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Falx and Interhemispheric Fissure on Axial CT: II.

Recognition and Differentiation of Interhemispheric Subarachnoid and Subdural Hemorrhage

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Interhemispheric hyperdensity or unenhanced computed tomography was originally considered a sign of subarachnoid hemorrhage, the "falx sign." It has since been identified as a normal feature and has also been seen with interhemispheric subdural hemorrhage. To determine the differential features of interhemispheric hemorrhage, 50 patients with subarachnoid hemorrhage and 32 patients with interhemispheric subdural hematomas were reviewed. Subarachnoid hemorrhage produced anterior interhemispheric hyperdensity only, with a zigzag contour and extension from the calvarium to the rostrum of the corpus callosum. Interhemispheric subdural hematomas produce unilateral crescentic hyperdensities that are largest in the posterior superior part of the fissure, behind and above the splenium of the corpus callosum. Interhemispheric hyperdensity in children is more complex. Because the anterior part of the fissure is narrow in younger patients, subarachnoid hemorrhage may go undetected. Likewise, interhemispheric subdural hematomas in children are smaller and more difficult to recognize. They produce asymmetric thickening of the falx shadow with extension over the tentorium. They are, however, of great significance since they are generally seen in abused patients and carry a poor prognosis.

Interhemispheric hyperdensity on unenhanced computed tomography (CT) was originally described as a sign of subarachnoid hemorrhage [1-4] (the "falx sign") [4], but other causes of interhemispheric hyperdensity have since been identified, including the normal falx [5-7] and interhemispheric subdural hemorrhage [8]. We have also noted transient interhemispheric hyperdensity in children with cerebral edema. To determine the differential CT features of each of these conditions, 50 patients with subarachnoid hemorrhage, 32 patients with interhemispheric subdural hematomas, and three children with cerebral edema were evaluated. On the basis of this material, criteria have been developed for the differentiation of these processes from the normal falx.

Materials and Methods

Subarachnoid Hemorrhage

CT scans of 50 patients with subarachnoid hemorrhage were retrospectively evaluated. Criteria for selection were: (1) initial scan within 3 days of ictus; (2) spinal tap indicative of hemorrhage; and (3) CT evidence of blood within subarachnoid spaces (other than the interhemispheric fissure). In addition, at least one of the following criteria was met: (1) angiographic evidence of a source of hemorrhage (e.g., aneurysm or arteriovenous malformation [AVM]) or (2) surgical and/or postmortem confirmation of hemorrhage. There were 23 males and 27 females, varying from 3 months to 81 years of age. Serial scans were obtained in 24 patients. The various causes of hemorrhage are listed in table 1. Findings in this group were compared with those in 200 normal patients (see part 1 of this article [5]).

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TABLE 1: CT Recognition of Interhemispheric Hemorrhage Relative to Cause of Bleeding

Etiology of Hemorrhage	No. (%)			
	Definite	Equivocal	Negative	Totals
Aneurysms:				
Anterior cerebral-pericallosal	11	0	1	12
Internal carotid (postcommunicating/anterior choroidal)	6	2	2	10
Middle cerebral trifurcation	0	0	1	1
Vertebrobasilar	0	0	2	2
Subtotals	17	2	6	25
Trauma:				
Adult, blunt	4	2	2	8
Child, blunt	2	3	3	8
Penetrating	0	2	1	3
Subtotals	6	7	6	19
Arteriovenous malformation	0	1	4	5
Agonal hemorrhage	1	0	0	1
Totals	24 (48)	10 (20)	16 (32)	50

Edema

Three patients, all under 5 years of age, with acute cerebral edema had a CT pattern suggestive of interhemispheric subarachnoid hemorrhage. One child had pathologically proven subacute sclerosing panencephalitis with an acute exacerbation. Two children had edema secondary to prolonged hypoxia. In each case, subarachnoid hemorrhage was excluded on the basis of clinical, laboratory (spinal tap negative for hemorrhage), and/or pathologic findings despite CT signs suggestive of hemorrhage.

Interhemispheric Subdural Hematoma

Thirty-two patients with interhemispheric subdural hematomas were evaluated. Surgical and/or pathologic confirmation was available in 11 of 25 adults and three of seven children. Angiographic confirmation was available in three other cases.

Results

Subarachnoid Hemorrhage

Interhemispheric hyperdensity was present in all patients, but since it was also seen in 90% of normal patients [5], it alone was not a reliable sign of subarachnoid hemorrhage. The mean density of the interhemispheric fissure was 61 Hounsfield units (H) in the hemorrhage group as compared with 46 H in the normal group [5]. There was, however, considerable individual variation and significant overlap of mean and peak densities. Therefore, although a mean interhemispheric density of greater than 65 H is suggestive of hemorrhage, the measurement of interhemispheric density alone does not reliably differentiate subarachnoid hemorrhage from the normal falx.

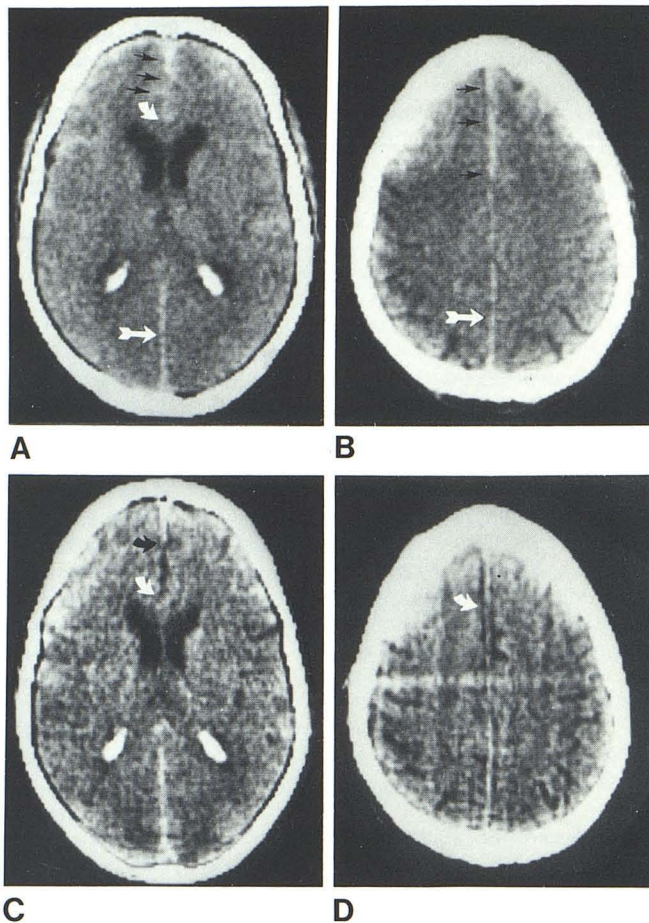
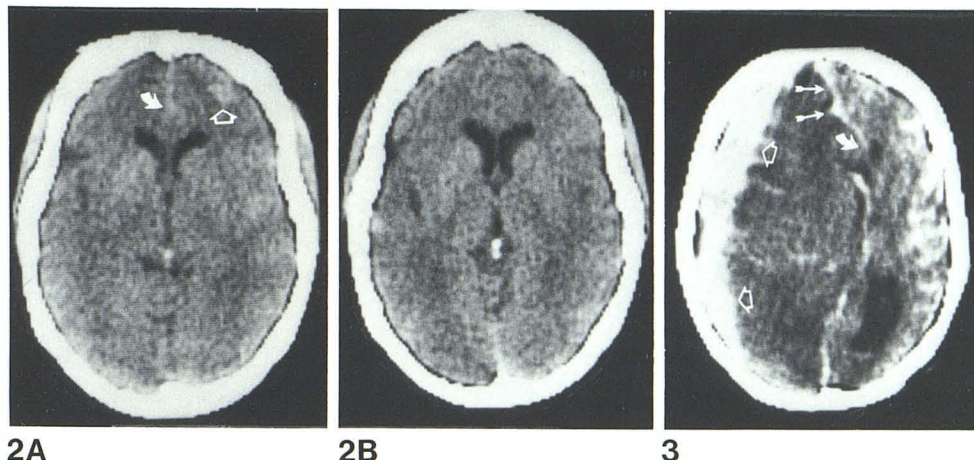


Fig. 1.—Anterior interhemispheric subarachnoid hemorrhage. A and B, 36 hr after rupture. A, Anterior interhemispheric hyperdensity (black arrows) with zigzag configuration extending from calvarium to genu of corpus callosum (curved white arrow). Posterior interhemispheric hyperdensity (straight white arrow) has configuration of normal posterior falx. B, Supracallosal cut. Anterior hyperdensity (black arrows) projects into medial sulci and represents subarachnoid hemorrhage. Posteriorly (white arrow), line is thin and straight and represents dense falx. C and D, 2 weeks later. Resolution of anterior interhemispheric hyperdensity. C, Thin, straight line (black arrow) represents normal falx in anterior one-half of this part of fissure with CSF density extending to genu of corpus callosum (white arrow). Fissure has zigzag configuration. Retrocallosal interhemispheric density is unchanged. D, Supracallosal cut. Anterior part of falx (arrow) is surrounded by CSF density. Posteriorly, there is no change when compared with initial study.

The recognition of subarachnoid hemorrhage is dependent on distinguishing the interhemispheric hyperdensity produced by opacification of the fissure by hemorrhage from that seen with the normal hyperdense falx. This differentiation requires separate evaluation of each segment of the falx/fissure combination (precallosal, retrocallosal, and supracallosal segments). In the precallosal segment of the interhemispheric fissure, hemorrhage produces hyperdensity with the configuration of the fissure rather than that of the falx. Thus, the linear hyperdensity was broader than the falx, had a zigzag outline, and extended from the calvarium to the genu of the corpus callosum (figs. 1A and 1B). On follow-up examination, resolution of the interhemispheric hyperdensity confirmed that it was due to hemorrhage as the precallosal falx became visible in the anterior part of the

Fig. 2.—Anterior subarachnoid hemorrhage. **A**, 1 day after rupture of AVM. Anterior interhemispheric hyperdensity begins at calvarium and approaches corpus callosum (solid arrow). Because fissure is narrow, it is difficult to differentiate hyperdensity of hemorrhage from anterior falx. Parenchymal hematoma at site of AVM (open arrow). **B**, 2 weeks later. Resolution of interhemispheric hyperdensity confirming subarachnoid hemorrhage.

Fig. 3.—Posttraumatic subarachnoid hemorrhage. CT scan several hours after trauma. Massive left acute subdural hematoma (open arrows). Anterior interhemispheric subarachnoid hemorrhage (straight solid arrows) extends posteriorly reaching corpus callosum (curved arrow). Fissure is distorted by mass effect.



interhemispheric fissure as a thin straight line extending toward but not reaching the genu of the corpus callosum (figs. 1C and 1D). Anterior interhemispheric subarachnoid hemorrhages were identified in 21 (42%) of the 50 cases (table 2). In eight other cases, anterior interhemispheric hyperdensity was seen, but the differentiation between a normal deep falx and subarachnoid hemorrhage could not be reliably made (fig. 2). In the other 21 cases, interhemispheric hyperdensity was either absent or attributable to that produced by the normal falx alone.

Subarachnoid hemorrhage did not alter the appearance of the posterior falx/fissure combination. In both the hemorrhage and normal groups, a thin hyperdense line extended from the calvarium to the splenium of the corpus callosum and no cerebrospinal fluid (CSF) density collection was identified (figs. 1–3). No change was seen on serial examination and, thus, we concluded that the hyperdensity represents the normal falx. To substantiate this opinion, the posterior falx/fissure combination was evaluated in five patients after the intrathecal injection of metrizamide for evaluation of the suprasellar cistern spaces. In these five cases, there was complete opacification of all visualized subarachnoid spaces, both infra- and supratentorially. The appearance of the posterior falx/fissure combination in these patients did not change, suggesting again the small size of the subarachnoid space in this region.

Superior interhemispheric subarachnoid hemorrhages were noted in 14 (28%) of the 50 patients. Anterior and superior to the corpus callosum, the findings were identical to those observed in the contiguous precallosal interhemispheric fissure. More posteriorly, the superior interhemispheric hyperdensity had the configuration of the normal falx (fig. 1B). Subarachnoid hemorrhage in the superior part of the fissure was identified as an isolated finding in only two patients (both with head trauma).

The overall incidence of identifiable interhemispheric subarachnoid hemorrhage was 48% (24 of 50 patients). In another 10 patients, the configuration and density of the anterior part of the interhemispheric fissure was suggestive but not definitively diagnostic of subarachnoid hemorrhage. The recognition of interhemispheric subarachnoid hemor-

TABLE 2: CT Recognition of Subarachnoid Hemorrhage Relative to Location of Bleeding

Location	No. (%)		
	Definite	Equivocal	Negative
Anterior	21	8	21
Posterior	1	0	49
Superior	14	4	32
All sites	24 (48)	10 (20)	16 (32)

rhage was influenced by patient age. Seven of the 10 patients in whom the diagnosis was equivocal were under 30 years of age. In younger patients, the precallosal segment of the fissure is narrow and, therefore, hemorrhage was either not seen or produced interhemispheric hyperdensity with a configuration indistinguishable from that of a normal deep falx. The other major factor that determined the visibility of interhemispheric hemorrhage was the proximity of the site of the hemorrhage to the fissure. Thus, ruptured aneurysms most often produced interhemispheric subarachnoid hemorrhage (17 cases, 68%), especially (11 of 12 cases) with aneurysms of the anterior cerebral or pericallosal arteries. Interhemispheric subarachnoid hemorrhage was identified in six (32%) patients with trauma, but it was only seen equivocally in one of five patients with arteriovenous malformations (table 1).

Diffuse Edema Mimicking Subarachnoid Hemorrhage

Three patients under 5 years of age had CT scans demonstrating diffuse cerebral edema and interhemispheric hyperdensity mimicking subarachnoid hemorrhage. Two were studied within 24 hr of an hypoxic episode and the third had an acute exacerbation of histologically proven subacute sclerosing panencephalitis (fig. 4). In each case, there was marked ventricular compression, decreased attenuation of the cerebral hemispheres (frontal lobe mean density of 26 H), and a loss of normal demarcation between cortical gray and white matter. In each case, interhemispheric hyperdensity with a configuration typical of subarachnoid hemorrhage, but with a mean density of 40 H, was identified. Two patients had serial examinations, and, in both, the

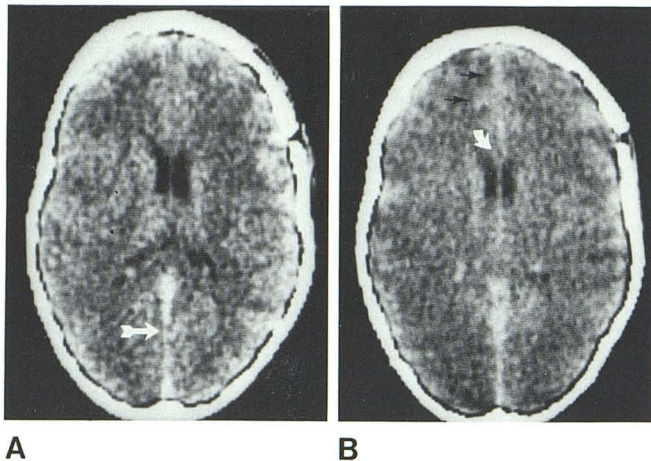


Fig. 4.—Acute edema mimicking interhemispheric subarachnoid hemorrhage in 13-year-old child with biopsy-proven subacute sclerosing panencephalitis. **A**, During acute deterioration. Diffuse white matter hypodensity. Ventricles not compressed. Posterior interhemispheric hyperdensity of normal falx (arrow). No hyperdensity in anterior interhemispheric fissure. **B**, Nonenhanced scan 11 days later. Marked progression of edema in gray and white matter. Posterior interhemispheric hyperdensity unchanged from previous study. Anterior interhemispheric hyperdensity (black arrows) extending from calvarium to genu of corpus callosum (white arrow) mimics subarachnoid hemorrhage. Postmortem examination 24 hr later showed only severe edema.

interhemispheric hyperdensity was transiently seen, another finding suggestive of hemorrhage. The hyperdensity was present when the edema was at its maximum and either appeared as the edema progressed or disappeared as the edema regressed (fig. 4). Postmortem examination within 24 hr of CT scans in two patients demonstrated no evidence of hemorrhage. Severe edema was present with sparing of the parasagittal cortical gray matter.

Interhemispheric Subdural Hematoma

Interhemispheric subdural hematomas were identified in 25 adult patients (figs. 5–7). These collections were characteristically parasagittal and had a crescentic configuration with a flat medial border and a convex lateral border. This configuration reflected the predilection of these collections to occur unilaterally within the fissure (22 cases, 88%). In two of the three patients with bilateral interhemispheric subdural hematomas, the lesions were the result of penetrating trauma with rupture of the falx. Interhemispheric subdural hematomas always involved and were largest within the posterior superior part of the fissure behind and above the splenium of the corpus callosum (figs. 5–7). Inferior extension over the medial superior margin of the

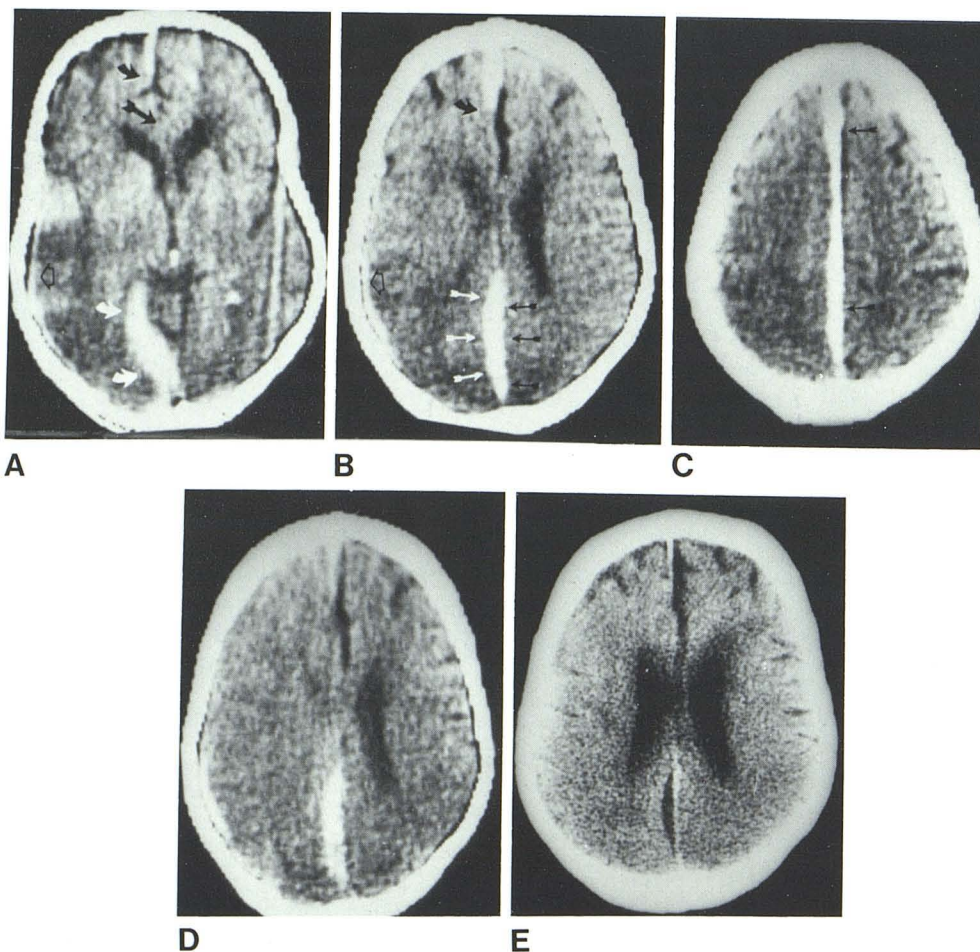


Fig. 5.—Interhemispheric subdural hematoma. **A–C**, Initial study. Posterior interhemispheric hematoma with flat medial border (straight solid black arrows, **B** and **C**). Lesion bulges laterally, compressing ipsilateral parasagittal cortex (white arrows, **A**), and extends anteriorly (curved black arrows, **A** and **B**). Gap between subdural hematoma and corpus callosum (straight solid black arrow, **A**). Above corpus callosum (**C**), hematoma produces continuous interhemispheric hyperdensity. Small convexity subdural hematoma is also seen (open arrows, **A** and **B**). **D**, 10 days later. Anterior part nearly resolved and posterior part is smaller and less dense. Thin cleavage plane is now visible between medial aspect of subdural hematoma and hyperdense falx. **E**, After 1 month. Small hypodense posterior interhemispheric subdural hematoma resolved.

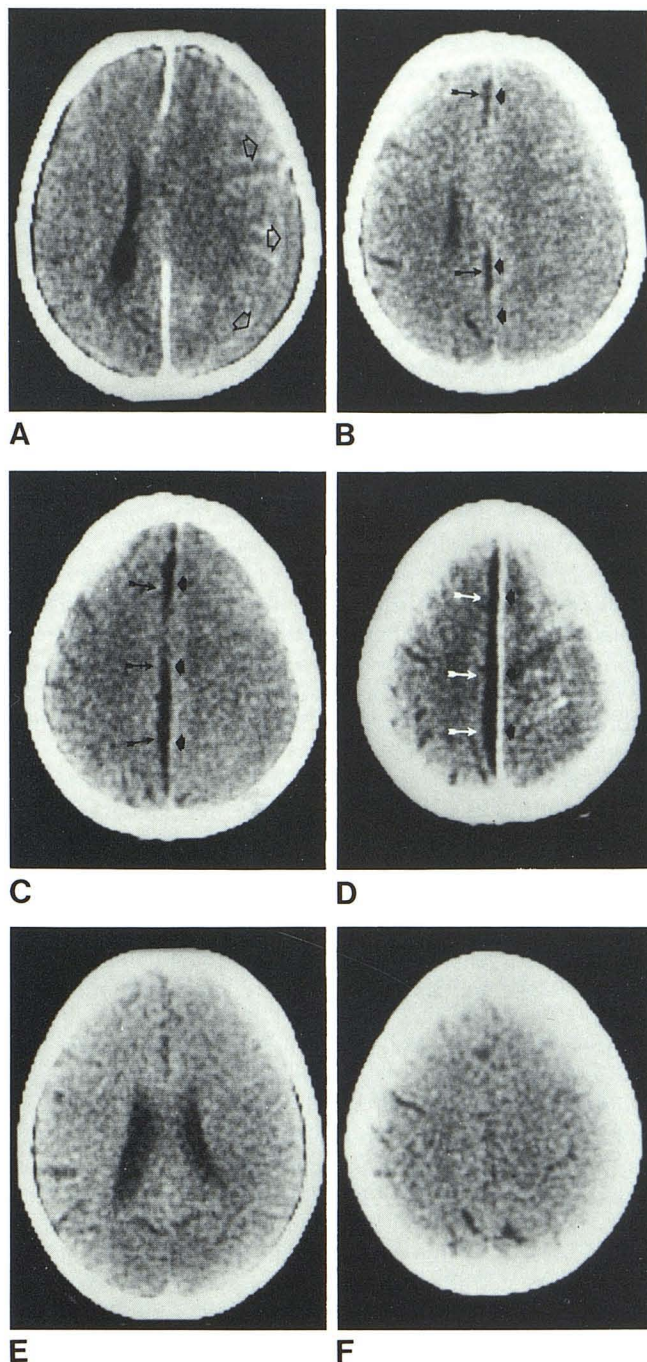


Fig. 6.—Chronic interhemispheric subdural hematoma. **A**, Initial scan. Isodense convexity hematoma (arrows). No interhemispheric collections. **B–D**, 10 days later. Crescentic hypodense contralateral interhemispheric subdural collect (long arrows) with flat medial border (short arrows). Sulci adjacent to collection are compressed rather than dilated as they would be in focal atrophy. **E** and **F**, 2 months later. Resolution of both convexity and interhemispheric subdural hematomas. Lack of interhemispheric hypodensity confirms previous hematoma, not atrophic dilatation of fissure.

tentorium was frequently observed (18 cases, 72%). Anterior extension was less common (11 cases, 44%). When present, the anteriorly situated interhemispheric subdural hematoma occupied that part of the fissure containing the falx and, thus, never extended completely from the calvar-

Fig. 7.—Interhemispheric subdural hematoma with layering. Typical curved lateral contour. Slight convexity to medial border due to large size of hematoma and displacement of falx. Fluid-fluid level (arrow) between hypodense antedependent and hyperdense dependent parts of hematoma.



ium to the genu of the corpus callosum (fig. 5A). Associated convexity subdural hematomas were identified in 23 cases. The ipsilateral convexity was involved in 11 (44%) cases, both convexities in two (8%) cases, and the contralateral convexity in six (24%) cases. Contusions and subdural hematomas were also seen in four cases and contusions alone in one case. Serial examination in 13 patients demonstrated that there was a progressive decrease in the density of the hematomas that occurred most rapidly at the periphery of the lesion (fig. 5D). The extent and width of the hematoma also decreased with time. Total resolution without residua was seen in 12 of 13 patients, including eight patients without surgical intervention. When initially seen, 15 (60%) of the hematomas were hyperdense (fig. 5), four (16%) showed dependent layering of the denser elements and a fluid-fluid level (hematocrit effect) (fig. 7). Six (24%) of the hematomas were hypodense (fig. 6). These hypodense collections retained the characteristic configuration and location of interhemispheric hematomas. Focal atrophic dilatation of the interhemispheric subarachnoid space was excluded in these cases, since the retrocallosal and posterior superior callosal interhemispheric fissure rarely underwent atrophic dilatation [5] and the collections had the characteristic configuration of interhemispheric subdural hematomas. No isodense interhemispheric subdural hematomas were identified, despite careful evaluation for direct and indirect (mass or displacement) CT findings.

Evaluation of seven children with interhemispheric subdural hematomas demonstrated the same location and configuration as that seen in the adult population. There were, however, major differences from the adult patients. In the children, the hematomas were smaller, presenting as subtle areas of asymmetric thickening of the posterior falx shadow (fig. 8). Differentiation between the normal posterior falx and a small interhemispheric hematoma was facilitated by evidence of inferior extension of the hematoma over the tentorium (five of seven cases) (fig. 8A). The linear unilateral hyperdensity along the course of the tentorium produced by this extension is not seen under normal circumstances [5], and is, therefore, more unequivocally pathologic than simple thickening of the posterior falx shadow. Although associated lesions were present in six cases, these lesions also differed

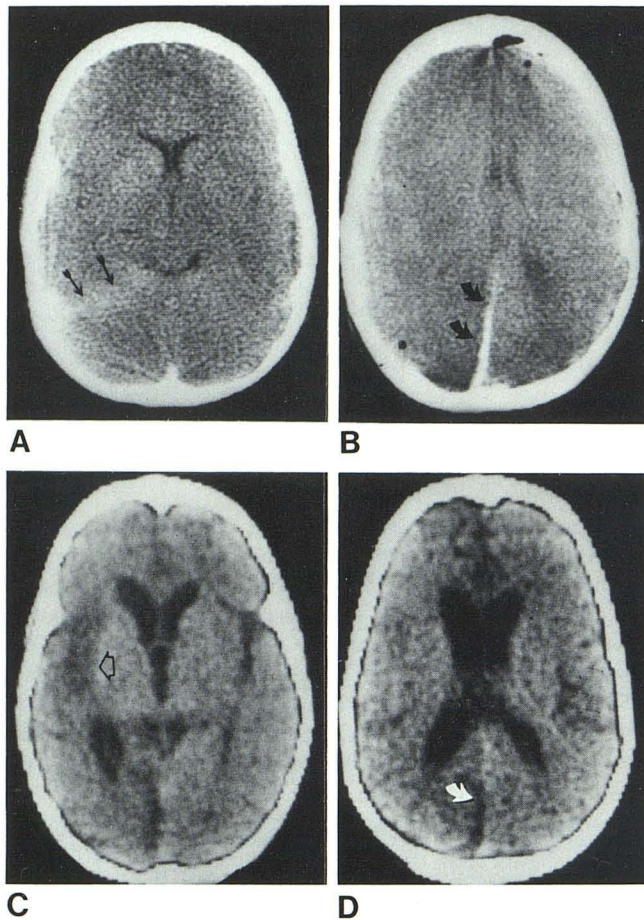


Fig. 8.—Interhemispheric subdural hematoma in abused child. **A** and **B**, Initial study. Subtle hyperdensity (arrows) along left superior tentorial surface (**A**) and left side of falx (**B**). Poorly defined mass effect due to large area of nonhemorrhagic contusion. **C** and **D**, Hydrocephalus has developed and there is posttraumatic encephalomalacia in temporal lobe (open arrow). Hypodense posterior interhemispheric subdural hematoma (white arrow). The child was severely demented and had a fixed neurologic deficit.

from those seen in adult cases. In five cases, regions of parenchymal damage of varying size and density were identified. These contusions were nonhemorrhagic and produced poorly defined areas of hypodensity, and, therefore, the extent of the parenchymal damage was often difficult to determine on the initial CT, and typically the only definite abnormality visualized was a small interhemispheric subdural hematoma. Even though the initial study showed only subtle abnormalities, serial scans over several months demonstrated significant posttraumatic residua in five patients, including hydrocephalus, focal or diffuse posttraumatic encephalomalacia, and persistent unilateral interhemispheric hypodense collections (four cases) (figs. 8C and 8D). Complete resolution of parenchymal and interhemispheric abnormalities was identified in only two patients.

Discussion

Hyperdensity in the interhemispheric fissure may have several causes, including: (1) the normal falx [5–7]; (2) dural calcification [9]; (3) subarachnoid hemorrhage [1–4]; (4)

interhemispheric subdural hematoma [8]; and (5) diffuse cerebral edema. Differentiation of these processes is possible if one considers the falx and fissure as a single unit divided into three segments by their relationship to the corpus callosum. Each process produces a specific pattern of density with a characteristic configuration and location within the fissure.

In the anterior (precallosal) part of the fissure, hyperdensity may be due to the normal falx, subarachnoid hemorrhage, or subdural hemorrhage. As previously described [5], the normal falx is seen as a thin, straight, dense line extending posteriorly from the calvarium for a variable distance. It does not reach the rostrum of the corpus callosum. In subarachnoid hemorrhage, there is opacification of the cisternal spaces by unclotted blood, which produces a dense cast of the fissure. Thus, the interhemispheric hyperdensity has the configuration of the fissure, and extends from the calvarium to the genu of the corpus callosum with a zigzag contour due to extension of the hemorrhage into the medial sulci of the brain. In younger patients, the fissure is narrow, and, therefore, hemorrhage produces a thin, hyperdense line that is relatively straight and may be difficult to differentiate from a normal deep falx. In older patients (over 40 years of age), the fissure is wider [5] and, therefore, hemorrhage into the fissure is more easily identified. Identification of interhemispheric subarachnoid hemorrhage may be more difficult in the presence of significant mass effect, which distorts the anatomy of the fissure. Another factor that influences the recognition of interhemispheric subarachnoid hemorrhage (table 2) is the proximity of the origin of the hemorrhage to the fissure. Because of all of these factors, interhemispheric subarachnoid hemorrhage was most frequently seen after ruptured aneurysms (68%), was less frequently seen after trauma (32%), and was not definitely seen after ruptured AVMs.

Anterior interhemispheric subdural hemorrhage was not observed as an isolated finding in our series. It was always secondary to extension of a large posterior superior interhemispheric subdural hematoma. When present, it was seen as a unilateral crescentic collection with its medial border formed by the falx. Since the subdural space extends into the fissure with the falx, interhemispheric subdural hematomas involve only that part of the fissure where the falx is present, and, therefore, the hematoma never extends from the calvarium to the genu of the corpus callosum [5].

The differential diagnosis of posterior interhemispheric hyperdensity is limited to the normal falx and interhemispheric subdural hematoma, since subarachnoid hemorrhage does not alter the appearance of the posterior falx/fissure combination. In normal patients, the falx is seen as a thin high hyperdense line extending completely from the calvarium to the corpus callosum [5]. The falx occupies virtually the entire posterior fissure. The subarachnoid space around it is narrow and virtually invisible. The fissure does not dilate with age because the medial parietal cortex does not undergo significant atrophy. Thus, with subarachnoid hemorrhage, the volume of blood that can be present in this part of the fissure is too small to alter the appearance or density of the falx/fissure combination. A similar phenomenon can be observed after the intrathecal injection of

metrizamide. Despite the density of the contrast material flooding the subarachnoid space, there is no change to the appearance of the posterior falx/fissure combination.

Interhemispheric subdural hematomas have highly characteristic features, determined by the specific pathophysiologic formation of these collections and local anatomic features. They are produced by torsion forces (whiplash injuries) and are secondary to rupture of large and relatively fixed bridging veins between the parietooccipital cortex and the superior sagittal sinus [10, 11]. Once rupture has occurred, hemorrhage is confined to the interhemispheric subdural space, which does not freely communicate with other parts of the subdural space. We believe this is due to the pacchionian granulations preventing free communication between the interhemispheric and ipsilateral convexity subdural space [10]. We also believe that there is no communication between the right and left interhemispheric subdural spaces. Thus, the lesions are most often seen and are largest at their site of origin in the posterior superior part of the fissure behind and above the splenium of the corpus callosum. They cannot extend into the contralateral interhemispheric fissure and, therefore, have a flat medial border formed by the falx. The pacchionian granulations impede their extension into the convexity subdural space and, therefore, they bulge into the adjacent ipsilateral hemisphere. (The convexity subdural hematomas that are usually observed in these patients arise from separate bleeding sources. Since the convexity and interhemispheric subdural collections are separate, they undergo independent changes in size and density with time and/or surgical intervention.)

Interhemispheric subdural hematomas frequently extend inferiorly over the tentorial surface producing a band of hyperdensity that conforms to the contour of that structure. Less often, they extend into the more distant anterior interhemispheric fissure conforming to the course and location of the anterior portion of the falx. Interhemispheric subdural hematomas were very difficult to diagnose before the advent of CT and, thus, were believed to be rare [12]. Evaluation of our material would suggest that they are relatively common lesions. Clinical findings may be absent or attributed to the associated convexity collection. The hematomas typically undergo spontaneous resolution and, therefore, in the absence of focal findings may be treated conservatively.

On serial examination, interhemispheric subdural hematomas decrease in density more rapidly at their periphery than their center, appearing to "melt away" as the hematoma ages. Hypodense interhemispheric subdural hematomas can be differentiated from atrophic dilatation of the interhemispheric subarachnoid space since the posterior part of the fissure rarely dilates atrophically. When atrophic dilatation does occur, it is generally bilateral, and parasagittal sulci are prominent. Hypodense interhemispheric subdural hematomas are unilateral and compress the adjacent medial sulci producing a smoothly marginated crescentic configuration. Interhemispheric subdural empyemas may have the same configuration as subdural hematomas, but the empyemas have marked thickening of the lateral medial margins of the lesion, which enhance after injection of contrast material [13].

Evaluation of interhemispheric hyperdensity in children presents several problems. Because of the narrowness of the anterior part of the fissure, subarachnoid hemorrhage may go undetected, or may be impossible to differentiate from the normal falx (fig. 2). Diffuse cerebral edema may mimic subarachnoid hemorrhage because the cerebral hemispheres become hypodense. Those structures spared by the edema (the falx, the pericallosal vessels, and the parasagittal cortex) are rendered relatively hyperdense and have the same configuration as that seen in subarachnoid hemorrhage. As with hemorrhage, the finding is transient, appearing as the edema progresses and disappearing as the edema regresses. Therefore, in the presence of severe edema (especially in head trauma), interhemispheric hyperdensity is not a reliable sign of subarachnoid hemorrhage.

In children, posterior interhemispheric hyperdensity has the same causes as those seen in the adult population. Differentiation between the normal falx and interhemispheric subdural hematoma is, however, more complicated because the hematomas are generally smaller and more difficult to visualize. They usually produce only subtle asymmetric thickening of the falx shadow, and, therefore, identification of inferior extension over one tentorial surface; a more clearly pathologic finding is often the key to confirming the diagnosis. Although the hematomas are smaller in children, their diagnosis is paradoxically of greater significance than in adults. Hematomas are usually produced by trauma and are the most common cranial sequelae of child abuse [14-16]. Thus, the medicolegal implications of visualization of even a small interhemispheric subdural hematoma in a child are obvious. In addition, the lesions occur in association with contusions, rather than convexity subdural hematomas, and the nonhemorrhagic contused brain may be difficult to visualize on the initial CT scan. Thus, a small interhemispheric subdural hematoma may be the only obvious abnormality on the initial CT scan in a patient whose serial scans reveal the development of severe cerebral damage and large residual interhemispheric collections. The prognosis in children with interhemispheric subdural hematomas is poor [16] and permanent neurologic impairment is usually seen. This poor prognosis is a reflection of the multiple episodes of trauma to which these abused children are subjected and, therefore, the diagnosis of even a small interhemispheric subdural hematoma is extremely important for identifying the abused child and possibly preventing further episodes of cranial trauma.

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