ISSN 1753 - 044X

IJOCS

DOCENDO AC DISCENDO

Volume 3 Issue 3
September 2009

INTERNATIONAL JOURNAL OF CLINICAL SKILLS

A Peer Reviewed International Journal for the Advancement of Clinical Skills

- 'docendo ac discendo' - 'by teaching and learning'

In this issue:

Executive Board

International Journal Of Clinical Skills
P O Box 56395
London
SE1 2UZ
United Kingdom

E-mail: info@ijocs.org
Web: www.ijocs.org
Tel: +44 (0) 845 0920 114
Fax: +44 (0) 845 0920 115

Published by SkillsClinic Ltd.

Acknowledgements

I would like to take this opportunity to show appreciation to all those involved with the production of the International Journal of Clinical Skills (IJOCS). Many thanks to all members of the Editorial and Executive Boards. A special thank you Dr Ronald Woro and Dr Dora Affam for their inspirations.

Our sincere gratitude for the kind support from Sir Liam Donaldson, the Chief Medical Officer for England, United Kingdom.

The International Journal of Clinical Skills looks forward to contributing positively towards the training of all members of the healthcare profession.

Contents

Editorial Board for the International Journal of Clinical Skills

Disclaimer & Information

Foreword

A Message from the Chief Medical Officer for England, United Kingdom

The systematic and safe acquisition of high quality clinical skills is an essential part of modern medical training as highlighted in my Annual Report published in March 2009. I wish the International Journal of Clinical Skills every success in highlighting research and knowledge in this important area.

Sir Liam Donaldson
The Chief Medical Officer for England

Examination of the patient with a brainstem lesion

Aravinthan Varatharaj
Final Year Medical Student
University of Oxford

William Rattenberry
Final Year Medical Student
University of Oxford

Correspondence:
Aravinthan Varatharaj
University of Oxford
Pembroke College
Oxford
OX1 1DW
UK

E-mail: aravinthan.varatharaj@pmb.ox.ac.uk
Tel: +44 (0) 845 0920 114
Fax: +44 (0) 845 0920 115

Keywords:
Brainstem
Neurology
Examination
Cranial nerves

Abstract

Background: Examination of the patient with a brainstem lesion may seem complicated but is a useful skill to learn.
Objective: To provide a coherent system by which to examine patients with brainstem lesions.
Methods: The authors provide a concise review of the important aspects of brainstem anatomy and describe the clinical features that allow the localisation of lesions. A brief outline of possible causes is also discussed.
Outcome: Readers should be able to apply the methods described to perform a logical examination of the patient with a brainstem lesion.

Introduction

Clinical neurology is founded on the principles of logical history taking and examination. Although non-specialists may practice the application of these principles to lesions in the cerebral cortex, spinal cord and peripheral nervous system, the brainstem is often covered in less detail. This article aims to show that localising lesions in the brainstem is not a special art, but a useful skill which can be achieved by combining a knowledge of the salient features of brainstem anatomy with the logical application of the principles of clinical neurology. The objectives of this paper are shown in Box 1.

Box 1: Learning objectives

- recognise that a lesion is in the brainstem
- identify the side of the lesion
- deduce position in the midbrain, pons or medulla
- suggest if the lesion is medial or lateral
- describe likely causes

Applied anatomy of the brainstem

The brainstem contains several structures which make it of special importance (Box 2). Multiple structures in a compact space mean that brainstem pathology often produces multiple signs. The biggest clue is usually cranial nerve involvement.

Box 2: Key structures in the brainstem

The main three
descending pathways (corticospinal)
ascending pathways (dorsal column and spinothalamic)
cranial nerve nuclei III-XII

Also consider
cerebellar pathways
autonomic control centres
special nuclei and pathways concerned with eye movements
'diffuse systems' involved in consciousness

It is convenient to think of the brainstem in sets of three zones. The midbrain, pons and medulla are three zones from cranial to caudal ('top' to 'bottom'). We can also imagine three zones from ventral to dorsal ('front' to 'back') which contain the 'main three' features listed in Box 2. The corticospinal pathway is ventral, the dorsal column and spinothalamic pathways are in the middle, and the cranial nerve nuclei are dorsal [1].

Descending pathways

Corticospinal pathway

Anatomy: The ***corticospinal pathway*** conveys voluntary motor commands through upper motor neurons (UMN) which descend from the motor cortex through the brainstem, decussate in the lower medulla (the medullary 'pyramids') and then pass down the lateral columns of the spinal cord. These fibres terminate in the anterior horn where they synapse with lower motor neurons (LMN) which exit the spinal cord [2].

Clinical correlate: Brainstem lesions above the medullary pyramids may damage the corticospinal pathway before it has decussated, resulting in signs of an UMN lesion on the contralateral half of the body.

Ascending pathways

Dorsal column pathway

Anatomy: The ***dorsal column pathway*** conveys sensory information regarding light touch, proprioception and vibration. The pathway ascends in the ipsilateral dorsal column of the spinal cord, decussates behind the pyramids in the lower medulla and then ascends to the thalamus [2].

Clinical correlate: Brainstem lesions above the lower medulla may damage the dorsal column pathway after it has decussated, resulting in loss of light touch, proprioception and vibration sense on the contralateral half of the body.

Spinothalamic pathway

Anatomy: The ***spinothalamic pathway*** conveys sensory information regarding pain and temperature. Fibres decussate in the spinal cord around the level at which they entered, then ascend in the anterolateral column and pass up through the brainstem into the thalamus [2].

Clinical correlate: Brainstem lesions may damage the spinothalamic pathway after it has decussated, resulting in loss of pain and temperature sensation on the contralateral half of the body.

Cranial nerve nuclei

Anatomy: The ***cranial nerves III - XII*** have their nuclei arranged in approximate order down the brainstem, with motor nuclei medial to the sensory nuclei, as shown in Figure 1. The nerves emerge ventrally and supply structures on the ipsilateral side, with the exception of IV, which emerges dorsally and decussates behind the brainstem to supply the contralateral superior oblique muscle [2].

Clinical correlate: Brainstem lesions may cause cranial nerve abnormalities, as shown in Table 1. By identifying the levels of abnormality, the upper and lower bounds of the lesion may be determined.

Figure 1: schematic diagram of cranial nerve nuclei in the brainstem

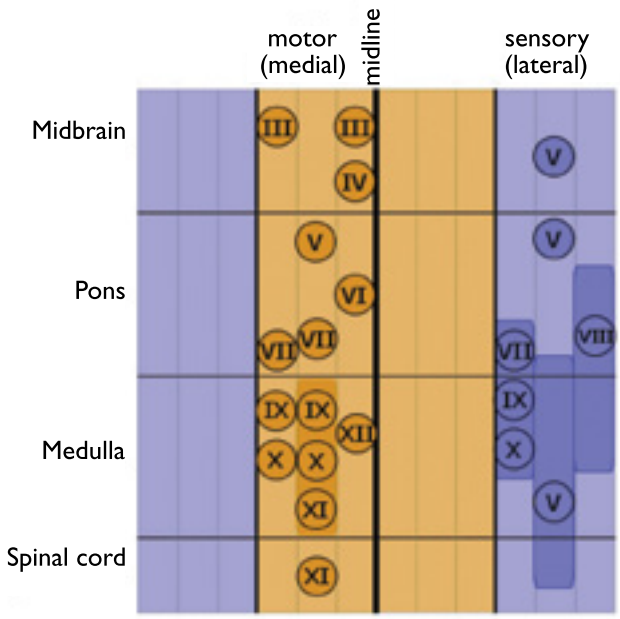


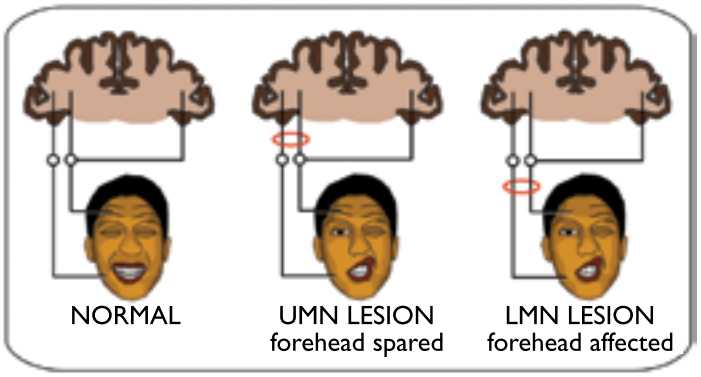
Table 1: Clinical features of cranial nerve lesions in the brainstem. IX and XI are excluded due to difficulties in examination.
*The afferent and efferent pathways of the corneal reflex are V and VII respectively, and there is a consensual component such that stimulation of one eye elicits closure of both.

	Lesion in nucleus	Clinical features
Midbrain	III	ipsilateral third nerve palsy; ptosis, fixed dilated pupil, and paralyses of eye movements with sparing of abduction (VI) and depression (IV)
	IV	fourth nerve palsy in contralateral eye; inability to look towards nose
Pons	V (principal)	loss of light touch and vibration sensation on ipsilateral half of face. Absence of corneal reflex, but can be elicited by stimulating opposite eye if ipsilateral VII is intact*
	V (motor)	weakness of muscles of mastication. Brisk jaw jerk if the lesion involves only corticobular fibres above the nucleus
	VI	ipsilateral sixth nerve palsy; inability to abduct eye
	VII	ipsilateral facial palsy, of LMN type if lesion is in or below nucleus, of UMN type if lesion is above nucleus. Absence of corneal reflex, but stimulation elicits blink in opposite eye if ipsilateral V is intact*
	VIII	nystagmus, vertigo, nausea, vomiting, loss of hearing
Medulla	V (spinal)	loss of pain and temperature sensation on ipsilateral half of face
	X	dysarthria, dysphagia, asymmetrical palatal elevation (uvula points away from side of lesion)
	XII	paralysis of ipsilateral half of tongue; on protusion deviates to that side

The motor nuclei are controlled by descending fibres from the motor cortex, the **corticobulbar pathway**. These fibres decussate roughly at the level of the nucleus they supply. In the same way that a UMN lesion in the corticospinal tract gives brisk tendon reflexes, a lesion to corticobulbar fibres supplying the trigeminal (V) motor nucleus gives a brisk jaw jerk. Such a lesion must be in the midbrain or above.

The corticobulbar innervation is largely bilateral, each nucleus receiving fibres from left and right motor cortices. VII is a special case because the parts of the nucleus supplying the lower face receive innervation from the contralateral motor cortex only. As shown in Figure 2, this means that a LMN lesion paralyses the whole contralateral face whereas a UMN lesion spares the forehead. As the cranial nerve nuclei are dorsal, the facial nerve has to travel forward through the brainstem before it exits the ventral surface, and therefore it is possible for a lesion inside the pons to cause an LMN facial palsy.

Figure 2: UMN and LMN palsy of facial (VII) nerve. The patient is being asked to screw up his eyes and show his teeth



Cerebellar pathways

Anatomy: Fibres from the cerebellum interweave through the brainstem via the superior, middle and inferior cerebellar peduncles, whilst the spinocerebellar pathway ascends through the brainstem and conveys proprioceptive information to the cerebellum [2].

Clinical correlate: Brainstem lesions may produce an ipsilateral cerebellar ataxia.

Autonomic functions

Anatomy: The brainstem contains numerous autonomic control centres. Of most clinical relevance is the lateral medulla, which contains the descending fibres of the sympathetic system.

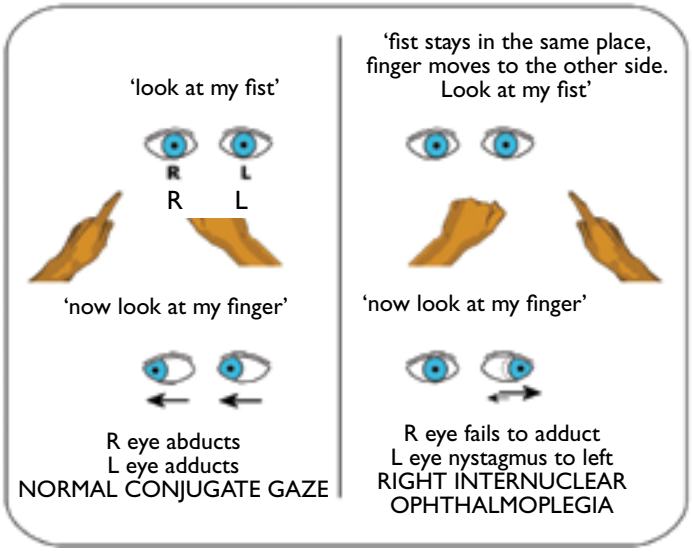
Clinical correlate: Lesions in the lateral medulla (eg. Wallenberg's syndrome) can damage descending sympathetic pathways. This may manifest as Horner's syndrome, with ipsilateral ptosis, meiosis (small pupil) and facial anhidrosis (lack of sweating).

Special nuclei and pathways for eye movements

Anatomy: The paramedian pontine reticular formation (PPRF) lies next to the abducens (VI) nucleus in the medial pons, and manages horizontal gaze. The vertical gaze centre and the vergence centre (which converges the eyes, for example whilst reading) lie in the medial midbrain, next to the oculomotor (III) nucleus. The medial longitudinal fasciculus (MLF) connects all these special nuclei with the nuclei of III, IV and VI to allow the co-ordinated control of eye movements [2].

Clinical correlate: Lesions to the PPRF (medial pons) or to the vertical gaze and vergence centres (medial midbrain) cause specific gaze palsies. Complex nystagmus featuring upward or rotatory movements may also be present. Lesions to the MLF result in internuclear ophthalmoplegia, manifest as slowed or absent adduction of the eye on the same side as the lesion, with nystagmus in the opposite eye as it attempts to abduct (Figure 3).

Figure 3: Internuclear ophthalmoplegia, in this case with a lesion in the right MLF. By asking the patient to look quickly between finger and fist, it is possible to elicit subtle slowing of adduction



Diffuse systems

Discussion of these features is outside the scope of this paper, however, they are of enormous importance as lesions may result in coma and brainstem death.

Causes of brainstem lesions

In the 19th century, Adolf Wallenberg and numerous other neurologists were busy applying their names to describe newly recognised syndromes of brainstem lesions. At this time *meningovascular syphilis* was a common cause of brainstem pathology. Today, *cerebrovascular* disease is the most common cause. Pathology in specific vascular territories results in recognisable syndromes [3], some of which are detailed in Table 2.

Table 2: Vascular syndromes in the brainstem

Vessel	Territory	Structures affected
posterior inferior cerebellar artery (Wallenberg's syndrome)	cerebullum lateral medulla	cerebullar pathways spinothalamic tract sympathetic tract IX and X V
superior cerebellar artery	cerebellum lateral midbrain	sympathetic tract spinothalamic tract
anterior inferior cerebellar artery	cerebellum anterolateral pons	sympathetic tract spinothalamic tract emerging VII nerve V nerve nucleus
basilar artery - paramedian branch occlusion	midbrain, pons and/or medulla	*midbrain:* III nerve, red nucleus *pons:* VI nerve, VII nerve, medial lemniscus *medulla:* pyramidal tract, medial lemniscus, XII nerve

The brainstem is susceptible to demyelination in *multiple sclerosis*. The median longitudinal fasciculus (MLF) is particularly so; therefore the presence of internuclear ophthalmoplegia makes MS a probable diagnosis. When bilateral, this is almost pathognomonic of MS.

Other causes of brainstem lesions include metabolic disorders. One such is *Wernicke's encephalopathy,* where axonal and myelin damage due to thiamine deficiency produce a triad of ophthalmoplegia, ataxia and confusion [3]. *Central pontine myelinolysis* is a second metabolic disease affecting the brainstem. It is caused by the rapid correction of hyponatraemia. It can present with bulbar weakness and tetraparesis and may develop into *locked-in syndrome*, coma and death.

Tumours of the brainstem are rare but should be considered in a patient with brainstem signs who is young or who has neurofibromatosis type I. Signs of raised intracranial pressure may also be present [3].

Congenital diseases of the brainstem include syringobulbia. This is a syringomyelic cavity that extends into the brainstem. Another congenital condition is the Chiari malformation of the medullary-spinal junction.

Box 3: Causes of brainstem lesions

- cerebrovascular disease
- demyelination
- metabolic
- tumours
- congenital

Conclusions

Brainstem lesions can be localised by combining a knowledge of anatomy with thorough clinical examination. When synthesised with an awareness of possible causes, the clinician is able to elucidate differential diagnoses. MRI has rapidly advanced our ability to accurately diagnose brainstem lesions but such technology requires the direction of a sound question based on clinical suspicion. Therefore the ability to localise brainstem lesions through the logical sequence of history and examination remains a useful skill.

References

1. Standring S, Ellis H, Healy J, Johnson D, Williams A. (2005). Gray's Anatomy, 39th Edition. London, Churchill Livingstone.
2. Joseph J T. (2005). Functional Neuroanatomy. New York, Wiley-Liss.
3. Patten J. (1996). Neurological Differential Diagnosis. London, Springer.

If you would like to subscribe to IJOCS, please contact subscription@ijocs.org

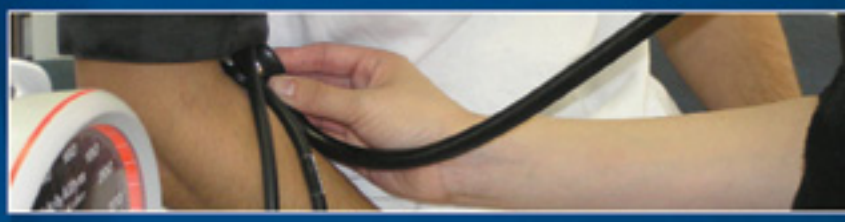
INTERNATIONAL JOURNAL OF CLINICAL SKILLS

IJOCS

DOCENDO AC DISCENDO

If you wish to submit material for publication, please email info@ijocs.org

Clinical Skills Lab (CSL)



The Clinical Skills Lab database will comprise information on over 200 clinical skills, broadly separated into:

- ➔ History taking skills
- ➔ Communication skills
- ➔ Clinical examination/interpretation skills
- ➔ Practical skills

Not only will this valuable resource provide material to students as a learning tool and revision aid, for example, OSCEs, it will also offer educational materials for teachers from all disciplines, allowing some standardisation of practice. The Clinical Skills community will also be encouraged to contribute, making this database interactive.

CSL is a free not for profit database. Visit **www.ijocs.org** for access