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*Stroke*. 2003;34:2264-2278; originally published online August 21, 2003;

doi: 10.1161/01.STR.0000087786.38997.9E

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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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## Vascular Syndromes of the Thalamus

Jeremy D. Schmahmann, MD

**Background**—This article reviews the anatomy, connections, and functions of the thalamic nuclei, their vascular supply, and the clinical syndromes that result from thalamic infarction.

**Summary of Review**—Thalamic nuclei are composed of 5 major functional classes: reticular and intralaminar nuclei that subservise arousal and nociception; sensory nuclei in all major domains; effector nuclei concerned with motor function and aspects of language; associative nuclei that participate in high-level cognitive functions; and limbic nuclei concerned with mood and motivation. Vascular lesions destroy these nuclei in different combinations and produce sensorimotor and behavioral syndromes depending on which nuclei are involved. Tuberothalamic territory strokes produce impairments of arousal and orientation, learning and memory, personality, and executive function; superimposition of temporally unrelated information; and emotional facial paresis. Paramedian infarcts cause decreased arousal, particularly if the lesion is bilateral, and impaired learning and memory. Autobiographical memory impairment and executive failure result from lesions in either of these vascular territories. Language deficits result from left paramedian lesions and from left tuberothalamic lesions that include the ventrolateral nucleus. Right thalamic lesions in both these vascular territories produce visual-spatial deficits, including hemispatial neglect. Inferolateral territory strokes produce contralateral hemisensory loss, hemiparesis and hemiataxia, and pain syndromes that are more common after right thalamic lesions. Posterior choroidal lesions result in visual field deficits, variable sensory loss, weakness, dystonia, tremors, and occasionally amnesia and language impairment.

**Conclusions**—These vascular syndromes reflect the reciprocal cerebral cortical–thalamic connections that have been interrupted and provide insights into the functional properties of the thalamus. (*Stroke*. 2003;34:2264-2278.)

**Key Words:** cerebral cortex ■ cognition ■ diaschisis ■ language ■ memory ■ thalamus

In the first detailed account of the behavioral consequences of thalamic hemorrhage, Fisher<sup>1</sup> described 13 patients of a total of 102 cases of intracerebral hemorrhage pathologically studied. Sensory deficits were associated with neglect (“modified anosognosia and hemiasomatognosia”), “dysphasia” was global and moderate in severity, and confusion, delirium, visual hallucinations, and peduncular hallucinosis taking the form of colorful objects, animals, and flowers were described. Later reports of thalamic hemorrhage confirmed and extended the relationship between thalamic lesions and cognitive deficits.<sup>2–5</sup> Hemorrhage is seldom confined to the thalamus itself, however, and remote effects include pressure of the hemorrhagic mass, effect of surrounding edema on adjacent structures, and toxic effects of the blood products. Similarly, accounts of deficits in adults<sup>6</sup> and children<sup>7</sup> with thalamic tumors have helped to shape the understanding of the behavioral manifestations of thalamic lesions, but the structure-function analysis is too complex to establish with certainty that the behavior is a consequence of involvement of the thalamus alone. A patient with a penetrating injury of the left thalamus (intralaminar, dorsomedial, ventral lateral, and ventral anterior nuclei and mamillothalamic tract) developed

amnesia for verbal material, but the lesion also involved the posterior hypothalamus and both mamillary bodies.<sup>8</sup> Conclusions concerning thalamic functions in humans have also been derived from thalamotomy and thalamic stimulation studies (referred to below) and more recently from functional imaging studies that fall outside the scope of this review.

More precise anatomic-clinical correlations have been derived from studies of thalamic stroke. This article reviews the vascular syndromes resulting from thalamic lesions and uses these clinical manifestations along with connective studies in monkeys to formulate putative functional attributes of the thalamic nuclei.

### Thalamic Blood Supply and Vascular Syndromes

The details of thalamic vascularization were first studied by Duret<sup>9</sup> and Foix and Hillemand<sup>10</sup> and subsequently by Lazorthes<sup>11</sup> and Plets et al.<sup>12</sup> The subject was reevaluated by Percheron,<sup>13–17</sup> and subsequent reports helped to simplify the clinical-anatomic considerations.<sup>18–27</sup>

There are 4 major thalamic vascular territories, each with a predilection for supplying particular groups of nuclei. In the nomenclature used here, these are the (1) tuberothalamic,

Received March 13, 2003; accepted May 5, 2003.

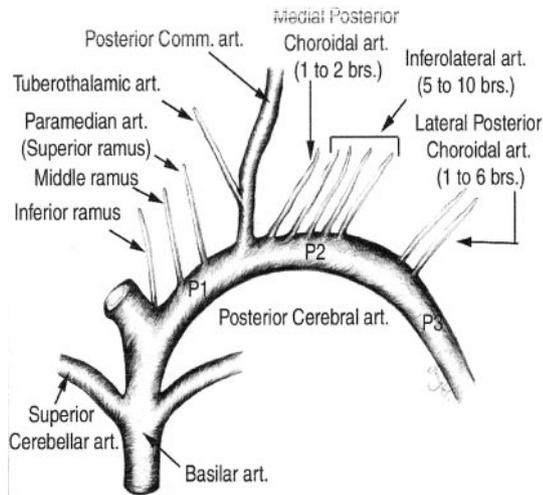
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*Stroke* is available at <http://www.strokeaha.org>

DOI: 10.1161/01.STR.0000087786.38997.9E



**Figure 1.** Artist's rendition of origin of arteries to thalamus arising from the vertebrobasilar system. Note that the medial posterior choroidal artery (art.) may arise before (P1) or after (P2) the origin of the posterior communicating artery. The inferolateral arteries may arise individually or from a common pedicle.

(2) inferolateral, (3) paramedian, and (4) posterior choroidal vessels (Figures 1 and 2; see Table 1 for alternative names of these blood vessels). The thalamic arteries vary between individuals with respect to the parent vessel from which each branch arises, the number and position of the arteries and their tributaries, and the nuclei supplied by each vessel (Figure 3). This situation is analogous to the lenticulostriate branches of the middle cerebral artery that irrigate the basal ganglia and internal capsule.<sup>18,24</sup>

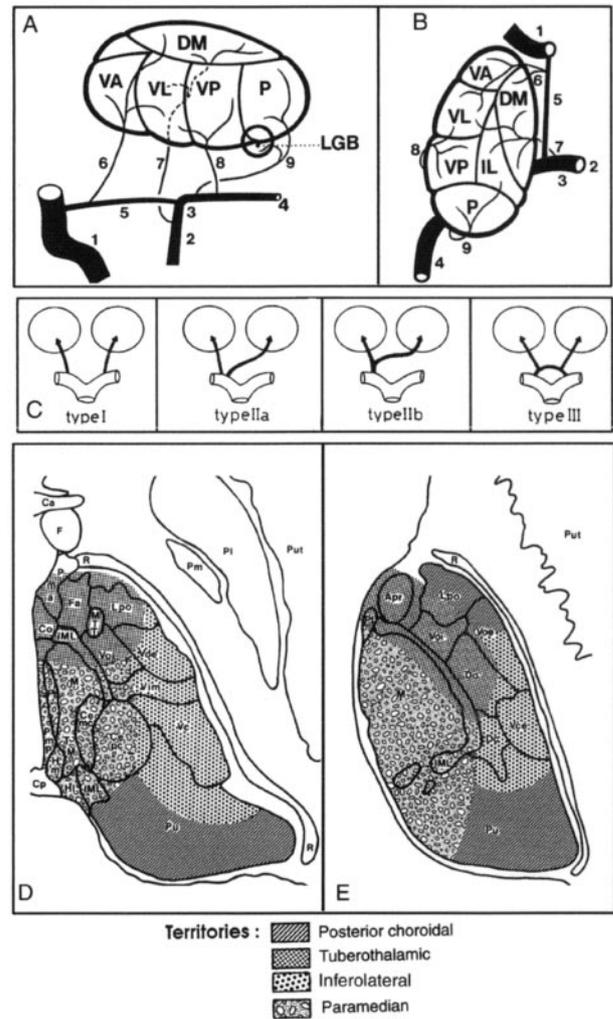
The optimal method for lesion-deficit correlations is comparison of the clinical symptoms with the location of the lesion on pathological examination of the brain. This is seldom possible, however, and much of the current understanding of the role of thalamus in behavior is derived from *in vivo* neuroimaging. The anatomic precision of MRI, including diffusion-weighted imaging, represents a considerable advance over CT, but the strength of the conclusions must nevertheless be tempered by the realization that determining the exact limits of the infarct and which neural components are damaged is not as precise with the use of these technologies as with serial histological sections examined under the microscope.

**Thalamic Nomenclature**

Recent histological analyses<sup>28,34</sup> have resolved the dissimilarities between human<sup>29,35</sup> and monkey<sup>36-39</sup> thalamic nomenclature and made it possible to describe human thalamus using nomenclature readily interchangeable with that of other species in which experimental work has been performed (Figure 4). This revised nomenclature is used in this analysis.

**Tuberothalamic Artery Infarction**

The tuberothalamic artery originates from the middle third of the posterior communicating artery. Within thalamus it follows the course of the mamillothalamic tract. It is absent in approximately one third of the normal population, in which case its territory is supplied by the paramedian artery.<sup>22</sup> The



**Figure 2.** Thalamic vascular supply. Schematic diagram of lateral (A) and dorsal (B) views of the 4 major thalamic arteries and the nuclei they irrigate, according to Bogousslavsky et al.<sup>23</sup> Numbers indicate the following: 1, carotid artery; 2, basilar artery; 3, P1 region of the posterior cerebral artery (mesencephalic artery); 4, posterior cerebral artery; 5, posterior communicating artery; 6, tuberothalamic artery; 7, paramedian artery; 8, inferolateral artery; and 9, posterior choroidal artery. Terminology not defined in the Appendix is as follows: DM, dorsomedial nucleus; IL, intralaminar nuclear complex; P, pulvinar; and VP, ventral posterior complex. C, Percheron's<sup>15</sup> representation of the varying patterns of origin of the paramedian artery off the mesencephalic/P1 artery. The illustrations in D and E, from De Freitas and Bogousslavsky,<sup>33</sup> are an adapted version of the conclusions of Von Cramon et al.<sup>21</sup> regarding the patterns of irrigation by the thalamic arterial supply to the thalamic nuclei. Thalamic terminology in this diagram is from Hassler<sup>29</sup>; see Figure 4.

tuberothalamic artery irrigates the reticular nucleus, ventral anterior nucleus (VA), rostral part of the ventrolateral nucleus (VL), ventral pole of the medial dorsal nucleus (MD), mamillothalamic tract, ventral amygdalofugal pathway, ventral part of the internal medullary lamina, and anterior thalamic nuclei: anteromedial (AM), anteroventral (AV), and anterodorsal (AD). Percheron<sup>13</sup> concludes that the anterior nuclear group is not supplied by this tuberothalamic artery but rather by the posterior choroidal artery, an assertion reflected in the discussion of Von Cramon et al.<sup>21</sup> This relation-

**TABLE 1. Thalamic Arterial Supply and Principal Clinical Features of Focal Infarction**

Thalamic Blood Vessel	Prior Designations	Nuclei Irrigated	Clinical Features Reported
<b>Tuberthalamic artery</b> <sup>22</sup> (arises from middle third of P.Comm)	Premamillary branch of thalamotuberian pedicle <sup>10</sup> Polar artery <sup>13,14</sup> Premamillary branch of P.Comm <sup>30</sup> Anterior internal optic artery <sup>9</sup> Inferior thalamic peduncle <sup>31</sup> Anterior thalamoperforating artery <sup>18,20</sup> Anterior thalamosubthalamic paramedian artery <sup>13</sup>	Reticular, intralaminar, VA, rostral VL, ventral pole of MD, anterior nuclei (AD, AM, AV), ventral internal medullary lamina, ventral amygdalofugal pathway, mamillothalamic tract	Fluctuating arousal and orientation Impaired learning, memory, autobiographical memory Superimposition of temporally unrelated information Personality changes, apathy, abulia Executive failure, perseveration True to hemisphere: language if VL involved on left; hemispatial neglect if right sided Emotional facial, acalculia, apraxia
<b>Paramedian artery</b> <sup>22</sup> (arises from P1)	Thalamoperforating pedicle <sup>10</sup> Retromamillary pedicle <sup>10</sup> Posterior thalamosubthalamic artery <sup>13</sup> Interpeduncular profundus artery <sup>32</sup> Superior ramus of interpeduncular artery <sup>26</sup>	MD, intralaminar (CM, Pf, CL), posteromedial VL, ventromedial pulvinar, paraventricular, LD, dorsal internal medullary lamina	Decreased arousal (coma vigil if bilateral) Impaired learning and memory, confabulation, temporal disorientation, poor autobiographical memory Aphasia if left sided, spatial deficits if right sided Altered social skills and personality, including apathy, aggression, agitation
<b>Inferolateral artery</b> <sup>22</sup> (arises from P2)	Thalamogeniculate pedicle <sup>10</sup> Posterior and external arteries of thalamus <sup>9</sup>		
Principal inferolateral branches		Ventroposterior complex: VPM, VPL, VPI Ventral lateral nucleus, ventral (motor) part	Sensory loss (variable extent, all modalities) Hemiataxia Hemiparesis Postlesion pain syndrome (Dejerine-Roussy): right hemisphere predominant
Medial branches		Medial geniculate	Auditory consequences
Inferolateral pulvinar branches		Rostral and lateral pulvinar, LD nucleus	Behavioral
<b>Posterior choroidal artery</b> (arises from P2)			
Lateral branches		LGN, LD, LP, inferolateral parts of pulvinar	Visual field loss (hemianopsia, quadrantanopsia)
Medial branches		MGN, posterior parts of CM and CL, pulvinar	Variable sensory loss, weakness, aphasia, memory impairment, dystonia, hand tremor

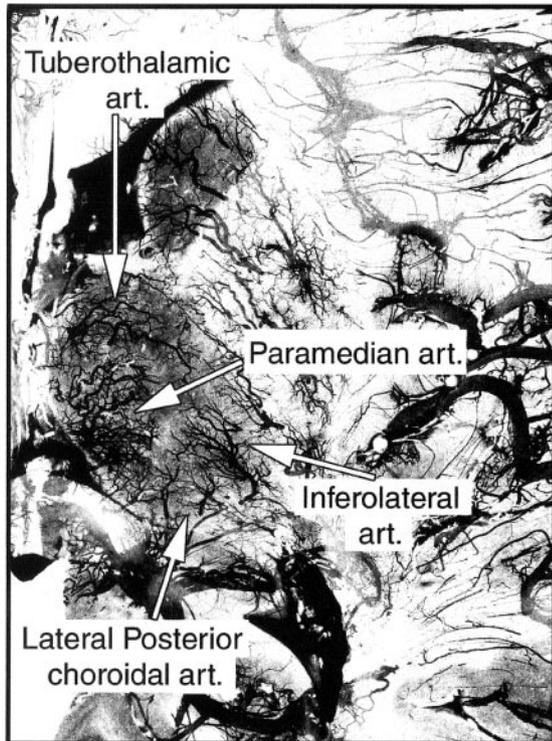
P.Comm indicates the posterior communicating artery.

ship is questionable, however, given the anatomic proximity of the tuberthalamic artery and the mamillothalamic tract to the AM/AV/AD nuclei and the demonstration<sup>25,27</sup> of involvement of the anterior nuclei along with the VA, VL, mamillothalamic tract, and internal medullary lamina, which are known to be supplied by the tuberthalamic artery.

The clinical syndrome resulting from infarction in the territory of the tuberthalamic artery (Figure 5) is characteristic, with the principal manifestation being severe and wide-ranging neuropsychological deficits.<sup>19,20,22,23</sup> In the early stages of infarction, patients exhibit fluctuating levels of consciousness and appear withdrawn. Persistent

personality changes include disorientation in time and place, euphoria, lack of insight, apathy, and lack of spontaneity. Emotional unconcern may be prominent.<sup>41</sup>

The major features of tuberthalamic infarction are impairment of recent memory, impairment of new learning, and temporal disorientation. These are more prominent with left-sided lesions, which also involve both verbal and visual memory impairments. Visual memory impairments are seen after right-sided lesions, and these in general result in less pervasive cognitive impairment. Von Cramon et al<sup>21</sup> considered the amnesic syndrome to represent a disconnection between anterior thalamic nuclei and hippocampal formation



**Figure 3.** Illustration of thalamic vascular complexity within the 4 major vascular territories, as shown by injection of tracer substance into postmortem human blood vessels by Salamon.<sup>40</sup> See Appendix for abbreviations.

by virtue of the disruption of the mamillothalamic tract and between amygdala and anterior nuclei by damage to the amygdalothalamic projections passing through the internal medullary lamina. This conclusion was supported by the patient of Graff-Radford et al,<sup>42</sup> who developed a severe amnesic syndrome after infarction of the internal medullary lamina. These authors used a monkey tract tracing study to confirm that the ventral amygdalofugal pathway that links the amygdala with the medial dorsal thalamic nucleus runs through the internal medullary lamina.

Language disturbances occur in left hemisphere lesions. These are characterized by anomia with decreased verbal output and impaired fluency, impairment of comprehension, and fluent paraphasic speech that may be hypophonic and lacking meaningful content. Semantic and phonemic paraphasic errors occur, with occasional neologisms and perseveration. Reading may be relatively preserved, although the comprehension of what is being read may be poor. In striking contrast, repetition is well preserved. These general characteristics of language impairment are consistent in different reports of thalamic aphasia, although the component features may vary between individuals, and the specific contribution of each thalamic nucleus to the impaired linguistic elements remains unresolved. Left thalamic lesions are also associated with acalculia.

Visual spatial processing deficits occur after right tuberothalamic lesions, in addition to visual memory deficits and hemispatial neglect.

“Emotional central facial paralysis” is characterized by good facial movement with volition but pronounced facial

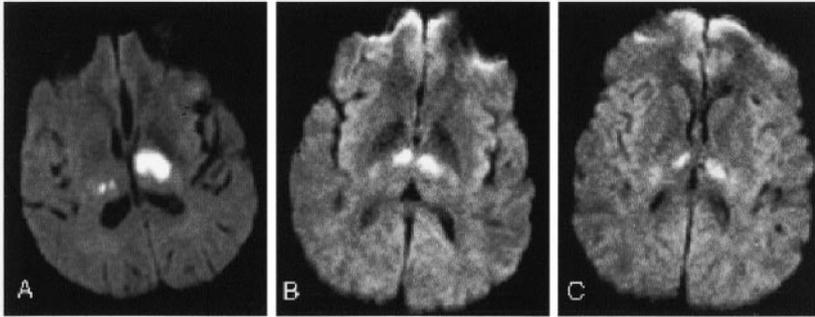
asymmetry during emotional displays such as laughing or crying. Constructional apraxia is seen after thalamic lesions on either side. Buccofacial and limb apraxia, in addition to severe anterograde amnesia, may be observed after left tuberothalamic infarction.<sup>43</sup> Mild to moderate contralateral weakness or clumsiness is seen in most cases, but sensory disturbances are rare, minimal, and transient.

In some patients with tuberothalamic territory infarction, the stroke is confined to the most anterior region of thalamus. In these rostral, or polar, infarctions within the tuberothalamic artery territory, the VL and MD nuclei appear to be entirely spared, and the lesion involves the anterior nuclei, VA, paramedian nuclei, mamillothalamic tract, and internal medullary lamina. The presentation in these patients is quite different from those with the larger tuberothalamic infarctions. Memory is severely impaired, but language is relatively spared. In the series of Ghika-Schmid and Bogouslavsky,<sup>27</sup> dysarthria, hypophonia, anomia, and decreased verbal and nonverbal fluency were noted, but comprehension, writing, reading, and repetition were normal. The major clinical feature in all patients, however, was persistent deficits in new learning, as well as apathy. Impaired planning and motor sequencing were evident, and severe perseverative behaviors were noted in thinking, spontaneous speech, memory, and executive tasks. Patients displayed superimposition of temporally unrelated information, producing a state of parallel expression of mental activities that the authors term *palipsychism*. The patient of Clarke et al,<sup>25</sup> with left polar thalamic infarction, had no aphasia apart from decreased verbal fluency. The case was marked by global amnesia, persistent verbal memory deficits, inattention, disorientation, and perseveration. Autobiographic memory and newly acquired information were disorganized with respect to temporal order. Impaired emotional engagement was noted in association with hypometabolism in the posterior cingulate cortex measures with positron emission tomography.

### Paramedian Artery Infarction

According to Percheron,<sup>13</sup> the paramedian arteries “represent a special differentiation of the highest of the group of paramedian arteries which can be found all along the neuraxis.” They arise from the P1 section of the posterior cerebral artery, to which the term “mesencephalic artery” may correctly be applied, as it is “the proximal stretch of the posterior cerebral artery from the bifurcation of the basilar to its junction with the posterior communicating” artery.<sup>44</sup> Tatu et al<sup>26</sup> adopt a useful approach of grouping the interpeduncular branches that arise from the mesencephalic, or P1 artery, into inferior, middle, and superior rami. The inferior ramus of the interpeduncular arteries that arises from the basilar bifurcation, as well as the middle ramus, which Percheron<sup>13</sup> together called the paramedian mesencephalic pedicle, irrigate the pons and midbrain<sup>18,26</sup> and can produce the “locked-in” component of the “top of the basilar” infarction.<sup>45</sup> The superior ramus that irrigates the thalamus corresponds to the posterior thalamo-subthalamic paramedian artery of Percheron<sup>13</sup> or, in the nomenclature adopted here, the paramedian artery. The paramedian arteries can arise as a pair from each P1, but they may arise equally from



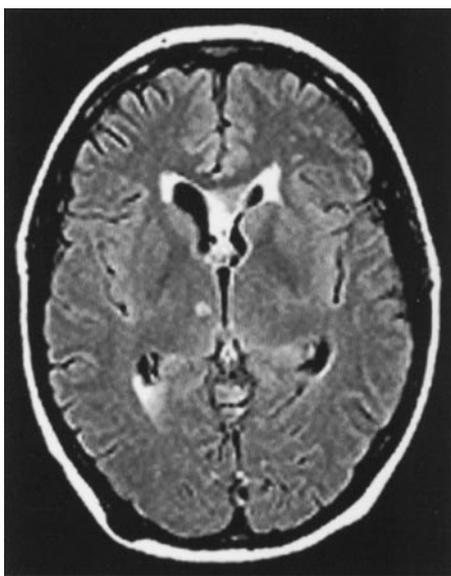


**Figure 5.** Diffusion-weighted MRI of acute thalamic infarction in 2 patients. A, Extensive tuberothalamic artery territory infarction on the left (right of image), as well as small lesions in the territory of the inferolateral arteries on the right. B and C, Bilateral infarction in a teenager with patent foramen ovale. The infarcted region is in the tuberothalamic artery territory, although this pattern of bilateral stroke is more usually seen after paramedian artery infarcts. This case is likely an example of the paramedian artery irrigating both the paramedian and the tuberothalamic “territories.” The patient made a complete recovery, and follow-up MRI scans revealed only minute lesions.

a common trunk off 1 P1, thus supplying thalamus bilaterally (Figure 2C).

The paramedian artery ascends within thalamus from its medial and ventral aspect to its lateral and dorsal part. It supplies a variable extent of thalamus but principally the dorsomedial nucleus, internal medullary lamina, and intralaminar nuclei: central lateral (CL), centromedian (CM), and parafascicular (Pf). The paraventricular nuclei, postero-medial part of VL, and ventromedial part of the pulvinar may also be supplied. The lateral dorsal (LD), lateral posterior (LP), and VA have been involved in some instances. When the tuberothalamic artery is absent, the paramedian artery may assume that territory as well, and thus infarction in this vascular territory can be devastating.

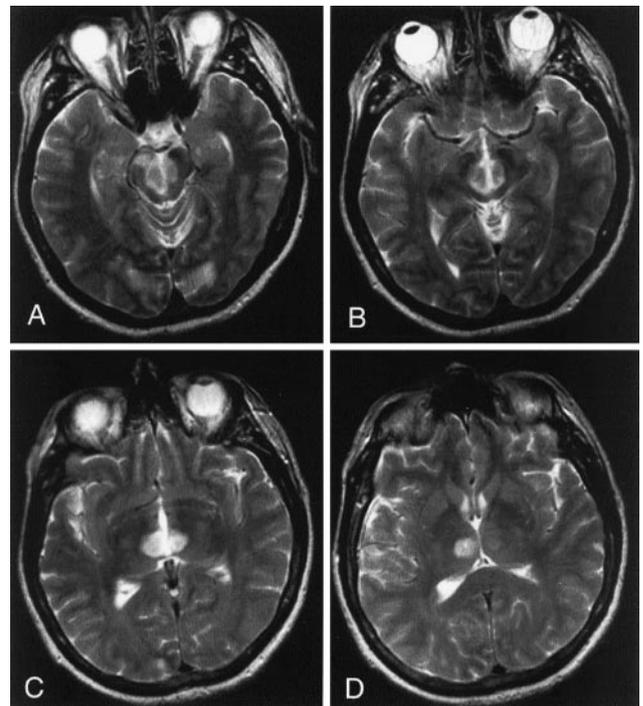
Unilateral thalamic infarction in the territory of the paramedian artery (Figure 6) produces neuropsychological disturbances predominantly in the areas of arousal and memory. A left-right asymmetry is evident in language versus visual-spatial deficits. Impairment of arousal with decreased and fluctuating level of consciousness is a conspicuous feature in the early stages, lasting for hours to days. Confusion, agitation, aggression, and apathy may be persistent features.<sup>18–20,23</sup>



**Figure 6.** Fluid-attenuated inversion recovery (FLAIR) MRI revealing right paramedian thalamic infarction in a patient with abrupt onset of confusion, confabulation, and autobiographical memory impairment in the setting of underlying ventriculitis after placement of a ventriculoperitoneal shunt for hydrocephalus.

Speech and language impairments are characterized by hypophonia and dysprosody, with frequent perseveration, markedly reduced verbal fluency, but generally preserved syntactic structure with occasional paraphasic errors and normal repetition: the *adynamic aphasia* of Guberman and Stuss.<sup>46</sup>

Bilateral infarction in the paramedian artery territory (Figure 7) may result in an acutely ill and severely impaired patient. Disorientation, confusion, hypersomnolence, deep coma, “coma vigil” or akinetic mutism (awake unresponsiveness), and severe memory impairment with perseveration and confabulation are prominent behavioral features, often accompanied by eye movement abnormalities.<sup>18–20,47</sup> The anterograde and retrograde memory deficit and apathy can be severe and persistent. The syndrome may be characterized in the late stages by inappropriate social behaviors, impulsive aggressive outbursts, emotional blunting, loss of initiative, and a reported absence of spontaneous thoughts or mental activities (see Reference 46 for case reports and early



**Figure 7.** T2-weighted MRI of bilateral paramedian thalamic infarction in a patient with a top of the basilar embolus involving midbrain and hypothalamus. Horizontal sections from ventral (A) to dorsal (D).

literature), conceptualized as loss of psychic self-activation.<sup>48,49</sup> In 1 case, Hodges et al<sup>50</sup> noted severe and systematic distortion of personally relevant autobiographical memory but relative sparing of knowledge of famous people and public events, suggesting a “thematic retrieval” memory disorder resulting from disconnection of frontal and medial temporal memory systems. Prominent disorientation in time is observed,<sup>18,42</sup> similar to the report of Spiegel et al<sup>51</sup> that introduced the term *chronotaraxis* to describe the phenomenon. Apraxia and dysgraphia are noted in some cases.<sup>18</sup> Complete recovery from bilateral paramedian thalamic infarction has occasionally been reported.<sup>52</sup>

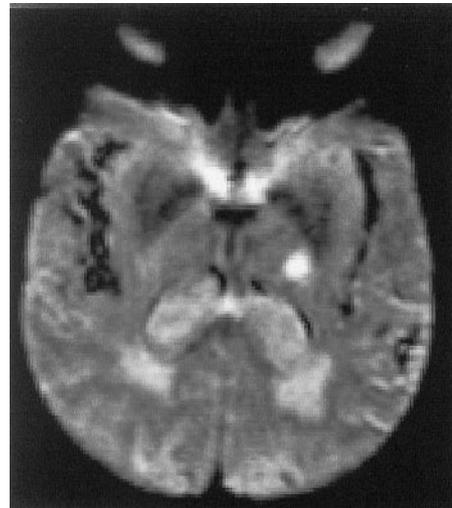
The amnesic syndrome resulting from paramedian territory infarction is similar to the thiamine-deficient Korsakoff syndrome that destroys the medial dorsal thalamic nuclei along with the mamillary bodies,<sup>53</sup> but the addition of the other behavioral features produces a constellation that led to the term *thalamic dementia*,<sup>44</sup> used also to describe the behavioral and cognitive consequences of thalamic involvement in Creutzfeldt-Jakob disease and fatal familial insomnia.<sup>54</sup> The phenomenon of transient global amnesia is thought to be related to transient ischemia in the paramedian thalamus; however, with the exception of rare case reports supporting this assertion anatomically,<sup>55–57</sup> there is no pathological verification of this association.

Elementary neurological signs from lesions of the paramedian artery include asterixis, complete or partial vertical gaze paresis, loss of convergence, pseudo-sixth nerve palsies, bilateral internuclear ophthalmoplegia, miosis, and even intolerance to bright light.<sup>23,46</sup> These are among the findings reported by Fisher<sup>1</sup> in cases of medially situated thalamic hemorrhage and are likely to result from lesions involving midbrain nuclei supplied by the inferior and middle rami of the interpeduncular arteries that arise from the P1 medial to the paramedian artery.

### Inferolateral Artery Infarction

The inferolateral arteries<sup>13,23</sup> are composed of 5 to 10 arteries that arise from the P2 branch of the posterior cerebral artery, ie, after the level of the posterior communicating artery. There are 3 main groups: the medial geniculate, principal inferolateral, and inferolateral pulvinar arteries. (1) The medial branch supplies the external half of the medial geniculate nucleus. (2) The principal inferolateral arteries, the “most voluminous, longest, most vertical of the short branches of the posterior cerebral artery,”<sup>13</sup> penetrate between the geniculate bodies, ascend in the lateral medullary lamina, and supply the major part of the ventral posterior nuclei (lateral [VPL], medial [VPM], and inferior [VPI]), as well as the ventral and lateral parts of the VL nucleus more rostrally. In the material of Percheron, the CM nucleus is not supplied by these vessels, as suggested by Foix and Hillemand<sup>10</sup> and Plets et al.<sup>12</sup> (3) The inferolateral pulvinar branches are posteriorly situated among the inferolateral arterial group and supply dorsal and posterolateral regions, including the rostral and lateral parts of the pulvinar and the LD nucleus.<sup>13,23,24,26</sup>

Patients with inferolateral artery infarction present with the thalamic syndrome described by Dejerine and Roussy,<sup>58</sup> namely, sensory loss to a variable extent, with impaired



**Figure 8.** Diffusion-weighted MRI of acute infarction in the left inferolateral artery territory causing the clinical triad of ataxia, mild hemiparesis, and hemisensory loss on the contralateral side.

extremity movement, sometimes with postlesion pain. The details of this presentation have been explored in numerous reports.<sup>24,59–61</sup> In the report of Bogousslavsky et al,<sup>23</sup> for example, sensory loss included all modalities but not necessarily in the same patient. Touch, temperature, and pin sense were decreased in 5 of 17 patients, whereas the remainder also had impairment of position and vibration sense. Ataxia with hemiparesis is also noted in this group,<sup>10,24,58,62,63</sup> and indeed, the combination of sensory loss with ataxic hemiparesis is strongly indicative of a thalamic lesion, although not pathognomonic (Figure 8). The thalamic pain syndrome of Dejerine and Roussy occurs following lesions of this region of thalamus, particularly the right thalamus,<sup>64</sup> but more complex behavioral syndromes have not been reported. The “thalamic hand” of Foix and Hillemand,<sup>10</sup> produced by lesions of the inferolateral artery, is flexed and pronated, with the thumb buried beneath the other fingers.

The complexity of the penetrating arteries that constitute the inferolateral arteries explains why small-vessel disease in this territory can have distinctly different presentations. Pure sensory stroke affecting face, arm, and leg in whole or in part results from variable involvement of the VPM (head and neck) or VPL nuclei (trunk and extremities). Infarction in those VL regions that convey cerebellar fibers to the motor-related cortices adds an ataxic component to the presentation. Cognitive and psychiatric presentations are notably missing from the descriptions of infarction restricted to the ventral posterior nuclei. The pulvinar is strongly associative, and LD is related to limbic cortices, but reports of the clinical manifestations of infarction in the inferolateral pulvinar vessels restricted to, or predominantly involving, these nuclei have yet to appear. Similarly, whereas the VL nucleus receives some supply from the inferolateral arteries, it is primarily supplied by the tuberothalamic artery territory, as described above. Incoordination is noted in patients with inferolateral territory infarction when it involves the VL, but the language disturbances that accompany VL lesions in

tuberothalamic artery infarction are not commonly seen after stroke in the inferolateral artery territory. Karussis et al<sup>65</sup> reported that 2 patients with inferolateral territory infarcts (no neuroimaging published) had aphasia with impaired fluency, comprehension, and naming, and Graff-Radford et al<sup>19</sup> described impaired fluency in patients with thalamocapsular infarction. This only occasional involvement of language in inferolateral territory lesions may reflect the fact that different nuclei may be involved within the territory of the infarcted inferolateral arteries. This is to be expected on the basis of the number of vessels that constitute the inferolateral constellation, normal variability in the arteriolar and capillary supply within thalamus, and the effect of slowly evolving lipohyalinosis on the zone of supply of the remaining intact blood vessels. The involvement of the different subcomponents of the VL, in particular, which link deep cerebellar nuclei with motor, premotor, prefrontal, and posterior parietal regions of the cerebral hemispheres, may also have markedly different clinical consequences.

### Posterior Choroidal Artery Infarction

The posterior choroidal arteries, like the inferolateral, arise from the P2 segment of the posterior cerebral artery and are made up of a number of branches. One to 2 medially placed branches arise adjacent to the origins of the posterior communicating artery (ie, at the distal P1 or proximal P2 segment of the posterior cerebral artery). These supply the subthalamic nucleus and midbrain, the medial half of the medial geniculate nucleus, the posterior parts of the intralaminar nuclei CM and CL (densocellular part of MD in the terminology of Olszewski,<sup>36</sup> CL in the terminology of Jones<sup>28</sup>), and the pulvinar nuclei.<sup>13,17,18,23,26</sup> The conclusion of Percheron<sup>13</sup> that it also supplies the AD, AV, and AM components of the anterior nuclear group is not universally shared.<sup>26</sup> In the lateral group of posterior choroidal arteries, 1 to 6 branches arise from the distal P2 segment of the posterior cerebral artery. Some of these supply medial temporal structures, but the thalamic arteries are destined for the lateral geniculate nucleus (LGN), the inferolateral region of the pulvinar, the lateral dorsal nucleus, and the lateral posterior nucleus. The LGN may also receive supply from the anterior choroidal artery,<sup>26</sup> although Percheron<sup>13,17</sup> could not confirm this in his material, and thalamus was not involved in the series of anterior choroidal artery infarcts reported by Decroix et al.<sup>66</sup>

There is only limited information on the clinical manifestations of infarction confined to thalamus in the distribution of the posterior choroidal arteries. The Lausanne group<sup>23</sup> reported 3 patients with LGN infarcts in whom quadrantanopsia was detected, along with impaired fast phase of the optokinetic response to the side opposite the lesion. One patient had contralateral hemibody numbness and mild aphasia. These authors<sup>67</sup> subsequently described 20 patients (10 personal, 10 previously published) in whom lateral posterior choroidal artery territory infarcts were associated with homonymous quadrantanopsia or horizontal "sectoranopsia," hemisensory loss, transcortical aphasia, and memory deficits, whereas medial infarcts produced eye movement disorders not suggestive of thalamic involvement. Three of their patients<sup>68</sup> with infarcts reportedly restricted to the pulvinar

developed a delayed complex hyperkinetic motor syndrome, including ataxia, rubral tremor, dystonia, myoclonus, and chorea, a constellation they termed the *jerky dystonic unsteady hand*. Sensory dysfunction in all 3 patients and a thalamic pain syndrome in 2 raise the question of whether the lesion involved other nuclei in addition to the pulvinar, however. The observation of incongruous homonymous hemianopic scotoma after lateral posterior choroidal artery stroke was also reported by Wada et al.<sup>69</sup> Spatial neglect was associated with lesions located primarily in the right pulvinar in the study of Karnath et al,<sup>70</sup> although the LD and VL were involved as well.

### Recovery of Function After Thalamic Infarction

The prognosis after thalamic hemorrhage is correlated with the volume of the hematoma, level of consciousness at onset, extent of motor weakness at presentation, and presence of intraventricular extension and hydrocephalus.<sup>4,71</sup> Prognosis after thalamic infarction is generally regarded as being rather good compared with lesions of the cerebral cortex or other subcortical structures, in both adults<sup>72,73</sup> and children,<sup>74,75</sup> but this generally applies to the low incidence of mortality and the good recovery from motor deficit. Persistence of cognitive and psychiatric manifestations after tuberothalamic or paramedian artery stroke is reported, but systematic longitudinal analyses have not been performed. Similarly, the incidence and long-term outcome of poststroke thalamic pain are not well established. Thus, the incidence of cognitive impairment and alterations of mood and personality after thalamic infarction are not known.

### Thalamic Fiber Systems

In attempting to understand the effects of thalamic lesions on behavior, it is necessary to consider the roles of the fiber systems that link thalamic nuclei with other brain regions and of the fiber systems that pass through the thalamus because some of the observed clinical phenomena may result from lesions of these fiber pathways rather than from lesions of the nuclei themselves.

Two intrathalamic fiber systems are particularly relevant for learning and memory. The mamillothalamic tract (of Vicq d'Azyr) connects the anterior thalamic nuclear group with the mamillary body, which in turn is linked with hippocampus and entorhinal cortex. This tract, along with the fornix, binds the anterior thalamic nuclei into the neural system that subserves learning and memory. The ventral amygdalofugal pathway, in contrast, links the amygdala with the medial part of MD, and damage may therefore contribute to amnesia as well as to emotional dysregulation.

Lesions of medial thalamus disrupt the corticofugal fibers that lead from motor and premotor cortices to the nucleus of Darkschewitsch and the interstitial nucleus of Cajal in the midbrain that are concerned with vertical gaze (up and down). They also disrupt the fibers to the rostral nucleus of the medial longitudinal fasciculus in the tectal region, which is concerned chiefly with downgaze.<sup>76</sup> Disruption of these pathways may be responsible for the aberrations of oculomotor control after thalamic lesions.

The thalamic “peduncles” at the superior, medial and inferior, and lateral aspects of thalamus convey information in and out of thalamus. Lesions restricted to these small white matter tracts occur rarely, if at all, but their trajectory to cerebral cortex and basal ganglia is relevant in considering the anatomic basis of thalamic effects on behavior. The anterior limb of the internal capsule conveys the bulk of the prefrontal and anterior cingulate interaction with thalamus (mostly the medial dorsal and the anterior thalamic nuclei), and focal lesions of the anterior limb, or genu, of the capsule essentially disconnect these nuclei from their cortical targets, producing a complex behavioral syndrome.<sup>77–79</sup> The anterior limb also conveys prefrontal input to the cerebrocerebellar system, and therefore the effects of this lesion are not exclusively on the thalamocortical interaction.

### A Note on Diaschisis

The observation that thalamic lesions produce alterations of higher-order behavior in addition to sensory and motor deficits raises for consideration the phenomenon of diaschisis. According to this hypothesis,<sup>80</sup> a lesion in one brain region (in contemporary parlance, one node in the distributed neural system subserving cognition) produces functional impairment in a distant but interconnected brain region. This manifests not only as loss of the behavior subserved by that circuit but also as altered metabolism on functional neuroimaging techniques (positron emission tomography and single-photon emission CT) that evaluate the integrity of the circuit. Depressed levels of metabolic activity in cerebral hemispheres have been observed after discrete thalamic infarction<sup>81,82</sup> and thalamotomy.<sup>82</sup> This diaschisis phenomenon depends on thalamocortical interconnections and provides corroborating physiological evidence for the hypothesis of distributed neural circuits and a mechanism for the behavioral deficits in patients with focal thalamic lesions.

### Putative Behavioral Roles of Thalamic Nuclei

Together with physiological studies in patients and connective neuroanatomic investigations in monkeys, clinical-anatomic correlations provide insights into the possible functional roles of the individual thalamic nuclei. Some of the conclusions are necessarily tentative, but they provide a useful framework for understanding the different clinical manifestations of focal thalamic lesions (Table 2).

#### *Reticular/Intralaminar Nuclei*

The reticular nucleus surrounds thalamus and conveys afferents from cerebral cortex exclusively into thalamus. It is critical for arousal and attention, maintains normal rhythmicity of thalamic neuronal firing, and is central in the consideration of the pathophysiology of epilepsy and related disorders<sup>83</sup> and in the discussion of the neural substrates of conscious awareness.<sup>84</sup>

The intralaminar nuclei include the paracentral (Pcn), central lateral (CL), centromedian (CM), and parafascicular (Pf) and a number of “midline” nuclei, such as the paraventricular, rhomboid, and reunions. These nuclei play a role in autonomic drive, and they may provide the striatum with attention-specific sensory information important for condi-

tional responses. They receive afferents from brain stem, spinal cord, and cerebellum and have reciprocal connections with cerebral hemispheres. The CM/Pf nuclei have strong reciprocal connections with the basal ganglia, arranged as tightly connected functional circuits. According to Sidibe et al,<sup>85</sup> a “sensorimotor” circuit links the putamen with the CM through the ventrolateral part of the internal segment of the globus pallidus (GPI); a “limbic” circuit links the ventral striatum with the Pf through the rostromedial GPI; and cognitive circuits link the caudate with Pf through the dorsal GPI and through the pars reticulata of the substantia nigra. The midline nuclei receive input from the periaqueductal gray as well as the spinothalamic tract and constitute the medial thalamic system involved in processing the motivational-affective components of nociceptive information.<sup>86–88</sup>

#### *Limbic Nuclei*

The limbic thalamic nuclei are defined by their reciprocal anatomic connections with limbic structures in the cingulate gyrus, hippocampus, parahippocampal formation, entorhinal cortex, retrosplenial cortex, orbitofrontal and medial prefrontal cortices, and subcortical structures, including the mammillary bodies and amygdala.<sup>89–92</sup> They include the anterior nuclear group (ventral, medial, and dorsal [AV, AM, and AD nuclei]) and the dorsally and more posteriorly situated lateral dorsal (LD) nucleus. The mamillothalamic tract and ventral amygdalothalamic tract, which link the anterior nuclei with other limbic regions, pass through the anterior thalamus. Other nuclei, not traditionally thought of as limbic, have subcomponents that are reciprocally interconnected with the cingulate gyrus and other structures in the limbic system and may be considered limbic as well. These are the magnocellular division of the medial dorsal nucleus (MDmc), and parts of the medial pulvinar and VA nuclei. Like their cortical and other subcortical counterparts,<sup>93,94</sup> the limbic thalamic nuclei are critical for learning and memory, emotional experience and expression, drive, and motivation.

#### *Specific Sensory Nuclei*

The specific sensory nuclei include the medial geniculate nucleus (MGN), lateral geniculate nucleus (LGN), and ventroposterior nuclear group (lateral, medial, and inferior [VPL, VPM, and VPI]).

The functions of the MGN are confined to the auditory realm. By virtue of its connections with both primary and association auditory cortices, it is likely to play a role in higher-level auditory processing as well as in simple audition.<sup>95–97</sup>

The LGN, a critical component of the visual system, projects in a highly ordered manner to the primary and secondary visual cortices.<sup>98</sup> It also receives projections back from the visual areas,<sup>99</sup> indicating that higher-order processing can influence visual perception at an early stage.

The VPL and VPM nuclei are reciprocally interconnected with the primary somatosensory cortices. VPL serves body and limbs, and VPM serves head and neck.<sup>100</sup> Gustatory function is subserved by VPMpc, the parvicellular (small cell) division of VPM.<sup>101</sup> The somatotopy of these nuclei is precise, and lesions of VPL and VPM produce focal sensory deficits in the affected regions. The VPI nucleus has anatomic

**TABLE 2. Behavioral Role of Thalamic Nuclei\***

Major Functional Grouping	Thalamic Nuclei	Putative Functional Attributes
Reticular	Reticular	Arousal, rhythmicity, role in epileptogenesis
Intralaminar	CM, Pf, CL, Pcn, midline (reunions, paraventricular, rhomboid)	Arousal, attention, motivation, affective components of pain
Limbic	Anterior nuclear group (AD, AM, AV), lateral dorsal nucleus Other: MDmc, medial pulvinar, ventral anterior	Learning, memory, emotional experience and expression, drive, motivation
Specific sensory	Medial geniculate	Auditory
	Lateral geniculate	Visual
	Ventroposterior	
	Lateral (VPL)	Somatosensory body and limbs
	Medial (VPM)	Somatosensory head and neck
	Medial, parvicellular (VPMpc)	Gustatory
Effector	Inferior (VPI)	Vestibular
	Ventral anterior	
	Reticulata recipient	Complex behaviors
	Pallidal recipient	Motor programming
	Ventral medial	Motor
	Ventral lateral	
	Ventral part	Motor
Dorsal part	Language (dominant hemisphere)	
Associative	Lateral posterior	High order somatosensory and visuospatial integration - spatial cognition
	Medial dorsal	
	Medial, magnocellular (MDmc)	Drive, motivation, inhibition, emotion
	Intermediate, parvicellular (MDpc)	Executive functions, working memory
	Lateral, multiform (MDmf)	Attention, horizontal gaze
	Pulvinar	
	Medial	Supramodal, high-level association region across multiple domains
	Lateral	Somatosensory, visual association
	Inferior	Visual association
	Anterior (pulvinar oralis)	Intramodality somatosensory association, pain appreciation

\*See list of abbreviations in the Appendix.

connections with the rostral inferior parietal lobule and SII, ie, the second somatosensory area in the parietal operculum.<sup>102,103</sup> By virtue of its anatomic connections with the frontal operculum and physiological studies, VPI is also implicated in vestibular functions.<sup>104</sup>

Spinothalamic and trigeminothalamic inputs and the presence of topographically organized wide dynamic neurons and nociceptive-specific, or high-threshold, neurons in the ventroposterior nuclei (VPL, VPM, and VPI) facilitate the role of the sensory nuclei in the lateral, or specific, component of the pain system.<sup>88</sup>

#### **Effector Nuclei**

The effector or, more commonly, “motor” nuclei include the VA, ventromedial (VM), and VL nuclei. The VA nucleus, according to Ilinsky and Kultas-Ilinsky,<sup>37</sup> incorporates the ventral lateral anterior nucleus (VLa) of Jones.<sup>28</sup> Afferents to

specific and segregated subregions within VA are derived from the pars reticulata of the substantia nigra<sup>39,105</sup> and from the internal globus pallidus.<sup>37</sup> The reticulata recipient VA nucleus is linked with premotor, supplementary motor,<sup>106</sup> and prefrontal cortices,<sup>107</sup> as well as with the caudal parts of the posterior parietal cortices<sup>103</sup> and the rostral cingulate gyrus.<sup>108</sup> The pallidal recipient VA is linked with premotor cortices.<sup>28</sup> The functions of the VA nucleus are not well established. The dystonic component of motor syndromes from rostral thalamic lesions may result from involvement of VA neurons that are linked with motor and premotor cortices, whereas complex behavioral syndromes seen in lesions of anterior thalamus may be accounted for in part by VA regions linked with cingulate, prefrontal, and posterior parietal association and paralimbic cortices.

The VL nucleus has traditionally been regarded as relaying information from cerebellum to motor cortex, but this view

needs to be expanded. The posterior part of VL (VLp according to Jones<sup>28</sup>; VL according to Ilinsky and Kultas-Ilinsky<sup>37</sup>) has complex connections. Its ventral part is linked with the motor cortex<sup>109</sup> and is likely responsible for the ataxia and mild motor weakness after thalamic stroke. The dorsal part, however, has projections through its caudal and pars postrema subdivisions to the posterior parietal cortices,<sup>103</sup> prefrontal cortices,<sup>110–112</sup> and upper bank of the superior temporal sulcus.<sup>113</sup> Stimulation studies of VL suggest a role in articulation and language (perseveration from stimulation of left medial VL and misnaming and omissions with stimulation of left posterior VL), as well as in the encoding and retrieval of verbal (left) and nonverbal (right) information.<sup>114–117</sup> Early thalamotomy studies directed at VL (see reports<sup>115,117–119</sup>) indicated that left-sided lesions produced aphasic syndromes including anomia, reduced verbal output, semantic and phonemic paraphasic errors, agrammatism, perseveration, and alexia, but comprehension was relatively preserved and repetition was normal. Impaired articulation, including stuttering, and abnormalities of rhythm and modulation also resulted from left VL lesions, while bilateral lesions resulted in hypophonia. The precision of these early lesions is questionable because the postoperative course was complicated by impaired consciousness, “chronic organic brain syndrome,” hemiparesis, lateropulsion, impaired gait, and hemiballismus. Contemporary MRI-guided thalamotomy lesions in Parkinson’s patients aim for the posterior part of VL and the anterior part of the VP nucleus<sup>120</sup> and do not produce worsening of preexisting cognitive deficits<sup>121</sup> and only mild decline in verbal fluency and in executive functions.<sup>122</sup>

### **Associative Nuclei**

The associative thalamus includes the lateral posterior, medial dorsal, and pulvinar nuclei. The lateral posterior (LP) nucleus has strong anatomic links with the posterior parietal cortices,<sup>102,103,123</sup> medial and dorsolateral extrastriate cortices,<sup>124</sup> and paralimbic regions in the posterior cingulate and medial parahippocampal regions.<sup>92</sup> These connections suggest that its role is an integrative one in the intramodal and multimodal associative somatosensory and visual systems. It may be expected to participate in higher-order somatosensory and visual-spatial function, such as in goal-directed reaching<sup>125</sup> and possibly in conceptual and analytical thinking.

The medial dorsal (MD) nucleus has strong reciprocal connections with the prefrontal cortex.<sup>107,126–129</sup> The frontal behavioral syndromes following thalamic lesions routinely involve MD, but the unique architectonic features of the different MD sectors that are linked with different regions of the prefrontal cortex are likely to determine the precise nature of the behavioral deficit.

The medial part (magnocellular MD [MDmc]) is linked with paralimbic regions (medial and orbital prefrontal cortices) as well as with the amygdala, basal forebrain, and olfactory and entorhinal cortices.<sup>42,130</sup> Apathy, abulia, disinhibition, and failure to inhibit inappropriate behaviors are likely to result from MDmc lesions, but the extent to which the amnesic<sup>53</sup> and aphasic disorders<sup>23</sup> following medial thalamic lesions are a result of MDmc involvement is

unresolved because lesions are seldom confined to this subnucleus.

The intermediate part of MD (parvicellular MD [MDpc]) is linked with the dorsolateral and dorsomedial prefrontal cortices, areas 9 and 46. The properties of these cortical areas suggest that the executive function problems stemming from MD lesions, including poor working memory and perseveration, may be a result of involvement of MDpc.

The laterally placed multiformis part of MD (MDmf) is linked with area 8 in the arcuate concavity, and therefore the impairments of volitional horizontal gaze, as well as deficits in attention, are possibly related to involvement of this sector of MD.

Early evidence for the role of thalamus in cognition was provided by the demonstration of anomia in patients after stimulation of the pulvinar nucleus.<sup>114</sup> These investigators subsequently observed that stimulation of the left pulvinar disrupted verbal memory processing, and right pulvinar stimulation disrupted nonverbal memory processing. The pulvinar, like the MD nucleus, is not a homogeneous structure because it has subcomponents with unique architecture and connective properties; it is even more diverse than MD with respect to its connections and possibly with respect to its functional attributes as well.

Different subregions within the medial pulvinar (PM) are linked with the prefrontal cortex,<sup>92,131,132</sup> posterior parietal lobe,<sup>102,103,131</sup> auditory-related<sup>96</sup> and multimodal cortices of the superior temporal region,<sup>133</sup> paralimbic cingulate and parahippocampal cortices,<sup>92</sup> and the limbic insula.<sup>134</sup> Some reports have suggested that pulvinar contributes to language processing<sup>114</sup> and that pulvinar lesions result in spatial neglect<sup>70</sup> and affective and psychotic manifestations.<sup>135</sup> These anatomic connections and clinical features suggest that the medial pulvinar is truly “associative” thalamus.

The lateral pulvinar (PL) is linked with posterior parietal,<sup>103,131</sup> superior temporal,<sup>133</sup> and medial and dorsolateral extrastriate cortices,<sup>124</sup> as well as with superior colliculus.<sup>136</sup> It may contribute to the integration of somatosensory and visual information.

Inferior pulvinar (PI) is a strongly visual region, linked both with temporal lobe areas concerned with visual feature discrimination and with ventrolateral and ventromedial extrastriate areas concerned with the analysis of visual motion.<sup>124,137</sup> It also receives direct visual input from retinal ganglion cells<sup>138</sup> and from the visual neurons of the superior colliculus.<sup>136</sup>

The anterior pulvinar, or pulvinar oralis (PO), is interconnected with the intramodality somatosensory association cortices in the rostral part of the posterior parietal region and with SII, the second somatosensory region.<sup>102,103,125,131</sup> The PO nucleus may also be important in the appreciation of pain, among other possible functions.

Other nuclei such as the supragenulate, limitans, and posterior nuclei are small in size but may be important as part of the “posterior nuclei of thalamus,”<sup>39</sup> thought to be responsible for the perception of pain in the animal model.

### **Conclusions**

Clinical presentations of thalamic stroke fall into 4 principal vascular syndromes (paramedian, tuberothalamic, inferolat-

eral, and posterior choroidal arteries), but there is variation within each of these syndromes because of factors related to vascular anatomy and pathology. The stroke syndromes are not specific to individual nuclei because even small, focal ischemic lesions are seldom confined within nuclear boundaries. The functional properties of the different thalamic nuclei are inferred from these clinical-anatomic observations and from the reciprocal connections with behaviorally defined regions of the cerebral cortex. The further study of clinicopathological correlations, the more sophisticated use of diaschisis as a method of analysis of human corticothalamic connectivity, and functional imaging of thalamus in normal subjects and experimental animals will be important future directions in the ongoing effort to understand the normal thalamus and the consequences of its disruption.

## Appendix

### Selected Abbreviations

AD indicates anterior dorsal thalamic nucleus; AM, anteromedial thalamic nucleus; AV, anteroventral thalamic nucleus; CeM, central medial thalamic nucleus; CL, central lateral thalamic nucleus; CM, centromedian thalamic nucleus; Csl, central superior lateralis thalamic nucleus; F, fornix; GM, medial geniculate nucleus; GMpc, medial geniculate nucleus, parvicellular part; Gpe, globus pallidus, external segment; Gpi, globus pallidus, internal segment; H, habenula; IML, internal medullary lamina of thalamus; L/Li, limitans thalamic nucleus; LD, lateral dorsal thalamic nucleus; LGN/LGd/LGB, lateral geniculate thalamic nucleus; LP, lateral posterior thalamic nucleus; MD, medial dorsal thalamic nucleus; MDdc, medial dorsal thalamic nucleus, densocellular part; MDmc, medial dorsal thalamic nucleus, magnocellular part; MDmf, medial dorsal thalamic nucleus, multiform part; MDpc, medial dorsal thalamic nucleus, parvicellular part; MGN/MG, medial geniculate thalamic nucleus; MTT, mamillothalamic tract; P1, P2, P3, first, second, and third divisions of posterior cerebral artery; Pcn, paracentral thalamic nucleus; Pf, parafascicular thalamic nucleus; PI, inferior pulvinar thalamic nucleus; PL/Pil, lateral pulvinar thalamic nucleus; PM/Plm, medial pulvinar thalamic nucleus; PO/Pla, anterior pulvinar (pulvinar oralis) thalamic nucleus; Po, posterior thalamic nucleus (Jones); Pt, paratenial thalamic nucleus; Pv, paraventricular thalamic nucleus; R, reticular thalamic nucleus; Re, reunions thalamic nucleus; RN, red nucleus; SG/Sg, supragenulate thalamic nucleus; Sm, stria medullaris; SN, substantia nigra; ST, subthalamic nucleus; VA, ventral anterior thalamic nucleus; VAF, ventral amygdalofugal pathway; VL, ventral lateral thalamic nucleus; VLa, ventral lateral anterior nucleus (Jones)=VLo (Olszewski); VLc, ventral lateral thalamic nucleus, pars caudalis (Olszewski)=VLP (dorsal part) (Jones); VLo, ventral lateral thalamic nucleus, pars oralis (Olszewski)=VLa (Jones); VLP, ventral lateral posterior nucleus (Jones); VLps, ventral lateral thalamic nucleus, pars postrema (Olszewski)=VLP (posterodorsal part) (Jones); VM, ventromedial thalamic nucleus; VMb, ventromedial thalamic nucleus, basal part; VP, ventral posterior thalamic nucleus; VPI, ventral posterior inferior thalamic nucleus; VPL, ventral posterolateral thalamic nucleus; VPLa, ventral posterior lateral nucleus, anterior division (Jones)=VPLc (Olszewski); VPLc, ventral posterolateral thalamic nucleus, pars caudalis (Olszewski)=VPLa (Jones); VPLo, ventral posterolateral thalamic nucleus, pars oralis (Olszewski)=VLP (ventral part) (Jones); VPM, ventral posteromedial thalamic nucleus; and VPMpc, ventral posteromedial thalamic nucleus, parvicellular part.

Comparisons of nomenclature for ventral tier thalamic nuclei are according to Olszewski<sup>36</sup> and Jones.<sup>28</sup>

### Acknowledgments

This work was supported in part by the McDonnell Pew Program in Cognitive Neuroscience and the Birmingham Foundation. The in-

valuable assistance of Charlene DeMong, BA, and Jason MacMore, BS, is gratefully acknowledged.

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