Long-term epicardial ventricular pacing from endocardial bipolar pacemaker lead: perforation of right atrial wall

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Lees, D. A. R. and Green, G. D. (1977). Thorax, 32, 370-372. Long-term epicardial ventricular pacing from endocardial bipolar pacemaker lead: perforation of right atrial wall. One of the hazards of endocardial cardiac pacing is that the pacemaker lead may perforate the myocardial wall or interventricular septum, although the incidence of such perforations is believed to be small. This paper describes what is believed to be a unique case in which a pacemaker lead perforated the *atrial* wall at implantation (or possibly shortly afterwards) and yet gave satisfactory right ventricular epicardial pacing for more than five years. The perforation was discovered during a routine postmortem examination but earlier *lateral x-ray* examinations would probably have identified the abnormal position of the electrodes. Moreover, the present implantation technique would not have allowed perforation of the atrial wall at implantation to go undetected.

One of the hazards of endocardial cardiac pacing is that the pacemaker lead might perforate the myocardial wall. Schwedel *et al.* (1960) first reported lead perforation during a period of temporary pacing. Chardack and colleagues (1965) were the first to report such incidents with leads designed for long-term pacing. The incidence of perforations is believed to be small, but reports on perforations continue to appear from time to time (Berman 1975; Grögler *et al.*, 1975; Sorkin *et al.*, 1976; Vera *et al.*, 1976).

This report describes a case in which a pacemaker lead perforated the right *atrial* wall.

Case report (patient No. 170. MB)

A Medtronic endocardial bipolar pacemaker (pulse generator, Type 5842, pacemaker lead, Type 5818) was implanted on 18 December 1970. This patient, then aged 68, had sustained a posterolateral myocardial infarction earlier that year and had subsequently suffered from variable second-degree heart block. The tip of the lead was apparently sited in the apex of the right ventricle. Re-operation took place in July 1974 when a failing generator was replaced. The patient

was readmitted to hospital in April 1976 in congestive cardiac failure and died later that month.

At postmortem examination, on opening the pericardial sac, the pacemaker lead was visible on the posterior surface of the heart in close proximity to the posterior interventricular branch of the right coronary artery. Further dissection revealed the lead enclosed in fibrous adhesions as it passed through the right atrium. However, instead of leaving via the tricuspid valve, the lead was seen to perforate the posterior atrial wall at a point close to, but clearly distinct from, the opening of the coronary sinus (Fig. 1). Thereafter it lay beneath the epicardium, in intimate apposition to the middle cardiac vein, its tip lying near the apex of the right ventricle (Fig. 2).

Histological examination of transverse sections from the posterior aspect of the interventricular septum suggested that the lead had dissected the wall of the middle cardiac vein along the greater part of its length and in this way had come to lie beneath the epicardium. It seems likely that the lead had perforated the posterior atrial wall, passing directly through loose connective tissue until, coming into contact with the middle

Fig. 1 View of opened right atrium. Pacemaker lead is seen superiorly surrounded by adhesions. Site of perforation is also seen, adjacent to which is the opening of the coronary sinus.

Fig. 2 Posterior ventricular surface with epicardium over pacemaker lead opened and posterior interventricular coronary artery displayed.

cardiac vein, it passed by dissection of the vessel wall down the posterior surface of the septum towards the apex of the right ventricle. Dissection of

the vessel wall from its point of entry into the coronary sinus seems unlikely as the coronary sinus ostium was seen clearly separate from the point of perforation of the lead (Fig. 1).

Discussion

The hazards which can arise from perforation of the myocardial wall are haemopericardium, pericardial tamponade, left diaphragmatic twitching, and loss of pacing.

In the case reported here not only were there no complications, but routine investigations at pacemaker clinics failed to suggest that perforation had occurred.

Electrocardiogram limb-lead records show that the paced ECG pattern had not changed from 29 January 1971, that is, from about six weeks after first emplacement (an earlier recording shows the pacemaker being inhibited). Therefore it must be assumed that the atrial wall was perforated on first emplacement, or that during the first six weeks after first emplacement the lead became displaced into the atrium and thereafter perforated the atrial wall. It then took up a stable position, the tip of the lead lying in contact with the epicardial surface of the right ventricular wall.

Conclusions

This apparently unique case of atrial perforation did not prevent satisfactory right ventricular epicardial pacing for over five years. In retrospect, had pacing problems arisen, lateral x-ray

examinations (which are now done routinely) would probably have indicated that the electrodes were outside the heart wall.

Perforation of the right atrial wall during implantation would no longer go un-noticed because with present implantation techniques the tendency is to pass the lead into the right ventricular out-flow tract before final emplacement.

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