

Bachmann bundle block occurring during radiofrequency ablation at the inter-atrial septum

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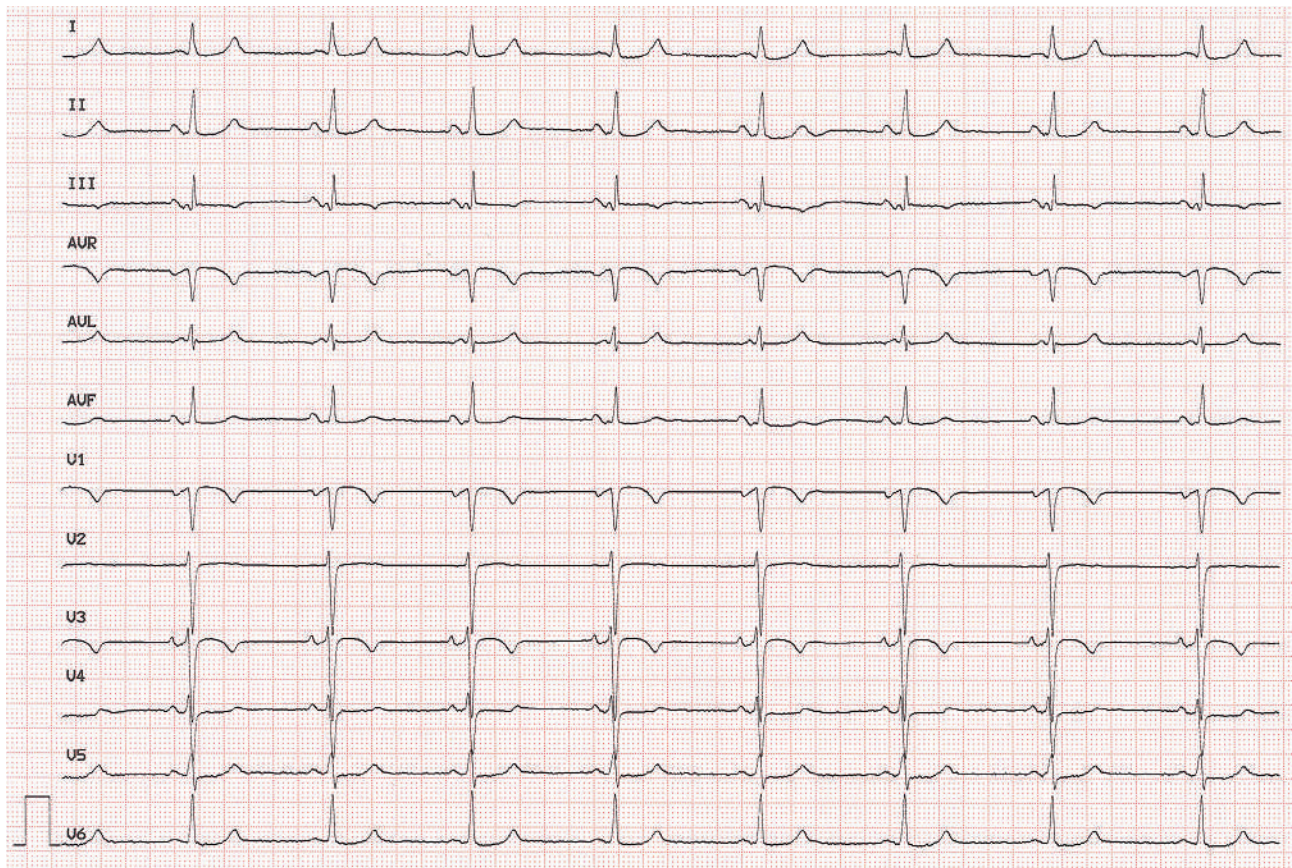
Case presentation

A 69-year-old woman was admitted for an elective radiofrequency ablation (RFA) of symptomatic paroxysmal atrial fibrillation, without concomitant structural heart disease except for mild left atrial dilatation. The baseline electrocardiogram (ECG) is shown in figure 1.

The catheter ablation was performed under general anaesthesia, with a right femoral endovascular access. After the transeptal puncture, complete isolation of the four pulmonary veins was performed including ablation of an atrial tachycardia/flutter originating from the left common ostium. During burst stimulation another atypical flutter was provoked with a critical

Figure 1

The baseline ECG at admission. Of note, the baseline prolonged P wave duration is evident in inferior leads as well as a small terminal negative deflection in lead III which reflects prolongation of inter-atrial conduction time.



Funding / potential competing interests:

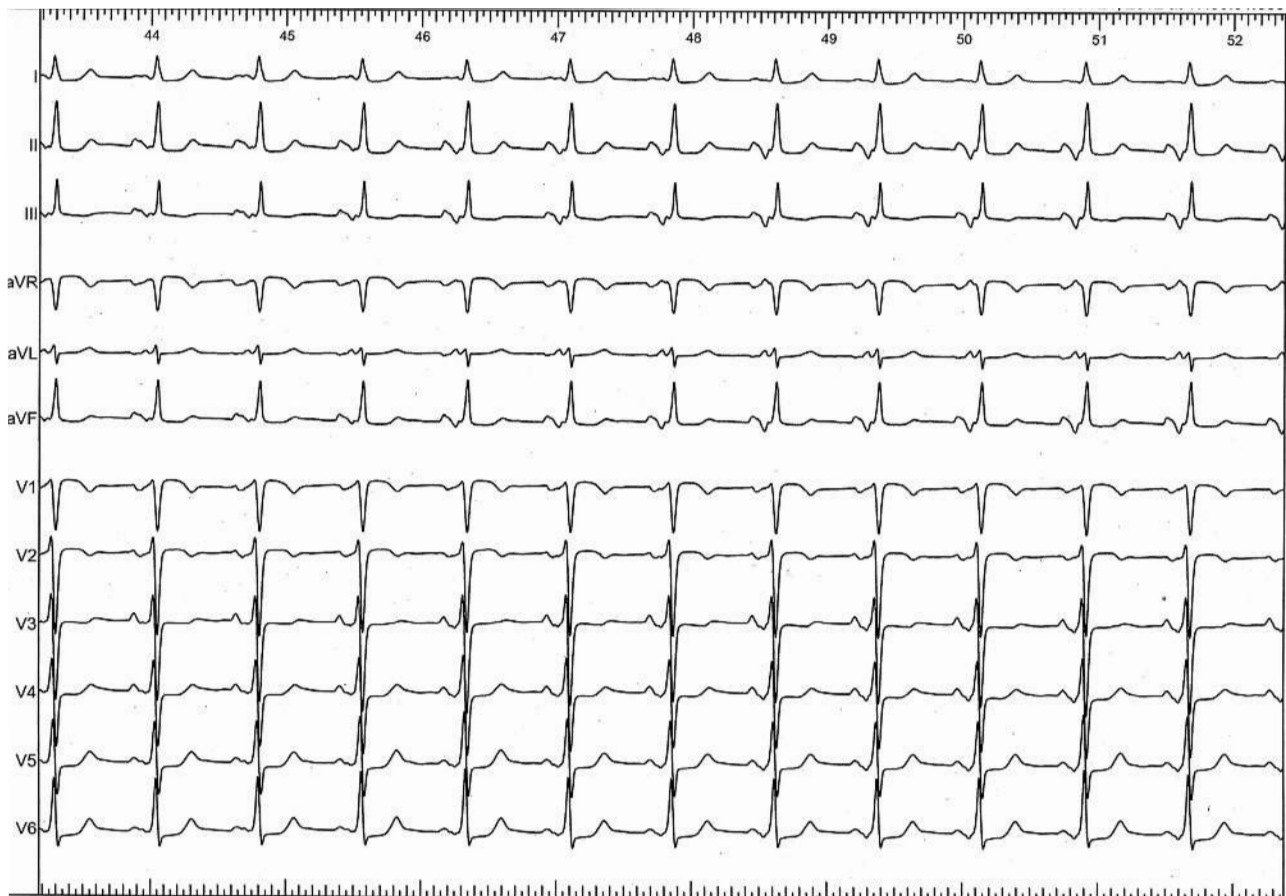
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Figure 2

ECG was recorded during RFA on the left inter-atrial septum and shows the progressive generation of biphasic, prolonged P waves in inferior leads and lateral leads starting from the fourth complex; a BB block that will likely be permanent in our patient.



isthmus localised on the left side of the high inter-atrial septum by entrainment mapping. Two radiofrequency ablation lesions were performed at this point during sinus rhythm. The continuous electrocardiogram eight seconds after the start of the first ablation lesion is shown in figure 2.

Questions

1. What is the diagnosis?
2. What is the mechanism of the modification?
3. What are the implications of the ECG changes?

Commentary

Radiofrequency ablation (RFA) of atrial fibrillation (AF) implies a transeptal left atrial access with creation of thermal lesions around the four pulmonary veins in the left atrium to interrupt the mechanism of transmission of the arrhythmia.

If the tachyarrhythmia circuit or the critical isthmus of a re-entry circuit is localised to the left side of the inter-atrial septum, as was the case with this pa-

tient, it may be necessary to deliver RF lesions in this particular anatomical zone. Lesions in the inter-atrial septum may, as in this case, create damage to the major impulse-conducting pathways of inter-atrial conduction.

Although there is no histopathologic evidence of specialised conduction tracts, three zones of preferentially rapid conduction across the human inter-atrial septum have been described [1]. The upper zone corresponds to the location of the Bachmann's bundle (BB), the middle is localised to the fossa ovalis and the inferior zone corresponds to the ostium of the coronary sinus and connections of the proximal coronary sinus.

The BB, first described by Bachmann in 1916 [2], is thought to be responsible for the rapid conduction of impulses from the right atrium (RA) to the LA during sinus rhythm, synchronising atrial activation. It emerges from the anterior-superior-septal aspect of the right atrium to form a trapezoid-shaped bundle of parallel fibres, courses along the superior quadrant of the inter-atrial sulcus from the RA to the LA, traverses the curvature of the atrial wall across the inter-atrial septal roof and then courses leftward and bifurcates to

encircle the neck of the left atrial appendage. Most frequently, the fibres of this inter-atrial muscular bridge are separate in orientation and perpendicular to the transverse atrial myocardial fibres.

Typically, the median bundle measurements are 4 mm in thickness and 9 mm in height with upper and lower bundle lengths of 10 mm and 3 mm, respectively.

In our patient, the baseline prolonged P wave duration (160 ms) with notching (see lead I) as well as a small terminal negative deflection in lead III reflects prolongation of inter-atrial conduction time with minor changes in the terminal P wave vector. This correlates well with major change in the second half of the P wave produced by a localised RFA lesion in the region of the BB's insertion. This lesion probably completely blocked or severely prolonged conduction through the superior inter-atrial septum (predominantly through BB) forcing inter-atrial conduction to proceed during sinus rhythm via the lower inter-atrial connection and producing an upward vector best seen in the inferior leads as a negative deflection. This upward vector indicates depolarisation of the left atrium from below in an upwards direction during sinus rhythm (fig. 2).

Normal duration of the P wave is <110 ms and P waves in BB conduction block are often ≥ 110 ms, and in our case 160 ms at baseline which prolonged to 180 ms after BB block. Figure 2 was recorded during RFA on the left inter-atrial septum and show the progressive generation of biphasic, prolonged P waves in II, III and aVF leads and in V_3 to V_6 leads starting from the fourth complex, a BB block that will likely be permanent in our patient.

The IAB is now believed to be precursor for atrial tachyarrhythmias and LA electromechanical dysfunction, and may be an important mechanism predisposing to, and even maintaining, AF [3].

References

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