CASE REPORT AND LITERATURE REVIEW

Autonomic effects of radiofrequency catheter ablation

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Radiofrequency (RF) catheter ablation has become the treatment of choice for a variety of supraventricular tachycardias. Autonomic dysfunction may occur during application of RF current; these abnormalities resolve quickly when current delivery is terminated. We present a case of sinus node arrest and AV block in a 69-year-old woman induced by RF catheter ablation of an AV nodal slow pathway. The proposed mechanisms are a Bezold-Jarisch-like phenomenon or paradoxical activation of cardiac C fibres by direct neural sympathetic stimulation or RF-induced myocardial injury. Based on a review of previously published reports, autonomic effects of RF ablation are discussed.

Key Words: Radiofrequency ablation, AV nodal reentrant tachycardia, slow pathway, asystole, autonomic dysfunction.

Introduction

Radiofrequency (RF) catheter ablation has been widely accepted as the treatment of choice for a variety of supraventricular tachycardias, including atrioventricular (AV) nodal reentrant tachycardia[1–3]. It is a safe procedure in experienced centres and definitive cure is obtained in the vast majority of cases. A potential, but rare complication is complete heart block requiring implantation of a permanent pacemaker[4,5]. Selective ablation of the slow pathway is less likely to be associated with conduction disturbances, and therefore it has been the favoured approach in most centres. Nevertheless, transient AV block or abrupt slowing of the sinus node suggesting autonomic dysfunction may occasionally occur during RF current application to sites remote from the sinus node and the AV node[6]. Direct stimulation of parasympathetic fibres travelling to the sinus node and the AV node, or alternatively, stimulation of autonomic receptors that by reflex mediate a change in autonomic tone affecting the sinus node and AV node function are among the proposed mechanisms for these observations[6].

Cardiac asystole, an extreme manifestation of neurocardiogenic reaction, may also rarely occur during RF current application. Cases of RF ablation-induced asystole during ablation of accessory pathways have been reported previously[7,8]. Profound sinus bradycardia and asystole may also occur during ablation of pulmonary tissues in patients with paroxysmal atrial fibrillation[9]. We present a case of sinus node arrest and AV block with resultant syncope induced during RF ablation of an AV nodal slow pathway.

Case history

A 69-year-old female patient with AV nodal reentrant tachycardia was referred to our institution for RF catheter ablation. The patient had experienced syncopal episodes during early adulthood, suggestive of neurocardiogenic syncope, and had suffered from recurrent, drug-resistant palpitations for more than 10 years. She had undergone RF catheter ablation 4 years before which revealed an AV nodal reentrant tachycardia with a rate of 125 beats.min⁻¹ that was no longer inducible after ablation of the slow pathway. Administration of isoprenaline during this procedure resulted in presyncope due to transient paradoxical slowing of the sinus rate (PP intervals up to 2 s) and transient complete AV block with a ventricular escape rhythm of 32 beats.min⁻¹ during atrial pacing. The ablation...
procedure was considered successful and antiarrhythmic medications were withdrawn.

Despite initial success, the patient reported palpitations several months after discharge. Her physical examination and the surface 12-lead ECG were normal. After informed consent had been obtained, the patient was admitted to the electrophysiological (EP) laboratory in the fasting, non-sedated state. Three multipolar electrode catheters (2-mm interelectrode space) were introduced from the right femoral vein and placed in the right atrium, His bundle area, and right ventricle. The clinical tachycardia with a rate of 120 beats.min\(^{-1}\) was induced during placement and manipulation of the electrode catheters and was also easily inducible during baseline EP study. The diagnosis of AV nodal reentrant tachycardia was established by tachycardia initiation dependent on an A-H delay of 70 ms and the earliest retrograde activation in the His bundle electrogram. Termination could be achieved easily with atrial or ventricular pacing. A 7-French deflectable quadripolar ablation catheter with a 4-mm tip and 2-mm interelectrode distance (Sulzer Ospyka Cerablate Plus TTwin 745, Grenzach-Wyhlen, Germany) was introduced. The tip of the ablation catheter was positioned along the posteroseptal right atrium, close to the tricuspid annulus, where the His deflection was no longer visible and the ratio of atrial to ventricular deflection was below 1. RF energy was applied with an Ospyka HAT 300 generator (Grenzach-Wyhlen, Germany) between the distal electrode of the ablation catheter and the cutaneous patch electrode placed over the left scapula. During initial delivery of the temperature-controlled RF current, progressive slowing of the sinus node was noted (Fig. 1). The second RF application in the same region during tachycardia resulted in sinus node arrest with resultant syncope after approximately 7 s of RF current delivery (Fig. 2). Atrial pacing showed complete AV block initially which recovered after approximately 10 s. Administration of atropine abolished the occurrence of sinus arrest and AV block during RF current delivery in the same region. The subsequent two attempts at the same location resulted in slowing of the sinus rate. The patient reported no discomfort or pain during the whole procedure. Careful assessment of sinus nodal and AV nodal function following asystole revealed no abnormalities. There was no detectable sudden increment in the A-H interval and the tachycardia was no longer inducible. The patient remained free of tachycardia and syncope recurrences 1 year after the procedure.

**Discussion**

It is known that autonomic dysfunction may occur as a consequence of RF catheter ablation of a variety of supraventricular tachycardias\(^6\). Acute effects during...
application of RF current include slowing of the sinus rate or transient AV block which usually resolve quickly when current delivery is terminated. Transient AV block may occur due to mechanical or RF injury in the junctional area.[10–12] Application of RF current along the tricuspid or mitral annulus, at sites distant from both the sinus node and the AV node, may also be associated with profound sinus bradycardia or transient AV block, suggestive of autonomic dysfunction.[6] It has been shown that atrial tissue in close proximity to the AV rings has a high concentration of autonomic nerve fibres. Therefore, direct stimulation of parasympathetic nerve fibres travelling from the site of RF application to the sinus node and the AV node and alternatively, a reflex-mediated change in autonomic tone are among the proposed mechanisms.[6]

In contrast to the above-mentioned transient effects, RF ablation may create chronic AV block or cause a long-lasting increase in sinus rate.[13–15] The precise mechanism for inappropriate sinus tachycardia, defined as a resting sinus rate >100 beats min⁻¹ in the absence of physiological precipitants, is unknown. A study based upon spectral analysis of heart rate variability showed a decrease in high-frequency components immediately after an ablation procedure, suggestive of reduced parasympathetic tone.[16] The abnormalities of heart rate and of heart rate variability resolved 1 to 6 months after ablation, with reappearance of the high frequency parasympathetic component, probably due to reinnervation. These changes were most obvious in patients undergoing AV node modification and ablation of a posteroseptal accessory bypass tract and were related to a possible concomitant destruction of vagal inputs.[16,17]

Transient asystole, preceded by a profound slowing of the sinus rate has been reported during RF ablation of accessory pathways.[7,8] Schlapfer et al. presented a case of sinus bradycardia followed by an 8-s asystole induced by RF current application via the coronary sinus for ablation of a left posteroseptal accessory pathway.[7] Tsai et al. reported a case of asystole for 5·5 s during RF application on the ventricular aspect of the mitral annulus for a left anterolateral accessory pathway.[8] Similar to the previous case, there was progressive slowing of the sinus rhythm. Asystole was terminated by right ventricular pacing and, after 15 s, the sinus rate gradually increased to the pre-ablation level. Both patients experienced near-syncope during the transient asystole. A reflex-mediated mechanism, the Bezold-Jarisch phenomenon, was proposed as an explanation in both cases. This inhibitory reflex originating in cardiac sensory receptors with vagal afferents produces both an increase in parasympathetic activity and a decrease in

![Figure 2](image-url)
sympathetic activity resulting in bradycardia, vasodilation and hypotension\(^{[18-23]}\). More recently, similar observations have been made during RF ablation of the pulmonary vein tissues in patients with paroxysmal atrial fibrillation\(^{[29]}\).

The anatomy of the autonomic innervation of the heart is complex\(^{[24]}\). The sympathetic and parasympathetic innervation of the sinus node and the AV node follow different courses. Parasympathetic pre-ganglionic neurones, located in the medulla oblongata of the brain stem, send fibres to the heart through the vagus nerves. Within the heart itself, pre-ganglionic fibres destined to innervate the sinus node terminate in the pulmonary vein fat pad, whereas those that innervate predominantly the AV node terminate in the inferior vena cava–inferior left atrial fat pad. Parasympathetic denervation by surgical dissection of the fat pads does not influence sympathetic control of sinus rate and AV nodal conduction. The human heart contains a variety of morphologically distinct nerve terminals that are distributed more widely than has been described in experimental animals\(^{[24]}\). Coronary sinus and epicardial nerve terminals arise only from myelinated nerve fibres, whereas endocardial terminals arise from either myelinated or non-myelinated fibres. The non-myelinated endocardial nerve terminals are believed to be responsible for reflex vasodilation and bradycardia, and correspond to those described electrophysiologically as C-fibres. In addition to intramyocardial pressure, C-fibres may also be activated by nicotine, catecholamines, neural sympathetic stimulation and myocardial infarction\(^{[25]}\). The unmyelinated afferent C-fibre terminals were found to be numerous in areas of the roof of the left atrium, around the four pulmonary veins, the lateral wall of the right atrium, and the posterior wall of the left ventricle\(^{[25]}\). Thus, profound sinus bradycardia and asystole occurring when RF energy was delivered to the regions described in the previously published report\(^{[27,28]}\) is concordant with the known high concentration of vagal afferent receptors in these areas.

Our case is, to the best of our knowledge, the first reported case of reflex-mediated sinus node arrest and AV block induced by RF ablation of an AV nodal slow pathway. A reflex mechanism in our case is suggested by the fact that asystole duration was longer than that of direct stimulation of efferent vagal fibres, which would only last for 1 to 2 s after cessation of stimulation\(^{[26]}\). Assessment of the sinus node and the AV nodal function following termination of asystole revealed no abnormalities, suggesting that the transient sinus arrest and the AV block had been the result of a change in autonomic tone. Atropine abolished reoccurrence of this phenomenon during subsequent RF applications, possibly due to its counteracting effects on the efferent parasympathetic limb of the reflex arc. The patient experienced no pain during RF current delivery, which could provoke a central type vasovagal response\(^{[27,28]}\). Previous history of syncopal episodes and a decrease in heart rate following isoprenaline administration in our case suggests a mechanism similar to that observed in neurocardiogenic syncope, a condition characterized by a paradoxical withdrawal of sympathetic activity and an increase in parasympathetic activity possibly in response to excessive stimulation of ventricular mechanoreceptors\(^{[29-33]}\). While the efferent limb of the reflex arc in our case is probably similar to that observed in neurocardiogenic syncope, the afferent limb was triggered by RF ablation and not by changes in intramyocardial pressure.

A Bezold-Jarisch-like phenomenon or paradoxical activation of cardiac C fibres by direct neural sympathetic stimulation, and RF-induced myocardial injury may be responsible in our case, but given the complexity of autonomic innervation in human hearts, the above mentioned causal mechanisms can only be speculative. However, our case might serve to call attention to the possibility that a reflex transient sinus node arrest and AV block may occur during RF ablation of an AV nodal slow pathway.

References


