Diploma in Fetal Medicine & ISUOG Educational Series
The 18 - 23 weeks scan
Gianluigi Pilu, Kypros Nicolaides, Renato Ximenes & Phillipe Jeanty

Chapter 13

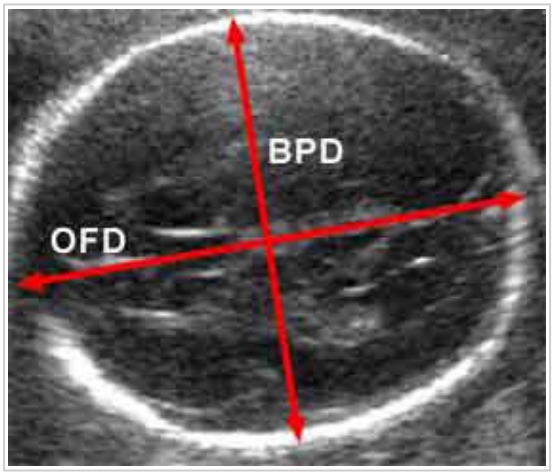
Small for gestational age

SMALL FOR GESTATIONAL AGE

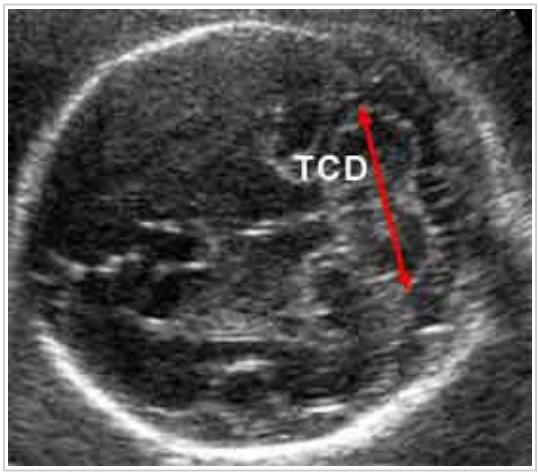
Small for gestational age fetuses are defined by the finding that the abdominal circumference is below the 5th centile for gestation. About 80% of such fetuses are constitutionally small, with no increased perinatal death or morbidity, 15% are growth restricted due to reduced placental perfusion and "utero-placental insufficiency", and 5% are growth restricted due to low growth potential, the result of genetic disease or enviromental damage.

Ultrasound findings

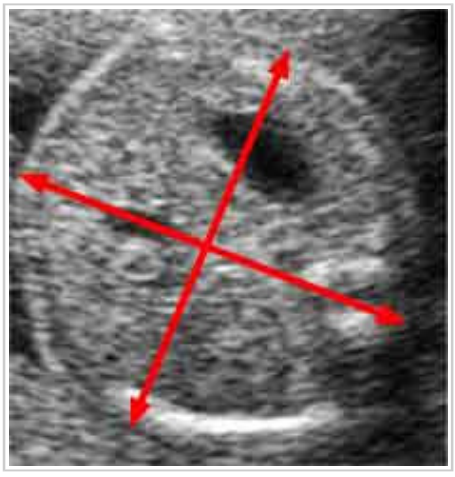
The finding of a small abdominal circumference should stimulate the sonographer to consider four possible causes: wrong dates, normal small, abnormal small or starving small fetus. Accurate measurements of the head and abdominal circumference, femur length and transverse cerebellar diameter should be taken and their various ratios should be examined. Additionally, a detailed examination should be carried out for the detection of any defects or markers of chromosomal abnormalities (mainly triploidy and trisomy 18), and for assessment of amniotic fluid and fetal activity.



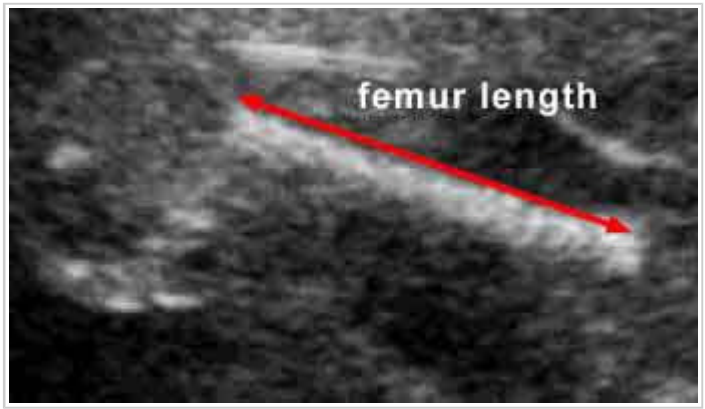
BPD / OFD / HC



TCD



AC



FL

In cases of wrong dates, there may be a suggestive history (uncertain last menstrual period, irregular cycle, conception within three months of stopping the contraceptive pill or breast feeding), all measurements symmetrically small, no obvious anatomical defects, normal amniotic fluid volume and fetal activity. A repeat ultrasound examination in two weeks will demonstrate an increase in fetal measurements and the rate of growth is normal (the lines joining the measurements are parallel to the appropriate normal mean for gestation).

In normal small fetuses, the mother is usually small (the main determinant of fetal size is maternal size), and the ultrasound findings are similar to pregnancies with wrong dates. However, a repeat scan in two weeks may demonstrate a further deviation from normal in the various fetal measurements.

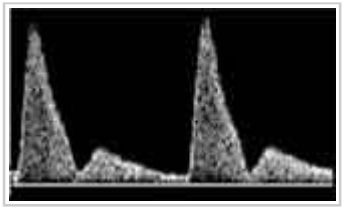
In starving small fetuses, the fetal measurements demonstrate asymmetry (the greatest deficit is observed in the abdominal circumference, then the femur length and finally the head circumference with the transverse cerebellar diameter being the least affected), there are no obvious fetal anatomical defects, the amniotic fluid and fetal movements are reduced, the placenta is often thickened with translucent areas (placental lakes) and there are abnormal Doppler waveforms in the uterine and / or umbilical arteries.

In abnormal small fetuses there may be anatomical defects suggestive of chromosomal abnormalities (in triploidy there may be a molar placenta or in the presence of a normal placenta the fetus demonstrates severe asymmetrical growth retardation, mild ventriculomegaly, micrognathia, cardiac abnormalities, myelomeningocoele, syndactyly, or 'hitch-hiker' toe deformity; trisomy is characterised by strawberry-shaped head, choroid plexus cysts, absent corpus callosum, enlarged cisterna magna, facial cleft, micrognathia, nuchal oedema, heart defects, diaphragmatic hernia, oesophageal atresia, exomphalos, renal defects, myelomeningocoele, growth retardation and shortening of the limbs, radial aplasia, overlapping fingers and talipes or rocker bottom feet). The amniotic fluid may be normal decreased or often increased. In congenital infection growth retardation may be associated with features of hydrops and brain abnormalities (ventriculomegaly, microcephaly or cerebral calcifications).

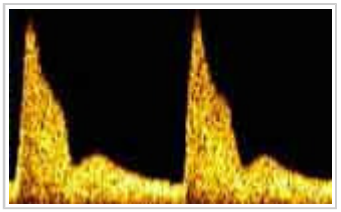
Doppler ultrasound

Doppler ultrasound provides a non-invasive method for the study of fetal haemodynamics. Investigation of the uterine and umbilical arteries provide information on the perfusion of the utero-placental and feto-placental circulations respectively, while Doppler studies of selected fetal organs are valuable in detecting the hemodynamic rearrangements that occur in response to fetal hypoxaemia. In normal pregnancy, impedance to flow in the uterine artery decreases with gestation and this presumably reflects the trophoblastic invasion of the spiral arteries and their conversion into low resistance vessels. Similarly, there is a decrease in impedance to flow in the umbilical arteries due to progressive maturation of the placenta and increase in the number of tertiary stem villi.

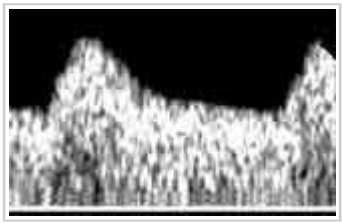
Normal Pregnancy - Development of the uterine artery



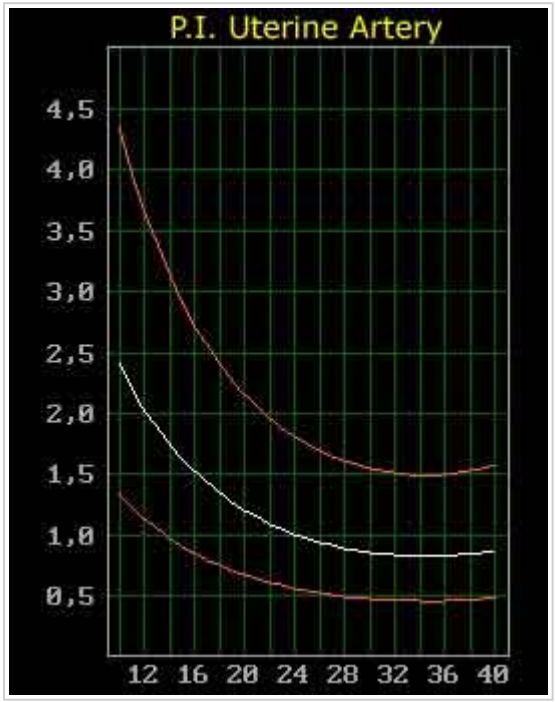
Normal impedance to flow in the uterine arteries in 1º trimester



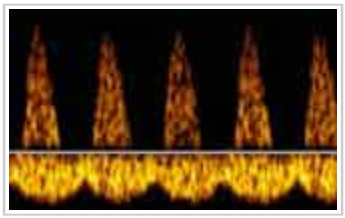
Normal impedance to flow in the uterine arteries in early 2º trimester



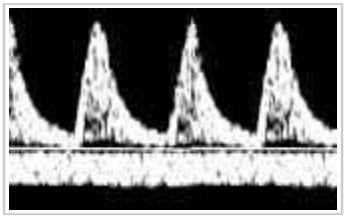
Normal impedance to flow in the uterine arteries in late 2º and 3º trimester



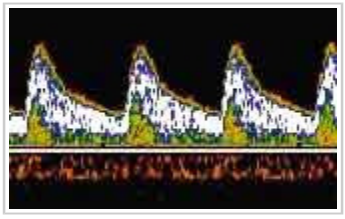
Normal Pregnancy - Development of the umbilical artery



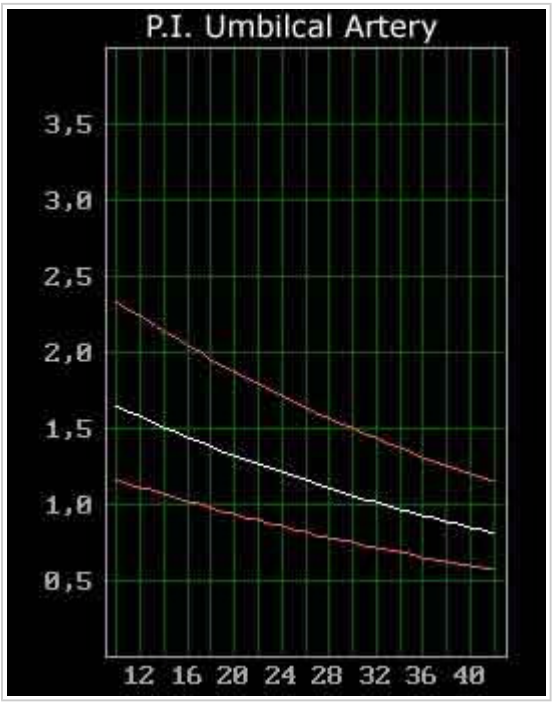
Normal impedance to flow in the umbilical arteries and normal pattern of pulsatility at the umbilical vein in 1º trimester



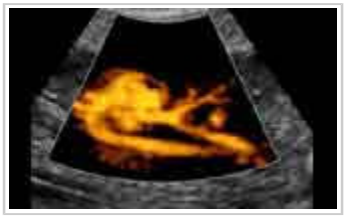
Normal impedance to flow in the umbilical arteries and umbilical vein in early 2º trimester



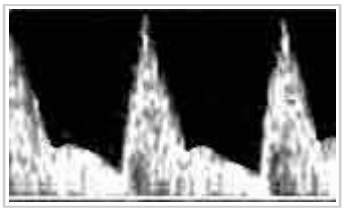
Normal impedance to flow in the umbilical arteries and umbilcal vein in late 2º and 3º trimester



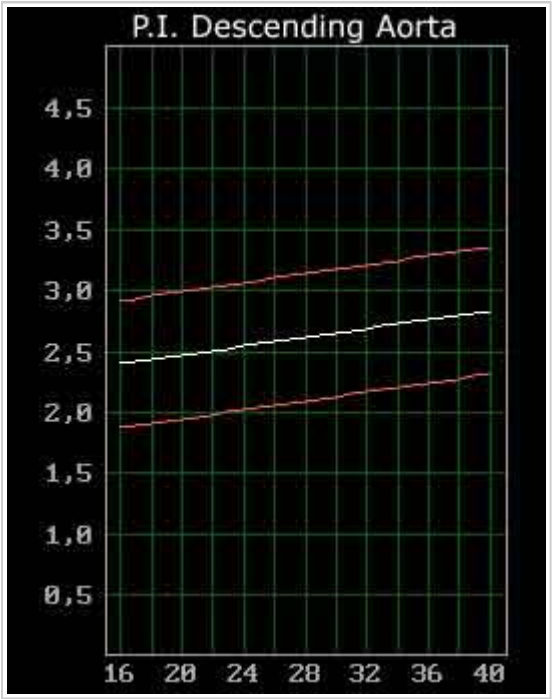
Normal Pregnancy - Development of the Descending Aorta



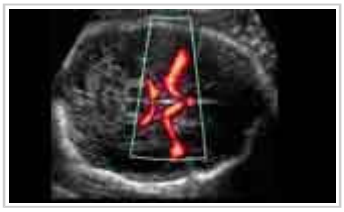
Color Doppler Energy with visualization of the Aortic Arch and descending thoracic aorta



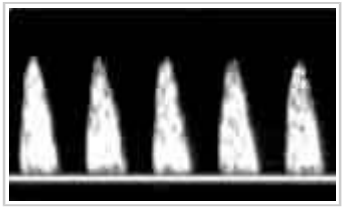
Normal flow of the descending thoracic aorta in 2º and 3º trimesters



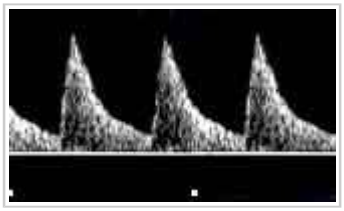
Normal Pregnancy - Development of the Middle Cerebral Artery



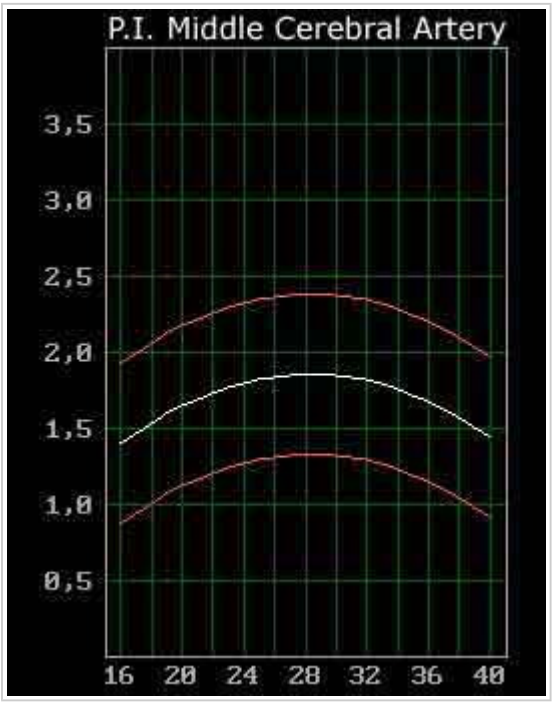
Color Doppler Energy with visualization of the Circle of Willis and the Middle Cerebral Artery

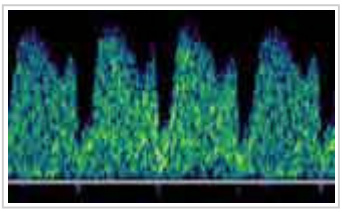


Normal flow of the Middle Cerebral Artery in 1º trimester

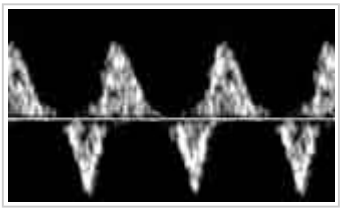


Normal flow of the Middle Cerebral Artery in 2º and 3º trimester





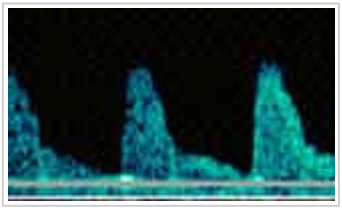
Normal flow of the ductus venosus



Normal flow of the inferior vena cava

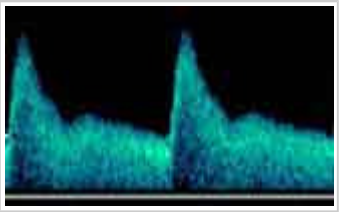
Normal flow of the ventricular valves

Color Doppler Energy with bifurcation of the renal arteries

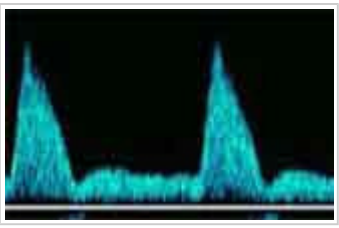


Normal flow of the renal artery

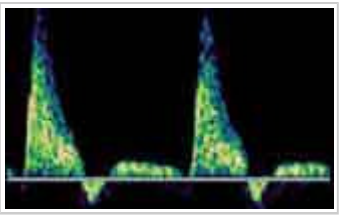
Abnormal Development of the uterine artery



Normal impedance to flow in the uterine arteries (with the characteristic waveform of early diastolic notching)

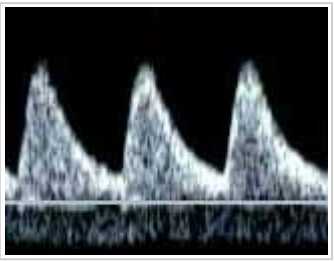


Increased impedance to flow in the uterine arteries (with the characteristic waveform of early diastolic notching)

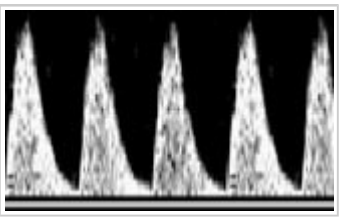


Very high resitance flow in the uterine arteries (with reverese diastolic flow)

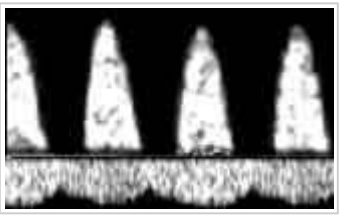
Abnormal Development of the umbilical artery



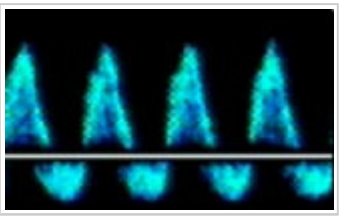
Umbilical arteries
- high pulsatility index



Umbilical arteries
- high pulsatility index



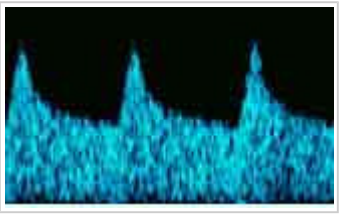
Umbilical arteries
- very high pulsatility index
- end diastolic velocity
- pulsation in the umbilical vein



Umbilical arteries
Severe cases absence of reversal of end diastolic frequencies

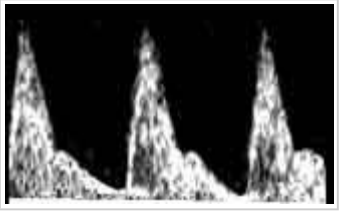
Redistribution of blood flow - Middle Cerebral Artery

Color Doppler Energy with visualization of the Circle of Willis and the Middle Cerebral Artery. Note the vascularization.

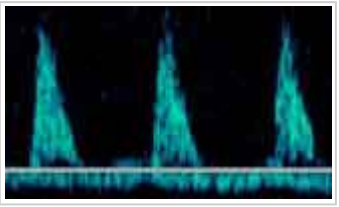


Decrease in impedance to flow in the middle cerebral arteries

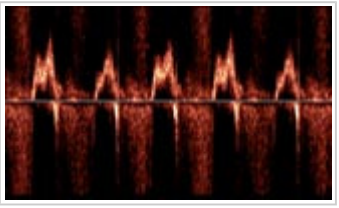
Redistribution of blood flow - Descending Aorta and Renal Artery



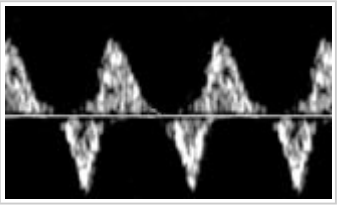
Descending Thoracic Aorta
- descrease the diastolic flow
- increase of the impedance



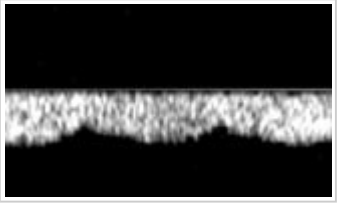
Renal artery
- end diastolic flow
- increase of the impedance



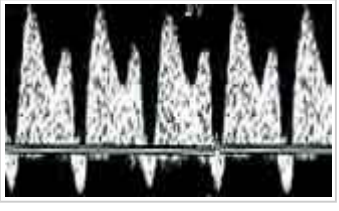
Severe fetal hypoxemia there is decompenation in the cardiovascular system and right heart failure



Peripheral vasoconstriction, as seen in fetal redistribution, causes an increase in ventricular afterload and thus ventricular end diastolic pressure increases. This results in retrograde blood flow in IVC with increase velocity in atrial contraction.



Umbilcal vein pulsations are also observed in severe hypoxia.



In severe fetal hypoxemia there is decompenation in the cardiovascular system and right heart failure. This is manifested by the absence or reversal of forward flow during atrial contraction in the ductus venosus and this is a sign of impending fetal death.

In constitutionally small fetuses Doppler studies of the placental and fetal circulations are normal. Similarly in growth restricted fetuses due to genetic disease the results are often normal. In growth restriction due to placental insufficiency there is increased impedance to flow in the uterine arteries (with the characteristic waveform of early diastolic notching) and umbilical arteries (high pulsatility index and in severe cases absence of reversal of end diastolic frequencies). These data support the findings from histopathologic studies that in this condition there is failure of the normal development of maternal placental arteries into low resistance vessels (and therefore reduced oxygen and nutrient supply to the intervillous space), and reduction in the number of placental terminal capillaries and small muscular arteries in the tertiary stem villi (and therefore impaired maternal-fetal transfer).

Doppler studies of the fetal circulation demonstrate decrease in impedance to flow in the middle cerebral arteries and increase in impedance in the descending thoracic aorta and renal artery. These findings suggest that in fetal hypoxemia there is an increase in the blood supply to the brain and reduction in the perfusion of the kidneys, gastro-intestinal tract and the lower extremities. Although knowledge of the factors governing circulatory readjustments and their mechanism of action is incomplete, it appears that partial pressures of oxygen and carbon dioxide play a role, presumably through their action on chemoreceptors. In severe fetal hypoxemia there is decompenation in the cardiovascular system and right heart failure. This is manifested by the absence or reversal of forward flow during atrial contraction in the ductus venosus and this is a sign of impending fetal death.

Chromosomal defects

Although low birth weight is a common feature of many chromosomal abnormalities, the incidence of chromosomal defects in small for gestational age neonates is less than 1-2%. However, data derived from postnatal studies underestimate the association between chromosomal abnormalitites and growth retardation, since many pregnancies with chromosomally abnormal fetuses result in intrauterine death. Thus in fetuses presenting with growth retardation in the second trimester the incidence of chromosomal abnormalities is 10-20%. The chromosomal abnormalities associated with severe growth restriction are triploidy, trisomy 18 and deletion of the short arm of chromosome 4.

The incidence of chromosomal defects is much higher in (a) fetuses with multiple malformations, than in those with no structural defects, (b) the group with normal or increased amniotic fluid volume, than in those with reduced or absent amniotic fluid, and (c) in the group with normal waveforms from both uterine and umbilical arteries, than in those with abnormal waveforms from either or both vessels. A substantial proportion of the chromosomally abnormal fetuses demonstrate the asymmetry (high head to abdomen circumference ratio), thought to be typical for uteroplacental insufficiency; indeed the most severe form of asymmetrical growth retardation is found in fetuses with triploidy.

Growth restriction can also be caused by confined placental mosaicism. In this condition, which is found in about 1% of pregnancies, the fetal karyotype is normal but there are two different chromosomal complements in the placenta (one is usually normal and the other an autosomal trisomy). Placental mosaicism is also associated with uniparental disomy (inheritance of two homologous chromosomes from one parent), which often results in growth restriction.