Fetal activity and fetal wellbeing: an evaluation

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Summary

The clinical value of the 12-hour daily fetal movement count (DFMC) as a test of antepartum fetal wellbeing was assessed. The lowest 2.5% of 1654 DFMCs recorded by 61 women who subsequently delivered healthy infants fell below 10 movements per 12 hours. This level was taken as the lower limit of normal for clinical purposes. A normal DFMC in a population at risk was associated with a satisfactory fetal outcome. A low DFMC was associated with a high incidence of fetal asphyxia, and when fetal death occurred fetal movements rapidly diminished and stopped 12 to 48 hours before death. The DFMC is a generally applicable method of monitoring fetal welfare during pregnancy which provides an inexpensive adjunct or even an alternative to the more expensive placental function tests in current use.

Introduction

The relation between diminished fetal activity and fetal asphyxia is the subject of increasing investigation. Sadovsky and Yaffe¹ observed in two patients that before intrauterine fetal death fetal movements decreased or stopped before the heart stopped. Mathews² suggested that a history of reduced fetal activity indicated serious fetal danger. Reinold,³ using ultrasound to record fetal activity, found that in cases of disturbed fetal welfare spontaneous movements were absent, infrequent, or sluggish. Maternal observations are subjective, with all the disadvantages that this implies, but about 87% of fetal movements recorded on an electromagnetic device were felt by the mother.⁴

We attempted to assess the clinical value of the daily fetal movement count (DFMC) using objective criteria. The study was conducted in three parts: (a) we established a normal range of fetal activity; (b) we determined the DFMC in pregnancies at risk; and (c) we determined the relation between DFMC and antepartum intrauterine death of the fetus.

Methods

ESTABLISHMENT OF NORMAL RANGE OF FETAL ACTIVITY

Pregnant women in whom there was no reason to suspect impaired placental perfusion were studied as outpatients. Each patient recorded the number of fetal movements occurring daily between 9.00 am and 9.00 pm from the 32nd week until delivery. The patients were instructed to decide for themselves what constituted a movement. Sixty-one patients subsequently delivered vigorous infants of above the 25th percentile birth weight for gestational age, sex, and parity.⁵ These patients comprised the study group.

A total of 1654 DFMCs recorded by these 61 mothers were analysed (fig 1). The median value fell progressively from about 90 movements per 12 hours at the 32nd week of gestation to 50 per 12 hours at the 40th week. About 2.5% of counts at each period of gestation fell below 10 per 12 hours. This level was used as the lower limit of normal for clinical purposes.

FIG 1—Percentile distribution of DFMCs below 50th percentile in 1654 counts from 61 patients between 32 and 40 weeks of pregnancy.

DFMC IN PREGNANCIES AT RISK

Most women who were antenatal inpatients between September 1973 and September 1975 recorded DFMCs. Altogether 122 of these patients (a) had at least two 24-hour total urinary oestrogen estimations performed in the week before delivery; (b) recorded DFMCs for at least seven days before delivery; and (c) underwent continuous fetal heart rate monitoring during labour. Maternal 24-hour total urinary oestrogen assay was the standard placental function test during this period; serial results of this test, together with clinical findings and biparietal cephalometry, primarily guided management.

The patients were divided into four groups. Group 1 consisted of 100 patients with normal DFMCs and normal 24-hour total urinary oestrogen excretion (less than 2 SDs from the mean for gestational age). Group 2 included eight patients with normal DFMCs but low urinary oestrogen values on at least two successive occasions in the week before delivery. Group 3 included 10 patients with normal urinary oestrogen values but low DFMCs for at least one day before delivery. Group 4 consisted of four patients with low urinary oestrogen values in the week before delivery and low DFMCs for at least one day before delivery.

FIG 2—Percentile birthweight and 1-minute Apgar scores of infants of 100 patients with normal DFMCs and normal 24-hour total urinary oestrogen excretion.

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Results

Group 1—The infants were analysed according to the one-minute Apgar scores and percentile birth weight for gestational age, sex, and parity⁵ (fig 2). Eighty-six had a one-minute Apgar score of 7 or more, and 14 scored 6 or less. The distribution of percentile birth weight was weighted towards the lower end of the percentile birth weight scale, as expected in a group at risk. The 14 infants scoring under 7 are detailed in table I. All these infants scored 10 at 10 minutes, but four required endotracheal intubation.

Group 2—Of these eight patients (see table II) five were submitted to elective caesarean section. Seven infants survived, and one died of respiratory distress due to prematurity. Labour was induced in cases 15, 18, and 22, and emergency caesarean section was required in cases 15 and 18 for fetal distress with acidosis. Four of the seven infants of certain gestational age were below the 5th percentile.

Group 3—Two infants, one with multiple abnormalities, died before labour (table II). Of the eight remaining patients, five required

caesarean section for fetal distress, four with acidosis. Only one infant's birth weight was above the 15th percentile.

Group 4—All four infants died (table II). The only infant born alive died of respiratory distress.

DFMC AND ANTEPARTUM INTRAUTERINE FETAL DEATH

Over the two-year period 11 infants died in utero of causes other than congenital abnormality. Details of four of these cases (cases 28, 33, 34, and 35) are given in table II; details of the remaining seven infants are shown in table III. Seven patients recorded DFMCs, and all showed a precipitate fall in the DFMC to levels below 10 per 12 hours before fetal death; movements thereafter stopped for 12 to 48 hours before the fetal heart stopped. DFMCs in four of these patients are shown in fig 3. Serial oestrogen estimations were performed in five of the 11 patients in the week before delivery, and only one patient (case 28; table II) had normal values.

TABLE I—Details of delivery for those infants in group 1 (normal oestrogen values and normal movements) with Apgar scores of under 7 at one minute

Case No	Pregnancy complication	Onset of labour	Mode of delivery	Indication	1-min Apgar score	Additional factors
1	Diabetes	Induced	Caesarean section	Fetal distress	3	Tight nuchal cord
2	Unstable lie	Spontaneous	Caesarean section	Prolapsed cord	5	
3	Hypertension	Induced	Forceps	Fetal distress	3	
4	Hypertension	Induced	Caesarean section	Fetal distress, obstructed labour	2	
5	Diabetes		Elective caesarean section	Breech	3	Excessive sedation
6	Diabetes		Elective caesarean section	Previous caesarean section	6	
7	Hypertension	Induced	Difficult forceps	Fetal distress	3	
8	Hypertension, placenta praevia, breech		Elective caesarean section	Placenta praevia	3	
9	Breech	Spontaneous	Breech with forceps to aftercoming head	Breech	5	Tight nuchal cord
10	Small for dates	Induced	Spontaneous	Severe haemorrhage	5	
11	Placenta praevia		Caesarean section	Placental abruption	2	
12	Hypertension	Induced	Caesarean section	Failed trial of forceps	1	
13	Polyhydramnios	Induced	Caesarean section		5	
14	Hypertension	Induced	Spontaneous		3	

TABLE II—Details of pregnancies and infants in groups 2, 3, and 4

Case No	Pregnancy complication	Maturity at delivery (weeks)	Type of delivery	Fetal acidosis†	Apgar score at 1 minute	Percentile birth weight	Outcome
<i>Group 2 (low oestrogen values; normal movements)</i>							
15	Hypertension	38	Emergency caesarean section	+	3	40th	Survived
16	Unstable lie	35	Elective caesarean section	ND	8	80th	Survived
17	Pre-eclamptic toxæmia	35	Elective caesarean section	ND	10	<5th	Survived
18	Small antepartum haemorrhages	about 38	Emergency caesarean section	+	6		Survived
19	Severe pre-eclamptic toxæmia	36	Elective caesarean section	ND	2	<5th	Survived
20	Small antepartum haemorrhages	35	Elective caesarean section	ND	9	20th	Survived
21	Small for dates, bad obstetric history	31	Elective caesarean section	ND	4	<5th	Neonatal death (respiratory distress)
22	Antepartum haemorrhage, abruptio placentae, small for dates	34	Forceps	ND	3	<5th	Survived
<i>Group 3 (normal oestrogen values; few movements)</i>							
23	Threatened premature labour	37	Forceps	(UA) +	8	15th	Survived
24	Hypertension and small antepartum haemorrhages	39	Spontaneous	ND	8	15th	Survived
25	Pre-eclamptic toxæmia	Probable term	Emergency caesarean section	(scalp) +	4		Survived
26	Small for dates	39	Assisted breech	ND	7	<5th	Neonatal death, large septal defect
27	Hypertension and small antepartum haemorrhages	37	Emergency caesarean section	(scalp) +	2	<5th	Survived
28	Severe pre-eclamptic toxæmia	37	Spontaneous			15th	Intrauterine death
29	Hypertension	39	Emergency caesarean section	Cord presentation	9	30th	Survived
30	Rheumatic heart disease	32	Forceps				Intrauterine death (multiple abnormalities)
31	Severe pre-eclamptic toxæmia	33	"Emergency" caesarian section*	+	2	7th	Survived
32	Moderate pre-eclamptic toxæmia	37	Emergency caesarean section	+	1	<5th	Survived
<i>Group 4 (low oestrogen values; few movements)</i>							
33	Severe hypertension	31	Spontaneous			10th	Intrauterine death
34	Severe hypertension	33	Spontaneous			<5th	Intrauterine death
35	Severe Rh isoimmunisation	32	Spontaneous			Invalid	Intrauterine death
36	Severe pre-eclamptic toxæmia	32	Emergency caesarean section	+	3	<5th	Neonatal death (respiratory distress)

*Antenatal cardiotocography showed fetal distress.

†Scalp blood: pH ≤ 7.25 ; umbilical artery (UA): pH ≤ 7.20 ; umbilical vein (UV) pH ≤ 7.27 . ND = Not done.

FIG 3—DFMCs of four patients whose infants died in utero before labour. Time of death is indicated (D). (See also tables II and III.)

Discussion

In normal pregnancies about 2.5% of DFMCs fell below 10 movements/12 hours. Intrauterine death was preceded by a rapid diminution of fetal movements, usually over three or four days, followed by cessation for 12 to 48 hours before death occurred. This latter observation corresponds to the "movement alarm signal" described by Sadovsky *et al.*⁶

Normal oestrogen values when combined with a normal DFMC were associated with good fetal outcome in a population at risk. On the other hand, a low DFMC was associated with a

TABLE III—Details of antepartum fetal deaths among inpatients not included in groups 2 and 3

Case No	Pregnancy complication	Maturity (completed weeks)	Percentile weight	Oestrogen excretion	DFMC
37	Pre-eclamptic toxæmia	31	<5th (1st twin)	Low	Low
38	Severe hypertension	29	800 g		
39	Mild hypertension, patient educationally subnormal	39	20th		
40	Severe Rh isoimmunisation	30	Invalid	Low	Low
41	Acute polyhydramnios	31	40th		
42	Severe Rh isoimmunisation	27	Invalid		
43	Diabetes, pre-eclamptic toxæmia, abruptio placentæ	28	660 g	Low	Low

high incidence of fetal asphyxia and intrauterine death even when urinary oestrogen levels were normal.

We believe that the DFMC is useful in the more accurate prediction of imminent fetal death. Of greater clinical importance is the observation that a normal fetal movement count was generally associated with a good outcome. The finding of a normal DFMC would thus reassure the obstetrician who wishes to defer delivery to gain further fetal maturity.

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References

- Sadovsky, E, and Yaffe, H, *Obstetrics and Gynecology*, 1973, **41**, 845.
- Mathews, D D, *Lancet*, 1973, **1**, 1315.
- Reinold, E, *Journal of Perinatal Medicine*, 1973, **1**, 65.
- Sadovsky, E, *et al*, *Lancet*, 1973, **1**, 1141.
- Thomson, A M, Billewicz, W Z, and Hytten, F E, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1968, **75**, 903.
- Sadovsky, E, Yaffe, H, and Polishuk, W Z, *International Journal of Gynaecology and Obstetrics*, 1974, **12**, 75.

Immunological studies in pre-eclamptic toxæmia

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Summary

Although five patients with severe pre-eclamptic toxæmia (PET) had increased anticomplementary activity in their serum, there was no evidence of complement activation in the plasma of four of the five patients. These results suggest that circulating immune complexes are not implicated in the pathogenesis of PET. No

significant correlation was found between anticomplementary activity and pregnancy-associated α_2 -glycoprotein.

Introduction

It is now established that in normal pregnancy there is a maternal immune response to trophoblastic antigens that involves both cell-mediated and humoral factors.¹⁻³ In pre-eclamptic toxæmia (PET) an immunological disturbance is suggested by studies showing a correlation between the incidence of the disease and the degree of maternal/fetal histoincompatibility.⁴ Immunoglobulin and complement have also been detected in the renal glomeruli of pre-eclamptic patients immediately after delivery.^{5,6}

Maternal antibody production to paternal transplantation antigens in a first pregnancy could be considered analogous to primary immunisation, whereas subsequent pregnancies would elicit a form of secondary immune response in which the antibody titre would be higher than in the first pregnancy. Part of the function of such antibodies might be to neutralise soluble

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