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**Callosomarginal infarction secondary to  
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# Callosomarginal Infarction Secondary to Transfalcial Herniation

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To evaluate the prevalence of anterior cerebral artery infarcts in the context of transfalcial herniation, 1100 cerebral CT scans were reviewed. Three patients had acute intracranial hemorrhages resulting in transfalcial herniation and subsequent focal ipsilateral paracentral lobule or superior frontal gyrus infarcts. The infarcts are presumed to have resulted from compression and compromise of the terminal portions of the callosomarginal artery against the falx.

Callosomarginal infarcts are reflective of severe, acute hemispheric insult. As such, they seem to portend a declining clinical condition, and thus may provide another CT indication of a poor clinical outcome.

When large enough, intracranial space-occupying lesions cause the brain to herniate under the falx, over the free tentorial margin, through the tentorial hiatus, over the sphenoid wing, or through the foramen magnum. Such herniation may be accompanied by vascular compression and compromise of blood supply. The most commonly recognized vascular syndromes are those caused by transtentorial herniation of the medial temporal lobe. Brainstem hemorrhage results from deformation of the paramedian and pontine arteries associated with distortion of the brainstem by the herniated hippocampal gyrus [1]. As such, this punctate hemorrhage usually contributes to the terminal phases of brain insult; it is not usually detected by CT or other neuroimaging techniques. Compression of the posterior cerebral artery between the hippocampus and the free tentorial margin may result in occipital lobe infarction [1]. This can often be detected on CT [2].

Although transfalcial herniation is quite commonly associated with cerebral mass, resultant vascular compression with hemorrhage or infarct is uncommon. The literature contains only a few pathologic descriptions of infarcts in the distribution of the anterior cerebral artery associated with this type of herniation [1, 3–5]. To date, however, there has been no specific CT documentation of such infarcts or the type of mass lesion with which they may occur. In a review of 1100 consecutive cerebral CT scans, we recognized three cases of anterior cerebral artery distribution infarcts occurring with acute transfalcial herniation secondary to intracranial hemorrhages. These are described below.

## Case Reports

### Case 1

A 69-year-old alcoholic man became comatose after a fall. Admission CT scans revealed a large, left hemispheric subdural hematoma; subarachnoid blood; and considerable transfalcial herniation to the right (Figs. 1A and 1B). Within hours, the subdural hematoma was evacuated, but the patient's level of consciousness did not improve significantly. CT scans 3 days later showed that the lateral ventricles returned to a normal position. Focal infarct was noted in the paracentral lobule and cingulate gyrus (Figs. 1C and 1D). The patient was without notable improvement until his death 2 weeks later.

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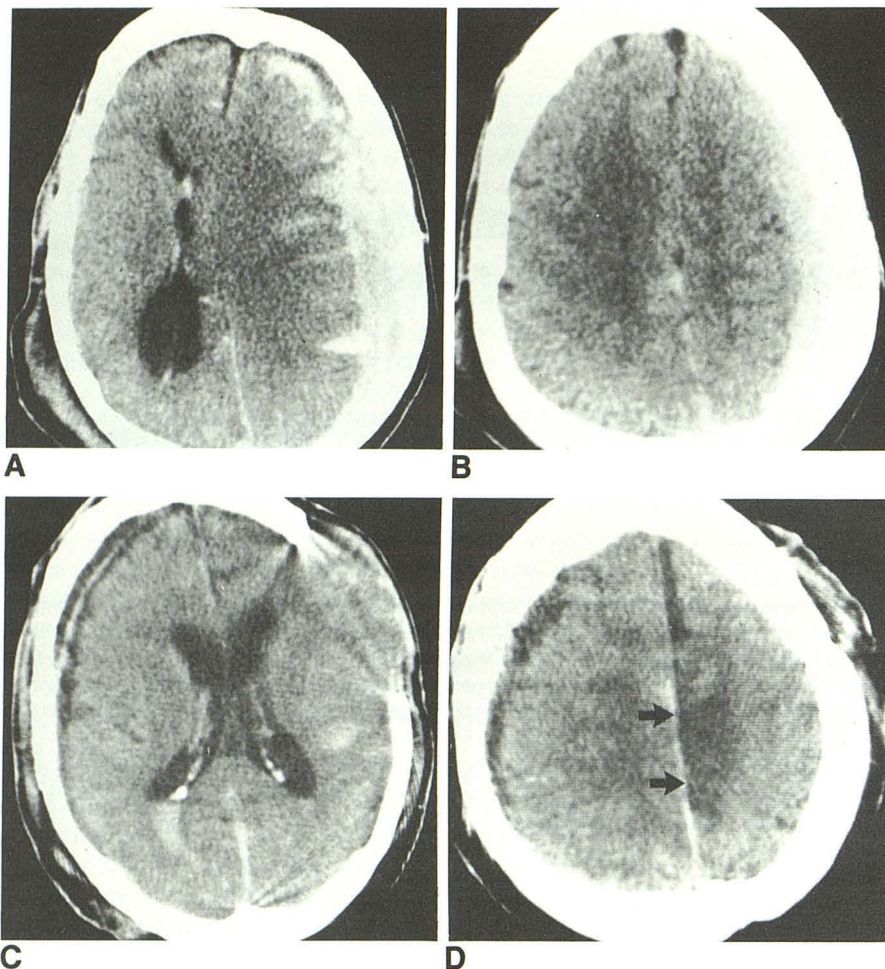


Fig. 1.—A and B, Large, heterogeneous, left hemispheric subdural hematoma associated with some subarachnoid bleeding and left hemispheric swelling. Considerable compression of left lateral ventricle, which is not well seen. Severe transfalcial herniation is present, with ventricles displaced well to the right. There is no evidence of anterior cerebral artery distribution infarct.

C and D, CT scans at comparable levels 3 days later show evacuation of subdural hematoma and decompression of brain with return of lateral ventricles to normal position. Infarct is seen in region of left paracentral lobule (arrows). Right frontal extracerebral collection and intraventricular blood have appeared in the interim as well.

#### Case 2

A 91-year-old hypertensive woman was found unconscious. CT scans demonstrated right subdural hematoma, large right cerebral hematoma, and marked right-to-left transfalcial herniation (Figs. 2A and 2B). Despite craniectomy with evacuation of the subdural hematoma and occipital lobectomy, intracranial pressure remained high. CT scans 1½ days later showed persistent transfalcial herniation and diffuse cerebral swelling, with infarcts of the right occipital, parietal, and temporal lobes and thalamus, as well as a right paracentral lobule/cingulate gyrus infarct (Figs. 2C and 2D). There was no clinical improvement, and the patient died 3½ weeks after admission.

#### Case 3

A formerly healthy 54-year-old man had sudden loss of consciousness from a large, spontaneous right basal ganglionic hemorrhage associated with intraventricular bleeding, hydrocephalus, and transfalcial herniation (Figs. 3A and 3B). The hematoma was emergently evacuated, along with the arteriovenous malformation that caused it. CT scans 2 days later revealed much less mass effect, the ventricles having returned to a nearly normal position. Infarcts of the superior frontal gyrus, cingulate gyrus, and paracentral lobule were demonstrated (Figs. 3C and 3D). Over the ensuing 3½ months, the patient remained hemiparetic and minimally responsive.

#### Discussion

In each of these cases there was an abrupt onset of increased intracranial pressure. Hematoma (intraaxial and/or

extraaxial) and brain swelling occurred over a wide area of one hemisphere, forcing it against and under the relatively unyielding falx. With herniation of the ipsilateral cingulate gyrus beneath the falx, anterior cerebral artery branches were presumably compressed. Enough compromise of blood flow ensued to cause frank infarction in the distribution of these branches.

On CT, these infarcts appeared along the medial aspect of the hemisphere in the region of the paracentral lobule or superior frontal gyrus and adjacent cingulate gyrus [6] (Figs. 1–3). These areas correlate with distributions of the supply of the paracentral or posterior internal frontal arteries [7].

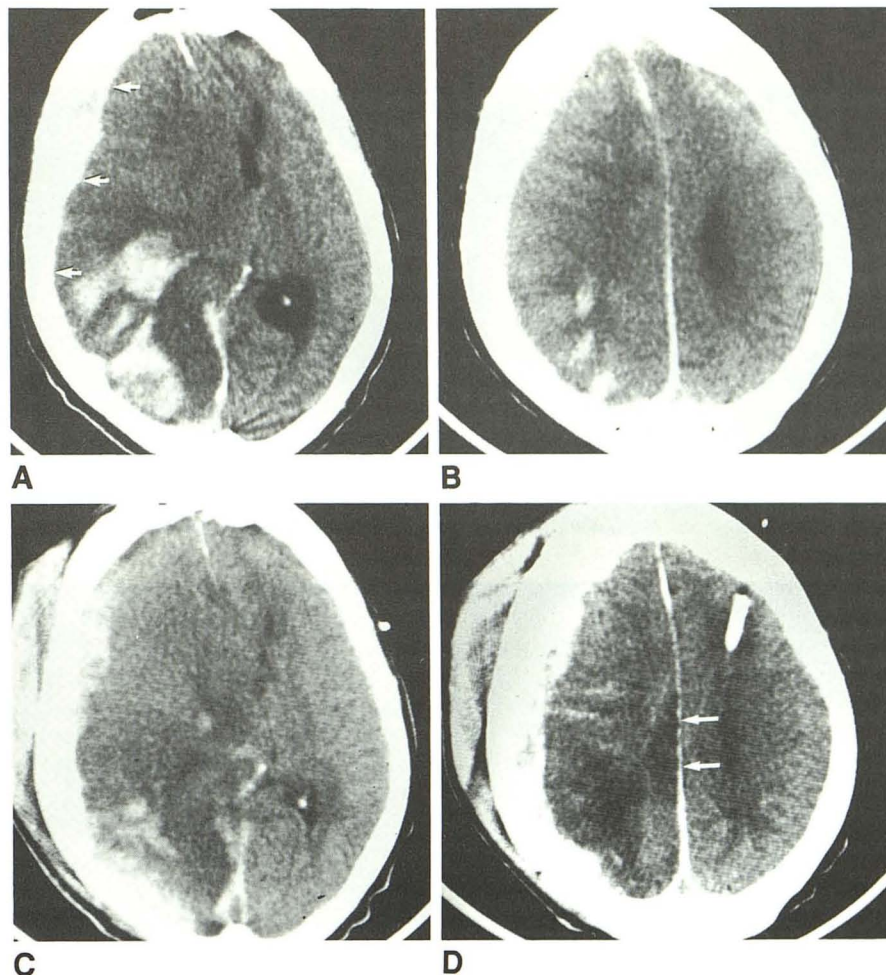
The posterior internal frontal artery supplies the posterior third of the medial surface of the superior frontal gyrus and the subjacent cingulate gyrus, while the paracentral artery supplies the paracentral lobule and subjacent cingulate gyrus [8]. Most commonly, these arteries arise as terminal divisions of the callosomarginal artery [8, 9]. It would seem from our cases that by virtue of its relationship to the falx, the (ipsilateral) callosomarginal artery may be the most susceptible of the anterior cerebral branches to compressive effects of transfalcial herniation [Fig. 4].

The falx, a fairly rigid dural curtain, is narrow in its anterior position and widens as it spreads posteriorly. As several authors have emphasized, there is considerable variability in the width of the falx, so there is considerable variability in the relationship of the falx to the corpus callosum, cingulate gyrus, and anterior cerebral artery branches [10, 11]. However, the



Fig. 2.—A and B, Admission CT scans show right hemispheric subdural hematoma (arrows) and large right posterior temporooccipitalparietal hematoma. Lateral ventricles are compressed and markedly shifted to left side under falx.

C and D, CT scans 1½ days later show some residual hematoma and persistent right cerebral swelling, despite craniectomy and hematoma evacuations. There are recent infarcts of right occipital and temporal lobes and right thalamus. A focal infarct involves right paracentral lobule (arrows) and subjacent cingulate gyrus (not shown).



greatest variance of depth is present in its anterior, or precallosal segment, with less variability recognized in the more posterior supra- and retrocallosal segments [10]. The corpus callosum and cingulate gyrus with their associated arteries converge toward the free edge of the falx posteriorly and intersect it at a (variable) point a few centimeters anterior to the splenium.

With transfalcine herniation, the posterior cingulate gyrus becomes trapped against the falx, while the anterior portion is more free to swing under it. The midportion, in a region of transition, must take a more abrupt course change to squeeze under the falx. The terminal callosomarginal artery above the gyrus is forced against the falx (Fig. 4). If the artery is compressed enough, blood flow is reduced and infarction results.

We believe the rapidity and extent of increased intracranial pressure were highly influential in the development of the infarcts described in these cases. Precipitous herniation occurred from large hematomas, resulting in abrupt compression of the callosomarginal artery. There was, therefore, no chance for collaterals to supply ischemic regions. The fact that we found no similar infarcts in other cases of transfalcine herniation from tumor, abscess, or middle cerebral infarct would support this contention. In fact, previously described cases occurred in acute trauma, massive infarction, or massive hemorrhage [3–5].

In all of our cases the focal infarct was separate and distinct from the primary lesion; in no case was it the primary lesion.

Thus, the infarct seemed to represent an aggravating insult to the brain that contributed to, but did not primarily cause, a worsening clinical course.

No angiograms were attempted on our patients; thus, we cannot totally exclude the possibility of intracranial or extracranial arteriopathy as a cause of these infarcts. Carotid artery injury and intracranial dissecting aneurysm are known causes of ischemic sequelae to trauma; however, symptomatology is frequently delayed [12–14]. The lack of hemorrhage makes it doubtful that the infarcts were venous in origin. Pressure necrosis could produce an appearance similar to infarction, but would not be expected to correspond to a clear-cut vascular distribution.

In summary, although uncommon, callosomarginal infarcts are reflective of severe, acute hemispheric insult. As such, they seem to portend a declining clinical condition, and thus may provide another CT indication of a poor clinical outcome.

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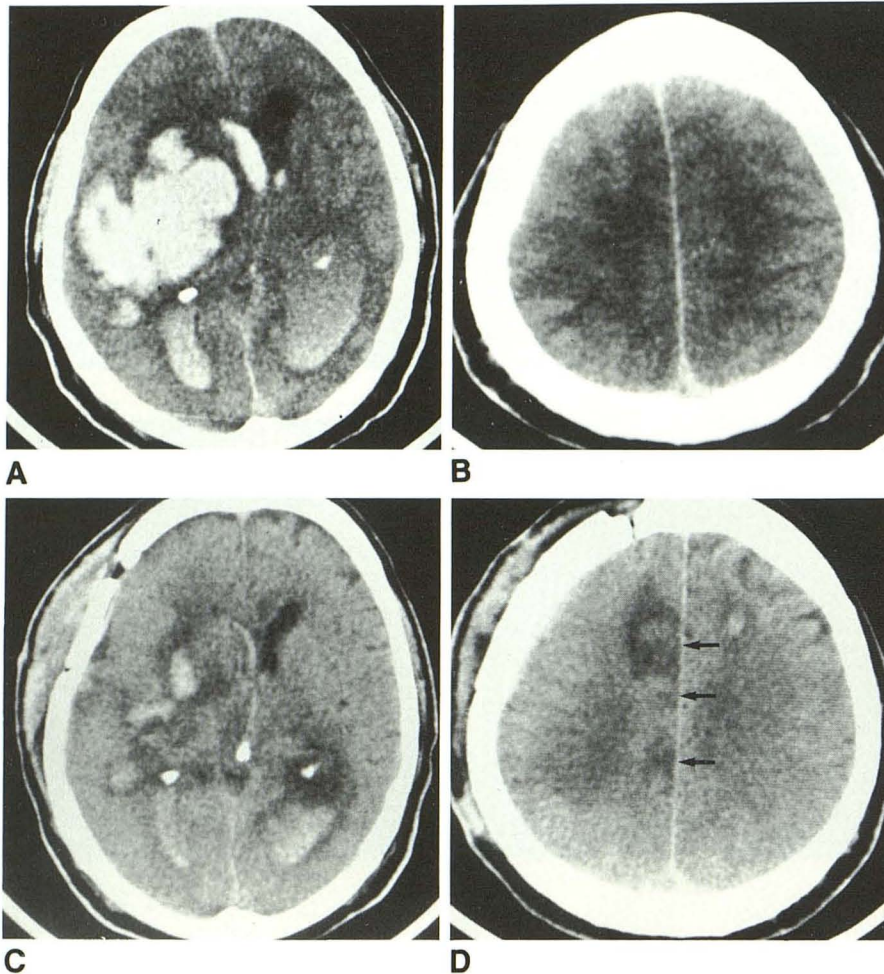


Fig. 3.—A and B, Admission CT scans show large right basal ganglionic-opercular hematoma. Blood is seen within lateral ventricles, which are displaced across midline beneath falx. Supraventricular sections showed no abnormality.

C and D, 2 days later there is evidence of craniectomy and evacuation of hematoma. Less mass effect is present and lateral ventricles are near normal position. Focal infarcts are seen along medial aspect of right hemisphere in region of superior frontal gyrus and paracentral lobule (arrows).

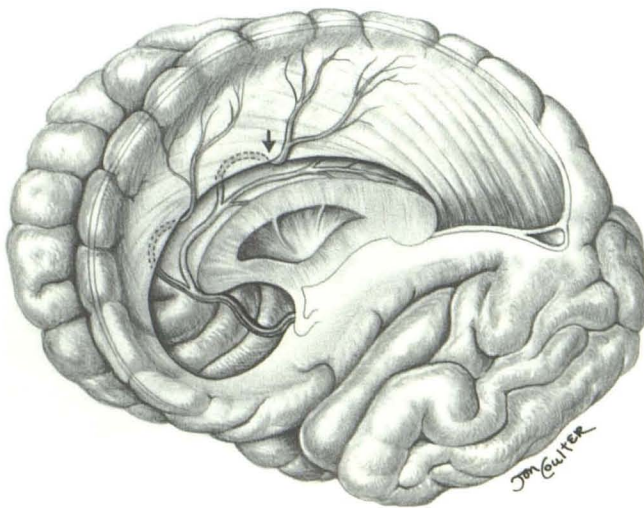


Fig. 4.—Schematic showing left cerebral mass displacing left anterior cerebral artery and its branches under the falx. Callosomarginal artery is kinked against free margin of falx (arrow).

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