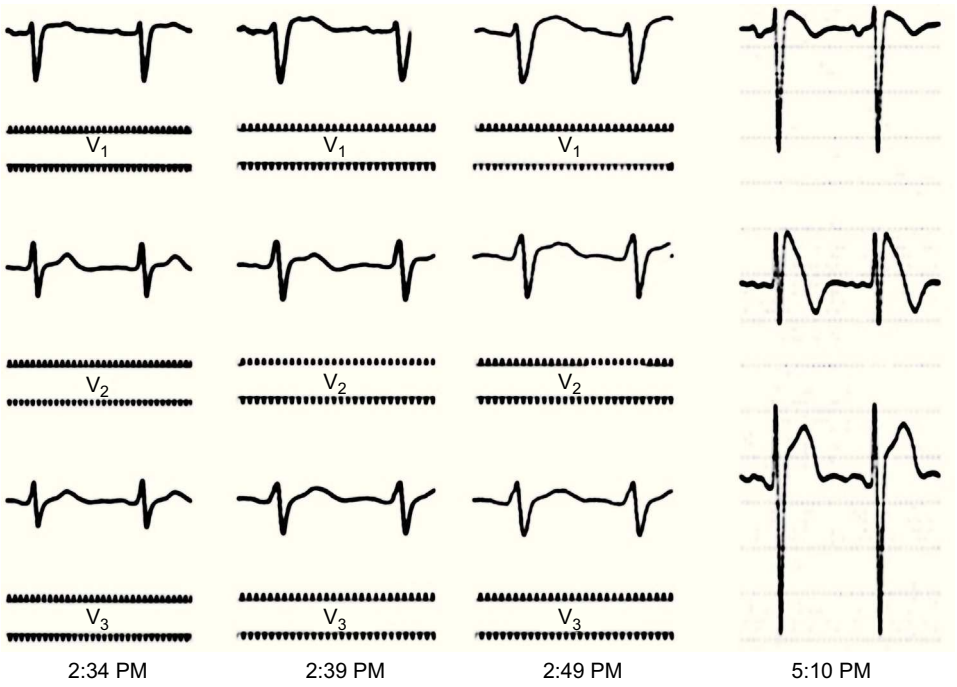

Figure. Right precordial leads (V_1-V_3) at 25 mm/s and 10 mV/mm. The tracings in the first 3 panels were obtained in the electrophysiology laboratory with an EP-WorkMate polygraph (St. Jude Medical). The fourth panel is a standard digital electrocardiogram obtained in the cardiology department.

currents at the end of phase 1 of the action potential that enables the unmasking of the B-ECG.

The case of a delayed positive response to flecainide reported here might indicate that results considered to be negative at the time of the challenge test may be false negatives, since the electrocardiographic monitoring time is usually 30 minutes or less.

Studies involving a larger number of patients should be carried out to examine whether the systematic prolongation of the monitoring period following a FT could contribute to reducing the number of false negatives.

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Infranodal Atrioventricular Block as a Possible Cause of Exercise-induced Cardiac Arrest

Bloqueo auriculoventricular infranodular como posible causa de parada cardiaca inducida por ejercicio

To the Editor,

Cardiac arrest that occurs during long-distance running races is most commonly attributed to hypertrophic cardiomyopathy or atherosclerotic coronary disease. Conduction system disease is an extremely rare cause of sudden death in athletes and the final cause remains unknown in a small proportion of cases.[1] We present a case of malignant bradyarrhythmia in a long distance runner.

A 46-year-old healthy man, with no previous cardiovascular history, had a cardiac arrest immediately after completing a half marathon. At the finish line, he fainted and was attended by the emergency medical services, which initiated advanced life support in the presence of severe bradycardia (23 bpm) and hypotension. A first electrocardiogram showed complete atrioventricular block (AVB) with slow wide QRS complex escape (Figure 1A). Intravenous atropine only increased sinus rate without improving the degree of AVB (Figure 1B) and therefore a transcutaneous

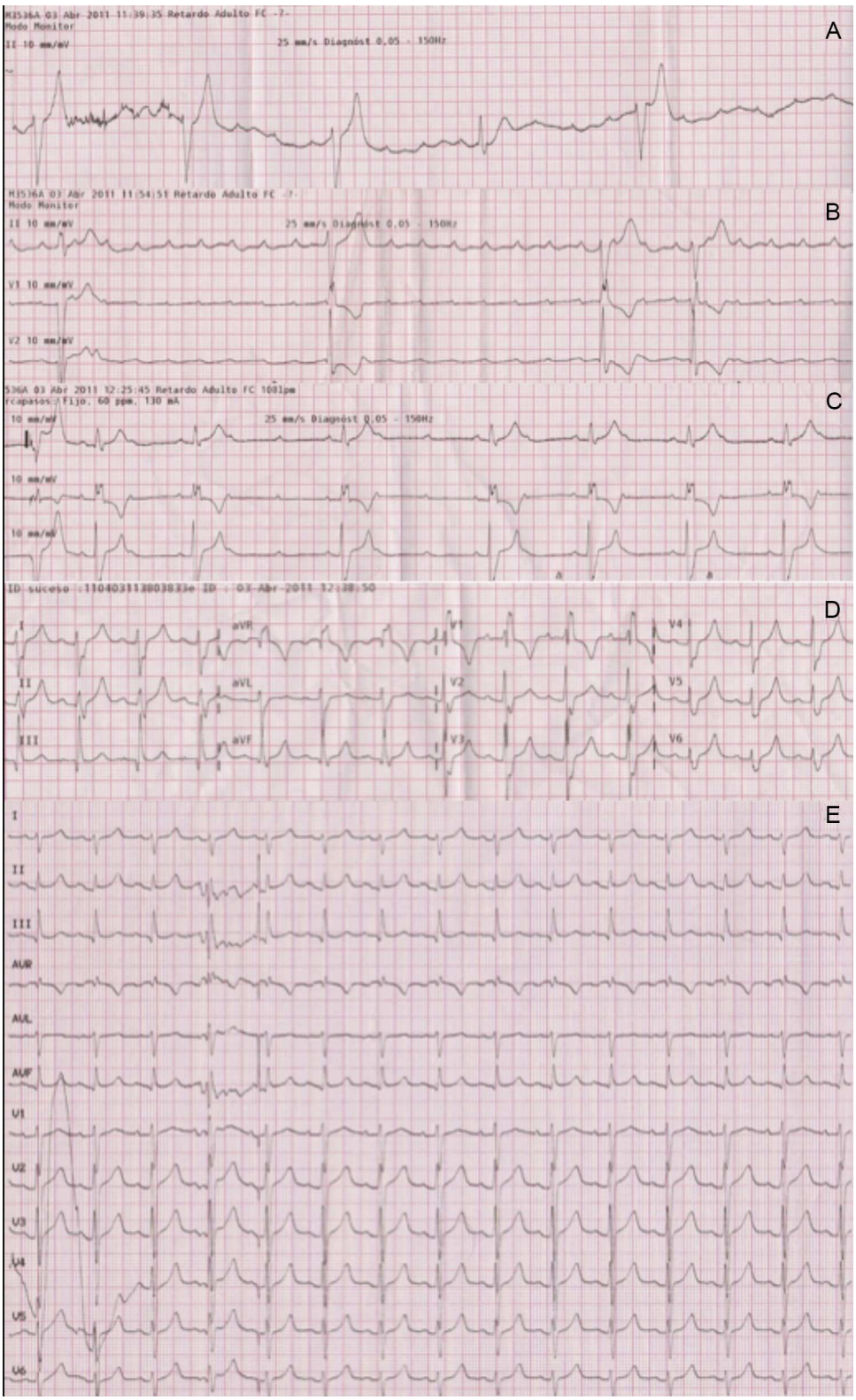

Figure 1. A: First electrocardiogram recorded following consciousness recovery showed complete atrioventricular block and wide QRS escape. B: Intravenous atropine increased sinus rate but did not improve atrioventricular conduction, strongly suggesting an infranodal location of the atrioventricular block. C: Partial atrioventricular conduction recovery 45 min after the cardiac arrest. D: Right bundle branch block was observed during the first few minutes following 1:1 atrioventricular conduction recovery. E: Electrocardiogram at hospital admission.

pacemaker was placed and the patient was transferred to our hospital. Forty-five minutes after cardiac arrest, atrioventricular (AV) conduction progressively recovered (Figure 1C), until 1:1 AV conduction resumed with first degree AVB and complete right bundle branch block (Figure 1D). At hospital admission, the electrocardiogram only showed a borderline prolongation of the PR interval (210 ms) (Figure 1E). Diagnostic work-up including blood analyses, echocardiogram, and coronary angiography ruled

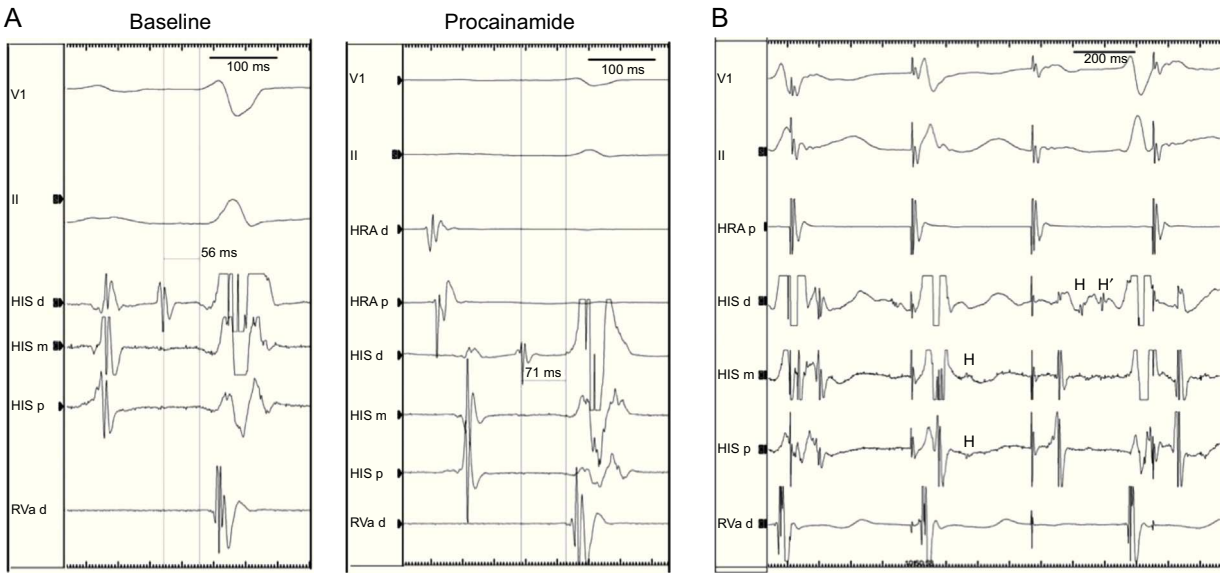

Figure 2. A: Electrophysiologic study performed 3 days after the index admission: slightly prolonged HV interval (56 ms) and notched His at baseline, which further prolonged following procainamide challenge. B: Atrial pacing 380 ms cycle length following procainamide infusion shows a proximal His bundle deflection (H) with intermittent failure to conduct to the distal His bundle (H') (second paced beat) and split His potentials (third paced beat).

out drug use, electrolyte disturbances, myocardial damage (maximum troponin T, 0.03 ng/mL), structural heart disease, and coronary lesions. An electrophysiologic study was performed documenting the presence of a bordeline HV interval (56 ms) and a notched wide His deflection (35 ms) (Figure 2A). After procainamide infusion (10 mg/kg), there was an HV interval prolongation up to 70 ms with further His widening (45 ms). Atrial pacing at progressively higher rates demonstrated the presence of intermittent conduction between the proximal (H) and the distal His bundle potentials (H') (Figure 2B).

With the diagnosis of second degree intra-His block unmasked by procainamide infusion,[2] a double chamber pacemaker was implanted. During the year following the episode, the patient continued running everyday free of symptoms. An exercise test performed 6 months after the episode showed a normal baseline electrocardiogram and the development of frequency-dependent right bundle branch block at a critical rate that resolved with sinus rate slowing. Genetic testing excluded mutations in the principal ion channel genes associated with progressive cardiac conduction disease (*SNC5A*, *KCNQ1*, *KCNH2*, *KCNE1*, and *KCNJ2*).

Hypertrophic cardiomyopathy and ischemic heart disease are the most common causes of cardiac arrest during long distance competitions. However, in a non-negligible proportion of cases (5%-10%), the ultimate cardiovascular cause of death remains unknown.[1] Exercise-induced complete AVB is an uncommon event and, to our knowledge, has not been previously reported as a cause of cardiac arrest during exercise.[1] Our case highlights the clinical importance of this phenomenon as a possible cause of sudden death in athletes. The prolonged duration of the AVB episode (>1 h) and the unstable and wide QRS complex escape suggest that, if the patient had not been immediately resuscitated, fatal asystole might have developed at any time during the episode.

Although exercise-induced ischemia in patients with coronary artery disease or coronary vasospasm can cause AVB, in other instances it may occur in the absence of ischemia in patients with an otherwise normal resting electrocardiogram.[3] In the latter group, infranodal location is the most common site of block.[2,3] The present case recapitulates the electrophysiological features of an intra-His conduction impairment unmasked by procainamide infusion and atrial pacing: prolonged intra-His duration, notched His deflection, and split potentials with intermittent conduction.[2] The development of rate-dependent right bundle branch block several months after the admission points to a progressive conduction disease etiology. However, the reasons for the development of these conduction disturbances in a previously healthy, middle-aged, well-trained patient remain unknown. Gene mutations have been implicated with the development of progressive cardiac conduction disease, and the number is ever-growing.[4] Cardiac sarcoidosis and giant cell myocarditis are responsible for some unexplained AVB in young adults,[5] but these possibilities seem unlikely in the absence of symptoms or myocardial damage. Recent magnetic resonance imaging studies have suggested a link between lifelong endurance exercise and myocardial fibrosis that could eventually have consequences on the conduction pathways[6] and could constitute an explanation for our findings.

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Correlation Between Agatston Scores Obtained by Cardiac Computed Tomography Studies With and Without Contrast in Asymptomatic Population

Correlación entre la puntuación de Agatston obtenida por tomografía computarizada cardiaca con y sin contraste en población asintomática

To the Editor,

The extent of calcification of the epicardial coronary arteries can be quantified using contrast-free coronary computed tomography angiography (CTA) by calculating the Agatston score (AS) to provide a measure of atherosclerotic load. Several studies have demonstrated that the AS is a strong predictor of cardiovascular events and adds prognostic information to the cardiovascular risk scales.[1] The AS has allowed individual cardiovascular risk to be refined, particularly in intermediate-risk patients. Recent guidelines on cardiovascular prevention recommend use of this scale. Currently, contrast-enhanced CTA during cardiac computed tomography can be used to measure the extent of coronary artery disease, whether or not associated with calcification, and up to 20% of noncalcified atherosclerotic lesions can be detected.[2] However, use of contrast enhancement in the study procedure hinders calculation of the AS which has traditionally been determined by contrast-free CTA. Several articles have validated different methodologies for obtaining this score during the same cardiac computed tomography procedure[3,4]; however, few of these studies used software currently available commercially. Recently, Otton et al[5] validated a new methodology that allows this score to be obtained during contrast-enhanced cardiac computed tomography with such software.

We present our experience with a study designed to assess the correlation of AS calculated from contrast-free and contrast-enhanced CTA by applying the above methodology. No such study has been published by other groups.

We retrospectively analyzed 50 individuals who underwent a voluntary medical check-up between December 2012 and September 2013. Tests included a contrast-free CTA to calculate the AS and a contrast-enhanced CTA. Patients were only included if some degree of coronary calcification was detected in both studies. No patient had to be excluded due to poor quality of the study (for example, excessive noise or insufficient coronary opacification). The mean (SD, standard deviation) age of the population studied was 58 (11) years and 86% were men. In addition, 47% were smokers, 45% had hypertension, 71% had dyslipidemia, 18% had diabetes, and their mean (SD) REGICOR score was 6.7% (3.7%). Both studies (with and without contrast) were performed consecutively with a Toshiba Aquilion One scanner and the results were analyzed on a Vitrea FX v3.1 workstation (Toshiba Medical Systems, Tokyo, Japan). The AS from contrast-free CTA was quantified using axial views and the VScore tool. Curved multiplanar reconstructions along the short axis (intravascular ultrasonography-like images) were used in contrast-enhanced CTA, with 75% acquired in the R-R interval and using the SurePlaque tool, which enables calculation of the total calcium volume (in mm^3) present in the coronary atherosclerotic plaques. According to the published methodology, in contrast-enhanced CTA the AS is derived from the product of a factor that represents the gradient of the linear regression fit of the AS obtained for both methodologies (3.13 HU/μL) and the total calcium volume, using a threshold of 320 HU to discriminate between noncalcified atheroma and contrast material.

In our study, the median AS was 66.5 (interquartile range, 233) in contrast-free CTA and 63.23 (interquartile range, 181) in contrast-enhanced CTA. In our linear regression model, the

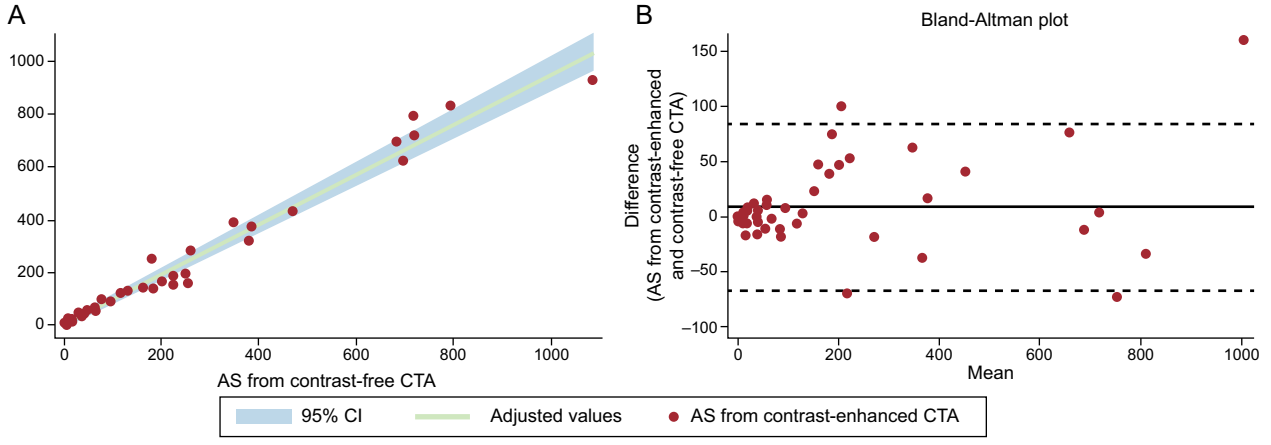

Figure. A: Correlation between Agatston scores obtained by contrast-free and contrast-enhanced computed tomography angiography (linear regression using Passing-Bablok method). B: Degree of agreement between Agatston scores (Bland-Altman plot). AS, Agatston score; CI, confidence interval; CTA, computed tomography angiography.