

Patterns of Perineural Tumor Spread in Head and Neck Cancer

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KEYWORDS

- Perineural tumor spread • Trigeminal nerve • Facial nerve • Adenoid cystic carcinoma
- Skin cancer metastases

KEY POINTS

- Perineural tumor may extend in a retrograde or antegrade direction and can have skip lesions. Inspection of the entire course of the nerve should be performed.
- Perineural tumor spread (PNS) is most common along branches of the trigeminal and facial nerves.
- PNS can spread from the facial to the trigeminal nerve and vice versa via the auriculotemporal or greater superficial petrosal nerves.

PATTERNS OF PERINEURAL SPREAD IN HEAD AND NECK CANCER

Perineural tumor spread (PNS) is a well-recognized entity in head and neck cancers and represents the dissemination of tumor from the primary site via the nerve and neural sheath. It is important to distinguish between perineural invasion and PNS of tumor. Perineural invasion represents small nerve involvement and is a histologic finding at the primary site, present when tumor cells lie within any of the layers of the nerve sheath or when tumor cells surround more than 33% of the circumference of the nerve.¹ Perineural invasion has significant negative prognostic value and correlates with recurrence. Perineural spread, however, describes extension of malignancy beyond the confines of the primary tumor via neural conduits and represents gross, radiologically evident large nerve involvement. It is a mode of metastasis rather than a histologic feature of the primary tumor. PNS most commonly occurs in a retrograde fashion, toward the central nervous system, but also can occur in

an antegrade direction. Like perineural invasion, PNS is also considered a poor prognostic factor and suggests aggressive tumor biology.

The common head and neck tumors that spread via the perineural route include cutaneous malignancies, mucosal primaries such as squamous cell carcinoma and salivary gland malignancies (especially adenoid cystic carcinoma), desmoplastic melanoma, nasopharyngeal carcinoma, myeloma, lymphoma, and leukemia. Various theories have been proposed for why PNS occurs; the nerve may represent the line of least resistance through anatomy otherwise difficult to traverse by tumor, or it may be caused by expression of neurotropic growth factors by tumors of the head and neck.² Among patients with PNS, 40% are asymptomatic, which puts the onus on the radiologist to be vigilant to its presence.³ If symptomatic, the patients present with pain, numbness, burning, and dysesthesia along the course of the affected nerve, and motor denervation changes. The latter is most commonly manifested as weakness of the muscles of mastication or facial paralysis. Another

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common presentation is multiple cranial neuropathies, which indicate involvement of the cavernous sinus and is an ominous sign.

Detection of PNS has important therapeutic implications. Accurate diagnosis of PNS allows complete tumor resection in some cases, and in others allows recognition of unresectable disease before a major ablative surgery is performed. It enables radiation fields to be expanded for complete tumor coverage. PNS also has prognostic significance; in the case of adenoid cystic carcinoma, perineural invasion detected histologically in a larger “named” nerve and skull base involvement predicted much worse rates of local control at 5 years: 12.5% compared with 90% in patients who were named nerve-negative.⁴ However, PNS in adenoid cystic carcinoma does not significantly impact long-term survival, which is more dependant on tumor size, TNM staging, histologic subtype, and expression of *p53*.⁵

In general, PNS occurs along branches of the facial and trigeminal nerves, although spread along the sixth cranial nerve⁶ and the greater auricular nerve⁷ has also been described. The greater auricular nerve originates from the cervical plexus and is composed of branches of spinal nerves C2 and C3. It provides sensory supply to the skin over the parotid gland, mastoid process, and outer ear. Additionally, the facial and trigeminal nerves have various interconnections that serve as conduits for widespread dissemination of neoplasm.

Relevant Anatomy

The trigeminal nerve arises from the pons and travels through the prepontine cistern to enter the Meckel cave, a cerebrospinal fluid-filled arachnoid recess posterolateral to the cavernous sinus (Fig. 1). The trigeminal gasserian ganglion resides along the lateral and anterior wall of the Meckel cave. Within the Meckel cave, the nerve divides into ophthalmic, maxillary, and mandibular divisions (Fig. 2). From the Meckel cave, the ophthalmic and maxillary divisions travel through the lateral wall of the cavernous sinus (Fig. 3). The ophthalmic division (V1), the smallest division of cranial nerve V, divides into the nasociliary, frontal, and lacrimal nerves just before it enters the orbit via the superior orbital fissure. The frontal nerve is the largest branch and divides into the supratrochlear and supraorbital branches, which provide sensory innervation to the skin of the forehead and upper eyelid and the mucosa of the frontal sinus. The nasociliary nerve supplies the nasoethmoid mucosa. The lacrimal nerve provides sensory innervation for the lacrimal gland, conjunctiva, and the lateral upper eyelids. From the cavernous sinus, the

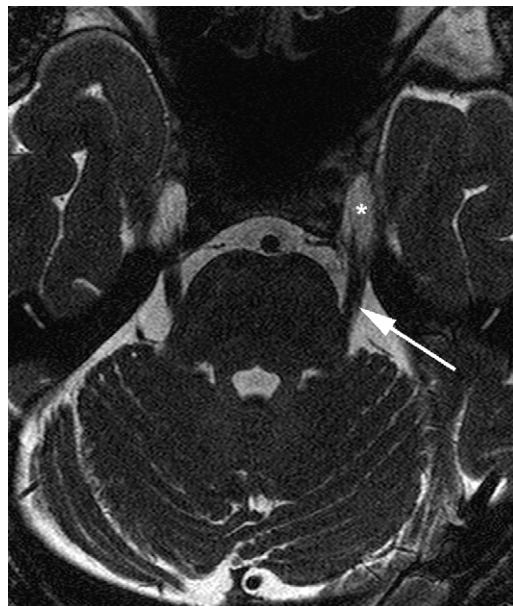


Fig. 1. Cranial nerve V anatomy. Axial 3-dimensional constructive interference in steady state (CISS) image shows the cranial nerve V traveling in the prepontine cistern (arrow) before entering the Meckel cave (asterisk).

maxillary division (V2) enters the pterygopalatine fossa (PPF) via the foramen rotundum (Fig. 4). Within the PPF, V2 gives rise to greater and lesser palatine branches, the zygomatic nerve (sensory to the skin of the temporal region and cheek), and the posterior superior alveolar nerve (supplying the maxillary sinus) before continuing on as the infraorbital nerve in the infraorbital canal (Fig. 5). The infraorbital nerve gives off the anterior and middle superior alveolar nerves to the maxillary teeth and gingiva before emerging from the infraorbital foramen to supply the skin of the midface and lateral nose. The greater and lesser palatine nerves descend from the PPF to the hard palate via the descending palatine canals (Fig. 6).

The mandibular division (V3) exits the skull base via the foramen ovale without entering the cavernous sinus (see Fig. 4B). It enters the masticator space, giving off branches to the muscles of mastication, the lingual nerve (sensory to tongue), auriculotemporal nerve (sensory to temporal scalp, secretomotor to parotid gland), and buccal nerve (sensory to buccal mucosa and molar teeth), and continues as the inferior alveolar nerve, which enters the mandibular canal and supplies the mandibular teeth and gingiva. At the level of the second premolar, the inferior alveolar nerve gives off the mental nerve, which exits via the mental foramen on the lateral aspect of the mandibular body and supplies the skin of the chin and lower

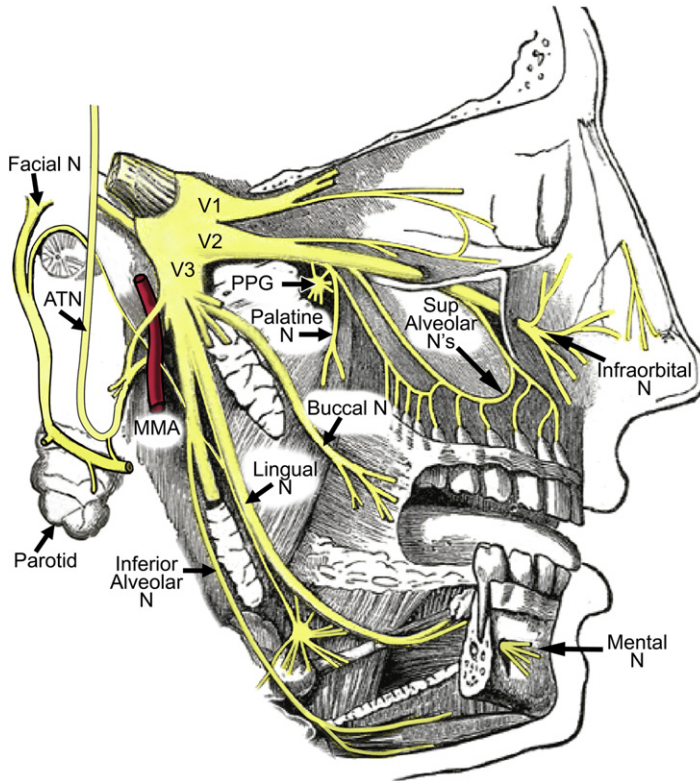


Fig. 2. Cranial nerve V anatomy. Cranial nerve V divides into the ophthalmic (V1), maxillary (V2), and mandibular (V3) branches in the Meckel cave. V1 passes through the cavernous sinus lateral wall and enters the orbit via the superior orbital fissure. V2 passes through the cavernous sinus and exits the skull via foramen rotundum. V3 exits the skull via foramen ovale and enters the masticator space, where it gives off sensory and muscular branches. The auriculotemporal nerve arises from V3 via two roots that encircle the middle meningeal artery and then form a single root, which enters the parotid gland and intermingles with branches of cranial nerve VII. ATN, auriculotemporal nerve; MMA, middle meningeal artery; N, nerve; PPG, pterygopalatine ganglion.



Fig. 3. Cavernous sinus anatomy. Coronal high-resolution postgadolinium image shows cranial nerve III (oculomotor), IV (trochlear), VI (abducens), and V2 (maxillary) traveling through the cavernous sinus. Cranial nerve V1 (ophthalmic) is not well seen on this image.

lip. The auriculotemporal nerve anatomy is described in greater detail in subsequent paragraphs. Tumor anywhere within the dermatomal distribution of the trigeminal nerve can perineurally spread via the pathways mentioned earlier through the skull base foramina into the cranial cavity.

The facial nerve arises from the pons, and its fibers loop around the abducens nucleus to form the facial colliculus in the anterior wall of the fourth ventricle. It crosses the cerebellopontine angle cistern (cisternal segment) and enters the internal auditory canal, where it resides in the superior anterior compartment (canalicular segment). The labyrinthine segment lies between the canalicular segment and the first genu and is so named because of its intimate relationship to the inner ear (labyrinth) (**Fig. 7A**). The tympanic segment travels posterolaterally from the first genu under the lateral semicircular canal (best appreciated on coronal images) (see **Fig. 7B**). From the second



Fig. 4. Cranial nerve V anatomy. Axial CT (A) image shows the foramen rotundum (arrows) though which V2 travels after exiting from the cavernous sinus. It continues forward and enters the pterygopalatine fossa (asterisk). At a slightly inferior level (B), the vidian canal (white arrows) can be seen extending forward from the foramen lacerum. Foramen ovale resides in the sphenoid wing (black arrows). Asterisks indicate the pterygopalatine fossa.

genu, close to the pyramidal process, the nerve travels straight down in the mastoid bone (mastoid segment) and exits the skull via the stylomastoid foramen to enter the parotid gland. The facial nerve has five branches in the parotid gland: temporal, zygomatic, buccal, marginal mandibular, and cervical. Presence of a perineural venous plexus can cause normal enhancement beyond the first genu of the nerve. If the enhancement is thick and asymmetric, even in the parts of the nerve that can normally enhance, one should suspect pathology in the appropriate clinical

setting. Any enhancement of the nerve proximal to the first genu is very suspicious.

Connections exist between the facial and trigeminal nerve that facilitate tumor traveling between these nerves. The greater superficial petrosal nerve is a branch of the facial nerve that provides parasympathetic innervation to the lacrimal gland, palate, nasal cavity, and nasopharynx. It contains facial nerve fibers originating in the nervus intermedius, exits the geniculate ganglion at the facial hiatus, passes close to the Meckel cave, and joins with the deep petrosal



Fig. 5. Cranial nerve V anatomy. The infraorbital foramen and fissure is noted on this sagittal CT image (arrows). Skin cancers in the infraorbital region can travel along this pathway to the pterygopalatine fossa.



Fig. 6. Palatine canals. The palatine canals carrying the greater and lesser palatine nerves extend superiorly toward the pterygopalatine fossa from the hard palate (arrows).

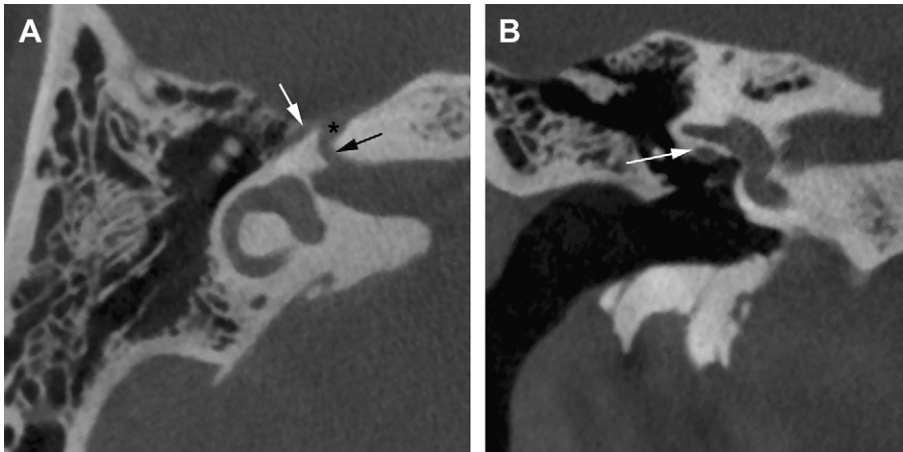


Fig. 7. CN VII anatomy. Axial cone beam CT image (A) shows the labyrinthine segment (*black arrow*) extending into the geniculate ganglion (*asterisk*). The proximal tympanic segment is also noted (*white arrow*). On the coronal image (B) the tympanic segment of the facial nerve (*arrow*) is seen traveling below the lateral semicircular canal.

nerve to form the vidian nerve, which passes through the vidian canal to the pterygopalatine fossa where its preganglionic fibers synapse in the pterygopalatine ganglion. Some of the postganglionic fibers join branches of V2 in the PPF (**Fig. 8**).⁸ PNS along the greater superficial petrosal nerve (GSPN) usually occurs after the tumor has reached the PPF, from where retrograde extension via the vidian nerve to GSPN can occur (**Fig. 9**). Also, tumor in the Meckel cave may extend directly to the GSPN because of their proximity.

The auriculotemporal nerve arises as two roots from the posterior division of V3 soon after it exits the skull base via the foramen ovale. The two roots encircle the middle meningeal artery (see **Fig. 2**) and then converge to form a single nerve, which passes into the parotid gland along the posterior border of the mandibular ramus/condylar head. The nerve gives off intraparotid secretomotor

branches and eventually provides sensory innervation to the auricle and skin of the temporal region. Rami of the auriculotemporal nerve connect it to the facial nerve within the parotid gland.⁹ Tumor may spread from cranial nerve V to VII (or vice versa) along this route (**Fig. 10**).

Finally, the chorda tympani (branch of cranial nerve VII in the temporal bone) joins the lingual nerve (V3 branch) after it exits from the skull and supplies sensation to the anterior two-thirds of the tongue and secretomotor innervation to the submandibular and sublingual glands (see **Fig. 2**).

Fat is generally present where the cranial nerves exit from skull base foramen. These fat pads can be seen at the superior orbital fissure (V1), the PPF (V2), inferior to the foramen ovale (V3; the trigeminal fat pad), and the stylomastoid foramen. Obliteration of these juxtaforaminal fat pads is a key element in detecting PNS.

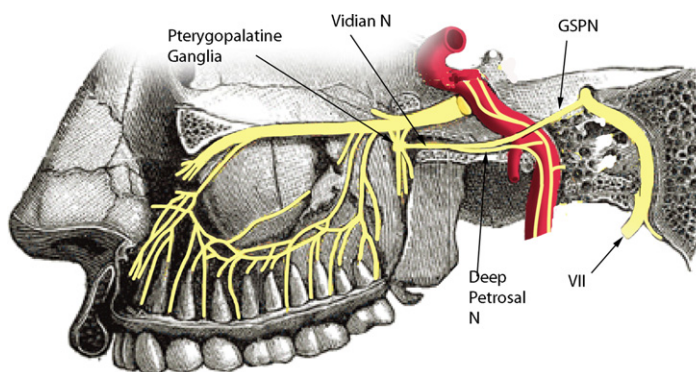


Fig. 8. Greater superficial petrosal nerve (GSPN) anatomy. The GSPN arises from the first genu of the facial nerve in the temporal bone, travels anteriorly and joins with the deep petrosal nerve to form the vidian nerve in the vidian canal. The vidian nerve then joins V2 branches in the pterygopalatine ganglion residing in the pterygopalatine fossa. N, nerve.

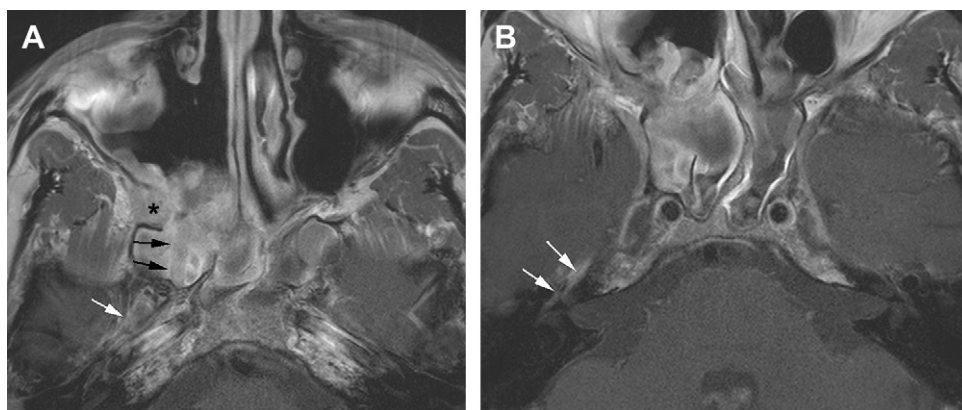


Fig. 9. PNS along the GSPN in a patient with a history of maxillary sinus squamous cell carcinoma. Axial T1-weighted postgadolinium image (A) shows tumor in the PPF (asterisk), enlargement of the vidian N (black arrows), and enhancement along the foramen lacerum (white arrow). (B) Enhancement extends back to the GSPN and proximal tympanic segment of the facial nerve (arrows); this is one of the connections between the cranial nerves V and VII, which facilitate PNS between the nerves.

Imaging approach

CT better detects the bony changes associated with PNS, but MRI is by far the preferred study for evaluation of PNS. CT findings that should raise concern for PNS include obliteration of juxtaforaminal fat pads and fat planes along the paths of cranial nerves, and foraminal enlargement and destruction (see **Fig. 10**; **Fig. 11**). Attention should be specifically directed to the pterygopalatine fossa, the foramen ovale, and the stylomastoid foramen. Although MRI is more sensitive for detecting PNS, patients frequently present for imaging without a history of primary tumor, and recognition of these findings on CT can diagnose the cause of symptoms and indicate a likely site

of tumor origin. High-resolution MRI (3 mm sections, small field of view) can then be performed for the most accurate depiction of the extent of PNS. Precontrast T1-weighted images are helpful in detecting obliteration of fat along the nerves or in the juxtaforaminal fat. Fat is your friend!¹⁰ At the authors' institution, high-resolution postgadolinium 3-mm axial and coronal images without fat suppression are performed. Other head and neck radiologists advocate postgadolinium images with fat suppression for evaluation of PNS, but one must be aware of artifacts associated with fat suppression at the skull base when reviewing the images. On MRI, obliteration of fat planes and foraminal enlargement may also be appreciated,

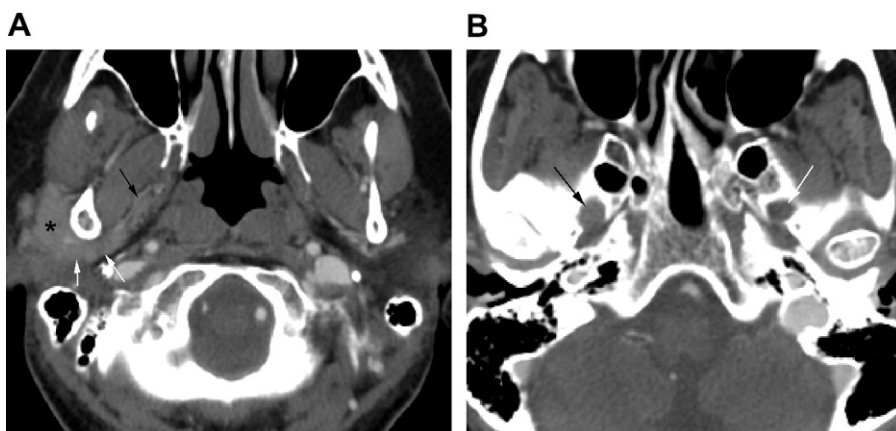


Fig. 10. PNS along auriculotemporal nerve in a patient with carcinoma ex pleomorphic adenoma of the right parotid gland. Axial contrast-enhanced CT (A) shows a mass in the superficial lobe of the right parotid gland (asterisk). Curvilinear soft tissue thickening is seen along the back of the mandibular ramus (white arrows) indicating tumor involving the auriculotemporal nerve, which continues anteromedially to join the trunk of V3 in the masticator space (black arrow). (B) Enlargement of the right foramen ovale is seen (black arrow) with loss of fat compared with the left (white arrow).

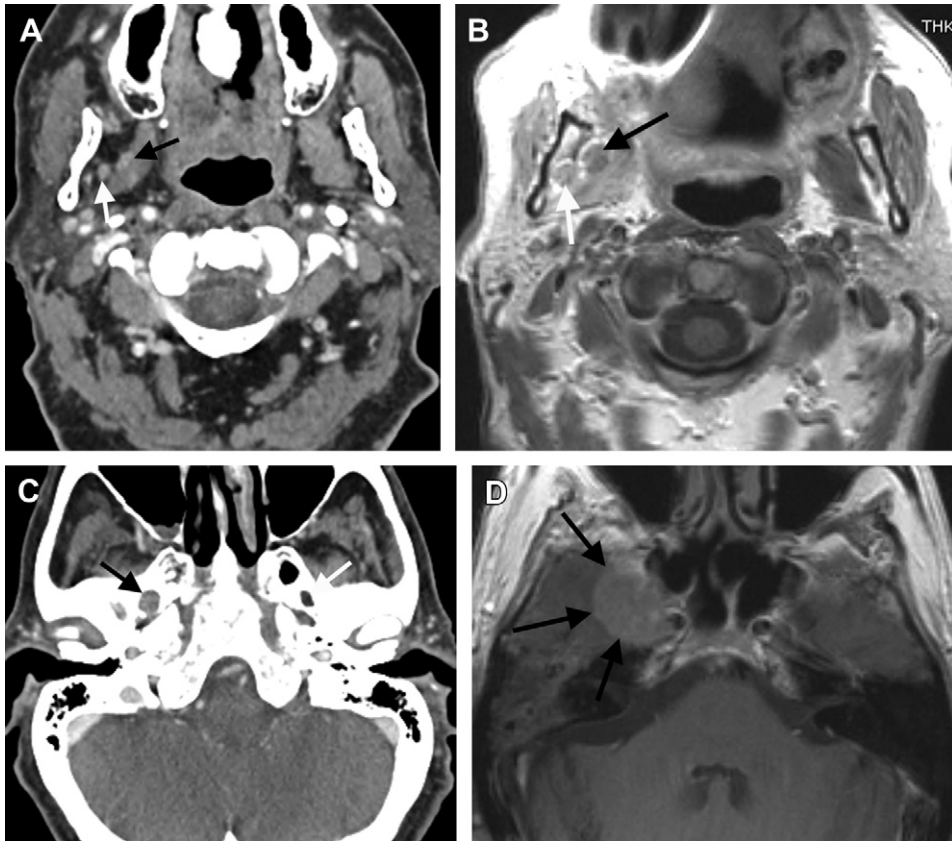


Fig. 11. PNS from adenoid cystic carcinoma of the submandibular gland. Axial CT image with contrast (A) shows thickening and enlargement of the right lingual nerve (*black arrow*) and inferior alveolar nerve (*white arrow*). The same findings are seen on the axial T1-weighted image with gadolinium (B). Tumor extension to the foramen ovale is seen (*black arrow*), which is enlarged with replacement of the normal fat on axial CT image with contrast. Normal left foramen ovale (*white arrow*) (C). Tumor extension is noted into the right Meckel cave and middle cranial fossa (*arrows*) on the axial T1-weighted postgadolinium image (D).

but subtler findings of nerve enhancement and nerve enlargement also may be seen.¹¹ Intracranial findings of PNS along the trigeminal nerve include bulging of the wall of the cavernous sinus, enhancement of nerves in the cavernous sinus or basal cistern, and enhancing soft tissue in the Meckel cave (see **Fig. 11**; **Figs. 12** and **13**).¹² Intracranial findings of PNS along the facial nerve include enhancement in the internal auditory canal and in the cerebellopontine angle cistern. The radiologist should be aware of skip lesions with PNS and should perform an inspection of the nerve in its entirety and look for antegrade lesions. Once tumor has accessed the PPF from any route, antegrade PNS may occur along the infraorbital nerve (see **Fig. 13**). Similarly, once tumor reaches the cavernous sinus or Meckel cave via V3, it can easily spread antegrade along V1 and V2 and retrograde to the pons. One should also look for secondary signs, such as denervation changes in the

muscles supplied by the nerves (**Fig. 14**). On MRI, denervated muscles undergo a characteristic pattern of change. Acute changes (<1 month) cause T2 hyperintensity and enhancement with increased volume. Subacute changes (up to 12–20 months) are heralded by increase in T1 hyperintensity because of fat deposition but no volume loss. Chronic changes (after 12–20 months) are characterized by increased fatty atrophy and volume loss of the affected muscle.¹³ Although PNS may be evident on CT, in the authors' opinion MRI using skull base protocol (including axial thin-section non-fat saturated T1-weighted images) should be the preferred modality for complete evaluation and should be performed even if the CT is positive for PNS.

Knowledge of the site of primary tumor allows careful examination of the nearest large nerve for nerve enhancement, nerve thickening, and foraminal enlargement. **Table 1** provides a

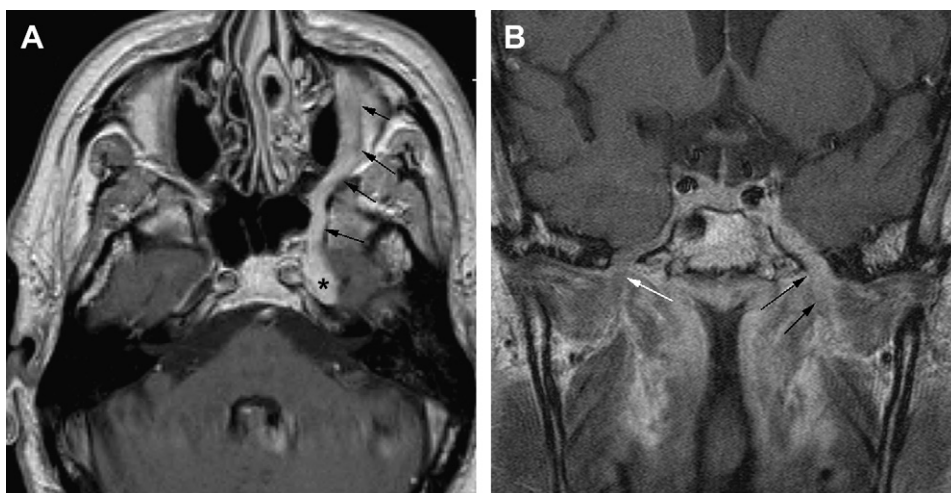


Fig. 12. PNS along V2 in a patient with desmoplastic melanoma of the cheek. Axial postcontrast T1-weighted image (A) shows thickening and enlargement of V2 (arrows) extending to PPF, foramen rotundum, and Meckel cave (asterisk). The coronal postgadolinium image (B) shows PNS into the left V3 (black arrows) via foramen ovale. Note the normal right V3 (white arrow).

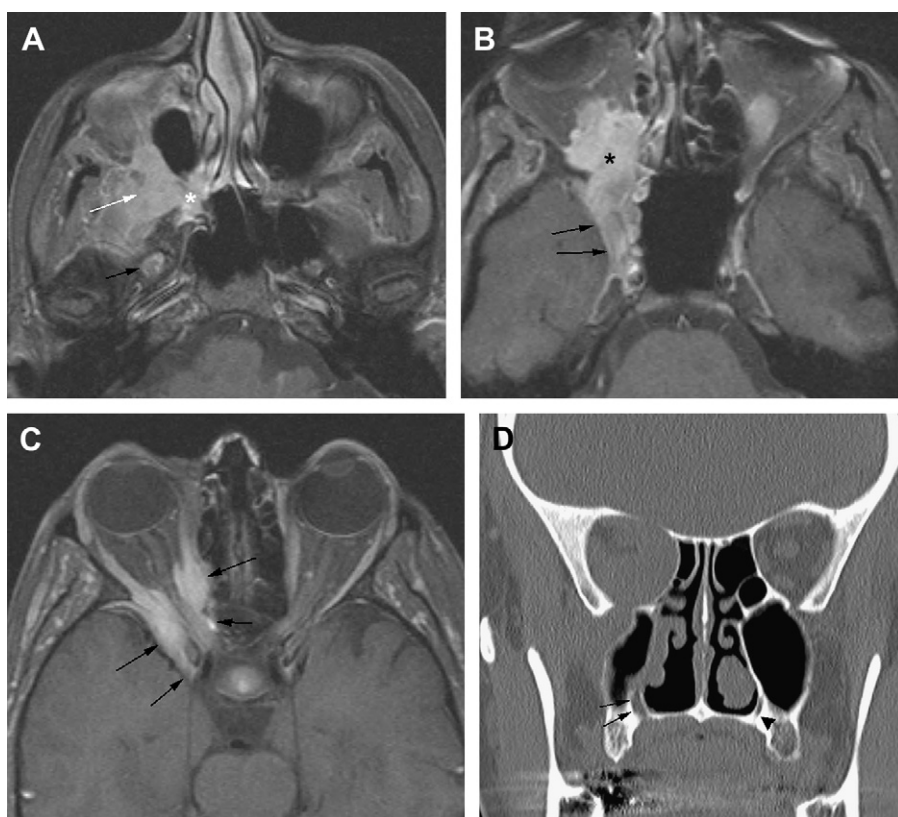


Fig. 13. PNS from adenoid cystic carcinoma of the hard palate via the palatine nerves. Axial T1-weighted postgadolinium image (A) shows tumor in the PPF (white asterisk) and pterygomaxillary fissure/masticator space (white arrow). Enlargement and enhancement of the right foramen ovale is seen (black arrow) from direct involvement of V3 via the masticator space. (B) PNS into the infraorbital nerve (asterisk) via antegrade spread from the PPF is noted. Tumor spread is also seen into the cavernous sinus (black arrows). At a slightly higher level, axial T1-weighted postgadolinium image (C) shows tumor extending into the orbit via direct extension from the PPF with encasement of the optic nerve. The enlargement of the right palatine canals (black arrows) is better appreciated on the coronal CT image (D); note the normal sized left palatine canal (black arrowhead).

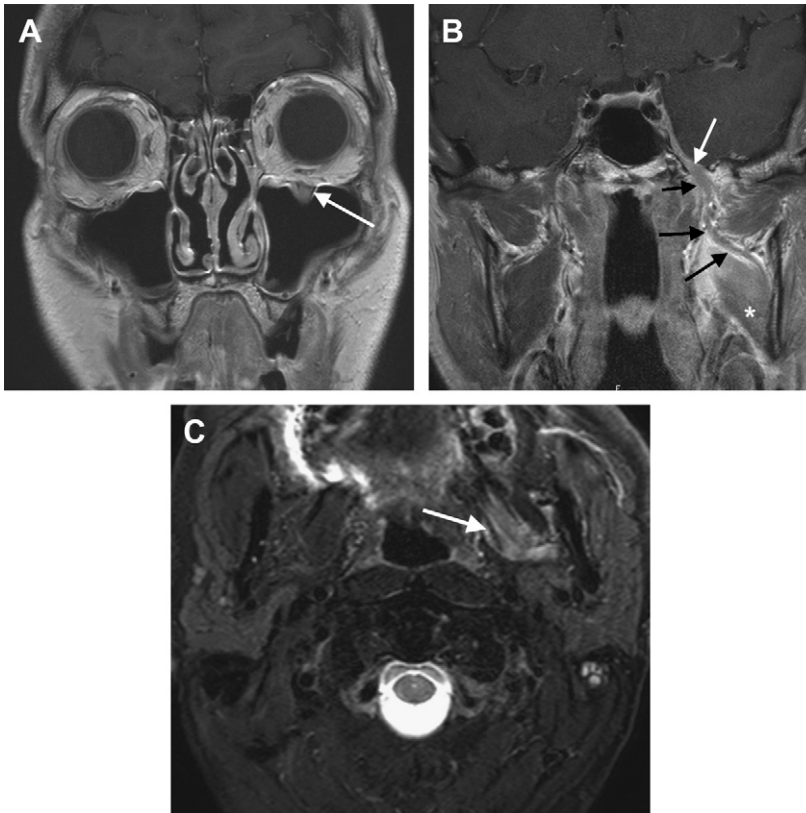


Fig. 14. PNS from cutaneous squamous cell carcinoma of the left face. Coronal T1-weighted image postgadolinium (A) shows a thickened enlarged infraorbital nerve (arrow). (B) Tumor extended retrogradely to the PPF and cavernous sinus with antegrade spread into the left V3 (black arrows), which exits the skull base via the foramen ovale (white arrow). Also note the enhancement of the left muscles of mastication (asterisk), with increased T2 signal (white arrow) on the axial T2-weighted images (C) compatible with acute denervation changes.

comprehensive list of primary tumors and nerves at risk. If the primary site is a cutaneous malignancy of the skin of the nose or cheek, attention should be directed to the infraorbital nerve (see **Figs. 12** and **14**). For orbital primaries, one should inspect the supratrochlear nerve/supra-orbital nerves with attention to the orbital apex/superior orbital fissure/cavernous sinus for potential spread along V1 (ophthalmic nerve). If the primary site is the palate, then the greater and lesser palatine foramina and PPF should be evaluated carefully (see **Fig. 13**). Retromolar trigone cancers can involve the lingual or inferior alveolar nerve, and inspection of the V3 trunk is essential. Submandibular tumors should prompt inspection of the lingual and hypoglossal nerves (see **Fig. 11**), and mandibular tumors should prompt inspection of the inferior alveolar nerve. V2 involvement is seen with maxillary sinus tumors. Ethmoid sinus or medial orbital tumors may spread along V1 (ophthalmic N) branches (**Fig. 15**). The facial and auriculotemporal nerves

should be examined in all parotid malignancies (see **Fig. 10**; **Fig. 16**). Nasopharyngeal carcinoma can involve V3 through either perineural spread or direct extension to the main trunk of V3 in the masticator space. Nasopharyngeal carcinoma can also directly extend to the PPF and then involve V2 perineurally (**Fig. 17**).

In all cases, the combined V3 trunk should be evaluated within the trigeminal fat pad to the level of foramen ovale. Evaluation of a parotid tumor should include inspection of the stylomastoid foramen and the path of the auriculotemporal nerve posterior to the mandibular ramus into the trigeminal fat pad and foramen ovale.

Radiologists, treating physicians, and patients must be aware that after treatment, the imaging findings of PNS may persist indefinitely despite clinical evidence for improvement. Evidence of radiographic or clinical progression will thus become the indicator of recurrent tumor in the setting of previously noted PNS on imaging, not the mere persistence of enhancing nerves.

Table 1 Major head and neck tumors and their at-risk nerves for PNS		
Primary Tumor	At-Risk Distal Nerve Branch for PNS	At-Risk Main Trunk for PNS
Cutaneous malignancy, forehead	Supratrochlear, supraorbital branch of frontal nerve	V1
Cutaneous malignancy, ear	Greater auricular nerve, auriculotemporal nerve	VII
Cutaneous malignancy, temple, upper lateral face	Zygomatic nerve Frontal nerve	V2, VII
Cutaneous malignancy, cheek	Infraorbital nerve, branches, zygomatic nerve, distal facial nerve	V2, VII
Cutaneous malignancy, nose	Infraorbital nerve	V2
Cutaneous malignancy, upper lip	Infraorbital nerve Buccal branches of facial nerve	V2, VII
Cutaneous malignancy, lower lip, chin	Mental nerve Marginal mandibular nerve	V3, VII
Floor of mouth	Lingual nerve	V3
Submandibular gland	Lingual nerve, hypoglossal nerve	V3, X11
Mandible	Inferior alveolar nerve	V3
Retromolar trigone	Lingual nerve, inferior alveolar nerve	V3
Buccal cancer	Buccal branches	V3, VII
Base of tongue	Hypoglossal nerve	XII
Palate	Greater and lesser palatine nerves	V2, PPF
Maxillary sinus	Superior alveolar nerve	V2
Ethmoid sinus	Nasociliary nerve, optic nerve	V1, II, III, IV, abducens
Frontal sinus	Supraorbital nerve	V1
Parotid gland	Facial nerve branches, auriculotemporal nerve	V3, VII
Nasal cavity	PPF via sphenopalatine foramen	V2



Fig. 15. PNS from lacrimal sac adenoid cystic carcinoma. Axial T1-weighted postgadolinium image (A) shows the primary lesion in the right lacrimal sac (*asterisk*) and enlargement of the right cavernous sinus (*arrow*). The coronal postgadolinium (B) image shows the PNS to the orbital apex (*white arrows*) and foramen rotundum (*black arrow*).

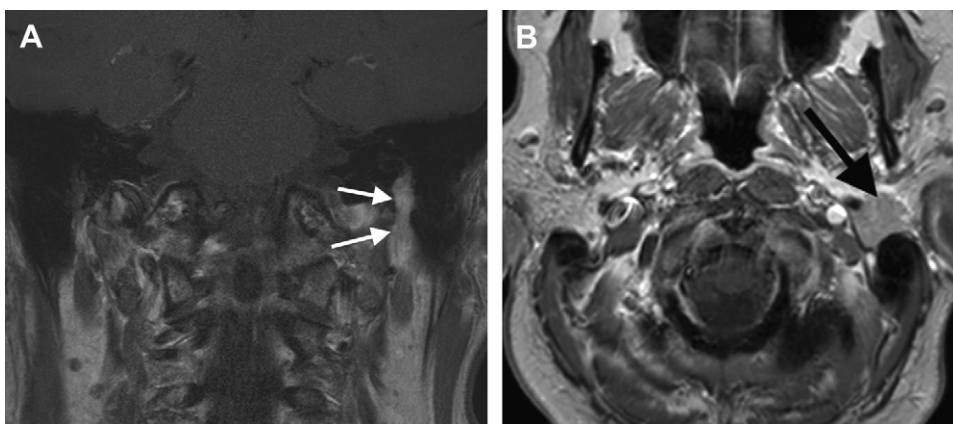


Fig. 16. PNS from adenoid cystic carcinoma of the parotid. Coronal T1-weighted image after contrast (A) shows a thickened enhancing left mastoid segment of the facial nerve (arrows). (B) Axial T1-weighted image with gadolinium shows the primary neoplasm in the parotid (arrow) extending into the stylomastoid foramen, which was pathologically proven to be adenoid cystic carcinoma.

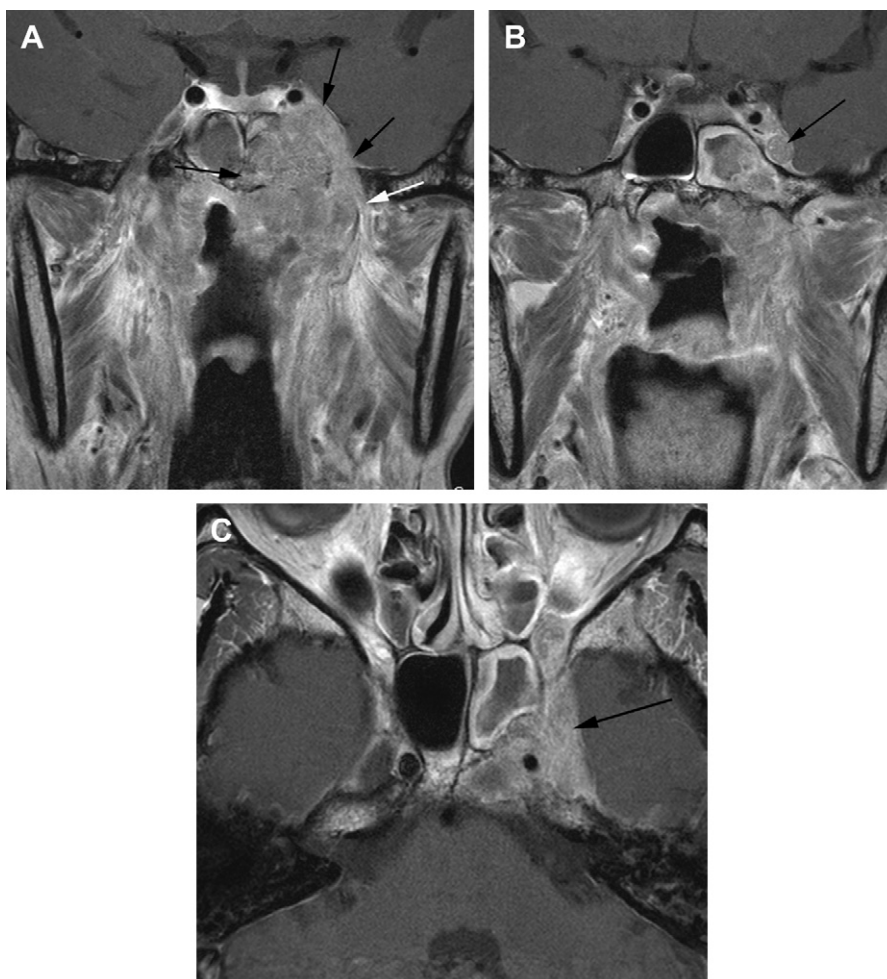


Fig. 17. Nasopharyngeal carcinoma with PNS. Coronal T1-weighted post gadolinium images show (A) left nasopharyngeal carcinoma with direct extension to the V3 trunk in the masticator space (white arrow) and into the Meckel cave and sphenoid sinus (black arrows). (B) Coronal T1-weighted post gadolinium images shows tumor extension into the sphenoid sinus, enlargement of the left cavernous sinus, and PNS along V2 (black arrow). PNS along V2 (arrow) is also well seen on the axial postgadolinium T1-weighted image (C) that also shows involvement of the Meckel cave and encasement of the internal carotid artery.

The imaging differential for PNS includes viral neuritis, carcinomatosis, sarcoid, meningioma, schwannoma, and rhinocerebral mucormycosis, and imaging findings may be very similar. In most cases clinical history itself is the most useful factor in differentiating these entities. At initial imaging presentation, PNS is recognized by the presence of tumor outside of the confines of the cranium, whereas in leptomeningeal carcinomatosis, sarcoid tumor, and meningioma the bulk of disease will lie in the intracranial compartment. In the case of a schwannoma disease along the nerve tends to be bulkier and the nerve acts as the epicenter of the disease process. Patients with invasive fungal sinus disease have a much more acute presentation than do patients with perineural tumor spread and disease will be clustered about the paranasal sinuses with inflammatory change in the sinuses themselves.

Multiple studies have attempted to assess the diagnostic ability of MRI for PNS. In general, research is somewhat hampered by the low prevalence of tumors demonstrating PNS, with multiple reports featuring small numbers of patients. In addition, the presence of PNS into the skull base often mitigates against surgery in many centers, precluding histologic confirmation of imaging findings. Most studies attempt to determine the sensitivity and specificity of imaging for PNS in a population of patients known to have cancer. In this group, with higher pretest probability than the general population, MRI performs well, with reported sensitivities of 100%; however, the extent of PNS is typically underestimated.^{14,15}

SUMMARY

Knowledge of the anatomy of the nerves commonly involved with PNS in the head and neck and appropriate use of imaging modalities will enable early detection and positively impact management of head and neck cancer. In searching for PNS, "fat is your friend," and special attention should be paid to the T1-weighted images.

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