Editorial

Electrical pacing in heart block complicating acute myocardial infarction

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Complete atrioventricular (AV) block occurs in up to 8 per cent of patients with acute myocardial infarction who are admitted to hospital (Julian, Valentine, and Miller, 1964). Any consideration of the treatment of this important complication must take account of the variations in clinical and electrocardiographic features which depend upon the site of the infarct which caused the block.

In most instances the infarction is inferior (Paulk and Hurst, 1966), following occlusion of the right coronary artery or occasionally of a dominant left circumflex artery (Sutton and Davies, 1968). Block results from inflammation and oedema in the region of the AV node which is situated posteriorly near the crest of the interventricular septum. Complete AV block occurring at this level is typically preceded by prolongation of the PR interval, and later by second degree block of the Wenckebach type (Norris, 1969). When complete block supervenes, lower junctional tissue within the conduction system takes over pacemaking function and in most patients the QRS complexes retain a configuration similar to that of the previously conducted beats. The ventricular rate is usually in the range 40 to 60 beats a minute, and adequate acceleration can be obtained by infusions of isoprenaline if the spontaneous rate is too slow to maintain a satisfactory cardiac output. Asystole is unlikely to occur because junctional pacemakers provide a reliable escape mechanism. The mortality is only slightly higher than that of inferior infarction not complicated by block. The indications for pacing are relative and include such factors as an unusually slow escape rate, abnormal intraventricular conduction indicating an idioventricular rather than a junctional escape rhythm, congestive heart failure, critically low cardiac output, and

recurrent ventricular arrhythmias which do not respond readily to drug therapy. Most patients with inferior infarction and block do not require pacing. In some cases of inferior infarction a junctional pacemaker accelerates to a rate more rapid than that of the sinus mechanism. This type of AV dissociation, in which the ventricles are beating *faster* than the atria, should not be classified as complete AV block (Pick, 1963).

The sequence of events is quite different in cases of anterior infarction. An occlusion in the anterior descending branch of the left coronary artery causes necrosis in the left ventricle and anterior part of the interventricular septum remote from the AV node (Sutton and Davies, 1968). Block occurs only if both bundle-branches are involved, and for this to happen the area of muscle damage must be extensive. Many patients have evidence of previous inferior infarction (Friedberg, Cohen, and Donoso, 1968). Prolongation of the PR interval is not usually seen (Norris, 1969): in the bundle-branches impulses tend either to be conducted without delay or to be blocked completely. However, complete block may be heralded by the onset of right or left bundle-branch block. The sudden development of left axis deviation indicates a conduction defect in the superior division of the left bundle-branch (Samson and Bruce, 1962); right bundle-branch block plus left axis deviation is therefore a particularly important warning sign. If second degree block does occur it is of Mobitz type II rather than Wenckebach type, in which one or more P waves abruptly fail to conduct to the ventricles. After the development of complete heart block, any escape rhythm must be idioventricular. The QRS complexes are therefore wide and of abnormal configuration. Idioventricular pacemakers are usually slow, sometimes irregular, and always unreliable. Adams-Stokes attacks or sudden death may occur at any time as a

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result of asystole or asystole followed by ventricular fibrillation. Isoprenaline is relatively ineffective as a means of increasing heart rate. Pacing is mandatory, but even with the reduced risk of asystole and the improved cardiac output which may accrue, the mortality remains extremely high. This is in part a reflection of the extensive muscle necrosis which is present in these patients, and most of those who are successfully paced die of cardiogenic shock (Lassers and Julian, 1968). Many more patients die before electrical pacing can be established.

If the incidence of asystole is to be reduced in patients with anterior myocardial infarction, those at special risk of developing AV block must be recognized, and a pervenous pacing electrode positioned as a precautionary measure. The prime indication is evidence of impairment of conduction in both bundle-branches, especially the development of right bundle-branch block with left axis deviation. Consideration should also be given to patients who develop left bundle-branch block, for the pattern also frequently heralds complete AV block or asystole (Hunt and Sloman, 1969). An electrode must be introduced without delay if second degree block of Mobitz type II occurs.

Several venous routes have been recommended for the introduction of pacing electrodes, and all have disadvantages. The easiest route is from a medial antecubital vein, but arm movements may subsequently dislodge the tip of the electrode. Binding the arm to the chest wall obviates this difficulty at the cost of inconvenience to the patient. The external jugular route provides a stable electrode position, though entry into the vein and manipulation from this site are slightly more difficult. The percutaneous subclavian route is very satisfactory in skilled hands. When the tip is correctly positioned, preferably in the apex of the right ventricle, only a gentle curve of electrode should be visible in the atrium, for a redundant loop will increase the risk of penetration of the myocardium. The position of the electrode tip is satisfactory only if the threshold for pacing is low, preferably less than 1 volt, and remains stable during deep inspiration. Treatment with isoprenaline should be discontinued during manipulation of the electrode, and a DC defibrillator must be ready for immediate use because ventricular fibrillation is a common complication of the procedure (Paulk and Hurst, 1966).

If screening facilities are not available, a fine Teflon-coated platinum-tipped stainless steel wire electrode (Kimball and Killip, 1965)[1]

[1] Available from Davis and Geck Division, Cyanamid Ltd., Bush House, Aldwych, London W.C.2.

can be introduced through a small catheter extending to the superior vena cava. The electrode can be manoeuvred into the ventricle by monitoring an intracardiac electrogram recorded from its tip; a striking change in the tracing occurs as soon as the tricuspid valve is traversed. It is important, of course, that the electrical characteristics of the electrocardiograph should conform to the recommendations of the Ministry of Health (Hospital Technical Memorandum No. 8, 1963) if intracavity electrograms are to be recorded. Any other piece of electrical equipment connected to the patient should also comply with these recommendations and share a common earth with the electrocardiograph to avoid "earth loops". Fine electrodes can also be used for atrial pacing to increase heart rate in patients with normal conduction who have refractory arrhythmias or bradycardia.

The pacing unit used for a patient with myocardial infarction should always be of the demand or standby type to reduce the risk of ventricular fibrillation as a result of competition between paced beats and spontaneous beats. This is important even in cases of complete block, for in survivors AV conduction almost always recovers, and the threshold for the electrical induction of ventricular fibrillation is reduced (Wiggers, Wégria, and Piñera, 1940). The pacemaker unit is set at approximately twice the output necessary to capture the ventricle, to allow for small changes in stimulating threshold. This should be checked daily and the output reset if necessary. Small increases in threshold are common, but an unnecessarily high output setting increases the risk of pacemaker-induced arrhythmias. Ideally, a ventricular electrogram should be recorded from the pacing electrode before it is secured to ensure that the signal is stable and also strong enough to inhibit the demand pacemaker, because low intracardiac potentials may occur in patients with myocardial infarction (Chatterjee, Sutton, and Davies, 1968). A signal of 2·0 mV is adequate for most units. There is no general agreement on the length of time for which the demand pacemaker should be left in place following recovery of AV conduction. Some patients with anterior infarction die from a recurrence of asystole late in the course of their hospital admission. If pacing has been uncomplicated, the unit should probably be left connected, but inactive at a rate setting below that of the sinus, for at least a week after conduction has returned. This precaution is of course unnecessary in most of the patients with inferior infarction who require pacing for one of the indications discussed above.

The rate at which the ventricular pacemaker is set depends upon the degree of impairment of the circulation and upon the stability of the paced rhythm. Cardiac output is enhanced by ventricular pacing in myocardial infarction with AV block, and within limits will continue to improve as rate is increased (Lassers *et al.*, 1968). On the other hand, myocardial oxygen requirement becomes progressively greater with faster rates. In practice, pacing rates of 60 to 80 beats a minute are usually chosen, but faster rates may be necessary to help counteract shock or to suppress ventricular arrhythmias.

Ventricular pacing has one major disadvantage: the atria and the ventricles continue to beat independently and at different rates. The beneficial effect of atrial contraction therefore occurs only in those beats in which the AV intervals fall by chance within the physiological range. This causes beat-to-beat changes in stroke output; arterial pressure tracings recorded under these conditions reveal a phasic variation in systolic and pulse pressures. The variation suggests that output could be increased by the restoration of normal relationships, possibly improving the prognosis in patients with low output states. We have recently compared the haemodynamic effects of ventricular pacing and sequential atrioventricular pacing in 9 patients with heart block complicating myocardial infarction (Chamberlain *et al.*, 1970). Sequential pacing was achieved by the use of a double electrode system. One electrode was positioned in the right ventricle and the other in the right atrium; both were connected to a coupled pulse generator which could be used either for ventricular pacing or for AV pacing at the same heart rate. The pacing rate was set just above sinus rate in order to maintain atrial capture. The restoration of normal AV relationships resulted in an increase in cardiac output in each case, the average values being 2·9 l./min. during ventricular pacing, and 3·6 l./min. during sequential pacing, an increment of 24 per cent (p < 0·001). Significant rises in systolic arterial pressure (88 mm. to 105 mm. Hg) and falls in venous pressure were also obtained. Despite an improvement in cardiac output, all 4 patients with anterior infarction in our series died, 2 of them in the third hospital week, after they had regained AV conduction.

Sequential pacing is not difficult to initiate and carries no immediate hazard beyond those of simple ventricular pacing, but the technique has two disadvantages in myocardial infarction. The first disadvantage lies in the difficulty in maintaining atrial capture over a long period of time using conventional bipolar electrodes which readily drift away from any position on the atrial wall. This may be overcome by the recent development of special electrodes designed to lodge in the atrial appendage. The second disadvantage is the need to pace slightly faster than sinus rate in patients who may already have a considerable sinus tachycardia. However, sequential pacing is not the only technique that can restore normal AV relationships in heart block. A second method is by synchronous pacing in which the spontaneous atrial depolarization (P wave) is used to trigger a ventricular pacemaker after a preset delay. With this method, ventricular rate is slightly lower when pacing is initiated, and may be expected to fall spontaneously as the sinus rate responds to an improvement in the haemodynamic state. No report has yet appeared on the use of synchronous AV pacing in myocardial infarction with block. In this situation the pulse generator must have the facility to continue demand ventricular pacing at a preset rate if atrial sensing should fail.

Though a reliable estimate of the effect of artificial pacing on the mortality rate in myocardial infarction has never been obtained, there is little doubt that ventricular pacing can improve the prognosis (Scott *et al.*, 1967). A small decrease in the high mortality presently associated with anterior infarction and block may be achieved if ventricular electrodes are routinely placed at the first indication of bilateral branch involvement. Sequential or synchronous atrioventricular pacing may also make a contribution to the management of patients in whom cardiac output is critically reduced.

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