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Anatomic Considerations in Vestibular Neuritis

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Hypothesis: The authors believe that anatomic differences render the superior division of the vestibular nerve more susceptible to injury during vestibular neuritis. The purpose of the study was to investigate anatomic differences between the superior vestibular nerve and singular nerve canals.

Background: Previous studies of temporal bones have revealed vestibular nerve degeneration in patients with vestibular neuritis. Although the cause of this degeneration has not been established, it has been noted that the superior division of the vestibular nerve is preferentially affected, with sparing of the inferior division. The superior vestibular nerve and the singular nerve, a branch of the inferior vestibular nerve, both pass through canals interlaced with bony networks before reaching the peripheral receptors.

Methods: The authors performed histologic analysis of 40 normal temporal bones randomly selected from their temporal bone library. With a micrometer, measurements were taken of the individual canals. The ratio of the total bony spicule component to the total canal width was obtained for both the superior vestibular nerve and the singular nerve. The length of the canals was also measured. Arteriole:arteriolar canal ratios of the superior vestibular nerve and singular nerve were obtained.

Results: The bony channel of the singular nerve had an average length of 0.59 mm, and the average length of the superior vestibular nerve was 2.30 mm ($p < 0.001$). The ratio of total bony spicule width to total canal width was significantly smaller ($p < 0.05$) for the singular nerve (0.30 mm) compared with the superior vestibular nerve (0.34 mm). The arteriole: arteriolar canal ratio was significantly smaller ($p < 0.05$) for the singular nerve (0.45 mm) than for the superior vestibular nerve (0.54 mm).

Conclusion: The bony canal of the superior vestibular nerve is longer than the singular nerve canal. Additionally, the superior vestibular nerve and arteriole travel through a relatively narrower passage than the singular nerve and its vascular supply. From an anatomic standpoint, this renders the superior division of the vestibular nerve more susceptible to entrapment and possible ischemic labyrinthine changes. **Key Words:** Vestibular nerve—Singular nerve—Vestibular neuritis—Temporal bone.

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Vestibular neuritis, a disorder of unknown cause, is described as an acute episode of severe vertigo and nausea lasting for days to weeks. It is the second most common cause of vertigo other than benign paroxysmal positional vertigo. Other characteristic findings include the absence of auditory symptoms, markedly reduced or absent caloric response in the affected ear, and the absence of other neurologic symptoms or findings. The natural course of the disease varies. In most patients, complete recovery of vestibular signs and symptoms occurs within 6 months after the acute episode (1).

A single prolonged attack is the most common presentation, but this diagnosis has been applied to some patients who have had recurring similar episodes. These are usually less intense and may occur years after the initial episode (1,2). Bilateral vestibular neuritis has also been described (3). Synonyms of vestibular neuritis include epidemic vertigo (4), vestibular paralysis (5), and vestibular neuronitis (6). Differential diagnosis includes vestibular schwannoma, herpes zoster oticus, multiple sclerosis, vascular occlusion, and Ménière's disease.

Although there is no consensus about the cause of vestibular neuritis, viruses have largely been implicated, including herpes simplex virus 1 (HSV-1) (7–10). Another infectious agent, *Borrelia burgdorferi,* has been named to play a causative role (11,12). Conclusive evidence in human beings, however, is lacking.

Histopathologic examination of the temporal bones in patients known to have had vestibular neuritis has revealed degeneration of peripheral vestibular nerve fibers and the neuroepithelium of the peripheral receptors. This has largely been seen in the superior division of the vestibular nerve, involving the lateral and superior semicircular canals (2,13). Furthermore, a more recent study using three-dimensional vestibuloocular reflex testing in patients with vestibular neuritis suggests that vestibular neuritis preferentially affects the superior division of the vestibular nerve while sparing the inferior division (14). The cause of this discrepancy is unknown.

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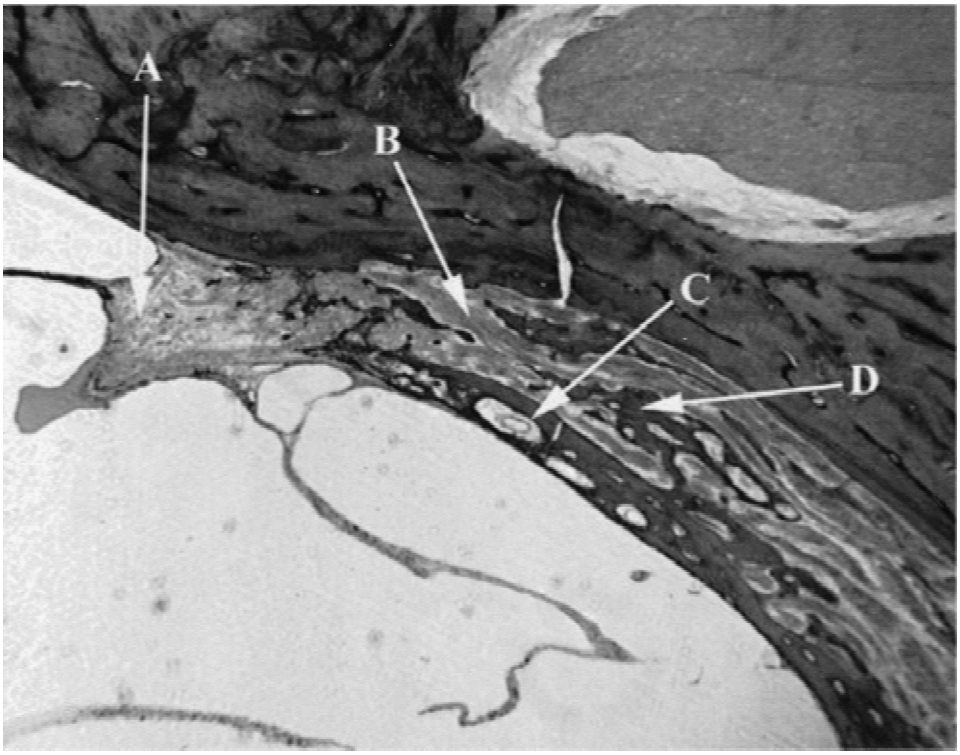

FIG. 1. Photomicrograph of superior vestibular nerve canal. A, ampulla of lateral semicircular canal; B, superior vestibular nerve; C, arteriole of superior vestibular nerve; D, reticulated bony canal. (Hematoxylin and eosin; original magnification ×40.)

Inherent anatomic differences exist between the neuronal and arteriolar courses of the superior vestibular nerve and singular nerve, a branch of the inferior vestibular nerve. This may render the superior division and the accompanying vascularity more vulnerable to compression and ischemia triggered by perineuritis. Before reaching their respective end organs, the superior vestibular nerve and singular nerve pass through canals par-

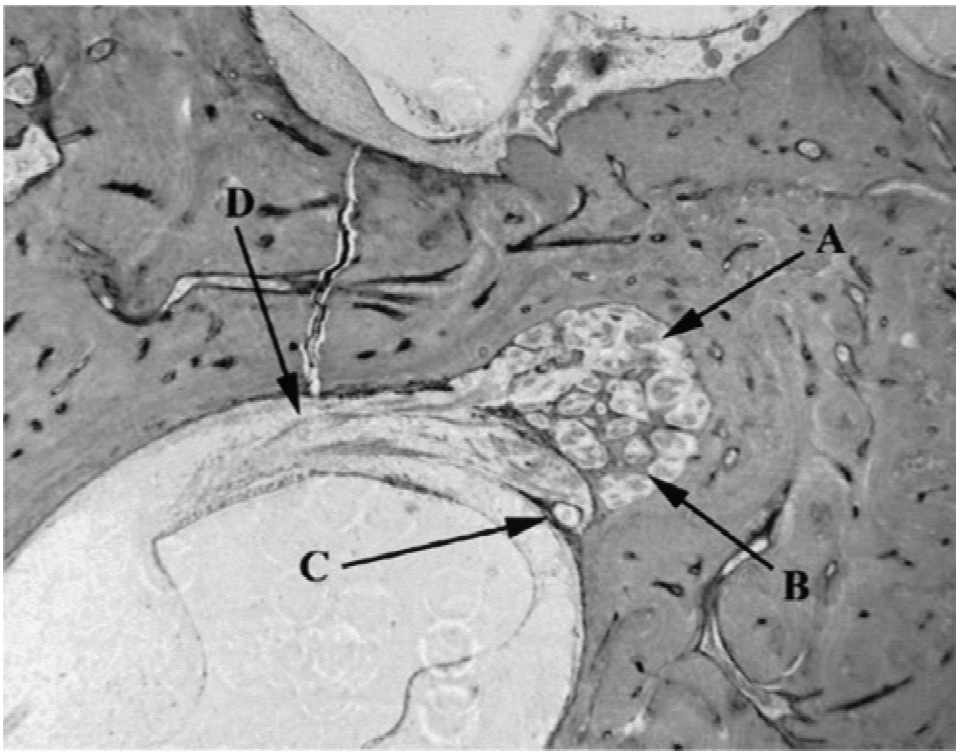

FIG. 2. Photomicrograph of singular nerve canal. A, singular nerve; B, reticulated bony canal; C, arteriole of the singular nerve; D, posterior semicircular canal. (Hematoxylin and eosin; original magnification ×40.)

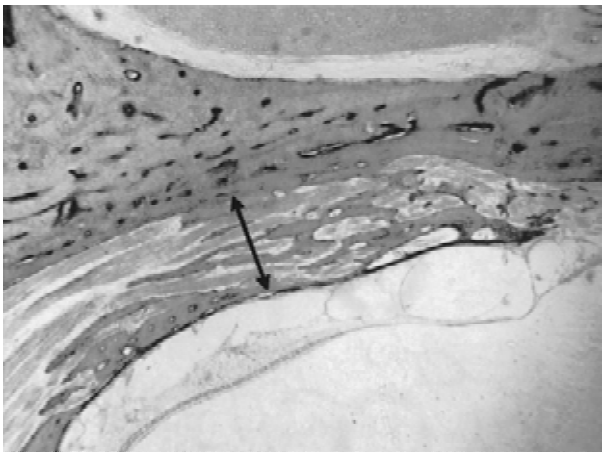

FIG. 3. Photomicrograph of superior vestibular nerve and canal. The arrow marks the midpoint of the canal length and the site of measurement of the superior vestibular nerve canal width. (Hematoxylin and eosin; original magnification ×40.)

tially occluded with a bony meshwork. The arterioles accompanying the nerves also pass through a bony channel. The following analysis was performed to demonstrate key anatomic differences between the superior and inferior divisions of the vestibular nerve.

MATERIALS AND METHODS

A histologic analysis was performed on 40 randomly selected normal temporal bones from the Tulane University School of Medicine temporal bone library. By use of a light microscope and a micrometer, specific measurements were taken of the distal portions of the superior vestibular nerve and singular nerve. The area of measurement was focused on the bony channels, which the nerves pass through before reaching the peripheral end organ (Fig. 1 and 2). Successive coronal slices for each temporal bone were examined at ×40 and ×100 magnification. Values for each subject were averaged and recorded.

First, the length of the bony channels of each vestibular nerve division was measured. Next, measurements were taken to elucidate the proportion of the bony component of the bony channels of the vestibular nerves. The individual bony spicules were summated and recorded, and the total width of the bony canal was recorded. This was figured into a ratio of bony spicule component to total canal width (BC:TW). For each temporal bone, successive specimens were averaged. Measurements were taken at the midpoint of the length of the bony canals (Fig. 3 and 4). Ratios of arteriolar width to arteriolar canal width (A:AC) were also measured and averaged for both vestibular nerve divisions of each temporal bone (Fig. 5 and 6).

RESULTS

The average length of the bony canal of the singular nerve was 0.59 mm, and the average length of the bony canal of the superior vestibular nerve was 2.30 mm. The results were compared by use of a paired *t* test and found to be highly statistically significant ($p < 0.0001$) (Table 1).

The average ratio of the bony spicule component to canal width (BC:CW) of the singular nerve was 0.30, and the average BC:CW ratio of the superior vestibular nerve was 0.34. By use of a paired *t* test, this was also found to be statistically significant ($p < 0.05$) (Table 2). The percentage of specimens with values 0.30 or more was calculated. There were 52.5% singular nerve specimens in this group, and the superior vestibular nerve specimens contained 69% (Fig. 7).

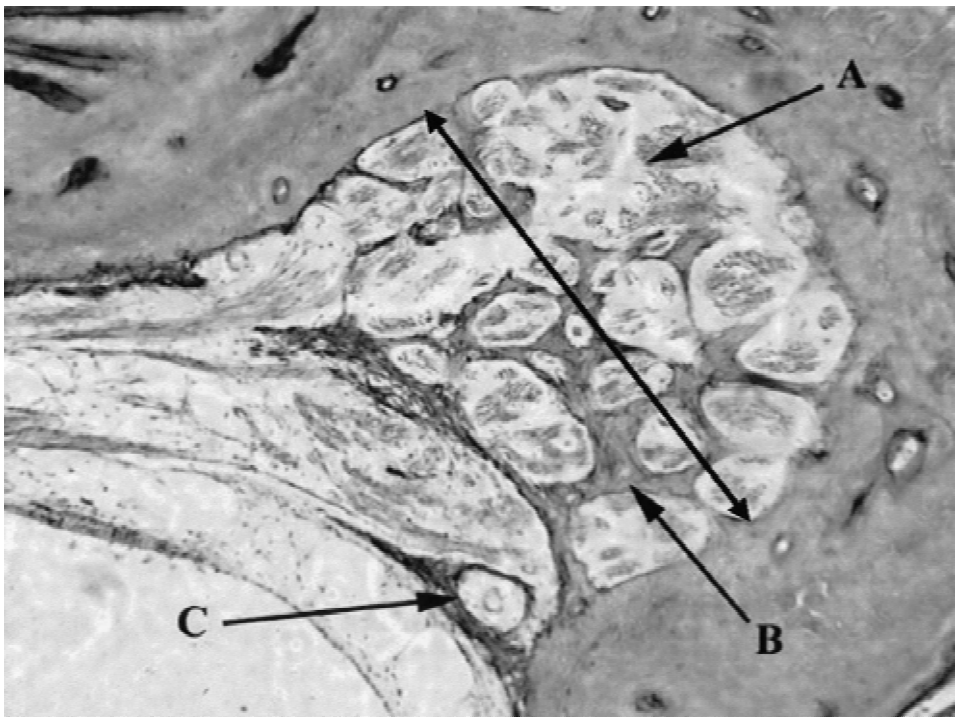

FIG. 4. Photomicrograph of the singular nerve and canal. The arrow marks the site of measurement of the singular nerve canal width. A, singular nerve; B, reticulated bony canal; C, arteriole of the singular nerve. (Hematoxylin and eosin; original magnification ×100.)

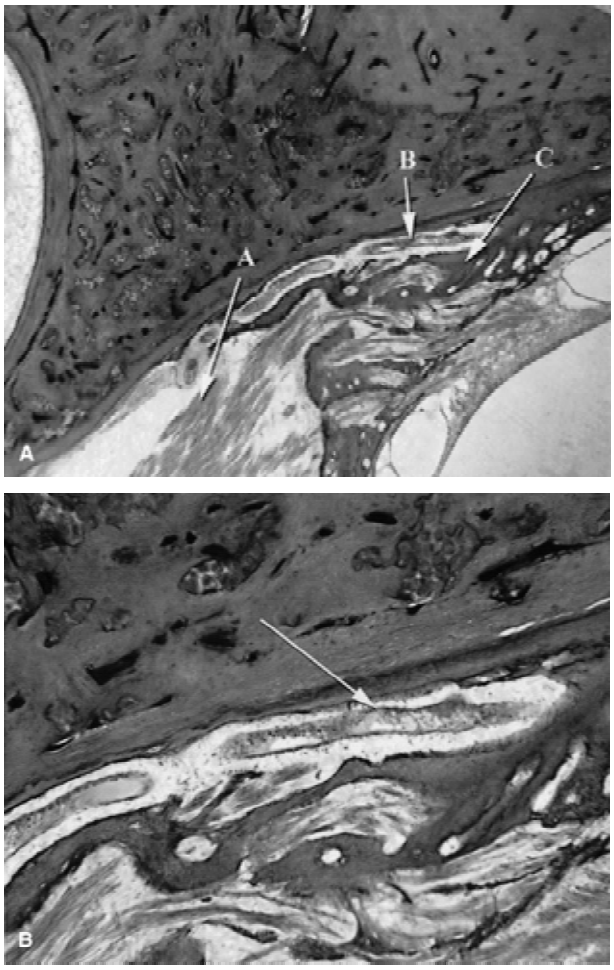

FIG. 5. **A:** Low-power view of the superior vestibular nerve (A), arteriole (B), and bony reticulated network of the superior vestibular nerve canal (L). (Hematoxylin and eosin; original magnification ×40.) **B:** Higher-power view of the superior vestibular nerve arteriole marked by the arrow. (Hematoxylin and eosin; original magnification ×100.)

The average ratio of arteriole to arteriolar canal width (A:AC) of the singular nerve was 0.45, and the average A:AC ratio of the superior vestibular nerve was 0.54. Again, by use of a paired *t* test, this difference was statistically significant ($p < 0.05$) (Table 3). The groups were also stratified into the percent of specimens with values 0.50 or more. According to Figure 8, only 23.7% of the singular nerve specimens had values of 0.50 mm or more, whereas 76.3% of superior vestibular nerve specimens fell into this category.

DISCUSSION

To our knowledge, this study is the first to provide a histologic explanation for selective superior vestibular nerve compression after vestibular neuritis. Previous studies have characterized the histologic changes that accompany neuritis, including atrophy of the superior nerve distal processes and variable degeneration of the lateral canal ampulla and utricle (2,13). Furthermore, three-dimensional rotational studies by Fetter and Dichgans have clearly demonstrated normal function of the ipsilateral posterior canal and abnormal function of the ipsilateral lateral and anterior canal (14). The dilemma, therefore, was to explain how an inflammatory process such as neuritis could cause such selective injury and functional loss.

Benign positional vertigo is common after an episode of vestibular neuritis. Furthermore, the pathophysiology of benign positional vertigo is dependent on the intact function of the posterior semicircular canal. In 1956, Lindsay and Hemenway described seven elderly patients with vertigo, which they attributed to anterior vestibular artery occlusion (15). In those cases, loss of both the distal superior nerve fibers and the end-organ sensory epithelium of the lateral canal ampulla and utricle suggested a mechanism of ischemic injury to both the nerve and the end organ alike. The authors also noted the his-

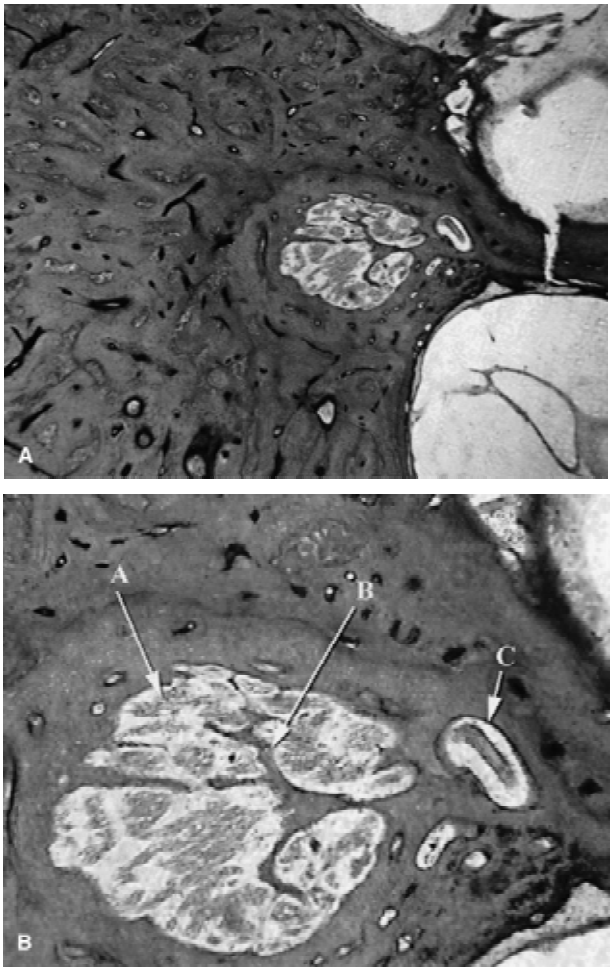

FIG. 6. **A:** Low-power photomicrograph of the singular nerve and canal. (Hematoxylin and eosin; original magnification ×40.) **B:** Higher-power view of the singular nerve (A), reticulated bony canal (B), and arteriole of the singular nerve (C). (Hematoxylin and eosin; original magnification ×100.)

TABLE 1. *Bony canal lengths (mm) of the singular nerve (SN) and the superior vestibular nerve (SVN) (in mm)*

SN*		SVN†	
1. 0.48	21. 0.63	1. 1.56	21. 2.26
2. 0.39	22. 0.56	2. 1.56	22. 2.47
3. 0.60	23. 0.48	3. N/A	23. 2.82
4. 0.52	24. 0.53	4. 1.76	24. 2.67
5. 0.55	25. 0.54	5. 1.91	25. 3.12
6. 0.37	26. 0.55	6. 1.98	26. 3.38
7. 0.43	27. 0.64	7. 2.17	27. 2.46
8. 0.52	28. 0.54	8. 2.39	28. 1.30
9. 0.64	29. 0.40	9. 2.02	29. 2.11
10. 0.69	30. 0.36	10. 2.43	30. 2.41
11. 0.92	31. 0.43	11. 1.80	31. 2.47
12. 0.65	32. 0.89	12. 2.38	32. 2.89
13. 0.67	33. 0.52	13. 2.35	33. 2.19
14. 0.52	34. 0.64	14. 2.34	34. 1.90
15. 0.53	35. 0.81	15. 2.31	35. 1.76
16. 0.57	36. 0.61	16. 2.64	36. 2.22
17. 0.32	37. 0.68	17. 2.24	37. 2.32
18. 0.33	38. 0.85	18. 2.43	28. 2.56
19. 1.00	39. 0.24	19. 2.28	39. 2.30
20. 1.42	40. 0.46	20. 3.22	40. 2.21

*N = 40; range 0.24–1.42 mm; average 0.59 mm.
†N = 39; range 1.30–3.38 mm; average 2.30 mm.
N/A, not applicable because of inadequate histologic preservation.

torical appearance of benign positional vertigo weeks after the prolonged first attack of vertigo. Schuknecht studied these seven cases and four others and concluded that loosened otoconia secondary to utricular injury could migrate toward the posterior canal ampulla to cause positional vertigo (16) (Fig. 9).

In 1981, Schuknecht and Kitamura described temporal bone findings in clinical cases of vestibular neuritis (2). In some cases, superior vestibular nerve atrophy was seen without end-organ injury. In other instances, however, both neural atrophy and end-organ degeneration was evident. Despite the suspicion of vascular occlusion, there was no evidence of arteriolar thrombus or intralabyrinthine hemorrhage. They concluded that neuritis preferentially damaged the superior nerve and end organ but did not offer a plausible explanation for this selectivity.

The initial insult in vestibular neuritis has for some time been attributed to a viral infection of the nerve sheath or Scarpa's ganglion. Although the damage seen in Schuknecht's cases was consistent with a viral cause, no direct evidence of viral particles was found in the ganglion cells. However, Furata et al. reported 6 of 10 temporal bones studied with evidence of HSV in Scarpa'a ganglion, using polymerase chain reaction (8). They believed that vestibular neuritis was caused by an HSV-1 ganglionitis. Further evidence for viral infection of both Scarpa's ganglion and the geniculate ganglion was provided by Arbusow et al. (10). They reported HSV-1 in 66% of geniculate and 60% of vestibular ganglia in 35 temporal bones. They believed that all areas of Scarpa's ganglion were equally affected and that the sparing of the posterior canal may be caused by the presence of dual innervation via distinct peripheral processes seen in some specimens.

TABLE 2. *Bony spicule component width: total canal width ratios for the singular nerve (SN) and the superior vestibular nerve (SVN) (in mm)*

SN*	SVN†
1. 0.12	1. 0.44
2. 0.35	2. 0.35
3. 0.36	3. N/A
4. 0.20	4. 0.41
5. 0.32	5. 0.24
6. 0.43	6. 0.27
7. 0.18	7. 0.26
8. 0.25	8. 0.27
9. 0.32	9. 0.27
10. 0.30	10. 0.25
11. 0.26	11. 0.34
12. 0.32	12. 0.34
13. 0.22	13. 0.36
14. 0.39	14. 0.33
15. 0.29	15. 0.39
16. 0.25	16. 0.39
17. 0.39	17. 0.31
18. 0.46	18. 0.47
19. 0.15	19. 0.37
20. 0.20	20. 0.27
21. 0.24	21. 0.26
22. 0.21	22. 0.24
23. 0.29	23. 0.23
24. 0.31	24. 0.40
25. 0.31	25. 0.33
26. 0.31	26. 0.26
27. 0.33	27. 0.37
28. 0.39	28. 0.42
29. 0.39	29. 0.43
30. 0.48	30. 0.55
31. 0.28	31. 0.39
32. 0.30	32. 0.33
33. 0.33	33. 0.40
34. 0.31	34. 0.35
35. 0.29	35. 0.31
36. 0.41	36. 0.35
37. 0.18	37. 0.27
38. 0.22	38. 0.31
39. 0.24	39. 0.39
40. 0.24	40. 0.33

*N = 40; average 0.30 mm.
†N = 39; average 0.34 mm.
N/A, not applicable because of inadequate histologic preservation.

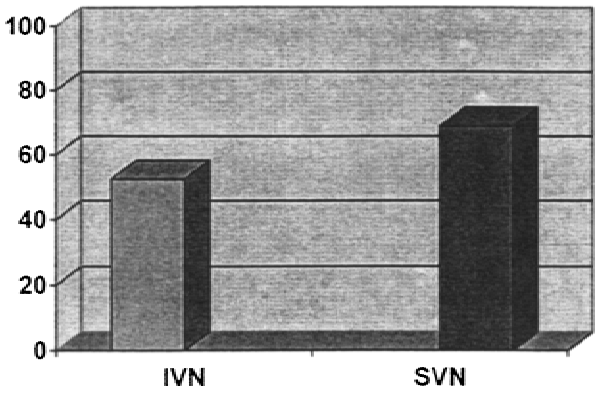

FIG. 7. Percent of specimens of each vestibular nerve having a ratio of bony spicule component width to total canal width of 0.30 or more. SN, singular nerve; SVN, superior vestibular nerve.

TABLE 3. *Ratio of arteriole width to arteriolar canal width for the singular nerve (SN) and the superior vestibular nerve (SVN) (in mm)*

SN*	SVN†
1. 0.36	1. 0.46
2. 0.55	2. 0.52
3. N/A	3. N/A
4. 0.37	4. 0.60
5. 0.42	5. 0.47
6. 0.40	6. 0.63
7. 0.69	7. 0.40
8. 0.37	8. 0.62
9. 0.48	9. 0.48
10. 0.33	10. 0.49
11. 0.34	11. 0.55
12. 0.40	12. 0.63
13. 0.43	13. 0.50
14. 0.42	14. 0.47
15. 0.54	15. 0.45
16. 0.43	16. 0.50
17. 0.42	17. 0.57
18. 0.60	18. 0.61
19. N/A	19. 0.55
20. 0.48	20. 0.52
21. 0.43	21. 0.48
22. 0.44	22. 0.59
23. 0.45	23. 0.63
24. 0.34	24. 0.55
25. 0.38	25. 0.75
26. 0.50	26. 0.52
27. 0.48	27. 0.41
28. 0.44	28. 0.55
29. 0.48	29. 0.52
30. 0.41	30. 0.50
31. 0.50	31. 0.55
32. 0.45	32. 0.63
33. 0.43	33. 0.50
34. 0.35	34. 0.55
35. 0.53	35. 0.51
36. 0.60	36. N/A
37. 0.42	37. 0.52
38. 0.44	38. 0.59
39. 0.50	39. 0.53
40. 0.50	40. 0.57

*N = 38, average 0.45 mm.
†N = 38, average; 0.54 mm.
N/A, arteriole/arteriolar canal not visualized in the histologic specimens.

In light of the current study, the following hypothetical scenario is proposed: (1) vestibular neuritis is initially a viral ganglionitis causing transient neural dysfunction and swelling of the nerve sheath; (2) in mild to moderate cases, minimal entrapment occurs in either the superior or inferior distal nerve processes, and the patient quickly recovers with minimal residual injury; (3) in more severe cases, neural entrapment occurs involving the superior division within its longer, narrower bony passage. End-organ degeneration does not occur because the vascular supply is not compromised by the compressive neuropathy; and (4) in severe cases, entrapment leads to vascular compromise of the anterior vestibular artery branches because of their smaller arteriolar channels, causing additional ampullary and macular injury and loosening of otoconia. Clinical evidence of lateral canal dysfunction

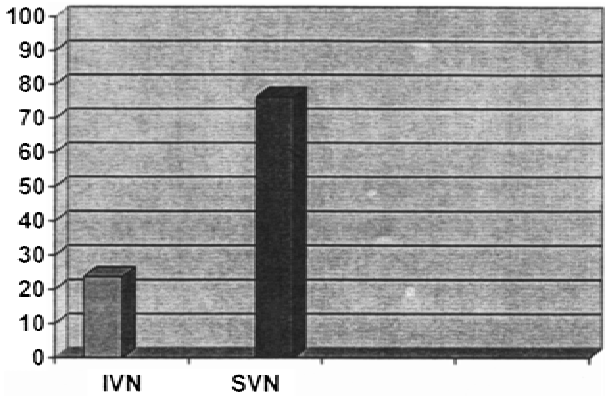

FIG. 8. Percent of specimens of each vestibular nerve having a ratio of arteriolar width to arteriolar canal width of 0.50 mm or more. SN, singular nerve; SVN, superior vestibular nerve.

(absence of caloric response, positive head thrust) is seen, and postneuritic positional vertigo appears as the otoconial debris settles into the posterior canal. Hence, the entire clinical spectrum can be explained on a continuum of neural entrapment and variable vascular involvement.

It should be noted, however, that the ultimate evidence for vascular compromise in cases of neuritis is still lacking. To date, there is no clear-cut histologic evidence of arteriolar thrombi accompanying the changes seen in the

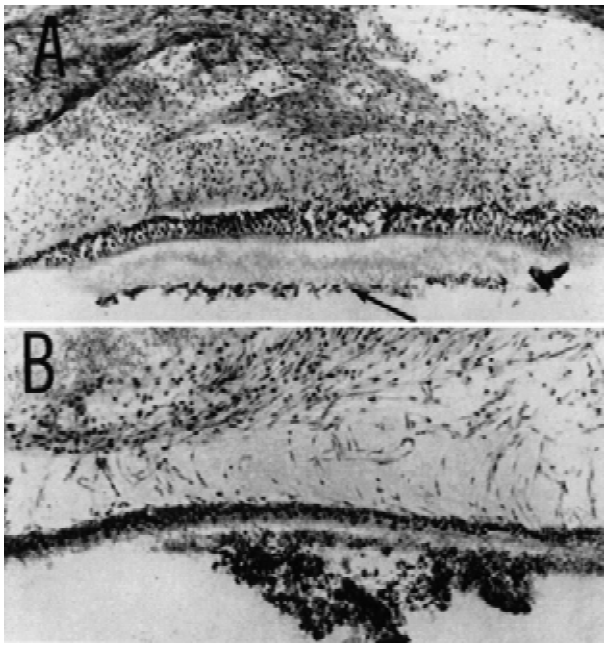

FIG. 9. **A:** Normal utricular macula with its otolithic membrane and otoconia (arrow). **B:** Degenerating utricular macula and otolithic membrane with partial detachment of otoconia following section of the anterior vestibular artery. (Reproduced with permission from *Annals of Otology, Rhinology, and Laryngology*. Taken from Schuknecht HF. Mechanism of inner ear injury from blows to the head. *Ann Otol Rhinol Laryngol* 1969;78:253–62.)

nerve and end organ. However, vascular occlusion experiments in cats have demonstrated similar neural and end-organ degeneration, which is not seen with sectioning of the nerve alone (16). It seems, therefore, that some form of vascular compromise is essential to the development of end-organ damage.

Further studies are planned to clarify the relationship of neuritis and positional vertigo with the histologic picture of end-organ degeneration. If, indeed, direct evidence of vascular occlusion can be proved, then vestibular neuritis could be viewed as a potentially treatable event, and steps can be taken to avoid excessive compression and possibly even end-organ degeneration.

CONCLUSIONS

1. The distal superior vestibular nerve travels through a longer, more reticulated bony channel than the singular nerve.
2. The arterioles following the superior vestibular nerve course through smaller bony channels than those associated with the singular nerve.
3. These anatomic differences predispose the superior vestibular nerve and accompanying blood supply to compressive injury and possibly even end-organ ischemia secondary to swelling caused by vestibular neuritis.

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