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Respiratory Changes in the Pulse-Oximetry Waveform Associated with Pericardial Tamponade

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Summary

Background: Timely identification of hemodynamic compromise in patients with acute pericardial effusion and tamponade is critical in patient management. Respiratory variability in pulse-oximetry waveforms has been correlated with pulsus paradoxus, but has not been reported with cardiac tamponade in adult patients.

Hypothesis: This study describes changes in respiratory variability in pulse-oximetry waveform pre and post pericardiocentesis in patients with hemodynamically significant pericardial effusions.

Methods: A single-center, catheterization laboratory hemodynamic database was reviewed for all patients who underwent pericardiocentesis for clinically suspected tamponade and had continuous digital pulse-oximetry, electrocardiographic, and respiration waveforms recorded during the procedure. Phasic respiratory changes in pulse-oximetry waveform amplitude (maxima—minima) were expressed as an expiratory/inspiratory ratio and compared pre and post pericardiocentesis.

Results: The study population consisted of 12 patients (6 men:6 women, age 60 ± 10 years) with pericardial effusion documented by echocardiography on the day of pericardiocentesis. Phasic respiratory variability in the pulse-oximetry waveform was evident in all patients prior to aspiration (respiratory ratio = 1.9 ± 0.5). Following pericardiocentesis (aspirat-

ed volume: 650 ± 300 ml), the respiratory ratio decreased in all patients (1.2 ± 0.1 , $p = 0.001$). Receiver operator characteristic curve analysis suggests that pulse-oximetry respiratory ratios ≥ 1.5 should raise suspicion of hemodynamic compromise in high-risk populations.

Conclusions: Pulse-oximetry is a commonly used tool for monitoring critically ill patients. The present study suggests that increased respiratory variability in the pulse-oximetry waveform should raise suspicion for hemodynamic compromise in patients at risk for pericardial effusion.

Key words: pulse oximetry, cardiac tamponade, pericardial effusion

Introduction

Pericardial tamponade is a life-threatening condition characterized by decreased cardiac filling secondary to increased pericardial pressure. The normal pericardium has a small degree of elasticity and contains a thin layer of fluid. A variety of conditions can lead to accumulation of fluid within the pericardial space and an increase in pericardial pressure with resultant limitation in cardiac inflows and equalization of mean diastolic pericardial and chamber pressures. Prompt identification of tamponade and treatment with pericardiocentesis is essential.¹

Two-dimensional echocardiography remains the modality of choice for the diagnosis of large pericardial effusions and can demonstrate impaired cardiac filling before the development of frank tamponade. In austere environments, however, echocardiography may be difficult to obtain in a timely fashion or may yield suboptimal imaging in patients with significant lung or mediastinal disease. Clinical signs of cardiac tamponade such as pulsus paradoxus are highly suggestive but may be difficult to detect.

Pulse-oximetry is a commonly employed modality for the noninvasive monitoring of physiologic status in critically ill patients. Studies in pediatric populations have shown that pulse oximetry is useful for detecting the pulsus paradoxus associated with respiratory distress and airflow obstruction,^{2–7} as well as congestive heart failure in neonates with supraventricular tachycardia.⁸ A recently published study suggested

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that pulse oximetry may be of value in detecting hemodynamically significant pericardial effusions;⁹ Tamburro *et al.* reported an inspiratory reduction of the plethysmographic peak of the pulse-oximetry waveform in seven pediatric patients, which was partially or completely reversed after pericardiocentesis.⁹ To date there have been no reports of respiratory pulse-oximetry waveform changes in adult patients with pericardial tamponade.

Methods

The catheterization laboratory hemodynamic database at Brooke Army Medical Center was reviewed from 2003–2005 for all adult patients who underwent pericardiocentesis for clinically suspected tamponade and had continuous digital pulse-oximetry, electrocardiographic (ECG), and respiration waveforms recorded during the procedure. Case information recorded included demographic and diagnostic information and vital signs during presentation and at the time of pericardiocentesis. Pulsus paradoxus was defined as an inspiratory decrease in systolic pressure ≥ 10 mmHg. Other data recorded included the volume of fluid aspirated and the etiology of the effusion. All patients had echocardiographic documentation of pericardial effusion and the clinical diagnosis of symptomatic pericardial effusion and/or cardiac tamponade prior to referral for pericardiocentesis.

Pulse-oximetry, respiratory, electrocardiographic, and hemodynamic waveforms were obtained from a full-disclosure hemodynamic recording system (Physiolog, Camtronics Inc, Hartland, Wisc., USA). Respiratory and beat-to-beat changes in pulse amplitude were measured from continuous recordings of a digital waveform archive (500 S/s per channel) pre and post pericardiocentesis.

The pulse-oximetry waveform was recorded from the analog output (millivolts) of the pulse oximeter (Masimo Corporation, Irvine, Calif., USA). The amplitude of the pulse-oximetry wave was defined as the number of units displaced from the peak to the trough of the wave. Respiratory pulse-oximetry waveform variability was expressed as the expiratory/inspiratory ratio of pulsatile changes (maximum—minimum waveform amplitude). This retrospective study was approved by the institutional review board (IRB) at Brooke Army Medical Center.

Data Analysis

Data are presented as mean $\pm$ standard deviation (SD). A paired *t*-test was used to compare pulse-oximetry pulsatile respiratory ratio pre and post pericardiocentesis (Sigma Stat version 3.11, Systat Software Inc., Richmond, Calif., USA). *P* values < 0.05 were considered significant. Given the limitations of referral bias in this case series, a receiver operator characteristic (ROC) curve and plot of sensitivity and specificity were employed to identify a threshold for pulse-oximetry respiratory ratio, which may raise the suspicion in future studies for hemodynamically significant pericardial effusion. To define specificity, it was assumed that the patient's improved clinical and hemodynamic status post pericardiocentesis reflected a trend toward normal.

Results

Twelve patients were identified who met inclusion criteria (Table I). All patients had symptomatic pericardial effusions, and all but one had a preprocedure clinical diagnosis of peri-

TABLE I Summary results

Patient number	Age (years), gender (M/F)	Diagnosis (type of effusion)	Aspirated fluid (ml)	Pulsus paradoxus (mmHg)	Respiratory variation Initial SBP (mmHg)	Pulse-ox Ratio		Respiratory variation Final SBP (mmHg)
						Pre	Post	
1	57 M	Post AVR/CABG	1,000	N/R	N/R	1.67	1.17	N/R
2	62 F	Malignant	500	N/R	10–15	1.55	1.27	5–8
3	61 F	Malignant	500	N/R	N/R	1.81	1.31	N/R
4	72 F	Malignant	850	18	20–25	2.23	1.49	5–8
5	63 M	Malignant	500	20–25	20–30	1.5	1.07	8–10
6	45 F	Post CABG	300	Palpable ^a	N/R	2.3	1.16	N/R
7	75 M	Post AVR	1000	35–40	N/R	2.71	1.22	N/R
8	57 M	Post CABG	600	N/R	N/R	1.22	1.05	N/R
9	64 M	Malignant	1200	N/R	35–40	2.52	1.07	8–12
10	42 F	Malignant	800	13–14	30–40	2.42	1.12	6–8
11	52 F	Post LA myxoma resection	200	N/R	N/R	1.67	1.45	N/R
12	71 M	Post pacemaker	650	14	N/R	1.29	1.2	N/R

^a Refers to loss of palpable radial pulse during inspiration as noted in the medical record.

Abbreviations: AVR = aortic valve replacement, CABG = coronary artery bypass grafting, SBP = invasive central aortic systolic blood pressure, LA = left anterior, N/R = not recorded.

FIG. 1 Changes in the electrocardiogram (ECG), femoral artery blood pressure (BP), pulse-oximetry (pulse-ox) and respiratory (Resp) waveforms in a 42-year-old patient prior to (Pre) and following (Post) aspiration of 800 cc pericardial fluid. Inspiration is noted by a downward deflection of the respiratory waveform.

cardiac tamponade defined by hypotension, tachycardia, elevated jugular venous pressure, respiratory distress, positive pulsus paradoxus, or echocardiographic evidence of impaired cardiac filling. The patients ranged in age from 42 to 71, with mean age of 60 years. Male and female patients were equally represented. The most common cause for pericardial effusion in this group was malignancy (50%). All patients were taken urgently to the cardiac catheterization laboratory for pericardiocentesis. All patients exhibited respiratory variability in both peak and pulsatile amplitudes in their pulse-oximetry waveforms that decreased following pericardiocentesis (mean aspirated volume = 650 ± 300 cc).

Figure 1 illustrates a representative case of a 42-year-old woman who was taken to the cardiac catheterization laboratory with the clinical diagnosis of cardiac tamponade from a malignant pericardial effusion. Prior to pericardiocentesis (left panel), the patient demonstrated pulsus paradoxus with an inspiratory fall in arterial blood pressure of 30–40 mmHg. Associated with this was an inspiratory fall in both peak and

FIG. 2 An average decrease > 32% in the pulse-oximetry respiratory ratio with pericardiocentesis is shown.

amplitude of the pulse-oximetry waveform, which returned to baseline as the patient exhaled. In this patient, the respiratory pulse-oximetry ratio was calculated to be 2.4 prior to pericardiocentesis. Following removal of more than 800 cc of pericardial fluid, the pulse-oximetry ratio decreased to 1.1 (right panel). This was associated with attenuation in the presentation of pulsus paradoxus in arterial systolic blood pressure (10–15 mmHg). The persistent respirophasic variation in systolic pressure was suspected to be from effusive-constrictive physiology, which has been reported in cases of malignant effusion in the absence of radiation therapy.^{10, 11}

Figure 2 illustrates the changes in pulse-oximetry ratios pre and post pericardiocentesis in all patients. The pulse-oximetry ratio decreased by an average of $32.3 \pm 17.8\%$ ($p = 0.001$) following pericardiocentesis (pre 1.9 ± 0.5 vs. post 1.2 ± 0.1). It is noteworthy that the ratio decreased in all patients following pericardiocentesis and that these changes were associated with improvement or resolution of clinical and hemodynamic derangements. It was observed (Fig. 2) that patients with higher initial values had a more dramatic decrease in respiratory variability in their pulse-oximetry waveform following effusion drainage.

Figure 3 depicts the sensitivity and specificity for various cut-off values of the pulse-oximetry ratio. In this selected group of patients with symptomatic pericardial effusions and with caveat that specificity was defined from pulse-oximetry postpericardiocentesis, a pulse-oximetry ratio threshold of 1.5 yielded a sensitivity and specificity of 83 and 100%, respectively, for detecting clinically significant pericardial effusions.

Discussion

The pulse oximeter was first introduced in the 1970s and uses pulsatile variations in optical density of tissues in the red

FIG. 3 A plot of sensitivity and specificity versus pulse-oximetry respiratory ratio suggesting that, as the ratio approaches 1.5, the sensitivity and specificity are 83 and 100%, respectively, for hemodynamically significant pericardial effusions. See text for the caveats used to define this index from the sample population.

and infrared wavelengths to compute arterial oxygen saturation without the need for calibration.^{3, 12} It also acts as a plethysmograph by measuring the changes in blood volume across a pulsating vascular bed.³ While the primary use for pulse oximetry continues to be for noninvasive monitoring of patients' arterial oxygen saturation, recent work has shown it to be useful in other clinical situations. For example, the pulsatile variation in the light transmission detected by the device is believed to be directly related to arterial blood pressure.¹³ This feature has led to research that demonstrated the ability of pulse oximetry to detect pulsus paradoxus in pediatric patients in the settings of obstructive airway disease and pericardial effusion. The present study extends observations of respiratory variability in pulse-oximetry waveforms to adults with symptomatic pericardial effusion and/or cardiac tamponade.

In 1988, Ryan reported respiratory variability in the pulse-oximetry signal in a 12-year-old child with status asthmaticus and a 40 mmHg pulsus paradoxus. As the child's clinical condition improved with treatment, the respiratory variability lessened and eventually resolved.³ In 1992, Chadwick *et al.* evaluated 15 healthy volunteers and demonstrated that the presence of respiratory variability in the pulse-oximetry waveform was equivalent to the clinical detection of pulsus paradoxus; they suggested its potential for use in infants with airway obstruction.⁴ Steele *et al.* observed similar findings in healthy adults in whom pulsus paradoxus was induced by breathing through a one-way valve⁶ and concluded that significant pulsus paradoxus should be suspected when large respiratory variations in the amplitude of pulse-oximetry waveforms are observed. Hartert *et al.* studied 26 adults admitted to the intensive care unit with obstructive airway disease and noted that pulse-oximetry waveform variability was useful for determining the severity of airflow obstruction.⁵

A prospective study by Frey and Butt evaluated the relationship between intra-arterially measured pulsus paradoxus and changes in the pulse-oximeter waveforms in 63 nonintubated children, both with and without respiratory disorders. They reported that the magnitude of photoplethysmographic fluctuations in the pulse-oximetry waveform correlated well with the severity of intra-arterially measured pulsus paradoxus.² The presence of pericardial effusion was not evaluated in the study despite inclusion of 33 patients post cardiac surgery. The same authors observed large fluctuations of the pulse-oximetry waveform associated with a 20–30 mmHg pulsus paradoxus in a 19-day-old neonate with congestive heart failure secondary to long-lasting supraventricular tachycardia.⁸ There was near complete resolution of these fluctuations after the patient was converted to normal sinus rhythm.⁸

More recently, Clark *et al.* reported that pulse-oximetry waveform changes compared favorably with traditional auscultation to detect pulsus paradoxus in children.⁷ Tamburro *et al.* demonstrated that pulse-oximetry waveform analysis was useful in the detection of pulsus paradoxus in pediatric patients with large pericardial effusions and evidence of cardiac tamponade.⁹ They noted an inspiratory drop in the peak of the pulse-oximetry wave that lessened after pericardiocentesis.⁹

The present case series is the first to extend observations of pulse-oximetry variability to an adult population with hemodynamically significant pericardial effusion and tamponade. We also confirmed in this population that respiratory variability in pulse-oximetry waveform decreased following pericardiocentesis. Results from this study also suggest that respiratory changes in the amplitude of the pulse-oximetry waveform ≥ 1.5 may raise a suspicion of hemodynamically significant effusion in high-risk patients. Although the retrospective nature and selected study population are limitations in precisely defining the true sensitivity and specificity of pulse oximetry for detecting cardiac tamponade, the observations of increased respiratory variability may alert providers to the need for further evaluation and a life-sustaining intervention.

Conclusion

Patients with tamponade frequently present with nonspecific symptoms, the detection of pulsus paradoxus by auscultation is often difficult, and echocardiography is not always readily available for definitive diagnosis. The results of this study, in addition to those in previous reports, suggest that respiratory pulse-oximetry amplitude changes may be a useful marker for raising the index of suspicion for significant pericardial effusion and impending hemodynamic collapse. Additional prospective studies would be helpful in further defining the value of pulse oximetry for detecting early pericardial tamponade.

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