VIEWPOINT

Heart rate variability: why do spectral analysis?

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Measurement of heart rate variability (HRV) by spectral analysis has become a hot issue in cardiology in recent years. Originally this technique was promoted by well-known clinicians and physiologists who reported that HRVgave insights into sympatho-vagal balance in autonomic outflow.[1–3] This led to it being applied to the assessment of mental[4] or physical stress[5] on the one hand and the diagnosis of autonomic neuropathy[6] on the other. Later it became apparent that HRV analysis might help in risk assessment of patients, for instance after myocardial infarction.[7] It was found that decreased HRV points to a poor prognosis, probably related to the decreased cardioprotective activity of the parasympathetic system. Despite difficulties with the interpretation and in application of the techniques,[8] many are using HRV analysis in clinical research.

Heart rate variability is mainly a reflection of the influence of the autonomic nervous system on the sinus node of the heart. The heart rate alters with many of the changes in demand on the cardiovascular system that are related to changes in respiration, posture, and physical or mental activity. These changes are invoked by the control mechanisms that coordinate the total pattern of activity in the individual. We need information on this total activity to interpret HRV correctly. For instance, unless we were aware of the highly emotional conversation that took place, a sudden increase in blood pressure and heart rate in someone apparently sitting quietly at his desk would be incomprehensible.

Data on blood pressure changes are essential for the understanding of short-term HRV because much of it is under baroreflex control.[9] In the blood pressure to heart rate reflex loop the vagus nerve induces fast, brisk changes in the activity of the sinus node.[10] This to a large extent explains why a decrease in vagal outflow as observed after myocardial infarction induces lower HRV and lower baroreflex sensitivity scores.[7 11]

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Glossary
HRV = Heart rate variability—continual change in heart rate even under stable conditions
HF = High frequency—the variations in heart rate at a rate from about one oscillation per six seconds and faster. HF variations are mostly coupled to respiration
LF = Low frequency—variations in heart rate at slower rhythms than HF. Prominent LF oscillations at a rate of about one per 10 seconds are seen, but slower rhythms are also seen

Use of HRV measurement

Measurement of HRV and heart rate changes in response to known stressors is used for various purposes. The technique should be adapted to the aim of the investigation.

In the diagnosis of cardiovascular autonomic neuropathy a combination of three classic tests—blood pressure and heart rate responses to deep breathing, standing up, and Valsalva's manoeuvre—gives a quick and clinically useful indication of the site and extent of impairment. There are age-related limits of normal responses for these tests.[12] The Finapres technique for the measurement of continuous blood pressure has helped to improve the diagnostic power of these tests.[13] In recent years spectral analysis of HRV in the supine and standing position has been proposed for the diagnosis of diabetic autonomic neuropathy.[14 15] However, the wide variance in the results of these HRV tests and the required lengths of stationary ECG data sets has limited use of these newer techniques.

HRV analysis in risk assessment—for example, in patients after myocardial infarction—requires techniques and data sets other than the relatively short-lasting records (5 to 10 minute recordings) that are required for investigation of momentary autonomic state. Here the availability of 24 h Holter recordings seems to have opened a new era in biomathematics.[16 17] The very low frequency variability at frequencies below those that are generally considered the result of properties of the baroreflex control loop seems to be an especially good indicator of general health.[18] But analysis techniques other than Fourier analysis of harmonic content or more classic statistical approaches are also being considered. These include Poincaré plots,[19] fractal dimension,[20] or apparent entropy.[21] These techniques take a more global look at HRV than those that interpret HRV in terms of sympathetic and parasympathetic (baroreflex) control of the cardiovascular system. The HRV signal is considered more or less as a chaotic signal with hidden features. Because of the long data

stretches that are required for these analyses few researchers have applied them to recordings of heart rate with continuous blood pressure. The technique for 24 h continuous blood pressure has been limited to a few centres which use portable intra-arterial recording techniques. Presently the portable version of Finapres (Portapres) intended for 24 h use is gradually coming out of the laboratory.[22]

Guidelines in HRV: the task force's report

Recently a task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology has delivered its report on proposed standards of measurement, physiological interpretation, and clinical use for heart rate variability.[23] This group of experts in mathematics, engineering, physiology, and clinical medicine has reviewed a wide area of techniques and applications for this new tool in cardiology. The emphasis in the report is very much on measurement and computational techniques. The physiological mechanisms underlying HRV are only touched on. This may give the impression that much of the physiology in HRV is settled and that we only have to consider the choice of the right computational technique for a specific study.

Critique

The origin and meaning of HRV are much debated in various circles of researchers. Some have tried to fit the experimental observations to simple physiological models.[9 24] The task force report adopts a more global view, emphasising the distinction between low frequency (LF) and high frequency (HF) HRV and viewing LF as sympathetic and HF as parasympathetic cardiovascular control. This is an oversimplification and detracts from the unsolved fundamental problems in HRV analysis. Basic physiology indicates that short-term HRV is to a large extent baroreflex-mediated. Various techniques have been proposed to extract measures for baroreflex sensitivity (BRS) from the combined spontaneous fluctuations in beat-to-beat blood pressure and heart rate.[25 26] Some members of the task force applied these measures themselves[27] or stressed the importance of BRS measurement to risk assessment in patients after myocardial infarction.[11 28–30] From this perspective the task force's report seems to have overemphasised computation, possibly to avoid being too opinionated.

In 1995 Sleight *et al* asked "Is power spectral analysis largely an index of baroreflex gain?".[31] They showed that the model of DeBoer *et al*[9] was probably correct in explaining LF (0·1 Hz) blood pressure variations as a resonance phenomenon in the blood pressure response to peripheral resistance baroreflex. In this model HRV is a consequence of the baroreflex modulation of the sinus node by cardiac parasympathetic and sympathetic activity. This relates HF and LF HRV to heart rate control by the vagus nerve (HF) and combined vagus and sympathetic activity (LF). The existence of LF oscillations in blood pressure is explained as a result of sympathetic vasomotor activation probably originating in the baroreflex to sympathetic feedback loop. This model does not preclude other mechanisms inducing oscillations in blood pressure in the LF band; however, under normal conditions the feedback mechanism of the baroreflex is sufficient to explain the experimental observations. In addition, it explains why parasympathetic blockade by atropine not only abolishes HF HRV but also strongly reduces LF HRV.

For testing the autonomic state by spectral analysis short-term stable recordings of continuous blood pressure and heart rate can be sufficient; short-term, as proposed in the task force report, being at least five minutes. Moreover, the cardiovascular control system must be challenged to show its regulatory capacity. Therefore one recording only in the supine position will, on most occasions, be insufficient. When they are supine most people need little contribution from the sympathetic nervous system to cardiovascular control. Orthostatic stress will unveil a shift from the mainly parasympathetic supine state to a sympathetically dominated upright position.[32 33]

However, in many cases of cardiovascular disease the required recordings are very hard to obtain. Frequent cardiac arrhythmias can make it impossible to obtain a clean recording of sufficient data length to do any spectral analysis. Moreover, the classic tests mentioned above (deep breathing, Valsalva's manoeuvre and, if conditions allow, a standing test) will give most of the required data on the condition of autonomic cardiovascular control. If the sensitivity of the baroreflex needs to be assessed, the classic phenylephrine test can be used to demonstrate its capacity to influence heart rate (expressed as ms interval prolongation per mm Hg increase of systolic blood pressure). Intra-arterial recording can be replaced by Finapres.[34]

Limited and cautious interpretation of HRV spectra is required. For diagnostic use in the autonomic function laboratory spontaneous HRV as measured in conventional Holter recordings should be combined with responses to classic manoeuvres. Among these are the blood pressure and heart rate responses to standing, Valsalva's manoeuvre, and the deep breathing test. We must avoid making unwarranted claims for clinical tests, as were made for Vmax some years ago where modelling and computing seemed to receive more attention than clinical sense and sound physiology.

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