Heart Rate Variability
Just a Surrogate for Mean Heart Rate?

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See related article, pp 1334–1343

In this issue of Hypertension, Monfredi et al address a phenomenon that has been repeatedly described in the literature but, nevertheless, was 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 al 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 al is that it explores the physiological mechanisms underlying the relationship between HR and HRV 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 to 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 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-interval and not from HR time series. As pointed out in a recent review article by Sacha, 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). This is illustrated in Figure 1 because a given perturbing current acting on the diastolic depolarization is already large (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. 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. 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, the majority of more recent studies have largely ignored this relationship and, thus, are difficult to interpret.
Figure 1. 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.15–18 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 al1 published in this issue of Hypertension1 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 al1 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 al1 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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is cardiovascular health improvement.\(^{[24]}\) However, with autonomic training and heart rate reduction of concern, heart rate variability can be considered. At one time, the effects of heart rate variability in terms of cardiovascular risk have been observed to be independent of heart rate, thus, improving the quality and interpretation of future studies. However, this approach is not yet established and would require further testing.

Figure 1. Differences in heart rate variability, baroreflex sensitivity, and anxiety-like behavior were demonstrated that to interpret HRV parameters correctly, it is necessary to account for the mean level of HR. However, the study by Monfredi et al.\(^{[1]}\) demonstrates that to interpret HRV parameters correctly, it is necessary to consider the mean level of HR.

In conclusion, HRV is not just a surrogate of mean HR. HRV is a cardiovascular risk factor that can provide useful information on autonomic cardiac function. HRV is still an independent predictor of cardiovascular risk beyond the information that can be gained simply by the mean level of HR. However, the study by Monfredi et al.\(^{[1]}\) demonstrates that to interpret HRV parameters correctly, it is necessary to account for the mean level of HR.

Figure 2. A fixed angle was applied to each data sample. The hope is that the study by Monfredi et al.\(^{[1]}\) will be published in this issue of "J Am Coll Cardiol". 1994;2:1539–1546.

Disclosures

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