Editorial:
Status of Surgery for Ventricular Arrhythmias

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IN 1913, MINES1 demonstrated that a circulating wave of excitation in a closed ring of myocardial tissue could be interrupted by dividing the ring at some point. He foretold the role of surgery for reentrant rhythms when he stated, "... the best test for a circulating excitation is to cut through the ring at one point... the vigorous circulating wave and its instantaneous arrest by section of the ring is a sight not easily forgotten."2 The clinical demonstration of this phenomenon came in 1969 when Sealy and co-workers divided an accessory pathway in a patient with Wolff-Parkinson-White syndrome subjected to recurrent reentrant supraventricular tachycardia.3 Interest has continued in devising methods to localize and divide circuits of reentry that underly other reentrant rhythms in man.4 Excluding surgical treatment of the preexcitation syndromes, interest has centered on the potential for surgery to ablate or modify the site of origin of recurrent ventricular arrhythmias. This communication will consider the status of surgical treatment for ventricular arrhythmias.

We will consider the mechanisms underlying ventricular arrhythmias, clarify the method used for the localization, and discuss the rationale of surgical approaches based on the mechanism and location of the arrhythmia. Our understanding in each of these areas is incomplete. Judging from animal models, for example, a spectrum of different mechanisms of ventricular arrhythmias may exist, depending on the etiology of the arrhythmia and the stage of the disease process. Although such phenomena as reentry, automaticity and "triggered" activity experimentally appear reasonable to consider as mechanisms, our ability to discriminate between them at the clinical level is limited. Nonetheless, there appears to be a growing tendency, however unjustified, to ascribe tentatively the mechanism of reentry to rhythms that can be initiated and terminated by programmed stimulation. Empirically, this differentiation has value in that it provides 1) a means to assess medical management, 2) a rationale for pacemaker devices, and, most relevant to this discussion, 3) some assurance that the rhythm can be evoked for investigative purposes at the time of surgery. It further appears that programmed stimulation may identify patients in whom surgery is likely to be successful. One possible but unproved explanation is that the ability to initiate and terminate ventricular tachycardia by programmed stimulation generally requires a macroreentry circuit rather than a microreentry circuit. (The experimental model of "triggered" activity in isolated cells would clearly be an exception.) The larger size of the reentry circuit would thus permit a less precise surgical approach to be effective. Rhythms that cannot be induced and terminated by programmed stimulation are often thought to be caused by automaticity and are therefore not only more capricious but also more demanding of a precise intervention.

Substantial progress has been made in investigating and treating reentrant ventricular arrhythmias associated with cardiomyopathies unrelated to coronary artery disease. This work has been largely pioneered by Guiraudon and Fontaine in Paris.5,6 Using mapping of the earliest area of epicardial activation during ventricular tachycardia as a guide to the site of reentry, these workers demonstrated that a simple transmural ventriculotomy at the site so identified can be effective in abolishing further episodes of ventricular tachycardia. Patients are selected who demonstrate the reentry phenomenon. The success of simple ventriculotomy in cases where the tachycardia can be manipulated by programmed stimulation and the failure of the technique in patients who do not demonstrate this characteristic support the previous hypothesis concerning the potential size of the reentry circuit. When the same investigators attempted to apply the method of simple ventriculotomy to the treatment of ventricular arrhythmias related to coronary artery disease, the results were disappointing.6

Because of the greater incidence of ventricular arrhythmias related to coronary artery disease, considerable effort has been expended to identify the underlying mechanisms. Despite extensive experimental research, no detailed study has yet depicted a definitive three-dimensional representation of the reentry circuit of sustained ventricular tachycardia related to coronary artery disease. Nonetheless, a growing body of evidence tends to support the hypothesis that, at least in chronic infarction, reentry is related to the border zone.

Recently Guiraudon7,8 and co-workers described an innovative surgical technique, encircling endocardial ventriculotomy (EEV), that was formulated on the hypothesis that reentry theoretically can be sustained at any portion of the border zone of chronic ischemia and infarction. Their initial group of patients presented with resistant ventricular arrhythmias 1 month to 8 years after suffering myocardial infarction, and all demonstrated reentry during programmed stimulation preoperatively. An EEV was performed along the limits of the endocardial fibrosis that

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separated the abnormal from the normal myocardium. Over the parietal portion of the ventricular wall, the EEV was a full-thickness section, sparing only the epicardium and the coronary vessels. When the septum was involved, the ventriculotomy was approximately 1 cm deep and nontransmural. One patient received a bypass graft to a stenosed coronary artery and two patients underwent myocardial resection of aneurysmal tissue located at the apex and on the inferior wall, respectively. There were no operative deaths. Antiarrhythmic therapy was discontinued postoperatively and no recurrence was noted during a follow-up of 6–24 months.

Josephson, Harken and co-workers describe a technique for the surgical treatment of ventricular tachycardia related to ventricular aneurysm. They report 12 patients in whom the tachycardia could be initiated and terminated by programmed stimulation preoperatively. Intraoperative mapping was used to localize the apparent site of origin of tachycardia to the border zone. On the basis of the intraoperative mapping, endocardial resection in the area of apparent origin was performed. The combined early and late mortality in this series was 25%. The survivors have remained free of sustained ventricular tachycardia without antiarrhythmic agents during the 9–20 months of follow-up.

The above series are still relatively small, and a long-term follow-up is not available. The success that has been achieved appears related in part to the inclusion of the border zone and the septal areas in the surgical intervention. There are advantages and disadvantages to both approaches.

The EEV (Guiraudon et al.) directs an action at the entire border zone and theoretically is more complete. Mapping is not essential. The procedure assumes that the extent of infarction is generally greatest on the endocardial aspect of an infarction and endocardial fibrosis thus becomes the key to defining the limits of the border zone. (This relationship may not be true with infarcts that include the posterobasal regions of the left ventricle.12) The acumen of the surgeon may clearly be taxed in determining discrete limits to the fibrosis. It would appear that the EEV requires more technical skill, especially when performed in a relatively normal-sized heart. The closure obtained would appear technically more secure. The EEV theoretically could diminish ventricular function in that the incision and closure may compromise normal myocardium situated in the epicardial layers overlapping the borders of fibrosis.

Endocardial resection (Josephson et al.) is clearly more limited in scope, and is directed at only one portion of the border zone. Eleven of the 12 patients operated on had ventricular aneurysms. Detailed epicardial and endocardial mapping is required to locate the area for resection. This requires not only that the patient remain in a stable ventricular tachycardia after ventriculotomy, but also that the electrograms recorded from the ischemic border allow identification of local activation during tachycardia. Clearly, Josephson selected patients whose tachycardia could be readily induced and terminated preoperatively. This, however, is no guarantee that the ventricular tachycardia will be induced in the operating room. Anyone who has mapped for any time under clinical operative conditions appreciates the remarkable antiarrhythmic properties of general anesthesia and of cardiopulmonary bypass. Electrograms recorded from the border zone of aneurysms or infarcts are often very difficult to interpret. Furthermore, there is no guarantee that a neighboring area of the border zone might not begin to sustain reentry later although it was not evident at the time of operation. The extensive undermining of the ventricular wall required clearly leads to thinning of the myocardium at the site of resection; one case of late pseudoaneurysm with rupture has already been encountered. The overall hemodynamic effect of adding endocardial resection to routine aneurysmectomy would appear to be minimal. This is confirmed by the postoperative studies described by Josephson.

In summary, both EEV and endocardial resection direct attention to the border zone and to the septal areas. However, both suppose the presence of a localized area of pathology, i.e., endocardial fibrosis or aneurysm, and not diffuse myocardial disease as a substrate of the ventricular arrhythmia. Both also prolong the duration and the complexity of the operative intervention.

The long-term sequelae of both procedures must be defined in regard to hemodynamic function, the structural integrity, and the electrophysiologic stability of the ventricle. Added to this is the uncertain natural history of a dynamic disease process, such as coronary artery disease, which has not been essentially altered. The difficulty in assessing the significance of any late arrhythmia after such surgery is obvious. Even if the QRS morphology of any recurrent arrhythmia is different from the preoperative rhythm disorder, might this not be the same reentrant rhythm with a different pattern of propagation to the myocardium brought about by the surgical intervention? The surgical interventions described here focus on the existence of a substrate of ventricular arrhythmia that is largely static, and thus amenable to modification or ablation. This emphasis minimizes the role of dynamic variables such as spasm, reperfusion, stretch, and circulating catecholamines in the genesis of ventricular arrhythmias.12

Surgery of this type is still investigative and should be considered only if conventional treatment has failed. Since mapping may sometimes improve the outcome, these operations should be performed only in institutions performing electrophysiologic investigations preoperatively and intraoperatively. Newer mapping techniques are being developed13 that may permit mapping of rapid, unstable or evanescent arrhythmias. The availability of newer antiarrhythmic agents14 may diminish the need for surgical treatment. However, much of the pioneering work in the surgical treatment of ventricular arrhythmias is flourishing in countries with ready access to potent antiarrhythmic agents such as amiodarone.
References

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