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In Reply: Drs Aspray and Unwin raise questions about the difficulty in extrapolating evidence from younger to older patients. It is important to consider more broadly that clinicians must make a calculated guess when translating the average effect of an intervention measured for a population studied in a clinical trial to an individual patient in a practice, irrespective of the age difference.¹

Nevertheless, I agree that this problem is especially true for the older adult population that is particularly heterogeneous with respect to comorbidity, frailty, and disability. This "calculated guess" or judgment remains a poorly understood cognitive process. Clinical trials must include older patients who are representative of those seen in practices, and tools must be developed for assessing where individuals fit in the clinical spectrum to more accurately individualize medical advice.

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RESEARCH LETTER

Atypical Trigeminal Neuralgia Associated With Tongue Piercing

To the Editor: Tongue piercing is an increasingly common form of self-expression. Reported important complications of tongue piercing include endocarditis,¹ tetanus,² and brain abscess.³ We describe a patient with trigeminal neuralgia that developed after a tongue piercing and that resolved shortly after its removal.

Report of a Case. An 18-year-old woman presented with a 2-month history of neuropathic facial pain described as severe, constant, and paroxysmal. This had started 1 month after a piercing of her tongue and insertion of a bispherical metal stud. A typical episode began with right-sided paroxysmal pain in the maxillary (V2) and mandibular (V3) region, followed by hypoesthesia 30 seconds later. The episodes were described as "electrical shocks," lasted from 10

to 30 seconds, and recurred 20 to 30 times each day, increasing in frequency and severity during the latter weeks. Episodes rarely occurred when she was chewing or talking and did not awaken her at night.

On examination, she had mild hypoesthesia of the skin over the right maxilla and mandible, as well as dysarthria in an attempt to guard against the pain trigger. Touching the skin in a 2-cm × 2-cm area lateral to the right nostril evoked pain in the right V2 and V3 distributions, without glossalgia. No other trigger point was found. Oral examination revealed a bispherical metal stud in the anterior third of the tongue (FIGURE, A). The area surrounding the stud appeared normal. Taste and swallowing were normal. Her neurologic examination was otherwise normal, as was the dental examination and remainder of the physical examination.

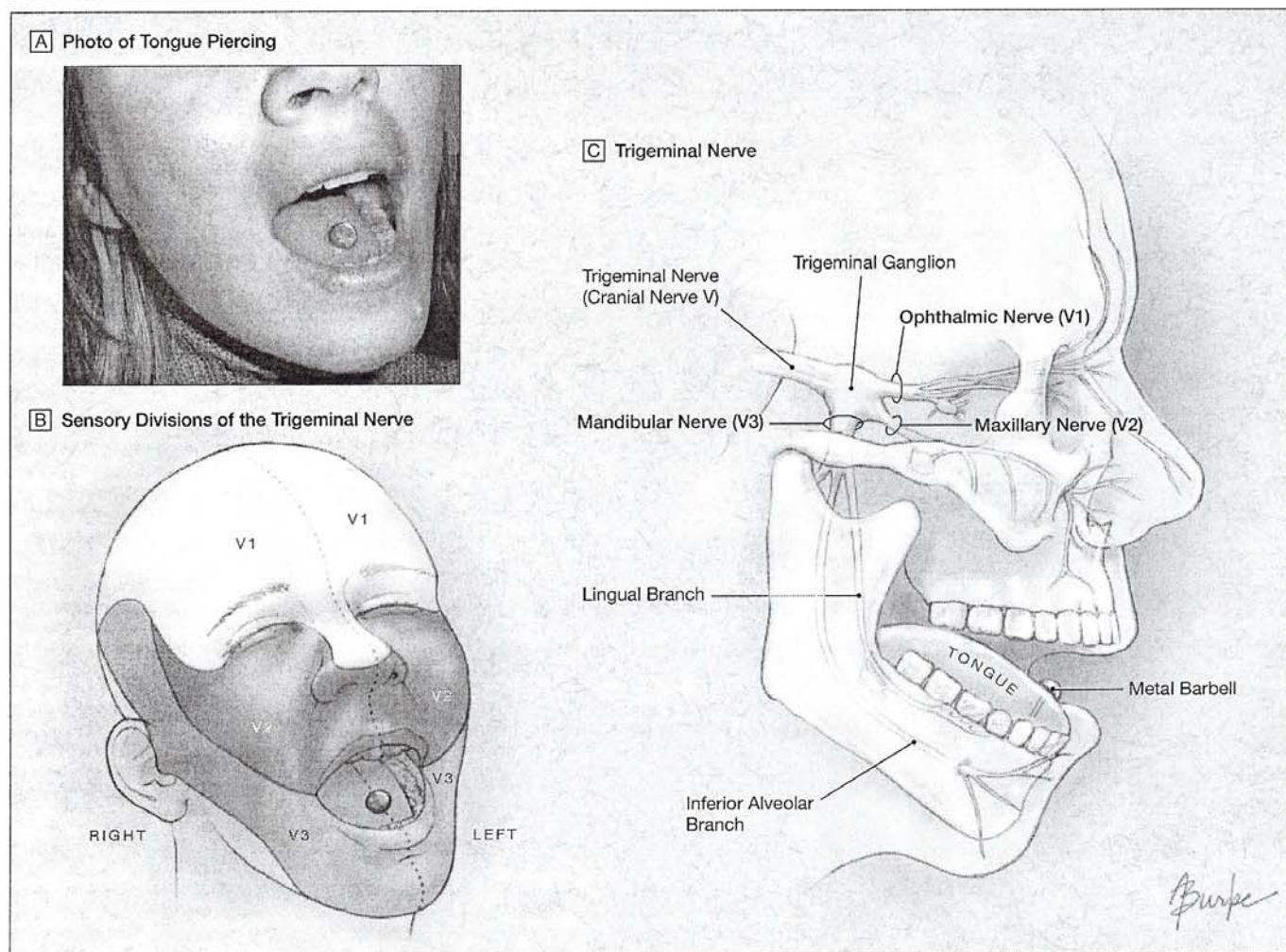
She had a normal C-reactive protein level and erythrocyte sedimentation rate. A cerebral angiogram and magnetic resonance imaging scan performed 2 weeks following the initial examination were unremarkable (the stud was removed for the duration of the scan). Treatment with acetaminophen, codeine, and dexamethasone did not improve the symptoms. The diagnosis of trigeminal neuralgia was considered, and she then started carbamazepine, 200 mg twice daily. The severity of the pain did not change, but it occurred less frequently. After 1 week of carbamazepine, the trigeminal neuralgia recurred with previous characteristics despite a therapeutic drug level; carbamazepine was discontinued after 2 weeks. The stud was removed that day, and 48 hours later her symptoms resolved completely. One year later, she continues to be free of all symptoms, with normal findings on physical examination.

Comment. Piercing of the tongue disrupts the skin, mucous, and muscle barriers and may produce local infections secondary to the introduction of buccal microflora into the muscles. Because the intrinsic muscles of the tongue possess a rich network of vessels, there is a secondary risk of sepsis¹ or formation of a distant abscess.³

Cranial nerve involvement with right facial weakness has been reported secondary to cephalic tetanus after tongue piercing.² The lingual branch of the mandibular division of the trigeminal nerve provides general sensation to the anterior two thirds of the tongue (Figure, B) and, as in the patient we describe, constitutes the afferent limb for the spread of sensory impulses, resulting in secondary neuralgia. Our patient had trigeminal neuralgia of atypical type, given that there was hypoesthesia and no significant response to carbamazepine.⁴

The syndrome was probably secondary to a lingual metallic implant, and although findings indicate involvement of the trigeminal system, the location of the piercing and implant should not have resulted in trigeminal injury (Figure, C). The causal mechanism may involve mechanical or chemical nerve irritation with secondary sensitization of central and peripheral type. Another possibility is galvanic phenomena,⁵ in which intraoral galvanism is thought to generate electric currents that may elicit neuralgia. Trigeminal

Figure. Tongue Piercing With Insertion of Metal Stud, Sensory Divisions of the Trigeminal Nerve, and Anatomy of Atypical Trigeminal Neuralgia Secondary to Insertion of Stud



Insertion of the stud in this case may have provoked unilateral trigeminal neuropathic pain and associated hypoesthesia. The stud may have irritated the lingual branch of the mandibular nerve (V3); after possible thalamic and cortical projections, a secondary afferent response was manifested in the maxillary nerve (V2) and the inferior alveolar branch of mandibular nerve (V3). A basis for this phenomenon may include mechanical, chemical, or galvanic irritation of the lingual nerve.

neuralgia after a dental alloy implant, possibly triggered by galvanism, has been reported.⁶ Our patient described her pain in terms of electricity.

To our knowledge, this is the first published report of atypical trigeminal neuralgia associated with tongue piercing. This should be considered in the differential diagnosis of such symptoms.

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Acquisition of data: Mercuri, Galarza.

Drafting of the manuscript: Gazzeri, Mercuri, Galarza.

Critical revision of the manuscript for important intellectual content: Galarza.

Study supervision: Gazzeri, Mercuri, Galarza.

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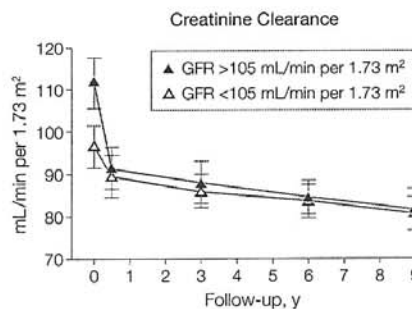
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CORRECTIONS

Incorrect Formula: In the Special Communication entitled "Reporting of Noninferiority and Equivalence Randomized Trials: An Extension of the CONSORT Statement" published in the March 8, 2006, issue of *JAMA* (2006;295:1152-1160), a formula for a 2-sided confidence interval (CI) used to assess noninferiority of a new treatment compared with a standard one with regard to an outcome was incorrect. On page 1158 in the subsection titled "Elaboration" that reads "the upper bound of the 2-sided $(1-\alpha/2) \times 100\%$ CI for the treatment effect has to be below the margin Δ to declare that noninferiority has been shown . . ." should read "the upper bound of the 2-sided $(1-2\alpha) \times 100\%$ CI for the treatment effect has to be below the margin Δ to declare that noninferiority has been shown . . ."

Error in Figure: In the Original Contribution entitled "Long-term Renal Outcomes in Patients With Primary Aldosteronism" published in the June 14, 2006, issue of

JAMA (2006;295:2638-2645), an error occurred in **FIGURE 3**. In the left panel, which reports creatinine clearance values, the scale of the y-axis is not correct, and the data are therefore not plotted correctly. The correct graph appears below.



No. of Patients						
GFR <105	25	25	25	14	8	
mL/min per 1.73 m ²						
GFR >105	25	25	25	14	8	
mL/min per 1.73 m ²						