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Heart Rate Variability

Summary prepared by Ichiro Kawachi in collaboration with the Allostatic Load Working Group. Last revised 1997.

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Definition

Heart rate variability (HRV) refers to the beat-to-beat alterations in heart rate. Under resting conditions, the ECG of healthy individuals exhibits periodic variation in R-R intervals. This rhythmic phenomenon, known as respiratory sinus arrhythmia (RSA), fluctuates with the phase of respiration — cardio-acceleration during inspiration, and cardio-deceleration during expiration. RSA is predominantly mediated by respiratory gating of parasymphathetic efferent activity to the heart: vagal efferent traffic to the sinus node occurs primarily in phase with expiration and is absent or attenuated during inspiration. Atropine abolishes RSA.

Reduced HRV has thus been used as a marker of reduced vagal activity. However, because HRV is a cardiac measure derived from the ECG, it is not possible to distinguish reduced *central* vagal activity (in the vagal centers of the brain) from reduced peripheral activity (the contribution of the target organ — the sinus node — or the afferent/efferent pathways conducting the neural impulses to/from the brain).

What aspect of allostasis does HRV potentially measure?

Although our understanding of the meaning of HRV is far from complete, it seems to be a marker of both dynamic and cumulative load. As a *dynamic* marker of load, HRV appears to be sensitive and responsive to acute stress. Under laboratory conditions, mental load — including making complex decisions, and public speech tasks — have been shown to lower HRV. As a marker of *cumulative* wear and tear, HRV has also been shown to decline with the aging process. Although resting heart rate does not change significantly with advancing age, there is a decline in HRV, which has been attributed to a decrease in efferent vagal tone and reduced beta-adrenergic responsiveness. By contrast, regular physical activity (which slows down the aging process) has been shown to raise HRV, presumably by increasing vagal tone.

In short, HRV appears to be a marker of two processes, relevant to the conceptualization of allostatic load: (1) *frequent activation* (short term dips in HRV in response to acute stress); and (b) *inadequate response* (long-term vagal withdrawal, resulting in the over-activity of the counter-regulatory system — in this case, the sympathetic control of cardiac rhythm).

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How is HRV measured?

Originally, HRV was assessed manually from calculation of the mean R-R interval and its standard deviation measured on short-term (e.g., 5 minute) electrocardiograms. The smaller the standard deviation in R-R intervals, the lower is the HRV. To date, over 26 different types of arithmetic manipulations of R-R intervals have been used in the literature to represent HRV. Examples include: the standard deviations of the normal mean R-R interval obtained from successive 5-minute periods over 24-hour Holter recordings (called the SDANN index); the number of instances per hour in which two consecutive R-R intervals differ by more than 50 msec over 24-hours (called the pNN50 index); the root-mean square of the difference of successive R-R intervals (the rMSSD index); the difference between the shortest R-R interval during inspiration and the longest during expiration (called the MAX-MIN, or peak-valley quantification of HRV); and the base of the triangular area under the main peak of the R-R interval frequency distribution diagram obtained from 24-hour recording; and so on. So far, experimental and simulation data appear to indicate that the various methods of expressing HRV are

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largely equivalent, and there is no evidence that any one method is superior to another, provided measurement windows are 5 minutes or longer.

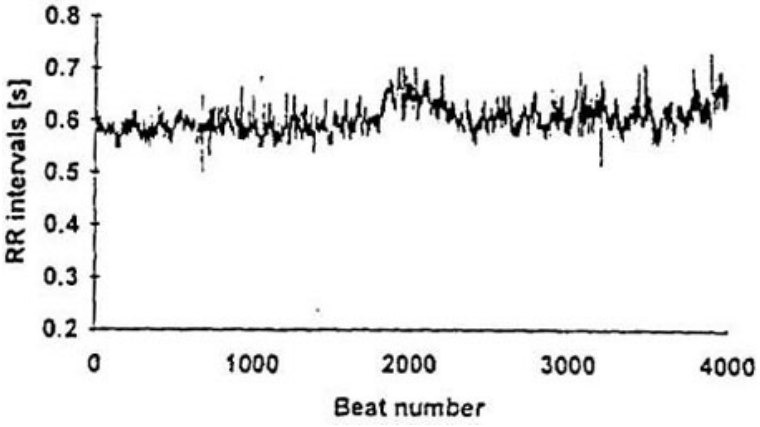

Figure 1. Tachogram

In contrast to the so-called *time domain* measures of HRV cited above, recent developments in microprocessor technology has enabled the calculation of *frequency* measures based on mathematical manipulations performed on the same ECG-derived data. Frequency measures involve the spectral analysis of HRV. Briefly, R-R interval data are represented on a tachogram (Figure 1), in which the y-axis plots the R-R intervals, and the x-axis the total number of beats. Spectral analysis of the tachogram transforms the signal from time to frequency on the x-axis, by representing the signal as a combination of sine and cosine waves, with different amplitudes and frequencies (Figure 2).

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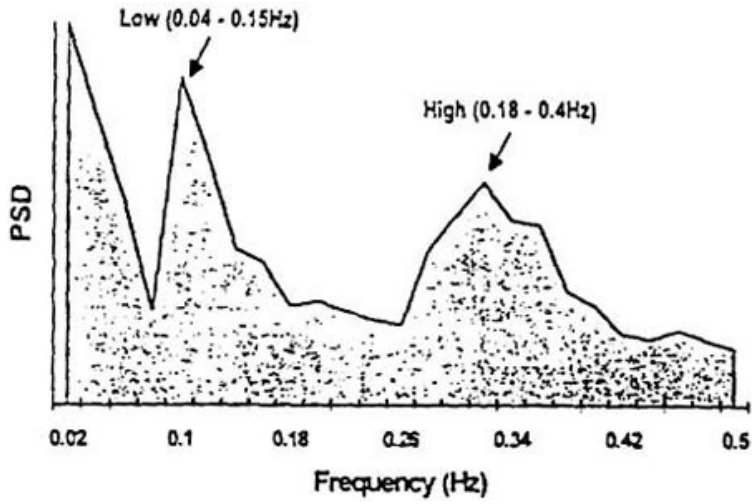

Figure 2. Power spectrum of HRV (PSD = power spectral density)

The approach uses Fourier transforms. The HRV spectrum contains two major components: the high frequency (0.18-0.4 Hz) component, which is synchronous with respiration and is identical to RSA. The second is a low frequency (0.04 to 0.15 Hz) component that appears to be mediated by both the vagus and cardiac sympathetic nerves. The power of spectral components is the area below the relevant frequencies presented in absolute units (square milliseconds). The total power of a signal, integrated over all frequencies, is equal to the variance of the entire signal. Some investigators have used the ratio of the low-to-high frequency spectra as an index of parasympathetic-sympathetic balance; however, this remains controversial because of our lack of complete understanding of the low frequency component (which seems to be affected by centrally generated brainstem rhythms, baroreceptor feedback influences, as well as both sympathetic and vagal input).

As a measure of vagal activity, spectral analysis of the high-frequency component probably offers no additional information over time-domain measures of RSA. On the other hand, the meaning and utility of the low frequency component deserves further investigation.

In sum, the analysis of HRV (whether by time-domain or spectral approaches) offers a non-invasive method of evaluating vagal input into cardiac rhythm. The measurement of

HRV is becoming increasingly standardized (e.g., see report of the Task Force of the European Society of Cardiology, 1996). Although, the assessment of HRV requires electrophysiologic expertise, the equipment is not prohibitively expensive, requiring only ECG equipment, microprocessors, and relevant software for carrying out Fourier analyses.

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The DTH Response as a Predictor of Disease

The major reason for the interest in measuring HRV stems from its ability to predict survival after heart attack. Over half a dozen prospective studies have shown that reduced HRV predicts sudden death in patients with MI, independent of other prognostic indicators such as ejection fraction. Reduced HRV appears to be a marker of fatal ventricular arrhythmia. Moreover, a small number of studies have begun to suggest that reduced HRV may predict risk of survival even among individuals free of CHD.

Does HRV vary with psychosocial factors?

Several studies have now suggested a link between negative emotions (such as anxiety and hostility) and reduced HRV. Kawachi et al (1995) reported a cross-sectional association between anxiety and reduced HRV (as assessed by two time-domain measures) in 581 men. Offerhaus (1980) observed lower HRV in individuals who were "highly anxious" according to the Minnesota Multiphasic Personality Inventory. Yeragani et al. (e.g., 1990; 1993) have published a series of reports indicating reduced HRV (using both time domain and spectral measures) among DSM-III diagnosed panic disorder patients. In turn, at least three prospective epidemiologic studies (Haines et al, 1987; Kawachi et al, 1994a; Kawachi et al, 1994b), and one case-crossover study (Mittleman et al, 1995) have suggested a relationship between high levels of anxiety and risk of CHD.

Sloan et al (1994) reported reduced high-frequency power among 33 healthy volunteers who scored high on the Cooke-Medley Hostility scale. The association between negative affect and reduced HRV may thus provide a potential mechanism linking chronic stress to disease outcomes (e.g., risk of CHD).

Further Readings

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