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NEONATAL HYPOCALCEMIA ASSOCIATED WITH MATERNAL HYPERCALCEMIA. Burton P. Fine, Barbara A. Glista, and Franklin C. Behrle. New Jersey Medical School, Martland Medical Center, Dept. of Pediatrics, Newark, N.J.

An infant born to a mother with hypercalcemia secondary to metastatic breast cancer developed severe hypocalcemia during the first week of life. Maternal calcium and phosphorous levels ranged from 14-17 mg/dl and 2-2.5 mg/dl, respectively, prior to delivery, and the infant's calcium level was 15.0 mg/dl at birth. During the first 9 days of life the infant's serum Ca level decreased to 4.8 mg/dl and P rose to 8.4 mg/dl. The serum Mg decreased to 0.7 mg/dl. Administration of $MgSO_4$ intramuscularly raised the serum Mg to normal levels, but hypophosphaturia and hypocalcemia persisted. Administration of PTH was followed by return of Ca to normal levels and an apparent increase in P excretion. When PTH was stopped, serum Ca and Mg again decreased, but the phosphaturia persisted. Ca and Mg levels returned to normal spontaneously by the fourth week of life.

The hypocalcemia of this infant supports the hypothesis of transient neonatal hypoparathyroidism resulting from exposure to relatively high levels of maternal Ca in utero and contradicts the theory that it is related to Mg deficiency and end-organ unresponsiveness to PTH.

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HYPOCALCEMIA IN THALASSEMIA MAJOR. Alan R.Fleischman, John F. Rosen, Eva G. Radel, Joseph A. Kochen, (Spon. by Laurence Finberg), Albert Einstein Coll.Med.,Montefiore Hospital and Medical Center, Dept.Ped., The Bronx, NY.

A ten year old patient with thalassemia major who had been maintained on a chronic hypertransfusion regimen for 8 years, presented with a tonic-clonic seizure. Admission laboratory data included a serum level of total calcium of 5.7 mg/dl, ionized calcium 2.08 mg/dl, inorganic phosphorus 7.8 mg/dl, and alkaline phosphatase 1.2 BLU. Although liver function was abnormal with elevated transaminases, serum 25 hydroxyvitamin D level was normal at 21.8 ng/ml. Concentration of immunoreactive parathyroid hormone was relatively decreased at 15 µlEq/ml. Skeletal radiographs were normal. Evaluation of other endocrine, renal and gastrointestinal functions, were normal. Urinary excretion of calcium and cAMP were 43 mg/24 hrs. and 2.75 nmol/mg creatinine/24 hrs. respectively, which are both markedly decreased from normal. All studies indicated hypoparathyroidism presumably due to iron deposition in the gland. 1,25-dihydroxycholecalciferol in an oral dose of 0.5-0.75 µg/d, in this 22 kg child, corrected the hypocalcemia within 7 days of initiating treatment. Serum ionized calcium level has remained normal for 100 treatment days with no evidence of hypercalcemia or hypercalcuria on a dose of 1.0-1.5 µg/d of the hormone.

Four other children maintained on chronic hypertransfusion for thalassemia major had no evidence of hypocalcemia. This form of hypoparathyroidism is recorded to alert the clinician to a significant complication and effective new treatment plan in thalassemia major.

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SERUM 25-HYDROXYVITAMIN D (25OHD) LEVELS IN INFANTS OF DIABETIC MOTHERS. Alan R. Fleischman, John F. Rosen, Gerald Nathenson, Albert Einstein Coll.Med.,Montefiore Hosp. and Med. Ctr., Department of Pediatrics, Bronx, NY.

Thirty-five mothers with previously documented diabetes and their infants born at term were studied. Thirty infants remained normocalcemic (serum ionized calcium > 3.0mg/dl) and 5 infants became hypocalcemic during the first 4 days of life. Serum levels of 25OHD from these diabetic mothers and their infants were compared to values obtained from 30 normal term pregnancies. Serum levels of 25OHD in ng/ml are expressed as mean ± 1SD:

	Normal Preg.	Diabetic Preg.	
		Normocal.	Hypocal.
Maternal	22.2 ± 7.2	13.4 ± 4.0	8.6 ± 3.0
Neonatal-Day 1	20.6 ± 3.4	12.7 ± 2.8	9.3 ± 2.4
Neonatal-Day 14	-	16.1 ± 4.4	11.9 ± 3.1

Serum 25OHD values from normal term mothers and their neonates were greater ($p<0.001$) than values from diabetic mothers and their infants. Serum levels of 25OHD in diabetic mothers and their infants who became hypocalcemic were significantly lower ($p<0.01$) than values from diabetic mothers and their infants who remained normocalcemic. Serum 25OHD level increased ($p<0.05$) in both groups of infants of diabetic mothers from day 1 to day 14 of life with levels in the normocalcemic group remaining greater than the hypocalcemic group ($p<0.05$).

These data indicate that pregnancy in diabetics is associated with a low serum level of 25OHD and suggest that infants born to diabetics with the lowest concentrations of 25OHD are at considerable risk to develop early neonatal hypocalcemia.

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THE USE OF ORAL 25-HYDROXYCHOLECALCIFEROL ($25OHD_3$) IN PREMATURE NEONATES. **Alan R. Fleischman, John F. Rosen, Gerald Nathenson, Albert Einstein College of Medicine, Montefiore Hospital & Med. Cent., Dept. of Ped., The Bronx, NY**

Oral 25-hydroxycholecalciferol ($25OHD_3$) was used in this study to assess its effectiveness in preventing early neonatal hypocalcemia. Thirty-one premature neonates were studied prospectively from birth. Twenty-two prematures constituted a control group; 9 prematures were treated orally with $25OHD_3$ (2 µg/kg/day) for 5 days beginning within the first 12 hours of life. 11 of the 22 prematures in the control group (50%) became hypocalcemic with serum ionized calcium level (Ca^{++}) less than 3.0 mg/dl and total calcium level (Ca_T) less than 7.0 mg/dl on day 2 of life. Only 1 of the 9 prematures treated with $25OHD_3$ became hypocalcemic. Serum 25-hydroxyvitamin D as well as Ca^{++} and Ca_T levels increased during the first 5 days of life in treated neonates and were significantly greater than levels in control neonates. No untoward effects of this treatment were observed.

These data indicate that vitamin D metabolism plays an important role in perinatal calcium homeostasis and that oral $25OHD_3$ is effective in preventing early neonatal hypocalcemia.

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ESTIMATION OF LEAN BODY MASS FROM ARM CIRCUMFERENCE AND SKINFOLD THICKNESS. Gilbert B. Forbes, University of Rochester Medical Center, Department of Pediatrics, Rochester, NY.

Assays for lean body mass (LBM) by ^{40}K, body water, or densitometry require specialized techniques. Simpler methods for estimating this metabolically active component of the body are needed. We assayed LBM by ^{40}K counting in 251 males and 206 females 8-60 years old, and calculated the circumference of the muscle-bone component (M+B) of the arm from measurements of arm circumference and the triceps and biceps skinfold thickness. The correlation coefficients are 0.959 for males and 0.800 for females, and the respective standard errors of estimate are 5.2 and 4.8 kg LBM.

LBM is also related to stature (Forbes, Am. J. Clin. Nutr. 27,1073). A multiple regression analysis of LBM on stature and arm M+B circumference reduced the s.e. of estimate to 4.0 kg for males and 3.4 kg for females, and yielded correlation coefficients of 0.975 and 0.906, respectively.

Hence these simple measurements can yield a reasonable estimate of LBM.

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BONE MINERAL TURNOVER IN OSTEOGENESIS IMPERFECTA. Gilbert B. Forbes, Frank A. Smith, Donald Taves, Robert W. Kilpper, University of Rochester Medical Center, Departments of Pediatrics and Radiation Biology and Biophysics, Rochester, NY.

A young boy with the severe form of osteogenesis imperfecta was treated with NaF for 8 years (1 mg F/kg/d). The frequency of fractures decreased somewhat, but we could detect no gross improvement in bone density. Based on F balance studies and analysis of a bone biopsy, his body F burden was estimated to be about 5 gm at the end of the treatment period (age 14 yr).

NaF treatment was abruptly stopped, and during the next 4 1/2 years a number of 24hr urine samples were analyzed for F. After the first few days, during which only 28 mg F were excreted, fluoride excretion declined exponentially: 10% of the body F burden was lost with a half-time of 5 months, 90% with one of 9 years. This latter value is within the range observed by others for the elimination of F and other "bone-seekers" (Sr, Pb, Ra) in normal subjects.

Since approximately 99% of body F is located in the mineral phase of bone this element can serve as a tracer for bone mineral. Based on the extended observations on our patient, it would appear that bone mineral turnover is normal in this disease.