Webster) with all four PVs electrically isolated verified by a PV mapping catheter. Linear ablation was performed in the roof and from the left inferior PV to mitral valve isthmus and fractionated potentials were targeted in the posterior mitral valve ring. The patient remained in AF at the end of the procedure despite DC cardioversion.

Within days after the procedure the patient became breathless and centrally cyanosed. A repeat TOE showed a persistent ASD with a maximum diameter of 6 mm with bidirectional shunting and the patient underwent magnetic resonance imaging (Figure 1). The patient was referred for closure of the iatrogenic ASD that was performed using a 25 mm Amplatz® patent foramen ovale (PFO) occluder (AGA Medical). Arterial oxygen saturations improved from 86 to 96%. After further DC cardioversion, she remained free from AF for over 3 months.

The persistence of iatrogenic ASDs following transeptal procedures has been reported. In one study, 42 patients underwent TOE 9 months following PVI, no ASDs were seen in patients with a two-sheath double-puncture method, but an ASD was seen in 30% (n = 8) of those with a two-sheath single-puncture method (P = 0.01).2 Patent foramen ovales with significant right to left shunting and hypoxaemia have been described in normal hearts and transcatheter closure has been used to close the defect.3

In patients with congenital heart disease, high RA pressures and iatrogenic ASD right to left shunting may occur. Our patient was at increased risk because of the long procedure time, elevated RA pressure with the Fontan circulation, and a single-puncture, two-sheath technique.

We recommend that a double-transseptal puncture or a single-sheath technique is used with early sheath withdrawal back into the RA for left atrial ablation procedures, in patients with raised right heart pressures or complex grown up congenital heart disease, to minimize the risk of reverse shunting through an iatrogenic ASD. We have reported successful transcatheter closure of this defect as treatment for this complication.

Conflict of interest: none declared.

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References

CASE REPORT

Three-dimensional electroanatomic entrainment map in atypical atrial flutter late after heart transplantation

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Atrial flutter in the donor part of orthotopic heart transplants has been reported and successfully treated by radiofrequency ablation of the cavo-tricuspid isthmus, but mapping and ablation of atypical flutter circuits may be challenging.1 Entrainment mapping has been used in combination with activation mapping to define the mechanism of atypical atrial flutter. Here, we report a case where colour-coded three-dimensional (3D) entrainment mapping allowed us to accurately determine and visualize the 3D location of the reentrant circuit and to plan the ablation of a left atrial flutter without the need for activation mapping.

Case presentation
A 69-year-old patient developed symptomatic persistent atypical flutter 5 years after heart transplantation with bi-atrial anastomosis. The arrhythmias occurred shortly after electrical cardioversion (Figure 1A). Mean ventricular rate over 24 h was 105/min despite atrio-
ventricular node slowing agents and there was a decrease in left ventricular ejection fraction from 55 to 35%. Both atria were severely enlarged. Graft rejection was excluded by right ventricular biopsy. The electrophysiological study was performed using the Carto System and a 7 F Navistar catheter (Biosense Webster, Diamond Bar, CA, USA). A decapolar 6 F diagnostic catheter was placed in the coronary sinus (CS) for time reference (Bard Electrophysiology, Billerica, MA, USA). The host right atrium was in sinus rhythm, whereas the donor atrium had a cycle length (CL) of 230 ms.

Discussion
Activation mapping requests generation of complete and dense maps and correct annotation of electrograms may be challenging, e.g. when activation of the entire atria lasts more than the CL of the tachycardia in patients with important conduction delays. Therefore, in this case with very abnormal atrial conduction, we exclusively used entrainment mapping to delineate the reentry circuit and displayed post-pacing intervals (PPI–CL) values on an electroanatomic three-dimensional (3D) map (Figures 1B and 2), as recently described by Esato et al. Entrainment mapping showed long PPIs in the right lateral atrium and at the lateral cavotricuspid isthmus, and short PPI at the septum and in the CS (PPI – CL ≤ 20 ms). Consequently, transseptal puncture and electroanatomic entrainment mapping of the left atrium were performed. In addition, anatomical reconstruction of the host left atrium (HLA) in sinus rhythm (in green) and of the left pulmonary veins (PVs) is shown (Figure 2).

Points with short PPI (equal or less than CL + 20 ms) were found (in red) around the mitral annulus, in the CS, at the lower part of the right atrial septum (Figures 1B and 2). Thus, entrainment data are consistent with a macroreentry around the mitral annulus, with a reentry front between the left atrial anastomosis and the mitral annulus. Activation within the CS showed a clockwise activation.
A line of radiofrequency lesions was constructed at the infero-lateral aspect of the mitral annulus between the atrial suture and the mitral annulus. After extensive ablation along this line (exclusively in the left atrium, max power 50 W), the atrial flutter terminated with an impedance rise with a ‘pop’. Thereafter, atrial flutter was not inducible (1–3 extrasystoles with two different pacing CL and burst pacing). The procedure time was 113 min, fluoroscopy time 19 min, and ablation time 44 min.

However, the arrhythmia recurred 1 week later with identical morphology in 12-lead ECG, with the same CL, and the same mechanism according to entrainment mapping. Several additional ablations completing the ablation line in the left atrium were performed without effect. Finally, flutter termination was obtained with ablation from within the CS (max power of 30 W), completing the mitral isthmus line from the epicardial side. Mitral isthmus block was documented with differential pacing. In the following months, the patient had no recurrence.

This case illustrates the importance of entrainment mapping in the evaluation of atypical flutter. Colour-coded 3D entrainment mapping allows to accurately understand the 3D location of the reentrant circuit without the need of activation mapping.

Acceleration or termination of macroreentrant atrial arrhythmia induced by programmed electrical stimulation has been described in several situations. However, such modification of the clinical tachycardia by careful entrainment pacing (10–20 ms below CL, reduction of stimulation output) is uncommon.

Conflict of interest: none declared.

References


CASE REPORT

Intrinsic neural reflexes in the post-transplant human heart

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Radiofrequency ablation of post-transplant flutter in a centrally denervated donor atrium at a site remote from the AV node resulted in transient worsening of AV nodal conduction, with absent central vagal reinnervation. This could be an electrophysiological marker of intact innervation to the donor AV node from the intrinsic cardiac neuronal plexus, not demonstrated in human hearts earlier.

Introduction

There are no reports of the electrophysiological effects of intact intrinsic innervation to the AV node post-transplant. The intrinsic cardiac neuronal system has not been well elucidated in humans.

Case

In a 40-year-old man, post-atrioatrial cardiac transplantation for non-ischaemic cardiomyopathy mapping revealed isthmus-dependent donor atrial clockwise flutter and dissociated recipient atrial sinus rhythm (Figure 1). During four consecutive radiofrequency ablations (8 mm tip non-irrigated, 50 W, 50–60°C) at the cavo-tricuspid isthmus at 6.30 o’clock on the left anterior oblique projection remote from the AV node and anteriorly at the tricuspid annulus with no local His electrograms on the ablation catheter, there was reproducible transient worsening of atroventricular conduction from 2:1 to 3:1 and 4:1. There was no significant pain during energy delivery. The patient was in 2:1 AV block prior to the start of ablation. No spontaneous change in AV conduction was seen before or after...