without recurrence of the hepatitis. This supports the concept that acute hepatitis complicating intravenous amiodarone is related to the diluent rather than the drug. On this basis we suggest amiodarone can be safely administered by the oral route even in patients who develop hepatitis with the intravenous preparation, provided hepatic function is closely monitored.

1 Lupon-Roses J, Simo-Canonge R, Lu-Cortez L, Permanyer-Miralda G, Allende-Mondus H. Probable

early acute hepatitis with parenteral amiodarone. *Clin Cardiol* 1986;9:225-9.

2 Pye M, Northcote RJ, Cobbe SM. Acute hepatitis after parenteral amiodarone administration. *Br Heart J* 1988;59:690-1.

3 Stephenson RNS, Nayam TH, Davies JR. Acute hepatic dysfunction following parenteral amiodarone administration. *Postgrad Med J* 1989;65:707-8.

4 Rhodes A, Eastwood JB, Smith SA. Early acute hepatitis with parenteral amiodarone: a toxic effect of the vehicle? *Gut* 1993;34:565-6.

5 Ruter HD. The E-ferol syndrome solved? In: Dukes MNG, Beeley L, eds. *Side effects of drugs, annual 12: a worldwide survey of new data and trends*. Amsterdam: Elsevier, 1988:329.

IMAGES IN CARDIOLOGY

Endothelial dysfunction in the absence of coronary atheroma causing Prinzmetal's angina

Angiography was performed on a 54 year old woman with no previous history of ischaemic heart disease who was admitted complaining of increasing chest pain occurring frequently at rest. She reported that her symptoms had begun two months previously following withdrawal of hormone replacement therapy (Prempak C; Wyeth, UK) which she had taken for over nine years. An electrocardiogram on admission showed minor inferolateral ST segment depression but later she developed further severe chest pain with 3 mm of acute ST elevation in leads II, III, and aVF. Urgent cardiac catheterisation was performed within 30 minutes of this episode by which time her pain and electrocardiographic changes had resolved in response to a high dose intravenous nitrate infusion. Angiography revealed completely normal coronaries with a large dominant right coronary artery (fig A) and normal left ventricular function. Subsequent provocation

tests involving intracoronary infusion of acetylcholine at concentrations of 10^{-7} and 10^{-6} M demonstrated severe diffuse spasm of the right coronary artery with virtual obliteration of the distal branches (fig B). These changes were associated with anginal pain and marked electrocardiographic ST segment depression in the inferior territory. Intravascular ultrasound imaging of the right coronary artery demonstrated that it was free from atheromatous plaque.

These images provide a dramatic illustration of Prinzmetal's angina. Diffuse distal spasm associated with endothelial dysfunction in the right coronary artery were demonstrated in the absence of subangiographic atheroma. It was appealing to speculate that oestrogen withdrawal may have contributed to the evolution of vasospasm in this case.

ID COX
J C KASKI
J R CLAGUE