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Spectral analysis of heart rate variability predicts mortality and instability from vascular injury

Kiavash R. Koko, MD, Brian D. McCauley, MD, John P. Gaughan, PhD, Marc W. Fromer, MD, Ryan S. Nolan, MD, Ashleigh L. Hagaman, MD, Spencer A. Brown, PhD, and Joshua P. Hazelton, DO*

Cooper University Hospital, Camden, New Jersey

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ABSTRACT

Background: Spectral analysis of continuous blood pressure and heart rate variability provides a quantitative assessment of autonomic response to hemorrhage. This may reveal markers of mortality as well as endpoints of resuscitation.

Methods: Fourteen male Yorkshire pigs, ranging in weight from 33 to 36 kg, were included in the analysis. All pigs underwent laparotomy and then sustained a standardized retrohepatic inferior vena cava injury. Animals were then allowed to progress to class 3 hemorrhagic shock and where then treated with abdominal sponge packing followed by 6 h of crystalloid resuscitation. If the pigs survived the 6 h resuscitation, they were in the survival (S) group, otherwise they were placed in the nonsurvival (NS) group. Fast Fourier transformation calculations were used to convert the components of blood pressure and heart rate variability into corresponding frequency classifications. Autonomic tones are represented as the following: high frequency (HF) = parasympathetic tone, low frequency (LF) = sympathetic, and very low frequency (VLF) = renin-angiotensin aldosterone system. The relative sympathetic to parasympathetic tone was expressed as LF/HF ratio.

Results: Baseline hemodynamic parameters were equal for the S ($n = 11$) and NS groups. LF/HF was lower at baseline for the NS group but was higher after hemorrhage and the resuscitation period indicative of a predominately parasympathetic response during hemorrhagic shock before mortality. HF signal was lower in the NS group during the resuscitation indicating a relatively lower sympathetic tone during hemorrhagic shock, which may have contributed to mortality. Finally, the NS group had a lower VLF signal at baseline (e.g., [S] 16.3 ± 2.5 *versus* [NS] 4.6 ± 2.9 $P < 0.05$,) which was predictive of mortality and hemodynamic instability in response to a similar hemorrhagic injury.

Conclusions: An increased LF/HF ratio, indicative of parasympathetic predominance following injury and during resuscitation of hemorrhagic shock was a marker of impending death. Spectral analysis of heart rate variability can also identify autonomic lability following hemorrhagic injuries with implications for first responder triage. Furthermore, a decreased VLF signal at baseline indicates an additional marker of hemodynamic instability and marker of mortality following a hemorrhagic injury. These data indicate that

* *Corresponding author*. Cooper University Hospital, 3 Cooper Plaza Suite 411, Camden, NJ 08103. Tel.: (714) 336-0837; fax: (856) 365-7582.
E-mail address: koko-kiavash@cooperhealth.edu (K.R. Koko).
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continuous quantitative assessment of autonomic response can be a predictor of mortality and potentially guide resuscitation of patients in hemorrhagic shock.

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Background

Uncontrolled hemorrhage remains the most common cause of preventable death among trauma patients who survive long enough to receive treatment in a hospital.[1] There is a bimodal distribution of traumatic deaths associated with hemorrhage which necessitates rapid and accurate triage of patients in hemorrhagic shock. Currently, the field triage of patients in occult hemorrhagic shock relies on interpretation of traditional vital signs such as heart rate and blood pressure along with identifying signs of hemorrhage. Unfortunately, physiologic compensatory response to hemorrhage may prevent the early changes of vital signs, thus inspiring the search for alternative methods of early detection of hemorrhagic shock.[2-7]

Hypovolemia, secondary to hemorrhagic shock, is compensated primarily by the autonomic nervous system.[3] Animal models of hemorrhagic shock monitored through direct invasive neural activity have shown that sympathetic tone initially drives the hemodynamic compensation of early hemorrhagic shock; however, as hemorrhage progresses, the sympathetic tonal response becomes overwhelmed. Therefore, decreasing neural regulatory activity is a marker of irreversible hemorrhagic shock.[8,9] This physiologic phenomenon highlights the potential role of monitoring neural regulation and cardiovascular autonomic regulation to help guide the resuscitation of patients in traumatic hemorrhage.

Applying heart rate variability to quantify the autonomic compensatory response to traumatic hemorrhage has revealed several important insights. Cooke *et al.*[3,10] revealed higher parasympathetic than sympathetic modulation in traumatic injuries, and that heart rate variability is inversely correlated to decreased central volume. Various models and data acquisition techniques have also emerged to further investigate this application in hemorrhagic shock.[11,12] Beyond sympathetic and parasympathetic tone, Ryan *et al.*[5] revealed that decreased very low-frequency (VLF) signal which corresponds to contributions from the renin-angiotensin aldosterone system (RAAS) is an independent predictor of mortality and morbidity in hemodynamically stable trauma patients. Continuously and noninvasively measuring the autonomic compensatory response to traumatic hemorrhage may provide further valuable insights into the physiology of hemorrhagic shock, whereas previous quantification mechanisms relied on invasive methods of monitoring sympathetic nerve activity such as microneurography, which involves invasive electrode placement in various nerves such as the peroneal nerve or renal sympathetic nerves.[9,13,14] Power spectral analysis provides a noninvasive method to quantify autonomic compensatory response using simple electrocardiogram (ECG) or continuous blood pressure monitoring. Power spectral analysis of heart rate variability involves analyzing the R-R interval oscillations from an ECG tracing into frequency components which represent sympathetic and parasympathetic response. Quantitatively analyzing the autonomic response may provide prognostic information to guide the management of patients after traumatic injury.[3,15-18]

Evaluation of the parasympathetic to sympathetic tone has been used as a predictor of mortality in the critically ill patient population.[15] Heart rate variability has also been applied to aide in prehospital triage of traumatically injured patients.[10] The analysis of heart rate variability has not been widely applied to the resuscitation of patients in hemorrhagic shock; however, it may potentially detect the early changes in the compensatory autonomic response to hemorrhage that precede the changes in vital signs.

The purpose of our study is to apply power spectral analysis of heart rate variability to noninvasively identify early markers of mortality in a penetrating traumatic major vascular hemorrhage model, thus allowing us to facilitate early effective intervention for hemorrhagic shock. Furthermore, we aim to apply power spectral analysis to quantitatively assess autonomic compensation and the adequacy of resuscitation in a swine hemorrhage model. We hypothesize that quantifying autonomic compensation to hemorrhagic shock will allow ongoing assessment of the efficacy of resuscitation of an animal in hemorrhagic shock.

Methods

Experimental design

This study is a prospective observational preclinical study conducted with the approval of the University of Sciences Institutional Animal Care and Use Committee (Philadelphia, PA). All animals were cared for according to the National Institutes of Health Guide for the Care and Use of Laboratory Animals.[19] Fourteen male Yorkshire swine, each weighing 34.4 ± 2.2 (95% confidence interval, 30.5-38.0), were used in this study. Animals were housed indoors in enclosed groups of two. A complete corn-soybean meal–based ration was fed at a 2.0 kg/pg per day, and water was available *ad libitum*. All animals received care in strict compliance with the Guide for the Care and Use of Laboratory Animals (National Research Council, 1996).

Specific details regarding the swine hemorrhage model are described further by Koko *et al.*[20] Animals were sedated using an intramuscular injection of ketamine (25 mg/kg), midazolam (0.5 mg/kg), and glycopyrrolate (0.008 mg/kg.) The swine were placed on an operating table with continuous ECG and pulse oximetry. A 20-gauge peripheral intravenous line was placed into an ear vein through which they received a 500 mL bolus of 5% dextrose and lactated ringer's solution to prevent hypotension and hypoglycemia on induction of anesthesia. Anesthesia was induced with 4% bolus of isoflurane via face cone, and the swine were intubated with a size

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