Asymptomatic malposition of a pacing lead in the left ventricle: the case of a woman untreated with anticoagulant therapy for eight years

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We report the case of a woman with a low-rate atrial fibrillation and a wire lead inadvertently inserted in the left ventricle through an ostium secundum defect. The malposition of the lead was diagnosed 8 years after the procedure on the basis of the presence of a right bundle branch block pattern of the paced QRS, at echocardiography and at chest X-ray. The patient, who was neither on antiaggregant nor anticoagulant therapy, was asymptomatic.

Biventricular pacing is a useful tool in the therapy of patients with severe chronic heart failure and intraventricular conduction delay but the insertion of the lead in the left ventricle through the coronary sinus is not always feasible. Because a patent foramen ovale is a frequent occurrence in the adult population, we suggest that in case of an unsuccessful catheterization of the coronary sinus, the physician should search for a patent foramen ovale through which to introduce the wire lead into the left ventricle, especially if the patient is already receiving anticoagulant therapy.

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Introduction

The inadvertent positioning of the wire lead in the left ventricle is a rare and often undiagnosed complication of pacemaker implantation. In such cases, the most common route of lead malposition is through the atrial septum[1,2] and most of the patients are either asymptomatic or else experience neurological manifestations related to embolic events. Management is not uniform but anticoagulant therapy seems to be useful to avoid neurological complications[1]. We describe the case of an asymptomatic woman with a pacing lead inadvertently implanted in the left ventricle through a large ostium secundum interatrial defect 8 years previously and not treated with anticoagulant therapy.

Case report

A 74-year-old woman with a permanent atrial fibrillation, left bundle branch block and symptomatic low ventricular rate was referred to our cardiac unit, where a VVI-R pacemaker was implanted using a left subclavian transvenous endocardial approach. A flanged lead was utilized. The pacing and sensing thresholds and the lead impedance measured at the end of the implantation and at the time of discharge were satisfactory. The thresholds were evaluated using a monitor with a single ECG lead and the 12-lead ECG performed at the time of discharge did not record any paced QRS complex, because at the moment the spontaneous ventricular rate exceeded the programmed pacing rate. Although the pacing lead showed an unusually high curvature at chest X-ray, it was not suspected to be within the left ventricle.

The patient left Rome after discharge and the pacemaker was checked elsewhere. The pacemaker was checked at our Center 8 years after the procedure: the patient, who was only on digoxin, was asymptomatic and the pacing parameters were normal. A malposition of the lead in the left ventricle was suspected because the ECG showed a paced QRS with right bundle branch block (Fig. 1). This suspicion was confirmed at chest X-ray and at echocardiography. At the antero-posterior and lateral chest X-ray the ventricular lead appeared to be displaced superiorly and laterally (Fig. 2). A transthoracic echocardiogram showed the ventricular lead in the left atrium and ventricle, inserted into the later-

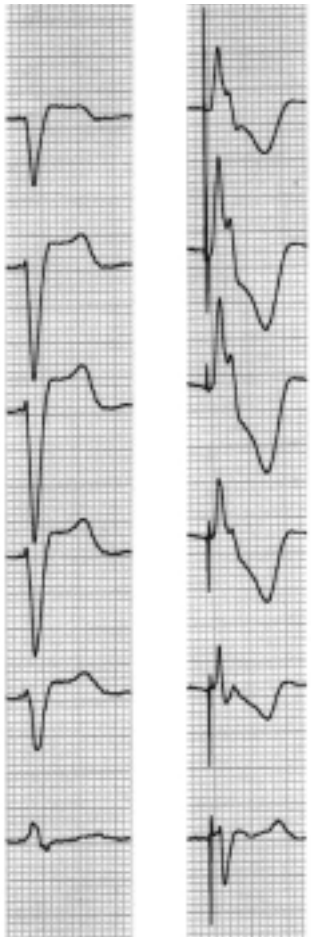

Figure 1. *Precordial ECG leads showing a spontaneous QRS complex with a left bundle branch block configuration* (left) *and a paced QRS with a right bundle branch block pattern* (right).

al wall below the posterior mitral leaflet. The atrial and ventricular sizes and the ventricular function were normal; a mild mitral insufficiency was present. A transesophageal echocardiogram confirmed the position of the lead in the left ventricle, crossing an ostium secundum defect (maximum diameter 1.5 cm). The lead appeared stable and there seemed to be no thrombotic material (Fig. 3). Computed tomographic scan did not reveal any sign of cerebral embolization.

As the patient had been asymptomatic for 8 years, the position of the lead was not corrected and anticoagulant therapy with warfarin was started.

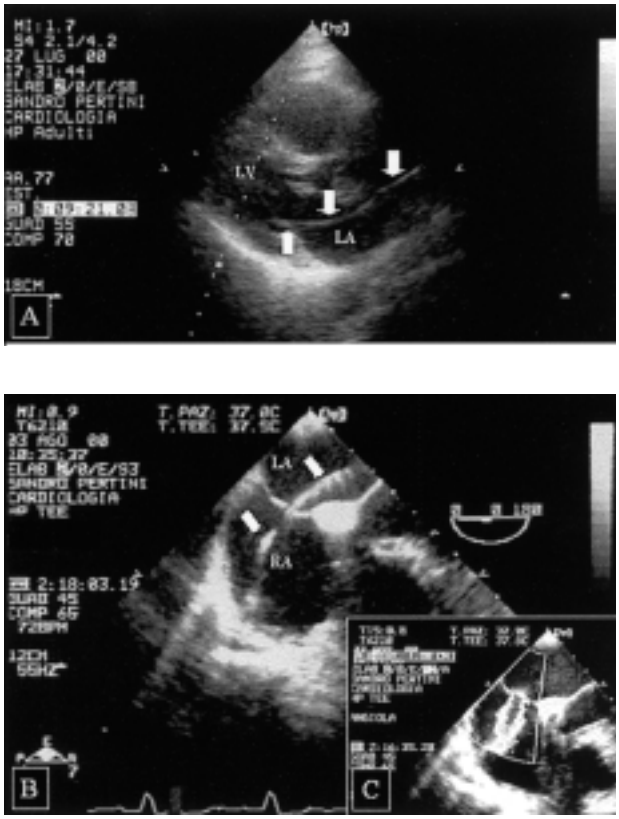

Figure 3. A: *transesophageal echocardiogram (long-axis view) showing the position of the distal portion of the lead* (arrows) *in the lateral wall of the ventricle, crossing the left atrium (LA) and the mitral valve.* B: *transesophageal echocardiogram showing the position of the lead* (arrows) *through the atrial septal defect* (C). *LV = left ventricle; RA = right atrium.*

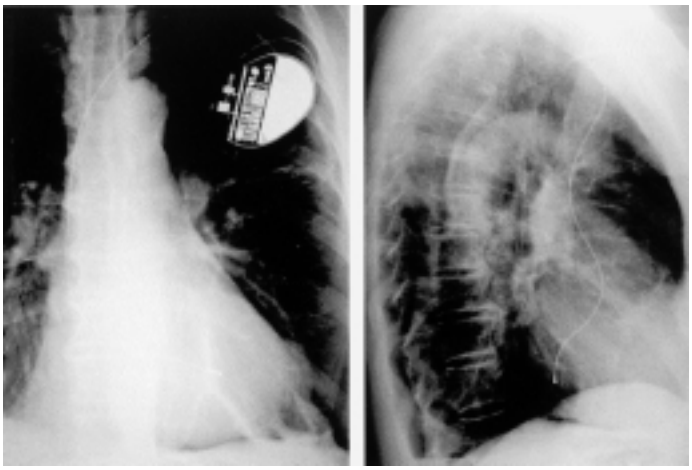

Figure 2. Left panel: *postero-anterior chest X-ray. Note the unusually high curvature of the lead as it crosses the atrial septum.* Right panel: *lateral chest X-ray. The lead appears to be displaced posteriorly and lies in the left atrium.*

Discussion

The malposition of the pacing lead in the left ventricle is rare: in a recent review[1] only 27 patients with permanent endocardial leads unintentionally placed in the left ventricle have been reported. The most common route for endocardial left ventricular pacing is the atrial septum, through a patent foramen ovale[1,3-5], an atrial septum defect[5,6] or a sinus venosus defect[7].

The ability to pass the lead up into the pulmonary artery confirms the passage into the right ventricle and this maneuver should always be performed. All patients with left ventricular pacing exhibit a right bundle branch block morphology during ventricular pacing and this finding should be considered an indication to verify the position of the lead. The correct diagnosis may be confirmed by means of an antero-posterior and lateral chest X-ray and especially by means of echocardiography. Conventional two-dimensional echocardiography may allow for the direct visualization of the lead in the left ventricle; transesophageal echocardiography permits the precise localization of the lead as it crosses from the right to the left heart and may reveal the presence of a thrombus on the wire lead.

Most patients are asymptomatic, but the incidence of thromboembolism is high, reaching a level of 37%[1]. The embolic symptoms are limited to neurological manifestations: amaurosis fugax, dizziness, syncope, aphasia, and hemiplegia[1,2]. Sharifi et al.[2] reported no thromboembolic event 3 years after the implantation of a pacemaker, perhaps because of endothelialization of the lead; Van Gelder et al.[1] reported embolic symptoms manifesting between 6 months and 6.5 years after the procedure. No patient treated with warfarin presented with embolic events.

There is increasing evidence that synchronous biventricular pacing improves the systolic function in patients with severe chronic heart failure and an intraventricular conduction delay[8,9]. The left ventricular lead is usually placed via the coronary venous system but this procedure is time-consuming and not always feasible because of technical difficulties and a long-term increase in the pacemaker threshold is often observed. Recently transseptal catheterization has been proposed as an alternative approach to left heart pacing in patients in whom attempts to enter the coronary sinus or its branches were unsuccessful[10,11]. However, this technique requires a specific competence that most physicians who implant pacemakers do not have.

Atrial septum defects are rare in adults, but a patent foramen ovale is thought to be present in approximately 25% of the adult population[12]. The reported experiences regarding the inadvertent implantation of pacing leads in the left ventricle through the atrial septum and in particular via a patent foramen ovale suggest that this approach is feasible and apparently free of complications if anticoagulant therapy is prescribed. The patient that we studied, although neither treated with anticoagulant nor with antiaggregant therapy for 8 years, did not experience any embolic event and the wire lead did not cause any serious damage to the mitral valve.

We suggest that a patent foramen ovale could be considered as a valid route to introduce the wire lead into the left ventricle in case of unsuccessful catheterization of the coronary sinus of patients in whom biventricular pacing is indicated, especially if they are already receiving anticoagulant therapy. Since this approach has never been described before, a study on the feasibility and safety of the procedure is needed.

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