
بسم الله الرحمن الرحيم

MANANGMENT OF IUGR

&

DOPPLER

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Perinatalogist

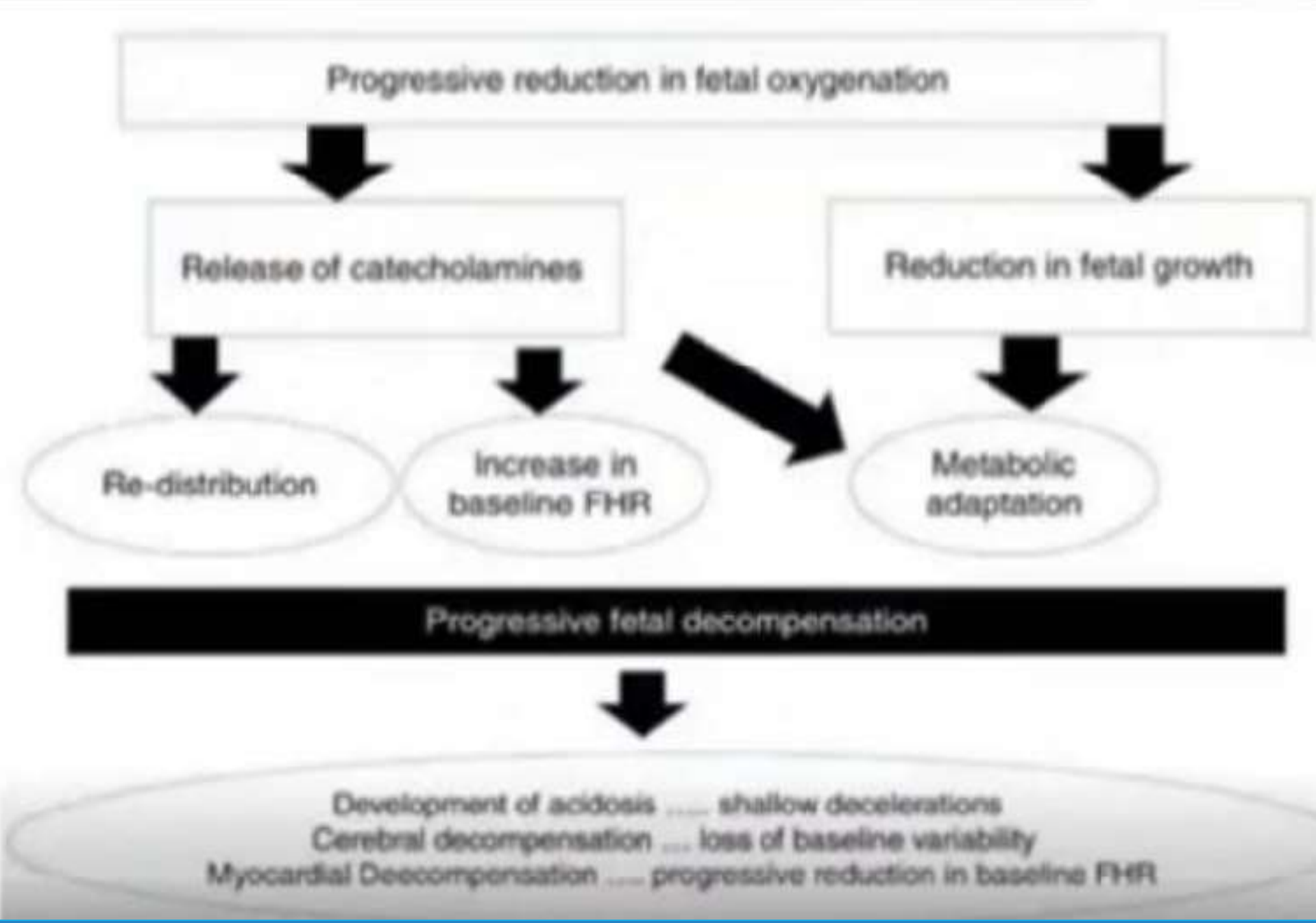
Table 1 Main clinical characteristics of early- and late-onset fetal growth restriction (FGR)

Characteristic	*Early-onset FGR*	*Late-onset FGR*
Main clinical challenge	Management	Detection
Prevalence	30%	70%
Gestational age at manifestation	< 32 weeks	≥ 32 weeks
Ultrasound findings	Fetus may be very small	Fetus not necessarily very small
Doppler velocimetry	Spectrum of Doppler alterations that involves umbilical artery, middle cerebral artery and ductus venosus	Cerebral blood-flow redistribution
Biophysical profile	May be abnormal	May be abnormal
Hypertensive disorders of pregnancy	Frequent	Not frequent
Placental histopathological findings	Poor placental implantation, spiral artery abnormalities, maternal vascular malperfusion	Less specific placental findings, mainly altered diffusion
Perinatal mortality	High	Low
Maternal cardiovascular hemodynamic status	Low cardiac output, high peripheral vascular resistance	Less marked maternal cardiovascular findings

Table 2 Definitions for early- and late-onset fetal growth restriction (FGR) in absence of congenital anomalies, based on international Delphi consensus

Early FGR: *GA < 32 weeks, in absence of congenital anomalies*	*Late FGR:* *GA ≥ 32 weeks, in absence of congenital anomalies*
AC/EFW < 3rd centile *or* UA-AEDF *Or* 1. AC/EFW < 10th centile *combined with* 2. UtA-PI > 95th centile *and/or* 3. UA-PI > 95th centile	AC/EFW < 3rd centile *Or at least two out of three of the following* 1. AC/EFW < 10th centile 2. AC/EFW crossing centiles > 2 quartiles on growth centiles* 3. CPR < 5th centile *or* UA-PI > 95th centile

*Growth centiles are non-customized centiles. AC, fetal abdominal circumference; AEDF, absent end-diastolic flow; CPR, cerebroplacental ratio; EFW, estimated fetal weight; GA, gestational age; PI, pulsatility index; UA, umbilical artery; UtA, uterine artery. Reproduced from Gordijn *et al.*[16].


Progressive reduction in fetal oxygenation
Release of catecholamines
Reduction in fetal growth
Re-distribution
Increase in baseline FHR
Metabolic adaptation
Progressive fetal decompensation
Development of acidosis ..... shallow decelerations
Cerebral decompensation .... loss of baseline variability
Myocardial Deecompensation ..... progressive reduction in baseline FHR

Tools for diagnosis, surveillance and fetal Management of growth restriction

Fetal growth velocity

Customized growth charts

Dappler velocimetry

Biophysical profile scoring

Cardiotocography and short-term variation

Biomarkers

DOPPLER VELOCIMETRY

Blood flow velocity measured by Doppler ultrasound reflects downstream impedance . For growth-restricted fetuses, several fetal vascular circuits including the umbilical artery, middle cerebral artery, and ductus venosus have been evaluated as diagnostic tools for fetal well-being . Maternal uterine artery Doppler velocimetry has also been assessed as a modality to predict placental dysfunction, with the goal to balance stillbirth against the risks of preterm delivery . . Even the effects of sildenafil in pregnant sheep have been evaluated using Doppler velocimetry (Alanne, 2017). The rationale is that sildenafil would improve placental blood flow in the presence of placental insufficiency. This proved untrue, as sildenafil was associated with detrimental effects on fetal cardiovascular dynamics.

Doppler Blood Flow Velocity

Waveforms were first studied in the umbilical arteries late in pregnancy, and abnormal waveforms correlated with placental villous hypovascularity. Of the small placental arterial channels, 60 to 70 percent need to be obliterated before the umbilical artery Doppler waveform becomes abnormal. Such extensive placental vascular pathology has a major effect on fetal circulation. According to Trudinger (2007), because more than 40 percent of the combined fetal ventricular output is directed to the placenta, obliteration of placental vascular channel increases afterload and leads to fetal hypoxemia. This in turn leads to ventricular dilation and redistribution of middle cerebral artery blood flow. Ultimately, pressure rises in the ductus venosus due to afterload in the right side of the fetal heart (Baschat, 2004). Clinically, abnormal Doppler waveforms in the ductus venosus are a late finding in the progression of fetal deterioration due to chronic hypoxemia.

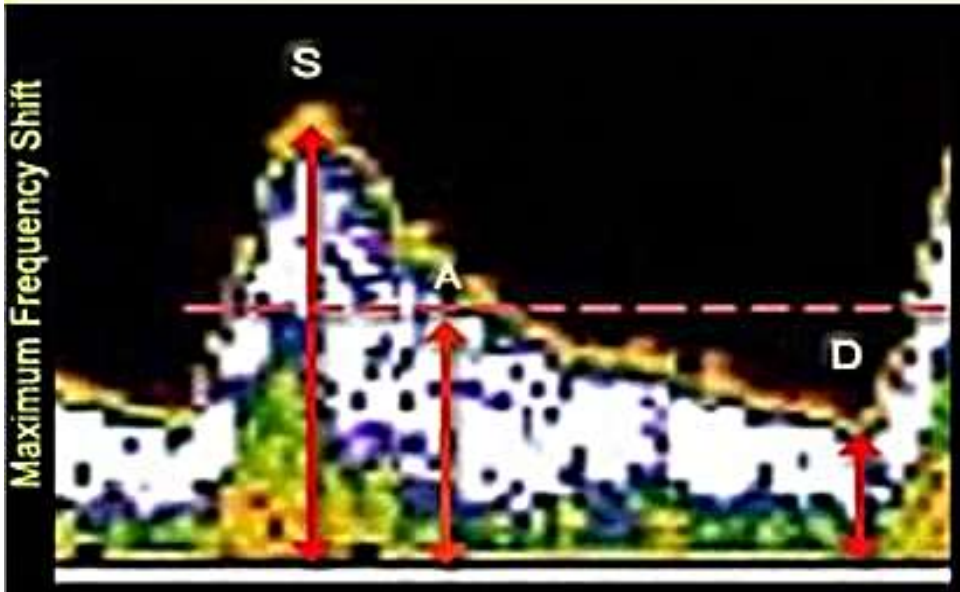
Umbilical Artery Velocimetry

The umbilical artery systolic-diastolic (S/D) ratio is considered abnormal if it is >95th percentile for gestational age or if diastolic flow is either absent or reversed (Chap. 10, Doppler). Absent or reversed end-diastolic flow signifies greater impedance to umbilical artery blood flow (Fig. 44-8). It is reported to result from poorly vascularized placental villi and is seen in extreme cases of fetal-growth restriction (Todros, 1999). According to Zelop and colleagues (1996), the perinatal mortality rate for absent end-diastolic flow was about 10 percent, and for reversed end-diastolic flow, it approximated 33 percent. studied neurodevelopmental outcome at 2 years of age in 266 growth-restricted fetuses delivered between 24 and 35 weeks' gestation. Of infants who had shown absent or reversed umbilical artery flow, 8 percent had evidence of cerebral palsy compared with 1 percent of those in whom Doppler flow had been normal.

Which measurement to use?

Angle independent indices

Angle < 90 degrees



S = systolic peak (max. velocity)
D = end diastolic flow
Vm = mean velocity
A = Temporal average frequency over 1 cardiac cycle

Doppler Indices

RI = (S - D) / S (Pourcelot, 1974)

PI = (S - D) / A (Gosling, 1976)

S/D Ratio = S/D (Stuart & Drumm, 1980)

pulsatility index (PI) preferred

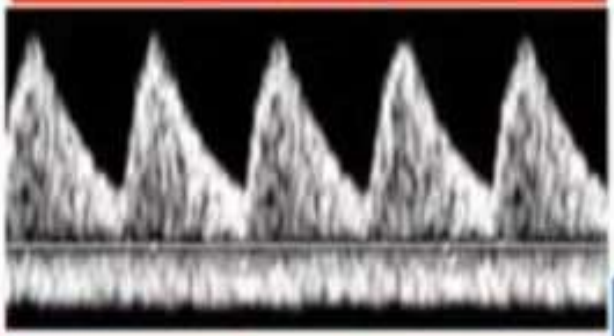
Middle Cerebral Artery

As discussed, at this time, Doppler velocimetry interrogation of the middle cerebral artery (MCA) to detect fetal compromise is not recommended. Still, the technology has received particular attention because of observations that the hypoxic fetus attempts *brain sparing* by reducing cerebrovascular impedance and thus increasing blood flow. Such brain sparing in growth-restricted fetuses has been documented to undergo reversal (Konje, 2001). Investigators reported that 8 of 17 fetuses with this reversal died. Ott and coworkers (1998) randomized 665 women undergoing modified biophysical profile evaluation to either the profile alone or combined with middle cerebral and umbilical artery velocity flow assessment. Pregnancy outcomes between these two study groups did not differ significantly.

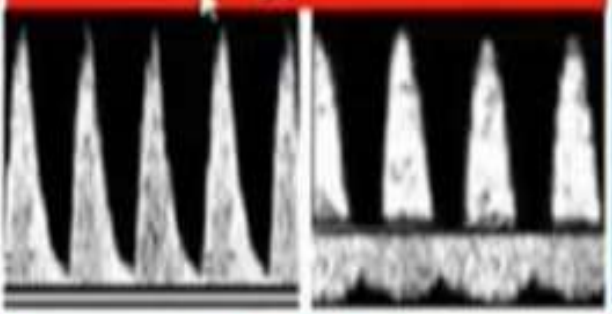
Middle cerebral artery Doppler velocimetry has proven valuable to detect severe fetal anemia in 165 fetuses with d-antigen alloimmunization. Oepkes and colleagues (2006) prospectively compared serial amniocentesis for measurement of bilirubin levels with Doppler measurement of peak systolic velocity in the middle cerebral artery. These investigators concluded that Doppler could safely replace amniocentesis in the management of alloimmunized pregnancies. And as discussed in Chapter 15 (Management of the Alloimmunized Pregnancy), this technique has been reported to be useful for detection and management of fetal anemia of any cause (Moise, 2008).

Fetal Doppler in FGR: Umbilical and Middle cerebral artery

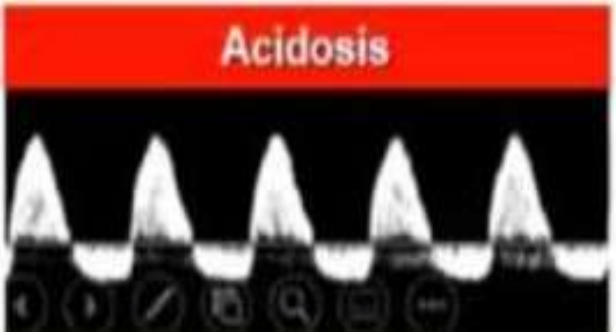
Normal

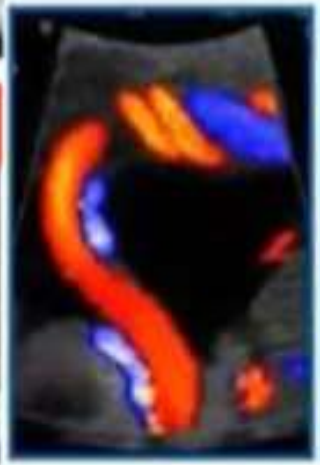


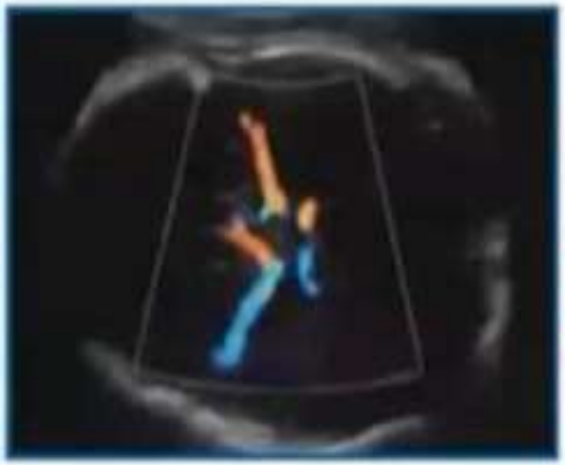
Hypoxia



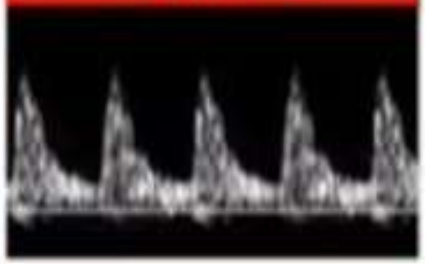
Acidosis



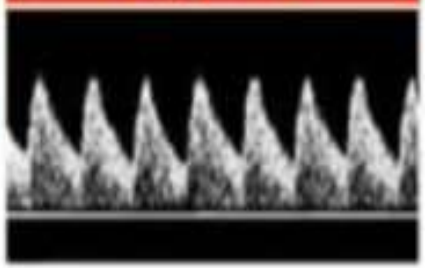




Normal



Hypoxia



Ductus Venosus

Doppler ultrasound has also been used to assess the fetal venous circulation. Bilardo and colleagues (2004) prospectively studied umbilical artery and ductus venosus Doppler velocimetry in 70 growth-restricted fetuses at 26 to 33 weeks' gestation. They concluded that ductus venosus velocimetry was the best predictor of perinatal outcome. Importantly, negative or reversed flow in the ductus venosus was a late finding because these fetuses had already sustained irreversible multiorgan damage due to hypoxemia. Also, gestational age at delivery was a major determinant of perinatal outcome independent of ductus venosus flow. Specifically, 36 percent of growth-restricted fetuses delivered between 26 and 29 weeks' gestation succumbed compared with only 5 percent delivered from 30 to 33 weeks.

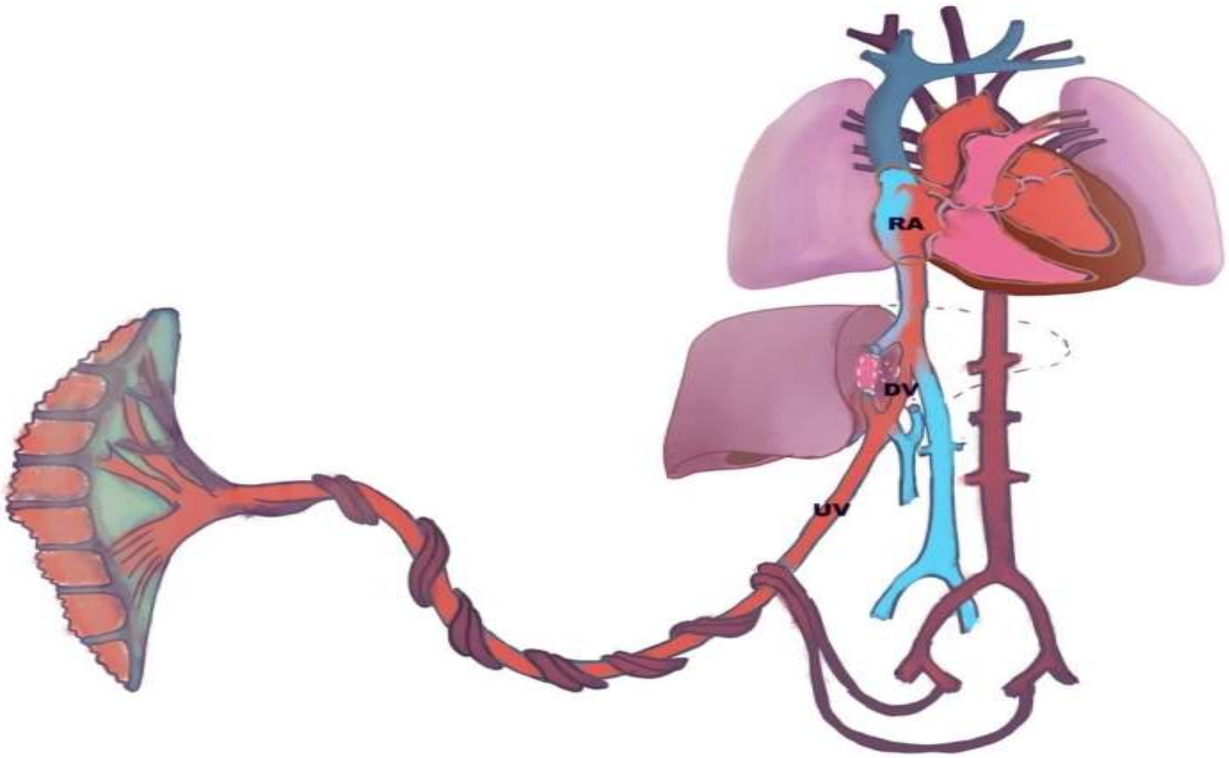
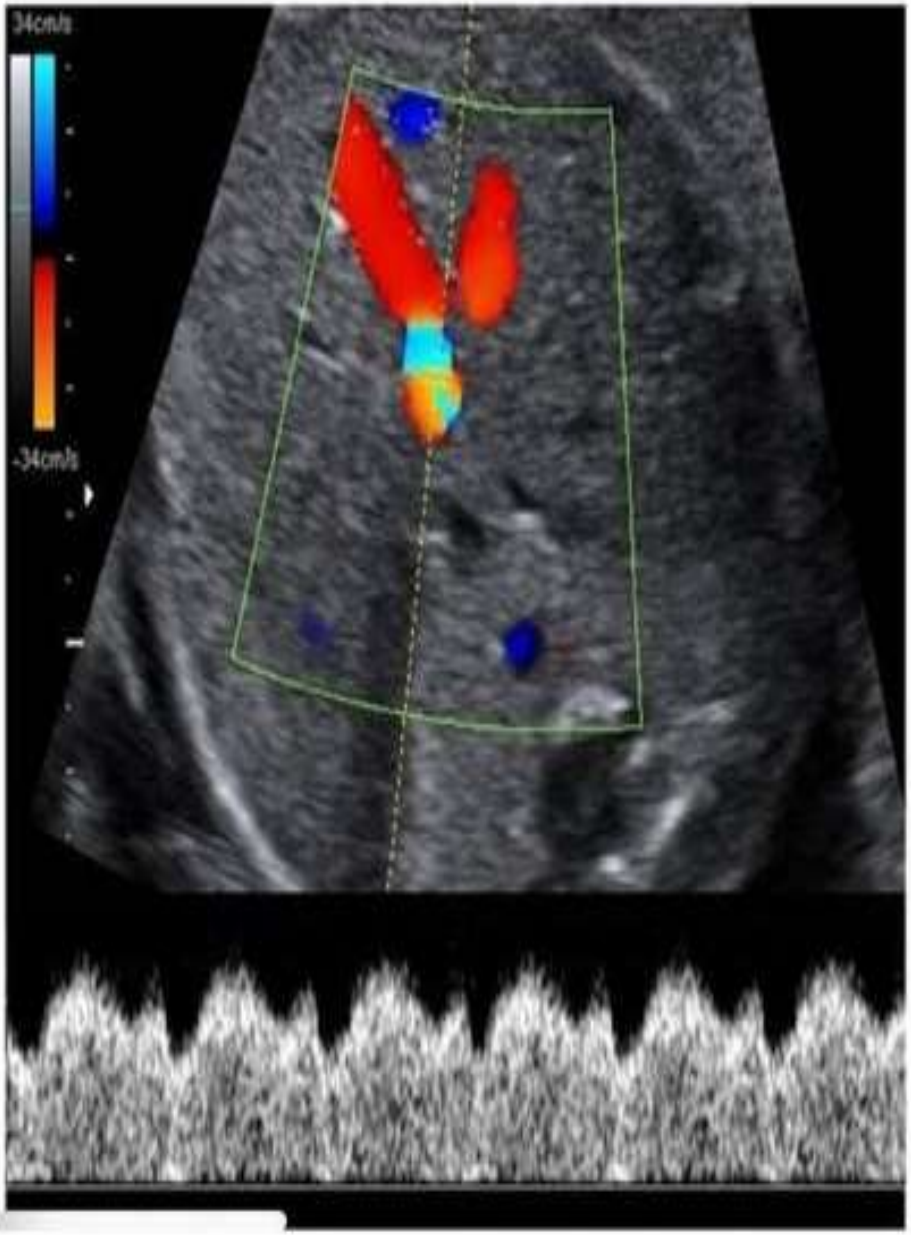


Figure 1. Anatomy of the ductus venosus (DV). The **DV** is a vascular shunt situated within the fetal liver parenchyma connecting the umbilical vein (UV) to the inferior vena cava and right atrium (RA).


34cm/s
-34cm/s


34cm/s
-34cm/s

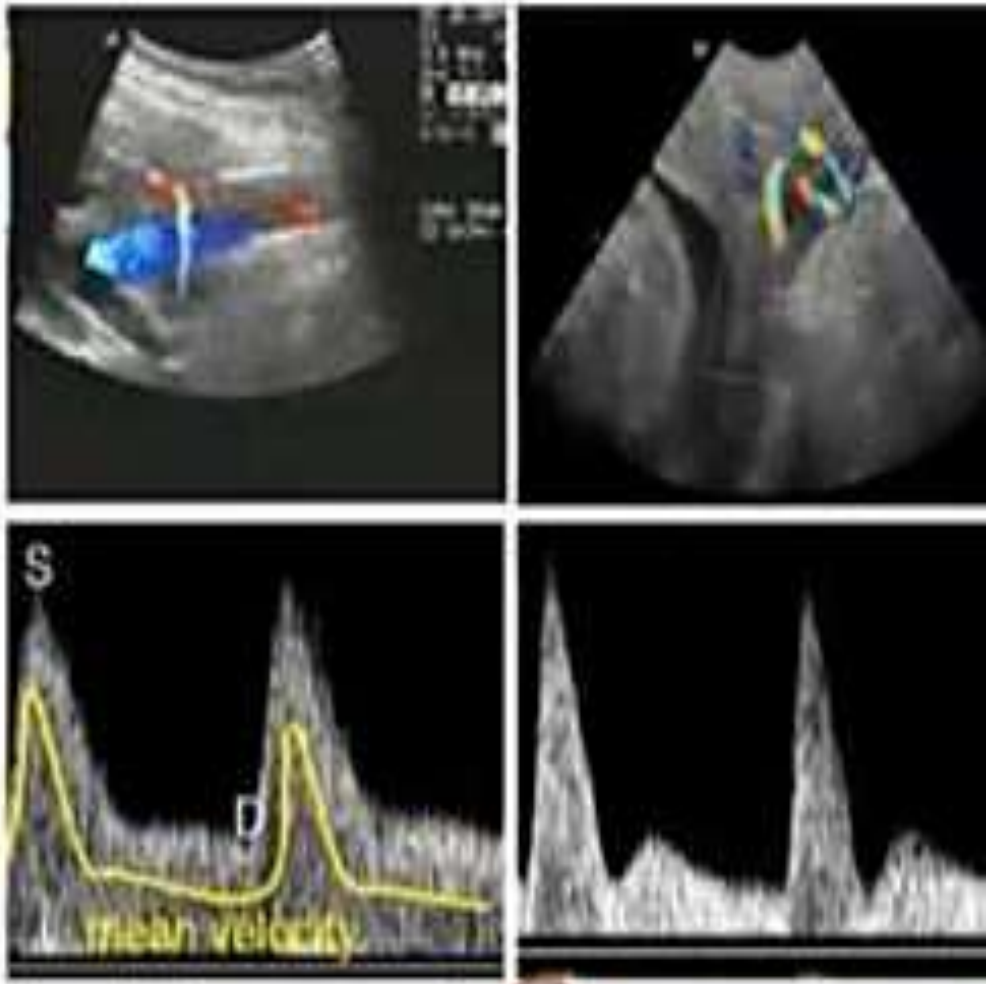
Uterine Artery

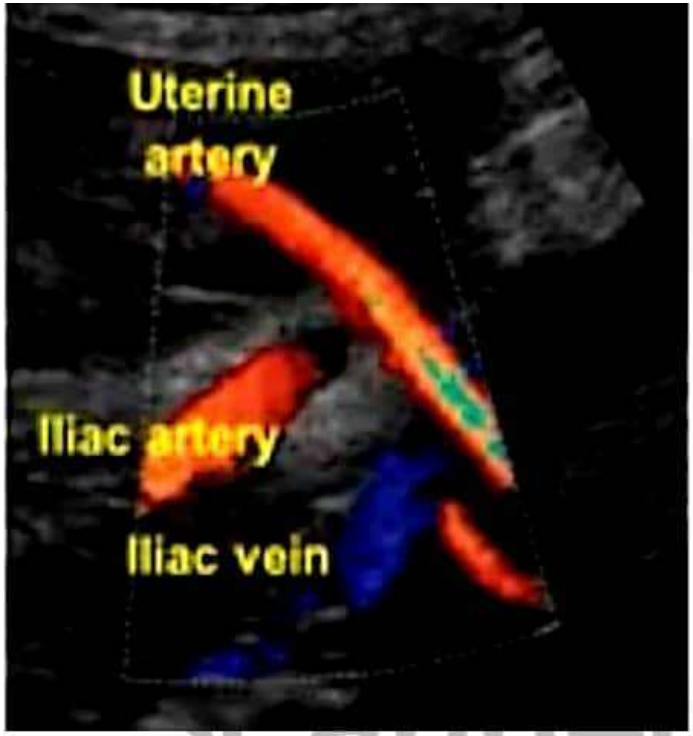
Vascular resistance in the uterine circulation normally decreases in the first half of pregnancy due to invasion of maternal uterine vessels by trophoblastic tissue (Chap. 5, Endometrial Cycle). This process can be detected using Doppler flow velocimetry, and uterine artery Doppler may be most helpful in assessing pregnancies at high risk of uteroplacental insufficiency (Abramowicz, 2008). Persistence or development of high-resistance patterns has been linked to various pregnancy complications (Lees, 2001; Yu, 2005). In a study of 30,519 unselected British women, Smith and colleagues (2007) assessed uterine artery velocimetry at 22 to 24 weeks' gestation. The risk of fetal death before 32 weeks, when associated with abruption, preeclampsia, or fetal-growth restriction, was significantly linked to high-resistance flow. This has led to suggestions for continued research of uterine artery Doppler velocimetry as a screening tool to detect pregnancies at risk for stillbirth (Reddy, 2008). Sciscione and Hayes (2009) reviewed the use of uterine artery Doppler flow studies in obstetrical practice. Because standards for the study technique and criteria for an abnormal test are lacking, they noted that uterine artery Doppler studies should not be considered standard practice in either low- or high-risk populations.

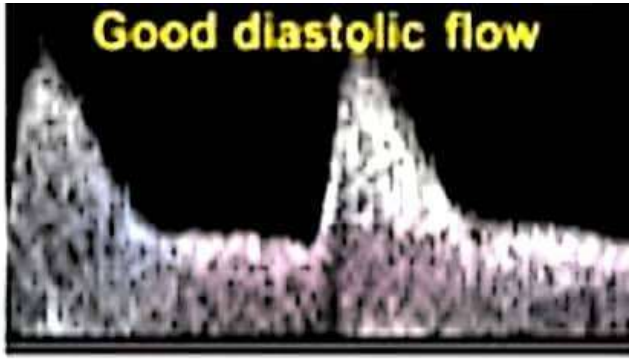
Mean uterine artery PI should be the Doppler index of choice (GRADE B)

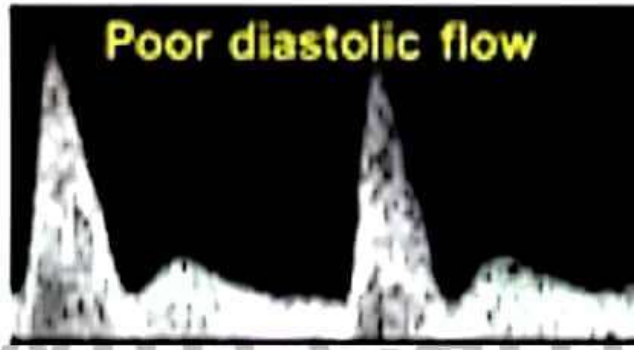
- PI includes in its calculation the *averaged value of all maximum velocities during the cardiac cycle*, rather than just two points in the cardiac cycle as for RI

- PI is *more stable* and it does not approach infinity when there are absent or reversed diastolic values



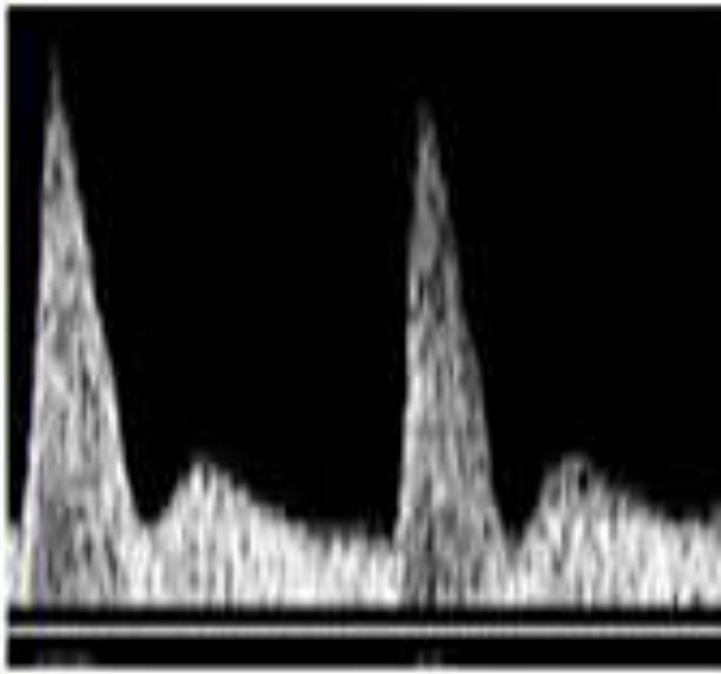


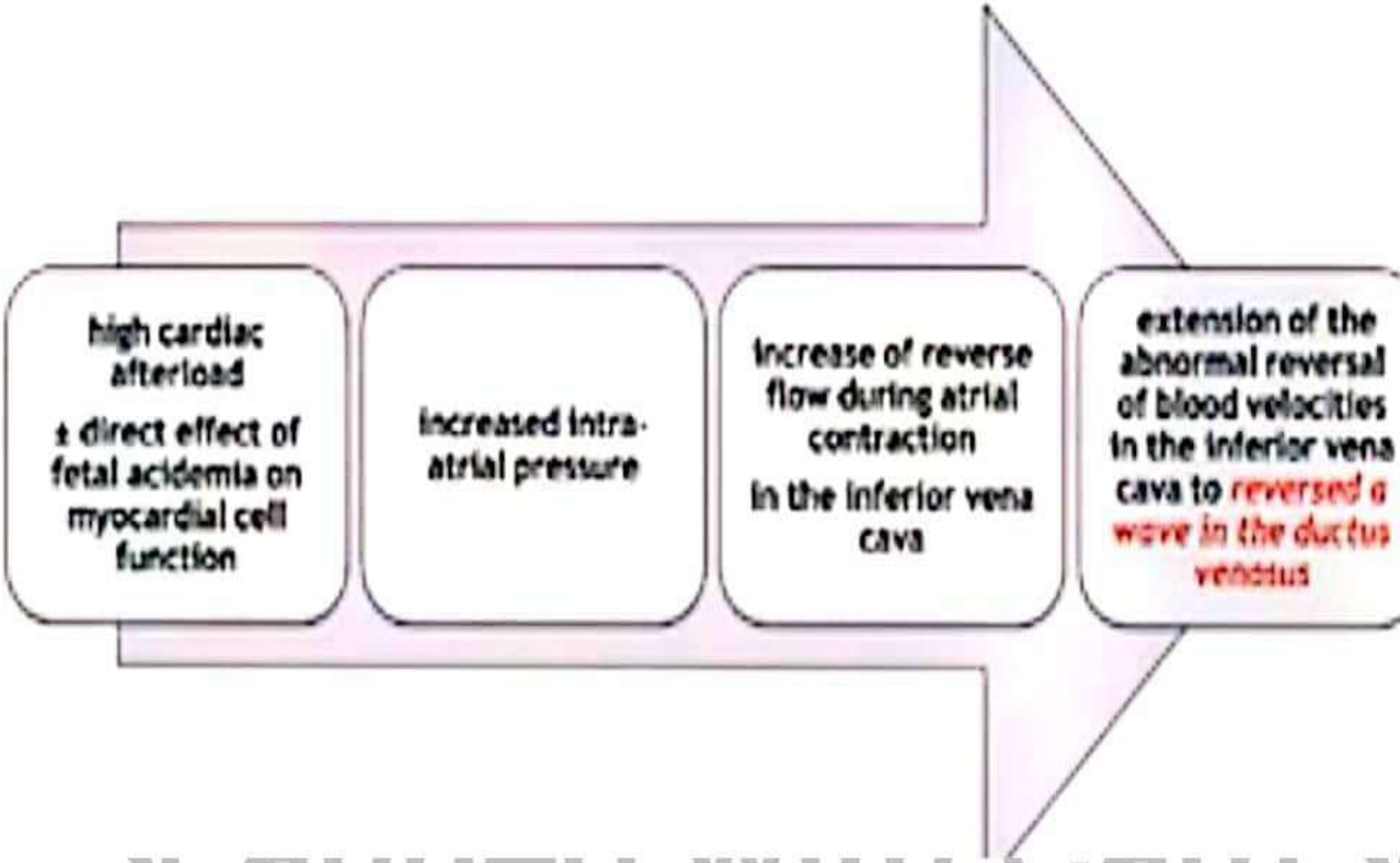




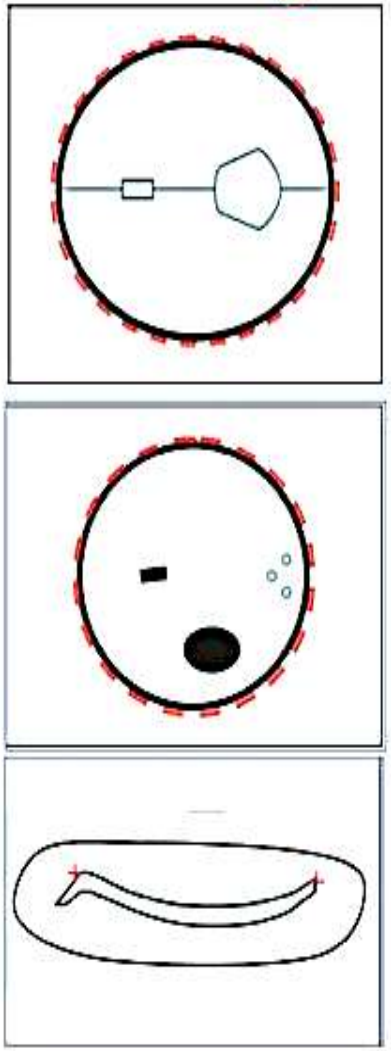
Uterine artery Notching

- Subjective finding
- Bilateral notching is present in 1st trimester in 43% of normal pregnancies (↓specificity)
- Notching in the 2nd trimester: higher screen-positive rate than PI

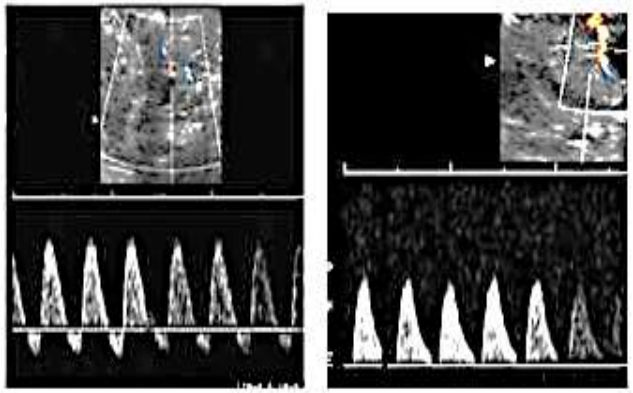




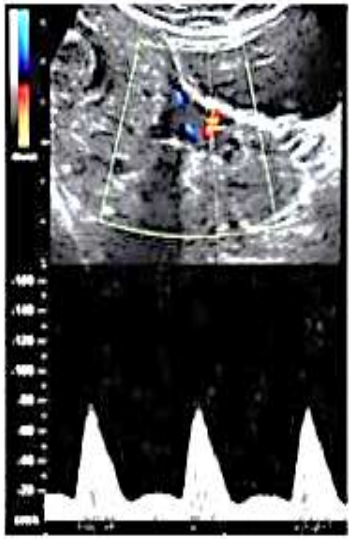
Distinguishing between FGR and SGA



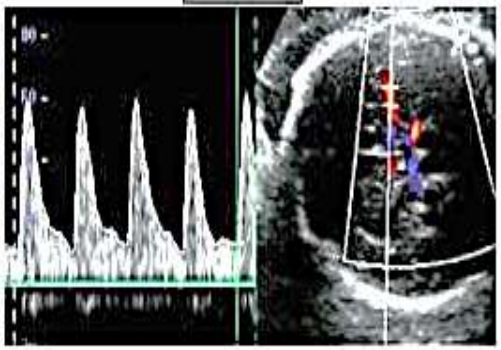
umbilical artery



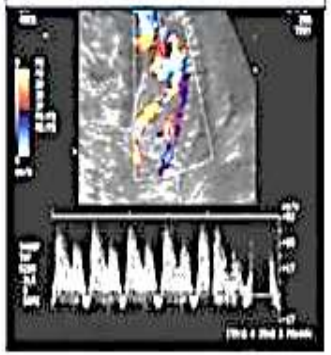
uterine artery

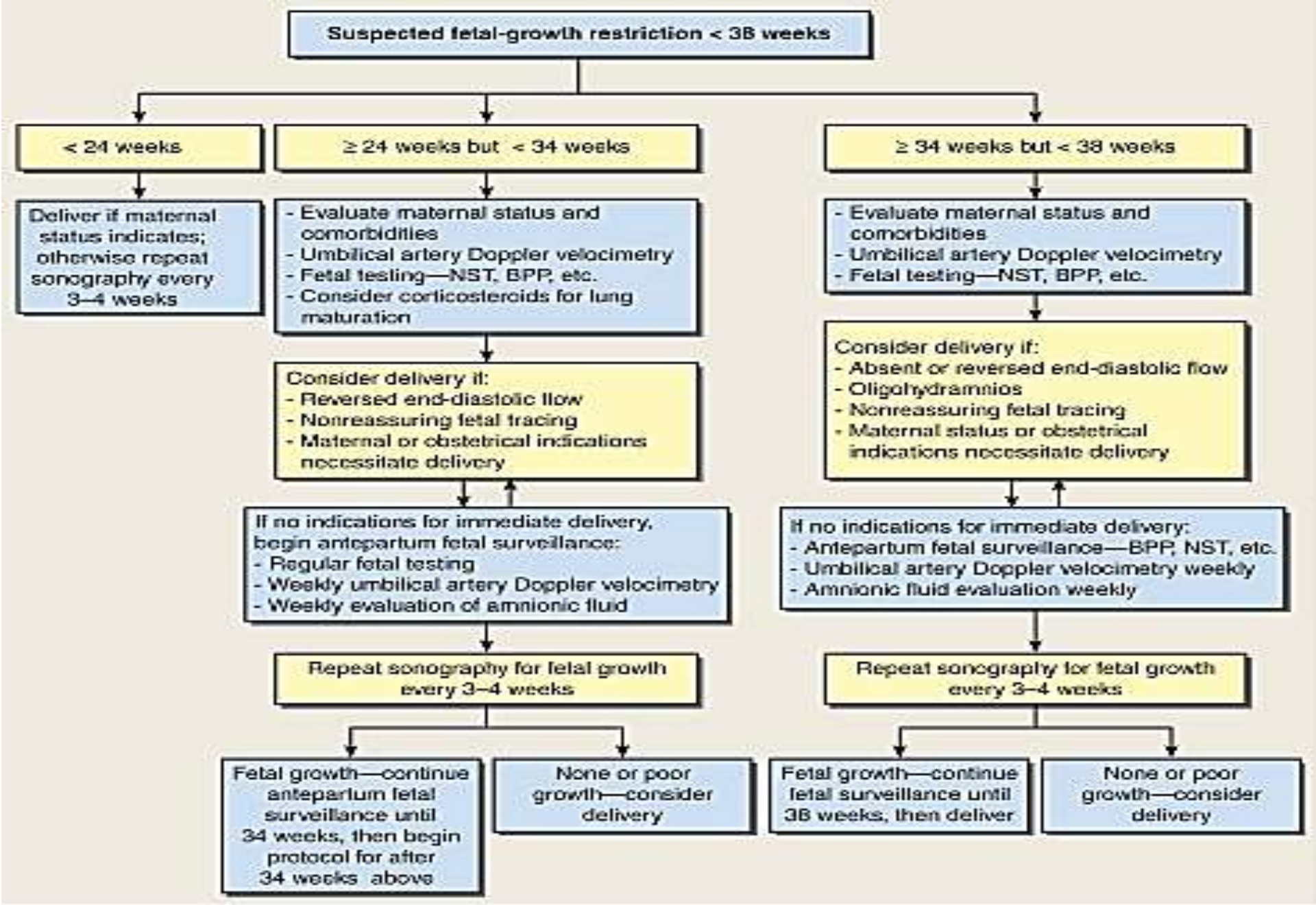


MCA

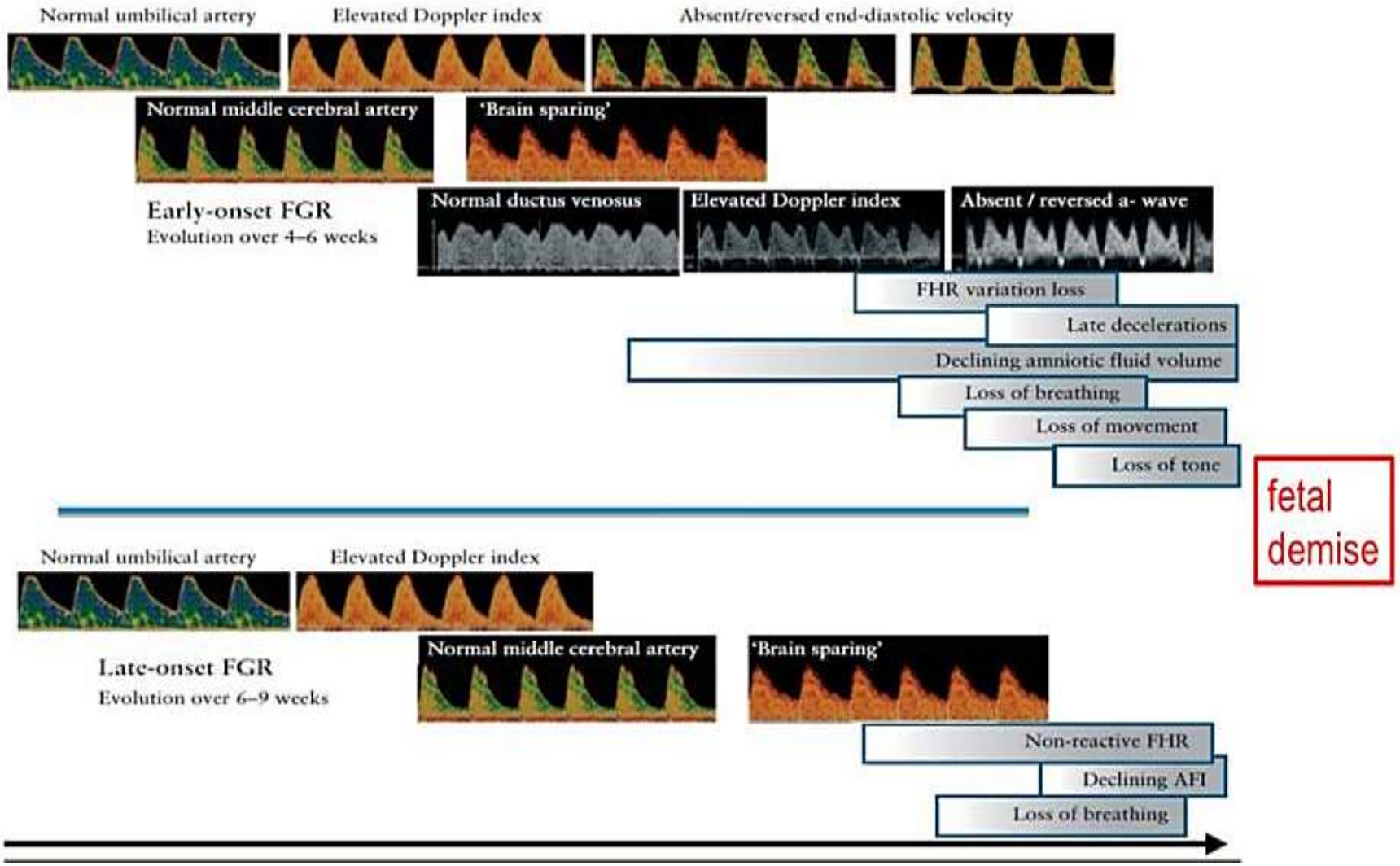


ductus venosus



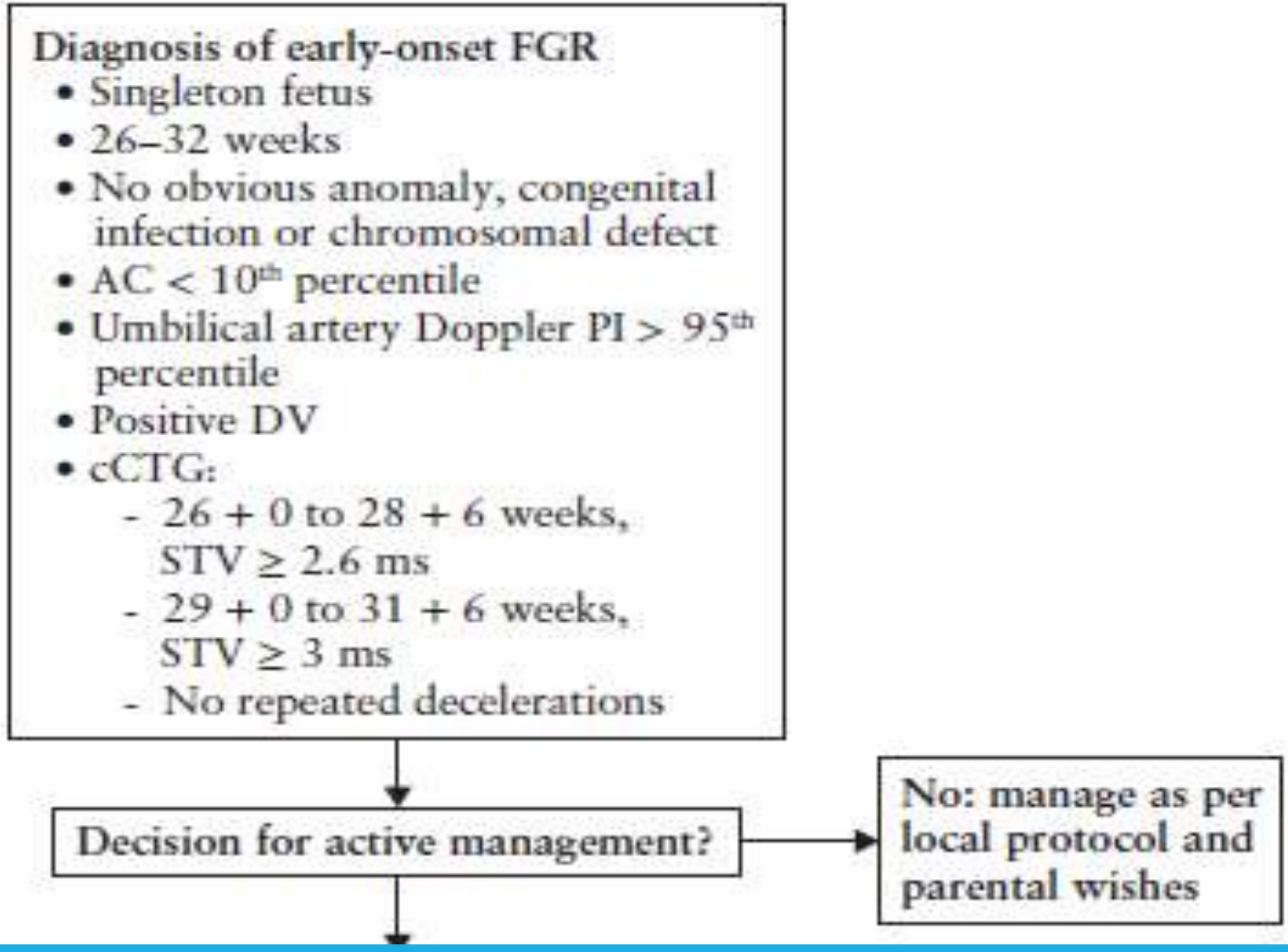

Suspected fetal-growth restriction < 38 weeks
< 24 weeks
≥ 24 weeks but < 34 weeks
≥ 34 weeks but < 38 weeks
Deliver if maternal status indicates; otherwise repeat sonography every 3–4 weeks
- Evaluate maternal status and comorbidities
- Umbilical artery Doppler velocimetry
- Fetal testing—NST, BPP, etc.
- Consider corticosteroids for lung maturation
- Evaluate maternal status and comorbidities
- Umbilical artery Doppler velocimetry
- Fetal testing—NST, BPP, etc.
Consider delivery if:
- Reversed end-diastolic flow
- Nonreassuring fetal tracing
- Maternal or obstetrical indications necessitate delivery
Consider delivery if:
- Absent or reversed end-diastolic flow
- Oligohydramnios
- Nonreassuring fetal tracing
- Maternal status or obstetrical indications necessitate delivery
If no indications for immediate delivery, begin antepartum fetal surveillance:
- Regular fetal testing
- Weekly umbilical artery Doppler velocimetry
- Weekly evaluation of amniotic fluid
If no indications for immediate delivery:
- Antepartum fetal surveillance—BPP, NST, etc.
- Umbilical artery Doppler velocimetry weekly
- Amniotic fluid evaluation weekly
Repeat sonography for fetal growth every 3–4 weeks
Repeat sonography for fetal growth every 3–4 weeks
Fetal growth—continue antepartum fetal surveillance until 34 weeks, then begin protocol for after 34 weeks above
None or poor growth—consider delivery
Fetal growth—continue fetal surveillance until 38 weeks, then deliver
None or poor growth—consider delivery

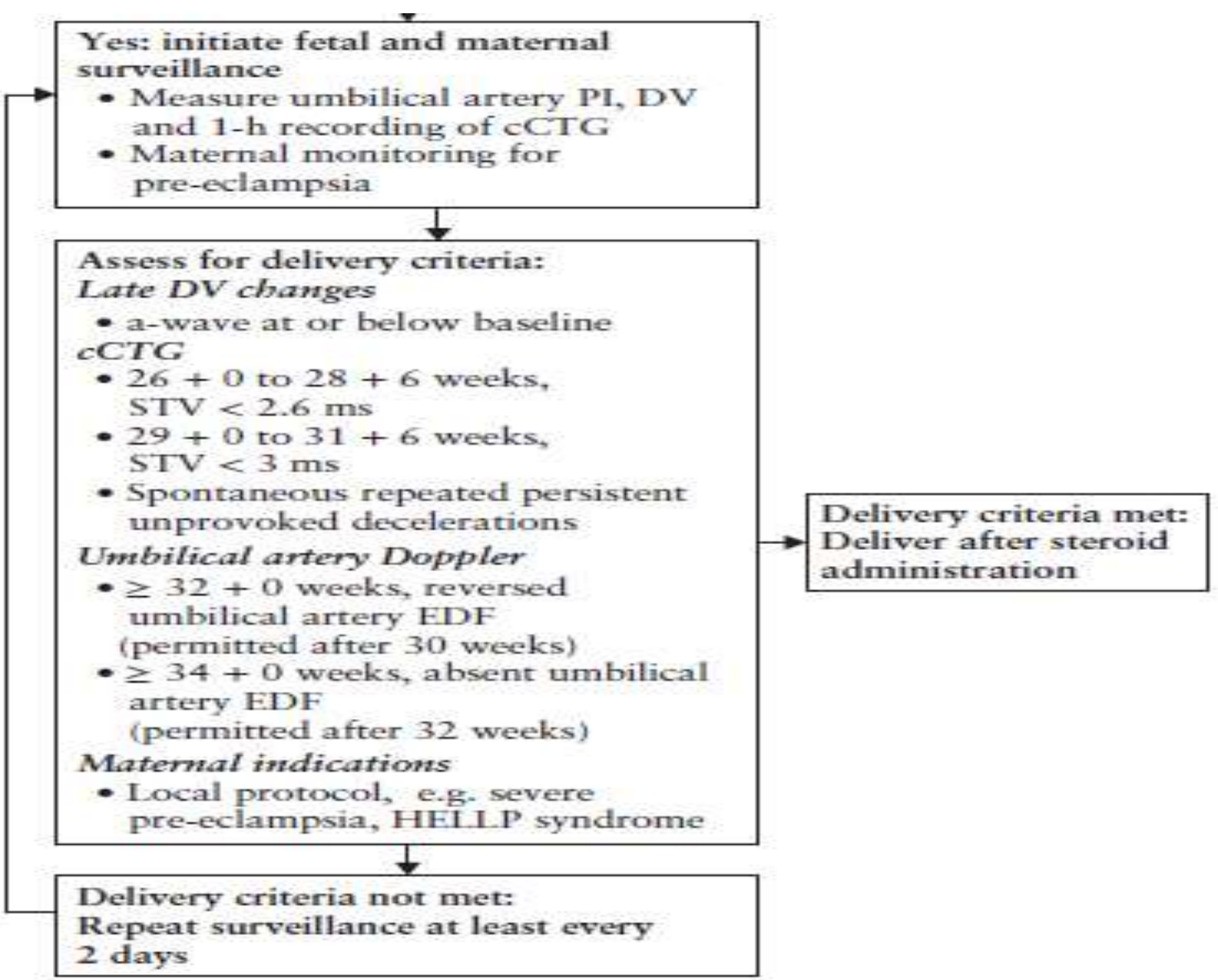
Placental insufficiency, early and late response



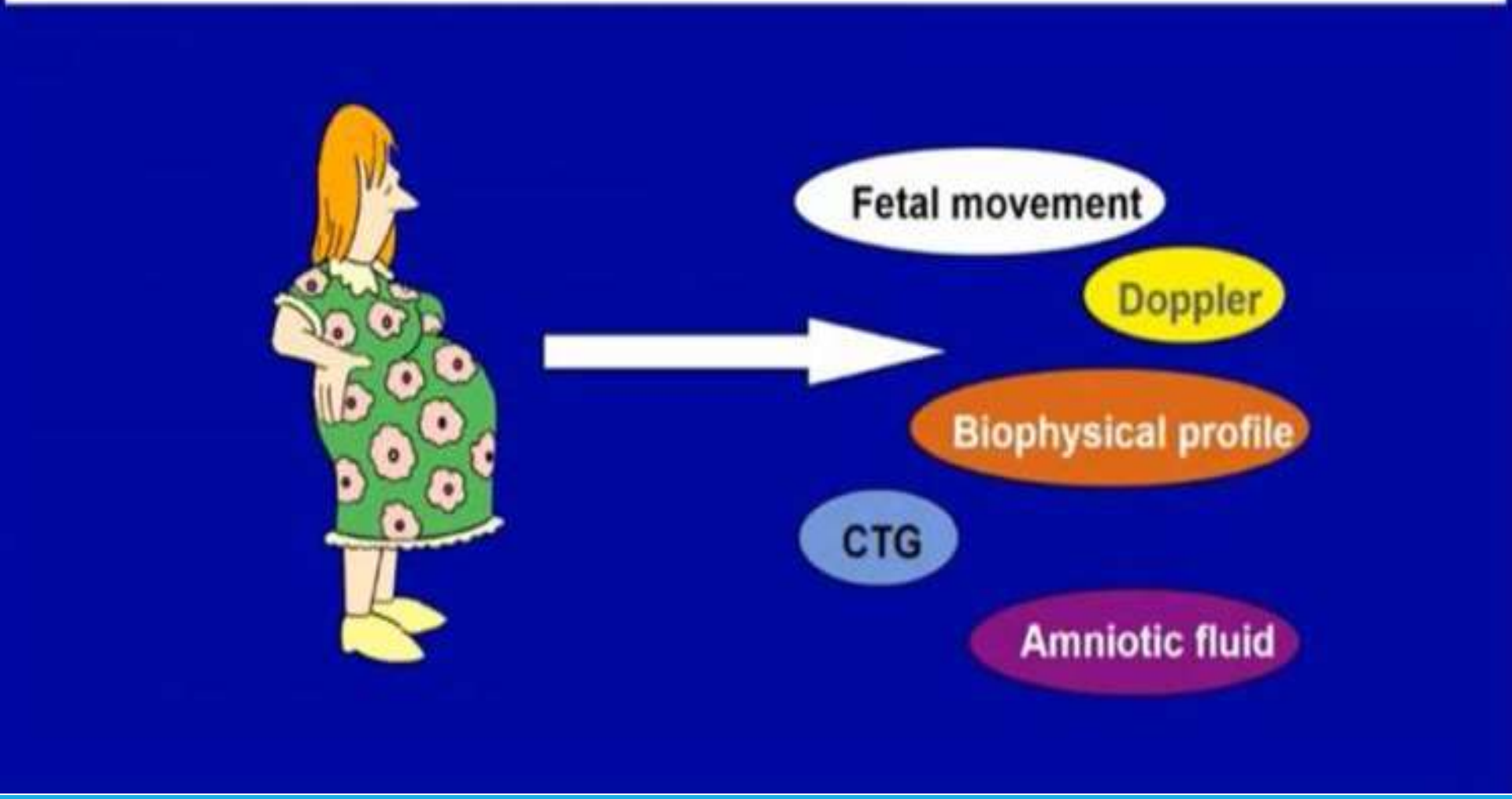
Corticosteroid prophylaxis

There is a lack of consensus between guidelines with respect to corticosteroid prophylaxis between 34 and 36weeks' gestation. Most guidelines on FGR recommend corticosteroid prophylaxis if the birth is likely to occur before 34+0weeks70–74, however, the RCOG recommends corticosteroid prophylaxis up to 35+6weeks67.

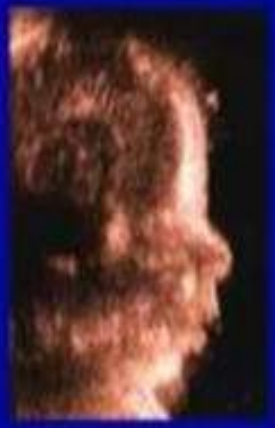

Diagnosis of early-onset FGR
• Singleton fetus
• 26–32 weeks
• No obvious anomaly, congenital infection or chromosomal defect
• AC < 10th percentile
• Umbilical artery Doppler PI > 95th percentile
• Positive DV
• cCTG:
- 26 + 0 to 28 + 6 weeks, STV ≥ 2.6 ms
- 29 + 0 to 31 + 6 weeks, STV ≥ 3 ms
- No repeated decelerations
Decision for active management?
No: manage as per local protocol and parental wishes


Yes: initiate fetal and maternal surveillance
• Measure umbilical artery PI, DV and 1-h recording of cCTG
• Maternal monitoring for pre-eclampsia
Assess for delivery criteria:
Late DV changes
• a-wave at or below baseline
cCTG
• 26 + 0 to 28 + 6 weeks, STV < 2.6 ms
• 29 + 0 to 31 + 6 weeks, STV < 3 ms
• Spontaneous repeated persistent unprovoked decelerations
Umbilical artery Doppler
• ≥ 32 + 0 weeks, reversed umbilical artery EDF (permitted after 30 weeks)
• ≥ 34 + 0 weeks, absent umbilical artery EDF (permitted after 32 weeks)
Maternal indications
• Local protocol, e.g. severe pre-eclampsia, HELLP syndrome
Delivery criteria met: Deliver after steroid administration
Delivery criteria not met: Repeat surveillance at least every 2 days

Placental insufficiency: Which test?



Response to Fetal Hypoxaemia



Worsening Fetal hypoxaemia

Chemoreceptor stimulation

Arterial redistribution

Increased blood flow:
- brain
- adrenals
- heart

Decreased blood flow:
- kidneys (oliguria)
- lungs (RDS)
- GUT (NEC)
- Liver (IUGR)

CNS cellular dysfunction

- Non-reactive CTG
- Absent fetal breathing
- Fetal hypotonia
- Absent fetal movements

Summery:

- IUGR fetuses demonstrate progressive hemodynamic changes.
- Umbilical artery pulsatility index is the first variable to become abnormal, followed by the middle cerebral artery, right diastolic indices (right E/A, ductus venosus), right systolic indices, and finally, both diastolic and systolic left cardiac indices.
- It appears that there is an earlier and more pronounced right than left and diastolic than systolic fetal cardiac function deterioration in growth-restricted fetuses