

Using Age-Specific Upper Limits for Normal TSH Slightly Reduces the Incidence of Subclinical Hypothyroidism in the Elderly

Kahapola-Arachchige KM, et al.

ANALYSIS AND COMMENTARY ● ● ● ● ●

A recent reanalysis of the NHANES III data, using age-, sex- and ethnicity-specific TSH reference limits, eliminated 144 of 13,344 subjects in the “Reference Population” because they had a TSH >10 or <0.1 mU/L, leaving 4671 whites for analysis (2). The 97.5th percentile for TSH values in whites 70 to 79 years old was 5.6 mU/L, and for those over age 80 it was 6.6 mU/L. The reference population was known not to be overtly hyperthyroid or hypothyroid, was not taking medications known to affect thyroid-function tests, and was specifically known to have no detectable antithyroid antibodies, and thus is closest to the Western Australian population studied, with the exception that anyone who had had antithyroid antibodies ordered was excluded from the Australian study. In the 14,347 Australian subjects who were 70 to 79 years old, the 97.5th percentile for TSH was 4.5 mU/L; for the 8417 subjects over age 80, it was 5.0 mU/L. Thus, the increase in TSH with increasing age in Australia was not as great as that found in the reanalysis of the NHANES III data. One major difference between the two studies is the much greater sample size in the Australian study, but the differences between the TSH assays used, in geography, and in ethnic backgrounds also need consideration.

Until recently, manufacturers of TSH assays indicated that values between 0.4 to 4.0 mU/L were normal, although it is now clear that the TSH level

in a healthy individual does not vary by that much. Some of the genes that contribute to the TSH set point have been identified, but as an individual ages, there could be changes in 1) TSH bioactivity, 2) thyroidal TSH responsiveness, 3) factors regulating thyroid hormone uptake and metabolism, 4) thyroid hormone receptors, and/or 5) cofactors that modulate the T₃-responsiveness of a given gene in a given tissue. A recent study of patients over 65 years of age who were able to function normally in a community and were not taking thyroid hormone indicates that subclinical hypothyroidism commonly persists for at least 4 years, but if a patient’s TSH is below 7 mU/L (as determined by Elecsys 2010 analyzer, Roche) and the anti-TPO titer is normal, the TSH is more likely to normalize within 2 years (3). Undoubtedly, administering L-T₄ to elderly patients with mild subclinical hypothyroidism can alter physiological and biochemical parameters. (For example, Dr. Mestman discusses such a report in this issue of Clinical Thyroidology on patients under age 75 with subclinical hypothyroidism plus chronic kidney disease, in which giving L-T₄ reduced the rate of decline in renal function [4]). In general, replacement of T₄ in elderly patients with subclinical hypothyroidism should be gradual and monitored closely, to avoid overreplacement. Evidence that such therapy improves mortality remains meager, however, particularly in those over age 65 (5).

— Stephen W. Spaulding, MD

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
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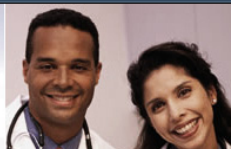
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