

**193** The Significance of hypoxic stress as stimuli for erythroid production in the fetus. H. Tanaka, S. Nishijima, N. Masaoka, Y. Miyake, H. Sakata, K. Satoh, Dept. Obst. and Gynec., Nihon Univ. Sch. Med., Tokyo,

Percutaneous umbilical blood sampling was performed in 29 subsequently normal and 6 IUGR pregnancies. In normal fetuses, all umbilical venous PO<sub>2</sub> and pH were found to be within the normal range of our previous report. In normal fetuses, Hb and erythropoietin (EPO) increased with gestational age, but PO<sub>2</sub>, erythroblasts and reticulocytes decreased respectively. There was a negative correlation between PO<sub>2</sub> and EPO in the normal group. However, in IUGR, PO<sub>2</sub> in 3 out of 6 cases were below the normal range. All 3 hypoxemic fetuses showed elevated EPO values (26–72mU/ml) compared to the normal and 3 nonhypoxemic IUGR fetuses. When including all normal and IUGR cases, EPO increased significantly as the PO<sub>2</sub> were below 20–25 mmHg, and Hb and EPO showed a positive correlation. These findings suggest that 1) hypoxemia in utero may stimulate EPO production in the fetus as in adults, and this may induce the fetal erythroid production. 2) the presence of O<sub>2</sub> receptor for EPO synthesis in the fetus, and its threshold is in the range of from 20 to 25 mmHg. 3) an elevation of fetal blood EPO values can be used as a marker for chronic hypoxemic states in utero.

**194** The pitfall of FHR monitoring in asymmetrical IUGR. Comparison with fetal blood gases and umbilical arterial blood flow velocity waveforms. S. Shin, H. Ogawa, T. Iwasaki, K. Takeuchi, K. Kijima, H. Katoh, T. Koshino, T. Kawamura, T. Araki. Dept. Obst. Gynec. Nippon Medical School, Tokyo.

Transabdominal umbilical blood sampling under ultrasound guidance was performed on 30 toxemia induced asymmetrical IUGR fetuses just after admission (28 to 34 weeks of gestation) and within one week before delivery. Antepartum fetal heart rate records and umbilical arterial blood flow velocity waveforms were made every day during admission.

The IUGR group of umbilical pO<sub>2</sub> 20mmHg or less (pH=7.29±0.053, pCO<sub>2</sub>=51.8±5.7mmHg, pO<sub>2</sub>=16.6±3.1mmHg), ten out of 16 cases showed reactive NST but high pulsatility index (PI) and low Apgar score. The other group (14 cases) which pO<sub>2</sub> 20mmHg or high, all cases showed reactive NST, normal PI and high Apgar score.

NST did not always point out chronic hypoxxygenation, so we must turn our attention to fetal blood gases.

Fetal blood gases is a most excellent examinations for fetal welfare assessment.

**195** Effect of 24-hour hypoxic hypoxemia on fetal epinephrine (E), norepinephrine (NE) and vasopressin (V) concentrations in goat. H. Sameshima, T. Ikenoue, H. Sakamoto\*, Y. Ijuin. Dept. Obst. Gynec., Kagoshima Municipal Hospital, Kagoshima, \*Div. Vet. Med., Kagoshima Univ.

Effect of 24-hour hypoxemia on fetal plasma concentration of E, NE, and V was studied on 7 chronically instrumented fetal goats at 120–135 days of gestation. Fetal hypoxemia was made by infusing N<sub>2</sub> gas through 3.5 Fr. tube implanted in maternal trachea through tracheotomy. Adequate flow of N<sub>2</sub> gas was determined by a stepwise increase until the fetus showed typical bradycardia in response to acute hypoxia, the level of whose PaO<sub>2</sub> was then kept almost constant for 24 hours. E, NE, and V were measured at control (C), 1 (H1), 6 (H6), 24 (H24) hours of hypoxemia, and recovery (R). Fetal PaO<sub>2</sub> was significantly decreased from 28.5 ± 5.1 mmHg (C) to 19.6 ± 5.2 (H1) and kept relatively constant throughout experiment, while PaCO<sub>2</sub> and pH did not apparently change. E was increased significantly from 21 ± 10 pg/ml (C) to 83 ± 38 (H1) and stayed high during hypoxemia. NE was also elevated from 260 ± 140 pg/ml (C) to 650 ± 380 (H1) and stable during hypoxemia. On the other hand, V showed different pattern from E and NE in that it increased from 1.9 ± 2.5 (C) to 11.6 ± 5.8 (H1) and 8.3 ± 7.8 (H6) with return to 0.9 ± 0.3 (H24) during hypoxemia. We conclude that V is a relatively short-term initial stress hormone in response to prolonged hypoxemia compared with E and NE. (m ± sd)