Power Spectrum Analysis of Heart Rate Variability in Human Cardiac Transplant Recipients

The study by Sands et al.1 reported promising results with the mathematical technique of power spectrum analysis of heart rate variability to detect graft rejection. We were surprised, however, to learn that they did not find spectral peaks in transplant recipients. In a similar study,2 we detected peaks at the so-called Mayer wave frequency (0.1 Hz) as well as at the frequency of respiratory sinus arrhythmia (RSA). Furthermore, we found that the RSA peak had better repeatability at two weeks (r=0.81) than the Mayer wave frequency and was significantly lower during rejection periods (p=0.039).

We suspect that part of the difference in our findings may be attributable to the different techniques used. Specifically, Sands et al. used a smoothing technique that, in effect, created a low-pass filter. Although this is acceptable, this often results in large, low-frequency spectral peaks that may “swamp” or interfere with small, higher-frequency peaks such as the RSA.3 To avoid this problem, we high-pass filtered the data at approximately 0.04 Hz to remove the low-frequency drift or aperiodic trend. This technique may also introduce spurious peaks near the corner frequency of the filter. However, we believe it highly unlikely that this filtering procedure affected the RSA peaks because the RSA frequencies were sufficiently higher. Furthermore, one of our subjects demonstrated clear, unequivocal Cheyne-Stokes respirations that modulated the heart rate and were clearly evident on the tachogram.4,5 We agree with Sands et al. that this technique is of potential benefit to transplant recipients and that larger, more-controlled studies are warranted.

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References

Reply
Drs. Zbilut and Lawless report finding 0.1-Hz heart rate fluctuations as well as respiratory sinus arrhythmia in heart transplant recipients, which differs from our characterization of heart rate fluctuations in transplant patients as broad-band.1 They attribute these differences to our signal processing methodology, claiming that spectral smoothing might cause low-frequency power to spill over into the respiratory frequency range and obscure the true variability in that range. Furthermore, they report that their measure of variability, peak spectral power of respiratory sinus arrhythmia (PSP-RSA), declined during rejection episodes, whereas our measure of variability (total, 0.02–2.0-Hz power) increased during rejection episodes.

We turn first to the question of whether heart rate fluctuations in transplant patients have discrete peaks or are broad-band. The striking finding in transplant patients compared with normal controls is that variability at all frequencies up to roughly 0.5 Hz is greatly reduced. Indeed, a comparison of Figure 1 (a normal subject) with Figures 3 and 4 (transplant patients with and without rejection) from Zbilut et al.2 demonstrates the same finding. Broad-band spectra may appear to have peaks at many frequencies, including the respiratory frequency. However, we found that we could not clearly distinguish a peak at the respiratory frequency from the background noise; this contrasts sharply from the situation with spectra from control subjects in which a distinct peak appears at the respiratory frequency. Zbilut et al.3 define the respiratory peak to be the tallest spectral peak found between 0.12 and 0.5 Hz. Their method guarantees the presence of “respiratory sinus arrhythmia” and begs the question of whether respiration has a measurable effect on heart rate variability in transplant recipients. The presence or absence of true respiratory sinus arrhythmia can be ascertained only if a respiratory signal is actually measured and in some way correlated with the heart rate signal. It is interesting to ask whether some small residual respiratory sinus arrhythmia is present in transplant patients and, if so, whether it is caused by sinus node stretch, to electrocardiograph axis shift resulting from respiratory movements, or to some other mechanism. If any respiratory sinus arrhythmia exists in transplant recipients, its mechanism surely differs from that in control patients in whom autonomic modulation of the vagal innervation to the heart is implicated.4

Interestingly, sympathetically and parasympathetically denervated, anesthetized, mechanically ventilated animals have very small residual respiratory frequency variations in heart rates (power reduced 99.8% from control).5 The presence of lower-frequency oscillations is a separate finding of interest. Since publication of our study, we have seen data from a small minority of transplant recipients exhibiting low-frequency oscillations (Phil Saul, personal communication), although the level of variability is still much lower than the low-frequency variability observed in normal subjects.

We agree with Drs. Zbilut and Lawless that the analysis of data with very low levels of heart rate variability as observed in
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