Termination of Spontaneous Tachycardia In a Patient with WPW Syndrome

To the Editor:

Multiple mechanisms of spontaneous termination of supraventricular tachycardia (SVT) in patients with Wolff-Parkinson-White (WPW) syndrome have been reported. The association of dual AV nodal pathways in patients with the WPW syndrome has been well documented, and in two reports, dual AV nodal pathway was the mechanism for spontaneous termination of AV reentrant SVT (in patients with manifest WPW). We present a similar case, however, with a different pattern of termination. To our knowledge, this mechanism of spontaneous SVT termination has not been described previously.

The patient is a 35-year-old man with idiopathic dilated cardiomyopathy and the WPW syndrome. He was placed on therapy with atenolol (400 mg/day) because of recurrent orthodromic SVT utilizing a left lateral accessory pathway for retrograde conduction. He did well for several months until palpitations lasting only minutes recurred. A quadrupolar electrode catheter was percutaneously advanced to the high right atrium and, using programmed atrial extra-stimulation, multiple episodes of nonsustained SVT were induced which replicated the patient's symptoms.

Figure 1 shows the reproducible mechanism of spontaneous termination of SVT. The first five beats of SVT show a regular R-R interval (cycle length of 320 ms). Retrograde conduction to the atria is over a free wall accessory pathway (significant increase in VA interval with ipsilateral bundle branch block) with retrograde P wave occurring after the QRS. The P wave following the fifth QRS has a shorter VA interval representing either conduction over a second accessory pathway or a premature atrial beat.

Thereafter, antegrade conduction to the ventricles shows a sudden jump in AV interval (presumably AH interval) representing a jump from fast to slow AV nodal pathway. Retrograde conduction switches to the retrograde fast AV nodal pathway (now available for conduction) producing an echo beat with the P wave inside the QRS (typical echo beat of AV nodal reentry). The same pattern of conduction continues (antegrade slow and retrograde fast AV nodal reentry), however, with a further prolongation in conduction through the antegrade slow pathway until the next beat where it blocks antegradely (Wenckebach of the antegrade slow pathway) and terminates the SVT.

COMMENTS

Two previous reports have demonstrated dual AV nodal pathways as the causative mechanism of spontaneous SVT termination in the WPW syndrome. Westveer et al reported a patient with WPW and non-sustained antidromic SVT, where SVT termination was related to retrograde dual AV nodal pathways. Retrograde conduction of the SVT occurred over the slow retrograde AV nodal pathway with concealment into the retrograde fast pathway. Once retrograde concealed penetration of the fast pathway disappeared, the fast pathway became capable of antegrade conduction and a reentrant AV nodal ventricular echo occurred terminating the SVT. Adams et al reported a patient with WPW and orthodromic SVT. The SVT utilized the accessory pathway for retrograde conduction and a slow AV nodal pathway for antegrade conduction. In this case, spontaneous SVT termination was related to a sudden jump in antegrade conduction from the slow to the fast pathway, advancing the SVT so that block occurred in the accessory pathway or atrium. In our case, spontaneous termination of SVT was related to a sudden jump from antegrade fast to slow pathway. The conduction through the slow pathway was slow enough to allow recovery of excitability of the fast pathway which became available for retrograde conduction (closing the AV nodal circuit) and preempted the conduction over the left accessory pathway (most probably for anatomic reasons). The SVT finally terminated when Wenckebach block occurred in the slow pathway.

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REFERENCES


Figure 1. Example of spontaneous termination of supraventricular tachycardia. See text for explanation. A = mid right atrial electrogram.