Systolic Venous Waves Cause Spurious Signs of Arterial Hemoglobin Desaturation

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Pulse oximetry has found extremely widespread use in the perioperative period,1−3 and its routine use has been recommended.4−6 During cardiac surgery invasive hemodynamic monitoring supplements oximetry and other noninvasive perioperative monitors. Recently, two patients were identified who had abnormal venous waves and coincident apparent arterial hemoglobin desaturation indicated by ear probe pulse oximetry. The combination of pulse oximetry, blood sampling, and observation of the intravascular pressure waveforms allowed the elucidation of an unusual but clinically important problem inherent to pulse oximetry.

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CASE REPORTS

Patient 1. A 78-yr-old woman with severe mitral stenosis, pulmonary hypertension, and tricuspid regurgitation was scheduled for mitral commissurotomy. Perioperative monitoring included electrocardiography (ECG), mean arterial pressure (MAP), pulmonary arterial pressure (PAP), and pulse oximetry ($S_{PO_2}$) with an ear probe (Ohmeda Biox 3700), Ear Probe model #8122-003, Boulder, Colorado; Pulse oximetry equipment was identical in patient 2.) Apparent arterial hemoglobin desaturation ($S_{PO_2} < 80\%$) was observed during induction of anesthesia despite the appearance of bright red blood drawn from the arterial catheter. At the time $PAP = 67/25$ mmHg and central venous pressure (CVP) = 14 mmHg with large systolic v-waves present (fig. 1). A sample of arterial blood revealed a $PaO_2 = 470$ mmHg (Stat Profile 4, Nova Biomedical, Waltham, Massachusetts). Simultaneously, mixed venous blood sampled from the pulmonary artery showed $PvO_2 = 39$ mmHg, calculated $SvO_2 = 75.8\%$.

**FIG. 1.** Hemodynamic recordings from a patient with severe holo-systolic tricuspid insufficiency, similar to patient 1. The large systolic wave of tricuspid insufficiency (v-v) in the CVP trace temporally overlaps the systolic arterial pulse wave recorded at the radial artery (ART). The dashed line marks the ECG R-wave for reference. Vertical scales are in mmHg. Note that the CVP c-v wave is truncated due to the limits of the chart recorder.

Patient 2. A 72-yr-old man with severe coronary artery disease, biventricular dysfunction, and a history of congestive heart failure was scheduled for myocardial revascularization. Perioperative monitoring was established as in patient 1. The ECG showed junctional rhythm and left bundle branch block, MAP = 91 mmHg, cardiac index (CI) = 1.0 l·min$^{-1}$·m$^{-2}$, mean PAP = 56 mmHg, and CVP = 20 mmHg with large early systolic waves (fig. 2A). The pulse oximeter connected to an ear probe consistently showed marked arterial hemoglobin desaturation ($S_{PO_2} = 60-80\%$). The pulse oximeter probe was changed to the finger (Finger Probe model #8122-001), where the saturation readings were 100%. Arterial and mixed venous blood were sampled immediately thereafter and showed $PaO_2 = 425$ mmHg, $PvO_2 = 38$ mmHg, and calculated $SvO_2 = 71.1\%$.

After cardiopulmonary bypass, oximetry monitoring resumed with two devices, one using the ear probe and one the finger probe. Both showed arterial saturation to be 100%. Circulation had improved at this time; the ECG showed AV sequential pacing, MAP = 68 mmHg, CI = 2.2 l·min$^{-1}$·m$^{-2}$, mean PAP = 55 mmHg, and CVP = 8 mmHg with normal a- and v-waves (fig. 2B).

**FIG. 2.** Hemodynamic recordings from patient 2. A. Recording done during junctional rhythm (AV dissociation). A large systolic wave (*) is inscribed in the CVP trace and temporally overlaps the systolic arterial pressure recorded in the radial artery (ART). B. Recording done during AV sequential pacing when normal AV synchrony has been restored. A normal CVP trace with small a- and v-waves is present. | and † denote the atrial and ventricular pacing spikes, respectively. Other notations are similar to figure 1.

**DISCUSSION**

These observations highlight a clinically important problem with ear probe pulse oximetry in critically ill patients with prominent systolic venous pressure waves. By indicating arterial hemoglobin desaturation, these spuriously low readings monitored via ear probe could lead to incorrect therapy. Observation of the CVP tracing provided insight into the mechanism of the problem, allowing appropriate action to be taken.
Despite near-universal acceptance as a clinically useful and reliable monitoring technique, pulse oximetry is subject to a variety of technical artifacts that generate erroneous information. Most common are motion artifacts and peripheral vasoconstriction, both of which alter the pulse signal sufficiently to warn that the saturation values displayed should be questioned. Vital dyes, such as methylene blue, indigo carmine, and indocyanine green, may alter light absorption in the critical range monitored by the pulse oximeter and influence readings by the device. Other sources of error include recognition of dysfunctional hemoglobin species (carboxy- and met-) and subsequent overestimation of the proportion of oxyhemoglobin present. None of these issues seemed pertinent to the cases reported here. Furthermore, the oximeters and probes functioned normally without modification on subsequent patients.

Pulse oximeters examine the vascular bed interposed between the light source and detector and assume that the observed pulsations arise from the arterial system. Although Kim et al. have suggested that pulsations detected by pulse oximetry were mostly venous in origin, these pulsations were ascribed to arterial blood shunted across open arteriovenous anastomoses. More recently, a true venous artifact has been reported to occur during pulse oximetry with a nasal probe. In this instance, phasic venous congestion during mechanical ventilation was recorded as a slow pulse wave and erroneously indicated arterial desaturation.

The venous pulsations described in the present report are of a different nature. In these cases, CVP was elevated and pulsatile, with a prominent venous pressure peak occurring during systole, near the time that the arterial pressure peak was occurring. In the first instance, a prominent c-v wave of holosystolic tricuspid insufficiency was caused by retrograde systolic ejection of venous (right ventricular) blood through the regurgitant tricuspid valve. The second patient also manifested a prominent systolic venous wave, although the pathophysiologic mechanism was different. Here atrioventricular dissociation accompanying junctional rhythm produced a transient pressure peak in early systole, again temporally overlapping the arterial waveform (fig. 2A). This prominent early systolic wave has been called either c-n wave, due to atrial contraction during systole against a closed tricuspid valve, or c-wave of transient tricuspid insufficiency, due to atrioventricular dysynchrony. Whatever the functional cause, this abrupt systolic rise in venous pressure is recognized clinically by venous pulsations in the neck and presumably was recognized as a systolic vascular pulsation by the oximeter ear probe as well. As a result, the oximeter mechanism was thwarted, identified venous blood, and reported saturations closer to venous values. Presumably, pulse oximetry using a finger probe will still recognize arterial blood (as in patient 2) because the central venous pulsations may be dampened and attenuated by external pressure on the limbs, venous valves, and distance between the heart and veins of the fingertip.

In summary, patients with prominent systolic venous pulsations may have artfactually low pulse oximeter recordings of arterial hemoglobin saturation when ear probes are utilized. Unlike the experimental subjects studied by Severinghaus and Naifeh, these patients with cardiac abnormalities may have more accurate arterial hemoglobin saturation recorded with finger probes. Finally, the central venous waveform is readily available when a CVP catheter is in place. Critical continuous monitoring of the venous waveform can elucidate circulatory disturbances and help explain other confusing pathophysiologic events.

REFERENCES