31

THE ROLE OF ELECTRICAL PACEMAKERS IN THE TREATMENT OF UNEXPECTED CARDIAC ARREST

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DURING THE PAST 8 YEARS cardiac pacemakers have found an important use in the immediate and prolonged treatment of postoperative or spontaneously occurring heart block in which the idioventricular rate has become too slow to provide adequate cardiac output. Scarcely a major hospital in the country lacks one of these electronic devices. For the treatment of Stokes-Adams disease with heart block the role of pacemaker therapy is well established, but in other situations the popularity of pacemakers has sometimes outstripped their value. Ill-conceived efforts at cardiac stimulation can result in deaths either by direct injury or because the effort at pacemaking supplants more effective means of therapy. The commonest of these abuses have occurred in the treatment of patients with cardiac arrest.

Some patients with cardiac arrest have been resuscitated with pacemaker therapy. This group of patients should serve as a guide in determining the limitations and indications for electrical stimulation in cardiac arrest. In virtually all successful cases the possibility of cardiac arrest was recognized in advance, either because of preexisting abnormalities such as heart block or because of capricious behavior of the heart under anesthesia. Because of the advance warning, the pacemaker was available at the patient's side and often the electrodes were in position on the chest. When arrest occurred, stimulation was immediately performed. Pacemaking warranted a trial under these circumstances both because this is the time when stimulation is most apt to succeed and because its use if unsuccessful would not materially delay the institution of some form of cardiac massage.

If pacemaker therapy cannot be immediately applied, it is likely to fail even in the most favorable cases. The capacity for electrically evoked contraction decreases with every second of continued asystole so that in 2 or 3 minutes an unresponsive myocardium develops in a favorable candidate for stimulation. In other cases, the assumption that cardiac arrest is due purely to failure of the conduction system may not be warranted. Almost invariably the arrest is the final result of a bout of anoxia and hypercapnia in which the myocardium has also been damaged. In other instances, fibrillation may have occurred, or a heart beat may be present but so feeble that no blood pressure is obtainable. When cardiac arrest is announced, the patient's chance for prompt massage may be delayed by frantic and time-consuming efforts to procure and apply a pacemaker. When electrical stimulation is finally begun in such patients with unexpected arrest, the myocardium will usually be incapable of contraction.

The primary emphasis in the therapy of unexpected cardiac arrest should still be on cardiac massage, carried out directly or conceivably by the recently described method of external compression. If electrical pacemaking is to be tried, it should be under circumstances which will not significantly delay the application of standard methods of treatment.

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