Functionless retained pacing leads in the cardiovascular system

A complication of pacemaker treatment

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SUMMARY Twenty one patients with retained endocardial pacemaker leads were followed during a total observation period of 1097 months to assess the incidence of complications. Two patients developed thrombosis and occlusion of the superior vena cava, which was relieved by the development of a collateral venous circulation. In one patient the broken tip of the lead migrated to a pulmonary artery but did not cause overt complications. The remaining patients were free of symptoms. One patient died for reasons unconnected with pacemaker treatment.

The good toleration of retained pacemaker leads by most patients indicates that major surgical procedures to remove the lead should be reserved for patients with life threatening complications, such as persistent infection or dangerous migration of the lead or both.

Complications of pacemaker treatment such as infection, lead fracture, insulation break, threshold increase (especially with non-programmable pacemakers), and electrode displacement may need corrective surgery to insert a new pacing lead. Removal of primary, now functionless and often defective, leads cannot always be achieved. As a result, there are two or sometimes even more leads in the cardiovascular system, which increases the risk of thrombus formation, infection, or migration of the lead. To assess this risk we studied patients with retained pacing leads.

Patients, methods, and results

The clinical records of permanent pacemaker implantations from January 1972 to January 1982 were reviewed. In 21 patients (17 men, four women; mean age 64.9 (range 51-85) years) implantation of a new pacing lead was necessary in 23 instances because of various forms of malfunction (Table). In all 23 instances the removal of the functionless lead was

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Table *Reasons for implantation of a new pacing lead (23 implantations in 21 patients)*

	No of patients
Pacemaker pocket infection	9
Electrode displacement	5
Mechanical penetration	2
Threshold increase	2
Lead fracture	1
Insulation break	1
Unspecified	3

impossible. In 18 instances the lead remained fixed to the right ventricular wall; in five instances it migrated into the right atrium (three cases), the right internal jugular vein (one case), and one of the proximal branches of the right pulmonary artery (one case).

CASE HISTORIES

Case 1—A 63 year old man underwent insertion of the Elema-Schönander EMT 588 lead in January 1972 for complete atrioventricular block. The pacing lead became dislodged on the tenth day after insertion. Repositioning was not achieved, and a new EMT lead was introduced. A third lead was inserted in August 1982 because of extrusion of the box through the skin. Both functionless electrodes remained in the cardiovascular system despite attempts to remove them;

Fig 1 Case 1: chest radiograph showing three pacing leads in the cardiovascular system; one electrode is visible in the right internal jugular vein and two in the right ventricle.

the first lead migrated into the right internal jugular vein (Fig 1).

Case 2—An EMT 588 lead was inserted in August 1972 in a 60 year old man because of complete atrioventricular block. The lead became dislodged two months later; repositioning and removal were impossible, and a second EMT 588 lead was implanted. In May 1983 a third lead was introduced because of late infection of the pacemaker pocket three months after replacement (Fig. 2).

Case 3—A 54 year old man underwent implantation of the LEM lead for intermittent second degree atrioventricular block in March 1976. Pacing ceased in September 1982 because of a threshold increase. During attempted withdrawal the lead broke leaving the tip in the heart. Subsequently, the electrode migrated into one of the proximal branches of the

Fig 2 Case 2: chest radiograph showing three pacing leads in the cardiovascular system; the electrodes are visible in the right ventricle.

right pulmonary artery without producing any clinical symptoms and signs either immediately after the event or later during 21 months follow up (Fig. 3).

FOLLOW UP AND OUTCOME

All the patients were followed up to the end of June 1984. The total observation period for patients with two pacing leads was 1097 (range 16–127) months. Mean observation time was 52.2 (median 37) months. Two patients with three leads were followed for 11 and 22 months after the insertion of a third lead.

In two patients, previously reported major venous thrombosis occurred 11 and 24 months after insertion of a new pacing lead.¹ In both cases symptoms and signs of thrombosis and occlusion of the superior vena cava were relieved by the development of collateral

Fig 3 Case 3: chest radiograph showing a migrated electrode (arrow) in the intermediary part of the right pulmonary artery.

venous channels. One patient died 58 months after the insertion of a second lead as a result of gastrointestinal haemorrhage. Two patients were lost to follow up after 16 and 23 months of observation. At the final examination in June 1984 all the remaining patients were free of symptoms and had no signs of thrombosis or infection. Chest x ray films excluded the possibility of serious migration of the pacing leads in all patients.

Discussion

Insertion of a pacemaker lead may predispose to thrombosis or occlusion in axillary, subclavian, or innominate veins, or in the superior vena cava.^{2,3} The venographically confirmed incidence of venous thrombosis in patients with an inserted pacing lead is high and may vary from 28.5%⁴ to 78.5%.⁵ Nevertheless, in most instances the effective circulation is well preserved because of the development of collaterals; thus, the vena cava syndrome or oedema of the arms occurs rarely.⁶⁻⁸

Only one case of overt thrombosis occurred out of a total number of 1201 patients reported by Lagergren *et al*,⁹ Furman *et al*,¹⁰ and Kalmar *et al*.¹¹ The incidence of clinically overt venous thrombosis does not seem to be high, in absolute terms, even in patients with two pacing leads. It was not noted in probably the largest reported series of Rettig *et al*,¹² who fol-

lowed 46 cases for a mean of 21.4 months. On the other hand, as many as half the overall number of thrombotic complications due to pacemaker treatment relate to patients with two pacing leads.¹³ The incidence of subclinical venous thrombosis is also significantly higher in patients with two leads than in those with one.¹⁴ In our experience the successful removal of a functionless lead does not increase the incidence of thrombotic complications. Such a findings has not been reported. Symptoms and signs of venous thrombosis may be encountered as early as 23 days and as late as seven years after the insertion of a pacing lead¹³; more often, they tend to appear after a latent period of approximately 12 months.¹⁵

The possibility of sepsis culminating in endocarditis is a serious risk in patients with retained leads after surgical treatment for infective complications. In Rettig *et al*'s series there were four deaths due to septic complications, and infection persisted in two cases out of 21 with probably infected leads.¹² There were no infective complications in our series either because the numbers were small or because we adopted the policy of early surgical intervention in case of infection. The risk of fatal infection perhaps justifies attempts to remove infected leads in selected cases by traction.^{11,16,17} Such removal may avoid cardiectomy, which may, however, be necessary in cases of life threatening infection.^{12,17} Traction, especially when performed too forcibly, is a hazardous procedure which may cause severe arrhythmias and damage to the heart structures.^{12,17,18} It should therefore be performed under close clinical and electrocardiographic surveillance.

Removal of an implanted pacing lead is often impossible because of the development of a fibrous sheath around the lead which extends along it as far as the superior vena cava.¹⁹ Nevertheless, even when firmly secured a retained lead sometimes frees itself and migrates. The seriousness of this rare complication depends on the size of the portion of the migrated lead and where it became lodged. Migration into the inferior vena cava^{12,20,21} and the pulmonary vascular system^{12,22} has been reported.

A pacing lead in the inferior vena cava was removed by the angiographic snare catheter technique.²⁰ Migration of a portion of lead into the pulmonary vascular bed may be fatal.¹² In contrast, in a patient described by Theiss and Wirtzfeld²² there were no noticeable clinical consequences after migration, which is similar to one of our cases (case 3). Accordingly, in both instances the dislodged fragments were left in situ.

HK died recently.

References

- 1 Krug H, Zerbe F. Major venous thrombosis—a compli-

- cation of transvenous pacemaker electrodes. *Br Heart J* 1980; 44: 158-61.
- 2 Sethi GK, Bhayana JN, Scott SM. Innominate venous thrombosis: a rare complication of transvenous pacemaker electrodes. *Am Heart J* 1974; 87: 770-2.
- 3 Williams EH, Tyers GFO, Shaffer CW. Symptomatic deep venous thrombosis of the arm associated with permanent transvenous pacing electrodes. *Chest* 1978; 73: 613-5.
- 4 Balau J, Buysch KH, Marx E, Seling A, Knierem HJ. Thrombose der vena subclavia nach tranvenöser Schrittmacher-implantation. *Radiologie* 1971; 11: 50-3.
- 5 Stoney WS, Addlestone RB, Alford WC Jr, Burrus GR, Frist RA, Thomas CS Jr. The incidence of venous thrombosis following long-term transvenous pacing. *Ann Thorac Surg* 1976; 22: 166-70.
- 6 Griep RB, Daily PO, Shumway NE. Subclavian-axillary vein thrombosis following implantation of a pacemaker catheter in the internal jugular vein. *J Thorac Cardiovasc Surg* 1970; 60: 889-92.
- 7 Williams DR, Demos NJ. Thrombosis of superior vena cava caused by pacemaker wire and managed with streptokinase. *J Thorac Cardiovasc Surg* 1974; 68: 134-7.
- 8 Crook BRM, Gishen P, Robinson CG, Oram S. Occlusion of the subclavian vein associated with cephalic vein pacemaker electrodes. *Br J Surg* 1977; 64: 329-31.
- 9 Lagergren H, Johansson L, Schüller H, *et al.* 305 cases of permanent intravenous pacemaker treatment for Adams-Stokes syndrome. *Surgery* 1966; 59: 494-7.
- 10 Furman S, Escher DJW, Solomon N. Experience with myocardial and transvenous implanted cardiac pacemakers. *Am J Cardiol* 1969; 23: 66-72.
- 11 Kalmar P, Bally K, Bleese N, *et al.* Clinical complications due to pacemaker system failures and their management. In: Schaldach M, Furman S, eds. *Advances in pacemaker technology*. New York: Springer-Verlag, 1975: 153-74.
- 12 Rettig G, Doenecke P, Sen S, Volkmer I, Bette L. Complications with retained transvenous pacemaker electrodes. *Am Heart J* 1979; 98: 587-94.
- 13 Blair TP, Seibel J Jr, Goodreau J, Groves R, Ashworth HE, Baker WP. Surgical relief of thrombotic superior vena cava obstruction caused by endocardial pacing catheter. *Ann Thorac Surg* 1982; 33: 511-5.
- 14 Pauletti M, Di Ricco G, Solfanelli S, Marini C, Contini C, Giuntini C. Venous obstruction in permanent pacemaker patients: an isotopic study. *PACE* 1981; 4: 36-42.
- 15 Fritz T, Richeson JF, Fitzpatrick P, Wilson G. Venous obstruction. A potential complication of transvenous pacemaker electrodes. *Chest* 1983; 83: 534-9.
- 16 Imparato AM, Kim GE. The trapped endocardial electrode. Removal by prolonged graded skin traction. *Ann Thorac Surg* 1972; 14: 605-8.
- 17 Imparato AM, Kim GE. Electrode complications in patients with permanent cardiac pacemakers. Ten years experience. *Arch Surg* 1972; 105: 705-10.
- 18 Bilgutay AM, Jensen NK, Schmidt WR, Garamella JJ, Lynch MF. Incarceration of transvenous pacemaker electrode. Removal by traction. *Am Heart J* 1969; 77: 377-9.
- 19 Furman S, Escher DJW. Retained endocardial pacemaker electrodes. *J Thorac Cardiovasc Surg* 1968; 55: 737-40.
- 20 Ramo BW, Peter RH, Kong Y, Morris J Jr. Migration of a severed transvenous pacing catheter and its successful removal. *Am J Cardiol* 1968; 22: 880-4.
- 21 Toumbouras M, Spanos P, Konstantaras C, Lazarides DP. Inferior vena cava thrombosis due to migration of retained functionless pacemaker electrode. *Chest* 1982; 82: 785-6.
- 22 Theiss W, Wirtzfeld A. Pulmonary embolisation of retained transvenous pacemaker electrode. *Br Heart J* 1977; 39: 326-9.