# Coronary spasm provocation testing: still useful?

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## Summary

This short and interesting case demonstrates very aptly the potential clinical utility of provocation testing for coronary artery spasm and briefly discusses the uncertainty encountered in interpreting the test.

*Key words: coronary artery spasm; cardiac arrest; provocation testing*

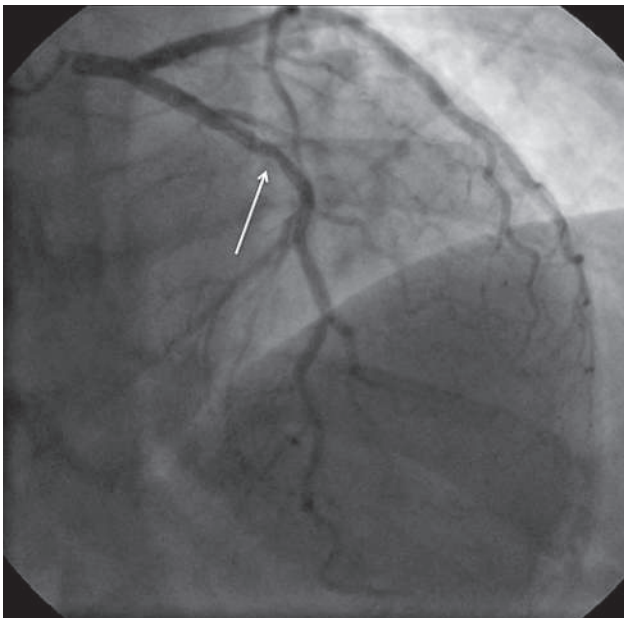
## Case report

A 47-year-old New Zealand European female presented to hospital after two syncopal events, both preceded by transient chest discomfort. She had a history of mild asthma and dyslipidaemia and was an active smoker. Her only current medication was omeprazole. She did not drink alcohol, nor consume illicit drugs and had immediately prior to her presentation been well. The cardiovascular examination was unremarkable and baseline haematological and biochemical investigations, including high sensitivity troponin T, were all within the normal range. The 12-lead electrocardiogram (ECG) was normal, but during the course of her admission she had two nonsustained runs of monomorphic ventricular tachycardia (NSVT) not associated with symptoms. No ST-segment change was evident, either prior to, or immediately following, the arrhythmia.

During selective coronary angiography, mild diffuse plaque was noted in all major epicardial coronary arteries (fig. 1). No regions of myocardial bridging were observed. Magnetic resonance imaging (MRI) with gadolinium enhancement demonstrated no structural cardiac abnormality, nor evidence of infiltration, oedema or fibrosis. The history of possible angina pectoris prior to the syncopal events raised concern regarding the possibility of coronary spasm. She therefore underwent an ergonovine challenge. Following 150 µg intracoronary ergonovine, abrupt occlusion of the proximal left anterior descending (LAD) coronary artery was observed (fig. 2). This was associated with ST-segment elevation in the anterior ECG leads, but no arrhythmia. There was complete resolution of the ECG changes and restoration of coronary flow following administration of intracoronary nitroglycerine.

The patient was subsequently treated with high-dose oral nitrates and a calcium channel blocker, and underwent dual chamber implantable cardioverter defibrillator (ICD) insertion. At follow-up she remains well with no further events.

**Figure 1 (movie 1[1])**
Cranial 40° Projection.
Note: mild diffuse plaque affecting left main stem and left anterior descending artery (LAD).



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[1] You can find the movies on http://www.cardiovascmed.ch/for-readers/multimedia/

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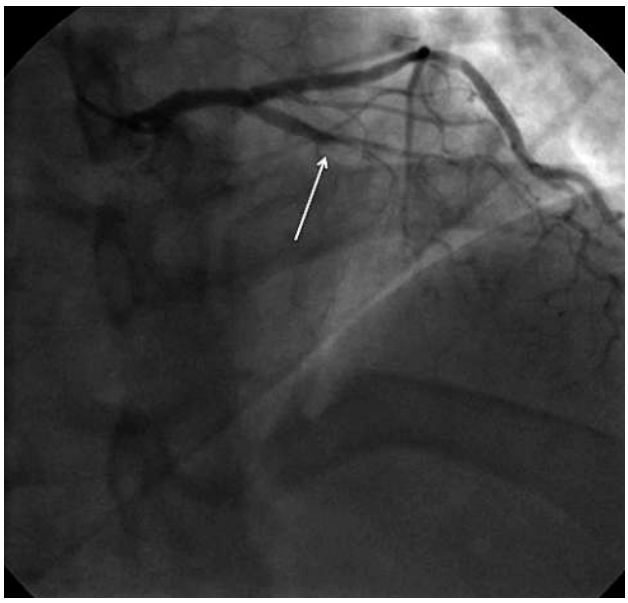
**Figure 2 (movie 2[1])**
Cranial 40° Projection.
Note: abrupt occlusion of mid left anterior descending (LAD) artery at site of moderate plaque (arrow).



## Discussion

Coronary artery spasm is a syndrome of reflex reduction in the coronary arterial lumen in response to neurological, chemical or mechanical stimulation. This can result in angina pectoris, often unpredictably at rest and sometimes termed Prinzmetal or variant angina. Coronary spasm is often not recognised. This is concerning, particularly given that beta-blockers (frequently prescribed for angina) are contraindicated, but also because spasm has significant potential for both morbidity and mortality as it can be associated with extensive infarction and malignant arrhythmia [1].

The clinical utility of provocation testing for suspected coronary artery spasm has long been contentious owing to uncertainty regarding lack of a reference standard and potential for harm. The most commonly administered agent is ergonovine (ergometrine), a primary ergot alkaloid with profound alpha-adrenergic effects. When administered into the coronary circulation, ergonovine acts via alpha-1A receptors, leading to a marked increase in vascular tone and, in some instances, profound vasospasm as seen in the present case, with total, focal spasm associated with ST-segment elevation.

The incidence of spasm determined by provocation testing varies dramatically between published series. High rates of a positive provocation test are observed in Asian populations [2]. The ergonovine challenge is more likely to be positive in patients with rest rather than exertional angina pectoris [3]. A further small study showed that sensitivity of ergonovine challenge in a subgroup of patients with daily attacks of variant angina was 100% and much lower (55%) in patients with sporadic attacks [4]. In one large series, an ergonovine challenge was more likely to provoke spasm in patients with rest angina pectoris and unlikely when chest pain was considered to be 'atypical' [5]. However, the correlation between a positive provocation test and the development of variant angina due to coronary vasospams remains uncertain. Of further interest, there appears to be overlap between the observation of coronary spasm and coronary artery disease, as in about 50% of cases with a positive provocation test, atheromatous lesions are found at the site of spasm, in keeping with the original report of variant angina from Prinzmetal [6].

Despite potential for risk, a large study found the rate of major complications to be low, even with sequential administration of ergonovine [7]. Nonetheless, given rare occurrence of serious complications including death, it is suggested that where there is moderate clinical suspicion of coronary vasospasm, medical therapy (oral nitrates / calcium channel blockers) be used as an initial approach. In refractory cases, provocation testing may be applied for targeting possible stent sites – an approach that can sometimes successfully reduce the propensity towards spasm at the treated site [2].

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