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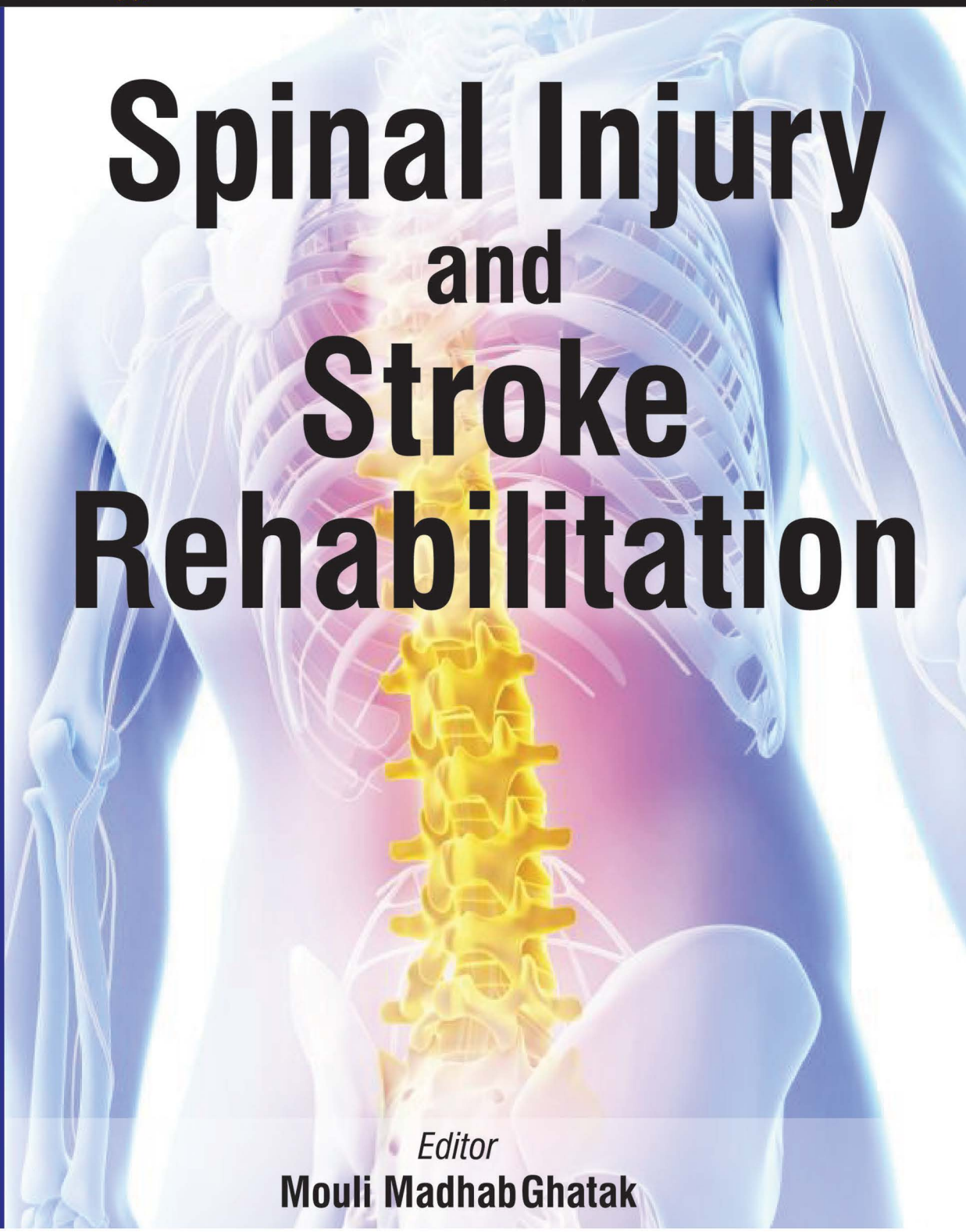
Spinal Injury and Stroke Rehabilitation



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2

Clinical Presentation of Stroke Syndromes

Souvik Sen

Introduction

Stroke is the sudden onset neurological deficit of vascular origin. It is broadly classified into ischemic and hemorrhagic type, and the clinical presentation depends on the type of stroke and the territory of the brain affected. Physicians involved in management of acute stroke are generally interested in distinguishing between these two categories of stroke, as well as differentiating the etiological and prognostic subtypes, as the management may differ based on them. Although sometimes it is possible to distinguish clinically between ischemic and hemorrhagic stroke, the method is often not reliable and frequently need the assistance of imaging assessment such as non-contrast computed tomography (CT) scan or magnetic resonance imaging (MRI).

In order to distinguish clinically between ischemic and hemorrhagic stroke at the bedside, scoring systems have been designed and validated such as the Allen score, the Siriraj stroke score and the Besson score. However since 80% of strokes are ischemic and only 20% hemorrhagic, it is hard to find a score that is reliable greater than 90%. Determination of the cause of ischemic stroke in patients is important for the clinical management. The diagnostic test pursued and the stroke prevention strategy advocated is often dependent on the suspected cause of ischemic stroke. Ischemic stroke etiology may be classified using the Trial of Org 10172 in acute stroke treatment (TOAST) criteria. Cardioembolic etiology was considered in patients, with a major brain artery or a branch of cortical artery occlusion, who had at least one cardiac source identified, according to the TOAST criteria (example-atrial fibrillation). Potential large-artery atherosclerosis the source of thrombosis or embolism was assessed by carotid ultrasound and/or MRA. Small vessel occlusive disease is a consideration when a patient present with one of the lacunar stroke syndrome, with a corresponding lacunar infarct on CT/MRI

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and absence of any identified cardioembolic or large artery atherosclerosis. Uncommon causes of stroke identified on testing include CNS vasculitis, a hypercoagulable state, and "watershed infarct" from a hypoperfusion state or a traumatic dissection of a cerebral blood vessel. Stroke may be classified according to their clinical presentation, using the Oxfordshire community stroke project (OCSP) criteria. In brief: Lacunar infarcts (LACI) are diagnosed if patients present clinically with pure motor, sensorimotor or ataxic hemiparetic findings, confirmed with an appropriate CT/MRI lesion; total anterior circulation infarcts (TACI) are diagnosed if patients present with a cortical disorder (such as dysphasia, dyscalculia) and/or homonymous hemianopsia and/or ipsilateral sensory or motor findings involving at least two regions (with CT/MRI confirmation); posterior circulation infarct (POCI) are diagnosed in the presence of cranial nerve palsies and contralateral motor or sensory deficits, or bilateral motor or sensory findings or disorders of eye movement or cerebellar dysfunction. Partial anterior circulation infarct (PACI) is diagnosed if patients present with only two of the three components of the TACI syndrome and CT/MRI confirmation of an appropriate lesion.

Substantial progress has been made in recent times in stroke prevention strategies and treatment of acute ischemic stroke. Most of the stroke prevention strategies can be advocated after an initial assessment and may be instituted at a small cost, such as antithrombotic therapy, management of stroke risk factors such as hypertension, diabetes, smoking and hypercholesterolemia. These strategies may be implemented with minimal resources available in the setting of primary care. The more advance strategies such as carotid endarterectomy, carotid angioplasty and stenting may only be accomplished in the setting of an advanced tertiary referral center with infrastructure for vascular surgery and interventional neuroradiology. The treatment of acute ischemic stroke includes strategies to promote recanalization of an occluded artery such as thrombolysis and mechanical revascularization and ancillary management of cardiac and pulmonary comorbidities, blood pressure management, management of cerebral edema, anticoagulation and antiplatelet therapy and prevention of complications such as deep venous thrombosis, bedsore, stress ulcers nutritional care. The approach requires a massive multidisciplinary team approach and infrastructure that is often expensive and unaffordable. It is feasible that these strategies may be partly or wholly implemented and with adequate rehabilitation may achieve modest to good stroke outcome. Stroke outcome measures have been reliably assessed utilizing clinically based stroke scales such as the NIH stroke scale (NIHSS), the Canadian Neurological Scale (CNS), the Scandinavian Neurological stroke scale (SCNSS) and European stroke scale (ESS). These scales have the greatest utility in assessing severity of the stroke, monitoring stroke outcome and possibly prognosticating stroke outcome.

In this chapter, we shall emphasize specifically three aspects of the clinical assessment of stroke syndromes. Early clinical diagnosis of acute ischemic and hemorrhagic stroke, clinical stroke syndromes and stroke scales used to assess and prognosticate stroke at the time of presentation.

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Clinical Diagnosis of Acute Ischemic and Hemorrhagic Stroke

The management of ischemic and hemorrhagic strokes is significantly different which makes it important to distinguish the two types of stroke in the acute setting. The typical symptoms of a hemorrhagic stroke are related to acute increase in intracranial pressure such as headache, vomiting, decrease in level of consciousness and occasionally signs of meningeal irritation. Lacunar stroke syndromes and resolution of stroke symptoms do not necessarily point towards an ischemic or hemorrhagic stroke.

Neuroimaging modalities such as a non-contrast CT or MRI can reliably distinguish between ischemic and hemorrhagic stroke. However, access to such diagnostic test are not available in a timely fashion to clinicians stressing the importance of clinical scoring system. In such a setting, there is need for a scoring system that is easy, reproducible and performed immediately after a stroke. The lack of a scoring system that is accurate and reliable, is a major setback to clinical scoring system. Nevertheless, such scoring systems have roles in assessment of the acute stroke. The three scoring systems those have used, are briefly summarized in **Table 2.1**.

Table 2.1: Clinical scores for differentiation of ischemic compared with hemorrhagic stroke

Allen score	*Siriraj stroke score*	*Besson score*
1. Apoplectic onset	1. Level of consciousness	1. History of hypertension
2. Level of consciousness	2. Vomiting	2. History of TIA
3. Plantar responses	3. Headache	3. History of hyperlipidemia
4. Diastolic blood pressure	4. Diastolic blood pressure	4. Alcohol consumption
5. Atheroma markers	5. Atheroma markers	5. Headache
6. History of hypertension		6. Peripheral arterial disease
7. Previous TIA or stroke		7. Atrial fibrillation on admission
8. Heart disease		8. Plantar response

The Allen score is a validated, weighted, linear, clinical score that was developed from a multivariate analysis of the clinical data in large groups of patients. It was developed to help clinicians without immediate access to CT or MRI. A score greater than 6 is more likely to be secondary to ischemic stroke than a score of less than 6. The Siriraj stroke score was developed and validated in Thailand. It uses five elements and is easier to use than the Allen score. The Besson score is a simple validated score with a high positive predictive value. Factors such as history of transient neurological deficit, peripheral arterial disease, hyperlipidemia and atrial fibrillation lead to the diagnosis of ischemic stroke. Alcohol consumption, extensor plantar response, a history of hypertension and headache lead to 'uncertain' diagnosis. The score is computed using the formula (2 × alcohol consumption) + (1.5 × plantar response) + (3 × headache) + (3 × hypertension) – (5 × transient neurological

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deficit) – (peripheral arterial disease) – (1.5 × hyperlipidemia) – (2.5 × atrial fibrillation) <1 translates to a likely diagnosis of nonhemorrhagic stroke. The three scores have not been validated in the same patient group. The scores have their best utility in prioritizing patients for neuroimaging study such as CT scan or MRI scan and is best used to conjunction with such imaging. CT scan remains the test of choice for early exclusion of hemorrhagic.

Clinical Syndromes and Arterial Distribution of Stroke

Stroke is defined as a sudden onset of focal neurological deficit that is vascular in origin and typically the symptoms last more than 24 hours. Traditionally symptoms lasting less than 24 hours have been referred to as Transient Ischemic Attack or TIA. With the advent of advanced neuroimaging modalities such as Magnetic Resonance Diffusion Weighted Imaging (MR-DWI) the traditional definition has been muddied by the "DWI-positive TIA". Independent of the definition the sudden onset of focal neurological signs such as hemianopsia, hemiparesis and aphasia should tip off towards a possibility of a cerebrovascular syndrome. Although the focus is on the suddenness in onset, it must be pointed out that occasionally it may be rapidly progressive or even fluctuating and stuttering in onset. Ischemic stroke typically manifests itself as a syndrome that relates to its localization in terms of the part of the brain it affects and its arterial supply. In the following section, we will describe some of the traditional stroke syndromes those are based on a particular arterial supply.

Middle Cerebral Artery Stroke Syndrome

Physical findings in patients with middle cerebral artery (MCA) stroke syndrome depend on the location of middle cerebral artery occlusion and dominant versus nondominant hemisphere involvement. In most of the persons, the left MCA territory controls speech and language function.

Proximal MCA occlusion leads to contralateral hemiplegia, eye deviation away from the side of the weakness, contralateral hemianopsia, and contralateral hemisensory loss. Proximal MCA occlusion of the dominant hemisphere causes global aphasia, whereas in the nondominant hemisphere causes impaired perception of deficits (anosognosia) resulting from the stroke. Superior division of MCA strokes lead to contralateral deficits with weakness of the upper extremity and face greater than the lower extremity. Inferior division of MCA stroke on either side can result in a superior quadrantanopsia or homonymous hemianopsia. In the dominant hemisphere, it can lead to Wernicke's aphasia. In the nondominant side, it may lead to a visual neglect. MCA stroke syndrome rarely cause decrease in level of consciousness at onset of symptoms, which if present should raise suspicion for other reasons such as seizures, swelling from edema or hemorrhagic transformation leading to herniation and brainstem compression, or brainstem ischemia from vertebrobasilar ischemia.

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The pattern of weakness sometimes provides a clue to a MCA territory stroke. Typically MCA stroke weakness produces weakness in the face and arm greater than in leg, with facial weakness of the upper motor neuron type (affecting lower half of stroke only). The face, arm and leg weakness are all on the same side. Occasionally, the face arm and leg are densely paralyzed suggestive of internal capsular involvement supplied by the lenticulostriate branches of the proximal MCA. Uncommonly the presentation is of faciobrachial paresis (the weakness of the lower face) and the upper extremity distal hand affected greater than the proximal forearm. Such deficits result from stroke in the insula and operculum.

Unilateral movement disorders such as hemiathetosis, hemichorea, and hemidystonia are rarely seen MCA stroke syndromes and reflect, involvement of the basal ganglia.

Visual deficits: Field cut or hemianopsia can occur with MCA stroke syndromes. Quadrantanopsia can be attributed to a parietal infarct affecting a division of the MCA (superior or inferior) supplying the deep fibers of the upper or lower optic radiation respectively.

Neglect or inattention is MCA stroke syndrome reflecting ischemic injury to the parietal lobe. Two types of neglect are commonly tested. Visual and sensory neglect are tested by double simultaneous stimulation of the visual and sensory modalities. Patients with visual neglect will frequently ignore objects or person approaching the patient from that side. They may have difficulty in naming objects presented on the affected side. Motor neglect with underutilization of the side contralateral to cerebral insult appears much like a hemiparesis. Special efforts must be made by the examiner to encourage the patient to demonstrate strength. Typically, the patient has delayed withdrawal to noxious stimuli, fails to place the affected hand in the lap when seated, and falls heavily to the affected side with no apparent effort to minimize impact.

Left-MCA (dominant) stroke syndrome: The left cerebral hemisphere is dominant for speech and language in vast majority of right-handed individuals. Defining cerebral dominance for left-handed and ambidextrous individuals is more difficult.

Aphasia or language disturbance is typically seen in strokes resulting from the dominant MCA (left MCA in a right-handed person). They are broadly classified as global, expressive or nonfluent, receptive or fluent and conduction aphasias.

Global aphasia, refers to a large entire MCA territory stroke affecting both expressive and receptive components of speech. Typically, these patients are mute. Global aphasia needs to distinguish from anarthhria resulting from paralysis of the oropharyngeal muscles. Although the patients appear mute their comprehension is spared.

Broca's or expressive or motor aphasia, results in difficulty in expression of either spoken or written language. Comprehension of written and spoken language is spared. Typically, such form of aphasia is seen with strokes affecting the insula and frontoparietal operculum.

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Wernicke's or receptive or sensory aphasia, results in difficulty in comprehension of written and spoken language. Expression of either spoken or written language is spared resulting in a fluent speech with few understandable words and frequent paraphasic errors. It is caused most often by stroke affecting the lower division of the MCA bifurcation or one of its branches. The Wernicke's aphasia, localizes to a stroke affecting the dominant posterior temporal, inferior parietal, and lateral temporo-occipital areas.

Conduction aphasia results from stroke affecting the arcuate fasciculus connecting the motor and sensory area of speech. This hard to elucidate syndrome, both receptive and expressive component of speech are preserved. It is the inability to put the written or spoken language into action. They have difficulty in repetition of spoken sentences and often try to correct the misspoken words.

Specific Aphasia Syndromes

Alexia: It is the inability to recognize or comprehend written language. It is associated with lesion in the left occipitotemporal cortex.

Agraphia: It is inability to write, is associated with left occipital lesion.

Apraxia of speech: Results from inability to use the oropharyngeal and respiratory muscles necessary for speech resulting in a hesitant and telegraphic speech output. This syndrome is associated with lesion in the rolandic area.

Agrammatism is described when the individual uses fewer words to communicate an idea. This is associated with lesion in the Broca's area.

Transcortical aphasia: It is characterized by inability to repeat, may be motor, sensory or mixed, resulting from isolation of the individual speech areas in the cortex.

Apraxia refers to the inability to perform a previously learned tasks despite preserved strength, vision, and coordination. The condition is due to an insult to the dominant hemisphere.

The most common form of apraxia is ideomotor apraxia. On verbal command, the patient is unable to perform simple tasks, such as imitating the use of a common object such as combing the hair. Apraxia results from a stroke resulting in disconnection between the cortex containing plans for movement and the cortex responsible for execution.

Ideational apraxia results in inability to complete an idea and is tested by examining the ability to complete complex multistep tasks, such as taking a piece of paper, folding it into two and leaving it on the table.

Besides ideomotor and ideational apraxia, few specific form of apraxias have been described. Limb-kinetic apraxia results in impaired clumsy manipulation of objects in such tasks as combing one's hair. Oral-buccal-lingual apraxia results in ability to perform complex movements involving the described muscles.

In addition to the above-mentioned symptomatology, it is important to recognize that the dominant hemispheric strokes more common than the

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nondominant hemisphere strokes are associated with depression. This often bears impact on the overall prognosis as well as prognosis for rehabilitation.

Right MCA (nondominant) stroke: Strokes of the nondominant hemisphere can lead to behavioral abnormalities. The patients are confused, agitated and disoriented. General confusion and delirium often are more appreciated in patients with nondominant hemisphere stroke. The right hemisphere is responsible for attention, vigilance, and distinguishing stimuli. Such stroke presentations are predictive of an unfavorable long-term outcome after rehabilitation.

Extinction describes inattention to one stimulus when double simultaneous stimuli are applied. Generally, the ignored stimulus is on the left side. The stimulus may be visual or auditory or tactile.

Impersistence is used to describe an inability to persist in performing motor tasks and often is accompanied with visuomotor and visuospatial deficits. This indicates an unfavorable rehabilitation outcome.

Dressing apraxia is seen with right-hemispheric stroke and results in difficulty distinguishing right from left and up from down. The patient is unable to dress without assistance despite having no apparent weakness or coordination difficulties that would prevent performing this task.

Memory deficit, which occurs when individuals become lost in familiar surroundings, often accompanies nondominant hemisphere stroke. Confabulation of information is due largely to inability to recognize errors, disinhibition, and memory deficits.

Constructional apraxia defines difficulty in manipulating objects in space. This kind of apraxia can be appreciated by having affected patients copy designs or build-3 dimensional models.

Aprosody, lack of intonation in speech, and affective agnosia refer to the inability to comprehend emotional intonation of speech. The two deficits correlate with stroke in the right temporoparietal region.

Lacunar Stroke Syndrome

The five classical lacunar syndromes have symptoms that are relatively specific to it. Occasionally, cortical infarcts and intracranial hemorrhages can mimic a lacunar syndrome. Cortical signs and symptoms (e.g. hemianopsia, aphasia, neglect) are absent.

Pure motor stroke/hemiparesis: This is the most common of all the lacunar syndromes. It consists of hemiparesis or hemiplegia that typically affects the face, arm, and leg equally. Sensory symptoms, dysarthria and dysphagia may also be present. The lacunar stroke is usually present in the posterior limb of the internal capsule or the basis pontis.

Ataxic hemiparesis syndrome: This is the next most common lacunar syndrome, consisting of weakness and clumsiness on one side of the body. The onset of symptoms may stutter over hours or days. The most frequent site of infarction is the posterior limb of the internal capsule, basis pontis, and corona radiate.

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Dysarthria clumsy hand syndrome: The main symptoms are dysarthria and clumsiness of the hand, which often is most prominent when the patient is writing. It results from a small infarct in the dorsal basis pontis just below the medial lemniscus.

Pure sensory stroke: This syndrome consists of persistent or transient numbness and/or paresthesias (pins and needles) on one side of the body. Occasionally, it is associated with pain, burning, or other unpleasant sensation. The stroke is usually in the thalamus (VPL and VPM nuclei).

Mixed sensorimotor stroke: This syndrome consists of a combination of hemiparesis and sensory impairment on one side of the body.

The stroke is usually in the thalamus and adjacent posterior limb of the internal capsule.

Anterior Cerebral Artery Syndromes

Stroke in the anterior cerebral artery (ACA) territory is uncommon, commonly resulting from atherothrombosis of the proximal segment of the ACA. The ACA supplies the whole of the medial surfaces of the frontal and parietal lobes, majority of the corpus callosum, the frontobasal cerebral cortex, the anterior diencephalons, and the deep structures including the head of the caudate nucleus. ACA strokes often go unrecognized because of paucity of detectable signs and symptoms. ACA strokes can lead to weakness in the legs typically out of proportion to leg weakness, apathy and abulia. It can cause personality changes and urinary incontinence in bilateral ACA infarcts. It is not uncommon to have an anatomical variation where a single ACA supplies both sides of the brain. In such a case, ACA stroke can result in paraparesis.

Anterior choroidal artery (AChA) is a branch of the ACA. It supplies the lateral thalamus and posterior limb of the internal capsule. AChA stroke is rare and can result in hemiparesis and hemisensory loss. It also supplies the lateral geniculate body resulting in certain unusual homonymous field abnormalities.

The "recurrent artery of Heubner" is a branch of the ACA that supplies the head of the caudate nucleus and the adjacent internal capsule. Stroke in this specific distribution can result in hemiparesis.

Internal Carotid Artery Syndromes

The internal carotid artery (ICA) supplies the MCA, ACA and less commonly a fetal posterior cerebral artery (PCA). Occlusion of ICA commonly results from atherothrombosis, less commonly from dissection, embolism and other causes. This commonly results in a combination of MCA syndrome and ACA syndrome with homonymous hemianopsia.

Ophthalmic artery is a branch of the ICA. Ischemia in the distribution of the ophthalmic artery is transient in the setting of symptomatic internal carotid artery occlusion (i.e. transient monocular blindness) is common, but central retinal artery ischemia is relatively uncommon, presumably because of the efficient collateral supply.

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Vertebrobasilar Insufficiency Syndromes

Patients with vertebrobasilar artery thrombosis typically have a waxing and waning course of symptoms. When caused by atherothrombosis, it is frequently preceded by prodrome that includes TIAs and rarely a "herald hemiparesis". In contrast, embolic events are sudden, without prodrome or warning, with acute and dramatic presentation. Commonly reported symptoms associated with the vertebrobasilar insufficiency (VBI) include vertigo, nausea, vomiting decrease in the level of consciousness, oculomotor signs, dysarthria, dysphagia, dysphonia, lower motor neuron pattern (both upper and lower halves) weakness of facial muscles and tongue, sensory loss, ataxia, crossed motor weakness and sensory loss, bladder or bowel incontinence and visual-field defects. Common clinical findings observed in patients include abnormal level of consciousness and hemiparesis or quadriparesis, which is usually asymmetric. Papillary abnormalities and oculomotor signs are common, and bulbar manifestations, such as facial weakness, dysphonia, dysarthria, and dysphagia, occur less commonly.

Oculomotor signs are usually reflex involvement of the abducens nucleus, the horizontal gaze center located in the pontine paramedian reticular formation (PPRF), contiguous to the abducens nucleus, and/or the medial longitudinal fasciculus (MLF). Lesions to these structures result in ipsilateral lateral gaze or conjugate gaze palsy. Ocular bobbing is described as a brisk downward movement of the eyeball with a subsequent return to the primary position. This deficit localizes the lesion to the pons. Other reported signs of pontine ischemia include ataxia and tremor associated with mild hemiparesis. The signs described can occur in different combinations presenting a diagnostic challenge in lesion localization.

Certain constellations of findings may serve as clues for narrowing down the search, including the following examples. Midbrain syndromes (Coarse tremor on one side of the body, cranial nerve III lesion or vertical gaze palsy), Pontine syndrome (Clue-Cranial nerve VI lesion, horizontal gaze palsy, or VII nerve palsy), medullary syndrome (crossed pain and temperature loss, Horner syndrome, ataxia, paralysis of the tongue, soft palate, vocal cord or sternocleidomastoid muscle), posterior cerebral artery (macular sparing hemianopsia).

A variety of specific neurologic syndromes have been described based on constellations of findings. Some examples are listed below:

Wallenberg (lateral medullary) syndrome: It is the most common of all the posterior circulation syndromes and is due to vertebral artery occlusion. Patients present with nausea, vomiting, vertigo, dysarthria and gait unsteadiness. Ipsilateral clinical features include ataxia and dysmetria, due to damage of the inferior cerebellar peduncle and cerebellum; Horner syndrome (including ptosis, miosis, hypohidrosis or anhidrosis, enophthalmos), due to the damage to descending sympathetic fibers; facial pain and temperature loss; reduced corneal reflex from damage to the descending spinal tract and trochlear nerve nucleus resulting in nystagmus, hypoacusis (cochlear

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nucleus), dysarthria, dysphagia, paralysis of the pharynx, palate, and vocal cord; and loss of taste from the posterior third of the tongue (nuclei or fibers of glossopharyngeal and vagus nerves). The prognosis of patients with the lateral medullary syndrome is usually benign with good functional outcome. However, patients may die in the acute phase from aspiration pneumonia, and death has been reported from sleep apnea in a number of cases.

Dejerine (medial medullary) syndrome: This syndrome is due to occlusion of anterior spinal branches of the vertebral artery and it involves the pyramid, the medial lemniscus, and the hypoglossal nerve.

The clinical features include ipsilateral paresis of the tongue with deviation towards the lesion (lower motor neuron lesion of the hypoglossal nerve), contralateral hemiplegia with sparing of the face (corticospinal tract), and loss of ipsilateral vibration and proprioception (medial lemniscus).

Cerebellar infarction: A stroke involving the cerebellum may result in a lack of coordination, clumsiness, intention tremor, ataxia, dysarthria, scanning speech, and occasionally difficulties with memory and motor planning. Early diagnosis of cerebellar infarctions is important since swelling may cause brainstem compression or hydrocephalus.

Locked-in syndrome: This dramatic clinical syndrome occurs when there is an infarction of the upper ventral pons. Locked-in syndrome results from occlusion of the proximal and middle segments of the basilar artery, or hemorrhage involving that region, and also may be caused by trauma, central pontine myelinolysis, encephalitis, or tremor. Bilateral ventral pontine involving both corticospinal and corticobulbar tracts lead to quadriplegia. The patient is unable to speak, or produce facial movement (damage to the corticobulbar tracts) as well as look to either side (horizontal eye movement is impaired due to a lesion of bilateral abducens nerve nuclei). Because the tegmentum of the pons is spared, the patient's consciousness is preserved, with the patients fully awake, sensate, and aware. The only movements preserved are vertical eye movements and blinking. The patient is paralyzed completely and communicates only by blinking. Some recovery of facial muscle movement and horizontal gaze may occur with time or in an incomplete form of this syndrome.

Top of the basilar syndrome: This syndrome is the manifestation of upper brainstem and diencephalic ischemia caused by occlusion of the rostral basilar artery, usually due to an embolism. The syndrome is characterized by visual, oculomotor, and behavioral abnormalities, often without significant motor dysfunction. Rostral brainstem infarction produces oculomotor and pupillary signs that are identical to those in thalamic hemorrhage. Somnolence, vivid hallucinations and dreamlike behavior may also accompany rostral brainstem infarction. Temporal and occipital infarctions are frequently accompanied by hemianopia with distinctive characteristics, fragments of the Balint syndrome, amnestic dysfunction, and agitated behavior.

Internuclear ophthalmoplegia: Clinically, internuclear ophthalmoplegia (INO) is a horizontal gaze palsy resulting from a brainstem lesion affecting the MLF between the nuclei of abducens and oculomotor nerves, most

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commonly in the pons. When a patient with a lesion in the right MLF attempts to look to his/her left (i.e. away from the involved side), he/she shows no adduction of the right eye and full abduction of the left eye with the end-point abduction nystagmus. By the same logic, in the case of bilateral INO, there is no adduction to either side with nystagmus of the abducting eye in both directions. Convergence is preserved since both nuclei of oculomotor nerve and peripheral innervation of the medial recti muscles are intact. Since horizontal gaze requires coordinated activity of the ipsilateral oculomotor nerve and communication pathway (i.e. MLF) between the nuclei of oculomotor nerve (in the midbrain) and abducens nerve (in the pons) results in the inability of the eye ipsilateral to the lesion to adduct and contralateral eye to exhibit abduction nystagmus when looking away from the involved side. In elderly patients, INO is caused most often by occlusion of the basilar artery or its paramedian branches. In younger adults, it may occur due to multiple sclerosis, commonly with bilateral involvement and occasionally partial INO.

One-and-a-half syndrome: This syndrome is caused by a lesion affecting the PPRF and MLF simultaneously, resulting in ipsilateral conjugate gaze palsy and INO. The patient with a lesion in the ipsilateral PPRF or abducens nucleus and MLF connecting to the contralateral abducens nerve exhibits horizontal gaze palsy when looking towards the side of the lesion (one), and INO when looking away from the side of the lesion (half). Associated features may include vertical nystagmus, exotopia of the contralateral eye, and skew deviation. Vertical gaze and convergence generally are preserved. A patient with this syndrome is completely unable to move the ipsilateral eye, and he/she is able only to abduct the contralateral eye, with resulting nystagmus.

Millard-Gubler (Ventral pontine) syndrome: This syndrome occurs after paramedian infarction in the pons and results in ipsilateral lateral rectus palsy (abducens nerve) with diplopia, complete facial paresis (unilateral facial nerve palsy), and contralateral hemiparesis/hemiplegia (corticospinal tract involvement). It is also referred to as "crossed hemiplegia".

Raymond-Céstan (Upper dorsal pontine) syndrome: This syndrome is due to obstruction of flow in the long circumferential branches of the basilar artery. This occlusion results in ipsilateral ataxia and coarse intention tremor (indicating involvement of the superior and middle cerebellar peduncles), weakness of mastication and sensory loss in the face (suggesting sensory and motor trigeminal nuclei and tracts), and contralateral loss of all sensory modalities due to damage to medial lemniscus and spinothalamic tract) with or without facial weakness and hemiparesis (corticospinal tract). Horizontal gaze palsy also may occur.

Foville (Lower dorsal pontine) syndrome: This syndrome may result from lesions to the dorsal tegmentum of the lower pons. The patient exhibits ipsilateral paresis of the whole face (nucleus and fibers of facial nerve), horizontal gaze palsy on the ipsilateral side (PPRF ± abducens nucleus), and contralateral hemiplegia (corticospinal tract) and sensory loss.

Weber (Ventral midbrain) syndrome: Weber syndrome occurs with an occlusion of the median and/or paramedian perforating branches of the

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basilar artery. Typical clinical findings include ipsilateral oculomotor nerve palsy, ptosis, and mydriasis with contralateral hemiplegia.

Benedikt (Dorsal midbrain) syndrome: This syndrome is due to lesion in the midbrain tegmentum from occlusion of paramedian branches of either the basilar artery, PCA, or both. The patient demonstrates ipsilateral oculomotor palsy, ptosis and mydriasis (like in Weber syndrome), along with the contralateral involuntary movements, such as intention tremor, ataxia, or chorea (due to the involvement of the red nucleus).

Posterior cerebral artery infarct: The most common finding in occipital lobe infarction leading to contralateral hemianopsia with macular sparing. Clinical symptoms associated with occlusion of the PCA vary depending on the location of the occlusion and may include the thalamic syndrome, thalamic perforate syndrome, Weber syndrome, cortical blindness, and color blindness, failure to see to-and-for movements, verbal dyslexia, and hallucinations.

Clinical Stroke Scales

The introduction of specific modalities of treatment for acute stroke has emphasized the interest in the clinical measurement of stroke severity. A number of quantitative stroke scores have been devised to enable outcome measurement in clinical trials, prognosis stratification and assess quantitative changes in neurological status of stroke patients. The commonly used stroke scales are listed in **Table 2.2.**

Table 2.2: Clinical stroke scale

CNS	*SNSS*	*ESS*	*NIHSS*
1. Level of consciousness	1. Level of consciousness	1. Level of consciousness	1. Level of consciousness
2. Orientation	2. Eye movement	2. Comprehension	2. Orientation
3. Speech	3. Arm power	3. Speech	3. Comprehension
4. Motor weakness	4. Hand power	4. Visual field	4. Gaze
5. Motor response	5. Leg power	5. Gaze	5. Visual field
	6. Orientation	6. Facial movement	6. Facial palsy
	7. Speech	7. Arm position	7. Limb weakness
	8. Facial palsy	8. Leg raising	8. Ataxia
	9. Gait	9. Wrist extension	9. Sensory
		10. Finger strength	10. Language
			11. Neglect

Bibliography

1. Adams, et al. Guidelines for the early management of adults with ischemic stroke: A guideline from the American heart association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and

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Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. Circulation. 2007;115(20):e478-e534.
2. Celani, et al. Comparability and validity of two clinical scores in the early differential diagnosis of acute stroke. British Medical Journal. 1994;308(6945): 1674-6.
3. Chalela, et al. Magnetic resonance perfusion imaging in acute ischemic stroke using continuous arterial spin labeling. Stroke. 2000;31(3):680-87.
4. Coull, et al. Nderestimation of the early risk of recurrent stroke: evidence of the need for a standard definition. Stroke. 2004;35(8):1925-9.
5. Edwards, et al. Unified neurological stroke scale is valid in ischemic and hemorrhagic stroke. Stroke. 1995;26(10):1852-8.
6. Flossmann, et al. Reliability of clinical diagnosis of the symptomatic vascular territory in patients with recent transient ischemic attack or minor stroke. Stroke. 2008;39(9):2457-60.
7. Gérard Besson. Is it clinically possible to distinguish nonhemorrhagic infarct from hemorrhagic stroke? Stroke. 1995;26:1205-09.
8. Goldstein, et al. Improving the clinical diagnosis of stroke. Stroke. 2006;37(3): 754-5.
9. Goldstein, et al. Improving the reliability of stroke subgroup classification using the trial of org 10172 in acute stroke treatment (TOAST) criteria. Stroke. 2001;32(5):1091-7.
10. Hideaki, et al. Deteriorating ischemic stroke in 4 clinical categories classified by the Oxfordshire community stroke project. Stroke. 2000;31(9):2049-54.
11. Higashida, et al. Should interventional radiologists be involved in acute stroke intervention? Re: Emergency interventional stroke therapy: A statement from the American Society of Interventional and Therapeutic Neuroradiology and the Society of Cardiovascular and Interventional Radiology. Journal of Vascular and Interventional Radiology. 2001;12(2):145-6.
12. J Bogousslavsky, et al. The Lausanne stroke registry: Analysis of 1,000 consecutive patients with first stroke. Stroke. 1988;19:1083-92.
13. Jager HR. Diagnosis of stroke with advanced CT and MR imaging. British Medical Bulletin. 2000;56(2):318-33.
14. Kobayashi, et al. Oxfordshire community stroke project clinical stroke syndrome and appearances of tissue and vascular lesions on pretreatment CT in hyperacute ischemic stroke among the first 510 patients in the third international stroke trial (ist-3). Stroke. 2009;40(3):743-8.
15. Mallen C. Predicting the outcome of acute stroke: A prognostic score. Journal of Neurology, Neurosurgery, and Psychiatry. 1984;47:475-80.
16. MG Celani. Comparability and validity of two clinical scores in the early differential diagnosis of acute stroke. British Medical Journal. 1994;308:1674-6.
17. Pelz, et al. Stroke review: Advances in interventional Neuroradiology 2004. Stroke. 2005;36(2):211-4.
18. Peter D Schellinger. A standardized MRI stroke protocol comparison with CT in hyperacute intracerebral hemorrhage. Stroke. 1999;30:765-8.
19. Phillips, et al. Clinical diagnosis of lacunar stroke in the first 6 hours after symptom onset: Analysis of data from the glycine antagonist in neuroprotection (gain) Americas Trial. Stroke. 2007;38(10):2706-11.
20. Qureshi, et al. New grading system for angiographic evaluation of arterial occlusions and recanalization response to intra-arterial thrombolysis in acute ischemic stroke. Neurosurgery. 2002;50(6):1405-15.

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21. Sato, et al. Baseline NIH stroke scale score predicting outcome in anterior and posterior circulation strokes symbol. Neurology. 2008;(70):2371-7.
22. Thanvi, et al. Early neurological deterioration in acute ischaemic stroke: predictors, mechanisms and management. Postgraduate Medical Journal. 2008;84(994):412-7.
23. Toni, et al. Pure motor hemiparesis and sensorimotor stroke: Accuracy of very early clinical diagnosis of lacunar strokes. Stroke. 1994;25(1):92-6.
24. Wilterdink, et al. Effect of prior aspirin use on stroke severity in the trial of org 10172 in acute stroke treatment (TOAST). Stroke. 2001;32(12):2836-40.

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