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Power spectral analysis of heart rate variability and baroreflex gain

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Experimental open-loop studies [1] indicate that when a sinusoidal pressure function is forced onto the arterial baroreceptors, a sinusoidal oscillation is reflexly transferred onto the heart rate to induce a synchronous component in the spectrum of the heart period variability.

Sleight et al. [2] addressed this issue in man with an elegant approach employing frequency domain analysis. They showed that a slow (0.1 Hz) sinusoidal oscillation is capable of inducing a baroreceptor reflex gain.

3. Experimental coronary artery occlusion, causing an excitatory sympathetic reflex and decreasing baroreflex gain, is accompanied by a consistent increase of the LF component of heart rate variability.

In conclusion, given the extreme complexity of central and peripheral mechanisms capable of contributing to the generation of cardiovascular rhythms, as indicated by the experimental findings that LF and HF components are present in both sympathetic and vagal efferent activities [3], it is our opinion that the physiological characterization of the various rhythmic components should be addressed with more of an observational approach considering rhythms as markers of functional states (as in the EEG studies), rather than pursuing inadequate oversimplifications.

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Power spectral analysis of heart rate variability and baroreflex gain: author’s reply

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We thank Professor Malliani and his colleagues for their comments on our paper. We are not attacking their prior contributions but rather were attempting to provide a hypothesis to explain some discrepancies that have arisen from uncritical application of power spectral techniques, particularly when applied to non-normalized R–R interval spectra.

We agree with the points they make; we also have stated previously [1] that the baroreflex is not the only source of heart rate variability. However, our paper does suggest that the amount of heart rate variability is related to baroreflex gain. Our interest was initially stimulated by those conditions, moderate to severe exercise, and also heart failure, where sympathetic tone is known to be high, but where power spectral techniques do not show this in absolute terms.

We agree with Malliani’s group, and indeed stated in our paper that there are many other instances where the gain of the baroreflex arc is reduced and where power spectral markers of sympathetic activity are increased (and vice versa). Therefore, their points 3, 4 and 5, are what one would expect from the de Boer/Karemaker model.

With regard to their first point, quadriplegic patients present very complex and variable abnormalities, with sympathetic as well as vagal over-activity. We find it quite reasonable that LF components in the power spectrum can appear during recovery. These might, as suggested by Malliani et al., be due to spinal rhythmicity in sympathetic discharge, but could equally perhaps be due to baroreflex modulation of heart rate (via the still intact vagus) interacting with mechanical respiratory modulation of venous return and hence blood pressure. Similarly, it is quite possible to envisage influences from a central oscillator, together with the above non-neural mechanisms, in the case of sino-aortic denervation (their second point).

In summary we do not disagree with their letter and certainly did not wish to be oversimplistic; conversely, we have provided a possible explanation for the apparent contradictions arising from uncritical application of power spectral data, and provided evidence to support the use of R–R interval normalized spectra, as suggested by Malliani’s group [2].

REFERENCES
