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 documented 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.3

**Conflict of interest:** none declared.

**References**


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**CASE REPORT**

**Intrinsic neural reflexes in the post-transplant human heart**

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**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 cavotricuspid 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 atrioventricular 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 ablation.

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.

**Intrinsic neural reflexes in the post-transplant human heart**

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Ablation. The transient AV nodal block could be either due to vagal reinnervation or a local intrinsic cardiac neuronal reflex. The absence of recipient atrial sinus slowing and donor atrial flutter cycle lengthening in the presence of AV nodal block suggested vagal denervation. During Valsalva manoeuvre and head-up tilt test (on and off isoprenaline) which depend on intact cardiovagal integrity, the baseline rhythm was sinus tachycardia, with no significant changes in heart rate or blood pressure indicating a centrally denervated heart. Transient AV nodal block was likely mediated by the intrinsic cardiac neuronal system, the ‘brain’ of the heart, which may have either been intact or may have reinnervated the donor AV node.

Discussion
The status of reinnervation post-cardiac transplant is not clear.\(^1\) Lack of response to atropine and loss of heart rate variability post-transplant indicate that the transplanted heart may not reinnervate. At the same time, electrical stimulation of cardiac autonomic nerves, responses to tyramine-induced release of norepinephrine stores, and scintigraphic assessment of myocardial tracer uptake support autonomic reinnervation.\(^1,2\)

In an animal study, vagal input to the AV node has been shown to be via the ganglion in the inferior vena cava-inferior left atrium fat pad which may partly be above the coronary sinus, and retained in the donor heart.\(^3\) However, vagal effects like AV block observed during ablation have not been previously reported in a donor heart. It is unlikely that the radiofrequency lesions affected an extension of the AV node since the burns were made in an anterior location at the tricuspid annulus, and away from the interatrial septum. Cardiac transplantation therefore provides an ideal model to study intrinsic neural reflex loops.

Conclusion
The intrinsic cardiac neuronal plexus may retain innervation or may reinnervate post-transplantation making the heart capable of responding to local stimuli.

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References