Successful pregnancy soon after oral contraceptive-associated malignant hypertension

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Summary
A woman who developed malignant hypertension while taking a very low oestrogen oral contraceptive underwent an uncomplicated pregnancy conceived 3 months later. Her BP was well controlled with propranolol alone.

Introduction
Successful pregnancy after the development of malignant hypertension has been reported on 2 occasions (Kincaid-Smith, McMichael and Murphy, 1958; Weir and Willocks, 1976). In these patients conception occurred 4 and 3 years respectively after control of the malignant phase. The authors report a successful pregnancy in a patient who developed malignant hypertension while taking an oral contraceptive and who conceived 3 months later. Throughout pregnancy her BP was well controlled with propranolol.

Case report
A 21-year-old woman had taken Micropogen 30 (Schering) (0·15 mg levonorgestrel and 0·03 mg ethinylestradiol) for 3 years. She was admitted in June 1977 with a history of severe headache for one week and vomiting for one day. The BP was 230/170 mmHg and bilateral papilloedema, haemorrhages and soft exudates were seen on fundoscopy. Microscopic haematuria was present. Investigations revealed a creatinine clearance of 40 ml/min with a serum urea of 3·8 mmol/l and a serum potassium of 3·2 mmol/l. The latter returned to normal following treatment. ECG showed left ventricular hypertrophy. IVP, isotope renogram, urinary VMA and catecholamine excretion were all normal.

The BP was initially controlled with i.v. diazoxide. Oral propranolol was started and the patient was discharged 3 weeks later taking propranolol 640 mg daily. The dose of propranolol was increased to 960 mg following discharge and subsequent BP control was satisfactory. Two months after ward discharge her BP was 140/94 mmHg (standing values quoted) and the fundi appeared normal apart from a few old exudates. The contraceptive pill was discontinued on admission to hospital.

Three months later the patient was found to be 9 weeks pregnant. She had been strongly advised to avoid pregnancy and some contraception had been organized. At this ante-natal visit her BP was 120/80 mmHg, fundoscopy normal, and creatinine clearance 103 ml/min. During pregnancy her BP was maintained between 110–143/75–80 mmHg on a propranolol dose of 640 mg. Serum urea, uric acid, blood platelet count, and urinalysis for protein were normal throughout the pregnancy.

Fetal growth was monitored biochemically and by ultrasound. Serial urinary total oestrogens and plasma placental lactogen (HPL) remained within normal range until 38 weeks' gestation when the HPL fell to 3·4 mg/l. A reduction in fetal growth as measured by serial cephalometry was initially noted at 34 weeks and continued over the following 4 weeks. During the 37th week a reduction in fetal activity was reported and the patient was immediately admitted to hospital. Cardiotacography (external) revealed a baseline fetal bradycardia of 100–115 beats/min but this rate responded by more than 20 beats/min on fetal movement. The maternal resting heart rate was between 56–64 beats/min. In view of the reduction in fetal activity and objective evidence of fetal growth retardation, delivery was planned during the 38th week of gestation. Propranolol was stopped 36 hr before delivery without loss of BP control. The fetal heart rate 24 hr after propranolol had been stopped was 120–130 beats/min. Vaginal examination revealed a grossly unfavourable cervix (Bishops score 2) for induction and delivery by elective Caesarean section under epidural anaesthesia was undertaken.

The delivered male infant was small for dates, weighed 2·6 kg (5th centile weight for gestation) and had an Apgar score of 9 and 10 at one and 5 min.
The cord venous blood pH was 7.34 and the base deficit 2.1. The placenta weighed 420 g and was histologically normal. The infant's blood sugar was presumably related to the β-blocker. The bradycardia was reversed by fetal activity and disappeared soon after stopping the drug. Omission of propranolol for 4 days did not affect BP control, a feature recorded by Kristensen, Steiness and Weeks (1978). In retrospect, however, stopping the β-blocker before Caesarean section seems unnecessary.

It appears that a past history of malignant hypertension is not a contra-indication to pregnancy when renal function is good and the BP is well controlled.

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**References**


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