5 Thalamic and Other Posterior Cerebral Artery Stroke Syndromes

Stefania Nannoni and Patrik Michel

Introduction

Approximately 20–40% of all ischaemic events in the brain affect posterior circulation [1, 2]. According to registry-based studies, isolated posterior cerebral artery (PCA) territory strokes occur in 5–10% of cerebral infarctions [3, 4], while up to 40% of patients have concomitant infarcts elsewhere in the posterior circulation or in the carotid territory [4, 5]. Among patients with pure PCA territory infarction, the confined involvement of deep structures (i.e. the thalamus and midbrain) varies from 34 to 64% across studies, with the ventrolateral thalamus emerging as the most frequently affected structure [4, 5]. The proportion of pure PCA cortical infarcts ranges from 14% to 51% of the total PCA infarctions, with the occipital lobe being most frequently involved [6, 7].

In the Acute Stroke Registry and Analysis of Lausanne (ASTRAL), from 5120 acute ischaemic stroke patients collected between 2003 and 2018, 184 (3.6%) patients presented with multilevel posterior stroke involving the PCA territory, whereas 336 (6.6%) patients exhibited pure PCA territory infarctions. Of these, 226 (67.3%) cases were authentic thalamic strokes (unpublished results from the ASTRAL registry) [2].

S. Nannoni · P. Michel (✉)
Stroke Centre, Neurology Service, Lausanne University Hospital, Lausanne, Switzerland
e-mail: Patrik.Michel@chuv.ch

The involvement of supratentorial posterior structures supplied by the posterior cerebral arteries (PCAs) causes typical stroke syndromes. Knowledge of the clinical features allows characterizing the diverse spectrum of symptoms associated with the vascular topography of lesions.

Clinical identification of supratentorial PCA syndromes has several clinical implications: Precise localization to the anterior vs. posterior circulation may allow (a) correlating findings on arterial and cardiac workup with the stroke location; (b) making inferences on stroke mechanism in recurrent events, especially if a parenchymal lesion is not evident on imaging (such as in transient ischaemic attacks, TIA); and (c) deciding on acute recanalization in cases of multiple occlusions in pre- and intracranial arteries.

However, posterior circulation ischaemia can be challenging to recognize, particularly in patients with a TIA, and discriminating between anterior and posterior vascular territory may be difficult on a purely clinical basis. Studies comparing anterior vs. posterior circulation stroke symptoms have shown that vestibulo-cerebellar signs (including nystagmus and oculomotor palsy), visual field abnormalities and crossed sensory-motor deficits are very specific to posterior strokes, while dysarthria, hemiparesis and cognitive symptoms are not [8, 9].

In some situations, clinical distinction between a stroke in the middle cerebral artery (MCA) and PCA is impossible [10–12]. This is typically the case with an acute proximal PCA occlusion,

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where contralateral hemiparesis results from ischaemia in the cerebral peduncle and hemispheric symptoms from the large-volume thalamic ischaemia. It has been shown, however, that clinical distinction between anterior and posterior circulation strokes are very reliable in patients with large (proximal) artery occlusions that are eligible for acute endovascular treatment [13].

This chapter will review the major clinical syndromes associated with posterior circulation ischaemia in the thalamus as well as other supratentorial structures. The clinical features of midbrain infarctions are described in Chap. 4.

Anatomy of the Posterior Cerebral Circulation

The posterior cerebral arteries (PCAs) are the terminal branches of the basilar artery (BA) and supply blood to the midbrain (rostral part), thalamus (medial and posterolateral regions), hippocampus, occipital lobes, temporal lobes (inferior and medial portions) and partially to the parietal lobes (posterior and inferior portions) [14].

Each PCA originates from the bifurcation of the BA at the pontomesencephalic junction and is traditionally divided into four segments: P1, from the termination of the BA up to the posterior communicating artery (PCom); P2, between the PCom and the posterior part of the midbrain; P3, from the pulvinar to the anterior limit of the calcarine fissure; and P4, the cortical segment within the calcarine fissure becoming the calcarine artery [15].

Classically, the PCAs have two main territories of vascular supply: a proximal or deep PCA territory, including the thalamus, and a distal or superficial PCA territory, including the hemispheric occipital and temporoparietal lobes [16, 17].

Blood Supply to the Thalamus

The thalamus receives most of its blood supply from four arterial pedicles that arise from the proximal portions of the PCAs and the P-com. Consequently, the vascular territories of the thalamus can be divided into four major regions: (a) the anterior region supplied by the polar or tuberothalamic artery; (b) the medial region supplied by the paramedian or thalamic–subthalamic arteries; (c) the inferolateral region supplied by the thalamogeniculate arteries; and (d) the posterior region supplied by the posterior choroidal arteries [15, 18]. Similar to the lenticulostriate arteries that irrigate the basal ganglia and the internal capsule, the thalamic arteries may show wide interindividual variation, regarding the origin, the number of arteries and the supplied nuclei [15, 18].

Cortical Branches of the PCA

As the PCAs reach the dorsal surface of the midbrain, they divide into four cortical branches: the anterior temporal, posterior temporal, parieto-occipital and calcarine arteries.The anterior temporal arteries arise first from the distal P3 segment; then, the posterior temporal arteries arise and course laterally, travelling along the hippocampal gyrus. The posterior temporal arteries course between the tentorium and the medial temporal lobe, including the fusiform gyrus. The parieto-occipital arteries usually originate from the P4 segment and supply the occipital and medial inferior parietal lobes, usually giving off the posterior pericallosal arteries, which circle the splenium of the corpus callosum. Usually, the calcarine arteries arise as single branches from the P4 segment, travelling at first lateral to the parieto-occipital arteries and then following a winding course, medially along the calcarine fissure [17, 19].

Important anatomical variants of the posterior circulation are frequent but commonly asymptomatic. Although typically discovered incidentally, their clinical significance is important so as not to mistake them for pathological findings and may be of relevance when determining stroke aetiology.

A carotid or foetal (fPCA) origin of the PCA refers to a PCA arising directly from the intracranial internal carotid artery (ICA) and occurs in 10–29% of the population [20, 21]. In this variant of the circle of Willis, the internal carotid artery contributes to the PCA via a patent

Pcom, while the connection of the PCA to the BA is hypoplastic or even aplastic. fPCA is mainly unilateral and is either partial or complete depending on whether or not a hypoplastic P1 segment is present. Bilateral fPCAs are associated with a small calibre BA, as the BA does not contribute to mesencephalic, temporal or occipital lobe flow.

Even though there is no established association between unilateral or bilateral fPCAs and stroke risk, such a foetal anastomosis may allow thromboemboli from the carotid artery to pass into the PCA [22]. Therefore, the etiologic evaluation of occipital stroke in patients with an ipsilateral fPCA should include an assessment of carotid artery disease. Moreover, in patients with haemodynamically significant carotid occlusive disease, the ipsilateral fPCAs and a non-functioning anterior communicating artery may be particularly vulnerable to ischaemia and infarction due to haemodynamic failure.

Another form of foetal anastomosis with a prevalence of 0.1–0.6% is the persistent trigeminal artery that connects the carotid to the basilar artery [23]. This artery originates from the internal carotid artery after its exit from the carotid canal and anastomoses with the mid-basilar artery. The part of the basilar artery caudal to the anastomosis is usually hypoplastic. In such patients, atherosclerotic carotid stenosis may lead to bilateral occipital infarction [24].

The artery of Percheron refers to an anatomical variant of thalamus supply, characterized by a single thalamic perforating artery arising from the proximal PCA (P1 segment) and suppling the rostral mesencephalon and both paramedian thalami [15]. Hypoplastic or absent P1 segments are more likely to be seen with this variant. The occlusion of this artery leads to bilateral infarction of the paramedian thalami, with or without rostral midbrain involvement [25].

Thalamic Stroke Syndromes

Ischaemic strokes involving the thalamus can give rise to a large variety of syndromes due to the complex anatomy and vascularization of this structure.

Isolated thalamic infarctions are traditionally classified into four territories (i.e. anterior, paramedian, inferolateral and posterior infarctions), which correspond, respectively, to the vascular territory of the polar, paramedian, thalamogeniculate and posterior choroidal arteries. This classification was initially based on neuroanatomical and neuropathological data [15] and later confirmed by imaging techniques (CT and MRI) [18, 26]. Some variant topographic patterns of thalamic infarction with distinct clinical manifestations, including the anteromedian, central and posterolateral infarct types, were also described [15, 27]. These result from variations in the thalamic arterial supply or reflect border-zone ischaemia.

The main types of thalamic infarctions, with their vascular supply and typical clinical presentation, are described below and summarized in Table 5.1. Examples of thalamic strokes are depicted in Fig. 5.1.

Table 5.1 Thalamic infarcts, vascular supply and corresponding clinical syndromes, including the four classical thalamic stroke syndromes (a-d), less frequent variants (e-g) and the syndrome from occlusion of the artery of Percheron (h)

Thalamic infarct type	Frequency	Main aetiology	Vascular supply	Clinical stroke syndrome
(a) Anterior	11–13% [26, 27]	SVD 60% [26]	Polar arteries	– Personality changes, apathy, aboulia – Executive failure, perseverations – Superimposition of temporally unrelated information (*palipsychism*) – Anterograde amnesia – Aphasia if left; hemispatial neglect if right-sided – Emotional facial paresis, acalculia, apraxia

(continued)

Table 5.1 (continued)

Thalamic infarct type	Frequency	Main aetiology	Vascular supply	Clinical stroke syndrome
(b) Paramedian	23–27% [26, 27]	CE 33%, SVD 33% [26]	Paramedian arteries	– Decreased or fluctuating arousal – Impaired learning and memory, confabulation, temporal disorientation – Altered social skills and personality, including apathy, aggression, agitation – Vertical gaze paresis – Aphasia if left-sided, spatial deficits if right-sided
(c) Inferolateral	27–45% [26, 27]	SVD 33%, LAA 33% [26]	Thalamogeniculate arteries	– Sensory loss (variable extent, all modalities) – Hemiataxia – Hemiparesis – Post-lesion painful syndrome (Dejerine–Roussy)
(d) Posterior	6% [26]	SVD 33% [26]	Posterior choroidal arteries	– Visual field loss (hemianopia, quadrantanopia, sectoranopia) – Variable sensory loss, weakness, aphasia, memory impairment, dystonia, hand tremor
(e) Anteromedian	13% [27]	CE 56% [27]	Variant anteriomedian arteries	– Cognitive and memory impairment – Decreased consciousness – Vertical eye paresis – Aphasia (left-sided lesion)
(f) Central	6% [27]	SVD 50% [27]	Border-zone vascular territory	– Hypoaesthesia – Memory impairment
(g) Posterolateral	11% [27]	SVD 38%, LAA 38% [27]	Variant posterolateral arteries	– Hypoaesthesia – Ataxia – Hemiparesis – Aphasia and executive dysfunction (left-sided)
(h) Bilateral paramedian	12% [26]	40% CE [26]	Artery of Percheron	– Disorders of vigilance – Anterograde and retrograde memory deficit – Behavioural changes (with a mixture or irritability and apathy) – Vertical gaze palsy

Legend: *SVD* small vessel disease, *CE* cardioembolic, *LAA* large artery disease

Anterior Thalamic Infarct

Anterior thalamic infarct is caused by the occlusion of the polar artery (also known as the tuberothalamic artery) [28]. This artery originates from the middle-third of the PCom artery but is absent in about one-third of cases (in these cases, the anterior territory is supplied by the paramedian arteries from the same side). It irrigates the reticular nucleus, ventral anterior nucleus, rostral part of the ventrolateral nucleus, ventral pole of the medial dorsal nucleus, mamillothalamic tract, ventral amygdalofugal pathway, ventral part of the internal medullary lamina and anterior thalamic nuclei. The anterior thalamic nuclei receive projections from the mamillothalamic tract and are connected to the anterior limbic system.

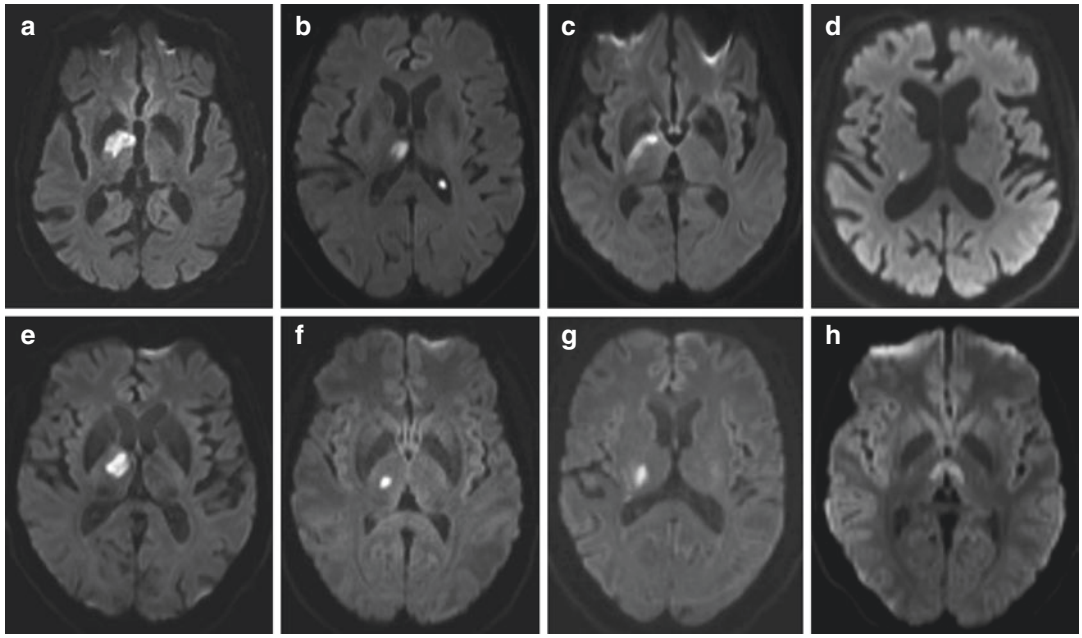

Fig. 5.1 Diffusion-weighted MRI showing a right thalamic infarction in anterior (**a**), paramedian (**b**), inferolateral (**c**) and posterior (**d**) infarctions. Localization in the anteriomedian (**e**), central (**f**), posterolateral (**g**) and bilateral paramedian (**h**) territories are also shown. (From Department of Radiology, Lausanne University Hospital)

Anterior thalamic infarcts account for about 13% of all thalamic infarcts but are rarely isolated, being most often part of anteromedian infarctions [26]. The most frequent aetiology is small vessel disease.

The clinical syndrome of anterior thalamic strokes is dominated by severe and various neuropsychological deficits [28, 29]. For this reason, such strokes are often underdiagnosed or missed initially. Patients can exhibit changing levels of consciousness in the early stages, while persistent personality changes are seen in the later course. They typically appear apathetic and aboulic, with a lack of spontaneity and emotional concern [28]. Disorientation in time and place, executive failure and perseverations can be present. Moreover, patients may show increased sensitivity to interference and have improper superimposition of temporally unrelated information (the latter called "palipsychism") [29, 30].

Another common finding in anterior thalamic infarct is the impairment of anterograde memory, likely from involvement of the mamillothalamic tract, with inability to make new memories. Left-sided lesions tend to affect more often the verbal and right-sided, the visual memory function [29, 31, 32].

Language disturbances, named transcortical motor aphasia, have been described in anterior thalamic lesions affecting the dominant hemisphere, i.e. typically left-sided lesions. They are characterized by impaired verbal fluency, anomia, semantic and phonemic paraphasia, with impaired comprehension but well-preserved repetition.

Right-sided lesions may be associated with hemispatial neglect.

"Emotional central facial paralysis," characterized by impaired activation of face muscles with emotion but normal voluntary activation, is also described [18].

Paramedian Thalamic Infarct

Paramedian thalamic infarct is due to the occlusion of the thalamic–subthalamic arteries (also called the paramedian or thalamoperforating arteries), which arise from the proximal P1 seg-

ment of the PCA, mainly as a pair from each P1. In about one-third of cases, these small arteries both originate from one side (asymmetric variant) or from a common pedicle of one P1 (artery of Percheron). They supply the paramedian parts of the upper midbrain and the posteromedial thalamus, including the posteroinferior portion of the dorsomedial nucleus and the intralaminar nuclei. When the polar artery is absent, the paramedian artery supplies this territory as well, and thus, infarction in this vascular territory is clinically severe.

Paramedian infarctions are the second most frequent after lateral infarcts and may occur unilaterally or bilaterally, accounting overall for about 35% of all thalamic infarcts [26]. The main aetiology is embolism.

Patients with unilateral infarcts of the paramedian arteries are described by a classic triad of symptoms: an acute decrease of consciousness, neuropsychological disturbances and abnormalities of vertical gaze [28, 33]. Impairment of arousal, with patients being lethargic, hypersomnolent or even comatose, is a dominant feature during the early stages. It is probably related to involvement of the intralaminar nuclei and the rostral portion of the midbrain reticular activating system [30]. As the impairment of consciousness diminishes and patients become more alert, memory and behavioural disturbances may be more evident. Amnesia is predominant, with patients being unable to learn and make new memories. Confusion, agitation, aggression and apathy are common personality disturbances and may persist in the long term [33, 34]. It has been suggested that memory loss and behavioural syndromes are related to the interruption of the mamilothalamic tract or ventral amygdalofugal pathway [31].

Vertical gaze palsy is characteristically present, with up-gaze palsy or combined up- and down-gaze palsy, depending on the volume of the lesion, its bilaterality and the degree of rostral midbrain involvement. Skew deviation is also common, with the eye elevated on the side of the lesion. Also, speech and language impairments are described in left-sided infarction. They are characterized by hypophonia and dysprosody, with frequent perseveration and markedly reduced verbal fluency, but normal repetition and preserved syntactic structure. This was named the adynamic aphasia of Guberman and Stuss [25]. Temporary and spatial neglect may be observed in patients with right-sided infarction.

An alternate syndrome characterized by central Horner syndrome (i.e. ptosis, myosis, pseudoenophthalmos and hypohidrosis of the ipsilateral hemibody) and contralateral mild ataxic hemiparesis has been described in ischaemic stroke patients due to paramedian (and anterior) thalamic lesions [35]. In these patients, infarction extended to the hypothalamic or rostral paramedian mesencephalic region, also irrigated by branches arising from the P1 segment. This syndrome likely results from impairment of the sympathoexcitatory and motor pathways in the thalamic–hypothalamic–rostral mesencephalic region [35, 36].

Inferolateral Thalamic Infarct

This type of thalamic stroke is caused by occlusion of the inferolateral arteries (also known as thalamogeniculate arteries), a group of 6–10 arteries that arise from the P2 segment of the PCA after the level of the PCom. They supply the ventrolateral thalamus, including the ventrolateral and ventroposterior nuclear groups, the lateral part of the centromedian nucleus and the rostrolateral portion of the pulvinar [15].

Infarcts in the inferolateral territory are the most common type of ischaemic stroke in the thalamus, accounting for about 45% of all thalamic infarcts [26]. Their major aetiology is small vessel disease.

The clinical features of inferolateral artery infarction were initially described as “thalamic syndrome” by French neurologists Dejerine and Roussy, with intense central post-stroke pain as the most characteristic symptom [37]. Patients may present a pure sensory stroke, a sensorimotor stroke or, in cases of extensive involvement of lateral thalamus, a sensorimotor stroke with abnormal movement patterns.

A pure sensory stroke is due to the selective involvement of the ventrolateral nucleus. It usu-

ally starts with paraesthesia or numbness on the contralateral side, followed by an isolated hemisensory deficit. Sensory loss may involve all modalities of sensation, though a dissociated loss, with sparing of pain and temperature, may be observed. A typical distribution in face-arm-leg is suggestive of a lateral thalamic infarction, even if a predominant acral distribution may occur [38, 39]. Some patients may develop an intense and delayed pain in the affected area, usually unrelieved by analgesics. This post-stroke painful syndrome has an apparent preference for right thalamic infarcts [40].

In sensorimotor stroke, the above-mentioned sensory disturbances are associated with hemiparesis on the same side, due to the extension of the infarcted area to the posterior limb of the internal capsule.

Abnormal movement patterns, such as ataxic hemiataxia or hemydystonia, result from the interruption of cerebellar or extrapyramidal tracts that synapse in the lateral thalamus. In some patients, an inability to stand and walk is predominant, and Masdeu and Gorelick called this "thalamic astasia" [41]. Another motor abnormality was described by Foix and Hillemand as flexed and pronated hand, with the thumb tucked under the other fingers, called the "thalamic hand" [42].

Cognitive and behavioural performances are usually preserved in inferolateral thalamic infarcts, although mild transcortical motor aphasia with reduced fluency is occasionally reported in dominant hemisphere lesions [30].

Posterior Thalamic Infarct

Posterior thalamic infarct is caused by the occlusion of the posterior choroidal arteries, also arising from the P2 segment of the PCA, just after the inferolateral arteries. They consist of a group of small vessels, with 1–2 branches (medial) arising adjacent to the origin of the Pcom artery and 1–6 branches (lateral) originating from the distal P2 segment of the PCA. They supply the pulvinar and lateral dorsal and posterior nucleus, the geniculate bodies and partially the anterior nucleus. Infarction limited to the dorsal part of the thalamus is rare [26], and the most characteristic clinical findings are visual field defects (due to involvement of the lateral geniculate body) [43].

Medial posterior choroidal artery infarction causes visual field cuts including upper or lower quadrantanopia, whereas involvement of the lateral posterior choroidal artery causes horizontal wedge-shaped or tubular sectoranopias [44].

Involvement of the pulvinar and posterior nuclei can produce numerous less-specific symptoms, including impairment of ipsilateral pursuit, contralateral saccades, mild hemiparesis or hemisensory abnormalities, abnormal dystonic movement and neuropsychological disturbances (such as aphasia, amnesia, aboulia and visual hallucinosis) [30].

A delayed complex hyperkinetic motor syndrome that includes myoclonus, ataxia, chorea, pseudorubral tremor, dystonic posture of the fingers and worsened by voluntary activities, termed the "jerky dystonic unsteady hand," was also observed in a small subset of patients with infarcts restricted to the pulvinar according to CT or MRI assessment, raising the question of additional nuclei involved [18, 45].

Variant: Anteromedian Thalamic Infarct

This stroke involves the posterior part of the anterior territory and the anterior part of the paramedian territory [27]. It is likely related to the occlusion of variant anteromedian arteries, originating from the proximal segment of the PCA [15]. Similar to paramedian thalamic strokes, cardioembolism is the most frequent aetiology [27].

The dominant feature is a wide range and severe neuropsychological disturbance [27]. Severe anterograde amnesia is a common feature, particularly prominent when involving the anterior part of the dorsomedian nucleus and intralaminar nuclei. Loss of initiative and executive dysfunction is frequently found. Contrary to infarcts restricted to the anterior territory, patients with anteromedian territory infarcts do not exhibit issues with perseverance. Instead, the

main behavioural change in anteromedian infarct is a severe loss of self-activation, requiring constant external stimulation. Aphasic troubles with word-finding difficulties, reduced fluency and denomination are also described. Decreased consciousness is uncommon in unilateral anteromedian territory infarcts, but a frequent finding in bilateral lesions. Vertical gaze palsy has been reported and postulated to be due to involvement of fronto-cortical fibres that may be decussating in the medial thalamus [46].

Variant: Central Thalamic Infarct

Infarct of the central territory is characterized by the involvement of parts of all four adjacent classic territories. It may be expression of a border-zone infarction between adjacent territories [27].

In the four patients observed by Carrera et al., hypoesthesia was a common feature, likely due to involvement of the medial portion of the ventroposterolateral nucleus. Anterograde amnesia and short-term memory impairment are also dominant and more severe than in anteromedian territory infarcts [27]. Ataxia, vertical gaze paresis and neuropsychological signs are also described in patients with bilateral lesions.

Variant: Posterolateral Thalamic Infarct

The posterolateral territory is formed by combining the posterior portion of the inferolateral territory and the anterior portion of the posterior territory. This is likely supplied by variant posterolateral arteries. Microangiopathy is the predominant stroke aetiology, as well as for inferolateral infarcts [27].

The clinical picture is characterized by contralateral hypaesthesia and ataxia, with transient hemiparesis. Compared to patients with inferolateral infarcts, an unusual finding in posterolateral territory infarct is the impaired cognition from a left-sided lesion. Aphasia with impaired repetition that resembles cortical motor aphasia is described; this differs from transcortical aphasia due to anteromedian infarcts. Executive dysfunction may also be seen because of the disruption of thalamocortical fibres arising from the posterolateral nuclei of the thalamus [27, 47].

Variant: Bilateral Paramedian Thalamic Infarct

Occlusion of the artery of Percheron leads to bilateral infarction of the paramedian thalami, with or without rostral midbrain involvement, and causes severe stroke [15, 33]. Asymmetric thalamic involvement is seen in two-thirds of cases, and midbrain infarction is present in over half [48]. Artery-to-artery embolism or cardioembolism are thought to be the most common aetiology of stroke in patients carrying this anatomic variant [49].

The most typical clinical features of bilateral paramedian thalamic infarction are altered sensorium such as stupor or coma, prominent memory impairment, behavioural changes and vertical gaze palsy. Overall, the neuropsychological disturbances are more severe than in those with unilateral infarcts and can be persistent [25, 34, 50].

Patients are usually apathetic and aboulic, with reduced spontaneity and increased inertia. Disorientation, confusion and akinetic mutism (i.e. awake unresponsiveness) can be observed. Patients may show perseveration and a marked tendency to confabulate. A compulsive use of objects out of a behavioural context, as observed in patients with frontal-lobe lesions, is also described [48, 51].

The amnestic syndrome resulting from paramedian territory infarction is similar to thiamine-deficient Korsakoff's syndrome, destroying the medial dorsal thalamic nuclei and the mammillary bodies. The addition of the other behavioural features produces a constellation of symptoms that led to the term "thalamic dementia" [18, 52].

Non-thalamic PCA Stroke Syndromes

The syndromes of infarction in the PCA territory are conventionally associated with homonymous visual field defects. However, patients with PCA

territory infarcts often present clinically with multiple symptoms and signs, including sensory and motor abnormalities and cognitive and neuropsychological deficits [5, 7, 16, 19]. These strokes can simulate strokes in the middle cerebral artery (MCA) territory, especially in the presence of significant motor deficits [10–12].

In this section, we first describe typical signs and symptoms found in infarcts affecting the PCA territory and not related to thalamic involvement, followed by a description of the main clinical syndromes associated with proximal and distal PCA occlusion.

Clinical Features in PCA Strokes without Thalamic Involvement

Hemispheric infarctions in the PCA territory potentially involve the occipital, posterior temporal and parietal lobes, with variable clinical manifestations. The most frequent symptom is visual field abnormality, which is reported in more than 90% of patients with cortical PCA infarctions [4, 6, 7]. Among cognitive deficits, memory impairment and aphasia are reported in 18 and 15% of patients, respectively [6]. Cognitive deficits associated with visual function, such as visual neglect or visual agnosia, seem less common in clinical practice, being reported in less than 10% of patients with cortical PCA infarctions [6].

Visual Field Defects

Homonymous hemianopia is the most frequent visual field defect after unilateral PCA infarctions, involving either the two right or two left-halves of the visual fields of each eye [16, 53]. It is caused by contralateral lesions of the optic radiations (also called geniculocalcarine tracts) in the occipital lobe and/or by contralateral lesions in the cerebral visual (occipital) cortex (Brodmann area 17). Hemianopia from PCA infarctions is traditionally described as sparing the macula, i.e. the central or medial part of the visual field is preserved [4, 6, 7].

Homonymous hemianopia is often disabling, causing difficulties with reading and visual scanning. Patients usually fail to notice relevant objects or avoid obstacles on the affected side, causing collisions with approaching people or cars. The visual defect is often described as a void, blackness, or a limitation of vision to one side, and patients recognize after some training that they must focus extra attention on the hemianopic field.

Hemianopia is in most cases complete, but upper or lower quadrantanopsia can also be found. A *superior quadrant field defect* (*"pie in the sky"*) is seen if the infarct is limited to the lower-bank of the calcarine fissure (the lingual gyrus), or if it affects the inferior (temporal) radiations of the optic tract. An *inferior quadrantanopia* results if the lesion affects the cuneus on the upper-bank of the calcarine fissure, or the upper (parietal) optic radiations [6].

Cortical bilateral visual field defects including complete "cortical" blindness are found as a result of bilateral PCA territory strokes, usually after "top of the basilar" embolism [54, 55]. Interestingly, such patients may exhibit *visual anosognosia* for their blindness, despite the sparing of parietal and thalamic structures. This so-called "Anton's syndrome" is characterized by the patients' affirmation of seeing normally despite objective evidence of blindness [56]. If patients do not admit that they cannot see, they may use confabulation or increased verbosity to try to compensate for the lack of visual input.

Neuropsychological Features in Dominant PCA Strokes

Language-related disorders such as dysphasia, dyslexia (without dysgraphia), dyscalculia and colour anomia may occur when the dominant PCA territory is infarcted (usually the left side) [16, 57].

Aphasia can be due to an infarction large enough to cover the left parietal lobe or temporal lobe [58]. "Transcortical sensory aphasia," similar to Wernicke's sensory aphasia but with preserved repetition, is caused by infarctions into the parietal–occipital region on the left side. The patient may alternatively show "amnestic (or anomic) aphasia" (inability to name but repetition and comprehension intact) due to infarction to the left temporal lobe of the PCA territory.

Alexia refers to difficulty in reading, with patients being unable to read single letters or

numbers, while writing, speaking and other language functions are preserved. In less-severe deficit, patients may need more time to read, depending on sequential identification of letters. Alexia without agraphia (pure alexia) is caused by a lesion of the dominant occipital lobe and splenium of the corpus callosum and is often accompanied by right homonymous hemianopia. The pathophysiological basis is a disconnection between the visual information and the language-processing area [59]. In patients with extensive infarction that damages the left angular gyrus, *alexia with agraphia* will develop, but oral-language functions are still preserved [60].

Elements of "Gerstmann's syndrome" (i.e. dyscalculia, dysgraphia, finger agnosia and right–left disorientation) may be found in patients with inferior parietal lobe lesions (especially involving the angular gyrus and adjacent structures) [61].

Bilateral or unilateral dominant PCA infarction may produce significant *memory impairment* by damaging the hippocampus, parahippocampus and connecting fibres [19, 57, 62]. Patients demonstrate the impaired acquisition of new memories (anterograde amnesia), while the retrieval of memories encoded prior to the onset of the infarction (retrograde amnesia) is usually less affected.

Amnesia in patients with unilateral lesions is generally transient on bedside examination, lasting a few days, but may not be detected at all. Sometimes, patients appear frankly confused. They cannot recall what has happened recently, and when given new information, do not recall it moments later. They often repeat statements and questions spoken only minutes before.

Clinically isolated amnesia from stroke may be difficult to distinguish from transient global amnesia (TGA). The latter often shows a small punctate DWI lesion ("pixel") in the hippocampus at 12–48 h after symptom onset. Still, TGA is not considered an ischaemic stroke and its pathophysiology remains uncertain [63]. As described by our group, a typical TGA presentation is very rare due to ischaemic stroke [64]. Red flags that may indicate stroke include associated focal neurological symptoms and signs, such as visual field deficits or transient hemisyndromes. Also, a very long or very short duration of amnesia and the presence of major stroke sources increase the likelihood of an ischaemic origin of amnesia, which are located in or close to the Papez circuit [64].

Radiologically, DWI lesions from TGA are located in the CA1 region of the hippocampus and are usually unique. In 10–15%, a second similar lesion in the same or contralateral hippocampus may occur [65]. On the contrary, patients with ischaemic hippocampal lesions often have other acute lesions in the same or other territories, with lesions of a larger size that are visible more quickly (i.e. within 12 h) and that tend to enhance with gadolinium if repeat MRI is performed beyond 5–7 days [66].

After the acute phase, amnestic signs in PCA territory strokes may persist up to 6 months on a detailed neuropsychological exam. However, in bilateral medial temporal lobe lesions, amnesia may be permanent and severe.

Neuropsychological Features in Non-dominant PCA Strokes

Disorders of visual cognitive functions with visual agnosia including prosopagnosia, spatial disorientation, dyschromatopsia and palinopsia may be found in patients with non-dominant hemisphere (usually right-sided) infarcts [16, 19].

Visual agnosia is the inability to recognize visually presented objects despite the preservation of elementary visual function. This is usually found in patients where the PCA supplies adjacent parietal structures and PCA branch occlusions cause a disconnection between language and visual systems [67, 68]. Patients have difficulty in understanding the nature and use of objects presented visually, but they can name objects when they touch them or when the objects are described to them. Two forms of visual agnosia are described: "apperceptive" agnosia involves poor perception and ability to understand, while "associative" agnosia involves poor ability to match and use. Close to associative agnosia, patients may present *optic aphasia,* i.e. a naming deficit confined to the visual modality [69]. Patients typically present with extensive left

PCA territory infarction with right homonymous hemianopia.

Prosopagnosia is a form of visual agnosia characterized by difficulty in recognizing previously familiar faces. It is due to lesions in the inferior occipital areas, the lingual and fusiform gyri and the anterior temporal cortex. In the literature, this deficit is described as associated with the right PCA territory [70].

Achromatopsia refers to difficulty perceiving colours [71]. It is due to infarctions in the ventral occipital cortex and/or infracalcarine. The patient may present with hemiachromatopsia if the infarction is unilateral.

Visual hallucinations are uncommon but may develop from PCA strokes on any side of the brain, often during the recovery phase [6]. They can be either simple or complex and usually criticized by the patient.

Palinopsia refers to images persisting even after the image has been removed from the visual fields. Infarctions can be in the lingual and fusiform gyri [72].

Spatial and geographic disorientation and an inability to recall routes or to read or visualize the location of places on maps are also common. This is named *topographagnosia* and may comprise heterogeneous manifestations, including difficulty identifying familiar environmental landmarks such as buildings and street corners. This deficit is associated with the right posterior parahippocampal gyrus and the anterior part of the lingual and fusiform gyri [73].

Moreover, unusual aggressive behaviour can be caused by PCA strokes as well, especially with the involvement of the right occipital lobe. These patients may become anxious, aggressive and frustrated when they are stimulated by the environment [74].

PCA Stroke Syndromes According to Occlusion Site

Patients with PCA occlusion present with clinical stroke syndromes that vary according to the site of occlusion and to the corresponding location and extent of infarction. As described above, cognitive symptoms are often side-related [7, 16, 75, 76].

Depending on the location of the vascular occlusion, we identify three groups of unilateral PCA infarctions and a heterogeneous syndrome associated with bilateral infarction, which are listed below and summarized in Table 5.2.

Table 5.2 Infarct topography and clinical findings of the main PCA stroke syndromes

PCA infarction	Vascular site of occlusion	Infarct location	Clinical stroke syndrome
Unilateral			
Proximal	(a) Proximal P1 segment (near its origin from the basilar artery)	*Deep and superficial infarct*, involving: medial midbrain, posterolateral thalamus, and hemispheric occipito-parieto-temporal PCA territory	– Decrease in consciousness – Lethargy, aboulia – Oculomotor abnormalities (partial or complete ipsilateral third nerve palsy, bilateral ptosis and vertical gaze palsy) – Contralateral hypoaesthesia and hemiplegia – Visual agnosia, colour anomia, visual hallucinations
	(b) P2 segment (before the branching of the thalamogeniculate arteries)	*Deep and superficial infarct*, involving: lateral thalamus and hemispheric PCA territory	– Severe contralateral sensory loss – Hypotonia, clumsiness and abnormal movements (but not hemiplegia) – Short-term memory impairment – Variable language and visual cognitive dysfunctions

(continued)

Table 5.2 (continued)

PCA infarction	Vascular site of occlusion	Infarct location	Clinical stroke syndrome
Distal	(c) Single or multiple PCA branch(es)	*Superficial infarct*, involving the calcarine, parieto-occipital and posterior temporal artery territories	– Homonymous hemianopia – Language disorders including dysphasia, dyslexia, dyscalculia, colour dysnomia (dominant hemisphere) – Visual cognitive dysfunctions including visual agnosia, dyschromatopsia and spatial disorientation (non-dominant hemisphere)
Bilateral	(d) Bilateral PCA occlusion from embolus or fragmentation of a thrombus in the basilar artery	*Deep and superficial infarct*: variable extended bilateral hemispheric infarction with thalamic involvement	– Cortical blindness – Possible visual anosognosia and confabulation (Anton's syndrome) – Amnesia and cognitive dysfunctions – Emotional and behavioural disturbances (agitated delirium) – Visual field defects with visual cognitive abnormalities (for bilateral inferior-bank infarct) – Optic ataxia, oculomotor apraxia, simultagnosia (Balint's syndrome) (for bilateral superior-bank infarct)

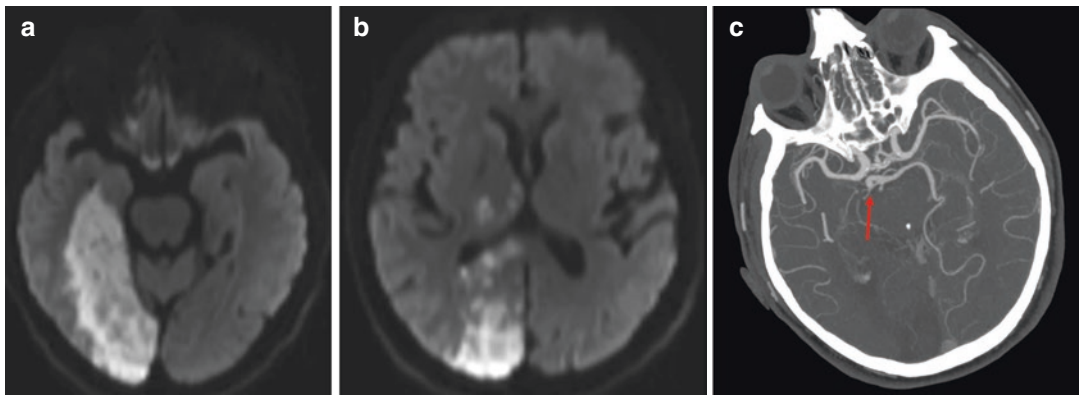

Fig. 5.2 Diffusion-weighted MRI showing a large right PCA infarction involving the temporal lobe (**a**), the occipital lobe and the median and lateral thalamus (**b**). CT-angiography shows a right-side P1 occlusion (**c**, red arrow). (From Department of Radiology, Lausanne University Hospital. Right P1 occlusion on CTA)

(a) *Occlusion of the very proximal PCA (P1-segment occlusion)*: Occlusion near its origin from the BA causes a total PCA territory infarction, which includes the cerebral peduncle of the midbrain, lateral thalamus and the hemispheric territory (Fig. 5.2).

The midbrain infarction can also be bilateral, even when only one PCA is occluded, if penetrating arteries to the bilateral paramedian rostral brainstem structures arise from one PCA. In this case, patients often exhibit prolonged stupor or coma, or, later, hypersomnolence and vertical gaze palsies [17].

Occlusions of the proximal PCA origins are usually embolic (from the heart, aorta or proximal vertebrobasilar arteries) [4].

Sensory and motor abnormalities are described in approximately 70% of patients, with sensory deficits being more common. In

patients with PCA territory ischaemia, lateral thalamic infarction is likely the major reason for sensory symptoms and signs. A severe hemiparesis or hemiplegia is mainly due to infarction in the lateral midbrain as a result of involvement of the corticospinal and/or corticobulbar tracts in the cerebral peduncles.

Partial or complete ipsilateral third-nerve palsy, bilateral ptosis, loss of vertical gaze, lethargy and aboulia are also variable features associated with proximal PCA occlusion. Other signs of posterior hemispheric cortical involvement, such as visual agnosia, colour anomia, visual hallucinations or illusions can also be present.

Such proximal P1 occlusion can also produce the historical syndromes of Weber [77] or Parinoud [78].

(b) *Occlusion of the P2 segment of the proximal PCA*, beyond the origin of the posterior communicating artery but before the branching of the thalamogeniculate arteries, will lead to a combined deep and superficial infarction mainly involving the inferolateral thalamus and the hemispheric PCA territory [16, 17]. The pulvinar (posterior choroidal artery) may also be affected.

The combination of infarctions of the lateral thalamus and cortical PCA branches (with or without involvement of the pulvinar) will lead to a variable combination of contralateral sensory hemisyndromes (sometimes with ataxia and minor corticospinal signs). Abnormal spontaneous contralateral limb movements may occur, and pain may develop weeks or months after the stroke, as described above. Dejerine–Roussy syndrome may occur after the acute phase. Consciousness disturbances are more frequent than in patients with pure cortical PCA infarctions [7]. Ischaemia in superficial PCA branches will lead to variable combinations of homonymous visual field defects and cognitive signs, as described above. Visual inattention and prosopagnosia are seen in patients with right temporal and parieto-occipital branch involvement, while patients with left-side lesions show mostly transcortical sensorial aphasia [7].

Clinically, it is sometimes impossible to distinguish whether signs stem from the thalamic or superficial lesions; similarly, strokes from P2 occlusion may imitate occlusions of parietal and temporal (posterior) branches of the MCA.

(c) *Occlusions of cortical PCA branches* (from the P3 segment of the PCA) mainly affect the calcarine arteries, leading to homonymous visual field defects [5, 17]. Parieto-occipital branch occlusions will lead to visual associative agnosia and visual neglect with right-sided lesions. Anterior temporal branch occlusions will affect mainly memory function specific to the side of the lesion, with language disturbance if localized in the dominant hemisphere, as described above. Occlusions of the posterior temporal branch lead to spatial/geographic disorientation and prosopagnosia, mainly if located on the right. Simple and complex visual hallucinations may occur with an occlusion of any of these branches, typically beginning hours to days after the stroke, and usually disappear spontaneously [4, 7].

Occlusion of multiple cortical PCA branches leads to variable combinations of signs described in this chapter.

(d) *Bilateral PCA territory infarction* occurs in about 6–13% of all PCA strokes [6, 7, 76]. We already described bilateral thalamic stroke from a single occlusion of the paramedian thalamic artery (artery of Percheron) [15]. Moreover, in "top of the basilar" syndrome, thalamic and/or superficial PCA territory lesions are frequently bilateral and occur simultaneously [54] (Fig. 5.3).

In such extensive lesions, patients usually have decreased levels of consciousness. In cases of bilateral occipital involvement, they present "cortical" blindness, with or without Anton's syndrome, i.e. anosognosia of blindness. Confabulations are commonly used to compensate blindness. Less-severe visual field defects, like bilateral hemianopia or bilateral scotoma, may be observed in cases of compensatory blood supply, including from the middle cerebral artery.

Cognitive dysfunction from bilateral thalamic involvement may dominate the clinical

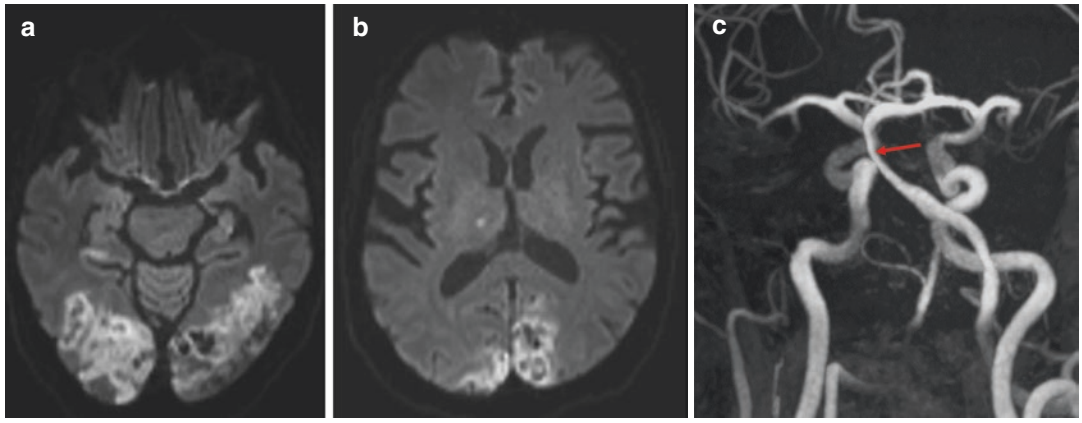

Fig. 5.3 Diffusion-weighted MRI showing a bilateral PCA infarction involving the occipital lobe (**a**) and the central thalamus (**b**). MR-angiography shows a focal stenosis in the mid-basilar artery (**c**, red arrow). (From Department of Radiology, Lausanne University Hospital)

picture in the long term ("thalamic dementia") [52]. Amnesia may also be a permanent sequela of bilateral infarction of the medial temporal lobes.

The most frequent structures involved in bilateral PCA strokes are those located below the calcarine fissure, including both occipital and temporal lobes [17]. These produce upper quadrantopsia, achromatopsia, apperceptive visual agnosia and prosopagnosia. Patients may present hyperactive and restless behaviour, with motor agitation and aggressive reactions, especially when stimulated. This agitated delirium seems to be attributed to the limbic system involvement in patients with bilateral inferior temporal lobe infarction [19, 74].

The infarction of the bilateral occipitoparietal border may result in features of Balint's syndrome. This presents with optic ataxia (inability to reach targets under visual guidance), oculomotor apraxia (inability to intentionally move eyes towards an object) and simultagnosia (inability to synthesize objects within a visual field) [79].

Moreover, patients with bilateral upper-bank lesions may have difficulties in recognizing where objects, people or places are topographically [80].

Prognosis of PCA Stroke

After thalamic infarction, prognosis is generally regarded as more favourable compared with lesions of the cerebral cortex or other subcortical structures [26, 81]. This generally reflects the low incidence of mortality and the good recovery from motor deficit. However, patients with tuberothalamic or paramedian artery stroke could be affected by the persistence of cognitive and psychiatric manifestations, even if systematic longitudinal analyses have not been performed [18].

Similarly, the functional outcome of patients with superficial PCA territory infarction is usually good [16]. Compared with MCA infarctions, they showed higher frequency of symptom-free at discharge and lower in-hospital mortality rate [3, 4]. Early mortality in PCA infarction is low, ranging from 0% to 7% in different series [1, 3, 4, 6, 75]. Recurrent vertebrobasilar ischaemia, myocardial infarction, sudden unexplained death and pneumonia are the main reported causes of death [3, 7].

Unlike MCA territory infarction, malignant infarctions of the PCA with cerebral edema, mass effect and transtentorial herniation are rarely reported, given the more limited vascular territory that is involved. Gogela et al. described three cases of unilateral occipital infarction, which resulted in massive edema and herniation [82],

while Pfefferkorn et al. reported one case of extensive bilateral PCA infarction, which produced fatal herniation [83].

Long-term prognosis after PCA strokes seemed heavily associated with the localization of the infarct: patients with extensive involvement of deep structures, especially the midbrain, showed worse prognosis than those with infarction limited to the superficial territory [84]. Similarly, PCA-plus patients (i.e. patients with coincident infarct outside the PCA territory) had increased disability at 6 months and long-term mortality compared to pure PCA strokes [85]. In a retrospective cohort study of PCA strokes from our institution, we observed a trend for a lower cognitive, visual and functional disability at 3 months in patients treated with intravenous thrombolysis and/or mechanical thrombectomy compared to conservative treatment [86].

Stroke recurrence is an important cause of morbidity and mortality after posterior ischaemic stroke. Patients with PCA infarcts of atherothrombotic aetiology showed a higher risk of recurrence compared to other etiologies [16]. Moreover, patients with PCA stroke and proximal large artery disease (i.e. BA and intracranial vertebral artery disease) demonstrated a higher risk of a second ischaemic event than patients with intrinsic PCA atherosclerosis [7].

Similarly to anterior circulation stroke, functional outcome after PCA stroke mainly depends on recovery from motor dysfunction (due to the involvement of the midbrain or internal capsule). Also, the size of infarction of the dominant hemisphere is crucial for the persistence and severity of neuropsychological deficits [16]. Moreover, patients with PCA stroke might be specifically affected by long-term sequelae concerning visual field defects, sensory deficits and involuntary movements.

Visual field defects after PCA stroke can result in significant disability and reduction in quality of life [87]. The impact of visual impairment on daily activities can be wide ranging, including a general reduction in mobility, reduced ability to judge distance, higher risk of falls, reading impairment and inability to drive [88]. Spontaneous visual field improvement can occur post-stroke in varying degrees, mostly depending on the severity of the initial severity of symptoms and lesion extension. This has been reported in up to 50% of patients, usually within the first 3–6 months, mostly due to the resolution of cerebral edema and the recovery of neurotransmission [89]. After this period, spontaneous recovery is possible, but usually at a much slower rate and likely related to improvement in the patient's functional ability despite persistent defects [89].

Sequelae related to sensory dysfunction are relatively common after thalamic infarction. Central post-stroke pain syndrome (CPSP) is a debilitating sequelae that can follow laterothalamic sensory stroke. It has been originally described as part of the "thalamic syndrome of Dejerine and Roussy" [37], even if it is now recognized that strokes occurring anywhere along the spinothalamic or trigemino-thalamic pathways (including lateral medullary stroke and parietal cortical stroke) can produce similar symptoms [90]. Frequency of CPSP appears to depend upon lesion location: in inferior lateral thalamic infarctions, 17% to 18% of cases were described to develop CPSP, while this percentage was higher in the lateral medullary infarction, and much lower in parietal cortex infarction [90]. At the nuclear level, CPSP following thalamic stroke seemed to be critically related to the damage of the ventral posterolateral nucleus [91], although the exact pathogenesis of this delayed sensory syndrome remains unclear. Symptoms usually develop weeks or months after the onset of stroke and affect the areas where the sensory deficits were the most severe in the acute phase. Patients may describe sharp, stabbing or burning pain and experience hyperpathia and especially allodynia [92, 93]. Pharmacological therapy, magnetic stimulation and invasive electrical stimulation are therapeutical options [94].

Patients with thalamic stroke may also develop post-stroke involuntary movements, such as asterixis, dystonia, chorea/athetosis, tremors, and myoclonus [95]. These represent a rare complication of posterior or inferolateral thalamic strokes, usually appearing several months after the event and following full recovery of initially severe motor deficits. Affected patients often show a

complex combination of hyperkinetic movements, also described as "jerky dystonic unsteady hand" syndrome [45]. Dystonia and choreoathetosic patterns have been associated with severe positional sensory deficits, whereas tremor/myoclonus patterns were related to severe cerebellar ataxia. Therefore, it has been proposed that these involuntary movements result from failure of the proprioceptive sensory and cerebellar inputs in addition to successful, but unbalanced, recovery of the motor dysfunction [96].

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