Delayed Response to Radiofrequency Ablation of Accessory Connections

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DICK, II, MACDONALD, ET AL.: Delayed Response to Radiofrequency Ablation of Accessory Connections. *This article summarizes delayed interruption in anomalous conduction through accessory connections following radiofrequency ablation attempts in three patients. The time course of the delayed interruption in accessory connection conduction suggests that such an effect is unlikely to occur after the first week following unsuccessful radiofrequency ablation. (PACE, Vol. 16, November 1993)*

ablation, children, radiofrequency, supraventricular tachycardia, Wolff-Parkinson-White syndrome

Introduction

Recent articles have confirmed the efficacy and safety of radiofrequency energy to interrupt accessory atrioventricular connections in the human heart.[1–5] Interruption of conduction in the accessory connection typically occurs within 3–4 seconds of application of the radiofrequency pulse at the mapped accessory pathway site.[5] In this article, we outline our experience with three patients who demonstrated transient interruption of the accessory connection conduction during radiofrequency energy application, followed by a variable period of return of conduction, ending finally with permanent loss of conduction, in effect achieving successful delayed radiofrequency ablation of the accessory connection.

In our 85 patients with accessory connections, 55 patients with left-sided pathways, 3 patients demonstrated transient interruption of a left-sided accessory connection during radiofrequency energy application, followed by late permanent loss of conduction. These three patients (Table I) form the basis of this article.

Patient 1 demonstrated persistent junctional reciprocating tachycardia with a long RP interval and a short PR. Patient 2 demonstrated preexcitation and orthodromic reciprocating tachycardia. Patient 3 demonstrated a normal ECG and orthodromic reciprocating tachycardia. The three patients were brought to the electrophysiological laboratory in the postabsorptive state. Under general anesthesia, electrophysiological study and radiofrequency ablation were performed as previously described.[5] Radiofrequency energy was delivered through a 7-French electrode catheter with a 4-mm electrode tip (Mansfield Webster, Watertown, MA, USA) The energy source was a Radionics RFG-3C radiofrequency lesion generator (Radionics, Inc., Burlington, MA, USA). Radiofrequency energy was delivered at 60 volts with a power output of 25–40 watts for 6–60 seconds. Delivery of current was terminated after 6–10 seconds if the accessory pathway conduction was not interrupted; energy was continued for 30 seconds if pathway conduction was terminated.

Patient 1 experienced transient loss of conduction for 7 minutes after application #15 and for 15 minutes after application #26. However, persistent junctional reciprocating tachycardia returned and was present when he left the electrophysiological laboratory. In patient 2 preexcita-

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Received February 18, 1993; revision May 4, 1993; accepted May 25, 1993.

Table I.

PT #	Age/Yr	Sex	Session	RF Trials	Volts	Seconds	AC Site	ACERP Anterograde	Long-Term EP Result	Months F/U After Ablation	Follow-Up Clinical Status
1	3	M	1	28	60	10–60	Posterior	Concealed	Negative	30	Well-No SVT
2	14	M	1	3	60	10–60	Posterior	310	Negative	27	Well-No SVT
3	15	M	1	18	60	10–60	Left Lateral	Concealed	—	8	Well-SVT
3	15.5	M	2	24	60	10–60	Left Lateral	Concealed	Negative	21	Well-No SVT

AC = accessory connection; EPS = electrophysiological study; ERP = effective refractory period; F/U = follow-up; M = male; RF = radiofrequency; SECS = seconds; YR = years.

tion disappeared during the second application of radiofrequency energy and accessory connection conduction was absent. Patient 3 required a second session of radiofrequency ablation because of failure at first session. The patient experienced transient (seconds to 1 minute) interruption of accessory connection conduction during radiofrequency applications at both sessions; however, accessory connection conduction returned and the patients left the electrophysiological laboratory with inducible supraventricular tachycardia (SVT) and intact accessory connection conduction following both sessions.

In two patients an estimate of the time course for the delayed effect could be determined. In patient 1, electrocardiographic telemetry disclosed spontaneous cessation of this incessant tachycardia 12 hours after the procedure, confirmed by Holter tracing 3 days later. In patient 2 electrocardiographic telemetry and a 12-lead ECG 4 hours after ablation demonstrated preexcitation identical to that observed prior to radiofrequency ablation. Two hours later, a repeat ECG showed normal atrioventricular conduction without preexcitation.

Patients 1, 2, and 3 underwent electrophysiological study 6 months, 2 days, and 12 months, respectively, following the final session of radiofrequency ablation. Accessory connection conduction was absent in all three patients and SVT could not be induced, even during isoproterenol infusion. The three patients have been free of symptoms suggestive of SVT with normal atrioventricular conduction on the ECG for 11–30 months.

Discussion

This experience demonstrates a permanent delayed response to radiofrequency ablation of accessory connections; in our laboratory it was noted in 3 of 85 patients (3 of 55 patients with left-sided pathways). In contrast to Leitch and associates[6] who have reported not only a delayed response to radiofrequency ablation in four patients, but also, most importantly, a later return of anomalous conduction in these same four patients; we have confirmed permanent interruption in anomalous conduction with a much longer follow-up interval. Furthermore, there was no difference in accessory connection conduction properties or patient age, weight, or energy delivered per subject between these three patients and our other 52 patients with left-sided pathways. This experience indicates that a delayed response may, in fact, herald permanent cure.

Successful interruption of an accessory connection by radiofrequency energy is characterized by an abrupt termination of conduction in the anomalous pathway. This result is predicated upon the creation of a lesion of desiccation necrosis at the catheter tip site, destroying myocardium at the ventricular or atrial side of the accessory connection. This lesion, when delivered sufficiently close to the pathway, produces abrupt cessation of conduction in the anomalous pathway, usually within 3–4 seconds in the permanently successfully ablated pathway; in pathways that are only transiently ablated and in which conduction returns within seconds to minutes, fine adjustment of the catheter electrode tip may lead to suc-

cessful ablation, presumably because of a closer proximity to the pathway.

The lesion created is dependent on a number of factors, including proximity to the target pathway, contact of the electrode to the myocardial tissue, the temperature achieved in the electrode tip, duration of energy delivery and surface area of the electrode tip.[7–9] Of those several factors, only electrode tip size and duration of energy delivery can currently be precisely controlled. Using a 4-mm long electrode tip, lesions 4 × 5 mm in dimension, as well as 2–3 mm deep are created; these lesions take the form of craters with a coagulum in the center and a donut shaped circle of necrotic myocardium, sharply demarcated from healthy tissue. This sharp demarcation and small lesion size account, in large part, for the abrupt cessation of conduction in the anomalous pathway when the electrode tip is placed sufficiently adjacent to the pathway. When the ablating electrode tip is placed adjacent but not close enough to the area of the anomalous pathway, only transient interruption of conduction may occur. However, recent evidence suggests that the lesion created extends beyond the ablated necrotic area and involves the micro circulation of the tissue immediately adjacent to the necrotic zone, potentially extending the lesion size.[10] Such extension of the primary necrotic lesion may account for the delayed effect of radiofrequency energy interrupting conduction in anomalous pathways. Based on the disappearance of accessory connection conduction within 4 hours in patient 1 and within 12 hours in patient 2, a delayed successful response, if it is to occur, will do so within several days.

Conclusion

This experience underscores the possibility of delayed interruption of conduction in accessory connections by radiofrequency energy and is compatible with extension of the radiofrequency lesion over time by involvement of the micro circulation. However, the time course described by 2 of the 3 patients suggests that delayed interruption in anomalous pathways is unlikely to occur after the first week following unsuccessful radiofrequency ablation.

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