Plan B Manual

Heart Rate Variability (HRV)/ RSA

The autonomic nervous system is the primary regulator of heart rate in presence of sinus rhythm. Under normal resting conditions, there is little efferent sympathetic neural input to the sinus node; however, there is substantial efferent parasympathetic input from the vagus nerves which slows resting sinus node rate to 55-75bpm in healthy adults. Resting HR is determined by both sympathetic and parasympathetic. tone.

HRV measures the fluctuations in the beat to beat intervals related primarily to autonomic control but does not measure autonomic tone directly. HRV actually reflects the complex interplay of all feedback loops - autonomic and non autonomic which regulate sinus node pacemaker activity and facilitate matching cardiac output to the needs of the body.
Analysis of HRV patterns from continuous EKGs of at least 24hrs show the predominant physiologic rhythm that accounts for most HRV is the circadian rhythm with increased sympathetic activity associated with higher heart rate during the daytime and increased vagal activity associated with lower heart rates during the night.

Rhythmic fluctuations in the frequency of impulse conduction along the vagus nerves, modulated by the rate and depth of breathing result in, among supine subjects with normal autonomic function, substantial variations in beat to beat intervals known as respiratory sinus arrhythmia (RSA). RSA can be exaggerated with meditation-related slow breathing. Beat to beat fluctuations are also affected by mental or physical activities and responses to environmental stress which reduce activity from the vagus nerves and, as effort or stress increases, increase the activity of the sympathetic nervous system.

Chronically increased sympathetic activity and elevated plasma catecholamines can be found in many conditions including heart disease and can predispose to potentially serious heart rhythm issues.

The parasympathetic nervous system through vagal innervation may exert important anti arrhythmic effects by reducing heart rate and counteracting the potential of the SNS to cause arrhythmias.

Based on the polyvagal theory perspective, higher parasympathetic activity is NOT always better since excess vagal activation can result in syncope/collapse and serious heart rhythm issues (via activation of the unmyelinated, phylogenetically older dorsal vagus

The clinical use of HRV is limited because of a number of technical issues but is a promising modality.

Ref: "Evaluation of heart rate variability," A.Goldberger, MD, Phyllis Stein, PhD, Up to Date