Comments on 1/f^a power spectrum of the QRS complex revisited

Dear Sir:

This letter addresses the note of P. J. Lewis and M. R. Guevara entitled "A 1/f^a power spectrum of the QRS complex does not imply fractal activation of the ventricles." In 1985, we proposed the initial description of the His-Purkinje system as a fractallike structure (1) with a mathematical model suggesting two consequences of depolarizing the ventricles via such an irregular, self-similar branching network: (*a*) that the spectrum of the resultant depolarization waveforms (QRS complexes) should have a broad profile and (*b*) that this spectrum should exhibit power-law scaling. (Furthermore, the model predicted a relative decrease in the contribution of higher frequency potentials if the fractal network were disrupted.) Fourier analysis of electrocardiographic data from 21 healthy men was consistent with this hypothesis. This was, to our knowledge, the first attempt to dynamically link nonlinear (fractal) structure and function in a stable physiologic system.

In their letter, Lewis and Guevara attempt to demonstrate that the broadband, inverse power-law spectrum of the QRS complex does not require activation of the ventricles by a fractal process and that fractal activation in the ventricles cannot be inferred from analysis of the power spectrum of the QRS complex. They support their contention with two signal processing arguments. First, they show that the pulse wave resulting from one dimensional propagation in an ionic model of ventricular tissue has a frequency spectrum similar to that of the QRS complex despite the absence of any fractal structure. Second, they show that the power spectrum of a simple triangular pulse has an inverse power-law decay.

However, neither of these observations contradicts the premise or the validity of the model we proposed. We did not claim that all pulses with inverse power-law spectra are generated by fractal conduction. Obviously, the QRS complex represents the complex spatial and temporal summation of multiple myocardial potentials, not simply the electrical activity of an individual fiber. Our model was the first to suggest explicitly that the frequency content of the QRS complex was related not just to impulse transmission in the myocardium, but also to the sequence and timing of ventricular activation. Indeed, in our initial model, we deliberately excluded ventricular conduction and based our argument solely on the effects of having a fractal network distribute the activation pulses to the ventricles. Whereas transmission of a depolarization wave down a single myocardial fiber may generate an electrogram with a broadband, inverse power-law spectrum, the frequency spectrum of the QRS complex (representing the summation of multiple potentials) clearly depends on the sequence and timing of activation. For example, the broadband spectrum of the QRS complex generated by activation of the ventricles via the His-Purkinje network under normal conditions is quite different from that generated by nonfractal activation of the ventricles during ectopic ventricular arrhythmias (1) or ventricular pacing. Atrial activation via a nonhierarchical mechanism also fails to generate a broadband spectrum, as is apparent by comparing the *P* wave to the QRS complex.

Therefore, according to this fractal theory, the frequency content of the QRS complex is related to the macroscopic structure of the His-Purkinje system and not exclusively to the microscopic nature of the Purkinje-myocardial interactions or to local wave front propagation (2). Support for this counterintuitive notion has been provided by computer modeling studies (3–5) not cited by Lewis and Guevara in which a self-similar conduction system has been used to depolarize a three-dimensional network of cells. Such computer experiments reveal that with 9 or 10 generations of conduction system branchings, one can simulate QRS complexes that are essentially indistinguishable from those seen clinically. (Fig. 1) Furthermore, the simulated QRS complexes have a broadband frequency spectrum comparable to that observed physiologically (Fig. 1). These models also confirm that changes in the geometry of the branching conduction system may alter the frequency content of the QRS complexes, independent of any changes in myocardial conduction. Furthermore, Abboud and colleagues have simulated "late" potentials by showing conduction velocity in the distal part of the anterobasal conduction system or at the left ventricular apex in their model (4, 5). These investigators also showed more subtle changes in high frequency QRS content associated with altered conduction. To our knowledge, the fractal model is the only one to replicate normal QRS morphology, its frequency content and scaling pattern, as well as subtle abnormalities associated with certain pathologies (1–3).

The fact that the QRS complex and a triangular waveform share some similarities in overall scaling between higher and lower frequencies is to be expected given the fact that the QRS is indeed a triangularlike wave pulse. However, the QRS is clearly more complex in its morphology than that of a simple triangle and its frequency spectrum is correspondingly richer. From a physiologic point of view, more interesting questions are: what gives the QRS its triangularlike appearance and what accounts for the differences in morphology and frequency content between a simple triangular waveform and that of the actual QRS complex? Answers may be provided by simulations using a fractal model of the conduction system.

As noted by the authors, the full implications of having a fractallike conduction system remain uncertain. We proposed that this self-similar branching structure provides an essential component of electrical stability. Lewis and Guevara's suggestion that a branching network of this kind gives a mechanism for the rapid activation of the ventricular muscle is certainly consistent with our initial hypothesis. Furthermore, their suggestion that this rapid activation allows the depolarization wave to travel in an approximately one-dimensional fashion from the endocardium to the epicardium is also consistent with our hypothesis and suggests a mechanism to explain how a

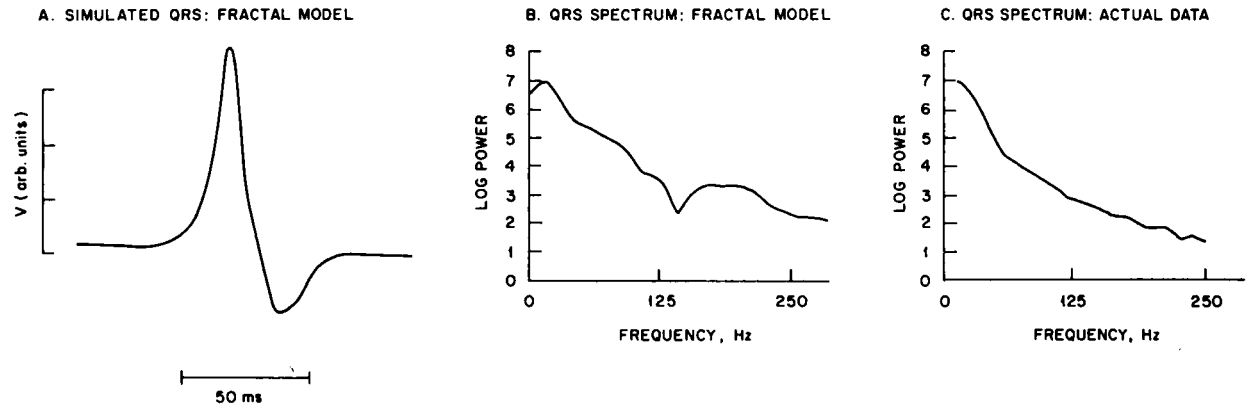

FIGURE 1 Activation of a three-dimensional network of myocardial "cells" by a self-similar conduction system in a computer model generates realistic QRS complexes (*left*) with a broadband frequency spectrum (*middle*) comparable to that obtained from actual ECG data in healthy men (*right*). Computer model QRS and spectrum are from the work of Berenfeld, Sadeh, and Abboud (3–5), clinical data are from (1). Note that the normal QRS complex is not a simple triangular waveform and that the broadband frequency content of the real and simulated QRS is richer than that of the discrete spectrum of a pure triangular signal. (Adapted from [2]).

complex waveform such as the QRS, the resultant of multiple wavefronts, may approximate the pulse generated in a one-dimensional cable model.

In summary, the fractal model of the cardiac electric conduction system provides: (*a*) a mechanism for explaining the normal activation sequence of the ventricles; (*b*) a mechanism for generating triangularlike QRS complexes with a broadband inverse power-law spectrum different from that seen if a large number of myocardial cells are depolarized in a nonhierarchical fashion; (*c*) a new means of studying perturbations of normal ventricular activation with simulation of late potentials and alterations of high frequency QRS potentials observed with certain clinically relevant pathologies.

REFERENCES

1. Goldberger A. L., V. Bhargava, B. J. West, and A. J. Mandell. 1985. On a mechanism of cardiac stability: the fractal hypothesis. *Biophys J.* 48:525–528.
2. Goldberger A. L. 1991. Fractal mechanisms in cardiac electrophysiology. *In* Cardiac Electrophysiology, Circulation, and Transport. S. Sideman, R. Beyar, and A. G. Kleber, editors. Kluwer Academic Publishers, Norwell, Massachusetts. 125–134.
3. Eylon, D., D. Sadeh, Y. Kantor, and S. Abboud. 1988. A model of the heart's conduction system using a self-similar (fractal) model. *Computers in Cardiology.* IEEE Computer Society. 399–402.
4. Berenfeld, O., D. Sadeh, and S. Abboud. 1989. Simulation of late potentials using a three-dimensional model of the heart's ventricles with fractal conduction system. *Computers in Cardiology.* IEEE Computer Society. 137–141.
5. Abboud, S., O. Berenfeld, and D. Sadeh. 1991. Simulation of high resolution QRS complex using a ventricular model with a fractal conduction system: effect of ischemia on high frequency QRS potentials. *Circ. Res.* 68:1751–1760.

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