gamete transfer to avoid missing such abnormalities. If an accessory ostium is found, the catheter must be seen to pass beyond the junction of the two ostia to ensure that the gametes are not deposited in a blind pouch.

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Relation between maternal-to-fetal blood glucose gradient and uterine and umbilical Doppler blood flow measurements

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In fetal blood obtained by cordocentesis the mean umbilical vein blood glucose concentration in appropriately grown fetuses is higher than in the umbilical artery, indicating that there is fetal glucose uptake from the placenta (Economides & Nicolaides, 1989). Similarly, the maternal glucose concentration is higher than the fetal and the levels in the two compartments are significantly associated, confirming that the fetus is supplied with maternal glucose (Economides & Nicolaides, 1989). Furthermore, cordocentesis has shown that some small for gestational age (SGA) fetuses are hypoglycaemic and that this is probably not due to decreased gluconeogenesis or increased glucose consumption. A possible mechanism for the low fetal glucose values is reduced utero-placental and/or feto-placental blood flow.

The aim of the present study was to investigate the relation between the maternal to fetal blood glucose gradient (maternal venous minus umbilical vein blood glucose concentration) and Doppler ultrasound measurements of the impedance to flow in the uterine and umbilical circulations in pregnancies with SGA fetuses.

Patients and methods

Uterine artery and umbilical artery flow velocity waveforms (FVWs) were obtained by continuous wave Doppler ultrasound within 30 min before cordocentesis in 30 pregnancies with SGA fetuses. The patients were referred for fetal karyotyping and blood gas analysis because of suspected severe fetal growth retardation. Ultrasound examination in our unit confirmed that the fetal abdominal circumferences were 2·5–10·3 SD below the normal mean for gestation. In all the pregnancies included in this study the fetuses were morphologically and

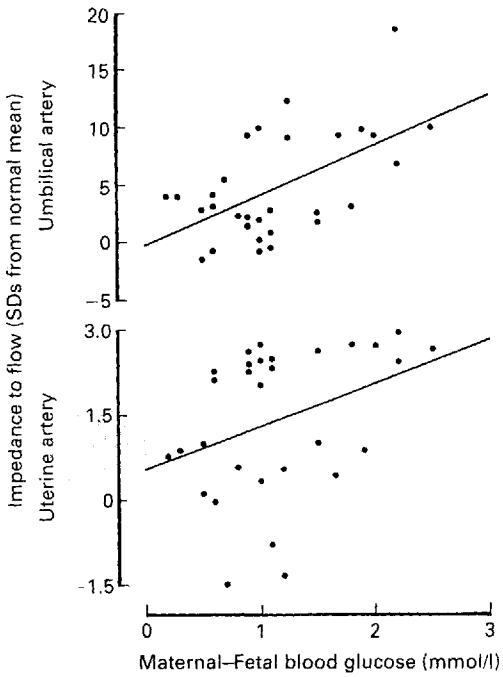

Fig. 1. Relation of maternal to fetal blood glucose concentration gradient and the impedance to flow (in SDs from the normal mean for gestation) in the umbilical and uterine arteries (the diagonal lines are the regression lines).

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chromosomally normal and the diagnosis of SGA was confirmed subsequently by a birthweight below the 3rd centile (Yudkin *et al.*, 1987). Indeed 10 pregnancies ended in perinatal death reflecting the severity of the growth retardation.

FVW were recorded from both the right and left uterine arteries (Aristidou *et al.*, 1990) and umbilical artery (Nicolaides *et al.*, 1988), and the resistance index (RI) and pulsatility index (PI) were calculated respectively, using the Doptek Spectrascan analyser 9000 (Doptek Ltd, Chichester, UK).

Cordocentesis was performed as an outpatient procedure without maternal fasting and sedation or fetal paralysis (Nicolaides *et al.*, 1986). Umbilical vein blood (25μl) was collected using sodium fluoride as an anticoagulant (Vacutainer, Rutherford, New Jersey, USA) and the blood glucose concentration was measured using a Yellow Springs analyser (Yellow Springs Instrument Co, Yellow Springs, Ohio 45387, USA). The blood glucose concentration was also measured in maternal blood taken from an antecubital vein immediately before fetal blood sampling.

Since the uterine artery (UA) RI and the umbilical artery (UmbA) PI change with gestation (Aristidou *et al.*, 1990), the values in the SGA fetuses were expressed as the differences in SDs from the normal mean for gestation (UAΔRI and UmbAΔPI respectively). Regression analysis was used to determine the relation between the maternal to fetal glucose gradient and the UAΔRI and UmbAΔPI. The differences between SGA and AGA pregnancies were tested by the Mann–Whitney *U*-test.

Results

The mean maternal-to-umbilical vein blood glucose gradient in the SGA pregnancies was 1·2 (SD 0·65) mmol/l, which was significantly higher (z=5·52, P<0·0001) than the values from 91 pregnancies with AGA fetuses (mean 0·51, SD 0·34 mmol/l), reported by Economides & Nicolaides (1989) and the gradient was significantly associated with both the UAΔRI (Fig. 1; r=0·37, n=30, P<0·05), and the UmbA△PI (Fig. 1; r=0·55, n=30, P<0·01). Multiple regression analysis demonstrated that UmbAΔPI and UAΔRI explain 39·9% of the variance in maternal-to-fetal glucose gradient [gradient = 0·587 + 0·068 (UmbAΔPI) + 0·16 (UAΔRI); residual SD=0·469].

Discussion

Some SGA fetuses have high maternal-to-umbilical vein blood glucose gradient and the degree of hypoglycaemia is proportional to the degree of abnormality in the blood flow impedance measurements in both the uterine and umbilical circulations. These findings provide supportive evidence that fetal hypoglycaemia may result from reduced fetal glucose uptake due to impaired blood flow on both sides of the placenta. However, the degree of fetal hypoglycaemia is associated more significantly with the umbilical than uterine blood flow impairment. This is not surprising in animal studies microsphere embolization of the placenta will only result in growth retardation (Clapp *et al.*, 1980) and reduced glucose uptake (Clapp *et al.*, 1981) when umbilical blood flow is also reduced.

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