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by Irwin B. Levitan and Leonard K. Kaczmarek, 603 pp, ill, New York, NY, Oxford University Press, 2002, \$59.95

**Neuroplasticity, Development, and Steroid Hormone Action**

edited by Robert J. Handa, et al., 386 pp, ill, New York, NY, CRC Press, 2002

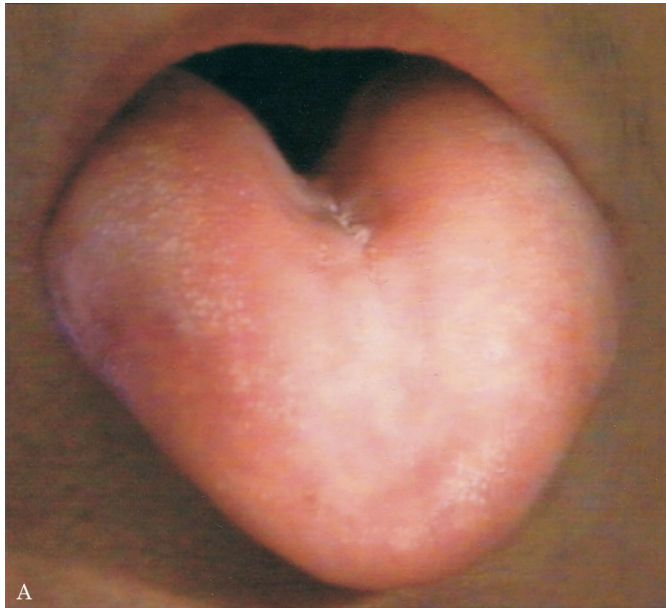
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by Kim J. Burchiel, 1,008 pp, ill, New York, NY, Thieme Medical Publishers, 2002, \$249

**Neuro Images**



*Figure. (A) A 61-year-old woman had an enlarged tongue. At age 58 she had restrictive cardiomyopathy and 2 years later proximal muscular weakness developed. In addition, EMG demonstrated a prominent motor axonal neuropathy. A monoclonal serum protein was identified leading to a diagnosis of amyloidosis, which was confirmed by endomyocardial biopsy. (B) A 53-year-old woman reported paresthesiae and dysesthesia in the feet and showed a severely atrophic tongue with continuous fasciculations. At age 37 she presented with loss of vision caused by amyloid infiltration of the vitreous body. Neurologic examination showed a mild sensory neuropathy and EMG confirmed axonal polyneuropathy. Molecular studies revealed the Pro36 TTR mutation.*

**Tongue involvement in amyloidoses**

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Light chain amyloidosis and transthyretin (TTR) amyloidosis both have peripheral neuropathy as a prominent neurologic feature.<sup>1</sup> We report two patients with amyloidotic

peripheral neuropathy and remarkably different abnormal appearance of the tongue. One showed macroglossia (figure, A) secondary to deposition of amyloid between the muscle fibers,<sup>2</sup> whereas the other showed tongue atrophy (figure, B) presumably caused by TTR amyloid deposit in the hypoglossal nerves.<sup>1</sup>

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