Spectrotemporal Analysis of the Electrocardiogram

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There are many simple solutions to complex problems, and most of them are wrong.

H.L. Mencken

Analysis of the tail end of the signal-averaged QRS (so-called late potentials) can predict arrhythmic events, such as after myocardial infarction, a benign course being indicated by a negative test. However, measurements have not been standardized, especially with regard to spectral analysis. Spectral analysis—looking at the frequency distribution of signals' content (voltages, in the case of the QRS) rather than their time course—is an informative way to further process signals. Spectral analysis has been around a long time, and even the Fourier transform, commonly used to look at late potentials, was developed almost 200 years ago. With the availability of computer processing, there has been recent discussion as to the value of applying spectral analysis to the electrocardiographic diagnosis of coronary artery disease1-3 as well as to evaluation of late potentials.4-6

The computer enables the physician to do very complicated mathematics, which can be incorrectly applied. The portion of the QRS to be examined is one source of controversy. The broader the segment, the finer the resolution of the examination but the lesser the focus on the region of interest. Additionally, the Fourier transform presumed a periodic repetition of the examined window, but the electrocardiogram is discontinuous and manipulations of the data at the edges of the period examined (“windows”) are used to account for this, along with subtraction of mean data to establish the baseline.

The latest twist on spectral analysis is use of the computer to overcome the conceptual difficulty of comparing a frequency representation to a time representation of the QRS by showing both graphs together. One sees the energy in the various frequency bands at sequential time intervals. This time-varying spectrum or three-dimensional approach (voltage versus time versus frequency), sometimes called a “running Fourier transform” or “spectrotemporal mapping,” was also described some time ago.8 Time-varying spectra published in the cardiology literature have used the Fourier transform,7,9-11 although preliminary reports have used other techniques12,13 that perhaps are useful for more detailed analysis.14 Generally, multiple overlapping spectral curves are made from limited electrocardiogram segments, successively moving the data segments by small intervals, to create a three-dimensional impression.

The graphics of spectrotemporal mapping are entertaining—late potentials are “foothills” after the large wave forms or spectral mounds of the QRS15—but have not yet substantially contributed to our understanding of late potentials. They provide an easy way to look for and perhaps discount contaminating noise,10 possibly even leading to single-beat analysis. An incentive for looking at the breadth of the QRS is that the basis for ventricular tachycardia (VT) is often a focus of slowed and late anterior depolarization that can be obscured by subsequent normal inferobasal forces16 if one looks merely in the time dimension, or by normal right ventricular depolarization in the case of right bundle branch block from anteroseptal infarction.

A more important advance may be the use of these approaches to diagnose coronary disease before chronic infarction (or with hibernating myocardium), or to make a rapid diagnosis of acute infarction while deciding about thrombolytic therapy in the emergency room. Specifically, coronary ligation causes major alterations in spectral content of the QRS by the 500th beat in dogs or during angioplasty balloon inflation in patients; coronary stenosis can be identified by high-frequency electrocardiogram in patients without Q waves,17 and the high-frequency changes can be resolved by angioplasty.3 Perhaps anti-ischemic drugs can also modulate the electrocardiogram.

In this issue of Circulation, Cain et al18 report on spectral examination (Fourier transform analysis) of the entire cardiac cycle (not just the late potential) in patients who did and did not have VT after infarction and in normal subjects. The study is contrived in part...
to address prior criticisms of their use of mathematics. For example, they aim to obviate subtracting out mean data by analyzing a complete cycle with isopotential points as boundaries; this may be inadequate if the mean of all data samples is not zero. They avoid the vagueness of data segments bounded by T wave onset; however, segments based on cycle length change with each beat and from one patient to another. They avoid applying mathematical manipulations, such as the Blackman-Harris window function generally used by others, to account for edge discontinuities. This “rectangular” window reduces the signal-to-noise ratio, and, also, the authors averaged only 100 beats, suggesting a less-than-ideal signal-to-noise ratio—but noise is broadband and can disproportionately increase the fraction of the voltage that is thought to be high frequency because the QRS is mostly less than 40 Hz. However, the point of this editorial comment is not to critique the mathematics but to add perspective. The analysis by Cain et al was another way of relating frequency changes to time (of spectrotemporal mapping) by subsequently applying an inverse Fourier transform to look at the temporal distribution of those frequency bands that systematically distinguished patients with VT from those with no VT. Until the publication of this report, there has been an inadequate look at the QRS that precedes late potentials. In this report we learn that VT patients have proportionately less high-frequency voltage (i.e., normalized to total QRS voltage) throughout their QRS. Parenthetically, this discrimination was done by a test that depends on a normal distribution of voltage data, and this may not be the case, at least for late potential voltage. At any rate, postinfarction patients without VT actually had an increased portion of their QRS that was high frequency as compared with normal subjects, at variance with other reports (perhaps reflecting different methodology), and this may be the most important observation in the long run. Specifically, the portion of the voltage in two frequency bands (13–56 Hz and 70–128 Hz) was less in VT patients than in no-VT patients. This result is consistent with empirical observations that the voltage in these very bands is relatively increased at the end of the QRS (in the late potentials) of patients with VT. That is because the high-frequency bands were “eccentric”—dispersed, or displaced later in time. Perhaps slowed conduction of high-frequency forces contributes to the low-amplitude late potentials in VT patients. There are problems with the analysis of the data by Cain et al. One wonders if the harmonics of line current (60 Hz and 120 Hz) added noise and might explain the inadequate separation of VT from no-VT patients at 57–69 Hz and above 120 Hz. Not only could important signals of brief duration be diluted by analysis of the entire cycle, but segments of different lengths were examined in each individual, which can dramatically alter the results. Cycle lengths were shorter on average by 18 msec in the VT patients but their QRS durations were longer by an average of 21 msec (indeed, seven of the 40 VT patients had bundle branch block and nine an intraventricular conduction delay, compared with one and three, respectively, of the no-VT group). Thus, the QRS represented an average of 15.44% of the data segment in the VT patients, 12.45% in the no-VT patients, and 11.43% in normal subjects. This could not only alter the areas calculated by the Fourier transform; the temporal displacement of voltages in VT patients could have reflected conduction system disease rather than VT. Another difference between the two patient groups was that the no-VT controls had higher ejection fractions and fewer had anterior infarcts (one presumes they had less muscle damage). Was that why they had more high-frequency voltage throughout their QRS, rather than because they did not have VT? It is enticing to consider temporal displacement of high-frequency voltage as being due to slowed conduction, which in turn might explain why there are late potentials. Only examples are provided, however. Indeed, tabulation of data might indicate that different leads reflect different infarct locations, or that inferobasal depolarization could in some leads obscure the temporal displacement of high-frequency voltages in VT (or all) patients after anterior infarction. Finally, before this work is assigned clinical implications, the validity of the technical issues (avoidance of subtracting out mean data, avoidance of defining regions of interest by computer algorithm, avoidance of applying a window function) and the very usefulness of the identified frequency bands need to be dealt with in terms of the bottom line: positive predictive value in diagnosing VT. The computer offers seemingly unending opportunities to analyze signals from the heart (e.g., electrical and ultrasonic signals) using advanced mathematical techniques. It becomes easy to fall into pits of mathematical uncertainty or clinical obscurity. Cain et al are to be congratulated for bravely risking these pitfalls to resolve issues in spectral analysis so as to increase the positive predictive value of the signal-averaged electrocardiogram. It is difficult to know at this point whether their manipulations are correct and provide practical refinements. As with the pilot work of Cain et al that introduced us to analysis of the signal-averaged electrocardiogram in the frequency domain, this investigation is seminal to diagnosis not only of arrhythmia risk but also of coronary disease per se. It encourages prospective high-fidelity or high-frequency electrocardiography of the entire QRS (not just late potentials), not just in search of VT but in prospective diagnosis of the substrate of VT. Perhaps someday we might anticipate electrocardiographic diagnosis of hibernating myocardium, ischemia, or mere coronary stenosis. The importance of the latter is that thrombolytic agents and angioplasty alter the potential for a substrate for VT.
References
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