COLLAPSE DURING OPERATION FOLLOWING I.V. ERGOMETRINE
A case report

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SUMMARY
Peripheral pulses became impalpable following the administration of ergometrine during Caesarean section. The subsequent clinical course suggested sensitivity to ergometrine in a patient with Raynaud's disease.

CASE REPORT
A female Caucasian patient in her third pregnancy was booked, at 9 weeks of gestation, for hospital confinement. Her previous confinements were unremarkable. Asthma, which had responded to desensitizing agents, was noted in her previous medical history. Her sensitivity to oxytocic drugs was not known. Pre-eclamptic toxaemia, noted at 34 weeks of gestation, responded to bed rest until 39 weeks of gestation when oedema precipitated a bilateral carpal tunnel syndrome. Antenatal glycosuria was present as a result of a reduced renal threshold.

At 39 weeks of gestation, labour was induced by forewater amniotomy, and oxytocin (Syntocinon) was given to augment labour. Four hours following induction of labour, foetal bradycardia (40 beat/min) occurred. A prolapsed cord was confirmed and a Caesarean section was performed.

Anaesthesia was induced with methohexitone 80 mg followed by suxamethonium 75 mg. Following endotracheal intubation, pancuronium 5 mg was given. Trichloroethylene 0.4% decreasing to 0.2% in 60% nitrous oxide in oxygen was delivered from a Manley ventilator for the maintenance of anaesthesia. No narcotic analgesic was administered. At the commencement of anaesthesia the arterial pressure was 125/80 mm Hg and it settled at 110/75 mm Hg following the induction of anaesthesia.

Ergometrine 0.5 mg was administered i.v. following delivery of the child. Shortly afterwards, the peripheral arterial pulses were noted to be absent, although the carotid pulse remained palpable at 80 beat/min and the e.c.g. signal was regular in rhythm and pattern. The peripheral perfusion remained adequate with warm, pink hands and feet. Atropine 0.6 mg was administered and the operation was completed. The myoneural block was antagonized successfully with atropine 1.2 mg and neostigmine 2.5 mg.

Following operation the patient remained drowsy but responsive. A neurological examination revealed no abnormality, but the peripheral pulses remained impalpable and the arterial pressure was unrecordable. The carotid pulses were palpable and the jugular venous pulse was normal. The apex beat had increased to 100 beat/min without a gallop rhythm. The respiratory frequency was normal and there were no abnormalities on auscultation of the chest. There was no cyanosis. There was no evidence of an abnormal blood loss and subsequently the haemoglobin concentration was found to be 12.9 g/d litre. The e.c.g. and chest x-ray revealed no abnormality and arterial Po2, Pco2 were within the normal ranges.

Two hours after the collapse all the peripheral pulses had returned, the patient's condition remaining satisfactory. A history elicited from her husband was suggestive of Raynaud's phenomenon occurring before this pregnancy. Therefore the possibility of sensitivity to ergometrine was considered. The recovery after operation was unremarkable although at follow-up the patient described further episodes suggestive of Raynaud's phenomenon.

DISCUSSION
Ergot alkaloids act centrally and peripherally and it is accepted that they may induce peripheral vasoconstriction (Johnstone, 1972; Goodman and Gilman, 1975). This may occur following therapeutic dosage, especially if hepatic disease, pruritis, pre-eclamptic toxaemia or peripheral vascular disease co-exist (Kenney, 1946; Johnstone, 1972; Goodman and
Gilman, 1975). The precipitation of Raynaud's phenomenon in sensitive subjects has been suggested previously (Mann, 1961). These effects have been described only rarely in association with ergometrine (Bross et al., 1963), although one death has been associated with the use of i.v. ergometrine (Report, 1970-72). However, ergometrine has been shown to have a vasoconstrictive effect even in normal subjects (Brook and Robinson, 1970). More recently, these effects have been confirmed in vitro and in vivo with a possible potentiation of effect in association with some of the anaesthetic agents (Wassef, Lal and Pleuvry, 1974).

The patient described in this report developed absent peripheral pulses for which no definite cause was found. Recovery was complete and the timing of events suggests that ergometrine sensitivity was the causal factor. However, this interpretation can only be speculative. The other ampoules from the same box had been used without abnormal effect.

We feel that patients with a history of Raynaud's phenomenon should be observed carefully following the administration of ergometrine and that caution should be exercised in the administration of this drug routinely in the third stage of labour.

REFERENCES