dromotropic, and inotropic properties.[2] Reports of diltiazem overdose are rare but well documented.[2–9] The diagnosis of diltiazem overdose is based on clinical features affecting the central nervous system (for example, confusion, lethargy, coma, respiratory arrest), gastrointestinal system (nausea, vomiting), cardiovascular system (hypotension, bradycardias, and a variety of differing degrees of heart block), and respiratory system (non-cardiogenic pulmonary oedema).[2 3] Metabolic effects include hyperglycaemia and lactic acidosis.[2 3] The haemodynamic features in diltiazem overdose are a low systemic vascular resistance in the presence of good cardiac output.[4 5] Serum diltiazem concentrations will confirm the diagnosis, but the assay is neither readily nor rapidly available.[4] The treatment for diltiazem overdose is essentially supportive, although early haemoperfusion with intravenous calcium and charcoal may have some beneficial effect.[2–9]

Many patients associate a single drug with a single therapeutic action. They are often unaware of any of its other potential actions. Our patient perceived his drug to be predominantly for the relief of angina. This perception was reinforced after his initial self treatment with a large quantity of isosorbide mononitrate. He was indirectly aware of the coronary vasodilation that nitrates can cause but was unaware of the drug's effects on systemic vasodilation. It is likely that nitrate tolerance was the only factor that prevented his admission with hypotension on that occasion. Subsequently, the significance of the amount of diltiazem was not appreciated by the patient nor shown in a cursory drug history. Inevitably the patient's failure to respond to treatment was ascribed to insufficient treatment rather than inappropriate treatment by the admitting team.

Two lessons can be learnt from this case. Firstly, an adequate drug history should include not just the list of drugs prescribed for a patient but also the patient's compliance with them—that is, whether insufficient or excessive quantities of prescribed drugs are being taken. A more detailed drug history might have led to the earlier institution of appropriate treatment and avoided the development of potentially fatal multiple organ failure. Secondly, the earlier use of haemodynamic monitoring would have increased the suspicion of a diagnosis other than ischaemic left ventricular dysfunction. Haemodynamic monitoring by Swan-Ganz catheterisation is a widely available tool.[10] It is especially useful in hypotensive patients when the cause of haemodynamic compromise is uncertain, in certain cases of pulmonary oedema associated with hypotension, and, as in this patient, when the response to treatment is poor.[10] Confirmation of the diagnosis by measurement of haemodynamic variables prevented inappropriate, extremely high risk surgery being performed.

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Correction

Drug treatment in heart failure

Two errors occurred in this regular review by Eva Lonn and Robert McKelvie (29 April, pp 1188-92). The first sentence of the section on angiotensin converting enzyme inhibitors should read: "Angiotensin converting enzyme (ACE) inhibitors prevent the conversion of angiotensin I to angiotensin II and prevent [not cause] the degradation of bradykinin." The penultimate sentence of the penultimate paragraph should read: "The aldosterone receptor antagonist spironolactone [not aldosterone] was recently shown to improve considerably the outcomes of patients with severe heart failure."