Cynics on Sugar Loaf mountain

granite stack at 30 metres a day, spending most of their time bivouacked in portable platforms hanging from a single hook. From the city you could just see their banner, tiny against the grey green rock face, creeping higher day by day. It was like a barometer for the negotiations. Today they would reach the top.

As Edwin Drummond and Tess Burrows finally scrambled over the ledge the idealism in their sunburnt faces contrasted starkly with the cynicism being expressed below. The people hugging them were idealists too. "The easiest thing in the world is to complain," said Robert Swan, the UN ambassador for environment and youth. "But what has happened in Rio is positive, there's no question about it. The time for confrontation is over. We have to give young people hope. Unless we inspire them now they won't bother to turn up next time."

Sunday 14 June

Maurice Strong's closing speech was undiplomatic, fast, and passionate. While we had been in Rio the process of deterioration had continued, he said. Five thousand tons of carbon dioxide had been pumped into the atmosphere every minute. More than 250 000 children had been born, "most of them poor, all facing an uncertain future." Twenty eight had died of hunger every minute, three quarters of them under 5 years old.

He called for urgent ratification and implementation of the two framework conventions and for rapid action on Agenda 21 to be monitored by a high level commission on sustainable development. He strongly criticised the lack of financial commitment. New sources of funding were needed, he said, such as energy taxes and user and emission charges based on the principle that the polluter pays.

The world, he said, would not be the same after Rio. Nor would the environment development dialogue. In future all talk of the environment would have to include discussions about poverty, terms of trade, and flow of resources.

Boutros Boutros-Ghali closed the conference with talk of post-Rio man. Rio meant, he said, a change in individual behaviour on a massive scale. His words echoed those of Wangari Maathai, spokesperson for the Kenyan NGO, Greenbelt, and for me the most inspiring speaker in a fortnight of high flown words. A gently spoken woman in her 50s, and recently beaten by Kenyan police, she had told us that the challenge to change started with each one of us. "We have come a long way," she said. "We should speak more of hope. We should not discourage each other. We must continue pushing for a more just world." Our governments were doing what they thought we wanted, she said. "If they thought that their people had decided otherwise a different discussion would be happening at Rio."

Monday 15 June

A few remaining journalists staggered about looking shellshocked. In the previously bustling telecommunications room staff were watching an American B movie with blank faces. Already Rio was looking shabbier. There were beggars on the streets. A man with no legs—God knows how he got there—lay face up on a pile of rags. Later a woman in very short shorts approached a friend as we walked in a group along the sea front. She grabbed him by the genitals, took the money from his back pocket, and walked off.

The two week conference had cost the UN an estimated $1·3 billion and Global Forum about $11 million. Add to that the cost, both environmental and financial, of plane flights for the tens of thousands of foreign delegates and journalists, taxis and buses to and from the conference centre, and the piles and piles of paper.

But 153 countries signed the two legally binding conventions on climate change and biodiversity, and all countries agreed on Agenda 21 and the broad statement of principles laid down in the Rio Declaration. These were achievements unthought of at the beginning of the fortnight.

Back in London: traffic jams, one person per car, and trains that won't allow bikes on board. A change of individual behaviour on a massive scale? No sign of it yet.

Lesson of the Week

Severe "silent" mitral regurgitation after myocardial infarction: a clinical conundrum

John Efthimiou, Maxton Pitcher, Oliver Ormerod, Fiona Harper, Stephen Westaby, David Grahame-Smith

Papillary muscle rupture causing severe mitral regurgitation may be "silent" on auscultation but be detectable by transoesophageal echocardiography

Correspondence to:
Dr Efthimiou.

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Papillary muscle dysfunction complicating acute myocardial infarction is well recognised. We present a case of papillary muscle rupture causing severe but auscultatorily "silent" mitral regurgitation and cardiogenic shock.

Case report

A 61 year old man was admitted to hospital with a four hour history of severe central chest pain associated with sweating, nausea, and dyspnoea. He had no risk factors for ischaemic heart disease. On examination he was found to be in cardiogenic shock with blood pressure 90/50 mm Hg, heart rate 90/min, and jugular venous pressure raised 4 cm. On auscultation a third heart sound was detected but no murmurs, and inspiratory crackles were heard bilaterally. The findings were confirmed by two cardiologists, who advised on the case throughout.

An electrocardiogram confirmed an acute inferior myocardial infarction with posterior extension. Cardiac enzyme values were raised, with a peak aspartate transaminase concentrations of 138 IU/l and lactate dehydrogenase concentrations 328 IU/l. The chest radiograph showed diffuse alveolar shadowing consistent with pulmonary oedema. The patient was treated with oxygen, morphine, frusemide, aspirin, and a streptokinase infusion. He remained hypotensive and became oliguric within 24 hours of admission. The central venous pressure was 12 cm H_2O, and intravenous dopamine and dobutamine were begun. His condition failed to improve and a Swan-Ganz catheter was inserted, which disclosed a pulmonary artery wedge pressure of 20 mm Hg.

John Radcliffe Hospital, Oxford OX3 9DU
John Efthimiou, *senior registrar in medicine*
Maxton Pitcher, *senior house officer in medicine*
Oliver Ormerod, *senior registrar in cardiology*
Fiona Harper, *senior house officer in cardiothoracic surgery*
Stephen Westaby, *consultant in cardiothoracic surgery*
David Grahame-Smith, *professor of clinical pharmacology*

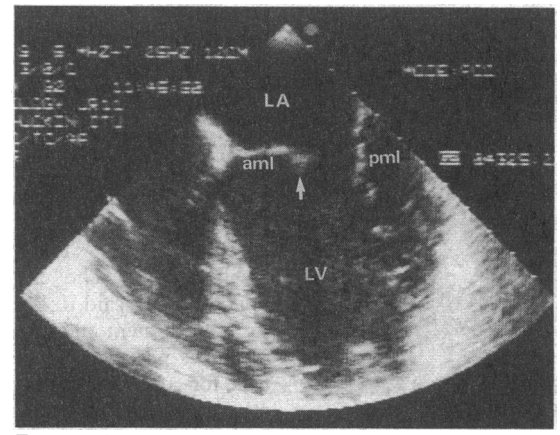
Forty eight hours after admission his serum creatinine concentration had risen from 151 to 675 μmol/l, confirming acute renal failure. He had several small haemoptyses of fresh red blood and his chest radiograph showed worsening bilateral alveolar shadowing with obvious air bronchograms. Arterial blood gas values confirmed severe type I respiratory failure with arterial oxygen pressure 6·2 kPa breathing 74% oxygen. Two dimensional transthoracic echocardiography showed good left ventricular systolic function with inferolateral hypokinesia and no evidence of mitral regurgitation. He was subsequently transferred to the intensive care unit, where haemofiltration was instituted and inotropes changed to noradrenaline and dopamine. His temperature rose to 38·9°C but cultures of blood and bronchoalveolar lavage fluid grew no pathogens. Treatment was begun with intravenous cefuroxime, gentamicin, and erythromycin. In view of the worsening shock a further transthoracic echocardiogram was obtained, which confirmed vigorous left ventricular function with no evidence of ventricular septal defect or mitral regurgitation. He later deteriorated with distressing dyspnoea and worsening hypoxaemia and was intubated and ventilated. Up to 300 ml blood was sucked up his endotracheal tube each day, but a full clotting screen showed nothing abnormal and antineutrophil cytoplasmic antibodies and antiglomerular basement membrane antibodies were not detected.

Seven days after admission his pulmonary oedema was worsening and he required a fractional inspired oxygen concentration of 100% and positive end expiratory pressure ventilation of 10 cm H_2O to maintain an arterial oxygen pressure of 10 kPa. Despite the continued absence of a heart murmur transoesophageal echocardiography was performed, which showed a flail anterior mitral valve leaflet with a ruptured papillary muscle attached (figure). Severe mitral regurgitation was evident on Doppler colour flow study. At emergency operation severe haemorrhagic pulmonary oedema was seen with a flail anterior mitral valve leaflet and ruptured papillary muscle and inferoseptal myocardial infarction caused by occlusion of a dominant right coronary artery. This was bypassed by using a reversed saphenous vein graft and a Starr-Edwards mitral valve prosthesis inserted. After initial improvement he died 48 hours after surgery.

Discussion

Papillary muscle rupture in acute myocardial infarction occurs in only 1-5% of cases.[1] Usually rupture occurs two to seven days after infarction, and half the patients die within 24 hours due to cardiogenic shock and pulmonary oedema.[2] Postmortem studies in patients with papillary muscle rupture show that the infarct is often small and usually related to single vessel coronary disease.[1] Left ventricular systolic function is usually good and surgical intervention successful provided that a prompt and definitive diagnosis is made.[3]

The sudden development of a systolic murmur after acute myocardial infarction is much more often due to papillary muscle dysfunction than to papillary muscle rupture or a ventricular septal defect.[4] Acute severe mitral regurgitation after papillary muscle rupture may result in pressure equalisation between the left atrium and left ventricle.[5] The mitral valve is then in effect functionally absent and there may be no murmur at all, as was the case in our patient. Such patients thus appear as if they have massive myocardial infarction with pump failure.



Transoesophageal echocardiogram showing left atrium (LA), left ventricle (LV), and mitral valve with anterior mitral valve leaflet (aml) and posterior mitral valve leaflet (pml). Arrow indicates ruptured papillary muscle mass attached to end of anterior mitral valve leaflet. Posterior mitral valve leaflet opened normally—that is, into left ventricle during diastole, whereas anterior mitral valve leaflet did not

Transthoracic echocardiography is a useful, non-invasive technique for diagnosing some complications of acute myocardial infarction but is not reliable for mitral regurgitation. In addition, in critically ill patients transthoracic echocardiography has several limitations, including suboptimal positioning and increased interference with ultrasound transmission when the patient is mechanically ventilated.[6,7] Two transthoracic echocardiographic examinations of our patient failed to disclose mitral regurgitation, which was subsequently diagnosed by using bedside transoesophageal echocardiography, a technique which is well recognised as providing better resolution of the mitral valve apparatus.[8-10]

The golden rule is that if in the presence of cardiogenic shock echocardiography shows vigorous left ventricular function, then there must either be a leaky mitral valve, a shunt such as a ventricular septal defect, or rarely a large aneurysm.

In conclusion, early diagnosis of papillary muscle rupture, particularly in the absence of a murmur, may be greatly facilitated by transoesophageal echocardiography, and early surgery to correct such severe mitral regurgitation is essential if the high mortality from this condition is to be reduced.

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