HEART RATE VARIABILITY AS A PREDICTOR OF CARDIAC DYSRHYTHMIAS DURING WEANING FROM MECHANICAL VENTILATION

By Muna H. Hammash, RN, PhD; Debra K. Moser, RN, DNPsc; Susan K. Frazier, RN, PhD; Terry A. Lennie, RN, PhD; and Melanie Hardin-Pierce, RN, APRN, ACNP-BC, DNP

Background  Weaning from mechanical ventilation to spontaneous breathing is associated with changes in the hemodynamic and autonomic nervous systems that are reflected by heart rate variability. Although cardiac dysrhythmias are an important manifestation of hemodynamic alterations, the impact of heart rate variability on the occurrence of dysrhythmias during weaning has not been specifically studied.

Objectives  To describe differences in heart rate variability spectral power and occurrence of cardiac dysrhythmias at baseline and during the initial trial of weaning from mechanical ventilation and to evaluate the impact of heart rate variability during weaning on occurrence of dysrhythmias.

Method  Continuous 3-lead electrocardiographic recordings were collected from 35 patients receiving mechanical ventilation for 24 hours at baseline and during the initial weaning trial. Heart rate variability was evaluated by using spectral power analysis.

Results  Low-frequency power increased \((P = .04)\) and high-frequency and very-low-frequency power did not change during weaning. The mean number of supraventricular ectopic beats per hour during weaning was higher than the mean at baseline \((P < .001)\); the mean of ventricular ectopic beats did not change. Low-frequency power was a predictor of ventricular and supraventricular ectopic beats during weaning \((P < .001)\). High-frequency power was predictive of ventricular and supraventricular \((P = .02)\) ectopic beats during weaning. Very-low-frequency power was predictive of ventricular ectopic beats \((P < .001)\) only.

Conclusion  Heart rate variability power spectra during weaning were predictive of dysrhythmias. \(\text{American Journal of Critical Care. 2015;24:118-127}\)
Cardiac dysrhythmias are a common and important clinical problem in patients receiving or being weaned from mechanical ventilation. Cardiac dysrhythmias can induce myocardial ischemia, impair myocardial contractility, and decrease cardiac index, eventually leading to unsuccessful weaning. Unsuccessful weaning is associated with prolonged duration of mechanical ventilation and increased intensive care unit (ICU) mortality, complications, and costs.

Changes in intrathoracic pressure and lung volume during weaning may induce marked hemodynamic changes in right and left ventricular preload and afterload; and intrathoracic blood volume and flow. In response, the autonomic nervous system (ANS) is activated and induces alterations in ventricular afterload, contractility, and heart rate to maintain cardiac output and tissue perfusion. The balance between the 2 components of autonomic tone, sympathetic and parasympathetic innervation, can be evaluated by measuring heart rate variability (HRV). HRV, the beat-to-beat variation in heart rate, is due primarily to ongoing changes in sympathetic and parasympathetic input to the sinoatrial node. During weaning, changes in the ANS with a shift to sympathovagal imbalance (ie, increased sympathetic and/or reduced vagal tone) may occur in patients who have underlying cardiovascular dysfunction and impaired compensatory mechanisms, resulting in decreased HRV. Sympathetic dominance is associated with decreased duration of myocardial action potential, and increased myocardial excitability, which may enhance abnormal automaticity, triggered activity, and reentry and produce cardiac dysrhythmias.

Numerous experimental and clinical studies have revealed a relationship between reduced HRV and the genesis of malignant ventricular dysrhythmias, paroxysmal atrial fibrillation, and atrial flutter, especially in patients with heart failure, myocardial ischemia, or myocardial infarction. Although dysrhythmias that develop during weaning are often clinically important and can lead to unsuccessful weaning, little research has been done on the relationship between HRV and occurrence of dysrhythmias during weaning. Therefore, the purpose of this study was to evaluate the impact of HRV as a noninvasive reflection of ANS tone during the initial weaning trial on occurrence of cardiac dysrhythmias. Clinically important cardiac dysrhythmias such as ventricular ectopy, ventricular tachycardia or fibrillation, atrial fibrillation or flutter, supraventricular tachycardia, and supraventricular beats were the focus of the study because of their potential adverse consequences in patients who are undergoing weaning trials. The specific aims of the study were to determine whether spectral power analysis indicated differences between HRV at baseline and during the initial weaning trial; determine whether occurrence of cardiac dysrhythmias at baseline and during the initial weaning trial differed; and determine the association between HRV measured during weaning and the occurrence of cardiac dysrhythmias, after controlling for baseline HRV measured during mechanical ventilation.

Methods

Design, Sample, and Setting

A descriptive correlational design was used to evaluate the impact of HRV during initial weaning from mechanical ventilation on the occurrence of cardiac dysrhythmias. Data were collected on cardiac dysrhythmias and HRV twice, at baseline (for 24 hours during mechanical ventilation) and during the initial weaning trial (up to 2 hours). A total of 35 adult patients from the medical, surgical, cardiac care, and trauma ICUs at the University of Kentucky Chandler Medical Center, Lexington, Kentucky, were enrolled in the study between 2008 and 2009. Patients were included in the study if they were 18 years or older and received mechanical ventilation for a minimum of 24 hours via an endotracheal tube.

About the Authors

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Patients were excluded if they had experienced a myocardial infarction within the preceding 3 months or were admitted with a myocardial infarction, were undergoing cardiac surgery or had cardiac surgery within the previous 3 months, had neurological trauma or stroke within the previous 3 months or were admitted with neurological trauma or stroke, were in atrial fibrillation or had paroxysmal atrial fibrillation, had a pacemaker or implanted cardioverter defibrillator, or had a pulmonary artery catheter or central venous catheter that was not in the proper position as confirmed by radiography.

Measures

Sociodemographic Characteristics. Data on age, sex, marital status, ethnicity, and type of insurance were abstracted from the medical records at baseline when a patient was enrolled in the study.

Clinical Data. Clinical data on previous need for mechanical ventilation, history of cardiac diseases and dysrhythmias, score on the Acute Physiology and Chronic Health Evaluation (APACHE) IV, physiological status (temperature, score on the Glasgow Coma Scale, results of arterial blood gas analyses, electrolyte levels, complete blood cell count), pulmonary status (respiratory rate, tidal volume, minute volume, airway pressure and resistance), cardiovascular status (blood pressure, heart rate, central venous pressure), and intake and output were collected from the medical records twice, at baseline at the time of enrollment during mechanical ventilation and during the initial weaning trial.

Dysrhythmias. Three-lead electrocardiographic (ECG) Holter (Evo, Del Mar) recordings of cardiac electrical activity in leads I, II, and V$_1$ were evaluated for cardiac dysrhythmias at baseline during mechanical ventilation and during the initial weaning trial. The Del Mar Holter Analysis System (CardioNavigator Plus version 3.05.0116, SpaceLabs Healthcare) was used to scan all Holter recordings, and all types of heart beats were confirmed by 2 of the investigators (M.H. and D.M.). Ventricular and supraventricular ectopic beats were identified, frequency determined, and calculated as beats per hour. Episodes of atrial fibrillation or flutter and ventricular tachycardia or fibrillation were evaluated and scored as present or not present.

Heart Rate Variability. HRV is the beat-to-beat alterations in heart rate produced by modulation of the ANs and is detected by variations in R-R intervals in the ECG. Frequency domain analysis of HRV was used to assess the autonomic modulation of sinus node activity during mechanical ventilation and the initial weaning trial. After confirmation of all normal R-R intervals obtained via Holter recordings, artifacts were filtered out and R-R tachograms (normal-to-normal R-R intervals) were generated. The software then used a fast Fourier transformation (FFT) to produce the frequency domain measures of the power spectrum. These variables are valid measures of the frequency bands that provide distinct indications of vagal, as well as sympathetic, modulation of the R-R intervals. This analytical technique provides quantification of the frequency bands in the power spectrum. The heart rate signal is dismantled into sine waves. The amplitude of each sine wave is then converted to power and plotted against the frequency of the sine wave to give the power spectrum. The FFT analysis separates the heart rate signal into its frequency components. These bands are quantified by their intensity, which is also known as power; thus, the greater the intensity of one band, the greater is the power in that band. The result is an evaluation of the power spectral density of the heart rate signal. High-frequency (HF), low-frequency (LF), very-low-frequency (VLF), and ultra-low-frequency (ULF) bands were included in the evaluation. The HF band (0.15-0.40 Hz) represents the vagal control to the heart, modulated by respiration. The LF band (0.04-0.15 Hz) has contributions from both vagal and sympathetic modulation of the heart. Although many investigators use the LF band as an index of sympathetic modulation, this practice is somewhat controversial because of the additional contribution of parasympathetic tone. VLF power (0.003-0.04 Hz) represents the sympathetic activity.

ECG recording periods were divided into consecutive, nonoverlapping 5-minute epochs by the HRV software (Del Mar). Artifacts, atrial fibrillation or flutter, and abnormal complexes along with the preceding and succeeding R-R intervals were excluded from the analysis, and linear interpolation was used to estimate the spectrum before applying an FFT. Segments in which more than 80% of the R-R intervals were not normal were excluded. The mean power spectra from the 5-minute epochs recorded during the 24-hour baseline period of mechanical ventilation and during the initial weaning trial were used in the data analysis.

Procedure

The study was approved by the University of Kentucky medical institutional review board.

Electrocardiographic Holter data were evaluated for dysrhythmias at baseline and during the initial weaning trial.
appropriate surrogate for each patient provided informed consent. At baseline, upon enrollment in the study, demographic and baseline clinical data were abstracted from medical records. Electrodes for a 3-lead Holter monitor were positioned to record data from leads I, II, and V2, and ECG readings were continuously recorded for 24 hours during the baseline period.

Patients receiving mechanical ventilation were evaluated daily in the morning by respiratory therapists and an attending physician for readiness to wean. Readiness was determined by using the following criteria in the institution’s protocol: resolution of the reason for which the patient was intubated, adequate oxygenation (oxygen saturation by pulse oximeter ≥ 90% with positive end-expiratory pressure < 8 cm H₂O, fraction of inspired oxygen 0.40), stable hemodynamic status without need for high-dose vasopressor treatment, pH greater than 7.35, body temperature less than 38.9°C (102°F), and minimal sedation or without need for sedation. Once the weaning trial was prescribed, the Holter monitor was placed and ECG values were continuously recorded during the initial weaning trial. Clinical data were then collected from the medical record for the weaning day.

Data Analysis

Data were evaluated for normality before statistical analyses were done. Frequency distributions were performed to detect errors, outliers, and missing data. Data transformation with log 10 was required for the HRV variables before statistical analysis; an inverse transformation was used to normalize distributions of beats per hour. Data were analyzed by using SPSS for Windows, version 20.0 (IBM SPSS). Descriptive statistics were used to characterize the sample. Repeated-measures analysis of variance was used to determine if mean values measured at baseline (during mechanical ventilation) differed from mean values measured during the initial weaning trial. Clinical data were then collected from the medical record for the weaning day.

Table 1
Characteristics of the sample (N = 35)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Valuea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>53.3 (14.6)</td>
</tr>
<tr>
<td>Men</td>
<td>23 (66)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>34 (97)</td>
</tr>
<tr>
<td>African American</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>17 (49)</td>
</tr>
<tr>
<td>Single/divorced/widowed</td>
<td>18 (51)</td>
</tr>
<tr>
<td>Type of insurance</td>
<td></td>
</tr>
<tr>
<td>Government</td>
<td>23 (66)</td>
</tr>
<tr>
<td>Commercial/self</td>
<td>12 (34)</td>
</tr>
<tr>
<td>Score on Glasgow Coma Scale, mean (SD)</td>
<td>8.4 (2.6)</td>
</tr>
<tr>
<td>Score on Acute Physiology and Chronic Health Evaluation IV, mean (SD)</td>
<td>69.5 (24.8)</td>
</tr>
<tr>
<td>History of pulmonary diseases</td>
<td>11 (31)</td>
</tr>
<tr>
<td>History of cardiovascular disease</td>
<td>17 (49)</td>
</tr>
<tr>
<td>History of cardiac rhythm disturbances</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Reason for current mechanical ventilation</td>
<td></td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>25 (71)</td>
</tr>
<tr>
<td>Neurological disorders</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Surgical conditions</td>
<td>6 (17)</td>
</tr>
<tr>
<td>Outcomes of initial weaning trials (n=30)</td>
<td></td>
</tr>
<tr>
<td>Success</td>
<td>7 (23)</td>
</tr>
<tr>
<td>Failure</td>
<td>23 (77)</td>
</tr>
<tr>
<td>Number of weaning trials (n=30), mean (SD) [range]</td>
<td>2.8 (2.6) [0-11]</td>
</tr>
<tr>
<td>Duration of mechanical ventilation, mean (SD) [range], d</td>
<td>11.6 (8.4) [2-33]</td>
</tr>
</tbody>
</table>

*Values are number (%) of patients unless indicated otherwise. Because of rounding, not all percentages equal 100.

This technique maintained the statistical power to detect differences in HRV measures.31 Separate models were developed to test the impact of HRV during weaning in the frequency domain (HF, LF, and VLF frequencies) on each of the following outcomes: ventricular ectopic beats per hour, supraventricular ectopic beats per hour, episodes of atrial fibrillation, episodes of atrial flutter, episodes of ventricular fibrillation, and episodes of ventricular tachycardia.

Results

Sample Characteristics

The 35 patients in the study were primarily men (66%), and the mean age was 53.3 (SD, 14.6) years (Table 1). The mean duration of mechanical ventilation was 11.6 (SD, 8.4) days, and the mean length of stay in the ICU was 15.6 (SD, 9.2) days. A total of 31% of the sample had a history of chronic pulmonary disease, and 49% had a history of cardiovascular disease. Of those patients with previous
The mean score on the Glasgow Coma Scale increased from 8.7 (SD, 2.3) to 10.3 (SD, 1.3) \( (P = .001) \), indicating an increase in consciousness and responsiveness. Serum levels of calcium and magnesium both increased significantly over time; mean calcium levels increased from 7.6 (SD, 0.8) mg/dL (to convert to millimoles per liter, multiply by 0.25) to 7.9 (SD, 0.8) mg/dL \( (P = .02) \), and mean magnesium levels increased from 1.8 (SD, 0.4) mg/dL to 2.0 (SD, 0.4) mg/dL \( (P = .007) \). However, values at both time points were within the reference range for these variables. Thus, although the increases are statistically significant, the changes are not clinically significant. Hemoglobin and hematocrit levels significantly decreased over time: hemoglobin, from 10.1 (SD, 1.7) g/L to 9.6 (SD, 0.9) g/L \( (P = .05) \); hematocrit, from 29.1% (SD, 4.8%) to 28.0% (SD, 2.6%) \( (P = .05) \). This change most likely reflects iatrogenic anemia, a common result of frequent blood sampling. Cumulative fluid balance increased by nearly 3 L over time: baseline, 12,364.4 (SD, 10,558.7) mL to 15,340.1 (SD, 13,764.5) mL \( (P = .03) \). No other differences in clinical variables occurred over time.

### Changes in Cardiac Rhythm

Data on cardiac rhythm were collected at baseline at the time of enrollment in the study for 24 hours and for 2 hours during the initial weaning trial. All patients experienced either ventricular or supraventricular ectopic beats or both during the ECG recording sessions (Table 3). When compared over time, the mean number of supraventricular ectopic beats per hour

### Table 2

**Comparison of clinical data at baseline during mechanical ventilation and on day of initial weaning trial \( (n = 30) \)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline during mechanical ventilation</th>
<th>Day of initial weaning trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score on Glasgow Coma Scale</td>
<td>Mean (SD) 8.7 (2.3) Range 3.0-11.0</td>
<td>Mean (SD) 10.3 (1.3) Range 6-14</td>
</tr>
<tr>
<td>Score on APACHE IV</td>
<td>Mean (SD) 69.8 (25.7) Range 26.0-125.0</td>
<td>Mean (SD) 61.0 (20.4) Range 16-111</td>
</tr>
<tr>
<td>Arterial blood gases</td>
<td></td>
<td>P</td>
</tr>
<tr>
<td>pH</td>
<td>Mean (SD) 7.4 (0.1) Range 7.2-7.6</td>
<td>Mean (SD) 7.4 (0.1) Range 7.3-7.6</td>
</tr>
<tr>
<td>$P_{\text{O}}_2$, mm Hg</td>
<td>Mean (SD) 97.3 (39.0) Range 34-243</td>
<td>Mean (SD) 109.5 (35.6) Range 34-179</td>
</tr>
<tr>
<td>$P_{\text{CO}}_2$, mm Hg</td>
<td>Mean (SD) 35.3 (10.1) Range 23-71</td>
<td>Mean (SD) 35.4 (10.3) Range 18-69</td>
</tr>
<tr>
<td>Serum sodium, mmol/L</td>
<td>Mean (SD) 139.0 (5.5) Range 127-150</td>
<td>Mean (SD) 139.0 (4.1) Range 131-148</td>
</tr>
<tr>
<td>Serum potassium, mmol/L</td>
<td>Mean (SD) 3.9 (0.6) Range 2.9-5.7</td>
<td>Mean (SD) 3.9 (0.7) Range 3.0-6.2</td>
</tr>
<tr>
<td>Serum calcium, mg/dL</td>
<td>Mean (SD) 7.6 (0.8) Range 5.3-9.3</td>
<td>Mean (SD) 7.9 (0.8) Range 5.3-9.3</td>
</tr>
<tr>
<td>Serum magnesium, mg/dL</td>
<td>Mean (SD) 1.8 (0.4) Range 1.3-3.6</td>
<td>Mean (SD) 2.0 (0.4) Range 1.4-3.1</td>
</tr>
<tr>
<td>Hemoglobin, g/L</td>
<td>Mean (SD) 29.1 (4.8) Range 20.3-41.5</td>
<td>Mean (SD) 28.0 (2.6) Range 24.0-33.2</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>Mean (SD) 29.1 (4.8) Range 20.3-41.5</td>
<td>Mean (SD) 28.0 (2.6) Range 24.0-33.2</td>
</tr>
<tr>
<td>Cumulative fluid balance, mL</td>
<td>Mean (SD) 12364.4 (10558.7)</td>
<td>Mean (SD) 15340.1 (13764.5)</td>
</tr>
</tbody>
</table>

**SI conversions:** To convert calcium to mmol/L, multiply by 0.25; to convert magnesium to mmol/L, multiply by 0.5.

### Table 3

**Comparison of frequency of dysrhythmias at baseline during mechanical ventilation and during the initial weaning trial \( (n = 25) \)**

<table>
<thead>
<tr>
<th>Cardiac rhythm</th>
<th>Mean (SD)</th>
<th>Weaning</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean supraventricular beats per hour</td>
<td>40 (82.2)</td>
<td>366 (1726.3)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean ventricular beats per hour</td>
<td>15 (37.4)</td>
<td>14 (27.2)</td>
<td>.68</td>
</tr>
</tbody>
</table>

* Supraventricular and ventricular beats per hour were inverse transformed before analysis.
during weaning was significantly greater ($P < .001$) than the mean number per hour at baseline: weaning, 366 (SD, 1726.3) and baseline, 40 (SD, 82.2). Ventricular ectopic beats per hour were not different over time (Figure 1). Only 1 patient had a single episode of ventricular tachycardia during weaning; none of the patients had ventricular or atrial fibrillation.

**Changes in HRV**

HRV components at baseline (LF=82.9 ms$^2$, HF=78.8 ms$^2$, VLF=79.4 ms$^2$) were reduced compared with normalized values published by the Task Force of the European Society for Cardiology and the North American Society of Pacing and Electrophysiology.$^{33}$ When baseline and weaning values were compared (Figure 2), LF power was significantly increased ($P = .04$) during weaning: baseline mean, 82.86 (SD, 126.43) ms$^2$; weaning mean, 202.62 (SD, 391.27) ms$^2$. No significant changes in HF or VLF power occurred during weaning (Table 4).

**Relationship Between HRV During Weaning and Occurrence of Cardiac Dysrhythmias**

HRV measures were entered into a linear regression with APACHE IV scores to control for the severity of illness, a confounding variable. In regression analyses, the LF power accounted for 41% of the variance in occurrence of ventricular ectopic beats ($\beta = 0.60; P = .002$), and 36% of the variance in occurrence of supraventricular ectopic beats ($\beta = 0.61; P = .002$) during weaning (Table 5). Although neither HF nor VLF power changed significantly during weaning, in the multiple linear regression analyses, HF power accounted for 68% of the variance in occurrence of ventricular ectopic beats ($\beta = 0.79; P < .001$) and for 29% of the variance in occurrence of supraventricular ectopic beats during weaning ($\beta = 0.53; P = .02$). The VLF power accounted for 63% of the variance in occurrence of ventricular ectopic beats ($\beta = 0.78; P < .001$), but did not explain variance in supraventricular beats during weaning ($P = .53$).

**Discussion**

HRV in this small group of critically ill patients at baseline was lower than published normative values, indicating sympathetic dominance. We found a significant increase in supraventricular beats per hour during weaning in our patients: approximately 9 times as many supraventricular beats per hour during weaning as during the baseline measurement period. The number of ventricular beats per hour at baseline did not differ significantly from the number during weaning. HF and LF HRV were predictive of supraventricular ectopy during weaning, whereas all
3 components of HRV (HF, LF, and VLF) were predictive of ventricular ectopy.

Transition from mechanical ventilation to spontaneous breathing during weaning induces marked hemodynamic changes in intrathoracic vascular volume, preload, and afterload. The ANS responds to hemodynamic alterations by increasing the activity of the sympathetic nervous system, reducing the activity of the parasympathetic system, or doing both simultaneously to maintain adequate cardiac output and tissue oxygenation. The ANS response to hemodynamic changes is often altered in patients with impaired cardiac function. Altered ANS is primarily reflected by decreased HRV, with a shift toward sympathetic dominance, rather than the normal parasympathetic predominance. Using frequency domain measures of HRV in a group of 6 healthy anesthetized canines with normal ventricular function, Frazier et al found a significant increase in activity of the sympathetic nervous system and a significant decrease in activity of the parasympathetic nervous system with exposure to a combination of pressure support 10 cm H2O and continuous positive airway pressure 10 cm H2O. In our study, almost half of our patients (49%) had a history of cardiac disease. We found that exposing patients to a combination...
of pressure support (range, 8-15 cm H₂O) and con-
tinuous positive airway pressure of 5 cm H₂O during
weaning led to an increase in sympathetic tone and
a concomitant decrease in parasympathetic tone.
Sympathetic dominance resulted in more rapid
sinus node depolarization and a reduced R-R interval.
Thus, HRV was reduced in response to weaning from
mechanical ventilation in these patients.

Decreased HRV has been described as arrhyth-
mogenic.16,17,36-40 In previous studies,16,17,36-40 increased
sympathetic activity or decreased parasympathetic
activity or both occurred before paroxysmal atrial
fibrillation and atrial flutter in patients with under-
lying heart disease. Although our patients did not
experience significant changes in VLF or HF power
during weaning, VLF and HF power during weaning
were predictive of the occurrence of supraventricular
and ventricular dysrhythmias. We also found that
at baseline, HRV spectral components were lower
than published norms,33 indicating reduced HRV
and sympathetic dominance. During weaning,
HRV was further reduced; the consequence was a
significant increase in supraventricular dysrhythmias.
Consistent with previous researchers,16,17,36-40 we found
that LF power and HF power were predictive of the
occurrence of supraventricular and ventricular ectopic beats, whereas VLF power was predictive of only the
occurrence of ventricular ectopic beats. Huikuri et al37
reported a significant decrease in HF power before
the occurrence of ventricular tachycardia in patients
with ischemic heart disease. Fei et al39 reported sig-
ficant changes in HRV immediately before the
occurrence of episodes of idiopathic ventricular tachycardia. Thus, our findings are consistent
with, and add to the support for, the association
between reduced HRV and serious dysrhythmias.

The prevalence of cardiac dysrhythmias during
weaning from mechanical ventilation has only been
described in a pilot study by Frazier et al,1 who
reported that the number of supraventricular ectopic beats per hour during weaning was almost double
the number at baseline and that the number of ven-
tricular ectopic beats per hour decreased by nearly
two-thirds in 39 patients exposed to a combination
of pressure support of 10 cm H₂O and continuous
positive airway pressure of 10 cm H₂O during
weaning attempts. Our results are consistent with
the observed change in supraventricular, but not ventricular, ectopic beats. We found no change in
ventricular ectopic beats per hour during weaning.
These findings may be attributed to the greater
proportion of patients in our study with preexisting
cardiac disease; half of our patients had a previous
diagnosis of cardiovascular disease compared with
only 21% in the study by Frazier et al.1 Thus, previous
structural alterations may have influenced the
effect of reduced HRV on the action potential and
arrhythmogenesis, particularly for supraventricular
dysrhythmias. We also studied patients whose severity
of illness did not decrease between the baseline measure and the day of their initial weaning trial
(APACHE IV score, 70 at baseline and 61 at weaning), whereas patients in the study by Frazier et al1 had
significant reductions in their APACHE II score,
from 26 at baseline to 14 the day of initial weaning.

In our study, supraventricular ectopic beats
during weaning may have been induced by a mecha-
noelectrical feedback mechanism. We found 9 times
more supraventricular ectopic beats during weaning
than at baseline. During weaning, increased atrial
and ventricular end-diastolic volume and afterload
can be arrhythmogenic because of this feedback
mechanism.42 Acute cardiac mechanical loading
with greater blood volume, and distension of the
atria and ventricles potentially induced mechanical
changes that reduced duration of myocardial action
potential and altered myocyte excitability. This
mechanoelectrical feedback is mediated by cardiac
myocyte stretch-activated channels and selective-ion
channels.43-46 Because we found a mean cumulative
fluid increase of nearly 3 L, we inferred that preload
was also increased to some degree and may have
stimulated the mechanoelectrical feed-
back mechanism. The result was a significant
increase in supraventricular ectopic beats. This
increase is consistent with findings in patients
with chronic congestive heart failure.47

This study was the first investigation of the
predictive power of HRV, characterized by using
spectral power analysis, for dysrhythmias during
weaning from mechanical ventilation. We found
that the results of power spectral analysis of HRV
explained 29% to 68% of the variance in the
occurrence of supraventricular and ventricular
ectopy. Our findings provide evidence that HRV
measures may be useful as a clinical indicator of
autonomic dysfunction in critically ill patients;
interventions to address autonomic balance could

Heart rate variability measures may be
useful as a clinical indicator of autonomic
dysfunction.
reduce the prevalence of cardiac dysrhythmias while patients are being weaned from mechanical ventilation. The use of HRV by clinicians could improve patients’ outcomes, because autonomic dysfunction and reduced HRV are predictive of greater morbidity, mortality, and complications in patients with a variety of health issues.40-52

Limitations

Our study has some limitations. First, the sample size was small. However, the percentages of baseline scores were used to maintain the power of the study to detect differences. Larger sample sizes are recommended for future studies. Second, we did not use other measures to collect data on autonomic activity, such as plasma catecholamine levels or baroreflex sensitivity. However, power spectral analysis of HRV is an accepted, valid, and reliable noninvasive indicator of ANs balance. Third, the majority of patients were male (67%) and white (97%), characteristics that reduce the generalizability of the study findings.

Conclusion, Clinical Implications, and Recommendation for Future Research

We found that HRV was significantly predictive of the occurrence of cardiac dysrhythmias during weaning from mechanical ventilation in a heterogeneous sample of critically ill patients. HRV was reduced at baseline and further decreased with weaning. Early detection of ANs changes and effective management of cardiac dysrhythmias during weaning are of paramount importance; these alterations can contribute to seriously unstable hemodynamic status, unsuccessful weaning, and the need for longer duration of mechanical ventilation. HRV analysis and clinical use could contribute to improved clinical decision making about readiness for weaning and subsequently lead to improved outcomes for patients.

FINANCIAL DISCLOSURES

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REFERENCES

11. Frazier SK, Moser DK, Stone KS. Heart rate variability and hemodynamic alterations in canines with normal cardiac function during exposure to pressure support, continuous positive airway pressure, and a combination of pressure support and continuous positive airway pressure. Biol Res Nurs. 2001;3(3):167-174.
Heart Rate Variability as a Predictor of Cardiac Dysrhythmias During Weaning From Mechanical Ventilation
Muna H. Hammash, Debra K. Moser, Susan K. Frazier, Terry A. Lennie and Melanie Hardin-Pierce

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