complications in this study. This risk factor should be value to practitioners counselling women older than 35.

FCO27

ST WAVEFORM OF THE FETAL INTRAPARTUM ELECTROCARDIOGRAM FOR THE DIAGNOSIS AND PREVENTION OF PERINATAL ASPHYXIA

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Intrapartum hypoxia is a recognized cause of fetal morbidity and mortality. However we recognize that the consequences of a severe lack of oxygen will vary from one fetus to another and the capacity of fetuses to handle hypoxia may differ greatly depending also on the situation prior to the actual hypoxic event. It is recognized that cardiotocography does not provide all the information we require to specifically interpret fetal reactions to labour stress. Fetal blood sampling can be used along with CTG monitoring to assess fetal acid-base status during labour and can reduce operative intervention but it requires additional expertise, is time consuming, gives only intermittent information and is therefore not widely used. Fetal pulse oximetry is focused on recording the actual level of fetal hypoxemia. However at present the ability of CTG plus pulse oximetry to provide diagnostic capacity on fetal metabolic acidosis have not yet been demonstrated. Extensive experimental work indicate that analysis of changes in ST waveform provide continuous information on metabolic events occurring within myocardial cells which allow cardiac function to be maintained during hypoxia. Clinical studies have shown that ST analysis of the fetal ECG provide useful information on fetal reaction to labour. Randomized controlled trials have provided conclusive evidence that ST waveform analysis can safely reduce the number of obstetric operative intervention with a parallel improvement in fetal outcome. In a European Commission supported project, involving ten European perinatal centres, the clinical introduction of ST waveform analysis has been accompanied by a specifically developed model of teaching, training and staff accreditation. The results of the project show a significant improvement in fetal outcome with the combined use of CTG and ST waveform analysis. These results show that, through the appropriate use of proven technology and specific models of training and management, a safe reduction in the risk of babies being affected by oxygen deficiency during labour can be achieved with a significant reduction in the need for operative interventions.

FCO28

MATERNAL MORTALITY DUE TO AMNIOTIC EMBOLISM

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The cause of sudden and unexpected death of mother in normal pregnancy, delivery and postnatal period is difficult to estimate. Among the possible causes the leading role belongs to death due to amniotic embolism.

To study pathogenesis and pathomorphological changes of amniotic embolism, had been investigated 9 cases of death due to amniotic embolism and 26 cases of death due to bleeding from uterus. Experimental study was performed on 55 adult healthy female rabbits. The animals had been divided into four groups: I – control group; II – with injection of filtrated amniotic fluid into the auricular marginal vein; III - with injection of not-filtrated but clear amniotic fluid; IV - with injection of not-filtrated and not-clear amniotic fluid.

The study of dissection and experimental material revealed that obstetrical coagulopathic bleedings in majority of cases are the complications of amniotic embolism. The experimental study showed different clinical variants of amniotic embolism: 1. Infusion of filtrated amniotic fluid with mild picture, causes the anaphylactic reaction, discirculatory and coagulopathic changes, and only rarely causes the death. 2. Infusion of not-filtrated but clear amniotic fluid causes embolic discirculatory processes in lungs together with allergic damages and intensive coagulopathic changes - equal to clinical obstetrical chock with col-

lapse and later afibrinogemical bleeding. 3. Embolism with contaminated amniotic fluid always causes the death and resembles to those clinical cases with true diagnosis "Amniotic Embolism", when the cause of death is allergic reaction complicated by embolic discirculatory processes in lungs.

FCO29

OXITOCINONE, INDUCTING FETUS MATURATION

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Objectives: The aim of this study was to present the possibility of making earlier maturation of fetus lungs, by provocating the "stress phenomena".
Study Methods: In the last three months, we started an interesting study of provocating faster production of lecitin L and sphingomieline S in fetus lungs by giving mothers the infusions of sintocinone in the course of 7 days, in low doses, looking after cardiothocography, ultrasound and doppler, and making the analysis of L/S ration after and before the experiment.
Results: We have tested 30 women between 35wg-37wg, with diabetes mellitus gestational in 24 of them and insulin dependent in 3 cases. The L/S ratio was 1,5/1 and they had 0,7-1,1 x10 cells. After 5-7 days of oxitocinone infusions at 6-8 hours intervals in 0,9%NaCl solutions we have checked their ansimes concentration and in 87% (26 cases) it was for planning delivery, L/S=2/1 and we had 1,2 to 2,1 x 10 cells. in 13% the L/S ratio was 1,75/1 and it is nearly enough for delivery.
Conclusions: We wanted to suggest a possible way of speeding fetus lung maturation, using oxitocinone infusions, and initiating stimulus to realise endogenous TRH and T3, by making fluctuations in fetus PO2. This is a pilot idea, but very successful, and needs more experience.

FCO30

DETERMINATION OF FETAL NUCHAL THICKNESS IN 2ND TRIMESTER OF PREGNANCY IN PREGNANT WOMEN RESIDE IN GEORGIA REGION

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Background: In earlier studies the high risk of chromosomal anomalies has been detected in fetuses with Fetal Nuchal Thickness (FNT) more than 6 mm and there are the appreciable peculiarities of FNT specified of local and ethnic difference. The aim of our study was to determine the FNT in Georgia resident pregnant women in 2nd trimester of pregnancy.
Methods: From 1994 to June 2002 the 4750 pregnant women at 15 to 27 weeks of pregnancy were prospectively studied by routine ultrasound and any abnormalities of fetuses were observed.
FNT measurement was obtained by standard ultrasound section in transverse plane of the fetal head. Under procedure of examination the 5th, 50th and 95th percentiles were determined.
Results: The mean index of FNT increased from 15 to 22 weeks of gestation (M ± 2SD):

15 weeks – 3,0 ± 0,27mm	19 weeks – 3,5 ± 0,81 mm
16 weeks – 3,0 ± 0,19 mm	20 weeks - 3,8 ± 0,45 mm
17 weeks – 3,3 ± 0,90 mm	21 weeks – 3,8 ± 0,35 mm
18 weeks – 3,5 ± 1,90 mm	22 weeks – 4,0 ± 0,27 mm

The mean index of FNT from 23 to 27 weeks of gestation varied (M ± 2SD):

23 weeks – 4,0 ± 0,20 mm	26 weeks – 3,9 ± 0,27 mm
24 weeks – 3,8 ± 1,0 mm	27 weeks – 4,0 ± 0,16 mm
25 weeks – 3,9 ± 0,14 mm	

Conclusions: The mean measurement of FNT in our study varied but in all cases was not more then 5 mm in normal fetuses at 15 to 27 weeks of gestation. The obtained FNT mean index can be useful in routine ultrasound screening program to detect the genetic disorders as a selective test before the basic genetic examination.