

## Exercise-induced right bundle branch block progressive resolution

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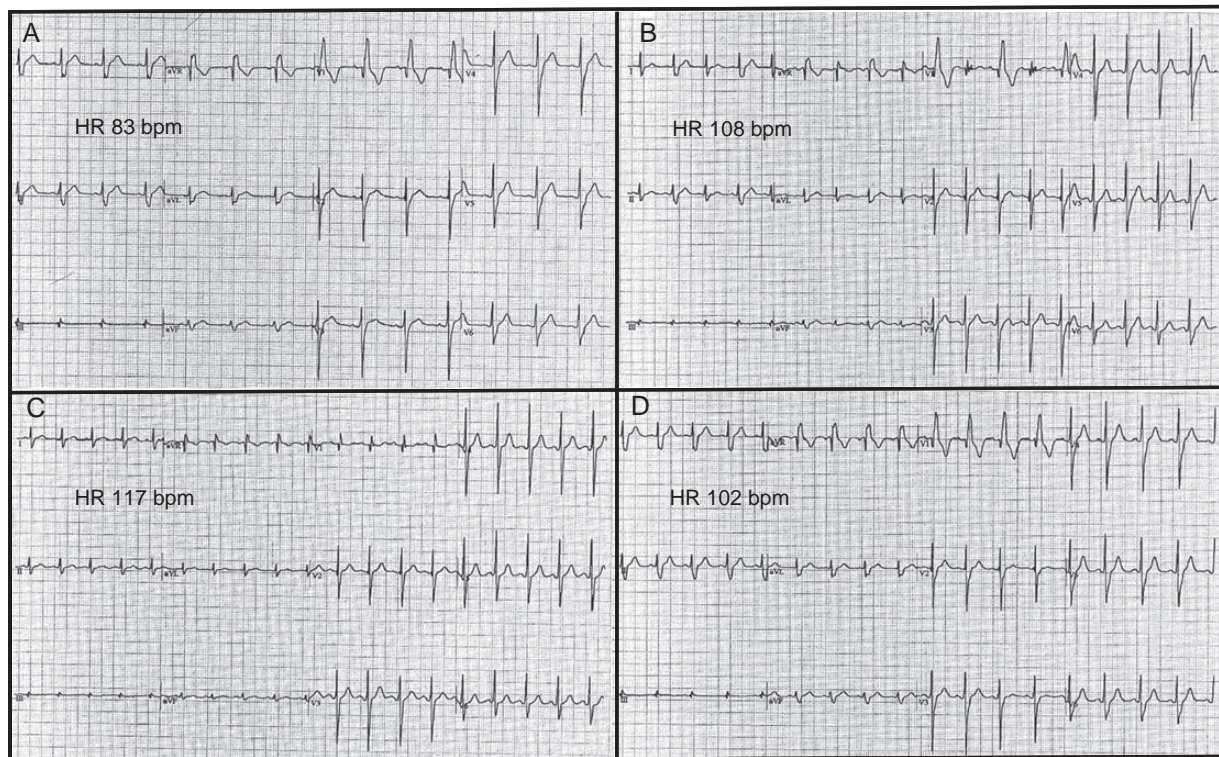
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A 67-year-old male was presented to the consultant with atypical chest pain. A stress test was performed. A persistent right bundle branch block progressively disappeared during exercise. What are the possible mechanisms?

### Case presentation

A 67-year-old male was presented to the consultant with atypical chest pain. Physical examination was unremarkable. He was not taking any medications. As part of his work up, a stress test was performed. *Figure 1A* shows a 12-lead electrocardiogram (ECG) immediately before the test, depicting a normal sinus rhythm at 83 b.p.m. and right bundle branch block (RBBB). *Figure 1B* shows the heart rate increasing to 108 b.p.m. and a brief period of RBBB in a 2 : 1 fashion. Panel C shows a maximum heart rate at 117 b.p.m. with almost complete resolution of RBBB (a minimum defect in the right bundle branch still persists). *Figure 1D* shows the immediate post-exercise period with a heart rate of 102 b.p.m. and reappearance of RBBB.



**Figure 1** Treadmill stress test (Modified Bruce protocol) at rest (A), stage 3 (B), stage 4 (C) and recovery period (D).

### What are the possible mechanisms?

First, one should speculate that during exercise a rapid and progressive increment of plasmatic catecholamines occur, leading to a progressive shortening of the right bundle branch refractory period facilitating 2 : 1 conduction first, and then complete normalization of conduction through the right bundle branch at maximum heart rate. Secondly, possible supernormality of conduction (SNC) over the right bundle branch may be occurring, given that the PR interval remains stable. This is very difficult to prove without a complete screening of the diastolic period at different heart rates.<sup>1</sup> One may need to apply different manoeuvres to increase but mostly to decrease the heart rate to check the behaviour of conduction over the right bundle branch evaluating the response of the QRS width at different rates. Even when 1 : 1 conduction was seen for a short period time, persistence of SNC for several beats is unusual. Thirdly, Phase 4 RBBB needs to be ruled out. Several previous ECGs at lower heart rates over the prior years depicted RBBB identical to the one observed during the stress test, making this diagnosis very unlikely. Finally, two other possible explanations include a frequency-dependent delay in the proximal atrioventricular

conduction or a frequency-dependent delay in the left bundle branch leading to a QRS complex narrowing. However, in these two situations one would expect a prolongation in the PR interval. Detailed analysis of ECG recordings during the stress test helps the physician in speculating intrinsic complex electrophysiological mechanisms; however, this case is a clear example of the difficulties to determine the exact electrophysiological mechanism underlying an intermittent intraventricular block when a complete scanning of the cardiac cycle is not available, including the characteristics of impulse propagation at lower heart rates.<sup>2,3</sup>

Given the lack of symptoms and no evidence of ischaemia, no further action was required.

**Conflict of interest:** none declared.

## References

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