

# Supernormal conduction and linking in an accessory AV pathway

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Received: 19 April 2009 / Accepted: 18 June 2009  
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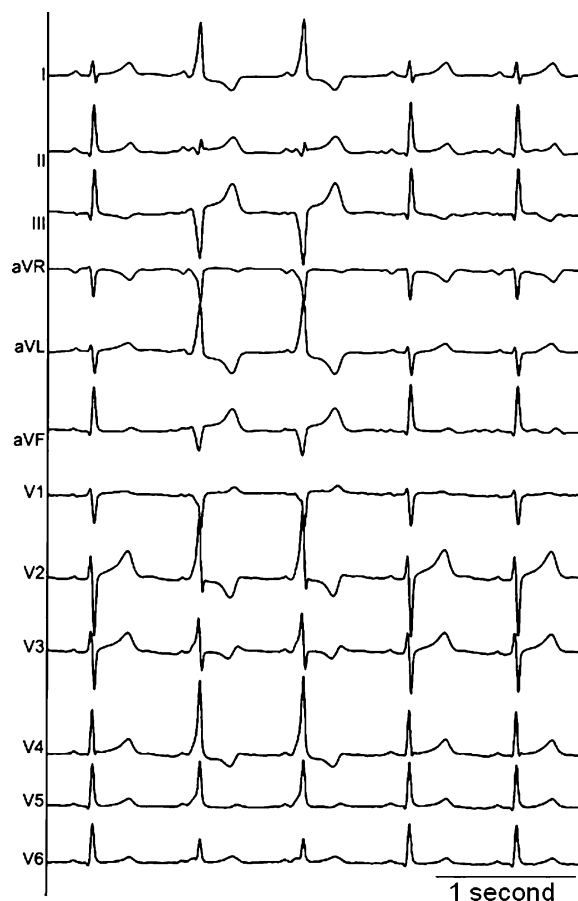
## 1 Introduction

Spontaneous intermittent block over an accessory pathway manifested as alternating QRS complexes of pre-excited and normally conducted beats suggests a prolonged antegrade refractory period [1]. Similarly, sudden loss of the delta wave during exercise [2] is also considered evidence of prolonged refractoriness over an accessory A–V pathway. However, the antegrade refractory period of an accessory A–V pathway is not the only determinant of conduction over bypass tracts. Two other important but frequently overlooked electrophysiologic mechanisms modulate conduction over accessory A–V pathways: supernormal conduction [3] and linking [4] between the normal conduction system and the accessory A–V pathway.

We present a patient that, despite intermittent preexcitation at rest and loss of preexcitation during exercise, had an accessory A–V pathway able to conduct at fast rates as demonstrated during the electrophysiology study. Both supernormal conduction and the linking phenomenon accounted for this unexpected behavior.

## 2 Case report

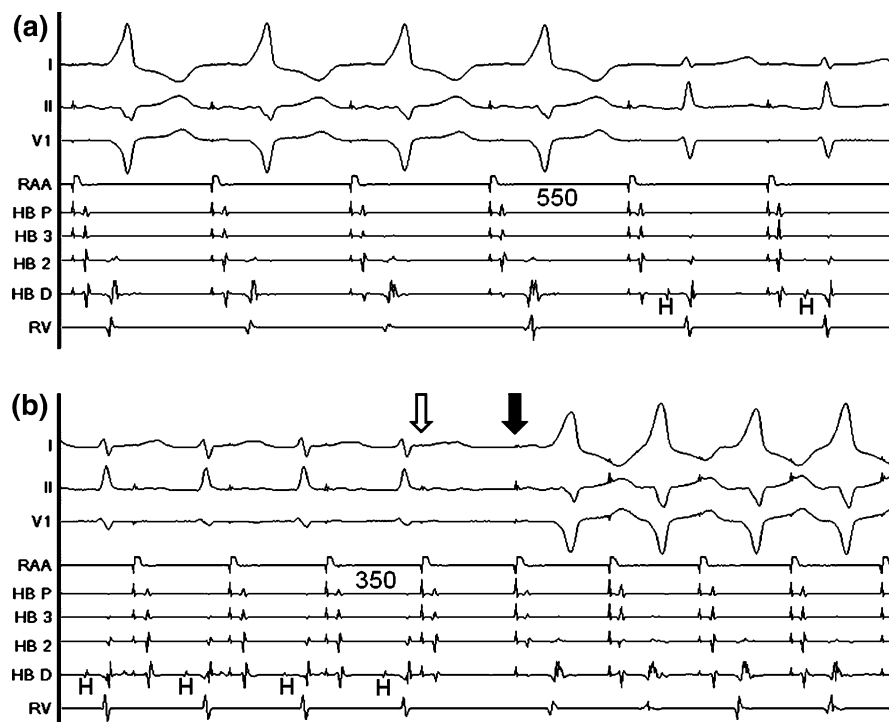
An 18-year-old asymptomatic male with a structurally normal heart was found to have intermittent ventricular



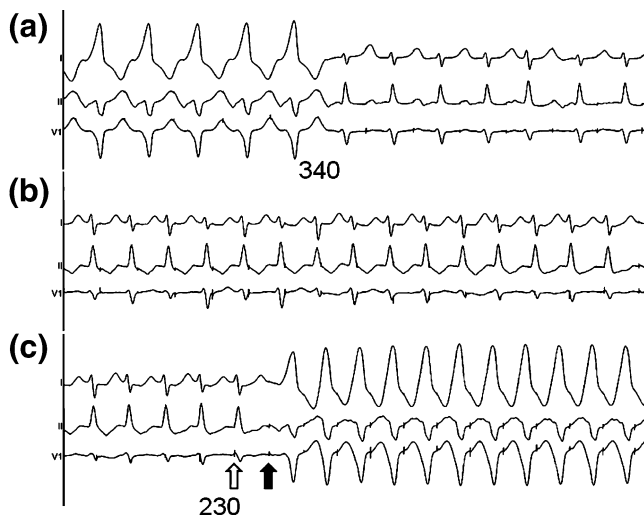
**Fig. 1** Spontaneous sinus rhythm with a cycle length of 775 ms is associated with intermittent ventricular preexcitation consistent with a right inferior or infero-paraseptal accessory A–V pathway

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**Fig. 2** (A) Decremental atrial stimulation results in antegrade block over the accessory A–V pathway at a cycle length of 550 ms. One to one antegrade conduction over the AV node was maintained with cycle length between 550 and 360 ms (B). At a cycle length of 350 ms, antegrade block in the AV node is followed by resumption of antegrade conduction over the accessory A–V pathway at cycle length between 350 and 320 ms



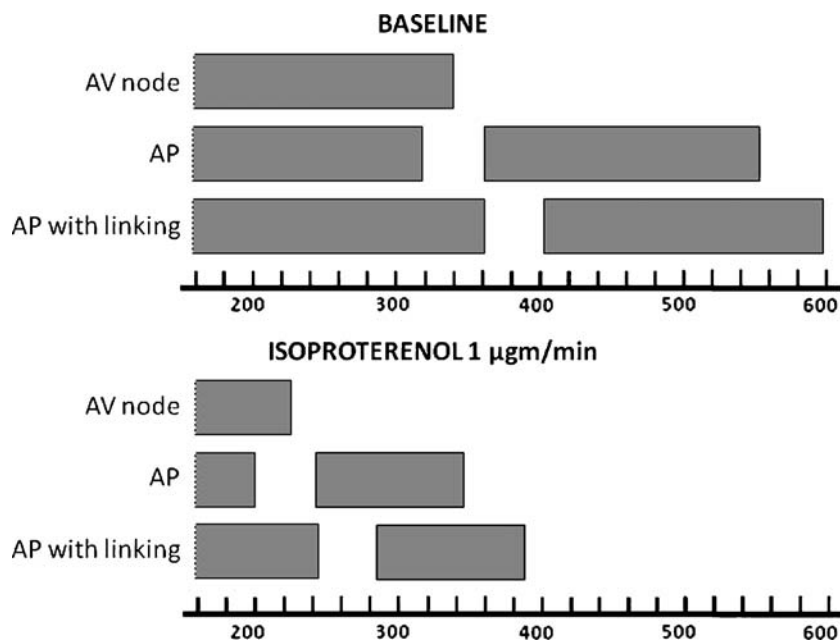
preexcitation at rest (Fig. 1) and sudden loss of preexcitation during exercise. Despite these findings, an electrophysiology study was performed because of his participation in competitive sports and his desire to become



**Fig. 3** (A) During administration of isoproterenol 1  $\mu\text{g}/\text{min}$ , decremental atrial stimulation results in antegrade block over the accessory A–V pathway at a cycle length of 340 ms. One to one antegrade conduction over the AV node was maintained with cycle length between 340 and 230 ms (B and C). At a cycle length of 230 ms, antegrade block in the AV node is followed by resumption of antegrade conduction over the accessory A–V pathway at cycle length between 230 and 200 ms

a pilot. The procedure was performed with the patient in the fasting state under sedation with intravenous propofol. Multipolar electrode catheters (Cordis Webster, Diamond Bar, CA) were introduced percutaneously and positioned in the right atrial appendage, right ventricle, His bundle region, and coronary sinus. To record His bundle activation, a 7-French deflectable catheter with four closely spaced pairs of electrodes was used. A deflectable catheter with eight pairs of electrodes (5 mm between pairs) was advanced into the coronary sinus, with the most proximal pair of electrodes positioned at the coronary sinus ostium. Bipolar electrograms (30 to 500 Hz) and unipolar electrograms (0.5 to 500 Hz) were displayed and stored using a digital recording system (Bard Electrophysiology, Lowell, MA). During decremental atrial stimulation, antegrade block over the accessory pathway occurred at a cycle length of 550 ms (Fig. 2(A)) and was maintained at progressively shorter cycle lengths until the refractory period of the AV node was reached at a cycle length of 350 ms. Following block over the AV node (Fig. 2(B), open arrow), the next paced atrial complex (filled arrow) unexpectedly conducted antegradely through the accessory A–V pathway between pacing cycle lengths of 350 and 320 ms. During administration of isoproterenol 1  $\mu\text{g}/\text{min}$ , the same phenomenon was repeated although at shorter cycle lengths. Initial loss of AP conduction occurred at a cycle length of 330 ms (Fig. 3), with resumption of conduction with cycle length between 230 and 200 ms. Ventricular stimulation with intermittent

**Fig. 4** Diagram represents the refractory periods of the AV node, the accessory A–V pathway (AP), and the accessory A–V pathway during linking (AP with linking). The supernormal phase of conduction is shown hidden inside the refractory period and represented as a gap in the horizontal bars. Values are given in milliseconds during the baseline state and during isoproterenol administration. Note the displacement to the right of the refractory period and supernormal phase of conduction during the linking



His bundle capture and differential ventricular stimulation before and during isoproterenol administration (1 to 2 µg/min) showed absence of retrograde conduction over an accessory A–V pathway. Atrial and ventricular stimulation did not induce an arrhythmia. Due to the presence of supernormal conduction over the accessory pathway resulting in rapid conduction, ablation was performed. Mapping of the tricuspid annulus and the coronary sinus identified earliest ventricular activation on the anterior wall of the coronary sinus approximately 0.5 cm from the ostium. One application of radiofrequency current rapidly (2.5 s) eliminated conduction over the accessory A–V pathway.

### 3 Discussion

Antegrade conduction over accessory AV pathways is considered the main determinant of the risk of sudden cardiac death [2]. This risk can be estimated by noninvasive methods including the resting ECG and the behavior of ventricular preexcitation during exercise. A minimally invasive procedure like esophageal pacing can be used to directly measure the antegrade refractory period of an accessory A–V pathway, induce reentrant tachycardias, and assess the effect of antiarrhythmic agents [5]. However, a complete electrophysiologic study although invasive, is the most reliable method to assess antegrade and retrograde conduction over an AP. There is a close correlation between the antegrade effective refractory period of an accessory A–V pathway and the shorter cycle length associated with 1:1 antegrade conduction and the shortest preexcited RR interval [6]. However, this information does not take into account the possible presence of supernormal conduction [7, 8] or the

modulation of antegrade conduction over the accessory A–V pathway by the normal conduction system [4].

As observed in the present case, if further testing is not performed once the refractory period is reached, the presence of supernormal conduction can easily be overlooked. True supernormal conduction (as opposed to the gap phenomenon) is defined as a conduction that occurs at a time when block is expected [9]. It can occur in the abnormal HIS-Purkinje system as well as in accessory pathways with prolonged refractory periods as observed in the present case.

Linking in an accessory A–V pathway occurs when antegrade conduction over the normal AV conduction system modifies the conduction properties of an accessory pathway through retrograde concealed activation [4]. This retrograde invasion of the pathway extended the duration of the antegrade refractory period and the range of supernormal conduction in the present case (Fig. 4). Sudden block over the AV node (Figs. 2, 3, and 4) eliminated this interaction resulting in conduction during the supernormal phase and antegrade conduction over the accessory pathway at rapid rates. This case emphasizes the need for a detailed electrophysiologic evaluation whenever the properties of the accessory A–V pathway need to be accurately assessed.

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