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This information is current as of May 8, 2024.

Dyke award. Influence of fiber tracts on the CT appearance of cerebral edema: anatomic-pathologic correlation.

A R Cowley

Dyke Award

Influence of Fiber Tracts on the CT Appearance of Cerebral Edema: Anatomic-Pathologic Correlation

A. Ronald Cowley[1]

Four monkeys received cold-induced lesions provoking cerebral edema. The edema–Evans blue complex was shown in these animals to migrate along known short and long association bundles. Computed tomographic findings in 12 human cases are discussed and correlated; all exhibit unencapsulated fluid (edema, blood, or tumor) to track along the association pathways. Major fiber bundles are separated into those that permit the spread of edema and those that seem to be resistant. The radiologic and diagnostic significance of the association bundles is discussed and their connections are reviewed.

The advent of computed tomography (CT) has allowed the various neuroscientific disciplines an unparalleled view of intracranial anatomy and pathology. The remarkable ability of the technique to distinguish minor variations of attenuation coefficients has led, for the first time [1, 2], to direct imaging of cerebral edema and other nonencapsulated fluid masses (blood, fat, and tumor), which could only be inferred by use of previous modalities. There have been numerous reports of the CT appearance of characteristic configurations of various fluid masses such as subdural and epidural hematomas. In addition, Gado et al. [3] have clearly elucidated the anatomic pattern of the various cortical areas in CT terms, yet no current CT accounts of the association bundles can be found in the literature.

In reviewing numerous examples of edema, primarily of the vasogenic type so well seen in meningiomas and metastases, the author has become aware of characteristic patterns of orientation of nonencapsulated fluid masses. It appeared that the deep white matter edema was not random in its configuration, and that its progression through the brain seemed to follow the courses of the short and long association bundles, which have been clearly shown in earlier anatomic literature. Clearly, these bundles may provide structural substrates for the spread of fluid masses between gyri, lobules, and, indeed, even between lobes of the cerebrum. The bundles have often been shown to allow false localization of clinical symptoms [4, 5], that is, frontal lobe system symptoms from temporal lobe lesions, because of interconnections along such bundles as the uncinate fasciculus. Various major, well known bundles have seemed to provide cleavage planes or line of least resistance along which fluid elements can move freely [6–8].

To provide basic evidence for this tenet, an animal model was needed. A nonhuman primate was chosen because it possesses a well developed temporal lobe and because excellent descriptions of the association bundles are readily available. Reproducible vasogenic edema is easily produced with a cold-lesion technique of Klatzo et al. [9]. This study does not attempt to address the mechanism of Evans-blue spread. Clearly visible Evans blue–albumin edema material leaks from the disrupted blood-brain barrier and can be accurately recorded photographically. The animal findings are then correlated with parallel
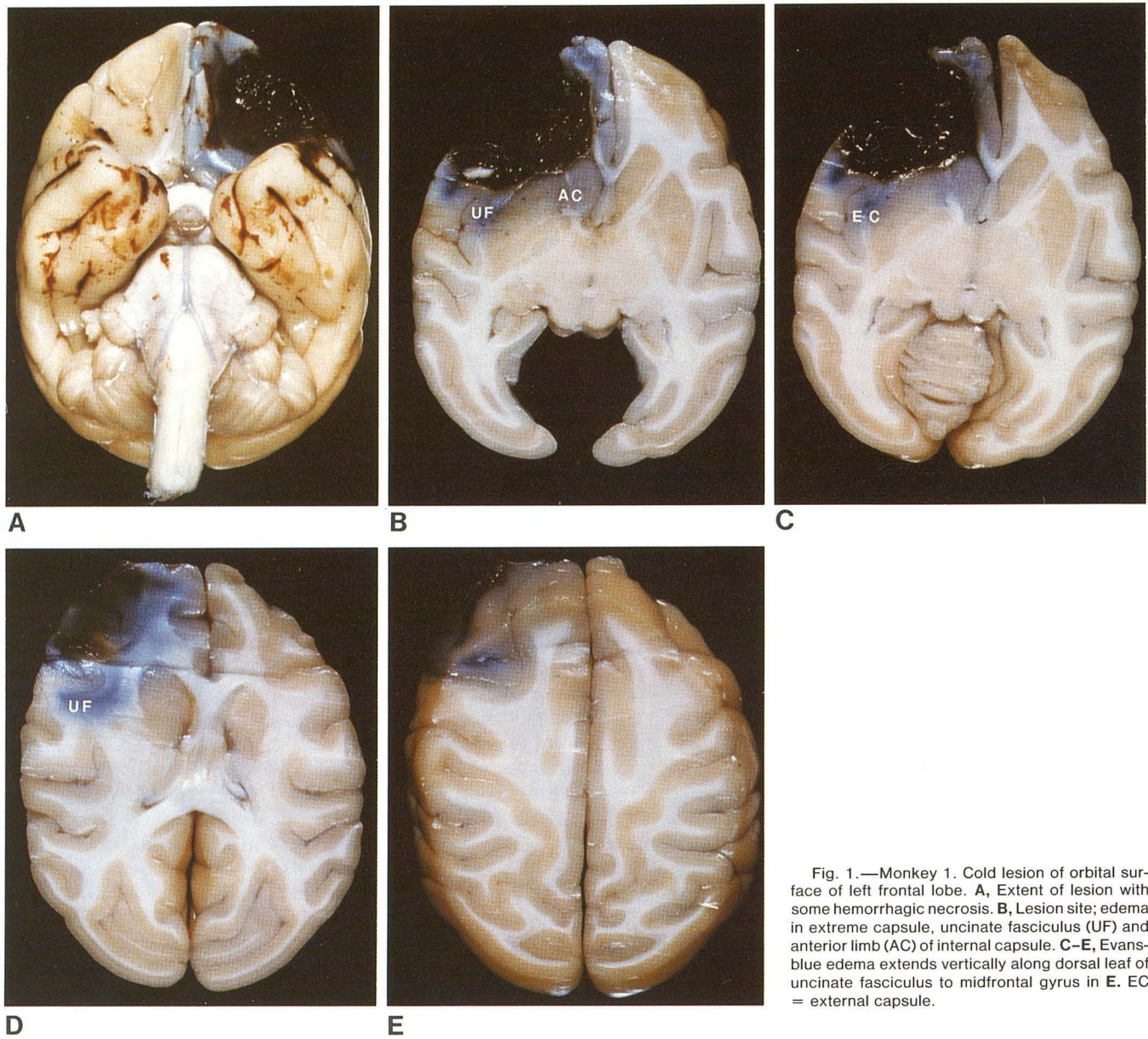
Fig. 1.—Monkey 1. Cold lesion of orbital surface of left frontal lobe. **A,** Extent of lesion with some hemorrhagic necrosis. **B,** Lesion site; edema in extreme capsule, uncinate fasciculus (UF) and anterior limb (AC) of internal capsule. **C–E,** Evans-blue edema extends vertically along dorsal leaf of uncinate fasciculus to midfrontal gyrus in **E.** EC = external capsule.

human cases to effect the description of the major association pathways seen daily in the CT suite.

Recent evidence [1] suggests that the spread of edema occurs in three main directions from the site of the cold-induced lesions: (1) from the lesion to adjacent gyri, (2) into the deep white matter, and (3) toward the ventricles and corpus callosum without ever crossing the corpus itself. For this reason, in three of the animals, separate cold lesions were placed in each hemisphere in an asymmetric pattern. All lesions were made purposely large to evoke as much edema as possible. In so doing some cortical necrosis and staining also undoubtedly developed. The generalized gray-matter staining seen in several of the monkey brain sections is most likely secondary to Evans-blue edema complex pooling with the plethora of small nerve fiber strata in the laminae of the cerebral cortex.

Materials and Methods

Four *Macaca fascicularis* monkeys averaging 3 kg each were used for the study. Under ketamine anesthesia (15 mg/kg) and maintenance with pentobarbital, the animals were placed in a stereotaxic head frame and craniectomies performed with careful preservation of the dura. In all but one animal, bilaterally assymetric lesions were made using a cryo-probe cooled to −50°C ± 2°. The probe was left in place for 1 min and its dural attachment loosened by gentle saline irrigation. One animal only received a single lesion to the exposed orbital surface of the frontal lobe through a cruciate opening in the dura; the dura was subsequently closed. Before lesion production, each animal received Evans blue intravenously at a rate of 2 ml/kg of a 2.5% aqueous solution. The scalp in all animals was closed with silk and the animals were maintained for 48 hr with no untoward effects.

At the time of sacrifice, the animals were immobilized with ketamine and anesthesia maintained with pentobarbital. A midline lap-
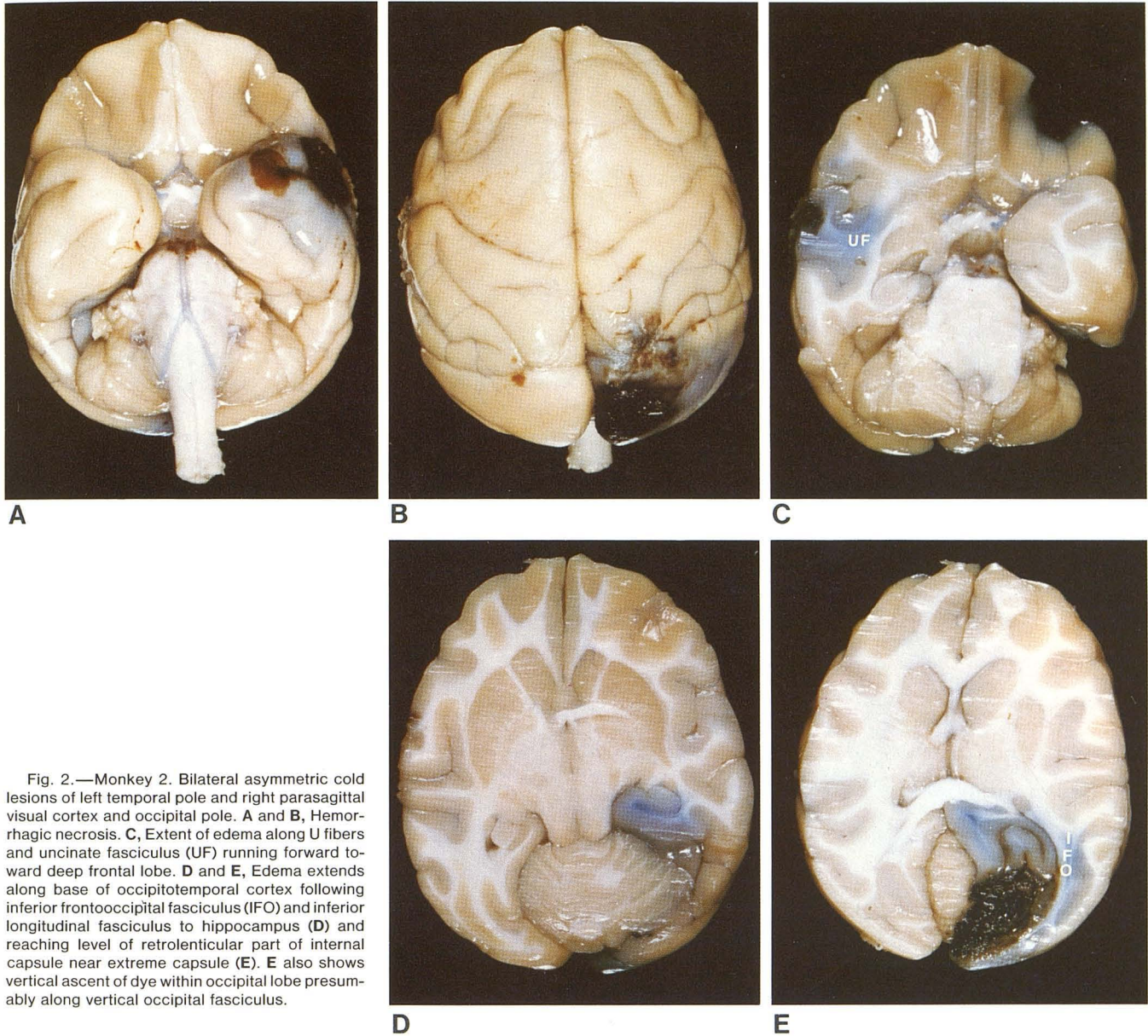
Fig. 2.—Monkey 2. Bilateral asymmetric cold lesions of left temporal pole and right parasagittal visual cortex and occipital pole. **A** and **B,** Hemorrhagic necrosis. **C,** Extent of edema along U fibers and uncinate fasciculus (UF) running forward toward deep frontal lobe. **D** and **E,** Edema extends along base of occipitotemporal cortex following inferior frontooccipital fasciculus (IFO) and inferior longitudinal fasciculus to hippocampus (**D**) and reaching level of retrolenticular part of internal capsule near extreme capsule (**E**). **E** also shows vertical ascent of dye within occipital lobe presumably along vertical occipital fasciculus.

arotomy was performed and the inferior vena cava incised. Animals were killed by exsanguination, and the pericardial cavity opened through an incision in the tendinous part of the diaphragm. The cardiovascular system was initially perfused for 10 min with a 0.1 M phosphate buffer (ph 7.2) through an 18 gauge needle placed in the left ventricle. The entire cardiovascular system was then perfused with 10% neutral buffered formalin at 100 ± 10 mg of mercury. The inferior vena cava was ligated and arterial pressure maintained at 100 mm Hg for 1 hr, at which time fixation was judged to be complete. The animals were decapitated and the dura exposed by scattered burr holes. All brains were removed after 1 week and stored for two more weeks before sectioning and photography. Gross-section brains were photographed with standard macro equipment for the descriptive material. Brains were sectioned by freehand technique to approximate the orbitomeatal line. CT scans of the monkey brains were attempted before sacrifice and after removal of the brain, but our current equipment did not allow adequate resolution of the edema patterns in these small 200 g brains.

Results

Animal Studies

Monkey 1 sustained a 2 × 2 cm cold lesion of the left orbital surface of frontal lobe, which resulted in some local hemorrhagic necrosis (fig. 1A). From the lesion, edema could be traced upward along vertical fiber systems from the orbital to the middle frontal gyrus (figs. 1D and 1E) and posteriorly into the extreme and external capsules (figs. 1B and 1C). Blue dye traveling with the uncinate fasciculus ultimately reaching the medial temporal region could also be seen (fig. 1B). On gross inspection of the brain, there
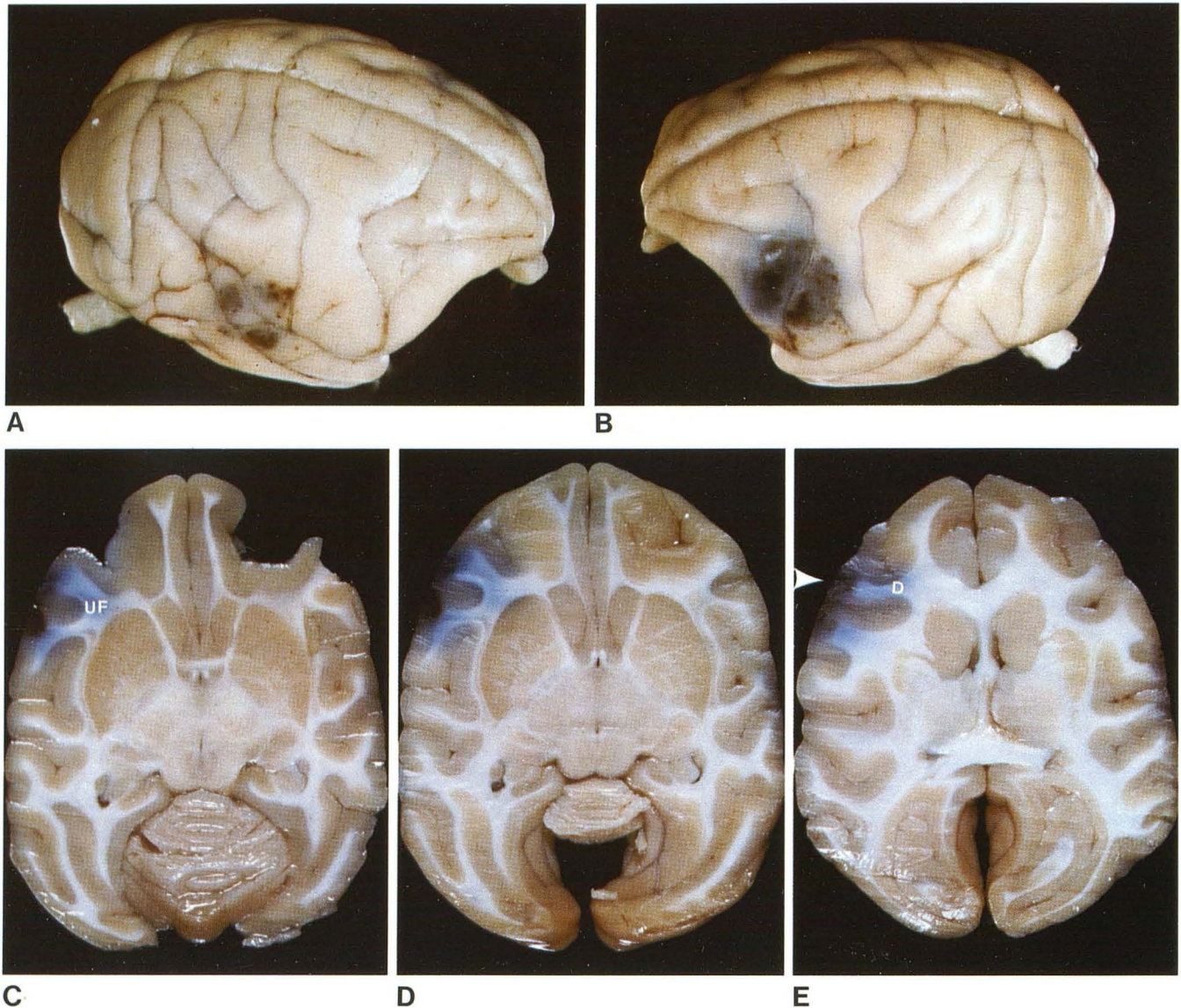
Fig. 3.—Monkey 3. Cold lesion of right parasylvian region and left mid and opercular frontal region. A and B, Hemorrhagic necrosis. C-E, Dye follows along uncinate fasciculus (UF) from frontal lobe lesion (*arrowhead*) in E downward along uncinate fasciculus to temporal tip seen in C and D. In E, edema spreads in an ascending fashion along dorsal leaf of uncinate fasciculus (D).

was diffuse gyral swelling and sulcal effacement with midline shift as a general manifestation of frontotemporal edema. Even though the forceps minor of the corpus callosum was mildly injured, there was little spread of Evans blue–labeled edema. A small amount of blue dye accumulated in the anterior limb of the internal capsule, probably following the course of the anterior thalamic radiations from prefrontal cortex to dorsomedial thalamic nucleus (figs. 1B and 1C).

Monkey 2 received two asymmetric cold lesions (figs. 2A and 2B). One, a 2 cm lesion in the right paramidline occipital region, was located in the visual projection and association area about 2 cm above the transverse sinus (fig. 2B). Edema spread from this lesion coursed predominately in the direction of the inferior occipitofrontal fasiculus (figs. 2D and 2E), however, it could not be traced as far forward as the frontal lobe. Its course included the fusiform and parahippocampal gyrus region, as seen in figure 2D. The vertical ascent of edema was also noted in the occipital lobe along fiber systems, which have been described within the occipital lobe itself, the so-called vertical occipital fasciculus (see fig. 8). Although the lesion was rather extensive, no spread of Evans blue could be found in the region of the splenium of the corpus callosum.

Lesion 2 in this animal was in the temporal polar region including the superior and middle temporal gyrus (fig. 2A). This lesion induced edema that spread into the external capsule and into the uncinate region as well as along the small U-shaped short association bundles primarily of the temporal lobe (fig. 2C).

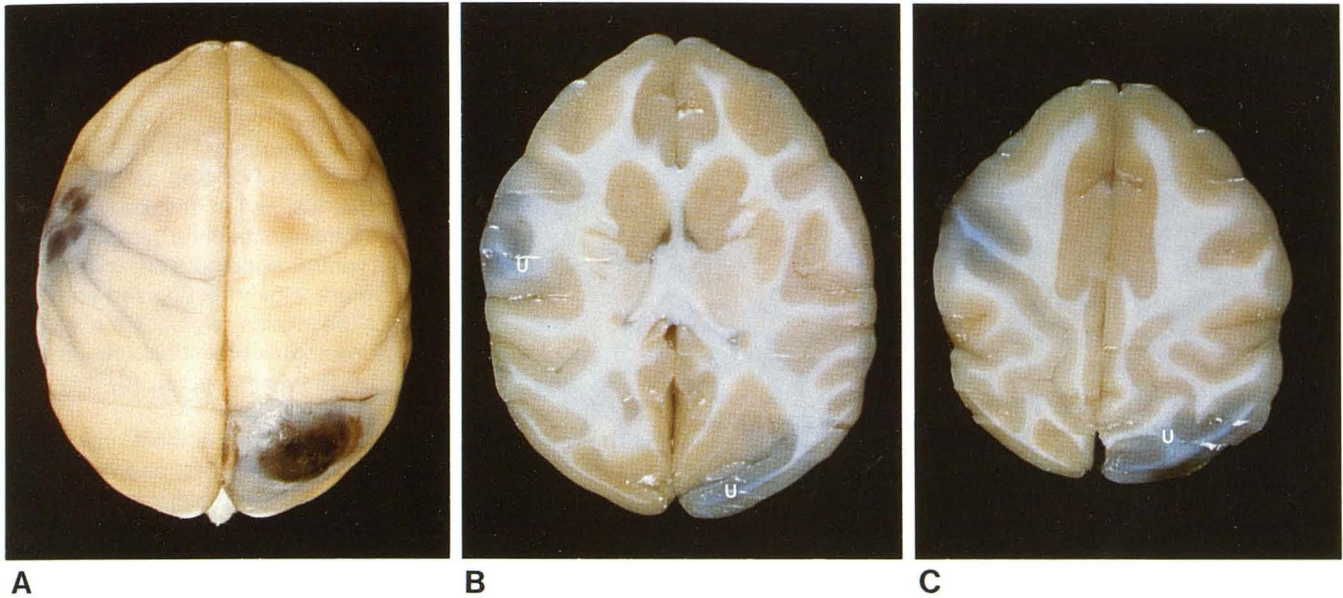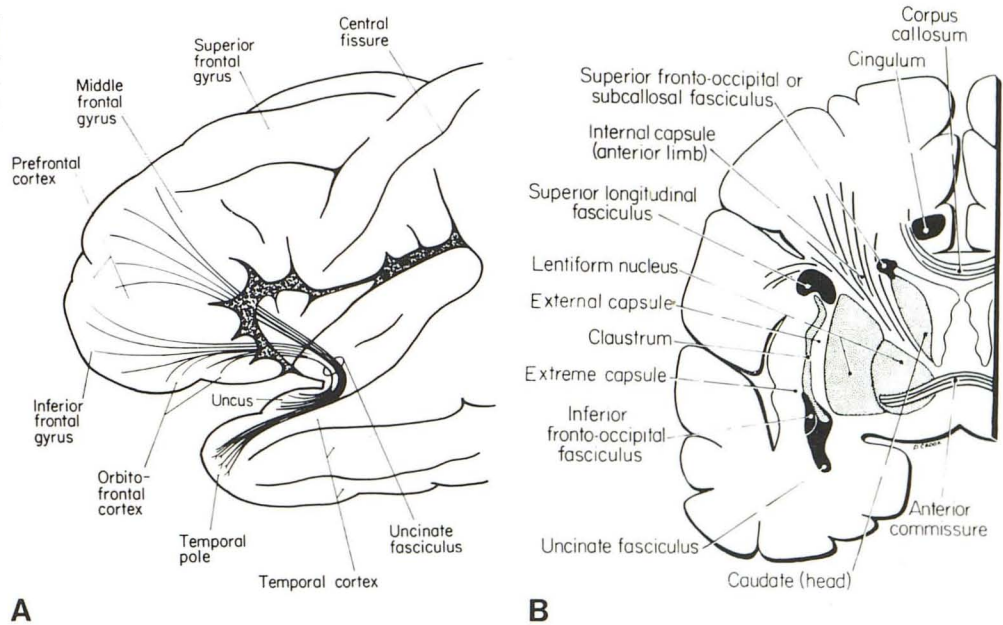Animal 3 received two asymmetrically placed lesions: one

A   B   C

Fig. 4.—Monkey 4. Cold lesions of left frontal lobe operculum and right posterior parietal area. **A,** Surface shows hemorrhagic necrosis. **B,** Edema only, occupying arcuate fasciculi (U) without longitudinal fiber spread. **C,** Edema spreads vertically along vertical occipital fasciculus. This series provides evidence that basal lesions alone produce edema in major basal long fibers, whereas higher convexity areas are connected with other association bundles.

Fig. 5.—**A,** Course of uncinate fasciculus including its dorsal and ventral leaves (not separately labeled). **B,** Coronal brain section demonstrates positions of uncinate fasciculus, extreme capsule, and inferior frontooccipital fasciculus. (Reprinted from [13].)

A   B

involving the left opercular (fig. 3B) middle and inferior frontal gyrus and measuring 1 × 1 cm; the other, a right-sided lesion (fig. 3A) straddling the sylvian fissure and encompassing most of the superior temporal gyrus near the posterior part of the sylvian fissure. Edema from the right superior temporal lesion was rather inconsequential and could not be followed with any clarity. However, the left frontal region demonstrated Evans-blue edema complex to follow the uncinate fasciculus including the dorsal leaf, which is more vertical in its orientation (figs. 3D and 3E),

and a ventral leaf from the middle frontal gyrus to the temporal tip (fig. 3C). The site of lesion placement can best be seen on figure 3D and the dorsal extent of the edema is evident on figure 3E.

Animal 4 received two asymmetrically placed lesions, one in the right occipital area in the visual association cortex and the other a low frontal opercular lesion over the midsylvian fissure in the pre- and post–central gyrus zone (fig. 4A). Apparently, only minimal connections are given over longitudinal association bundles since this lesion only dem-
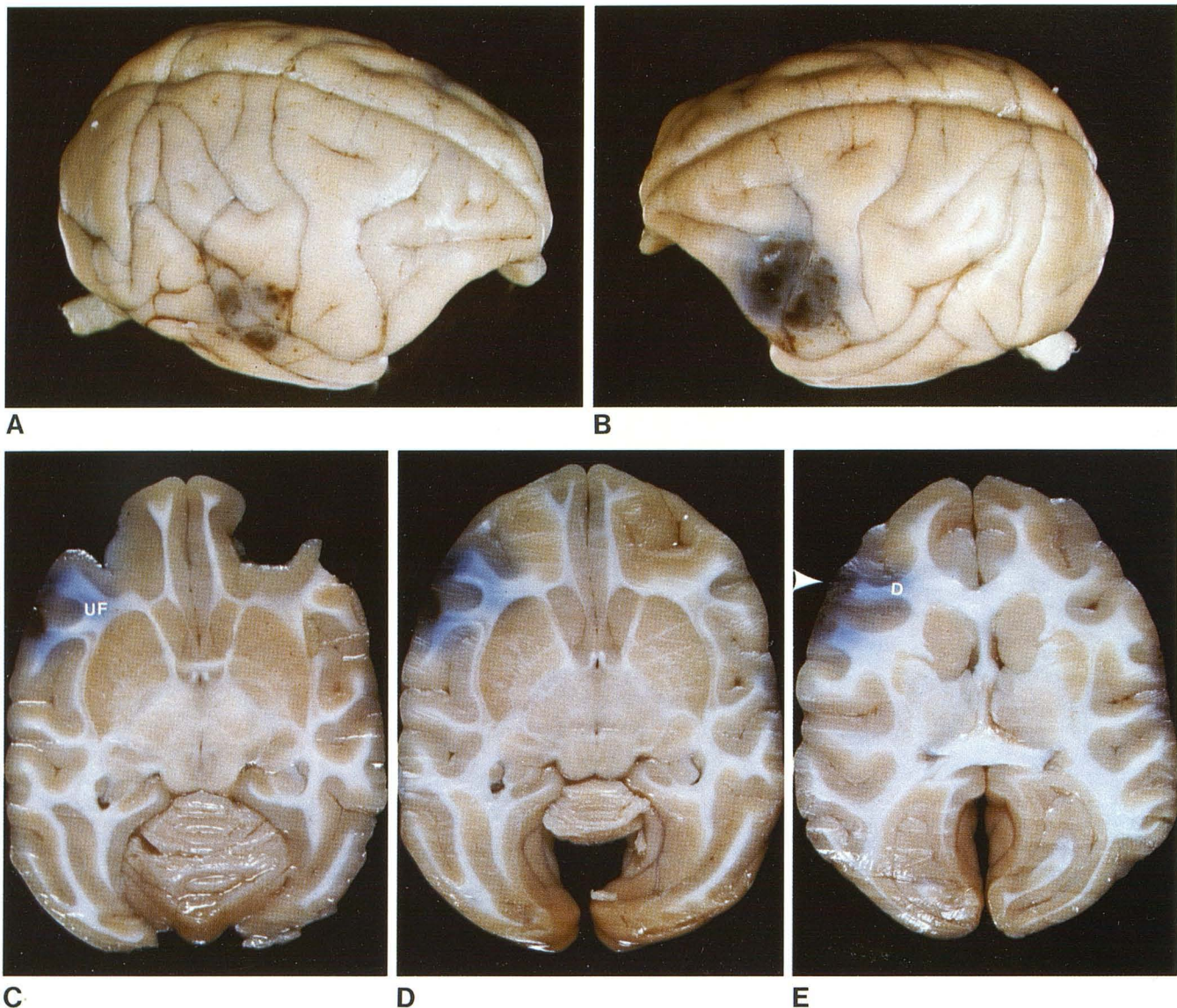
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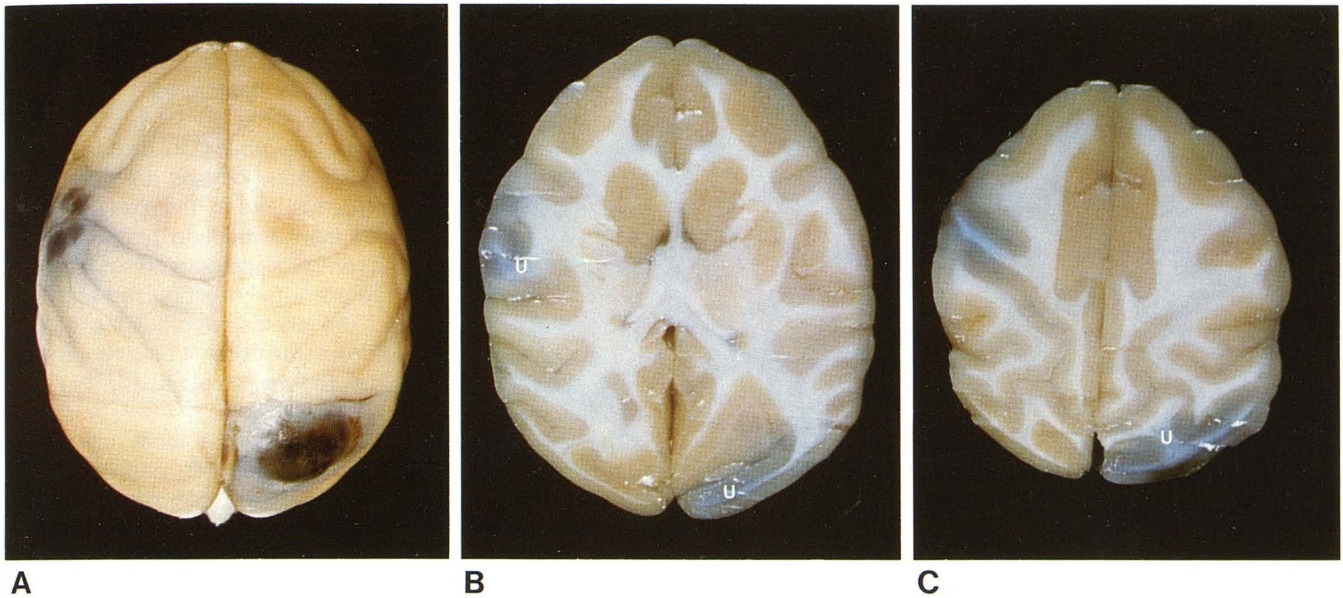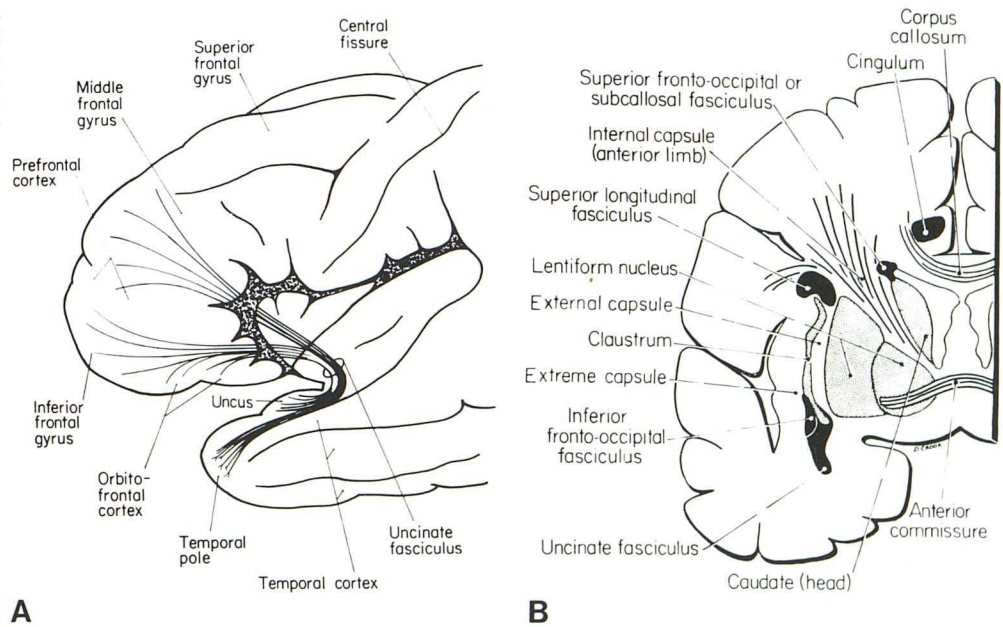Animal 3 received two asymmetrically placed lesions: one

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onstrated short U fiber connections in the frontal lesion and vertical intralobar U fiber connections in the occipital lobe. No other significant deep white-matter spread could be seen in this particular animal.

These observations suggest that the major association bundles provide an anatomic substrate or scaffolding along which cerebral edema·and presumably even blood, tumor, or other unencapsulated fluid masses can spread within the cerebrum. From a review of the literature and this study, there seem to be two categories of fiber systems: those that readily allow edema to spread and those that do not. Seemingly, the former systems include the major longitudinal and short association bundles including the U fiber systems, and the inferior frontooccipital fasciculus, the extreme capsule, and the uncinate fasciculus. The systems not allowing spread appear to include the corpus callosum, unless directly involved by a lesion, and the commissural systems, such as anterior and hippocampal commissures. The visual radiations have been shown in previous studies [8] to be rather resistant to the spread of edema, and they are included in this category. The reasons for the relative resistance to edema spread in certain fiber bundles remains enigmatic and should be further studied. The most likely underlying mechanism probably resides in the microvascular level.

Human Cases

Various human examples of spread of unencapsulated fluid masses are illustrated throughout the Discussion.

Discussion

The major bundles can be considered historically and with reference to the current animal studies and various clinical examples obtained from the neuroradiology files. There are many longitudinal association bundles, and for the most part they comprise groups of fibers that can be dissected among sheets of intertwining neural fibers [11]. Many bundles have been described, and a full discussion of these is neither desirable nor warranted here. Only a brief account of the important ones is provided.

Longitudinal Association Bundles

Uncinate fasciculus. This bundle, recognized as early as 1890 by Schnopfhagen [12] has been clearly shown to interconnect the orbital surface of the frontal lobe with the hippocampus and possibly the amygdala [13]. Its dorsal and lateral leaves are well documented [13] and are illustrated profusely in the literature (fig. 5A) and in this study (figs. 3, 6, and 7). Dorsal fascicles interconnecting temporal with middle frontal gyrus and ventral bundles from the orbital surface to the temporal tip are widely recognized and discussed (figs. 5A and 8). The clinical significance of this bundle in allowing false frontotemporal localizations has been shown repeatedly by Schneider et al. [5, 14] and Crosby et al. [13].

The uncinate fasciculus is possibly the most universally accepted of the major association bundles, and its course through the lower part of the external capsule has been shown in humans and monkeys by Bucy and Klüver [15].

The uncinate fasciculus is exemplified in the current monkey studies in animals 1, 2, and 3 as discussed above (figs. 1, 2, and 3C–3E). Clinical examples gathered recently (see figs. 6, 7, 9–12) demonstrate vasogenic edema from various causes spreading in the region of the external capsule and through the uncinate fasciculus from the temporal to the frontal regions or vice versa. It also seems likely that tumor can spread along the same fiber system, as evident in figure 13. This case is that of proven glioblastoma multiforme, in which the tumor appears initially to be multicentric. The author believes, however, that this may represent unifocal glioblastoma in the temporal lobe, which has followed the uncinate fasciculus as a means of spread from the temporal lobe to the frontal lobe. In fact, many so-called multifocal glial tumors may demonstrate this phenomenon. The cystic temporal lobe mass is well appreciated and several radiodense parts of tumor are best evidenced in the deep frontal region exactly along the course of the uncinate fasciculus on the lowest slice.

Figure 6A demonstrates a low midtemporal fossa meningioma with edema coursing along the inferior surface of the temporal lobe following the course of inferior frontooccipital fasciculus (fig. 6B). Figure 6C shows a fingerlike pattern of
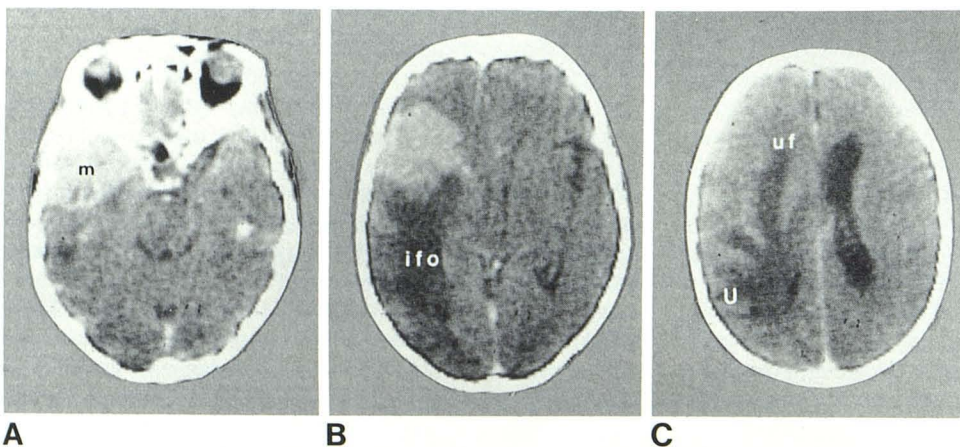

Fig. 6.—Meningioma in floor of left middle fossa. **A,** Tumor mass (M). **B,** Edema in inferior frontooccipital (ifo) and inferior longitudinal fasciculi. Anterior lucency extends along uncinate fasciculus and deep extreme capsule. **C,** Edema in arcuate fascicles (U) and in uncinate fasciculus (uf) in deep midfrontal lobe.

A   B   C

Fig. 7.—Meningioma of right olfactory groove. **A,** Edema extends along inferior frontooccipital fasciculus (ifo) into deep temporooccipital lobe. Uncinate fasciculus (uf) also involved. **B,** More abundant edema in same fascicles now in deep external capsule. **C,** Arcuate U fibers (U) spread among frontoparietal cortex.
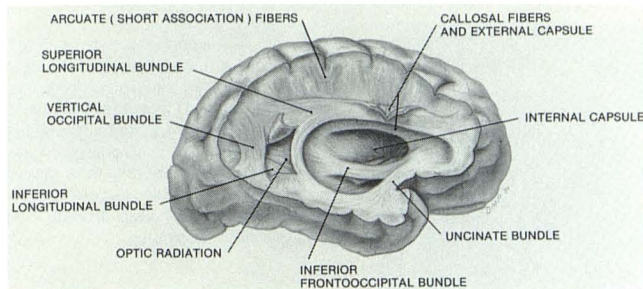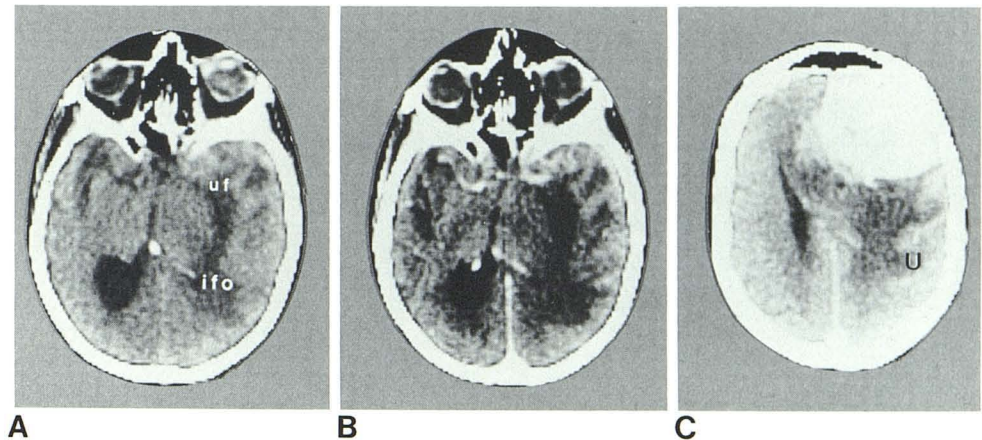

A   **B**   **C**

Fig. 8.—Several major association bundles are demonstrated. Note particularly uncinate, inferior frontooccipital, and vertical occipital fasciculi. (Modified from [10].).

edema filling the small U-shaped arcuate association fibers that are connecting the temporal lobe gyri and a large zone of edema coursing anteriorly through the region of the external capsule and through the uncinate fasciculus into the deep frontal lobar zone.

Figure 7C demonstrates enhancement in a large low-lying olfactory groove meningioma. Traced posteriorly and inferiorly from the tumor is a broad zone of edema ultimately arriving in figure 7B at the deep medial temporal lobe, presumably along the uncinate fasciculus. More posteriorly in the temporooccipital region, there is lucency along the inferior occipitofrontal fasciculus and the inferior longitudinal fasciculus.

The metastatic lung carcinoma seen in figure 9B provokes edema spreading in a fingerlike manner along the U fibers, along the inferior occipitofrontal zone, and through the external capsule in the region where the uncinate fasciculus lies. Figure 10 demonstrates a low-lying temporal meningioma provoking edema again through these very same fiber systems in a characteristic pattern.

At this juncture, it should be stated that the characteristic appearance of vasogenic edema requires the neuroradiologist to explain its origin. If the standard protocol for obtaining CT cuts has not provided evidence of an enhancing lesion, then further inferior or superior sections should be added to extend the search. An example is seen in figure 14, where, if one only obtained cuts through figure 14B, vasogenic deep white-matter edema would be identified; but

unless the cut of figure 14C were also obtained, the chloroma in this patient with chronic lymphocytic leukemia would be missed.

Although the preceding lesions were rather large in most cases, there was no commissural edema pattern in the anterior commissure or in the corpus callosum. From the studies of Rieth et al. [1] and others [8], it appears that unless the corpus callosum or other major commissural systems are directly involved, there appears to be no spread of edema between hemispheres. This is in contrast to the occurrence of lesions within the corpus callosum in which bilateral spread of tumor or edema can be seen. Except when butterfly glioma or other similar lesions occur primarily in the corpus callosum (fig. 15), it is rather uncommon to see bihemispheric patterns of edema. Such a pattern is usually caused by a vascular lesion or tumor in the corpus callosum.

Inferior longitudinal fasciculus and inferior frontooccipital fasciculus. These long association bundles have both been widely described in earlier anatomic literature [13, 16, 17]. The assertion by Davis [17] that the inferior longitudinal bundle is largely inseparable from the inferior frontooccipital fasciculus is confirmed by this study, and only the inferior frontooccipital bundle will be described and illustrated.

The inferior frontooccipital fasciculus has been well described by Schneider et al. [4], who reviewed its connections among frontal, occipital, and temporal lobes (fig. 5B). Also shown in their beautiful illustrations is the closely applied fascicle known as the inferior longitudinal fasciculus. The subclaustral course along the base beneath the external capsule can be seen in figure 5B.

The inferior frontoocipital fasciculus can be seen filled with edema in figures 2D, 2E, 6, 7, 9–11. On CT examinations, the bundle has a characteristic course lying obliquely along the inferior surface of temporooccipital regions lateral to the inferior horn with its posterior end inclined medially.

The clinical importance and false localizing effects of the inferior frontooccipital and inferior longitudinal fasciculi have been repeatedly shown by Schneider et al. [4] and Kahn et al. [16].

Extreme capsule. This band of fibers is insinuated between the insular cortex and the claustrum and is thought
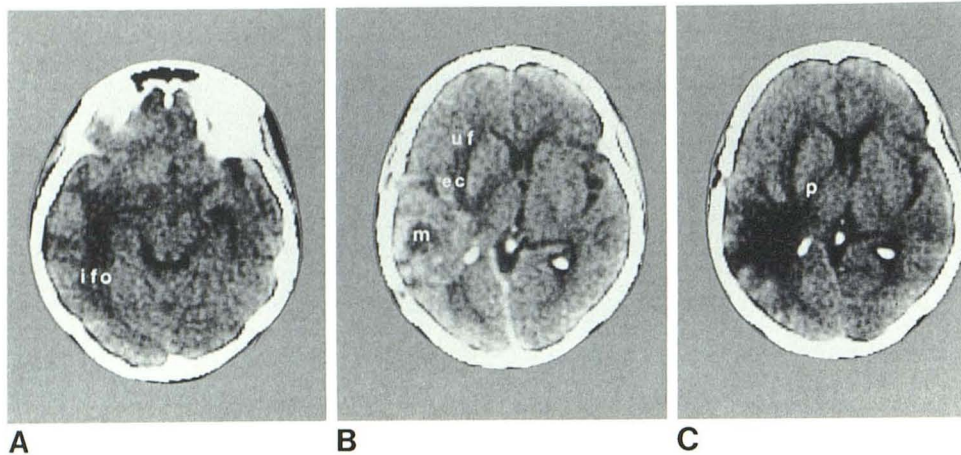
Fig. 9.—Adenocarcinoma of lung metastatic to left temporal operculum. **A,** Edema in inferior frontooccipital fasciculus (ifo) and extending forward into uncinate fasciculus. **B,** Tumor mass (m) with edema in uncinate fasciculus (uf) and extreme capsule (ec). **C,** Lucency in posterior limb of internal capsule (p).
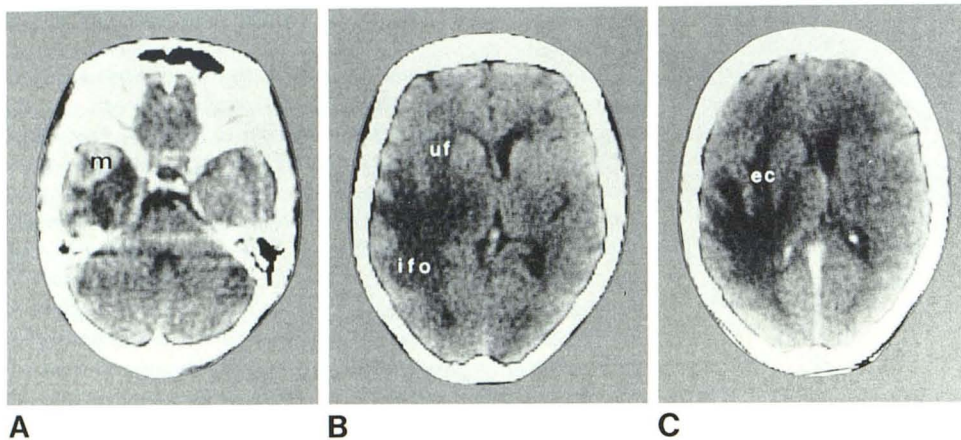

Fig. 10.—Meningioma in floor of left middle fossa again showing mass (m) in **A. B,** Massive edema in inferior frontooccipital fasciculus, U fibers (not labeled), and ventral limb of uncinate fasciculus (uf). **C,** Higher extent of edema in extreme capsule and medially in posterior limb of internal capsule. Lucency signifying edema in left deep frontal lobe along uncinate fasciculus.
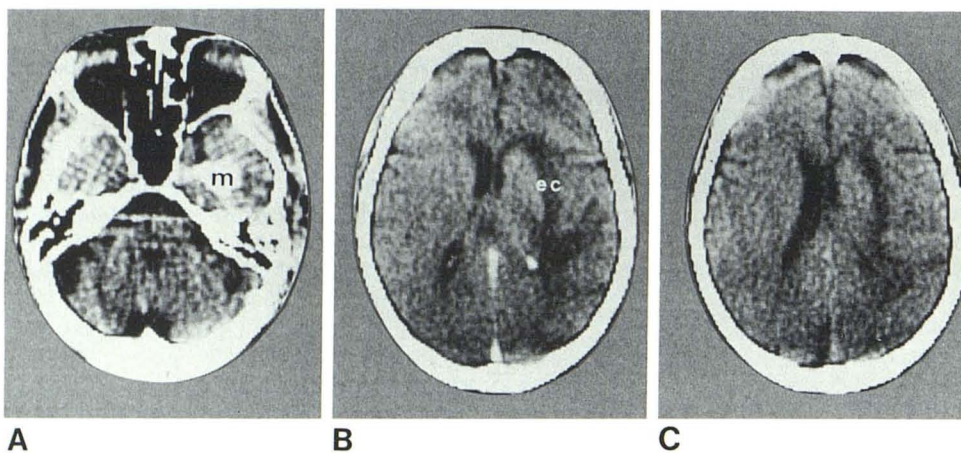

Fig. 11.—Right middle fossa floor meningioma. **A,** Mass (m) in middle cranial fossa. **B,** Characteristic vasogenic edema of extreme capsule (ec). **C,** Highest extent of extreme capsular edema.

by some authors to provide interconnections among the various island cortices [13, 18]. Numerous connections among superior temporal gyrus and the insula as well as frontoinsular connections have been described. Crosby et al. [13] have indicated that, at least for the macaque, it provides frontotemporal opercular interconnections. It is illustrated in figures 1C, 2C, and 5B. It can be seen filled with edema in figures 6C (just posterior to the uncinate fasciculus), 9B, 10C, and 11. It is inseparable from the uncinate fasciculus in the low subventricular levels (fig. 5B), but at levels through midventricles can be seen as a distinct entity (figs. 9B and 10C).

Fig. 12.—Adenocarcinoma of lung metastatic to deep left frontal lobe. **A,** Massive deep frontal edema with extension into region of uncinate fasciculus (uf). **B** and **C,** Higher cuts. Enhancing tumor nodule and arcuate fasciculi filled with edema (not labeled).
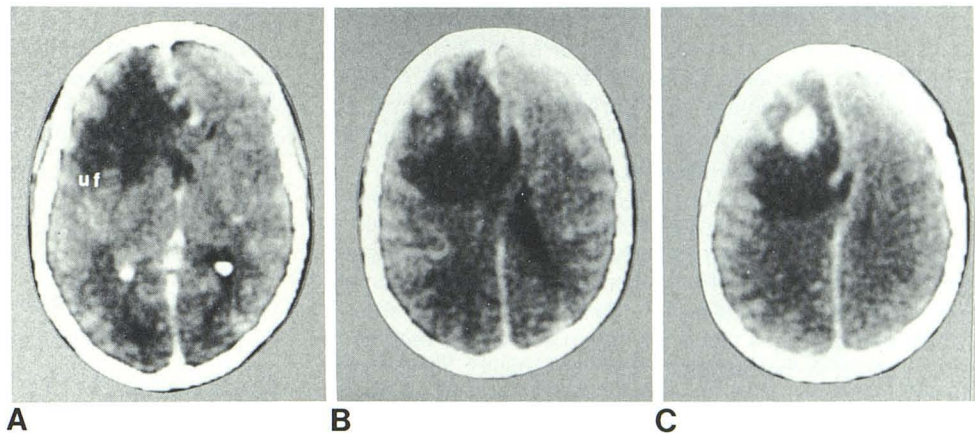

Fig. 13.—Glioblastoma multiforme in right temporal and frontal lobes. **A,** Large "necrotic" tumor mass in right midtemporal lobe. Second density in frontal lobe in region of uncinate fasciculus (uf). **B** and **C,** Respectively higher planes reveal more tumor along course of uncinate bundle.
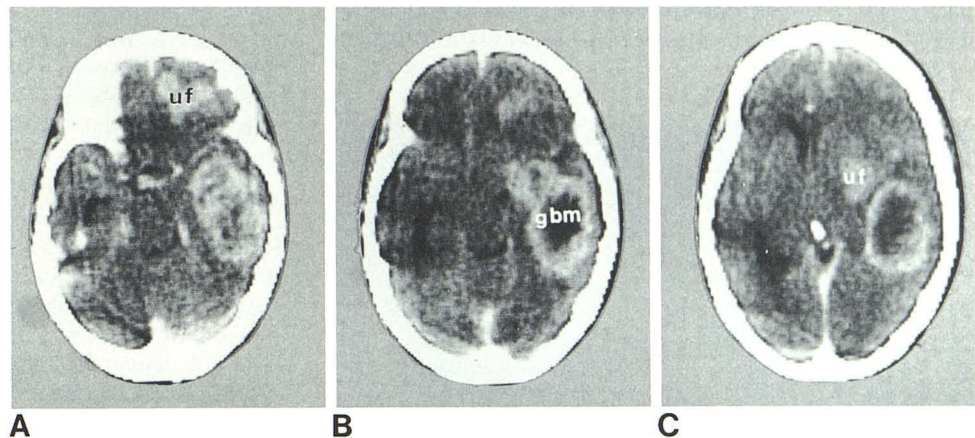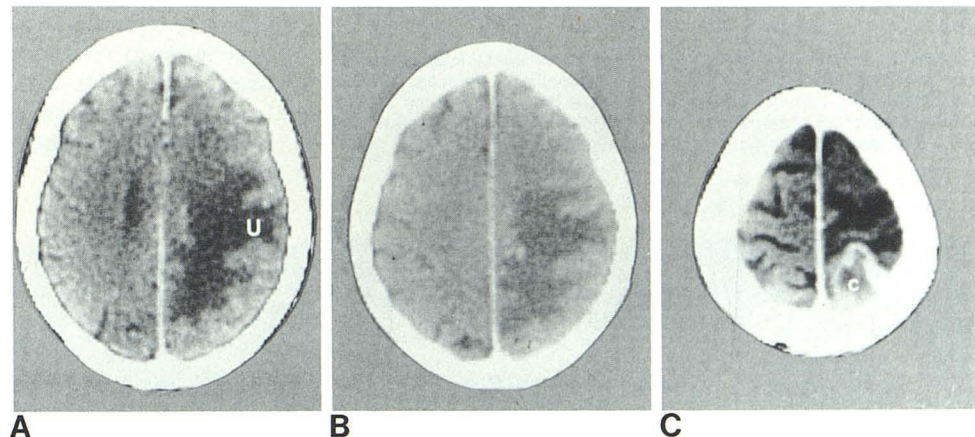

Fig. 14.—Chloroma with chronic lymphocytic leukemia. **A** and **B,** Edema in centrum semiovale extending along arcuate U fibers (U). **C,** Mass of chloroma (c) seen only on highest vertex view.

Short Association Fibers

Arcuate fibers. These comprise an almost limitless array of short intra- and intercortical fibers. They may even be interlobar, as seen in the figure 8 U draped around the central sulcus [13, 16]. Their orientation allows a characteristic configuration on CT and allows us to recognize white-matter disease patterns. Their multitudinous pattern of arrangement can present confusing patterns, however, as in cases of hemorrhage following a closed U bundle, presenting as a ring shadow in the proper tangent. Arcuate fascicles are seen in figure 16 filled with edema from a metastasis.

Vertical occipital fasciculus. One of many lesser bundles, the vertical occipital fasciculus connects dorsomedially with

ventrolateral regions of occipital lobe and more rostrally joins the fusiform gyrus with posterior parietal cortex [19]. The fibers can be seen in diagram form in figure 8. Monkey material showing Evans-blue spread includes figures 2D, 2E, 4B, and 4C. They are inferred in figure 9, as edema traverses 2 cm of occipital lobe in this patient.

In the cases collected over a 5 year period at this institution, the single lesion showing spread across commissural fibers, other than a corpus callosum glioma or hematoma, is shown in figure 17. This woman had multiple hematomata secondary to a coagulopathy. Only one image is shown, which demonstrates blood extending across the cerebellar commissure in the fastigium from the primary hemorrhage in the dentate nucleus. No autopsy proof could be obtained.

Posterior limb of the internal capsule. Several of the clinical cases (figs. 6C, 9C, and 10C) showed edema in the region of the posterior limb of the internal capsule. As projection fibers are thought to be resistant to edema [1, 8] and association fibers tend to promote spread [20], it ap-

pears that the edema seen in these studies may represent occipitotemporopontine connections and other relays from association cortices coursing along the posterior limb of the internal capsule. They may as well include the fibers of visual radiations, although these have been reported to be refractory to edema [8]. Berke [18] showed some relay of degenerating fibers into this area from a similar lesion involving the opercular frontal zones.

General Conclusions

This study confirms that the major association bundles of the brain provide a pattern of scaffolding for the spread of edema. The neuroradiologist must learn to recognize the characteristic pattern of vasogenic edema, obtain sufficient tomographic cuts, and use contrast enhancement to delineate the cause. This is particularly important in small lesions lying on the middle fossa floor and in lesions occupying the high vertex zone. Such lesions are highly characteristic, showing fingerlike projections of edema oriented along major bundles. Fiber-tract edema should not be dismissed as infarction or white-matter degenerative processes without thorough study.

Major fiber bundles may provide the modes of spread of both intra- and interlobar tumor. It is likely that certain cases presumed to be multicentric glioma may indeed be unicentric with spread along these bundles. We have shown such a case which we believe to exemplify this fact in figure 13.

Furthermore, since from the literature and the current study it seems that the corpus callosum is refractory to transmission of edema from a remote lesion, it must be surmised that the corpus itself is the lesion site in cases of bifrontal edema patterns. We have not seen examples of fluid masses spreading along the anterior commissure, the hippocampal commissure, but we have seen "butterfly glioma" of the posterior commissure presenting with hydrocephalus and an enhancing tectal and posterior thalamic mass. We believe that the low incidence of commissural lesions of all kinds relates to the smaller proportion of total white matter relegated to these systems.
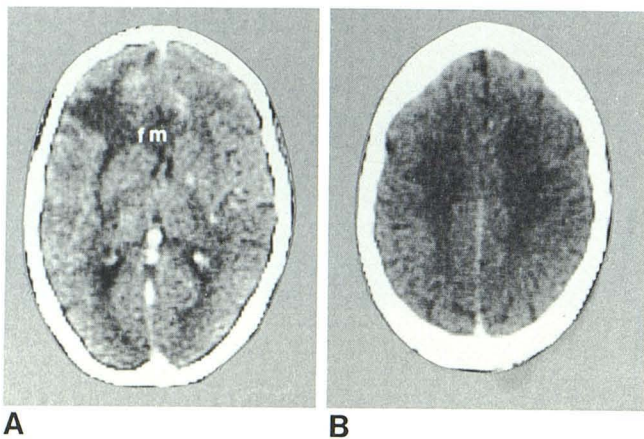

A   **B**

Fig. 15.—Metastatic carcinoma of breast to left frontal lobe with superimposed progressive multifocal leukoencephalopathy. **A,** Parasagittal frontal metastasis with bifrontal edema on left more than right. Lesion involves forceps minor (fm) of corpus callosum provoking bifrontal edema pattern. **B,** High centrum semiovale lucency of leukoencephalopathy.
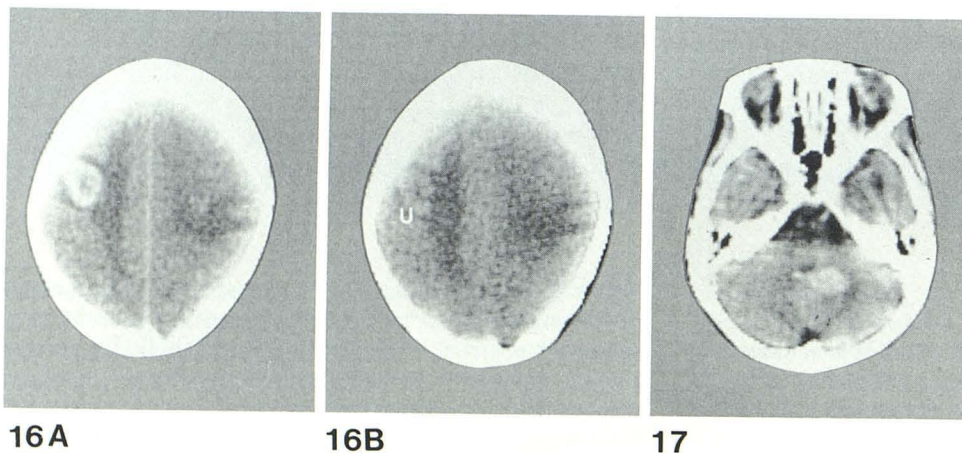

16 A   **16 B**   **17**

Fig. 16.—Metastasis from carcinoma of breast to left frontal lobe. **A,** Enhancing metastasis with edema in U fibers **B,** u) and along centrum semiovale. Second right-sided, less obvious mass and edema.

Fig. 17.—Primary hematoma in right dentate nucleus secondary to coagulopathy. Tongue of blood density crossing midline in region of cerebellar commissure in fastigium.

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