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small amounts of xenobiotics applied as part of a therapeutic program^{279,280} may also be produced by this same process.⁸⁴

Cholinergic hyperresponsiveness has been noted in some cases of asthma and chronic obstructive lung disease, vasculitis, arrhythmia, and vascular spasm, which would be a type of denervation neural sensitization response. Hyperresponsiveness of the autonomic nerve may be congenital or acquired but fit with Gunn's³¹⁴ model of denervation hypersensitivity, which would render the entire bronchopulmonary organ hypersensitive after an injury. The whole bronchial tree appears to become supersensitive after the initial damage occurs, rendering the individual to be more sensitive to inhaled irritants, foods, and chemicals. At times, only one branch of the nerve is injured because

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inappropriately because of environmental overload. Additionally, the plexi can be augmented. Then they function abnormally, yielding alterations in the dynamics of homeostasis.

A denervation hypersensitivity (Chapter 2 of *Reversibility of Chronic Degenerative Disease and Hypersensitivity: Regulating Mechanisms of Chemical Sensitivity*) condition either by newly sprouted out nerve growth (regeneration of neurons) or damaged nerves occurs with solvent or traumatic injury. As described by Gunn,²⁷⁹ the whole organ supplied by the damaged peripheral sensory and dorsal root nerve (esophagus, stomach, small, or large intestine) and its autonomic nerve can become super responsive to autonomic discharges and the effects of neurotransmitters like norepinephrine or acetylcholine. In addition to autonomic discharge, the vanilloid peptidergics that