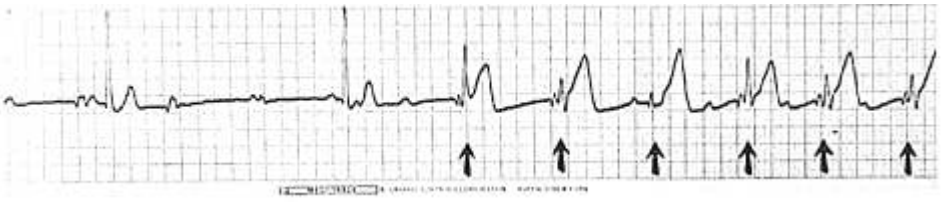

FIG. 3. Underlying rhythm after approximately 30 s of precordial thumping showing high grade A-V block with occasional supraventricular beats. Precordial thumping was resumed (arrows) again resulting in QRS complexes and patient awakening.

tors are examples of natural mechanicoelectrical transducers. Coughing (which may generate 1–25 joules of energy[5]) has been successfully used by patients to pace or cardiovert themselves after asystole or ventricular tachycardia.[6]

The technique of precordial chest thumping has been used by this author and associates to temporarily pace severe sinus bradycardia or sinus arrest occurring at induction of anesthesia or during laryngoscopy. All such attempts resulted in successful temporary circulatory support until an adequate sinus rhythm was restored.

When compared to standard cardiopulmonary resuscitation and its attendant complications (rib fracture, liver laceration, myocardial and pulmonary contusions),[8] pacing by precordial chest thump appears to be a benign and effective initial means of resuscitation in witnessed bradycardic or asystolic arrest. The potential for inducing a more malignant rhythm with chest thumping has been reported to be as high as 44% when used without regard to duration or magnitude of cardiac dysfunction.[9] However, a closely monitored patient in an operating room is an ideal candidate for resuscitation by precordial chest thumping, should this become necessary. This means of resuscitation should be considered early in the management of monitored asystolic or bradycardic arrest in the perioperative period.

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Respiratory Artifact during Pulse Oximetry in Critically Ill Patients

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Pulse oximetry is a valuable monitor,[1–3] but problems are associated with its use.[4–7] We report a case during which the pulse oximeter failed to detect the usual pulse wave during mechanical ventilation with high peak inspiratory pressures, but did work when controlled ventilation was briefly interrupted.

REPORT OF A CASE

A previously healthy 22-yr-old man sustained trauma resulting in anoxic brain damage, bilateral hemopneumothoraces, and multiple fractures. Massive transfusions of blood and crystalloid were required in the immediate resuscitation period. Bilateral chest tubes were placed prior to emergent abdominal exploration. The patient arrived in the operating room mechanically ventilated and comatose (Glasgow 4), with a sinus tachycardia, hypothermia (34.5° C), and an arterial blood pressure of 110/50 mmHg.

During surgery, a Siemens™ 900C anesthesia machine was used to control ventilation at a rate of 20 breaths per minute, a tidal volume

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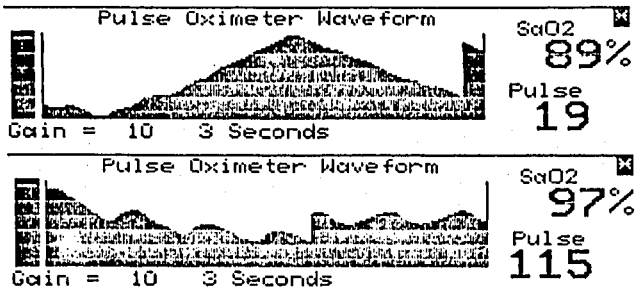

FIG. 1. *Top.* Waveform during positive pressure ventilation. Displayed rate is equal to respiratory rate; displayed saturation is low. *Bottom.* Waveform during expiratory pause in ventilation. Displayed rate is equal to heart rate; displayed saturation is normal.

of 1100 cc (12 cc/Kg), and a positive end-expiratory pressure (PEEP) of 15 cm H_2O. Peak airway pressures ranged from 45 cm H_2O to 55 cm H_2O. Oxygen saturation was measured with a Nellcor™ N-100 pulse oximeter interfaced to a System for Anesthetic and Respiratory Analysis (SARA™) monitor that continually displayed the pulse oximeter waveform and saturation values. Because a signal was not obtained with a finger probe, a nasal probe was used. The waveform shown in figure 1 (top) was obtained during mechanical ventilation. The rate of 19 per minute corresponds to the rate of ventilation. When mechanical ventilation was halted at end expiration, the pulse oximeter promptly sensed a pulse wave at the same rate as the electrocardiogram and displayed a saturation value (fig. 1, bottom). This maneuver was repeated several times during surgery with similar results. The saturation values obtained from the oximeter during expiratory pause were consistently 93–95%.

DISCUSSION

The Nellcor™ N-100 pulse oximeter emits light at two different wavelengths from light-emitting diodes. The light traverses tissues and is sensed by a photodetector. The resultant signals are resolved into alternating and direct components; the alternating component represents the pulsating vascular bed and the direct component the non-pulsatile blood and tissue. The direct component of the signal is used to scale the alternating component. The alternating (pulsatile) component is further processed to yield a saturation value which ideally represents arterial hemoglobin saturation with oxygen.

In the case described above, failure to sense a pulse using a finger probe was likely due to severe peripheral vasoconstriction of the cool extremities. Vascular pulsations in the bridge of the nose may persist in instances when finger pulsations do not.[1] For that reason, the nasal probe was tried and demonstrated an interesting phenomenon which we have since noticed in other cases. The upper tracing in figure 1 demonstrates an instance where the pulse oximeter recorded the respiratory pulse wave and displayed a rate equal to the respiratory rate and a saturation value of 89%. When mechanical ventilation was halted at end expiration, the oximeter promptly generated a waveform that coincided with the patient's heart rate and displayed a higher saturation value. PEEP was maintained during these maneuvers. We suggest the high airway pressures required to ventilate this patient caused phasic venous congestion that was recorded by the oximeter as a pulse wave. Additionally, phasic changes in both systolic and mean arterial blood pressure with ventilation may also have contributed to the waveforms we observed. The relative importance of venous *versus* arterial pressure changes is yet to be studied.

Kim *et al.*[3] contend that pulsations in arteriovenous anastomoses, rather than arterial pulsations, are sensed by the pulse oximeter. The abundance of arteriovenous anastomoses in the cutaneous circulation allows arterial blood to be shunted into veins, bypassing the capillaries, prior to being sensed by the pulse oximeter. Venous congestion at the sensing site may have contributed to a greater proportion of venous blood in the pulse waves sensed by the oximeter.

Partridge has suggested using pulse oximetry as a noninvasive indicator of intravascular volume.[8] Figure 1 shows that the baseline variation due to increases in intrathoracic pressure may overshadow the pulse waveform variation due to cyclical changes in arterial blood pressure. Therefore, useful information may be gained even in the absence of typical arterial pulse waves.

We conclude that during mechanical ventilation requiring high positive pressures, artifact due to venous congestion and fluctuating arterial blood pressure may occur. This artifact may be circumvented and useful information gained if ventilation is interrupted for brief periods. PEEP need not be discontinued during this maneuver.

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