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EDITORIAL

The purpose of heart rate variability measurements

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Dear Editor,

It is well known and has been repeatedly published [1, 2] that analyses of heart rate variability (HRV) offer an insight into the autonomic modulations of cardiac periodicity and might thus be used to study autonomic responsiveness. This makes HRV investigations useful for the characterization of autonomic dysfunction as well as for risk stratification.

Both these goals have distinct specifics. Detection of primary autonomic abnormalities is best achieved by well-controlled short-term recordings obtained under provocative conditions, such as the head-up tilt test, the postural test, and other well-defined autonomic provocations during which continuous electrocardiographic recordings (ECG) are made. In most quantifications of primary autonomic dysfunction, it is also advantageous to accompany continuous ECG by simultaneous continuous recordings of blood pressure, e.g. by plethysmography, and of respiration, e.g. by end-tidal CO_2, chest belt or chest impedance measurements. If these signals are obtained under stationary conditions of different autonomic provocative stages (e.g. different tilt inclinations), standard spectral [1] and coherence analyses of the ECG and blood pressure records, as well as novel signal processing advances [2], allow not only the quantifying of the differences between the different provocation stages but also measuring the influences that the provocations have on the separate limbs of the autonomic nervous system, on the baroreflex, and on other autonomic-based regulatory processes.

Since the autonomic nervous system reflects the homeostasis of the whole organism, HRV suppression in controlled short-term recordings (e.g. the absence of respiratory sinus arrhythmia) also signifies increased risk. In most situations, however, more powerful risk stratification is achieved by the HRV analysis of long-term, e.g. 24-h, recordings. This is because risk-related homeostasis disturbances also restrict the responses to inputs from the surrounding environment, which are, together with the day–night differences and levels of physical activity, a major contributor to 24-h heart rate fluctuations. Hence, if clinically well-defined patients with the same or similar underlying diagnosis are recorded within a uniform environment, such as during hospital stay, those who are clinically poorer will react less actively to surrounding mental and physical stimuli, which will be reflected in lower long-term HRV, independently of whether the underlying clinical condition leads to a primary autonomic disorder. This is the reason why reduced long-term HRV also indicates increased risk in non-cardiovascular conditions.

This also means that a primary autonomic dysfunction cannot be properly diagnosed from global measures of long-term HRV (e.g. 24-h SDNN), since responses to environmental stimuli cannot be sufficiently controlled regardless of any protocol instructions. Only dedicated analyses of long-term recordings can be interpreted in terms of autonomic abnormalities. One such analysis is that

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of heart rate turbulence (HRT) which measures the responses to internal stimuli of ectopic beats [3]. The autonomic consequences of HRT mechanisms are more or less independent of the external environment. At the same time, HRT-related fluctuations of cardiac periods influence long-term global HRV measurements, making it difficult to compare HRV values derived from recordings that differ in the ectopic frequency.

In this issue of *Clinical Autonomic Research*, Bienias and colleagues describe a study that aimed to detect autonomic dysfunction in patients with myotonic dystrophy [4]. The authors have to be commended for addressing a population with such a challenging disease, but unfortunately used an inappropriate type of recordings for their stated purpose. The myotonic dystrophy debilitating effects of muscle stiffness and cognitive decline make the cardiovascular responses to environmental challenges very different from those in healthy subjects. Consequently, comparisons of overall 24-h HRV measurements in patients with myotonic dystrophy and healthy controls mean very little. We note the reported differences in HRT but the values found in myotonic dystrophy patients are well above the ranges that are normally seen in patients with impaired baroreflex [3]. The exact number of ectopics used in HRT evaluations is not reported, but since this was clearly larger in myotonic dystrophy patients, the comparisons of 24-h HRV are even further compromised.

Bienias et al. also try to rekindle the long discredited and abandoned concept of QT dispersion (QTD) [5]. It is now well understood that, rather than heterogeneity of ventricular repolarization, QTD only reflects inaccuracies in QT interval measurement and the differences in lead-specific isoelectric projections of the T wave vectorcardiographic loop. Indeed, since leads III, aVR, aVL, and aVF are simple algebraic combinations of leads I and II, having the QT interval shorter in these leads than in leads I and II is only possible by imprecision and/or loop projections. Thus, if any further proof is needed of the nonsensical nature of the QTD concept, it can be found in table 4 of Bienias' manuscript.

Undoubtedly, cardiac muscle abnormalities in myotonic dystrophy lead to repolarization changes and, perhaps next time, these should be properly addressed by the battery of focused and validated methods ranging from the spatial QRS-T angle to the T wave temporal variability [6]. However, in any case, we do not understand why any spatial cardiac electrophysiology abnormalities should be interpreted as a suggestion of autonomic dysfunction. Some of the temporal beat-to-beat variations of ventricular repolarization might be reasonably related to ventricular control by the autonomic nervous system. However, these must not be confused with spatial static irregularities, for which there is no evidence of any autonomic implications. Beat-to-beat modulations of cardiac electrophysiology, haemodynamics, blood pressure, and other beat-to-beat measurements are, under controlled conditions, suitable for studying autonomic influences. However, QTD is a static spatial measurement obtained within one beat or the equivalent to one beat. Linking such static measurements to cardiac autonomic function is an obvious misunderstanding of the autonomic control of the heart.

Compliance with ethical standards

Conflict of interests None.

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