Syncope caused by cardiac asystole during dobutamine stress echocardiography

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Abstract
Syncope caused by cardiac asystole during dobutamine stress echocardiography occurred in a 60 year old woman presenting with chest pain and a non-diagnostic exercise test. Cardiac asystole was not associated with myocardial ischaemia and was attributed to a powerful cardioinhibitory vagal reflex elicited by the stimulation of the drug of cardiac and aortic mechanoreceptors. Cardiac asystole was promptly reversed by the administration of atropine with no significant sequelae.

(Heart 1996; 75: 320-321)

Keywords: dobutamine stress echocardiography; asystole; syncope

Ventricular arrhythmias are the most common side effect of dobutamine stress echocardiography. They are related to the adrenergic activity of dobutamine. A syndrome characterised by dizziness, bradycardia, and hypotension has occasionally been described. It is thought to be mediated by a vagal activation caused by the stimulation of mechanoreceptors of the myocardial wall and of the aortic root. Usually it is not associated with myocardial ischaemia. When it causes severe symptoms the test may have to be stopped. Until now, cardiac asystole has not been described during dobutamine stress echocardiography in more than 1700 tests.

We report a case of syncope during dobutamine stress echocardiography caused by the abrupt development of cardiac asystole in a woman with chest pain who was being tested for coronary artery disease.

Case report
A 60 year old woman was admitted for evaluation of chest pain occurring both at rest and on effort. A basal electrocardiogram showed normal sinus rhythm and slight T wave abnormalities. Physical examination and chest x ray were normal. A cross sectional echocardiogram showed normal left ventricular wall thickness, normal segmental wall motion, and systolic function. A supine bicycle exercise stress test was interrupted after four minutes of exercise because of dyspnoea and fatigue at an heart rate of 100 beats/min (62% of the age-predicted maximum heart rate) with no significant ST-T changes.

A dobutamine stress echocardiographic test was then performed according to a previously described protocol. At baseline her heart rate was 63 beats/min and her blood pressure was 180/100 mm Hg. At a dobutamine dose of 20 µg/kg for two minutes the heart rate...
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increased to 82 beats/min and blood pressure to 195/90 mm Hg. Diffuse left ventricular hyperkinesia with increased systolic thickening of all myocardial segments was apparent. During the third minute of this step heart rate decreased to 60 beats/min; then cardiac asystole caused by sinus arrest suddenly developed and lasted more than eight seconds (figure) causing abrupt loss of consciousness. External cardiac massage was started immediately and an intravenous bolus of atropine (1 mg) was given. Sinus rhythm was rapidly restored and she regained consciousness with no neurological sequelae. Sinus tachycardia (145 beats/min) followed the administration of atropine with no electrocardiographic or echocardiographic signs of ischaemia. The patient refused coronary arteriography and electrophysiological evaluation of sinus function.

Discussion

The syndrome characterised by hypotension, bradycardia, and dizziness occurring during dobutamine administration has been thought to be due to vagal activation caused by the stimulation of mechanical receptors in the aorta or myocardium by the vigorous myocardial contraction induced by the drug. This side effect was detected in some patients performing dobutamine stress echocardiography, ranging from 14% in the study of Mazeika et al. to 0-3% in the study of Mertes et al. In a few cases only it was associated with myocardial ischaemia or impaired left ventricular systolic function. To the best of our knowledge, however, there are no earlier reports of syncope in patients undergoing dobutamine stress echocardiography. Although we cannot be certain that there were no important coronary obstructions, the diffuse hyperkinetic wall motion seen during dobutamine infusion and the absence of chest pain and ST-T changes during dobutamine and exercise tests suggest that myocardial ischaemia was not the cause of this syndrome in our patient. A powerful cardioinhibitory vagal reflex elicited by exaggerated cardiac activity of the mechanoreceptors and concomitant stimulation of the aortic receptors by the increase of systolic blood pressure is the most likely mechanism of syncope in this woman.

Although dobutamine stress echocardiography is generally regarded as a safe test with a low incidence of major side effects, this case report suggests that the test should be performed with caution especially in patients with a history of dizziness or syncope and that atropine should be immediately available to counterbalance the vagal activation induced by dobutamine.

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*Heart* 1996 75: 320-321
doi: 10.1136/hrt.75.3.320

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