Editorial

Heart Rate Variability Analysis as a Patient Investigation Won't Be an Ideal Test for Assessing Autonomic Dysfunctions

Gopal Krushna Pal

Autonomic dysfunctions have been associated with many clinical disorders. Dysregulation of autonomic functions is reported to be involved in etiopathogenesis of cardio-metabolic diseases such as diabetes, hypertension, heart diseases and so on. Therefore, recently autonomic function tests (AFTs) have become part of routine patient investigations in medical practice. Standard autonomic assessments include conventional AFTs, spectral analysis of heart rate variability (HRV) and blood pressure variability (BPV). Recently, HRV analysis has become a more popular test in investigating autonomic disorders. Nevertheless, an investigator should be aware of the limitations of HRV analysis, especially that of short-term HRV, especially when it is done as a single investigation of the AFT. Therefore, in this editorial we analyse the merits and demerits of spectral analysis of HRV, used for patients' investigations in clinical practice.

The biological systems exhibit complex patterns of variability that can be described by mathematical computation and analyses. Heart rate variability (HRV) consists of changes in the time intervals between consecutive heartbeats called interbeat intervals (IBIs). The oscillations of a healthy heart are complex phenomena, which allow the cardiovascular system to rapidly adjust to sudden physical and psychological challenges to homeostasis. There are multiple perspectives on the mechanisms that generate 24 hr, short-term (5 min), and ultra-short-term (<5 min) HRV and their implications in health and diseases. The widely used HRV indices are time-domain, frequency-domain, and non-linear metrics. Time-domain indices quantify the amount of HRV observed during monitoring periods that may range from about 2 min to 24 hr.[1] Frequency-domain values calculate the absolute or relative amount of signal energy within component bands. Non-linear measurements quantify the unpredictability and complexity of a series of IBIs. There are many measurement contexts including recording period length, subject age and gender and respiratory trainings that influence the baseline HRV values.

Power spectral analysis of the beat-to-beat variations of heart rate or the heart period (R–R interval) has become widely used to quantify cardiac autonomic regulation. The total variance (the "total power") of a continuous series of beats in its frequency components identifies three main peaks: Very Low Frequency (VLF) <0.04Hz, Low Frequency (LF) 0.04–0.15Hz, and High Frequency (HF) 0.15–0.4Hz. The HF peak is widely believed to reflect cardiac parasympathetic nerve activity while the LF, although more complex, is often assumed to have a dominant sympathetic component. Based upon these assumptions, ratio of LF to HF (LF-HF ratio) has been used to quantify the strength of sympathetic and parasympathetic nerve activities, i.e., the sympathovagal balance in health and diseases.[2] However, this concept of LF and LF-HF ratio representing sympathetic drive and sympathovagal balance has been challenged many a times. Despite serious and largely under-appreciated limitations, the LF-HF ratio has gained wide acceptance as a tool to assess cardiovascular autonomic regulation where increase in LF/HF are assumed to reflect a shift to "sympathetic dominance" and decrease in this index correspond to a "parasympathetic dominance."[3] Therefore, it is vital to provide a critical assessment of the assumptions upon which this concept is based. The hypothesis that LF/HF accurately reflects sympathovagal balance rests upon several interrelated assumptions:[3] (i) cardiac sympathetic nerve activity is a major, if not the exclusive, factor responsible for the LF peak of the heart rate power spectrum; (ii) cardiac parasympathetic is exclusively responsible for the HF peak of the heart rate power spectrum; (iii) disease or physiological challenges provoke reciprocal changes in cardiac sympathetic and parasympathetic nerve activity (i.e., increases in cardiac parasympathetic nerve activity are always accompanied with corresponding reductions in cardiac sympathetic nerve activity and *vice versa*); and (iv) there is a simple linear interaction between the effects of cardiac sympathetic and cardiac parasympathetic nerve activity on heart rate variability (HRV). However, there are body of evidence that all these assumptions do not work in real clinical and critical situations.[3] Thus, LF-HF ratio, the major index of HRV does not always reflect the functional status of autonomic tone of the subject in health and disease.

The LF peak of the heart rate power spectrum is reduced by at least 50% by either cholinergic antagonists or selective parasympathectomy.[4] Importantly, this peak is not fully abolished by the combination of selective denervation and

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beta-receptor blockade; 25% of the peak remains after this treatment. Therefore, LF/HF often actually increases from baseline values when both parasympathetic and adrenergic nerve activity have been blocked.[4] Finally, direct recording of sympathetic nerve activity failed to correlate with LF power in either healthy subjects or patients with heart failure, a condition known to increase cardiac sympathetic drive. Thus, the LF component of HRV does not provide an index of cardiac sympathetic drive but rather reflects a complex and not easily discernible mix of sympathetic, parasympathetic, and other unidentified factors with parasympathetic factors accounting for the largest portion of the variability in this frequency range. As a consequence, the physiological basis for LF/HF is difficult to discern.[4] Further, reports also that suggest that HF power cannot be solely attributed to changes in cardiac vagal efferent nerve traffic, further compromising an accurate interpretation of HRV indices, especially the LF/HF ratio.[5]

Apart from the ambiguities and the limitations of HRV as discussed above, the HRV recordings unless done for 24 hr or at least for 12 hr, does not provide the concrete knowledge of long-standing fluctuations or variations in the R-R interval, the heart rate. A short-term HRV recorded for 5 to 10 min following 10-15 min supine rest does not assess the adequate oscillations in interbeat intervals and therefore cannot reflect the strength of autonomic drives or confirm the impending cardiovascular (CV) risks. Especially, the strength of short-term HRV in detecting the extent of autonomic dysfunctions is quite less, though at best it can help in prediction of CV disease risks. Moreover, the HRV does not assess the autonomic responses to various stimuli or stressors, as it assesses only the cardiac autonomic drives.[6] The sympathetic and parasympathetic reactivities to various stimuli such as orthostatic stress, handgrip or cold-pressor pain etc. reflect the magnitude of autonomic functions and dysfunctions.[6] Therefore, these conventional reactivity tests are always considered as the gold standards of autonomic function (or dysfunction) assessments. Hence, conventional AFTs should be performed along with HRV analysis to detect the nature and degree of autonomic dysfunctions in various clinical disorders.

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