(ACT 333 s, aPTT > 180 s), thrombus was still present. Attempted aspiration through the sheath was unsuccessful. At this point, a second TSP was performed and a steerable introducer (Agilis, St Jude Medical, Minnetonka, MN, USA) was deployed in the LA. Its distal end was positioned at the distal aspect of the Lasso (Figure 1C and D) and vigorous aspiration resulted in abrupt disappearance of the thrombotic image. Verification of the extracted blood revealed elements of fragmented fresh clot. No suspect image was visualized by TEE during an additional observation time of 10 min, and the ablation procedure resumed. The patient remained asymptomatic and no embolic physical sign was seen.

Discussion
Systemic (cerebrovascular) embolization is a serious potential adverse event of AF ablation, reported in up to 2% in different series.1 Prior anticoagulant therapy is mandatory and TEE to exclude LA thrombus is systematically performed. Thromboembolic complications are prevented by maintaining strong anticoagulation levels during the procedures2 and by minimizing the procedure time.

In our case, TSP was difficult and facilitated by a brief RF burst. This manoeuvre in itself had no complications in an initial small series. Two possible mechanisms of thrombus formation were invoked: in situ formation in the LA or previous formation inside the sheath (and therefore pushed inside the LA by the Lasso).

Thrombus formation on the tip of the transseptal sheath is frequent (9% in a 90 patient group3); removal of thrombus was successfully performed in all these patients by vigorous aspiration of the ‘guilty’ sheath.

In our case, since the thrombus did not dissolve despite strong anticoagulation and since aspiration by the Lasso’s sheath was unsuccessful, several options were considered. Complete withdrawal of the Lasso might have resulted in dislodgement of the thrombus. Fragmentation and embolism were feared in case of thrombolysis. Open heart thrombus removal seemed a heavy option. We chose to insert a second sheath inside the LA for a thromboaspiration attempt. Steerability of the sheath was crucial for placing the tip of the sheath exactly at the site of the thrombus anchor (the distal end of the Lasso).

Conflict of interest: none declared.

References

A life-threatening arrhythmia induced by inappropriate activation of an implantable cardioverter deﬁbrillator

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We describe an unusual case of near fatal inappropriate implantable cardioverter defibrillator therapies due to atrial oversensing by a newly implanted ventricular lead. Chest X-ray revealed dislodgement of the active fixation lead to the tricuspid annulus area explaining the atrial oversensing and intermittent ventricular therapies.

Our patient, a 62-year-old man, with an old myocardial infarction and moderate left ventricular dysfunction, had an implantable cardioverter defibrillator (ICD) implanted in 2005 for recurrent ventricular tachycardia (VT). In 2007 and in 2008, he experienced several shocks, which turned out to be inappropriate and were attributed to ‘noise’ from a malfunctioning ventricular electrode. In July 2008, the ICD was replaced, the old malfunctioning electrode was capped, and a new ventricular active fixation lead (true bipolar) was inserted. The following day a routine chest X-ray (CXR) showed proper placement of the implanted electrode.

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Two weeks after the re-implantation, the patient experienced out-of-hospital cardiac arrest. Interrogation of his ICD revealed that prior to the event the patient was in sinus rhythm. Electrogram readings from the ventricular channel demonstrated that the ventricular electrode sensed some electrical activity additional to the ventricular electrical activity, the timing of which corresponded to oversensed atrial electrical activity.

The device misinterpreted the oversensed P-waves as QRS complexes, and therefore referred to the PR interval as RR interval, which corresponded to a very short coupling interval between alleged successive QRS complexes. This interval matched the definition of VT, and the device activated its antiarrhythmic therapies—initially antitachycardia pacing (ATP), which resulted in the induction of VT (Figure 1), and in response, a shock was delivered which accelerated the VT, then matching the definition of VF. The patient received several shocks from his ICD and finally from an external automated defibrillator which terminated the arrhythmia. The device was re-programmed cancelling all antiarrhythmic therapies, in order to avoid induction of further arrhythmias.

A CXR demonstrated that the recently inserted ventricular electrode was dislodged and was located adjacent to the tricuspid valve. This location explains both the oversensing of the atrial electrical activity and the failure of several shocks to terminate the ventricular arrhythmias.

The new ventricular electrode was repositioned to a proper location in the right ventricle. Several days after the event, the patient was discharged, suffering from minimal cognitive neurological deficits.

Discussion

Common causes of inappropriate shocks include misinterpretation of cardiac rhythms (e.g. identifying supraventricular tachyarrhythmia as ventricular tachyarrhythmia), lead failure and over-sensing of T-waves and diaphragmatic myopotentials. Fatal or almost fatal ICD-induced events are exceedingly rare.

In the literature, there are reports of ventricular arrhythmias that were induced by ICDs, due to ventricular oversensing from ventricular lead ‘noise’ or undersensing of atrial activity (disrupting A-V discrimination). In addition, there is a report of inappropriate ICD shocks due to far-field oversensing of atrial electrical activity or ‘noise’, however, without induction of a ventricular arrhythmia.

Our patient, in contrast, developed life-threatening arrhythmias that proved to be almost fatal. Lead dislodgement, therefore, should be considered in the differential diagnosis of inappropriate shocks, especially those occurring relatively early after the ICD implantation.

Conflict of interest: none declared.
References

CASE REPORT
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Unsuccessful left ventricular lead implantation in two first-degree relatives. Is the coronary venous anatomy similar in both cases?
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We reported on two unsuccessful implantations of the left ventricular lead in two first-degree relatives due to inability to cannulate the coronary sinus (CS). The anatomy of the coronary venous system investigated by means of dual source computed tomography showed several similarities in both patients: narrowing of the proximal part of CS and a small number of CS tributaries.

Introduction
The variations and abnormalities of the coronary venous anatomy remain one of the principal limitations of cardiac resynchronization therapy (CRT). We reported on two unsuccessful implantations of the left ventricular (LV) lead in two relatives (mother and son) with heart failure. In both cases, the implantations failed due to abnormalities of the coronary venous system.

Case report
A 23-year-old man was admitted to our hospital with symptoms of drug refractory heart failure. The patient had undergone surgical atrial septal defect closure (ostium secundum type) at 4 years of age. In 2004, a dual chamber pacemaker was implanted for the treatment of complete heart block. Recent echocardiogram showed global LV hipokinesis with ejection fraction (EF) estimated at 15%, and LV end-systolic diameter (LVESD) of 75 mm. There were no symptoms of intra-atrial shunting. The patient was qualified for the pacemaker upgrade to CRT-D. The implantation of the LV lead failed due to the inability to advance the sheath through the coronary sinus (CS) ostium, despite the angiographic guidewire engagement and the use of various sheaths. The venous phase of the coronary angiography performed during the implantation revealed the atypical coronary venous system anatomy: there was a dominant middle cardiac vein (MCV) and a relatively small great cardiac vein (GCV). The MCV drained into the GCV very closely to the right atrial orifice (Figure 1). The dual source computed tomography (DSCT) performed several days later revealed further details of the coronary venous anatomy (Figure 2): there was no typical CS (defined as a vessel formed by the connection of the GCV and the main posterior lateral vein or Marshall’s vein),1,2 there was a narrowing between MCV and GCV junction and the right atrial orifice, and finally, the number of either GCV or MCV tributaries was scarce. Only one lateral vein appeared on both the angiography and the DSCT. Since the patient’s mother was also diagnosed with heart failure, we performed a similar investigation in her. The DSCT showed a similar anatomical situation, which led us to recommend against further attempts at CRT-D implantation in the family.

Figure 1 Venous phase of coronary angiography performed during CRT implantation in the first patient. The Amplatz catheter (AL) is positioned in the left coronary artery ostium. The middle cardiac vein (MCV) and the great cardiac vein (GCV) joined close to the right atrial orifice (RA). Note the small number of tributaries of both veins. Pacemaker leads are positioned in the right atrium and right ventricular apex. The left heart delivery sheath is visible in the right atrium.