Cardiology in the Young

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Transient block during radiofrequency ablation well away from the His–Purkinje system: atrioventricular nodal artery trauma?

Victor Grech[1], Elizabeth Grech[2] and Joseph DeGiovanni[1]

[1]Mater Dei Hospital, Tal-Qroqq, Malta and [2]University of Malta, Msida, Malta

Brief Report

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Author for correspondence:
Elizabeth Grech, University of Malta, Msida, Malta. E-mail: elizabeth.v.grech.18@um.edu.mt

Abstract

Cardiac arrhythmias occur at all ages. Cardiac mapping and ablation are established methods for curing arrhythmia substrates; however, complications may occur. We report a patient with transient Wenckebach heart block during radiofrequency ablation in the setting of Wolff Parkinson White syndrome despite the ablation catheter being well away from the atrioventricular node, and we speculate on the potential mechanism.

Cardiac arrhythmias occur at all ages. Wolff Parkinson White Syndrome is one of the commoner causes of childhood tachyarrhythmias. Cardiac mapping and ablation are standard, but complications may occur.[1]

We report a patient with transient Wenckebach heart block during ablation of a right mid-septal accessory pathway in the setting of Wolff Parkinson White syndrome despite the ablation catheter being well away from the atrioventricular node, and we speculate on the potential mechanism.

Case report

Our patient was a 5.5-year-old boy who had presented with narrow complex supraventricular tachycardia, with pre-excitation after conversion to sinus rhythm. The standard algorithm indicated a mid-septal pathway.[2] He was maintained on propranolol and underwent mapping and ablation of a right mid-septal accessory pathway under general anaesthesia. Optimal signals were obtained with an intracardiac V signal preceding the surface ECG by 19 msec and a favourable unipolar signal. Pathway ablation occurred after 1–2 beats from the onset of energy delivery. Recurrence occurred and four further applications were made with early ablation but with some non-conducted atrial beats and intermittent and variable heart block, mostly of the Wenckebach type (Figs 1 and 2). Intravenous atropine and dexamethasone were given, and the escape rate was 65 bpm, but he spontaneously reverted to sinus rhythm after 1 hour and remained so subsequently and with no pathway conduction. He was discharged after 24 hours on aspirin.

Discussion

Atrioventricular node conduction may be compromised during radiofrequency ablation of septal pathways or later. Late block may be related to radiofrequency lesion extension, involving the artery or the node. The degree of block can be of varying severity and is usually transient although some are permanent and require permanent pacing, in around 2% for the overall group.[3]

Many believe that the mechanism of the atrioventricular block is direct damage to the atrioventricular node itself, although many of these pathways, apart from the antero-septal and Para-Hisian, are anatomically far away from the His signal where the compact atrioventricular node is considered to be located.[3] Because of this and the transient nature in most cases, we propose an alternative mechanism, namely, trauma to the atrioventricular node artery, enough to cause spasm but not complete occlusion. Once the spasm disappears, we assume blood flow is restored to the atrioventricular node artery, and functionality of the node is restored.

It is possible that the right free wall pathways associated with block are more likely to be related to right coronary artery spasm rather than direct atrioventricular node damage. While the circumflex coronary artery travels close to the coronary sinus, the atrioventricular node itself does not lie in close proximity to this area.[4] Atrioventricular block can also occur after classical isthmus ablation for atrial flutter and, again, this is not close to the atrioventricular node but can be close to the right coronary artery, so the same mechanism may apply.[5] In addition, mid-septal accessory pathways have a high risk of AV nodal injury with transient block due to their close proximity to the node and due to the small amplitude of the atrial signals.

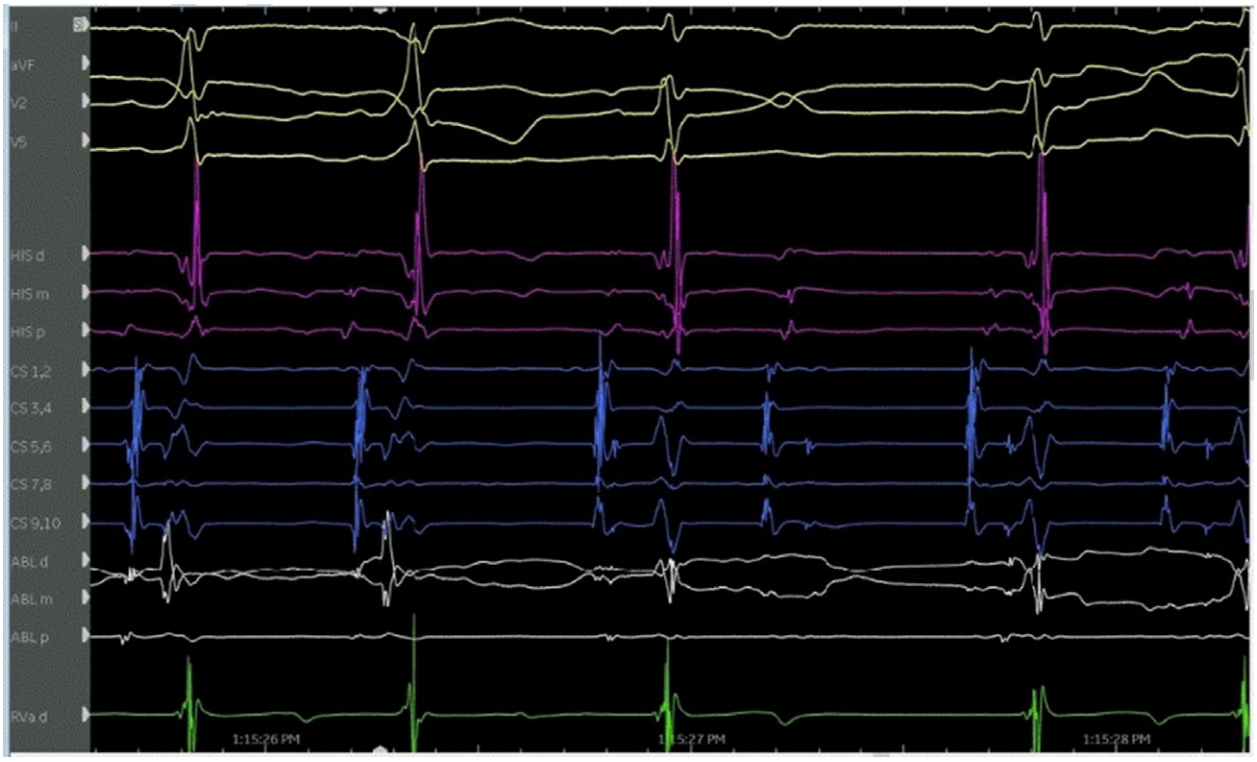

Figure 1. Surface and intracardiac ECG showing early pathway ablation followed by sinus rhythm and normal PR interval.

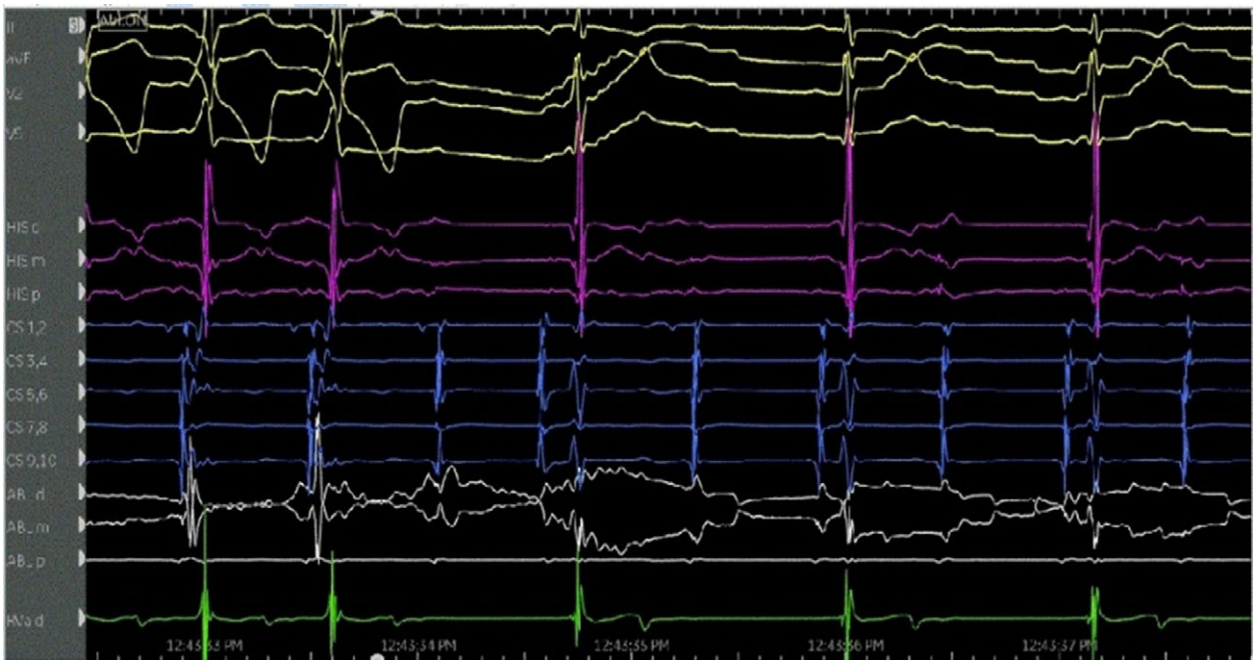

Figure 2. Surface and intracardiac ECG showing progression from 1:1 conduction to second degree atrioventricular block.

Should non-conducted atrial beats or blocks occur, radiofrequency energy must be stopped immediately. After returning to sinus rhythm, one may consider continuing with a lower energy output ablation which can be titrated whilst observing the intracardiac electrograms. Temperature can also be monitored and titrated to try and achieve pathway ablation without compromising atrioventricular node function.

The atrioventricular node artery courses in the posterior pyramidal space from its origin to termination in the central fibrous body. It commonly arises not only from the right coronary

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artery but may also arise from the posterior descending coronary artery and the left coronary artery via the circumflex branch. Some have a dual supply from both coronary arteries.[6]

Irrespective of the origin of the atrioventricular node artery, the relationship to the mid-septal area is close and the same, but the compact atrioventricular node itself is usually well away anatomically.[6] It is thus reasonable to propose that atrioventricular block in ablation of mid-septal pathways is more likely to be related to injury to the atrioventricular node artery, resulting static irreversible damage or progressive obstruction, or transient spasm and not injury to the node itself.[7] If the heart block is of a high degree, such as Mobitz Type II or complete, and if it is prolonged, the assumption is that the coronary artery damage is physical, whereas if the heart block is of a lower degree and transient, spasm is a more likely mechanism.[8] If the heart block occurs late, usually weeks after the ablation, this is potentially secondary to oedema/intimal hyperplasia with stenosis, which is static and can be progressive. Some require stenting to restore coronary flow or to abolish symptoms or ventricular dysfunction.[9]

When heart block occurs during ablation, and particularly if this and any ST segment elevation is transient, then spasm is more likely.[3] Intermittent return to sinus rhythm alternating with block can be due to spasm and consideration has to be given to selective coronary angiography.[9] In our experience, if spasm is still suspected, intra-coronary nitrate should be given and angiography repeated in addition to intravenous atropine and dexamethasone. If the narrowing fails to respond to nitrate administration, stenting may be indicated. Coronary artery angiography should, therefore, be selective and not routine. Spasm should be treated medically with nitrates to try and restore normal flow and atrioventricular node function.

In our patient, part of the ablation was done in what is known as the cavotricuspid isthmus, part of the right atrium which is a bridge of right atrial myocardium that is located between the inferior vena cava ostium and the tricuspid valve, a common target for atrial flutter ablation, and the area from which the atrioventricular node artery commonly arises.[6]

We speculate that in our patient, reversible trauma to the atrioventricular node artery due to radiofrequency application in the cavotricuspid isthmus may have caused a transient block. The literature is sparse on this topic, and to the best of our knowledge, this is the first such report with a proposed mechanism.

Cryoablation as opposed to radiofrequency ablation is increasingly being adopted as a first-line energy source in younger patients as it is safe, effective, and has comparable success compared with radiofrequency. Although late recurrences are more commonly associated after cryoablation, the avoidance of permanent atrioventricular block makes cryoablation a generally safer treatment option.[10]

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Conflicts of interest. None.

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