



## ST elevation on the exercise ECG: only severe stenosis?

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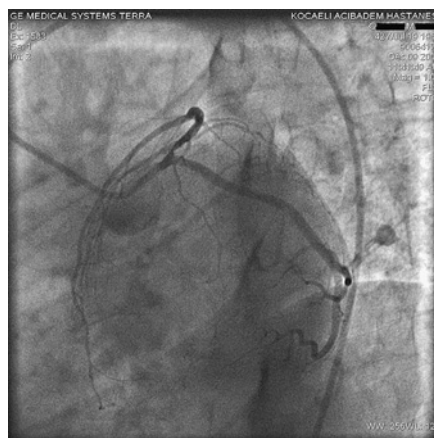
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## FEATURED CORRESPONDENCE

## ST elevation on the exercise ECG: only severe stenosis?

**To the Editor:** We read with interest the article by Murphy *et al* on the importance of ST elevation on the exercise ECG.<sup>1</sup> In this article, the authors suggest that severe coronary artery stenosis was an almost exclusive cause for ST elevation during exercise in a white population. Just a few days after I read this article, a patient came to my office who was referred for coronary angiography as a result of ST elevation on exercise electrocardiography (ECG). The patient was a 42-year-old man who had experienced resting chest pain 2 days ago. He presented at an emergency clinic. After obtaining negative results for cardiac markers, he had undergone exercise ECG. During the second stage of the Bruce protocol, exercise ECG showed up to 5 mm of ST elevation at leads V<sub>1</sub>–V<sub>3</sub> together with chest pain (figure 1). Keeping the article of Murphy *et al* in mind, I planned urgent coronary angiography. During angiography, the first injections in the left coronary artery revealed a severe stenosis at the proximal left anterior descending coronary artery (figure 2). However, also keeping in mind that ST elevation during exercise might be related to coronary spasm, I administered intracoronary nitroglycerin and realised that the stenosis was a coronary spasm (figure 3).

We admire the work of Murphy *et al*, but we have some concerns regarding the interpretation of the data, probably as a result of well-known limitations of retrospective study design. There are several reports indicating that



**Figure 2** Coronary angiogram at left anterior oblique 39° caudal 30°.

coronary vasospasm may mimic obstructive coronary artery disease and unnecessary coronary interventions; even coronary bypass surgery may be performed in case there is lack of suspicion of coronary spasm.<sup>2</sup>

Therefore, in our view, there is a possibility of unrevealed coronary spasm in this large group of patients presented by Murphy *et al* if routine intracoronary nitroglycerin administration was not performed. We believe that clinicians should be more suspicious about coronary spasm if there is transient ST elevation in the history of the patient, and routine use of intracoronary nitroglycerin may be helpful in identifying these patients. In addition, to clarify the meaning of ST elevation on exercise ECG, a prospective angiographic study evaluating the role of coronary spasm is needed.



**Figure 3** The same view after intracoronary injection of nitroglycerin.

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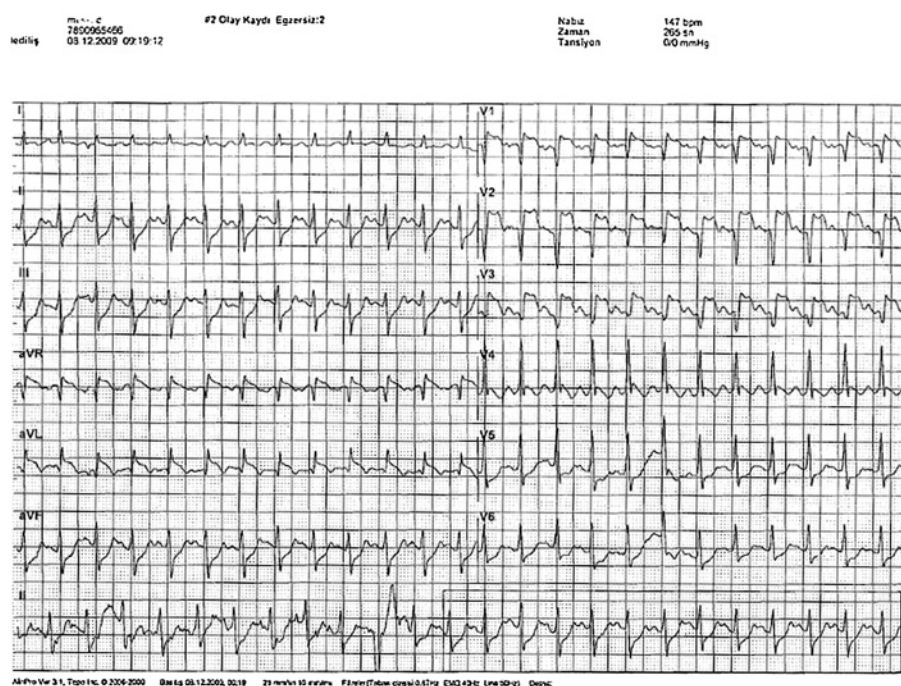
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**The Authors' reply:** We thank Norgaz *et al* for their comments on our article<sup>1</sup> and their demonstration of coronary spasm during cardiac catheterisation in a male patient who had ST-segment elevation (STE) during exercise stress testing.<sup>2</sup> Their report does not suggest the possibility that the spasm was induced by catheter instrumentation or whether the phenomenon reproduced symptoms of chest pain and STE on the electrocardiogram (ECG) at coronary angiography. Furthermore, the exercise ECG demonstrates STE in leads V<sub>1</sub>–V<sub>3</sub> and leads aVR and aVL, with pathological Q waves present in the anteroseptal leads. This indicates the probability of a previous anteroseptal infarction. Our patients did not have a prior history of myocardial infarction or significant Q waves on their ECG.

We indicated<sup>1</sup> that coronary artery spasm can cause exercise-induced STE and has been well described in the literature.<sup>3–4</sup> This can occur in both atheromatously diseased vessels and coronary arteries that otherwise appear normal. We concluded that all patients with



**Figure 1** Exercise ECG recording during the second stage of the standard Bruce protocol.