Frontiers of Neurology and Neuroscience
Editor: J. Bogousslavsky
Vol. 30

Manifestations of Stroke

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Frontiers of Neurology and Neuroscience

Vol. 30

Series Editor

J. Bogousslavsky Montreux

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46 figures, 9 in color, and 14 tables, 2012

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Library of Congress Cataloging-in-Publication Data

Manifestations of stroke / volume editors, M. Paciaroni ... [et al.].
p. ; cm. -- (Frontiers of neurology and neuroscience, ISSN 1660-4431 ; v. 30)
Includes bibliographical references and indexes.
ISBN 978-3-8055-9910-8 (hard cover : alk. paper) -- ISBN 978-3-8055-9911-5 (e-ISBN)
I. Paciaroni, M. (Maurizio) II. Series: Frontiers of neurology and neuroscience ; v. 30. 1660-4431
[DNLM: 1. Stroke--complications. 2. Emergency Treatment--methods. 3. Stroke--diagnosis. 4. Stroke--therapy. W1 MO568C v.30 2012 / WL 356]
616.81--dc23

2011050950

Bibliographic Indices. This publication is listed in bibliographic services, including Current Contents® and Index Medicus.

www.karger.com
Printed in Germany on acid-free paper by Kraft Druck GmbH, Ettlingen
ISSN 1660–4431
e-ISSN 1662–2804
ISBN 978–3–8055–9910–8
e-ISBN 978–3–8055–9911–5

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Preface

What is a stroke? The latest definition by WHO is simply stated as 'a rapidly developing loss of brain function(s) due to disturbances of the blood supply to the brain'. Within this definition lies a wide variety of symptoms and signs that clinicians sometimes fail to detect. In fact, it is believed in the collective imagination and by many physicians that stroke is solely characterized by a loss of strength, language disturbances, and vertigo and/or sensitivity loss which lead to varying degrees of disability. Currently, emergency stroke management depends heavily upon stroke scores to quantify the damage and to speed up the diagnosis process. In fact, stroke management presently requires determining time of onset, the presence of vessel occlusions, in order to decide on thrombolysis treatment. Moreover, the development of high-resolution neuroimaging technologies has become the 'third' clinical eye of physicians. These are the hallmarks of acute stroke treatment, which are light-years away from the typical academic discussions we had with our mentors which focused on the question 'Where are the lesions'? Unfortunately, several important stroke syndromes are not taken into consideration in these currently used stroke scores and for this reason they tend to be overlooked and not treated. In the 21st century neurologists need to reach a compromise between contemplative neurology and current emergency stroke medicine. In this regard, every chapter deals with the principal clinical pictures of stroke syndromes in order to provide present and future stroke physicians a 'snapshots handbook'. Being so, the aim of this work is to provide an overview on current stroke syndromes which together with stroke scores will lead to more thorough assessments in emergency settings.

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Paciaroni M, Agnelli G, Caso V, Bogousslavsky J (eds): Manifestations of Stroke.
Front Neurol Neurosci. Basel, Karger, 2012, vol 30, pp 1–3

Motor Syndromes

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Abstract

Motor disturbances alone or associated with other focal deficits are the most common symptoms suggesting a neurovascular event. An appropriate clinical assessment of these signs and symptoms may help physicians to better diagnose and to both better treat and predict outcome. In this paper the main clinical features of motor deficit are described together with other motor-related events such as ataxia and movement disturbances.

During the first evaluation of conscious patients with a suspicion of stroke, the initial steps should include an assessment of motor performance as well as impairment. Regarding the latter, its distribution and whether it is isolated or not need to be accurately determined. We urge assessing the movements in each of the limbs while at same time looking for any spontaneous movements and asymmetries. When the patient is unable to cooperate we suggest eliciting movements with painful stimuli (sternum, inner end eyebrow, squeezing nail bed of a digit in each limb).

Most motor deficit profiles in stroke patients (75%), according to many studies, are characterized by uniform weaknesses of the hand, foot and hip [1, 2]. Other neurological conditions which need to be ruled out include: neglect, ataxia, apraxia and/or anosognosia. These groups of conditions may result in varying motor performance without any direct damage to the corticospinal structures. The main clinical patterns of motor syndromes, together with their probable topographic locations, are reported in table 1.

A faciobrachial motor weakness pattern is usually seen in events involving the superficial branch of the middle cerebral artery (MCA), while monoparesis may be more often observed in smaller infarcts of the cortex and centrum ovale [3]. Non-lacunar etiologies of stroke (large artery disease and cardioembolism) are the main causes of stroke not involving lower limb motricity.

The weakness distribution found in capsular or brainstem strokes reminds us of the local somatotopic motor pathway organization. The internal capsule leg-directed fibers can be found more dorsolaterally while the arm-directed fibers lie ventromedially. A proximal-distal clinical correlate in motor deficit patterns is the effect of the gradient in the corticospinal tract descending down the spinal cord. Specific motor deficits more often involve the axial musculature than intrinsic limb musculature. A mostly proximal limb paresis is more often observed in infratentorial lesions while the distal limb weakness pattern is more often characteristic of supratentorial events, and these two have different prognostic effects [4].

A relevant number of stroke victims develop a faciobrachial weakness without any involvement of the lower limb. This is generally due to

Table 1. Motor syndromes and more frequent vascular event location

Segmental weakness	Stroke location	Arterial territory
FUL	Deep infarct	MCA
FU	Superficial infarct	MCA
UL	Variable	ACA/VB
U	Superficial/deep	MCA/watershed
L	Superficial	ACA
F	Superficial/deep	MCA/VB

F = Facial; U = upper limb; L = lower limb; MCA = middle cerebral artery; ACA = anterior cerebral artery; VB = vertebrobasilar.

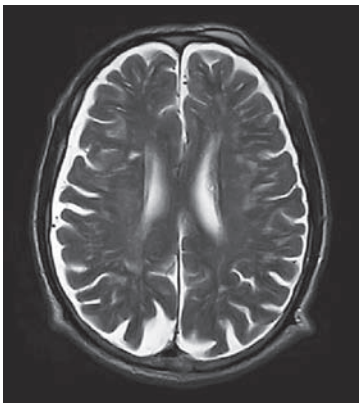

Fig. 1. Magnetic resonance study (T_2-weighted sequences) in a patient suffering from a minor lower facial paresis. The study demonstrated a new ischemic lesion in the supratentorial right hemispheric white matter.

an involvement of the MCA territory, but even if less frequent it can be seen also in basal ganglia and rarely in anterior cerebral artery (ACA) and brainstem infarctions.

A pure motor hemiparesis was first reported in the autopsy studies by Fisher in the 1960s and such clinical presentations later led to the development of the 'lacune' concept [5]. Taking into account various hospital-based registries, the prevalence of this clinical picture has been reported to be between 9 and 24% [1, 5, 6].

An isolated monoparesis is a rare symptom in stroke patients, around 1–2% of cases, and is often caused by either small artery disease or small hemorrhages [7]. Lower limb isolated distal weakness has also been reported to be associated with cortical infarcts [8].

An isolated facial paresis with abrupt onset and no clear involvement of limb motricity is an uncommon finding, generally due to damage of the internal capsule genu. Furthermore, strokes involving the corona radiata have been reported to lead to lower face palsy triggered by upper motor neuron damage (fig. 1). A larger facial paresis including the upper face may be found in pontine events. Such clinical presentations, when mimicking a partial Bell's palsy, from a diagnostic point of view are challenging for stroke physicians [9].

When motor syndromes are associated with an overlapping sensory loss the topography of the stroke is expected in the internal capsule. This is true also if an ataxic disturbance of the paretic limb is reported (ataxic hemiparesis), together with a pontine or corona radiate topography.

A segmental weakness involving contralateral limbs (without facial involvement) may be caused by ACA infarctions. While a distal leg predominant deficit associated with ACA events has been observed in 25% of cases. The ACA supply territory if damaged may lead to mono- or bilateral medial frontal damage and thereby provoking disturbances to upper extremities including bimanual weakness and lack of coordination.

Movement Disorders and Ataxia

Hypo- or hyperkinetic movement disorders may occur in acute stroke of which the more frequent forms include hemichorea and hemiballism.

In hypokinetic disorders related with stroke, vascular parkinsonism is the most widely reported even if this clinical entity is still a debated. While the clinical picture of an atypical parkinsonism is

usually associated with multiple vascular damage to neuroimaging, being so the condition of vascular parkinsonism should be ruled out. Any lack of response to standard antiparkinsonian drugs renders it difficult to distinguish between extrapyramidal disturbances such as those included in the group of multiple systemic atrophies. Other conditions even more rarely reported include: asterixis, dystonia, tremor and myoclonus [10, 11]. Usually, hyperkinetic stroke-related disturbances are transient and more often related to either basal ganglia or thalamic damages.

Ataxic disturbances have generally been reported in vascular cerebellar damage cases or those involving the cortico-ponto-cerebellar and/or dentate-thalamic pathways [12]. According to the traditional features of cerebellar structures, (i) archicerebellum, (ii) paleocerebellum and (iii) neocerebellum as parallel syndromes are useful. While the vermian involvement leads to both head and trunk-postural ataxia, an anterior lobe stroke will result in ataxia of stance and gait. Any damage of the cerebellar hemisphere is correlated with a major hypotonic compromise of limb movements, with asynergia and/or dysdiadochokinesis, as long as it is not associated with a concurrent weakness that may impede any movement analysis.

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Paciaroni M, Agnelli G, Caso V, Bogousslavsky J (eds): Manifestations of Stroke.
Front Neurol Neurosci. Basel, Karger, 2012, vol 30, pp 4–8

Sensory Syndromes

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Abstract

Somatosensory deficit syndromes represent a common impairment following stroke and have a prevalence rate of around 80% in stroke survivors. These deficits restrict the ability of survivors to explore and manipulate their environment and are generally associated with a negative impact on quality of life and personal safety. Sensory impairments affect different sensory modalities in diverse locations at varying degrees, ranging from complete hemianesthesia of multiple modalities to dissociated impairment of somatosensory submodalities within a particular region of the body. Sensory impairments induce typical syndromal patterns which can be differentiated by means of a careful neurological examination, allowing the investigator to deduce location and size of the underlying stroke. In particular, a stroke located in the brainstem, thalamus, and the corticoparietal cortex result in well-differentiable sensory syndromes. Sensory function following stroke can be regained during rehabilitation even without specific sensory training. However, there is emerging evidence that specialized sensory interventions can result in improvement of somatosensory and motor function. Herein, we summarize the clinical presentations, examination, differential diagnoses, and therapy of sensory syndromes in stroke.

Perception and interpretation of somatosensory information is a general requirement for human life. Mental registration and interpretation of sensory stimuli is dependent on information from several sensory systems. Hence, the somatosensory network is closely interlinked to all other sensory structures, including the higher functional areas in the brain, and the motor system. Due to this tight relationship, somatosensory function is not only susceptible to damages to somatosensory brain areas, but also vulnerable to impairments of other brain systems. Accordingly, most stroke survivors suffer several somatosensory deficits (body senses such as touch, temperature, pain, and proprioception). Reported prevalence rates appear to vary between 65 and 100% [1–4]. Even in patients diagnosed with pure motor stroke via neurological examination, sensory dysfunction was found in 88% of cases [3]. Impaired sensory function is often notably underdiagnosed, though it hinders the ability to explore and manipulate one's environment and negatively affects the quality of life as well as personal safety. Therefore, correct diagnosis and appropriate treatment of sensory syndromes has raised much attention in recent years.

Clinical Presentations

Stroke affects one or more of the sensory modalities in varying degrees, ranging from complete hemianesthesia of multiple modalities to dissociated impairment of somatosensory submodalities within a particular body region.

Studies report an impairment of elementary sensory modalities such as touch, pressure, pain, vibration, and temperature in 53–64% survivors after stroke [2]. Impaired proprioception occurs at a similar frequency [2, 5]. Moreover, the majority of studies consistently regard stereognosis (tactual object recognition) as being the most common somatosensory impairment following stroke [2, 6]. However, pure or predominant somatosensory symptoms in stroke patients are also not uncommon. These are reported as representing the most frequent lacunar syndromes [7–9]. In most cases, the lacuna was found in the thalamus [10–12], but also brainstem [13, 14], capsular [15], and parietal [6] lesions are described as causing predominantly somatosensory symptoms. The degree of impairment of sensory function correlates closely to stroke severity (NIHSS score) and extent of lesion [2, 5]. In contrast, larger strokes almost always result in non-sensory symptoms due to the tight relationship of the somatosensory network to other systems.

Although somatosensory impairment varies widely according to location of stroke within the CNS, the investigator can suspect stroke site via particular patterns of presentation:

Sensory impairment due to brainstem stroke results mostly from small infarcts or hemorrhages in the medulla or pons. Lateral brainstem strokes in medulla and pons often cause a loss of pain and temperature sensation. Most lateral, they affect the ipsilateral face and the contralateral lower body (type I of Stopford's classification [16]). Mediolateral lesions can affect only the upper part of the body (type II), while large strokes in both of these regions can result in a combination of crossed and unilateral pattern (type III).

Paramedian infarcts are found more frequently in the pons than in the medulla and affect elementary sensations (most often vibration and position sense) and often show dominance in the cheiro-oral or leg region. Somatosensory symptoms of the facial or perioral region due to paramedian pons lesions frequently occur bilaterally. Aside from sensory deficits, most patients also suffer dizziness and gait ataxia [13].

Sensory impairment owing to thalamic stroke is predominantly caused by lacunar infarcts in the ventroposterior nucleus of the thalamus, again mostly affecting elementary sensations showing a faciobrachiocrural distribution. A typical constellation of symptoms for thalamic strokes include numbness and paresthesia, although dysesthesia and pain also commonly occur. The latter can develop directly following stroke, or subacutely a few days later (2–15 days) [12].

Corticoparietal stroke mainly involves discriminatory modalities of sensation like proprioception, stereognosis, or texture recognition which are usually limited to one or two parts of the body, sparing the trunk [6, 17]. In particular, a combination of impaired discriminating modalities with a preserved vibration sense can be considered as being characteristic for cortical strokes [6]. Since this pattern arises mainly due to lesions in the superior-posterior parietal cortex, it is referred to as the cortical sensory syndrome. However, a lesion in the inferior-anterior parietal cortex (parietal operculum, posterior insula) can mimic a thalamic sensory syndrome and is designated as the pseudothalamic syndrome [6]. A corticoparietal stroke resulting in a pseudothalamic syndrome cannot be differentiated from a thalamic stroke on the basis of sensory deficits alone. In cases of left hemispheric parietal strokes, neuropsychological dysfunctions usually involve language impairment, while right hemispheric lesions lead to visuoconstructive and visuospatial disturbances [6, 18–20]. In addition, somatosensory impairment due to cortical strokes is accompanied by some motor dysfunction in over 90% of cases [21].

Somatosensory impairment is more frequent in right hemispheric than in left hemispheric stroke [22]. Several studies report significant sensory impairment of the ipsilateral body side with an incidence of 17% following unilateral stroke [2, 3, 23]. The border zone of sensory symptoms on

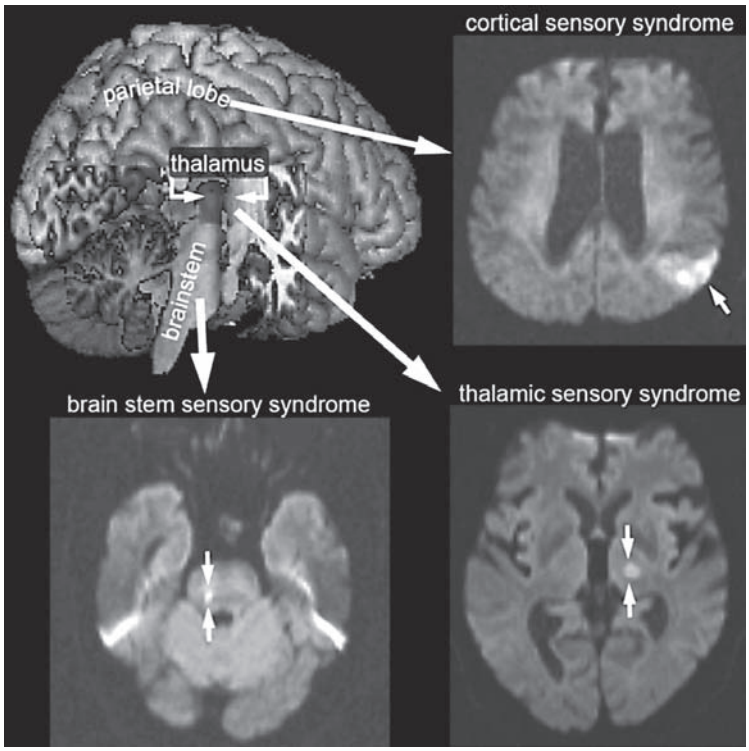

Fig. 1. MRI lesion examples (diffusion-weighted imaging) for different sensory syndromes.

the trunk and face are expected to be paramedian due to the 1–2 cm sensory function overlap of the intercostal nerves (fig. 1).

Diagnosis of Somatosensory Syndromes

Study results pertaining to the occurrence of somatosensory impairments after stroke vary widely [1, 2, 4, 24]. It is thought that the incidence is often underestimated since differential sensory modality assessments are limited in their scope. By assessing a single elementary somatosensory modality like touch, an impairment was found in only ~25–40% of stroke survivors, whilst a multimodal testing of elementary sensory modalities revealed dysfunction in ~60% of cases [2, 3]. However, good agreements between different body areas were found within each modality, indicating redundancy of testing between adjacent body regions [2]. The highest sensitivities for sensory impairments after stroke were found by testing discriminatory sensations, such as stereognosis, texture discrimination, position sense, and two-point discrimination. By such testing, impairments were found in 85–89% of stroke survivors [2, 3]. Many studies have described sensory functions using largely subjective scales such as 'absent' versus 'impaired' versus 'normal', thereby restricting interpretability and comparability between studies. New measures have been developed for improved standardized clinical somatosensory testing. The two most frequently used test batteries comprise the Nottingham Sensory Assessment (NSA) [25, 26] and the Rivermead Assessment of Somatosensory Performance (RASP) [26]. Both tests aim to identify sensory deficits after stroke and to monitor their recovery from stroke. The NSA employs eight quantifiable

subtests that can be used in a clinical setting without additional instruments. However, inter-rater reliability is relatively poor even in the revised NSA [25]. There are attempts to further modify the NSA for a better inter-rater reliability [27]. In contrast, the RASP has good inter-rater reliability, but requires additional tests that are not common in clinical practice [26].

Therapy and Prognosis

The main complaint of stroke survivors is loss of somatosensory function. Most patients undergo rehabilitation to regain and relearn lost skills. In the past, rehabilitation training has focused mainly on motor recovery, whereas somatosensory recovery has received less attention. This is probably due to the assumption that loss of sensation is less important for motor recovery. Recent studies, however, have shown that impaired sensory function is associated with the quality of upper limb movement, force control, manipulation of fine-graded objects, and sensory ataxia [4, 23]. The resulting sum of impairments predict poor functional outcome after stroke, including independence in activities of daily life, and even mortality [4, 5]. These findings have increased interest in underlying mechanisms of somatosensory recovery after stroke. It has long been recognized that sensory function following stroke improves during typical rehabilitation training without specific sensory training [2, 28]. Thus, multiple studies were performed to investigate the effectiveness of specialized sensory rehabilitation training [29–32]. In general, these interventions used sensory discrimination tasks [1, 30, 33] and sensory stimulation approaches involving tactile [29], electrical [32], thermal [31], and magnetic stimuli [34]. There is emerging evidence that specialized sensory interventions can result in increased recovery of somatosensory function as well as an improvement of other impairments such as motor function [for review, see 35].

Acknowledgement

We thank Nasim Kroegel for carefully reviewing the manuscript.

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Paciaroni M, Agnelli G, Caso V, Bogousslavsky J (eds): Manifestations of Stroke.
Front Neurol Neurosci. Basel, Karger, 2012, vol 30, pp 9–12

Headache

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Abstract

Headache can be a symptom of vast pathologies, and common secondary headache including head or neck trauma, cranial or cervical vascular disorder, non-vascular intracranial disorders headache related to a substance or its withdrawals, infection, disorders of homeostasis, disorders of cranium or facial mouth or cranial disorders, and headache attributable to psychiatric. Stroke-related headache has been reported between 7 and 65% and headache is also the most frequent symptom of cerebral venous thrombosis, which is present in nearly 90% of patients.

Headache can be a symptom of vast pathologies, and common secondary headache including head or neck trauma, cranial or cervical intravascular disorder, non-vascular intracranial disorders headache related to a substance or its withdrawals, infection, disorders of homeostasis, disorders of cranium or facial mouth or cranial disorders, and headache attributable to psychiatric [1]. The reported frequency of stroke-related headache varies from 7 and 65% and can occur in ischemic stroke, in transient ischemic attack, and in non-traumatic intracranial hemorrhage, including intracerebral and subarachnoid hemorrhage [2]. Headache is also the most frequent symptom of cerebral venous thrombosis, which is present in nearly 90% of the cases [3].

Acute Ischemic Cerebrovascular Disease

Transient Ischemic Attack (TIA)

The International Classification of Headache Disorders (ICHD) [1] requires the onset of a new type of headache which develops simultaneously with onset of focal deficit. Two other points of the criteria include focal neurological deficit of ischemic origin lasting <24 h and headache resolves within 24 h. However, it is likely that an update of the criteria will be introduced according to the new definition of TIA [4] in the third edition of the ICHD [5]. The headache is more common with basilar than carotid territory TIA, and it is very rarely a prominent symptom of TIA. The differential diagnosis between TIA with headache and an attack of migraine with aura may be particularly challenging [1]. Fisher reported that [6] 7 of 20 patients with vertebrobasilar ischemic attacks experienced headache, usually occipital or occipitalfrontal in distribution. The headache that accompanied TIA was described in 6 patients who subsequently developed a complete internal occlusion. The pain was mild, usually frontal, and sometimes radiating to the occiput and lasted only for the duration of the neurologic deficit. The author also questioned 58 patients with transient monocular blindness (amaurosis fugax) but none felt pain or discomfort with the attack. Grindal and Toole [6, 7] reviewed the case histories of 240 patients with TIA

and selected 58 who had a definite, recorded history of headache. The headache was a prominent symptom in 25% of patients and was the presenting complaint in nearly one third. Of 33 patients with carotid insufficiency, the headache was not localized or generalized in 13 patients and mainly frontal or retro-orbital in 19 patients. Headache in vertebrobasilar insufficiency was a constant feature in the majority of patients with recurrent vertebrobasilar insufficiency and affected the occipital or neck in 15 of 23 cases. Tentschert et al. [8] prospectively studied 2,196 patients with the diagnosis of acute ischemic stroke or TIA. Of the patients with acute ischemic stroke, 27% had headache at stroke onset, and that this was bilateral in the majority of the patients (61%). The study confirmed previous studies which found an association of headache at stroke onset with younger age patients and/or those with a history of migraine. The authors suggested a careful evaluation of young patients with a focal neurological deficit and a history of migraine to avoid the misclassification as 'complicated migraine'. In a recent study by Amort et al. [9], 303 patients were prospectively studied with suspicion of TIA. In summary, about 1 out of 5 patients had TIA mimics and epileptics (43.7%), and migraine attacks (23.6%) were the most common diagnoses among this group; paresis suggested TIA; memory loss, headache and blurred vision were significantly more frequent in patients with TIA mimics.

Acute Ischemic Stroke

The ICHD [1] requires the onset of a new type of headache that develops simultaneously or in very close temporal relation to signs or other evidence of ischemic stroke. Ischemic stroke can cause a migraine syndrome in patients who previously did not have a history of migraine, or can precipitate a migraine attack in patients who are prone to migraine. In addition, patients who have migraine after stroke might continue to experience recurrent migraine attacks. Headaches are more common after major strokes and significantly more frequent in patients with vertebrobasilar territory ischemia than those in the anterior circulation, probably because vessels in the posterior circulation are more densely innerved by nociceptive afferents than those in the anterior circulation. Most aspects of onset headache are debated and there is not a precise definition, though this type of headache might be an indication of the initial vascular occlusion and resultant ischemia [10].

Cervical Artery Dissection

Headache is reported by 60–95% of patients with carotid artery dissections an in about 70% of patients with vertebral artery dissections. Although headaches most commonly have a gradual onset, 20% of patients present with thunderclap onset [11]. According to the ICHD [1], headaches secondary to cervical artery dissection must be ipsilateral to the dissected artery. Headaches due to carotid artery dissection most commonly involve the jaw, face, ears, periorbital, and frontal or temporal region. Headaches due to vertebral artery dissection are commonly located in the occipital-nuchal region. Neck pain is present in 50% of patients with vertebra-artery dissections and 25% of patients with carotid-artery dissection. Associated neurological symptoms and signs include amaurosis fugax Horner's syndrome, pulsatile tinnitus, dysgeusia, diplopia, or other stroke manifestations [11]. In a recent case series of patients with spontaneous cervical artery dissection, pain was the only symptom in 20 out of 245 (8%), and in those particular cases, 12 had vertebral artery dissection, 3 had internal carotid dissection and 5 had multiple dissections. The headache was mainly throbbing in quality and the pain to the neck was mainly constrictive in quality [12].

Intracranial Hemorrhage

Subarachnoid Hemorrhage

The ICHD [1] requires severe headache of sudden onset that develops simultaneously with hemorrhage. The headache is the most common symptom of subarachnoid hemorrhage [11], and

typically the presentation is sudden and severe pain with nausea, vomiting, neck pain, photophobia, and loss of consciousness [2]. The sudden onset of the pain is the most characteristic feature, in fact in 3 out of 4 patients the onset is within a split second or a few seconds. The headache is the only symptom in about a third of the patients in general practice. Headache is generally diffuse and often described by patients as the most severe headache they have ever had. However, the crucial feature is the suddenness of onset and not the severity of the pain. The headache usually lasts 1–2 weeks, something longer. How short in duration a headache from subarachnoid headache can be is not known. No single or combined features of the headache exist that distinguish reliably, and at an early stage, between subarachnoid hemorrhage and non-hemorrhagic thunderclap headache [13].

Intracerebral Hemorrhage

The ICHD [1] requires the onset of a new acute type of headache which develops simultaneously with, or in very close temporal relation to intracerebral hemorrhage. The frequency of headache varies from 13% for putaminal hemorrhage to 50% in the case of cerebellum. 55% of patients with intraparenchymal hemorrhage experienced headache, usually unilateral and severe, at onset. In most patients with cerebral hemorrhage, the headache is overshadowed by the rapid onset of a devastating neurologic deficit, drowsiness, or vomiting [6]. One study prospectively studied 124 patients. 61% of the patients had ischemic stroke and 39% had hemorrhagic (not including subarachnoid hemorrhage type). Headache after acute stroke is frequent, starts usually on the first day of stroke, lasts about 3.8 days and is most frequently continuous, pressure-type in nature, more often located in the anterior cranial region and bilateral. Headache is more severe on the first day of stroke, and on the same day association with nausea and vomiting is frequent. Patients with hemorrhagic strokes had more severe headache and that they start more frequently before other neurological signs, whereas headache in ischemic stroke occurs more frequently in association with other focal signs and it is progressive in onset. A previous history of headaches was documented in 71 patients and the headache in acute stroke was a reactivation of previous headache in 38–53% of the patients, with the trend to complain of the same type of headache if the headache was frequent in comparison with those that had sporadic headache [14].

Cerebral Venous Thrombosis (CVT)

The ICHD [1] requires the onset of a new headache which develops in close temporal relation to CVT. Headache, generally indicative of an increase in intracranial pressure, is the most common symptom in patients with CVT and is present in nearly 90% of patients. The pain is typically diffuse and often progresses in severity over days to weeks. A minority of patients may present with thunderclap headache, suggestive of subarachnoid hemorrhage, and migraine-like pain has been described. Isolated headache without focal neurological findings or papilledema occurs in up to 25% of patients with CVT and presents a significant diagnostic challenge. CVT is an important diagnostic consideration in patients with headache and papilledema or diplopia (caused by sixth nerve palsy) even without other neurological focal signs suggestive of idiopathic intracranial hypertension. The superior sagittal sinus is most commonly involved, which may lead to headache, increased intracranial pressure, and papilledema. For lateral sinus thromboses, symptoms related to an underlying condition (middle ear infection) may be noted, including fever and ear discharge. Additionally, pain in the ear or mastoid region and headache are typical. Headache is also a common complaint during the follow-up sessions with CVT patients, occurring in about 50% of cases. In general, however, the headache is primary and not related to CVT [3].

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Paciaroni M, Agnelli G, Caso V, Bogousslavsky J (eds): Manifestations of Stroke.
Front Neurol Neurosci. Basel, Karger, 2012, vol 30, pp 13–16

Eye Movement Abnormalities

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Abstract

Generation and control of eye movements requires the participation of the cortex, basal ganglia, cerebellum and brainstem. The signals of this complex neural network finally converge on the ocular motoneurons of the brainstem. Infarct or hemorrhage at any level of the oculomotor system (though more frequent in the brainstem) may give rise to a broad spectrum of eye movement abnormalities (EMAs). Consequently, neurologists and particularly stroke neurologists are routinely confronted with EMAs, some of which may be overlooked in the acute stroke setting and others that, when recognized, may have a high localizing value. The most complex EMAs are due to midbrain stroke. Horizontal gaze disorders, some of them manifesting unusual patterns, may occur in pontine stroke. Distinct varieties of nystagmus occur in cerebellar and medullary stroke. This review summarizes the most representative EMAs from the supratentorial level to the brainstem.

Eye Movement Abnormalities in the Cerebral Hemispheres and the Basal Ganglia

Transient conjugate eye deviation (CED) toward the side of the lesion (Prévost's sign), whether accompanied by a concomitant ipsilateral head deviation or not, may occur in up to one-third of acute supratentorial infarcts. CED has more often been associated with right-side infarcts (probably because of lateralized representation of spatial attention in the right hemisphere), more severe neurological deficits and large cortical and subcortical infarcts (particularly on the left side) [1]. Ipsilesional CED may also occur, although less frequently, with small right frontal infarcts and right basal ganglia infarcts. Contralateral CED ('wrong-way eyes') may occur in striatocapsular hemorrhages.

Eye Movement Abnormalities in Thalamic Stroke

Eye movement disorders in thalamic infarcts are common. Combined up- and downgaze palsy and selective upgaze palsy can be due to paramedian thalamic-subthalamic infarcts (more often bilateral than unilateral) and to concomitant ischemia of the rostral midbrain. Complete ophthalmoplegia (loss of all extraocular movements with bilateral ptosis), which suggests neuromuscular junction disorders, may also result from bilateral paramedian and midbrain infarcts. Pure downgaze palsy is seen with bilateral paramedian thalamic infarcts.

Bilateral internuclear ophthalmoplegia (INO) with ptosis, pseudo-sixth nerve palsy (thalamic esotropia) and 'vertical one-and-a-half syndrome' (OAAH) may be seen, though rarely, in unilateral