Clinical paper

Vital sign patterns before shock-related cardiopulmonary arrest

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Abstract

Objective: Traditional vital sign thresholds reflect an increased risk of mortality, which may occur hours, days, or weeks following illness/injury, limiting immediate clinical significance to guide rescue therapy to avoid arrest. Our objective is to explore vital sign patterns prior to arrest due to shock.

Design: This retrospective observational analysis utilized physiological data from adult helicopter patients suffering provider-witnessed arrest. Pre-arrest values for systolic blood pressure (SBP), mean arterial pressure (MAP), heart rate (HR), shock index, and end-tidal carbon dioxide (EtCO2) were modeled against time using polynomial linear regression. The "terminal inflection point" beyond which arrest was imminent was identified where slope equals 1.0 (shock index) or -1.0 (SBP, MAP, HR, EtCO2) and was then compared to initial values.

Setting: Air ambulance services.

Patients: 70 helicopter patients over age fourteen suffering cardiac arrest.

Results: SBP and MAP demonstrated a gentle decline followed by acceleration beyond the inflection point (SBP 80.7 mmHg, MAP 61.9 mmHg). HR demonstrated an increase followed by a terminal drop, but inflection point values fell within normal range. Shock index increased gradually from a mean of 0.9 to the inflection point of 1.1. Initial EtCO2 values declined gradually from normal (34.4 mmHg) to the inflection point (24.7 mmHg), then dropped precipitously into arrest. All inflection points occurred 2–5 min prior to arrest.

Conclusions: Vital sign patterns were defined for SBP, MAP, HR, shock index, and EtCO2 with clear inflection points identified 2–5 min prior to arrest. These patterns may help guide therapy to reverse deterioration and prevent arrest.

Keywords: Shock, End-tidal CO_2, Cardiopulmonary arrest, Pre-arrest patterns, Vital sign patterns, Capnography

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Introduction

Deterioration from shock is common in both the out-of-hospital and inpatient environments. Furthermore, a substantial proportion of provider-witnessed cardiopulmonary arrests appear to have a shock etiology, with outcomes remaining abysmal.[1,2] Thus, the identification of critical thresholds for various physiological metrics– beyond which arrest is imminent– has great appeal to suggest the use of aggressive rescue therapies. These include the use of hypertonic fluids, blood transfusion, push-dose vasopressors, and even extra-corporeal membrane oxygenation (ECMO).[3–6] However, these therapies are not without significant resource requirements as well as adverse effects, underscoring the importance of accurate identification of impending arrest.

Our preliminary work explored systolic blood pressure (SBP) patterns during deterioration.[7] Using observational data from helicopter emergency medical services (HEMS) arrest victims, we identified a clear SBP inflection point at 80 mmHg, beyond which arrest was imminent. However, SBP was limited by a lack of differentiation between the inflection point and antecedent values, challenging the use of SBP alone as an indicator for aggressive rescue therapy. In addition, the time between the inflection point and cardiac arrest was potentially too short to allow rescue.

This pilot analysis motivated a search for additional hemodynamic variables that may better indicate early deterioration as well as impending arrest due to shock. We identified several desirable qualities for the ideal physiological parameter: (a) consistency in the pre-arrest pattern across multiple patients, (b) a gradual change from a relatively normal baseline over 30–45 min prior to arrest, and (c) a clear pre-arrest inflection point after which deterioration accelerates but still leaves adequate time for intervention. We hypothesized that another physiological parameter or the combination of multiple parameters (shock index) would have more utility for early identification and potential reversal of deterioration.

Methods

Design

This was a retrospective observational analysis which utilized data archived from a cohort of adult patients experiencing HEMS-witnessed cardiopulmonary arrest. Waiver of informed consent was granted by the Air Methods Institutional Review Board.

Setting

Air Methods Corporation is a privately-owned company providing air medical emergency transport services to patients requiring intensive medical care. Helicopter crews consisting of a flight nurse and critical care paramedics respond to both scene and interfacility calls from 160 bases located in 48 states. Crews document clinical variables in an electronic patient care record (ePCR). In addition, performance improvement data for patients meeting specific criteria (invasive airway management, advanced procedure, HEMS-witnessed arrest) are entered into the Air Methods continuous quality improvement (CQI) database for additional analysis and tracking.

Subjects

Eligible patients were identified in the Air Methods CQI database between November 2015 and June 2016. Inclusion criteria are as follows: adult patients (age >14 years) experiencing HEMS-witnessed cardiopulmonary arrest due to a presumed perfusion-related etiology as per the Advanced Resuscitation Training (ART) taxonomy.[8] This included patients with suspected sepsis, hemorrhage, congestive heart failure, massive pulmonary embolus, tension pneumothorax, or pericardial tamponade. These patients had a diagnosis related to these etiologies as well as pre-arrest evidence of hemodynamic instability. Patients experiencing arrest due to dysrhythmia, asphyxia, or a fatal brain injury were excluded.

Data collection and analysis

Clinical data for eligible subjects were abstracted from both the ePCR as well as the Air Methods CQI database. This included demographic data (age, gender), clinical category (trauma, burn, congestive heart failure, sepsis, other non-traumatic diagnosis), pre-arrest vital signs (SBP, heart rate, respiratory rate, Glasgow Coma Scale score, end-tidal CO_2), and therapeutic interventions (including fluids, vasopressor therapy, insertion of an advanced airway). Vital signs are acquired every five minutes (or more frequently as indicated) by protocol. Recorded values are stored in the monitor and printed out for later entry into the ePCR. Values for $EtCO_2$ are obtained in all patients undergoing positive-pressure ventilation, either during bag-valve-mask ventilation or following insertion of an advanced airway.

The following hemodynamic parameters were selected for inclusion in this analysis: Systolic Blood Pressure (SBP), mean arterial pressure (MAP), heart rate (HR), shock index (heart rate/SBP), and end-tidal CO2 ($EtCO_2$). Other physiological measures (respiratory rate, Glasgow Coma Scale score) were considered but excluded due to the high percentage of patients who were intubated prior to arrest. For each of the included physiological parameters, measurements for all patients were plotted versus time with the moment of arrest defined as "time zero". Data up to 45 min prior to arrest were included. Polynomial regression was then used to create a best-fit curve for each parameter. Optimal goodness-of-fit was quantified by using the absolute value of "r" for various degrees for the polynomial equation. Inflection points on a curve are defined mathematically as transition points between concavity and convexity or between horizontal and vertical. For this analysis, we were interested in the latter, with horizontal (absolute value of slope <1) representing relative stability and vertical (absolute value of slope >1) representing instability. Thus, the derived equations were used to identify the point at which slope = −1 for SBP, MAP, HR, $EtCO_2$ and slope = 1 for shock index. In addition, the first and last recorded pre-arrest values for each hemodynamic parameter were compared using paired t-test. StatsDirect™ (Cheshire, UK) was used for all statistical calculations. Statistical significance was assumed for a p-value less than 0.05.

Results

A total of 70 patients with HEMS-witnessed arrest due to shock etiology with complete vital signs available during the study period were identified. Demographic and clinical data are displayed in Table 1. Polynomial curves were generated for each of the

Table 1 – Demographic and clinical data.

Parameter	Mean or % (95% confidence intervals)
Age (years)	43.2 (37.7–48.8)
Male gender (%)	67.1 (55.9–78.4)
Traumatic mechanism (%)	71.4 (60.6–82.3)
Positive-pressure ventilation prior to arrest (%)	90.0 (80.8–95.1)
Intubated prior to arrest (%)	47.1 (35.9–58.7)
Fluid therapy prior to arrest (%)	90.0 (80.8–95.1)
Vasopressor therapy prior to arrest (%)	28.6 (19.3–40.1)

hemodynamic parameters. All inflection points were identified 2–5 min prior to arrest (See Figs. 1–5). The SBP inflection point of 80.7 mmHg was lower than the initially recorded SBP value of 96.2 mmHg (Fig. 1). The MAP inflection point of 61.9 mmHg was only slightly lower than the initially recorded MAP of 65.3 mmHg (Fig. 2) and was encompassed by the 95% confidence intervals. Similarly, the HR inflection point of 78.2 bpm was slightly lower than the initial HR of 82.1 bpm (Fig. 3) but was also encompassed by the 95% confidence intervals. In addition, both the initial and inflection point values fall within the normal HR range. The shock index inflection point of 1.1 was higher than the initial value of 0.9 (Fig. 4) but represents the upper boundary of the 95%

confidence intervals. The $EtCO_2$ inflection point of 24.7 mmHg was well below the initial $EtCO_2$ value of 34.4 mmHg (Fig. 5) and was not encompassed by the 95% confidence intervals.

Discussion

Vital signs have always been critical in risk stratifying, triaging, and managing patients.[9–13] In addition to reflecting the severity of illness, identifying parameters that reliably predict impending cardiac arrest should trigger the use of aggressive therapies designed to prevent further decompensation and associated morbidity and mortality. Here we explore pre-arrest patterns for five physiological parameters in a
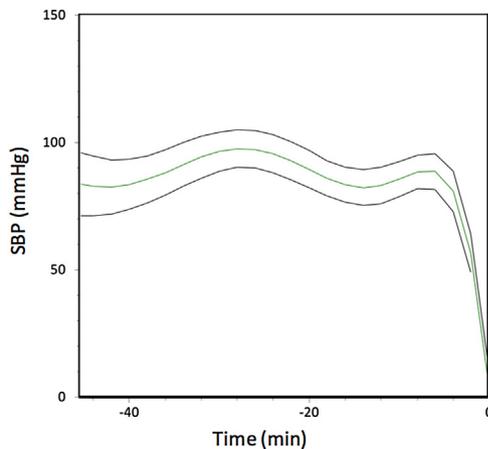

Fig. 1 – Systolic blood pressure versus time to arrest. Initial value 96.2 mmHg. Inflection point 80.7 mmHg. Inflection time −4 min. The outer lines represent the 95% confidence intervals around the best-fit curve.

Fig. 3 – Heart rate versus time to arrest. Initial value 82.1 BPM. Inflection point 78.2 BPM. Inflection time −2 min. The outer lines represent the 95% confidence intervals around the best-fit curve.
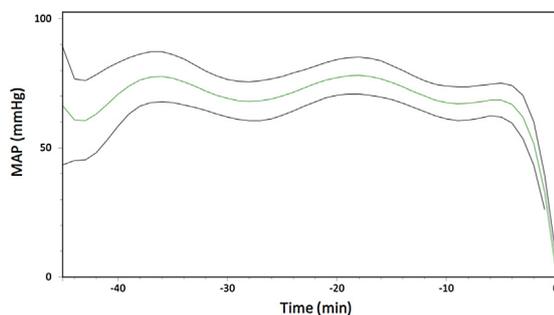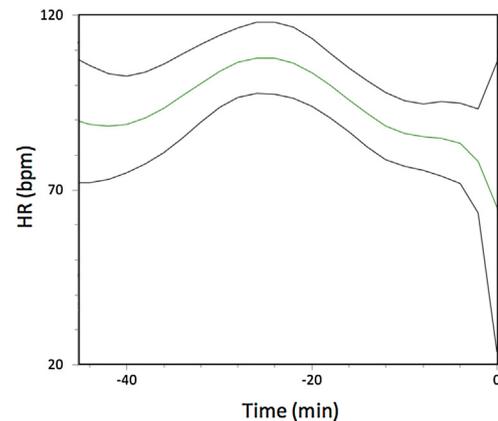

Fig. 2 – Mean arterial pressure versus time to arrest. Initial value 65.3 mmHg. Inflection point 61.9 mmHg. Inflection time −3 min. The outer lines represent the 95% confidence intervals around the best-fit curve.
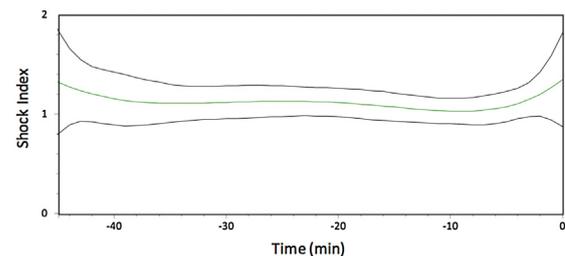

Fig. 4 – Shock index versus time to arrest. Initial value 0.9. Inflection point 1.1. Inflection time −4 min. The outer lines represent the 95% confidence intervals around the best-fit curve.
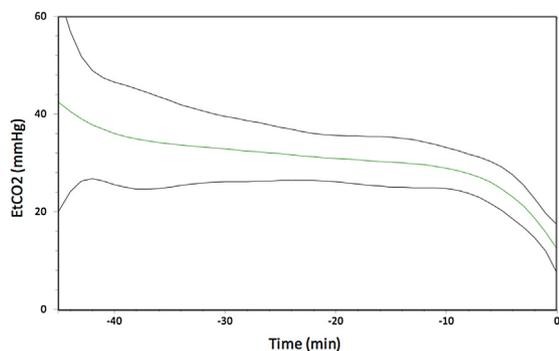
Fig. 5 – End-tidal CO$_2$ versus time to arrest. Initial Value 34.4 mmHg. Inflection point 24.7 mmHg. Inflection time −5 min. The outer lines represent the 95% confidence intervals around the best-fit curve.

cohort of air medical patients with a presumed shock-related etiology of arrest. For each parameter, the deterioration pattern was non-linear, with a clear inflection point identified 2–5 min prior to arrest. This supports the feasibility of identifying a point where more aggressive therapies should be employed. However, initially recorded values were similar to inflection point values for most parameters. This makes it difficult to determine when a change that crosses the inflection point threshold is only temporary versus a "point of no return" with impending arrest. The notable exception was EtCO2, which displayed a gradual descent from normal over 30–40 min, followed by an acceleration in deterioration to arrest once the threshold of 25 mmHg was crossed.

It is notable that the blood pressure parameters (SBP, MAP) demonstrated relatively small differences between initial and inflection point values. This may reflect the physiology of compensation, with natural and clinical mechanisms employed that defend pressure above critical thresholds.[14] The HR pattern included an anticipated rise then terminal drop.[15,16] However, initial and inflection point values were similar and fell into the normal range for HR, limiting the utility of this parameter in identifying impending collapse. Shock index displayed a desirable pattern of steady increase with terminal acceleration, but initial values may not differ enough from inflection point values to be clinically useful.[17,18] In addition, vital sign frequency may not allow rapid identification of a terminal descent prior to arrest. These patterns may justify a transition towards more continuous vital sign acquisition.

The favorable EtCO$_2$ pattern warrants additional investigation using this parameter to guide monitoring and treatment of critical patients. Carbon dioxide is a byproduct of cellular metabolism, traveling through the venous system to be delivered to the lungs and diffused across the alveolar membrane for exhalation. This requires adequate cardiac output and appropriate pulmonary ventilation-perfusion (V/Q) matching. With progression of shock, an increasing proportion of lung is not perfused, resulting in functional dead space that "dilutes" measured EtCO$_2$ values.[19] Thus, EtCO$_2$ is a reasonable surrogate for cardiac output and tissue perfusion.[20–23] Kheng et al. observed good correlation between EtCO$_2$ and blood pressure as well as serum lactate, bicarbonate, and base excess in 103 hypotensive patients.[22] In addition, Chandrasekharan et al. demonstrated that EtCO$_2$ relates to chest compression quality and tissue perfusion in a large mammalian model of asphyxial arrest.[24] Current arrest guidelines define EtCO$_2$ as a marker of compression quality as well as a

potential indicator of return of spontaneous circulation, with the threshold of 25 mmHg identical to the inflection point identified here.[25] We believe that EtCO$_2$ should be used in all critically ill and injured patients, not only to guide ventilation but also as a measure of perfusion. In addition, future investigations should explore the combination of EtCO$_2$ with other vital signs to improve the ability to predict deterioration or reflect the appropriateness of therapeutic efforts.

Most prior efforts to identify critical vital sign thresholds have focused on longer-term outcomes, such as death before hospital discharge, rather than imminent cardiac arrest.[9–12,26–31] These studies balance sensitivity and specificity for prognostication or triaging decisions, such as primary transport to a trauma or cardiovascular center by EMS providers. Vital sign-based decision rules generally err on the side of sensitivity to avoid preventable deaths, since overtriage generally does not carry the risk of patient harm.[32] Even with identification of critical thresholds beyond which the risk of death increases sharply, mortality generally remains quite low despite the vital sign abnormalities. In contrast, we focused on pre-arrest physiology, with all patients suffering eventual cardiac arrest. Thus, it is not surprising that our SBP inflection point is lower than the traditional 90 mmHg to define hypotension or with recent studies suggesting increased mortality with only mildly diminished SBP values.[9–12,26–31] Similarly, our previous work with EtCO$_2$ in trauma patients identified increased mortality at a higher threshold of 30 mmHg, and our shock index inflection point is higher than the traditional threshold of 1.0.[18,33,34]

Limited previous investigations have focused on pre-arrest SBP values. Brunauer et al. observed a mean SBP of 47 mmHg in critically ill ICU patients immediately prior to arrest.[35] While they did not focus on pre-arrest patterns or identification of an inflection point signifying a terminal slide into arrest, examination of the figures from their study suggests an inflection point of 75–80 mmHg, which is consistent with our data. Of note, they employed a novel definition of arrest that included a precipitous drop in HR, which is consistent with the pattern we observed here.

These results must be considered in light of study limitations. The study cohort was relatively small and somewhat heterogeneous, although we limited the analysis to patients with cardiac arrest due to shock. In addition, abstracted data were restricted by input by providers into the ePCR. Not all patients had complete data for the entire 45-min pre-arrest period. This was particularly true for EtCO$_2$, which required the use of positive-pressure ventilation to acquire. This resulted in wider confidence intervals for certain parameters, particularly for earlier measurements.

Only patients suffering cardiac arrest were included in this analysis. To validate the predictive ability of these identified patterns, all patients with hemodynamic shock would need to be studied to determine their specificity in identifying impending arrest. Alternatively, the significance of these findings includes identification of opportunities to prevent arrest. Thus, these patterns have been incorporated into educational materials as well as protocols for rescue therapies, such as push-dose vasopressors.

The accuracy of particular vital signs may also be challenged in the pre-arrest state. While HR is fairly reliable when acquired via electrocardiogram, most non-invasive blood pressure systems are calibrated for accuracy at pressures much higher than the pre-arrest inflection points explored here. In addition, EtCO$_2$ is affected by ventilation rate and tidal volume, which we did not attempt to account for here, although standardized ventilation parameters designed to

achieve adequate lung inflation and to avoid hyperventilation were employed. Furthermore, we did not acquire $EtCO_2$ values in spontaneously breathing patients. Although most patients had positive-pressure ventilation prior to cardiac arrest, this may have been performed late in the course of deterioration and affected recorded $EtCO_2$ values.

We did not attempt to account for the influence of various therapeutic interventions. All patients included in this study had various combinations of fluids, medications, and other disease-specific interventions as part of resuscitative efforts. The protocols employed during the study period did not include push-dose vasopressor therapy, blood product administration, or ECMO. This may limit generalizability to the hospital setting, where these therapies are used routinely. However, the use of intravenous fluids, vasopressor drips, and needle/tube thoracostomy clearly influence perfusion status but were not incorporated into our mathematical models. Finally, we did not attempt to determine long-term outcomes for these patients, who have generally poor prognoses with non-dysrhythmic arrests. Ultimately, the use of these parameters to help guide resuscitative therapies is the most important measure of their value.

Conclusions

Vital signs followed predictable pre-arrest patterns in a cohort of air medical shock victims. Blood pressure values (SBP, MAP) hovered above critical thresholds, with patients rapidly deteriorating into arrest once these were violated. The pre-arrest pattern for HR included a gradual rise followed by a terminal drop as anticipated, although initial and inflection point values fell within a physiologically normal range. Shock index increased slightly during the 45-min pre-arrest period. The $EtCO_2$ appeared most useful, with a gradual decline from normal and a sharp acceleration once values decreased below 25 mmHg. Future research should explore the use of vital sign thresholds as a trigger for more aggressive rescue therapies.

Conflicts of interest

None.

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