Comment

Duodenal atresia results from failure of canalization of the primitive gut or from a vascular accident disrupting blood supply to a bowel segment with subsequent atrophy.[1] Both events occur early in embryonic life, at approximately 11 weeks of gestation. However, most cases of duodenal atresia have not been diagnosed until well into the third trimester of pregnancy. Early diagnosis is essential, because untreated duodenal atresia may result in death caused by emesis, aspiration, and electrolyte imbalance. Early diagnosis of duodenal atresia is also important because of its association with trisomy 21 (30% of cases), bowel malrotation (22%), congenital heart disease (20%), and other abnormalities. Nelson et al.[2] reported their experience with serial ultrasonography in the detection of duodenal atresia in two patients. In both patients results of initial ultrasonographic examinations performed at 13 and 24 weeks of pregnancy were normal. Duodenal atresia was not diagnosed until 29 weeks of pregnancy in the first case and 32 weeks in the second. The authors proposed two possible explanations for such an occurrence: either inadequate skill of the sonographer or the possible lack of fetal swallowing activity to cause dilatation of the stomach until late in pregnancy or both. The earliest diagnosis of duodenal atresia was reported in a 19-week fetus by Romero et al.[1] They emphasized that caution should be exercised in the diagnosis of duodenal atresia, because a prominent incisura angularis gastris can mimic the double-bubble appearance. This error can easily be avoided by obtaining stomach images in the transverse plane. Another potential but rare source of confusion is a choledochal cyst, mimicking a double-bubble sign. Lack of communication between the two bubbles is helpful in making a correct diagnosis.[1] This report confirms the possibility of a first-trimester diagnosis of duodenal atresia, allowing ample time for patient counseling, genetic testing, and decision making.

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Artery-to-artery anastomosis in monochorionic twins

Kurt Hecher, MD,[a] Eric Jauniaux, MD, PhD,[a] Stuart Campbell, MD,[a] Collin Deane, PhD,[b] and Kypros Nicolaides, MD[a]

London, United Kingdom

We describe Doppler signals in artery-to-artery anastomoses of two monochorionic twin pregnancies. Flow velocity waveforms demonstrated cyclic changes in systolic velocities with intermittent reversal of end-diastolic velocities. A computer model showed that this was the result of two opposing pulsatile blood flow waveforms with different velocities and frequencies. (Am J Obstet Gynecol 1994;171:570-2.)

Key words: Color Doppler, prenatal diagnosis, monochorionic twins, communicating placental vessels, twin-twin transfusion syndrome

Vascular connections between the two cord insertions are a frequent postnatal finding in monochorionic twin placentas. We describe the prenatal diagnosis of artery-to-artery anastomoses.

From the Department of Obstetrics and Gynecology[a] and the Department of Medical Engineering and Physics,[b] King's College School of Medicine and Dentistry.
Supported by a grant (J628-MED) from The Austrian Science Foundation (K.H.).
Received for publication July 21, 1993; revised December 20, 1993; accepted March 11, 1994.
Reprint requests: Kurt Hecher, MD, Department of Obstetrics and Gynecology, King's College Hospital, Denmark Hill, London, United Kingdom SE5 8RX.

0002-9378/94 $3.00 + 0 6/1/55805

Case reports

Case 1. In this case the ultrasonographic examinations were part of our routine monitoring of twin pregnancies. Serial scans every 2 weeks from 20 to 35 weeks revealed normal growth velocity in both fetuses, but the abdominal circumference of one was on the 10th percentile and that of the other was on the 70th percentile. There was also a discordancy in amniotic fluid volume between the two sacs. At 35 weeks the

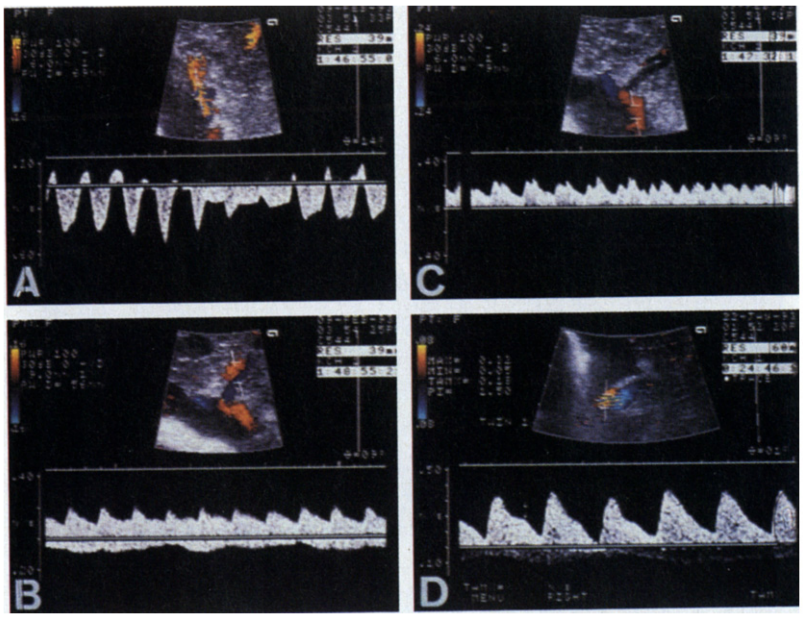

Fig. 1. A, Superficial arterial vessel on side of bigger twin, showing cyclic changes of flow velocity waveforms with reversal of end-diastolic velocities. The color image shows aliasing. **B,** Arterial and venous blood flow within shared cotyledon. **C,** Superficial arterial vessel on side of smaller twin. The flow velocity waveform shows a notch coincidental with maximum systolic velocities of the cotwin. **D,** Blood flow in free cord loop of smaller twin.

deepest pool for the smaller fetus was 30 mm and for the other it was 95 mm. An elective cesarean section was performed. The birth weights of the female infants were 2000 and 2440 gm. The hemoglobin of the smaller neonate was 17.5 gm/dl and that of the larger was 15.9 gm/dl.

Case 2. This patient was seen at 28 weeks' gestation with polyhydramnios. She had drainage of amniotic fluid at 28, 30, and 33 weeks. Ultrasonographic examination demonstrated discordancy for fetal size and amniotic fluid volume. The larger fetus had measurements on the 80th percentile, and the other had values on the 20th percentile; the respective measurements for the deepest pool of amniotic fluid at 28 weeks were 105 mm and 15 mm. At 36 weeks elective cesarean section for breech presentation was performed. The birth weights of the male infants were 2920 and 2090 gm with hemoglobin concentrations of 19.1 and 14.5 gm/dl, respectively. The neonatal course was uncomplicated for all babies.

In both cases, color Doppler imaging (Acuson 128, Acuson, Mountain View, Calif.) of the placental branches of the umbilical arteries from 28 weeks onward demonstrated a chorionic plate artery crossing the intertwin membrane and running toward an artery from the other fetus. In the first case the communication was located on the placental surface with branches going from there into the cotyledon (Fig. 1, *A* to *C*). In the second case the arterial communication was found near the placental edge and running over a thin bridge of placental tissue connecting the two placental masses. Flow velocity waveforms from both sides of the communication demonstrated cyclic changes every fvc or six heartbeats (Fig. 1, *A* and *C*). There was progressive increase followed by a decrease in systolic velocities, accompanied by opposite changes and intermittent reversal of diastolic velocities (Fig. 2). The umbilical artery waveforms in the cord of the smaller twin in the first case were also affected but much less so than in the placental arteries (Fig. 1, *D*).

The waveform was simulated by means of a computer model on an IBM PC computer (IBM, London) and mathematics software (PC-MATLAB, The MathWorks, South Natick, Mass.). Two pressure waveforms of different amplitudes and frequencies representing those of the fetal umbilical arteries were simulated. The opposing action of these waveforms produced a resulting waveform (Fig. 2) that closely matched the flow waveform obtained by Doppler ultrasonography, thus demonstrating the presence of artery-to-artery communication.

Macroscopic examination of the placenta confirmed vascular connections in both cases. In the first case the only connection was from a single artery to artery, whereas in the second case perfusion studies showed four cotyledons supplied by arteries from both sides.

Comment

This report demonstrates the successful application of color flow mapping and flow velocimetry in the

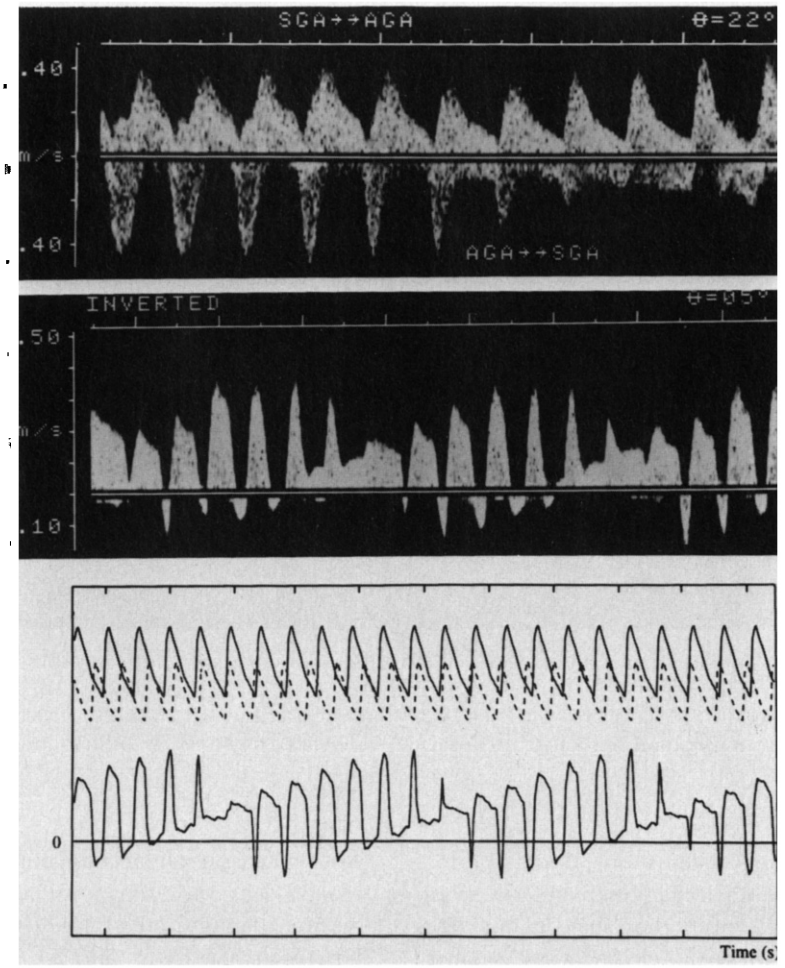

Fig. 2. *Top,* Simultaneous recording of blood flow in superficial placental vessels from smaller toward bigger twin *(SGA→→AGA)* and from bigger toward smaller twin *(AGA→→SGA). Middle,* Cyclic changes in pulsatility of arterial blood flow in superficial vessel on placental surface of bigger twin. *Bottom,* Simulation of waveforms. The diagram depicts the resulting waveform arising from two opposing pressure waves. The pressure waves are shown at the *top* of the diagram. The *solid line* represents a pressure waveform with a heart rate of 124 beats/min, and the *broken line* represents one of lower magnitude with a heart rate of 142 beats/min. The *lower line* represents subtraction of the lower pressure *(broken line)* from the higher pressure *(solid line).*

prenatal diagnosis of artery-to-artery anastomoses in monochorionic twin pregnancies. Erskine et al.[1] have previously described a monochorionic twin pregnancy with cyclic changes of umbilical artery Doppler signals of the smaller twin. This fetus died suddenly at 36 weeks' gestation, and the authors suggested that death was the consequence of raised cardiac workload caused by an arterial anastomosis to a dominant twin.

Although there may be differences in the systemic pressure of the two fetuses, in our cases the waveforms in both communicating arteries demonstrated net forward flow, and it can be presumed that there was no net shift of blood from one to the other. However, acute twin transfusion syndrome may occur if one fetus suddenly becomes hypotensive. This can occur with death of one fetus. It is also possible that even when both fetuses are alive there are intermittent episodes of pressure imbalance of the two circulations, resulting in cerebral necrosis caused by transient episodes of unidirectional flow of blood across vein-to-vein or artery-to-artery anastomoses.[2]

The development of new techniques such as laser ablation of the communicating vessels may prevent the catastrophic consequences of death or brain damage as a result of an unpredictable sudden episode of pressure imbalance in the two circulations.

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