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**Types of Disturbances of Mineral Metabolism Associated
With Bone Dystrophies.**

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In addition to rickets arising from diets deficient in vitamin D or salts of calcium or phosphorus, bone dystrophy has been noted in various disturbances of mineral metabolism which lead to imperfect deposition of the mineral constituents of bone, or to their excessive mobilization. Because of the similarity of clinical picture and the mechanics of production, the term rickets may justifiably be applied to the whole of this diverse group of bone dystrophies. Those cases arising from deficient absorption of inorganic bone constituents should be considered of exogenous origin, whether the deficient absorption be due to inadequate intake of minerals or of vitamin D, to inadequate ultraviolet irradiation, or to other cause. Those instances in which the metabolic disturbance is not dependent upon inadequate absorption are secondary to some endogenous disturbance of metabolism.

Several patients with endogenous rickets have been studied intensively in this clinic, and while their bone lesions were very similar, the metabolic data indicate that their anomalies were not dependent upon a common metabolic defect. The nature of the metabolic data indicated that in one group of patients the condition was secondary to chronic acidosis, due in one instance to persistent ketosis, and in another to constant reabsorption of urine. In a second group the bone changes were dependent upon hyperparathyroidism. The remaining group must be termed idiopathic, until further studies serve to establish the etiology of their condition. This third group is very similar in some respects to infantile rickets of the usual, or exogenous, type; it is distinguished from it in its failure to respond satisfactorily to adequate calcium and phosphorus ingestion, to vitamin D in the form of cod liver oil or viosterol, or to ultraviolet irradiation.

The accompanying table presents a correlation of the nature of the changes in the metabolism of phosphorus and calcium observed in this clinic, together with data collected from the literature.

TABLE I.—*Bone Dystrophies of Metabolic Origin.*

Classification	Remarks	Metabolic Manifestations					
		Serum*		Urine†		Feces†	
		Calcium	Phosphorus	Calcium	Phosphorus	Calcium	Phosphorus
I. Exogenous Infantile rickets Starvation osteomalacia ¹	Due to deficiency of Ca, P, vitamin D, or ultraviolet light	10-12 mg. 5-7 mg.	1-4 mg. 1-3 mg.	low low or normal	normal low or normal	high high	high high
II. Endogenous a. Endocrine dysfunction Hyperparathyroidism ² Hyperthyroidism ³	Parathyroid adenoma; diffuse rarefaction, cyst formation Exophthalmic goiter, toxic adenoma; diffuse rarefaction	10-18 mg. 9-12 mg.	1-3.5 mg. 3-4 mg.	high high	high high	normal or high normal or high	normal or high normal or high
b. Chronic Acidosis Renal Insufficiency ⁴	“Renal rickets”; deficient calcification, diffuse rarefaction Base deficit acidosis; deficient calcification	5-9 mg.	6-17 mg.	low	low	normal or high	high
Ureteral Transplantations ⁵ Chronic Ketosis ⁶	Atypical diabetes; deficient calcification and diffuse rarefaction Similar to “exogenous” group, but do not respond to adequate diet and ultraviolet light	10-12 mg. 8.6-11 mg.	2-4 mg. 1.5-4 mg.	low ‡	low ‡	‡ ‡	‡ ‡
c. Idiopathic ⁷		10-12 mg.	1-4 mg.	low	normal or high	high	high

* These values do not necessarily cover absolute range of variation, but are quoted from literature or authors' data.

† Absolute values would be dependent upon intake, and so are not quoted.

‡ Urine-feces partition impossible.

¹ Miles, L. M., and Peng, C. T., *J. Exp. Med.*, 1925, xli, 137.

² Boyd, J. D., Milgram, J. E., and Stearns, G., *J. Am. Med. Assn.*, 1929, xciii, 684. Gold, E., *Mittel. a. d. Grenzgeb. d. Med. u. Chir.*, 1928, xli, 63. Barr, D. P., Bulger, H. A., and Dixon, H. H., *J. Am. Med. Assn.*, 1929, xcii, 951. Wälder, R. M., *Endocrinol.*, 1929, xiii, 231.

³ Aub, J. C., Bauer, W., Heath, C., and Ropes, M., *J. Clin. Invest.*, 1929, vii, 97.

⁴ Schoenthal, L., and Burpee, C., *Am. J. Dis. Child.*, (in press). ⁶ Authors' data, unpublished.

⁵ Boyd, J. D., *Proc. Soc. Exp. Biol. and Med.*, 1928, xxvi, 181. ⁷ Authors' data, unpublished.