myocardium located between the site of impulse emergence (from the right branch) and the recording electrodes. In acute “complete” RBBB with normal H-V intervals the H-RVA interval was significantly prolonged since it now represented conduction time from His bundle to left ventricular endocardium, and from the latter site to the RVA (across the septum in a left-to-right direction). In some cases of acute “complete” LBBB in which the H-RVA interval maintained the preblock value, the H-V interval increased by 12–20 msec. This is shown in figure 1. The first QRS complex without a “complete” LBBB morphology had a small q wave in lead I. The corresponding H1-V1 and H1-RVA1 intervals measured 50 and 70 msec, respectively. In the second beat (with a “complete” LBBB pattern) the H-V increased to 70 msec but the H-RVA remained unchanged, indicating that a conduction delay had not occurred through the right branch.

These findings corroborate studies performed from surface electrocardiograms,6 in revived human hearts,7 and the Elizari preparation.8 For instance, the left-sided schematic in figure 2 indicates that the preblock H-V interval represented conduction time from His bundle to left ventricular endocardium. On the other hand when “complete” LBBB occurred, the H-V interval increased slightly because of the (normally) longer conduction time through the right branch, without necessarily implying the presence of an additional conduction delay within this structure.

Probably the 20-msec increment of the H-V interval is artifactually long (due mainly to the incorrect assessment of the onset of the QRS complex). Nevertheless, the importance of the H-VRVA interval warrants further studies of the RVA electrogram as already is being done in some laboratories.9

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References


The authors reply:
To the Editor:

Dr. Castellanos’ data suggest that the prolonged H-V intervals frequently noted in patients with left bundle-branch block partially reflect a slightly longer conduction time in the right bundle-branch system. This is in keeping with Durrer’s demonstration that the onset of left ventricular activation precedes that of the right by 5–10 msec (Castellanos’ reference 7).

These observations contrast with our experience in patients with rate-dependent left bundle-branch block. In two previously reported cases1 and in four cases studied recently (Rosen KM, Wu D, Denes P, Dhandhar R: Unpublished data), H-V intervals were similar during normal conduction and left bundle-branch block. These results suggest that left bundle-branch block does not unmask a longer conduction time in the right bundle branch.

The apparent conflict between our data and that of Castellanos may reflect patient-to-patient variability in the degree of asynchrony between onset of left and right ventricular activation. Further electrophysiologic studies, with endocardial mapping, should clarify the patterns of normal and abnormal ventricular activation.

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Circulation, Volume XLVII, May 1973
Letters to the Editor

Reference

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 impactability 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.2,3 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 electroshock4 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 pacemaker 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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