

Pacemaker Failure in Complete Heart Block

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■ The role of the electrical pacemaker with myocardial electrodes in the treatment of atrioventricular conduction blocks is an important one. Various observers, however, have reported a progressive increase in power needed to stimulate the heart and eventual failure of stimulation. The reason for this stimulation failure has been reported as due to increased electrical resistance from scar formation about the electrodes.¹⁻⁴ This study is an attempt to identify causes of failure of myocardial stimulation.

Methods

Atrioventricular conduction blocks were produced in mongrel dogs (group 1) by the technique of Taufic, Bashour, and Lewis.³⁻⁵ One or two wires were sutured into the right ventricular myocardium and an indifferent electrode placed in the musculature of the thoracic wall. All wires were brought out through stab wounds and identified as to location of implantation by color of insulation. Transistor-type electrical stimulators were used to pace the heart in the immediate post-operative period until the animals were well stabilized. The pacemakers were used daily from one to three weeks; then at weekly intervals until the heart failed to respond. Those animals which developed cardiac failure were placed on continuous electrical cardiac stimulation until the cardiac failure was relieved, usually a matter of two or three days; then they were returned to the schedule of intermittent stimulation. When the heart no longer responded to the electrical pacemaker, exploration was done in an effort to determine the cause of failure of stimulation.

In another group of dogs (group 2), attempts were made to produce myocardial scarring by electrocoagulation of the right ventricular myocardium and by ligation of the anterior descending coronary artery. These animals were allowed to recover from the scar-producing procedure; then atrioventricular conduction blocks were produced.

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A myocardial electrode was introduced into the area of scarring and another into normal myocardium. The indifferent electrode was placed in the muscle of the chest wall. Electrical pacemaking of the heart was carried out in a manner similar to those of the first series.

In five of the animals of group 1, exploration showed scarring and granulation tissue about the previously implanted wires. This area of scarring was used for implantation of a new myocardial electrode, care being taken not to bring the electrode in contact with normal myocardium but to limit contact to scar only (group 1a). A normal myocardial electrode and an indifferent electrode were also implanted just as was done in group 2.

Canvas binders were placed on all dogs to prevent their pulling out the wires or chewing them in two.

Results

Autopsies of the animals with myocardial scarring produced by coronary ligation or electrocoagulation (group 2) showed so much normal myocardium in the area of scarring that this group of animals must be included with the normal myocardium (group 1). In the discussion below, the reference to animals with scarring includes only those five group 1 animals with a large build up of scar and granulation tissue into which the electrodes could be placed without any contact with normal myocardium (group 1a).

The findings in all animals were essentially similar, the differences being only in the amount of current necessary for cardiac stimulation. The initial current necessary to drive the heart with an electrode in normal myocardium and one in the chest wall (group 1) was 3 to 6 ma. With both electrodes in normal myocardium (group 1), the initial current for cardiac pacemaking was between 1.5 and 3 ma. With one electrode in scar tissue and one in the chest wall (group 1a), the initial driving current was from 4 to 9 ma. Stimulation through the electrode implanted in scar tissue using the electrode implanted in normal myocardium as an indifferent electrode required 3 to 6 ma. Stimulating through the

electrode in normal myocardium with the indifferent electrode in the scar required only 1.5 to 3 ma.

During the first 10 days, there was a gradual increase in current necessary to stimulate the ventricle. The increase was from 2 to 7 ma., and at the end of this period, a rather stable plateau was reached and no further increase of current was necessary. The increase occurred with all combinations of electrode placement but was least with both electrodes in normal myocardium. In a few cases, a sharp increase over a 24-hour period, from very low current to the full limit of the pacemaker, was needed to stimulate the ventricle. After 24 hours, no stimulation occurred even with full power. Failure of stimulation occurred from two days to one year after implantation of the electrodes.

Exploration and evaluation showed that the cause of failure in each case, without exception, was loss of contact between the electrical pacemaker and the heart. The electrode wires were either broken or had pulled out of the heart.* The most common site of breakage was the junction of the insulated portion and the bare wire at the point of implantation into the heart. This was true even though a portion of the insulated wire was also embedded in the myocardium. The second most common site of breakage was in the chest wall at skin level. Failure of stimulation under these circumstances was, within the limits of the experiment, sudden and complete. Breakage of the wire within the insulation as the wire traversed the chest cavity resulted in a sharp increase of current necessary to drive the heart. After approximately 24 hours of increased current there was complete failure of stimulation in these cases.

In every case the heart could be driven con-

*Originally, the electrode wires were placed in the myocardium as a simple suture and held in place with additional silk sutures. It was soon learned that the wires pulled out of the heart in as short a time as one to four weeks. From then on, the myocardial wires were placed as double loop or figure-of-8 sutures with a lock. These could not be pulled out of the heart.

tinuously, well within the limits of the standard commercial pacemakers, until wire breakage or loss of myocardial contact occurred.

Discussion

Electrical ventricular pacing in patients with atrioventricular conduction block may be necessary for prolonged periods of time. The electrical pacemakers which have been developed and are sold commercially seem to be reliable for long periods and require little care except for infrequent battery changes. Nevertheless, failure of stimulation has occurred after varying times in patients with implanted myocardial electrodes. This failure has, in the past, been attributed to increasing resistance because of scar tissue around the electrodes. More recently, several observers have pointed out that wire breakage may be the important factor.⁶⁻⁸ This study indicates that ventricular stimulation can occur through scar tissue and that peak resistance is probably reached within the first two to three weeks after implantation. Furthermore, the power necessary for ventricular stimulation, even with severe scarring, is well within the range of the standard commercially produced pacemaker. This study further indicates that the major limiting factor in myocardial stimulation is the durability of the wire electrodes since all cases of failure were due to wire breakage or loss of wire contact with the heart.

There is little doubt that wires which can resist breakage due to prolonged continued flexing can be produced. It is doubtful, however, that such wires could be relied upon for 5 to 10 years, a period conceivably required by some patients with atrioventricular conduction blocks. At present, all available pacemakers require wires to stimulate the heart, and all such wires can break. One apparent solution to the problem of wire breakage is a wireless pacemaker. These are now in the experimental stage, but their development in this day of miniaturization should be possible so that a small button-sized receiver can be placed on the myocardium and the pacemaker signals transmitted from

a small pacemaker worn on the chest or elsewhere on the patient's body.

Summary

Atrioventricular conduction blocks were produced in dogs, and ventricular stimulation was accomplished by the use of electrodes implanted either into normal myocardium or scar tissue on the myocardium. Stimulation through scar tissue requires several times the power necessary for stimulation through normal myocardium, but both are well within the limit of standard, commercially available, transistorized pacemakers. Failure of stimulation of the ventricle was not due to excessive scarring. Failure of stimulation was in every instance due to wire breakage or actual loss of contact between the wire and the myocardium.

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