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ABSTRACT: Compartmental models for the various aspects of human iodine metabolism are reviewed, emphasizing the role of Mones Berman in the development of this field. The review first presents published submodels for the peripheral distribution of inorganic iodine, for the thyroidal iodide trapping function, and for the peripheral distribution and metabolism of the thyroid hormones. Approaches to improving understanding of the physiology of the thyroid gland itself through compartmental modeling techniques are then discussed in more detail. The three submodels described above are incorporated into overall models of thyroid iodine metabolism after being simplified to various degrees. Previously published models for thyroidgland radioiodine metabolism, as well as current work in progress, are illustrated by attempting to fit the models to data from a single (previously unpublished) detailed prolonged feeding experiment in a normal human subject. Published thyroid gland models reviewed include: (1) the usual presentation, where the thyroid is a single homogeneous iodine compartment; (2) the model of DeGroot and colleagues, where thyroidal iodine is presented as MIT, DIT, 13. and T4, each with an active and linked storage compartment; (3) the thyroid model developed by Berman and colleagues, with less chemical subcategorization but incorporating a delay compartment, in which a fraction of the iodinated material in the thyroid is partially or completely inaccessible to secretion during the delay; and the later updating cf Berman' model to include a thyroidal iodide recirculation 0001.

The experimental data presented fits most of these models for the first 1-2 weeks, but the fit could not be extended to longer data collection times. To overcome this shortcoming, a new thyroid gland model is introduced. It is based on the latest Berman model but describes thyroglobulin metabolism as incorporating multiple delay compartments of various time periods. The overall fit of the long term data is better with this model construct than with any of the published models. It appears that a complex thyroidal substructure, such as that of the multidelay model under development, will be required to account for overall thyroid iodine metabolism.