Increased placental resistance and late decelerations associated with severe proteinuric hypertension predicts poor fetal outcome

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Summary

The flow velocity wave forms generated by Doppler ultrasound examination of the umbilical artery were correlated with feto-placental blood flow and numerically expressed as a ratio between the systolic (A) and the end-diastolic point (B). The technique is non-invasive and simple to perform. A cohort analytical study was done to see whether useful information could be obtained from the A/B ratio that could help in the management of patients with severe proteinuric hypertension. Fifty patients with severe proteinuric hypertension at less than 34 weeks' gestation were studied and serial Doppler ultrasound examinations of the umbilical artery were performed. No ultrasound results were made available to the clinician. An A/B ratio of 6 or greater was regarded as increased. Twenty-eight of the patients had an increased A/B ratio; in this group these 14 infants were small for gestational age, 14 developed late decelerations and there were 12 perinatal deaths. The remaining 22 patients had an A/B ratio of less than 6 and only 3 produced infants which were small for gestational age; 2 fetuses developed late decelerations and there was 1 perinatal death. A significant difference was found between the two groups in respect of these results. The group with an abnormal A/B ratio also experienced more neonatal morbidity. The A/B ratio of the umbilical artery wave form may assist in planning delivery of patients with severe proteinuric hypertension more accurately.

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The perinatal morbidity rate in infants born to mothers with severe proteinuric hypertension is very high, ranging from 71/1 000 deliveries[1] to 240/1 000.[2] The deaths are due mainly to complications of growth retardation, prematurity and asphyxia. Timing of delivery in these cases is often extremely difficult, since there are maternal complications on one hand and fetal complications on the other. Modern fetal heart monitoring techniques and ultrasound examinations often make it possible to prevent intra-uterine death.[3] The problem is thus transferred to the paediatrician, since premature and growth-retarded infants are most at risk of neonatal death and morbidity.[4]

Uteroplacental blood flow is one of the major factors determining fetal growth and well-being. Recently it has become possible to examine umbilical blood flow by a simple non-invasive means, namely Doppler ultrasound examination. A definite difference has been reported between the flow velocity wave forms from the umbilical artery of a growth-retarded fetus and those from one which is appropriately grown.[5,6] When an appropriately grown fetus is examined the flow velocity wave form is characterised by high diastolic flow velocity, attributed to low placental resistance. When the fetus is growth-retarded the wave form is characterised by low diastolic flow velocity, attributed to increased placental resistance. To describe the flow velocity wave form a ratio between the peak systolic flow velocity (A) and the end-diastolic velocity (B) has been formulated.[5] A high A/B ratio has been found to correlate with a growth-retarded fetus.[5,6] Examination of the placentae of babies with high A/B ratios has revealed obliteration of the small arteries of the tertiary villi.[7]

Doppler ultrasound examination of the umbilical artery to determine the A/B ratio and consequently obtain a measure of placental resistance could be used to identify the potentially compromised fetus. This information may influence the timing of delivery, which can be planned so that the baby is handed over to the paediatrician in the best possible condition, thus improving the perinatal mortality rate. An analytical study was performed on a group of 50 patients with severe proteinuric hypertension to determine whether measuring the A/B ratio has a role to play in the management of this condition.

Patients and methods

From March 1986 to January 1987, 50 patients with severe proteinuric hypertension (blood pressure 160/110 mmHg and $\geqslant$ 2+ proteinuria on dipstick examination (Ames) on two occasions 6 hours apart) were studied. All were admitted to a high-risk obstetric ward where they were monitored intensively until 34 weeks' gestation, unless maternal or fetal indications necessitated earlier delivery. The management protocol has been described in detail elsewhere.[3] Non-stress tests (NSTs) were performed at least 3 times a day.

The gestational age was derived from the date of the last menstrual period and, where available, an early ultrasound examination. Gestational age was expressed as completed weeks. A small-for-gestational-age (SGA) baby was defined as having a birth weight less than the 10th percentile for gestational age on the growth curves of Lubchenco *et al.*[8] Deaths were classified as intra-uterine, early neonatal (in the first week), late neonatal (between 7 and 28 days) and perinatally related (including all deaths after 28 days). The neonatal records were analysed by a paediatrician (G.K.) and all neonatal complications were recorded.

Doppler ultrasound examination of the umbilical artery was performed using an ATL MK 500 ultrasound system which combined high-frequency imaging with pulsed Doppler flowmetry and real-time spectral analysis. A wall filter of 200 Hz was used. The probe was 3 MHz. The method used is described by Erskine and Ritchie.[9] Doppler ultrasound examination was performed 3 times a week and hard copy of all the flow velocity wave forms was kept. All results of ultrasound examination were kept 'blind' to the obstetricians and paediatricians.

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Placental resistance was measured using the peak systolic flow (A)/diastolic flow (B) ratio.[5] For the purposes of the study placental resistance was regarded as severely raised when the A/B ratio was 6 or more. This value is far above the 95th percentile of the normal A/B ratio curve of the umbilical artery described by Trudinger *et al.*[5] and represented the upper limit of their scale.

The last ultrasound examination performed and the last three NSTs were used for analysis. All patients had their last ultrasound examination 3 days or less before delivey and their last NST on the day of delivery.

Statistical analysis was performed using Student's *t*-test to compare means of normally distributed data and the chi-square test for comparing proportions. A difference of $P < 0,05$ was regarded as significant.

Results

One hundred and ninety-nine Doppler ultrasound examinations of the umbilical artery were performed on the 50 patients (median 3, range 1 - 15). In 22 cases the last A/B ratio was less than 6 (group 1) and in the remaining 28 it was 6 or more (group 2).

The median age of the group 1 patients was 24 years (range 17 - 41 years) and that of the group 3 patients 25 years (range 20 - 41 years). Ten patients in group 1 and 7 in group 2 were nulliparous and 12 in group 1 and 21 in group 2 were multiparous.

Reasons for delivery

In group 1, 6 (27%) of the infants were delivered before 34 weeks' gestation for fetal reasons and 10 (45%) for maternal reasons, 5 (23%) were induced at 34 weeks' gestation, and 1 was delivered because of intra-uterine death caused by abruptio placentae. In group 2, 15 (54%) of the infants were delivered before 34 weeks' gestation for fetal reasons and 7 (25%) for maternal reasons; labour was induced at 34 weeks in 2 cases (7%), and 1 patient went into labour spontaneously. The remaining 3 fetuses in group 2 died *in utero* (see 'Correlations with perinatal mortality').

Correlation with NSTs

Two (9%) of the fetuses in group 1 developed late decelerations; 1 was born after abruptio placentae had developed and the other had meconium ileus. In group 2, 14 (50%) of the fetuses developed late decelerations, a significant difference ($P < 0,01$) when compared with group 1. In group 2, 10 of the fetuses which developed late decelerations died in the perinatal period, whereas only 2 of the remaining 14 died. In 19 group 2 patients no flow was present in the diastolic period. In 4 of these cases no diastolic flow was recorded for more than 3 weeks, the maximum being 6 weeks; all 4 of these fetuses eventually developed late decelerations and 3 died in the neonatal period. More than one ultrasound examination had been done on 11 of the 14 group 2 fetuses with late decelerations. In 10 of these cases the second last ultrasound examination revealed an A/B ratio of greater than 6, indicating that this change preceded the occurrence of late decelerations.

Correlation with SGA infants

The mean birth weight (± SD) in group 1 was 1 459 ± 366 g, compared with 1 110 ± 336 g in group 2 ($P < 0,01$). Although gestational ages tended to be lower in group 2 (30,6 ± 2,3

TABLE I. INFANT DEATHS

Patient No.	Reason for delivery	Gestation (wks)	Birth weight (g)	A/B ratio	NSTs	Cause of death	Days survived	SGA/AGA
Group 1								
1	Intra-uterine death	Unknown	1260	5,6	Non-reactive	Abruptio placentae	—	Unknown
2	Fetal distress	29	1070	3,9	Late decelerations	Meconium ileus	38	AGA
3	Imminent eclampsia	29	1090	3,6	Non-reactive	Necrotising enterocolitis	42	AGA
4	Imminent eclampsia	28	1165	3,7	Non-reactive	Necrotising enterocolitis	67	AGA
5	Imminent elcampsia	33	1390	2,9	Non-reactive	Septicaemia	67	SGA
Group 2								
6	Intra-uterine death	27	640	NDF	Late decelerations	Asphyxia	—	SGA
7	Imminent eclampsia	27	700	8,1	Non-reactive	Severe prematurity/asphyxia	—	SGA
8	Intra-uterine death	31	610	7,2	Late decelerations	Asphyxia	—	SGA
9	Fetal distress	28	648	NDF	Late decelerations	Asphyxia	1	SGA
10	Imminent eclampsia	28	725	NDF	Late decelerations	Asphyxia	3	SGA
11	Fetal distress	28	705	NDF	Late decelerations	Pulmonary bleeding	3	SGA
12	Fetal distress	30	930	Reversed flow	Late decelerations	Pneumothorax	2	SGA
13	Imminent eclampsia	29	835	NDF	Non-reactive	Respiratory distress syndrome	7	SGA
14	Fetal distress	30	920	NDF	Late decelerations	Pulmonary bleeding	7	SGA
15	Fetal distress	28	840	NDF	Late decelerations	Intracranial haemorrhage	8	SGA
16	Fetal distress	28	820	NDF	Late decelerations	Intracranial haemorrhage	11	SGA
17	Fetal distress	32	1025	NDF	Late decelerations	Septicaemia	16	SGA
18	Fetal distress	28	1125	NDF	Late decelerations	Necrotising enterocolitis	29	AGA

NDF = no diastolic flow; AGA = appropriate for gestational age; SGA = small for gestational age.

weeks) than in group 1 (31,9 ± 2,2 weeks), the difference was not significant.

In 2 cases in each group the gestational age was unknown. Of the remaining 20 group 1 infants 3 (15%) were SGA, compared with 14 (54%) of the remaining 26 in group 2 ($P < 0,01$).

Correlations with perinatal mortality

A total of 5 group 1 infants died; 1 intra-uterine death was due to abruptio placentae and 4 deaths were perinatally related (after 28 days) (Table I). One of the latter (case 2) was due to meconium ileus with bowel perforation at birth. The other 3 deaths were all very late and were caused by complications of nosocomial infections acquired in the neonatal wards.

In group 2 a total of 13 infants died. There were 3 intra-uterine deaths, 6 early neonatal deaths, 3 late neonatal deaths and 1 perinatally related death (Table I). The 3 intra-uterine deaths were expected. In 2 cases (Nos 6 and 8) the fetus developed severe late decelerations but was considered too small to survive in the neonatal unit. In case 7 the mother developed imminent eclampsia and since the baby was considered too small to survive the pregnancy was terminated vaginally. If the perinatally related deaths which occurred after 28 days are excluded there were significantly more perinatal deaths in group 2 ($P < 0,01$).

Correlation with neonatal complications

The neonatal complications are set out in Table II. Necrotising enterocolitis was diagnosed if an abdominal radiograph showed intestinal pneumatosis or if bowel perforation occurred. Perforation occurred in 2 cases in group 2. Septicaemia was diagnosed if blood culture was positive. Six infants in group 1 had respiratory distress syndrome and 2 needed intermittent positive-pressure ventilation, while 11 in group 2 had respiratory distress syndrome and 5 needed intermittent positive-pressure ventilation.

Discussion

In patients with severe proteinuric hypertension at more than 28 weeks' but less than 35 weeks' gestation, there were significant correlations between increased placental resistance and late decelerations of the fetal heart rate, growth retardation and perinatal death. It also appears that increased placental resistance precedes the occurrence of late decelerations, confirming previous reports.[5,6,10,11] Conversely, late decelerations were not associated with an A/B ratio of less than 6 unless an acute incident occurred, e.g. abruptio placentae, or a congenital abnormality was present.

Caution must be exercised when interpreting neonatal outcome because although the difference between gestational ages in the two was not statistically significant it might have attained significance if there had been a larger number of patients. Prematurity also played a role in neonatal outcome in group 2 which could not be quantified. On the other hand, placental resistance is a measure of the severity of disease and as such is a very good prognostic indicator.

With this proviso in mind, some of the neonatal complications in the group with increased placental resistance could be explained by the pronounced redistribution of blood flow that occurs in the fetus in response to chronic hypoxia. Hacket *et al.*[11] postulated that severely raised placental resistance might cause permanent damage in the organ systems most severely affected by the diminished blood flow. For example, the decreased flow to the lungs could diminish the production of surfactant, increasing the incidence of respiratory distress syndrome. The decreased flow to the gastro-intestinal tract could lead to a greater predisposition to early-onset necrotising enterocolitis. Our finding that necrotising enterocolitis and septicaemia were more common in the group with relatively normal placental resistance could be explained by the 12 intra-uterine or early deaths in group 2.

The findings in this study have two major implications with respect to the management of severe proteinuric hypertension. Firstly, when placental resistance is found to be relatively normal management could be more conservative, thus improving the chances of a premature baby. This finding might also help with the interpretation of suspicious NSTs. However, the fetus would still have to be monitored intensively because normal placental resistance does not preclude abruptio placentae.

Secondly, if placental resistance is severely increased delivery could be expedited if the fetus is viable, preventing the occurrence of late decelerations and asphyxia. Caution must be exercised since the interval between the onset of severely abnormal placental resistance and that of late deceleration is unknown, but can be up to 6 weeks. Since the extremely preterm fetus may benefit from further intra-uterine development, careful monitoring is necessary to allow it to develop further, especially when neonatal services have little experience in the management of extremely low-birth-weight infants. Fetal blood gas sampling by cordocentesis might also help in assessing the condition of the fetus in these cases.[12]

TABLE II. NEONATAL COMPLICATIONS

	Group 1 (21 infants)		Group 2 (25 infants)	
	No.	%	No.	%
Apgar score < 6 at				
1 min	4	19	9	36
5 min	2	10	3	12
Necrotising enterocolitis	5	24	3	12
Septicaemia	9	43	4	16
Respiratory distress syndrome	6	29	11	44
Intracranial haemorrhage	—	—	3	12
Exchange transfusion	—	—	3	12
Pulmonary haemorrhage	—	—	2	8
Pneumothorax	1	5	1	4
Patent ductus arteriosus	2	10	—	—

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Dorsal ganglion of the wrist – pathogenesis and biomechanics

Operative v. conservative treatment

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Summary

It is shown that the dorsal ganglion arises as a herniation from the dorsal scapholunate ligament. This herniation increases in size (according to La Place's law) owing to the unidirectional pinchcock effect of the mucosal folds of the duct and the pressure of the overlying extensor retinaculum until the distending pressure inside the ganglion equals the overlying tissue pressure. Wrist gangliography, retrograde wrist arthrography, histology and nuclear magnetic resonance were used to prove this conclusively. Bearing the pathogenesis in mind, the best clinical results were obtained by excision of the ganglion with 0,5 cm^2 of dorsal scapholunate ligament and closure of the dorsal capsule with a 3/0 Vicryl purse-string suture. Non-surgical sclerotherapy led to severe inflammation and sepsis and a recurrence rate of 45%. Conservative therapy is illogical since the communicating duct remains and synovial fluid from the scapholunate joint will cause a reherniation and recurrence of the ganglion.

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The dorsal ganglion of the wrist is a synovial-lined cystic swelling communicating with and arising from the wrist joint. It is the most common soft-tissue tumour in the hand.[1] At Tygerberg Hospital 9,3% of all hand surgery is for dorsal wrist ganglion. The sex ratio for men and women is 1:1,4, while the vocational predominance of manual over sedentary work is 2,5:1 (Fig. 1).

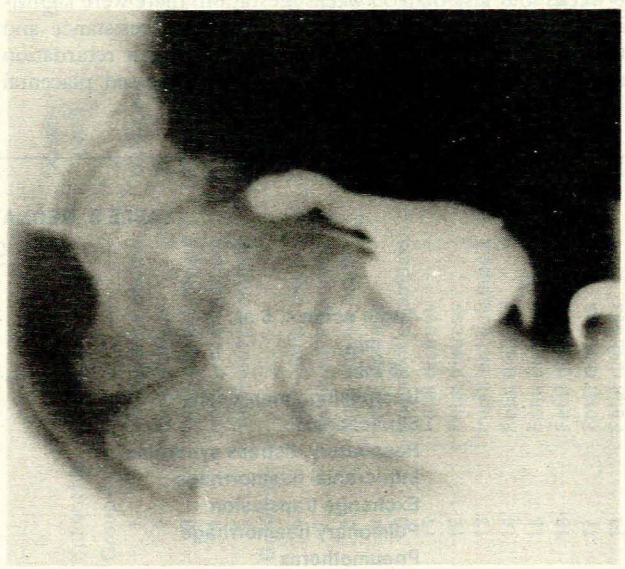

Fig. 1. Retrograde gangliogram of dorsal wrist ganglion (note proximal unidirectional valve).