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Ductus venosus opens in high-risk pregnancies without signs of increased central venous pressure

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ABSTRACT

Objectives: It has been belived that changes in diastolic blood velocities in the fetal ductus venosus were due to increased central venous pressure secondary to increased fetal heart strain during hypoxia or heart failure. There have been recent reports of changes in ductus venosus blood velocity without signs of increased fetal heart strain. The aim of this evaluation was to compare blood velocity in the right hepatic vein as a marker of increased central venous pressure in relationship to changes in ductus venosus blood velocity.

Material and methods: Fifty pregnancies suspected of fetal growth resitriction were evaluated by Doppler ultrasound. Blood velocity was recorded in the right hepatic vein, ducus

venosus and in the umbilical vein. Placental blood flow was also recorded in the uterine and umbilical arteries as well as the fetal middle cerebral artery.
Results: Increased umbilical artery pulsatility index was recorded in 19 fetuses and 20 has signes of brain sparing according to recordings in the middle cerebral artery. Abnormal blood velocity in the ductus venosus was recorded in 5 fetuses, none of these fetuses had an abnormal pulsatility in the right hepatic vein.
Conclusions: Opening of the ductus venosus is not only related to fetal cardiac strain. This might indicate that the ductus venosus does not primarily open due to increased central venous pressure in moderate fetal hypoxia. Increased fetal cardiac strain might be a late event in the process of chronic fetal hypoxia.

Key words: ultrasound; Velocity vector imaging; fetal heart; strain; Doppler; ductus venosus; umbilical vein; pulsations; hypoxia; pregnancy

INTRODUCTION

About 20 to 30% of umbilical venous blood flow passes the fetal ductus venosus (DV). The main part of the blood stream from the DV passes through the oval foramen for the left heart atrium [1]. During fetal hypoxemia the DV opens giving away more of the oxygenated blood from the umbilical vein towards the left part of the heart and thus for the coronaries and brain as a part of a redistribution regulation. As much as 70% of the umbilical venous blood flow has been reported to pass the DV in severe fetal hypoxia [2].

Fetal central venous blood flow and flow in the DV has a pulsating pattern, which reflects pressure within the right atrium. Increased reversal of blood flow during diastole has been demonstrated during fetal heart failure [3]. Blood flow in the umbilical vein is normally without pulsations. A pulsating blood flow pattern has been demonstrated in severe fetal hypoxia and heart failure presumably because of opening of the ductus venosus due to increased central venous pressure, which has been believed to be secondary to increased strain on the fetal heart [3, 4].

Studies of fetal heart function have not given signs of strain during moderate chronic hypoxia using analysis of speckle tracking of the heart walls or during analysis of waveforms in the hepatic veins or inferior vena cava entering the right atrium which might indicated increased central venous pressure. Fetal heart function has been shown normal even during abnormal blood flow pattern in the ductus venosus [5–8]. Ductus venosus might thus open without an increase in fetal central venous pressure. The ductus might thus react by opening

due to hypoxia, as a part of redistribution of oxygenated blood to the vital organs heart and brain. This redistribution of oxygenated blood from the placenta might maintain fetal heart function for some time during moderate chronic hypoxia.

The aim of this study was to examine blood flow in the DV and hepatic veins in pregnancies suspected of chronic hypoxia. Signs of DV opening without changes in hepatic venous blood velocity should proof that opening of the ductus during moderate chronic hypoxia might occur without an increased in central venous pressure due to increased fetal heart strain.

MATERIAL AND METHODS

Singleton pregnancies suspected fetal growth restriction and at risk of developing chronic hypoxia were examined. Fetal size was evaluated by ultrasound and related to normal reference levels [9]. The amount of amniotic fluid was also evaluated. Fetal condition was evaluated by cardiotocography and by ultrasound Doppler recording of the umbilical, uterine and middle cerebral arteries. Blood velocity was also recorded by Doppler ultrasound in the right hepatic vein, in the DV and in the umbilical vein in the cord and intra-abdominally. Blood velocity waveforms recorded were analysed for pulsatility index (PI) and related to normal reference values.

The umbilical artery blood flow velocity was recorded from a free-floating central part of the umbilical cord at a zero-degree angle. Pulsatility index was analysed during three consecutive cardiac cycles and compared with normal reference values [10]. The uterine artery blood velocity was recorded from both uterine arteries after the vessels have been located by color flow mapping in an oblique scan with the sample volume placed in the artery just cranial to the crossing of the external iliac vessels. Three even blood velocity waveforms were analysed for pulsatility index. Increased uterine artery vascular impedance was defined as PI > 1.20. A pattern of an early diastolic notch in the uterine blood velocity spectrum was also documented. Umbilical venous blood flow was recorded in the central intra-abdominal part of the vein and in a free-floating part of the vein in the cord. The DV blood velocity was recorded in either a midsagittal or transverse view, positioning the Doppler gate at the isthmic portion. The DV blood velocity pattern was analysed for pulsatility index for veins (PIV) related to normal reference levels [11]. Blood flow velocity was also be recorded in the right hepatic vein before its entrance into the inferior vena cava. The blood velocity pattern was evaluated for PIV and related to normal reference levels as a marker of increased central venous pressure [12]. Blood flow in the middle cerebral artery (MCA) was also be recorded

about 1 cm from the circle of Willis and analysed for PI and related to normal reference limits for signs of brain sparing [13].

Outcome of pregnancy was evaluated as interventions during labor such as: Caesarean section; operative delivery for fetal distress (ODFD) indicated by abnormal cardiotocography tracing and/or fetal scalp blood pH. The gestational age at birth was noted as well as birthweight. A small-for-gestational-age (SGA) infant were defined as having a birthweight below 10^{th} percentile for the corresponding gestational age [9].

The neonatal condition was evaluated by the Apgar score at 1, 5 and 10 minutes, umbilical artery and venous cord blood gases at birth, the frequency of admission to a neonatal intensive care unit (NICU), the need for artificial ventilation and perinatal mortality was also be noted.

RESULTS

Fifty pregnancies were included in the study suspected of fetal growth restriction. The mean gestational age at admission was 33.9 weeks (range 24–39) and the mean growth percentily deviation was 3.22 ± 22.7 %.

Nineteen of the pregnancies had an increased PI in the umbilical artery, one having absent flow in diastole. There were 9 cases with abnormal PI in the right uterine artery and 14 in the left artery. There were 20 fetuses with signs of brain sparing in the middle cerebral artery. Increased PIV in the DV was recorded in 5 fetuses, none having absent or reversed flow in end-diastole. None of the fetuses had signs of pulsations the umbilical vein. Blood flow veliocity in the right hepatic vein was normal in all these cases.

Gestational age at birth was 35.5 weeks (range 25–39). There were 23 abdominal deliveries, 6 due to breech presentation and 17 due to suspected fetal distress. Umbilical cord pH was normal in all the newborns. There were 13 admissions for neonatal intensive care. The avarage gestational age at birth in these cases was 30.5 weeks (range 25–37), the avarage birth weight was 1164 g (range 490–1870), the avarage number of days on NICU was 23 days (range 2–80). Twelf of the newborns needed respiratory support. Seven newborns needed mechanical ventilation first and then continuous positive airway pressure (CPAP). The avarage gestational age at bith in that group was 28.5 weeks (range 25–31) and the avarage birth weight was 844 g (range 490–1300). The avarage time on mechanical ventilation was 13 days (range 5–41) and on CPAP 26 days (range 2–56). One newborn born in 25 weeks‘ gestation with birth weight of 490 g died after 5 days of mechanical ventilation. Five newborns needed only CPAP after being admitted to NICU. The avarage gestation age at birth

in that group was 32 (range 30–35) and the avarage birth weigt was 1470 g (range 1100–1770). The avarage time on CPAP was 7.6 days (range 3–14). There was no correlation between any blood velocity or PIV recorded by Doppler ultrasound in the hepatic vein or in the DV and the need of any respiratory support in newborn infants.

DISCUSSION

These resulsts support the theory that the ductus venosus can open in moderate chronic hypoxia without there being signs of increased central venours pressure due to increased cardiac strain.

Increased venous pulsatility has been demonstrated in fetal heart failure and severe hypoxia as a sign of increased central venous pressure [3, 4]. The increase in fetal heart failure and severe hypoxia has been show to be transmitted to the ductus venosus and even to the umbilical vein where the steady flow becomes pulsating due to opening of the ductus venosus. We belived based on this knowledge, that changes in ducus venosus PIV was a reflection of increased cardiac strain during chronic fetal hypoxia. However, Doppler research on blood flow in the central fetal veins like the hepatic veins and inferior vena cava have not been able to demonstrate changes in pulsatility in the central venous pulsatility in cases suggested in moderate chronic hypoxia [5, 6]. The same applies for evaluation of fetal heart wall motility. Increased blood flow pulsatilify in the ductus venousus could not be demonstraited by using spectral tracking of fetal ventricel walls and the right atrial wall movements where reduced motility would be a sign of increased cardiac strain [7, 8]. These findings might suggest that the ductus venosus functions as a redistributional organ facilitating increased flow of oxygenated blood to the left side of the heart and thus the coronaries and brain without there being an increase in central venous pressure in moderated fetal hypoxia.

The DV Doppler is presently increasingly being used for fetal surveillance in early onset fetal growth restriction before 30–32 weeks of gestation. The results indicate that DV PIV measurement is a good predictor of perinatal outcome and may be useful in determining the timing of the delivery in of early FGR fetuses [14].

CONCLUSIONS?

The use of DV Doppler in conbination with computerized CTG on deciding on delivery because of worsening fetal condition has given reasuring results with a surveival rate of about 90% without major neurology handicap [15, 16]. The results suggest that the optimal timing of delivery of fetuses with early intrauterine growth restriction may be best determined

by monitoring them longitudinally, with both DV and CTG monitoring [17]. Admission to NICU and the need of respiratory support was seen in the preterm infants group without any correlation with any blood velocity or PI recorded by Doppler ultrasound in the hepatic vein and in the DV. This tends to confirm the fact that respiratory condictions are the most common reason for admissions tu NICU [18, 19] and are mostly related to the gastational age of the newborn infant [20].

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Conflict of interest

All authors declare no conflict of interest.

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