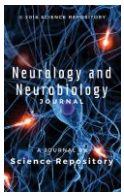

Available online at www.sciencerepository.org

Science Repository

Review Article

# Review Article: Clinical Symptoms and Structures Involved in Isolated Infarcts in the Branches of the Anterior, Middle, and Posterior Cerebral Arteries

***Melissa Martinez Johnson[1*] and Mohammed Teleb[2]***

*[1]Department of Neurology, The University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA*
*[2]Department of Neurology, Banner Desert Medical Center, Phoenix, Arizona, USA*

ARTICLE INFO

*Article history:*
Received: 16 July, 2022
Accepted: 2 August, 2022
Published: 24 August, 2022

Keywords:
*isolated infarct*
*branch occlusion*
*clinical symptoms*

ABSTRACT

**Introduction:** This review article outlines the clinical symptoms associated with isolated occlusions in the major branches of the anterior, middle, and posterior cerebral arteries.
**Methods:** Review article
**Conclusion:** A comprehensive understanding of isolated branch infarcts will deepen clinical acumen, aid in precise infarct localization, and optimize diagnostic management.



## Introduction

An understanding of the individual branches of the anterior, middle, and posterior cerebral arteries is paramount to the precise localization of an infarct. We aim to clearly name the major individual branches of the anterior, middle, and posterior cerebral arteries and list the structures they supply, in order to correlate the individual branches to the clinical symptoms. The current literature reviewed does not comprehensively collate the information in this format. A precise understanding of isolated branch infarcts will deepen clinical acumen and optimize diagnostic management.

## Anterior Cerebral Artery

The anterior cerebral artery (ACA) consists of the following branches, named for their destination: orbital frontal, frontal polar, anterior, middle, and posterior internal frontal, paracentral, superior parietal, and pericallosal arteries [1].

### I Orbital Frontal Artery

The orbital frontal artery supplies the gyrus rectus, olfactory bulb and tract, and orbital surface of the frontal lobe [2]. An isolated infarct in the orbital frontal artery will result in hand grasping or groping, and behavioural abnormalities, including euphoria and inappropriate laughing [3-5].

### II Frontopolar Artery

The frontopolar artery supplies parts of the medial and lateral surfaces of the frontal pole [2]. Although rare, one case report described conflictual task impairment as a result of an isolated infarct in the frontopolar artery [5].

### III Anterior Internal Frontal Artery

The anterior internal frontal artery supplies the anterior part of the superior frontal gyrus and cingulate gyrus [2]. An isolated infarct in the anterior internal frontal artery will result in transient akinesia, mutism, coordination impairment, and faciobrachial and faciobrachialcrural weakness [4, 5].

*Correspondence to: Melissa Martinez Johnson, Department of Neurology, The University of North Carolina at Chapel Hill, Physician's Office Building, 170 Manning Drive, Unit 3195-F, Chapel Hill 27517, North Carolina, USA; E-mail: Melissa.Johnson@unchealth.unc.edu*


http://dx.doi.org/10.31487/j.NNB.2022.03.01

**IV Middle Internal Frontal Artery**

The middle internal frontal artery supplies the middle part of the superior frontal and cingulate gyri [2]. An isolated infarct in the middle internal frontal artery would affect the supplementary motor cortex, which will result in transcortical motor aphasia, reduced verbal fluency, and the "alien hand sign" which has been described as a psychomotor disturbance with forced grasping, motor perseveration, and purposeful movements that are dissociated from conscious volition [5, 6]. A bilateral supplementary motor cortex lesion will result in gait apraxia and neuropsychologic disturbance, including dysexecutive impairment in planning and memory, confabulation, amnesia, and hemiballismus [7].

**V Posterior Internal Frontal Artery**

The posterior internal frontal artery supplies the posterior part of the superior frontal gyrus and cingulate gyrus [2]. An isolated infarct in the posterior internal frontal artery will result in leg predominant hemiparesis and limb apraxia [5].

**VI Paracentral Artery**

The paracentral artery supplies the paracentral lobule and the cingulate gyrus [8]. An isolated infarct in the paracentral artery will result in lower limb predominant hemiparesis and upper limb ataxia [9].

**VII Superior Parietal Artery**

The superior parietal artery supplies the superior part of the precuneus [2]. An isolated infarct in the superior parietal artery will result in lower limb predominant hemianaesthesia [5].

**VIII Pericallosal Artery**

The pericallosal artery supplies the rostrum, genu, body, and splenium of the corpus collosum [2]. An isolated infarct in the pericallosal artery will result in leg predominant hemiparesis due to involvement of the supplementary motor cortex [2].

**Middle Cerebral Artery**

The middle cerebral artery (MCA) branches are separated into the superior and inferior divisions. The superior division branches are the orbitofrontal, prefrontal, precentral, and central. The inferior division branches are the anterior parietal, posterior parietal, angular, temporo-occipital, posterior temporal, middle temporal, anterior temporal, and polar [1].

**I Superior Division**

**i Orbitofrontal and Prefrontal Arteries**

The orbitofrontal and prefrontal arteries supply the Broca speech area, frontal eye fields, and the premotor strip [1]. An isolated infarct in the orbitofrontal artery would result in a grasp reflex, contralateral extensor plantar response, gaze deviation, and altered consciousness [2]. A dominant hemisphere lesion in the orbitofrontal and prefrontal areas will also result in expressive aphasia and transcortical motor aphasia [1, 10]. A nondominant hemisphere lesion in the prefrontal artery will result in motor hemineglect [10].

**ii Precentral Artery**

The precentral artery supplies the lower part of the precentral gyrus [11]. A dominant hemisphere infarct in the precentral artery would result in transcortical motor aphasia, non-fluent aphasia, proximal hemiparesis, agraphia, and premotor syndrome, or difficulty switching from one motor sequence to another [10, 12].

**iii Central Artery**

The central artery supplies the central sulcus [13]. An isolated infarct in the central artery will result in hemisensory loss, ataxia, asterixis, tremor, and myoclonus [13]. A dominant hemisphere infarct in the central artery would also result in motor deficits, non-fluent aphasia, or Broca's aphasia [1, 10, 12].

**II Inferior Division**

**i Anterior Parietal Artery**

The anterior parietal artery supplies the posterior postcentral gyrus, parasagittal part of the central sulcus, anterior part of the inferior parietal gyrus, supramarginal gyrus, and parts of the upper and middle temporal gyri [13]. A dominant lobe lesion in the anterior parietal artery would result in hemianaesthesia, conduction aphasia, alexia, and agraphia [13, 14]. A nondominant hemisphere lesion in the anterior parietal artery will result in motor hemineglect [13].

**ii Posterior Parietal Artery**

The posterior parietal artery supplies the posterior parts of the superior and inferior parietal lobules, and the supramarginal gyrus [13]. An infarct in the posterior parietal artery will result in upper limb predominant hemianaesthesia, dysgraphesthesia, dysstereognosis, position sensory loss, visual field deficit, faciobrachial predominant motor weakness, kinetic ataxia, and hemiextinction [13]. A dominant lobe lesion in the posterior parietal artery would result in ideational apraxia, fluent aphasia, constructional apraxia, altered mentation, alexia with agraphia, leftward eye tracking impairment, and anomia [2, 10, 13].

**iii Angular Artery**

The angular artery supplies the angular gyrus [13]. An infarct in the dominant hemisphere angular artery would result in alexia, contralateral arm numbness, and Gertsmann's syndrome, or acalculia, agraphia, right-left disorientation, and finger agnosia [1, 2, 12]. An infarct in the nondominant hemisphere angular artery will result in hemineglect, visuospatial disturbances, asomatognosia, constructive apraxia, optic ataxia, and constructional apraxia [10, 12]. In bilateral angular artery lesions, Balint's syndrome can result with gaze paralysis, visual inattention, and optic ataxia [12].

**iv Temporo-Occipital Artery**

The temporo-occipital artery supplies the inferior part of the lateral occipital gyrus, and the posterior portions of the middle and inferior gyri [13]. A dominant hemisphere infarct in the temporo-occipital artery would result in hemianopia, fluent aphasia, or Wernicke's aphasia [10, 12].

**v Posterior Temporal Artery**

The posterior temporal artery supplies the posterior temporal area [2]. An isolated infarct in the posterior temporal artery would result in pure word deafness [2].

**vi Middle Temporal Artery**

The middle temporal artery supplies the superior temporal gyrus, the middle part of the middle temporal gyrus, and the middle and posterior portions of the inferior temporal gyrus [13]. An isolated infarct in the middle temporal artery would result in altered mentation, including agitation, confusion, delirium, hyperactivity, hallucinations, restlessness, and distractibility [13].

**vii Anterior Temporal Artery**

The anterior temporal artery supplies the anterior and lateral portions of the temporal lobe [1]. One case report of an isolated occlusion in the anterior temporal artery did not result in a notable clinical deficit [15].

**viii Polar Artery**

The polar artery supplies the anterior nuclei which have connections to the limbic system [16]. An infarct in the polar artery would result in abulia, apathy, amnesia, clumsiness, slow rapid alternating movements, drift, facial asymmetry, and paresthesias [13].

**Posterior Cerebral Artery**

The posterior cerebral artery (PCA) branches are the posterior communicating, anterior thalamoperforator, posterior thalamoperforator, thalamogeniculate, tuberothalamic, paramedian, medial posterior choroidal, lateral posterior choroidal, perisplenial, parieto-occipital, calcarine, and posterior temporal [1].

**I Anterior and Posterior Thalamoperforator Arteries**

The anterior thalamoperforator arteries supply the thalamic nuclei, the posterior aspect of the optic chiasm, the proximal part of the optic radiations, the posterior hypothalamus, and part of the cerebral peduncle [1]. The posterior thalamoperforator arteries supply the thalamus, subthalamic nucleus, and the nuclei and tracts of the upper midbrain, including the substantia nigra, red nucleus, oculomotor and trochlear nuclei, posterior portion of the internal capsule, and the cisternal segment of the oculomotor nerve [1]. An infarct in the anterior or posterior thalamoperforator arteries will result in cranial nerve III and IV palsies, hemiplegia, hemiballismus, cerebellar ataxia, and choreiform movement disorders [1].

**II Thalamogeniculate Artery**

The thalamogeniculate artery supplies the ventrolateral and ventroposterior thalamus [2]. The spinothalamic tracts run through these locations. An infarct in the ventrolateral thalamus will result in hemiataxia and dysarthria without hemisensory loss [2]. An infarct in the ventroposterior thalamus will result in hemisensory disturbance, occasionally limited to the proximal limbs and trunk and spare the face and distal limbs, and loss and of taste sensation [2]. If the infarct is limited to the medial parts of the ventroposterior thalamus, this will result in an isolated trigeminal sensory loss [2].

**III Tuberothalamic Artery**

The tuberothalamic artery supplies the ventral anterior nucleus of the thalamus, the rostral part of the ventrolateral thalamus, and the medial dorsal thalamus [2]. These areas connect to the hippocampus and amygdala and include the posterior cingulate cortex. An infarct in the tuberothalamic artery will result in neuropsychological deficits, fluctuating levels of consciousness, reserved or withdrawn behaviour, impaired memory formation, amnesia, euphoria, lack of insight, and lack of spontaneity [2]. Right-sided lesions result in hemineglect, and visual memory impairments. Left-sided thalamic lesions will additionally result in anomia, decreased verbal output, impaired fluency, impaired comprehension, paraphasic speech, acalculia, and limb apraxia [2].

**IV Paramedian Artery**

The paramedian artery supplies the paramedian parts of the upper midbrain and thalamus, including the intralaminar and dorsomedial nuclei [2]. This includes connections to the reticular activating system, mammillothalamic tract, and ventral amygdalafugal pathway [2]. An infarction in the paramedian artery will result in fluctuating levels of consciousness, somnolence, memory deficits, confusion, agitation, aggression, and apathy [2]. Left-sided lesions will result in hypophonia, reduced verbal fluency, perseveration, paraphasic errors, and apraxia [2]. A bilateral paramedian artery infarction can occur if one pedicle supplies both sides and will result in stupor, disorientation, dementia, unresponsiveness, non-verbal, apathy, memory impairment, confabulation, inappropriate social behaviours, impulsiveness, aggression, emotional blunting, vertical gaze palsy, convergence failure, pseudo-sixth cranial nerve palsy, pupillary changes, internuclear ophthalmoplegia, ocular tilt reaction, diplopia, dizziness, and gait difficulties [2].

**V Medial and Posterior Choroidal Arteries**

The medial posterior choroidal artery supplies the midbrain, tectal plate, pineal gland, posterior thalamus, habenula, medial geniculate body, the choroid plexus of the third ventricle, and the paramedial nuclei [1, 17]. The lateral posterior choroidal artery supplies the lateral geniculate body, the inferolateral region of the pulvinar, the lateral dorsal nucleus, the lateral posterior nucleus, and the anterior nucleus [2, 17]. An infarct in the posterior choroidal arteries will result in homonymous hemianopia, quadrantanopia, or sectoranopia (a wedge-shaped defect on each side of the horizontal meridian), hemisensory deficits, nystagmus, or impaired fast-phase optokinetic response to the opposite side of the

lesion [2, 17]. Dominant hemisphere infarcts will result in aphasia, paraphasia, and word finding difficulties [2]. Non-dominant hemisphere infarcts will result in spatial neglect due to involvement of the pulvinar [2].

### VI Perisplenial Artery

The perisplenial artery supplies the splenium and the posterior corpus callosum [18, 19]. An infarct in the left perisplenial artery would result in alexia without agraphia [17].

### VII Parieto-Occipital Artery

The parieto-occipital artery supplies the splenium and portions of the precuneus and cuneus [2]. An infarct in the parieto-occipital artery would result in hemianopia and visual neglect [17].

### VIII Calcarine Artery

The calcarine artery supplies the calcarine cortex, the medial surface of the occipital lobe to the occipital pole, the striate visual cortex, and the calcarine fissure [2, 17]. An infarct in the calcarine artery would result in hemianopia due to infarction of the striate visual cortex or interruption of the geniculo-calcarine tract as it nears the visual cortex, a superior quadrant field defect if only the lower bank of the calcarine fissure is involved, or an inferior quadrantanopia if the cuneus in the upper bank of the calcarine fissure is involved [17]. A bilateral calcarine artery infarct would result in cortical blindness, or Anton's syndrome, described as the patient cannot admit or recognize their loss of vision and experiences hallucinations in their blind fields [20].

### IX Posterior Temporal Artery

The posterior temporal artery supplies the posterior part of the parahippocampal and occipitotemporal gyri, and the lingual gyrus [21]. An infarct in the posterior temporal artery would result in hemianopia and visual neglect [18].

### Conclusion

In conclusion, isolated infarcts of the ACA, MCA, and PCA are rare. More often, multiple or all branches will be affected. However, a clear understanding of the vascular territories, the structures they supply, and their clinical syndromes will aid in localization of the lesion. Furthermore, a clear understanding of the most commonly described distributions will aid in the identification of anomalous vascular variations. Further research could focus on the variations of the vascular supplies and their clinical syndromes.

## REFERENCES


1. Morris P (2013) Practical Neuroangiography Third Edition.
2. Mohr J (2016) Stroke: Pathophysiology, Diagnosis, and Management - 6th ed.
3. Kumral E, Bayulkem G, Evyapan D, Yunten N (2002) Spectrum of anterior cerebral artery territory infarction: clinical and MRI findings. *Eur J Neurol* 9: 615-624. [Crossref]
4. Nagaratnam N, Davies D, Chen E (1998) Clinical Effects of Anterior Cerebral Artery Infarction. *J Stroke Cerebrovasc Dis* 7: 391-397. [Crossref]
5. Bogousslavsky J, Regli F (1990) Anterior Cerebral Artery Territory Infarction in the Lausanne Stroke Registry: Clinical and Etiologic Patterns. *Arch Neurol* 47: 144-150. [Crossref]
6. Goldberg G, Mayer NH, Toglia JU (1981) Medial frontal cortex infarction and the alien hand sign. *Arch Neurol* 38: 683-686. [Crossref]
7. Bejot Y, Caillier M, Osseby GV, Didi R, Salem DB (2008) Involuntary masturbation and hemiballismus after bilateral anterior cerebral artery infarction. *Clin Neurol Neurosurg* 110: 190-193. [Crossref]
8. Rothfus WE, Goldberg AL, Tabas JH, Deeb ZL (1987) Callosalmarginal infarction secondary to transfalcine herniation. *AJNR Am J Neuroradiology* 8: 1073-1076. [Crossref]
9. Bogousslavsky J, Martin R, Moulin T (1992) Homolateral ataxia and crural paresis: a syndrome of anterior cerebral artery territory infarction. *J Neurol Neurosurg Psychiatry* 55: 1146-1149. [Crossref]
10. Dunitz M (1997) Acute Stroke Treatment.
11. Frigeri T, Paglioli E, de Oliveira E, Rhoton Jr AL (2015) Microsurgical anatomy of the central lobe. *J Neurosurg* 122: 483-98. [Crossref]
12. Delgado MG, Bogousslavsky J (2012) Superficial Middle Cerebral Artery Territory Infarction. *Front Neurol Neurosci* 30: 111-114. [Crossref]
13. Caplan L, Gijn J (2012) Stroke Syndromes.
14. Bogousslavsky J (1991) Topographic Patterns of Cerebral Infarcts. *Cerebrovasc Dis* 1: 61-68.
15. Harrigan MR, Leonardo J, Gibbons KJ, Guterman LR, Hopkins LN (2005) CT Perfusion cerebral brain flow imaging in neurological critical care. *Neurocrit Care* 2: 352-366. [Crossref]
16. Chen XY, Wang Q, Wang X, Wong KS (2017) Clinical Features of Thalamic Stroke. *Curr Treat Options Neurol* 19: 5. [Crossref]
17. Caplan L. (2009) Caplan's Stroke: A Clinical Approach.
18. Kakou M, Destrieux C, Velut S (2000) Microanatomy of the pericallosal arterial complex. *J Neurosurg* 93: 667-675. [Crossref]
19. Krings T (2015) Neurovascular Anatomy in Interventional Neuroradiology: A Case-Based Approach.
20. Gilman S (2003) Essentials of Clinical Neuroanatomy and Neurophysiology 10th edition.
21. Haegelen C, Berton E, Darnault P, Morandi X (2012) A revised classification of the temporal branches of the posterior cerebral artery. *Surg Radiol Anat* 34: 385-391. [Crossref]