Concentrations of thyroid gland hormones in maternal and fetal serum and in amniotic fluid after intraamnial application of thyroxine

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Studies performed on rats, rabbits and lambs have shown that thyroxine stimulates the phospholipid production in the fetal lung. In human beings intraamnial instillation of thyroxine should increase lung maturity and lower the number of cases of membrane syndrome.

The aim of our examinations on 22 pregnant patients with a gestational age of under 35 weeks, was to follow the course of the thyroid gland hormone concentration in amniotic fluid, and in maternal and fetal serum after giving an intraamnial dosage of 500 µg levothyroxine. The hormone determinations were done radioimmunologically and the lecithin was determined encymatically.

Groups were drawn up according to the interval between the instillation of T_4 in the amniotic fluid and fresh amniotic fluid withdrawal, normally when the membranes ruptured at the start of labour, in this way a limit of 5 days was empirically created. In 12 cases the interval was the same or less than 5 days (Group A), in the remaining 10 cases the time interval was more than 5 days (Group B).

Amniotic fluid: The mean value of the T_4 concentration rises in Group A 18 times as much; in Group B the concentration is no different from the original level. The mean value of the rT_3 concentrations after T_4 injections into the amniotic fluid points to a significant increase, about 13 times as much. In Group B no difference can be seen.

Umbilical cord blood: Whilst the T_4 and rT_3 concentrations in Group A and B are significantly different, the T_3 concentrations are similar in both groups.

Maternal serum: The concentrations of T_4, T_3, and rT_3 were measured before, 1/2 an hour after and 24 hours after T_4 injections into the amniotic fluid. No significant changes were to be seen in maternal blood.

The results must be interpreted so that the T_4 injection into the amniotic fluid leads to an increase of T_4 in the fetal blood. This clearly shows that the T_4 injected into the amniotic fluid is resorbed by the fetus. However in the group where the interval between injection and delivery was more than 5 days, we observed that the T_4 level had normalised again.

The thyroid gland hormones resorbed by the fetus will exercise their effect on various fetal organs. As an expression of the cardial effect of the thyroid gland hormones after thyroxine injection we found a moderate fetal tachycardia in 5 cases two days after the injection.

It seems that there is no paraplacental passage of the T_4 injected into the amniotic fluid into the maternal blood.

In addition to the hormone examination we also tested the lecithin concentrations in amniotic fluid before and after T_4 injections; we observed a significant increase of the median values from 3,5 mg/100 ml to 5,2 mg/100 ml after injection. The mean value in Group A was 5,6 mg/100 ml and in Group B 4,3 mg/100 ml. The induction of surfactant production by means of thyroxine probably creates a reversible effect mechanism.

In this connection the membrane syndromes diagnosed by neonatologists are important: Whilst in our group about 8 cases of membrane syndrome were to be expected, this diagnosis had only been made in 2 cases.

It must be said that the risks involved in giving thyroxine with regard to the increase in oxygen output are not certain, whereby this increase does not necessarily have to be the same in all organs. The question of increased oxygen output of T_4 injection would be of particular importance when dealing with fetuses with respiratory placental insufficiency.

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