On the Nature of Ventricular Tachycardia in Coronary Heart Disease

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The prerequisites for the development of uniform ventricular tachycardia (VT) late after myocardial infarction (MI) have been fairly well understood for many years. Compared with survivors of cardiac arrest or patients evaluated for nonsustained VT, patients with uniform VT have more extensive infarction, more profound left ventricular dysfunction, and more abnormal endocardial activation during sinus rhythm.1,2 The slow conduction required for formation of VT circuits is caused by tissue disruption secondary to myocyte death and secondary fibrosis, as well as electric remodeling (eg, altered connexin expression) of the surviving cells.3,4 Although infarct-related damage may be transmural, the importance of the endocardial substrate for the formation of VT circuits has been established by histological-electrophysiological correlation3,5,6 and by the effects of catheter and surgical ablation. Given the importance of the topic, it is puzzling that there have been few data collected on the influence of modern cardiovascular care on the development of VT. There is a general sense that current pharmacological therapy and appropriate revascularization have reduced the incidence of VT after MI, but it is not clear how these therapies might affect the underlying pathophysiology.

Article see p 1887

In this light, the study by Wijnmaalen and coworkers in this issue of Circulation offers considerable promise.7 In this study, 36 consecutive patients with VT late after MI (mean 13±9 years) were carefully evaluated to compare the influence of acute reperfusion of the infarct-related artery on the characteristics of the spontaneous/induced VT and the underlying VT substrate. There were 14 patients in the reperfused group and 22 who were not reperfused. The baseline characteristics of the 2 groups were similar, although the reperfused group presented with VT earlier after MI and were slightly younger. The majority of patients had New York Heart Association class I heart failure, and the mean left ventricular ejection fraction was 30±13%. Electrophysiological study, including left ventricular endocardial voltage mapping and VT mapping, was performed in all patients; catheter ablation was attempted in 27. In 10 patients, ventricular restoration with intraoperative anatomic cryoablation and transmural biopsy was performed.

Compared with the nonreperfused group, patients in the reperfused group had more rapid VT (after exclusion of amiodarone: 254±51 versus 334±65 ms) and were more likely to have very rapid VT induced (cycle length <250 ms: 71% versus 23%). Although the total endocardial scar area was similar (65±48 versus 85±46 cm²), the area of voltage <0.5 mV was smaller in the reperfused group (21±25 versus 42±32 cm²), and these patients were more likely to have patchy endocardial scars (71% versus 14%). Biopsy samples from reperfused patients often demonstrated nontransmural fibrosis and often had significant areas of viable myocytes. On the basis of this information, it seems clear that the authors’ hypothesis is correct: “Early reperfusion during [acute] MI results in smaller and less homogeneous scars . . . and faster spontaneous and inducible VT . . . late after MI.”

As suggested by the somewhat archaic-sounding title, I was hopeful that this valuable set of observations would provide greater insight into the relationship of the VT circuit to the underlying VT substrate, particularly in patchy infarcts. As the authors point out, much of our understanding of VT in healed infarction is based on dense infarction, in animal models as well as patients treated with surgical ablation, who typically had aneurysm formation. Unfortunately, although the study provides detailed characterization of the infarct substrate, there is limited characterization of where or even whether the VT circuit exists within the architecture of the infarct. Attempts were made to establish reentry isthmus sites, but few data were presented to support the success of this endeavor (which is notoriously difficult in poorly tolerated VT) or the relationship of these sites to voltage abnormalities. The authors do state that loosely defined circuit isthmus sites were found within the infarct-related artery territory in 33 of 36 patients (presumably for the presumed clinically dominant VT morphology); in 3 other patients, the VT QRS morphology was “compatible with” an exit site within the infarct-related artery territory. The usual manner of clinical proof of circuit isthmus site location requires detailed mapping during VT, which results in VT termination and subsequent noninducibility, ideally with a single radiofrequency lesion. In poorly tolerated VT, a successful procedure (often requiring extensive ablation) is proposed as a logical surrogate. In the present study, the acute success of both catheter and surgical ablation was rather disappointing. In the catheter ablation group, only 14 of 27 patients had complete success, with 4 patients having persistent inducibility of the presumed clinical VT; although not statistically significant, there appeared to be a trend toward less successful procedures in the reperfused group (which included 3 of the 4 failed
procedures). This suggests the possibility that there was an incomplete understanding of the relationship of the circuit to the underlying anatomy, which was not the major focus of the study but is what I was hoping for.

To my interpretation, this wish was also expressed by the authors’ statement that “reentry circuit locations and VT characteristics vary greatly among patients and are influenced by the 3-dimensional geometry of infarcted areas.” Catheter mapping studies, albeit in patients with extensive voltage abnormalities representative of a nonreperfused group, have demonstrated important relationships of the circuit and the infarct substrate: (1) Critical isthmus sites (defined by concealed-entrainment criteria and single-site ablation) are typically located within the dense scar (bipolar electrogram voltage $<$0.5 mV). (2) Circuit exit sites (defined by local activation coincident with the onset of the QRS) are observed in the infarct border zone as described by voltage mapping. (3) A spatially finite connection between central isthmus and exit sites can often be detected, even during sinus rhythm (eg, long stimulus to QRS pace mapping or “corridors” of relatively preserved voltage or conduction within the dense infarct). These patterns are observed more often than not in relatively slow, uniform reentrant VT in the setting of chronic infarction; they cannot necessarily be expected in more rapid VT or VT due to a nonreentrant mechanism.

Are VT circuits in patients with reperfused infarcts different? I would expect that even in the setting of patchy scars, VT circuits would exist within the area of lowest voltage, similar to the situation in more confluent scars (Figure). Alternatively, it must be considered that even in patients with coronary disease, VT does not necessarily occur because of scar produced by an infarct. Some patients with extensive

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**Figure.** Electroanatomic mapping (posteroanterior view) of the left ventricular endocardium in sinus rhythm in a patient with ischemic cardiomyopathy and a patchy posterior wall voltage abnormality. The color pattern is identical to that used in the study by Wijnmaalen et al: bipolar voltages $<$0.5 mV (dense scar) are shown in red, normal tissue ($\geq$1.5 mV) in purple, and intervening voltages in the other colors. Although there are areas of preserved voltage within the infarct, the area critical for the VT circuit (as judged by entrainment mapping and response to ablation) was within the dense scar. Pacing from sites within the scar duplicated the VT morphology and had a long stimulus to QRS interval. Radiofrequency energy delivery at a site within the dense scar resulted in prompt termination of the clinical VT.
infarction have idiopathic VT, defined as nonreentrant VT that arises from normal tissue. Some patients with coronary disease and even clinical MI have reentrant VT that is not due to a coronary disease substrate. The possibility is suggested in at least some of the voltage maps in the reperfused group (eg, the third example in the reperfused group in Figure 2 in the report by Wijnmaalen et al). How is it possible to have dramatic reduction of left ventricular function, as well as VT, secondary to coronary disease in the absence of extensive endocardial scar? It is at least plausible to suggest that both the left ventricular dysfunction and the VT are secondary to nonischemic cardiomyopathy, in which case the abnormal voltage substrate is more likely epicardial.

The authors are to be congratulated on a wonderful effort, managing a huge data set over a long time period, which provides important insight into the effect of acute reperfusion on VT substrate in humans. As with most worthwhile studies, the work by Wijnmaalen and coworkers raises yet more questions to be addressed. Answers to these questions regarding the nature of VT in healed infarction are essential to our understanding of the disease process and for development of rational treatment strategies in our patients.

Disclosures

None.

References


Keywords: Editorials ■ ablation ■ electrophysiology ■ myocardial infarction ■ tachyarrhythmias
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_Circulation_. 2010;121:1881-1883; originally published online April 19, 2010;
doi: 10.1161/CIR.0b013e3181e0b226
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2010 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circ.ahajournals.org/content/121/17/1881

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