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A Case Report

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Inadvertently Placed Pacing Lead: A Case Report

N. Schön

■ Introduction

Inadvertently placed pacing and implantable cardioverter defibrillator leads in the left ventricle are rare complications of device implantation and require a high index of suspicion for proper diagnosis. Management is not uniform in the reports on this subject as most concern only a limited number of patients.

Stimulation of the left ventricle has been reported due to

- passage of the pacing lead through an atrial septum defect, a sinus venous defect [1, 2] or an open foramen ovale [3] and mitral valve into the left ventricle [4–9];
- perforation of the intraventricular septum and migration of the lead into the left ventricle [10, 11];
- erroneous introduction of the pacing lead into the subclavian artery and passage into the left ventricle across the aortic valve along a retrograde course [5, 6, 12–14];
- perforation of the right ventricular apex by the pacing lead, which migrates through the pericardium towards the left ventricular epicardium.

The misplacement of the lead is diagnosed on the basis of the presence of a right bundle branch block pattern of the paces QRS, via echocardiography and via chest X-ray.

If timely removal of a malpositioned lead in the left ventricle is not performed, lifelong anticoagulation with phenprocoumon can be recommended as first-choice therapy and lead extraction kept as a stand-by in case of failure or during concomitant surgery.

■ Case Report

I report the case of a permanent pacemaker lead inadvertently placed through the right subclavian artery, across the aortic valve into the left ventricle. The 86-year-old woman had a single-pass VVI pacemaker system implanted in a neighbouring hospital in May 1990 because of sick sinus syndrome (model: Vitatron Typ Finesse 201, pacing lead: Ventrikel Focus TMT 83F IS-1 UNI). In April 1991, the patient experienced a transient ischemic attack, which recurred after treatment with aspirin. A chest X-ray (Figs. 1, 2) and the electrocardiogram (Fig. 3) suggested a pacemaker catheter malposition. No thrombotic material attached to the lead was detected by echocardiography (Figs. 4, 5). Anticoagulation was discussed but not started. The patient who was on antithrombotic therapy (aspirin) was asymptomatic up to December 2003. Then there was new evidence of neurological deficiency which also recurred. At that time, the patient was again in hospital where cranial computertomography, duplex of the carotid arteries and echocardiography showed no pathology. The patient was put on clopidogrel 75 mg once a day. One month later, the patient was presented in a university hospital to determine the future therapy. Using transesophageal echocardiography the pacemaker lead was shown to cross via the right subclavian artery through the slightly calcified aortic valve without regurgitation or stenosis and implant in the left ventricular endocardium. Again, no thrombotic material was detected attached to the lead, corresponding to the patient's uneventful course for a surprising period of 12.5 years without evidence of neurological deficiencies or of peripheral embolic

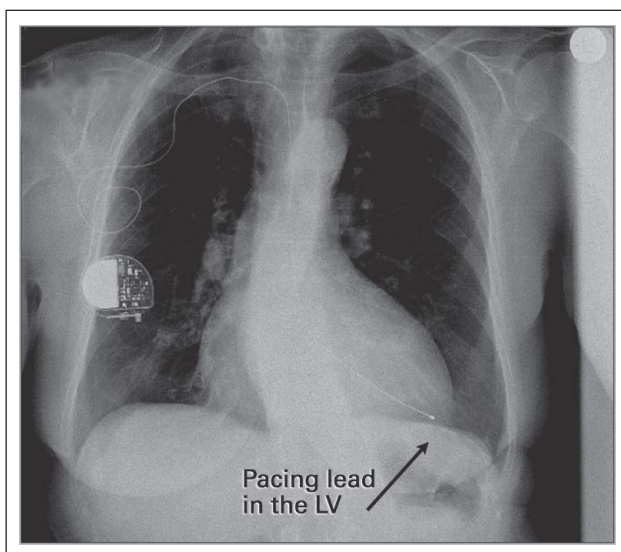


Figure 1: Chest x-ray in anterior-posterior view

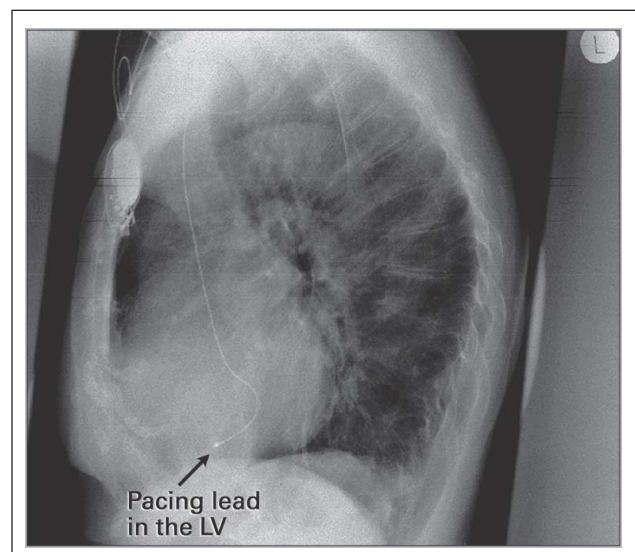


Figure 2: Chest x-ray in lateral view

phenomena. So she had two episodes of transient neurologic deficits, possibly secondary to embolic events, anticoagulation with phenprocoumon, however, was initiated. No clinical events have been recorded during a 3-year follow-up until now. Life-long full anticoagulation with an internationally normalized ratio (INR) level 2–3 is mandatory to prevent recurrence.

Discussion

In a review of the literature available, we found 31 patients with permanent endocardial lead placed unintentionally in the left ventricle.

11 patients [1, 4, 5, 7, 8, 15–19] experienced thromboembolic complications from 1 month to 6.5 years after implantation

(average 32 months). Neurological symptoms varied from *amaurosis fugax* [15, 19] to aphasia and hemiplegia [1, 4, 5, 17, 18]. Four of them had pre-treatment with aspirin [5, 8, 18], 6 no medication [1, 4, 5, 7, 15–17] and 1 was not known [19]. After the event, 6 of these 11 patients had surgical removal because of planned bypass surgery [1, 7, 15, 17–19] and one percutaneous lead replacement [8]. A thrombus was present on the removed lead in only 3 patients [7, 15, 18]. In only 2 patients, a thrombus was diagnosed via echocardiography [5, 15]. The remaining 4 patients were on anticoagulant therapy after the event [4, 5, 16]. During follow-up, there was no recurrence of symptoms after removal of the lead or conservative treatment with an anticoagulant regimen.

20 patients [2–4, 6, 7, 9, 10, 12–14, 20–26] had no thromboembolic events from 2 weeks to 17 years after implantation

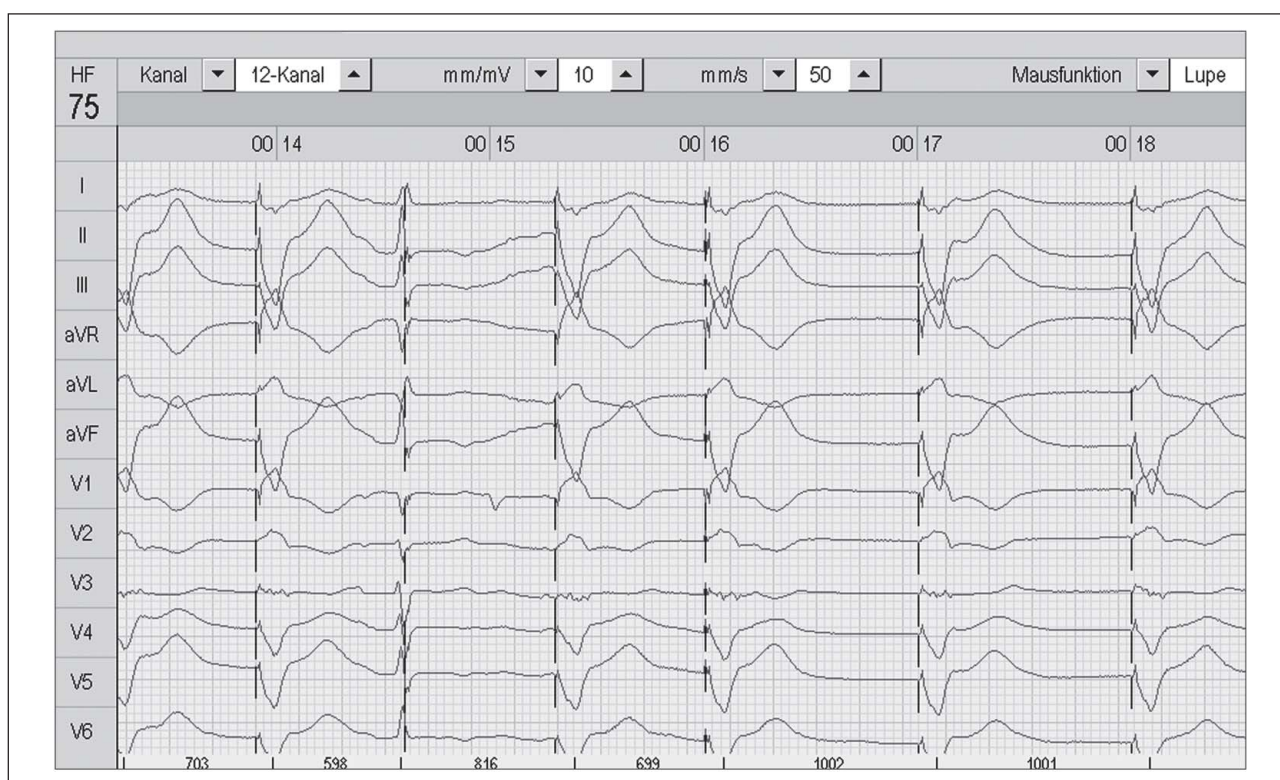


Figure 3: Pacemaker ECG

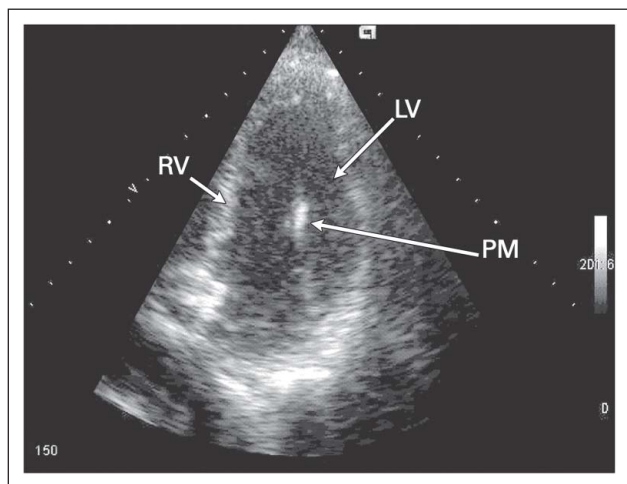


Figure 4: 4-chamber-view in echocardiography. PM = pacemaker lead

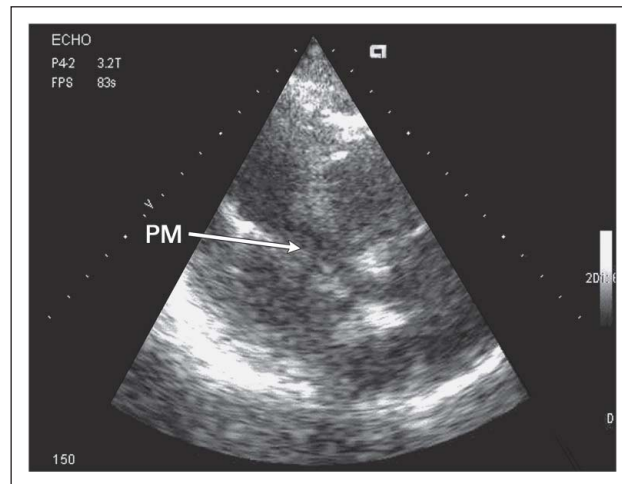


Figure 5: Long axis view in echocardiography. PM = pacemaker lead

(average 8 years). Twelve patients [2, 4, 6, 7, 9, 12, 13, 24, 25] had no medication, 5 patients had pre-treatment with aspirin [3, 4] or warfarin [6, 14, 22] and for 3, treatment was not known [10, 21, 26]. The lead was removed [7, 13, 20, 23, 24] in 5 patients, only 1 had a thrombus on the right heart side [24], the others no thrombus on the removed lead.

Conclusion

The incidence of thromboembolic events associated with leads inside the left ventricle cannot be concluded from the patient data of these few case reports. But published cases with thromboembolic events are very often reported when pacemaker or defibrillator leads have been implanted in the left ventricle.

Van Gelder [6] reached the following conclusion: if timely removal of a malpositioned lead in the left ventricle is not performed, life-long anticoagulation with warfarin can be recommended as first-choice therapy and lead extraction kept as a stand-by in case of failure or during concomitant surgery.

Sharifi [5] reached another conclusion: patients who have remained completely asymptomatic for ≥ 3 years may be kept under careful surveillance with no therapy. For asymptomatic patients diagnosed before this time period, they recommend empiric therapy with antiplatelet agents or low-dose warfarin (INR 1.5–2) with attentive observation for any symptoms. Patients developing any neurologic symptoms should be sent for transcatheter or surgical lead extraction after a period of anticoagulation or, if this is not possible, chronic anticoagulation with warfarin (INR > 2.5) must be initiated.

Our case report with a 17-year follow-up showed two clinical events, the latter after a period of 14 years. So we prefer the conclusion reached by van Gelder. Antiplatelet therapy is not the ideal safeguard against thromboembolic events which can be recommended as primary therapy [8, 18, 19].

There is very little experience of lead extraction by percutaneous lead extraction [8]. The risk of dislodging thrombi or fibrotic adhesions and causing systemic emboli is unknown when sheaths are advanced over the lead. A pre-existing thrombus will probably not be dissolved by anticoagulant therapy.

Transthoracic and transesophageal echocardiography cannot reliably detect the presence or absence of thrombi. This diagnostic method therefore cannot be used in deciding about prescribing anticoagulation.

Interestingly, this pacing lead in the left ventricle is responsible for failure in resynchronisation therapy in placing the lead in the coronary sinus. Life-long anticoagulation therapy is advisable.

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