Virchows Arch [Pathol Anat] (1985) 407:43–57

Virchows Archiv A

Morphological changes in human myocardium during permanent pacing

P. Châtelain, R. Adamec, and J.N. Cox
Department of Pathology and Medicine, University of Geneva, C.M.U., 1 rue Michel-Servet, CH 1211 Geneva 4, Switzerland

Summary. We have reviewed 74 cases of patients with permanent pacing, using different types of pacing leads in order to determine what morphological changes are produced by this therapy. Macroscopic examination of the heart was performed and slides at the implantation site reviewed when available.

Severe chronic inflammation, scarring and a myocardial response were the prominent findings. From a strictly morphological aspect, the initial trauma at implantation time, the chronic foreign body reaction and myocardial response to chronic trauma, are both much less significant in endovenous than in epimyocardial pacing.

Key words: Heart – Pacemaker – Morphological changes

Introduction

Permanent pacemaker lead implantation has now become a frequent form of therapy in most cardiac centres. The morphological changes at the site of lead implantation and complications encountered with both endovenous and epimyocardial implantation have been well documented in animal models (Cummings et al. 1973; Fishbein et al. 1977; Roy et al. 1969) and to some extent in man (Becker et al. 1972; Contini et al. 1973; Furman and Escher, 1968; Haupt et al. 1963; Lagergren et al. 1966; Parsonnet et al. 1966; Robboy et al. 1969) and as a result there have been several modifications in the type, form and size of the leads, in order to improve their efficacity and reduce complications (Harthorne 1984; Mac Gregor et al. 1983; Robicsek et al. 1981; Roy et al. 1969). Due to recent developments in venipuncture techniques there has been an awakening of the discussion on the merits of epicardial versus the endocardial mode of implantation (Oliver 1983). To the best of our knowledge, there have been no comparative

Offprint requests to: J.N. Cox at the above address

studies of the tissue reactions secondary to endo- or epimyocardial lead insertions, or of other complications. We have therefore reviewed a group of patients wearing a pacemaker coming to post-mortem and have attempted to evaluate the morphological changes at the site of implantation in the different modes of permanent pacing.

Material and methods

Between March 1972 and December 1980 post-mortem was performed on 74 patients who carried a permanent pacemaker. There were 54 males and 20 females. Two patients were 5 and 8 years old, the remainder had ages ranging between 46 and 90 years with an average of 75 years. There were 37 endocardial and 40 epimyocardial implantations (1 or 2 leads). Three patients had both endo- and epicardial electrodes. At post-mortem, the heart and entire pacemaker were removed, X-rayed in some cases, then fixed in 10% formalin after removal of the battery and the lead, leaving the electrode in place. Once properly fixed, the portion of the heart with the implanted electrode was removed for histological study. Several other sections were also taken from the heart including some from the conducting systems. These were embedded in paraffin wax, stained with haematoxylin-eosin, van Gieson-elastin and Congo red when necessary. All sections were examined histologically, but for this study only lesions at the site of pacemaker implantation will be considered.

There were 74 cases carrying 77 leads of which 41 (57.7%) were retained. The histological sections of these were all taken by the same observer (JNC). The epicardial leads were gently unscrewed and the sections taken in the middle along the long axis of the coil; the endocardial leads were also sectioned in the middle along the long axis of the electrode. The two groups, endocardial and epicardial, were further subdivided into 6 subgroups depending on the lapse of time between implantation and the time of death (Table 1 and 2).

Group I: Epimyocardial implantation

Clinical data. Thirty-one sutureless (29 screw-in Medtronic 6917 and 6917A by subxiphoid approach and 2 Cordis) and 9 sutured electrodes were implanted by thoracotomy in this group of patients. The duration of pacing varied between 1 day and 101 months. Current threshold at the time of implantation ranged from 0.4 mA to 4.3 mA and new threshold measurement was performed during battery replacement in 6 cases with a lapse of time between 2 and 7 years (average time: 4.3 years). All but one measurement showed a significant rise in threshold.

7/40 patients (17.5%) leading epicardial leads experienced pacemaker dysfunction secondary to lead breakage (3 cases), rise in threshold of unknown origin (3 cases) or pericardial infection (1 case).

Anatomopathology

Gross anatomy. In this group (40 cases), gross examination of the heart and pericardium disclosed various pathological lesions directly related to lead implantation. Pericardial adhesions were observed in 22 cases (55%), fibrinous pericarditis in 7 cases (17.5%) associated in one case with purulent pericarditis 3 days after implantation; a serosanguinous effusion was present in 4 cases (10%).

Histopathology. Twenty-four specimens were retained for this study. Microscopic examination of the implantation site revealed various lesions depending on the duration of implantation.

Subgroup I (0–7 days): 3 cases. The epicardial lead implantation site showed thick fibrinous deposits around the electrode. Occasional fibroblasts were seen in the fibrin network (Fig. 1). Myocardial fibres within the axis of the coil and around the electrode area were stretched,

Table 1. Epicardial implantation

Duration of implantation		Total number	Documented histologically	Fibrin deposits	Necrosis	Fibrosis	Myocytolysis
I	(0–7 days)	5	3	+ +	+ + +	none	present
II	(8–14 days)	2	2	+ + to + + +	+ + +	none	present
III	(15 days–3 months)	8	4	+ to + +	none	+ + to + + +	present
IV	(3–6 months)	3	1	none	none	+ + +	present
V	(6 months–2 years)	13	9	+ +	+ +	+ + +	present
VI	(more than 2 years)	9	5	none	none	+ + +	absent

Measurements were taken at the level of the tip and about the middle of the coil of the electrodes; the average was taken as the thickness

< 60 µm = +
60–180 µm = + +
> 180 µm = + + +

Table 2. Endocardial implantation

Duration of implantation		Total number	Documented histologically	Fibrin deposits	Necrosis	Fibrosis	Myocytolysis
I	(0–7 days)	1	1	+ +	+ +	none	absent
II	(8–14 days)	0	0	–	–	–	–
III	(15 days–3 months)	4	1	+ + +	none	+ +	absent
IV	(3–6 months)	3	2	none	none	+ +	absent
V	(6 months–2 years)	19	8	none	none	+ + to + + +	present
VI	(more than 2 years)	10	5	none	none	+ +	absent

Measurements were taken at the level of the tip of the electrodes

< 60 µm = +
60–180 µm = + +
> 180 µm = + + +

curved and showed extensive coagulative necrosis or myocytolysis with numerous polymorphonuclear cells (Fig. 2). The epicardium located under the insulating sleeve showed an inflammatory reaction made up of fibrin, haemorrhage and an acute inflammatory reaction. In one case, an epicardial abcess accompanying purulent pericarditis was located under the dacron sleeve (Fig. 3).

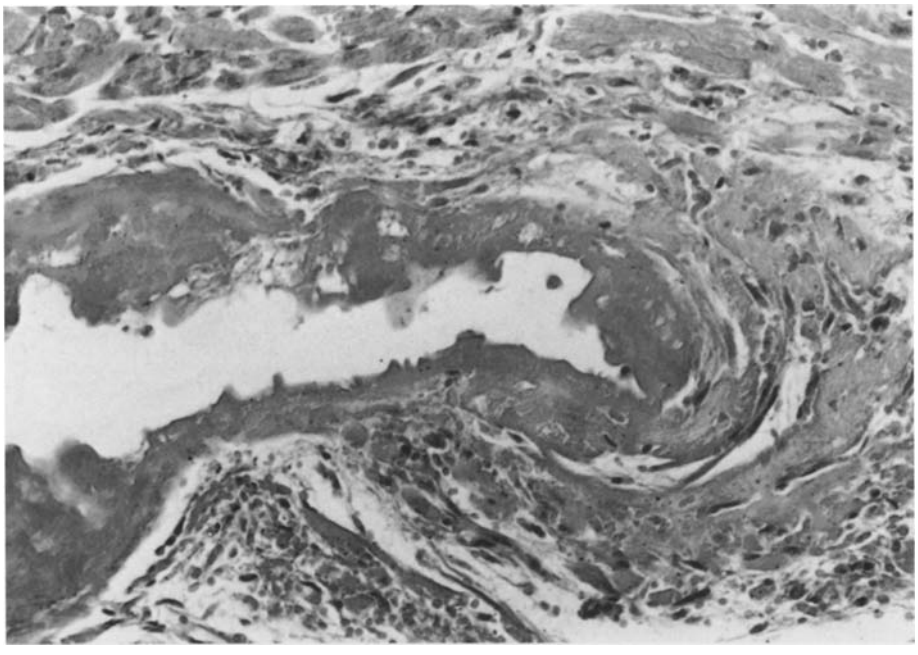

Fig. 1. Marked fibrin deposit around a sutureless electrode lead 3 days after implantation (VGE1, 64 ×)

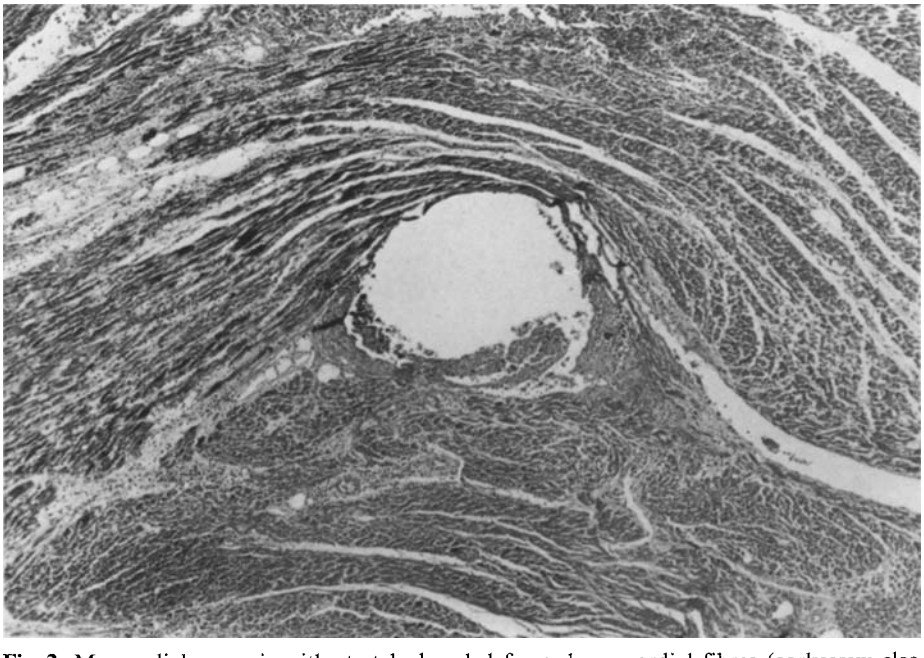

Fig. 2. Myocardial necrosis with stretched and deformed myocardial fibres (corkscrew electrode) 3 days after implantation (HE, 10 ×)

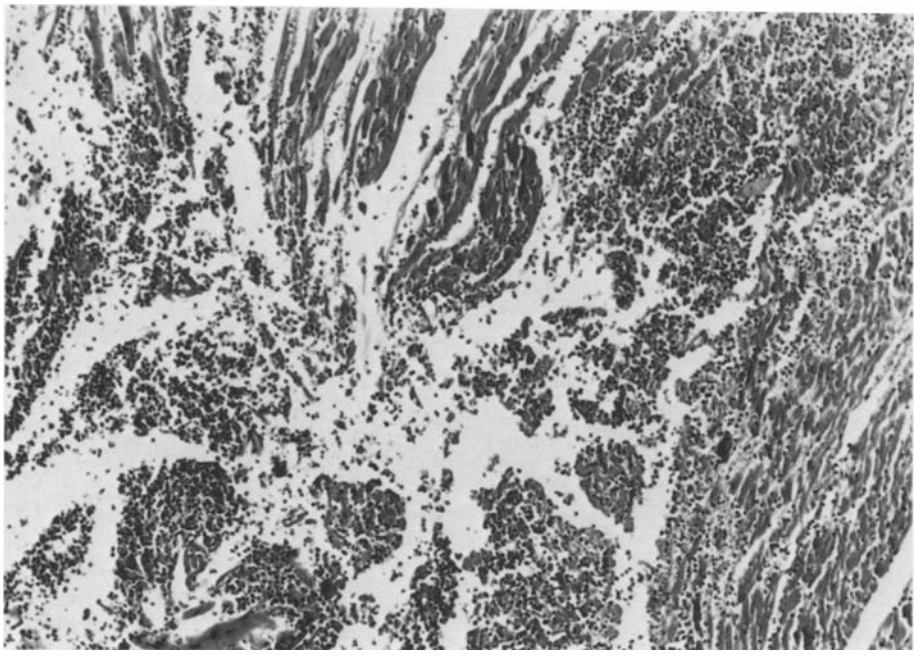

Fig. 3. Myo-epicardial abscess 3 days after implantation of epicardial electrode (HE, 25 ×)

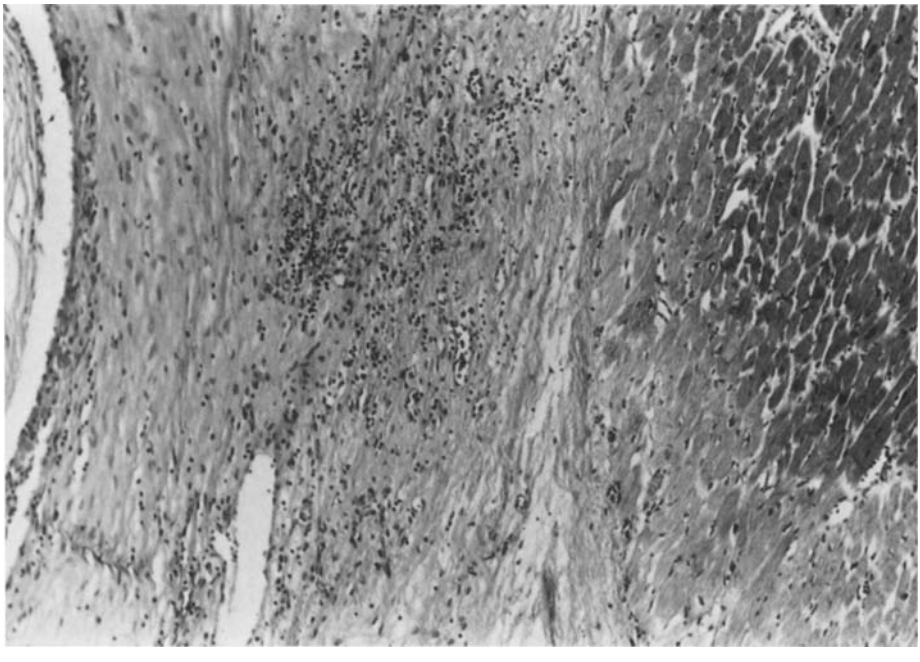

Fig. 4. Coagulative necrosis and myocytolysis in the vicinity of epicardial lead implantation – 12 days (HE, 25 ×)

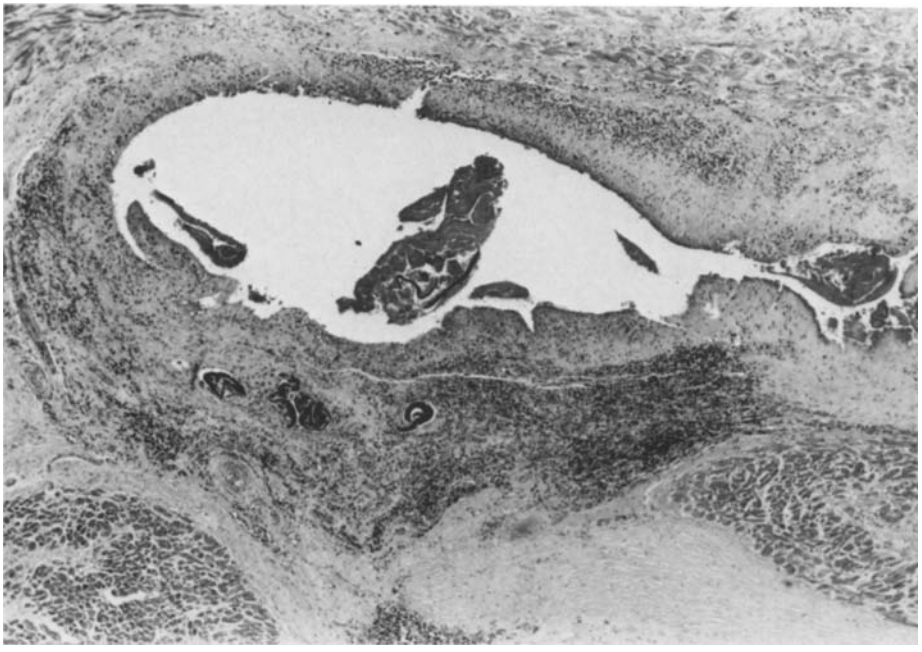

Fig. 5. Silicone chips free or incorporated in the surrounding granulation tissue of epicardial lead (VGE1, 4×)

Subgroup II (8–14 days): 2 cases. The fibrin deposits were still present around the electrode. The myocardium and the epicardium close to the implantation site were replaced by granulation tissue with numerous fibroblasts, fibrocytes and vessels. Chronic inflammatory cells were numerous. Myocytolysis and, surprisingly coagulative necrosis were still present in the surrounding myocardium (Fig. 4).

Subgroup III (15 days to 3 months): 4 cases. A thin fibrin layer which was more or less organized was still present around the electrode. No coagulative necrosis was seen in the myocardium, however, myocytolysis was a common feature. In one case, chips of silicone (broken insulating material) were incorporated in the granulation tissue with giant cell formation (Fig. 5). The dacron net had induced a foreign body type granuloma in the epicardium.

Subgroup IV (3 to 6 months). Only one case was available for study. The implantation site showed myocardial sclerosis and pericardial fibrosis with giant cell granuloma. No fibrin was visible around the electrode in this case.

Subgroup V (6 months to 2 years): 9 cases. There was persistance of granulation tissue around the electrode coil (Fig. 6) and in 2 cases foreign body granulomas induced by broken insulating material. In 2 other cases, there were fibrinoid deposits in the myocardium made up of fresh and partially organized fibrin containing few fibroblasts and new vessels (Fig. 7). Surprisingly, even at this late stage myocardial fibres in and about the granulation tissue showed coagulative necrosis. Myocytolysis was a frequent finding in the myocardium in the vicinity of the scar tissue (Fig. 8). Amyloid deposits were observed in the myocardium in one case.

Subgroup VI (beyond 2 years): 5 cases. A chronic inflammatory reaction was still present at the implantation site with only a moderate lymphoplasmacytic infiltrate. No fibrinoid material nor myocardial necrosis was observed close to the implantation site. Diffuse cardiac amyloid-

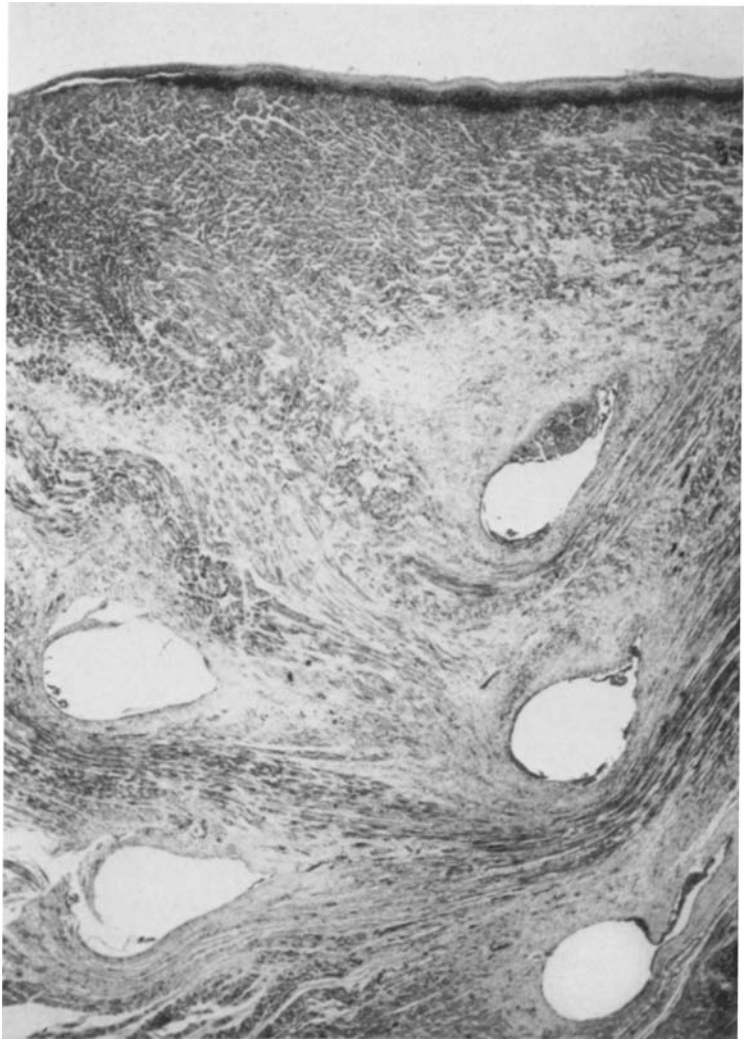

Fig. 6. Diffuse fibrosis around the site of implantation of the epicardial lead with fibrin deposits around the electrode – 18 months (HE, 3 ×)

osis was seen in three cases (whose ages were 67, 75 and 82) and in one of these was some broken insulating material around the coil with a foreign body giant cell reaction. In the pericardium there was a marked fibrous reaction especially around the rare sutured coil (1) without myocardial involvement whereas the sutureless electrodes (4) were surrounded by a variably cellular intramyocardial scar tissue.

Group II: Endocardial implantation

Clinical data. In this group, pacemakers were carried by patients from 3 days to 115 months. At the time of implantation current threshold varied between 0.4 mA and 4.8 mA. Nine thresholds have been recalculated during battery replacement 5 months to 6 years after implantation

Fig. 7. Fibrinoid material around epicardial electrode 21 months after implantation (HE, 10×)

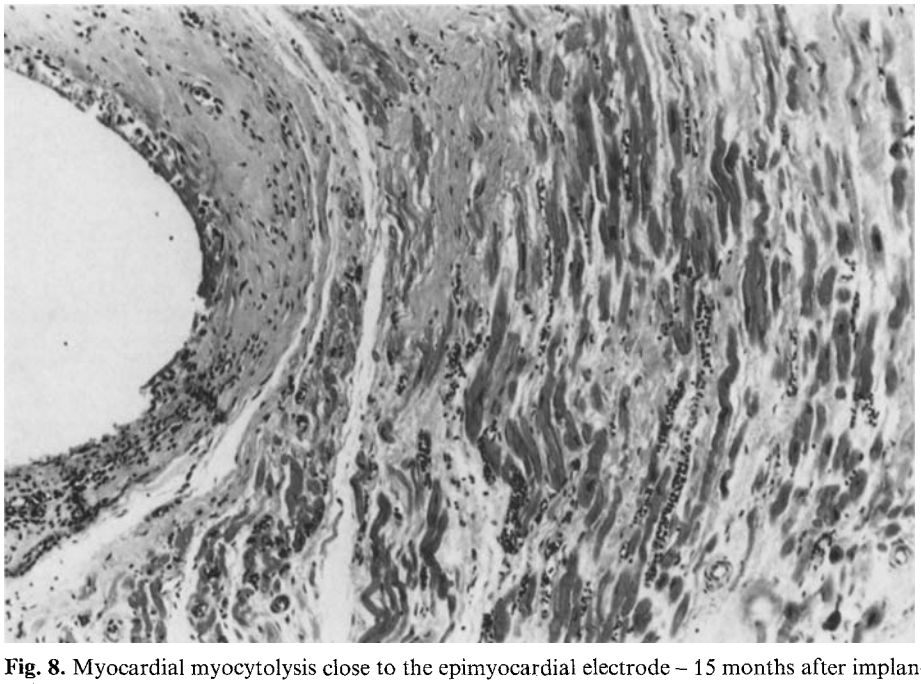

Fig. 8. Myocardial myocytolysis close to the epimyocardial electrode – 15 months after implantation (HE, 25×)

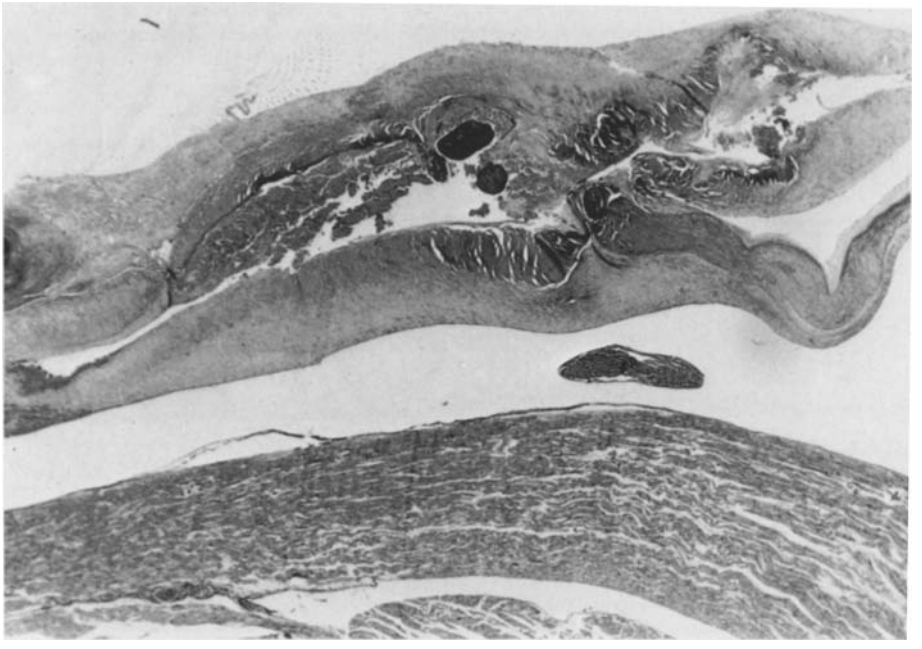

Fig. 9. Tricuspid leaflet perforation by endovenous lead (HE, 4×)

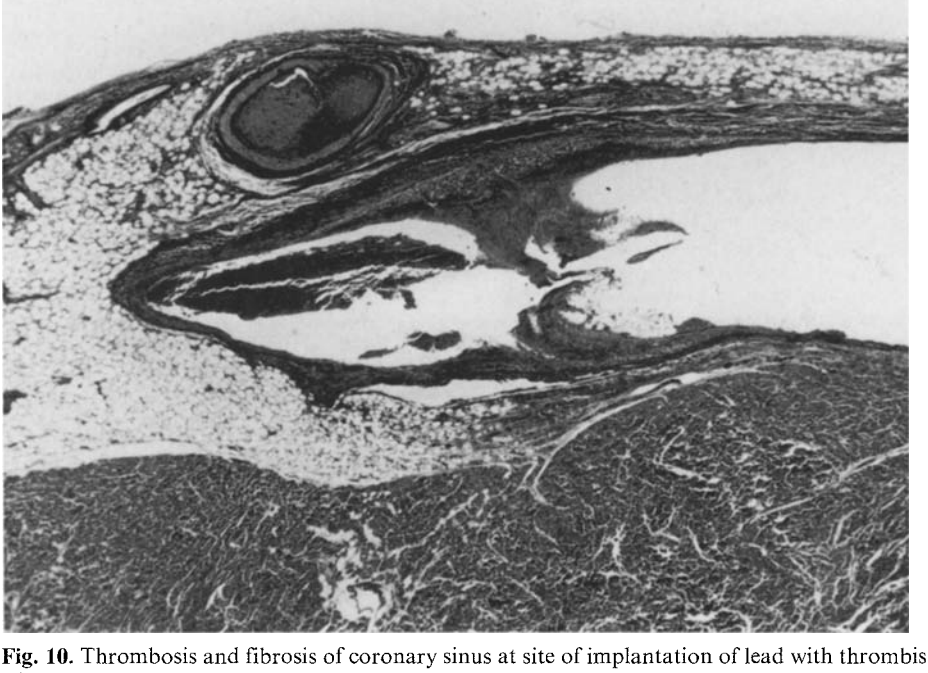

Fig. 10. Thrombosis and fibrosis of coronary sinus at site of implantation of lead with thrombis of tributary (VGE1, 4×)

(average time: 3 years). All but one value showed a rise. Clinical data revealed pacemaker dysfunction secondary to lead displacement (1 case) or malposition in the coronary sinus.

Anatomopathology

Gross anatomy. There were 37 patients in this group. At post-mortem, gross pathological findings secondary to lead implantation were tricuspid leaflet perforation (Fig. 9) in 5 cases (13.5%) with fixation in 3 cases (8.1%) without appearant haemodynamic consequences as far as could be determined from the available clinical data. Leads in the coronary sinus (Fig. 10) were found in 2 cases (5.4%) and this was associated with pacemaker dysfunction in one case (see above). In the other case, current threshold had been recalculated 3 years after implantation and was 5.8 mA (threshold at implantation: 1.0 mA). In all cases there were patchy fibrin thrombi along the leads which were sometimes adherent to the atrial wall.

Histology. Seventeen cases were retained for this study, the remaining 20 although showing similar histological lesions in regard to duration were insufficiently documented.

Subgroup I (0–7 days): 1 case. There was a fibrinoid deposit surrounding the lead with some degree of organisation.

Subgroup II (8–14 days): there was no case in this subgroup.

Subgroup III (15 days to 3 months): 1 case. A fibrin layer was still present around the electrode which was inserted in the trabeculae. Neither necrosis nor granulation tissue was seen in the adjacent myocardium.

Subgroup IV (3 to 6 months): 2 cases. In one case the electrode was located in the coronary sinus and was surrounded by granulation tissue. Thrombosis was not observed. In the second case, a discreet fibrous layer surrounded the electrode which was close to a parietal thrombus secondary to myocardial infarction of the right ventricle. There was no fibrinous material in either case.

Subgroup V (6 months to 2 years): 8 cases. Myocardial reaction around the electrode was moderate and was composed chiefly of fibrous material with some monoclear cells. Fresh fibrin associated with myocytolysis was seen in two cases and in two others foreign body granuloma secondary to broken insulating material was present.

Subgroup VI (beyond 2 years): 5 cases. The leads and electrodes were embedded in a narrow sparsely cellular fibrous tissue connecting few trabeculae. Fibrin and myocardial necrosis were invariably absent and there was no inflammatory reaction in the vicinity of the electrodes.

Discussion

Both endo- and epimyocardial modes of implantation induced a local tissue reaction which resulted in the formation of scar tissue around the electrode.

In the period immediately following implantation there was an eosinophilic fibrinoid layer around the electrode as described by several authors, either in experimental animals (Akyurekli et al. 1979; Fishbein et al. 1977; Mac Carter et al. 1979) or in man (Becker et al. 1972; Contini et al. 1973; Robboy et al. 1969). This material was gradually invested and incorporated by fibroblasts and myofibroblasts, becoming a partly hyalinised mantle by the sixth month around the endocardial lead, fixing it in place as suggested by some authors (Contini et al. 1973; Haupt et al. 1963; Lagergren et al. 1966). In the epimyocardial electrode the fibrinous coat was observed, in

some cases, for as long as 2 years after implantation. This fibrin deposit may be the expression of lead instability in endovenous pacing (Robicsek et al. 1981) or continuous trauma as in epimyocardial pacing (Akyurekli et al. 1979; Furman and Escher 1968).

In the hours following implantation there was coagulative necrosis of the myocardial tissue around the electrode followed by an inflammatory response rich in polymorphonuclear leucocytes within the first 12 h of implantation. This was present not only in epicardial leads as we have observed but also with endocardial leads as described by others (Althoff, 1978; Furman and Escher 1968; Lagergren et al. 1966; Robboy et al. 1969).

With time granulation tissue was formed, containing numerous macrophages with haemosiderin pigment. These macrophages were more numerous around the epicardial leads where they were accompanied by lipofuchsin granules among the collagen bundles and fibroblasts. In addition, myocytolysis was present in and about the scar tissue. Myofibrosclerosis was a persistent feature about the epicardial leads at all times. These lesions are partially responsible for the scar tissue formation as observed already in the subgroup III and subsequent subgroups. This reaction was less important in the cases with endocardial leads. The explanation for this difference may be found in the nature of the initial trauma. The whirling, with stretching and necrosis of the myocardial fibres within the axis and about the cockscrew epicardial lead would strongly suggest an initially traumatic origin (Akyurekli et al. 1979; Cummings et al. 1973; Furman et al. 1968). The endocardial self-anchoring leads appear to be less traumatic but probably less stable, as suggested by Robicsek et al. (1981).

Colliquative myocytolysis is an unusual form of cardiac cell necrosis present in myocardial infarction (Cantin and Leone 1981; Edwards et al. 1984). It may be observed more than 7 years after the initial insult in case of persistent angina or cardiac failure. Its significance and mechanism remain unclear but it is probably related to chronic hypoxia. When associated with fibrous tissue reaction close to the pacemaker electrode many months after implantation, it might be taken to be an indicator of persistent local trauma or ischaemia which may contribute to chronic inflammation and scar tissue extension. The concept that electrical stimulation is responsible for the scar tissue formation around pacemaker electrodes and for the persistent fibrin deposit and myocytolysis, has now been abandoned (Akyurekli et al. 1979). We did observe these changes, although to a lesser degree, around the three epicardial leads which were not submitted to electrical stimulation and therefore other explanations must be considered. Apart from the lesions observed around the electrodes, the other lesions and complications in the two modes are not comparable. We were unable to find in the literature any morphological description of lesions which could explain pericardial rub, a clinical complication of transvenous pacing, described by Hynes et al. (1983) nor did we find anything relevant in our material. Purulent pericarditis, although a rare complication of epicardial mode implantation (Firor et al. 1978; Tegtmeyer et al. 1974) was observed in one case (1/40) and this, associated with acute endocartditis and secondary sepsis

resulted in a fatal outcome in this patient, a sequence of events observed by other authors also in cases of transvenous pacing (Furman 1966; Lyons 1982; Schwartz and Pervez 1971). Pericardial adhesions, however, are not an uncommon finding and were observed in 55% of cases with sutureless or sutured electrodes in this series. They are considered to be the consequence of pericardial section and epicardial trauma associated with local haemorrhage as generally observed after surgery (Weibel and Majno 1973). Endocardial implantation may be the cause of other complications which are not encountered with epimyocardial implantation (Austin et al. 1982; Hynes et al. 1983; Ong et al. 1981; Parsonnet et al. 1966; Parsonnet 1983; Robboy et al. 1969). Cardiac perforation has been observed in some studies (Austin et al. 1982; Hynes et al. 1983; Lyons, 1982) but was not seen in our material, although in one case the tip of the electrode had penetrated into the epicardial fatty tissue. Cardiac perforation has been reported to be the cause of haemopericardium with cardiac tamponade especially in the early period after pacemaker insertion (Kalloor 1974).

Cases of electrode dislodgment have been documented in the literature (Austin et al. 1981; Charles et al. 1977; Furman et al. 1983; Parsonnet et al. 1979) but this complication was observed clinically only in one case in this series, but this patient died from other causes. In some cases of dislodgment the electrode may make a loop into the right ventricle outflow tract or may float freely in the right ventricle or right atrium (Lagergren et al. 1966; Parsonnet et al. 1979; Robicsek et al. 1981). Dislodgment of an epicardial lead may occur but was not observed in this group.

Fibrin deposits along the electrical lead were observed within the first 24 h after pacemaker insertion and with time formed a fibro-hyaline mantle about the electrical lead, either in focal areas or almost throughout its entire length as described by other authors (Lagergren et al. 1966; Robboy et al. 1969; Becker et al. 1972). In 7 cases the electrical lead was fixed by thrombus either to the right atrial wall (posterior or septal) or to the superior vena cava or both, but there were no cases with extensive lesions which could have caused occlusion of these structures and lead eventually to swelling of the arms (Mitrovic et al. 1982; Pauletti et al. 1983) or the more severe complication of the superior vena cava syndrome (Gundersen et al. 1982; Krug and Zerbe 1980). From these thrombi, pulmonary thromboembolism may ensue (Kinney et al. 1979) as has been observed in a number of our cases which presented with repeated distal thromboembolism. Tricuspid leaflet perforation (5/37 or 13.5%) mainly of the septal and posterior leaflet, with complete fixation of the valve to the fibrinous covering of the lead in 3 cases, did not provoke clinical haemodynamic disturbances nor apparent malfunction of the valve and this correlates well with the findings of other authors (Gould et al. 1974; Lagergren et al. 1966; Petterson et al. 1973; Robboy et al. 1969).

Malposition into the coronary sinus was observed in two cases one of which presented with pacemaker dysfunction while the other had an increase in current threshold as described in the literature (Meyer and Miller 1969; Spitzberg et al. 1969). One electrode was accompanied by extensive throm-

bosis of the vessel and its tributaries while the second showed secondary scarring and thickening of the vessel wall.

In the cases (endo- and epicardial) documented radiographically there were no lead fractures. This complication is well known to be the cause of pacing disturbances especially with epicardial leads, and may be the result of failure, especially in children (Fleming et al. 1983; Parsonnet et al. 1973).

Fragmentation of insulating material from around the electrode with formation of a foreign body type granulation tissue was observed in 4 cases wearing an epicardial pacemaker. Fragments were also observed in two cases wearing endocardial pacemaker but with little tissue reaction. This complication is known to be associated with a high current threshold (Doenecke et al. 1975; Fisher et al. 1976) as was observed in these patients. In one of our cases the tip of the epicardial electrode (6917 Medtronic) was free within the right ventricular cavity without damage to the insulating material.

Our morphological studies would indicate that the tissue reaction secondary to electrode implantation is significantly different in the endo- and epimyocardial mode of pacing. The acute and chronic local morphological changes taking place after lead insertion are less important, qualitatively and quantitatively, in the endocardial mode. These studies show that the histological modifications at the site of implantation are more severe and widespread with the epimyocardial mode and that the lesions continue to progress for years after implantation. This would suggest that the endocardial mode of pacing which results in an early stabilized local reaction, may be the method of choice.

Acknowledgements. We thank Professor C.L. Berry for his criticisms and suggestions, Miss Claire-Lise Seignemartin for typing the manuscript, Miss Joanne Stalder for technical assistance and Messers Jean-Claude Rumbeli and Etienne Denkinger for the photographs.

References

Akyurekli Y, Taichman GC, White DL, Keon WJ (1979) Myocardial responses to sutureless epicardial lead pacing. In: Proceedings of the VIth World Symposium on Cardiac Pacing. Pacesymp. Montreal, chap. 33, 3

Althoff H (1978) – Gewebereaktionen auf implantierte Herzschrittmacher. Med. Klin **73**, 1468–1476

Austin JL, Preis LK, Crampton RS, Beller GA, Martin RP (1982) Analysis of pacemaker malfunction and complications of temporary pacing in the coronary care unit. Am J Cardiol 49:301–306

Becker AE, Becker MJ, Claudon DG, Edwards JE (1972) Surface thrombosis and fibrous encapsulation in intravenous pacemaker catheter electrode. Circulation 46:409–412

Cantin M, Leone A (1981) Morphology of myocardial infarction. Meth Achiev Exp Pathol 10:244–284

Charles RG, Clarke LM, Drysdale M, Sequeira RF (1977) Endocardial pacing electrodes design and rate of displacement. Br Heart J 39:515–516

Contini C, Papi L, Pesola A, l'Abbate A, Magini G, d'Angelo T, Cinacchi P, Maseri A, Donato L (1973) Tissue reaction to intracavitary electrodes: Effect on duration and efficiency of unipolar pacing in patients with A-V block. J Cardiovasc Surg 14:282–290

Cummings JR, Gelok R, Grace JL, Salkind AJ (1973) Long-term evaluation in large dogs

and sheep of a series of new fixed-rate and ventricular synchronous pacemakers. J Thorac Cardiovasc Surg 66:645–652
Doenecke P, Flöthner R, Rettig G, Bette L (1975) Studies of short- and long-term threshold changes. In: Schaldach M, Furman S (eds) Engineering in Medicine I. Advances in pacemaker technology. Springer, Berlin, Heidelberg, pp 283–296
Edwards GM, Said JW, Block MI, Herscher LL, Siegel RJ, Fishbein MC (1984) Myocytolysis (vacuolar degeneration) of myocardium: Immunohistochemical evidence of viability. Human Pathol 15:753–756
Firor WB, Lopez JF, Manson EM, Mori M (1968) Clinical management of infected pacemaker. Ann Thorac Surg 6:431–436
Fishbein MC, Tan KS, Beazell JW, Schulman JH, Hirose FM, Criley JM (1977) Cardiac pathology of transvenous pacemakers in dogs. Am Heart J 93:73–81
Fisher JD, Furman S, Parker B, Escher DJW (1976) Pacemaker failures characterized by continuous direct current leakage. Am J Cardiol 37:1019–1023
Fleming WH, Sarafian LB, Yarbrough JW, Kugler JD, Mooring PK (1983) Epicardial corkscrew lead fracture: an underreported cause of pacing failure? Ann Thorac Surg 35:535–537
Furman S (1966) Complications of pacemaker therapy for heart block. Am J Cardiol 17:439–442
Furman S, Escher DJW (1968) Retained endocardial pacemaker electrodes. J Thorac Cardiovasc Surg 55:737–740
Furman S, Parker B, Escher DJW, Solomon N (1968) Endocardial threshold of cardiac response as a function of electrode surface area. J Surg Res 8:161–166
Furman S, Pannizzo F, Campo I (1983) Rates and modes of failure for eight lead types in 703 implants: a four-year structured study. In: Proceedings of the VIIth World Symposium on Cardiac Pacing, Vienna. Steinkopff Verlag, Darmstadt, pp 417–422
Gould L, Ramana Reddy CV, Yacob U, Teich M, de Martino A, de Palma D, Gomprecht RF (1974) Perforation of the tricuspid valve by a transvenous pacemaker. J.A.M.A. 230:86–87
Gundersen T, Abrahamsen AM, Jorgensen I (1982) Thrombosis of superior vena cava as a complication of transvenous pacemaker treatment. Acta Med Scand 212:85–88
Harthorne JW (1984) Pacemaker leads. Intern J Cardiol 6:423–429
Haupt GJ, Myers RN, Perrill CV, Birkhead NC (1963) Human tissue response to long term implanted cardiac pacemaker electrode system. Surg Gynecol Obst 117:484–488
Hynes JK, Holmes DR, Harrison CE (1983) Five-year experience with temporary pacemaker therapy in the coronary care unit. Mayo Clin Proc 58:122–126
Kalloor GJ (1974) Cardiac tamponade: report after insertion of a transvenous endocardial electrode. Am Heart J 88:88–89
Kinney EL, Allen RP, Weidner WA, Pierce WS, Leaman DM, Zelis RF (1979) Recurrent pulmonary emboli secondary to right atrial thrombus around a permanent pacing catheter: a case report and review of the literature. Pace 2:196–202
Krug H, Zerbe F (1980) Major venous thrombosis: a complication of transvenous pacemaker electrodes. Br Heart J 44:158–161
Lagergren H, Dahlgren S, Nordenstam H (1966) Cardiovascular tissue response to intracardiac pacemaking. Acta Chir Scand 132:696–704
Lyons C (1982) Sepsis and pacemaker malfunction. Arch Intern Med 142:1931
Mac Carter DJ, Jenewein CG, Schyma DH (1979) Spontaneous subacute reduction in myocardial stimulation thresholds. In: Proceedings of the VIth World Symposium on Cardiac Pacing. Pacesymp. Montreal, chap. 4–6
Mac Gregor DC, Wilson GJ, Klement P, Pilliar RM (1983) Improved electrophysiological performance using porous-surfaced electrodes for atrial epicardial pacing. In: Proceedings of the VIIth World Symposium on Cardiac Pacing, Vienna. Steinkopff Verlag, Darmstadt, pp 345–352
Meyer JA, Miller K (1969) Malplacement of pacemaker catheters in the coronary sinus. J Thorac Cardiovasc Surg 57:511–518
Mitrovic V, Thormann J, Schlepper M, Neuss H (1983) Thrombotic complications with pacemakers. Internat J Cardiol 2:363–374

Ong LS, Barold SS, Craver WL, Falkoff MD, Heinle RA (1981) Partial avulsion of the tricuspid valve by tined pacing electrode. Am Heart J 102:798–799
Olivier AF (1983) Transvenous pacemaker implantation. Ann Thorac Surg 35:115
Parsonnet V, Zucker IR, Kaanerstein ML, Gilbert L, Alvares JF (1966) The fate of permanent intracardiac electrodes. J Surg Res 6:285–292
Parsonnet V, Gilbert L, Zucker IR (1973) The natural history of pacemaker wires. J Thorac Cardiovasc Surg 65:315–327
Parsonnet V, Bilitch M, Furman S, Fisher JD, Escher DJW, Myers G, Cassady E (1979) Early malfunction of transvenous pacemaker electrodes. A three-center study. Circulation 60:590–596
Parsonnet V (1983) Techniques for permanent transvenous pacemaker implantation: personal preferences. In: Proceedings of the VIIth World Symposium on Cardiac Pacing, Vienna. Steinkopff Verlag, Darmstadt, pp 441–447
Pauletti M, di Ricco G, Contini C (1983) Venous obstruction from temporary pacing through the subclavian vein. Intern J Cardiol 4:189–192
Petterson SR, Small JB, Reeves G, Kocot SL (1973) Tricuspid valve perforation by endocardial pacing electrode. Chest 63:125–126
Robboy SJ, Harthorne JW, Leinbach RC, Sanders CA, Austin WG (1969) Autopsy findings with permanent pervenous pacemakers. Circulation 39:495–501
Robicsek F, Tarjan P, Harbold NB, Masters TN, Robicsek SA (1981) Self-anchoring endocardial pacemaker leads: current spectrum of types, advances in design, and clinical results. Am Heart J 102:775–782
Roy OZ, Heffweit HA, Waddell W (1969) Canine myocardial thresholds and tissue responses to chronic pacing. Med Biol Eng 7:501–506
Schwartz IS, Pervez N (1971) Bacterial endocarditis associated with a permanent transvenous cardiac pacemaker. J.A.M.A. 218:736–737
Spitzberg JW, Milstoc M, Wertheim AR (1969) An unusual site of ventricular pacing during the use of a transvenous catheter pacemaker. Am Heart J 77:529–533
Tegtmeyer CJ, Hunter JG, Keates TE (1974) Bronchocutaneous fistula as late complication of permanent epicardial pacing. Amer J Roentgenol Radium Ther Nucl Med 121:614–616
Weibel MA, Majno G (1973) Peritoneal adhesions and their relation to abdominal surgery. Am J Surg 126:345–353

Accepted March 21, 1985