Surgery had included a wide dissection of the diaphragm. It is likely that the shoulder pain was from this and was not vagal in origin. The innervation of the central portion of the diaphragm is by the phrenic nerve, which contains nerve fibers from C_{3-5}. The periphery of the diaphragm is innervated by intercostal nerves from T_{6-9} or T_{10}. When the volume of the narcotic solution administered at the lumbar level was increased from 10 ml to 15 ml, relief of diaphragmatic pain from nerves originating in the cervical spinal cord was achieved along with relief of pain originating from the thoracic and lumbar spinal cord levels. Presumably, by using a larger volume of solution, there was either increased spread of the hydromorphone in the CSF or in the epidural space itself. Since relief of the referred shoulder pain occurred very shortly after injection of the larger volume, the latter explanation appears more probable. It is unlikely that a cumulative effect from the narcotics occurred, since the patient experienced incisional pain as well as shoulder pain before each "top-up" dose of epidural hydromorphone was given.

Since respiratory depression can occur if epidurally administered narcotics reach the brainstem, we were hesitant to use larger volumes initially. Our patient was monitored in the intensive care while receiving epidural narcotics and did not have any evidence of respiratory depression with either 10 or 15 ml of narcotic solution.

In summary, 10 ml of hydromorphone (1.5 mg) administered via an epidural catheter inserted at the L_{3-4} level provided thoracoabdominal incisional pain relief but did not provide analgesia for pain originating from the diaphragm. By increasing the volume to 15 ml, we were able to achieve complete analgesia. The larger volume of solution enabled the hydromorphone to reach the cervical spinal cord levels needed for complete analgesia in our patient, levels that apparently were not reached with 10 ml of solution administered at the lumbar level.

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Noninvasive Detection of Profound Arterial Desaturations Using a Pulse Oximetry Device

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Current monitoring of blood oxygenation in patients with respiratory failure requires analysis of arterial blood gases, which is invasive, expensive, and provides only intermittent information. Furthermore, this type of analysis takes several minutes (depending on institutional facilities), which could prove to be crucial in a given clinical situation. Devices like the pulse oximeter,[1] which purport to measure oxygenation noninvasively, are attractive because they provide continuous information that may result in improved patient care. Pulse oximetry is similar to classical oximetry in that discreet wavelengths of light are used to measure optical density of hemoglobin but is unique in that it can distinguish arterial blood from venous blood and tissue. Pulse oximeters are essentially multiple-wavelength plethysmographs. The pulse amplitude detected is a function of the arterial distension, hemoglobin oxygen saturation of the inflow of arterial blood, and the wavelength of light. When two wavelengths are utilized, the pulse amplitude of the two wavelengths will change relative to each other as the arterial hemoglobin oxygen saturation changes. A ratio of one pulse amplitude to the other will change directly as arterial hemoglobin oxygen saturation changes.[1] We evaluated finger pulse oximetry as a measure of arterial hemoglobin oxygen saturation in critically ill patients with respiratory distress or failure.

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Key words: Equipment: pulse oximetry. Oxygen: tension; saturation; blood levels.

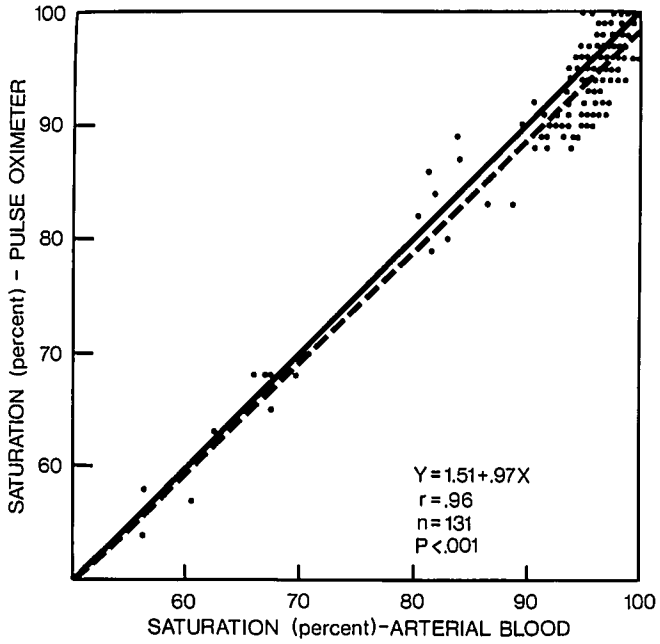

FIG. 1. *In vivo* pulse oximeter readings on the y-axis are compared with *in vitro* measurement of arterial oxygen saturation on the x-axis. The line of identity and linear regression line are the solid and dashed lines, respectively. The pulse oximeter correlated very well with *in vitro* measurements of arterial oxygen saturation.

METHODS

Eighteen patients were studied after informed consent and approval by our Human Subjects Committee. These patients were either those whose tracheas were not intubated, but who were in respiratory distress, or patients with respiratory failure who were being weaned from mechanical ventilatory support. Oxygen monitoring was accomplished by a pulse oximetry device that attached to the patient's finger and provided a continuous digital display of oxygen saturation (Nellcor Pulse Oximeter, Nellcor Corporation). The finger attachment of this oximetry system consists of two light-emitting diodes and a photo diode to receive transmitted light through the finger. The ratio of the maximum amplitudes of two wavelengths of light is measured and fed into a microprocessor that fits this information to an algorithm and then displays arterial saturation and heart rate based on an average of the last 5–7 beats.[1] The patients had in-dwelling arterial catheters through which blood samples were obtained intermittently and analyzed by a blood gas machine and a co-oximeter (I1 282). Values obtained were expressed as an average of triplicate measurements. At the time of blood sampling, *in vivo* arterial oxygen saturation, heart rate, mean arterial pressure, temperature, spontaneous or mechanical respiratory rate and tidal volume, and the presence or absence of anxiety, restlessness, confusion, cyanosis, diaphoresis, or new arrhythmias were recorded. Exhaled tidal volume was measured only in patients whose tracheas were intubated with an in-line Bournes spirometer. Statistical analysis utilized linear regression analysis with a level of significance at $P < 0.05$.

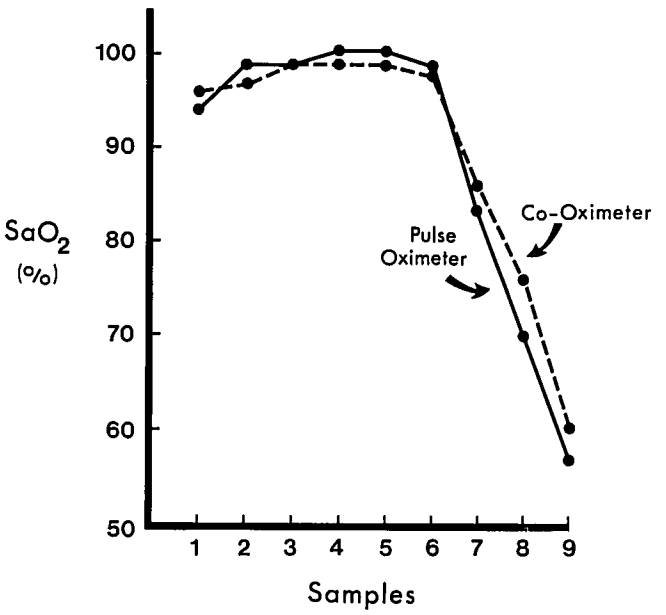

FIG. 2. One of the profound desaturation episodes are displayed in this study. Sequential samples are noted on the x-axis, while the y-axis shows arterial oxygen saturation, comparing the pulse oximeter readings with samples run on the I1 282 co-oximeter.

RESULTS

In 18 patients studied, 131 simultaneous comparisons between the pulse oximeter readings and analyzed arterial blood hemoglobin oxygen saturations were made. *In vitro* arterial hemoglobin oxygen saturations ranged from 56.2 to 99.9%. Carboxyhemoglobin and methemoglobin levels were less than 3.7 and 2.6%, respectively, for all patients studied. Analysis of linear regression demonstrated an excellent correlation between the pulse oximeter and the analyzed blood samples (fig. 1). During the study, three of the 18 patients developed potentially life-threatening arterial desaturation, defined as an arterial saturation less than 70%. Each of these episodes were detected accurately by the pulse oximeter and were not evident by clinical findings alone (fig. 2). Separate linear regression analysis of the samples obtained from these three patients also demonstrated an excellent correlation: $y = 1.00x - 0.76$, $n = 23$, $r = 0.99$, $P < 0.001$.

The patient population studied were elderly (age = 67 ± 10, mean ± SD). The primary diagnoses included the following: pneumonia (n = 5), chronic obstructive pulmonary disease (n = 4), lung cancer (n = 3), congestive heart failure (n = 2), sepsis (n = 2), cirrhosis (n = 1), and myasthenia gravis (n = 1). No

patient studied was hypothermic, with core temperatures for all patients ranging within 36.3–38.2° C. The site of attachment of the oximetry device was the index finger in 13 of the 18 patients. In the other five patients, other sites were used in order to obtain a better pulse signal (third finger — 2, thumb — 1, second toe — 2). Nine patients were receiving vasopressor drug infusions (dopamine — 8, neosynephrine — 1, epinephrine — 1, and four of these patients, plus another patient not receiving vasopressors, experienced episodes of hypotension (MAP < 60 mmHg) during the study. A total of nine of the 131 data sets were obtained during periods of hypotension. When analyzed separately, they demonstrated accurate pulse oximeter readings ($r = 0.97$, $P < 0.001$, $n = 9$).

There were four patients in whom signal failure occurred. None of the patients were hypotensive (MAP < 60 mmHg) or hypothermic (core temp < 36.3° C). In two signal failure occurred with initial monitoring attempts. In one of these patients, who had a history of vascular disease but was not receiving vasopressor drugs, signal failure occurred on two different days. The other patient who had initial signal failure was receiving dopamine and epinephrine infusions at the time. On another day when she no longer was receiving these drugs, a good signal was demonstrated and she was studied successfully. In the two other patients, signal failure occurred after initially obtaining an adequate signal. One of these patients was receiving a dopamine infusion, and the signal loss was associated with increasing the infusion rate to 7 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

Discussion

Transdigital noninvasive measurement of arterial hemoglobin oxygen saturation is accurate and reliable in normal volunteers in the range of 65–100%.[1,2] The results of this study demonstrate that noninvasive oxygen monitoring of critically ill patients with respiratory distress or failure is feasible and accurately can detect potentially life-threatening arterial hemoglobin oxygen desaturations. In the intensive care unit environment, such bedside information can provide increased safety in the weaning of ventilatory dependent patients. Adjustments of respiratory support can be made more rapidly, since the effects on oxygenation are immediately evident. This should shorten weaning periods and may decrease intensive care unit stays for some patients. In other situations, hypoxic events during weaning trials can be either prevented or identified and corrected with minimal insult to the patient.

The pulse oximeter has few limitations, the most significant being signal failure as a result of poor digital infusion. This oximeter may be unable to detect arterial saturations in patients who are hypothermic. Although none of the patients we studied were hypothermic, it must be recognized that large differences between extremity and core temperatures may exist and are particularly prone to develop during long surgical procedures in cold operating rooms. Other factors such as sympathetic tone, vasopressor drugs, hypotension, and peripheral vascular disease also may result in pulse oximeter signal failure, but the results of our study, as well as the nonquantifiable nature of some of these factors, prevents any clear-cut guidelines from being established. In this study, reliable data were obtained during all hypotensive episodes and in the majority of patients (seven of nine) on vasopressors (including one patient who was receiving neosynephrine infused at 425 $\mu g/min$).

The presence of dyshemoglobinemias (carboxyhemoglobin, methemoglobin, sulfhemoglobin) also may affect the oximeter accuracy, but this will not be an issue with most patients. Although none of the patients in the study had significantly elevated carboxyhemoglobin levels, caution is warranted when using this monitor in any patient suspected of smoke inhalation. The occurrence of acquired methemoglobin or sulfhemoglobin is an unusual event. Levels of these substances that are sufficient to cause cyanosis (1.5 g/dl and 0.5 g/dl, respectively) should not affect the pulse oximeter readings, however, levels may be very high in a rare patient. This monitor should not be used to replace laboratory blood analysis in a patient with unexplained cyanosis.

This type of monitoring combined with noninvasive monitoring of carbon dioxide elimination (*e.g.*, end-tidal or transcutaneous carbon dioxide measurement)[3] may reduce the need for blood gas analysis in many patients. These monitors might be particularly appropriate in patients without metabolic acid–base disturbances, where measuring blood *p*H would be unnecessary.

In summary, this study demonstrated the reliability of finger-pulse oximetry to detect clinically significant arterial desaturations that occurred in three of 18 critically ill patients. The value of the pulse oximeter lies in the possibility of not only reducing hospital costs of arterial blood analysis but also in the possibility of improving patient care by providing continuous information of arterial oxygenation.

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