CASE REPORTS

Unmasking of left free wall ventricular preexcitation by His bundle ablation

A G R Visman, R N W Hauer, E O Robles de Medina

Abstract

His bundle ablation was performed in a 48 year old man with drug refractory orthodromic atrioventricular reentrant tachycardia and paroxysmal atrial fibrillation. Reentry was caused by a left free wall concealed accessory pathway. Interruption of the His bundle by low energy direct current shock (25 J) was quickly followed by anterograde conduction via the accessory pathway, with various forms of advanced block. The combined effect of unfavourable anterograde conduction (prolonged anterograde effective refractory period and decremental conduction), the left lateral location of the accessory pathway and intra-atrial conduction delay on one side, and normal conduction via the atrioventricular node and His bundle with retrograde concealed conduction into the accessory pathway on the other side resulted in masking of the anterograde conduction capability of the anomalous pathway. This observation does not seem to invalidate the choice of His bundle ablation in similar cases.

Case report

A 48 year old man was referred to our institute for catheter ablation of drug refractory orthodromic atrioventricular reentrant tachycardia and paroxysmal atrial flutter and atrial fibrillation. The patient had had symptoms for about eight years. Eight trials of antiarrhythmic drugs, alone or in combination, had failed or had had to be discontinued because of side effects. Physical examination and routine non-invasive laboratory tests were normal. The electrocardiogram at rest showed sinus rhythm with an intra-atrial conduction defect characterised by widened (120 ms) notched P waves, but no signs of ventricular preexcitation. The PR interval during sinus rhythm measured 160 ms with an AH interval of 75 ms and an HV interval of 55 ms in the His bundle electrogram. Several paroxysms of regular narrow QRS complex tachycardia and atrial flutter and fibrillation had been recorded. Holter recordings showed that atrial flutter and fibrillation were initiated by premature atrial complexes and not by deterioration of regular narrow QRS tachycardia. The diagnosis of an atrioventricular reentrant tachycardia through a left free wall accessory pathway was based on the following observations: (a) during tachycardia RP was shorter than PR (b) atrial activation was eccentric in a left caudal to cranial direction as shown by inverted P' waves in electrocardiographic leads I, II, and III and distal coronary sinus activation preceding proximal coronary sinus and low right atrial septal activation in the intracardiac electrograms, and (c) evidence on Holter recording of shortening of the tachycardia cycle length by 40 ms after the disappearance of left bundle branch aberrancy.2

Inability of the accessory pathway to conduct in an anterograde direction was postulated because there was no evidence of ventricular preexcitation in any of the available resting and ambulatory electrocardiograms recorded in the eight years of follow up.
or during paroxysms of atrial fibrillation with RR intervals ranging from 300 to 1000 ms. However, the effects of carotid sinus massage and isoprenaline (isoproterenol) on anterograde accessory pathway conduction had not been evaluated. During the pre-ablation electrophysiological study, including decremental atrial pacing and the atrial extrastimulus method, no preexcitation was observed.

A single low energy direct current shock (25 J) was administered through a special low energy ablation catheter (4 mm distal electrode) that was positioned in the proximal His bundle area according to the electrophysiological criteria and technique reported by Gallagher et al. Persistent complete interruption of the His bundle was achieved and this was shortly followed by anterograde conduction over the accessory pathway. The characteristics of the surface electrocardiogram were consistent with maximal preexcitation of the ventricles via a left lateral bypass (monophasic slurred positive QRS complexes in lead V1 with marked right axis deviation) (fig A). The P-δ interval of the conducted P waves measured 160 ms. At a P width of 120 ms, this indicates delayed conduction over the accessory pathway. One to one anterograde conduction was later followed by 2:1 conduction over the accessory pathway depending on the duration of the RP interval. The shortest RP interval at which conduction was observed was 520 ms, corresponding to a sinus cycle length of 680 ms. During longer periods of 2:1 preexcitation the conduction pattern was further complicated by sudden failure of anterograde conduction. This was preceded by a 40 ms increment in the P-δ interval of the conducted P waves (fig B), indicating a superimposed type I block in the accessory pathway. The resultant pauses in the ventricular response were terminated by narrow His bundle escapes (R') and an escape rhythm resulting in atrioventricular dissociation (fig B and C).

Such complex patterns with apparent unexpected failure of preexcitation are best explained by assuming two levels of block in the accessory pathway: (a) a proximal level of block which is governed by a considerably prolonged effective refractory period, in which all P waves occurring at an RP interval

![Figure](image-url)
of 520 ms or less are blocked. P waves occurring outside this interval may excite the ventricles or undergo decremental conduction and be blocked at a more distal level in the accessory pathway (attempted 2:1 preexcitation). Concealed anterograde conduction into the distal level of block will tend to maintain the basic 2:1 conduction pattern across the proximal region, thus allowing for a sufficiently long pause in the ventricular excitation pattern to enable a His bundle escape. (b) Once a His bundle escape occurs the conduction pattern is further complicated by concealed retrograde conduction into the accessory pathway. This may cause perpetuation of anterograde block and maintenance of atrioventricular dissociation (fig C).

Whereas anterograde conduction over the accessory pathway was unstable and could already be completely blocked by intravenous (5 mg) ajmaline,4 retrograde conduction of His bundle escapes over the anomalous connection were preserved and occurred whenever the PR' interval was 360 ms or more (fig D).

Because accessory pathway conduction remained unstable, a backup VVIR pacemaker was implanted. During a follow up of 24 months the patient remained symptom free.

Discussion
In this patient two criteria determined that His bundle ablation rather than accessory pathway ablation would be used. First, Holter recording showed that paroxysms of atrial flutter and atrial fibrillation were triggered by atrial premature complexes and did not result from deterioration of orthodromic circus movement tachycardia. Second, the accessory pathway was assumed to be incapable of anterograde conduction. Ablation of the accessory pathway would only cure the atrioventricular tachycardia, but would not protect the ventricles against rapid ventricular responses during atrial fibrillation with conduction over the normal pathway. In contrast, His bundle ablation was expected to achieve both goals by creating a complete atrioventricular block. Much to our surprise, interruption of the His bundle was followed by 1:1 preexcitation and various forms of advanced block in the accessory pathway.

This observation accords with reports by Przybyslaki et al14 and Critelli et al15 that concealment of an accessory pathway does not necessarily indicate that anterograde conduction is not possible. Several mechanisms have been proposed to explain the presence of unidirectional anterograde block in an anomalous atrioventricular connection.1-3, 11

In the present case, the absence of preexcitation before His bundle ablation 68:221-31 can be explained by a combination of mechanisms. At relatively rapid sinu rates (cycle lengths <680 ms) the considerably prolonged anterograde effective refractory period of the anomalous bundle prevented preexcitation. At lower rates conduction over the accessory pathway was unfavourably influenced by the left lateral location of the anomalous bundle, the observed intra-atrial conduction delay, and the prolonged anterograde conduction time (P-Delta interval of 160 ms) with decremental characteristics. In contrast, conduction over the atrioventricular node-His bundle axis was unimpaired as shown by the relatively short AH interval and normal HV interval during sinus rhythm. The combined effect must have favoured conduction via the normal pathway with concealed retrograde activation of the anomalous bundle by the sinus impulse. These effects were abolished by His bundle ablation, leading to unmasking of the accessory pathway at relatively long diastolic intervals.

Since anterograde conduction via a previously concealed accessory pathway may obviate the need for backup pacing after His bundle ablation.4 Since the conduction capability of concealed bundles is probably limited4 the risk of rapid ventricular rates with conduction over the anomalous pathway during atrial fibrillation is probably small or absent. Thus, as in the present case, ablation of the His bundle seems to be an acceptable treatment for drug refractory symptomatic atrial and atrioventricular reentrant arrhythmias. In conclusion, we covered the unstable anterograde conduction characteristics of the accessory pathway. We chose VVIR pacing rather than DDD pacing because of the occurrence of paroxysmal atrial fibrillation, apparently unrelated to the atrioventricular reentrant tachycardias. In doing so, we accepted the potential disadvantage of VVI pacing with persistent retrograde atrial activation via the accessory pathway on haemodynamic function.

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