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Prediction of fetal acidaemia in pregnancies complicated by maternal diabetes mellitus by biophysical profile scoring and fetal heart rate monitoring

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ABSTRACT

Objective To determine whether computer assisted fetal heart rate analysis or the biophysical profile score can provide noninvasive prediction of fetal acidaemia.

Design Cross sectional study.

Setting Harris Birthright Research Centre for Fetal Medicine, King's College Hospital School of Medicine, London.

Subjects Forty-one women with pregnancies complicated by diabetes mellitus.

Interventions Fetal heart rate (FHR) monitoring with computer assisted analysis, biophysical profile score (BPS) and cordocentesis for measurement of umbilical venous blood glucose concentration and blood gases, up to 24 h before delivery at 27 to 39 weeks gestation.

Results The mean umbilical venous blood pH was significantly lower than the normal mean for gestation, and was below the 5th centile in 18 pregnancies, including all six cases where the mother had nephropathy and hypertension. The mean pO_2 was not significantly different from the normal mean for gestation. There were significant associations between fetal acidaemia and both the BPS ($r = 0{\cdot}46$, $P<0{\cdot}01$) and FHR variation ($r = 0{\cdot}42$, $P<0{\cdot}01$). However, of the 12 acidaemic fetuses of non-nephropathic mothers, nine had normal BPS and six had normal FHR variation.

Conclusions In pregnancies complicated by maternal diabetes mellitus, BPS and FHR variation are of limited value in the prediction of fetal blood pH.

In pregnancies complicated by maternal diabetes mellitus, analysis of blood samples obtained by cordocentesis has demonstrated that some fetuses are acidaemic (Bradley *et al.* 1991; Salvesen *et al.* 1992). The aim of the present study was to investigate whether computer assisted fetal heart rate (FHR) analysis or the biophysical profile score (BPS) can provide noninvasive prediction of the fetal blood gas results.

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Subjects and methods

In 41 women with established ($n = 31$) or gestational ($n = 10$) diabetes mellitus, umbilical venous blood was obtained by cordocentesis which was performed immediately after FHR monitoring and biophysical profile scoring, and up to 24 h before elective delivery at 27 to 39 weeks gestation. Ultrasound examination demonstrated normal fetal anatomy confirmed postnatally in all cases. All women gave their written informed consent to the study which was approved by our hospital ethics committee.

The women were recruited from our combined diabetic antenatal clinic and all were insulin treated. In the group

Table 1. Details of individual women showing: White class (White; GDM = gestational diabetes mellitus); gestation at delivery in weeks (GE): maternal glycosylated haemoglobin percentage (HbA_1); fetal heart rate variation (mean minute range in msec (MMR), *denotes those women with decelerations); fetal breathing movements (RM); amniotic fluid volume (AF); fetal body movements (BM); fetal tone (Tone); fetal heart rate reactivity (FHR); fetal umbilical venous blood glucose concentration (g/dl; GLUC); pH and pO_2 in mmHg at cordocentesis; and birthweight (BWT) in kg.

	Maternal			Biophysical profile score							Umbilical venous blood			
Case no.	White	GE	HbA_1	MMR	RM	AF	BM	Tone	FHR	Total	GLUC	pH	pO_2	BWT
1	GDM	39	9·2	42·4	2	2	2	2	2	10	3·1	7·411	36·4	3·81
2	GDM	38	9·3	33·5	2	2	2	2	2	10	5·3	7·430	36·1	3·64
3	GDM	38	8·8	55·8	2	2	2	2	2	10	3·5	7·322	36·0	3·19
4	GDM	39	8·9	26·9*	0	2	2	2	0	6	3·2	7·346	32·4	3·46
5	GDM	39	5·5	37·7	2	2	2	2	2	10	2·7	7·391	32·1	3·42
6	GDM	37	8·0	67·2	2	2	2	2	2	10	3·7	7·424	32·1	2·94
7	GDM	38	10·2	62·3	2	2	2	2	2	10	4·1	7·352	36·7	3·14
8	GDM	38	11·9	32·4	2	2	2	2	2	10	5·8	7·354	30·6	3·72
9	GDM	38	8·0	61·3	2	2	2	2	2	10	6·7	7·327	36·8	3·49
10	GDM	38	9·0	47·3	2	2	2	2	2	10	5·3	7·380	33·5	3·32
11	B	39	8·8	34·7	2	2	2	2	2	10	4·2	7·342	23·6	2·82
12	B	39	8·1	45·4	2	2	2	2	2	10	4·1	7·358	35·1	3·74
13	B	38	7·1	47·3	2	2	2	2	2	10	4·7	7·365	34·0	3·68
14	B	38	6·4	42·0	2	2	2	2	2	10	1·4	7·367	32·3	3·78
15	B	38	10·2	31·6*	2	2	2	2	0	8	3·4	7·390	35·5	3·16
16	B	37	8·9	17·9	2	2	2	2	0	8	3·5	7·342	21·6	3·80
17	B	38	10·8	43·9	2	2	2	2	2	10	3·0	7·391	42·2	3·40
18	C	36	6·3	44·4	2	2	2	2	2	10	4·3	7·369	33·8	2·95
19	C	39	8·9	32·5	2	2	2	2	2	10	3·0	7·383	30·2	3·42
20	C	38	7·9	70·1	2	2	2	2	2	10	2·8	7·389	35·2	3·80
21	C	39	11·1	51·0	2	2	2	2	0	8	1·2	7·403	33·5	3·74
22	C	38	9·0	58·1	2	2	2	2	2	10	2·2	7·426	39·3	3·40
23	C	39	6·6	40·6	2	2	2	2	2	10	2·5	7·395	36·2	3·70
24	C	36	8·4	61·5	2	2	2	2	2	10	8·0	7·335	38·5	4·21
25	C	38	7·6	45·5	2	2	2	2	0	8	6·0	7·403	33·2	3·30
26	C	37	9·2	41·2	2	2	2	2	2	10	3·1	7·340	33·2	5·32
27	C	38	8·5	28·9	2	2	2	2	2	10	8·8	7·301	27·9	4·10
28	C	37	9·9	23·6	0	2	0	2	0	4	5·6	7·273	26·7	2·14
29	C	38	7·8	28·8	2	2	2	2	2	10	5·6	7·394	37·5	4·20
30	C	38	10·6	48·0	2	2	2	2	2	10	4·8	7·347	38·1	3·58
31	C	38	10·7	24·6	2	2	2	2	0	8	2·9	7·406	31·7	3·38
32	C	38	7·6	42·4	2	2	2	2	2	10	5·5	7·356	37·3	4·04
33	C	38	7·2	17·5*	0	2	2	2	0	6	8·5	7·334	29·0	3·66
34	C	38	9·6	32·3	2	2	2	2	2	10	3·9	7·353	29·1	4·35
35	D	38	7·9	28·1	2	2	2	2	0	8	4·7	7·326	22·3	2·65
36	F	32	7·2	33·8	0	2	2	2	0	6	2·8	7·320	25·3	2·08
37	F	31	8·7	26·4	2	2	2	2	0	8	10·1	7·298	18·7	1·80
38	F/R	27	11·3	22·0*	2	2	2	2	0	8	9·5	7·370	24·5	0·84
39	F/R	30	11·2	32·3	2	2	2	2	0	8	1·7	7·362	31·4	1·42
40	F/R	35	7·4	23·3	2	2	2	2	0	8	10·3	7·228	15·0	1·98
41	F/R	27	7·4	20·6*	0	2	2	2	0	6	8·8	7·309	15·1	0·67

with established diabetes according to the White classification (White 1978), seven belonged to group B, 17 to group C, one to group D, two to group F and four to group F/R (Table 1). In two cases, elective delivery was undertaken at 36 weeks gestation as they previously had had late intrauterine deaths. In 33 pregnancies the antenatal course was uncomplicated and according to our current policy elective delivery was undertaken at 37 to 39 weeks gestation. In the other six, the women had nephropathy and delivery was undertaken at 27 to 35 weeks gestation because of worsening hypertension and proteinuria. Induction of labour was undertaken in 16 women; 11 had vaginal deliveries and five had emergency caesarean sections for suspected cephalopelvic disproportion ($n = 1$) or abnormal intrapartum FHR patterns ($n = 4$). In 25 women, elective caesarean section was performed because of previous caesarean delivery ($n = 15$), congenital abnormality of the maternal pelvis ($n = 1$), fetal macrosomia ($n = 1$), breech presentation ($n = 2$) or prematurity ($n = 6$). All the infants survived. During the same period (August 1990 to March 1992), there were another 31 diabetic women who were delivered at 37 to 39

weeks gestation who did not have cordocentesis, either because they did not agree to participate in the study (n = 15) or because they had spontaneous onset of labour (n = 16).

Gestational age was established from the maternal menstrual history and confirmed by an early ultrasound scan (n = 35) or by an early ultrasound scan in those with uncertain dates (n = 6). Cordocentesis was performed without fetal paralysis or maternal fasting or sedation and all procedures were uncomplicated (Nicolaides *et al.* 1989). Fetal blood was collected into a heparinised syringe (350 µl) for blood gas analysis (Radiometer ABL330, Copenhagen, Denmark), and for measurement of whole blood glucose concentration (YSI 23A, Yellow Springs Instruments, Ohio 45387, USA). Kleihauer-Betke testing confirmed that all samples contained only fetal blood. At cordocentesis, a maternal venous blood sample was also taken from the antecubital fossa for measurement of blood glucose concentration and glycosylated haemoglobin percentage (Corning scanner, Corning, Halstead, UK).

During the 90 min before cordocentesis, FHR monitoring and biophysical assessment (Manning *et al.* 1980) were performed. FHR was recorded for 60 min with the women in a semirecumbent position using a Hewlett-Packard 8040 cardiotochograph (Hewlett-Packard, Boblingen, Germany). The first 20 min of the FHR recording was analysed visually for use in the BPS. The BPS was considered normal, equivocal or abnormal if the score was ≥8, 6 or ≤4, respectively.

The Sonicaid System 8000 (Sonicaid Ltd, Oxford, UK) was used for on-line computer assisted analysis of the 60 min FHR trace to reduce the intra- and inter-observer variation associated with visual analysis, and to give a reproducible score for each FHR trace for statistical analysis (Dawes *et al.* 1991). In the first analysis, a baseline was fitted and each minute was divided into 16 epochs. The overall FHR variation was calculated by averaging the mean pulse intervals of individual epochs and expressed as the mean minute range (MMR) in msec (Dawes *et al.* 1991).

By comparison with established reference ranges, the mean FHR variation, blood gases and birthweights in the diabetic pregnancies were expressed as the number of standard deviations (SD) by which individual values differed from the appropriate normal mean for gestation (delta value) (Worth *et al.* 1985; Yudkin *et al.* 1987; Nicolaides *et al.* 1989; Snijders *et al.* 1990). Unpaired Students's t test was applied to determine whether the mean of the measured variables differed from that of the reference ranges for normal pregnancies. The correlation coefficients, derived from regression analysis, were then used to determine the significance of any associations between delta umbilical venous blood gases and fetal and maternal blood glucose concentrations, delta maternal glycosylated haemoglobin percentage, delta FHR variation and BPS.

Results

Maternal details, FHR variation, biophysical profile scores, umbilical venous blood gas results and birthweights are shown in Table 1. In pregnancies complicated by maternal diabetes mellitus, the mean birthweight and maternal glycosylated haemoglobin concentration were significantly increased (mean difference = 0·595 SD, t = 2·89, P<0·01; mean difference = 2·585 SD, t = 7·68; P<0·0001, respectively). The mean umbilical venous blood pH was lower, but the mean pO_2 was not signifi-

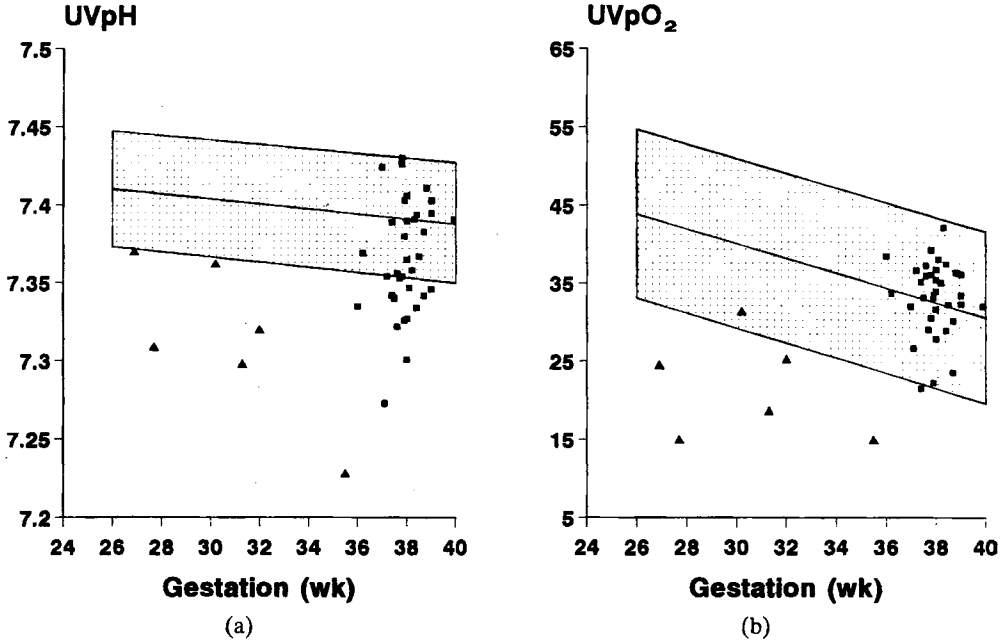

Fig. 1. Umbilical venous (UV) blood pH (a) and (b) pO_2 at cordocentesis in 41 pregnancies complicated by maternal diabetes mellitus, including six in which the mother had nephropathy and hypertension (▲), plotted on the appropriate reference range (mean, 95th and 5th centiles) for gestational age.

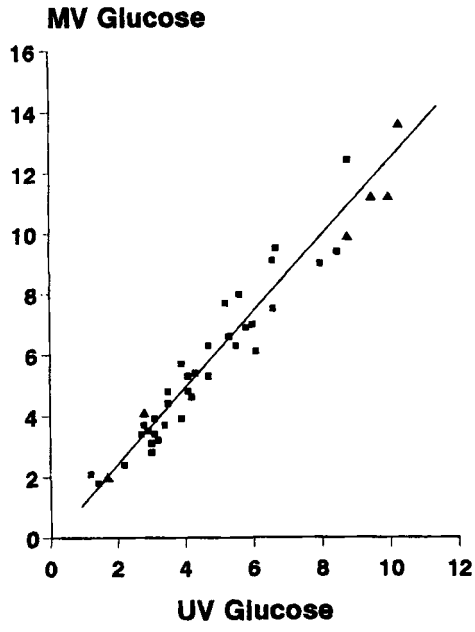

Fig. 2. Relation between maternal venous (MV) and umbilical venous (UV) blood glucose concentrations in 41 pregnancies complicated by maternal diabetes mellitus, including six in which the mother had nephropathy and hypertension (▲). r = 0·968 and $P<0·0001$.

cantly different from the appropriate normal mean for gestational age (mean difference = −1·38 SD, t = −5·00, $P<0·0001$ and mean difference = −0·327 SD, t = −1·66, P = 0·10, respectively) (Fig. 1).

Maternal blood glucose concentration was significantly associated with umbilical venous blood glucose (r = 0·968, $P<0·0001$) (Fig. 2). Both maternal and fetal blood glucose concentrations were significantly associated with umbilical venous blood delta pH: (r = −0·629, $P<0·0001$ and r = −0·603, $P<0·0001$, respectively) (Fig. 3); umbilical venous blood delta pO_2 (r = −0·506, $P<0·001$ and r = −0·536, $P<0·001$, respectively) (Fig. 4). There were no significant associations between maternal glycosylated haemoglobin concentration and either delta umbilical venous blood pH or pO_2 (r = 0·095 and r = 0·046, respectively).

The BPS was normal in 36 women, equivocal in four and abnormal in one (Fig. 5). Although there was a significant association between the BPS and delta umbilical venous blood pH (r = 0·46, $P<0·01$) (Fig. 5), the BPS was ⩾8 in 32 of the 35 pregnancies delivered at term, including nine of the 12 with acidaemic fetuses. The BPS was also normal in four of the six women with nephropathy that were delivered at 27 to 35 weeks; in all six pregnancies the fetuses were acidaemic or hypoxaemic, or both.

The mean FHR variation of the diabetic pregnancies was significantly lower than the normal mean for gestational age (mean difference = −1·08 SD, t = −4·15, $P<0·001$) (Fig. 6). Although there was a significant association between delta FHR variation and delta umbilical venous blood pH (r = 0·42, $P<0·01$) (Fig. 6), the FHR variation was normal in 24 of the 35 pregnancies delivered at term, including six of the 12 with acidaemic fetuses. The FHR was also normal in two of the six women with nephropathy.

Discussion

This study demonstrates that in pregnancies complicated by maternal diabetes mellitus some fetuses are acidaemic.

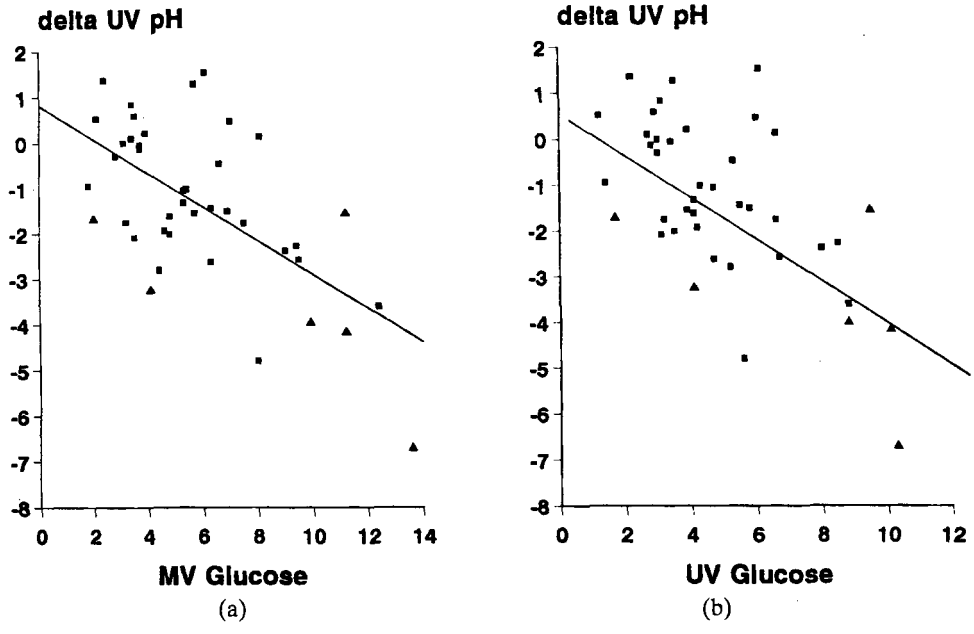

Fig. 3. Relation between (a) maternal venous (MV) (r = 0·629, $P<0·0001$) and (b) umbilical venous (UV) (r = 0·603, $P<0·0001$) blood glucose concentration and UV acidaemia (value of delta pH given as standard deviation (SD) from the normal mean for gestational age) in 41 pregnancies complicated by maternal diabetes mellitus, including six in which the mother had nephropathy and hypertension (▲).

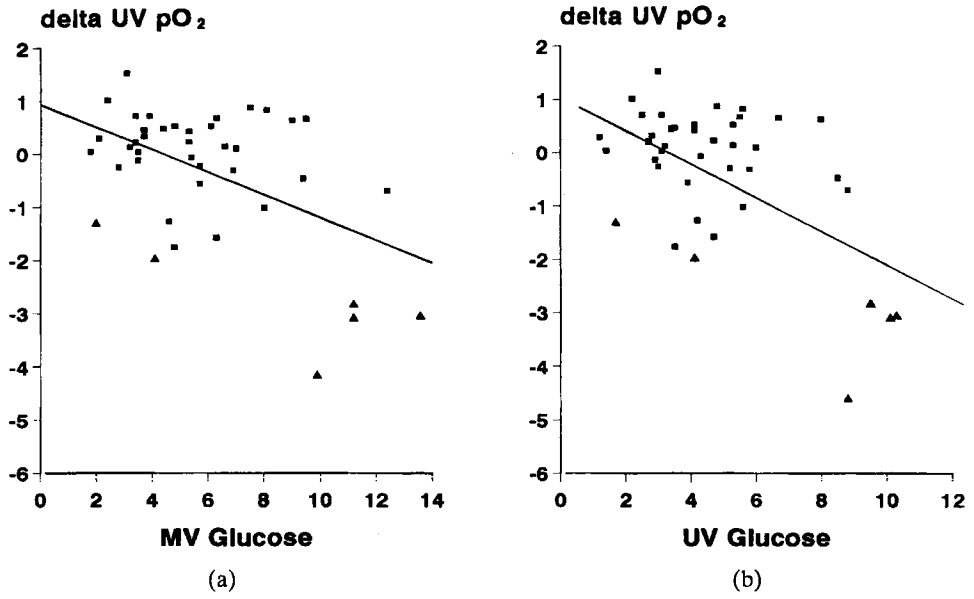

Fig. 4. Relation between (a) maternal venous (MV) (r = 0·506, $P<0·001$) and (b) umbilical venous (UV) (r = 0·536, $P<0·001$) blood glucose concentration and UV hypoxaemia (value of delta pO_2 given as standard deviation (SD) from the normal mean for gestational age) in 41 pregnancies complicated by maternal diabetes mellitus, including six in which the mother had nephropathy and hypertension (▲).

The finding of acidaemia in the absence of hypoxaemia is compatible with results of animal studies which demonstrated that minor elevations in fetal blood glucose are associated with acidaemia alone (Robillard *et al.* 1978). The finding of a significant association between fetal umbilical venous blood pH and both fetal and maternal glucose concentration, but not maternal glycosylated haemoglobin concentration, indicates that the degree of fetal acidaemia may be related to short term rather than long term glycaemic control.

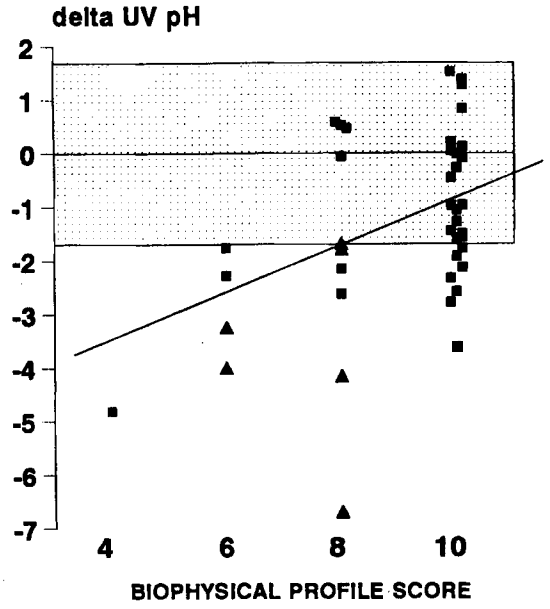

Fig. 5. Relation between umbilical venous (UV) acidaemia (value of delta pH given as SD from the normal mean of gestational age) and the biophysical profile score in 41 pregnancies complicated by maternal diabetes mellitus, including six in which the mother had nephropathy and hypertension (▲). The shaded area represents the reference range (5th–95th centile) of UV pH for normal pregnancies. r = 0·46, $P<0·01$.

In contrast to the 35 fetuses from uncomplicated diabetic pregnancies, fetal acidaemia was accompanied by hypoxaemia in five of the six fetuses of women with nephropathy and hypertension. Therefore, in the latter group the fetal acidaemia may be a consequence of placental insufficiency as well as the metabolic effects of maternal diabetes mellitus.

FHR monitoring and the BPS are widely accepted methods of antenatal fetal assessment in high risk pregnancies and several studies have demonstrated that in diabetic pregnancies a reactive nonstress test or a normal BPS before delivery reliably predict normal perinatal outcome as defined by survival, high Apgar scores, or normal neonatal metabolic balance (Ammala & Variniemo 1983; Golde *et al.* 1984; Olofsson *et al.* 1986; Dicker *et al.* 1988; Johnson *et al.* 1988).

The data of the present study indicate that both the BPS and FHR variation provide poor prediction of fetal acidaemia. One possible explanation for these findings is that the degree of fetal acidaemia was mild. Nevertheless, in intrauterine growth retardation similar degrees of fetal acidaemia were associated with suboptimal or pathological FHR patterns or fetal BPS (Ribbert *et al.* 1990, 1991; Visser *et al.* 1990). In uteroplacental insufficiency fetal nutrition and oxygenation are impaired, leading to growth retardation and acidaemia with hypoxaemia which are accompanied by reduced fetal breathing and urine production (Ribbert *et al.* 1990). In contrast, in diabetic pregnancies fetal acidaemia is associated with maternal

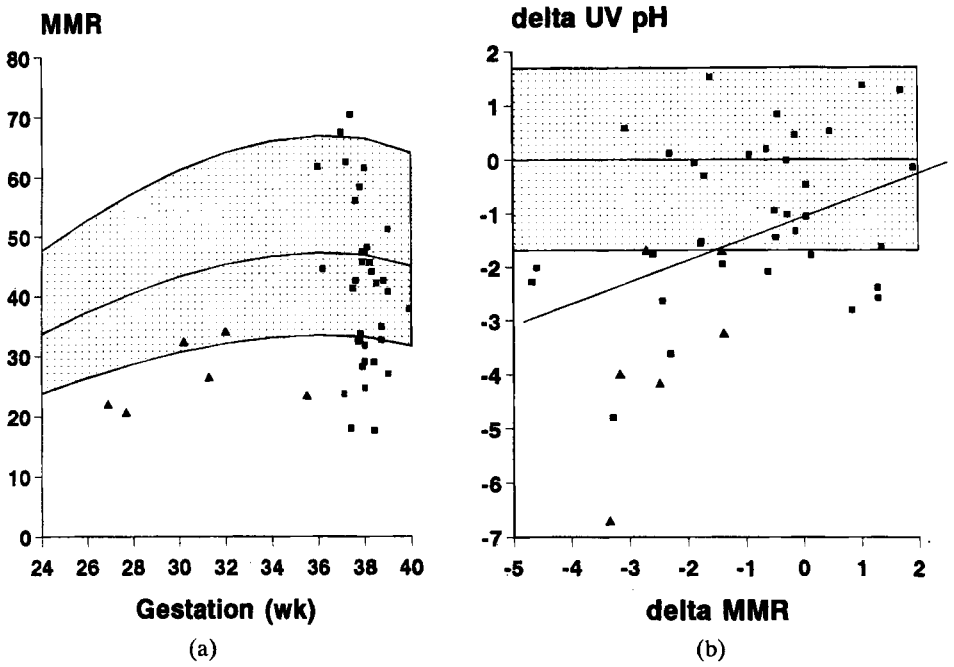

Fig. 6. Fetal heart rate variation (mean minute range in m sec, MMR) plotted on the appropriate reference range (mean, 95th and 5th centiles) for (a) gestational age (weeks), and (b) the relation between delta mean minute range (delta MMR) and delta umbilical venous blood pH (delta UVpH) (r = 0·42, $P<0·01$) in 41 pregnancies complicated by maternal diabetes mellitus, including six in which the mother had nephropathy and hypertension (▲). The shaded area in the graph on the right represents the reference range (5th–95th centile) of UV pH for normal pregnancies.

hyperglycaemia, and both the amniotic fluid volume and fetal breathing movements are either normal or increased (Roberts *et al.* 1980; Lunell 1986). In this respect, normal fetal breathing and amniotic fluid volume may provide false reassurance of fetal wellbeing, not only in uncomplicated diabetic pregnancies at term but also in diabetic pregnancies with the added complication of placental insufficiency.

Encouraged by normal tests of fetal wellbeing several authors have advocated that diabetic pregnancies should be managed expectantly, reducing the need for early delivery (Drury *et al.* 1983; Johnson *et al.* 1988). None of the studies, however, have had sufficiently large numbers to demonstrate that such a policy will not increase the occurrence of unexplained, late intrauterine deaths, since the incidence of this well recognised complication of diabetic pregnancies is less than four per 1000 births (Brudenell & Doderidge 1989). Indeed, there are several reports of apparently unexplained stillbirths within 24 h of reactive nonstress testing or a negative contraction stress test (Evertson *et al.* 1978; Shaxted & Jenkins 1981). Since there is a strong association between maternal glucose concentration and fetal blood pH, it is possible that acute elevations in maternal blood glucose could result in sudden, severe fetal acidosis and death. If this were true, it would not be surprising that fetal death may occur soon after normal assessment.

This study has demonstrated that in pregnancies complicated by maternal diabetes some fetuses are acidaemic. Furthermore, BPS and FHR variation are of limited value in the prediction of fetal blood pH. If unexplained fetal death is a consequence of acute acid base disturbance, then these methods of antenatal assessment are unlikely to identify reliably the women at risk and may indeed provide false reassurance of fetal wellbeing.

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