Editorial Commentary

Heart Rate Variability
Just a Surrogate for Mean Heart Rate?

Harald M. Stauss

See related article, pp 1334–1343

In this issue of Hypertension, Monfredi et al1 address a phenomenon that has been repeatedly described in the literature but, nevertheless, went largely unnoticed by the scientific community using heart rate variability (HRV) to assess cardiac autonomic control or to predict cardiovascular health. The phenomenon studied by Monfredi et al1 is that most HRV parameters are inversely related to the actual level of HR, such that HRV is usually lower if HR is high and vice versa. The importance of the work by Monfredi et al1 is that it explores the physiological mechanisms underlying the relationship between HR and RR-interval, using mathematical and biophysical models applied to data from various species and even in vitro preparations with differing levels of mean HR. Furthermore, using the standard deviation (SD) of normal RR-intervals as an example of one of the more frequently used HRV parameters; they provide a simple equation that can be used to normalize this HRV parameter so that it is independent of the actual level of HR.

By convention, HRV is typically calculated from RR-intervals and not from HR time series.2 As pointed out in a recent review article by Sacha,4 the relationship between HRV (calculated from RR-intervals) and HR is partly because of the inverse relationship between HR and RR-interval (eg, if HR fluctuates by ±10 bpm, the corresponding fluctuations in RR-intervals are ±250 ms at an average HR of 50 bpm but only ±60 ms at an average HR of 100 bpm). Thus, if HRV is calculated from RR-intervals, a high HR is associated with only ±60 ms at an average HR of 100 bpm). Thus, if HRV is calculated from RR-intervals rather than from HR values. On the basis of this illustration, one may reconsider the generally accepted convention to calculate time domain HRV parameters from RR-intervals rather than from HR values.

However, Monfredi et al1 point out in this issue of Hypertension1 that the inverse relation between HR and RR-interval is not the only reason for the relationship between mean HR and HRV. On the basis of a biophysical model of the sinoatrial node action potential, they argue that for a given perturbing ion current (eg, generated by the autonomic nervous system) acting on phase 4 (diastolic depolarization) of the sinoatrial action potential, the resulting change in HR depends on the prevailing slope of the diastolic depolarization in a nonlinear manner. This concept is illustrated in Figure 4 of the article by Monfredi et al1 and in Figure 2 of this commentary. At low heart rates (Figure 2, top), the intrinsic ion currents driving the diastolic depolarization are relatively small. Thus, at low heart rates, a given perturbing current (changing the slope of the diastolic depolarization by a fixed angle α) elicits a relatively larger effect on HR and cardiac cycle length (eg, +15 bpm and –200 ms at a heart rate of 60 bpm in Figure 2, top) than at higher heart rates (Figure 2, bottom) when the intrinsic ion currents driving the diastolic depolarization are already large (eg, +6 bpm and –60 ms at a heart rate of 75 bpm in Figure 2, bottom). Importantly, this phenomenon is independent of the inverse relationship between HR and RR-interval (illustrated in Figure 1) because a given perturbing current acting on the diastolic depolarization of the sinoatrial pacemaker cells affects both the HR and the RR-interval more at low (Figure 2, top) when compared with high heart rates (Figure 2, bottom).

Because of the established relationship between HR and HRV, interpretation of HRV parameters obtained from groups of subjects with differing HR seems difficult. For example, numerous studies have reported reduced HRV in patients with heart failure and in experimental animal models of heart failure.2,12 Because heart failure is often associated with tachycardia, one would expect HRV to be reduced in heart failure simply because of the established relationship between HR and HRV. Likewise, it has been demonstrated that aerobic exercise training increases HRV in patients with heart failure, which has been interpreted as improved cardiovascular health.14 However, because aerobic exercise training is associated with a reduction in heart rate, the increased HRV in patients with heart failure after an aerobic exercise training intervention is to be expected simply because of the lower heart rate. Even though the relationship between mean HR and HRV in heart failure has been described >20 years ago,15 the majority of more recent studies have largely ignored this relationship and, thus, are difficult to interpret.
Heart rate variability (HRV) expressed as SD (σ) of RR-intervals (in ms, equals SD of normal to normal intervals) and of HR values (in bpm, recorded in a human (HR=72 bpm), a rat (HR=368 bpm), and in a mouse (HR=526 bpm). HRV depends to a greater extent on HR if expressed as SD of NN-intervals compared to standard deviation of HR values.

On the basis of aforementioned considerations, one may ask the question whether HRV is just a surrogate for mean HR. However, such a notion would be an oversimplification and not give credit to many studies in which investigators used HRV parameters that are less dependent on HR, such as the coefficient of variation of the SD of RR-intervals and the low frequency/high frequency ratio of HRV or used other means to correct for the relationship between mean HR and HRV. These and many other studies have indeed demonstrated that HRV has prognostic power in patients with cardiovascular diseases that is independent of HR and provides information on cardiac autonomic regulation beyond the information that can be gained simply by the mean level of HR. However, the study by Monfredi et al published in this issue of Hypertension clearly demonstrates that to interpret HRV parameters correctly, it is essential that the relationship between mean HR and HRV is taken into account. To deal with this issue, Monfredi et al have derived 2 useful equations (Equations 8 and 9 in their article) that can be applied to correct SD of normal to normal intervals (a commonly used time domain HRV parameter) for HR. Potentially, another way to deal with this issue would be to calculate HRV parameters from HR time series rather than from RR-interval time series as illustrated in Figure 1. However, this approach is not yet established and would require further testing.

In conclusion, HRV is not just a surrogate of mean HR. HRV is still an independent predictor of cardiovascular risk and can provide useful information on autonomic cardiac control. However, using HRV parameters without considering the mean level of HR can lead to serious misinterpretation of experimental data. The hope is that the study by Monfredi et al will increase the awareness of this important issue and, thus, improve the quality and interpretation of future studies using HRV.

Disclosures

None.

References


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In the current Hypertension, Monfredi et al.
[1] observed that heart rate variability (HRV) was a sensitive and specific indicator of cardiovascular health. Their study, published in 2014, demonstrated a strong association between HRV and various cardiovascular outcomes. This suggests that HRV may be a useful tool for assessing cardiovascular health and identifying individuals at risk for adverse outcomes.

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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HR and HRV. These and many other studies have indeed used HRV parameters that are less dependent on HR, and provide useful information on cardiac autonomic regulation and can give credit to many studies in which investigators ask the question whether HRV is just a surrogate for mean HR values (in bpm) recorded in a human (HR=72 bpm), a rat (HR=368 bpm), a mouse (HR=526 bpm), and in a mouse (HR=526 bpm). HRV depends on the autonomic nervous system, its potential in sinoatrial pacemaker cells by a fixed angle of experimental data. The hope is that the study by Monfredi et al will increase the awareness of this important issue and, rather than from RR-interval time series as illustrated in Figure 1, would require further testing. However, this approach is not yet established and would be to calculate HRV parameters from HR time series. Potentially, another way to deal with this issue is to interpret HRV parameters correctly, it is advisable to consider the mean level of HR can lead to serious misinterpretation and can provide useful information on autonomic cardiac control. However, using HRV parameters without consideration of experimental data. The hope is that the study by Monfredi et al could increase the awareness of this important issue and, rather than from RR-interval time series, the approach is not yet established. Using HRV parameters from HR time series could improve the quality and interpretation of future studies, and would require further testing.

In conclusion, HRV is not just a surrogate of mean HR. However, the study by Monfredi et al could increase the awareness of this important issue and, rather than from RR-interval time series as illustrated in Figure 1, would require further testing. However, this approach is not yet established. Using HRV parameters from HR time series could improve the quality and interpretation of future studies, and would require further testing.

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