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Color Doppler Ultrasonography — A Wide use in Monitoring of Fetal Blood Flow

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Objective: To study the clinical value of monitoring of the fetal umbilical blood flow. Methods: 753 fetal umbilical blood flow velocity wave forms were measured in 682 pregnant women with SRF608 color Doppler ultrasonography, 589 cases were normal and the other 93 abnormal. 46 cases were found with intrauterine growth retardation and 23 cases with pre-eclampsia. The systolic/diastolic ratio(S/D), pulsatility index(PI), resistance index(RI) and fast blood velocity ratio(FBVR) were studied. Results: The wave forms of umbilical artery blood flow in normal pregnancy displayed regularly and equality, though S/D, PI, RI and FBVR changes regularly as the increase of the weeks of pregnancy. Fetal breathing movement may affect the wave forms of umbilical blood flow. The S/D(3.59± 1.17), PI(1.18± 0.29), RI(0.74± 0.18) in the group of IUGR were significantly higher than that of the normal group(S/D 2.68±0.76, PI 0.92± 0.22, RI 0.60± 0.09 respectively) (P<0.01). FBVR in the group of IUGR(0.51±0.06) was less than that of the normal group $(0.57\pm0.05)(P<0.0005)$. The changes of the wave forms in pre-eclampsia is similar to that in IUGR. The wave forms displayed an increase of fetal heart rate and FBVR with a decrease of S/D, PI, RI when fetal distress in the compensatory phase. An increase of S/D, PI, RI and a decrease of FBVR and fetal heart rate may occur when fetal distress in the phase of decompensation. A case of IUGR has an increasingly increase of S/D, PI, RI and the absent end-diastolic and recurrent arrhythmia before the fetal death. Some special abnormal wave forms of umbilical artery blood flow velocity were observed in 3 cases with fetal congenital heart disease. Conclusion: An increase of S/D, PI, RI with a decrease of FBVR may be observed in IUGR and pre-eclampsia. The absent or adverse of the end-diastolic flow is a risk signal of the fetus. Some special abnormal blood wave forms may be the represent of the fetal congenital heart disease.

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[Objective] Chromosomal abnormalities are associated with intrauterine growth retardation (IUGR). We have developed a mouse model for Down syndrome, Ts1Cje, which is trisomic for the segment of mouse chromosome 16 corresponding to human chromosome 21. In addition to Ts1Cje, segmental monosomy 16 (Ms1Cje) mice have been generated. In this study we investigated whether Ts1Cje or Ms1Cje mice would develop IUGR.

[Methods] Ts1Cje, Ms1Cje, and diploid control fetuses were obtained from the cross between the balanced translocation carriers [T(12;16)1Cje] and wild type animals. The number and weight of fetuses were measured and morphological studies were performed on days 13 to 19 of gestation.

[Results] The overall frequencies of Ts1Cje, and Ms1Cje fetuses were almost as same as that of the controls. The weight of Ts1Cje fetuses was significantly lower than that of the controls but higher than that of Ms1Cje at all stages studied. There were no apparent anomalies in either Ts1Cje or Ms1Cje fetuses. [Conclusion] Mice with either segmental trisomy (Ts1Cje) and monosomy (Ms1Cje) for mouse chromosome 16 develop significant IUGR. Monosomy 16 has much more deleterious effects on fetal growth than does trisomy 16. These animal models will be very useful to study the genetic control of fetal growth.