Intracranial Management of Perineural Spread in the Trigeminal Nerve

Michael J. Redmond^{1,2,3} Benedict J. Panizza^{1,3,4}

¹Queensland Skull Base Unit, Princess Alexandra Hospital, Brisbane, Queensland, Australia

²Department of Neurosurgery, Lady Cilento Children's Hospital, Brisbane, Queensland, Australia

³Department of Surgery, School of Medicine, University of Queensland, Brisbane, Queensland, Australia

⁴Department of Otolaryngology, Head and Neck Surgery, Princess Alexandra Hospital, Brisbane, Australia

Address for correspondence Michael J. Redmond, FRACS, Lady Cilento Children's Hospital, 501 Stanley Street South Brisbane, QLD, Australia 4101 (e-mail: Michael.Redmond@health.qld.gov.au).

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Abstract

Since the mid-1960s surgeons have attempted to cure intracranial perineural spread (PNS) of cutaneous malignancies. Untreated patients with trigeminal PNS die from brainstem invasion and leptomeningeal disease. It was understood that resection with clear margins was potentially curative, but early surgical attempts were unsuccessful. The prevailing wisdom considered that this surgery failed to improve the results achieved with radiation therapy alone and was associated with high morbidity. However, with improved imaging, surgical equipment, and better understanding of cavernous sinus (CS) anatomy and access, contemporary surgeons can improve outcomes for this disease. The aim of this paper is to describe a technique to access the interdural compartment of the CS and treat PNS of cutaneous squamous cell carcinoma (cSCC) in the intracranial trigeminal nerve and ganglion. It is based on the experience of the Queensland Skull Base Unit, Australia in managing PNS of cutaneous squamous cell carcinoma of the head and neck (cSCCHN).

Keywords

- ▶ perineural spread
- ▶ Meckel cave
- ▶ cavernous sinus
- ▶ interdural surgery

Introduction

Ballantyne et al in 1963 wrote that early recognition and interruption of intracranial perineural spread (PNS) of head and neck cancer in the peripheral nerves might avoid a “distressing sequence of events.”¹ In their 80 cases from a variety of tissues, 3 cases of cutaneous squamous cell carcinoma (cSCC) with intracranial extension were treated with resection of the trigeminal ganglion (TG). Their case 2 in 1956 was a 45-year-old man diagnosed with symptomatic PNS of cSCC in the maxillary nerve. Initial treatment was maxillary resection and orbital exenteration with a later craniotomy and removal of the involved TG. They commented that cure was a possibility if the tumor can be diagnosed and interrupted before intracranial spread and that “...efforts to arrest the spread of cancers which have gained access to the central nervous system by way of spread along cranial nerves have

been unsuccessful.”¹ It is notable that these procedures were performed 60 years ago when optimal radiologic examination was tomography and surgeons lacked the advantages offered by modern drills, retraction, magnification, and illumination. Two years later, Parkinson published his report of an exploration of the cavernous sinus (CS),² which commenced “the modern era in cavernous sinus surgery.”³ Goepfert et al in 1984 (from the same institute as Ballantyne et al) published their experience with PNS between January 1970 and December 1979 in 72 patients with cSCC.⁴ They reported six patients who underwent “temporal craniectomy and exploration of the intracranial nerve segments and trigeminal ganglionectomy.”⁴ Two were cancer free between 3 and 11 years postsurgery and they observed that “The indications for exploration of the middle fossa of the intracranial portion of the trigeminal nerve deserve further study.”⁴ Since then, there have been only small series.⁵ Trobe et al in 1982

reported one case of “unresectable gasserian ganglion tumour” and commented that “If the cranial nerve is involved up to the point of exit from the basal foramina of the skull, then radical surgery is not indicated.”⁶ Bourne, in his 1980 review of the Queensland, Australia experience with 13 cases of PNS of cutaneous squamous cell carcinoma of the head and neck (cSCCHN), commented “...once spread has occurred to the cranium, removal of the nerve is not indicated.”⁷

Much has changed since these opinions were expressed. The anatomy of the parasellar area and the lateral wall of the CS have been elaborated.^{8–16} Since Parkinson's work in the 1960s, research has enabled improved surgical access of the CS, the lateral wall of the CS and Meckel's cave (MC).^{12,17–21} Surgical approaches and variations thereof have been codified for the various parasellar regions and pathologies.²⁰ Without modern imaging, pioneer surgeons made difficult treatment decisions based on sometimes “bizarre” clinical findings.⁷ It is now possible for magnetic resonance imaging (MRI) to identify the proximal extent of PNS and whether it remains confined within the nerve, permitting more rational surgical decision making.^{22,23} Wound closure and security have been enhanced by use of free flaps. Modern surgical drills, microscopes, navigation systems, and instrumentation give aid denied to our forebears. Understandably, surgeons are reconsidering surgical interruption of the intracranial spread of perineural cutaneous malignancy⁵ but lack evidence-based treatment guidelines.²⁴

The term “skip lesion” is used by some investigators to describe apparent gaps in the continuity of PNS seen on histologic examination^{5,25–28} and are said to be common.^{29,30} The surgical implication is that, although the intraoperative histopathology of a nerve with PNS is clear of tumor, the tumor may have “skipped” proximally making surgery futile. Rodin et al,³¹ (colleagues of Larson et al³² who found that there were no lymphatics in the endoneurium) examined surgical prostatectomy samples and tumor-inoculated rodent nerve samples. As they saw no “skip areas of tumour growth” in either sample, they concluded that, if perineural lymphatics were present, skip metastases were conceivable, but as tumor spread is actually through the perineural space, it was unlikely.³¹ Panizza et al reviewed 51 operative specimens with PNS of cSCCHN finding no evidence of skip lesions and concluded that the concept should be dismissed in “deciding how far beyond a radiologically or histologically involved nerve should be treated.”³³ Though this view is not universally accepted, it is considered to be a sufficient justification to proceed with surgery with curative intent if the disease looks resectable with current MRI neurographic imaging.

PNS can extend significant distances longitudinally without circumferential outbreak through the perineurium and epineurium, which act as a barrier.^{1,27,33,34} It is known that extraneural outbreaks from trigeminal nerves can result in tumor masses in the superior orbital fissure (SOF) or CS,³⁵ and these outbreaks are largely identifiable with MRI.³⁶ Consequently, when preoperative MRI depicts disease, the epineurium/dura is considered to be a margin for resection.³³

A multidisciplinary team with otolaryngologic and neurosurgical skull base surgeons, plastic reconstructive surgeons,

radiologists, and radiation oncologists collaborates in treatment decision making. Intensity-modulated radiation therapy has shown improved outcomes in a related disease and may prove beneficial with PNS.³⁷ Published outcomes^{38,39} suggest that survival is improved with surgical treatment of PNS up to the TG when supplemented with radiation therapy, without significant operative mortality and morbidity.

Surgical Anatomy

Surgical proficiency in the parasellar region requires familiarity with the complex anatomy, particularly the dural layers.^{13,40} When describing the two layers of the dura, the terms *meningeal* or *dura propria* refer to the layer proximate to the cerebral cortex, the other being the endosteal or pericranial layer. The lateral wall of the CS consists of two layers: the outer of which is the *dura propria* and the inner layer is derived from endosteal layer of dura.¹¹ As the two-layered dura of the middle fossa floor reaches the lateral edge of the CS, the *dura propria* forms the outer layer of the lateral wall of the CS. At this point the endosteal layer divides, with the more superficial leaf forming the inner layer of the lateral wall and the other forming the floor of the CS.¹¹ The inner layer of the lateral wall can also be conceptualized as the oculomotor nerve (cranial nerve [CN] III), trochlear nerve (CN IV), and the ophthalmic division (V1) of the trigeminal nerve (CN V) ensheathed with the endosteal layer¹⁶ and having a “membrane of reticular texture” between them.¹⁴ The abducens nerve (CN VI) courses deep to the inner wall lateral to the internal carotid artery. It is stationed inferiorly in the posterior part of the CS, moving more superiorly as it approaches the SOF. The meningeal layer of the lateral CS wall can be separated from the inner endosteal layer that merges with the outer dura of MC.¹⁶ As the endosteal dura communicates with the extracranial pericranium at all skull base foraminae,¹⁵ an extradural approach requires that the endosteal layer be divided at those foramina included in the approach, notably at the SOF, foramen rotundum, and foramen ovale, with sharp division of the attachment more likely to correctly access the surgical plane than blunt.^{21,41,42}

As cranial nerves pass through the *dura propria* of the skull base, they receive dural sheaths and subarachnoid cisterns of varying size.⁴³ MC is an extension of posterior fossa arachnoid, bounded by dural outpouching that accompanies the trigeminal nerve into the floor of the middle fossa.^{8,15,43} The trigeminal cistern ends anteriorly where the arachnoid and *dura propria* of MC adhere to the TG and it does not extend into the trigeminal divisions.⁸ Trigeminal ganglionectomy necessitates opening the trigeminal cistern in MC, increasing the risk of postoperative cerebrospinal fluid (CSF) fistula, which is further discussed in this article. MC overlies the posterolateral aspect of the CS in its upper one-third and the petrolingual ligament and internal carotid artery laterally.¹¹

The venous element of the CS consists of vascular sinuses and veins, and though surgeon anatomists contest the proportions and disposition of these structures,^{11,44,45} the result for surgeons is unchanged. Important to surgeons is the 40% chance of deficiency in the inner lateral wall¹⁴ and a “rather

large vein" that can cross both layers of the lateral wall to enter the CS between V1 and the trigeminal maxillary division (V2).⁴¹ The pericavernous venous plexuses (PCVP) are paired dural venous structures abutting and communicating with the lateral edge of the respective CS, accompanying V2 and the trigeminal mandibular division (V3) and passing through the skull base foramina to anastomose with the infratemporal pterygoid venous plexus vessels.^{11,46} The most clinically relevant is that which accompanies the mandibular nerve through foramen ovale, but they also communicate through foramen rotundum, foramen lacerum, and the foramen of Vesalius.⁴⁶

At the anterior limit of the CS, the inner endosteal layer of the lateral wall continues through the SOF as periorbita and separates from the outer dura propria layer of the lateral wall. This occurs at the lateral end of the SOF and the lateral point of fusion of these two layers is the meningo-orbital fold.⁴² At this point, surgical access to the interdural space is possible with the anterolateral extradural approach. The lateral extradural approach is also used to access the interdural space by elevating the dura propria from the posterolateral CS and MC.

Surgical Planning

Surgery to the lateral sellar compartment developed slowly due to the complex regional anatomy, risk of damage to the internal carotid artery and cranial nerves, and of "voluminous haemorrhage."^{2,18} Browder pioneered CS surgery in 1937 followed by Parkinson in the early 1960s.^{2,47} Parkinson, informed by 200 autopsy examinations of the cavernous carotid artery, performed a lateral intradural approach to occlude a carotid-cavernous fistula.² This venture into "the last area of uncharted territory in neurosurgery"³ stimulated anatomic dissections to clarify the complex regional anatomy and to improve surgical access. Sekhar et al proposed a classification of the approaches to the CS but conceded that "none of the operative approaches provided optimal exposure of all regions of the CS. A combination of the superior and lateral approaches provided adequate exposure of all spaces of the CS."¹⁹ This is in accord with Dolenc's commentary on Muto et al:

When splitting the dura by the so-called anterolateral interdural approach it is necessary to go also further around and to come to the petrous bone to peel the dura away from the anterior surface of the petrous bone as this is an integral part of the exposure of the gasserian ganglion from all the aspects: anterior, lateral and posterior.⁴⁸

Chotai et al reviewed the approaches to the CS commenting that there are "several approaches and a plethora of modifications for lesions involving the middle fossa and CS.... The sheer number of reports has led to much confusion in the literature in terminology."²⁰ They suggested the following simplified classification: (1) anterolateral extradural approach, (2) anterolateral intradural approach, (3) lateral extradural approach, and (4) lateral intradural approach.²⁰ el-Kalliny et al classified tumors of the CS into the following:

type 1, intracavernous; type 2, interdural; and type 3, invasive.⁴⁹ With PNS of cSCC to the inner lateral wall of the CS, the disease is usually limited to the nerves and conforms to el-Kalliny type 2. The exceptions are those cases where PNS has escaped the epineurium invading and filling the CS, and in these cases it is felt that the dural barriers should be left intact to allow for targeted radiotherapy. The preferred surgical approach to intraneural PNS of the CN V and TG is the interdural approach, derived from Dolenc's approach to trigeminal neurinomas, combined with a lateral extradural approach.¹⁷ This extradural technique gives adequate access to CS and MC while limiting temporal lobe damage.

In treating PNS of the intracranial CN V, MRI provides an estimate of the proximal extent of PNS to inform surgical planning.^{22,23} This proximal advance of PNS in the CN V is staged using a zonal classification. Zone 1 describes spread proximally to the respective skull base foramen. Zone 2 disease extends from the skull base foramen to the TG. Zone 3 disease is proximal to the ganglion.²⁶ Patients with zone 1 disease are referred for subcranial resection to the skull base foramen with curative intent followed by radiation therapy.³⁸ Patients with intraneural zone 2 disease are referred for en bloc resection of the extra- and intracranial disease with curative intent followed by radiation therapy. Patients with PNS in zone 3 and those with zone 2 gross extraneural disease in the CS are best not operated on and are treated with radiation therapy. Patients diagnosed on MRI with zone 2 intraneural disease not extending to the TG may be found at surgery to have unexpected infiltration of the TG and presurgical planning should include this contingency. A lateral extradural approach gives excellent access to V2, V3 and MC but less so to V1 or the SOF.¹¹ The anterolateral extradural approach gives good access to the CS roof and anterolateral wall and V1 but less so to MC.²⁰ Consequently, an exposure combining anterolateral extradural and lateral extradural approaches is preferred.^{17,19,21,48} If the clinical and radiologic assessment confirm PNS restricted to V2 or V3 and not involving the ganglion, a lateral extradural approach may suffice; however, if the intraoperative histopathology shows positive margins, invasion may extend beyond the permitted surgical access. Although orbitozygomatic craniotomy has been used, it is rarely necessary, even in cases requiring orbital exenteration. The zygomatic arch may be removed if access to the posterosuperior CS is necessary but is not routinely performed.⁴⁸ Surgeons contemplating this exposure are encouraged to become conversant with it in the anatomy laboratory.^{41,50} Endoscopic endonasal surgery may be an alternative approach and is considered elsewhere in this volume.

Surgical Technique

The patient is positioned supine with the head elevated and neck extended and rotated 40 degrees to the nonsurgical side in a pinion head frame. As sequential craniotomy, orbital/facial resection, and free-flap reconstruction are required, the surgical table should be adjustable in pitch and roll. A free-flap donor site, if required, is prepared and draped at the outset. Cranial nerve monitoring has been advocated^{42,51} but has not been universally adopted. Neuronavigation is not

essential as the surgeon refers to anatomical features for situational awareness. The surgical exposure uses a conventional frontotemporal preparation and incision. A pterional free-flap craniotomy is performed extending to the root of the zygoma low in the middle fossa permitting a lateral extradural exposure.^{41,48} The anterior burr hole is based on McCarty's point, 5 mm behind and 7 mm above the frontozygomatic suture.⁵² By directing the drill in a posterosuperior direction, the floor of the anterior fossa is reached. Accurate location of the second burr hole in the temporal squama permits sufficient lateral access yet it avoids violation of the pneumatized portion of the temporal bone, a risk for CSF fistula. This point is as low as possible in the temporal squama near the attachment of the zygomatic process but anterior to the attachment of the most posterior temporalis muscle fibers (→**Fig. 1**). Options to minimize postsurgical temporal recession are described by Dolenc et al, with alternative burr hole location and osteoplastic craniotomy.⁴¹ As the median age of patients with clinical PNS is 66 years,⁵³ the dura is often adherent to the cranial vault and the risks of inadvertent durotomy and CSF fistula increased. This risk may be reduced by using the drill rather than the craniotome to raise the bone flap. The procedure is completely extradural⁴¹ and dissection is performed with a rigid sharp instrument⁴² such as a Freer septum elevator (V Mueller, San Diego, California, United States). Under the operating microscope, the dura is raised from the lesser and greater sphenoid wings in the anterior and middle fossae. The lateral sphenoid wing is drilled using a diamond tip, blue lining the orbit to the lateral point of the SOF, averaging 30.7 mm (range 21.2–35.7) from McCarty's point. At the lateral end of the SOF, the thick lesser wing and the eggshell-like greater wing are removed to expose the anterior and posterior aspects of the SOF. At the lateral point of the SOF is the meningo-orbital fold, which marks the

Fig. 1 Right pterional craniotomy showing the location of the burr holes. Note the anterior burr hole at McCarty point, flush with the anterior cranial fossa floor. The rear burr hole is above the most posterior fibers of temporalis, which course beneath the zygomatic arch. (Cadaver dissection.)

Fig. 2 After removal of the right greater and lesser wings of the sphenoid from around the lateral end of superior orbital fissure, the meningo-orbital fold is displayed, with tips of forceps on either side. (Cadaver dissection.) FL, frontal lobe; MOF, meningo-orbital fold; TL, temporal lobe.

conjunction of the periorbita with the two dural layers of the middle fossa and is the portal to the interdural plane (→**Fig. 2**). This is divided using scissors with the curve posterior.^{11,17,41,42} If the meningo-orbital fold is divided too posterior, the middle fossa subdural space may be entered and, if too anterior, the SOF is entered where the lacrimal and other nerves are vulnerable to injury.^{17,41,54} The anterior clinoid process (ACP) is not usually removed as this entails time and risk to the optic nerve and internal carotid artery while not contributing to the exposure essential for the task at hand. In fact, the retained ACP has a useful role, providing counter pressure to the dissector when exposing the upper lateral wall of the CS and helping to develop and maintain the interdural surgical plane (→**Fig. 3**). The foramen rotundum averages 47.6 mm (range 36–61.5) from McCarty's point in a straight line and is inferior to the SOF. To elevate the meningeal dura off V2, the endosteal layer that follows the nerve through the foramen is divided sharply. While the interdural plane may now be extended posterior to display the entire interdural space, it can be helpful to begin the lateral extradural approach and later join the anterior and lateral extradural tunnels, especially if confounding issues such as PCVP ooze or indecision about the surgical plane arise. The lateral extradural approach displays the middle meningeal artery in a bony groove that is followed medially to the foramen spinosum (→**Fig. 4**). The artery is cauterized and divided preserving, if possible, the branch to the greater superficial

Fig. 3 The right interdural plane is developed. The anterior clinoid process (ACP) is exposed showing the access to the upper and anterior CS with this approach. (Cadaver dissection.) FL, frontal lobe; TL, temporal lobe.

Fig. 4 The commencement of the right lateral extradural approach with the dissector tracing the middle meningeal artery to the foramen spinosum. (Cadaver dissection.) MMA, middle meningeal artery.

petrosal nerve (GSPN).^{42,55} The foramen ovale and V3 are 4 mm anteromedial to foramen spinosum.¹² The hiatus fallopii of the GSPN is 9.9 mm posteromedial to foramen spinosum.⁵⁵ It is suggested that the GSPN be deliberately divided to prevent facial nerve palsy from traction on the geniculate ganglion.⁵⁰ The GSPN can be inadvertently divided in the course of this exposure resulting in neurotrophic keratitis,⁵⁵ so others strive to preserve it.⁴¹ Removal of the GSPN is appropriate if there is suspicion of PNS from the pterygopalatine ganglion or from the geniculate ganglion. After dividing the endosteal layer at the foramen, the plane between the dura and V3 is developed to display MC. The

entire medial middle fossa is now accessed by joining the anterior and lateral exposures (►Fig. 5). Maintaining the dissection within the interdural plane may be difficult especially with adhesions between the layers at the junction of V2 and V3 particularly if venous bleeding from the PCVP is abundant. The inner layer of the lateral wall of the CS is now on display and planned resection can proceed (►Fig. 6).

Before commencing surgery and informed by clinical and MRI findings, the surgical team determines the likely extent of the trigeminal resection. There are two typical scenarios. More commonly, as V2 and V3 PNS are more frequent than V1 PNS,³³ the V2 and V3 nerves and possibly the respective portion of the TG will be considered for resection. If V3 appears on MRI to be involved to the TG, V3, V2, and the maxillomandibular portion of the TG are also slated for removal with clear surgical margins to prevent retrograde PNS in V2. Similarly, if V2 and potentially the TG appear involved on MRI, a similar surgical staging back to the TG is necessary with potential resection of V3 as well. Where there is no preoperative evidence of V1 involvement, the ophthalmic nerve and the ophthalmic component of the TG and sensory root are spared to avoid neurotrophic keratitis (though clinically this has not been problematic³⁹). The ophthalmic ganglion cells are limited to the superomedial region of the ganglion.⁵⁶ When dividing the TG between the ophthalmic and maxillomandibular areas, there are no anatomical landmarks apart from the lateral edge of V1 at the TG. To preserve corneal sensation when treating trigeminal neuralgia (TN), Tiffany (1894) described the resection of V2, V3, and the maxillomandibular TG while sparing V1 and ophthalmic portion of the TG⁵⁷ (►Fig. 7). Dooley and ophthalmic portion of the TG⁵⁷ (►Fig. 7). Dooley and Browder found while sectioning the lateral two-thirds of the trigeminal sensory root for TN preserved corneal sensation, sectioning three-fourths jeopardized corneal sensation.⁵⁷ If, despite the resection of V2, V3, and the maxillomandibular TG, the surgical margin is not clear of tumor, it is necessary to remove the whole TG and the sensory root up to the porus trigeminus. Although pursuit of involved sensory root to the brainstem has been reported,⁵ it is avoided if there is visual evidence of extraneural disease as this is believed to possibly result in CSF contamination and leptomeningeal disease. If intraneural disease remains in the trigeminal root proximal to the porus trigeminus, an option is to pursue it from the middle fossa, (►Fig. 8) but the preferred alternatives are radiation therapy, or staged retrosigmoid removal of the diseased trigeminal root plus radiation therapy.

The less common but more problematic second scenario arises when V1 and orbit are involved. If the intracranial V1 disease is shown to be extraneural on MRI, filling the CS, intracranial surgery is not justified. V1 PNS approximately 1 to 2 cm beyond the supraorbital foramen necessitates orbital exenteration, and if there is MRI evidence of V1 intraneural zone 2 disease, consideration is given to CS clearance with internal carotid artery sparing for which exposure of the upper CS and ACP is necessary. This procedure involves initial mobilization of the orbital contents with intraorbital division of the optic nerve and ophthalmic artery. After surgical

Fig. 5 Schematic drawings of the right middle cranial fossa, which demonstrate the surgical access to the interdural space via the anterior extradural (A and B) and lateral extradural (C and D) approaches. (A and B) An oblique axial section along a line through the right superior orbital fissure illustrating the access to the interdural space with the anterior extradural approach. At the lateral edge of the superior orbital fissure, the periorbita joins the sphenoidal pericranial layer and must be divided to permit access to the interdural plane. (C and D) Oblique coronal section from the porus trigeminus to the foramen ovale illustrating the access to the interdural space with the lateral extradural approach. Drawings C and D are adapted with permission of Mayfield Clinic from Youssef S, Kim E-Y, Khaled AMA, et al. The subtemporal interdural approach to dumbbell-shaped trigeminal schwannomas: cadaveric prosection. *Neurosurgery* 2006;59(4 Suppl 2):270–277.⁴⁰

Fig. 5 (Continued)

Fig. 6 Exposure of the right interdural space is complete. (Cadaver dissection.) GG, gasserian ganglion; SOF, superior orbital fissure.

exposure of MC and CS, a clear surgical margin is obtained at the sensory root or ganglion, which is then mobilized with the CS and reflected anteriorly. The meningo-hypophyseal trunk is divided and en bloc removal of the CS, CN V complex, and orbital contents is performed (→Figs. 9 and 10).

Lumbar drainage of CSF is not usual practice as both the CSF and intact temporal dura are protective against retraction injury to the temporal lobe.^{20,41,42} Fixed retraction of the temporal lobe is also avoided, and when necessary, intermittent handheld retraction by the surgical assistant or stay sutures on the temporal dura are used.^{17,41} Despite these precautions, anterior temporal encephalomalacia may be a

Fig. 7 Tiffany's operation for trigeminal neuralgia. The hatched area describes the portion of the trigeminal root, ganglion, and divisions removed totally. (After Dooley and Browder.⁵⁷)

Fig. 8 A cadaver dissection study exposing the right trigeminal root in the posterior fossa with division of the tentorium and petrous apicectomy. GG, gasserian ganglion; PA, petrous bone with apicectomy; PT, pars triangularis of trigeminal nerve.

late result, though with minimal clinical consequence, especially after radiation therapy to the parasellar area.

Parkinson's description of the hemorrhage from the CS as "voluminous hemorrhagic flooding" may have caused even sanguine neurosurgeons to pause.² However, the bleeding in his sentinel case was from a carotid cavernous fistula for which open surgery is now obsolete. No instances of internal carotid artery injury have occurred in the series of cSCCHN cases, but it remains a low percentage risk. The usual source of hemorrhage with interdural surgery is venous hemorrhage from the CS and PCVP.¹¹ Venous blood loss can be reduced by elevating the head when positioning the patient before surgery. CS hemorrhage occurs when the dura propria is dissected from the lateral wall inner layer, which is frequently deficient¹⁴ and also from those venous sinuses that communicate with the CS during CS resection. These bleeding points are managed by packing the CS and its tributaries with small pieces of gelatin sponge^{17,42} soaked in human recombinant thrombin. It is important to use a blunt-tipped microbayonet forceps at this time as sharp-tipped forceps may injure the carotid artery or nerves and to be patient. An alternative haemostatic is gelatin matrix and human-derived thrombin (FLOSEAL, Baxter Healthcare, Fremont, California, United States) though the manufacturer advises against intravascular injection. Bleeding from the PCVP can be as problematic as that from the CS proper, especially at foramen ovale.¹¹ If the PCVP is damaged during elevation of the dura, venous ooze is difficult to control with bipolar coagulation so packing with gelatin sponge and thrombin is useful though hemorrhage can resume if the packing is dislodged.

In published reports of these procedures, perioperative deaths have been avoided and the recorded complications of extradural hematoma, CSF fistula, wound infection, and deep venous thromboses are uncommon, with no epileptic seizures reported.^{38,39} The cranial nerves in the CS are usually protected by the inner lateral wall and injury to them is pathology specific.^{3,17} Resection of the entire TG has not been

Fig. 9 Surgical specimen from an en bloc resection of the right CN V from porus trigeminus, CS, and orbital contents. The dark area at the superior orbital fissure (SOF) is ink. TR, trigeminal root; V2, maxillary division; V3, mandibular division.

Fig. 10 A still photograph taken from an intraoperative microscope video. It shows the right parasellar space and cavernous carotid artery (CC) following a resection of the specimen in shown in ►Fig. 9.

associated with ophthalmic complications.³⁹ Unintended ocular cranial nerve injury with the interdural approach for trigeminal PNS³⁸ is rare and is the subject of current review. The most vulnerable nerve is CN VI as it passes lateral to the internal carotid artery where it can be damaged if the inner lateral wall is breached during dissection. Overzealous packing of the CS with gelatin sponge to control bleeding may damage CN VI by compression. No attempt has been made to preserve the trigeminal motor root.

CSF fistula is possible when CSF from the opened trigeminal cistern or optic nerve stump can exit through opened skull base air sinuses. Locations of concern are the ACP, anterior temporal cells near the attachment of zygomatic arch, and well-pneumatized sphenoid sinus that can be violated while resecting V2. Careful obliteration of any open air cells is imperative to avoid a CSF fistula.¹⁷ MC is the most likely source of CSF loss and can be sutured closed

using a fine monofilament suture, though it is difficult to achieve a watertight seal. A fascial patch can be laid over MC and covered with fibrin glue.^{17,42} If unplanned durotomy results from the surgical exposure, watertight repair is advisable. Finally, free-flap closure, described elsewhere in this publication, is the last line of defense. This safe and reliable technique has improved outcomes through reduced CSF leaks, tissue bulk replacement, and better cosmesis.

Conclusion

In the treatment of intracranial PNS of cSCCHN, there is a role for surgery to supplement radiation therapy in improving survival in selected cases by restricting central spread of the disease.^{38,39} This paper outlines the surgical considerations and techniques that have been used. The measures used to minimize the surgical risks are described and, despite the complex neurovascular anatomy, this surgery can be achieved with infrequent major complications. Familiarity with this approach gained in the anatomy laboratory is to be commended.

Note

This paper has not been presented or published elsewhere.

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