EFFECT OF SIGETIN ON THE FETAL HEART BEAT FOLLOWING BLOOD LOSS IN THE PREGNANT ANIMAL

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UDC 618.3-06:618.14-005.1-085.361.651-07:618.33:616.12-071.6

A factor of great importance in the mechanism of fetal injury following acute blood loss in the pregnant animal is the disturbance of the circulation in the uterus and placenta [7]. For the prevention and treatment of fetal injury in these circumstances, substances capable of producing a rapid increase in the circulation in the uterus should be used. One such substance is sigetin* – a synthetic water-soluble estrogen.

When injected intraveneously into the mother, sigetin has a beneficial effect on fetuses in a state of asphyxia [1, 4, 6, 7].

The object of the present investigation was to study the effect of sigetin, injected intravenously into the pregnant animal, on the state of the fetuses following acute blood loss.

EXPERIMENTAL METHOD

In chronic experiments the electrocardiogram was recorded simultaneously from a pregnant rabbit and from 2-6 fetuses by the method of unipolar detection of the cardiac potentials. The electrodes were implanted on the 27th day of pregnancy [3].

Blood was taken from a rabbit fixed in the usual position in a special stand on the 29th day of pregnancy. The volume of blood lost was 30% of the total blood volume of the animal and the time taken was 24-30 min.

Sigetin was injected into the auricular vein in a dose of 20 mg in one group of experiments 5 min before bleeding began, and in the other group 5 min after bleeding ended – at the moment of maximal change in the fetal heart rate.

The investigation was carried out on 25 rabbits and 84 fetuses. Sigetin was injected before blood loss into 5 rabbits (21 fetuses investigated), and after the end of bleeding into 10 rabbits (36 fetuses); in 10 control experiments (27 fetuses) no sigetin was injected.

EXPERIMENTAL RESULTS

In the control group of experiments, 16 min after the beginning of bleeding, when 15% of the rabbit's total blood volume had been withdrawn, the fetal heart rate began to slow, reaching a minimum (a mean fall of 60.2 beats/min) when the blood flow amounted to 24% of the blood volume. Somewhat earlier, when 10% of the total blood volume had been lost, the fetal heart rate began to increase. Restoration of the heart rate began 50-60 min after the blood loss but took place very slowly, and the initial level had not been regained 1 h after the end of the blood loss. In similar experiments, an increase in the variations of the heart rate within the minute when it was being counted was also observed 3-6 min from the beginning of the blood loss.

The preliminary injection of sigetin into the pregnant animals modified the reaction of the fetuses to blood loss. They began to develop bradycardia later, not until the blood loss had reached 20-23% ($P < 0.001$), and it was more severe (the heart rate slowed by 109.2 beats/min). The variations in the heart rate within the period of 1 min began to increase somewhat later (when the blood loss was equal to 13% of the total blood volume) and they also were more severe. They amounted to 25.4 beats/min, compared with 15.5 beats/min on the average in the control experiments ($P < 0.001$).

*The dipotassium salt of meso-4,4-diphenylhexane – Publisher's note.

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Variations in the fetal heart rate within the period of 1 min increased after the injection of sigetin given before the blood loss. Before the injection, for instance, these variations amounted on the average to 8.4 beats/min, rising 2-4 min after the injection to 11.6 beats/min ($P < 0.001$). This action of sigetin was similar to that of the natural estrogens, which give rise to rhythmic changes in the circulation in the uterus and placenta.

In the experiments in which sigetin was injected into the pregnant animal 5 min after the end of the blood loss, the fetal heart rate returned to its initial value faster than in the control experiments. By 50 min after the injection the mean fetal rate was 253.3 beats/min (initial heart rate 262.3 beats/min). In the control experiments at this time bradycardia was still present (211.3 beats/min).

In the pregnant rabbits in all the groups of the experiments the fetal heart rate was not substantially changed during blood loss, and its mean value was 260 beats/min; by the end of bleeding the animals often lay down and appeared thirsty, but within a few hours their behavior was indistinguishable from that of healthy animals.

The most effective action of sigetin was observed in experiments in which it was given 5 min after the blood loss. According to V. E. Kogan's findings [2], the indices of the functional state of fetuses in similar experiments (injection of sigetin into pregnant rats immediately after blood loss) showed a marked improvement, as determined by the reaction of the fetuses to hypoxia.

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