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神经病学双语教材

主编 朱榆红

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前　言

我国医学教育50年来一直采用前苏联的模式，基本上可以概括为“结构式课堂教学”。具体讲，就是重概念、轻实践，重理论、轻方法，重教师传授、轻学生参与，随着时代的发展，越来越显示出其存在的弊端和不足。创新精神和能力培养是医学教育的灵魂和目标，而案例教学在医学教育中，是培养高素质、创新型和实用型医学人才的有效途径。

神经病学是以研究中枢神经系统、周围神经系统和骨骼肌疾病发病机制、临床表现、诊断、鉴别诊断、治疗、预防和康复为主要内容的一门临床二级学科。是医学生必修课程，也是医师资格考试课程。该课程与神经解剖学等多个学科密切相关，新研究和新进展层出不穷，国际间的交流与合作会日益频繁，双语教学早已成为神经病学教学的重要方面。国内一直缺少一本适合医学本科生学习的神经病学双语教材，这是神经病学双语教学的瓶颈之一，所以，编写引领医学教材发展趋势的双语案例版教材势在必行。

我们结合双语教学实践，根据本科学生英语水平的实际情况及医学专业教育的特殊性，参考或选用了国外教材的部分内容以及现有的指南等，编写了这本教材。教材内容符合我国学生的认识能力，不追求多而全，仍然结合大纲，突出重点，主要收录了临床最常见的神经系统疾病和相关的神经解剖知识。篇幅适当，编排尽量适合教学课时需要。每个章节均分别采用中英文进行编写，方便学生对照学习。在各章节内容中，引入病因学、发病机制、诊断技术及治疗方法等方面国内外最新研究成果。在篇幅允许的范围内，努力做到既能反映神经病学领域的经典内容，又能反映当前研究的最新成就，拓展学生的视野和知识面。

本教材编写不改变现有教学体制，其教学核心内容不变，在教材中增加临床真实病例或标准化病例，是本教材有别于其他教材的特色。借鉴国外PBL教学模式，融典型案例于教材中，案例引导教学，建立起神经病学的临床思维。组织教学时，既可以按传统模式讲授，病例作为补充，供学生阅读使用；也可以以临床病例为先导进行教学。英文案例不仅提高了学生双语学习的兴趣，在医学实践中也具有很强的实用性。本教材内容从一开始的“活页版”，到现在的集合成册出版，试用期间不断完善，很好地解决了双语教学与神经病学之间的交叉与联系问题。

特别感谢美国新泽西州赛诺菲·安万特公司蔡宏容、李黎教授对本书章节的校对指正。

由于作者经验水平有限，本书难免有不少错误和疏漏，真诚希望得到读者的批评和指正，今后不断修改完善。

朱榆红

2010年12月

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Chapter 1
Anatomy and Clinical Correlation of Nervous System

Ⅰ Cranial nerves

The cranial nerves (CNs) are composed of twelve pairs of nerves that emanate from the nervous tissue of the brain. In order to reach their targets they must ultimately exit/enter the cranium through openings in the skull. Hence, their names are derived from their association with the cranium. Only the first and the second pair emerge from the cerebrum, the remaining 10 pairs emerge from the brainstem.

The cranial nerves are divided into different types because of their different functions. CNs Ⅲ, Ⅳ, Ⅵ, Ⅺ and Ⅻ are motor nerves. CNs Ⅰ, Ⅱ and Ⅷ are sensory nerves. CNs Ⅴ, Ⅶ, Ⅸ and Ⅹ are mixed nerves. CNs Ⅲ, Ⅶ, Ⅸ and Ⅹ are also accompanied with the fibers of parasympathetic nerve.

The corticobulbar tract innervates cranial motor nuclei bilaterally with the exception of the lower facial nuclei which are innervated only contralaterally (below the eyes) and CN Ⅻ, which is innervated contralaterally as well.

The olfactory nerve (CNⅠ)

【Anatomy】

The olfactory nerve is the first cranial nerve. It consists of unmyelinated axons of bipolar neurons that are located in the nasal mucosa and the olfactory epithelium. It enters the skull through the cribriform plate of the ethmoid bone.

【Clinical correlation】

Lesions of the olfactory pathway often result from trauma(*e. g.* skull fracture) and olfactory groove meningiomas. These lesions cause ipsilateral anosmia (localizing value). Lesions that involve the parahippocampal uncus may cause olfactory hallucinations.

The optic nerve (CN Ⅱ)

【Anatomy】

The optic nerve is usually associated with the charge of vision and pupillary reaction to light. Ganglion cells of the retina form the optic nerve. They project from the nasal hemiretina to the contralateral lateral geniculate body and from the temporal hemiretina to the ipsilateral lateral geniculate body. The optic nerve projects from the lamina cribrosa of the scleral canal, through the optic canal, to the optic chiasm. The optic chiasm contains decussating fibers from the two nasal hemiretinas and noncrossing fibers from

the two temporal hemiretinas. Then it projects fibers to the suprachiasmatic nucleus of the hypothalamus. The optic tract contains fibers from the ipsilateral temporal hemiretina and the contralateral nasal hemiretina. It projects to the ipsilateral lateral geniculate body, pretectal nuclei, and superior colliculus. The geniculocalcarine tract (visual radiation) projects through two divisions to the visual cortex. The upper division projects through to the upper bank of the calcarine sulcus, the cuneus. The lower division loops from the lateral geniculate body anteriorly (Meyer's loop), then posteriorly, to terminate in the lower bank of the calcarine sulcus, the lingual gyrus. The visual cortex is located on the banks of the calcarine fissure (Figure 1 - 1).

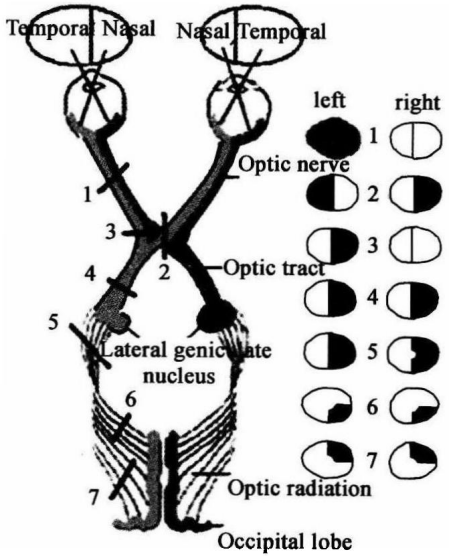

Figure 1 - 1 Common visual field defects and their anatomical bases

1. Total blindness of the left eye from a complete lesion of the left optic nerve. 2. Bitemporal hemianopia caused by pressure exerted on the optic chiasm by a pituitary tumor. 3. Left nasal hemianopia caused by a perichiasmal lesion (*e. g.* calcified internal carotid artery). 4. Right homonymous hemianopia from a lesion of the left optic tract. 5. Right homonymous hemianopia (with macular sparing) resulting from posterior cerebral artery occlusion. 6. Right homonymous inferior quadrantanopia caused by partial involvement of the optic radiation by a lesion in the left parietal lobe. 7. Right homonymous superior quadrantanopia caused by partial involvement of the optic radiation by a lesion in the left temporal lobe (Meyer's loop).

【Clinical correlation】

1. Transection of optic nerve Ipsilateral blindness, with no direct papillary light reflex.

2. Lesions of optic chiasm Midsagittal transection (often from a pituitary tumor) causes bitemporal hemianopia. Bilateral lateral compression causes binasal hemianopia (calcified internal carotid arteries).

3. Lesions of optic tract Transection causes contralateral hemianopia.

4. Lesions of geniculocalcarine tract (visual radiation) Transection of upper divi-

sion causes a contralateral lower quadrantanopia. Transection of lower division causes a contralateral upper quadrantanopia.

5. Lesions of visual cortex Lesions cause contralateral hemianopia with macular sparing.

6. Papilledema It is a noninflammatory congestion of the optic disk as a result of increased intracranial pressure. It is most commonly caused by brain tumors, subdural hematoma, or hydrocephalus. It usually does not alter visual acuity, but it may cause bilateral enlarged blind spots.

The oculomotor nerve, the trochlear nerve, and the abducent nerve (CN Ⅲ, CN Ⅳ, and CN Ⅵ)

【Anatomy】

1. The oculomotor nerve It moves the eyes, constricts the pupils, takes charge of the accommodation reflex and the convergence reflex. It exits the brain stem from the interpeduncular fossa of the midbrain, passes through the cavernous sinus, and enters the orbit through the superior orbital fissure. It innervates four extraocular muscles and the palpebralis. The medial rectus muscle adducts the eye. The superior rectus muscle elevates, intorts, and adducts the eye. The inferior rectus muscle depresses, extorts, and abducts the eye. The inferior oblique muscle elevates, extorts, and abducts the eye. The palpebralis elevates the upper eyelid.

2. The trochlear nerve It is a pure motor nerve that innervates the superior oblique muscle. This muscle depresses, intorts, and abducts the eye. It arises from the contralateral trochlear nucleus of the caudal midbrain, decussates beneath the superior medullary velum of the midbrain and exits the brain stem on its dorsal surface, caudal to the inferior colliculus. At last, this nerve encircles the midbrain within the subarachnoid space, passes through the cavernous sinus, and enters the orbit through the superior orbital fissure.

3. The abducent nerve It innervates the lateral rectus muscle, which abducts the eye. This nerve arises from the abducent nucleus that is found in the dorsomedial tegmentum of the caudal pons. Nerve root descends with the corticospinal tract. It passes through the pontine cistern and cavernous sinus and enters the orbit through the superior orbital fissure.

【Clinical correlation】

1. Peripheral ophthalmoplegia

(1)Oculomotor paralysis is seen in the transtentorial herniation (*e. g.* tumor, subdural or epidural hematoma). Lesions result in the following neurologic deficits: ptosis (denervation of the palpebralis), eye to look "down and out" (denervation of the extraocular muscles), diplopia when the patient looks in the direction of the paretic muscle (oculomotor palsy). Interruption of parasympathetic innervation results in a dilated, fixed pupil and paralysis of accommodation.

(2)The trochlear nerve paralysis results in the following conditions: extorsion of the eye and weakness of downward gaze, vertical diplopia, head tilting to compensate for extorsion. Head injury and tumors in the anterior medullary velum can result it.

(3) The abducent nerve paralysis is the most common isolated palsy that results from the long peripheral course of the nerve. It is seen in patients with meningitis, subarachnoid hemorrhage, late-stage syphilis and trauma. Abducent nerve paralysis results in the following defects: convergent strabismus and horizontal diplopia.

2. Internuclear ophthalmoplegia(Figure 1 - 2) There is damage to the medial longitudinal fasciculus (MLF) between the abducent and oculomotor nuclei. It causes medial rectus palsy on attempted lateral conjugate gaze and monocular horizontal nystagmus, while the adduction with convergence preserves. This syndrome is most commonly seen in multiple sclerosis.

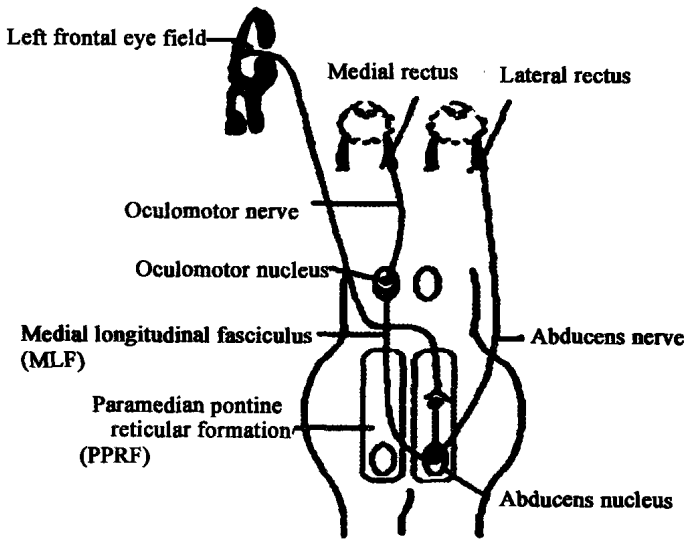

Figure 1 - 2 Internuclear ophthalmoplegia

3. One-and-a-half syndrome It consists of lesions in the bilateral MLF and the unilateral abducent nucleus. On attempted lateral conjugate gaze, the only muscle that preserving functions is the intact lateral rectus.

4. Argyll Robertson pupil It is the absence of a miotic reaction to light, both direct and consensual, with the preservation of a miotic reaction to near stimulus (accommodation-convergence). It occurs in syphilis and diabetes.

5. Horner's syndrome This syndrome is caused by transection of the oculosympathetic pathway at any level. It consists of miosis, ptosis, apparent enophthalmos, and anhidrosis.

6. Herniation of the cerebellar incisura tentori (or temporal lobe hernia) When supratentorial mass lesions produce herniation of the medial portion of the temporal lobe (the uncus) across the cerebellar tentorium, thus exerting direct pressure on the rostral brainstem, signs of oculomotor nerve compression such as ipsilateral pupillary dilation,

impaired adduction of the eye and loss of reactivity to light.

7. Diabetes mellitus It often affects the oculomotor nerve, damages the central fibers but spares the pupilloconstrictor fibers.

The trigeminal nerve (CN Ⅴ)

【Anatomy】

The trigeminal nerve has three divisions (Figure 1-3): ophthalmic, maxillary, and mandibular. The ophthalmic nerve lies in the wall of the cavernous sinus. It enters the orbit through the superior orbital fissure and innervates the forehead, dorsum of the nose, upper eyelid, orbit (cornea and conjunctiva), and cranial dura. The ophthalmic nerve mediates the afferent limb of the corneal reflex. The maxillary nerve lies in the wall of the cavernous sinus and innervates the upper lip and cheek, lower eyelid, anterior portion of the temple, oral mucosa of the upper mouth, nose, pharynx, gums, teeth and palate of the upper jaw, and cranial dura. It exits the skull through the foramen rotundum. The mandibular nerve exits the skull through the foramen ovale. Its sensory component innervates the lower lip and chin, posterior portion of the temple, external auditory meatus, and tympanic membrane, external ear, teeth of the lower jaw, oral mucosa of the cheeks and floor of the mouth, anterior two-thirds of the tongue, temporomandibular joint, and cranial dura. The motor component of the trigeminal nerve accompanies the mandibular nerve through the foramen ovale. It innervates the muscles of mastication, mylohyoid, anterior belly of the digastric, and tensors tympani and veli palatine. It innervates the muscles that move the jaw, the lateral and medial pterygoids.

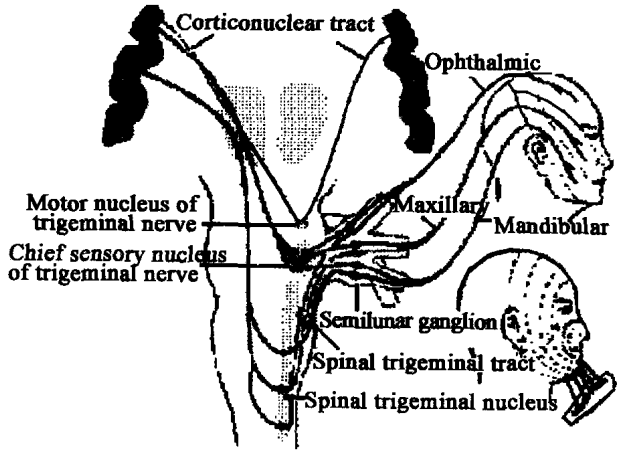

Figure 1-3 Anatomical bases of the trigeminal nerve

【Clinical correlation】

Lesions result in the following neurologic deficits.

(1) Loss of general sensation (hemianesthesia) from the face and mucous membranes of the oral and nasal cavities.

(2) Loss of the corneal reflex.

(3) Paralysis of the muscles of mastication.

(4)Deviation of the jaw to the weak side.

(5)Paralysis of the tensor tympani muscle, which leads to hypoacusis.

(6)Trigeminal neuralgia, which is characterized by recurrent paroxysms of sharp, stabbing pain in one or more branches of the nerve. It usually occurs in people older than 50 years of age, and it is more common for women than for men. Carbamazepine is the drug of choice for idiopathic trigeminal neuralgia.

The Facial Nerve (CN Ⅶ)

【Anatomy】

The facial nerve (Figure 1-4) is a general somatic afferent (GSA), general visceral afferent (GVA), special visceral afferent (SVA), general visceral efferent (GVE), and special visceral efferent (SVE) nerve. It mediates facial movements, taste, salivation, lacrimation, and general sensation from the external ear. The facial nerve exits the brain stem in the cerebellopontine angle. It enters the internal auditory meatus and the facial canal. It then exits the facial canal and skull through the stylomastoid foramen. The GSA component has cell bodies located in the geniculate ganglion and innervates the posterior surface of the external ear through the posterior auricular branch of the facial nerve. The SVA component projects centrally to the spinal trigeminal tract and nucleus and it also innervates the taste buds from the anterior two-thirds of the tongue. The GVE component is a parasympathetic component that innervates the lacrimal, submandibular, and sublingual glands. The SVE component arises from the facial nucleus, loops around the abducent nucleus of the caudal pons, and exits the brain stem in the cerebellopontine angle.

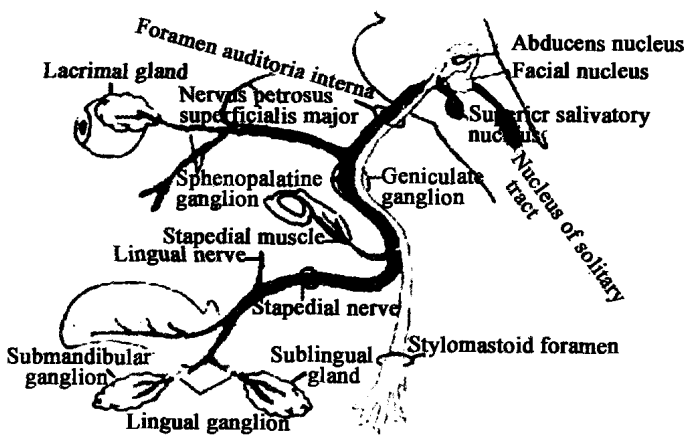

Figure 1-4 The functional components of the facial nerve

【Clinical correlation】

Lesions cause the following conditions.

(1)Flaccid paralysis of the muscles of facial expression.

(2)Loss of the corneal reflex, which may lead to corneal ulceration.

(3)Loss of taste from the anterior two-thirds of the tongue, which may result from

damage to the chorda tympani.

(4)Hyperacusis.

(5)Bell's palsy(peripheral facial paralysis), which is caused by trauma or infection. Peripheral facial paralysis shows that the wrinkles on the forehead in the affected side disappear, the ipsilateral eye can't shut voluntarily, the ipsilateral nasolabial fold becomes smooth (when a smile is attempted the angle of the mouth is drawn up on the unaffected side) and the protruded tongue deviates to affected side because of the action of the genioglossus in the unaffected side.

(6)Supranuclear (central) facial palsy, which results in contralateral weakness of the lower face, with sparing of the upper face. Supranuclear facial palsy shows that the nasolabial fold in the unaffected side becomes smooth but the bilateral wrinkles on the forehead exist. When a smile is attempted the angle of the mouth is drawn up on the affected side, the protruded tongue deviates to unaffected side but without atrophy of two sides of the tongue.

(7)Bilateral facial nerve palsies, which occur in the Guillain-Barré syndrome.

The vestibulocochlear nerve (CN Ⅷ)

The vestibulocochlear nerve has two functional divisions: the vestibular nerve, which maintains stability and balance, and the cochlear nerve, which mediates hearing. It exits the brain stem at the cerebellopontine angle and enters the internal auditory meatus. It is confined to the temporal bone.

The vestibular nerve

【Anatomy】

Its first-order sensory bipolar neurons are located in the vestibular ganglion in the fundus of the internal auditory meatus. The nerve projects its peripheral processes to the hair cells of the cristae of the semicircular ducts, the utricle and the saccule. It also projects its central processes to the four vestibular nuclei of the brain stem and the flocculonodular lobe of the cerebellum.

【Clinical correlation】

Lesions result in disequilibrium, vertigo, and nystagmus.

The cochlear nerve

【Anatomy】

The cochlear nerve's first-order sensory bipolar neurons are located in the spiral (cochlear) ganglion of the modiolus of the cochlea, within the temporal bone. The cochlear nerve projects its peripheral processes to the hair cells of the organ of Corti and projects its central processes to the dorsal and ventral cochlear nuclei of the brain stem.

【Clinical correlation】

Destructive lesions cause hearing loss (sensorineural deafness). Irritative lesions can cause tinnitus. An acoustic neuroma (schwannoma) is a Schwann cell tumor of the cochlear nerve that causes deafness.