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Fractal Analysis of Vasomotion

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Introduction

Random-like fluctuations have been observed in blood flow in the microvasculature. This we term flow motion, thus distinguishing it from the closely related phenomenon, vasomotion, which refers to fluctuations in vessel diameter. This intermittent perfusion of vascular beds has been observed in skeletal muscle,¹ skin,² mesentery,³ and brain.⁴ These fluctuations are usually tied to spontaneously occurring rhythmic diameter variations in the feeding arterioles.⁵ The observations in skeletal muscle microvasculature suggest a relaxation oscillator (pacemaker) behavior with tonic modulation by perfusion pressure.⁶ The random nature of these fluctuations, which have readily observable periodicities, however, make response measurements difficult in terms of conventional amplitude and frequency changes. Thus, the need exists for improved methods of dealing with such responses. The question that can be asked is whether this randomness represents noise or chaos produced by a low-dimensional nonlinear system.⁷

The evidence given by our analysis suggests that there may be a low-order chaotic attractor. One cannot make such a conclusion with confidence, however, because the data currently available for the analysis covered only about two minute time periods. While fulfilling the requirements suggested by Grassberger and Procaccia,⁷ some low-frequency power in the signals would be better analyzed if the data strings were ten times as long.

Methods

The approach used here was based on the estimation of correlation fractal dimension of a single time series according to the Grassberger–Procaccia⁷ algorithm. An embedding procedure based on sequential delays of the original time series was used as suggested by Takens.⁸ Random noise, by having an infinite dynamic dimension, always has an estimated dimension equal to the embedding dimension. A chaotic process would lead to a finite dimension, which is independent of embedding dimension. A good review of the methods

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for calculating and interpreting the correlation integral and the fractal or chaotic dimension is given by Kaplan.⁹

The analysis software was written in Fortran and optimized for the Sun 3/60 workstation. This software was used in combination with the MATLAB (Matrix Laboratory) package, which allowed convenient manipulation of vectors for spectral analysis, filtering, and graphic display. Program validation was accomplished by running a known test case of chaos; the logistic equation, $x_{n+1} = rx_n(1 - x_n)$.

The data on flow fluctuations were acquired by making simultaneous measurements of diameter, d , and of mean erythrocyte velocity, v , in arterioles of the rabbit tenuissimus muscle, as described by Slaaf *et al.*⁵ Two different sites were studied: the transverse arteriole (TA) and a first-order side branch (FOS). The TA was of the order of 20 μm , and the FOS was 5 μm in mean diameter.

A velocity-forced Van der Pol was used as a potentially analogous signal, and was subjected to the same analysis. The equation for the acceleration-driven Van der Pol is

$\ddot{x} - \gamma\dot{x}(1 - \beta x^2) + \omega_0^2 x = f_0 \cos \omega_1 t$. To use derivative or velocity forcing we used a state variable form:

$$\begin{aligned}\dot{x} &= 0.7y + 10x(0.1 - y^2) \\ \dot{y} &= -x + 0.25\sin(1.57t).\end{aligned}$$

Results

Figure 1 shows flow-motion data in terms of red-cell velocity in rabbit tenuissimus muscle vs. time. Note the general random character of the data, but with rapid and slow cyclic behavior.

The power spectral density of this record (after mean removal) shown in Figure 2 indicates low- (0.05-Hz) and high-frequency (0.45-Hz) peaks. These peaks appear to correspond to the peaks in the TA and FOS diameter spectra. The dominant peak in the TA diameter spectra is 0.05 Hz and that of the FOS diameter spectra is 0.45 Hz, as shown in Figures 3 and 4, respectively. It is interesting to note that arterial blood pressure with its main periodicity at the heart rate (>3 Hz), as shown in Figure 5, is not directly represented as a fundamental harmonic in these responses. The peaks at 1.5 and 1.8 Hz may be aliased harmonic components of the cardiac pressure signal whose fundamental was 3.2 Hz. Arterial pressure, however, does seem to have a low frequency (<0.1 -Hz) component that may serve as an “input” forcing. Figures 2, 3, and 4 illustrate the basis of a potential problem with the analysis to follow, namely that there is power in the frequency range below 0.05 Hz, and perhaps even below 0.01 Hz. This means that with a data string covering about 100 s, the spectrum is poorly defined in the low range, since only one or a few cycles were observable in the data string.

The two-dimensional phase-plane portrait of red-cell velocity using a delay of five samples (5/10 s) is shown in Figure 6. The length of the delay is arbitrary and is found empirically by choosing one that maximizes the area covered by the continuous curve. Not much structure can be seen in this figure, but a general elliptically shaped trajectory is suggested when observing the time evolution of the points combined with a shuffling of trajectories. With a one-dimensional attractor (for example, a sine function) this phase plane or “return plot” or state-space portrayal shows a single elliptical trajectory. When the process is higher dimensional, such as a sine superimposed on a very slowly changing function, then the center of the ellipse changes continuously along the line $y = x$ or $x(t) = x(t - 5t)$, confusing the picture. The apparent intersection of the lines is removed by embedding in high enough dimensional space to prevent overlap. The low-frequency power seen in Figures 2 to 4 causes the confusion in Figure 6. A chaotic attractor without fixed points or boundaries is suggested, but not proved, by such a picture. Structured noise can give a similar result, but white noise cannot.

The fractal dimension D estimated via the Grassberger–Procaccia method vs. embedding dimension M is shown for the red-cell velocity data in Figure 7. The plateau suggests a dimension of 4.0. Similar plateaus were observed for all red-cell velocity and diameter data (range 4–4.7). If the signal were simply noise, there would be no plateau. Thus, the flow-motion pattern differs from random noise in that a fourth-order chaotic process is suggested by fractal analysis.

A velocity-forced Van der Pol oscillator was studied in an initial exploration of models that might be analogous to vasomotion in their behavior. Figure 8 shows the velocity oscillator variable vs. time when a chaotic mode was produced by using the rate coefficients given previously. The power spectrum of this record is shown in Figure 9, which indicates a peak near 0.1 Hz. The forcing frequency was set at 0.25 Hz, so the peak occurs at about half this value. The two-dimensional phase-plane plot of position vs. velocity is shown in Figure 10. A general elliptical shape is seen, but with seeming randomness in loop-to-loop position (shuffling of trajectories), and two portions of the trajectory show twisted loops near the upper and lower points.

Vasomotion showed a similar randomness in loop-to-loop position, but did not clearly exhibit trajectories that looped over themselves.

Figure 11 shows the estimated fractal dimension vs. embedding dimension for the forced Van der Pol oscillator. A plateau is reached near a value of 2.6. This plateau could be changed by varying the magnitude of the sinusoidal level. The plateau level always remained below 3.0. A magnitude of zero led to the expected value of one for a periodic relaxation oscillator. Similarly, at very high levels of forcing, the oscillator became locked to the forcing and this also resulted in unity dimension. The maximum dimension of 3 is lower than what was estimated for vasomotion, suggesting the presence of more complex dynamics.

Conclusion

Fractal analysis of vasomotion data shows that the phenomenon involved is unlike random noise, and may involve chaos. The dynamic behavior appears to have a fractal dimension of at least four, and is thus more complex than the relaxation oscillator behavior of the single forced Van der Pol type. The data suggest that a model with at least two coupled relaxation oscillators is necessary to explain observations.

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FIGURE 1.

Flow-motion time-series data. Red-cell velocity (mm/s) measured in a transverse arteriole of rabbit tenuissimus muscle.

FIGURE 2. Power-spectral density $[S(f)]$ of flow motion in transverse arteriole. Spectrum computed from data shown in Figure 1.

FIGURE 3.

Power-spectral density $[S(f)]$ of transverse arteriole diameter changes during vasomotion. Spectrum computed from diameter variations measured simultaneously with velocity measurements shown in Figure 1.

FIGURE 4.

Power-spectral density $[S(f)]$ of first-order side-branch diameter changes during vasomotion. Spectrum computed from diameter variations measured simultaneously with velocity measurements from feeding transverse arteriole shown in Figure 1.

FIGURE 5.

Power-spectral density $[S(f)]$ of femoral artery pressure. The spectrum was computed from pressure measured simultaneously with the velocity measurements shown in Figure 1.

FIGURE 6.

Phase-plane portrait of flow-motion. A lag of five time samples (0.5 s) was used to produce the second spatial coordinate.

FIGURE 7.
Correlation fractal dimension (D) vs. embedding dimension (M) for flow motion in a transverse arteriole shown in Figure 1.

FIGURE 8.
Forced Van der Pol oscillator time series.

FIGURE 9.
Power-spectral density $[S(f)]$ of the forced Van der Pol oscillator.

FIGURE 10.
Phase-plane portrait of the forced Van der Pol oscillator.

FIGURE 11. Correlation fractal dimension (D) vs. embedding dimension (M) for the forced Van der Pol oscillator.