Modern evaluation of the hypertensive patient: autonomic tone in cardiovascular disease and the assessment of heart rate variability

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Analysis of heart rate variability (HRV) permits an assessment of sympathetic and parasympathetic activity from EKG recordings. Analysis of HRV may be performed in both the time and frequency domain by the application of mathematical principles of signal processing. HRV demonstrates abnormalities in myocardial infarction, sudden death, heart failure, autonomic neuropathy and hypertension. The technique is useful for assessing prognosis and for evaluating therapeutic interventions. *Blood Press Monit* 4 (suppl 1):S7–S14 © 1999 Lippincott Williams & Wilkins.

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Introduction

It has been widely recognized for centuries that variations in heart rate and blood pressure occur in a cyclical manner. Circadian, ultradian and infradian cycle variations have been observed in most biological systems; blood pressure and heart rate are no exception. Hales, in 1773, is credited with at least one of the first documented reports of beat-to-beat variation in arterial blood pressure [1]. Since 1965 variations in heart rate have attracted great interest since it has been determined to be an index of autonomic nervous system (ANS) function. Interest in heart rate variability (HRV) analysis has increased over the past two decades since it has been shown that a decrease in HRV is associated with increased cardiovascular mortality and that analysis of HRV provides insight into the function of the autonomic nervous system [2,3]. Cardiovascular conditions for which HRV measurements seem to be promising for determining prognosis and evaluation of therapeutic modalities are systemic arterial hypertension, ischemic heart disease and congestive heart failure (CHF).

The ANS has long been considered to play a role in the development of hypertension in some individuals [4–6]. This concept is based in large measure on the knowledge that the ANS is responsible for the short-term, i.e. minute-to-minute, control of blood pressure and heart rate. Thus, derangements in the ANS could easily be associated with sustained increases in blood pressure.

Abnormalities in the ANS are characteristic of patients with heart failure; abnormal activity of the parasympathetic nervous system (PNS) is one of the earliest abnormalities noted in the natural history of the syndrome [7,8,9]. Increased sympathetic nervous system (SNS) activity is associated with progressive deterioration and death either from progressive heart failure or sudden cardiac death, the latter often thought to be associated with cardiac arrhythmia.

Based on the understanding of the important role of the ANS in the pathophysiology of hypertension and heart failure, it is little wonder that much interest has been generated in HRV analysis hoping that this technique would add important information concerning the pathophysiology of heart failure, prognosis and treatment modalities.

Rationale for measuring HRV in hypertension and heart failure

Heart rate is determined by both the intrinsic firing of the pacemaker cells of the sino-atrial node and modulating influences of the ANS (Fig. 1). The ANS is composed of two divisions, the SNS, which innervates the sino-atrial node and which enhances firing rate, and the PNS, which exerts an inhibitory action, depressing spontaneous firing. Thus, the balance between the opposing ANS and PNS probably are the principal determinants of the heart rate.

HRV represents the continual fine-tuning of the beat-to-beat control mechanisms. In the human, the heart rate is usually under a tonic vagal control. Vagal afferent stimulation leads to reflex excitation of vagal efferent activity and inhibition of sympathetic efferent activity, whereas the opposite effects are mediated by the stimulation of sympathetic afferent activity. Central vasomotor and respiratory centers and peripheral oscillation in arterial pressure and respiratory movements add additional control. Analysis of HRV may provide a means to infer the state and function of the central oscillatory, the SNS and PNS activity, humoral factors and the sinus nod. These inter-relationships are demonstrated by the effect on HRV by tilt (Fig. 2).