

## CASE REPORT

## Myocardial stunning after streptokinase: what is the significance of the Q wave?

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### Abstract

A 58 year old woman presented with symptoms and electrocardiographic features consistent with acute infero-posterior myocardial infarction. The attempt at reperfusion with aspirin and thrombolytic treatment was deemed unsuccessful in view of Q wave development on ECG, a 48 hour period of hypotension and oliguria, and extensive wall motion abnormality on echocardiography. This was at variance with findings of a minimal cardiac enzyme rise. On the seventh day, list mode acquired, ECG gated, cine-images of perfusion and blood pool demonstrated strikingly normal perfusion despite continued contractile dysfunction. Six weeks later ECG, echocardiography, and radionuclide blood pool ventriculography had all normalised consistent with resolution of myocardial stunning. This practical method for the diagnosis of stunning, the significance of the ECG changes in particular the development of Q waves, and the time taken to resolution of this phenomenon are discussed.

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Keywords: myocardial stunning; thrombolysis; radionuclide; imaging.

A 58 year old woman awoke with central chest pain. The pain worsened over the next hour, becoming crushing in nature, radiated into both upper arms, and was accompanied by autonomic symptoms. Her distress was relieved only by intravenous opiate and anti-emetic in the accident and emergency department. The ECG was consistent with an acute infero-posterior myocardial infarction (MI) (fig 1A), and in the absence of contraindications she received aspirin and streptokinase infusion. Three and a half hours had elapsed since the onset of symptoms. The woman had smoked cigarettes all her adult life, but there were no other relevant features in the medical history.

Sustained hypotension ensued following thrombolytic treatment. Systolic blood pressure did not rise above 90 mm Hg. The woman denied symptoms. There were no signs of left ventricular failure, cardiac murmur, added heart sounds, or increase of jugular venous pressure.

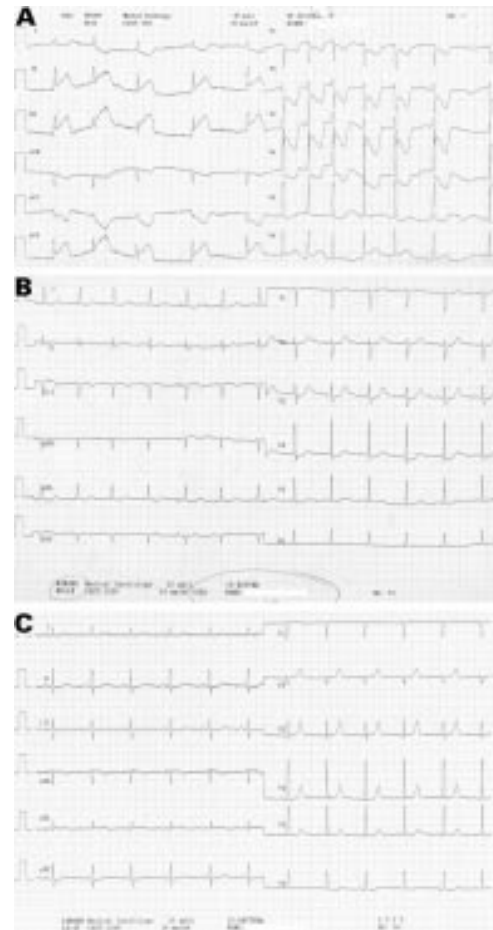


Figure 1 Twelve lead ECGs taken supine; (A) on admission; (B) 24 hours later; and (C) six weeks after admission.

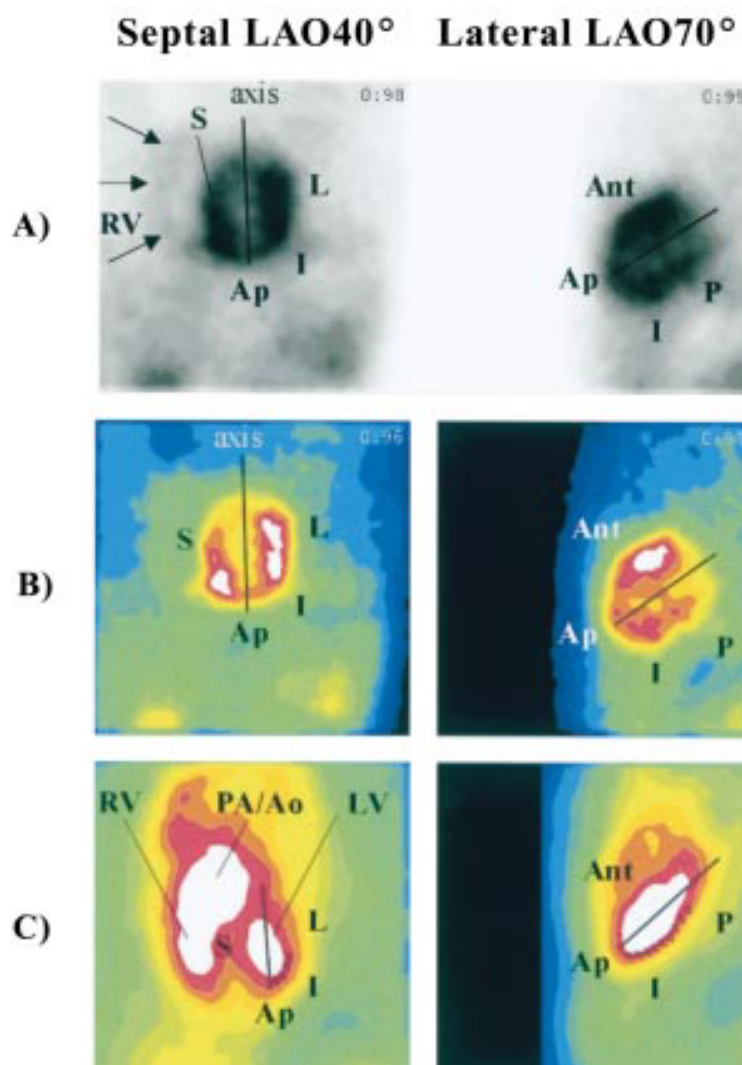
ECG taken after thrombolysis demonstrated a degree of ST segment resolution. However, by the second day, Q waves had developed in lead III and aVF (fig 1B). In view of ongoing hypotension, echocardiography performed on day two demonstrated akinesia of the infero-posterior myocardial segments, and hypokinesia of the lateral wall. At no stage were the inferior ECG changes associated with atrioventricular conduction abnormality.

Her condition deteriorated, blood pressure fell to 70/50 mm Hg, and she became oliguric. The clinical impression was one of right ventricular MI with left ventricular involvement.

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**Figure 2** Diagnostic criteria for myocardial stunning. Still frames from cine perfusion and blood pool images created by list mode acquisition of ECG and radionuclide data.<sup>1</sup> (A) Absence of right ventricular (RV) infarct. Monochrome cine thallium perfusion images demonstrate normal RV perfusion, dimensions and function. Myocardial segments are identified with respect to the cardiac axis. (B) Normal myocardial perfusion. Monochrome images are overlaid with a geographical colour scale. (Normal range, white; dark red; light red; and dark orange.) (C) Regional contractile dysfunction. Infero-posterior hypokinesia on the cineimages of the contracting technetium labelled intracardiac blood pool (denoted xxx.). (LAO, left anterior oblique; RV, right ventricle; LV, left ventricle; PA/Ao, pulmonary artery and aortic outflow overlap; S, septum; Ant, anterior; Ap, apex; L, lateral; I, inferior; P, posterior.)

Fluid challenge was instituted without invasive monitoring. Two litres of urine were passed within 24 hours with improved systolic blood pressure of 110 mm Hg. She was then started on an angiotensin converting enzyme inhibitor, receiving 5 mg of lisinopril by the fourth day.

There were two features strikingly at odds with the clinical course. First, despite her dramatic presentation, early ECG findings, development of Q waves, and wall motion abnormalities on echocardiography, only a mild increase of cardiac enzymes had occurred, with a peak creatine kinase of 660 (normal < 150 U). Second, the pathological Q wave in lead aVF had resolved on the supine 48 hour ECG.

Symptom limited, bicycle stress test with radionuclide imaging was performed on the seventh day. Twelve lead ECG, taken in the sit-

ting position before exercise, demonstrated sequential ST change without Q waves. The woman exercised for five minutes. The test was discontinued because of leg fatigue, but no additional ST segment change occurred.

Cine ECG gated thallium (<sup>201</sup>Tl) perfusion images unequivocally excluded right ventricular infarction (fig 2A). The technique of acquiring radionuclide and ECG data in “list-mode” to recreate moving images,<sup>1</sup> demonstrated the right ventricle to have normal perfusion, dimensions, and contractile function. Unexpectedly, perfusion of the left ventricle was also normal (fig 2). In contrast, the corresponding, cine gated technetium (<sup>99m</sup>Tc) blood pool images displayed infero-posterior hypokinesia, an observation entirely in accord with the previous echocardiogram (fig 2C). This combination of normal perfusion but myocardial contractile dysfunction is diagnostic of myocardial stunning.<sup>2</sup> Repeated investigations six weeks later found the ECG (fig 1C), echocardiogram, and cine-blood pool to have returned to normal.

Normalisation of the technetium blood pool “phase images” is further confirmation of the recovery of myocardial contractility. The phase image represents the changes in pixel counts with respect to time during the cardiac cycle.<sup>3</sup> A normal ventricle with normal electrical conduction will trigger almost instantaneous myocardial contraction across the ventricle. The initial day seven phase images, however, show activity in the infero-posterior region to be considerably out of phase with the rest of the ventricle. The images are normal six weeks later (fig 3).

## Discussion

Myocardial stunning is “the delayed recovery of regional myocardial contractile function after reperfusion, despite the absence of irreversible damage and despite restoration of normal flow.”<sup>2</sup> The concept was initially developed and characterised in experimental animal models,<sup>4</sup> then supported by clinical experience in cardiac surgery, and during interventional revascularisation for the treatment of acute coronary syndromes.

After thrombolysis for acute MI, reversible changes in contractile function have often been observed and loosely attributed to “stunning”. However, accurate diagnosis of stunning is rarely possible in clinical practice.<sup>5</sup> To critically distinguish stunning from necrotic, ischaemic, or hibernating myocardium, the criterion of completely restored perfusion must be fulfilled. Echocardiography may be used to observe wall motion abnormalities,<sup>6</sup> but thallium perfusion studies are necessary to demonstrate restored perfusion and metabolically active myocytes.<sup>7</sup> A practical difficulty is the orientation of both imaging modalities to characterise myocardial segments appropriately.

Stunning is an important phenomenon only when of such severity as to jeopardise ventricular function and cardiac output. Lesser degrees of stunning are not of clinical importance since by definition, reperfused, stunned myocardium will recover function with time. There is great

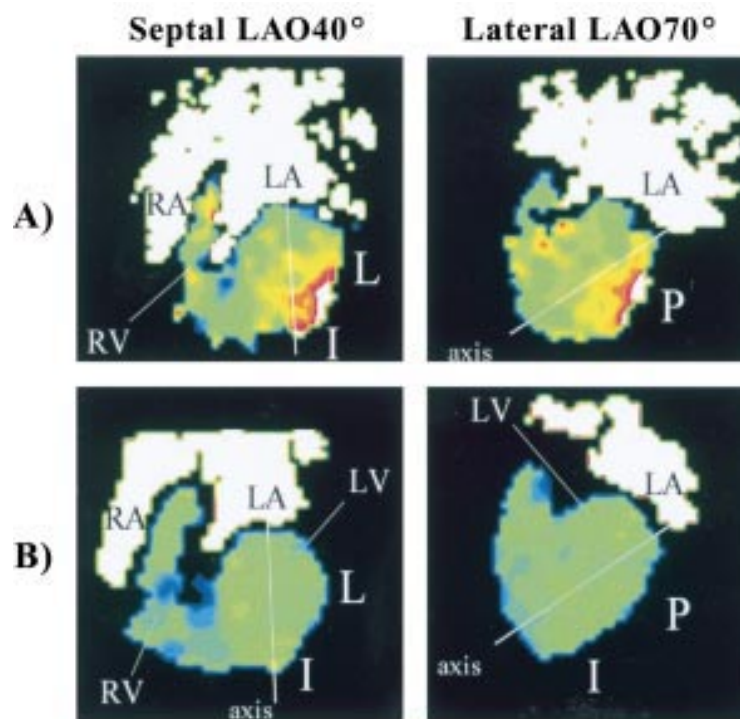


Figure 3 Phase images derived from intracardiac blood pool ventriculography. (A) Day seven activation in the infero-posterior region is out of phase with the rest of the ventricle. (B) After six weeks, all ventricular regions are moving normally "in phase" with each other during the cardiac cycle.

interest in both elucidating the mechanisms of stunning at a cellular level,<sup>8</sup> and developing therapeutic interventions. However, in clinical practice, the incidence of significant stunning after reperfusion, as distinct from extensive MI, is not known. An observational study of acutely compromised patients would be necessary to determine whether there is a clinical need for treatments to prevent myocardial stunning.<sup>9</sup>

The use of list mode acquisition of ECG and radionuclide data to create moving images of perfusion and blood pool in identical orientations,<sup>1</sup> has not been described for identification of myocardial stunning. This non-invasive technique could be performed at rest on the day of admission with a mobile gamma camera to characterise each myocardial segment in terms of perfusion and contraction. This would systematically delineate resting ischaemia or infarction from non-contracting but reperfused, stunned myocardium. There is no requirement for the incremental inotropic stimulation used for stress echocardiography, a technique that is deleterious in unstable patients with acutely ischaemic and infarcting myocardium.

The case report illustrates several further points:

- Thrombolytic treatment had been deemed unsuccessful in this case in view of Q wave development on ECG, a 48 hour period of hypotension and oliguria, and extensive wall motion abnormality on echocardiography. Had the minimal cardiac enzyme rise not been noted or radionuclide studies and further ECGs not performed, then the diagnosis would have remained one of un-

successful thrombolytic treatment, RV infarction with sizeable LV involvement.

- Stunning is clearly evident here, seven days after thrombolysis. This is attributable to the time of acute reperfusion because not only is resting perfusion normal, but also stress imaging. The possibility of exercise induced "repetitive stunning",<sup>6, 10</sup> due to an underlying critical coronary lesion is excluded. Coronary angiography was not indicated in view of the reassuring, asymptomatic exercise test without ECG changes and normal stress perfusion. The time course for recovery of stunning must lie between seven days and six weeks, although the initial 48 hour period of hypotension and oliguria after thrombolysis would appear to be the time when ventricular function is impaired as a consequence of myocardial stunning. This timescale greatly exceeds that seen in animal models.<sup>4</sup> However, a direct comparison between the effect of artificial coronary occlusions in healthy canines and undisclosed extremes of atherosclerotic disease and collateral vessel formation in human subjects, is inappropriate.
- The belief that abnormal Q waves are pathognomonic of MI has become accepted practice in cardiology since the 1930s. This case history documents the development of pathological Q waves in the absence of necrosis, which normalise days before the recovery of ventricular contractile function. There is a hypothesis that stunning of the cardiac electrical system as well as myocardium will occur during intense ischaemia.<sup>11</sup> The findings in this study suggest that development of extensive Q waves associated with hypotension will not always reflect extensive myocardial necrosis alone.

### Summary

This case report supports the finding that stunning of the cardiac electrical system, denoted by transient Q waves, occurs in clinical practice.<sup>11</sup> The presence of Q waves may not always equate with the presence of extensive myocardial necrosis. The electrical stunning seen here resolved before the myocardial stunning that had initially jeopardised cardiac output. Our experience of radionuclide imaging in the convalescent phase of MI suggests the presence of late stunning to be a rare phenomenon.

The incidence of stunning occurring in acutely compromised patients with apparent large MI at the time of thrombolytic treatment is not known. An observational study is required. List mode, ECG gating to create moving cineimages of perfusion, and blood pool have not been described for the identification of "myocardial stunning". The method is simple to perform, is reproducible, and can be applied to large numbers of patients.

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