

## Anatomic Differences in the Lateral Vestibular Nerve Channels and their Implications in Vestibular Neuritis

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**Hypothesis:** Anatomic differences may render the superior division of the vestibular nerve more susceptible to injury during vestibular neuritis.

**Background:** Neural degeneration has been identified in temporal bone studies of vestibular neuritis. Previous anatomic and physiologic studies of vestibular neuritis have demonstrated that the superior division of the vestibular nerve is preferentially affected, with sparing of the inferior division. A preliminary temporal bone study has implicated neural entrapment as a possible cause for this preferential injury.

**Methods:** Two independent unbiased observers performed histologic analysis of 184 temporal bones from our temporal bone library. Measurements of the medial, midpoint, and lateral portions of the superior vestibular, inferior vestibular, and the singular nerves and their bony channels lateral to the internal auditory canal were made. These measurements included the length and width of each bony channel and an estimated percent of each channel occupied by bony spicules at each location.

**Results:** The lengths of the bony channels of the singular nerve (0.598 mm) and the inferior vestibular nerve (0.277 mm) were significantly shorter than the average length of the superior vestibular channel (1.944 mm;  $p < 0.0001$ ). The total percent of the channel occupied by bone at the midpoint was significantly greater for the superior vestibular (28%) compared with either the singular (0%) or the inferior vestibular channel (18%) ( $p < 0.0001$ ).

**Conclusion:** The lateral bony channel of the superior vestibular nerve is seven times longer than the inferior vestibular and more than three times longer than the singular channel. There are a larger percentage of bony spicules occupying the superior vestibular compared with the inferior vestibular or singular channels. In addition, the superior nerve passes through a longer area of severe narrowing compared with the inferior or singular nerves. This anatomic arrangement of a longer bony channel with more interspersed bony spicules could make the superior vestibular nerve more susceptible to entrapment and ischemia. *Otol Neurotol* 26:489–494, 2005.

Vestibular neuritis has been described as an acute episode of vertigo lasting days to weeks. Its clinical course is characteristically absent of auditory findings or any other neurologic symptoms. Caloric response during electronystagmography testing is typically absent or markedly reduced, although the reduction is highly variable. There is generally complete recovery of symptoms for most patients within 6 months of the onset, whereas some patients experience lingering disequilibrium. Vestibular neuritis has generally been reported as a single, severe, prolonged attack of vertigo without recurrence occurring on one side; however, recurrent vestibular neuritis and bilateral vestibular neuritis have been described. The etiology of vestibular neuritis is

unknown, but the leading theory has been a viral inflammatory process with herpes simplex virus (HSV)-1 most commonly implicated (1–4).

Temporal bone histopathology in patients with vestibular neuritis has revealed varying degrees of degeneration of the peripheral vestibular nerve fibers and neuroepithelium. However, the pattern of degeneration has been mostly limited to the superior division of the vestibular nerve and, correspondingly, the horizontal and superior semicircular canals and the utricle (2,5). Three-dimensional vestibuloocular reflex testing of patients with vestibular neuritis have demonstrated findings that strongly implicate dysfunction of the ipsilateral horizontal and superior semicircular canal (superior division of the vestibular nerve) while sparing ipsilateral posterior canal function (inferior division) (6). In addition, the common clinical finding of benign paroxysmal positional vertigo (BPPV) after an episode of vestibular neuritis, in spite of absent caloric function, also implies sparing of

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the inferior division (posterior semicircular canal) with degeneration of the superior division of the vestibular nerve (utricle and horizontal semicircular canal).

Goebel et al. (7) recently reported a preliminary temporal bone study demonstrating anatomic conditions of the vestibular canals that would predispose the superior division to be more susceptible to injury from entrapment and ischemia that one might see as the result of viral neuritis. Specifically, the study revealed that the superior division of the vestibular nerve had to traverse a longer bony channel composed of more interspersed reticulated bone, allowing less room for swelling than the singular nerve. In addition, the arterial supply of the superior division and associated neuroepithelium had similar anatomic constraints. However, the study only compared the singular nerve/channel to the superior nerve/channel and did not include the inferior division of the nerve. The measurements were only made in one midsection of the channel and did not include the medial and lateral openings. In the current study, two independent, unbiased observers have analyzed a much larger number of temporal bones and included measurements of the superior division, the inferior division, and the singular nerve channels. Measurements of channel width included medial, lateral, and midpoint measurements and an estimation of percent bony impingement within each section of the channel.

## METHODS

Histologic analysis and measurements were made on 184 temporal bones from the Tulane University School of Medicine temporal bone library. All temporal bones had accompanying histories. All temporal bones with any known otologic pathology were excluded from the study. Using a light microscope and a micrometer, we made measurements of the vestibular nerve subdivisions and the surrounding bony channel lateral to the internal auditory canal. The temporal bone sections were oriented in the axial plane and were 10  $\mu\text{m}$  intervals. Measurements were made at  $\times 40$  and  $\times 100$  magnification. The sections measured were chosen to represent the largest cross-sectional width for each channel and nerve measured. Where two sections appeared to be appropriate for measurement, an average of the measurements from each of these sections was used.

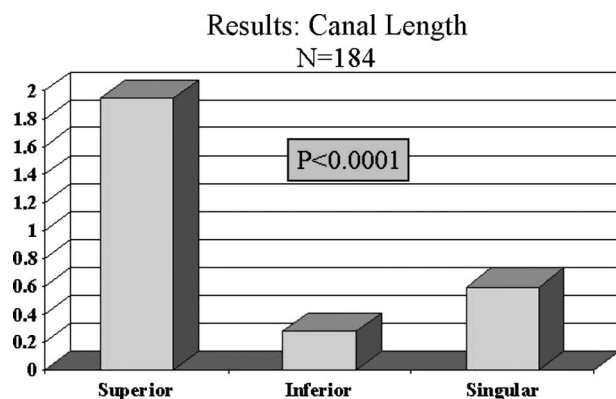
Specific measurements included the superior division, the inferior division, and the singular nerves and associated bony channels. The length for each channel was measured as well as the width for each channel and nerve. Nerve length was measured from the initial point where the nerve separates from the main vestibular nerve trunk to the exit into the inner ear. Width measurements were made at the lateral opening near the internal auditory canal, the midpoint, and the medial opening. The medial openings of each channel were defined as the point where the channel is distinct from the internal auditory canal; usually this was noted by an acute angle on one side of the bony channel and a more obtuse angle on the other side. The lateral channel openings were defined by the point at which the respective nerves entered the inner ear to innervate their respective neuroepithelium. The midpoint measurements were taken from the point exactly half the distance from the medial

to the lateral openings of each channel. In addition, at each of the width measurements, an assessment of the percent of bone occupying the canal was made. The assessment of percent bone occupying the channel was determined by subjective assessment by each observer. The width measurements and estimate of bone occupying the canal at the medial, lateral, and midpoints of each canal were not chosen as representative sections of the entire canal but only representative of the region measured. In addition, the ratio of nerve diameter to the entire free space (total width minus bony spicules) of the channel width was determined. Two independent unbiased observers made the measurements who were unaware of the study's hypothesis. The results were then averaged together for *t* test statistical analysis.

## RESULTS

One hundred eighty-four temporal bones were successfully analyzed. The average length of the bony channel of the superior division of the vestibular nerve (1.944 mm) was more than three times longer than the singular (0.598 mm) and seven times longer than the inferior nerve channel (0.277 mm) (Fig. 1). This finding was highly statistically significant ( $p < 0.0001$ ). However, the length of the superior nerve (3.18 mm) was significantly ( $p < 0.0001$ ) shorter than the singular nerve (3.8 mm). The inferior nerve was the shortest ( $p < 0.0001$ ) of the three (0.51 mm). In other words, the singular nerve is longer than the superior vestibular nerve, but the bony channel that the singular nerve courses through is shorter than the bony channel that the superior vestibular nerve courses through.

Bony channel width was wider medially for the superior and singular channels than either at the midpoint or laterally, but the inferior channel was slightly wider laterally than at the midpoint or medially (Fig. 2). The singular and inferior canals had easier lateral openings to measure than the superior canal because the superior canal innervates the superior and horizontal canal, utricle and, by way of Voit's anastomosis, the saccule compared with the single innervations of the singular (posterior semicircular canal) and inferior nerves (saccule). This



**FIG. 1.** Comparison of vestibular canal length. The superior vestibular canal was significantly longer than either the inferior vestibular canal or the singular canal.

Results: Canal Width

N=184

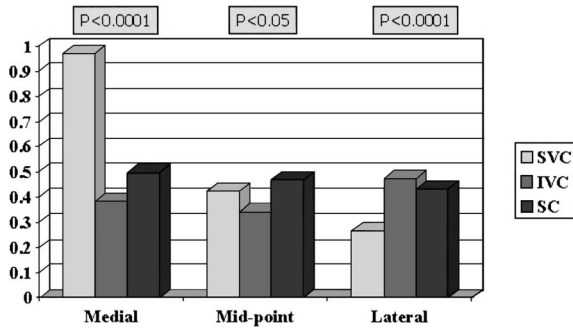


FIG. 2. Comparison of canal width at medial, midpoint, and lateral measurements. The canals were significantly different for each point of measurement. The progressive narrowing of the superior vestibular canal is notable compared with the other two canals.

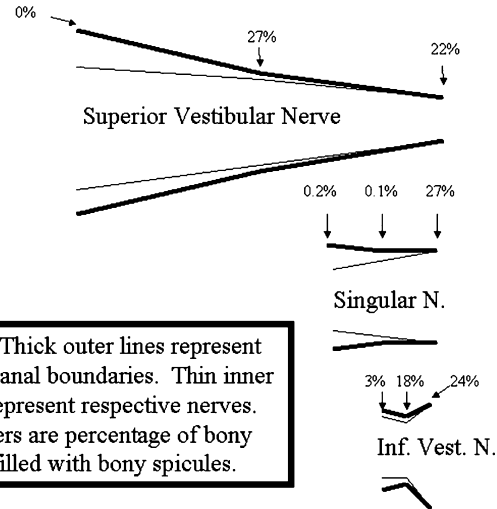
made comparisons of the lateral widths essentially not useful. However, the superior channel becomes comparatively narrower as it progresses laterally. The width of both the nerve and the bony channel (0.66 mm, 1.04 mm) were larger for the superior division at the medial opening compared with the inferior division nerve/channel (0.35 mm, 0.45 mm) and the singular nerve/channel (0.32 mm, 0.57 mm). The widths were very similar at the midpoint. The ratio of nerve width to free space width (channel width minus bony spicules) demonstrated more restrictive passage of the superior nerve compared with the inferior or singular nerves for the midpoint and lateral measurements. The inferior nerve had the most restrictive ratio when measured medially (Table 1). Although this would appear to demonstrate no significant differences in the ratios for the superior nerve channel compared with the others, when you consider the significantly longer length of the superior channel, you realize that the midpoint is measured at a point that would be medial to the medial opening of the singular or inferior channels (Fig. 3). Consequently, the superior nerve courses through a much longer segment of restriction in its bony channel than either of the other two nerves.

The percent of bone occupying the channel lumens was not significantly different at the medial and lateral opening, but they were significantly different at the midpoint measurement. The superior channel had a significantly ( $p < 0.0001$ ) higher percentage of bone occupying its lumen at the midpoint (28%) compared with either

TABLE 1. Nerve-free space ratio

	Superior nerve	Inferior nerve	Singular nerve
Medial	65.7	80.9	57.9
Midpoint	98.9	97.2	92
Lateral	99.1	97.3	91.9

Ratio = nerve width/(channel width-bony spicules).



Note: Thick outer lines represent bony canal boundaries. Thin inner lines represent respective nerves. Numbers are percentage of bony canal filled with bony spicules.

FIG. 3. Schematic outline of the vestibular nerves and channels that illustrates relative lengths and widths. Note the markedly longer superior division, which has a longer section of severe narrowing compared with the other divisions.

the inferior canal (18%) or the singular canal (0.13%) (Figs. 4 to 6).

DISCUSSION

The vestibular nerve innervates the five vestibular sense organs in each labyrinth. The vestibular nerve separates into three separate entities: the superior vestibular nerve, the inferior vestibular nerve, and the singular nerve. Laterally, each nerve passes through a bony channel interlaced with bony spicules before innervating its respective receptors (Figs. 7 to 9). The superior vestibular nerve innervates the superior semicircular canal ampulla, the horizontal (or lateral) semicircular canal ampulla, the utricle, and the saccule by way of Voit's anastomosis. The inferior vestibular nerve is the

Results: Percent of Canal Occupied by Bone – Medial

N=184

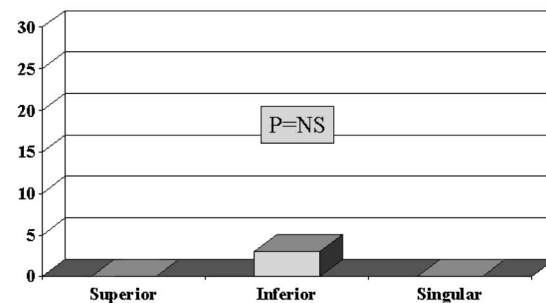
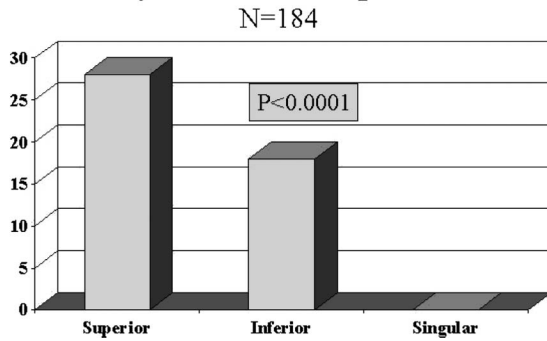


FIG. 4. Percent of canal occupied by bone: medial measurement. There was no significant difference among the canals.

Results: Percent of Canal Occupied by Bone – Midpoint



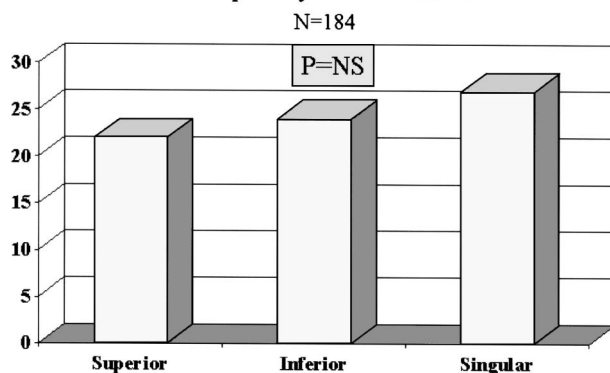
**FIG. 5.** Percent of canal occupied by bone: midpoint measurement. The superior vestibular canal had a significantly larger percentage of bone occupying its lumen than either the inferior vestibular or singular canals.

principle neural supply for the saccule, and the singular nerve innervates the posterior semicircular canal ampulla.

Schucknecht and Kitamura (2) studied temporal bone histopathology in clinical cases of vestibular neuritis and found selective superior vestibular nerve atrophy with neuroepithelial degeneration in some cases and selective superior vestibular nerve atrophy without receptor damage in other cases. They concluded that vestibular neuritis preferentially damaged the superior vestibular nerve or peripheral receptors. Although they were suspicious of a vascular compromise as the cause, there was no evidence of thrombosis or hemorrhage.

Three-dimensional vestibuloocular testing in patients with clinical vestibular neuritis has also demonstrated selective dysfunction of the ipsilateral superior vestibular nerve or its respective end organs. Fetter and Dichgans (6), using three-dimension rotational studies, demonstrated normal ipsilateral posterior semicircular canal function with abnormal ipsilateral horizontal and superior

Results: Percent of Canal Occupied by Bone – Lateral



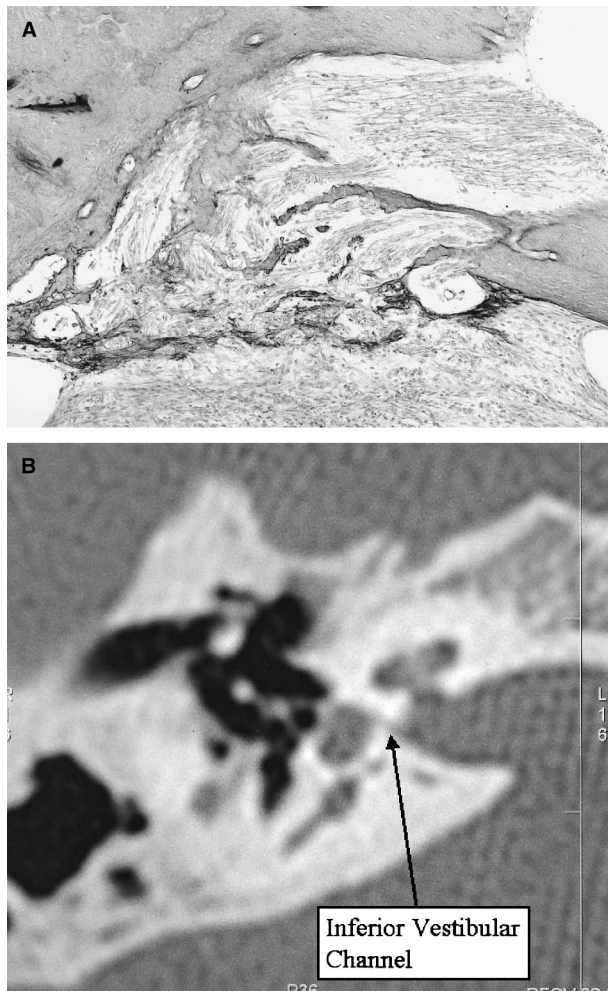
**FIG. 6.** Percent of canal occupied by bone: lateral measurement. There was no significant difference among the canals.



**FIG. 7.** Histologic section of the superior vestibular channel (A) and corresponding axial computed tomography scan (B). The entire canal is not shown. Note the ampulla of the lateral canal on the left and the significant amount of interspersed bony meshwork compared with the other canals (Figs. 8 and 9).

semicircular canal function after vestibular neuritis. These findings suggested preferential degeneration of the superior vestibular nerve. From a clinical standpoint, BPPV is frequently seen after vestibular neuritis. The presence of BPPV implies continued function of the posterior semicircular canal ampulla and the singular nerve because abolition of singular nerve or posterior semicircular canal function eliminates BPPV. The finding of BPPV in spite of absent caloric function in the same ear strongly implicates selective destruction of the superior vestibular nerve or horizontal semicircular canal function and preservation of posterior canal function.

Although the etiology of vestibular neuritis is still unknown, the preponderance of evidence implicates a viral inflammatory process, with HSV-1 the most likely offending organism. HSV-1 is ubiquitous. Serologic evidence of primary exposure by HSV-1 can be found in more than 80% of the adult population and has been identified in Scarpa's ganglion in 60% of temporal bone specimens by polymerase chain reaction (8–11). After primary infection, HSV-1 becomes dormant in a latent state among sensory neural ganglia. Reactivation of

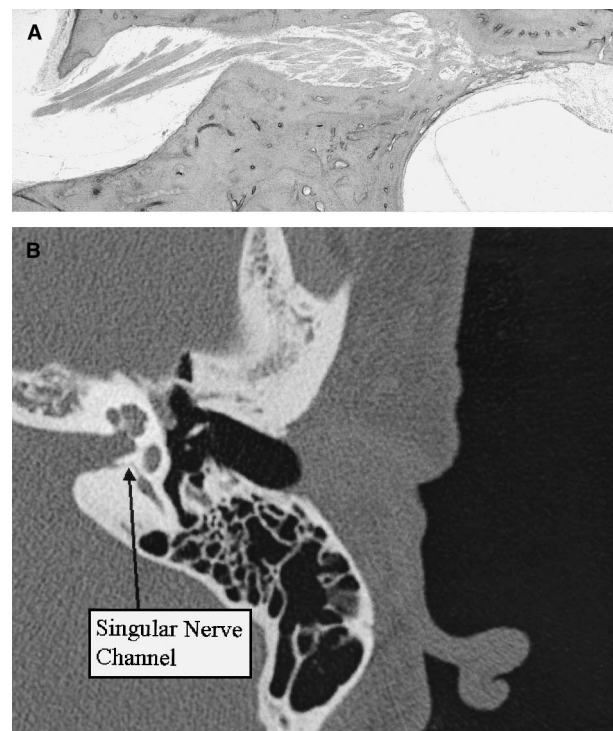


**FIG. 8.** Histologic section of the inferior vestibular channel (A) and corresponding axial computed tomography scan (B). The canal is much shorter and has less bony meshwork than the superior vestibular canal.

HSV-1 can result in focal involvement of a single nerve. Other neurotropic viruses can produce similar reactivation neuritides. It is believed by many authors that reactivation of a latent neurotropic virus is the etiology for vestibular neuritis.

Viral reactivation does not explain the preferential damage to the superior division of the vestibular nerve compared with either the inferior division or the singular nerves. Arbusow et al. (10) studied evidence of HSV-1 infection in both Scarpa's ganglion and the geniculate ganglion and found widespread distribution of the viral elements throughout both ganglia. They invoked a dual innervation theory of the posterior canal by way of an accessory singular nerve to explain posterior canal sparing. However, we believe the anatomic differences of the vestibular bony channels, as demonstrated in this study, may more easily explain the susceptibility of the superior division for degeneration. The superior vestibular nerve travels through a bony channel that is seven times

longer than the inferior and more than three times longer than the singular channel. Moreover, the superior channel has a larger percentage of interspersed bony meshwork occupying the canal lumen than either of the other two channels. As seen in Figure 3, the superior nerve passes through a "restricted area," with the nerve occupying 99% of the available space in the bony channel for a distance longer than either of the other two nerves. Although the singular and inferior nerves eventually pass through 92% and 97% restrictive areas, respectively, they do so for only a very short segment. In a previous study by Goebel et al. (7), the arterioles within the superior channel were found to have longer, narrower bony channels than the arterioles accompanying the singular nerve. If, as suspected, vestibular neuritis is a viral inflammatory process, the superior vestibular nerve would be more susceptible to entrapment and ischemia because of these anatomic differences. Furthermore, recurrent or reactivated ganglionitis may explain recurrent attacks of neuritis but still cannot explain selective preservation of posterior semicircular canal function seen in this disorder. However, the current study confirms the preliminary findings by Goebel et al. (7) and has provided additional anatomic data regarding the inferior division of the vestibular nerve and singular nerve that would render them less susceptible



**FIG. 9.** Histologic section of the singular channel (A) and corresponding axial computed tomography scan (B). Note the posterior semicircular canal ampulla on the right. The canal is much shorter and has less bony meshwork than the superior vestibular canal.

during bouts of neuritis to entrapment/ischemia than the superior division.

### CONCLUSIONS

The lateral bony channel of the superior division of the vestibular nerve is seven times longer than the inferior vestibular and more than three times longer than the singular channel. There is a larger percentage of bone spicules occupying the lumen of the superior channel compared with either the inferior or the singular channel at the midpoint of the canals, and the ratio of nerve to free space in the channel is more restrictive than either the singular or inferior channels. These anatomic differences—longer channel with more interspersed bony spicules and a relatively more narrow channel—make the lateral portion of the superior division of the vestibular nerve more susceptible to entrapment and ischemia than either the inferior or singular nerve. We propose that vestibular neuritis, although initiated by a viral process involving the entire vestibular nerve, may result in entrapment/ischemia that preferentially affects the superior division of the vestibular nerve.

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