even not at all, in certain clinical situations, lest we incur the risk of masking a patient's symptoms.

When I initially reviewed the paper by Fu and colleagues appearing elsewhere in this issue of CHEST (see page 1552), I was puzzled. The authors of this piece inveigh against the routine administration of oxygen in postoperative patients, pointing out that the presence of an oxygen-enriched breathing mixture is likely to abolish the emergence of arterial desaturation. To the extent that desaturation episodes might occur, those events would presumably be detected by means of pulse oximetry. At the outset, their argument appeared to be specious. Why criticize the practice of oxygen administration if and when it succeeds in preventing hypoxemia? Isn't that the very point of administering oxygen in the first place? Admittedly, we can't possibly detect an event (hypoxemia) that never happened (because of antecedent oxygen delivery), but why condemn a practice because of its virtues? A more careful reading of the article, however, brought their point home. The routine administration of inspired gases that have been only mildly supplemented (to an oxygen concentration of 25% or 30%) is likely to mask the emergence of ventilatory abnormalities, the presence of which can be crucially important to detect.

At first blush, some readers might be inclined to resist the suggestion by Fu and coworkers that supplemental oxygen should be routinely withheld from patients in the postanesthesia care unit. After all, oxygen is readily available in that environment, and, like Sir Edmund Hillary, shouldn't we use it "because it is there"? The answer, of course, is "not necessarily." To the extent that we might

be prompted to administer oxygen to a patient who has demonstrated a prior indication for it, oxygen delivery is both useful and intelligent. For example, it is commonplace to deliver oxygen-enriched breathing mixtures at night to hospitalized patients with a previous diagnosis of peripheral sleep apnea syndrome. This constitutes prophylactic oxygen use in a patient whose diagnosis has already been determined. But the reflex delivery of oxygen in the postanesthesia care unit to patients whose respiratory drive might be blunted as a result of the residual effects of anesthesia is quite a different story. I suspect that clinicians who adhere to this practice might have mistakenly assumed, as I did, that clinically important cases of ventilatory depression would be detectable by pulse oximetry, provided that the gas mixture delivered to those patients was only mildly elevated in oxygen. If this were the case, applying oxygen sparingly in this setting would allow us to discriminate between transient and clinically unimportant episodes of hypoventilation and more ominous cases of ventilatory dysfunction that we need to identify and treat. However, we are indebted to Fu et al for convincingly demonstrating that this is not the case. They make it abundantly clear that inspired oxygen concentrations of only 25% and 30% are capable of rendering hypoventilation undetectable in the face of continuous oximetry. Thus, they persuasively argue that the routine application of oxygen in this setting is tantamount to burying our head in the sand.



In a larger sense, this article might be used as evidence that pulse oximetry can sometimes conceal more than it reveals. No one can deny that the emergence of pulse oximetry as "the fifth vital sign" represents a boon to caregivers in most situations. Unfortunately, it is possible to become excessively, even slavishly, dependent on the digital readout displayed on the face of the pulse oximeter, to the exclusion of information supplied to us by other methods. Allow me to illustrate this point by describing a case drawn from my own experience. Several years ago, a patient in the ICU of a hospital that shall remain nameless was assigned to me. The practitioner from whom I received report noted that an arterial blood gas determination had not been performed, notwithstanding the fact that this was standard practice in that ICU. The patient, a 33-year-old woman, had been intubated and received ventilation for treatment of a drug overdose. An indwelling arterial line had not been placed, because it was anticipated that the patient's course of mechanical ventilation would be brief. The senior resident who was supervising the clinical team opined that a percutaneous arterial puncture was unnecessary, owing to the fact that the pulse oximeter readings were consistently  $> 95\%$ . By the time that an analysis was finally performed, some 36 h after the initial arterial blood gas, the  $\text{PaCO}_2$  was reported to be in the teens! This prompted the attending physician to roundly scold her resident, to reduce the ventilator respiratory rate by 50%, and to order a repeat arterial blood gas approximately 2 h later. Lo and behold, the  $\text{PaCO}_2$  appearing on the subsequent report was virtually identical. At some length, it became obvious to us what had occurred here. Because the patient had been hyperventilated for a protracted time period, carbon dioxide had been washed out of body stores, which are capacious (approximately 28 L in an adult patient of normal size). With the subsequent onset of hypoventilation, the  $\text{PaCO}_2$  did not rise promptly as we had expected. Instead, the hypoventilatory state elicited a gradual replenishment of the carbon dioxide in the patient's whole-body stores. Finally, some 7 h later, the  $\text{PaCO}_2$  rose to the mid-forties. I must admit that this case taught all of us in attendance that day a valuable lesson about the kinetics of carbon dioxide excretion. Nevertheless, it might certainly have been better for that patient if excessive confidence had not been placed in the pulse oximeter. In the final analysis (no pun intended), the article by Fu and coworkers teaches us that clinical tools are only as powerful as the judgement of the practitioner(s) applying and observing them.