Received: 26 February 2020 | Accepted: 8 March 2020

DOI: 10.1111/anec.12759

CASE REPORT

WILEY

The Bezold–Jarisch reflex in a patient with coronary spastic angina

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Abstract

Acute inferior myocardial damage can induce transient bradycardia and hypotension—the Bezold–Jarisch reflex, which is explained by the preferential distribution of vagal nerves in the inferior wall of the left ventricle. We report a 76-year-old man who showed a perfusion defect in the inferior wall with redistribution on exercise scintigraphy with thallium-201. Of note, during exercise at an intensity of 100 watts, the patient's heart rate transiently decreased from 122 to 95 bpm in sinus rhythm, accompanied by ST-segment depression. A diagnosis of coronary spastic angina was made since no stenotic lesions were observed on conventional coronary angiography.

KEYWORDS

Bezold–Jarisch reflex, bradycardia, coronary spastic angina, inferior wall

1 | INTRODUCTION

Acute inferior myocardial damage can provoke transient bradycardia and hypotension—the Bezold–Jarisch reflex. This phenomenon is explained by the preferential distribution of cardiac receptors along afferent vagal pathways in the inferior wall of the left ventricle (Aviado & Guevara Aviado, 2001; Mark, 1983). Here, we report a case of coronary spastic angina, in which the Bezold–Jarisch reflex was observed during inferior myocardial ischemia.

2 | CASE REPORT

A 76-year-old man was referred to the Department of Cardiology of Matsushita Memorial Hospital for further examination of chest pain. The chest pain developed in the morning 6 months before this presentation. The patient reported that the chest pain, which was radiating to the shoulder, developed in no relation to physical activities and improved spontaneously within 30 min. He had a history of hypertension and type 2 diabetes which was being treated with insulin. Other medications that the patient was taking included irbesartan, irsogladine maleate, suvorexant, and celecoxib. He did not smoke, drink, or use illicit drugs. There was no family history of cardiovascular diseases.

On examination, the blood pressure was 171/67 mm Hg, pulse was 70 bpm and regular, body temperature was 35.7°C, and oxygen saturation was 100% while he was breathing ambient air. Neither additional heart sounds nor murmurs were audible. An electrocardiogram showed normal sinus rhythm with a heart rate of 72 bpm, with no ST-segment changes or abnormal Q waves. A chest radiograph was normal and echocardiography showed normal chamber sizes and ventricular function. A routine blood examination was normal except for a slightly elevated C-reactive protein of 1.64 mg/dl. The brain natriuretic peptide level was <5.8 pg/ml (reference value, ≤18.4).

The patient underwent maximal symptom-limited exercise scintigraphy with thallium-201. The exercise began at a workload

of 25 watts, which was increased by 25 watts every 2 min, using a bicycle ergometer under continuous monitoring with the Mason-Likar lead system. Exercise was discontinued due to a horizontal ST-segment depression of 2 mm (Figure 1a). The maximal workload, heart rate, and double product were 100 watts, 125 bpm, and 20,000 bpm-mm Hg, respectively (Figure 1b). Of note, when the patient was performing exercise at 100 watts, the heart rate transiently decreased from 122 to 95 bpm (Figure 1c), accompanied by ST-segment depression (Figure 1d). This transient decrease in heart rate developed in sinus rhythm and lasted for approximately 10 s (Figure 2).

Thallium-201 (111 MBq) was injected intravenously at the peak of the exercise. A total of 36 images over a 180-degree anterior arc were acquired 5 min and 3 hr after tracer injections with a digital gamma camera equipped with a low-energy, high-resolution, and parallel-hole collimator. The acquisition lasted 50 beats per projection was stored in a matrix of 64 × 64 pixels, and the images were reconstructed using a Hanning filter without attenuation or scatter correction. Single-photon emission computed tomography and a bull's-eye map showed a perfusion defect in the inferior wall, with thallium-201 redistribution 3 hr after exercise (Figure 3), findings consistent with exercise-induced myocardial ischemia in the inferior wall of the left ventricle.

A diagnosis of coronary spastic angina was made since no atherosclerotic lesions were observed by conventional coronary angiography. Antispastic medication was recommended, but was declined according to the patient's wishes. He has been doing well without adverse cardiovascular events for >1 year although chest pains infrequently occur.

3 | DISCUSSION

The unique finding in the present case was a transient decrease in heart rate before the peak of the exercise during thallium-201 scintigraphy. Given the preferential distribution of cardiac receptors along afferent vagal pathways in the inferior wall of the left ventricle (Linden, 1973; Meller & Gebhart, 1992; Thorén, 1979), we speculate that the decreased heart rate was induced by inferior myocardial ischemia (i.e., the Bezold–Jarisch reflex) due to coronary spastic angina, although a spasm provocation test was not performed during cardiac catheterization.

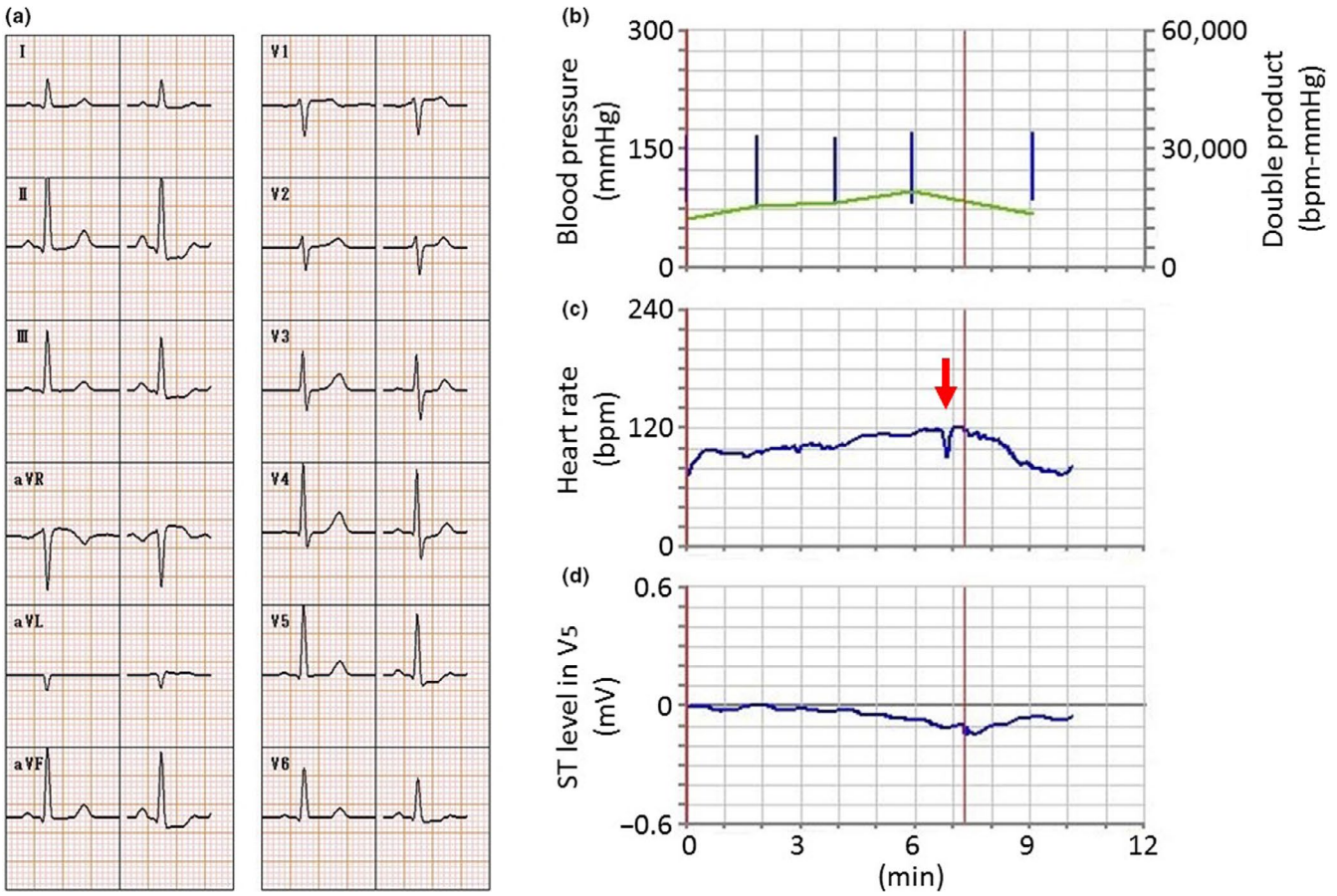

FIGURE 1 Electrocardiography shows horizontal ST-segment depressions in leads II, III, aV_F, and V_4 to V_6 at the peak of the exercise (a, right on each panel), compared with before exercise (a, left on each panel). The increase in blood pressure during the exercise test is not obvious (b); the double product decreases before the discontinuation of exercise (red vertical line). The heart rate transiently drops from 122 to 95 bpm (c, arrow) just before the peak of the exercise, along with an ST-segment depression (d)

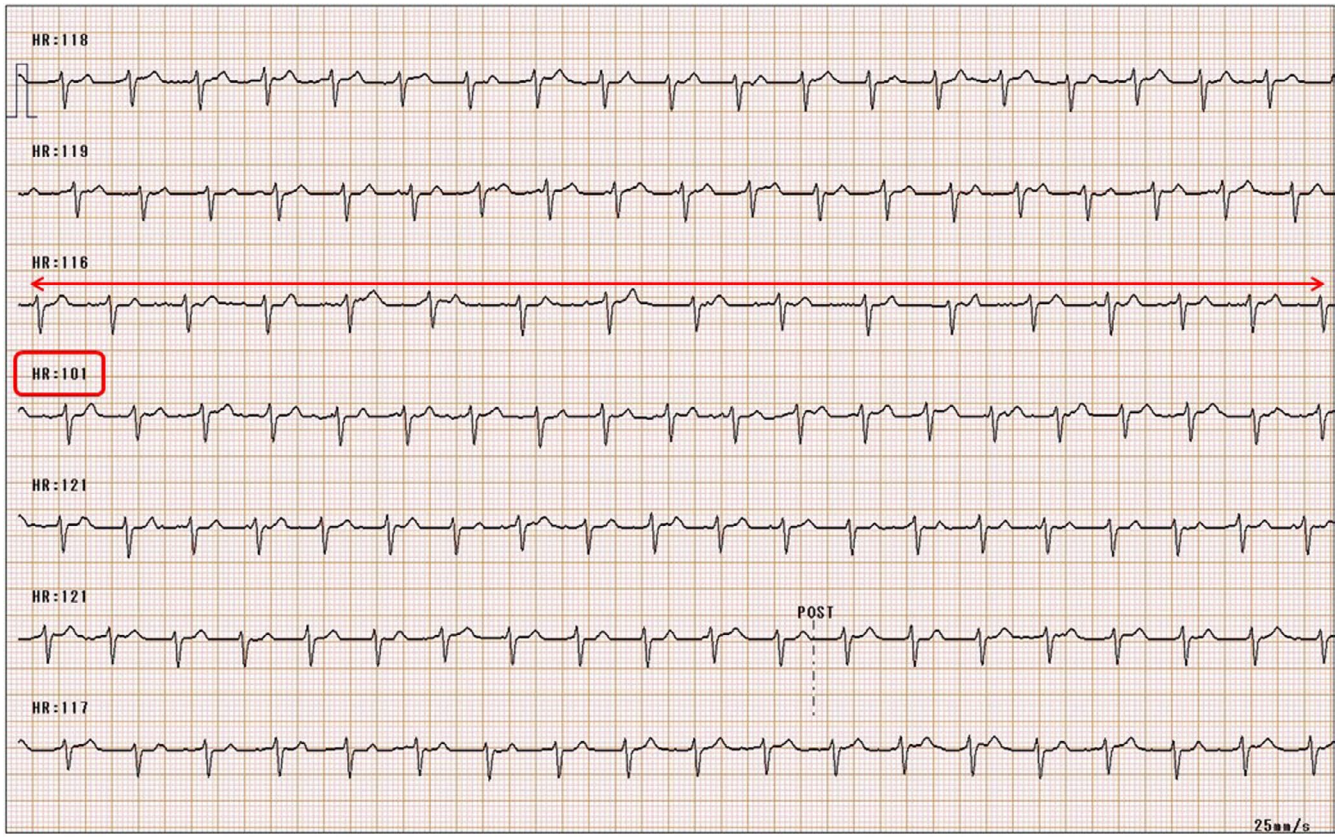

FIGURE 2 An electrocardiogram shows that the heart rate decreased from 116 to 101 bpm, followed by an increase to 121 bpm, approximately between 36 to 26 s (double-headed arrow) before the discontinuation of exercise (dotted line). HR denotes heart rate, which was averaged according to the prior 30-s interval

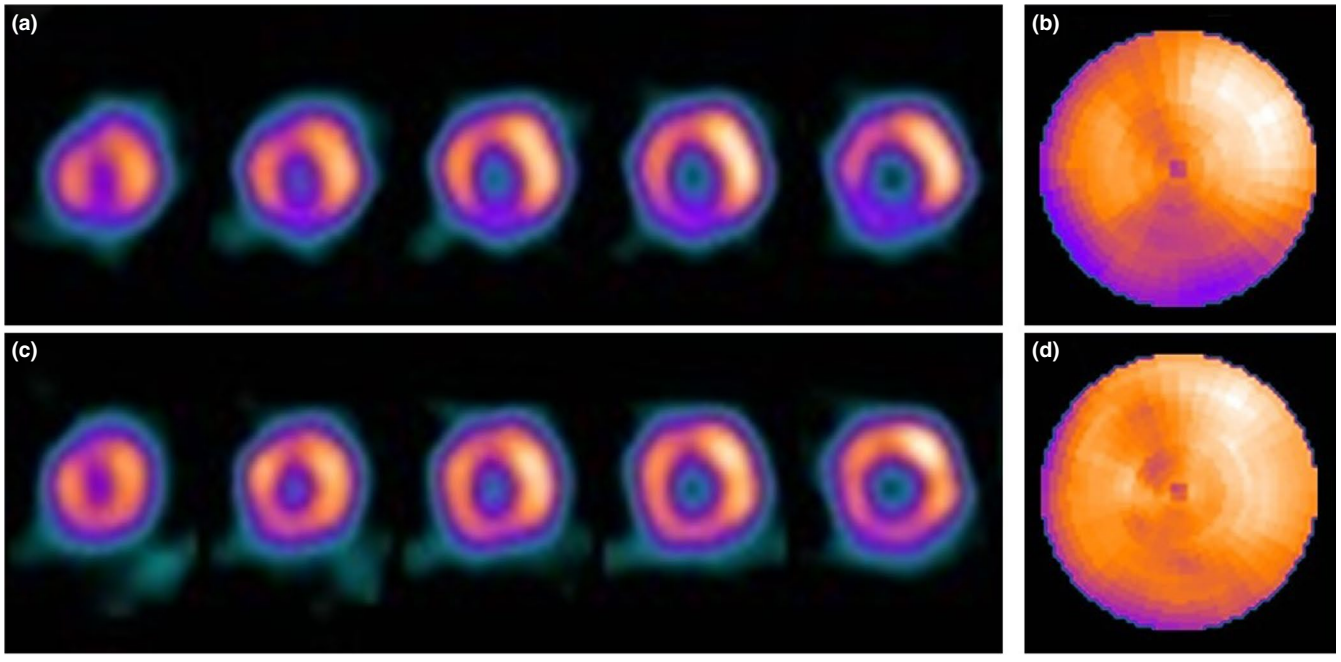

FIGURE 3 After exercise with thallium-201 exercise scintigraphy, the tracer uptake is decreased in the inferior wall of the left ventricle as determined by single-photon emission computed tomography (a) and a bull's eye map (b), with complete redistribution of images obtained 3 hr later (c and d)

The Bezold–Jarisch reflex was named after von Bezold AV and Jarisch A, both of whom conducted physiological experiments. von Bezold AV et al. observed a decrease in blood pressure and heart rate after an intravenous injection of veratrum alkaloids (von Bezold & Hirt, 1867) and Jarisch A et al. later examined the effect of veratridine on the cardiac vagus nerve (Jarisch & Richter, 1939a, 1939b). This phenomenon is the most likely explanation for the bradycardia and hypotension more commonly observed in acute inferior myocardial infarction, compared with acute anterior myocardial infarction (Mark, 1983; Webb, Adgey, & Pantridge, 1972). We previously reported that the Bezold–Jarisch reflex can be induced by inferior myocardial ischemia in patients with coronary artery stenosis (Kawasaki et al., 2006) and patients with residual inferior ischemia after inferior myocardial infarction (Kawasaki et al., 2009). To our knowledge, our case is the first report to demonstrate the Bezold–Jarisch reflex being induced by coronary spastic angina.

The exact incidence of the Bezold–Jarisch reflex associated with inferior myocardial ischemia remains unclear. It is reported that seven patients with significant narrowing of the right coronary artery displayed sinus deceleration, defined as ≥5 bpm during exercise in approximately 40,000 consecutive exercise tests (Miller, Gibbons, Squires, Allison, & Gau, 1993), although far more patients with right coronary lesions were included in their study. Thus, a decrease in sinus rate during exercise testing may be a useful indicator of whether or not the site of myocardial ischemia includes the inferior wall, as we described elsewhere (Kawasaki, Azuma, Kuribayashi, & Sugihara, 2011). It is worth noting that the Bezold–Jarisch reflex or enhanced vagal modulation related to inferior myocardial ischemia were more frequently detected when techniques for the assessment of the autonomic nervous system were performed such as a coefficient of component variance of high frequency by heart rate variability analysis (Kawasaki et al., 2006, 2009).

In our case, a decrease in heart rate did not persist until the resolution of inferior myocardial ischemia. This condition may be explained by a physiological reaction in which sympathetic excitation during exercise is translated into a functional response, that is, heart rate increase, although the mechanism remains to be elucidated based on physiological studies. Hypotension, which may be induced by the Bezold–Jarisch reflex, did not develop during exercise in our case, but it should be recognized that an adequate increase in blood pressure was not obtained during exercise. More frequent measurements or continuous monitoring of blood pressure may have detected transient hypotension related to the Bezold–Jarisch reflex.

In summary, our case highlights the importance of recognizing cardiac autonomic function, which may be a useful for determining the site of myocardial ischemia.

ACKNOWLEDGMENT

None declared.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Drs. Sakai and Kawasaki treated this patient; Dr. Sugihara performed exercise test; Dr. Matoba reviewed this manuscript.

ETHICAL APPROVAL

None declared.

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How to cite this article: Sakai C, Kawasaki T, Sugihara H, Matoba S. The Bezold–Jarisch reflex in a patient with coronary spastic angina. *Ann Noninvasive Electrocardiol*. 2020;25:e12759. https://doi.org/10.1111/anec.12759