Operative Techniques in Otolaryngology 30 (2019) 223–230

Operative Techniques in
Otolaryngology

Endoscopic-assisted surgical repair of superior canal dehiscence using a keyhole middle fossa craniotomy approach

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KEYWORDS
Endoscopic ear surgery;
Superior canal dehiscence;
Superior canal dehiscence syndrome;
Third window phenomenon;
Middle fossa;
Skull base

Superior canal dehiscence (SCD) is a bony defect of the superior semicircular canal that is called SCD syndrome (SCDS) when associated with vestibular and auditory dysfunction. Surgical management of SCD is reserved for patients with intractable auditory and/or vestibular symptoms. As direct visualization of an arcuate eminence defect is most easily achieved from above, the majority of cases use a microscope-assisted middle fossa craniotomy. However, approximately 30% of SCD cases have a medial arcuate eminence defect along a downsloping tegmen. These defects can be difficult to visualize without a large cranial window, drilling down a prominent lateral skull base ridge, and/or prolonged brain retraction. In line with recent development of endoscopic ear surgery, the endoscope has been employed at our institution via a middle fossa craniotomy approach to repair a SCD. We believe that skull base endoscopy is a safe and effective way to identify and repair a medial or blue-lined SCD when used with a middle fossa craniotomy approach. The angled endoscope enhances visualization and transillumination of the SCD and reduces temporal lobe retraction. The following chapter highlights an endoscopic-assisted middle fossa craniotomy repair of SCD.

Introduction

First characterized by Llyod Minor et al in 1998, superior canal dehiscence (SCD) is a bony defect of the superior semicircular canal (SSC) that is called SCD syndrome (SCDS) when associated with vestibular and auditory dysfunction[1]. Classic SCDS symptoms are aural fullness, pulsatile tinnitus, hyperacusis, and autophony.[2,3] The etiology of symptoms of SCD are believed to due to a "third window" phenomenon that decreases the cochlear impedance experienced by the stapes footplate. A reduction in the pressure across the cochlear partition and an increase in bone-conducted, cochlear-evoked potentials may also lead to hearing loss or conductive hyperacusis.[4-8] The most common subtype of SCD seen on high resolution computed tomography (CT) is a defect of the arcuate eminence (95% of cases) with the remaining involving the medial aspect of the SSC in close approximation to the superior petrosal sinus.[9] Surgical management of SCD is reserved for patients with intractable auditory and/or vestibular symptoms as most patients are followed conservatively.

As *direct visualization* of an arcuate eminence defect is most easily achieved from above, the majority of cases performed at the busiest SCD centers use a microscope-

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http://doi.org/10.1016/j.otot.2019.07.008
1043-1810/

assisted middle fossa craniotomy (MFC). However, approximately 30% of SCD cases have a medial arcuate eminence defect along a downsloping tegmen.[9] Consequently, these defects can be difficult to visualize without a large craniotomy, drilling down a prominent lateral skull base ridge, and/or prolonged brain retraction. Finally, the medial or nonampullated end of the defect is the most difficult to visualize and inadequate repair of the entire dehiscence could result in treatment failure or symptom recurrence.

As in the case of most otologic procedures, repair of SCD was developed employing binocular operative microscopy. The use of endoscopes in ear surgery has grown in popularity and provides alternatives for visualization during a SCD repair. Endoscopic technology affords a wide-field view, high resolution, magnification, and the ability to "look around corners". In line with recent development of endoscopic ear surgery, over the past several years, the endoscope has been employed at our institution via a MFC approach to repair SCD. We believe that skull base endoscopy is a safe and effective way to identify and repair a medial or blue-lined SCD when used with a MFC approach. The angled endoscope enhances visualization and transillumination of the SCD and reduces the need for prolonged temporal lobe retraction.

The following chapter highlights an endoscopic-assisted MFC repair of SCD developed at the Massachusetts Eye and Ear. This approach is used for visualization of the entire arcuate eminence defect regardless of bony skull base topography and for unequivocal identification and repair of the dehiscence.

Preoperative evaluation

The diagnosis of SCDS is based on standard otologic workup, which encompasses a comprehensive head and neck exam, audiometric and vestibular testing, including cervical vestibular evoked myogenic potential (cVEMP) testing, and high-resolution temporal bone CT. The differential diagnosis of SCDS includes labyrinthitis, Meniere's disease, BPPV, ototoxicity, perilymphatic fistula, and migraine-associated vertigo. Physical exam and ancillary testing are used to rule out potential causes of auditory and vestibular symptoms.

In addition to complete head and neck exam, physical exam includes otoscopy to rule out and/or middle ear disease and tuning fork evaluation. Weber lateralizes to the symptomatic ear in unilateral SCDS or the worse ear in cases of bilateral SCDS. Dix-Hallpike maneuver may also be performed to exclude benign paroxysmal positional vertigo. Additional physical examination testing may include presentation of intense low frequency sound, such as by using a Barany noise apparatus or positive pressure presented external auditory canal. Intense low frequency sound will lead to an excitatory stimulus in the dehiscent superior canal. Negative pressure presented to the external auditory canal, Valsalva maneuver against closed glottis or jugular compression will generate an inhibitory response of the dehiscent superior canal.

Pure tone audiometry should include bone conduction testing at −5 and −10 dB ("supranormal bone conduction") to evaluate for supranormal bone conduction, particularly at low frequencies such as 125, 250, and 500 Hz. An air-bone gap is commonly seen in SCDS patients. In addition, tympanometry and acoustic reflexes should also be evaluated to determine presence of middle ear disease, such as Eustachian tube dysfunction or ossicular fixation. An absent acoustic reflex suggests ossicular fixation but does not exclude SCD as more than one condition may be present.

cVEMP may be used to determine whether a SCD identified on imaging is associated with a physiologic disturbance of the inner ear. cVEMPs assay the vestibulospinal reflex mediated through the saccule and the inferior vestibular nerve and are evoked by intense low frequency auditory stimuli. In patients with SCDS, cVEMPs may show abnormally low thresholds (and elevated amplitudes although this measure is a less reliable indicator for cVEMP). It is important to note that not all patients with SCDS have abnormally low cVEMP thresholds. Consequently, normal cVEMP testing should not exclude a diagnosis of SCDS.

High-resolution CT of the temporal bone is critical in the diagnosis and surgical planning of SCDS.[9-12] HR-CT may demonstrate a dehiscent area of bone at varying locations along the arc of the SSC: lateral upslope of the SSC, arcuate eminence, medial downslope of the SSC, or the medial nonampullated limb associated with the superior petrosal sinus. As there are variations in thickness of the SCD depending on the precise location, it can be difficult to visualize the extent of the dehiscence or if the canal is dehiscent at all when relying only on coronal plane views. Our standard SCD protocol scan include Stenvers reformats that are perpendicular to the plane of the SSC and Pöschl reformats that are parallel to the SSC and 45° oblique to the coronal plane. Several classification schemes have been proposed to characterize radiographic features associated with SCD. There is still no standardized method of characterizing multiple radiologic findings associated with SCDS in the literature. We developed a radiologic classification system for identification of dehiscence location (Figures 1 and 2) and description of tegmen topography (Table 1) to guide surgical planning.[9] Of note, magnetic resonance imaging of the temporal bones is also routinely obtained to rule out central nervous symptom causes of vestibular symptoms, assess the intracranial anatomy along the skull base, and exclude a middle fossa neoplasm associated with the SCD.[13]

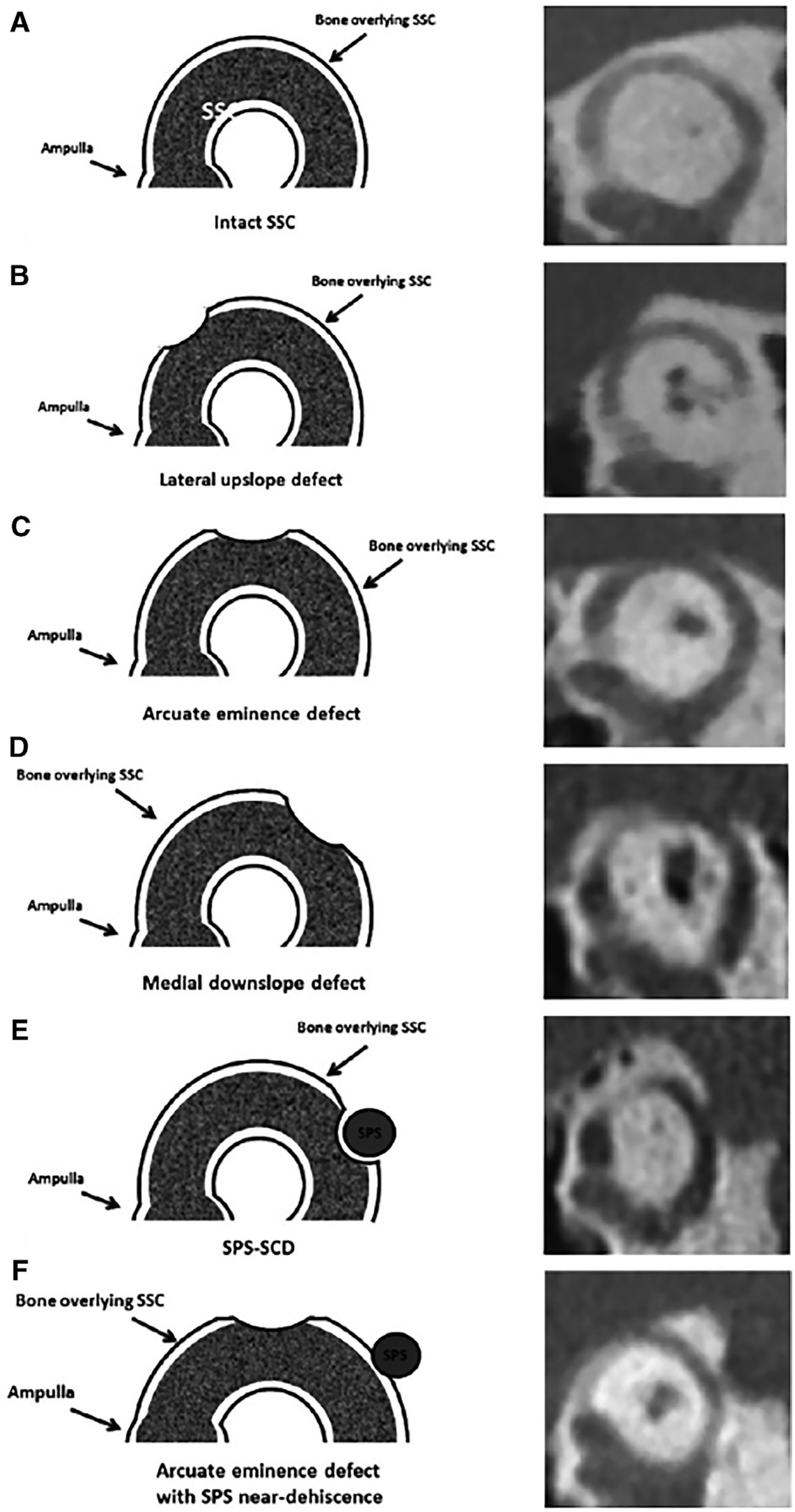

Figure 1 Radiologic classification of patients with superior canal dehiscence based on defect location. Left column: schematic of defect location relative to the ampullated end of the superior canal. Right column: CT images in the plane of Poeschl of corresponding defect location. (A) Intact superior semicircular canal (SSC). (B) Dehiscence on the lateral upslope of the SSC (arcuate eminence). (C) Dehiscence of the top of the arcuate eminence. (D) Dehiscence on the medial downslope of the SSC (arcuate eminence). (E) Superior petrosal sinus-associated SCD (SPS-SCD). (F) Arcuate eminence defect with a "near" dehiscent SSC associated with the superior petrosal sinus.[9] Adapted from Lookabaugh et al., 2014. Otology and Neurotology.

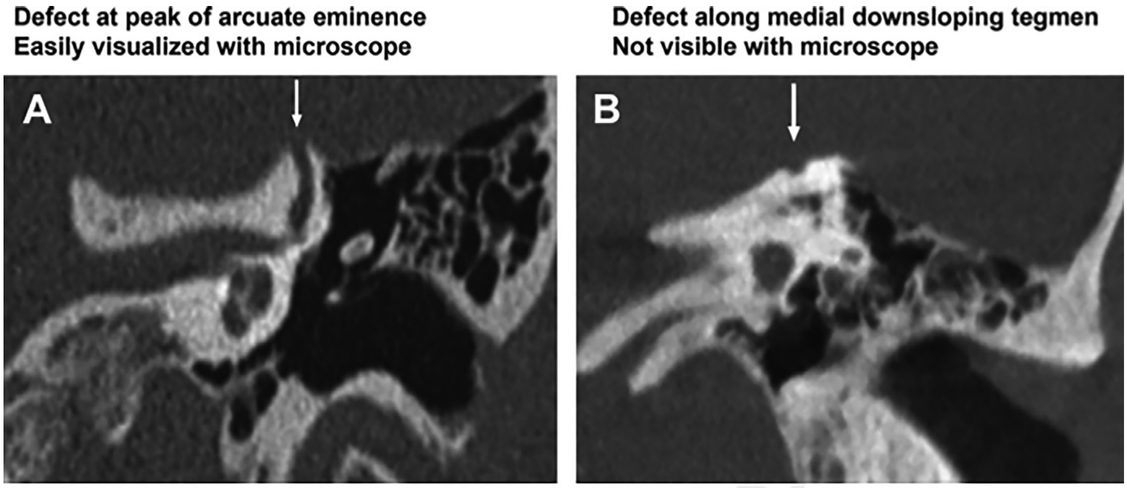

Figure 2 Anatomic location of superior canal dehiscence on preoperative computed tomography scans can help anticipate the need for the endoscope during middle fossa craniotomy repair. High-resolution computed tomography: coronal views of temporal bone. (A) Defect is located at the peak of the arcuate eminence (arrow). Middle fossa craniotomy using the binocular microscope should provide adequate visualization of the entire defect. (B) Defect along the downsloping tegmen medial to the peak of the arcuate eminence (arrow). Microscopy could not visualize any portion of this defect. A 30° endoscope was used to identify and repair the dehiscence in this case.

Table 1 Radiologic classification of patients with superior canal dehiscence: CT characteristics. *Adapted from Lookabaugh et al., 2014. Otology and Neurotology*.[9]

(1) Dehiscence of the superior semicircular canal (SSC).
  a. Intact SSC: clear bone overlying SSC in all slices in all views/reformats.
  b. Near dehiscence: thin bone overlying the SSC without questionable dehiscence OR only clear dehiscence in *1* slice in 1 view/reformat.
  c. Frank dehiscence: clear lack of bone overlying SSC in *at least 2* consecutive slices in at least 1 view/reformat.
(2) Tegmen dehiscence
  a. Lack of bone *without* soft tissue opacification in middle ear cavity.
  b. Lack of bone *with* soft tissue opacification in middle ear cavity; *no* ossicular contact.
  c. Lack of bone *with* soft tissue opacification in middle ear cavity; *with* ossicular contact.
  d. Intact tegmen.
(3) Low-lying tegmen
  In the coronal view:
  a. 75° or greater: flat/NOT low-lying.
  b. 74° or less: low-lying.
(4) Geniculate ganglion dehiscence.
  a. In the coronal view, clear lack of bone over geniculate ganglion on at least 2 consecutive slices.
(5) Superior petrosal sinus-associated superior canal dehiscence (SPS-SCD).
  a. Where SPS can clearly be distinguished by a bony groove in the petrous bone and SPS is juxtaposed to the site of SCD.
(6) Distance between outer table of temporal bone and arcuate eminence.
  a. Approximate location of the arcuate eminence, measured in a straight line from the outer table of temporal bone to the middle, or peak, of the arcuate eminence.

Indications and contraindications

Most SCD patients are asymptomatic and do not necessitate surgical intervention. Conservative treatment includes avoidance of triggers, as well as otologic examination and interval audiometric testing.[14,15] In SCD patients with incapacitating auditory and/or vestibular symptoms, however, surgical repair is a reasonable option.[14,16] Patients that meet the diagnostic criteria for SCDS require documentation of a comprehensive head and neck exam, high resolution CT, comprehensive audiometric testing, and cVEMP testing.

Absolute contraindications for SCD repair include comorbid conditions that would preclude general anesthesia and intracranial pathology that would increase the risk of a lateral skull base approach. Surgery for SCD should be avoided in patients where the bony defect affects the only hearing ear. MFC should be avoided in SCD associated with the superior petrosal sinus or in cases with a severely-downsloping skull base. In these cases, surgical approaches

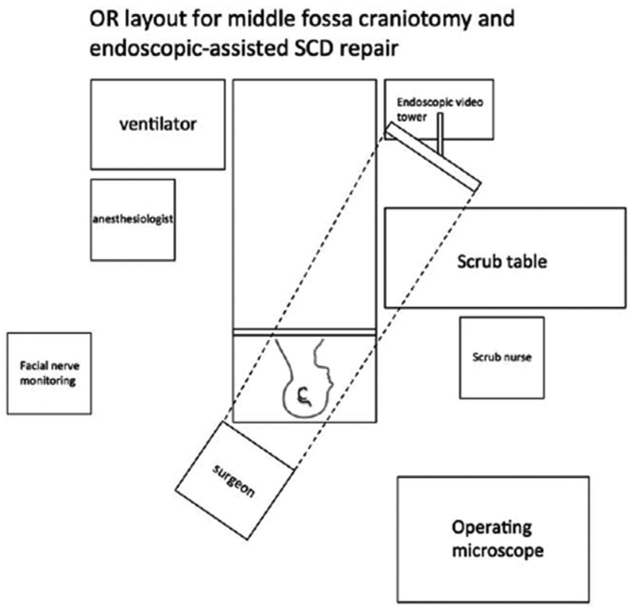

Figure 3 Operating room setup for endoscopic-assisted superior canal dehiscence (SCD) repair on the left ear. The high-definition video tower (or boom-mounted video screen) is placed directly opposite the surgeon for direct line of sight using rigid endoscopy. The scrub nurse is across from the surgeon. The microscope is placed to the right of the patient's head and is brought in as needed. Anesthesia is at the foot of the bed on the same side as the surgeon.[17] Adapted from Carter MS, et al. Laryngoscope, 2014.

may best be done using a transmastoid approach or with angled endoscopy. Additional relative contraindications include vestibular hypofunction in the contralateral ear, migraines, Ménière's disease, or large vestibular aqueduct in the SCD ear. In such cases, conservative management is most reasonable.

Surgical equipment

Equipment needed for endoscopic lateral skull base surgery includes a light source and rigid endoscope coupled to a 3 charge-coupled, high-definition (HD) camera and HD video monitor. The Hopkins rod-lens system comes in a range of diameters, lengths, and angles of view. At our institution, we routinely use a 3 mm diameter, 14 cm Hopkins rod, 30°, for endoscope-assisted lateral skull base surgery. Surgical ergonomics are critical. The HD monitor should be positioned at the foot of the bed, on the side contralateral to the craniotomy (Figure 3). The HD monitor should be placed as close as possible to eye-level to reduce neck strain. A moist gauze should be placed along the craniotomy edge or middle fossa retractor to stabilize the endoscope.

Surgical technique

There are 2 types of repair methods after exposure of the SCD via MFC: (1) "plugging" or "occluding" or (2) "recapping" or "resurfacing." Occluding the canal defect is achieved with bone wax, fascia, bone pate, hydroxyapatite cement, or a combination of 2 or more of these materials. Resurfacing of the defect is done using a bone chip, cartilage, or bone cement. As the defect is very small, achieving a durable resurfacing outcome may be challenging and many of these cases with stable outcomes are likely the result of "gentle" plugging as the materials used for resurfacing settle into the defect. High-resolution T2-weighted MRI scans have shown the lack of fluid signal in the region of cases that have been "resurfaced" suggesting this phenomenon (data not published). Finally, resurfacing approaches have been associated with a higher recurrence rate of symptoms compared to plugging approaches. At our institution, we routinely use the plugging approach.

An overview of the endoscopic-assisted middle fossa approach to SCD repair is as follows. The patient is positioned supine, with the anesthesiologist on the same side as the surgeon, and the scrub team placed opposite the surgeon. Preoperative intravenous broad-spectrum antibiotics, antivirals, and steroids are given. The operating microscope with the binocular teaching head mounted on the side ipsilateral to the surgical ear and endoscope set-up is planned before the patient is brought into the room to allow for the HD monitor to be placed contralateral to the surgical ear and at the foot of the bed, and as close to eye-level as possible.

General anesthesia is routinely performed. At the Massachusetts Eye and Ear, patients are preoperatively given levetiracetam, valacyclovir, vancomycin, ceftazidime, and dexamethasone. A Foley urinary catheter is placed following intubated. At the time of incision, mannitol and Lasix are given. Following intubation and a minimal hair shave, a 6 cm curvilinear incision is drawn, extending from 1 cm posterior and superior to the postauricular skin crease and curving anteriorly along the floor of the middle fossa and superiorly over the temporalis into the hairline anteriorly. Continuous facial nerve monitoring is employed. Next, about 15 cc's of 1% lidocaine with 1:100,000 epinephrine is infiltrated into the planned surgical field. The ear is prepped and draped in the usual sterile fashion.

The skin incision is then made *parallel* to the hair follicles (rather than perpendicular to the skin surface) to reduce the risk of alopecia along the surgical scar. The incision is made approximately 1 cm above the attachment of the pinna superiorly to avoid pain with wearing glasses that fall on the scar line postoperatively (Figure 4). Care is taken to not extend the incision past the anterior hairline. The skin flaps are elevated superficial to the temporalis fascia sharply. Bipolar cautery is used for hemostasis as monopolar cautery is avoided. A temporalis fascia graft is harvested centrally, allowing for a peripheral rim of fascia to facilitate closure (Figure 5).

An anteriorly-based muscle and periosteal flap is designed, carried down to the bone, and elevated anteriorly, as well as inferiorly, to expose the bony external auditory meatus. An inferiorly based temporalis muscle flap provides a pedicled flap for reconstructing the skull base

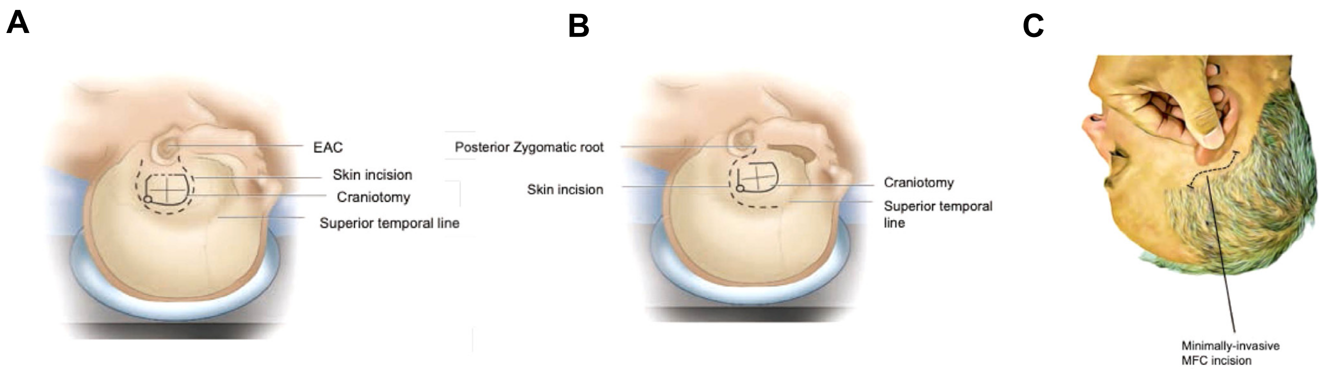

Figure 4 Traditional skin incisions used for middle fossa craniotomy. (A, B) Large C-shaped incision over the temporal skull to allow for a 5 × 5 cm middle fossa craniotomy (reproduced from: Seghrue et al. (2011), *Core Techniques in Operative Neurosurgery. Elsevier Inc. Retrieved 20 August 2017* (C) Illustration of minimally invasive soft-tissue approach to the middle cranial fossa described in this paper.

in large tegmental defects where poor vascularity is a concern, but is associated with greater pain issues postoperatively (Figure 5).

Next, a 2 × 3 cm bone flap is marked and centered on the bony external auditory canal (Figure 5). The bone flap is elevated using both cutting and diamond drill burrs. The dura is gently elevated off of the tegmen mastoideum and tegmen tympani. Careful attention is paid to dissecting the middle fossa dura and expose the lateral skull base from posterior to anterior along the petrous ridge (to initially avoid the geniculate ganglion). More anterior/medial skull base dissection can be performed with a nerve stimulator to avoid direct injury to the facial nerve (the geniculate ganglion is dehiscent in about 50% of patients with SCDS). If the intracranial pressure seems elevated, a controlled durotomy is performed along the lateral middle fossa dura to allow egress of cerebrospinal fluid (this can be closed primarily once the repair is completed).

Once the region of the arcuate eminence is visualized, a middle fossa retractor is placed under direct visualization, using a cottonoid to protect the dura and brain. When the arcuate eminence defect is not seen by standard binocular microscopy, either due to a downsloping tegmen or if there is difficulty visualizing a "blue-lined" or "near" dehiscence, the endoscope is brought into the operating field. At this point, a 3 mm diameter, 14 cm long 0, 30, or 45° endoscope is introduced into the craniotomy and stabilized with the nondominant hand to provide a view of the skull base.

Under endoscopic control, the dura is dissected from the medial aspect of the defect, and a gentle, slow occlusion of the ampullated and nonampullated ends is performed using bone wax. The bone wax is prepared as 1-2 mm spheres and placed under a warming lamp prior to use. The wax is carefully applied using either a Rosen knife or Freer elevator (Figure 6). An assistant may hold a fenestrated suction near the region of the defect to help clear the irrigation fluid and blood while the primary surgeon holds the endoscope and applies the wax. Over-occlusion can result in wax migration to the cupula or vestibule, resulting in vestibular dysfunction.

The temporalis muscle and deep cutaneous layers are closed in an interrupted fashion with 3-0 monofilament synthetic resorbable suture followed by a running locked nylon suture to ensure a watertight skin closure (Figure 5). No drain is placed. A mastoid dressing is applied for 5 days (and changed on postoperative day number 1 to examine the scalp flap and reduce the risk of pressure necrosis from the bandage).

Postoperative care

Patients are monitored in a monitored setting for 24 hours. If there is any concern about efficacy of the repair, a heavily weight T2 MRI of the superior canal may be obtained for confirmation. Headache and vertigo are managed with minimal pain medications to avoid masking neurological change and vestibular suppressants. Intravenous steroids are continued. Once the patient can ambulate and tolerate a diet, they can be discharged (average hospital stay is 2 days) on 60 mg of oral prednisone daily for 2 weeks followed by a 1-week taper. A 2-week course of antivirals may be used for prophylaxis. They are encouraged to ambulate without straining or lifting heavy objects for at least 2 weeks. Vestibular physical therapy consult is initiated during hospitalization for severe dizziness. About 1/3 of patients require outpatient vestibular physical therapy postoperatively. Further hearing loss (1%), cerebrospinal fluid leak (1%), temporal lobe injuries and hemorrhage, facial weakness, meningitis, persistent headaches, and persistent disequilibrium (worse in patients with a migraine history) are all complications of this procedure.

Pearls and pitfalls

- The endoscopic HD video monitor must be placed directly in front of the surgeon and as close to eye-level as possible to optimize surgical ergonomics.

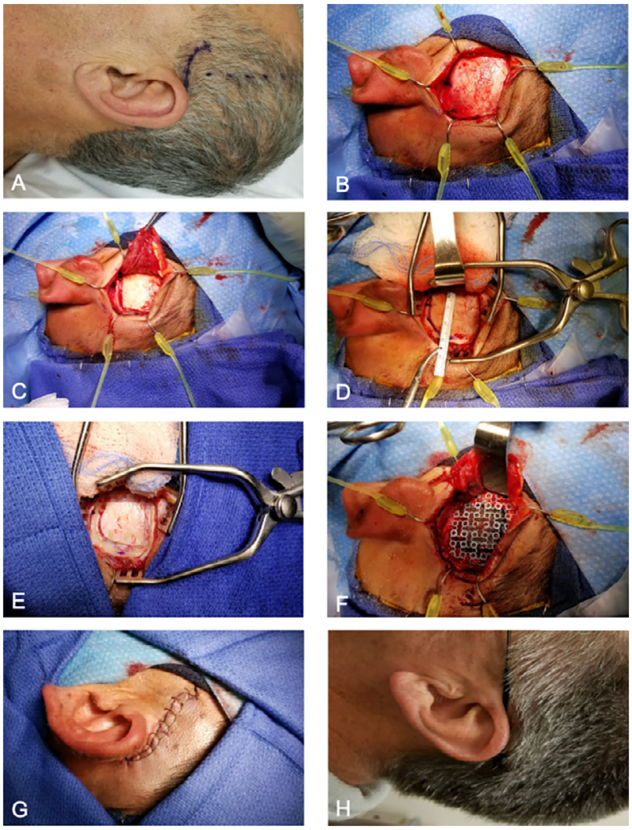

Figure 5 Skin and soft tissue approach during minimally invasive middle fossa craniotomy for superior canal dehiscence. (A, B) Representative image of S-shaped supra-auricular incision planning. Incision is centered on the external auditory meatus (dashed line) and is approximately 5-6 cm in length. Incision and flap elevation is made down to the level of the temporalis fascia. (C) Anteriorly-based temporalis muscle flap is created to achieve exposure of the calvarium. (D, E) Planning and execution of a temporal craniotomy approximately 2×3 cm for extradural dissection of the lateral skull base. (F) Titanium mesh plate cranioplasty. (G) The muscle flap and subcutaneous layers are closed with 3-0 Monocryl sutures followed by a running locked 3-0 nylon suture. A traditional mastoid dressing is applied and retained for 5 days postoperatively. (H) Representative image of healed incision 6 months following MFC for repair of superior semicircular canal dehiscence.

- Poor room setup, with a noneye level HD monitor that is not positioned in front of the operating surgeon to optimize the viewing angle, will make this approach difficult.
- Endoscopy for SCD surgery must be planned preoperatively (and not during surgery) and the OR set up with approval of the surgical team to optimize ergonomics.
- A middle fossa retractor is helpful to retract the dura during skull base endoscopy and 1-handed dissection.
- An arcuate eminence defect along a downsloping tegmen (especially the nonampullated or medial aspect of the defect) can be clearly visualized using angled rigid endoscopy, even when a keyhole MFC is performed.

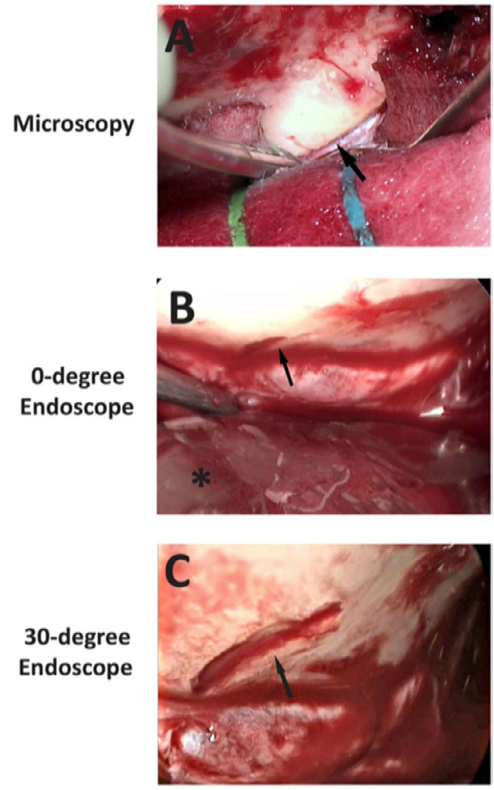

Figure 6 Intraoperative images during superior canal dehiscence (SCD) surgery, left ear. (A) Binocular microscopy in the region of the arcuate eminence (black arrow) and petrous ridge following middle fossa craniotomy. The canal defect was not seen. A middle fossa retractor is used to maintain surgical exposure. (B) In the left ear, improved view of the ampullated half of the superior canal defect using 0º endoscopy (black arrow). (C) In the left ear, both the ampullated and nonampullated ends of the dehiscence are seen with a 3 mm, 30º rigid endoscope, defining the exact limits of this arcuate eminence defect (thin arrow). The endosteum/membranous labyrinth of the inner ear can also be seen within the dehiscence.[17] Adapted from Carter MS, et al. Laryngoscope, 2014.

Acknowledgments

The authors would like to thank Dr. Ruwan Kiringoda and Dr. Osama Tarabichi for review of manuscript and assistance with figures.

Disclosure

The authors reported no proprietary or commercial interest in any product mentioned or concept discussed in this article.

References

1. Minor LB, Solomon D, Zinreich JS, et al: Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. Arch Otolaryngol Head Neck Surg 124:249–258, 1998.
2. Minor LB: Superior canal dehiscence syndrome. Am J Otol 21:9–19, 2000.

3. Minor LB: Clinical manifestations of superior semicircular canal dehiscence. Laryngoscope 115:1717–1727, 2005.
4. Rosowski JJ, Songer JE, Nakajima HH, et al: Clinical, experimental, and theoretical investigations of the effect of superior semicircular canal dehiscence on hearing mechanisms. Otol Neurotol 25:323–332, 2004.
5. Merchant SN, Rosowski JJ: Conductive hearing loss caused by third-window lesions of the inner ear. Otol Neurotol 29:282–289, 2008.
6. Songer JE, Rosowski JJ: A mechano-acoustic model of the effect of superior canal dehiscence on hearing in chinchilla. J Acoust Soc Am 122:943–951, 2007.
7. Songer JE, Rosowski JJ: The effect of superior-canal opening on middle-ear input admittance and air-conducted stapes velocity in chinchilla. J Acoust Soc Am 120:258–269, 2006.
8. Songer JE, Rosowski JJ: The effect of superior canal dehiscence on cochlear potential in response to air-conducted stimuli in chinchilla. Hear Res 210:53–62, 2005.
9. Lookabaugh S, Kelly HR, Carter MS, Niesten ME, McKenna MJ, Curtin H, Lee DJ: Radiologic classification of superior canal dehiscence: implications for surgical repair. Otol Neurotol 36(1):118–125, 2015. doi:10.1097/MAO.0000000000000523.
10. Belden CJ, Weg N, Minor LB, et al: CT evaluation of bone dehiscence of the superior semicircular canal as a cause of sound- and/or pressure-induced vertigo. Radiology 226:337–343, 2003.
11. Cloutier JF, Belair M, Saliba I: Superior semicircular canal dehiscence: Positive predictive value of high-resolution CT scanning. Eur Arch Otorhinolaryngol 265:1455–1460, 2008.
12. Nadaraja GS, Gurgel RK, Fischbein NJ: Radiographic evaluation of the tegmen in patients with superior semicircular canal dehiscence. Otol Neurotol 33:1245–1250, 2012.
13. Crane BT, Minor LB, Carey JP: Three-dimensional computed tomography of superior canal dehiscence syndrome. Otol Neurotol 29:699–705, 2008.
14. Minor LB: Superior canal dehiscence syndrome. Am J Otol 21:9–19, 2000.
15. Brantberg K, Bergenius J, Mendel L, et al: Symptoms, findings and treatment in patients with dehiscence of the superior semicircular canal. Acta Otolaryngol 121:68–75, 2001.
16. Vlastarakos PV, Proikas K, Tavoulari E, et al: Efficacy assessment and complications of surgical management for superior semicircular canal dehiscence: A meta-analysis of published interventional studies. Eur Arch Otorhinolaryngol 266:177–186, 2009.
17. Carter MS, Lookabaugh S, Lee DJ: Endoscopic-assisted repair of superior canal dehiscence syndrome. Laryngoscope 124(6):1464–1468, 2014. doi:10.1002/lary.24523.