The lingual nerve is at risk of injury during common dental procedures because of its proximity to the inferior alveolar nerve and the roots of the third molar tooth. Lingual nerve injury has been documented following extraction of wisdom teeth,1-3 dental anesthetic injections,4 and other endodontic procedures.5 We present a case of loss of somatosensory function and taste associated with the intriguing finding of fungiform papillae atrophy as a result of lingual nerve injury.

A 25 year old man with no previous medical history was evaluated in a general neurology clinic for numbness and loss of taste involving the right side of his tongue of two years duration. This began at the time of a difficult extraction of a right lower wisdom tooth with sudden, severe pain. Immediately thereafter, sensation and taste on the right half of the tongue were lost. This began to improve slowly about six months after the injury. However he still had altered sensation and taste when he was seen at our clinic two years later. Neurological examination revealed decreased sensation to pinprick over the anterior two thirds of the right side of the tongue and on the inner gingival mucosa of the right lower teeth. He was not able to identify salt or sugar applied to the anterior two thirds of the right side of the tongue. The number of fungiform papillae on the right side of the tongue was visibly reduced (Figure). The remaining examination of cranial nerves V and VII were normal. No other neurological deficits were found on exam.

The patient returned for follow up one year later (three years after injury) and reported some improvement in somatosensory function; unlike before, he could now appreciate whenever he bit the right side of the tongue. Hot and cold foods were detected more readily on that side. However, there was negligible change in taste function. Examination did not reveal any significant increase in the number of fungiform papillae oninspection. Sensation to pinprick was still reduced, but not as severe compared to the previous examination a year earlier. There was no recovery of taste function on the right side when tested again with salt and sugar.

The lingual nerve carries afferent somatosensory fibers from the anterior two thirds of the tongue, sublingual mucosa, and the lingual aspect of the lower gums. It also initially carries taste fibers from the anterior two thirds of the tongue which later enter the chorda tympani nerve. Therefore, lesions of the lingual nerve distal to its junction with the chorda tympani often result in altered sensation and loss of taste over the areas that they innervate. Our patient also had the interesting finding of atrophy...
of fungiform papillae. Fungiform papillae are localized predominantly to the anterior two thirds of the tongue, and they contain taste buds at their apices with a taste pore at the top. Fungiform papillae have dual innervation: the lingual nerve innervates the connective tissue and epithelium of surrounding taste buds and transmits somatosensory information; the chorda tympani innervation is confined to the taste buds and carries gustatory sensory information.

Zuniga et al. studied patients with partial to complete transection of the lingual nerve. They found that the density of fungiform papillae, as well as the ratio of pores per papillae were significantly reduced after lingual nerve transection. After their patients underwent lingual nerve repair by stump-to-stump coaptation, there was an increase in the number and density of fungiform papillae, taste pores, the number of pores per papillae, and some clinical recovery of taste.

In an elegant animal experiment, the chorda tympani nerve alone or chorda tympani – lingual nerve combined were transected followed by examination of the tongue at different time points. There were a portion of papillae with no taste buds in both groups, but the doubly denervated group had a significantly higher percentage of these papillae. This suggests that the lingual nerve formed collaterals to maintain neural support for some taste buds in the singly denervated group. Alternatively, the chorda tympani nerve might play a role in maintaining epithelial and connective tissue integrity. The maintenance of papillary structure is probably dependent on various trophic factors secreted by the lingual and chorda tympani nerves. A few candidate factors have been identified in animal models including calcitonin gene-related peptide (CGRP), neurokinin A (NKA), neural cell adhesion molecule (NCAM) and neuron specific enolase (NSE).

The persistence of papillary atrophy appears to be an indicator of the extent of lingual nerve injury and can be prognostic of functional recovery. When sensory function of the tongue did not recover more than six months after lingual nerve injury, there was also a noticeable loss of fungiform papillae on that side. Similarly, transient atrophy with return to normal morphology can coincide with the return of normal sensation and initial paresthesias in the absence of atrophy usually heralds a complete recovery.

This report serves to bring up an interesting clinical manifestation of a peripheral nerve injury that may be seen in general neurologic practice, namely loss of taste, somatosensory function, and visible atrophy of papillae on the tongue. Studies are warranted to better understand the pathophysiological mechanisms involved with an aim of developing viable therapeutic options. In particular, the therapeutic potential of human-specific gustatory trophic factors, perhaps in conjunction with surgical nerve repair, should be explored.

REFERENCES