

## REVIEW

### Improving the understanding of neural mechanisms and guiding targeted therapy for clinical syndrome after thalamic infarction with advanced neuroimaging

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## Abstract

Thalamic infarction can result in a diverse array of symptoms, including motor and sensory deficits, memory and attention difficulties, and alterations in mood and behavior; these symptoms are collectively referred to as thalamic infarction syndrome. The neural mechanisms underlying these symptoms are not yet fully understood, hampering the development of effective and individualized treatments. The current understanding of the pathology of thalamic infarction syndrome is mainly based on conventional computed tomography/magnetic resonance imaging scans, which reveal blood supply to various nuclei groups and clinical features. Thalamic infarction syndrome can be categorized into four groups according to the affected territory and associated vascular syndrome. Recent advancements in neuroimaging techniques, which enable the precise identification of affected pivotal thalamic subnuclei, altered brain structures, white matter pathway integrity, abnormal neural activity, and maladaptive states of brain networks, can enhance our understanding of the clinical mechanisms and inform the development of more effective therapeutic strategies. This review summarizes research on the pathological neural mechanisms of thalamic infarction syndrome and highlights future directions.

**Keywords:** Thalamic infarction, Clinical syndrome, Neural mechanism, Advanced neuroimaging

## Introduction

As a subtype of ischemic stroke, which is one of the leading causes of mortality and long-term disability worldwide [Wu et al., 2019], thalamic infarction accounts for 11% of posterior circulation infarcts and 3–4% of cerebral ischemic events [Gurley & Edlow, 2019; Saez de Ocariz et al., 1996] and represents a common and complex clinical entity.

The thalamus, which comprises several functional nuclei, plays an important role in a variety of functions, such as regulating sensory information; controlling motor signals; managing arousal, alertness, and attention; and supporting certain cognitive processes [Annoni et al., 2003; Emmanuel Carrera, 2006; Schmahmann, 2003; Sieveritz & Raghavan, 2021; Van Der Werf et al., 1999]. Its most critical role is serving as a relay center for the exchange of information between the cortex and other parts of the central nervous system (CNS) [H, 1874; Saez de Ocariz et al., 1996; Schmahmann, 2003]. According to conventional neuroimaging techniques (computed tomography angiography; digital subtraction

angiography), the thalamus is supplied by four major vascular territories, each preferentially providing blood to specific groups of nuclei. This distinct pattern of blood supply to the thalamus has important implications for the development of a vascular syndrome after thalamic infarction [Emmanuel Carrera, 2006; H, 1874; Saez de Ocariz et al., 1996; Schmahmann, 2003]. Depending on the affected supplying arteries and insulted nuclei, damage to the thalamus can cause a variety of clinical syndromes, including hemisensory deficits, hemiparesis, refractory central neuropathic pain, hemispatial neglect, neuro-ophthalmologic defects (such as visual field loss), and cognitive or neuropsychological impairments [Castaigne et al., 1981; Emmanuel Carrera, 2006; Saez de Ocariz et al., 1996; Schmahmann, 2003], all of which can have a profound impact on personal quality of life.

Given the unsatisfactory effects of conventional drug therapy on clinical symptoms and the lack of more effective individualized therapeutic methods, elucidating the neural mechanisms underlying these syndromes is crucial for developing more effective and targeted treatments [Bogousslavsky et al., 1988; Hosomi et al., 2015; Klit et al., 2009; Wu et al., 2019; C. Ye et al., 2022; Chen Ye et al., 2022]. Advanced neuroimaging techniques, such as functional magnetic resonance imaging (fMRI) [Bonkhoff et al., 2020, 2021; He et al., 2021; Salvalaggio et al., 2020; Wang et al., 2021], diffusion tensor imaging (DTI) [Chen et al., 2019; Jang et al., 2019; Li et al., 2011; Nemati et al., 2022] and positron emission tomography (PET) [Dieterich et al., 2005; Dieterich & Brandt, 2008; Kopelman, 2015; Rudolphi-Solero et al., 2022; Stenset et al., 2007; Weder et al., 1994; Willoch et al., 2004], have made important contributions to our understanding of these mechanisms. Thalamic infarction can lead to structural and functional changes in the brain, including altered white matter integrity [Chuo Li, 2011; Krause et al., 2012], cortical reorganization [He et al., 2021; Krause et al., 2014; Wang et al., 2021], and even brain network dysfunction [Favaretto et al., 2022; He et al., 2021; Hosomi et al., 2015; Wang et al., 2021]. These changes can affect both adjacent and distant brain regions, such as the basal ganglia (motor pathways) [Favaretto et al., 2022; Wang et al., 2021], prefrontal cortex [Krause et al., 2014], sensory pathways [He et al., 2021; Wang et al., 2021] and even vision pathways [Holly Bridge, 2011; Millington et al., 2014; Chen Ye et al., 2022], all of which may result in a wide range of clinical symptoms following thalamic infarction. As a result, there has been growing interest in developing neuroimaging-based targeted therapies for individuals affected by thalamic infarction [Elias et al., 2020; Guo et al., 2022; Hosomi et al., 2015]. One promising approach is the use of neuromodulatory techniques to enhance the function of undamaged brain regions and to promote plasticity in damaged regions, particularly in central poststroke pain patients after thalamic infarction [Elias et al., 2020; Guo et al., 2022; Hosomi et al., 2015; Urits et al., 2020].

In this review, we briefly summarize the patterns of blood supply to the thalamus and corresponding vascular syndromes according to conventional brain imaging techniques and outline progress in understanding the pathological mechanisms of clinical syndromes and the neurobiology underlying stroke recovery after thalamic infarction using advanced neuroimaging techniques. Neural mechanism-based targeted therapeutic options and future directions for studying thalamic infarction are proposed.

### **Thalamic vascular syndrome by conventional neuroimaging techniques**

As first reported by Duret [H, 1874] and Foix et al., [1925], the blood supply to the thalamus mainly comprises four major arteries [Bogousslavsky et al., 1986, 1988; Castaigne et al., 1981; Clarke et al., 1994; Ghika-Schmid & Bogousslavsky, 2000; Graff-Radford et al., 1984, 1985; Tatu et al., 1998; von Cramon et al., 1985], including the tuberothalamic artery, inferolateral artery, paramedian artery, and posterior choroidal artery (Figure 1A and B). As each vessel supplies specific nuclei groups, combining

clinical symptoms and lesion locations (according to conventional brain scanning, CT, or MRI) has been proposed as a method of diagnosing thalamic infarction syndrome in clinical practice [Schmahmann, 2003]. These distinct supply patterns can be summarized as four standard territories (Figure 1C) and three variants as follows [Emmanuel Carrera, 2006; Schmahmann, 2003]:

### **1. Anterior territory infarction**

The anterior portion of the thalamus is supplied by the tuberothalamic artery, which is also known as the anterior thalamoperforating artery [Castaigne et al., 1981; Graff-Radford et al., 1984] or polar artery [G, 1976; Percheron, 1973]. The tuberothalamic artery arises from the middle third of the posterior communicating artery (PComm), and the clinical features of this infarction are characterized by severe and widespread neuropsychological abnormalities. During the early stages of infarction in this area, one may observe varying degrees of consciousness and silence. As the condition progresses, persistent personality changes can occur, including disorientation in time and space, apathy, and lack of self-awareness. Emotional disruptions may also be present. Other common symptoms of anterior thalamic infarction include difficulty [Foix, 1925] forming new memories, language impairment (difficulty speaking and articulating words), and dysarthria, which is related to the connection with the mammillary bodies in the hippocampus.

### **2. Paramedian territory infarction**

This area is supplied by the paramedian artery, which is also known as the thalamoperforating pedicle or thalamoperforating artery [Foix, 1925]. It branches off from the P1 segment of the posterior cerebral artery (PCA) or the interpeduncular segment and passes through the posterior perforated substance. Approximately 1/3 of infarcts in these territories occur bilaterally, as the left and right vessel branches originate from the same trunk, referred to as type B artery of Percheron. The paramedian region encompasses parts of the anterior thalamus, medial and ventral thalamus, and part of the interlamellar area. The nuclei supplied by this artery include the reticular, limbic, effector, conjunctive, specific sensory, and lamella nuclei. Given the numerous nuclei in this region, the symptoms of infarction can include visual impairment, hemiparesis, memory loss, and dyskinetic mutism caused by ischemia of the midbrain reticular formation, posterior hypothalamus, mammillary body, and/or anteromedial nuclei of the thalamus.

### **3. Inferolateral territory infarction**

The thalamic geniculate artery, which arises from the P2 branch of the PCA, supplies this region and is located posterior to the PComm. Infarction of the lateral hypothalamic artery can result in thalamic pain syndrome, which is primarily characterized by intractable pain that is not alleviated by pain medications, potentially due to the dissociation of thalamic and cortical inhibition. This pain can have an immediate or delayed onset and affects approximately 80% of patients with infarctions in this area. Other symptoms of thalamic pain syndrome, include sensory loss and mild hemiparesis (sensorimotor stroke). Sensory disturbances can encompass all modalities, and pure sensory strokes can also occur.

### **4. Posterior territory infarction**

This region is supplied by the posterior choroidal artery, which arises from the P2 segment of the PCA and comprises a group of small vessels. Reports on infarctions in this area are scarce. According to the existing data, the most common symptom is quadrant blindness, which can be accompanied by loss of lateral sensation, aphasia, and memory impairment. A distinctive manifestation of posterior choroidal artery infarction is the ocular motor disturbance, which is relatively rare and not specific to thalamic infarction. Patients may also experience a complex hyperkinetic syndrome with a delayed onset, which can include ataxia, tremor, dystonia, myoclonus, sensory loss, and pain.

## **5. The other three variants**

The other three variant thalamic territories are the anteromedial, central, and posterolateral regions. Infarctions in the anteromedial region, which is located in the posterior part of the anterior region and the anterior part of the paramedian region, often result in symptoms such as vertical gaze palsy, anterograde amnesia, aphasia, lack of motivation, and apathy. The central area represents the intersection of the four standard thalamic territories, and infarcts in this area are rare. When present, patients may experience cognitive loss, vertical gaze palsy, loss of arousal, and ataxia. The posterolateral thalamus is located in the anterior part of the posterior region and the posterior part of the inferior lateral region. Infarctions in this area manifest as aphasia, ataxia, and impairments in executive functions.

### **Advanced neuroimaging findings on thalamic infarction syndrome**

After a thalamic infarction, the resulting behavioral symptoms can be diverse and can mimic many "cortical" disorders or brainstem syndromes [Emmanuel Carrera, 2006]. Additionally, behavioral disorders associated with anterior and paramedian damage to the thalamus can be difficult to distinguish from primary psychiatric conditions, particularly when there are limited indications of neurological dysfunction [Emmanuel Carrera, 2006; Schmahmann, 2003]. Advanced imaging techniques, such as high-resolution MRI, can provide more precise diagnoses of damaged thalamic nuclei and their related clinical features [Emmanuel Carrera, 2006; Rondina et al., 2016; Vogels et al., 2021; Xu et al., 2018]. Tractography with diffusion MRI [Behrens et al., 2003] and functional imaging methods, such as fMRI [Johansen-Berg et al., 2005; Puig et al., 2018] and PET [Dieterich et al., 2005; Dieterich & Brandt, 2008; Kopelman, 2015; Rudolphi-Solero et al., 2022; Stenset et al., 2007], can also shed light on the impact of the thalamic injury on the CNS and provide valuable insights into the altered connectivity underlying behavioral deficits after thalamic infarction [Emmanuel Carrera, 2006; Ward, 2023].

#### **1. More precise thalamic lesion localization**

High-resolution MRI is one of the most commonly used neuroimaging techniques in the past few decades to evaluate nuclei after thalamic infarcts. This technique provides detailed information about the anatomy of the thalamus and surrounding structures, which can help to identify the location and extent of the infarct [Rondina et al., 2016; Xu et al., 2018].

High-resolution MRI is particularly useful for the evaluation of microinfarcts [Tao et al., 2022] and small vessel diseases [Jiang et al., 2021; Y. Yan et al., 2022], which can be difficult to detect with conventional CT or MRI. In addition, a combination of high-resolution MRI and the voxel-based lesion-symptom mapping (VLSM) technique [Bates et al., 2003; Chris Rorden, 2007] provides better insight into the association between the thalamic lesion and the symptom, allowing precision diagnosis and treatment. Sprenger et al., [2012] identified the ventral-posterior pulvinar nuclei (VPL) as the most high-risk subregion for infarcts leading to central pain of thalamic origin, with an odds ratio of 81, through lesion-symptom mapping on high-resolution T1-weighted images. Furthermore, Krause and colleagues precisely located the key subregions for the occurrence of pain as belonging to more posterior, inferior, and lateral parts of the VPL [Krause et al., 2012]. In a study including all poststroke pain types, they also found that VPL and the pulvinar subdivisions of the thalamus were significantly associated with clinical symptoms [Delboni Lemos et al., 2022]. In a cohort study evaluating multidimensional neuropsychological impairments in thalamic infarction patients, verbal memory deficits were correlated with anterior thalamic lesions [Scharf et al., 2022]. Recently, in a study focusing on thalamic infarction syndrome inducing thalamic aphasia, the lesioned left mediodorsal thalamic nucleus was identified as significantly associated with language impairments via VLSM [Stockert et al., 2022]. These results indicate that the

occurrence of specific thalamic infarctions, particularly central post-stroke pain (CPSP) of thalamic origin, is spatially specific, and lesions in different thalamic subregions are highly linked to the occurrence of specific symptoms, providing a theoretical basis for early and precise identification of such syndromes and improvement in individualized interventions.

## **2. Structural alterations in adjacent and remote brain regions**

The thalamus is a relay station for information in the CNS [Salt, 2001], and accumulating neuroimaging evidence indicates alterations in a wide range of CNS regions after thalamic infarction, as shown in Figure 2.

### **2.1 Adjacent structures**

Adjacent structures closely connected to the thalamus, such as the basal ganglia, hippocampus, and white matter between the thalamus and cortex, are affected by thalamic damage, and these impacts are correlated with certain clinical symptoms [Aggleton et al., 2010]. Damage to the thalamus leads to changes in the activity and morphology of the basal ganglia, which can result in various motor and sensory deficits [Kalia & Lang, 2015; Meyer et al., 2016]. fMRI data indicated abnormal dynamic functional states in the basal ganglia and their connections with the thalamus [Favaretto et al., 2022], which indicates a crucial impact of this region on recovery from neurological impairment and long-term outcomes. Li et al. found that thalamic infarction led to abnormalities in subfields of the hippocampus that were associated with impairments in memory. The underlying neural mechanisms may involve interactions between the thalamus and the hippocampus [Aggleton et al., 2010]. Moreover, thalamoinsular opercular white matter was found to significantly correlate with central pain syndrome in thalamic stroke patients [Delboni Lemos et al., 2022].

### **2.2 Distant areas**

In addition to areas adjacent to the thalamus, there is increasing interest in alterations of remote regions and their potential relationship with specific clinical syndromes in thalamic infarction [Koh et al., 2021; Weishaupt et al., 2015]. On the one hand, one mechanism underlying this impact in remote regions might be the concept of disconnection, or diaschisis, which has been proposed for decades and is used to explain a series of behavioral abnormalities after stroke [Carrera & Tononi, 2014; Corbetta et al., 2015; Griffis et al., 2019; Langen et al., 2018; Salvalaggio et al., 2020; Xia et al., 2021]. From this perspective, damage to the thalamus, the central hub of the brain, may result in a wide range of effects on connected (yet remote) brain regions. Several pieces of evidence support this proposed mechanism. First, a neuroimaging study combining voxel-based morphometry analysis and tract-based spatial statistics revealed changes in volume and white matter integrity in remote regions, including the brain stem, medial frontal gyrus, precentral gyrus, superior temporal gyrus, supplementary motor area, and postcentral gyrus [Yin et al., 2013], after thalamic infarction. Second, a longitudinal study found significant white matter volume loss in various brain regions in patients after thalamic infarction [Conrad, Habs, Ruehl, Bogle, et al., 2022]. Third, a study observed abnormal gray matter morphometry in the transverse temporal gyrus and superior temporal gyrus that significantly correlated with cognitive dysfunction [S. Yan et al., 2022]. Fourth, remote changes, characterized by secondary impairment of central thalamic radiation and atrophied ipsilesional primary somatosensory cortex, were found in thalamic infarction patients with sensory deficits [Chen et al., 2019]. Moreover, a recent study using advanced diffusion MRI techniques showed that structural connectivity (SC) to regions remote from the lesions (such as the superior longitudinal fasciculus and corpus callosum to the sensorimotor portion) was affected, correlating with somatosensory deficits [Koh et al., 2021].

On the other hand, accumulating evidence has suggested that neurological damage can lead to various

neuronal microenvironments, such as neural excitotoxicity, neurovascular structural changes, neurotransmitter abnormalities, and inflammatory responses, which can trigger increased neuronal excitability [Bee & Dickenson, 2008; Finnerup, 2008; Klit et al., 2009; Vestergaard et al., 1995]; in addition, abnormal changes such as hyperresponsiveness, hypoperfusion, and burst electrical activity have also been found in other sites secondary to thalamic vascular damage [Elias et al., 2020]. These mechanisms may lead to alterations in the distal septum, causing specific clinical features through central sensitization and reorganization of the distal cortex [Klit et al., 2009], ultimately leading to the occurrence of specific syndromes.

Thus, these findings, obtained using advanced neuroimaging techniques, demonstrate that thalamic infarction results in changes to the structure of both nearby and distant regions of the brain. These results not only provide a theoretical basis for early and precise identification and individualized intervention for these diseases but also propose a new explanation for the neural pathogenesis of specific clinical syndromes after thalamic infarction.

### **3. Disorder of brain network reorganization**

Neuroimaging research, with a graph theoretical approach, has demonstrated that the human brain is an integrated and complex network of structurally and functionally interconnected regions, which can be divided into nodes or modules with information communicated among components [Bullmore & Sporns, 2009, 2012].

As discussed above, the damage to the thalamus and subsequent disconnection of the central hub of the brain [Guillery, 1995] suggest that clinical syndromes after thalamic infarction are brain network disorders. On the one hand, recent fMRI studies [Chen et al., 2019; He et al., 2021; Wang et al., 2021] have shown altered functional connectivity among certain brain regions in thalamic infarction patients with central somatosensory deficits, indicating secondary changes such as dysrhythmic or hyperexcitable patterns of neural activity, which have been suggested to cause various chronic pain symptoms [Baliki et al., 2014; Ducreux et al., 2006; Seghier et al., 2005]. On the other hand, alterations in SC revealed by diffusion imaging have also been observed in patients with thalamic or spinothalamic pathway lesions and are related to certain symptoms, such as somatosensory deficits and even central poststroke pain [Chen et al., 2019; Chuo Li, 2011; Jang et al., 2017, 2018; Kishi et al., 2009; Li et al., 2011]. More importantly, some reports have shown that structural changes may induce cortical alterations and reorganization of functional networks in thalamic infarction patients [Conrad, Habs, Ruehl, Boegle, et al., 2022; Stockert et al., 2022]. We propose that these observations denote a model of clinical syndromes after thalamic infarction, in which the brain undergoes a complex process of network reorganization rather than simple recovery from damage to a group of focal nuclei or vascular incidents, as shown in Figure 2.

### **Potential targeted therapeutic options**

Hyperacute therapies (thrombolysis and endovascular therapy) have improved dramatically over the last few decades for ischemic stroke patients; however, a considerable number of patients do not receive reperfusion treatments or experience sequelae after these therapies [Wu et al., 2019], particularly in the thalamic infarction population. A comprehensive exploration of possibilities for neural repair via drugs or neuromodulatory therapy based on the neural mechanisms underlying stroke-induced brain damage is needed, with future clinical studies using advanced neuroimaging techniques [Bonkhoff et al., 2020, 2021; Puig et al., 2018].

Advanced neuroimaging techniques have the potential to greatly enhance our understanding of the neural mechanisms underlying clinical syndromes after thalamic infarction and guide the development of future therapeutic options. The following are ways in which advanced neuroimaging techniques can guide therapeutic options:

1. Facilitate the early identification and accurate prediction of individuals at high risk of developing specific clinical syndromes after thalamic infarction, enabling early intervention to prevent the occurrence of symptoms. For example, in acute thalamic VPL infarct patients with somatosensory deficits, anti-neuropathic pain treatment such as pregabalin can be administered and might help to prevent the development of central poststroke pain. The next step is to conduct interventional studies in acute thalamic infarction patients before the onset of symptoms, based on advanced neuroimaging findings that suggest crucial lesions or other pathological changes in other regions in the pre-symptomatic stage. The disease course of most of these clinical syndromes, especially neuropathic pain syndromes and cognitive disorders, is highly heterogeneous, with most of them occurring weeks to months after the onset of infarction. Therefore, for most patients, there is a sufficient time window to initiate prophylactic treatment.
2. Identify the specific neural mechanisms that are disrupted in clinical syndromes after thalamic stroke. This information can be used to develop and improve existing therapies, such as anticonvulsants and analgesic drugs for central poststroke pain.
3. Monitor the effectiveness of neuromodulatory therapies with more accurate targets, such as repetitive transcranial magnetic stimulation and transcranial direct current stimulation. Moreover, advanced neuroimaging techniques can be used to monitor the effectiveness of different therapies over time and measure brain changes after stimulation therapy.
4. Guide the development of rehabilitation techniques. For example, DTI can be used to identify disruptions in white matter pathways, and fMRI can be used to identify brain regions that are activated during rehabilitation exercises. These imaging techniques can help to optimize rehabilitation techniques and ensure that they target the appropriate neural mechanisms.
5. Facilitate the development of personalized treatment approaches. Advanced neuroimaging data can be used to identify individuals who are likely to respond best to a particular therapy and to develop customized rehabilitation programs for each patient.

### **Conclusions and future directions**

In conclusion, thalamic infarction can result in profound impairments in motor, sensory, and even cognitive/neuropsychological functions. To date, the combination of conventional neuroimaging techniques (CT/MRI) and neurological examination may be unable to meet the precise diagnostic and individualized treatment needs of patients. The use of advanced neuroimaging techniques can enhance the understanding of the neural mechanisms underlying these complex clinical syndromes and can guide specific therapies to target these mechanisms, increasing the chances of favorable outcomes for patients with thalamic infarction.

As the interruption of thalamocortical white matter connections is an important component of thalamic infarction syndromes, future research is needed to further understand the neural mechanisms of thalamic infarction using advanced neuroimaging techniques and larger sample sizes with longitudinal designs. In addition, most previous studies have employed unimodal or single-dimensional imaging techniques, and the imaging sequences used in some studies have substantial limitations, such as low spatial resolution

or the inability to identify the intersection of multiple fibers within a single voxel in DTI models [Kaden et al., 2007]. These limitations may reduce the accuracy of the results and restrict the clinical applications of the findings. Last but not least, these advanced techniques are ineffective without consideration of patients' explicit needs and benefits. Future research should focus on subtle improvements in patients' behavioral deficits with close attention from clinicians and combine this information with neuroimaging data to develop a more effective strategy for mitigating the impact of post-thalamic infarction impairments.

#### **Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

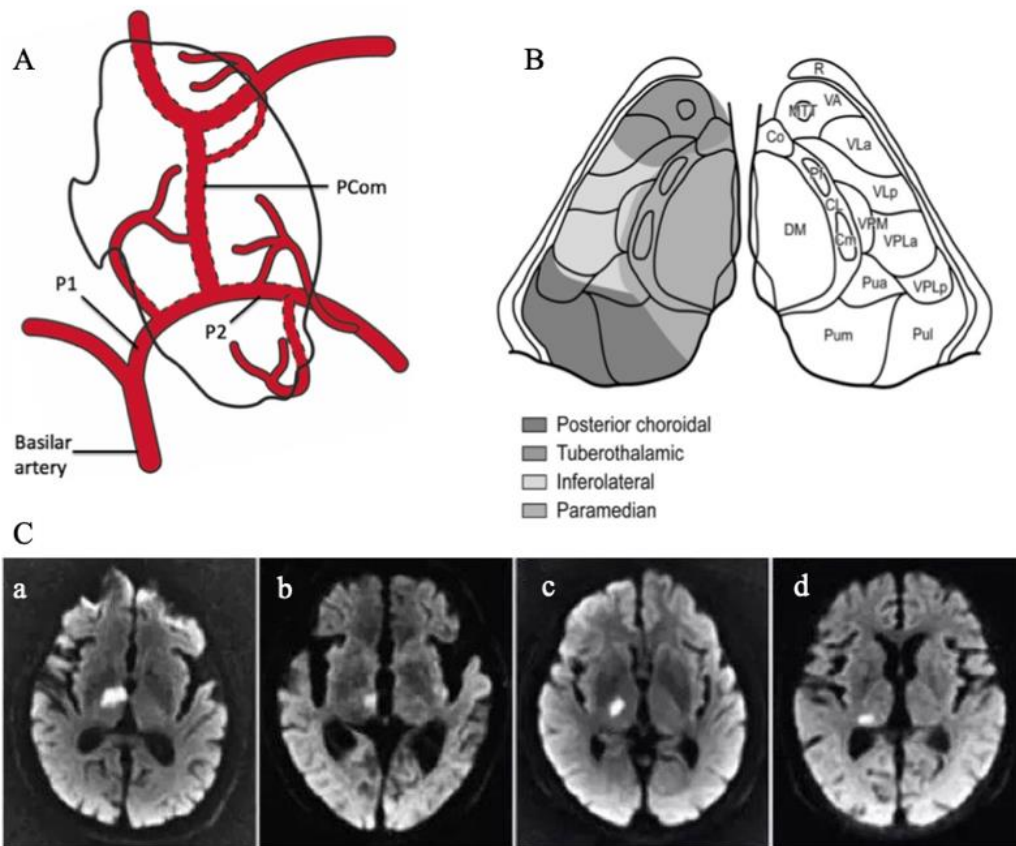
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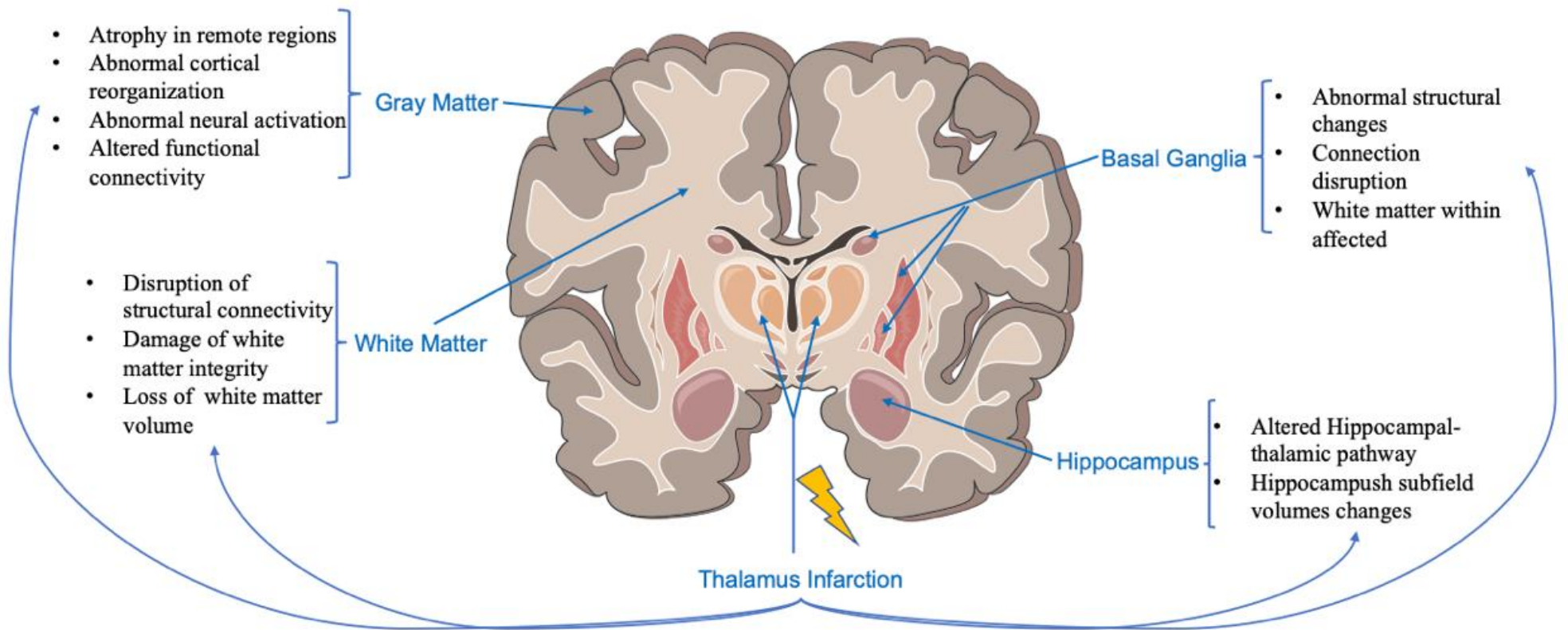
#### **Author Contributions**

C.Y. drafted the manuscript; C.Y. and R.P. performed the literature search; and B.W. supervised the research and revised the manuscript.





**Figure 1.** Schematic of the arterial supply of the thalamus and four diffusion-weighted MRI scans of standard infarction types. A, B. Illustration of the major supplying arteries of the thalamus and divisions of thalamic nuclei [Bordes et al., 2020; Emmanuel Carrera, 2006]. C. Diffusion-weighted images from four patients with standard infarction types: a. anterior territory infarction, supplied by the thalamic tuberosity artery; b. paramedian territory infarction, supplied by the paramedian artery; c. inferolateral territory infarction, supplied by the thalamic geniculate artery; and d. posterior territory infarction, supplied by the posterior choroidal artery. PComm: posterior communicating artery; P1: first segment of the posterior cerebral artery; P2: second segment of the posterior cerebral artery; CL: central lateral; CM: centromedial; Co: commissural; Cp: commissural posterior; DM: dorsomedial; MTT: mamillothalamic tract; Pua: pulvinar anterior; Pum: pulvinar medial; Pul: pulvinar lateral; Pf: parafascicularis; R: reticular; VA: ventral anterior; VLa: ventral lateral anterior; VLp: ventral lateral posterior; VPLa: ventroposterolateral anterior; VPLp: ventroposterolateral posterior; VPM: ventroposteromedial.



**Figure 1.** Illustration of affected brain regions after a thalamic infarction.

## References

- Aggleton, J. P., O'Mara, S. M., Vann, S. D., Wright, N. F., Tsanov, M., & Erichsen, J. T. (2010, Jun). Hippocampal-anterior thalamic pathways for memory: uncovering a network of direct and indirect actions. *Eur J Neurosci*, *31*(12), 2292-2307. <https://doi.org/10.1111/j.1460-9568.2010.07251.x>
- Annoni, J. M., Khateb, A., Gramigna, S., Staub, F., Carota, A., Maeder, P., & Bogousslavsky, J. (2003, Oct). Chronic cognitive impairment following laterothalamic infarcts: a study of 9 cases. *Arch Neurol*, *60*(10), 1439-1443. <https://doi.org/10.1001/archneur.60.10.1439>
- Baliki, M. N., Mansour, A. R., Baria, A. T., & Apkarian, A. V. (2014). Functional reorganization of the default mode network across chronic pain conditions. *PLoS One*, *9*(9), e106133. <https://doi.org/10.1371/journal.pone.0106133>
- Bates, E., Wilson, S. M., Saygin, A. P., Dick, F., Sereno, M. I., Knight, R. T., & Dronkers, N. F. (2003, May). Voxel-based lesion-symptom mapping. *Nat Neurosci*, *6*(5), 448-450. <https://doi.org/10.1038/nn1050>
- Bee, L. A., & Dickenson, A. H. (2008, Nov 15). Descending facilitation from the brainstem determines behavioural and neuronal hypersensitivity following nerve injury and efficacy of pregabalin. *Pain*, *140*(1), 209-223. <https://doi.org/10.1016/j.pain.2008.08.008>
- Behrens, T. E., Johansen-Berg, H., Woolrich, M. W., Smith, S. M., Wheeler-Kingshott, C. A., Boulby, P. A., Barker, G. J., Sillery, E. L., Sheehan, K., Ciccarelli, O., Thompson, A. J., Brady, J. M., & Matthews, P. M. (2003, Jul). Non-invasive mapping of connections between human thalamus and cortex using diffusion imaging. *Nat Neurosci*, *6*(7), 750-757. <https://doi.org/10.1038/nn1075>
- Bogousslavsky, J., Regli, F., & Assal, G. (1986, May-Jun). The syndrome of unilateral tuberothalamic artery territory infarction. *Stroke*, *17*(3), 434-441. <https://doi.org/10.1161/01.str.17.3.434>
- Bogousslavsky, J., Regli, F., & Uske, A. (1988, Jun). Thalamic infarcts: clinical syndromes, etiology, and prognosis. *Neurology*, *38*(6), 837-848. <https://doi.org/10.1212/wnl.38.6.837>
- Bonkhoff, A. K., Espinoza, F. A., Gazula, H., Vergara, V. M., Hensel, L., Michely, J., Paul, T., Rehme, A. K., Volz, L. J., Fink, G. R., Calhoun, V. D., & Grefkes, C. (2020, May 1). Acute ischaemic stroke alters the brain's preference for distinct dynamic connectivity states. *Brain*, *143*(5), 1525-1540. <https://doi.org/10.1093/brain/awaa101>
- Bonkhoff, A. K., Schirmer, M. D., Bretzner, M., Etherton, M., Donahue, K., Tuozzo, C., Nardin, M., Giese, A. K., Wu, O., V. D. C., Grefkes, C., & Rost, N. S. (2021, May). Abnormal dynamic functional connectivity is linked to recovery after acute ischemic stroke. *Hum Brain Mapp*, *42*(7), 2278-2291. <https://doi.org/10.1002/hbm.25366>

- Bordes, S., Werner, C., Mathkour, M., McCormack, E., Iwanaga, J., Loukas, M., Lammle, M., Dumont, A. S., & Tubbs, R. S. (2020, May). Arterial Supply of the Thalamus: A Comprehensive Review. *World Neurosurg*, *137*, 310-318. <https://doi.org/10.1016/j.wneu.2020.01.237>
- Bullmore, E., & Sporns, O. (2009, Mar). Complex brain networks: graph theoretical analysis of structural and functional systems. *Nat Rev Neurosci*, *10*(3), 186-198. <https://doi.org/10.1038/nrn2575>
- Bullmore, E., & Sporns, O. (2012, Apr 13). The economy of brain network organization. *Nat Rev Neurosci*, *13*(5), 336-349. <https://doi.org/10.1038/nrn3214>
- Carrera, E., & Tononi, G. (2014, Sep). Diaschisis: past, present, future. *Brain*, *137*(Pt 9), 2408-2422. <https://doi.org/10.1093/brain/awu101>
- Castaigne, P., Lhermitte, F., Buge, A., Escourolle, R., Hauw, J. J., & Lyon-Caen, O. (1981, Aug). Paramedian thalamic and midbrain infarct: clinical and neuropathological study. *Ann Neurol*, *10*(2), 127-148. <https://doi.org/10.1002/ana.410100204>
- Chen, L., Luo, T., Wang, K., Zhang, Y., Shi, D., Lv, F., Li, Y., Li, Y., Li, Q., Fang, W., Zhang, Z., Peng, J., & Yang, H. (2019). Effects of thalamic infarction on the structural and functional connectivity of the ipsilesional primary somatosensory cortex. *European Radiology*, *29*(9), 4904-4913. <https://doi.org/10.1007/s00330-019-06068-0>
- Chris Rorden, H.-O. K., and Leonardo Bonilha. (2007). Improving Lesion–Symptom Mapping. *Journal of Cognitive Neuroscience*
- Chuo Li, X. L., Sirun Liu, Anding Xu, Yusheng Zhang, Shihui Xing, Zhong Pei and Jinsheng Zeng. (2011). Early detection of secondary damage in ipsilateral thalamus after acute infarction at unilateral corona radiata by diffusion tensor imaging and magnetic resonance spectroscopy.
- Clarke, S., Assal, G., Bogousslavsky, J., Regli, F., Townsend, D. W., Leenders, K. L., & Bleicic, S. (1994, Jan). Pure amnesia after unilateral left polar thalamic infarct: topographic and sequential neuropsychological and metabolic (PET) correlations. *J Neurol Neurosurg Psychiatry*, *57*(1), 27-34. <https://doi.org/10.1136/jnnp.57.1.27>
- Conrad, J., Habs, M., Ruehl, R. M., Boegle, R., Ertl, M., Kirsch, V., Eren, O. E., Becker-Bense, S., Stephan, T., Wollenweber, F. A., Duering, M., Dieterich, M., & Zu Eulenburg, P. (2022, May). Reorganization of sensory networks after subcortical vestibular infarcts: A longitudinal symptom-related voxel-based morphometry study. *Eur J Neurol*, *29*(5), 1514-1523. <https://doi.org/10.1111/ene.15263>
- Conrad, J., Habs, M., Ruehl, R. M., Bogle, R., Ertl, M., Kirsch, V., Eren, O. E., Becker-Bense, S., Stephan, T., Wollenweber, F. A., Duering, M., Zu Eulenburg, P., & Dieterich, M. (2022, Feb 4). White

- matter volume loss drives cortical reshaping after thalamic infarcts. *Neuroimage Clin*, 33, 102953. <https://doi.org/10.1016/j.nicl.2022.102953>
- Corbetta, M., Ramsey, L., Callejas, A., Baldassarre, A., Hacker, Carl D., Siegel, Joshua S., Astafiev, Serguei V., Rengachary, J., Zinn, K., Lang, Catherine E., Connor, Lisa T., Fucetola, R., Strube, M., Carter, Alex R., & Shulman, Gordon L. (2015). Common Behavioral Clusters and Subcortical Anatomy in Stroke. *Neuron*, 85(5), 927-941. <https://doi.org/10.1016/j.neuron.2015.02.027>
- Delboni Lemos, M., Faillenot, I., Tavares Lucato, L., Jacobsen Teixeira, M., Mendonca Barbosa, L., Joaquim Lopes Alho, E., Bastos Conforto, A., Lilian de Lima Rodrigues, A., Galhardoni, R., Aparecida da Silva, V., Listik, C., Rosi, J., Peyron, R., Garcia-Larrea, L., & Ciampi de Andrade, D. (2022, Apr 1). Dissecting neuropathic from poststroke pain: the white matter within. *Pain*, 163(4), 765-778. <https://doi.org/10.1097/j.pain.0000000000002427>
- Dieterich, M., Bartenstein, P., Spiegel, S., Bense, S., Schwaiger, M., & Brandt, T. (2005, Sep). Thalamic infarctions cause side-specific suppression of vestibular cortex activations. *Brain*, 128(Pt 9), 2052-2067. <https://doi.org/10.1093/brain/awh551>
- Dieterich, M., & Brandt, T. (2008, Oct). Functional brain imaging of peripheral and central vestibular disorders. *Brain*, 131(Pt 10), 2538-2552. <https://doi.org/10.1093/brain/awn042>
- Ducreux, D., Attal, N., Parker, F., & Bouhassira, D. (2006, Apr). Mechanisms of central neuropathic pain: a combined psychophysical and fMRI study in syringomyelia. *Brain*, 129(Pt 4), 963-976. <https://doi.org/10.1093/brain/awl016>
- Elias, G. J. B., De Vloo, P., Germann, J., Boutet, A., Gramer, R. M., Joel, S. E., Morlion, B., Nuttin, B., & Lozano, A. M. (2020, Dec). Mapping the network underpinnings of central poststroke pain and analgesic neuromodulation. *Pain*, 161(12), 2805-2819. <https://doi.org/10.1097/j.pain.0000000000001998>
- Emmanuel Carrera, J. B. (2006). The thalamus and behavior Effects of anatomically distinct strokes. *Neurology*.
- Favaretto, C., Allegra, M., Deco, G., Metcalf, N. V., Griffis, J. C., Shulman, G. L., Brovelli, A., & Corbetta, M. (2022, Aug 29). Subcortical-cortical dynamical states of the human brain and their breakdown in stroke. *Nat Commun*, 13(1), 5069. <https://doi.org/10.1038/s41467-022-32304-1>
- Finnerup, N. B. (2008, Oct). A review of central neuropathic pain states. *Curr Opin Anaesthesiol*, 21(5), 586-589. <https://doi.org/10.1097/ACO.0b013e32830a4c11>
- Foix, C. (1925). Les artères de l'axe encéphalique jusqu'au diencéphale inclusivement. *Rev Neurol*, 2, 160-179.

- G, P. (1976). Les artères du thalamus humain, I: artère et territoire thalamiques polaires de l'artère communicante postérieure. *Rev Neurol (Paris)*, 132, 297–307.
- Ghika-Schmid, F., & Bogousslavsky, J. (2000, Aug). The acute behavioral syndrome of anterior thalamic infarction: a prospective study of 12 cases. *Ann Neurol*, 48(2), 220-227. <https://www.ncbi.nlm.nih.gov/pubmed/10939573>
- Graff-Radford, N. R., Damasio, H., Yamada, T., Eslinger, P. J., & Damasio, A. R. (1985, Jun). Nonhaemorrhagic thalamic infarction. Clinical, neuropsychological and electrophysiological findings in four anatomical groups defined by computerized tomography. *Brain*, 108 ( Pt 2), 485-516. <https://doi.org/10.1093/brain/108.2.485>
- Graff-Radford, N. R., Eslinger, P. J., Damasio, A. R., & Yamada, T. (1984, Jan). Nonhemorrhagic infarction of the thalamus: behavioral, anatomic, and physiologic correlates. *Neurology*, 34(1), 14-23. <https://doi.org/10.1212/wnl.34.1.14>
- Griffis, J. C., Metcalf, N. V., Corbetta, M., & Shulman, G. L. (2019, Sep 3). Structural Disconnections Explain Brain Network Dysfunction after Stroke. *Cell Rep*, 28(10), 2527-2540 e2529. <https://doi.org/10.1016/j.celrep.2019.07.100>
- Guillery, R. W. (1995, Dec). Anatomical evidence concerning the role of the thalamus in corticocortical communication: a brief review. *J Anat*, 187 ( Pt 3), 583-592. <https://www.ncbi.nlm.nih.gov/pubmed/8586557>
- Guo, S., Zhang, X., Tao, W., Zhu, H., & Hu, Y. (2022, Sep). Long-term follow-up of motor cortex stimulation on central poststroke pain in thalamic and extrathalamic stroke. *Pain Pract*, 22(7), 610-620. <https://doi.org/10.1111/papr.13137>
- Gurley, K. L., & Edlow, J. A. (2019, Nov). Avoiding Misdiagnosis in Patients With Posterior Circulation Ischemia: A Narrative Review. *Acad Emerg Med*, 26(11), 1273-1284. <https://doi.org/10.1111/acem.13830>
- H, D. (1874). Recherches anatomiques sur la circulation de l'encephale. *Arch Physiol Norm Pathol, series 2, vol 1:60–91, 316–354, 915–957.*
- He, M., Song, J., Luo, T., Wang, K., Li, Y., Lv, F., & Chen, L. (2021, Sep). Alteration of Resting-state Functional Connectivity in the Sensorimotor Network in Patients with Thalamic Infarction. *Clin Neuroradiol*, 31(3), 721-728. <https://doi.org/10.1007/s00062-020-00966-3>
- Holly Bridge, P. J., John Barbur, and Gordon T. Plant. (2011). Imaging Reveals Optic Tract Degeneration in Hemianopia. <https://doi.org/10.1167/iavs.10-5708/-/DCSupplemental>
- Hosomi, K., Seymour, B., & Saitoh, Y. (2015, May). Modulating the pain network--neurostimulation for



- central poststroke pain. *Nat Rev Neurol*, 11(5), 290-299.  
<https://doi.org/10.1038/nrneurol.2015.58>
- Jang, S. H., Kim, J., & Lee, H. D. (2018, Dec). Delayed-onset central poststroke pain due to degeneration of the spinothalamic tract following thalamic hemorrhage: A case report. *Medicine (Baltimore)*, 97(50), e13533. <https://doi.org/10.1097/MD.0000000000013533>
- Jang, S. H., Lee, J., & Yeo, S. S. (2017, Dec). Central post-stroke pain due to injury of the spinothalamic tract in patients with cerebral infarction: a diffusion tensor tractography imaging study. *Neural Regen Res*, 12(12), 2021-2024. <https://doi.org/10.4103/1673-5374.221159>
- Jang, S. H., Seo, J. P., & Lee, S. J. (2019). Diffusion Tensor Tractography Studies of Central Post-stroke Pain Due to the Spinothalamic Tract Injury: A Mini-Review. *Frontiers in Neurology*, 10. <https://doi.org/10.3389/fneur.2019.00787>
- Jiang, S., Cao, T., Yan, Y., Yang, T., Yuan, Y., Deng, Q., Wu, T., Sun, J., Wu, S., Hao, Z. L., Anderson, C. S., & Wu, B. (2021, Aug). Lenticulostriate artery combined with neuroimaging markers of cerebral small vessel disease differentiate the pathogenesis of recent subcortical infarction. *J Cereb Blood Flow Metab*, 41(8), 2105-2115. <https://doi.org/10.1177/0271678X21992622>
- Johansen-Berg, H., Behrens, T. E., Sillery, E., Ciccarelli, O., Thompson, A. J., Smith, S. M., & Matthews, P. M. (2005, Jan). Functional-anatomical validation and individual variation of diffusion tractography-based segmentation of the human thalamus. *Cereb Cortex*, 15(1), 31-39. <https://doi.org/10.1093/cercor/bhh105>
- Kaden, E., Knosche, T. R., & Anwander, A. (2007, Aug 15). Parametric spherical deconvolution: inferring anatomical connectivity using diffusion MR imaging. *NeuroImage*, 37(2), 474-488. <https://doi.org/10.1016/j.neuroimage.2007.05.012>
- Kalia, L. V., & Lang, A. E. (2015, Aug 29). Parkinson's disease. *Lancet*, 386(9996), 896-912. [https://doi.org/10.1016/S0140-6736\(14\)61393-3](https://doi.org/10.1016/S0140-6736(14)61393-3)
- Kishi, M., Sakakibara, R., Nagao, T., Terada, H., & Ogawa, E. (2009). Thalamic infarction disrupts spinothalamicocortical projection to the mid-cingulate cortex and supplementary motor area. *Journal of the Neurological Sciences*, 281(1-2), 104-107. <https://doi.org/10.1016/j.jns.2009.03.012>
- Klit, H., Finnerup, N. B., & Jensen, T. S. (2009, Sep). Central post-stroke pain: clinical characteristics, pathophysiology, and management. *Lancet Neurol*, 8(9), 857-868. [https://doi.org/10.1016/S1474-4422\(09\)70176-0](https://doi.org/10.1016/S1474-4422(09)70176-0)
- Koh, C. L., Yeh, C. H., Liang, X., Vidyasagar, R., Seitz, R. J., Nilsson, M., Connelly, A., & Carey, L. M. (2021, Jun 17). Structural Connectivity Remote From Lesions Correlates With Somatosensory Outcome Poststroke. *Stroke*, STROKEAHA120031520.

<https://doi.org/10.1161/STROKEAHA.120.031520>

- Kopelman, M. D. (2015, Jul). What does a comparison of the alcoholic Korsakoff syndrome and thalamic infarction tell us about thalamic amnesia? *Neurosci Biobehav Rev*, *54*, 46-56. <https://doi.org/10.1016/j.neubiorev.2014.08.014>
- Krause, T., Asseyer, S., Taskin, B., Flöel, A., Witte, A. V., Mueller, K., Fiebach, J. B., Villringer, K., Villringer, A., & Jungehulsing, G. J. (2014). The Cortical Signature of Central Poststroke Pain: Gray Matter Decreases in Somatosensory, Insular, and Prefrontal Cortices. *Cerebral Cortex*, *26*(1), 80-88. <https://doi.org/10.1093/cercor/bhu177>
- Krause, T., Brunecker, P., Pittl, S., Taskin, B., Laubisch, D., Winter, B., Lentza, M. E., Malzahn, U., Villringer, K., Villringer, A., & Jungehulsing, G. J. (2012). Thalamic sensory strokes with and without pain: differences in lesion patterns in the ventral posterior thalamus. *Journal of Neurology, Neurosurgery & Psychiatry*, *83*(8), 776-784. <https://doi.org/10.1136/jnnp-2011-301936>
- Langen, C. D., Cremers, L. G. M., de Groot, M., White, T., Ikram, M. A., Niessen, W. J., & Vernooij, M. W. (2018, Dec). Disconnection due to white matter hyperintensities is associated with lower cognitive scores. *NeuroImage*, *183*, 745-756. <https://doi.org/10.1016/j.neuroimage.2018.08.037>
- Li, C., Ling, X., Liu, S., Xu, A., Zhang, Y., Xing, S., Pei, Z., & Zeng, J. (2011, May 5). Early detection of secondary damage in ipsilateral thalamus after acute infarction at unilateral corona radiata by diffusion tensor imaging and magnetic resonance spectroscopy. *BMC Neurol*, *11*, 49. <https://doi.org/10.1186/1471-2377-11-49>
- Meyer, S., Kessner, S. S., Cheng, B., Bonstrup, M., Schulz, R., Hummel, F. C., De Bruyn, N., Peeters, A., Van Pesch, V., Duprez, T., Sunaert, S., Schrooten, M., Feys, H., Gerloff, C., Thomalla, G., Thijs, V., & Verheyden, G. (2016). Voxel-based lesion-symptom mapping of stroke lesions underlying somatosensory deficits. *Neuroimage Clin*, *10*, 257-266. <https://doi.org/10.1016/j.nicl.2015.12.005>
- Millington, R. S., Yasuda, C. L., Jindahra, P., Jenkinson, M., Barbur, J. L., Kennard, C., Cendes, F., Plant, G. T., & Bridge, H. (2014, Apr). Quantifying the pattern of optic tract degeneration in human hemianopia. *J Neurol Neurosurg Psychiatry*, *85*(4), 379-386. <https://doi.org/10.1136/jnnp-2013-306577>
- Nemati, P. R., Backhaus, W., Feldheim, J., Bonstrup, M., Cheng, B., Thomalla, G., Gerloff, C., & Schulz, R. (2022). Brain network topology early after stroke relates to recovery. *Brain Commun*, *4*(2), fcac049. <https://doi.org/10.1093/braincomms/fcac049>
- Percheron, G. (1973, Aug 29). The anatomy of the arterial supply of the human thalamus and its use for the interpretation of the thalamic vascular pathology. *Z Neurol*, *205*(1), 1-13.



<https://doi.org/10.1007/BF00315956>

- Puig, J., Blasco, G., Alberich-Bayarri, A., Schlaug, G., Deco, G., Biarnes, C., Navas-Marti, M., Rivero, M., Gich, J., Figueras, J., Torres, C., Dauris, I. E. P., Oramas-Requejo, C. L., Serena, J., Stinear, C. M., Kuceyeski, A., Soriano-Mas, C., Thomalla, G., Essig, M., Figley, C. R., Menon, B., Demchuk, A., Nael, K., Wintermark, M., Liebeskind, D. S., & Pedraza, S. (2018, Oct). Resting-State Functional Connectivity Magnetic Resonance Imaging and Outcome After Acute Stroke. *Stroke*, 49(10), 2353-2360. <https://doi.org/10.1161/STROKEAHA.118.021319>
- Rondina, J. M., Filippone, M., Girolami, M., & Ward, N. S. (2016). Decoding post-stroke motor function from structural brain imaging. *Neuroimage Clin*, 12, 372-380. <https://doi.org/10.1016/j.nicl.2016.07.014>
- Rudolphi-Solero, T., Trivino Ibanez, E. M., Aroui, T., Navidad, R. H., & Gomez-Rio, M. (2022, Apr 1). Dual-Phase 18F-Florbetaben PET/CT: Unsuspected Thalamic Infarction Mimicking Alzheimer Disease. *Clin Nucl Med*, 47(4), 333-335. <https://doi.org/10.1097/RLU.0000000000003955>
- Saez de Ocariz, M. M., Nader, J. A., Santos, J. A., & Bautista, M. (1996, Sep). Thalamic vascular lesions. Risk factors and clinical course for infarcts and hemorrhages. *Stroke*, 27(9), 1530-1536. <https://doi.org/10.1161/01.str.27.9.1530>
- Salt, T. (2001). EXPLORING THE THALAMUS.: By S. Murray Sherman and R. W. Guillery. 2000. New York: Academic Press. Price £39.95. Pp. 312. ISBN 0-12-305460-5. *Brain*, 124(10), 2120-2121. <https://doi.org/10.1093/brain/124.10.2120>
- Salvalaggio, A., De Filippo De Grazia, M., Zorzi, M., Thiebaut de Schotten, M., & Corbetta, M. (2020, Jul 1). Post-stroke deficit prediction from lesion and indirect structural and functional disconnection. *Brain*, 143(7), 2173-2188. <https://doi.org/10.1093/brain/awaa156>
- Scharf, A. C., Gronewold, J., Todica, O., Moeninghoff, C., Doeppner, T. R., de Haan, B., Bassetti, C. L. A., & Hermann, D. M. (2022, Jun). Evolution of Neuropsychological Deficits in First-Ever Isolated Ischemic Thalamic Stroke and Their Association With Stroke Topography: A Case-Control Study. *Stroke*, 53(6), 1904-1914. <https://doi.org/10.1161/STROKEAHA.121.037750>
- Schmahmann, J. D. (2003, Sep). Vascular syndromes of the thalamus. *Stroke*, 34(9), 2264-2278. <https://doi.org/10.1161/01.STR.0000087786.38997.9E>
- Seghier, M. L., Lazeyras, F., Vuilleumier, P., Schnider, A., & Carota, A. (2005, Mar). Functional magnetic resonance imaging and diffusion tensor imaging in a case of central poststroke pain. *J Pain*, 6(3), 208-212. <https://doi.org/10.1016/j.jpain.2004.11.004>
- Sieveritz, B., & Raghavan, R. T. (2021, Jun 9). The Central Thalamus: Gatekeeper or Processing Hub? *J*

*Neurosci*, 41(23), 4954-4956. <https://doi.org/10.1523/JNEUROSCI.0573-21.2021>

- Sprenger, T., Seifert, C. L., Valet, M., Andreou, A. P., Foerschler, A., Zimmer, C., Collins, D. L., Goadsby, P. J., Tölle, T. R., & Chakravarty, M. M. (2012). Assessing the risk of central post-stroke pain of thalamic origin by lesion mapping. *Brain*, 135(8), 2536-2545. <https://doi.org/10.1093/brain/aws153>
- Stenset, V., Grambaite, R., Reinvang, I., Hessen, E., Cappelen, T., Bjornerud, A., Gjerstad, L., & Fladby, T. (2007). Diaschisis after thalamic stroke: a comparison of metabolic and structural changes in a patient with amnesic syndrome. *Acta Neurol Scand Suppl*, 187, 68-71. <https://doi.org/10.1111/j.1600-0404.2007.00851.x>
- Stockert, A., Hormig-Rauber, S., Wawrzyniak, M., Klingbeil, J., Schneider, H. R., Pirlich, M., Schob, S., Hoffmann, K. T., & Saur, D. (2022, Oct 27). Involvement of Thalamocortical Networks in Patients With Poststroke Thalamic Aphasia. *Neurology*. <https://doi.org/10.1212/WNL.0000000000201488>
- Tao, W., Wang, Z., Liu, J., Li, J., Deng, Y., Guo, W., Wei, W., Wu, B., & Liu, M. (2022). Acute Cerebral Microinfarcts in Acute Ischemic Stroke: Imaging and Clinical Significance. *Cerebrovasc Dis*, 51(6), 755-763. <https://doi.org/10.1159/000524021>
- Tatu, L., Moulin, T., Bogousslavsky, J., & Duvernoy, H. (1998, Jun). Arterial territories of the human brain: cerebral hemispheres. *Neurology*, 50(6), 1699-1708. <https://doi.org/10.1212/wnl.50.6.1699>
- Urits, I., Gress, K., Charipova, K., Orhurhu, V., Freeman, J. A., Kaye, R. J., Kaye, A. D., Cornett, E., Delahoussaye, P. J., & Viswanath, O. (2020, Jul 15). Diagnosis, Treatment, and Management of Dejerine-Roussy Syndrome: a Comprehensive Review. *Curr Pain Headache Rep*, 24(9), 48. <https://doi.org/10.1007/s11916-020-00887-3>
- Van Der Werf, Y. D., Weerts, J. G., Jolles, J., Witter, M. P., Lindeboom, J., & Scheltens, P. (1999, Jan). Neuropsychological correlates of a right unilateral lacunar thalamic infarction. *J Neurol Neurosurg Psychiatry*, 66(1), 36-42. <https://doi.org/10.1136/jnnp.66.1.36>
- Vestergaard, K., Nielsen, J., Andersen, G., Ingeman-Nielsen, M., Arendt-Nielsen, L., & Jensen, T. S. (1995, May). Sensory abnormalities in consecutive, unselected patients with central post-stroke pain. *Pain*, 61(2), 177-186. [https://doi.org/10.1016/0304-3959\(94\)00140-A](https://doi.org/10.1016/0304-3959(94)00140-A)
- Vogels, V., Dammers, R., van Bilsen, M., & Volovici, V. (2021, Jul 27). Deep Cerebral Perforators: Anatomical Distribution and Clinical Symptoms: An Overview. *Stroke*, STROKEAHA120034096. <https://doi.org/10.1161/STROKEAHA.120.034096>
- von Cramon, D. Y., Hebel, N., & Schuri, U. (1985, Dec). A contribution to the anatomical basis of thalamic amnesia. *Brain*, 108 ( Pt 4), 993-1008. <https://doi.org/10.1093/brain/108.4.993>

- Wang, P., Zang, Z., Zhang, M., Cao, Y., Zhao, Z., Shan, Y., Ma, Q., & Lu, J. (2021). Longitudinal Changes of Sensorimotor Resting-State Functional Connectivity Differentiate between Patients with Thalamic Infarction and Pontine Infarction. *Neural Plast*, 2021, 7031178. <https://doi.org/10.1155/2021/7031178>
- Ward, N. (2023, Jan 10). Grasping the opportunity: better behavioural diagnoses will lead to better treatments for stroke. *Brain*. <https://doi.org/10.1093/brain/awad001>
- Weder, B., Knorr, U., Herzog, H., Nebeling, B., Kleinschmidt, A., Huang, Y., Steinmetz, H., Freund, H. J., & Seitz, R. J. (1994, Jun). Tactile exploration of shape after subcortical ischaemic infarction studied with PET. *Brain*, 117 ( Pt 3), 593-605. <https://doi.org/10.1093/brain/117.3.593>
- Weishaupt, N., Riccio, P., Dobbs, T., Hachinski, V. C., & Whitehead, S. N. (2015, Jun 17). Characterization of Behaviour and Remote Degeneration Following Thalamic Stroke in the Rat. *Int J Mol Sci*, 16(6), 13921-13936. <https://doi.org/10.3390/ijms160613921>
- Willoch, F., Schindler, F., Wester, H. J., Empl, M., Straube, A., Schwaiger, M., Conrad, B., & Tolle, T. R. (2004, Apr). Central poststroke pain and reduced opioid receptor binding within pain processing circuitries: a [11C]diprenorphine PET study. *Pain*, 108(3), 213-220. <https://doi.org/10.1016/j.pain.2003.08.014>
- Wu, S., Wu, B., Liu, M., Chen, Z., Wang, W., Anderson, C. S., Sandercock, P., Wang, Y., Huang, Y., Cui, L., Pu, C., Jia, J., Zhang, T., Liu, X., Zhang, S., Xie, P., Fan, D., Ji, X., Wong, K. L., Wang, L., & China Stroke Study, C. (2019, Apr). Stroke in China: advances and challenges in epidemiology, prevention, and management. *Lancet Neurol*, 18(4), 394-405. [https://doi.org/10.1016/S1474-4422\(18\)30500-3](https://doi.org/10.1016/S1474-4422(18)30500-3)
- Xia, C., Zhou, J., Lu, C., Wang, Y., Tang, T., Cai, Y., & Ju, S. (2021, Jul). Characterizing Diaschisis-Related Thalamic Perfusion and Diffusion After Middle Cerebral Artery Infarction. *Stroke*, 52(7), 2319-2327. <https://doi.org/10.1161/STROKEAHA.120.032464>
- Xu, T., Jha, A., & Nachev, P. (2018, Jul 1). The dimensionalities of lesion-deficit mapping. *Neuropsychologia*, 115, 134-141. <https://doi.org/10.1016/j.neuropsychologia.2017.09.007>
- Yan, S., Zhang, G., Zhou, Y., Tian, T., Qin, Y., Wu, D., Lu, J., Zhang, S., Liu, W. V., & Zhu, W. (2022, Mar). Abnormalities of Cortical Morphology and Structural Covariance Network in Patients with Subacute Basal Ganglia Stroke. *Acad Radiol*, 29 Suppl 3, S157-S165. <https://doi.org/10.1016/j.acra.2021.08.011>
- Yan, Y., Jiang, S., Yang, T., Yuan, Y., Wang, C., Deng, Q., Wu, T., Tang, L., Wu, S., Sun, J., & Wu, B. (2022, Mar 17). Lenticulostriate artery length and middle cerebral artery plaque as predictors of early neurological deterioration in single subcortical infarction. *Int J Stroke*,

17474930221081639. <https://doi.org/10.1177/17474930221081639>

Ye, C., Kwapong, W. R., Tao, W., Lu, K., Pan, R., Wang, A., Liu, J., Liu, M., & Wu, B. (2022). Alterations of optic tract and retinal structure in patients after thalamic stroke. *Front Aging Neurosci*, *14*, 942438. <https://doi.org/10.3389/fnagi.2022.942438>

Ye, C., Kwapong, W. R., Tao, W., Lu, K., Pan, R., Wang, A., Liu, J., Liu, M., & Wu, B. (2022). Characterization of Macular Structural and Microvascular Changes in Thalamic Infarction Patients: A Swept-Source Optical Coherence Tomography&ndash;Angiography Study. *Brain Sciences*, *12*(5), 518. <https://www.mdpi.com/2076-3425/12/5/518>

Yin, D., Yan, X., Fan, M., Hu, Y., Men, W., Sun, L., & Song, F. (2013, Jul). Secondary degeneration detected by combining voxel-based morphometry and tract-based spatial statistics in subcortical strokes with different outcomes in hand function. *AJNR Am J Neuroradiol*, *34*(7), 1341-1347. <https://doi.org/10.3174/ajnr.A3410>



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