

Umbilical Vessel Flow Velocity Waveforms in Cord Entanglement in a Monoamniotic Multiple Gestation

A Case Report

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An unusual flow velocity waveform pattern was seen in a case of cord entanglement in a monoamniotic multiple pregnancy. The umbilical artery systolic:diastolic ratio was abnormally high (>95th percentile for gestation), and diastolic notching was present. The umbilical vein flow velocity waveform was pulsatile, with flow absent during the diastolic phase of the cardiac cycle. The presence of that pattern strongly suggested a severe cord accident and mandated an immediate evaluation of fetal well-being.

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Introduction

Umbilical artery (UA) flow velocity waveform (FVW) analysis currently is the subject of intense investigation in several medical centers. The UA FVW is characterized by high diastolic flow velocity in normal pregnancy and low or absent diastolic flow in abnormal pregnancy.^{1,2} Umbilical vein flow velocity normally is continuous and nonpulsatile in the absence of fetal breathing. Below we report a case of an abnormal UA systolic:diastolic ratio, diastolic notching and pulsatile umbilical vein flow velocity pattern.

Case Report

A 25-year-old, black woman, gravida 5, para 2, abortus 2, with a triplet pregnancy, was hospitalized at 26 weeks' gestation for premature onset of labor. After 28 weeks' gestation the fetal status was evaluated with a nonstress test (NST) repeated every other day and with serial ultrasound measurements every three weeks to evaluate fetal growth. An early ultrasound examination at 22 weeks' gestation revealed the presence of three separate gestational sacs, a finding that was not verified in two subsequent ultrasound examinations. At 32 weeks' gestation the NST was unsatisfactory for two of the three fetuses, and an ultrasound examination demonstrated the death of one fetus. Reassuring biophysical profile scores were obtained on the two remaining fetuses. We then decided to follow the patient with daily NSTs.

The next day one fetus had a reactive NST and the other a nonreactive pattern. The nonreactive pattern was accompanied by several small variable decelerations. The biophysical profile score was 2; extended fetal observation did not change it. There was no explanation for the acute changes that caused the death of one fetus and the compromise of the second, although a cord accident was suspected.

The decision was made to deliver the infants for fetal distress. UA FVWs were obtained on both fetuses just before the cesarean section. Two separate segments of the umbilical cords of the two live fetuses were identified on real-time ultrasonography. A continuous wave Doppler device was used to obtain the flow velocity waveforms (Multigon 500A with 4-MHz transducer, Multigon Industries, Inc., Mount Vernon, NY). The fetus with the biophysical profile score of 10 had a normal UA FVW (Figure 1). The fetus with the nonreactive NST and biophysical profile score of 2 had an abnormal FVW with a systolic:diastolic ratio of 6.63 (Figure 2). There was a notch in the early diastole and pulsatile venous flow in the absence of fetal breathing. Reevaluation and

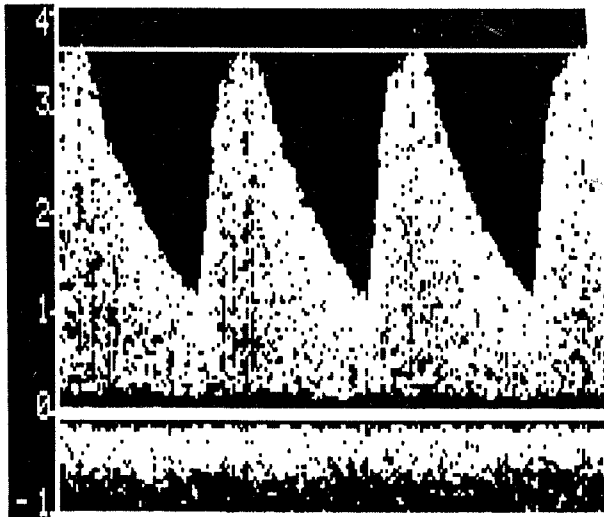


Figure 1
Doppler recording of normal characteristics in the healthy fetus. Note the normal amount of diastolic component and the non-pulsatile flow of the umbilical vein in the opposite direction.

verification of the origin of the Doppler signal were done with real-time ultrasonography. The venous flow started during the peak systole and ceased during the diastolic phase of the cardiac cycle. Those findings on the FVW, along with the variable decelerations, were thought to indicate severe persistent cord compression.

The patient underwent a primary low vertical cesarean section and delivered two living infants and one stillborn infant. Infant A weighed 1,650 g and had Apgar scores of 7 and 8 at one and five minutes, respectively; infant B weighed 1,820 g and had Apgar scores of 6 and 8 at one and five minutes, respectively. The umbilical arterial and venous pH for fetus B were 7.25 and 7.36, respectively. Infant C was stillborn and weighed 1,701 g. There were two separate placentas and two amniotic cavities. Infants B and C were contained in one sac, and their cords were entangled severely throughout their entire lengths. A tight true knot was present in the cord of the dead fetus.

Discussion

Acute cord compression is known to precipitate a number of cardiovascular changes in the fetus (hypertension, bradycardia, hypoxia).³ In our case the compression of the cord was prolonged and had started at least since fetus C was found to be dead and most likely before that. To our knowledge this is the first report of diastolic notching in the UA and absent flow in the umbilical vein during the diastolic phase

of the cardiac cycle. The temporal relationship of the arterial and venous waveforms left no doubt that this pattern did not represent a reverse diastolic flow or mirror image of the arterial waveform. The flow in the vein starts at the peak systole of the cardiac cycle and ends just before the appearance of the notch on the arterial waveform. In our case the increased UA systolic:diastolic ratio and the diastolic notching suggested increased resistance to blood flow.

For the UA flow to be continuous during diastole, the perfusion pressure must exceed the pressure gen-

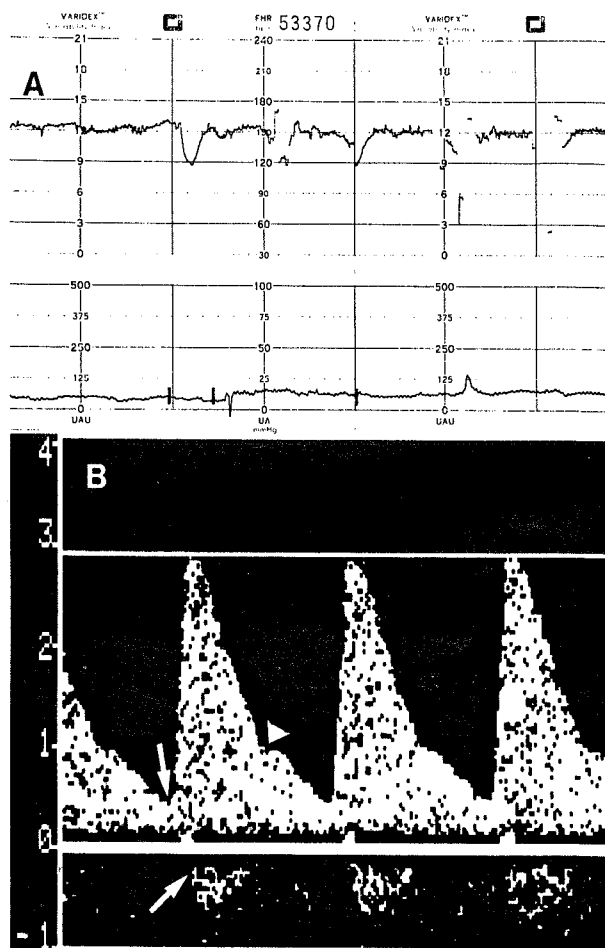


Figure 2
(A) Portion of the nonreactive tracing with variable decelerations. The Doppler recording was obtained right after the non-stress test was performed. The two arrows indicate the beginning of the flow rise in the umbilical artery and umbilical vein. The timing of the venous and arterial waveforms makes it clear that this is pulsatile venous flow and not a mirror image of the arterial waveform or a waveform originating in another arterial loop. (B) The arrowhead points to the notch during early diastole.

erated by UA compression. Whether the fetus responds to cord compression with arterial hypertension remains speculative. It is certain, however, that the arteriovenous pressure gradient in the placental vasculature is not high enough to overcome venous compression and maintain the venous flow during diastole. Blood flows into the arterial placental vasculature during diastole, but there it does not exit through the venous system. This placental congestion raises the placental resistance ("sluice mechanism"); it may be responsible for the appearance of the notching and further decline of the diastolic flow. During the peak systolic phase of the next cardiac cycle the perfusion pressure increases and pushes the blood forward into the venous system, so creating the pulsatile flow in the umbilical vein.

We suggest that the above finding be used to assist in diagnosing and decision making in the following

clinical conditions: (1) unexplained death of one fetus in multiple pregnancies, (2) presence of variable decelerations in multiple pregnancies, and (3) likelihood of cord compression in singleton pregnancies (oligohydramnios and nuchal cord). The presence of this FVW pattern should mandate an immediate evaluation of fetal status.

References

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