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Ed. Note - This overview was originally presented at the International Symposium on Clinical Disorders of Bone and Mineral Metabolism, May 9-13, 1983. The following list indicates the presentations given in this session at the Symposium and the contents of the corresponding chapter in the Proceedings of the Symposium published by Excerpta Medica. The numbers in parentheses refer to pages in this volume. Complete information about the contents of the Proceedings can be found at the back of this issue.

The general focus of this session was to highlight the time of life when development, rapid growth, and skeletal modeling occur. Interest in mineral homeostasis during this period of development has grown as exponentially as the rate of bone growth itself, but time constraints permitted only a representative sampling of work in the field. Organ development and growth are intimately linked with the ontogenesis of hormone biosynthesis, transport and response machinery. Since neonatologists are now able to save very premature infants, a whole new group of infants must make the transition from the placental guarantee of minerals in utero to the postnatal uncertainty of diets, infusions, and environments.

The placental transfer of calcium to the fetus provides a 20-30 g calcium content to the full-term infant. Calcium, magnesium and inorganic phosphorus concentrations are higher in fetal than in maternal blood in many species, including humans. Although a close correlation between maternal and neonatal vitamin D "nutrition" has been observed by many investigators, no evidence for placental crossover of parathyroid hormone has been presented (1). Depending on a variety of factors in utero and postnatally, the infant's adjustment to an independent mineral and skeletal homeostasis can be smooth and fairly rapid, or it can be troubled and protracted (2,3).

Anast (pp. 422 ff.) provided a concise review of recent studies dealing with normal and abnormal neonatal mineral homeostasis. Early and late neonatal hypocalcemia appear to be attributable to different mechanisms, since parathyroid responses differ, but at present, clear understanding of their pathogenesis is lacking. Neonatal hypercalcemic syndromes are described as well as recent experience with an infant whose study revealed features of both primary hyperparathyroidism and familial hypocalciuric hypercalcemia.

Both Anast and Marx and colleagues (pp. 431 ff.) addressed the issue of hypercalcemia in infancy and the importance of extending observations to renal handling of calcium and studies of family members. Although the basic defect(s) are not characterized in familial hypocalciuric hypercalcemia, clinical studies now available instruct us to be vigilant about this diagnosis.

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Hillman (pp. 427 ff.) discussed some important areas to be considered in assessing the difficulties of mineral and skeletal homeostasis during the neonatal period. The availability of minerals and sterols must be taken into account, and the issues of their appropriateness for infants represents a great challenge to investigators. The recent application of minerally-enriched formulae to the diet of premature infants appears to be an improvement over past practice. Optimal vitamin D supplementation awaits definition in small premature infants, and the frequency of multiple problems contributing to their osteopenia is worth our notice.

Although firmly entrenched in the literature, the concept of vitamin-D resistance is currently being reevaluated with newly available methodology. Glorieux (pp. 438 ff.) and Liberman et al (pp. 441 ff.) discussed new findings and therapeutic approaches in this area. With the recently available techniques in assay of hormones, receptors, hormone effects and histomorphometry, a more rational classification of apparent vitamin D resistance seems likely. Conversely, improved understanding of human diseases of vitamin D metabolism and action should provide clues to, and probes for, unraveling the mechanisms of vitamin D bioactivity.

References

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