Implanted Standby Defibrillators

To The Editor:

I am concerned about the implications of the Editorial by Drs. Lown and Axelrod which appeared in the October 1972 issue of Circulation. Although the authors rightfully point out the numerous technical problems still to be overcome in the development of such defibrillators, their conclusions in the last three paragraphs indicate a heavy bias in favor of “practical research.” Although Dr. Lown has been eminently successful with this approach, I believe he is unjustified in using this yardstick to evaluate the research activities of others. The authors take Drs. Mirowski et al. to task for a 1970 article in which they state: “It is too early to determine exactly the indications and contraindications of the standby automatic defibrillator.” Drs. Lown and Axelrod believe such a statement should be answered before social energies are expended on research and development of such a device.

Since when have answers to such questions been required before research is undertaken? Are Drs. Lown and Axelrod so clairvoyant that they can see the ultimate impracticability of someone else’s research energies thereby prematurely labelling that work “an imperfect solution in search of a plausible and practical application”? Fortunately, sincere investigators will continue to attack problems even when the prospect of solution is slight and when sensible people shake their heads.

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To the Editor:

We appreciate the editorial focus afforded by Circulation1 to our transvenous automatic defibrillator concept.2 In this Editorial, Drs. Lown and Axelrod raise a series of objections and emphasize the difficulties in implementing such an approach. While these admittedly great difficulties have not escaped our attention, the potential advantages, overlooked by the authors, amply justified the energies expended to explore this uncharted area.

Interestingly enough, after an intensive search for the real and hypothetic difficulties, Drs. Lown and Axelrod recognize that the technologic problems are subject to solutions. However, some of their assumptions are simply unwarranted. An example is the statement that energies required for catheter defibrillation in man would necessarily be higher than those in dogs. In fact, preliminary clinical results compare favorably to animal data, and this even under conditions of extreme ischemia.4, 6 As far as endomyocardial effects of catheter electroshock are concerned, the lesions, when present, are small, localized, and of little significance in view of the otherwise fatal outcome of the arrhythmia. Fortunately, Dr. Lown’s findings of myocardial damage due to transthoracic DC electroshock7 have not led to the abandonment of this technic by the medical profession.

In contrast to Drs. Lown and Axelrod, we do not foresee difficulties in identifying populations at particularly high risk of dying from ventricular fibrillation. The implantation of the transvenous automatic defibrillator in these patients may not necessarily be more “burdening” (using the authors’ expression) than drug therapy. In fact, at this time we are aware of no effective long-term antiarrhythmic regimen capable of reducing the present prohibitive toll of sudden coronary deaths.

The authors’ overcautious and negative attitude to the approach under investigation seems certainly premature at this experimental prototype stage. Would it not be more appropriate to postpone disqualification of this new way of approaching a major cause of mortality, however imperfect it may seem to be, until it faces the test of clinical trials?

Regrettably, almost three decades elapsed between the development of the first experimental model of the artificial cardiac pacemaker4 and its clinical acceptance. After an additional decade or more of extensive experience and continuous technologic advances, the pacemaker still remains a rather imperfect and occasionally a harmful device. Nevertheless, its reward-risk ratio has been sufficiently high to warrant its use. We are confident that much less time will be required to develop the transvenous automatic defibrillator, to bring it to an attractive reward-risk level, and, finally, to clinical acceptance.

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The authors reply:

To the Editor:

Drs. Mirowski and co-workers engage in minor polemics, but alas do not address themselves to the substantive criticisms raised in the Editorial. One essential and troublesome question relates to assessing operational adequacy of an implantable standby device in absence of the catastrophic condition for which it was intended. When such in vivo testing is impossible, how is one to be certain that the probe is properly positioned and not encased in thrombus or fibrous tissue so as to properly sense cardiac quiescence? What basis can there be for surmising discharge of its stored electrical energy at the appropriate time? What assurance is there that the electrical pulse will prove adequate? On different occasions, in the same animal, markedly disparate electrical energies may be required for achieving defibrillation. What a priori guidelines can exist for anticipating the defibrillating threshold in a particular patient? If the discharge is adequate the first time, what about recurrent arrhythmia, when energy release is set high in relation to the limited power stored in the device? And, what guarantee can one provide against the hazards of malfunction? Mirowski and co-workers imply abiding faith in the prowess of science; these commendable sentiments by themselves do not assure that these problems are soluble.

The clinical question still remains, for whom is such a device intended? If Mirowski and co-workers have found a method for precisely identifying the patient susceptible to sudden death, they remain remarkably modest in divulging such valuable knowledge. The undue enthusiasm for their device is exposed by the dismaying concept that the surgical insertion of a power plant within the body may not be more “burdening” than swallowing of pills.

But, there is a broader issue not dealt with in the Editorial or in these letters which relates to medical priorities. No society, whatever its wealth, can adequately respond to all social needs. Indeed within our own society the brief honeymoon between bountiful government and scientific investigative undertakings is coming to an end. It will be increasingly essential to define precisely the competitive position of health requirements to other legitimate societal goals. Within medicine itself support for investigative endeavor will need to be justified in relation not only to cost but to certain not problematic benefits. We continue to be unpersuaded that a complex, untestable, and costly electronic device provides a legitimate answer to the problem of sudden death justifying diversion of scarce social resources for its development.

In response to Dr. Moss’ letter we pretend no clairvoyant powers, but merely common sense, unfortunately the least common of the senses.

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Effect of Procaine Amide on Canine Purkinje Fibers

To the Editor:

The article by Drs. Rosen, Gelband, and Hoffman (Circulation 46: 528, 1972) describing their findings of the effect of procaine amide on the electrophysiologic properties of canine Purkinje fibers appears somewhat contradictory to previously published studies. By using a donor animal to perfuse isolated cardiac tissues, they correlated electrocardiographic and electrophysiologic effects with therapeutic and toxic plasma concentrations of procaine amide. In this preparation at therapeutic concentrations of 6–9 µg/ml the control action potential duration (APD) was prolonged from 230 to 270 msec and the effective refractory period (ERP) increased only from 200 to 210 msec. Therefore the ΔAPD/ΔERP ratio was greater than one with procaine amide.

However, two of the same authors have previously stated that, while both the APD and ERP are prolonged with procaine amide, the ERP is prolonged proportionately more than APD, thus producing a ΔAPD/ΔERP ratio of <1. They implied that this effect modifies the usual relationship between conduction and refractoriness in reentrant circuits and may be important inabolishing reentrant arrhythmias.1 Bigger also has recently stressed the same proportionately greater prolongation of ERP than prolongation of APD with procaine amide.2

In the article under discussion the authors noted a modifying effect on repolarization by the perfusate potassium level. They also speculated that the absence of early effects on conduction in the presence of the demonstrated early effects on automaticity may reflect a time lag in achieving tissue concentrations comparable to plasma levels. They did not comment specifically on their somewhat contradictory findings of the effects of procaine amide on the ΔAPD/ΔERP ratio.

Possibly the discrepant findings mentioned above may be explained by the effect of potassium or some artifact of the experimental preparation. It would seem important to resolve these differing results in an attempt to explain the useful effects of procaine amide on an electrophysiologic basis.

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