

Prediction of Fetal Acidosis by Blood Flow Velocity Waveform of the Fetal Descending Thoracic Aorta

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Abstract—Fetal descending thoracic aorta blood flow velocity waveforms were measured in 63 pregnancies. They were either delivered by elective C/S before labor (56 patients) or suffered an antefartum fetal death (7 patients). There was a significant correlation between the aorta waveforms and the cord arterial pH. A cord arterial pH less than 7.20 or an intrauterine fetal death were used as our criteria for fetal acidosis. Using the above criteria, the specificities and negative predictive values of these testings in predicting fetal acidosis were over 80%. When fetal compromise was defined as either fetal acidosis or fetal growth retardation, the positive predictive values of these studies in forecasting fetal compromise heightened (range 81% to 92%) compared to those in predicting fetal acidosis (range 56% to 75%), although other predictive values remained similar. In conclusion, Doppler measurement of fetal descending thoracic aorta flow velocity waveform may be a valuable adjunctive noninvasive method of fetal surveillance.

Key Words: *Doppler ultrasound, Aorta blood flow velocity waveform, Cord arterial pH, Fetal acidosis, Cesarean section*

INTRODUCTION

Doppler ultrasonic measurement of fetal blood flow velocity waveform (FVW) has been used as a clinical method for monitoring high risk pregnancies (Griffin *et al.* 1983; Rightmire, 1988). In pregnancies complicated by chronic hypoxia (Jouppila and Kirkinen, 1984; Lingman *et al.* 1986) or growth retardation (Griffin *et al.* 1984; Tonge *et al.* 1986; Arduini *et al.* 1987), characteristic changes in

the fetal descending aorta blood FVW, with a decrease in the diastolic velocity as a result of increased peripheral vascular resistance (McCallum, 1981; Hackett *et al.* 1987; Van Vugt *et al.* 1987), have been reported. Moreover, Doppler ultrasound has been reported to be able to detect fetal hypoxia in advance of cardiotocography (CTG) (Jouppila and Kirkinen, 1984; Lingman *et al.* 1986; Griffin *et al.* 1984; Hackett *et al.* 1987), and the relation of fetal umbilical blood gas levels in growth retardation to mean blood velocity in the fetal aorta was demonstrated (Soothill *et al.* 1986).

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This prospective study was carried out in order to determine the clinical value of blood FVW

measurement in the fetal descending thoracic aorta in the prediction of fetal acidosis or compromise. The fetal acid-base state was determined by cord arterial pH. To minimize the effects of labor on the fetus, cord blood was sampled in patients who had undergone elective cesarean section prior to having their onset of labor.

MATERIALS AND METHODS

Fetal descending thoracic aorta blood FVW was measured by Doppler ultrasound in 63 pregnancies without congenital anomaly. All of the patients had intact membranes and were not in labor. All were delivered by cesarean section without labor between 26 and 42 weeks of gestation, except for 7 cases in which intrauterine fetal death was noticed within 72 hours following Doppler examination.

Measurement of the fetal aorta FVW was carried out at the lower thoracic level of the fetal descending aorta using a 3 MHz real-time sector scanner equipped with 3.5MHz pulsed Doppler transducer (Diasonic SPA-1000). The lowest energy output setting possible was used. The angle of insonation of the vessel was kept below 70°. A high pass filter was set at 100Hz and the sample volume at 5-8 mm. The Doppler shifted frequencies, and their intensities against time were displayed on the spectrum analyzer. The peak systolic velocity (A) and end-diastolic velocity (B) were read using internal electronic calipers, and A/B ratio was calculated thereafter. The pulsatility index (PI) was measured automatically and displayed on the spectrum analyzer. At least 4 separate readings were made for each examination, and a mean value was obtained. The examination was performed with the patient in a semirecumbent position and the fetus in a quiet state. The end-diastolic velocity, A/B ratio, or pulsatility index were considered abnormal when they were greater or less than our previously reported normal limit (end-diastolic velocity < 9.7 cm/sec, A/B ratio > 9.53, and PI > 2.78) (Shin *et al.* 1989).

The Doppler examination was performed 1 to 16 hours prior to cesarean section in 56 cases, and the Doppler results were not used in any way in making clinically intervening decisions. The primary indications for cesarean section are shown

in Table 1. All operations were performed with the gravid in a left tilt position under either epidural anesthesia (71%) or general anesthesia (29%). Epidural anesthesia was performed with 0.5% bupivacaine, preceded by a preload of 500-1000 ml Ringer's lactated solution to prevent hypotension. Forty percent oxygen was given via face mask from the time when adequate anesthesia was achieved until delivery. General anesthesia was carried out after 2-3 minutes preoxygenation with 100% oxygen, which was reduced to 50% from the time of intubation until delivery.

Table 1. Primary indications for Cesarean section

| Indications | No. |
|---------------------|-----|
| Repeat cesarean | 31 |
| Fetal distress | 9 |
| Breech presentation | 8 |
| Fetal macrosomia | 2 |
| Placenta previa | 1 |
| Previous myomectomy | 1 |
| Others | 4 |
| Total | 56 |

Immediately following the delivery of the infant, a segment of the umbilical cord was doubly clamped, and blood was separately collected from the artery into heparinized plastic syringes. The syringes were capped with accompanying plastic covers, the specimens were placed in ice, and blood pH determinations were completed as soon as possible. Strickland *et al.* (1984) have shown that there is minimal change in arterial blood pH values if the analysis is completed in < 120 minutes. Other researchers have already shown that icing the cord blood samples does not change the rate of decrease in the arterial pH (Nhan *et al.* 1980). The cord blood gas analysis was done by means of an automatic pH/blood gas analyzer (AVL Model 995). For the purpose of this study, fetal acidosis was defined as an arterial pH < 7.20 or intrauterine fetal death without labor pain. Since oligohydramnioses were not found during sonographic examinations carried out just before fetal deaths, cord

compressions was not thought to be the causes of intrauterine fetal deaths. In addition, there was no evidence of abruptio placenta, and fetuses with congenital malformations were excluded from this study. Hence, we considered the possibility that all intrauterine fetal deaths occurred as a consequence of fetal hypoxia. The cord arterial pH was assumed to be 6.9 when intrauterine fetal death occurred within 12 hours following Doppler study, and 7.0 and 7.05 if the death occurred within 24 hours and 72 hours, respectively, following Doppler study. A fetus was classified as growth-retarded (IUGR) when the birth weight was below the tenth percentile for gestational age of the Korean birth-weight percentile curves (Hwang *et al.* 1987).

Statistical analysis was performed by using Student's t-test or chi-square test. Linear regression analysis was used in assessing the relation between the aorta FVW indices and the cord arterial pH. A Mann-Whitney U test was employed to compare the cord arterial pH between the patients delivered under epidural and those under general anesthesia.

RESULTS

A total of 63 patients were examined by Doppler ultrasound. The mean gestational age at examination, confirmed by dates and early sonography was 37.3 weeks (range 26-42 weeks).

Abnormal aorta FVWs were encountered in 17 patients. One case showed an abnormal peak velocity, end-diastolic velocity, A/B ratio, and PI in the fetal aorta FVW. In 8 cases, both abnormal end-diastolic velocity and PI in the aorta FVW were encountered. Both the abnormal end-diastolic velocity and A/B ratio in the fetal aorta FVW were encountered in 3 cases. Four cases showed only an abnormal A/B ratio in the fetal aorta FVW and another case showed only an abnormal PI in the fetal aorta FVW.

There were 14 acidotic fetuses (7 fetuses with a cord arterial pH < 7.20, and 7 fetuses died in utero within 72 hours following Doppler examination) at birth. Eleven of the 63 patients delivered an infant with IUGR. The frequency of fetal acidosis in the growth-retarded fetuses was 54% (6/11).

When fetal compromise was defined as either fetal acidosis or intrauterine growth retardation, there were 19 compromised fetuses. The mean cord arterial pH of fetuses with an abnormal A/B ratio or PI in the aorta FVW delivered under epidural anesthesia was not different from that of general anesthesia. This was also true in the fetuses with nor-

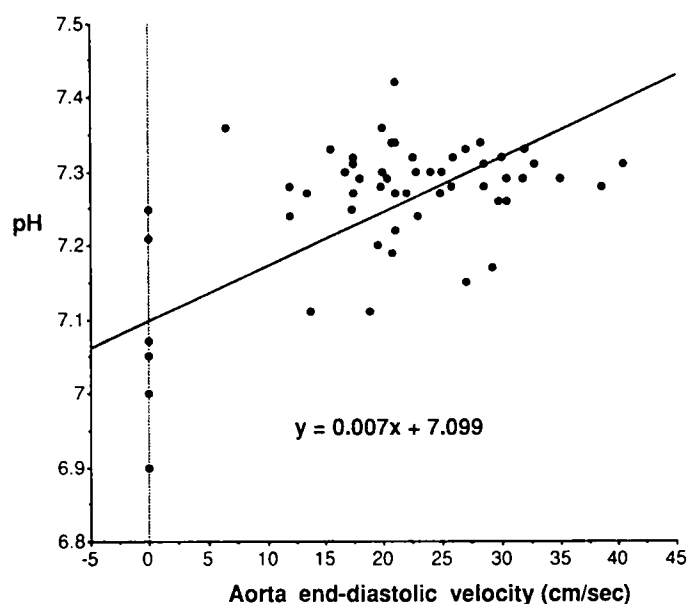


Fig. 1. Correlation of fetal aorta end-diastolic velocity and cord arterial pH. The lineal regression between the two parameters is highly significant ($r=0.67$, $n=63$, $p < 0.0001$).

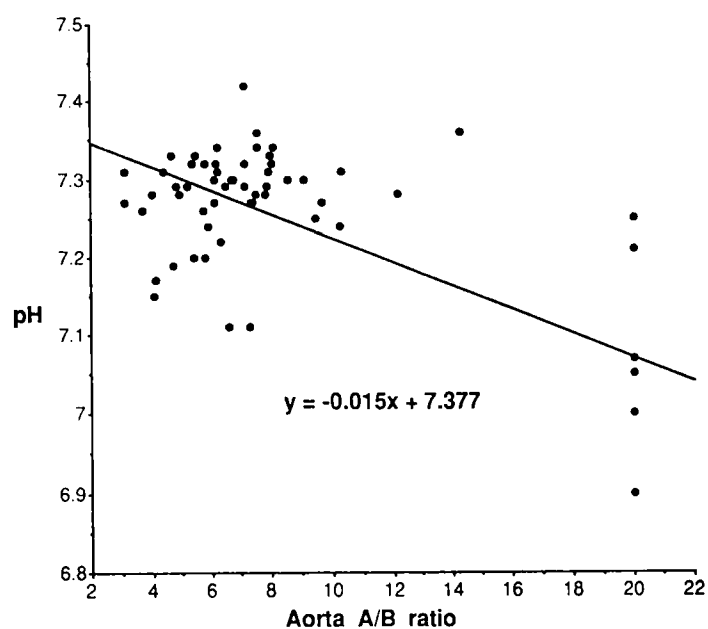


Fig. 2. Correlation of fetal aorta A/B ratio and cord arterial pH. The lineal regression between the two parameters is highly significant ($r=-0.70$, $n=63$, $p < 0.0001$).

Table 2. Efficacy of fetal aorta FVW in predicting fetal acidosis

| | End-diastolic Velocity | A/B ratio | PI |
|---------------------------|------------------------|-------------|-------------|
| Sensitivity | 64% (9/14) | 64% (9/14) | 50% (7/14) |
| Specificity | 94% (46/49) | 86% (42/49) | 93% (46/49) |
| Positive predictive value | 75% (9/12) | 56% (9/16) | 70% (7/10) |
| Negative predictive value | 90% (46/51) | 89% (42/47) | 87% (46/53) |

Table 3. Efficacy of fetal aorta FVW in predicting fetal compromise

| | End-diastolic velocity | A/B ratio | PI |
|---------------------------|------------------------|-------------|-------------|
| Sensitivity | 58% (11/19) | 68% (13/19) | 47% (9/19) |
| Specificity | 98% (43/44) | 93% (41/44) | 98% (43/44) |
| Positive predictive value | 92% (11/12) | 81% (13/16) | 90% (9/10) |
| Negative predictive value | 84% (43/51) | 87% (41/47) | 81% (43/53) |

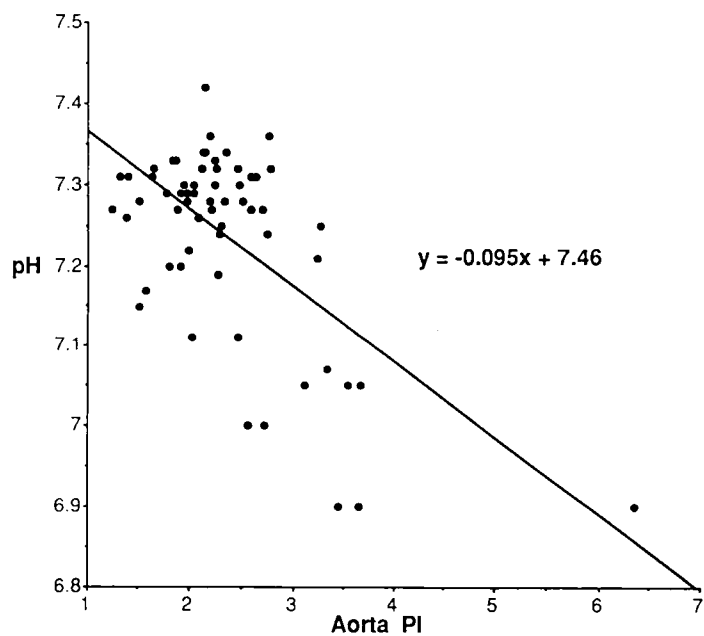


Fig. 3. Correlation of fetal aorta PI and cord arterial pH. The lineal regression between the two parameters is highly significant ($r = -0.61$, $n = 63$, $p < 0.0001$).

mal aorta FVW (Mann-Whitney U test, $p > 0.05$). There was a significant positive correlation between cord arterial pH and the end-diastolic velocity in the fetal aorta FVW ($r = 0.67$, $n = 63$, $p < 0.0001$) (Fig. 1). Also significant negative correlations were found between the cord arterial pH and the A/B ratio ($r = -0.70$, $n = 63$, $p < 0.0001$) (Fig. 2), along with the PI ($r = -0.61$, $n = 63$, $p < 0.0001$) (Fig. 3) of the fetal aorta FVW. The A/B ratio of the aorta

FVW resulted in a somewhat stronger correlation with cord arterial pH than the others, but each correlation's coefficients of the 3 graphs showed no significant differences statistically ($z < 2$, $p > 0.05$).

Intrauterine fetal death occurred in 7 cases among 11 fetuses with an absent end-diastolic velocity in the aorta FVW. When there was an absent end-diastolic velocity, 9 cases were acidotic, and the other 2 cases were growth-retarded, namely, all were compromised at birth. Tables 2 and 3 show the efficacy of the fetal aorta FVW in predicting fetal acidosis or fetal compromise.

DISCUSSION

With the use of real-time and Doppler ultrasound systems as a noninvasive method of measuring human fetal blood flow, quantitative evaluations on volume blood flow in the descending thoracic aorta were reported (Eik-Nes *et al.* 1980; Eik-Nes *et al.* 1982). However, the potential methodological errors (Tonge *et al.* 1983) in assessing vessel diameter and fetal weight by ultrasound caused a shift of the main interest to studies of flow velocity waveforms in the pulsatile fetal vessels. Soothill *et al.* (1986) demonstrate the association between umbilical venous blood gas levels and the mean velocity of blood flow in the fetal aorta measured by Doppler ultrasound.

In this study, we confirmed the correlation between the impedance indices in the fetal aorta (A/B ratio and PI) and cord arterial pH at elective cesarean section. The sensitivities of the fetal aorta A/B ratio and PI in predicting fetal compromise were 68% and 47% in each, and the specificities were 93% and 97%, respectively. The change in FVW is believed to mirror the impedance of the arterial vascular bed distal to the site of measurement. In the experimental animal fetus, hemodynamic redistribution has been demonstrated (Cohn *et al.* 1974; Peeters *et al.* 1980) in acute and chronic hypoxia. Constriction of the peripheral vessels- thus lowering the blood flow to the gut, kidney, and lungs with a simultaneous increase of blood flow to the brain, heart, and adrenals- was found in chronic hypoxic fetal lambs (Reuss *et al.* 1982). This redistribution may produce an increase in vascular resistance to the flow in the fetal aorta distal to the left common carotid artery, which is reflected in the change of aortic FVW. On the other hand, 40-60% of the blood flow in the fetal descending aorta is destined for umbilical circulation (Griffin *et al.* 1983; Tonge *et al.* 1983). Hence, the changes in aorta FVW of compromised fetus are an expression of an increased placental resistance in combination with vasoconstriction in the fetal lower body.

There was a high (up to 64% <7/11>) perinatal mortality in those fetuses with an absent aortic end-diastolic velocity. This result was higher than a report by Arabin *et al.* (1988), who found that perinatal mortality in infants with birth weights > 500 g, corrected for nonviable malformation, was 6/27 (22%). Laurin *et al.* (1987) proposes the blood flow class according to the combination of the pulsatility index and the configurational assessment of the diastolic part of the FVW. Our data is consistent with their concept, and the absence of end-diastolic velocity in aortic FVW should be considered an ominous sign of fetal compromise.

Aortic peak systolic velocity is considered to reflect myocardial contractility. Jouppila *et al.* (1986) report a significant reduction in aortic peak velocity in hypertensive pregnancies with fetal distress, but Cameron *et al.* (1988) report a much higher frequency of aortic diastolic flow abnormality than systolic flow reduction. Our study resulted in only 1 case

of decreased aortic systolic velocity with a concomitant decrease in diastolic velocity. As fetal hypoxia worsens, it seems to produce myocardial ischemia and a secondary decrease in aortic systolic velocity. Recently, Al-Ghazali *et al.* (1988) demonstrated that cardiac output is well maintained until late in the course of fetal compromise, as evidenced by the subsequent deaths of 6 of the 7 fetuses in whom diminished combined cardiac output was found.

In hypoxic fetuses, changes in the aortic FVW were usually detected before the occurrence of an abnormal cardiotocographic (CTG) finding, but the time relationships between the onset of the blood FVW change and the occurrence of CTG signs of fetal distress were not uniform. The reported time intervals between the 2 findings varied from 3 days (Jouppila *et al.* 1984; Lingman *et al.* 1986; Laurin *et al.* 1987) to 2-3 weeks (Griffin *et al.* 1984). The degree of abnormality of the aortic FVW correlates well with the cord arterial pH in the present study suggesting that hypoxia is already present by the time FVW changes are evident. Therefore, changes in the blood FVW can act as a guide as to the optimal time to deliver compromised fetuses, especially in cases with suspicious CTG findings, but aortic impedance indices (A/B ratio and PI) demonstrate a low sensitivity and positive predictive value of 50-70% in predicting fetal acidosis. It seems that individual fetal capability to tolerate impaired fetoplacental circulation and hemodynamic redistribution varies. Hence, making clinical decisions solely on the basis of fetal aortic hemodynamic change may be inappropriate. We recommend frequent follow-ups of the fetuses with abnormal velocity waveform, not only by Doppler examination but also by using other methods (e.g., evaluation of fetal structure and biophysical profiles by ultrasound, CTG tests, or cordocentesis to assess fetal acid-base status or for rapid karyotyping when needed).

The use of a real-time sector scanner in this study made it difficult to reduce the angle of insonation below 60°, so there might have been some errors in the FVW analysis. Had a combined real-time linear array and pulsed Doppler ultrasound system been used, this problem could have

been avoided. Nevertheless, from the results of this study, it can be concluded that Doppler measurement of aorta FVW may be a valuable adjunctive noninvasive method of fetal surveillance.

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= 국 문 초 록 =

태아 흉부 하강대동맥 혈류속도파형에 의한 태아의 산성증 예측

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Doppler 초음파에 의한 태아 흉부 하강대동맥 혈류속도파형을 이용하여 태아의 건강 상태를 얼마나 잘 파악할 수 있는지를 알아보기로 태아 건강의 객관적 지표인 제대동맥혈의 산도와 산전 시행된 Doppler 검사의 태아 흉부 하강대동맥 혈류속도파형간의 상관관계를 63명의 임신부를 대상으로 분석하였다.

분만진통이 태아혈액의 산도에 끼치는 영향을 배제하기 위하여 56명의 임신부를 대상으로 선택적 제왕절개술을 시행하기 1-16시간 전에 태아 흉부 하강대동맥 혈류속도파형을 측정하였으며 분만 직후 태아가 첫 호흡을 하기 전에 제대동맥혈을 채취하여 산도를 측정하였다. 나머지 7례에서는 Doppler 초음파 검사 후 3일내에 자궁내 태아사망이 발생하였다. 본 연구에서 제대혈 산도가 7.20 미만이거나 자궁내 태아사망이 발생한 경우를 태아산성증으로 정의하였고 흉부 하강대동맥 혈류속도파형의 A/B ratio나 PI치가 저자 등의 기준치 + 2x 표준편차인 경우 혹은 이완기말 속도가 저자 등의 기준치 - 2x 표준편차인 경우를 비정상으로 간주하였다. 흉부 하강대동맥 혈류속도파형과 제대동맥혈의 산도간에는 유의한 상관관계가 있었고 태아산성증의 진단 척도로서 흉부 하강대동맥 혈류속도파형의 진단 특이도 및 음성예측률은 80% 이상이었다. 본 연구결과 산전 시행된 Doppler 초음파 검사상 비정상 혈류속도파형을 보였으나 출생시 태아 산성증이 없던 태아는 모두가 발육지연태아임을 알았다. 이 점에 착안하여 태아 곤경상태를 태아 산성증이 있거나 태아발육지연이 있는 경우로 정의하였을 때 흉부 하강대동맥 혈류속도파형을 이용한 태아 곤경상태 예측의 양성예측율은 81-92%로 태아산성증의 진단 양성예측율 56-75%에 비하여 높은 예측률을 보였다.

이상의 결과로 보아 Doppler 초음파에 의한 산전 태아 흉부 하강대동맥 혈류속도파형을 이용하여 태아의 건강상태를 잘 파악할 수 있다고 판단되었다.