$1/f^{\alpha}$ Power spectral density of the cardiac QRS complex is not associated with a fractal Purkinje system

Dear Sir:

The so-called "fractal hypothesis" was proposed in this journal by Goldberger et al. (1), and was subsequently discussed in several reviews (2–6). According to that conjecture, a fractallike structure in the conduction system of the human heart determines the peculiar frequency content of the QRS complex as recorded in the electrocardiogram. However, to our knowledge, experimental validation of the role of a fractallike network in determining the frequency content of the QRS complex has not appeared. Here we compare the features of the human QRS complex in the frequency domain with those obtained in systems that do not have a morphological fractal substrate. Our results are clearly in conflict with the hypothesis of Goldberger et al. (1).

THE FRACTAL HYPOTHESIS (1–6)

In the mammalian heart, normal electrical activation starts in the sinus node; it spreads through the atria, and then reaches the atrioventricular node where it suffers a major delay. Once the impulse arrives at the His bundle, it then travels through the multiple branches of the Purkinje network and enters the right and left ventricular muscle. The subsequent activation of the ventricular mass is recorded on the surface electrocardiogram as a fast (50–100 ms) electrical deflection, the "QRS complex." The averaged power spectrum of the QRS complex shows a $1/f^{\alpha}$ falloff, where f is frequency and $\alpha \sim 4$ (1). The fractal hypothesis (1) attempts to explain such features by suggesting that "this process is mediated by current flow through a fractallike conduction network and therefore that the broadband spectrum of the depolarization waveform should be scaled as a power-law distribution" (1). Accordingly, only normal QRS complexes (i.e., those resulting from activation through the Purkinje network) should have $1/f^{\alpha}$ falloff. Abnormal ventricular activations resulting from "various types of bundle branch block in which the fractal depolarization sequence is also disrupted as well as in some patients with large, chronic myocardial infarction" (1) should not have a $1/f^{\alpha}$ power spectral density. Generalizing that concept, then any ventricular complex that results from a nonfractal activation network should not have $1/f^{\alpha}$ falloff.

Recent simulation studies (7) apparently support the hypothesis of Goldberger et al. (1). It has been shown that, when a numerical model of the ventricle is activated by a fractal network, the QRS frequency domain possesses a $1/f^{\alpha}$ spectral density distribution.

In this paper we report experimental results obtained in two different systems that clearly do not have any fractallike structure; yet they both have a $1/f^{\alpha}$ spectral decay. Indeed, recordings obtained in the frog ventricle during normal and premature ventricular activation, as well as in the sciatic nerve of the same species during propagation of the compound action potential, show a $1/f^{\alpha}$ falloff in their power spectral densities. Our results are in excellent agreement with the analytical and numerical arguments of Lewis and Guevara (8) that the $1/f^{\alpha}$ spectrum of the QRS complex is a result of its pulselike shape rather than of the temporal scale invariance that would be associated with fractal time series.

HUMAN VS AMPHIBIAN QRS (FRACTAL VS NONFRACTAL ACTIVATION?)

A normal QRS complex (limb lead I) recorded from a healthy individual during sinus rhythm is presented in *A* of Fig. 1. The computed first 32 harmonic amplitudes are plotted in *B*. The power-law falloff is apparent when amplitude vs harmonics number are plotted in a double logarithmic scale; the harmonic amplitudes are relatively well represented by a linear regression, where the slope of the fitted line is the exponent α (in this case $\alpha = 2.79$). It is important to note that the power spectrum in *B* was obtained from a single QRS complex; i.e., unlike in Goldberger et al. (1), no averaging was carried out, which might have improved the goodness of the fit.

Many years ago, Thomas Lewis (9) demonstrated that the frog heart does not have a His-Purkinje system. Moreover, to our knowledge, cell arrangement in the frog ventricle does not follow a fractallike structure, which makes this system ideal to test Goldberger's hypothesis. Ventricular activation in the frog heart starts at a rather diffuse area near the AV ring, which gives the QRS complex a slow initial rising phase (Fig. 2 *A*), and results in an appearance that resembles that in humans suffering from the so-called "preexcitation" or "Wolf-Parkinson-White" syndrome (10).

A normally activated frog QRS complex is presented in *A* of Fig. 2 (Frog SB), together with a premature ventricular complex (Frog PVC) initiated in the apex by an external stimulus applied to the epicardial surface. The PVC has a reverse polarity, denoting the inverse direction of propagation. The computed power spectra are plotted in *B* of Fig. 2. Both spectra show a very similar falloff with $\alpha = 2.02$ for the SB and $\alpha = 2.27$ for the PVC. From these results one may safely conclude that the $1/f^{\alpha}$ falloff is not related at all to any fractal activation network because SB and PVC have literally the same frequency content and scaling.

AN UNEQUIVOCALLY NONFRACTAL EXAMPLE: THE COMPOUND ACTION POTENTIAL

To provide definite proof that a waveform with a $1/f^{\alpha}$ spectrum need not reflect an anatomical fractal substrate,

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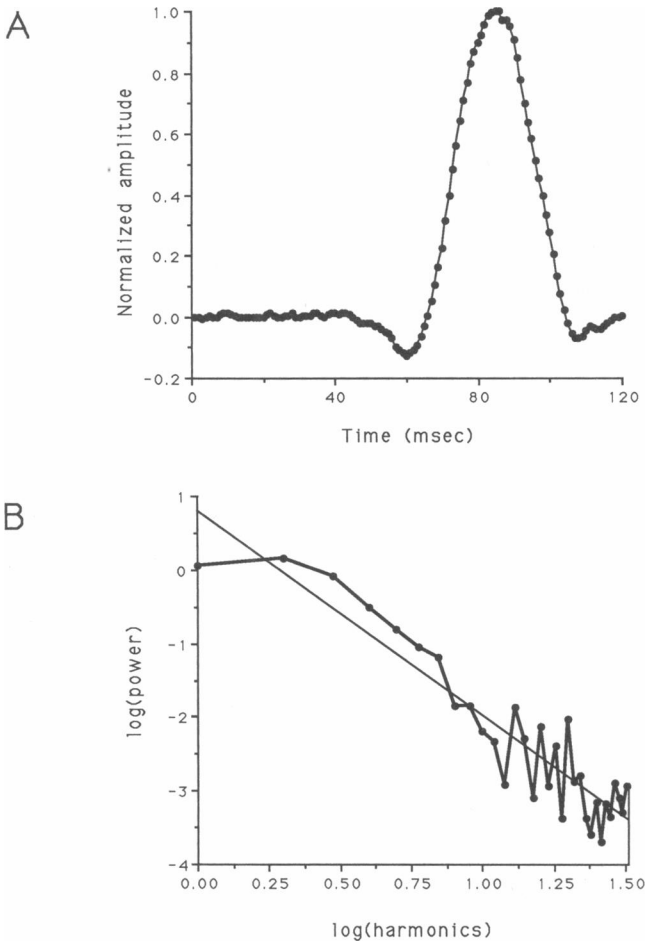

FIGURE 1 (*A*) A human QRS complex standard limb lead I. Peak amplitude has been normalized to one. (*B*). Log–log plot of the power spectrum computed from the QRS in *A*. Linear regression (*continuous line*) gives $y = 0.8 - 2.79x$, $R^2 = 0.87$. The electrocardiogram was recorded using an amplifier (Gould) (bandwidth, 0.05 – 1,000 Hz); it was taped (Neurocorder) and digitized off line (EGAA) at a sampling rate of 1,000 Hz. The 120 samples corresponding to the QRS complex were normalized to the positive peak amplitude. Power spectrum was computed using the fast fourier transform (FFT). Power was calculated taking the square of the amplitudes of the FFT terms; frequency is plotted as harmonics, the first harmonic being equal to 1/(window size) = 1/120 msec = 8.3 Hz and the last being equal to 235 Hz.

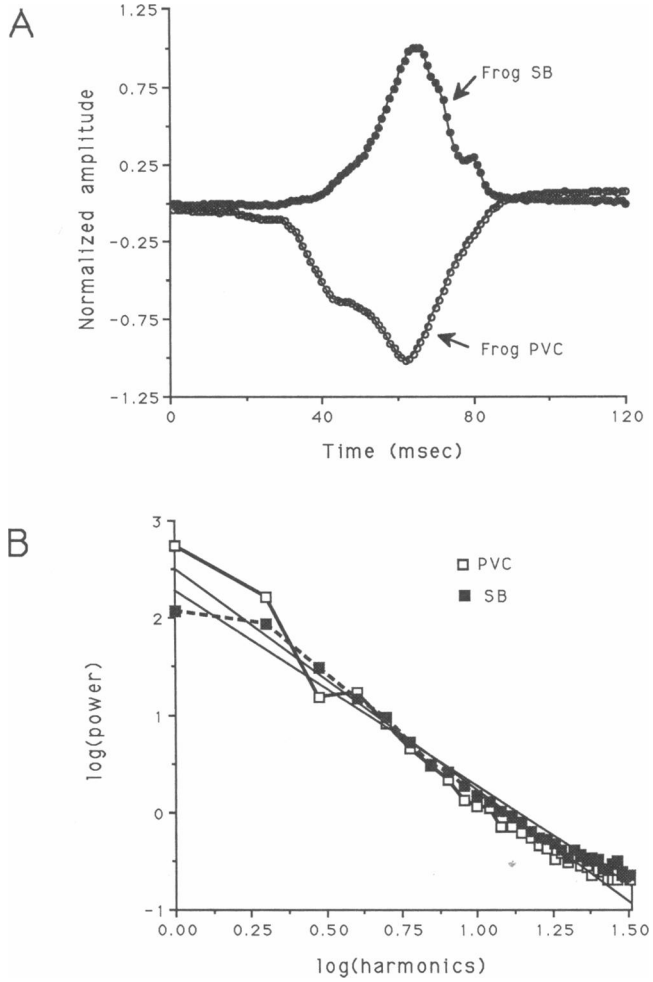

FIGURE 2 (*A*) Two frog QRS complexes. Upward deflection (*closed symbols*) corresponds to a normally activated sinus beat (Frog SB); downward recording (*open symbols*) is a premature ventricular complex (Frog's PVC) induced by an electrical stimulus applied at the apex through a small bipolar electrode. (*B*) Log–log plot of the power spectra computed from the two QRS complexes in *A*. Dashed line joining closed squares corresponds to the normally activated QRS. Continuous line joining open squares corresponds to the PVC. Fitted regression lines coefficients are $y = 2.28 - 2.02x$, $R^2 = 0.98$ for SB and $y = 2.5 - 2.27x$, $R^2 = .97$ for the PVC. Recording were obtained from a *Rana Catesbiana,* after pithing. One needle electrode was inserted subcutaneously in the right arm the other in the left foot. Same equipment, bandwidth and acquisition rate as those described in Fig. 1. Recordings were normalized to the positive (normally activated) or negative (PVC) peak amplitudes. Power spectra calculated as described for Fig. 1.

compound action potentials were obtained from frog sciatic nerve. When the multiple parallel fibers that comprise the sciatic nerve are synchronously excited by a supramaximal stimulus, a typical extracellular response is recorded, as depicted in *A* of Fig. 3. The reason for the pulselike shape of the response is well understood (11); it results from the passage of the individual action potential upstrokes beneath the extracellular recording electrode. Slight variations in its general morphology are associated with changes in the dispersion of the conduction velocity of individual fibers.

The computed power spectrum is presented in *B* of Fig. 3. Clearly the falloff of the harmonic amplitudes ($\alpha = 2.84$) is very similar to those obtained for the ventricular QRS complexes. Yet, here it is impossible to invoke a fractal activation network.

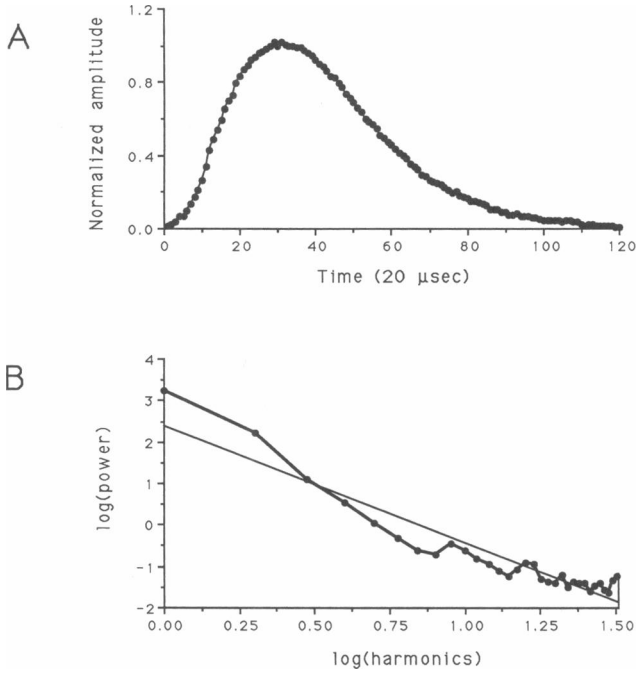

FIGURE 3 (*A*) Extracellular compound action potential recorded from a frog sciatic nerve. (*B*) Computed power spectrum for the trace in *A*. The regression line is: $y = 2.4 - 2.84x$, $R^2 = 0.9$. The sciatic nerve was dissected from a *Rana Catesbiana* (after stunning and pithing), and then mounted in a chamber for stimulation and recording. Supramaximal current pulses from a stimulus isolation unit (Grass) were applied to one end of the nerve. The reference electrode was placed near the other nerve ending which was crushed. The active recording electrode was located 3 cm from the stimulation electrodes. Same amplifier as in Fig 1, bandwidth .05 to 25 Khz, digitized every 20 μsec. Power spectrum computed as in Fig. 1. First harmonic here is equal to 1/(window size) = 1/2.4 msec = 0.42 Hz.

SUMMARY AND CONCLUSIONS

The results clearly demonstrate that the spectral features of the human QRS complex are not related to a fractal activation process. The frog heart data are conclusive; there is no His-Purkinje network in that species. Nonetheless, the spectral decay is similar to that in the human. Moreover, the fact that the spectrum of the PVC is identical to the normally activated QRS is an irrefutable argument against the fractal hypothesis. Finally, the results obtained in a cablelike structure such as the sciatic nerve, where no fractal anatomical substrate can be found, are also in conflict with Goldberger's conjecture. The results are in excellent agreement with the arguments of Lewis and Guevara (8), that the reason for the particular spectral decay of the QRS complex is related to its (large scale) pulselike shape and not to a fractal anatomical substrate of the His-Purkinje system.

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