Transvenous high energy shock for ablating atrioventricular conduction in man *Observations on the histological effects*

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SUMMARY Transvenous ablation of atrioventricular conduction by high energy shock through a pacing wire has been used as a method of controlling the ventricular response to rapid atrial arrhythmias. A patient in whom this technique successfully controlled refractory tachycardia was admitted with severe left ventricular impairment five months after the procedure and died. The heart was examined at necropsy. There was no sign of endocardial damage in the region of the atrioventricular node-His bundle. Histological sections failed to show any scarring, inflammation, or infiltration in the region of the atrioventricular node. On the contrary, there was no evidence of any surviving atrioventricular nodal tissue. The mechanism of this effect is not known. The lack of endocardial or myocardial damage further confirms the safety of this new method of controlling tachycardia.

Until recently, ablation of atrioventricular conduction could be achieved reliably and safely only by direct surgery.¹ This approach has been used in patients with refractory supraventricular arrhythmias that are unresponsive to medical treatment. Scheinman *et al*² and Gallagher *et al*³ have described a new simple technique of producing atrioventricular block by transvenous high energy shock. The histological effects of this shock have been studied in dogs and recently reported by Gonzalez *et al*⁴ and Bardy *et al*,⁵ but there are no data available from man. We report a case in which this technique was successful in controlling refractory tachycardia.

Case report

A 55 year old man was referred for investigation and treatment of refractory atrial tachycardia associated with severe left ventricular impairment as a result of several previous myocardial infarctions. Both digoxin and amiodarone had proved ineffective in controlling the tachycardia, which appeared to arise in the left atrium. The rate of tachycardia varied from 160 to 190

beats/min. Since further antiarrhythmic treatment might have jeopardised residual left ventricular function, we decided to perform transvenous ablation of atrioventricular conduction.

The ablation procedure was carried out as described by Gallagher *et al*.³ A pacing lead was positioned in the right ventricle. A standard 7F bipolar (USCI) pacing electrode was used to record His bundle activity and to deliver the shock. One shock of approximately 275 watt seconds was delivered to the region of the His bundle-atrioventricular node and this resulted in immediate atrioventricular block. Further studies were performed one week later before pacemaker implantation. A junctional escape rhythm was present. This showed normal QRS complexes with an axis of -30° , identical to that observed before shock (Fig. 1). After a permanent atrioventricular sequential pacemaker was implanted the patient was discharged. During follow up over five months there was no recurrence of a rapid ventricular rate and complete atrioventricular block persisted. He was admitted with severe left ventricular impairment 5.5 months after the shock and died despite resuscitative efforts. A necropsy was performed.

NECROPSY FINDINGS

The examination was confined to the heart at the request of the relatives. The heart weighed 510 g with

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Fig. 1 Recordings six days after transvenous ablation of atrioventricular conduction. The escape rhythm shows narrow QRS complexes with an HV interval of 50 ms and complete atrioventricular dissociation. HB, His bundle recording; H, His bundle electrogram; A, atrial electrogram; V, ventricular electrogram; CSM, carotid sinus massage, Paper speed = 100 mm/s.

a dilated left ventricle (septum 0.8 cm, posterior wall 1.1 cm, cavity diameter 5.9 cm). There was an aneurysmal anteroseptal healed myocardial infarction containing mural thrombus. Numerous areas of more than 85% stenosis by luminal area were present in all three main coronary arteries. Viewed from the right atrium the area immediately anterior to the coronary sinus and posterior to the insertion of the septal cusp of the tricuspid valve did not show endocardial thickening or scarring. Macroscopically there was no evidence that any form of His bundle ablation had been attempted. The atrial and ventricular conduction system was examined by a standard histological technique,⁶ sections being cut at 150 μ m intervals and stained both with haematoxylin and with trichrome.

Histological examination failed to show any surviving atrioventricular nodal myocardial tissue. The outline of the node (Fig. 2a and 2b) could be identified by its landmarks, and the nodal artery and accompanying venous sinusoids. The fibrous stroma of the node was condensed rather than increased in amount, and the nodal artery was morphologically normal. The endocardium overlying the node was normal, and no inflammatory cells were present. More anteriorly, the penetrating atrioventricular bundle was present and contained the normal numbers of conduction fibres (Fig. 3a and 3b). The bifurcating atrioventricular conduction system was also normal.

Discussion

The mechanism of producing atrioventricular block by transvenous high energy shock is not known. Gonzalez *et al*⁴ have recently reported the histological effects of their earlier experiments in dogs. The studies were performed three months after the initial shock procedure which caused atrioventricular block in all nine dogs. They noted extensive damage to the approaches of the atrioventricular node with variable degrees of fibrosis and fatty infiltration. The atrioventricular node itself was notably fibrotic in eight of the nine dogs. The penetrating bundle of His was similarly affected in all dogs. The bifurcation and the bundle branches were variably damaged with the most extensive lesions being in four dogs who had received more than one shock. These dogs also had fibroelastosis of the adjacent atrial and ventricular myocardium. Least damage was seen in those dogs who had had one shock only. There was no evidence of valvular or papillary muscle damage. Bardy *et al*.⁵ observed discrete fibrous lesions at the base of the septal tricuspid leaflet in all dogs with complete atrioventricular block after shock. In dogs in whom the technique failed to produce sustained atrioventricular block little evidence of damage was noticed. Although these studies provide important information, the results may partly be determined by a species

Fig. 2 (a) Site of the atrioventricular node identified by the nodal artery (arrow), large venous channels (V), the adjacent dense collagen of the central fibrous body (CFB), and endocardium (En) of the right atrium (RA). In the connective tissue surrounding the nodal artery there should be numerous small dark staining myocardial cells. (Trichrome stain $\times 64$). (b) At higher magnification the nodal artery can be seen to be normal without intimal thickening or obliteration of the lumen. In the adjacent connective tissue of the node there are only occasional isolated clumps of surviving myocardial cells (arrows). (Trichrome stain $\times 160$.)

difference both in the application of the technique—for example, catheter position in relation to conduction tissue—and in the tissue response, and they may not reflect what happens in man.

In the present case the morphological features were those of a selective loss of atrioventricular nodal conduction fibres not associated with any evidence of damage to the fibrous stroma, vascular system, or endocardium in the vicinity. As such, the appearances contrast strongly with those found after ablation of the conduction system by burning, cutting, freezing, or formalin injection, where death of all the tissue occurs with resultant inflammatory and fibrosing responses. Furthermore, the appearances were not those usually seen in ischaemic myocardial disease with associated atrioventricular conduction disturbances, when more distal tissue loss is present. Rarely, coronary artery disease does destroy the atrioventricular node but with obliteration of the

nodal artery and fibrous replacement of the entire area. The strongest reason for excluding the atrioventricular nodal destruction as a natural consequence of coronary artery disease lies in the manifest functional normality of the atrioventricular conduction system just before the ablative procedure.

Shocks of the type described in this report are known to generate transient high pressure waves. Tidd *et al*⁷ using a similar technique to disperse bladder calculi made high resolution pressure, cinematographic, and Schlieren recordings during the discharge. They concluded that the main instrument of damage was the pressure-time integral rather than the peak pressure. These results together with the observation that shocks of the order of 200–300 watt seconds may cause disruption of an isolated heart (unpublished observations) led us to speculate that the mechanism of ablation is barotrauma.⁸ Whether or not the atrioventricular nodal cells are selectively

Fig. 3 (a) This section is anterior to that in Fig. 2 by only 1 mm. The penetrating atrioventricular bundle of His (arrows) is encased in the collagen of the central fibrous body and contains numerous closely packed conduction fibres. (Trichrome stain $\times 40$.) (b) At higher magnification the penetrating atrioventricular bundle contains numerous small dark staining conduction fibres cut transversely. (Trichrome stain $\times 160$.)

susceptible to damage in this way is not known. Such an effect of high energy shock may cause atrophy and disappearance of tissue with no signs of burning. It is possible, however, that the "drop out" of nodal cells may merely reflect a non-specific response to trauma. Whatever the mechanism of abolition of atrioventricular conduction it would seem that the technique may be used with minimal damage to the heart provided that the number of shocks is small. As suggested by the canine studies reported by Gonzalez *et al*⁴ a single shock is preferable because this causes minimal damage.

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