The case is reported of an athlete who, during an exercise test, had a complete atrioventricular block without symptoms of cardiac output failure. Heart rate, stroke index, cardiac index, and myocardial contractility were monitored during the exercise by an impedance cardiograph. The most important findings of this report are the continuous increase in stroke index, which compensated for the lack of heart rate response, and the normal cardiac index values achieved during the exercise. This stroke index response was mainly due to an increase in myocardial contractility.

A 19 year old footballer (weight 65 kg; height 1.78 m) presented to our sports clinic for a medical examination. A physical examination showed no medical problems. The electrocardiogram at rest was normal, except for a sinus bradycardia. The subject then performed a progressive cycle ergometer test with incremental loads of 20 W/min, starting from 20 W, until exhaustion. We noted that during the exercise, the electrocardiogram (ECG) PR interval did not decrease compared with values at rest. This led to a complete atrioventricular block (CAV block), which occurred at the 80 W load and lasted until the first minute of recovery (fig 1A). During the entire period, the athlete showed no symptoms of an abrupt fall in blood pressure, so he was not stopped until exhaustion, which was reached at a load of 160 W. He was then informed that admission to hospital was advisable so that his heart function could be evaluated. He refused, and to date we do not know what caused the CAV block. The subject was also informed that his case would be submitted for publication and he agreed by signing a form.

During the cycle ergometer test, the subject was connected to an ECG, a sphygmomanometer, and an impedance cardiograph (NCCOM 3, BoMed Inc, Irvine, California, USA) for cardiodynamic evaluation.19 We show mean values for cardiodynamic variables during rest, exercise (shown as functions of the percentage of the maximum workload achieved), and five minutes of recovery. We also show, as a reference, the values (mean (95% of the confidence interval)) for seven normal controls (mean (SD) age 22 (2.6) years, height 176 (3.8) cm, weight 69 (4.6) kg) engaged in the same sport, who performed the same test.

Haemodynamic changes during exercise
In fig 1B, it can be seen that the heart rate of the subject was lower at rest and during exercise compared with the controls. Furthermore, whereas the heart rate of the controls increased linearly with respect to workload, that of the subject reached a plateau at a workload corresponding to 60% of the maximum—that is, after the CAV block had occurred—and increased no further. Figure 1C shows that the stroke index of the controls increased abruptly at a workload corresponding to 20% of the maximum achieved, then tended to reach a plateau. In contrast, the stroke index of the subject had a tendency to increase throughout the exercise, especially after the CAV block. This resulted in a stroke index for the subject that was about twice that of the reference group during 100% of maximum workload. In fig 1D it is evident that the cardiac index of the patient, despite the low heart rate and thanks to the increase in systolic index, was maintained within, or close to, the lower limit of 95% of the confidence interval throughout the exercise. Mean arterial pressure (fig 1E) started at a very similar level, but, after the CAV block, it tended to decrease in the subject, while there was a slight increase in the controls. Figure 1F shows the trend of the pre-ejection period/left ventricular ejection time ratio, which is related to myocardial contractility. It is evident that the myocardial contractility of the subject increased more sharply and reached higher values than that of the controls, especially after the CAV block.

DISCUSSION
Exercise induced paroxysmal CAV block is uncommon, even in patients with stable conduction disturbances.17,18 Haemodynamic responses to exercise induced cardiac rhythm disturbances in cardiac rhythm are of interest to physiologists and cardiologists because, despite relative bradycardia and if the venous function is normal, some compensatory changes enable these patients to maintain, at least in part, a satisfactory cardiac output. In 1994 Alexander et al17 found that the pressure response was maintained during one-leg static exercise in patients with a dual chamber pacing and sensing pacemaker placed for CAV block when the heart rate was stable at the resting rate. These authors stated that this response was caused by an increase in stroke volume and not by an increase in systemic vascular resistance. An increase in end diastolic volume and/or an increase in contractility may be the cause of this stroke volume increase. These findings were substantially confirmed by Nobrega et al.18

In our report, the cardiovascular response of the subject to exercise appears to be preserved. In fact, although bradycardia occurred after the CAV block, the cardiac index continued to increase in an almost linear way with respect to workloads, thanks to a continuous increase in stroke index. Moreover, mean arterial pressure values did not exhibit any abrupt falls. The continuous increase in stroke index was, in our opinion, the result of the sharper increase in myocardial contractility. As we could not obtain data on end diastolic volume, we were unable to see if the Frank-Starling mechanism was involved in this response.

In conclusion, this report shows that an increase in stroke volume may compensate for the lack of heart rate response and thereby maintain normal cardiac output during exercise in patients with CAV block.
An increase in stroke volume can compensate for the lack of heart rate response during exercise in patients with complete atrioventricular block. This stroke volume response is mainly due to an increase in myocardial contractility.

Take home message

REFERENCES