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Anterior Choroidal Artery: Angiographic Analysis of Variations and Anomalies

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In the first part of this investigation, 216 consecutive carotid angiograms from 108 patients were reviewed to analyze variations of the anterior choroidal artery (AChA) for the origin, size, and course of the stem and for the possible identification of the uncal branch, perforating branches, and plexal segment of the AChA. In the second part of our investigation, we analyzed anomalies of the AChA seen on angiography. The anomalies of this artery were divided into hypoplastic and hyperplastic types, with prevalences of 3% and 2.3%, respectively, as determined from 216 control carotid angiograms. Hypoplasia of the plexal segment of this artery might represent an evolutionary variant in which the artery ceased to acquire choroidal branches, thus remaining in the reptilian stage. Twenty-five hyperplastic anomalous arteries that supplied a part of the distribution of the posterior cerebral artery were found not only on the 216 control angiograms (five arteries), but also on 2000 carotid angiograms obtained over 8 years (20 arteries). The hyperplastic arteries were further classified into subtypes according to the distribution area and course of the vessel. The hyperplastic anomalies are considered to represent a situation in which the AChA has maintained, as a main pathway, an anastomosis with the posterior communicating artery and posterior cerebral artery.

Our detailed analysis of the variations and anomalies of the choroidal artery should be useful in the assessment of this territory.

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Occasionally, a well-developed artery other than the posterior communicating artery (PCoA) originates from the internal carotid artery (ICA) and supplies the inferomedial aspect of the temporooccipital lobe including the territory of the anterior choroidal artery (AChA) and a part of that of the posterior cerebral artery (PCA) [1-8]. In 1907, Blackburn [1] was the first to describe two occasions in which the AChA was the main supply to a large part of the territory of the PCA in a study of 220 brains. In both brains the PCoA and PCA were hypoplastic, mainly supplying the cerebral peduncle only, whereas the AChA was large, providing both choroidal branches and arteries to the territory normally supplied by the PCA. Since then, such arteries have been described by many authors and have been considered hyperplastic anomalies of the AChA [1, 2, 5, 6, 8], an anomalous temporal artery originating directly from the ICA [4], or two separate trunks of the PCA [3]. We have also noted such arteries and found that there are several forms of this kind of anomalous vessel. In addition, we have noted hypoplasia of the plexal segment of this artery. The absence and anomalous origin of this artery have been described, although hypoplasia of the plexal segment of this artery has never been clearly described in the literature. This prompted us to review the angiograms from patients with these types of anomalies.

In the first part of our investigation, we analyzed 216 consecutive carotid angiograms from 108 patients, with special attention to the origin of the artery and the size of the plexal segment; in the second part of our investigation, we analyzed

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anomalies of the AChA seen on angiography. The anomalies of the artery were classified into hypoplastic and hyperplastic types; hyperplastic types were further divided into subtypes.

Materials and Methods

For the control study, 216 consecutive stereoscopic carotid angiograms from 108 patients were reviewed. Most of the studies were performed by selective injection into the ICA (83%), while in 17% a common carotid injection was performed. The catheter used was 5 French in most cases; the contrast medium was 60% meglumine, 9 ml for the ICA and 12 ml for the common carotid injections, respectively. The angiograms were examined for the origin, the size of the stem 1 cm from the point of origin, the course of the cisternal segment of the artery, and the possible identification of the uncus branch and perforating branches. The size of the plexal segment of the AChA (both medial and lateral plexal branches) was evaluated subjectively as unidentifiable, small (barely visible), medium-sized (well visible but not large), or large (extending superiorly beyond the posterior pole of the thalamus). The size of the PCoA was also evaluated subjectively as unopacified; small (the trunk of the PCA unopacified); medium-sized, partially filling the PCA trunk; and large, directly connecting with the PCA trunk (fetal type). The measurement was corrected for the magnification ratio (1.3) of the angiograms.

The anomalies of the artery were analyzed and classified into hypoplastic and hyperplastic types. Hyperplastic anomalies of the AChA supplying the inferomedial temporooccipital region, including the territory of the AChA and part of the PCA territory, were found not only on the 216 control angiograms (five arteries) but also on 2000 carotid angiograms obtained over 8 years (20 arteries). Our analysis, therefore, included 25 hyperplastic anomalous arteries in 23 patients (the anomalous artery was paired in two patients). The angiograms of these 23 patients were observed in order to determine the course and territory of the blood supply for the AChAs and to determine the presence of choroidal branches from this artery. The size of the PCoA, development of the temporal branches of the PCA, and any associated vascular anomalies were also evaluated in these patients.

Results

Observation of Control Carotid Angiograms (Table 1)

Origin.—The AChA was identified in all 216 carotid angiograms; all AChAs originated from the posterior aspect of the supraclinoid portion of the ICA. The origin was just proximal to the ICA terminal bifurcation but distal to the origin of the PCoA when the latter vessel was filled (Figs. 1 and 2). In no case was there an anomalous origin of the artery from the middle cerebral artery or the PCoA.

Cisternal segment.—In about two-thirds of cases, the cisternal segment of the AChA took a gentle S-shaped course on the lateral view (Fig. 1); in the other one-third, it took an atypical course. The diameter of the cisternal segment, measured 1 cm distal to the origin, ranged from 0.38 to 2.0 mm (average, 0.75 mm) and was judged as large (0.8 mm or more) in 33.0%, medium-sized (0.6–0.8 mm) in 51.8%, and small (less than 0.6 mm) in 14.2%. The uncus branch and perforating branches were identified on 35.7% and 47.2% of carotid angiograms, respectively (Fig. 1). The uncus branch was hypertrophic on three carotid angiograms (1.4%), supplying the distribution area of the anterior temporal artery of the PCA. In one patient, the AChA was so prominent bilaterally

TABLE 1: Evaluation of the Anterior Choroidal and Posterior Communicating Arteries on 216 Carotid Angiograms

Artery Evaluated/Aspect Assessed	%
Anterior choroidal	
Origin from the supraclinoid internal carotid artery (distal to the posterior communicating artery)	100
Cisternal segment	
Course	
S-shaped	71.8
Atypical	28.2
Size	
Large (≥ 0.8 mm)	33.3
Medium (0.6–0.8 mm)	52.3
Small (< 0.6 mm)	14.4
Uncus branch	
Prominent	9.3
Barely visible	26.4
Unopacified	64.4
Perforator	
Prominent	11.1
Barely visible	36.1
Unopacified	52.8
Medial plexal branch ^a	
Large	43.8
Medium	44.8
Small	8.4
Unopacified	3.0
Lateral plexal branch ^b	
Prominent	18.2
Barely visible	45.4
Unopacified	36.4
Posterior communicating	
Size	
Large	14.4
Medium	32.9
Small	38.9
Unopacified	13.9

^a Six cases were excluded because the medial plexal branch was obscured and unidentifiable owing to overlapping posterior cerebral artery vessels.

^b 14 cases were excluded because the lateral plexal branch was obscured and unidentifiable owing to overlapping posterior cerebral artery vessels.



Fig. 1.—Angiographic appearance of normal anterior choroidal artery (lateral view) in patient with middle cerebral artery occlusion at its origin. Note typical S-shaped course of cisternal segment of anterior choroidal artery, prominent uncus branch (solid arrowhead), perforator (large arrow), and medial and lateral plexal branches (small arrows). Small posterior communicating artery (open arrowhead) is seen to partially opacify posterior cerebral artery system.

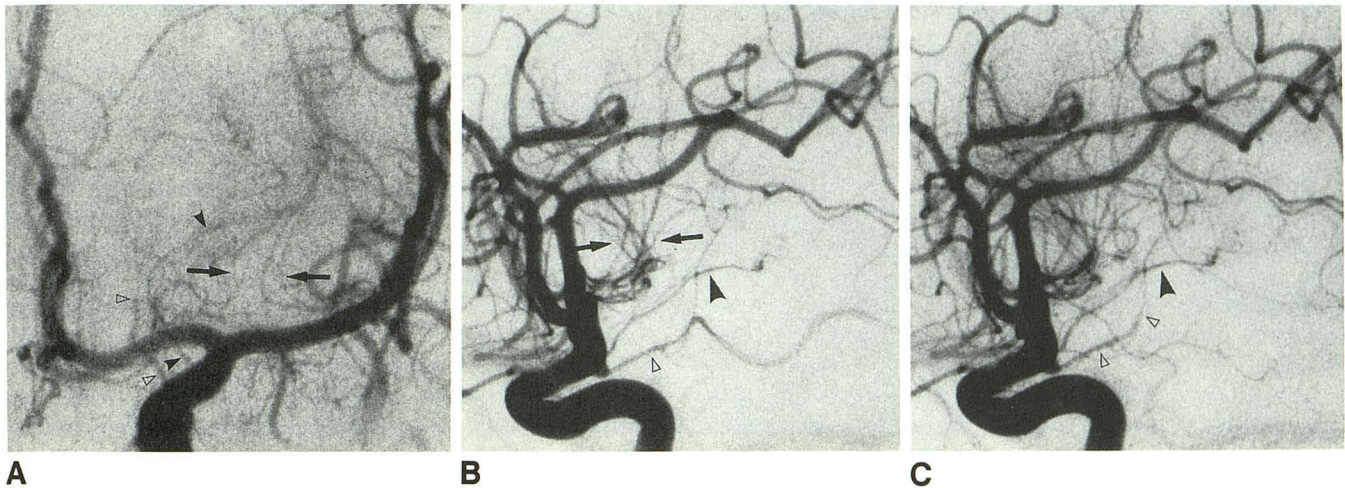


Fig. 2.—Angiographic appearance of normal anterior choroidal (AChA) and posterior communicating (PCoA) arteries.
 A–C, Anteroposterior (A) and lateral serial and stereoscopic (B and C), carotid angiograms. AChA (solid arrowheads) arises from supraclinoid portion of internal carotid artery just proximal to terminal bifurcation, but distal to origin of PCoA (open arrowheads). Because PCoA is small, trunk of posterior cerebral artery (PCA) is only transiently opacified on initial arterial lateral view (B). On film 0.5 sec later (C), however, PCoA and its perforator (i.e., thalamotubular artery) remain filled (open arrowheads) without opacification of PCA trunk. Perforator is often only branch of PCoA visualized when PCoA is so rudimentary. A very thin uncus branch and medial plexal branch of AChA are seen. Striate arteries, medial and lateral from anterior and middle cerebral arteries, enter brain at anterior perforated substance (arrows) [9].

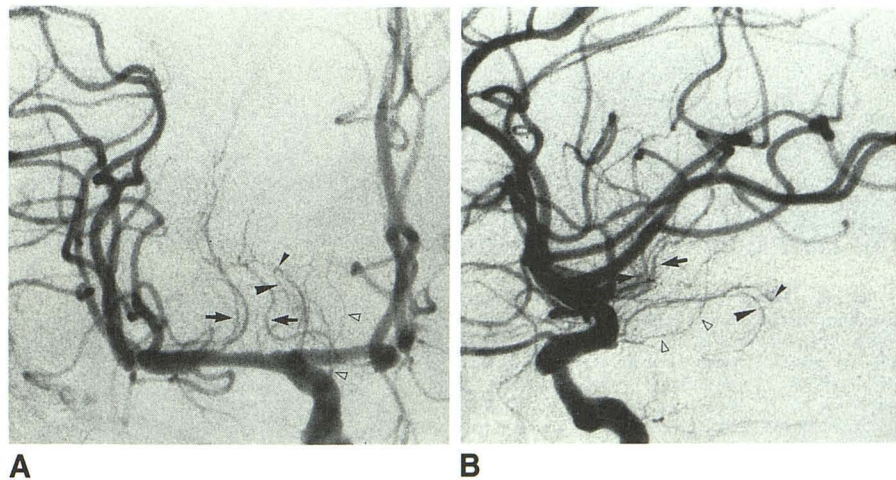


Fig. 3.—A and B, Hypoplasia of plexal segment of anterior choroidal artery (AChA). After uncus branch (large solid arrowheads), opacification of AChA ends at level of lateral geniculate body (small solid arrowheads) without visualizing plexal segment. Posterior communicating artery and its perforator or thalamotubular artery (open arrowheads), recurrent artery of Heubner, and lateral striate arteries (arrows) are noted. (Compare with Fig. 1.)

that it supplied a part of both PCA distributions in the medial temporal regions in addition to supplying the plexal branches (hyperplastic anomaly of the AChA, 0.5%).

Plexal segment.—Identification of the medial and lateral plexal branches was not difficult under stereoscopic observation, when both of them were opacified. When only one was opacified, the vessel was judged as a medial plexal branch or plexal stem artery, which is a posterior continuation of the cisternal segment along the attachment of the choroid plexus [3, 6]. When the PCoA was well developed and the PCA was opacified, it became difficult and sometimes impossible to distinguish the plexal segment (especially the lateral plexal branch) of the AChA from the lateral posterior choroidal artery of the PCA. The medial plexal branch could not be identified in 3.0%, while it was small (barely visible) in 8.4%, medium-sized (well visible but not large) in 44.8%, and large (extending superiorly beyond the posterior pole of the thala-

mus) in 43.8%. The lateral plexal branch could not be identified in 36.4%, was small or seen only as a faint blush in 45.4%, and was prominent in 18.2%. Either the medial or lateral plexal branch could not be identified in six (hypoplasia of the plexal segment of the AChA, 3.0%) (Fig. 3).

PCoA.—The PCoA was not opacified in 13.9% and was of very small size in 38.0%. When the PCoA was very small, the perforator from it (the thalamotubular artery [10]) was often the only branch of the PCoA visualized (Fig. 2). The PCoA was medium-sized, partially opacifying the PCA, in 31.0%, and large enough to opacify the PCA vessels well in 14.4% (fetal type or direct origin of the PCA).

Anomalies of the AChA

Hypoplasia of the plexal segment of the AChA.—The AChA was identified on all 216 control carotid angiograms. Opacification of the AChA ended at the level of the lateral geniculate

body (Fig. 2) without visualizing either the medial or lateral plexal branch (six [3%] of 216 carotid angiograms).

Hyperplastic anomalies of the AChA.—In each of the 25 hyperplastic anomalies, the artery arose from the supraclinoid portion of the ICA (a few millimeters above the origin of the PCoA) and took a course like the cisternal segment of the normal AChA, although some of the variations described below were seen in the course of the trunk of the anomalous artery. The choroidal branch was identified in all but one instance. The anomalous artery supplied varying parts of the PCA distribution. On the basis of the distribution area, the 25 examples of anomalous arteries were divided into four groups:

Type 1: Hypertrophic uncal branch, in which the uncal branch of the AChA looks more prominent than usual, prob-

ably covering the anterior temporal branch of the PCA (Fig. 4); this was found in five arteries (20%).

Type 2: Anomalous temporal artery, in which the vessel supplies most of the distribution area of the anterior and posterior temporal branches of the PCA. There were 15 arteries (60%) in this group, which were divided into three subtypes according to the course of the vessel: *type 2a*—in five arteries, the trunk of the anomalous vessel displayed the characteristic course of the uncal branch (Fig. 5); *type 2b*—in six arteries, the trunk took neither a characteristic course of the uncal branch nor a far lateral course (Fig. 6); and *type 2c*—in four arteries, the trunk took a far lateral course toward the inferior horn before reaching the PCA distribution (Fig. 7).

Type 3: Anomalous occipitoparietal artery, in which the anomalous vessel supplies the distribution area of the calcar-

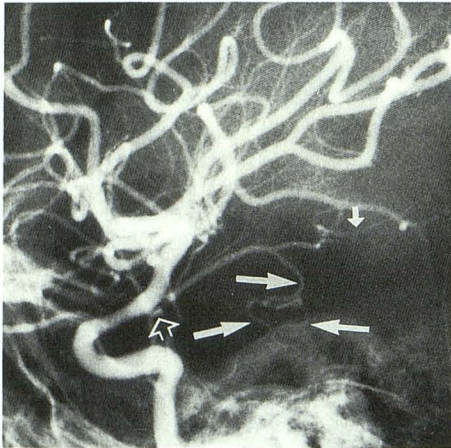


Fig. 4.—Hypertrophic uncal branch of anterior choroidal artery (AChA) (type 1). Uncal branch of AChA is so prominent that it covers territory of anterior temporal artery of posterior cerebral artery (large straight arrows). Plexal branch (small straight arrow) of the AChA is seen. Infundibular dilatation at origin of posterior communicating artery (open arrow) is present.

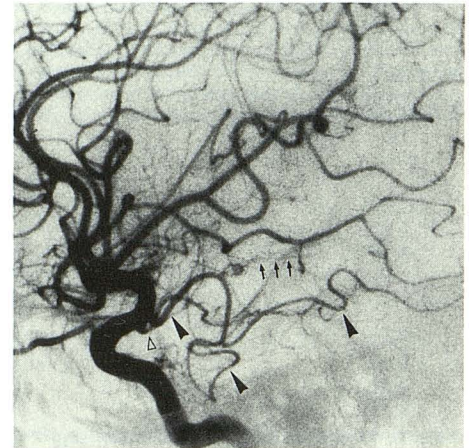


Fig. 5.—Anomalous temporal artery (type 2a). Anomalous artery (solid arrowheads) supplies territory of anterior and posterior temporal arteries of posterior cerebral artery. Note similarity in course of stem of anomalous vessel to usual form of uncal branch of anterior choroidal artery in Fig. 1. Also note plexal branch (arrows) and posterior communicating artery (open arrowhead).



A

B

Fig. 6.—Anomalous temporal artery (type 2b). A and B, Right carotid angiograms, antero-posterior (A) and lateral (B) views. Anomalous artery (arrowheads) distributes to medial temporal region, but is distinctly different from trunk of posterior cerebral artery (PCA). Saccular aneurysm (arrows) arises at origin of anomalous artery. This was the cause of this patient's subarachnoid hemorrhage, and the aneurysm was clipped. Preoperative vertebral angiography (not shown) did not opacify anomalous vessel. Postoperative carotid angiogram showed complete occlusion of vessel at its origin, in addition to disappearance of aneurysm. Vertebral angiography revealed anomalous artery opacified, probably via some potential anastomoses between vessel and posterior communicating artery-PCA system, explaining why patient did not have any serious symptoms postoperatively.

Fig. 7.—Anomalous temporal artery (type 3).
A and B, Anomalous artery courses far laterally deep in choroid fissure or into inferior horn of lateral ventricle (*arrowheads*), where branching plexal branch (*arrows*) reaches medial temporal region.

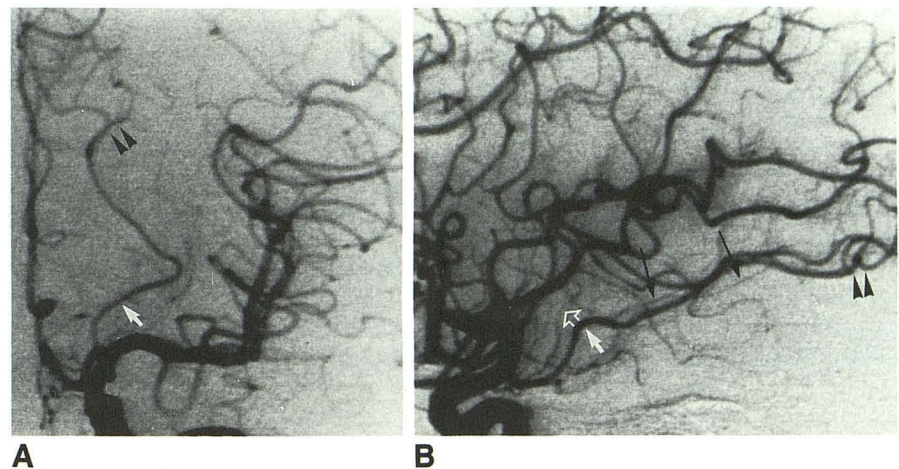
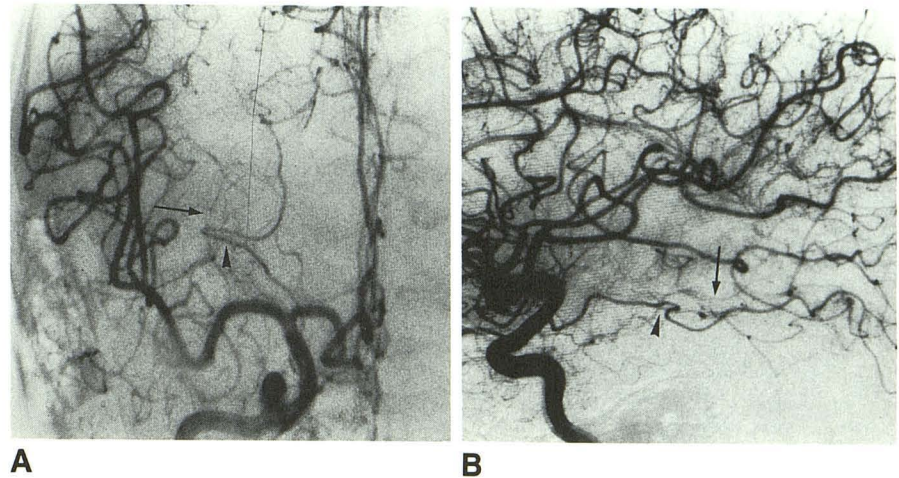
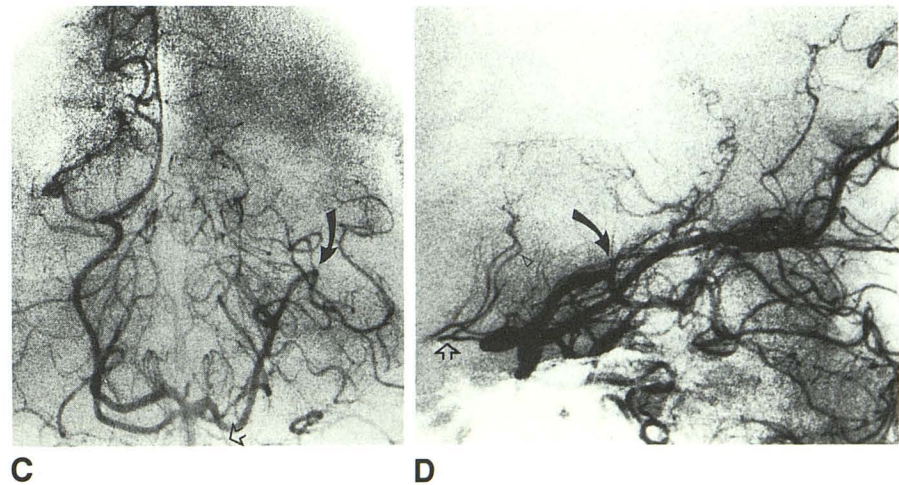


Fig. 8.—Anomalous occipitoparietal artery (type 3).

A and B, Carotid angiograms, anteroposterior (**A**) and lateral (**B**) views.

C and D, Vertebral angiograms, anteroposterior (**C**) and lateral (**D**) views.

Anomalous artery (*white arrows*) from internal carotid artery supplies plexal branch (*straight black arrows*) and calcarine and parietooccipital arteries of posterior cerebral artery (PCA) distribution (*arrowheads*). On vertebral angiography, left PCA gives off just anterior and posterior temporal branches, mimicking obstructed vessel (*curved arrows*). Calcarine and parietooccipital arteries are only faintly opacified from left PCA, probably via small anastomoses between anomalous vessel and left PCA proper. PCoA and its perforator are filled by reflux of contrast material on both sides (*open arrows*).



ine and occipitoparietal arteries (Fig. 8); this was found in two arteries (8%).

Type 4: Anomalous temporooccipitoparietal artery, in which the original PCA is almost totally replaced by the anomalous artery (Fig. 9); this was found in three arteries (12%). A case erroneously suggesting the absence of the normal AChA (Fig. 10) and another case of an erroneously appearing anomalous

origin of the vessel from the PCoA (Fig. 11) are considered to belong to this group. In the case erroneously suggesting the absence of the normal AChA, a hyperplastic AChA closely resembling the large fetal PCoA-PCA system was seen (Fig. 10).

In none of the cases of hyperplastic anomalies were additional branches looking like a normal AChA identifiable from

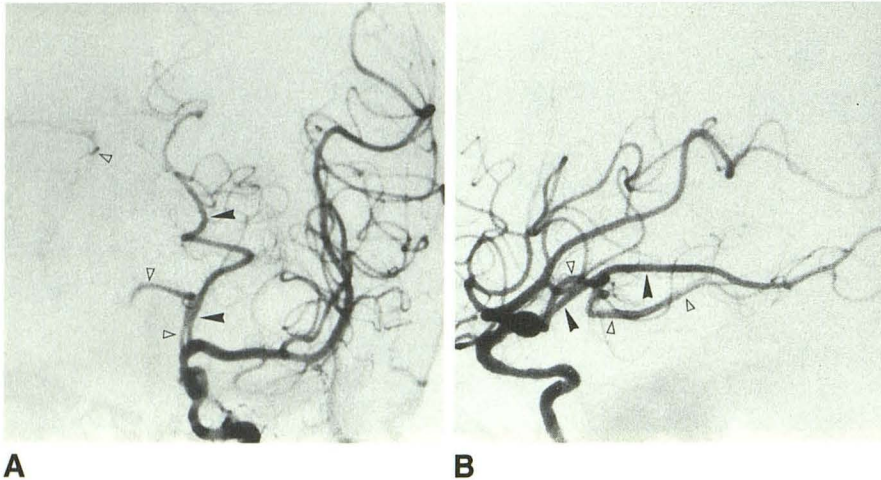


Fig. 9.—Anomalous temporooccipitoparietal artery (type 4).

A and B, Left carotid angiograms, anteroposterior (A) and lateral (B) views. Injection pressure makes proximally arising vessel, that is, posterior communicating artery (PCoA) and pre-communicating segment of posterior cerebral artery (PCA) (open arrowheads), transiently opacified. This in turn faintly filled contralateral PCA on earlier images (open arrowheads). Distally arising vessel is anomalous artery (solid arrowheads), although it looks like PCoA-PCA system of direct origin, supplying all of PCA distribution on this side. There may be a small communicating channel between PCoA and anomalous vessel at usual site of PCoA-PCA junction.

the supraclinoid part of the ICA. The PCoA was well developed on the side of the anomalous artery (Fig. 12) in three cases, was hypoplastic with only transient filling of a thin PCoA and/or its perforating artery in nine cases, and could not be identified at all in nine cases.

Vertebral angiograms were obtained in 13 patients, including the two patients with paired anomalous arteries. The PCA branches that were opacified from the anomalous artery on carotid angiograms (anterior and posterior temporal branches in most cases) were not filled or were only faintly opacified on vertebral angiograms. Thus, the PCA proper on the same side as the anomalous vessel appeared hypoplastic in most cases (Fig. 13), looking like it was being affected by obstructive changes, especially in type 3 (Fig. 8). There seemed to be some persistent anastomotic channels between the anomalous vessel and the PCA system in some cases (Fig. 6). In two patients, the anomalous artery was paired. One of these had aneurysms bilaterally at the origin of the anomalous arteries; one of the aneurysms, on the right, had ruptured (Fig. 6). Four aneurysms were found in four other patients, but none of them was associated with the anomalous artery. One of these patients died owing to aggravation of a vasospasm; the anomalous artery was confirmed at autopsy (Fig. 14).

Discussion

The AChA was identified on all of the 216 carotid angiograms. The artery was present in each of 778 hemispheres studied by Otomo [11] and also in each of 50 hemispheres studied by Rhoton et al. [12, 13], although its absence was reported in one (1.7%) of 60 hemispheres studied by Carpenter et al. [2]. Goldberg [3] also stated that the AChA may be very small or unrecognized if its territory is fed by branches of the PCoA or PCA. However, there has been no angiographic or photographic demonstration of absence of the AChA in the literature. Only one angiogram in our study (Fig. 10), which was not one of the 216 control carotid angiograms but was from those patients examined over an 8-year period, seemed to demonstrate absence of the AChA. However,

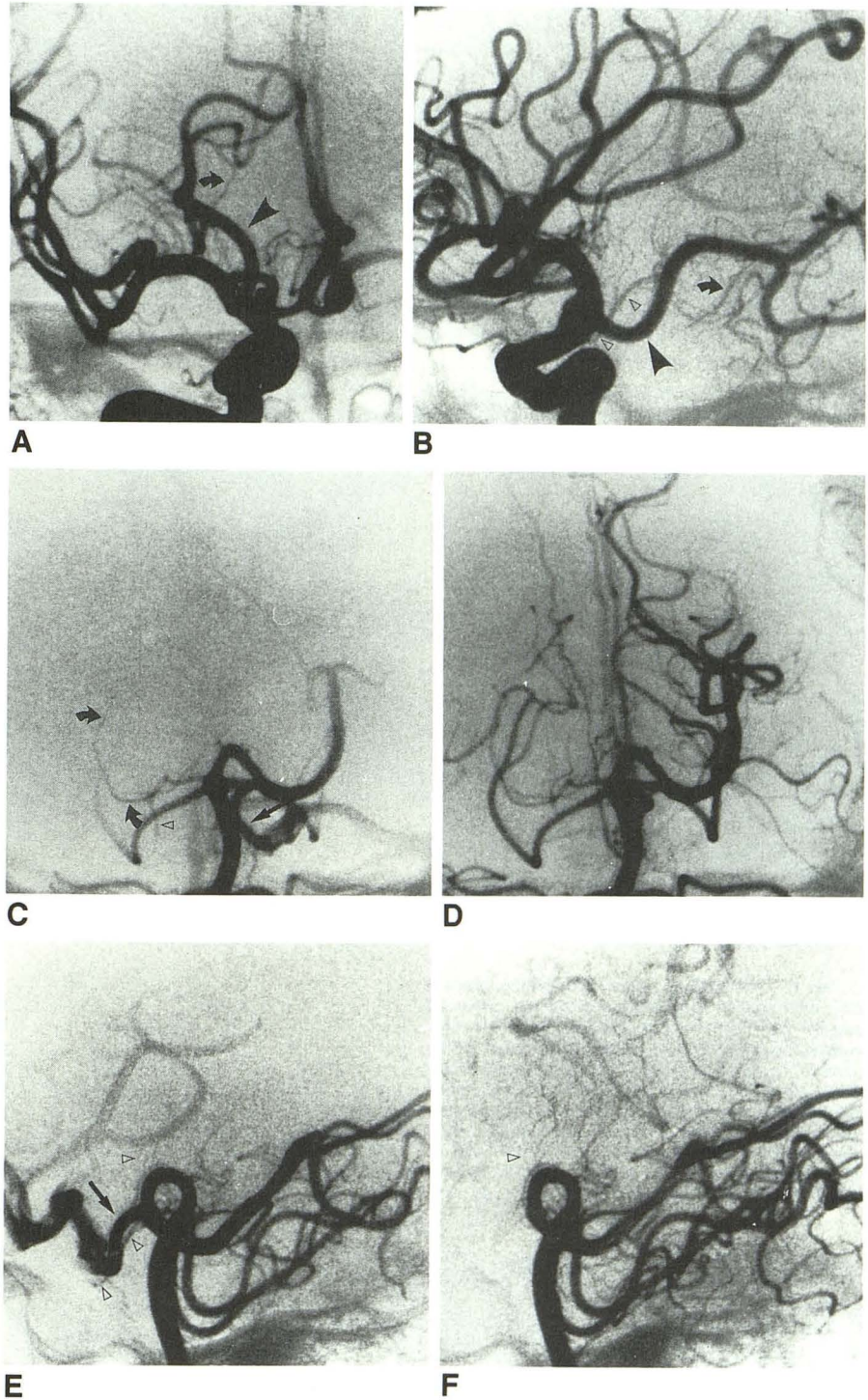
careful observation of the angiogram tells us that a tiny vessel from the supraclinoid ICA actually should be the original PCoA and its perforator, and the "PCoA-PCA system" is in reality a form of hyperplasia of the AChA. An anastomotic channel between the PCoA or the proximal PCA and the AChA persists as a main pathway, and the hyperplastic AChA totally supplies the PCA distribution. Thus, the absence of the AChA described in the literature may also be explained by a developmental mechanism for hyperplasia of the AChA.

There may be a racial variation in the origin of the AChA [3]. In anatomic studies in the United States, in which 60 hemispheres [2] and 44 hemispheres [14] were examined, 76.6% and 85% of the AChAs, respectively, arose from the ICA, whereas 11.7% and 8% arose from the middle cerebral and 6.7% and 0% from the PCoA. The artery originated from the ICA in 49 (98%) and from the PCoA in one (2%) of 50 hemispheres examined by Rhoton et al. [12]. In a larger Japanese series by Otomo [11], 99.2% of the AChAs arose from the ICA, 0.4% from the PCoA, and 0.4% from the junction of the ICA and the PCoA, but none from the middle cerebral artery. In two angiographic studies by Sjögren [14] and Morello and Cooper [15], the AChA was seen to originate from the PCoA in 3% and 8% of the angiograms, respectively, and from the middle cerebral artery in 2% and 1%. In Figure 11, the carotid angiogram seemingly suggests that the AChA abnormally originated from the PCoA. Anomalous origin of the AChA from the PCoA might safely be regarded as essentially the same anomaly as absence of the AChA; in both, the original PCoA has attenuated to a tiny channel, while the original AChA has enlarged to supply the PCA distribution. The difference between them in this study was simply in the site and mode of origination of the choroidal branch from the enlarged trunk of the AChA; the choroidal branch arose distally and coursed nearly parallel to the trunk of the PCoA-PCA in the case, erroneously suggesting the absence of the AChA, while the choroidal branch arose more proximally and more perpendicularly to the trunk and appeared to be a discrete vessel in the case of "anomalous origin" (compare Figs. 10 and 11). It is uncertain, however, whether the abnormal origination from the PCoA and absence of this vessel reported in the literature can also be explained by the same

Fig. 10.—Anomalous temporooccipitoparietal artery erroneously suggesting absence of anterior choroidal artery (AChA) (type 4).

A and B, Right carotid angiograms, anteroposterior (A) and lateral (B) views. Although no AChA seems to arise from supraclinoid internal carotid artery (ICA), a large vessel that seemingly looks like fetal type of posterior cerebral artery (PCA) is a hyperplastic anomaly of AChA (solid arrowheads). Just proximal to origin of this vessel, a tiny vessel is seen to arise posterosuperiorly from supraclinoid ICA, which is a hypoplastic PCoA that gives rise to its perforator (open arrowheads). Hypoplastic PCA trunk on right (curved arrows) is opacified via this small posterior communicating artery (PCoA).

C-F, Left vertebral angiograms, anteroposterior (C and D) and lateral (E and F) views, all of which are serial and stereoscopic. Small PCoA on right (open arrowheads) is transiently opacified from extremely attenuated right PCA trunk (curved arrows). This small PCoA is identical to tiny vessel from supraclinoid ICA (open arrowheads, A and B). Thus, the large vessel, which erroneously might be thought of as fetal type of PCA (solid arrowheads, A and B), should be considered an extremely hyperplastic form of AChA that almost totally replaced PCoA-PCA system on this side. PCoA is seen on contralateral side (arrows).



developmental mechanism, as we were unable to find any angiographic or photographic demonstrations of those cases.

The diameter of the stem of the AChA at 1 cm from the point of origin in our study ranged from 0.38 to 2.0 (average, 0.75 mm). The maximal diameter (2.0 mm) was noted in a hyperplastic anomaly of this artery. It is natural that the average value was smaller than the outer diameter of the artery, which has been reported [12] in studies using anatomic dissection in the range of 0.7–2.0 mm (average, 1.2 mm).

Hypoplasia of the plexal segment of the AChA, noted occasionally on the control angiograms, may be explained by the phylogeny of this artery. According to a phylogenetic study of forebrain arteries by Abbie [16], the AChA begins as a small vessel called the inferior cerebral artery of Dendy in the sphenodon, a phylogenically lower reptile (Fig. 15). The small vessel runs posteriorly along the optic tract and anastomoses with the caudal division (predecessor of the PCA) in the crocodile, a higher species of reptile. The vessel has no

choroidal branches, and thus is not a true choroidal artery at this stage. Because both the lateral ventricle and choroid fissure assume an arcuate form in mammals, the posterior part of the AChA comes to lie alongside the anteroinferior end of the elongated choroid fissure and acquires some choroidal branches from the PCA, thus completing the AChA in the true sense. Hypoplasia of the plexal segment of the AChA, which can be visualized to the level of the lateral geniculate body (Fig. 3), may represent an evolutionary variant in which the artery ceased to acquire choroidal branches, remaining in the reptilian stage.

Some authors have suggested that the anomalous artery is a hyperplastic variant of the AChA [1, 2, 5]. Takahashi et al. [8] reported seven such arteries (1.8%) in 640 bilateral carotid angiograms of 320 patients, and suggested that it should be considered as a hyperplastic variant of the AChA.

They listed the following angiographic features as the reasons for this conclusion: (1) the course of the proximal part of this artery is the same as that of the cisternal part of the AChA; (2) this artery gives rise to the choroidal branch, which supplies the choroid plexus of the trigone; (3) there is no other artery from the supraclinoid part of the ICA; and (4) the temporal and/or calcarine branches of the PCA are absent or hypoplastic. These four conditions were present in all but one case in our study; that is, in one case, no choroidal vessel was identified on angiography. Absence of a choroidal branch would not contradict the postulate, however, because the plexal segment of the AChA may normally be absent or hypoplastic, as demonstrated in this study. Thus, our results also strongly support the postulate that the anomalous artery is a hyperplastic variant of the AChA. However, there could be a wide spectrum of such arteries, which may represent

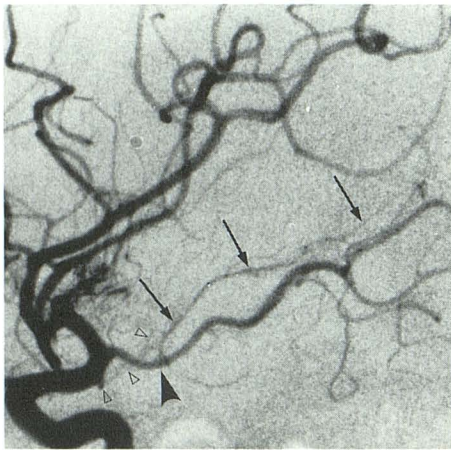


Fig. 11.—Anomalous temporooccipitoparietal artery erroneously suggesting anomalous origin of anterior choroidal artery (AChA) from posterior communicating artery (PCoA) (type 4). In this case, AChA (arrows) seems to originate from PCoA (solid arrowhead). However, there is a tiny vessel with an infundibular dilatation originating from carotid (open arrowheads) that actually is PCoA, which gives rise to a perforator. The main vessel (solid arrowhead) is the hyperplastic anomaly of AChA and its upward branch (arrows) is the plexal branch.

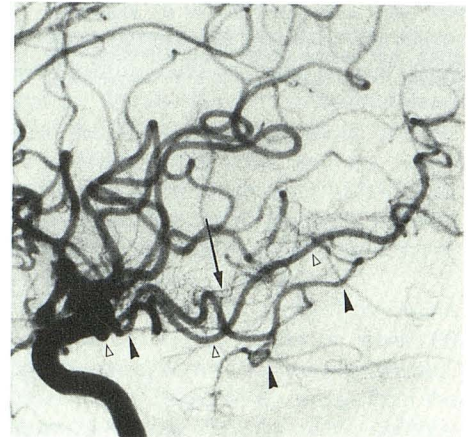


Fig. 12.—Well-developed posterior communicating artery (PCoA) on same side of anomalous artery (type 2c). Anomalous artery (solid arrowheads) is seen to give off plexal branch (arrow) and temporal branches, while well-developed PCoA-posterior cerebral artery system (open arrowheads) gives off parietooccipital and calcarine arteries.

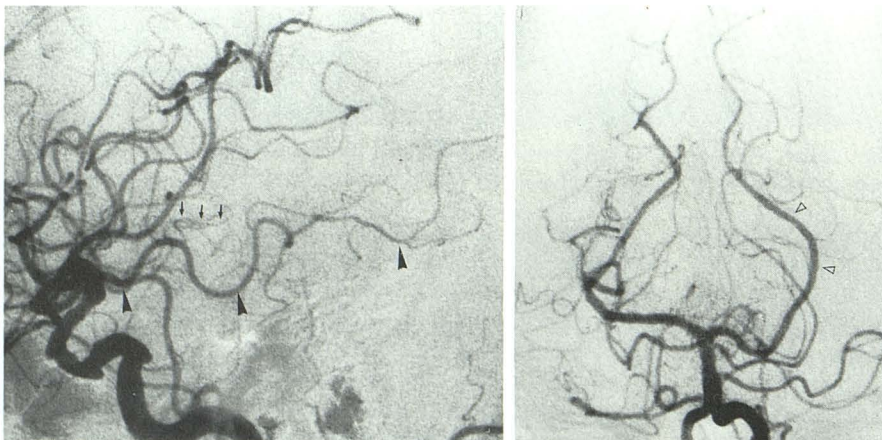


Fig. 13.—Hypoplastic posterior cerebral artery (PCA) not giving off temporal arteries (open arrowheads) on same side of anomalous artery (solid arrowheads) (type 2b) supplying a plexal branch (arrows) and temporal branches in corresponding region of PCA.

A, Left carotid angiogram, lateral view.
B, Left vertebral angiogram, anteroposterior view.

A

B

Fig. 14.—Anomalous artery (type 2c), confirmed at autopsy.

A and B, Right carotid angiograms, anteroposterior (A) and lateral (B) views. Anomalous temporal artery arises from supraclinoid internal carotid artery (ICA) and courses far laterally (large arrowheads) before going to medial posterior temporal region. Plexal branch (small arrows) and uncal branch (small arrowheads) are noted. A saccular aneurysm (large arrows) arises at anterior communicating artery with early findings of vasospasm around it. The patient died owing to aggravation of vasospasm; an autopsy was performed.

C and D, Axial sections of autopsied brain. D is superiorly contiguous to C. In C, just distal to origin of posterior communicating artery (white arrowhead), anomalous artery (straight arrow) is seen to originate. Posterior cerebral artery proper is artificially amputated (curved arrows), and it is disclosed that anomalous vessel (smaller straight arrows) goes laterally into choid fissure and gives off small twigs to choroid plexus of inferior horn of lateral ventricle (large arrow) and then distributes to medial posterior temporal region. BA = basilar artery; Ch = choroid plexus; ICA = internal carotid artery; M = midbrain; P = pons.

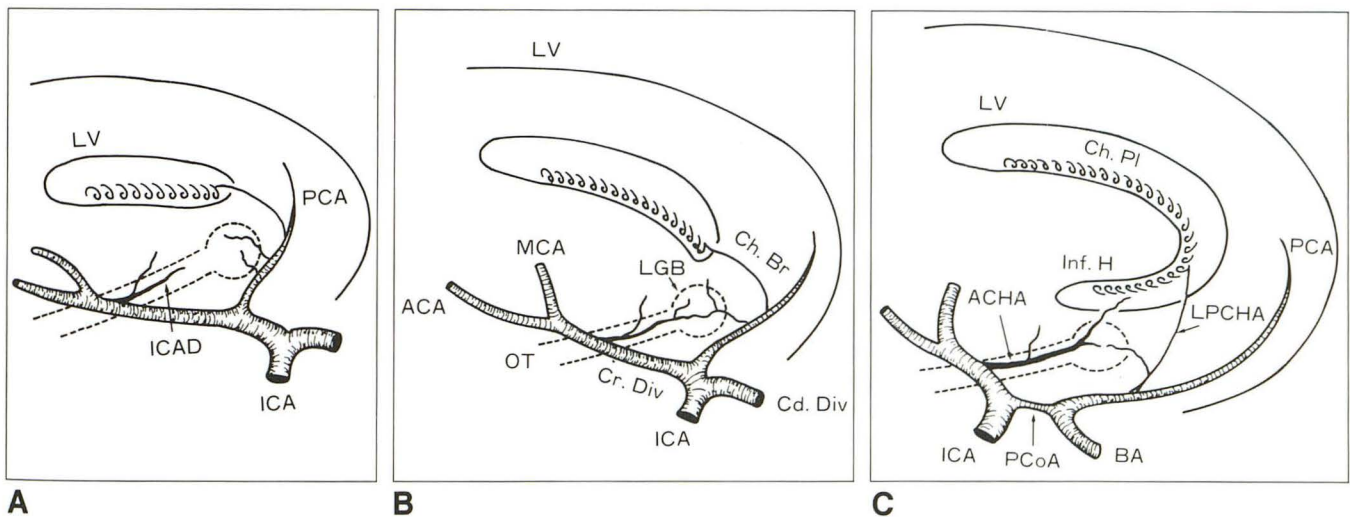
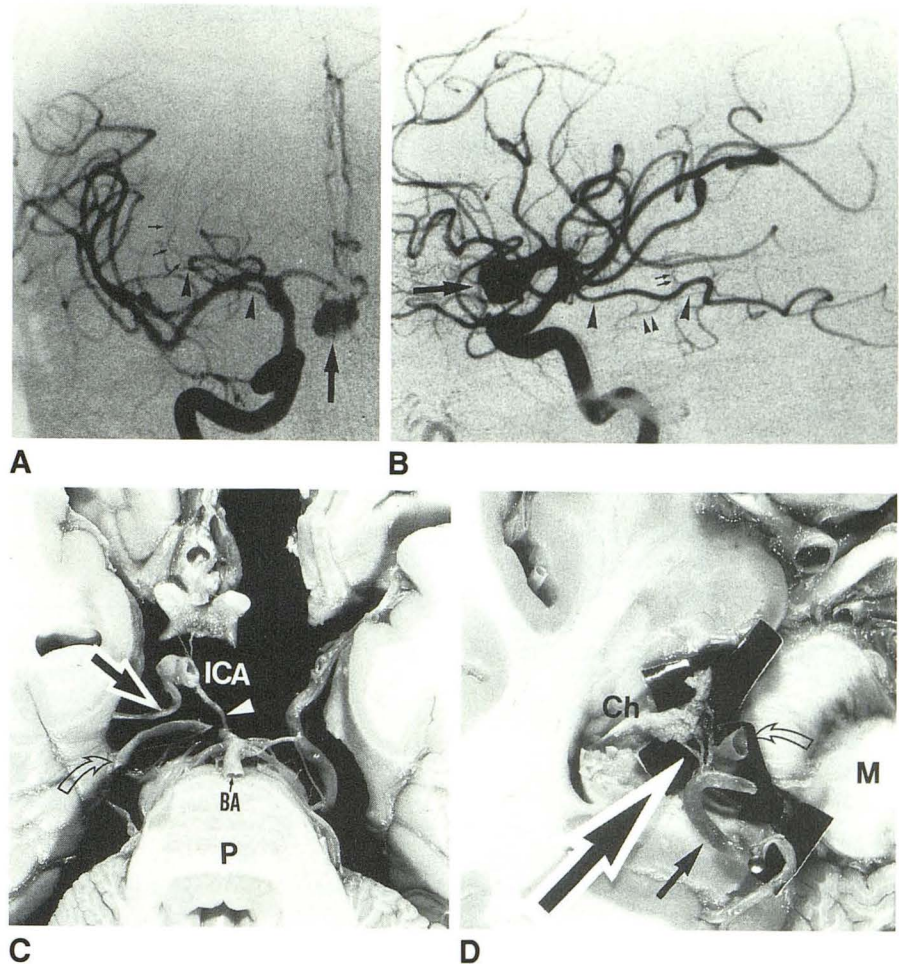


Fig. 15.—Schematic drawings show phylogenetic development of anterior choroidal artery (AChA). See key for abbreviations. (Adapted from Abbie [16].)
A, Sphenodon (phylogenically lower reptile). AChA begins as small vessel called inferior cerebral artery of Dendy, which arises from cranial division of internal carotid artery, courses along optic tract, and nourishes posterior part of optic tract, a part of amygdala and paleostriatum.

B, Crocodile (higher species of reptile). Inferior cerebral artery of Dendy runs more posteriorly along optic tract and terminates in caudal division. This vessel is predecessor of AChA, although it has acquired no choroidal branches yet. It must be remembered that lateral ventricle has no inferior horn in reptiles—the choroid fissure is limited to the region of the foramen of Monro—and the choroidal branches of the posterior cerebral artery pass into this region and suffice for its requirements.

C, Marsupial (a mammal). Both lateral ventricle and choroid fissure assume arcuate form with expansion and rotation of cerebrum. Thus, posterior part of AChA comes to lie alongside anteroinferior end of elongated choroid fissure, and it acquires some choroidal branches from posterior cerebral artery to anteroinferior part of choroid plexus, thus completing AChA. In ascending mammalian scale, in keeping with increasing growth of cerebrum and lateral ventricle and growing functional demands of enlarging choroid plexus, contribution of AChA to choroid plexus becomes greater as cerebrum progressively enlarges.

Key to Abbreviations Used in Figures 15–17

ACA	anterior cerebral artery
ACHA	anterior choroidal artery
Ant.TA	anterior temporal artery of the posterior cerebral artery
BA	basilar artery
Cd.Div	caudal division of the internal carotid artery
Ch.Br	choroidal branch
Ch.PI	choroid plexus
Cr.Div	cranial division of the internal carotid artery
ICA	internal carotid artery
ICAD	inferior cerebral artery of Dandy
Inf.H	inferior horn of the lateral ventricle
LGB	lateral geniculate body
LPCHA	lateral posterior choroidal artery
LV	lateral ventricle
MCA	middle cerebral artery
OC	optic chiasm
ON	optic nerve
OT	optic tract
PCA	posterior cerebral artery
PCoA	posterior communicating artery
PI.Br	plexal segment of the anterior choroidal artery
Post.TA	posterior temporal artery of the posterior cerebral artery
Temp.Br	temporal branch of the posterior cerebral artery
Unc.Br	uncal branch of the anterior choroidal artery

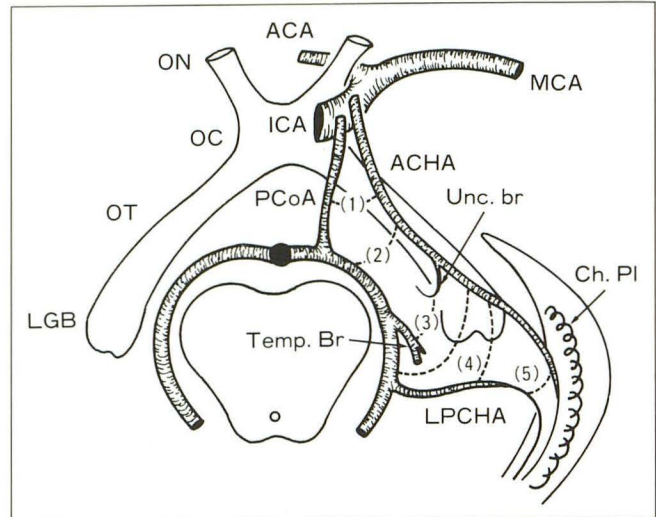


Fig. 16.—Anastomoses of anterior choroidal artery (ACHA) with posterior communicating artery (PCoA)-posterior cerebral artery (PCA) system. See key for abbreviations. ACHA has anastomotic channels (1) over optic tract with branches from PCoA; (2) over cerebral peduncle with proximal PCA; (3) over piriform cortex (uncal branches) with PCA branches (temporal and hippocampal branches); (4) over and around lateral geniculate body with PCA branches, including lateral posterior choroidal artery; and, finally, (5) in choroid plexus with posterior choroidal branches.

transitional forms between the normal ACHA and extreme hyperplasia and might totally replace the PCoA-PCA system on the same side (type 4).

In comparative anatomic studies, Shellshear [17] classified the cerebral arteries into two major categories: the end arteries and the major trunks. The end arteries, which are the first to develop during the embryonic stage, supply a constant functional territory, while the major trunks, which lead to the end arteries, have variations in their origin and course. The variations in the major trunks develop from anastomoses between them that should have derived from the capillary plexus over the surface of the brain, formed in Streeter's second stage of fetal development in the human cerebral vascular system [18]. Which anastomosis persists as a main pathway and what variation occurs may depend on the evolutionary direction of growth of an organ and on hemodynamic factors in the fetal development in individuals [16].

The normal ACHA also has potential anastomoses with its neighboring arteries, especially with the PCoA and PCA (Fig. 16) [2, 6, 19, 20]. Hyperplasia of the ACHA seems to represent a situation in which one of those anastomoses remains and enlarges as a main pathway of the artery, while a segment of

the PCA just proximal to the anastomosis eventually attenuates. Depending on which channel remains, different phenotypes of the hyperplasia may result (Fig. 17). Of the groups we have classified in this study, the hypertrophic uncal branch of type 1 may have maintained a persistent anastomosis between the uncal branch of the ACHA and the anterior temporal branch of the PCA. The anomalous temporal artery of type 2a may have maintained a communication between the uncal branch of the ACHA and a common stem of the temporal branches of the PCA, while type 2b may have preserved a communication somewhere in the surface area of the medial temporal region and type 2c may have maintained a communication deep in the choroid fissure at the inferior horn of the lateral ventricle. The anomalous occipitoparietal artery of type 3 may have maintained a persistent anastomosis with the PCA distal to branching of its temporal arteries. The anomalous temporooccipitoparietal artery of type 4 may represent a situation in which a persistent communication has remained at the level of the PCoA or at the anterior ambient segment of the PCA, resulting in a condition in which the PCA is almost totally replaced from its proximal part by the ACHA, including two cases in which the anomalous origin of the ACHA from the PCoA and total absence of the ACHA were erroneously suggested.

In conclusion, the normal angiographic anatomy and variations of the ACHA are described. Hypoplasia of the plexal segment of the ACHA was found occasionally. This may represent an evolutionary variation in which the artery ceased to acquire choroidal branches, thus remaining in the reptilian stage. Twenty-five examples of hyperplastic anomalies of the ACHA were also reviewed and were classified into four subtypes on the basis of their course and distribution area. The phenotypic variants of hyperplasia of the ACHA may represent

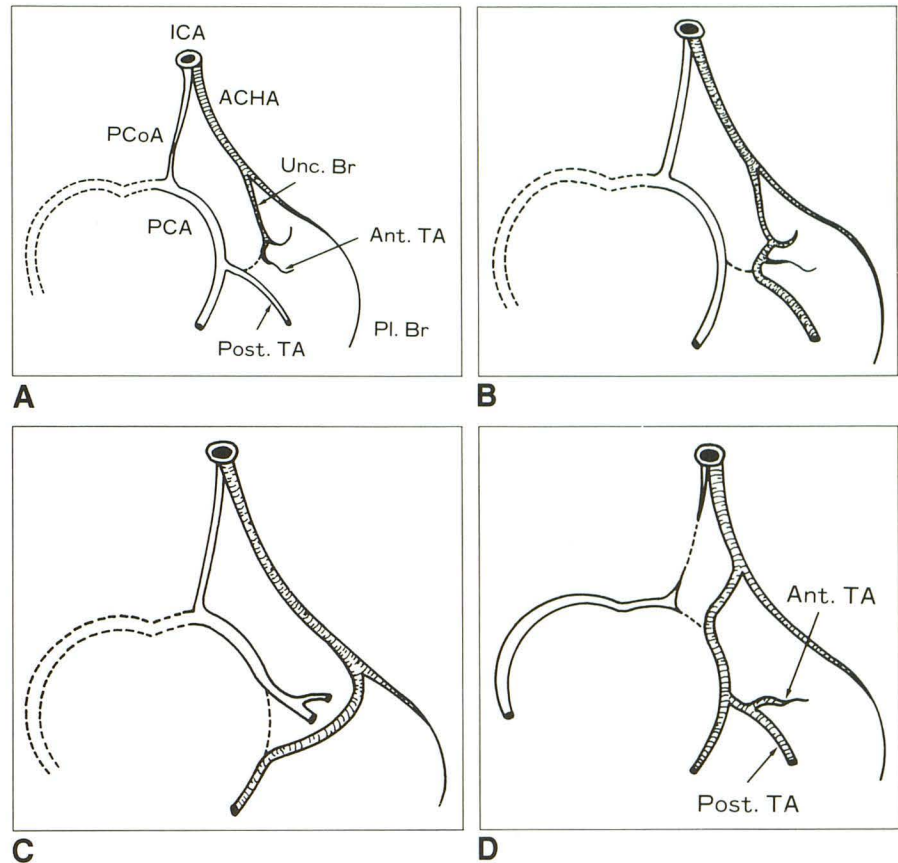
Fig. 17.—Diagrammatic representation of phenotypic variants of hyperplasia of anterior choroidal artery (AChA). See key for abbreviations.

A, Type 1: Hypertrophic uncal branch.—A persistent anastomosis between uncal branch and anterior temporal branch of posterior cerebral artery (PCA).

B, Type 2: Anomalous temporal artery.—A persistent anastomosis between AChA (uncal branch, and so forth) and common stem of temporal branches of PCA.

C, Type 3: Anomalous occipitoparietal artery.—A persistent anastomosis between AChA and PCA distal to branching of temporal arteries.

D, Type 4: Anomalous temporooccipitoparietal artery.—A persistent anastomosis at level of posterior communicating artery or at anterior ambient segment of PCA.



a situation in which the AChA adopted, as a main pathway, one of the anastomoses that the normal AChA has with the PCoA and PCA. Cases erroneously suggesting the absence and anomalous origin of the AChA from the PCoA seemed to represent essentially the same anomalies as hyperplasia of the AChA, in which the original AChA maintains well-developed communication with the proximal part of the PCoA-PCA system supplying all of the PCA vessels, while the original PCoA has attenuated into a minute vessel.

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