Semicircular Canal Dehiscence: Imaging, Diagnosis, Classification, Surgical Options, and Postoperative Imaging

S.M. Weindling and D.F. Broderick

CME Credit

The American Society of Neuroradiology (ASNR) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The ASNR designates this enduring material for a maximum of one AMA PRA Category one creditTM. Physicians should claim only the credit commensurate with the extent of their participation in the activity. To obtain credit for this activity, an online quiz must be successfully completed and submitted. ASNR members may access this quiz at no charge by logging on to eCME at http://members.asnr.org. Nonmembers may pay a small fee to access the quiz and obtain credit via http://members.asnr.org/ecme.

ABSTRACT

Since the association of sound- and/or pressure-induced vertigo and dehiscence of the superior semicircular canal was first described in 1998, imaging studies have been increasingly performed to exclude semicircular canal dehiscence in patients with “dizziness.” This article will first discuss normal semicircular canal bony roof development and then review the theorized pathophysiology, clinical signs and/or symptoms, and audiology examination findings of semicircular canal dehiscence. Semicircular canal dehiscence CT and MR imaging techniques and findings are reviewed. A semicircular canal dehiscence imaging classification system is proposed, which provides a standardized methodology for the interpretation and reporting of semicircular canal dehiscence imaging studies. Also, superior and posterior semicircular canal dehiscence surgeries and postoperative imaging findings are discussed.

Learning Objective: To identify and characterize deficiencies in the semicircular canal bony roofs using CT and MR imaging to review the clinical presentation and treatment options for patients with semicircular canal dehiscence syndrome and to discuss superior semicircular canal dehiscence postoperative imaging.

INTRODUCTION

In 1998, Minor et al1 described sound- and/or pressure-induced vertigo in patients who demonstrated superior semicircular canal dehiscence (SSCD) on CT imaging. Vertigo (illusion of motion), which may be induced by a loud sound (Tullio syndrome), pressure applied to the external auditory canal (Hennebert sign), or a sudden increase in intracranial pressure (Valsalva, heavy lifting), is the most common symptom ascribed to semicircular canal dehiscence (SCD). Autophony, the accentuated perception of self-generated sounds, such as heartbeat or breathing, and conductive hearing loss may also result from SCD. “Dizziness” is a common patient concern and, because the SCD syndrome has gained notoriety among all clinical specialties, imaging studies are increasingly ordered to exclude SCD as the underlying etiology. Because audiology testing may be inconclusive, otolaryngologists are deeply reliant on imaging to both accurately diag-
nose and characterize SCD before possible surgical intervention. Thus, a sound understanding of SCD is imperative for individuals who interpret temporal bone imaging studies.

**Superior Semicircular Canal Roof Development and Dehiscence Etiology**

The normal superior semicircular canal (SSC) roof (arcuate eminence) consists of 3 bony layers. From deep to superficial, these bony layers are the otic capsule, trabecular bone, and petrous ridge periosteal cortical bone which forms the middle and posterior fossa floors (Fig 1). Pneumatization of the central trabecular bony layer is variable. The arcuate eminence is uniformly thin in infants and progressively develops until approximately 3 years of age.2

SSCD is defined as the focal, complete absence of the bony roof; its exact etiology is uncertain. The bilateral nature of arcuate eminence thinning and dehiscence found on high-resolution CT3 and microscopic studies2 has led to proposals of insufficient postnatal development of the trabecular and periosteal cortical bony layers as the cause of abnormal SSC roof thinning. Because the arcuate eminence and tegmen tympani are contiguous, diminished postnatal development is similarly thought to play a role in coexistent thinning or dehiscence of SSC and tegmen tympani bony roofs (Fig 2), as seen at surgery4 and in cadaver studies.5

Most patients with SCD present in middle age,4 and an increased CT prevalence of SSCD in older age groups has been reported.6 A post-childhood insult has been postulated as a dehiscence inciting event, causing rupture of an abnormal developmentally thinned SSC bony roof.2 Triggering events previously reported in association with SSCD symptom onset include head trauma, weight lifting, coughing, and straining.4,7 Alternatively, systemic bone demineralization has been theorized to account for the increasing prevalence of SSCD observed with aging.6 Therefore, a “2-hit mechanism,” whereby an acute insult or temporal bone demineralization is superimposed on insufficient postnatal arcuate eminence development, is an intriguing possibility.

**Perilymph Pathophysiology**

In normal individuals, stapes inward motion at the oval window in response to sound initiates a perilymph fluid wave within the cochlea. This perilymph fluid wave travels from the scala vestibuli to the scala tympani, activating the organ of Corti contained within the central scala media. Because fluid is noncompressible, energy related to cochlear perilymph fluid motion is dissipated by compensatory outward motion at the round window (Fig 3 A). In the postulated mechanism for SSCD syndrome, the SSC bony roof deficiency creates a “third window,” which thereby allows transmission of sound-induced perilymph fluid wave pressure through the dehiscent SSC. This nonphysiologic SSC perilymph fluid wave causes firing of vestibular nerve afferent fibers, hence sound-induced vertigo (Fig 3B). In addition, the diversion of sound-induced perilymph fluid wave

---

**Fig 1. SSC roof development.** Temporal bone CT Pöschl view demonstrates the SSC posterior to the malleolus and incus within the epitympanic recess. The 3 layers of the SSC bony roof (arcuate eminence) are the otic capsule (arrowheads), trabecular bone (arrows), and the periosteal cortical bone (dashed arrows), which forms the middle and posterior fossa floor. Note the continuity of the arcuate eminence and tegmen tympani (block arrow).

**Fig 2. Coexisting SSC and tegmen tympani dehiscence.** A 52-year-old woman with a 1-year history of dizziness, imbalance, and loudness intolerance beginning after head trauma. Coronal CT (A) and T2-weighted MR imaging (B) images demonstrate dehiscence of the SSC (block arrow) and tegmen tympani (dashed arrow). A temporal lobe cephalocele (arrow) prolapses inferiorly into the epitympanic recess through the tegmen tympani defect.
Pressure from the cochlea to the SSC results in diminished organ of Corti activation and conductive hearing loss.

Physiologic and Audiology Testing

Patients with SCD may experience vertigo induced by loud noise (Tullio syndrome), pressure applied to the external auditory canal (Hennebert sign), or the Valsalva maneuver. Auditory manifestations of SSCD may include diminished conductive hearing within the affected ear, autophony (accentuated perception of one’s own heartbeat, breathing, or other self-generated noise), and hyperacusis of bone conducted sounds.7 Vestibular symptoms are more common than audiology manifestations in both SSCD7 and posterior semicircular canal dehiscence (PSCD).8 Objective signs for SCD include vertical and/or torsional nystagmus in the dehiscent semicircular canal (SC) plane induced by loud noise or pressure applied to the external auditory canal. Audiology testing may demonstrate a lowered threshold for vestibular evoked myogenic responses and low-frequency conductive hearing loss.

Clinical and audiometric examinations have variable findings in patients with SCD,7,9 and positive tests are not specific for SCD. At our institution, patients with audiology examination findings reported as consistent with SCD frequently have an intact arcuate eminence on temporal bone CT or nondehiscence pathologies that included vestibular schwannoma, labyrinthine dysplasia, and otosclerosis. SSCD-related conductive hearing loss may mimic that found with otosclerosis. In 1 study of patients with hearing loss clinically suspected as secondary to otosclerosis, SSCD was found on subsequent temporal bone CT in 5.3%.10

Thus, before entertaining possible surgical intervention, otolaryngologists are reliant on imaging to both confirm SSCD and characterize the arcuate eminence severity, size, and location.

Imaging Techniques and Reformations

CT is the current criterion standard for evaluation of SCD. Recent advances in CT detector technology have been leveraged under the guidance of our institution’s physicists to significantly decrease the mAs required for temporal bone CT helical acquisition. Our current multidetector CT scanner (Somatom-Definition Edge; Siemens, Munich, Germany) temporal bone dehiscence protocol uses a single helical acquisition with 0.6-mm collimation, 120 kV, 180 mAs, 1 rotation/s, and a 0.55 pitch. The resulting radiation dose index of 40 mGy is nearly half the American College of Radiology reference dose for a routine adult head CT (75 mGy). Using the thinnest available collimation and helical data reconstruction section thickness is imperative for attaining the optimal high resolution required to differentiate a severely thinned from a frankly dehiscent SSC. When available, 0.5-mm collimation is preferred. Our helical raw data, reconstructed with bone algorithm and a 10-cm field of view in the axial plane creating 0.6 mm thick slices at 0.2 mm intervals, yields 0.2 × 0.2 × 0.6-mm voxels. Multiplanar reformations in the axial, coronal, Stenvers, and Pöschl planes are then created from this axial reconstructed data (Fig 4).

The necessity of Stenvers and Pöschl plane reformations for the evaluation of SCD is controversial. Branstetter et al11 found no advantage to adding Stenvers and Pöschl reformations to conventional axial and coronal reformations for SSCD evaluation. However, in a retrospective review of 581 temporal bone CTs, Cloutier et al12 found that SSCD prevalence decreased from 10% to 4% when Pöschl plane reformations were included in the image analysis. In published studies that evaluated SSCD, most investigators used both Pöschl and Stenvers reformations3,5,13-15 or created these reformations in instances in which axial and coronal images were equivocal.6 Locke and Goh16 found no significant advantage of routine Pöschl reformations for

Fig 3. SSCD perilymph pathophysiology. (A) Sound-induced inward stapes motion at the oval window results in cochlear perilymph fluid wave, which moves from the scala vestibuli to the scala tympani (arrows), releasing energy by outward movement at the round window (B). Nonphysiologic perilymph fluid motion within the SSC (arrowheads) allowed by dehiscence of its roof (dashed arrow) causes vestibular afferent nerve firing, which is perceived as sound-induced vertigo.

Fig 4. Temporal bone reformations for the evaluation of SCD. Axial (A) and coronal (B) reformations parallel and perpendicular to the lateral SC are created from axially reconstructed helical raw data. Pöschl (C) and Stenvers (D) reformations are created with reference to the axial plane (A), bisecting anterior and posterior SSC limbs for Pöschl (solid line) and reconstructing 90° to the anterior and posterior SSC limbs for the Stenvers plane (dashed line).
SSCD evaluation “unless the visualized walls show questionable defects. In these cases, reconstructed images are probably advantageous and should be obtained and reviewed.” Visualization of very thin bone on CT is best accomplished by display perpendicular to the area of interest to minimize partial volume averaging.

In keeping with this tenet, we found that the Stenvers plane, perpendicular to the SSC roof long axis, was the best view for differentiating severely thinned and dehiscent SSC roofs on temporal bone CT. The Stenvers plane is also less susceptible to reformation errors, which may result in “pseudodehiscence” when Pöschl reformations are created oblique to the SSC long axis. Although the Pöschl reformations are typically the first images surveyed for arcuate eminence integrity, we found this plane more susceptible to partial volume averaging, especially when the petrous pyramid had a steep slope. For dehiscence temporal bone CTs, we believed that technologist reconstruction of axial, Stenvers, and Pöschl reformations for all studies both increased radiologist diagnostic confidence and expedited interpretation.

MR imaging, with heavily T2-weighted thin-section volumetric sequences, has been proposed as a possible alternative imaging technique for evaluating SCD.15,17 Perhaps most intriguing is the finding by Browaeys et al15 that thin-section T2-weighted coronal oblique reformations have a 100% negative predictive value for superior or posterior SCD, suggesting that the separation of the hyperintense SC perilymph from the inferior temporal lobe and hyperintense subarachnoid space CSF by intervening hypointense bone excludes dehiscence. Multiplanar reformations may be created from either axial or coronal T2-weighted thin-section volumetric sequences now incorporated within most internal auditory canal MR imaging protocols. Therefore, the potential exists to screen for SCD with MR imaging, which thereby reduces the number of patients who require both MR imaging and CT studies for the evaluation of “dizziness.”

We recently experimented with Stenvers and Pöschl reformations of a coronal thin-section volumetric T1-weighted inversion recovery sequence. Although not formally validated, these images seemed to provide improved discrimination of SC perilymph–bone–subarachnoid CSF/temporal lobe cortex when compared with T2-weighted reformations (Fig 5).

Classification and Localization
Previous studies demonstrated SCD in 0.5%–0.6% and severe thinning of the SSC roof (≤0.1 mm) in 0.6%–1.4% of routine anatomic specimens.2,5 Current CT helical acquisition techniques by using 0.5–0.6-mm collimation may achieve 0.1-mm in-plane spatial resolution.1,4 However, spatial resolution in the z-axis, most pertinent to resolving the SSC bony roof, is limited by the scanners minimal axial reconstruction thickness of 0.5 mm. Therefore, even with optimal CT imaging techniques, including routine Stenvers and Pöschl reformations, severe thinning (≤0.1 mm) of the SSC bony roof may be perceived as complete bone deficiency (dehiscence) due to partial volume averaging.

We previously proposed a SCD classification18 (Fig 6) devised to address the anatomically proved existence of severe SSC roof thinning and current temporal bone CT limitations in differentiating severe thinning from complete dehiscence. This dehiscence classification may be readily applied by using axial, Stenvers, and Pöschl reformations to the SSC or posterior SC (PSC) in which bony roof deficiencies are characterized as the following: grade 0, intact; grade 1, severely thinned; grade 2, dehiscent. In our experience, differentiating grade 1 and grade 2 SC deficiencies is best appreciated for the SSC by using the Stenvers reformations. The axial plane is best for evaluating the PSC deficiencies and is helpful for SSC posterior roof defects along the posterior petrous ridge. By using these reformations, grade 1 deficiencies have SC walls that converge as they contact the overlying middle or posterior fossa cortex, and grade 2
deficiencies have SC walls that are parallel or diverge as they contact the overlying cortex (Fig 6).

The SSCD location may influence whether a middle cranial fossa or a transmastoid approach is used for corrective surgery, depending on the otolaryngologist’s preferred surgical technique. An SSC defect may be localized as involving the anterior or posterior petrous ridge with Pöschl reformations. By using the Pöschl image which best demonstrates the bony

---

**Fig 6. SCD classification: CT images.** (Left column) grade 0, intact SC bony roof; (center column) grade 1, SC roof severe thinning (<0.1 mm) with converging SC walls on the Stenvers view (black arrows); (right column) grade 2, SC roof dehiscence, with parallel or diverging SC walls as they touch the middle or posterior fossa floor in the Stenvers plane (white arrows).

**Fig 7. SSC roof defect localization.** (A) A horizontal line drawn through the SSC equator allows its bony roof to be divided into equal thirds. (B) Proximal and distal SSC roof defect margins (arrows) are localized on Pöschl by cross referencing the defect locations from Stenvers reformations. (C) SSC roof lesion location is designated by which thirds of the SSC roof (anterior, middle or posterior third) is involved. Here, the SSCD involves the anterior third.

**Fig 8. Grade 1 SSCD: middle third.** The middle third of the SSC roof appears thinned (arrow) but is incompletely characterized on axial (A) and Pöschl (B) reformations. The Stenvers reformation (C) reveals converging SSC walls as they contact the middle cranial fossa floor cortex (arrows), consistent with a grade 1 lesion. Stenvers reformations are especially helpful in differentiating grade 1 and 2 lesions when the petrous ridge has a steep slope.
deficiency, the SSC is bisected at its equator and the 180° upper
SSC is divided into 60° equal thirds. The deficiency may then
be referenced as to its location within the anterior, middle
and/or posterior third of the SSC roof (Fig 7).

Although its clinical relevance is unproven, implement-
ing an SSCD classification and localization system into daily
practice is easy and establishes a simple lexicon that more
accurately reflects our current ability to resolve SSC roof
deficiencies given the limitations of CT. Furthermore, a for-
malized classification for evaluating SC roof integrity and
defect location provides a standardized methodology for
the review and reporting of dehiscence temporal bone CT

CT Prevalence and Limitations
Anatomic studies by Carey et al2 and Crovetto et al15 found
SSCD in 0.5% and 0.6% of anatomic specimens, respect-
ively. CT studies of patients with widely varying inner ear
symptoms report SSCD prevalence between 3.6% and
8%13,19; therefore, the prevalence of SSCD on temporal
bone CT may be overestimated by a factor of up to 16.
Previous CT studies did not attempt to discriminate severely
thinned (≥0.1 mm) (grade 1) from intact (grade 0) or com-
pletely dehiscent (grade 2) SSC roofs when evaluating for
SSCD. It is currently unknown if severe thinning and frank
dehiscence of the SSC bony roof distinguished on anatomic
specimens have differing symptoms and surgical results;
however, if these questions are to be critically addressed in
future studies, a more refined methodology for classifying
and localizing SC deficiencies will be required.

In the study of 1000 temporal bone specimens, Carey et
al2 described just 2 sites of SSC roof thinning or dehiscence:
the middle cranial fossa (Figs 8 and 9) and at the superior
petrosal sinus (Fig 10). We found additional cases of SSCD
in which the roof defect occurred along the posterior pe-
trous ridge separate from the superior petrosal sinus (Figs
11 and 12).

The prevalence of PSCD is less than that of SSCD, but its
reported incidence varies greatly within the literature. CT
temporal bone studies have most commonly reported the prevalence of PSCD between 0.3% and 0.8%. A single study found a 4.5% prevalence of PSCD, which nears the prevalence of SSCD. PSCD may be located at the posterior petrous ridge (Fig 13) or the jugular bulb (Fig 14). A PSC roof defect contiguous with a high-riding jugular bulb was found responsible for 70% of PSCD in 1 series.

**Our Experience**

By using the SCD classification detailed above, we reviewed 947 temporal bone CTs referred by the otolaryngology department for possible SSCD. Our study revealed SSC grade 1 deficiencies in 6.4% and grade 2 deficiencies in 5.1% of temporal bones. A prevalence of grade 1 deficiencies equal to or higher than grade 2 SSC roof deficiencies in our patient cohort was similar to that reported in previous anatomic series. Whether severe thinning and complete dehiscence of the SSC roof differ functionally in their association with SCD syndrome has yet to be determined. In our series, both grades 1 and 2 deficiencies involved the middle cranial fossa more commonly than the posterior fossa. Arcuate eminence thinning tends to occur symmetrically, and other investigators found bilateral SSCD in 60% of patients. In our patients with grade 2 SSCD temporal bones, contralateral arcuate eminence grade 2 and grade 1 temporal bone deficiencies were found in 14% and 43%, respectively.

In our patient cohort with vertigo and possible SCD, grade 2 deficiencies of the PSC were found in only 0.2% of patients. PSCD may coexist with SSCD (Fig 12). Although 1 study found coexisting SSCD and PSCD in 35%, we found a coexisting grade 2 PSCD in approximately 10% of our grade 2 SSCD temporal bones.

**Surgical Options**

Individuals without symptoms have a high incidence of SC severe thinning or frank dehiscence by temporal bone CT; therefore, surgical treatments of SCD are most commonly reserved for patients who also manifest debilitating symptoms and supportive objective tests. SSC dehiscence surgeries generally may be divided into SC plugging and SC roof resurfacing, and either technique may be performed from a middle cranial fossa or transmastoid approach. For the SC plugging technique, fascia, bone dust, wax, or hydroxyapatite is used to occlude the SC segment that con-
tains the bony roof deficiency. The plugged SC is then covered with fascia and a bone graft.4,7,9

Most SSC deficiencies characterized on CT imaging occur along the anterior petrous ridge and may be repaired by plugging from a middle cranial fossa approach. However, this approach does not afford direct visualization for plugging SSC deficiencies that occur along the posterior petrous ridge. Alternatively, SCD resurfacing surgery maintains SC membranous labyrinth patency while the SC roof defect is covered with fascia, cortical bone, or cartilage.4,7 At our institution, patients with either thinned (grade 1) or dehiscent (grade 2) SSC roof deficiencies may undergo resurfacing surgery. SSC roof defects along the anterior or posterior petrous ridge are addressed with a transmastoid resurfacing

Fig 14. Grade 2 PSCD: jugular bulb. A grade 2 PSCD with diverging walls (arrows) as it contacts a high jugular bulb on axial (A), coronal (B), and Stenvers (C) views. Careful scrutiny is necessary to avoid overlooking dehiscence in this location.

Fig 15. SSCD postoperative obliteration. A 28-year-old man with bilateral SSCD and persistent vertigo after bone wax plugging of the right dehiscence. Left temporal bone CT Pöschl (A) and Stenvers (B) reformations demonstrate grade 2 SSCD (block arrows) contralateral to the surgically treated right SSCD. Right temporal bone CT Pöschl view (C) reveals ossification within the SSC anterior third (arrow), whereas obliteration of the SSC middle third (dashed arrows) is uncertain. MR imaging T2-weighted Pöschl reformation (D) better demonstrates obliteration of SSC anterior and middle thirds, with loss of normal T2 hyperintense labyrinthine fluid.

Fig 16. SSC roof thickening after cartilage cap resurfacing. A 69-year-old woman with a middle third grade 1 SSCD (arrow) on preoperative CT Pöschl and Stenvers views (A). Increased thickness of the SSC bony roof 36 months after cartilage cap resurfacing is well demonstrated on CT (B), MR imaging T2-weighted (C) and T1-weighted inversion recovery (D) reformations. However, preservation of SSC endolymphatic fluid is only evident on MR imaging (C and D).
technique, with the exception of SSC deficiencies contiguous with the superior petrosal sinus. Both SSC plugging and resurfacing surgeries have demonstrated at least partial improvement in incapacitating SSCD symptoms, although plugging has also been shown to reduce vestibular function in the repaired canal. Hearing impairment related to SCD may improve or worsen after dehiscence surgery.

Either SSCD plugging or resurfacing by a middle cranial fossa approach requires neurosurgical team participation and immediate postoperative intensive care monitoring, although the duration of subsequent inpatient hospitalization may be shorter with the resurfacing technique. Recently, the stereotactic CT-guided placement of conchal or tragal cartilage over the SSCD after elevating the overlying dura through a tegmen minicraniotomy has been described. This technique, which may be performed on an outpatient basis, allows the surgeon to cover SSC roof deficiencies located on either the anterior or the posterior surface of the petrous ridge from a transmastoid approach. The SSCD operative technique and approach used by most surgeons are currently matters of personal preference. Ultimately, a large multicenter prospective trial will be required to prove which surgical technique is superior.

PSCD is surgically treated far less often than is SSCD. Case reports of PSC deficiencies at the jugular bulb treated by transmastoid plugging and resurfacing techniques reported secondary improvement in vestibular symptoms. When superior and posterior SC roof defects coexist, the SSCD is typically surgically repaired first. In most cases, the resulting improvement in vestibular symptoms makes subsequent PSCD surgical repair unnecessary.

Postsurgical Imaging
Imaging is not routinely performed after SCD surgery. CT or MR imaging is typically requested after dehiscence surgery for those patients with persistent vertigo or suspected contralateral dehiscence. If plugged with bone or calcium hydroxyapatite, or if secondary ossification occurs, the occluded SC segment may appear hyperattenuated with respect to the nonobliterated membranous labyrinth on CT. Plugging of the SC is readily demonstrated on T2-weighted thin-section Poschl reformations, where there is loss of usual hyperintense endolymphatic fluid signal intensity within the obliterated SC segment. Endolymphatic fluid T2 hyperintensity should be preserved within the SSC after SSCD cartilage cap resurfacing. In several cases, we saw increased thickness of the SSC bony roof on postoperative temporal bone CT and MR imaging studies after cartilage cap resurfacing. MR imaging thin-section volumetric sequences with Stenvers
and Pöschl reformations is advantageous because it may resolve both postoperative SSC roof thickening and preservation of SC labyrinthine fluid (Fig 17) with a single imaging technique.

Future Considerations

Differentiating severely thinned (≤0.1 mm) from completely dehiscent SSC roofs remains challenging despite using current optimal CT techniques and Stenvers reformations perpendicular to the SSC roof deficiency. The optimal technique for evaluating possible SCD is yet to be determined, and both multidetector and cone-beam CT have been proposed as superior techniques. When using a newer cone-beam CT system, Casselman et al27 found temporal bone cone-beam CT superior for SCD evaluation, resolving severely thinned bony roofs in cases in which multidetector CT images indicated complete dehiscence. However, due to the absence of a grid, cone-beam CT has increased scatter and hence, noise which degrades perceptibility of very thin bone. A recent quantitative and qualitative skull base imaging study that used phantoms and human skulls found multidetector CT superior to cone-beam CT for spatial resolution and image homogeneity.28 Few medical centers in the United States have cone-beam CT routinely available for temporal bone imaging. For most radiologists, evaluating possible SCD multidetector CT will remain the criterion standard imaging technique for years to come; it is hoped that future CT refinements will further improve spatial resolution.

Our proposed system for classifying and localizing SSC dehiscence provides a simple technique for attempting to differentiate severely thinned and completely dehiscent SC bony roofs given current limitations of CT, and in determining the surgical approach for treating the defect. Further studies are warranted to determine if significant clinical and audiometric differences between grade 1 and grade 2 defects exist, and whether or not this grading system has clinical utility in surgical selection or prognostic value for surgical outcomes. Addressing this question is of key clinical importance because anatomic studies have demonstrated the prevalence of SSC severe thinning to be equal to or greater than frank dehiscence.

MR imaging is commonly the initial imaging examination ordered for the evaluation of patients with dizziness, vertigo, and hearing loss. Primary clinical considerations frequently include posterior fossa ischaemia and/or infarction and vestibular schwannoma in addition to SCD. Stenvers and Pöschl reformations created from thin-section T2-weighted volumetric sequences now commonly incorporated within internal auditory canal MR protocols allow preliminary characterization of the SSC roof. Because MR imaging grade 0 SSC roofs have a 100% predictable value for SSCD further imaging with temporal bone CT is unwarranted, avoiding radiation and significant expense that result from a fixed dual MR imaging plus CT algorithm for evaluating such patients (Fig 17). The minority of patients with grade 1 or 2 deficiencies on initial MR imaging (approximately 10%), therefore, would require subsequent temporal bone CT for further SC bony roof characterization (Fig 18). Maximizing the value of each imaging study and minimizing redundant examinations with strategies such as this will become increasingly important in our evolving health care climate in which “quality per cost” is becoming the radiologist’s mantra.

CONCLUSION

Dehiscence of the superior or the posterior SC bony roof may be asymptomatic or may result in a wide range of symptoms, including vertigo, which may be sound and/or pressure induced; hearing loss; and autophony. Severe thinning (≤0.1 mm) or complete dehiscence of the SSC bony roof may potentially be differentiated on temporal bone CT or MR imaging studies that use both Stenvers and Pöschl reformations and by applying a dehiscence classification system. Improved imaging characterization of SC bony roof deficiencies should facilitate selection of optimal surgical candidates, determine the best surgical approach, and provide a foundation for future studies that explore possible functional differences between SC roof severe thinning and complete dehiscence.

REFERENCES

semi-circular canal. *Acta Otolaryngol* 2001;121:68–75. 10.1080/000164801300006308


