METHADONE WITHDRAWAL IN NEONATES. Fariborz Rahbar, (Intr. by Melvin E. Jenkins). Howard Univ. Col. of Med., Dept. of Ped., Washington, D. C.

Recently methadone has been used extensively for the treatment of heroin addiction. In adults, methadone produces a withdrawal syndrome similar to heroin. However, until very recently neonatal addiction to methadone was not recognized.

This study analyzes experience with both heroin and methadone withdrawal in newborns at Freedmen's Hospital.

Between September 1, 1971, and August 31 1972, 30 newborn infants born to addicted mothers were studied. Fourteen infants were born to mothers who were heroin users; and 16 newborn infants born to mothers who regularly received methadone.

The incidence of withdrawal in the methadone group was almost equal to the heroin group (75% and 72% respectively).

According to the severity of the symptoms, all infants were divided into 4 groups (asymptomatic, mild, moderate and severe). The incidence of asymptomatic infants was 25% for the methadone group and 28% for the heroin group. The incidence of mild symptoms was much higher in the heroin group compared with methadone group (6% vs. 42%). On the contrary, incidences of moderate and severe symptoms were increased markedly in the methadone group (50% and 18% vs. 14% and 7%).

In the methadone group higher doses of medication as well as combined (paregoric and phenobarbital) therapy were needed to control the symptoms. These findings suggest that methadone does cross the placenta and is addictive to the fetus. Withdrawal symptoms are more severe and more difficult to control than withdrawal symptoms due to heroin addiction.

HYPOXIA IN PRETERM INFANTS: EVIDENCE FOR CENTRAL RESPIRATORY DEPRESSION. Henrique Rigatto, Rafael de la Torre Verduzco, Donald B. Cates (Intr. by Victor Chernick). Univ. of Manitoba, Dept. of Ped., Winnipeg, Canada.

Hypoxia depresses ventilation in preterm infants. Because P_ACO_2 does not rise, the decrease in ventilation has been attributed to a decrease in metabolism rather than to the central depressant effect of hypoxia. To determine whether central depression is present, we measured the ventilatory sensitivity to CO_2 at varying inspired O_2 concentrations in preterm infants. Eight babies (B.W.1 to 2 kg; G.A.<36 weeks) were studied 10 times during the first 10 days of life. After breathing 21% O_2 for 3 minutes, they were given 15,21,40 or 100% O_2 for 4 minutes and then 2% CO_2 plus the various concentrations of O_2 for 4 minutes each. We determined respiratory minute volume and frequency, P_ACO_2, P_AO_2 and heart rate. Ventilation was measured with a nosepiece and screen flowmeter, using a constant flow-through to eliminate dead space and valves. We plotted minute ventilation against P_ACO_2 to assess the respiratory sensitivity at varying inspired O_2 concentrations. The mean slopes of the CO_2 response curves were 0.011, 0.022, 0.039 and 0.168 L/min/kg/mm Hg P_ACO_2 with 15,21,40 and 100% inspired O_2 respectively ($p<0.05$). Therefore, the more hypoxic the infant, the flatter was the response to CO_2. These findings suggest: a) the respiratory center is depressed during hypoxia in preterm infants despite a decrease in P_ACO_2; b) the response to inhaled CO_2 is the opposite to that seen in male adults, where the higher the inspired O_2 concentration, the flatter the response.

IMPACT OF FETAL HEART RATE MONITORING & BLOOD SAMPLING ON INFANT MORTALITY & MORBIDITY - ONGOING STUDY
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Since the introduction of fetal monitoring (heart rate & acid base) 10 years ago, first on a research basis, then as an adjunct to clinical care, the proportion of patients being monitored during labor has increased to 70% (2,000 patients/year). Associated with this there has been a gradual reduction in morbidity as reflected by better Apgar scores and in mortality, for infants weighing 1000 g or more. The proportion of infants with a 1 min. Apgar score of 6 or less prior to monitoring averaged 25%; this has fallen to approximately 12%. There was a comparable improvement in the 5 min. Apgar score. Neonatal death rate has been reduced by almost 40% and perinatal mortality by approximately 10%. Furthermore, of infants requiring intensive care in the neonatal period, those who were monitored during labor were found to require a significantly shorter period in the ICU than those who were not monitored. This difference was even more striking when only infants above 2500 g were considered. Four factors appear to be of major importance in the acceptance and institution of monitoring: an adequate number of monitors maintained in good working order at all times; continuing education of the medical and nursing staff in the use of the equipment and interpretation of records; education of patients; services of a knowledgeable bioengineer and acid-base technician on a regular basis.

LACTIC ACIDEMIA IN SICK NEONATES: Warren N. Rosenfeld, Howard A. Fox, Laszlo Sarkozi, (Intro. by Horace L. Hodes) Mount Sinai Sch. of Med., Depts. of Pediatrics and Chemistry, New York.

163 blood lactic acid (LA) determinations were performed on 42 infants admitted to the Newborn Special Care Unit. Mean values:

Diagnosis	Well	Pneumonia	CHD*	Fetal Distress	RDS*
No. of patients	7	5	7	5	17
Lactic Acid (mg% blood)	19 (9-31)	18 (9-29)	38 (9-111)	86 (23-111)	42 (6-153)

In the RDS group, all patients with LA >50mg% developed intraventricular hemorrhage (IVH). IVH appeared to be more closely correlated to periods of increasing LA rather than decreased $Pa0_2$ or $NaHC0_3$ therapy. No infant with LA>70mg% survived. All with >20mg% required assisted ventilation. Patients with rising LA had a poor prognosis. In cyanotic heart disease, a rise in LA was an early sign of decompensation and proved a valuable parameter for the timing of surgery. In general, increased LA was associated with increased serum osmolality, Na^+ and glucose, but this relationship was not absolute. $Pa0_2$ could not be used as an index of LA. While increases in LA did not occur in some patients with $Pa0_2 \leq 40$mm Hg, others with $Pa0_2 > 50$mm Hg developed severe lactic acidemia. Our data suggest that serial LA's provide both useful prognostic information and a more valid means of determining $Fi0_2$. LA determinations can be performed in 30 minutes.

*CHD = Cyanotic Heart Disease, *RDS = Respiratory Distress Syndrome.

UMBILICAL CORD SIZE AND FETAL MATURATION. C. Joan Richardson, Jeffrey J. Pomerance, and Louis Gluck, Univ. of Cal., San Diego, Sch. of Med., Dept. of Ped. Div. Perinatal Med. La Jolla

Our own random clinical observations suggested that premature infants with respiratory distress syndrome (RDS) have larger umbilical cords (UC) than term infants and prematures without RDS. That UC size might distinguish RDS susceptible prematures from prematures with prenatal stress sufficient to produce pulmonary biochemical maturation (no RDS), was prospectively studied. Over a 3-month period, UC's were examined on all admissions to Univ. Hosp. San Diego, Nursery. Weight, displacement volume, and largest diameter of ends and middle of a 10cm. UC segment were measured. Results are tabulated.

Group	Mean Wt.	Mean Vol.	Mean Diam.	Median Wt.	Median Vol.	Median Diam.
Premature with RDS N = 5	9.3gm.	9.5cc.	1.2cm.	7.6gm.	7.8cc.	1.0cm.
Premature without RDS N = 10	9.8gm.	9.8cc.	1.2cm.	8.4gm.	8.5cc.	1.2cm.
Full Term N = 59	7.4gm.	7.6cc.	1.1cm.	6.9gm.	7.0cc.	1.1cm.

Although premature UC's are larger than term UC's there is no difference in UC size of prematures with and without RDS.

LOWER WEIGHT GAIN AMONG SMOKERS EXPLAINS MOST OF THE EFFECT OF SMOKING ON BIRTHWEIGHT. David Rush (Intr. by L. Stanley James) Division of Epidemiology, Columbia U. Sch. Pub. Hlth., N.Y.C.

There has been no reported systematic exploration of the hypothesis that the low birthweight of infants whose mothers smoke in pregnancy is mediated by depressed caloric intake. Using gestational weight gain as an index of energy balance, we have studied this question among 162 mothers and their liveborn singleton infants, in a poor, black, urban American community. Smoking mothers had lower mean weekly weight gain (.73 vs. .90 lbs/w; $t=2.63$, $p< .01$), and a strong and highly significant gradient of decreasing weight gain with increased amount smoked (.017 fewer lbs/ w. gained per additional cigarette/d; $F=12.45$, $p< .005$). Women who stopped smoking before delivery had higher weight gain than those who continued; the difference was not significant, but numbers were small. Regression analyses were performed in order to quantitate the unique and joint contributions of smoking and change in maternal weight to birthweight. With all analyses, at least half, and usually closer to three quarters of the effect of smoking on birthweight was jointly shared with weight change. Smoking is in all likelihood depressing fetal growth, in large part, by depression of caloric intake, as reflected in lower maternal weight gain.

No other examined differences between smokers and non-smokers accounted for these differences.