Intravascular ultrasound in the diagnosis of the no-reflow phenomenon after primary angioplasty for myocardial infarction

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A 73 year old man was admitted with haematemesis and was found to have a pangastritis. Three days later he developed central chest pain, and ECG changes indicated an acute anterior myocardial infarction. In view of his recent haematemesis we performed emergency coronary angiography, which showed proximal occlusion of the left anterior descending (LAD) artery (TIMI 0 flow), with normal circumflex and right coronary arteries. We proceeded to primary percutaneous transluminal coronary angioplasty (PTCA) with a 7 French left 4.5 Judkins guide, a Magnum wire (Schneider UK, Staines, Middlesex, UK), and a 3.5 mm Viva balloon (Boston Scientific Ltd, St Albans, Herts, UK). Unfractionated intravenous heparin 10 000 units was given. The lesion was crossed with Magnum wire and injections at that time showed TIMI 1 flow. The site of occlusion was dilated with the 3.5 mm balloon to 6 then 8 atm with subsequent TIMI 0 flow. Dissection was thought to be the most likely cause of the no-reflow phenomenon and a 3.5 × 34 mm mounted Bard stent (Bard Ltd, Crawley, West Sussex, UK) was deployed at 13.5 atm. Subsequent injections showed TIMI 0 flow (fig 1) and 200 µg of intracoronary nitrate was given to exclude coronary spasm as a cause of the no-reflow phenomenon, with no improvement. At this stage it was impossible to find the cause of the no-reflow phenomenon and intravascular ultrasound (IVUS) was used to examine the distal vessel with an Ultracross 3.2 F 30 MHz IVUS probe (Boston Scientific Ltd). This showed segments of severe distal disease, a satisfactory appearance to the stented lesion proximally, and normal artery between these two areas (fig 2). The most severe segment was dilated with a 2.5 mm Viva balloon at 8 atm, and perfusion then improved. A 16 mm mounted Jo stent (Jomed UK Ltd, Knutsford, Cheshire, UK) was deployed at 11 atm and further inflations were made to additional distal lesions. TIMI 3 flow was thus achieved in the epicardial artery (fig 3). After the procedure, the patient was given ticlopidine; no other antiplatelet agents or anticoagulants were used. A blood sample taken at the end of the operation showed an activated partial thromboplastin time ratio of greater than 8.0.

Discussion
The no-reflow phenomenon is the inability to achieve myocardial reperfusion after the removal of a coronary artery occlusion. It was first seen in the coronary arteries of dogs,1 and has since been recognised in human patients as an uncommon complication of thrombolytic or mechanical reperfusion for acute myocardial infarction, and after percutaneous interventions with balloons and other devices. Microvascular injury is thought to be the cause of the phenomenon; possible mechanisms include capillary vasospasm from the release of vasoactive mediators, oxygen free radical mediated injury, neutrophil and erythrocyte plugging, intramural haemorrhage, perivascular oedema, and endothelial cell oedema secondary to ischaemic reperfusion. Intravenous and intracoronary glyceryl trinitrate, verapamil, thrombolytic agents, and heparin have been used to treat the condition and, in one series, intracoronary verapamil was used successfully to treat 67% of no-reflow cases.2 Intra-aortic balloon pumping can be used when the condition is unresponsive to other treatment to reduce myocardial afterload mechanically and increase coronary perfusion pressure. However, it is well documented that the no-reflow phenomenon is linked to a poorer prognosis, higher incidence

Figure 1 Left selective coronary angiogram in left anterior oblique cranial projection (48° LAO, 33° cranial) showing the no-reflow phenomenon in the LAD after stent deployment.
of in-hospital death, myocardial rupture, and larger infarct area.3

The diagnosis of the no-reflow phenomenon remains one of exclusion, and several angiographic views are usually taken to discount coronary dissection, distal embolisation, and prolonged spasm as the cause of failed reperfusion. However, poor opacification can mean that satisfactory examination of the distal vessel is difficult, and Sherman et al attempted to overcome this problem with an end hole infusion catheter to detect a pressure gradient across the obstruction and to allow distal contrast injections.4 We are not aware of any previously published descriptions of the use of IVUS to examine the distal vessel in a patient with the no-reflow phenomenon. This may, therefore, be a useful tool to detect residual high grade distal stenosis as a cause of no-reflow, which may be an underrecognised cause of failed reperfusion following balloon angioplasty for acute myocardial infarction. We suggest that consideration should be given to the use of IVUS before a diagnosis of the no-reflow phenomenon is made in such cases.

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Heart 1999 82: e3
doi: 10.1136/hrt.82.3.e3

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