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Heart Rate Variability as a Predictor of Death in Burn Patients

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Abstract

Objective—Heart rate variability (HRV), a noninvasive technique used to quantify fluctuations in the interval between normal heartbeats, is a predictor of mortality in some patient groups. The aim of our study was to assess HRV in burn trauma patients as a predictor of mortality.

Methods—We prospectively performed 24 hour Holter monitoring on burn patients and collected demographic information, burn injury details, and in-hospital clinical events. Analysis of HRV in the time and frequency domains was performed.

Results—A total of 40 burn patients with a mean age of 44 ± 15 years were enrolled. Mean % total body surface area burn was $27 \pm 22\%$ for the overall population and was significantly higher in those who died compared to those who survived ($55 \pm 23\%$ vs. $19 \pm 13\%$, $p < 0.0001$). There was a statistically significant inverse linear correlation between standard deviation of NN intervals (SDANN) and % total body surface area ($-r 0.337$, $R^2 = 0.113$, 95% CI -0.587 to -0.028 , 2-tailed $p = 0.034$), as well as with ultra low frequency (ULF) power and % total body surface area burn ($-r 0.351$, $R^2 = 0.123$, 95% CI -0.152 to -0.009 , $p = 0.027$). The receiver-operator characteristic showed the area under the curve for % total body surface area as a predictor of death was 0.82 ($p < 0.001$), for SDANN was 0.94 ($p < 0.0001$), and for ULF power was 0.96 ($p < 0.0001$).

Conclusions—Deranged HRV in the early post-burn period is a strong predictor of death.

Keywords

Heart Rate Variability; Burn Trauma

Introduction

Heart rate variability (HRV) is a noninvasive technique used to quantify fluctuations in the interval between normal heartbeats (1–3). These fluctuations are mediated by autonomic

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input to the sinus node and reflect spontaneous modulations in autonomic tone (4). For most clinical applications HRV is analyzed by time and frequency domain methods. Time domain analyses are simple statistical measures of the distribution of normal RR intervals (referred to as “NN” intervals). Frequency domain analyses provide information about how the variance (i.e. power) in the heart’s rhythm due to oscillations of heart rate distributes as a function of frequency. The standard approach, called power spectral density analysis, calculates the power within four bands: (1) high frequency power (0.15 to 0.40 Hz) which corresponds to vagal input, (2) low frequency power (0.04 to < 0.15 Hz) which corresponds to sympathetic and parasympathetic input, (3) very low frequency power (0.0033 to < 0.04 Hz), thought to represent the influence of the peripheral vasomotor and renin-angiotensin systems, and (4) ultra low frequency power (< 0.003 Hz) thought to reflect circadian and neuroendocrine rhythms (1). Diminished HRV has been documented in diverse conditions including myocardial infarction (5), diabetes (3), and congestive heart failure (5), and is associated with an increased cardiac mortality (3). Retrospective studies using short-term recordings have shown HRV to be a predictor of mortality in trauma patients who have sustained a wide range of injuries (6–9). There has been limited application, however, of HRV to predict clinical outcomes in burn patients, a population in whom myocardial dysfunction has been reported (10). The aim of our study was to assess cardiac autonomic modulation in the immediate post-burn period using long-term HRV monitoring and to determine whether it is a predictor of mortality.

Methods

Following IRB approval, we prospectively performed 24 hour Holter monitoring on consented patients 18 to 65 years old, admitted to an academic burn unit. We prospectively collected demographic information, details about the burn injury, and in-hospital clinical events. Exclusion criteria included patients with persistent atrial or ventricular arrhythmias or paced rhythms on admission, and those with such extensive burns located on the chest and back that placement of patches for the Holter monitor electrodes would be contra-indicated.

Variables assessed

The digitally sampled electrocardiographic data were transferred from the Delmar Avionics scanner (Holter Analysis System model 563, Irvine, California) to a computer (StrataScan, software version F, Delmar, Irvine, California) for analysis of HRV which was measured in accordance to previously described techniques (11). The computer program detected and labeled each QRS complex and rejected the RR intervals that differed in duration by >30% from the previously accepted interval to exclude artifact or ectopic beats. Only segments from which >90% of the RR intervals fulfilled these criteria were included in the final analysis. At least 20 hours of analyzable data were required for the Holter recording to be accepted for analysis. All Holter tapes were analyzed without knowledge of the patient’s clinical status.

Analysis of HRV in the time domain was performed for the entire recording (daytime and nighttime) using descriptive statistical methods. The SD of all NN intervals (SDNN), the SD of the average NN intervals over short periods (5-minute segments) for the entire recording

(SDANN), and the root-mean-square of successive differences between adjacent normal NNs (rMSSD) were measured in milliseconds.

Analysis of HRV in the frequency domain was accomplished by computing the power spectra on 5-minute epochs of each recording using Fast Fourier transformation, a mathematical algorithm that apportions the HRV signal into its frequency components and quantifies the power of each component. The sum of all components (total power spectral density) between 0.003 and 0.40 Hz was calculated. Subsequently, the power in 4 bands was quantified: 0.15 to 0.40 Hz (high frequency (HF) power, corresponding to vagal input), 0.04 to < 0.15 Hz (low frequency (LF) power corresponding to sympathetic and parasympathetic input), 0.0033 to < 0.04 Hz (very low frequency (VLF) power, thought to represent the influence of the peripheral vasomotor and renin-angiotensin systems), and 0.003 HZ (ultra low frequency (ULF) power thought to reflect circadian and neuroendocrine rhythms) (3).

Statistical analyses

Frequency domain variables without normal distribution were transformed using natural logs prior to statistical analysis, as previously described (12, 13). Categorical variables were expressed as the number and percentage of patients. Student's *t* test and *Chi* square test were used to compare variables between burn patients who survived and those who died. Linear regression was used to assess the correlation of time and frequency domain measures to % total body surface area burn. To evaluate the performance of % total body surface area burn, and HRV variables on predicting death, the area under the curve of the receiver-operating characteristic curve was calculated. The area under the receiver-operating characteristic curve was also calculated for combinations of variables hypothesized to be associated with death in burn trauma patients. Specifically, the area under the receiver-operating characteristic curve was calculated for logistic regression models of the following combinations: age plus % total body surface area burn, % total body surface area burn plus SDANN, and % total body surface area burn plus ULF power. The DeLong method was used to evaluate the differences between the area under the receiver-operating characteristic curves (14). Two-sided probability values were considered statistically significant if they were less than 0.05. Data were analyzed on a Macintosh computer using PRISM statistical software (Version 6.0, GraphPad Software, Inc. La Jolla, California, USA). Logistic regression models and area under the receiver-operating characteristic curve analyses were performed by R.C.S. using R version 3.0.2 (15).

Results

A total of 45 patients signed informed consent, but 5 were excluded due to insufficient HRV data. Therefore, a total of 40 burn patients with a mean age of 44 ± 15 years were included in the analysis (Table 1). Overall, co-morbid medical conditions including hypertension, diabetes and chronic obstructive lung disease were infrequent and not significantly different between those who survived and those who died. The majority of the injuries were secondary to flame, gasoline, and electrical burns. The average time from the burn injury to placement of the ECG patches and the start of the 24 hour Holter monitoring was 2.1 ± 1.4 hours; there was no difference in this timing variable between those who survived and those

who died. Mean % total body surface area burn was $27 \pm 22\%$ for the overall population and was significantly higher in those who died compared to those who survived ($55 \pm 23\%$ vs. $19 \pm 13\%$, $p < 0.0001$). In addition, burns involving the back were significantly higher in patients who died (67% vs. 26% , $p = 0.044$). The majority of the patients (78%) required admission to the burn intensive care unit (Table 2). Patients who subsequently died had longer ICU stays and also remained intubated longer than those who survived. Otherwise, there were no other significant differences between the two groups regarding in-hospital events. Of the 9 patients who died the cause of death was reported to be complication of wounds ($n=2$), respiratory failure ($n=1$), sepsis ($n=3$), and multi-system organ failure ($n=3$).

Correlations between % total body surface area burn and the time domain measures of HRV (SDANN, SDNN, and rMSSD) were calculated. There was a weak but statistically significant inverse linear correlation between SDANN and % total body surface area ($-r$ 0.337, $R^2=0.113$, 95% CI -0.587 to -0.028 , 2-tailed $p=0.034$) (Figure 1, left panel), whereas there was no correlation with SDNN or rMSSD. Correlations between % total body surface area burn and the frequency domain measures of HRV (ULF, VLF, LF and HF) showed a weak but a statistically significant inverse linear correlation between ULF and % total body surface area burn ($-r$ 0.351, $R^2=0.123$, 95% CI -0.152 to -0.009 , $p=0.027$) (Figure 1, right panel), whereas there was no correlation with LF, VLF or HF power. Because of the significant correlations with the time domain measure of SDANN and the frequency domain measure of ULF power with respect to % total body surface area burn, these two measures of heart rate variability were then assessed for their ability to predict death in our study population. The receiver-operator curves showed that the area under the curve for % total body surface area as a predictor of death was 0.82 ($p < 0.001$) (Figure 2, panel A), for SDANN was 0.94 ($p < 0.0001$) (Figure 2, panel B), and for ULF was 0.96 ($p < 0.0001$) (Figure 2, panel C). A model including % total body surface area burn and age was not significantly different than SDANN alone ($p=0.450$), or a model including % total body surface area burn and ULF power ($p=0.125$) (Figure 3, panels A and C). The area under the curve was higher for the model that combined SDANN and % total body surface area burn, but it was not statistically significant when compared to the model including % total body surface area burn and age (98% vs. 84% , $p=0.057$) (Figure 3, panel B).

Discussion

HRV is a non-invasive method of assessing autonomic modulation of the heart. Our results indicate that abnormal HRV during the immediate post-burn period is strongly predictive of death. While we found % total body surface area burn to be a predictor of death, consistent with other investigators, our data suggest that incorporating the HRV time domain parameter of SDANN and frequency domain parameter of ULF power may result in improved risk prediction in this population. We found statistically significant associations with death and low values of SDANN and ULF power.

The concept that impaired cardiovascular function adversely affects clinical outcomes following burn injury was first proposed more than 80 years ago (16) and since then has been studied by many investigators. Horton and her research group studied many factors thought to be associated with cardiac dysfunction in animal models of post-burn injury

including the inflammatory response and susceptibility to sepsis (17), the disruption of normal coronary endothelial function (18), and the effect of estrogen on burn-induced myocardial injury (19). One interesting feature is that while burn injury is associated with intrinsic myocardial contractile defects in animal models, in humans, cardiac output is usually maintained despite objective evidence of myocyte death (elevated troponin-I levels). In one study of otherwise healthy subjects without known coronary artery disease serum levels of troponin-I were elevated in all subjects who sustained a severe burn (>18% total body surface area) despite demonstrating hyperdynamic cardiac function and high cardiac index during the resuscitation period (20). Thus, finding alternative non-invasive methods to identify individuals who are at risk for a complicated post-burn course led to the study of HRV in this population.

The clinical relevance of HRV was first noted when investigators noticed that fetal distress was preceded by changes in beat-to-beat intervals before there was any appreciable change in the baseline heart rate (21). More recently investigators have used HRV to identify infants at high risk for sepsis and death (22, 23). In another condition triggered by severe emotional or physical stress called Takotsubo cardiomyopathy, patients present with depressed left ventricular function, elevated troponin levels, and angiographically normal coronary arteries (24). HRV in this condition has been assessed by several groups who both found that HRV was depressed during the acute setting, but significantly increased after recovery (25, 26). Results of these studies suggest that HRV is a marker of autonomic dysregulation that occurs as a result of severe illness.

While HRV has been studied in other trauma populations, little is known about HRV in burn trauma patients specifically. In a large, retrospective, single-center analysis of general trauma patients, investigators showed that worsening acidosis, coagulopathy and bleeding was associated with decreased HRV and increased mortality (8). While our results are consistent with their findings, there are several important differences between our studies. First, they measured HRV using short-term recordings from with the intensive care unit electrocardiographic monitoring system, not by obtaining 24 hour Holter monitoring. While these short-term recordings allowed them to calculate a variable similar to the time domain variable called SDANN, they were not able to measure other time domain variables or frequency domain variables which requires instantaneous heart rate to be acquired at every beat (1). Second, they did not provide information regarding medical co-morbidities and pre-admission medications. Third, they did not differentiate their study population according to the type of trauma sustained, making it impossible to gain any meaningful insight into the predictive value of HRV in burn trauma per se. In one small retrospective study of 13 burn patients, investigators assessed HRV using short-term electrocardiographic recordings upon admission and again at 8, 12, 24, and 36 hours to determine if it changed with resuscitation efforts (27). They found that HRV was abnormally low on admission and successful resuscitation was associated with progressive improvement in HRV.

The idea of altering autonomic tone to influence outcome in severe trauma has been studied. In one study consisting of a general trauma population, investigators showed that patients who were already on chronic beta-blocker therapy had significantly less severe initial base deficit than those who were not taking beta-blockers (28). In another study focused on a

burn trauma population, investigators showed that prior beta-blocker use was associated with decreased mortality (29). Both of these studies suggest that pre-injury beta-blockade may have a protective effect. In survivors of a remote myocardial infarction, beta-blocker therapy has been shown to enhance parasympathetic cardiac activity as assessed by time and frequency domain measures of HRV (30), suggesting that their influence on HRV is a reflection of their beneficial antiarrhythmic effect. It is uncertain whether this effect on HRV is similar in burn patients.

Our study has limitations. First, although prospective in design, ours is a small, single-centered study. Second, we do not have an assessment of left ventricular systolic function or levels of biomarkers reflecting myocardial damage such as troponin. Third, since few of the subjects were taking beta-blockers on admission, we were unable to assess whether beta-blockade played a role in HRV measures. Lastly, we do not have long-term follow-up data.

Conclusions

Deranged HRV in the early post-burn period is a strong predictor of death and may result in improved risk prediction when incorporated with % total body surface area burn. Larger studies of HRV in burn patients with longer clinical follow-up are needed to determine whether HRV assessment at the time of admission is prognostic of outcome. Importantly, this line of investigation may lend itself to possible therapeutic modalities including prophylactic administration of beta-blockers in this high-risk patient population.

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Figure 1.

Correlation between % total body surface area burn and the time domain variable SDANN (left panel), and the natural logarithm (Ln) of the frequency domain variable, ultra low frequency power (right panel). This figure shows a significant inverse correlation between % total body surface area burn and the heart rate variability measures of SDANN and ultra low frequency power demonstrating that as %total body surface area burn increases, both of these heart rate variability measures decrease. SDANN, standard deviation of the average NN intervals over short periods (5-minute segments) for the entire Holter recording.

Figure 2.

Receiver-operating characteristic curves for % total body surface area burn (panel A), the time domain variable, SDANN (panel B), and the frequency domain variable, ultra low frequency power (panel C) as predictors of death. This figure shows that the heart rate variability measures of SDANN and ultra low frequency power are comparable to % total body surface area burn with respect to predicting subsequent death. AUC, area under the curve; SDANN, standard deviation of the average NN intervals over short periods (5-minute segments) for the entire Holter recording.

Figure 3.

Receiver-operating characteristic curves for the combination of % total body surface area burn and age (panel A, $p=0.450$), the combination of % total body surface area burn and the time domain variable, SDANN (panel B, $p=0.057$), and the combination of % total body surface area burn and the frequency domain variable, ultra low frequency power (panel C, $p=0.125$) as predictors of death. This figure shows that compared to the receiver-operating characteristic curves for % total body surface area burn, SDANN, and ultra low frequency power (shown in Figure 2) there was no significant improvement in the prediction of death when age was added to %total body surface area burn, or when %total body surface area burn was added to the heart rate variability measures of SDANN and ultra low frequency power area. AUC, area under the curve; SDANN, standard deviation of the average NN intervals over short periods (5-minute segments) for the entire Holter recording; ULF, ultra low frequency.

Table 1

Baseline patient characteristics

Variable	Total n=40	Alive n=31	Dead n=9	p value
Mean age (years)	44 ± 15	44 ± 14	45 ± 18	0.768
Male gender	27 (68%)	23 (74%)	4 (44%)	0.120
White	10 (25%)	7 (23%)	3 (33%)	0.665
Black or African American	24 (60%)	18 (58%)	6 (67%)	0.717
Hispanic or Latino	6 (15%)	6 (19%)	0	0.307
Medical co-morbidities				
Hypertension	10 (25%)	8 (26%)	2 (22%)	1.000
Diabetes mellitus	5 (12.5%)	4 (13%)	1 (11%)	1.000
Tobacco use	11 (28%)	9 (29%)	2 (22%)	1.000
Chronic lung disease	3 (7.5%)	2 (6.5%)	1 (11%)	0.545
Beta-blocker use	5 (12.5%)	4 (13%)	1 (11%)	1.000
Admission pH < 7.3	6 (15%)	3 (10%)	3 (33%)	0.115
Height (inches)	67 ± 7	68 ± 7	66 ± 5	0.260
Weight (kilograms)	83 ± 20	84 ± 20	73 ± 19	0.352
Temperature (°celsius)	36.8 ± 0.9	36.9 ± 0.6	36.6 ± 1.9	0.202
Timing of burn (hours prior to arrival)	2.1 ± 1.4	2.22 ± 1.4	1.52 ± 1.2	0.256
Cause of burn				
Electrical	8 (20%)	7 (23%)	1 (11%)	0.655
Gasoline	9 (23%)	7 (23%)	2 (22%)	1.000
Hot water	2 (5%)	2 (6.5%)	0	1.000
Flame	15 (37%)	11 (35%)	4 (44%)	0.694
Motor vehicle accident	3 (7.5%)	3 (10%)	0	1.000
Explosion	3 (7.5%)	1 (3%)	2 (22%)	0.122
Area of Body Burned				
Face/neck	16 (40%)	11 (35%)	5 (56%)	0.441
Chest	15 (38%)	9 (29%)	6 (67%)	0.057
Back	14 (35%)	8 (26%)	6 (67%)	0.044
Abdomen	13 (33%)	8 (26%)	5 (56%)	0.120
Extremities	28 (70%)	22 (71%)	6 (67%)	1.000
Inhalation injury	8 (20%)	5 (16%)	3 (33%)	0.348
Total body surface area affected	27 ± 22%	19 ± 13%	55 ± 23%	<0.0001

Data shown as number, percentage (%), mean ± SD; p value reflects comparison between alive and dead.

Table 2**In-hospital events**

Variable	Total N=40	Alive N=31	Dead N=9	p value
Length of intubation (days)	5 ± 11	2 ± 7	17 ± 20	0.013
Length of stay in ICU (days)	8 ± 15	4.5 ± 12	24 ± 30	0.017
Length of total hospital stay (days)	18 ± 23	14 ± 18	37 ± 32	0.118
Requiring admission ICU	31 (78%)	22 (71%)	9 (100%)	0.089
Requiring vasopressors	2 (5%)	1	1	0.404
Requiring intubation	15 (37.5%)	10	5	1.000
Requiring multiple operations	8 (20%)	6	2	1.000
Electrolyte abnormalities *	10 (25%)	6	4	0.190
Pulmonary embolism	2 (5%)	1	1	0.404
Arrhythmias	4 (10%)	3	1	1.000
Sepsis	6 (15%)	4	2	0.602

Data shown as number, percentage (%), mean ± SD; p value reflects comparison between alive and dead. ICU= intensive care unit;

* includes hypernatremia, hyponatremia, hyperkalemia, hypokalemia, hypomagnesemia, and hypophosphatemia.