

$1/f^\alpha$ power spectrum of the QRS complex does not imply fractal activation of the ventricles

Dear Sir:

The activation of the ventricular myocardium normally occurs via the cardiac His-Purkinje network. The highly ramified nature of the Purkinje system has led to the proposal that it is a "fractal-like" structure (1) (see also [2–7]). Because the activation of the ventricular myocardium occurs through the Purkinje network, it has been further hypothesized that the activation of the ventricles via the Purkinje fibers leads to fractal (time-scale invariant) depolarization of the ventricles (1) (see also [2–5]). The wave of activation of the ventricular myocardium is reflected in the QRS complex on the electrocardiogram (ECG). The averaged power spectrum of single QRS complexes from different individuals shows a $1/f^\alpha$ falloff (where f is frequency) with $\alpha \sim 4$ (1). Because power spectral densities which fall off as $1/f^\alpha$ can sometimes be linked to fractal processes (8), the $1/f^\alpha$ power spectral density was used to support the "fractal depolarization hypothesis." Here, we demonstrate that the $1/f^\alpha$ spectrum of the QRS complex does not require the activation of the ventricles to be a fractal process and suggest that fractal activation of the ventricles cannot be inferred from analysis of the power spectrum of the QRS complex.

SCALE-INVARIANT DYNAMICS

The term fractal refers to geometric structures which have the property of self similarity at all size scales (8). This definition need not be reserved for deterministic structures, but may also apply to stochastic structures whose statistical properties exhibit scale-invariant self similarity. Furthermore, the concept may be extended to time series where there are fluctuations on multiple time scales. The most widely used technique to investigate whether a signal is time-scale invariant is spectral analysis. If a signal is fractal, and thus time-scale invariant, the amplitude of its power spectrum will decrease with increasing frequency, falling off as $1/f^\alpha$ (where α is a positive number), i.e., the power spectrum is scaled by an inverse-power law (8).

THE FRACTAL DEPOLARIZATION HYPOTHESIS (1–5)

As a result of the fractal-like anatomy of the His-Purkinje system, Goldberger et al. (1–4) have argued that as the cardiac impulse is conducted through the branched Purkinje network, it "shatters into a myriad of stimuli" (1) which have different arrival times at the Purkinje-myocardial interface. This distribution of arrival times was attributed to the variability in the conduction times along the conduction pathways (due to

differences in length and conduction velocity) at all levels of branching: as the impulse travels through the successive generations of the inhomogeneous Purkinje network, the component pulses in the different branches tend to desynchronize or decorrelate to an increasing degree. It was further suggested in (1–4) that should the distribution of arrival times be determined by a fractal process, the change in the "net voltage at the myocardium" (1) would be a superposition of the individual pulses and should therefore exhibit fractal dynamics. The statistical properties of the "net voltage at the myocardium" were assumed not to be modified during the conduction of individual pulses through the ventricular wall after emerging from the Purkinje network. Because the QRS complex of the ECG reflects the depolarization of the ventricular muscle, it was postulated that the power spectrum of the QRS waveform should demonstrate the inverse-power law that is characteristic of fractal dynamics (1–5).

To test this hypothesis, Goldberger et al. (1) obtained one QRS complex (time series duration of 128 ms) from each of 21 healthy males using the V1-V5 bipolar ECG lead (sampling rate 500 Hz; 64 points/QRS complex). The "broadband spectrum" of the normal QRS was obtained by averaging the 21 power spectra. The averaged power spectrum was well fit by power-law scaling ($\alpha = 4.3$, $|r| = 0.99$ where r is the correlation coefficient) (1). We collected ECGs from 10 individuals using standard bipolar limb lead II and computed the averaged power spectrum of the QRS complexes (see caption of Fig. 1 for details). Fig. 1 *a* shows a typical QRS complex from lead II. Fig. 1 *b* shows that the averaged power spectrum (all 10 subjects) falls off as $1/f^\alpha$ ($\alpha = 3.8$) as seen by the linear fit to the log-log plot (slope = -3.8 , $|r| = 0.98$). In each of the 10 subjects, we obtained spectra resembling the averaged power spectrum shown in Fig. 1 *b* with individual values of α ranging from 3.2 to 4.3 with $|r|$ values between 0.91 and 0.97. These findings are consistent with the results of the earlier study (1). In the following, we will demonstrate that the $1/f^\alpha$ power spectrum of the QRS complex does not imply scale invariance, being simply a direct result of the basic shape of the QRS complex, which bears a strong resemblance to a simple triangular pulse.

SPECTRAL ANALYSIS OF PULSE FUNCTIONS

Although analysis of the amplitude and phase spectra of single pulsatile waveforms can provide insight into the shape of the waveform, it does not provide a representation of the frequency content reflecting fluctuations as thought of in fractal dynamics (i.e., fluctuations on all time scales).

In general, pulse functions generate power spectra with ripples or lobes; the peaks of the lobes fall off as the frequency

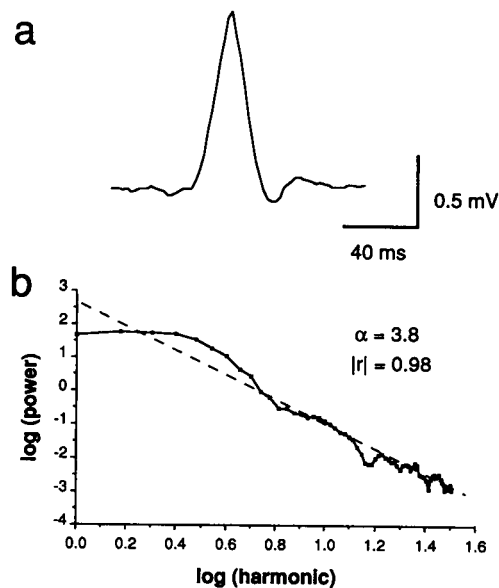


FIGURE 1 (a) A QRS complex from standard limb lead II. (b) A log-log plot of the averaged power spectrum of single QRS complexes from 10 male subjects. Linear regression (dashed line) gives a fit with a slope ($-\alpha$) of -3.8 and a y-intercept of 2.7 ($|r| = 0.98$). ECG signals were recorded with an isolation amplifier (0.05–250 Hz) and digitized at a sampling rate of 500 Hz (1). QRS complexes were extracted using a rectangular window of 128 ms (64 points) and the peak-to-peak amplitude was normalized to 1. Power spectra were then computed as the square of the modulus of the Fourier transform. Power is in arbitrary units and the frequency f is in harmonics, with 1 harmonic = $1/(\text{window width}) = 7.8$ Hz.

tends towards infinity (sometimes in a $1/f^\alpha$ fashion). For example, consider a simple triangular pulse function $g(t)$ (a nonscale-invariant waveform) of amplitude a and width $2b$ (Fig. 2 a):

$$g(t) = \begin{cases} (a/b)t + a & \text{if } -b < t \leq 0 \\ -(a/b)t + a & \text{if } 0 < t < b \\ 0 & \text{elsewhere.} \end{cases} \quad (1)$$

The power spectrum $S(f)$ of the function g , taken as the square of the modulus of the Fourier transform, yields:

$$S(f) = \left(\frac{4a}{b}\right)^2 \frac{\sin^4(bf/2)}{f^4}. \quad (2)$$

The term $\sin^4(bf/2)$ generates lobes, and the term $1/f^4$, with $\alpha = 4$, is responsible for the falloff of the peaks of the lobes. Note that the spacing of the lobes is dependent on the width $2b$ of the pulse, but the $1/f^4$ falloff is independent of the width. The $1/f^4$ falloff is clearly seen on a $\log[S(f)]$ vs. $\log(f)$ plot (Fig. 2 b).

The shape of the pulse determines the slope (and the goodness of fit) of the linear fit to the log-log power spectrum.

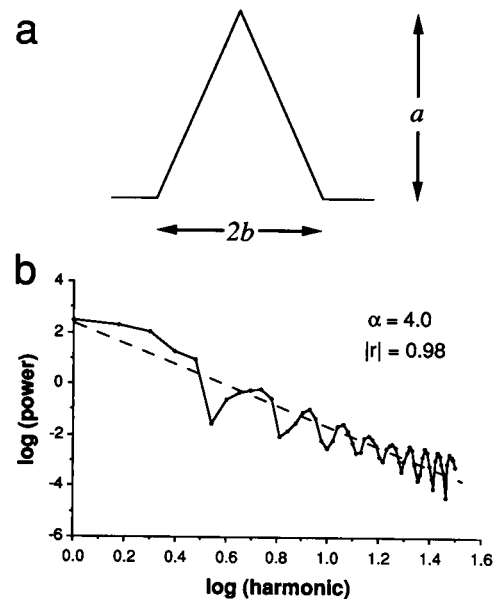


FIGURE 2 (a) A simple triangular pulse of amplitude a and half-width b . (b) A log-log plot of the power spectrum of the triangular pulse in (a). The power spectrum was numerically computed using a rectangular window containing 64 evenly spaced points. The pulse had an amplitude a of 1 and a width of 40 points ($b = 20$). Linear regression shows that the computed points are well fit ($|r| = 0.98$) by the dashed line with a slope ($-\alpha$) of -4.0 and a y-intercept of 2.4 , which agrees with the analytical result (Eq. 2). The depths of the lobes in the analytical power spectrum are of infinite depth on a log-log scale; however, the numerical algorithm does not show this due to the finite sampling. In fact, the frequency of the lobes in the analytical spectrum is much higher than that of the lobes shown here, as a result of aliasing. Note that averaging the power spectra of several triangles of slightly different widths would further diminish the presence of the lobes (see Fig. 1 b).

Pulses with sharper changes require a relatively larger contribution from higher frequencies, and therefore will have lower values of α . For example, it can be shown analytically that the power spectrum of a square pulse falls off as $1/f^2$. The power spectrum of a smoother pulse function will have larger low-frequency contributions, with smaller contributions from higher frequencies. This faster falloff will produce a higher value of α when a linear fit is computed. For example, the numerically calculated power spectrum of a sinusoidal pulse (one cycle of a sine wave from trough to trough with the troughs at 0) is fit well by a $1/f^\alpha$ falloff with $\alpha = 6.7$ ($|r| = 0.98$).

Note that the values of α found for the averaged power spectrum of single QRS complexes in Fig. 1 b ($\alpha = 3.8$) and in (1) ($\alpha = 4.3$) are close to the slope of the power spectrum of the simple triangular pulse ($\alpha = 4.0$). This is not surprising, because the QRS complex has an underlying shape that is triangular. The presence of lobes in the averaged power spectrum of Fig. 1 b (see also Fig. 1 in [1]) can still be detected, even though averaging the power spectra of QRS complexes of differing shapes and widths makes them less evident.

1/f^α POWER SPECTRUM FROM A NONFRACTAL CONDUCTION SYSTEM

To demonstrate that this type of 1/f^α falloff is not a property that can be used to distinguish fractal activation from nonfractal activation, we computed the power spectrum of an extracellular recording in an unambiguously nonfractal conduction system. One-dimensional propagation in an ionic model of ventricular tissue was modeled using the cable equation (9). The extracellular waveform was computed from the transmembrane current along the cable (10, 11). Fig. 3a shows a simulation of a unipolar recording taken as the upstroke of an action potential propagates down the cable. The power spectrum of the extracellular waveform in Fig. 3a shows that the amplitude falls off with increasing frequency (Fig. 3b). In fact, up to a frequency equivalent to the maximum frequency in (1), in terms of harmonics, the falloff is fit extremely well by 1/f^α (with α = 3.2, |r| = 0.99). This modeling result demonstrates that a 1/f^α power spectrum, similar to that of the QRS complex (Fig. 1b; Fig. 1 in [1]), is found in a system in which fractal conduction is excluded.

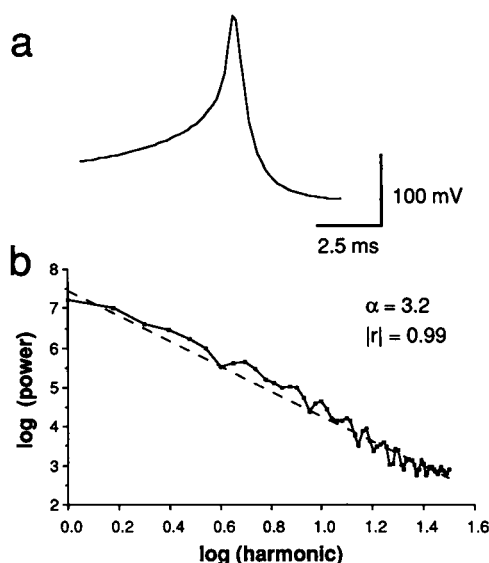


FIGURE 3 (a) An extracellular waveform of a propagated ventricular action potential computed from a one-dimensional cable model with the Beeler-Reuter formulation for the ionic currents (see [9] for details). The extracellular potential from a unipolar recording electrode was computed using the formula: $\Phi_e = \rho_e / 4\pi \int_V I_m / r dV$ where Φ_e is the extracellular potential at electrode, V is the volume of the cable, ρ_e is the extracellular resistivity (0.01 kΩ cm), and I_m is the transmembrane current at a distance r from the electrode (10). The cable has a length of 6.25 mm and a radius of 7.5 μm. The electrode was situated on the axis, 0.25 mm from the distal end of the cable. Window contains 64 points and the interval between points is 0.16 ms. (b) A log-log plot of the power spectrum of the extracellular waveform (a) reveals a 1/f^α spectrum, with linear regression giving a slope (−α) of −3.2 and a y-intercept of 7.4 (|r| = 0.99).

DISCUSSION

The evidence presented above shows that the 1/f^α spectral density of the normal QRS waveform does not imply fractal depolarization of the ventricles (1–5). This does not deny that the Purkinje system can be approximated by a fractal network, because the Purkinje fibers form a highly intertwined and interconnected branching structure (12–15). However, the above findings demonstrate that the 1/f^α spectra of the QRS complex is simply a result of its pulslike (large scale) shape and is not related to the temporal scale invariance that would be associated with fractal time series: scale-invariant activation remains to be shown.

Studying the QRS complex might not be a legitimate way to test the hypothesis of fractal activation of the ventricles via the Purkinje network, because the QRS complex is generated by current flow originating in ventricular muscle. The argument in (1) assumes that the “net voltage in the myocardium” consists of a superposition of the individual impulses from the Purkinje fiber branches, and that once an area of the ventricle is activated, conduction of the impulse in the ventricular muscle does not modify the statistics of the net voltage. However, the overall ventricular activation is influenced not only by the impulse arrival times at the distinct Purkinje-ventricular muscle interfaces (12), but by the geometry and electrical properties (including inhomogeneity and anisotropy of conduction velocity) of the ventricular myocardium. These complicating factors suggest that small variations in arrival times at different Purkinje-myocardial interfaces might not be preserved faithfully in the QRS complex.

The significance of a branched (perhaps fractal) conduction system remains unclear. It has been suggested that activation through a branching structure provides “electrical stability” (1). In our view, one simply needs a mechanism to rapidly and synchronously spread the wave of activation to the ventricular muscle, a task to which a branching network is well suited. This rapid sequential activation of a major part of the endocardium allows the wave of activation to travel in an approximately one-dimensional fashion from the endocardium outward to the epicardium (13), which may prohibit the collision of wavefronts and the evolution of refractoriness-activation profiles leading to the possible formation of reentrant rhythms resulting in ventricular arrhythmias.

Tim Lewis thanks the Fonds de la Recherche en Santé du Québec (FRSQ) and the Fonds pour la Formation de Chercheurs et l'Aide à la Recherche (FCAR) for support. Supported by a grant from the Medical Research Council of Canada.

Received for publication 3 April 1991 and in final form 3 June 1991.

REFERENCES

- Goldberger, A. L., V. Bharagava, B. J. West, and A. J. Mandell. 1985. On the mechanism of cardiac electrical stability: the fractal hypothesis. *Biophys. J.* 48:525–528.

2. Goldberger, A. L. 1990. Fractal electrodynamics of the heartbeat. *Ann. NY Acad. Sci.* 591:402–409.
3. Goldberger, A. L., and B. J. West. 1987. Applications of nonlinear dynamics in clinical cardiology. *Ann. NY Acad. Sci.* 504:195–213.
4. Goldberger, A. L., and B. J. West. 1987. Fractals in physiology and medicine. *Yale J. Biol. Med.* 60:421–435.
5. West, B. J. 1990. *Fractal Physiology and Chaos in Medicine*. World Scientific Publishing Co., Singapore. 67–76.
6. West, B. J., and A. L. Goldberger. 1987. Physiology in fractal dimensions. *Am. Sci.* 75:354–365.
7. Goldberger, A. L., D. R. Rigney, and B. J. West. 1990. Chaos and fractals in human physiology. *Sci. Am.* 262:42–49.
8. Mandelbrot, B. 1982. *The Fractal Geometry of Nature*. W. H. Freeman and Co., New York.
9. Lewis, T. J., and M. R. Guevara. 1990. Chaotic dynamics in a model of the propagated cardiac action potential. *J. Theor. Biol.* 146:407–432.
10. Plonsey, R. 1964. Volume conductor fields of action currents. *Biophys. J.* 4:664–673.
11. Roberge, F. A., A. Vinet, and B. Victorri. 1986. Reconstruction of propagated electrical activity with a two-dimensional model of anisotropic heart muscle. *Circ. Res.* 58:461–475.
12. Myerberg, R. J., H. Getland, A. Castellanos Jr., K. Nillson, R. J. Sung, and A. L. Bassett. 1976. Electrophysiology of endocardial intraventricular conduction: the role and function of the specialized conduction system. In *The Conduction System of the Heart*. H. J. J. Wellens, K. I. Lie, and M. J. Janse, editors. Lea and Febiger, Philadelphia. 336–359.
13. Spach, M. S., S. Huang, and C. R. Ayers. 1963. Electrical and anatomical study of the Purkinje system of the canine heart. *Am. Heart J.* 65:664–673.
14. Esmond, W. G., G. A. Moulton, R. A. Cowley, S. Attar, and E. Blair. 1963. Peripheral ramification of the cardiac conduction system. *Circulation.* 27:732–738.
15. Glomset, D. J., and A. T. A. Glomset. 1940. A morphological study of the cardiac conduction system in ungulates, dog, and man. II. The Purkinje system. *Am. Heart J.* 20:677–701.

Timothy J. Lewis and Michael R. Guevara

Department of Physiology and

Centre for Nonlinear Dynamics in Physiology and Medicine

McGill University

Montreal, Quebec H3G 1Y6 Canada