Fractionated electrical activity and continuous electrical activity: fact or artifact?

MARK E. JOSEPHSON, M.D., AND ANDREW L. WIT, PH.D.

ENDOCAVITARY MAPPING during chronic ventricular tachycardia in patients with healed myocardial infarction and/or ventricular aneurysms has been used to locate the region from which ventricular tachycardia originates. During the process of mapping, bipolar electrograms with unusual configurations have been recorded from the chronically ischemic or healed infarcted regions, both with electrode catheters and with hand-held probes. Electrograms consisting of multiple "high-frequency" components with low amplitudes (<1 mV) and long durations that may exceed 100 msec have been termed "fractionated." Although fractionated electrograms have these qualitative descriptors, quantitative criteria to distinguish among various types of electrograms have not been established. Such quantification, however, is limited by the fact that electrogram amplitude and width can vary depending on the interelectrode distance, contact, and degree to which the signal is amplified. We have recently undertaken studies in normal subjects and in patients with infarction and ventricular tachycardia to develop such criteria using a 1 cm interelectrode distance and a fixed gain of 1 mV = 1 cm. With these methods normal electrograms had amplitudes greater than 3 mV, durations of 70 msec or less, and amplitude/duration ratios of 0.046 or more. Electrograms outside these values were termed "abnormal." Multicomponent electrograms that fell beyond 1 SD from mean values of abnormal electrograms (amplitude 1.4 ± 0.9 mV, duration 93 ± 40 msec, ratio 0.017 ± 0.012) were termed "fractionated." Although the pathophysiologic significance of the relationship between multicomponent "abnormal" and "fractionated" electrograms is unclear, our arbitrary criteria for fractionated electrograms were an amplitude of 0.5 mV or less, a duration of 133 msec or longer, and/or an amplitude/duration ratio of 0.005 or less. Whether quantifying the electrograms provides useful information beyond the qualitative descriptors requires investigation.

Sometimes the total electrogram may occur throughout the entire cardiac cycle, a phenomenon that has been called "continuous electrical activity." Fractionated electrograms (but not continuous activity) can also be recorded during sinus rhythm, in which case they may extend beyond the QRS complex of the surface electrocardiogram.

The detection of fractionated electrograms and continuous electrical activity in patients with ischemic heart disease has prompted a number of proposals concerning their possible significance: (1) the occurrence of fractionated electrograms during sinus rhythm may help to identify those patients who are prone to develop ventricular tachycardia; (2) the site at which fractionated electrograms are recorded during sinus rhythm may indicate the site of origin of ventricular tachycardia; and (3) the region at which continuous electrical activity is recorded during tachycardia defines the site at which the reentrant circuit is located. These proposals are all based on the assumption that fractionated electrograms are caused by slow, inhomogeneous conduction, a property that can cause reentry. That continuous electrical activity is synonymous with the location of the reentrant circuit is based on the fact that, during certain kinds of reentrant excitation, the impulse is continuously conducting through a circumscribed region (reentrant pathway) and that an electrode placed on such a pathway would record activity from one or another part of this pathway throughout the cardiac cycle.

These proposals are also a source of controversy. It has been suggested that fractionated electrograms and continuous activity may be artifacts, resulting from movement between the electrode and myocardium. Artifacts caused by movement should be particularly
obvious because of the high amplification at which the
electrograms are recorded (because of their low ampli-
tude). Another suggestion has been that fractionated
activity does not represent local electrical events but
rather activity occurring far away from the recording
electrodes. To demonstrate these points, Ideker et al. recorded fractionated and continuous activity in a
sponge sutured to the anterior surface of the canine left
ventricle, and Gallagher et al. recorded continuous
activity from a bowl of Jell-o. Another suggestion has
been that fractionated electrograms may be a product
of the filtering characteristics of the amplifiers used to
record them. Some who concede that fractionated
electrograms and fractionated activity might not be
artifacts question whether their occurrence is predict-
tive of tachycardia or indicates the location of reentrant
circuits. It is obvious, therefore, that there are impor-
tant questions that must be resolved to determine the
significance of these abnormal signals.

Are fractionated electrograms artifacts dependent on
recording techniques?

The demonstration that fractionated electrograms
can be recorded from sponges or bowls of Jell-o does
not necessarily mean that those recorded from ischemic
myocardium are caused by motion artifacts or represent distant activity. Fractionated electrograms,
similar to those recorded in patients, have been found
in regions of acute and chronic infarction in dogs, with
both catheter electrodes and close bipolar electrodes
fastened directly to the heart. Fractionated electro-
grams have also been recorded with close bipolar elec-
 trodes or with unipolar electrodes from isolated superfused preparations of canine myocardium obtained
from areas of chronic or healed infarction in which
mechanical motion is negligible and where nearly per-
fected contact can be ensured between electrode and tis-
sue. This demonstrates that fractionated electrograms
can be real and can represent local activity under the
electrode. In the study in isolated tissues, alteration of
the filtering characteristics of the amplifiers did change
the morphology of fractionated electrograms by in-
cluding or excluding slow and high-frequency compo-
nents, but fractionation was still obvious in recordings
with a band pass above 0.6 Hz. Thus, although the
exact morphology of the electrograms may change at
the different filter settings, fractionation cannot be cre-
ated or abolished.

The demonstration that fractionated electrograms
are real under these experimental conditions does not
disprove suggestions that fractionated electrograms or
continuous activity recorded from patients might result
from motion artifact. Although catheter motion might
cause what appears to be fractionation, for the follow-
ing reasons we believe that in many instances frac-
tionated activity is not an artifact of electrode movement.
Fractionated electrograms, including continuous activity,
occurs almost exclusively in areas of scar tissue or
at borders of aneurysms. They can be recorded during
intraoperative mapping from the same regions where
they were recorded during catheter mapping, even
though electrode-tissue contact during intraoperative
mapping is expected to be much better than with a
catheter and the contraction of the empty ventricles on
cardiopulmonary bypass is less vigorous. It is hardly
likely that the same movement artifact would be ob-
served under these different conditions. Slight reposi-
tioning of recording electrodes further toward the bor-
der with more normal myocardium (as little as several
millimeters) usually results in a lesser degree of frac-
tionated activity or in its disappearance; slight catheter
movement would hardly be expected to alter motion
characteristics. The fact that the characteristics of
the electrogram can markedly change when the electrode
is moved short distances within the scarred region dur-
ing intraoperative mapping also argues against the
electrogram representing distant activity and not local
phenomena.

What causes fractionated electrograms?

If fractionated activity is not caused by motion, what
then do fractionated electrograms tell us about the
electrophysiologic traits of the regions from which they are
recorded? Fractionated electrograms per se cannot be
equated with slow conduction. In experiments on iso-
lated preparations of ventricular muscle or Purkinje
fibers, elevating K⁺ to depress the conduction velocity
simply reduced the amplitude and broadened the dur-
ation of the extracellular electrogram, but the slow con-
duction did not cause fractionated electrograms. In an
experimental study on electrical activity in the muscle
surviving on the epicardial aspect of canine in-
farcts, it was shown that fractionated electrograms
usually appeared as the infarcts aged and their appear-
ce corresponded to the ingrowth of fibrous tissue
during infarct healing. The connective tissue sepa-
rates the myocardial fibers, decreases their intercon-
nections, and distorts their orientation. Microelectrode
studies have shown that because of this separation on a
microscopic level, individual muscle bundles may be
activated asynchronously and the individual compo-
nents of the fractionated electrograms appear to repre-
sent electrical activity in the different surviving muscle
bundles. Since action potentials recorded in the vicini-
ty of fractionated electrograms often had fast upstrokes, these individual components had a "high frequency." Since only a few bundles embedded in large amounts of connective tissue were present, the extracellular electrical field was small and high amplification was needed to see them. Despite the nearly normal transmembrane potentials, activation of the region where fractionated activity was recorded was very slow. Slow conduction might be a result of the diminished intercellular coupling caused by the separation of the fibers. This finding of a structural basis for fractionated electrograms in infarcts should not be surprising, since Spach et al. have shown that similar fractionated electrograms can be recorded from the distal Purkinje system, where individual Purkinje bundles are separated from one another, and from nonuniformly anisotropic atrium, where connective tissue separates muscle bundles. In our recent study of the anatomy of subendocardial regions of patients with ventricular tachycardia, in which fractionated electrograms can be recorded, we have found this anatomic substrate — surviving myocardial muscle bundles separated by large amounts of connective tissue.

At the very least, therefore, fractionated electrograms indicate areas of abnormal anatomy and conduction that are suitable substrates for reentry. Experimental data suggest that the occurrence of fractionated electrograms depends on a specific anatomic substrate, one that produces nonuniform anisotropic conduction and slow regional activation. Myocardial infarction is a source of this substrate as well as a cause of ventricular tachycardia. However, the appearance of fractionated activity during sinus rhythm need not indicate a priori that the patient will have ventricular tachycardia. Fractionated activity might occur without the presence of reentrant circuits. Whether any specific measurements describing the fractionated activity (such as duration, number of components, etc.) will predict the eventual occurrence of tachycardia will require careful studies. Whether the site(s) at which fractionated electrograms are recorded during sinus rhythm in patients with histories of ventricular tachycardia indicate the sites where tachycardias originate must be determined by comparing these two in the same patients. Recent data from our laboratory have demonstrated that these abnormal electrograms are widespread and are not specifically predictive of the site of origin of ventricular tachycardia(s).

Is continuous electrical activity during ventricular tachycardia an indication of reentrant excitation?

In their studies on coronary arterial ligation in the dog, Waldo and Kaiser and Boineau and Cox showed that extension of fractionated electrograms into diastole often coincided with the appearance of ventricular tachycardias or fibrillation. More recently, El-Sherif et al. have used a composite electrode to record from a large area of an infarcted region in the dog and have found that electrical activity spanning the cardiac cycle was often associated with ventricular tachycardia. Mapping of activation sequences in regions where there was continuous activity sometimes showed reentrant excitation. Josephson et al., during endocavitary catheter mapping, found in some patients progressive fractionation of electrograms in areas of healed infarction or aneurysms during initiation of tachycardia and during sustained tachycardia. Initiation of the tachycardia was dependent on these electrograms spanning the cardiac cycle. This was the first demonstration of continuous activity in human beings. Of note was the finding that electrograms recorded from adjacent bipolar pairs on the same catheter did not show continuous activity, suggesting that this continuous activity was local. In addition, pacing at the same cycle length as the tachycardia failed to produce continuous electrograms. Furthermore, disturbance of the continuous electrograms by pacing during the tachycardia resulted in its termination. We have also noted similar observations during intraoperative mapping.

We agree that if one could record from an entire reentrant circuit, continuous electrical activity would be seen. If the circuit were relatively large, a composite electrode might be needed, but if the circuit were small it might fit under a relatively close bipolar electrode. However, this does not mean that finding continuous activity in a specific region indicates that the reentrant circuit is either present or located there, since continuous activity can also be caused by slow nonuniform conduction in the absence of reentry. Can it, then, be determined whether the site at which continuous activity is recorded is the location of the circuit? If continuous activity stops spontaneously during tachycardia or can be interrupted by pacing techniques that do not terminate the tachycardia or alter its cycle length or QRS morphology, then the continuous electrical activity cannot be related to the reentrant circuit. One must also exclude the possibility that continuous electrograms may merely represent fractionated electrograms that are present during sinus rhythm with a duration that approximates the ventricular tachycardia cycle length. Such electrograms would span the cardiac cycle during tachycardia and appear to be continuous.

We therefore suggest that the site at which continu-
ous activity is recorded might be the site of a reentrant circuit if the following criteria are met in an electrophysiologic study: (1) During protocols designed to initiate tachycardia there is an increase in fractionation and duration of the electrogram until, at some critical duration, tachycardia starts. (2) Continuous activity (with repetitive patterns) persists during tachycardia and interruption of continuous activity by pacing techniques changes tachycardia cycle length or QRS morphology, or terminates tachycardia. (3) During the cycles immediately preceding spontaneous termination of tachycardia there may be changes in the pattern of local continuous activity. (4) Continuous activity can be recorded only from a circumscribed area and not throughout the infarcted region. (5) When pacing the heart at the same cycle length as the tachycardia during which continuous activity was recorded, there is no continuous activity. This proof is more definitive if pacing a site close to the origin of tachycardia produces the same activation and contraction pattern as during the tachycardia. PACing is necessary to eliminate the possibility that continuous activity reflects nonspecific rate-dependent alterations in conduction. (6) Finally, cooling, compression, or resection of the region of continuous activity stops the ventricular tachycardia while similar interventions in other regions do not. All of the above are compatible with the interpretation that continuous activity indicates reentry and have been observed in humans. Ultimately, however, mapping excitation patterns with high-density electrode arrays in regions where continuous activity is recorded is required to demonstrate reentrant excitation.

Conclusion

We conclude that fractionated electrograms and continuous electrical activity are real phenomena that result from an anatomic substrate in which muscle fibers are separated by scar tissue to produce slow and nonuniform anisotropic conduction. Careful consideration of recording procedures can eliminate motion artifacts. All electrograms that are fractionated and/or continuous, however, may not be related to reentrant phenomena. Recognition of the limitations of recording methods and use of stimulation techniques with analysis of the response of the electrograms to these stimulation techniques can in many instances exclude "continuous electrical activity," which is unrelated to the mechanism of tachycardia. The demonstration of localized continuous electrical activity that is inextricably related to the initiation and maintenance of ventricular tachycardia as outlined above strongly suggests that such activity is a reflection of reentrant excitation.

References

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M E Josephson and A L Wit

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