Dear Sir,

Isolated right coronary artery lesion is rarely detected by standard ECG stress testing [1]. In the presence of exercise-induced myocardial ischaemia the cardiopulmonary exercise test shows blunted VO2 rise, early O2 pulse (VO2/heart rate) plateau, flattening of the VO2/Work relationship and a further heart rate increase to preserve cardiac output [2]. We report the peculiar behaviour at cardiopulmonary exercise test of a case of critical right coronary artery lesion.

A 54 year old overweight (Body Mass Index=31.1) man was referred to us because of light chest discomfort after intense physical activity. Clinical history was otherwise negative. Physical examination, blood pressure, resting ECG and cardiac ultrasound were normal. A cycloergometer cardiopulmonary exercise test was performed with a ramp protocol (20 W/min). ST was normal in active and recovery periods. The subject reported undefined chest discomfort from 140 W to the end of exercise. Increase in ventilation was normal (VE/VCO2 slope=22.5). VO2 increased normally with work-rate till 135 W (VO2/Work slope=11.2). Afterwards, flattening of VO2/Work relationship was observed (VO2/Work slope=4.1, Fig. 1A), together with an early O2 pulse plateau (Fig. 2A, open arrow). Ninety seconds after, heart rate plateau instead of a further increase was observed (Fig. 2A, full arrow). The patient was able to complete the exercise protocol biking for almost 3 more min.

On the basis of exercise test result, multidetector computed tomography coronary angiography (Light Speed 16 slices, General Electric Medical Systems, 0.4-s rotation time, 16 × 0.625-mm detector collimation) was performed. Monovascular disease with critical stenosis of middle right coronary artery was observed. The coronary lesion was treated by PTCA plus stent. Two weeks after PTCA, a new cardiopulmonary exercise test was performed. Normal VO2/work slope throughout the whole exercise and normal rise in heart rate were recorded (Figs. 1B and 2B) with an increase in exercise duration and peak VO2.

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In the present case myocardial ischaemia blunted the physiological VO2 increase with exercise in the absence of significant ECG abnormalities. Myocardial ischaemia was followed by a plateau in heart rate. This could have two explanations: 1) myocardial ischaemia was extended to the sinus node; 2) ischaemic stimulation of vagal receptor in the myocardial wall was able to counteract sympathetic activation. The former is unlikely because the right coronary artery stenosis was distal to the origin of the sinus node artery. The latter is likely because of the great density of vagal receptors in the inferior left ventricle wall [3]. Indeed, differently from anterior, inferior myocardial necrosis is often associated to bradicardia and hypotension [4,5]. Moreover, time course analysis of stress testing shows that myocardial ischaemia, detected by O2 pulse plateau, preceded heart rate impairment, meaning that heart rate stops to raise only when myocardial hypoxia is large enough to induce a significant activation of cardiac vagal reflexes.

Further evidence that VO2 and heart rate pattern was due to ischaemia, is that cardiopulmonary exercise test, performed 2 weeks after myocardial reperfusion, was normal.

References