Neurosurgical Review (2021) 44:1345–1355
https://doi.org/10.1007/s10143-020-01344-w

REVIEW

Lower cranial nerve syndromes: a review

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Received: 31 December 2019 / Revised: 9 June 2020 / Accepted: 26 June 2020 / Published online: 8 July 2020

Abstract

The purpose of this paper is to provide a comprehensive review encompassing the syndromes associated with the lower cranial nerves (LCNs). We will discuss the anatomy of some of these syndromes and the historical contributors after whom they were named. The LCNs can be affected individually or in combination, since the cranial nerves at this level share their courses through the jugular foramen and hypoglossal canal and the extracranial spaces. Numerous alterations affecting them have been described in the literature, but much remains to be discovered on this topic. This paper will highlight some of the subtle differences among these syndromes. Symptoms and signs that have localization value for LCN lesions include impaired speech, deglutition, sensory functions, alterations in taste, autonomic dysfunction, neuralgic pain, dysphagia, head or neck pain, cardiac or gastrointestinal compromise, and weakness of the tongue, trapezius, or sternocleidomastoid muscles. To assess the manifestations of LCN lesions correctly, precise knowledge of the anatomy and physiology of the area is required. Treatments currently used for these conditions will also be addressed here. Effective treatments are available in several such cases, but a precondition for complete recovery is a correct and swift diagnosis.

Keywords Accessory nerve · Brain injuries · Cranial nerves · Glossopharyngeal nerve · Hypoglossal nerve · Vagus nerve

Introduction

Impaired functioning of at least one of the twelve cranial nerves (CNs) is termed cranial nerve disease. The neurological physical examination (PE) has excellent localization value and can test the functioning of individual cranial nerves and differentiate among the specific presenting impairments. In this paper, we will address some of the lower cranial nerve (LCN) syndromes in one comprehensive review. The LCNs comprise the paired CN IX (glossopharyngeal), CN X (vagus), CN XI (accessory), and CN XII (hypoglossal) (Figs. 1 and 2). LCN lesions can be attributable to various causes, which need to be explored to optimize management and outcomes for patients.

It is more common for multiple LCNs to be affected than one LCN alone. For example, in upper cervical spine and skull base trauma settings involving occipital fractures, any LCN running along the jugular fossa or hypoglossal foramen can be affected. Causes of LCN lesions can be classified as vascular, traumatic, iatrogenic, immunological, infectious, metabolic, genetic, nutritional, degenerative, or neoplastic. Individual nerves can be affected, as in for example, Eagle's syndrome, where CN IX is compressed by the styloid process. Therefore, treatment of such lesions depends on the underlying cause, with many of these patients in need of addressing their swallowing and breathing issues with, for example, gastrostomy and mechanical ventilation. Although clinical examination is the best in determining which nerves are involved and thus which syndrome is present, imaging modalities such as MRI and CT are used to localize the pathology causing the

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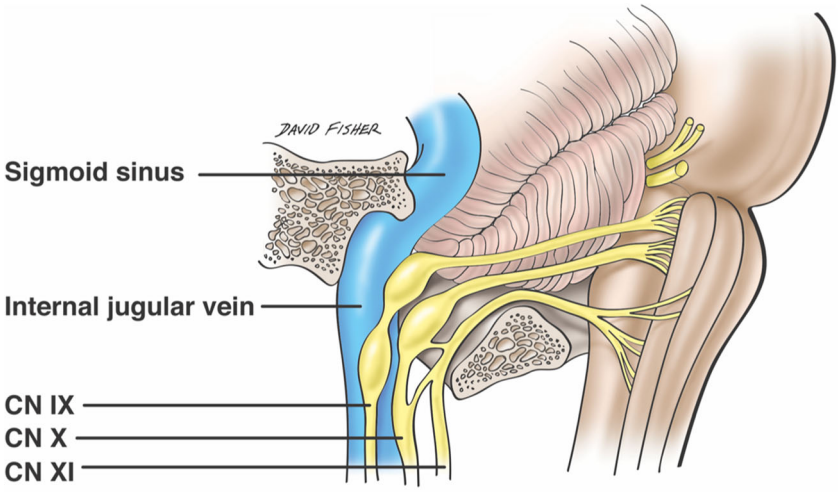

Fig. 1 Anterolateral view of the contents of the right jugular foramen

findings. Herein, we focus on syndromes affecting multiple cranial nerves at the same time so that, for example, isolated cranial nerve compression is not included. This review is an up-to-date analysis of such syndromes and will use detailed anatomical schematic drawings to better aid the reader in understanding this sometimes complicated anatomy.

As neurosurgeons will potentially encounter patients with such syndromes, a good working knowledge of their presentations is important. Therefore, the aim of this paper is to review and discuss seven syndromes that affect multiple LCNs: Avellis, Collet-Sicard, Tapia's, Vernet, Villaret, Schmidt, and Jackson (Fig. 3).

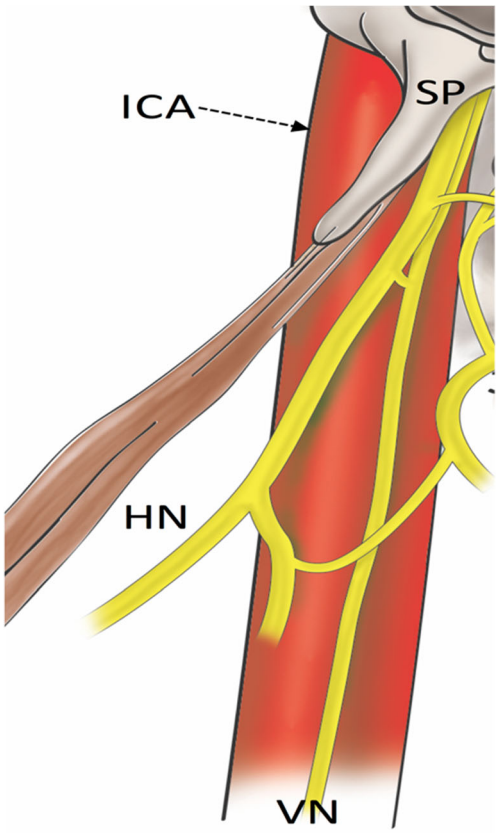

Fig. 2 Left view of the hypoglossal nerve (HN) and its relationships to the vagus nerve (VN) and internal carotid artery (ICA). Note the styloid process (SP) and the muscles arising from it. Connections between the hypoglossal nerve and adjacent cervical nerves are also seen

Anatomy

Jugular foramen

The jugular foramen (Fig. 4) is located in the posterior cranial fossa at the junction of the occipital bone with the petrous segment of the temporal bone [1]. It is separated from the hypoglossal canal by the jugular tubercle, which is highly relevant in skull base surgery, as it can be removed during a far-lateral surgical approach to allow for better visualization of the area around the brain stem [2]. The jugular foramen can be divided into two portions separated by a bony process in up to 20% of cases [3]. The presence of a bony septum as opposed to a fibrous septum or no septum can have implications on surgical planning. The neurovascular structures traversing this compartmented foramen and their relationship are essential for explaining the injuries that lead to jugular foramen syndromes. In its anteromedial division or pars nervosa, the foramen contains the inferior petrosal sinus, CN IX, and Jacobson's nerve (the tympanic branch of CN IX). Its posterolateral compartment or pars venosa contains the internal jugular vein, CNs X and XI, and the meningeal branches of the ascending pharyngeal and occipital arteries [1].

Hypoglossal canal

The hypoglossal canal (HC) (Fig. 5) is located in the anterior process of the occipital bone, lying inferomedial

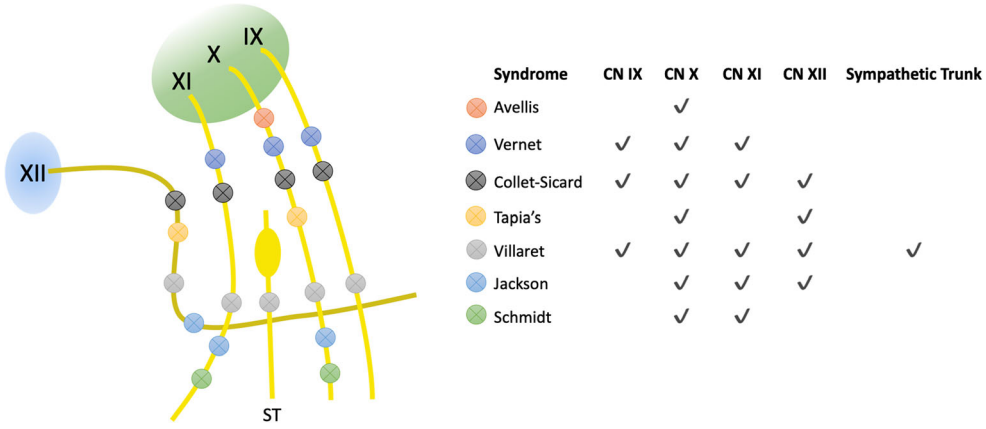

Fig. 3 The various lower cranial nerve syndromes and the nerves they affect. The green oval represents the jugular foramen, and the blue oval represents the hypoglossal canal

to the jugular foramen [1]. It is also segmented by a bony septum in up to 20–30% of cases [1]. The HC contains CN XII, the hypoglossal venous plexus, and the meningeal branch of the ascending pharyngeal artery in up to 45% of cases [4]. This artery can produce posttraumatic pseudoaneurysms that result in symptoms due to compression or to rupture [5]. The HC can also be affected by external invasion of nearby structures derived from the jugular foramen and by tumors such as paragangliomas, intradural meningiomas, and cystic schwannomas [1].

Cranial nerves

CNs IX, X, XI, and XII are complex nerves that originate in the brain stem and innervate muscles and viscera throughout

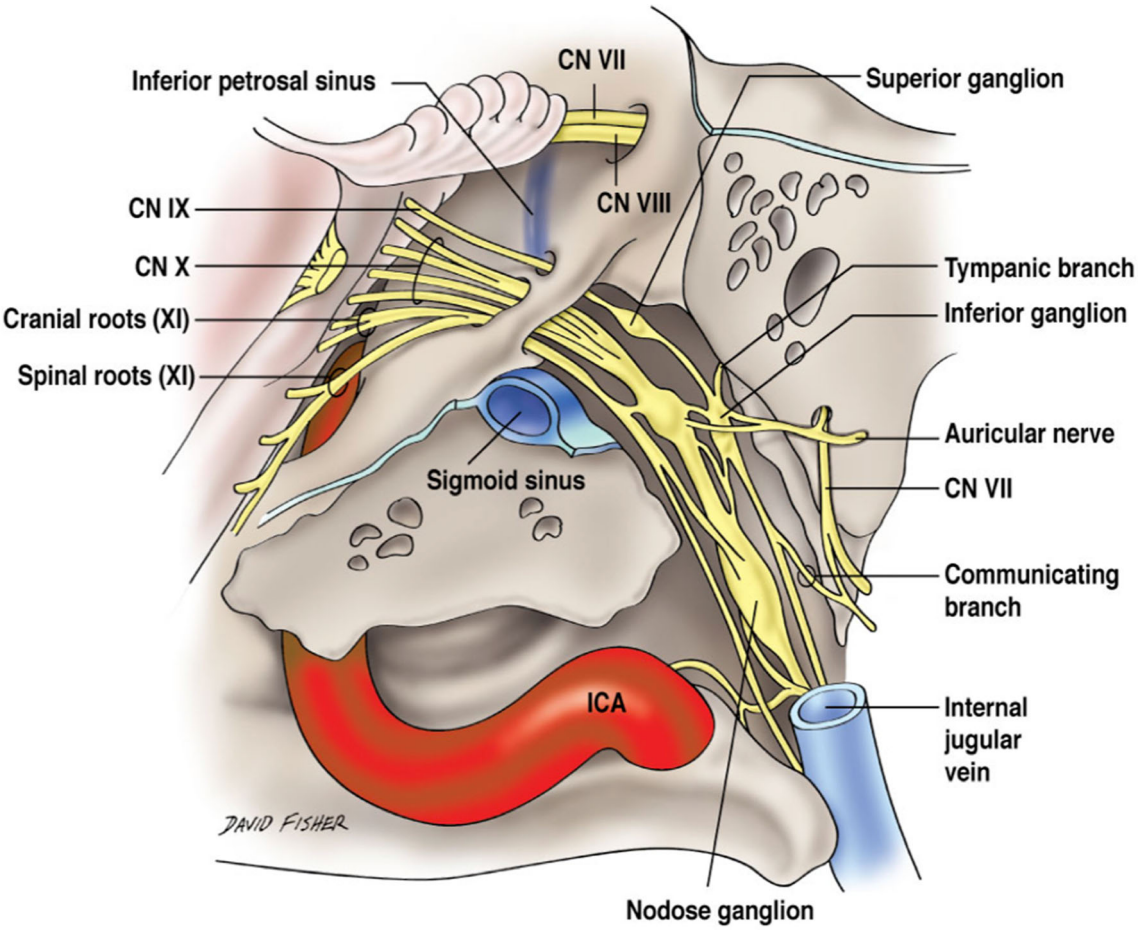

Fig. 4 Nerves of the right jugular foramen and their relationships

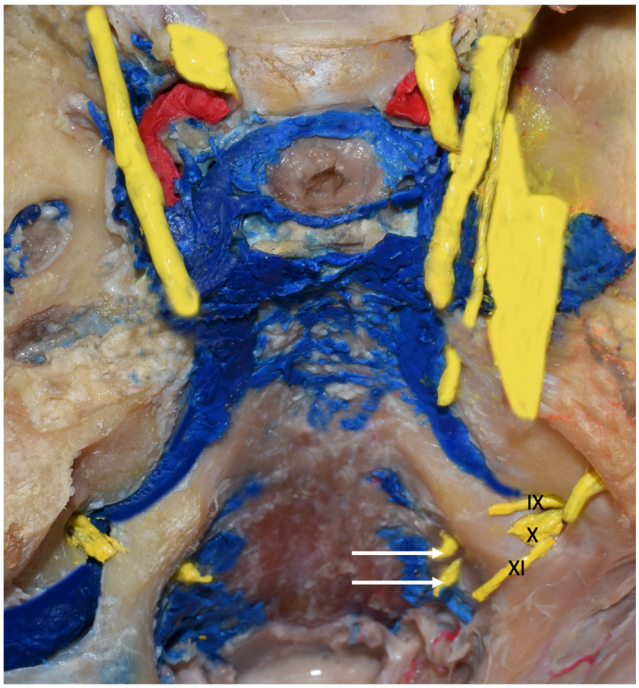

Fig. 5 Nerves and venous sinuses of the skull base noting the right IX, X, and XI cranial nerves. The hypoglossal nerve rootlets are seen at the arrows just prior to entering the hypoglossal canal

the body. Detailed anatomy of the LCNs is presented in Table 1 [6, 7].

Semiology

CNs IX, X, XI, and XII are mainly involved in the execution of crucial physiological functions such as swallowing, speech, and control of visceral organs through their motor and autonomic fibers (Table 2) [6, 8]. The principal functions of the CNs are important in considering the localization value that can be extracted from PE. Although the etiological factors surrounding a particular syndrome are difficult to elucidate using only PE, knowledge of the different functional and semiological highlights around each nerve can help us to diagnose the combination of impairments and establish an accurate description of the syndrome.

Cranial nerves and the sympathetic trunk

The motor function of CN IX (Fig. 6) is evaluated by assessing the patient's ability to swallow and also by looking for asymmetry of the soft palate at rest with movement elicited by vocalizing [6]. Somatic afferent fibers of CN IX are evaluated using pinprick tests in the soft palate and the posterior third of the tongue, and the special visceral afferent fibers are assessed by taste on the posterior third of the tongue. Reflexes of the glossopharyngeal nerve are assessed by the gag

Table 1 Origin, course, blood supply, and functions of the LCNs [6, 7]

	Origin	Course	Extracranial blood supply	Innervations
CN IX	Posterior lateral sulcus of the medulla oblongata dorsal to the inferior olive	Travels through the jugular foramen until it descends on the lateral pharynx	Ascending pharyngeal, occipital, descending palatine, sphenopalatine, ascending palatine, and dorsal lingual artery	Motor: stylopharyngeus Sensory: posterior 1/3 of tongue, soft palate, tonsillar region, pharyngeal wall Autonomic: parotid gland
CN X	Posterior lateral sulcus of the medulla oblongata dorsal to the inferior olive	Travels through the jugular foramen to the abdominal viscera; gives off branches to external ear, meninges, larynx, thoracic plexuses, abdominal plexuses	ICA, ECA, CCA posterior meningeal artery, vagal artery, vertebral artery, internal thoracic artery, bronchial branch, esophageal branch, aorta	Motor: soft palate except tensor veli palatini, pharynx, larynx Sensory: pinna, tongue Autonomic: visceral organs of thorax and abdomen
CN XI	Nucleus ambiguus of the medulla, lateral medulla, and accessory nucleus	Travels through the jugular foramen then branches to join the vagus as well as enter the posterior triangle of the neck	Occipital, lingual artery	Motor: SCM muscle, trapezius muscle
CN XII	Hypoglossal nucleus	Travels through the hypoglossal canal toward the tongue	Ascending pharyngeal, occipital, facial, lingual artery, ECA	Motor: all intrinsic muscles of tongue, hyoglossus, styloglossus, genioglossus

ICA, internal carotid artery; *ECA*, external carotid artery; *CCA*, common carotid artery; *SCM*, sternocleidomastoid

Table 2 Summary of the physiological functions of the LCNs [6]

	Efferent functions	Afferent functions	Primary reflex functions
CN IX	Motor: elevates pharynx for swallowing and speech Autonomic: parasympathetic fibers via otic ganglion to parotid glands for saliva secretion	Visceral sensory: baroreceptors and chemoreceptors of carotid sinus and body, respectively Somatic sensory: sensation from ear, tonsils, upper pharynx, posterior 1/3 of the tongue Special sensory: taste from posterior 1/3 of the tongue	Afferent limb of gag reflex
CN X	Motor: contracts soft palate, pharyngeal, and laryngeal muscles for swallowing and speech Autonomic: parasympathetic fibers for control of airway, heart rate, GI peristalsis, and sweating	Visceral sensory: heart and abdominal viscera Somatic sensory: sensation from external ear, larynx, pharynx	Efferent limb of gag reflex
CN XI	Motor: trapezius for elevating shoulders and sternocleidomastoid for rotating the head to the contralateral side	No physiologically significant afferent functions	No reflex functions
CN XII	Motor: extrinsic muscles of tongue for protrusion to contralateral side used in speech and food manipulation Autonomic: some sympathetic innervation to tongue vessels and oral mucosa glands	No physiologically significant afferent functions	No reflex functions

maneuver and the palatal reflex when the soft palate is stimulated by touch. Notably, CN IX carries the afferents involved in these reflex arches, while CN X (Fig. 1) carries the motor efferents. CN IX also contains autonomic fibers to the parotid gland to stimulate salivary secretions. The motor function of CN X is assessed by the ability to swallow and by phonation, as CN X innervates skeletal muscles of the pharynx and larynx. The soft palate must be symmetrical both at rest and during vocalization. Unilateral lesions cause deviation of the uvula and dysphagia, and unilateral flattening of the palatal arch. When the nerve is injured, the vocal cord adopts the midline position closing part of the glottis. Sensory information is hard to interpret because of the shared sensory territory with other nerves.

Reflexes are shared with CN IX. It is important to remember that CN X is the efferent limb of the reflex arch. The function of CN XI (Fig. 7) is assessed by rotation of the head, shoulder movement, and phonation, since it supplies the sternocleidomastoid, trapezius, and cricothyroid muscles [6]. Motor function of CN XII (Fig. 5) is assessed by movement of the tongue and the presence of lingual deviations both at rest and when the tongue is protruded. Unilateral lesions can result in tongue deviation toward the side of the lesion, atrophy, dysarthria, and tongue fasciculations [6]. The sympathetic trunk (Fig. 8) is responsible for sympathetic information to the pupils, lacrimal glands, sweat glands, and levator palpebrae muscle. When the sympathetic trunk is injured, patients present with Horner's syndrome, which consists of the triad of ptosis, miosis, and anhidrosis, and is also associated with facial blush thermal dysregulation [6].

The involvement of one or more of the nerves mentioned above indicates diagnostic suspicion of one of the following syndromes. Although rare, involvement of all or up to seven ipsilateral cranial nerves from tumors, usually of the nasopharynx, has been termed Garcin syndrome or Guillain-Alajouanine-Garcin syndrome.

Syndromes

Avellis syndrome

George Avellis, a German laryngologist, first described the condition that bears his name in a series of ten cases published in 1891 [9]. Since then, only approximately 30 cases have been reported in the available literature [9]. He described a so-called laryngeal hemiplegia, referring to an injury to the nucleus ambiguus that disturbed signals being sent to CN X, which controls the pharynx and larynx (Fig. 9) [8]. In this syndrome (Fig. 3; Table 3), there is ipsilateral paralysis of the vocal cords and soft palate and contralateral dissociated hemianesthesia including the extremities, trunk, and neck. It can result from occlusion of the vertebral artery in lesions affecting not only the nucleus ambiguus but also the

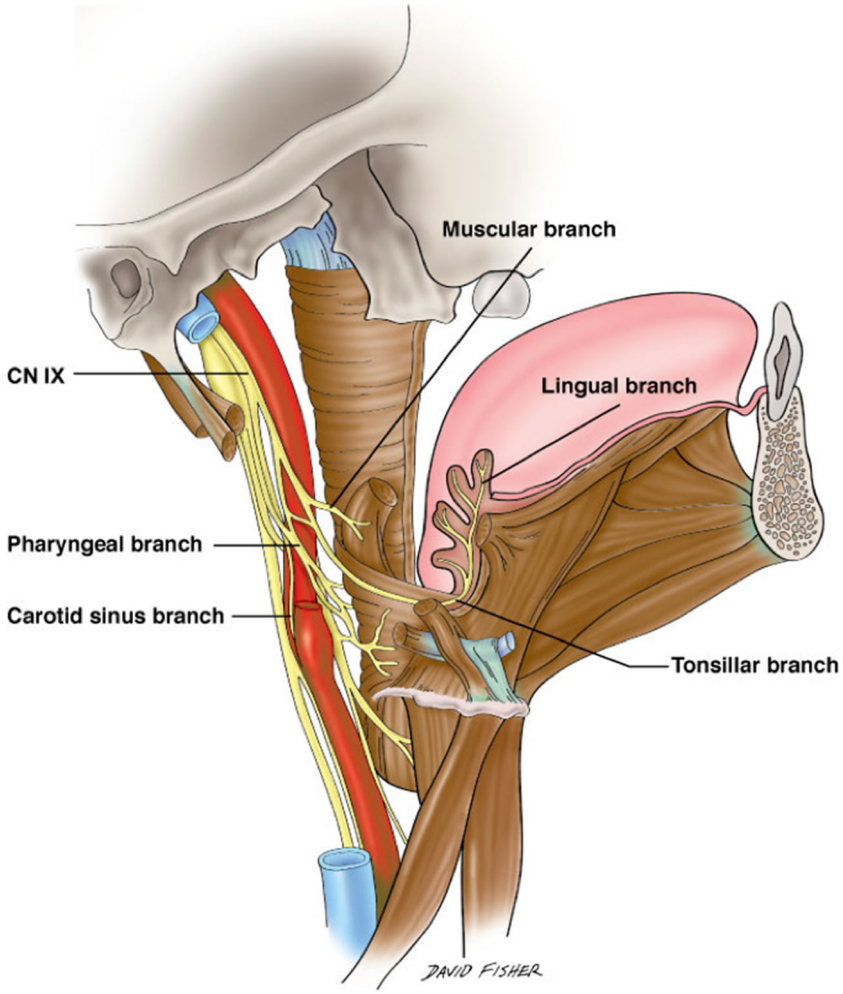

Fig. 6 Course of the right glossopharyngeal nerve and its relationships

pyramidal tract [8]. Thus, it represents a classical brain stem syndrome. Mass lesions compressing the jugular foramen have also been described as causing this syndrome; trauma is an infrequent cause [8, 9].

Surgical removal of the lesion and associated compressive effect in these cases has been shown to help alleviate symptoms [8]. Treatment with high-dose prednisolone, cyclosporin, and cyclophosphamide has proved fairly effective for Avellis syndrome, but the continuation of such medications may produce liver injury [25]. Patients can undergo swallowing rehabilitation treatment, such as oral and pharyngolaryngeal muscle strengthening exercises, swallowing reflex therapy, and functional electrical stimulation therapy at an outpatient clinic [17].

Vernet syndrome

Vernet syndrome (Fig. 3; Table 3) was named after Maurice Vernet (1887–1974), a French neurologist who described a motor paralysis of CNs IX, X, and XI. It is most often attributable to a malignant tumor, aneurysm, or skull base fracture [10]. It presents clinically as severe ipsilateral paralysis and atrophy of sternocleidomastoid and upper trapezius, with absent gag reflex and diminished posterior tongue pinprick sensation [13]. The usual causes of Vernet syndrome are primary tumors such as paraganglioma, meningioma, schwannoma, and metastatic tumors at the skull base. However, there are other causes such as meningitis, otitis externa, sarcoidosis, Guillain-Barré syndrome, and traumatic fractures [10]. In addition, there are rare cases related to varicella zoster virus [10], giant cell arteritis [26], and acute otitis media [27], all of which can present as Vernet syndrome. Vascular cases such as a large aneurysm of the extracranial internal carotid artery (ICA) and internal jugular vein thrombosis have also been known to manifest as Vernet syndrome [12]. Iatrogenic cases associated with carotid endarterectomy, causing posterior pharyngeal wall edema or swelling in the parapharyngeal space, have also been described [28].

Collet-Sicard syndrome

Collet-Sicard syndrome (Fig. 3; Table 3) was first described in 1915 by Frédéric Justin Collet, a French pathologist and otolaryngologist, who termed it "glossolaryngoscapulopharyngeal hemiplegia" [14]. It was called Collet-Sicard syndrome for

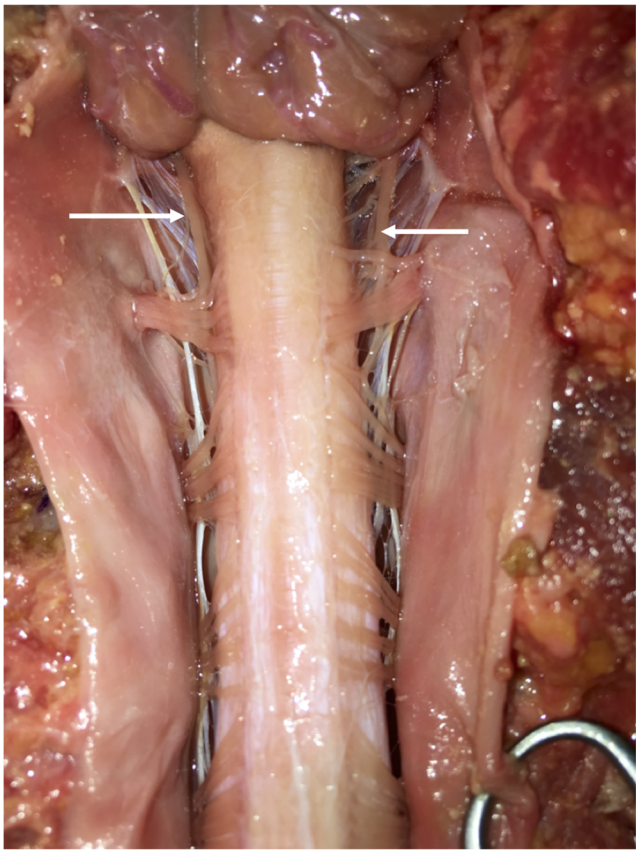

Fig. 7 Posterior cadaveric dissection of the accessory nerves (arrows). The white bands running longitudinally are the denticulate ligaments. Note the left and right cerebellar tonsils at the upper aspect of the image. The dura mater is reflected laterally

Jean-Athanase Sicard, a French neurologist and radiologist, who described the disorder independently of Collet [29]. It refers to unilateral paralysis of the last four cranial nerves (CNs IX, X, XI, and XII) [30, 31]. The causes are usually malignant lesions of the skull base and nasopharynx resulting in motor symptoms such as paralysis of the vocal cords, palate, and trapezius and sternocleidomastoid muscles [14], associated with sensory symptoms such as anesthesia of the larynx, pharynx, and soft palate. It has been described in traumatic contexts such as Jefferson fracture, idiopathic cranial polyneuropathy, multiple myeloma, ICA dissection, and Lyme disease, and is associated with skull base tumors of primary and metastatic (lung, breast, prostate, and renal) origins [15]. Occipital condyle fractures (OCF) are a rare cause of Collet-Sicard syndrome [16]. Although they are among the rarer traumatic lesions of the skull base, OCF were established as a distinct clinical entity owing to their specific features. The occipital condyles are closely related to the HC and the jugular foramen, which contains CNs IX, X, and XI [16]. They also have a vital anatomical relationship to the brain stem and vascular structures. Most craniocervical junction injuries, including OCF, are sustained in high-speed motor vehicle accidents or falls from a great height, and they can lead to immediate death [16].

Diagnosis of Collet-Sicard syndrome can be based on clinical findings, neurological examination, magnetic resonance imaging findings, a video fluoroscopy swallowing study, and electrodiagnosis [17]. After rehabilitation, the patient's swallowing difficulty and even shoulder weakness can improve [17].

Tapia's syndrome

Tapia's syndrome (Fig. 3; Table 3) was described in 1904 by Antonio Garcia Tapia, a Spanish otolaryngologist [18]. It is characterized by paralysis of CNs X and XII [18]. Roughly 69 cases have been reported in the literature [18]. Common symptoms can range from dysphonia to tongue deviation toward the affected side and from lingual motility disturbance to swallowing difficulty [18]. It is most commonly seen in patients as a rare complication of airway manipulation during a surgical procedure [18]. Hence, the simultaneous emergence of these symptoms in a patient who has undergone orotracheal intubation needs a correct diagnostic process to exclude Tapia's syndrome [18, 19]. In mild forms, the symptoms of Tapia's syndrome can be confused with discomfort triggered by surgery [18]. This is particularly relevant when the surgery involves anatomical areas close to CN X and CN XII [18], as can occur when shoulder surgery is performed in the sitting position. When the surgery is carried out in the fully upright sitting position, the patient can be liable to head misplacement [32]. Compression by the endotracheal tube caused by displacement of the head can easily cause injury to these CNs [32]. Keeping the head aligned can be difficult because the body is generally out of view and direct access is not easy [32]. To preclude such problems, specially designed shoulder operating tables have been made available to improve body alignment and the patient's comfort level [32].

Most patients recover their lost function within 3 months after diagnosis [18]. For proper rehabilitation of Tapia's syndrome, it is important to perform an airway endoscopy and institute a specific program of swallowing rehabilitation [18]. A multidisciplinary approach is necessary for the correct management of this pathology. Neurology, rehabilitation (dysphagia unit), and maxillofacial services can choose to collaborate closely to ensure proper management [18].

Villaret's syndrome

Villaret's syndrome (Fig. 3; Table 3) was described in 1917 by Maurice Villaret (1877–1946), a French neurologist [21]. It was first identified among injured soldiers as part of the jugular foramen syndrome in which multiple LCNs were injured [33]. Villaret is remembered for his studies and experiments involving the precise localization of vascular lesions of the brain. The syndrome results when CNs IX, X, XI, and XII have been affected in association with ipsilateral Horner's

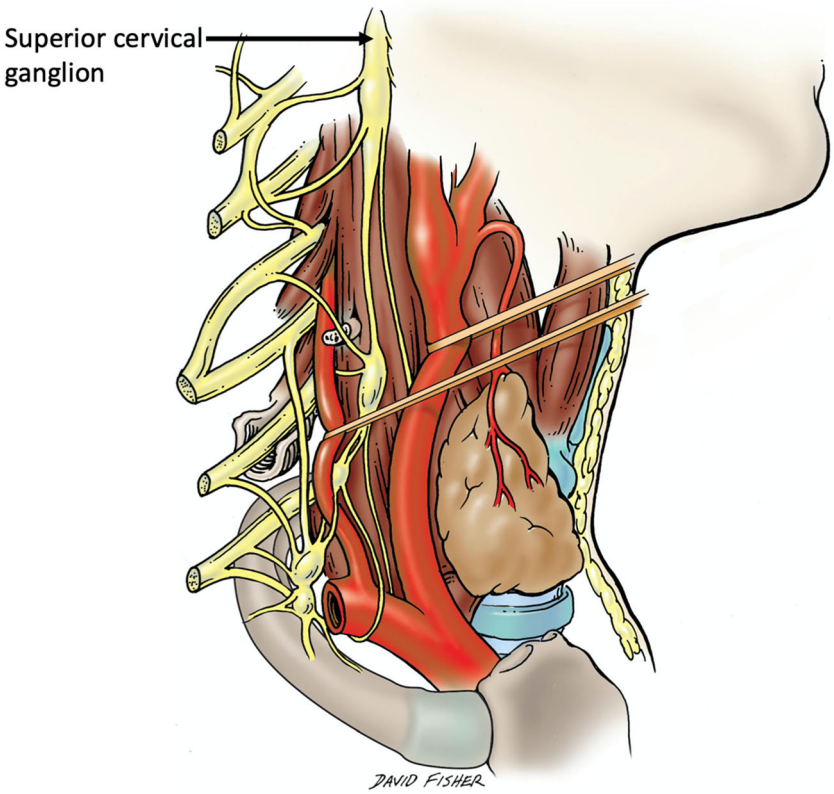

Fig. 8 Schematic drawing of the right sympathetic trunk and its relationships in the neck

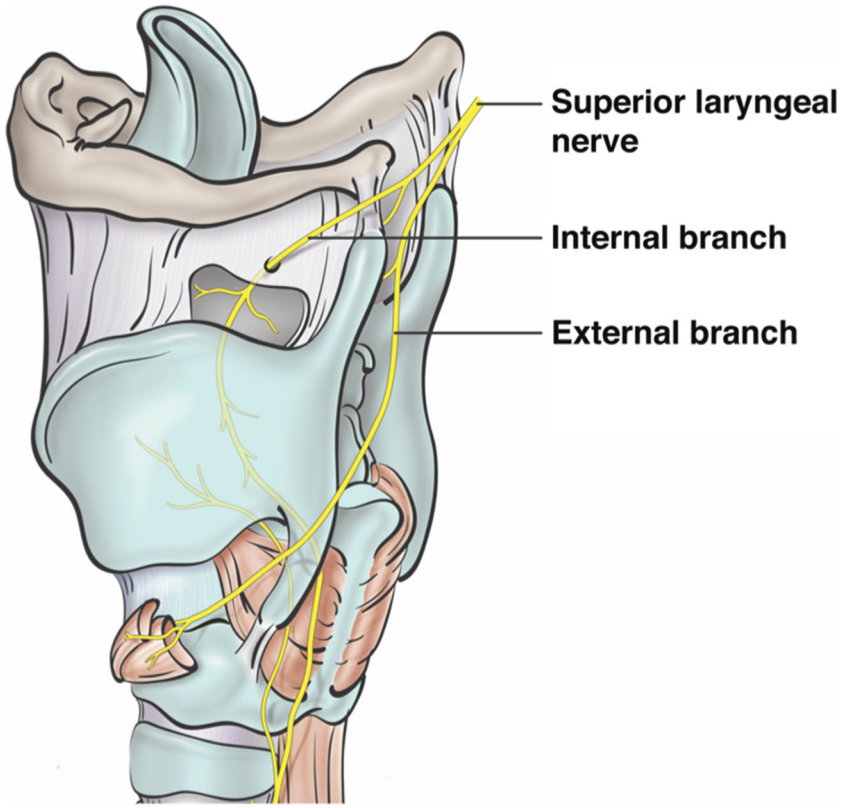

Fig. 9 Left superior laryngeal nerve and its branches, the internal and external laryngeal nerves

Table 3 Summary of cranial nerve syndromes discussed in this review

Syndrome	Etiology	CNs affected	Symptoms	Treatment
Avellis	Vertebral artery occlusion, jugular foramen tumors, trauma [5, 8, 9]	CN X	Ipsilateral paralysis of vocal cords, soft palate; contralateral dissociated hemianesthesia [8]	Steroids, cyclosporin, cyclophosphamide, rehabilitation [8]
Vernet	Jugular foramen tumors, aneurysms, infection, sarcoidosis, skull base fracture [10–12]	CNs IX, X, and XI	Ipsilateral paralysis of SCM, upper trapezius; absent gag reflex; diminished posterior tongue sensation [13]	Steroids, surgical elimination of inciting factor [10]
Collet-Sicard	Skull base tumors, trauma, occipital condyle fractures [14–16]	CNs IX, X, XI, and XII	Ipsilateral paralysis of vocal cords, palate, SCM, trapezius; anesthesia of larynx; pharynx, soft palate [14]	Rehabilitation [17]
Tapia's	Airway manipulation (intubation) [18]	CNs X and XII	Tongue deviation toward side of lesion, dysphagia, dysphonia [18]	Rehabilitation [18, 19]
Villaret	Primary retroparotid space base tumors, metastases from breast, aneurysms [18, 20]	CNs IX, X, XI, and XII, and sympathetic trunk	Horner's syndrome, loss of taste on posterior 1/3 of the tongue, loss of sensation from soft palate, ipsilateral deviation of the tongue, dysarthria [20, 21]	Surgical elimination of inciting factor [22]
Jackson	Tumors, trauma, aneurysms, iatrogenic [23]	CNs X, XI, and XII	Ipsilateral paralysis of soft palate, larynx, SCM, trapezius, and tongue [6, 24]	Surgical elimination of inciting factor [23]
Schmidt	Primary skull base tumors; metastases from prostate, breast, and cervix; skull fractures; ICA dissection; IJV thrombosis [24]	CNs X and XI	Ipsilateral paralysis of SCM, soft palate, larynx, trapezius, and the tongue [24]	Surgical elimination of inciting factor

SCM, sternocleidomastoid; *ICA*, internal carotid artery; *IJV*, internal jugular vein

syndrome [20]. The clinical manifestation includes loss of taste on the posterior third of the tongue, loss of sensory information from the soft palate, ipsilateral deviation of the tongue and dysarthria, and Horner's syndrome represented by the triad of ptosis, miosis, and anhidrosis [21]. It results from lesions of the four LCNs and the cervical sympathetic plexus fibers when they are compressed right at the skull base, particularly in the retroparotid space [20]. Although invasion of the central nervous system in patients with advanced lung cancer is frequent and well-known, a newly diagnosed lung adenocarcinoma presenting with Villaret's syndrome is extremely rare [20]. As a clinical subtype of jugular foramen syndromes, metastatic masses at the skull base cause this syndrome, breast cancer being the most common source of such metastases [34]. As a rare clinical entity, this constellation of signs should prompt the clinician to look for pathology in the retroparotid space, as this is the only area where the lower four cranial nerves and sympathetic fibers to the eye lie next to each other [35]. When the sympathetic fibers are spared, it is classified as Collet-Sicard syndrome, as previously mentioned. In a series of 29 patients with jugular foramen syndromes, only two had Villaret's syndrome [33].

The most common causes of Villaret's syndrome are masses in the retroparotid space such as tumors (melanoma, renal cell carcinoma, small cell carcinoma of the lung, meningioma, parotid gland tumor, and lymphoma) and aneurysms [22]. A unique case of metastatic prostate cancer as a cause has also been reported; the patient presented with dysphonia, dysphagia, and hemiparesis of the posterior pharynx, soft palate, and vocal cord, with ipsilateral deviation of the tongue and paresis of sternocleidomastoid and trapezius muscles associated with ipsilateral partial ptosis, miosis, and enophthalmos [22]. Treatment always consists of treating the cause of the compression by either surgery or radiation therapy [22]. In another case, a patient developed skull base osteomyelitis, a life-threatening condition similar to notoriously infectious diseases such as malignant otitis externa and necrotizing otitis externa [36]. *Pseudomonas aeruginosa* and *Proteus mirabilis* are the most common causative pathogens in skull base osteomyelitis. When the condition is caused by skull base osteomyelitis, it can be treated with adequate antibiotics, and the infection resolves to leave only mild sequelae. Vascular pathologies such as aneurysms of the ICA can also produce the clinical manifestations previously described [36].

Jackson syndrome

Jackson syndrome (Fig. 3; Table 3) was first described by the eponymous neurologist and psychiatrist in 1864 as paralysis of CNs X, XI and, XII. It presents with unilateral paralysis of the soft palate, larynx, sternocleidomastoid, trapezius, and tongue [6, 36]. Causes of the syndrome include tumors such

as metastases, epidermoid tumors [24], nasopharyngeal carcinoma, and also trauma, vascular etiologies, or surgical procedures.

Schmidt syndrome

Schmidt syndrome (Fig. 3; Table 3) was described in 1892 and described as paralysis of CNs X and XI. It presents with unilateral dysphonia and paralysis of the sternocleidomastoid muscle. Causes and etiologies include metastases from adenocarcinoma of the prostate, breast adenocarcinoma, carcinoma of the cervix, glomus jugular tumors, basilar skull fractures, and vascular lesions such as ICA dissection, thrombosis of the internal jugular vein, and ICA aneurysms [24].

Conclusion

LCN syndromes involving CN IX (glossopharyngeal), CN X (vagus), CN XI (accessory), and CN XII (hypoglossal) have been broadly discussed in this paper. All seven syndromes (Avellis, Collet-Sicard, Tapia's, Vernet, Villaret, Schmidt, and Jackson) present in various ways and share many different etiologies. Treatment is based on the etiology of the eliciting factors, and there can be many ways of rehabilitating the patient, which can sometimes take weeks to months in terms of their recovery process. A detailed understanding of the anatomy and functions of the lower cranial nerves can assist in diagnosing syndromes associated with their dysfunction [37–43]. Such knowledge can improve patient care and potentially decrease patient morbidity.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The protocol of the study did not require approval by the ethical committees or informed consent. The study followed the Declaration of Helsinki (64th WMA General Assembly, Fortaleza, Brazil, October 2013).

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