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Peritrigonal Echogenic "Blush" on Cranial Sonography: Pathologic Correlates

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Cranial sonography in neonates almost always reveals a hyperechoic "blush" just posterior and superior to the ventricular trigones on parasagittal views. This normal increased echogenicity resembles fine brush strokes. It is probably caused by the interface of numerous parallel fibers that are nearly perpendicular to the longitudinal axis of a sonographic beam passing through the anterior fontanelle. The same echogenicity is not seen on sonograms obtained through the posterior fontanelle because with that angulation the long axis of the sonographic beam and the fiber tracts are nearly parallel. Sonographic-pathologic correlation in 28 autopsy cases showed that abnormal, dense, globular, coarse, peritrigonal echogenicity was due to periventricular leukomalacia with hemorrhage. Cases with nonhemorrhagic periventricular leukomalacia or perinatal telencephalic leukoencephalopathy demonstrated the normal peritrigonal hyperechogenicity.

Recently, many articles on periventricular leukomalacia (PVL), an abnormality of the neonatal brain that has been discussed previously mainly in the pathologic literature, have appeared in the sonographic literature. We undertook a sonographic–pathologic correlative study to determine the nature of the commonly seen sonographic peritrigonal echogenic blush and to differentiate it from PVL, with or without hemorrhage, which occurs in the same region of the brain.

We began with two premises: The periventricular region is usually finely hyperechoic [1], and it is especially so posterolateral to the ventricular trigones (peritrigonal) on parasagittal sonographic scans obtained through the anterior fontanelle [2] (fig. 1). In an autopsy series where 88% of the dead, high-risk infants had pathologic proof of PVL, the involved areas always included the same peritrigonal region [3].

We wished to answer these questions: Is the commonly seen hyperechoic periventricular blush related to PVL? If not, is it related to myelin, hemorrhage, scanning artifact, blood vessels, or nerve fiber tracts? Can PVL be differentiated from normal brain on sonography?

Materials and Methods

We reviewed the cranial sonograms of 203 consecutive neonates and infants over a 1-year period, specifically looking for the peritrigonal, hyperechoic blush. All infants were scanned through the anterior fontanelle with sector real-time units equipped with transducers of 5.0, 6.0, or 7.5 MHz. We also reviewed 68 consecutive autopsies on live-born infants who died under 2 months of age and noted the prevalence of PVL in this group. In addition, cranial sonograms that were available on any of these autopsy patients as well as on any of the next year's 79 consecutive autopsies on similar live-born infants were reviewed (28 cases). We classified the peritrigonal echoes as absent, fine, or coarse. Fine, flame-shaped, peritrigonal echogenicity with poorly defined borders and the appearance of fine brush strokes was considered normal (figs. 1 and 2); globular, blotchy, coarse echoes with more defined edges were defined as abnormal (fig. 3).

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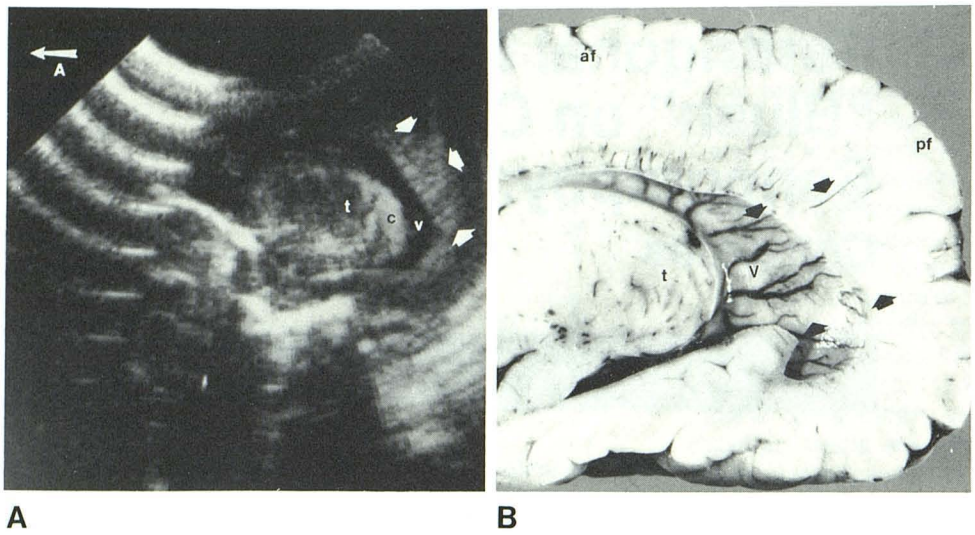

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Fig. 1.—**A,** Parasagittal sonogram (7.5 MHz) through anterior fontanelle of a 26-week estimated gestational age (EGA) infant. Normal, fine, peritrigonal echogenic blush (*arrows*). v = lateral ventricle; t = thalamus; c = choroid plexus; A = anterior. **B,** Parasagittal section of normal brain from 40-week EGA infant. Brain was sectioned to match usual parasagittal sonographic section (**A**). *Arrows* outline site of peritrigonal echogenic blush seen on sonography. Radial orientation of periventricular blood vessels is seen. A = anterior; V = trigone of lateral ventricle; t = thalamus; af = approximate site of anterior fontanelle; pf = approximate site of posterior fontanelle.

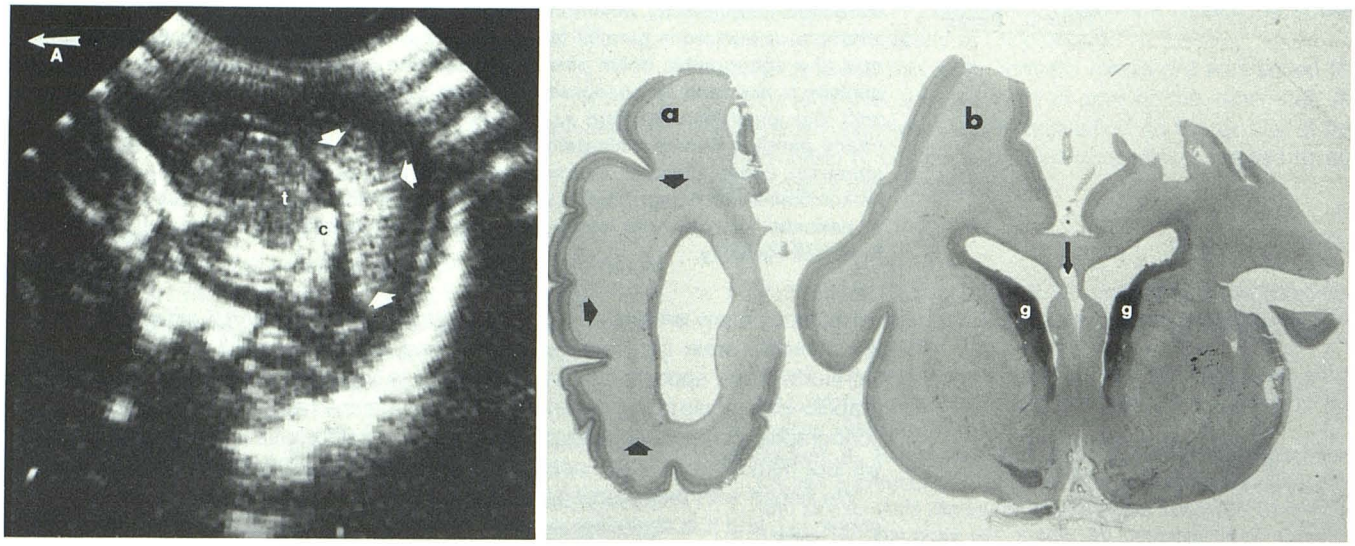

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Fig. 2.—**A,** Left parasagittal sonogram (7.5 MHz) through anterior fontanelle. Prominent but normal, fine, hyperechoic peritrigonal blush (*arrows*). Birth weight = 750 g; EGA = 26 weeks. A = anterior; t = thalamus; c = glomus of choroid plexus. **B,** Coronal brain section of infant in **A** at levels of left trigone (a) and bodies of lateral ventricles (b). Peritrigonal region (*short arrows*) is normal. Bilateral grade 1 germinal layer hemorrhage (g) and cavum septi pellucidi are seen (*long arrow*).

The brains of the 28 infants who during these 2 years had had recent cranial sonograms before death and/or postmortem sonograms within 6 hr of death (four patients) were carefully reexamined microscopically without knowledge of the sonographic findings. Sections 10 μm thick of frontal white matter dorsolateral to the lateral horns at the levels of the rostral basal ganglia, of the foramina of Monro, and of the midthalami were examined, as was the parietooccipital white matter dorsolateral to the trigones and back to the occipital horns. Sections were stained with hematoxylin and eosin and luxol fast blue (for myelin). We noted the presence of PVL, perinatal telencephalic leukoencephalopathy (PTL) (a form of generalized white-matter damage that may be an early prenecrotic form of PVL [4, 5]), parenchymal hemorrhage, myelin, and germinal matrix and intraventricular hemorrhage.

In contrast to another autopsy correlation study [6], we looked at infants dying at less than 2 months after birth. Our interest was in trying to distinguish the hyperechogenic, precavitary stage of PVL from the normal peritrigonal hyperechoic blush, and we wanted to be able to correlate the neuropathologic sections with recent sonograms.

Infants, some of whom were not part of the autopsy series, were scanned while power output and time gain compensation settings on the scanner were changed to see how this affected the appearance of the peritrigonal blush. Some infants were also scanned through the posterior fontanelle.

Results

Review of the cranial sonograms on 203 consecutive infants revealed that 159 patients had had adequate views of the peritrigonal region. Their mean estimated gestational age

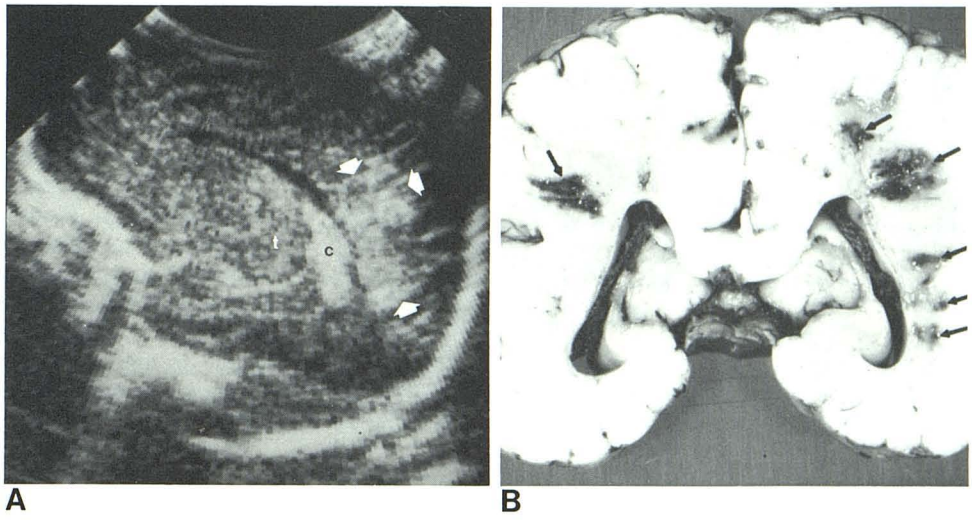

Fig. 3.—**A,** Right parasagittal sonogram (7.5 MHz) through anterior fontanelle. Abnormal, coarse, globular, hyperechoic peritrigonal echoes (*arrows*). EGA = 35 weeks. t = thalamus; c = glomus of choroid plexus. **B,** Coronal section at level of trigones of infant in **A,** who died 1 day after sonography. Peritrigonal hemorrhages (*arrows*) in microscopically defined areas of PVL were seen in **A.**

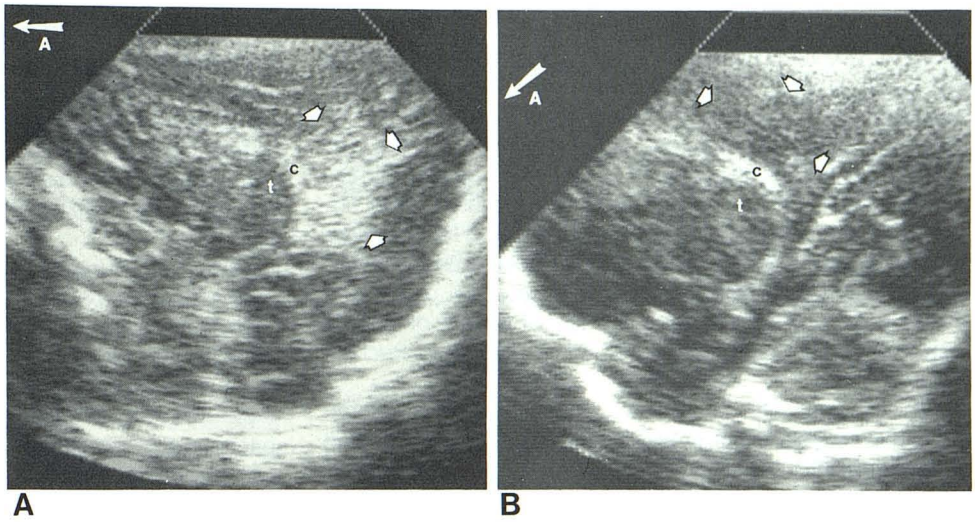

Fig. 4.—Parasagittal sonogram (5.0 MHz) through anterior (**A**) and posterior (**B**) fontanelles. Normal, fine, hyperechoic, peritrigonal blush (*arrows*) is seen through anterior but not posterior fontanelle, although that area is well demonstrated (*arrows*). A = anterior; c = choroid plexus; t = thalamus.

(EGA) at birth was 32 weeks, and their mean total (gestational and postnatal) age at the time of scanning was 33 weeks. Of these 159 patients, 154 (97%) demonstrated the peritrigonal hyperechoic blush. We have no explanation for the lack of a hyperechoic blush in the other five patients. The 44 patients with poor scans of the peritrigonal region tended to be older at the time of scanning, with a mean gestational age of 41 weeks. This group also included 10 patients between 4 and 9 months postnatal age. In our series, no patients over 4 postnatal months had adequate views of the peritrigonal region. We have seen exceptions to this since this study was completed. However, as a general rule, the peritrigonal region is seen less well in the older, more mature infant, probably because of the decreasing size of the fontanelle and poorer resolution of detailed structures in large heads where lower frequency transducers are needed for penetration.

In one case with a bright but fine peritrigonal echogenicity, the blush disappeared when scans were obtained through the posterior fontanelle. At pathology, this area was normal. In other cases, which were not part of the autopsy series, the normal, fine, prominent blush also disappeared when scanned through the posterior fontanelle (fig. 4). In contrast, in a case with coarse periventricular hyperechogenicity caused by bleeding, the abnormal echoes remained when scanned through the posterior fontanelle. Changing the power output and TGC curves did change the overall brightness or contrast of the normal peritrigonal blush but did not cause it to appear or disappear.

The pathologic prevalence of PVL in 68 consecutive liveborn infants dying under 2 months of age was 26%. This corresponds to other autopsy series cited in the literature [3, 7–9]. Fifteen (22%) of the 68 autopsied infants had evidence of PTL, and 27 (40%) had PVL or PTL or both (only PVL, 12; only PTL, nine; both, six). As high as these percentages are in our autopsy cases for 1 year, they are markedly below the almost uniform (97%) prevalence of the hyperechoic periventricular blush seen with sonography.

In 28 autopsy cases, sonographic-neuropathologic correlation was possible (table 1). In only one case was peritrigonal hemorrhage present without PVL or PTL. In that case the

TABLE 1: **Peritrigonal Echogenic Blush on Sonography: Autopsy Correlation**

Periventricular Leukomalacia on Autopsy	Sonographic Appearance	
	Coarse	Fine
Absent	0	14 (HEM, 1*; PTL, 3)
Present	5 (HEM, 5)	8 (HEM, 4)
Total	6†	22

Note.—HEM = hemorrhage; PTL = perinatal telencephalic leukoencephalopathy.
* Hemorrhage was microscopic.
† Includes one infant for whom pathologic sections through the trigone were unavailable. PTL was present anteriorly and posteriorly.

hemorrhage was microscopic and petechial; the infant was 25 weeks EGA and died at 1½ days. Germinal layer hemorrhage without intraventricular extension was present, and the sonogram showed a normal, fine peritrigonal echogenic blush. In the other nine peritrigonal hemorrhage cases, the hemorrhage accompanied PVL. The hemorrhage was either petechial, small, or large, but always within areas of necrosis. In no case was there a frank hematoma. Four of the nine cases with PVL and hemorrhage had normal-appearing peritrigonal echogenic blushes with petechial or small hemorrhages. In two of those four cases, the sonograms were obtained on the day of death. In one case, it was obtained on the day before, and in the other case 2 days before death. Hemorrhages may have occurred after the sonograms were obtained. In five cases with peritrigonal PVL and hemorrhage, the peritrigonal hyperechoic blush was coarse and irregular (fig. 3). Peritrigonal hemorrhages were petechial or small in three cases and large in two. No normal patient or patient with PVL without hemorrhage had this irregular hyperechoic pattern.

Four patients with peritrigonal PVL without hemorrhage had a normal peritrigonal hyperechoic blush. Three cases had PTL without PVL. Normal, fine, increased peritrigonal echoes were present in these three cases. In none of the 28 cases was myelin present microscopically in this region. Thus, myelinization is not the cause of the normal periventricular blush. In two cases, the germinal layer, usually limited at birth to the subependymal region over the head and body of the caudate nuclei, did extend out into the periventricular white matter but without hemorrhage.

Germinal layer or intraventricular hemorrhage was present at autopsy in nine (53%) of the 17 cases with evidence of PVL or PTL (three had the coarse peritrigonal echoes) and in six (54.5%) of the 11 cases with no evidence of PVL or PTL (54.5%) (fig. 2B). The mean postnatal age at death and the mean EGA for the six cases with the coarse peritrigonal echoes was 5 days and 35 weeks, respectively. The mean EGA for the patients with fine peritrigonal echoes was 32.4 weeks. The mean age at death for that group was 2.4 days, excluding five cases between 21 and 35 days at death.

Discussion

The peritrigonal echogenic blush, appearing on parasagittal sonograms as a grouping of fine, linear densities—almost like brush strokes—is virtually always present on cranial sonograms of premature babies (figs. 1 and 2). This was true in our series and in that of Grant et al. [1], who reviewed the sonograms of 180 premature neonates less than 33 weeks gestational age. Further, this sonographic finding was much less common in the older infants that we studied. We hypothesize that there are normal or abnormal factors, probably anatomic, which are peculiar to the immature brain and cause this appearance. Coincident with our review of pathologic material from autopsies of premature neonates was the publication of several papers that reported the sonographic appearance of PVL [6, 10–24]. Thus, we became interested in answering three questions: What causes the peritrigonal blush? Is the echogenic blush related to PVL? Is there a difference in the appearance of the blush between those infants with and without PVL?

The tendency for neonates to develop multiple small foci of necrosis in the periventricular region has been noted by neuropathologists for some time [25]; this was termed PVL by Banker and Larroche [7] in 1962. The propensity for these lesions to be peritrigonal in location has been attributed to the effects of hypotension and subsequent ischemia in the periventricular watershed region, an endarterial zone of perfusion [26]. It has also been recognized that severe hypoxic insult can produce large confluent areas of damage, which extend through white matter [8] and may be hemorrhagic [9]. This has been termed hemorrhagic PVL. In one autopsy series, however, 75% of PVL cases were without hemorrhage [9].

Gilles and Murphy [4] identified another type of injury to neonatal white matter that is characterized by the presence of hypertrophic astrocytes and proteinaceous amphophilic globular deposits. As the damage is more diffuse than PVL, does not necessarily include necrosis, and has been associated with prior exposure to endotoxins from Gram-negative bacteria, this entity was termed *perinatal telencephalic leukoencephalopathy.*

In most cases, the increased peritrigonal echogenicity is normal, but separating normal from abnormal is not always easy. The peritrigonal hyperechogenicity is so common that in one series [23] the authors chose to ignore it and only considered anterior hyperechogenicity superolateral to the external angles of the lateral ventricles at the level of the foramina of Monro as abnormal. Our incidence of PVL or PTL was not sufficiently high to account for the almost constantly noted peritrigonal echogenic blush. Correlation of autopsy and sonographic findings in the 28 children who underwent cranial sonography and autopsy showed that peritrigonal echogenic blushes were found in cases both with and without PVL or PTL. Yet, no case with normal peritrigonal brain sections had the course, globular, bright peritrigonal echoes. Five of those six cases with coarse peritrigonal echoes had pathologic sections of the peritrigonal region available. In all five, PVL with hemorrhage into the areas of necrosis without frank hematoma was present (fig. 3). In one series [21], surviving infants with small, linear, intraparenchymal, periventricular echoes extending a few millimeters into white matter had no or mild neurologic deficits, which was in contradistinction to the infants with large, globular, dense echoes. All survivors had moderate to severe neurologic deficits, and at

neuropathology, hemorrhagic infarction was present in those regions of the brain.

In another autopsy series [23], four of seven infants who died had increased periventricular echoes, although at the level of the foramina of Monro. Two of these four had PVL, but two did not (one was completely normal). Increased echogenicity in what was thought to be nonhemorrhagic PVL has been reported [14, 16, 17]. The possibility that such hyperechogenicity was due to edema [19] or venous congestion [12] was raised. PVL is actually a periventricular infarct, and one author prefers to call it such [25]. The earliest changes are microscopic. Secondary bleeding into the infarcted brain can vary from microscopic perivascular, to petechial, to linear streaky hemorrhage, and to large hemorrhages, which are more common in infants with a bleeding diathesis [9].

Cavitation with the appearance of small periventricular cysts can occur in 2–4 weeks [25]. At that stage the sonographic diagnosis of PVL is much easier. Our infants with PVL all died within 10 postnatal days except for one who died at 27 days and was last imaged at 23 days. No peritrigonal cysts were detected in our series.

The usual fine blush is not caused by white-matter damage, myelin, or hemorrhage. Also, extension of germinal matrix into this region occurred in only two of 28 cases, and is therefore not the cause. Although no venous congestion was noted, that or edema as a cause for peritrigonal echoes cannot be completely excluded.

The best explanation for the fine, hyperechoic peritrigonal blush is a consequence of the orientation of normal fiber tracts and their accompanying vasculature in the brain (fig. 1B). The beautiful specimens prepared by Gluhbegovic and Williams [27] graphically demonstrate the orientation of normal fiber tracts between cerebral cortex and diencephalon (fig. 5). The regular, almost parallel fibers would provide multiple interfaces to a perpendicular sonographic beam, especially within the gelatinous, less echogenic, watery neonatal brain. Because almost all cranial sonograms are obtained through the window provided by the anterior fontanelle, fibers in the area superior and posterior to the trigone would be perpendicular to the interrogating sonographic beam. The fibers anterior to the genu of the corpus callosum are likewise perpendicular to the beam, and a similar blush is often seen there. In those areas, the acoustic interfaces are maximal, the beam is best focused, and the echogenic blush is greatest.

In patients whose sonograms through both fontanelles were possible, the peritrigonal echogenic blush was evident only on scans through the anterior fontanelle (fig. 4A). When the parietooccipital area is scanned through the posterior fontanelle, the fibers are oriented parallel to the interrogating sonographic beam and the blush disappears (fig. 4B).

As infants age, the blush is less evident. The anterior fontanelle closes and the brain enlarges, making adequate demonstration of the peritrigonal area difficult. Also, myelination, decreasing water content, and especially the developing convolutions probably play a role in changing the sonographic appearance of the brain.

In summary, our material indicates that the fine, symmetric, peritrigonal, echogenic blush is present on sonograms of most, if not all, premature infants. It is probably related to the normal anatomy of fiber tracts and accompanying vasculature. Infants with this fine linear pattern of echogenicity may or may not have PVL. Coarse blotchy, periventricular echogenicity should be considered an abnormal finding, which correlated highly in our series with PVL and hemorrhage.

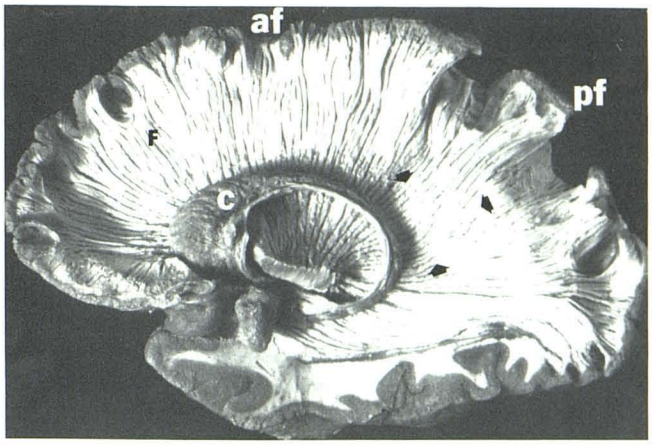

Fig. 5.—Sagittal section of normal brain shows radiating fiber tracts. F = frontal lobe; af = approximate location of anterior fontanelle; pf = approximate location of posterior fontanelle; peritrigonal region (*arrowheads*); c = caudate nucleus. (Reprinted from [27]).

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