INTRODUCTION
Pre-eclampsia and eclampsia are significant causes of maternal and fetal morbidity and mortality. Pre-eclampsia is pregnancy-induced hypertension in association with proteinuria (> 0.3 g in 24 hours), with or without oedema. Eclampsia is defined as the occurrence of one or more convulsions superimposed on pre-eclampsia. The most recent report of the confidential enquiry into maternal and child health found that pre-eclampsia and eclampsia were the second most common cause of direct maternal death in the United Kingdom. The most common cause was thrombosis and thromboembolism. Antihypertensive treatment should be started in pre-eclamptic women with a systolic blood pressure over 160 mmHg or a diastolic blood pressure over 110 mmHg. In the United Kingdom, methyldopa and labetalol are the most commonly used antihypertensive drugs. Labetalol, given orally or intravenously, nifedipine given orally or intravenous hydralazine have been recommended for the acute management of severe hypertension. Labetalol has the advantage that it can be given initially by mouth in severe hypertension and then, if needed, intravenously. Labetalol is a combined non-selective beta-blocker and alpha-blocker. When given intra-venously, the ratio of its beta-blocking effect to its alpha-blocking effect is approximately 7:1. In contrast, atenolol, a cardioselective (beta-1-selective) beta-blocker, is not recommended because it is associated with fetal growth restriction. Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor-blocking drugs (ARBs) are also contraindicated because of unacceptable fetal adverse effects.

CASE REPORT
A 42-year-old multigravida with severe pre-eclampsia had an emergency caesarean section under spinal anaesthesia. Peri-operatively, her arterial pressure was controlled with oral methyldopa and an intravenous infusion of labetalol. Post-operatively, in the Intensive Care Unit, she had recurrent episodes of hypoglycaemia which required treatment with intravenous glucose. These episodes resolved when the labetalol infusion was stopped. Clinicians should be aware of the potential of labetalol to cause hypoglycaemia.

ABSTRACT
A 42-year-old multigravida with severe pre-eclampsia had an emergency caesarean section under spinal anaesthesia. Peri-operatively, her arterial pressure was controlled with oral methyldopa and an intravenous infusion of labetalol. Post-operatively, in the Intensive Care Unit, she had recurrent episodes of hypoglycaemia which required treatment with intravenous glucose. These episodes resolved when the labetalol infusion was stopped. Clinicians should be aware of the potential of labetalol to cause hypoglycaemia.

Key words: Labetalol. Hypoglycaemia. Eclampsia. Pre-eclampsia. Beta-blockers.
cause of hypoglycaemia) and replaced by an intra-venous infusion of isosorbide dinitrate at 5-10 mg per hour (Figure 1). Two hours later, the blood glucose was 5.1 mmol/L and the intravenous glucose infusion was stopped. There were no further episodes of hypoglycaemia. The following day, the isosorbide dinitrate infusion was stopped and, subsequently, the arterial pressure was adequately controlled by oral methyldopa.

**DISCUSSION**

The sympathetic nervous system has multiple effects on carbohydrate metabolism, via activation of alpha-1, alpha-2 and beta-2 adrenergic receptors. Blockade of beta-2 adrenergic receptors would be expected to decrease glycogenolysis in the liver and in skeletal muscle and potentially cause a decrease in the plasma glucose level. In support of this, there have been many reports of hypoglycaemia secondary to therapy with propranolol, a non-selective beta-blocker. Propranolol has been shown to decrease muscle glycogenolysis and decrease glucagon secretion in response to hypoglycaemia. Hypoglycaemia may be more likely when there is hepatic glycogen depletion as a result of fasting.

Like propranolol, labetalol is a non-selective beta-blocker, but also has an alpha-blocking effect. When given intravenously, the ratio of beta-blockade to alpha-blockade is approximately 7:1. Blockade of beta-2 and alpha adrenergic receptors would both be expected to decrease glycogenolysis in the liver, while blockade of beta-2 adrenergic receptors would also be expected to decrease glycogenolysis in skeletal muscle. However, there have been few reports of labetalol-induced hypoglycaemia, other than an increased risk of neonatal hypoglycaemia secondary to maternal labetalol therapy. The British National Formulary states that beta-blockers, particularly non-selective beta-blockers, may interfere with the metabolic and autonomic responses to hypoglycaemia in diabetic patients, however, it does not state that hypoglycaemia may occur in non-diabetic subjects. Beta-blockers, particularly non-selective beta-blockers, may be associated with hypoglycaemia in non-diabetic subjects. In the patient described in this case report, glycogen depletion secondary to fasting and the high dose of intravenous labetalol may have increased the risk of hypoglycaemia. Clinicians should be aware of this potential side effect of labetalol.

**REFERENCES**