Discussion
The PVAC is effective for PV isolation in atrial fibrillation. Therefore, as shown by the present report it may be similarly useful for ablation of ATs originating from PVs. This technique offers some advantages over traditional methods for focal AT ablation, including safer positioning of the ablation catheter at the PV ostium rather than inside the vessel. Preselected energy delivery between neighbouring vein electrodes or between single electrodes and a large surface paddle creates a homogenous lesion of varying depth likely to result in long-term PV isolation. A main disadvantage is the need for a larger sheath, which may be difficult to insert and/or increase the risk during transseptal access.

Conflict of interest: none declared.

References

CASE REPORT

A dual-loop bi-atrial macro-reentry flutter during atrial fibrillation ablation

Songwen Chen, Shaowen Liu*, Gang Chen, Yiwen Yan, Weidong Meng, Ying Zhuge, and Feng Zhang

Department of Cardiology, Shanghai First People’s Hospital, Medical College, Shanghai Jiaotong University, No 85, Wuqin Road, Shanghai 200080, China

* Corresponding author. Tel: +86 21 63240090 ext. 3052. Email: shaowen.liu@hotmail.com

Dual-loop macro-reentry atrial flutter (AFL) is an atypical AFL, which has two loops of the reentry circuit usually localized within single atrium. In this case report, we present a double-loop bi-atrial flutter during atrial fibrillation (AF) ablation, in which the two reentry circuit loops were located around the inferior vena cava (IVC) and the mitral annulus (MA) respectively.

A 75-year-old woman was admitted to our hospital for catheter ablation in the treatment of persistent atrial fibrillation (AF). The patient had a history of symptomatic, drug-refractory persistent AF for 3 years and essential hypertension. Prior to the procedure, the echocardiography revealed a dilated left atrium (LA) of 51 mm and a normal left ventricular ejection fraction of (62%).

Case report
The procedure was performed under the guidance of a three-dimensional mapping system (CARTO-XP, Biosense-Webster, Inc., Diamond, CA, USA). A decapolar circular mapping catheter (Lasso) was used for pulmonary vein (PV) mapping. At the time of procedure, the patient was in AF. After the right PVs were isolated, AF was converted to atrial flutter during circumferential left PVs ablation, with a cycle length (CL) of 248 ms. The AFL persisted after left PV isolation and roof line ablation. The conduction block across the roof was confirmed by the activation mapping. Pacing at cavotricuspid isthmus (CTI) and mitral isthmus (MI) entrained
this flutter with a post pacing interval (PPI) much longer than the CL (308 ms in CTI and 338 ms in MI). During activation mapping, a counter-clockwise activation around the mitral annulus (MA) and several double-potential-points in inferior—anteri obr septum of LA were identified (Figure 1). Another activation mapping from the right atrium (RA), a macro-reentry AFL circuit around IVC was revealed (counter-clockwise in inferior view but clockwise in anterior view, Figure 1). During linear ablation at CTI, the CL prolonged to 273 ms suddenly without any change of the CS activation sequence. Another activation mapping of LA showed a macro-reentry AFL around MA with the same activation sequence as prior. Pacing at MI indicated concealed entrainment with a PPI of 272 ms. During the linear ablation from the right PVs to MA, the AFL was terminated and sinus rhythm was restored. Bidirectional block was evaluated and confirmed by pacing from the two sides of ablation lines. After the 30-minute observation period, it was confirmed that there was no recurrent conduction across ablation lines, the operation was completed successfully.

Warfarin was administered in the evening of the procedure and continued for 3 months with international normalized ratio (INR) controlled between 1.8 and 3.0. Low-molecular-weight heparin was bridged at a dose of 1 mg/kg twice a day until the INR was 1.8. Aspirin were continued (100 mg/perday) after the cessation of warfarin therapy. During the follow-up of 12 months via telephone interview and clinical visit, the patient remained in sinus rhythm without any tachycardia onset but with sporadic premature atrial contractions (PACs), documented by 24-h Holter and electrocardiogram. The patient took oral propafenone (200 mg/perday) irregularly to control PACs.

Discussion

Dual-loop reentry AFL was defined as concomitance of two simultaneously documented loops, each meeting the definition of reentry. Double-loop reentry AFL was reported to be frequent in post-surgical heart patients, usually late after open-heart correction of congenital heart disease, or be frequent in post AF ablation patients. In our case, two reentrant loops were identified simultaneously in LA and RA during activation mapping with a CL of 248 ms, each with the spatially shortest route of unidirectional activation encompassing the full range of mapped activation times (90% of the CL of the tachycardia) and returning to the site of earliest activation. After the bidirectional block of CTI, the CL of AFL was prolonged to 273 ms without termination. A macro-reentry flutter around MA was demonstrated with the same activation sequence as prior tachycardia. A linear ablation from right PVs ablation ring to MA terminated the LA flutter.

The challenge of this case is that pacing in both isthmuses failed to entrain concealedly the flutter with PPI equal to the CL at the beginning. The potential mechanism may be the interference of the two relatively- independent reentry loops that which have a linkage in the inferior atrial septum. As the activation time of each atrium exceeded 90% of CL with a common pathway in the inferior atrial septum, a hypothesis can be proposed that both atria were involved in the dual-loop flutter. After the change of CL following the bidirectional block of CTI, the same activation sequence of LA and concealed entrainment at MI confirmed the hypothesis.

Figure 1 The bi-atria flutter activation sequence was demonstrated using three-dimensional3D mapping system. The reentry circuit is around the IVC in right atrium (counter-clockwise in inferior view but clockwise in anterior view) and it is around the MA in left atrium (counter-clockwise). IVC, :=inferior vena cava; MA, :=mitral annulus; TA, :=tricuspid annulus.
Entrainment, such as from the atrial septum, may be helpful to identify the cause of the AFL, but it was laborious and time consuming because pacing from multiple sites was necessary. Therefore, with the activation mapping, lively displayed with the availability of a three-dimensional cardiac mapping system, the AFL’s mechanism can be understood more easily and exactly.

References

CASE REPORT

Acute severe mitral regurgitation as an early complication of pacemaker implantation

Rita Miranda*, Sofia Almeida, Luís Brandão, Carlos Alvarenga, Luciano Ribeiro, Ana Rita Almeida, and Manuel Carrageta

Department of Cardiology, Hospital Garcia de Orta, Avenida Torrado da Silva, 2801-951 Almada, Portugal

* Corresponding author. Tel: +35 193 613 2273, Fax: +35 121 295 7004, Email: ritasmiranda@gmail.com

A 70-year-old man developed drug refractory acute pulmonary oedema secondary to acute severe mitral regurgitation (MR) immediately after implantation of a dual-chamber pacemaker for complete heart block. Clinical improvement occurred after allowing the patient to resume his native rhythm. A new lead was positioned within the right ventricular outflow tract (RVOT). The echocardiogram during pacing at RVOT showed minimal MR.

Case report

A 70-year-old man, referred to our hospital for asymptomatic complete atrioventricular block, underwent the implantation of a transvenous dual-chamber pacemaker. Two passive fixation leads were positioned, respectively, at the right ventricular apex and right atrial appendage.

Immediately after implantation, the patient developed drug refractory acute pulmonary oedema. He underwent urgent Doppler echocardiographic examination that demonstrated preserved ventricular function, normal left atrial size, no organic mitral leaflet disease, and severe MR due to incomplete coaptation of mitral leaflets (Figure 1A and B).

The pacemaker was then reprogrammed to VVI at 30 bpm, minimizing pacing and allowing patient to resume his native rhythm. There was an immediate improvement in the patient’s symptoms.

The hypothesis of acute severe MR secondary to ventricular dyssynchrony induced by right ventricular apical (RVA) pacing was suspected in this patient.

It was decided to extract the ventricular lead, and a new active fixation ventricular lead was positioned within the RVOT (Figure 2). The echocardiogram after this last procedure and during ventricular pacing showed normal mitral leaflet coaptation and minimal mitral regurgitation (Figure 1C and D). The patient remained haemodynamically stable and made an uneventful recovery from the episode, remaining asymptomatic at follow-up.

Discussion

RVA pacing produces a number of unfavourable effects due to an abnormal left ventricular electrical-activation sequence, namely functional MR caused by left ventricular dyssynchrony and alterations in the timing of papillary muscle contractions. Most cases of pacemaker-induced, functional MR and congestive heart failure are described several months after pacemaker implantation, like in the article by de Guillebon et al. There are few reported cases of acute severe MR as an immediate perioperative complication of permanent pacemaker insertion, leading to acute haemodynamic deterioration.

Our case report demonstrates that RVA pacing may immediately induce severe MR and acute cardiac failure, even in patients with preserved left ventricular contraction. This case shows the clinical and echocardiographic improvement of acute mitral regurgitation induced by RVA pacing by RVOT pacing, probably by improving ventricular dyssynchrony, obviating the need of a more complex procedure for upgrading to a biventricular device in a patient with no previous symptoms of heart failure and with preserved left ventricular function.

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