Cranial Nerve XII: Hypoglossal

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23.1 Anatomy

The hypoglossal nerve is derived from the basal plate of the embryonic medulla oblongata. It arises from the hypoglossal nucleus within the posterior and inferior medulla. The efferent nerve fibers then course anteriorly through the medulla, exiting at the pre-olivary (ventrolateral) sulcus to exit from the anterior medulla, and extend laterally to enter the hypoglossal canal (Fig. 23.1), located inferior to the jugular foramen and jugular tubercle within the inferior occipital bone [1, 2]. This segment is accompanied by a prominent venous plexus. The fibers exit the skull and then descend within the carotid space between the internal jugular vein and the internal carotid artery. The nerve then courses toward the hyoid bone, medial to the posterior belly of the digastric muscle, and then along the hyoglossus muscle, where muscular branches provide motor innervation to the majority of the tongue musculature.

Intracranial course: Upper motor neurons originate from lateral precentral gyrus and descend along the corticobulbar tract via the corona radiata and the internal capsule to reach the hypoglossal nuclei bilaterally except for the genioglossus muscle which is crossed and unilateral.

Nuclei: The hypoglossal nuclei are located in the dorsal medulla.

23.1.1 Branches

- Intrinsic tongue muscles (GSE)
  - Vertical tongue muscle nerve
  - Transverse tongue muscle nerve
  - Superior longitudinal muscle nerve
  - Inferior longitudinal muscle nerve
- Extrinsic tongue muscles (GSE)
  - Genioglossus muscle nerve
  - Hyoglossus muscle nerve
  - Styloglossus muscle nerve

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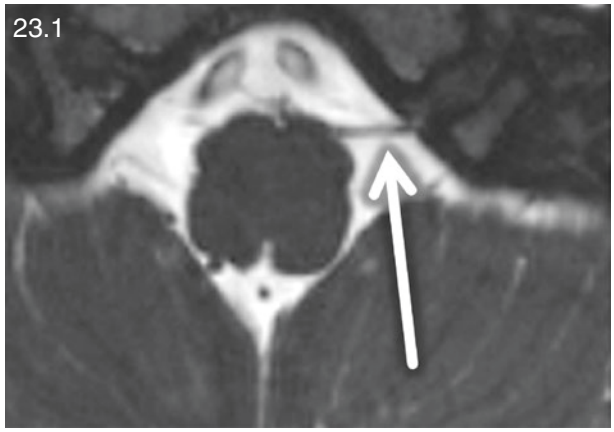

Fig. 23.1 Axial thin-section CSF bright sequence demonstrates the hypoglossal nerve (*arrow*) extending through the medullary cistern laterally toward the hypoglossal canal

23.2 Function

The hypoglossal nerve has only GSE motor fibers. It innervates the intrinsic and all of the extrinsic tongue muscles except the palatoglossus (which is innervated by CN X).

23.3 Pathology

Individual symptoms: Damage to the hypoglossal nerve results in the following symptoms depending on the location:

- *Supranuclear lesions*: Isolated lesions are rare and are typically associated with other neurologic findings. They are not associated with tongue fasciculations or atrophy. Spastic dysarthria and pseudobulbar palsy especially with bilateral cortical lesions are possible. When asked to protrude the tongue, the tongue will deviate away from the side of the lesion [3].
- *Lower medullary lesions*: Isolated lesions of the lower medulla such as ischemia, neoplasm, and/or demyelinating pathologies can result in hypoglossal nerve neuropathy. Pathology at this level will cause the tongue to deviate towards the side of the lesion.
- *Cisternal lesions*: Lesions within the medullary cistern may present with hypoglossal nerve neuropathy, such as meningiomas and/or metastatic disease.
- *Jugular foramen and extracranial lesions*: The most common lesion to involve the hypoglossal nerve is the hypoglossal schwannoma which accounts for only 5% of non-vestibular intracranial schwannomas. This lesion is a benign tumor of differentiated Schwann cells which may arise anywhere along the course of the hypoglossal nerve. A sharply marginated, fusiform mass which may exhibit a dumbbell shape is seen on CT or MRI [4, 5]. The most common MRI characteristics of this lesion include T1 isointensity to gray matter, T2 hyperintensity, and uniform enhancement. Intramural cysts may be identified if the lesion is large. Multiple lesions including skull base metastasis, glomus jugular paraganglioma, vascular variant persistent hypoglossal artery, or jugular foramen meningioma can mimic a hypoglossal schwannoma. Skull base traumatic fractures can also present with hypoglossal nerve dysfunction.
- *Others*: Hypoglossal nerve motor denervation can be secondary to hypoglossal schwannoma or any other skull base lesion causing mass effect on the hypoglossal nerve. Imaging demonstrates asymmetry of the tongue with linear demarcation corresponding to the hypoglossal nerve innervation. The appearance on CT and MRI of hypoglossal nerve motor denervation depends on the time course. In the initial stage, CT shows unilateral tongue swelling, while MRI demonstrates T2 hyper-

intensity and T1 hypointensity with variable enhancement consistent with edema. As the time course of denervation becomes chronic, there is resolution of the swelling and development of fatty atrophy of the affected half of the tongue. MRI demonstrates increasing T1 hyperintensity consistent with fatty atrophy of the tongue musculature and resolution of enhancement. Careful evaluation is recommended to not mistake this entity, with abnormal enlargement or enhancement of the tongue, for a tongue base tumor.

23.3.1 Syndromes

Dejerine syndrome (aka medial medullary syndrome): Occlusion of anterior spinal artery can cause ipsilateral loss of proprioception (damage to the medial lemniscus), ipsilateral paresis of the tongue (tongue deviates to the opposite side), and contralateral hemiplegia.

Pseudobulbar palsy: Bilateral supranuclear lesions can cause bilateral tongue paresis and dysarthria.

Collet-Sicard syndrome: See Chap. 20.

Schmidt syndrome: See Chap. 20.

Villaret's syndrome: See Chap. 20.

Jackson syndrome: Ipsilateral hypoglossal palsy, incomplete vagal paresis (dysarthria), and contralateral hemiparesis/plegia.

References

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