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High-Resolution Computed Tomography of the Basilar Artery: 2. Vertebrobasilar Dolichoectasia: Clinical-Pathologic Correlation and Review

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To better define the clinical significance of vertebrobasilar dolichoectasia, the clinical signs and symptoms and basilar artery parameters of diameter, height, and transverse position were evaluated in two groups of symptomatic patients. Ten patients had isolated involvement of the third, sixth, or seventh cranial nerves. The other 10 patients had multiple neurologic deficits including combinations of compressive cranial nerve deficits, both ischemic and compressive central nervous system deficits, and hydrocephalus. Although significant differences for mean basilar artery diameter and height exist between these two groups, the symptomatology and basilar artery parameters present as a spectrum. A symptomatic patient with a normal-caliber, but tortuous, basilar artery is more likely to have isolated cranial nerve involvement. Conversely, the patient with marked basilar artery dilatation (ectasia) is far more likely to present with multiple compressive or ischemic neurologic deficits. Conventional angiography in patients with dilated basilar arteries carries a significant risk for brainstem ischemia. Most authors agree that when vertebrobasilar dolichoectasia has been demonstrated by computed tomography, additional angiography, if required at all, should be performed by digital subtraction techniques.

Literally translated, *vertebrobasilar dolichoectasia* (*VBD*) means elongation (G. "dolichos") and distension (G. "ectasia") of the vertebrobasilar arteries. While this may, at first glance, appear to be a rather straightforward concept, attempts to extract reasonably consistent data from the literature are frustrating. Symptoms ascribed to elongated and/or distended vertebrobasilar vessels are presented under a variety of terms including megadolichobasilar artery or anomaly [1–10], aneurysmal malformation [11], dolichomegavertebralis anomaly [12], elongated basilar artery [13], megadolichovascular malformation [14], dolichoectasia [15–17], la dolicho-mega-basilare [18], ectasia [19–29], cirsoid aneurysms [30, 31], S-shaped aneurysms [32], aneurysms (including fusiform aneurysms) [33–40], wandering basilar artery [41], and tortuous basilar artery [42, 43].

Whether elongated, but normal-sized, vessels cause neurologic symptoms is controversial. Some authors maintain that it is unusual for an elongated vertebrobasilar vessel to extend to the cerebellopontine angle (CPA) and not cause cranial nerve palsies [42]. Others contend that no signs or symptoms should be attributed to elongated, normal-sized vertebrobasilar arteries [5]. The basic problem lies in the various criteria authors use to make the diagnosis of VBD.

Based on a previous study [44], we use the term *elongation* if the basilar artery, at any point throughout its course, lies lateral to the margin of the clivus or dorsum sellae or the artery bifurcates above the plane of the suprasellar cistern. *Ectasia* is diagnosed if the diameter of the basilar artery is greater than 4.5 mm [44].

We evaluated retrospectively 20 patients with a variety of clinical deficits ultimately attributed to VBD. We assessed the relation between clinical signs and symptoms and the diameter, height, and transverse position of the vertebrobasilar system. We also addressed the role of angiography in the evaluation of VBD and discuss the terminology currently used in reference to this condition.

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TABLE 1: **CT Findings in Patients with Vertebrobasilar Dolichoectasia**

Case No. (age, gender)	Hypertension	Cranial Nerve Involvement	Other	Diameter (in mm)	Height	Position
Isolated cranial nerve involvement:						
1 (69, M)	+	R-III (pupil-involving)	...	5.0	1	2R→0
2 (59, M)	−	L-IV (chronic ×12 years)		7.0	2	3L→2R
3 (57, F)	+	R-VI	Previous L-IV (now resolved)	3.4	1	2R→0
4 (60, F)	−	R-VII (HFS)	...	4.2	2	3R→2L
5 (65, M)	−	R-VII (HFS)	...	5.0	2	3R→2L
6 (72, M)	−	L-VII (HFS)	...	4.2	1	3L→0
7 (59, M)	+	L-VII (chronic palsy)	...	5.6	2	3L→1R
8 (60, M)	+	L-VII (HFS)	...	6.0	1	3L→1R
9 (71, M)	−	L-VII (HFS)	...	4.2	2	3L→0
10 (92, F)	−	R-VII (HFS)	...	4.6	1	3R→1L
Multiple deficits:						
11 (80, M)	−	R-V (TGN), R-VII (HFS)	...	5.8	2	3R→0
12 (64, M)	+	L-V, VII, VIII, IX, X, XII; R-V, VIII, IX, X	L 1½ syndrome, ataxic gait	14.0	3	3L→3R
13 (50, M)	+	L-V, VII, VIII, IX, X, XII; R-V, VII, VIII, IX, X, XII	"Locked-in" syndrome, R Horner syndrome	21.1	3	3L→2R
14 (50, M)	+	L-V; R-V, VI, VII, VIII, X	Wallenberg syndrome, R hemiplegia	10.0	2	3L→1R
15 (38, M)	+	L-VII	Syncopal episodes, visual blurring, dysarthria, L hemiparesis	10.0	3	3L→2R
16 (72, M)	+	R-V, VII, IX, X	Memory deficits, R hemihypesthesia, R hemiparesis, dysarthria, dysphonia	7.2	3	2R→2L
17 (82, M)	−	R-XII	Dizziness, lightheadedness, R hyperreflexia, tandem gait decreased	6.2	2	3L→0
18 (65, F)	+	R-VII (HFS)	Confusion, dementia, hydrocephalus	11.6	3	3L→2R
19 (70, M)	−	L-IX, X	R hemiparesis, aphasia, ataxia, dementia, incontinence	8.0	3	3L→0
20 (71, F)	+	None	Confusion, dementia, hydrocephalus, wide-based atactic gait	6.6	2	3L→2R

Note.—Normal basilar artery values: diameter, 3.17 mm (range, 1.9–4.5 mm); height, 0–1; position, 0–1. R = right; L = left; HFS = hemifacial spasm; TGN = trigeminal neuralgia. See Smoker et al. [44] for expanded definitions of height and position.

Materials and Methods

The radiographic studies and clinical histories of 20 patients with symptoms referable to VBD were reviewed. All patients initially presented to the neurology or neuroophthalmology services and were subsequently referred for neuroradiologic evaluation. There were 15 men and five women aged 38–92 years of age. All patients had high-resolution, contrast-enhanced CT examinations performed on a GE CT/T 8800, Picker 600, or Picker 1200 scanner immediately after drip infusion of 150 ml Conray 60. In addition, nine patients underwent either conventional or digital subtraction angiography (DSA). The diameter, height of the basilar bifurcation, and most lateral position of the vertebrobasilar arteries were assessed on CT scans according to parameters outlined in our companion study [44].

TABLE 2: **Mean Statistics for Basilar Artery Parameters in Vertebrobasilar Dolichoectasia**

	Mean	Standard Error	Standard Deviation
Isolated involvement:			
Diameter (mm)	4.93*	0.33	1.05
Height	1.50*	0.17	0.53
Position	2.80	0.13	0.42
Multiple deficits:			
Diameter (mm)	10.06*	1.49	4.71
Height	2.60*	0.16	0.52
Position	2.90	0.10	0.32

*$p < 0.01$.

Results

The findings in patients with isolated cranial nerve symptoms are presented in table 1. There was isolated involvement of the third (case 1), sixth (cases 2 and 3), or seventh (cases 4–10) cranial nerves. Of the patients with facial nerve involvement, there was one case of chronic facial palsy and six of hemifacial spasm (HFS). The left and right seventh cranial nerves were affected equally. Four of these 10 patients had a history of hypertension. The basilar artery diameter was normal in four patients and minimally increased in the other six. The level of the basilar bifurcation was normal in one-half of patients, while the transverse position was abnormal in every case.

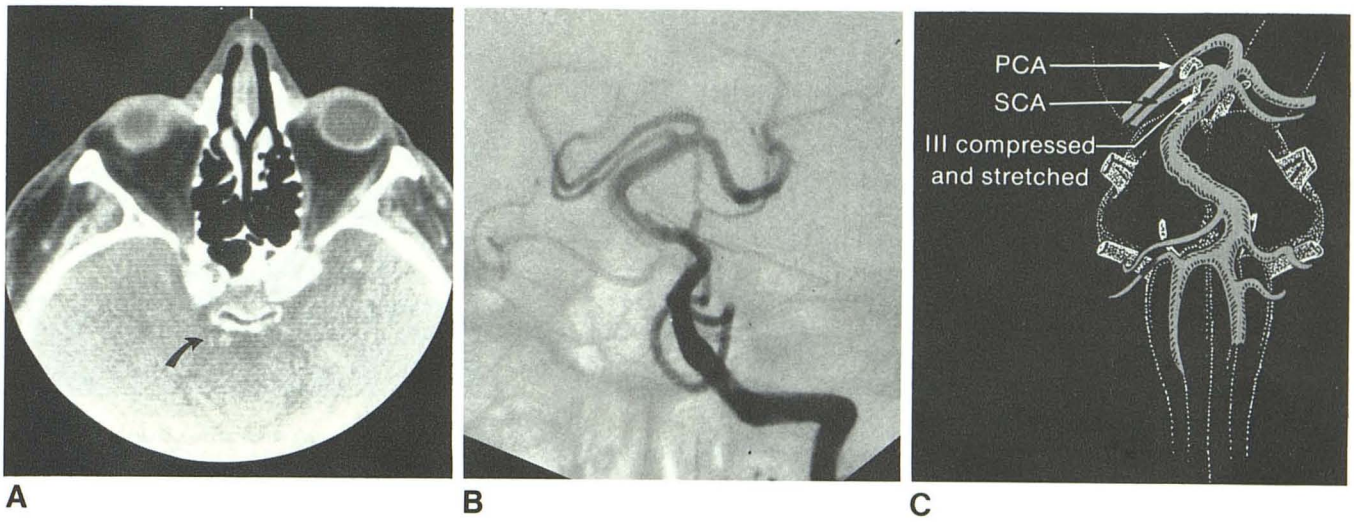


Fig. 1.—Case 1. **A**, CT scan. Minimally ectatic basilar artery positioned at right lateral margin of dorsum (*arrow*). **B**, Anteroposterior angiogram. Elongation and tortuosity of basilar artery resulting in elevation of right posterior cerebral and superior cerebellar arteries relative to left. **C**, Stretching of right third cranial nerve between elevated posterior cerebral (PCA) and superior cerebellar (SCA) arteries, in addition to possible direct compression from displaced distal basilar artery.

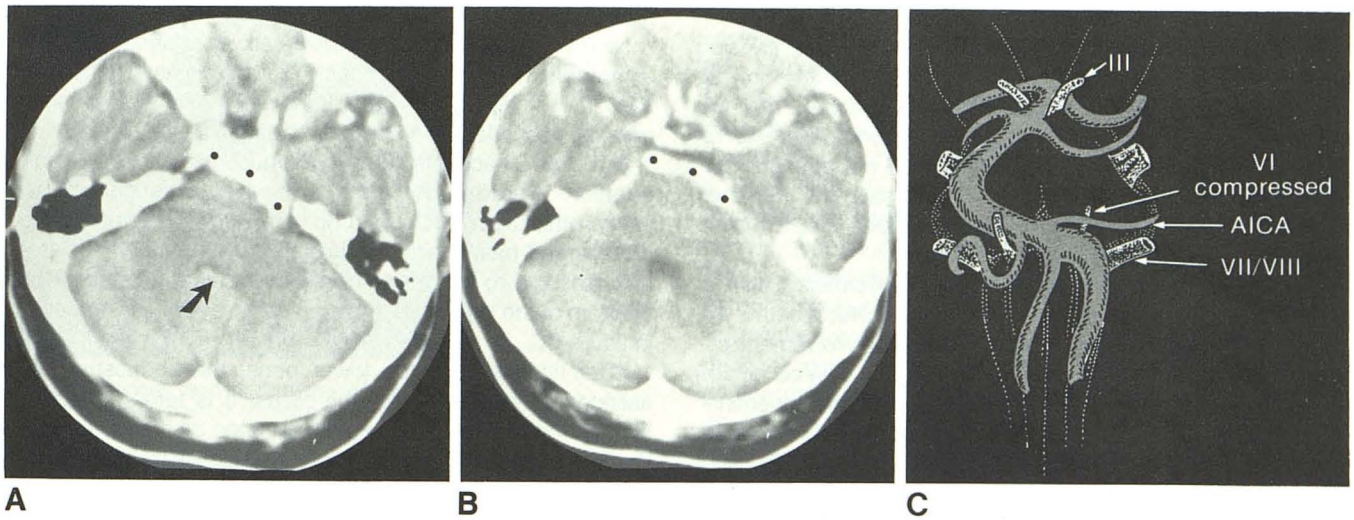


Fig. 2.—Case 2. **A** and **B**, Ectatic, tortuous basilar artery (*dots*) courses from left CPA cistern to right crural cistern. VBD has produced brainstem rotation, as evidenced by tilt of fourth ventricle (*arrow*). Also, there is vessel ectasia in anterior circulation. **C**, Direct compression of left sixth cranial nerve by distal left vertebral–proximal basilar artery. Brainstem rotation may have added element of stretch to etiology of chronic VI paresis.

Data for the other 10 patients are also presented in table 1. One patient had simultaneous right-sided trigeminal neuralgia (TGN) and HFS (case 11). The other nine patients had a wide variety of neurologic deficits. Three patients had symptoms referable to hydrocephalus (cases 18–20). The other six patients had brainstem or cerebellar signs and symptoms, most in association with multiple cranial nerve deficits. Bilateral cranial nerve involvement was encountered in three patients. Seven of this group of 10 had long-standing histories of hypertension. The diameter, height, and transverse position of the basilar artery were abnormal in every case. While the basilar artery was only minimally enlarged in two cases, it was markedly ectatic in the other eight cases.

The mean measurements for these two groups of patients are presented in table 2. The mean basilar artery diameter for the 10 patients with isolated cranial nerve involvement was 4.9 mm, just slightly above the upper limits of normal diameter (4.5 mm). However, the mean diameter of the patients with multiple deficits was 10.1 mm, twice the mean diameter of the isolated group. This represents a significant difference ($p < 0.01$). Likewise, a significant difference in height was present between these two groups. Position, however, was almost identical for the two groups (table 2).

Although the total number of symptomatic VBD patients in

this series is still small, it does represent the largest series yet reported. A number of trends are evident from analysis of our patients. A symptomatic patient with an isolated cranial nerve deficit is more likely to have an elongated but normal-sized, or minimally ectatic, basilar artery. If the artery is markedly ectatic, in addition to being elongated, the patient is much more likely to have multiple cranial nerve involvement, in association with a variety of central nervous system (CNS) deficits.

Discussion

The pathogenesis of VBD is controversial, but it is most likely caused by marked thinning or absence of the internal elastic lamina, thinning of the media secondary to smooth muscle atrophy, and hyalinization of connective tissue [17, 29, 45]. Since the elastic lamina is prominent in resisting the expansile effects of systolic blood pressure [46], prolonged systemic arterial hypertension may cause vessel dilatation and elongation [22]. Atherosclerotic changes are commonly superimposed [22, 29, 37].

Clinical Spectrum of VBD Symptomatology

Basilar artery ectasia was divided into three clinically distinct groups by Masson and Cambier [6]: (1) ischemic, (2) those caused by compression of cranial nerves, and (3) pseudotumoral. One must also add symptoms referable to hydrocephalus [13, 21, 23, 27]. We divided our patients into those with isolated and multiple compressive cranial nerve involvement, mixed ischemic and compressive CNS deficits, and those with hydrocephalus.

Isolated compressive cranial nerve involvement. From a literature review of 288 cases with sufficient clinical detail, we identified 74 cases of isolated cranial nerve involvement attributable to VBD. There were four cases of isolated oculomotor paresis [2, 40, 47], 27 cases of isolated trigeminal nerve deficits [3, 8, 22, 37, 47, 48], 42 cases of isolated facial nerve deficits (28 cases of HFS [3, 8, 12, 31, 42, 48–51] and 14 cases of facial paresis [8]), and one case of an isolated vestibulocochlear nerve paresis [43]. Ten of our patients had isolated cranial nerve deficits (table 1).

Oculomotor involvement by VBD has been documented in 32 reported cases [1, 2, 8, 17, 35, 39, 40, 47, 52]. While third-nerve involvement is not uncommon, it can hardly be regarded as pathognomonic, as suggested by Boeri and Passerini [2]. Four reported cases had isolated oculomotor involvement: Two were pupil-involving [47] and one was pupil-sparing [40]. Pupil involvement was not stated in case 8 reported by Boeri and Passerini [2].

Our case 1 had isolated, pupil-involving right third-nerve paresis. CT demonstrated a slightly enlarged, abnormally positioned basilar artery, coursing lateral to the margin of the dorsum (fig. 1A). Angiography demonstrated a tortuous basilar artery with elevated right posterior cerebral and superior cerebellar arteries (fig. 1B). After it emerges from the medial aspect of the cerebral peduncle, the oculomotor nerve crosses the interpeduncular cistern and courses between the posterior cerebral and superior cerebellar vessels [53]. We believe that elevation of these vessels on the right trapped and stretched the third nerve. A proposed mechanism of neural compression is presented in figure 1C.

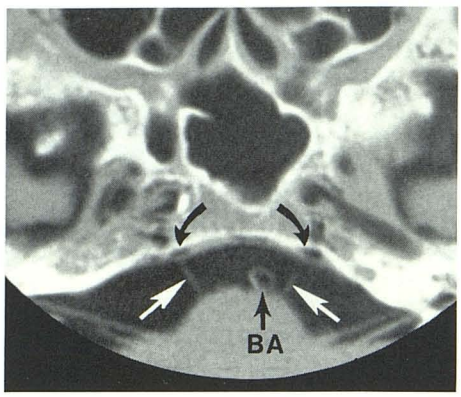


Fig. 3.—Cadaver air study shows course of abducens nerves (*straight arrows*) from origin at low pons, coursing anteriorly to enter Dorello canal (*curved arrows*). Note the proximity of basilar artery (BA), which is slightly paramedian in position, to left VI nerve.

Nine cases of *abducens involvement*, combined with other symptoms, have been previously reported in association with VBD [1, 17, 19, 26, 29, 36, 39]; however, isolated abducens paresis, secondary to VBD, has not been reported.

Our case 2 had an isolated left abducens palsy of 12 years' duration. Despite long-term extensive investigations, no cause other than VBD could be identified. CT demonstrated an enlarged basilar artery originating in the left CPA cistern and crossing to the right crural cistern (figs. 2A and 2B). A diagram of the proposed neurovascular relations is presented in figure 2C.

After it exits from the lower pons, the abducens nerve ascends intradurally along the clivus. It is tethered when it pierces the dura and it is again restricted as it passes under the Gruber ligament to enter the Dorello canal (fig. 3). Enlarged or displaced vessels may compress the nerve in the subarachnoid space or the nerve may be stretched secondary to brainstem rotation, as in our patient. Very large vessels may erode into and groove the pons, compressing nerves at their origins, as in the autopsy case detailed by Sacks and Lindenberg [17].

The *facial nerve* is the cranial nerve most often affected by VBD, in isolation or in combination with other cranial nerves. In our seven cases of isolated seventh nerve involvement, the basilar artery was normal in diameter in three cases and minimally enlarged in the other four (table 1). CT findings were consistently similar. In each case the basilar artery arose in the CPA cistern on the side of the affected facial nerve (fig. 4). It then coursed to a normal position in five patients or to the contralateral crural cistern in the other two.

Since our initial interest in VBD, we have evaluated 22 patients with isolated HFS. In all cases VBD was demonstrated by CT. Because of this high incidence, additional comments regarding HFS seem warranted.

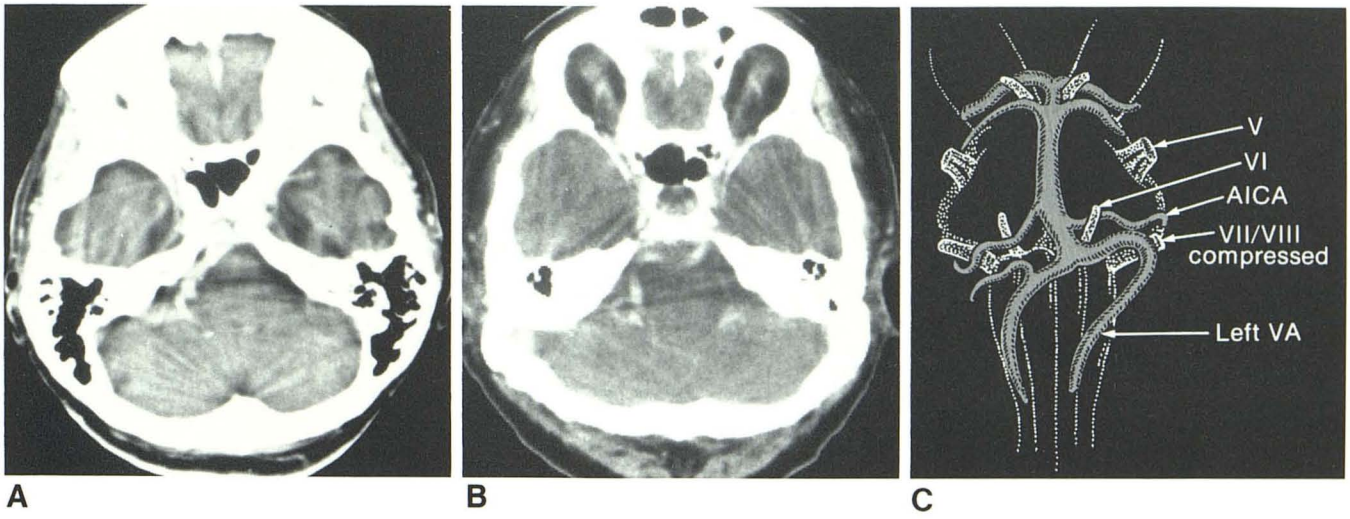


Fig. 4.—Basilar arteries in cases 4 (**A**) and 10 (**B**) are formed in right CPA cistern. Both patients had right-sided HFS. **C**, Compression of left seventh and eighth cranial nerves by distal vertebral–proximal basilar artery.

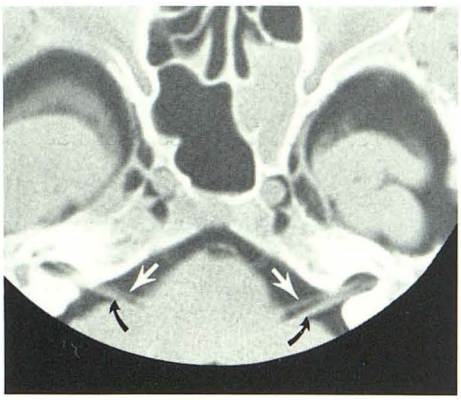


Fig. 5.—Cadaver air study illustrates course of seventh (*straight arrows*) and eighth (*curved arrows*) cranial nerves from their origins at low pons, crossing CPA cistern to enter internal auditory canals. Note more anterior and medial location of seventh cranial nerve.

Initially described by Gowers [54] and later detailed by Wartenberg [55] and Ehni and Woltman [56], HFS is an involuntary, irregular, intermittent, periodic contraction of the muscles on one side of the face. The contractions often begin in the orbicularis oculi muscle and slowly spread to involve all muscles innervated by the facial nerve. If the stapedius muscle is affected, an intermittent clicking noise may be heard. HFS is to be distinguished from blepharospasm, which is bilateral and involves only the muscles around the eye, and from facial myokymia, which appears as a wormlike, subcutaneous involuntary facial movement usually associated with spastic paretic facial contracture [57].

HFS is a symptom of hyperactive dysfunction, presumably caused by vascular compression of the facial nerve at the brainstem. Both the motor and sensory roots of the seventh nerve arise from the inferior border of the pons in the recess between the olive and inferior cerebellar peduncle. The motor root lies medial to the sensory root, while the vestibulocochlear nerve lies lateral to both of these roots [53] (fig. 5). Because of its medial location, the motor root is most susceptible to the compressive effects of displaced, tortuous vessels. The root entry zone is the point at which compression is most likely to produce disordered nerve function [58]. Anatomically, there is a discernible junction between thin (glial) central myelin and thicker (schwannian) peripheral myelin [58]. This junctional area may have a lower threshold to mechanical deformation [58].

Multiple compression cranial nerve deficits. Combined compressive cranial nerve deficits usually involve nerves that course through the CPA cistern [4, 10, 11, 26, 28, 39, 42, 49, 59]. Case 11 had right HFS and TGN, a commonly reported combination. CT and angiography revealed marked tortuosity of the vertebral and basilar arteries (fig. 6). The basilar artery was minimally enlarged. The CT examination was identical to those in patients with isolated facial nerve involvement (fig. 4).

The trigeminal nerve is second only to the facial nerve in reported involvement by VBD. After it emerges from the lateral aspect of the midpons, the trigeminal nerve courses anteriorly through the CPA cistern to pierce the dura and enter Meckel cave (fig. 7). As with HFS, some cases of TGN are thought to be caused by vascular compression at the root entry zone [60, 61]. At surgery, a tortuous loop of the superior cerebellar artery is the most frequently encountered offending vessel [62, 63]. Other vessels encountered include branches of the anterior and posterior inferior cerebellar arteries and venous structures [62, 63]. Isolated TGN secondary to direct compression by elongated and ectatic basilar arteries has been reported [16, 64]. A case of isolated TGN, in which the trigeminal nerve was pinched between an ectatic basilar artery and the superior cerebellar artery, has also been recorded

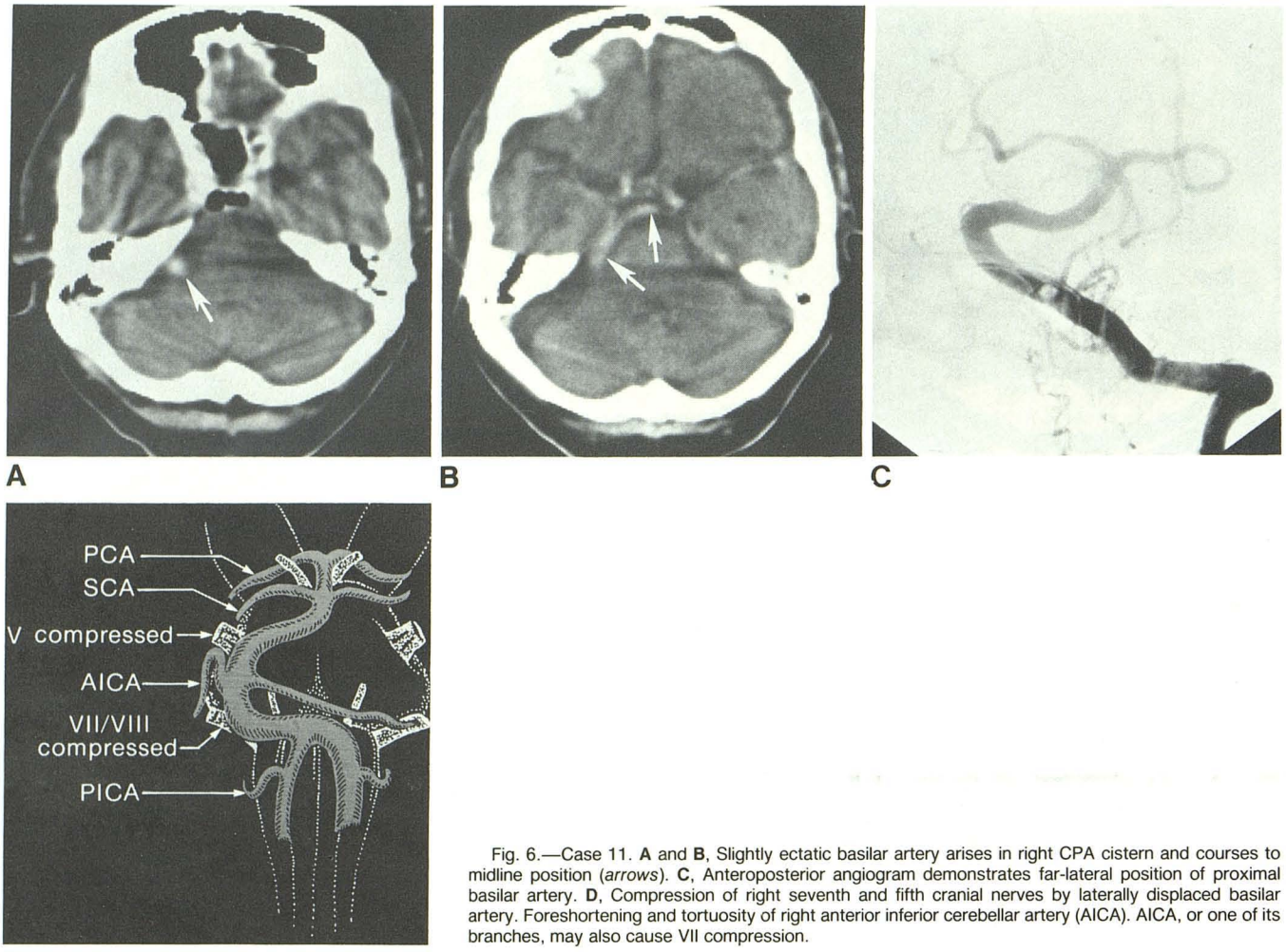


Fig. 6.—Case 11. **A** and **B**, Slightly ectatic basilar artery arises in right CPA cistern and courses to midline position (*arrows*). **C**, Anteroposterior angiogram demonstrates far-lateral position of proximal basilar artery. **D**, Compression of right seventh and fifth cranial nerves by laterally displaced basilar artery. Foreshortening and tortuosity of right anterior inferior cerebellar artery (AICA). AICA, or one of its branches, may also cause VII compression.

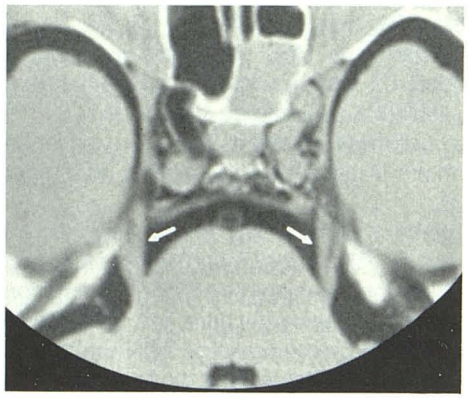


Fig. 7.—Cadaver air study shows course of trigeminal nerve (*arrows*) from origin at midpons across subarachnoid cisterns to enter Meckel cave.

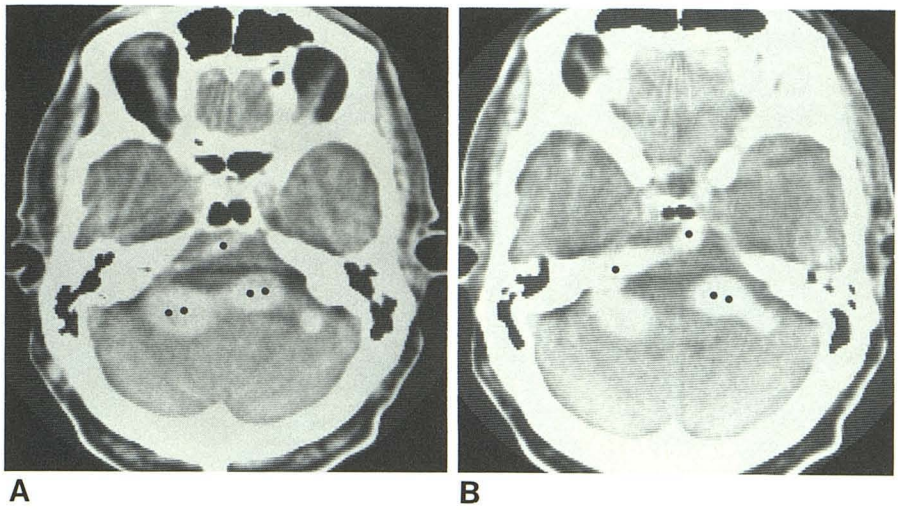
[65]. Campbell and Keedy [30] reported two cases of ipsilateral HFS and TGN secondary to compression by cirsoid aneurysms.

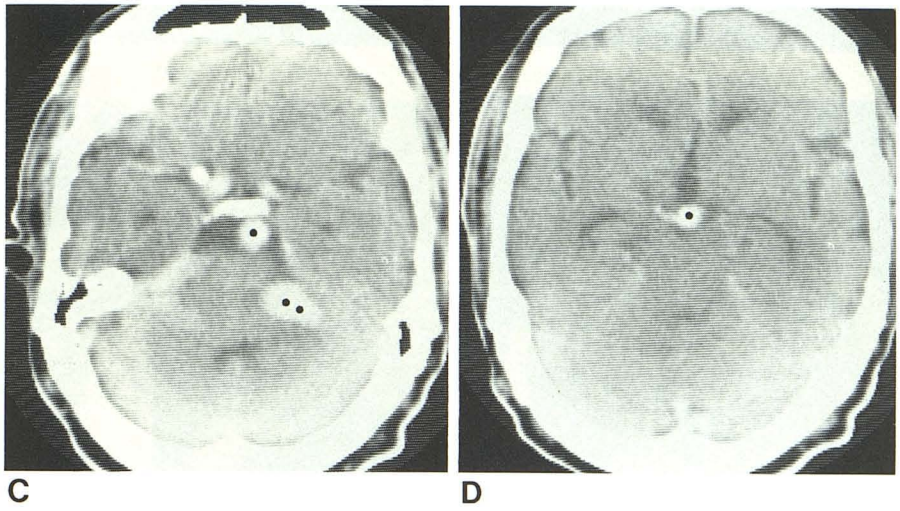
Mixed ischemic deficits. A variety of brainstem and cerebellar signs and symptoms are associated with severe VBD. These include isolated or combined nystagmus, vertigo, dysarthria, ataxia, hemiparesis, and seizures [2, 3, 8, 29, 47, 48]. The ischemia may be secondary to direct mass effect produced by the great ectasia. Hemodynamic changes, resulting from marked stasis of flow, may also be superimposed.

Six of our patients had a combination of neurologic signs most consistent with an ischemic etiology (cases 12–17) (table 1). Four deteriorated rapidly and died. As a group, these patients had the largest basilar artery diameters. Cranial nerve involvement was bilateral in three patients. The rapidly progressive downhill course of severe VBD is exemplified by the following history (fig. 8).

Case 12

A 64-year-old man developed gait disturbances, swallowing difficulties, and decreased hearing in the left ear 1 year before admission. Two months before admission he developed left seventh and twelfth cranial nerve deficits. On admission he had bilateral horizontal nystagmus, ataxic gait, left lateral gaze paresis, left V, VIII, IX, X, and XII and right paralytic lower motor neuron VII, IX, and X deficits. Early the next day he underwent vertebral angiography, and later that day he developed a left pontine infarct with "1½ syndrome" (left gaze





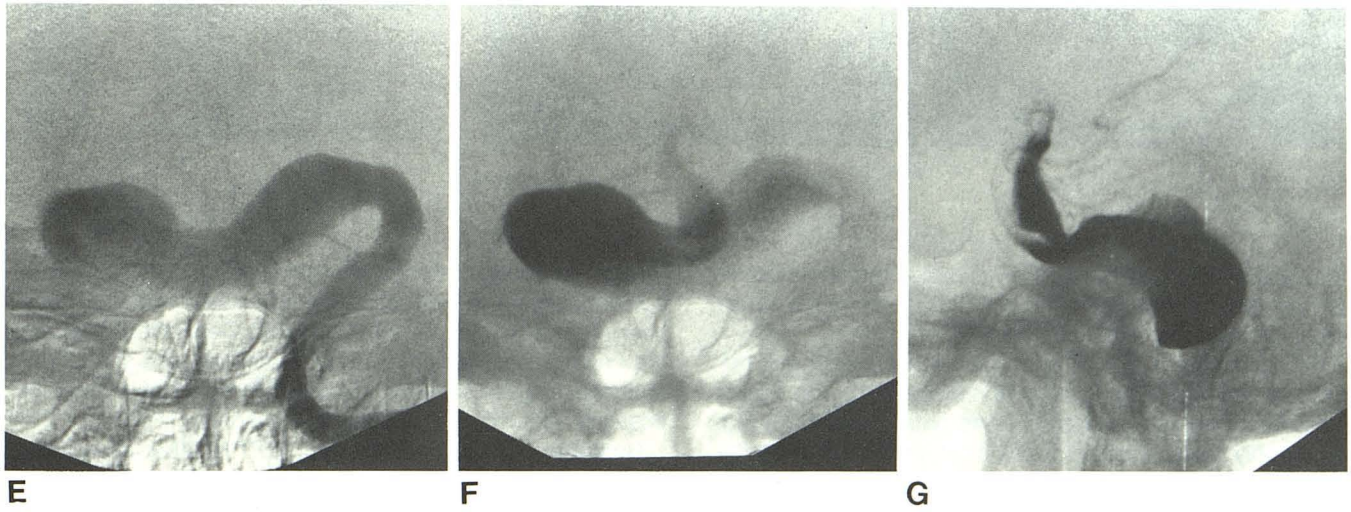


Fig. 8.—Case 12. **A–D**, CT sections. Marked ectasia and tortuosity of distal left vertebral (*double dots*) and basilar (*single dots*) arteries. Distal basilar artery indents floor of posterior third ventricle producing mild dilatation of anterior part (**D**). **E–G**, Angiograms. Stasis of flow manifested by persistence of contrast material within vertebral and basilar vessels late in venous phase (9 sec, **F** and **G**). Basilar artery branches are poorly opacified.

palsy and left internuclear ophthalmoplegia). Two days later he developed a left lower motor VII deficit and 3 days after that, right V_2 and V_3 numbness. Four days later, he underwent a posterior fossa decompressive craniectomy and left vertebral artery ligation. He died 13 days later. Permission for autopsy was denied.

CT and autopsy material from a similar patient (case 13) is presented in figure 9. The maximum basilar artery diameter was 21.2 mm, the largest in our series. His symptoms were clinically the most severe ("locked-in syndrome" or cortico-

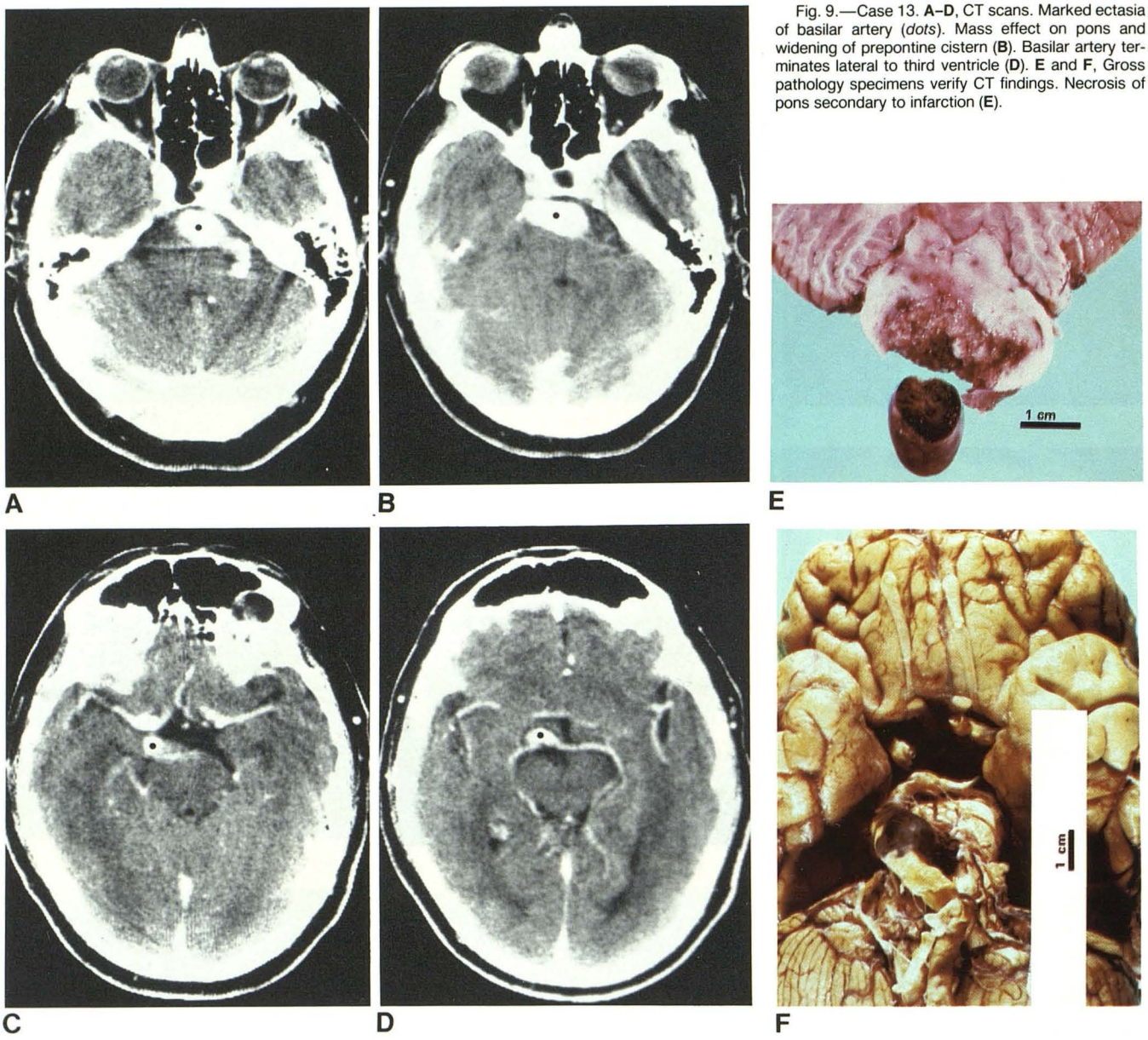


Fig. 9.—Case 13. **A–D**, CT scans. Marked ectasia of basilar artery (*dots*). Mass effect on pons and widening of prepontine cistern (**B**). Basilar artery terminates lateral to third ventricle (**D**). **E** and **F**, Gross pathology specimens verify CT findings. Necrosis of pons secondary to infarction (**E**).

medullospinal disconnection). The "pseudotumoral" nature of the markedly ectatic basilar artery was seen in the gross pathologic specimen (figs. 9E and 9F).

Hydrocephalus. Three of our patients had symptoms referable to hydrocephalus (cases 18–20) (table 1). The basilar artery was markedly enlarged in one patient (case 18) (fig. 10) but only mildly enlarged in the other two. In all three the basilar artery was very elongated, indenting the floor of the third ventricle.

In 1953 Sjögren [66] reported that an anomaly of the basilar artery could cause indentation of the third-ventricle floor, demonstrable by pneumoencephalography. In 1954 Greitz and Löftstedt [45] detailed the relation of the third ventricle to the basilar artery and presented five cases of hypertensive individuals who had hydrocephalus secondary to third ventricle compression by elongated basilar arteries. Since then a number of cases of hydrocephalus associated with VBD have been reported [7, 13, 21, 23, 24, 27, 67]. Depending on where the basilar artery indents the third ventricle, the site of obstruction may be anterior, near the foramen of Monro, or posterior, simulating aqueduct obstruction.

Hydrocephalus in patients with VBD is often associated with anatomically patent cerebrospinal fluid (CSF) pathways and normal mean CSF pressures. It is therefore termed *functional hydrocephalus* [68]. The mechanism of hydrocephalus in these patients is most likely a combination of increased CSF pulse pressure and impairment of outward CSF flow by "countercurrent pulsations" of the basilar artery [21, 23, 68, 69]. True obstructive hydrocephalus, in association with VBD, has also been reported [25, 27].

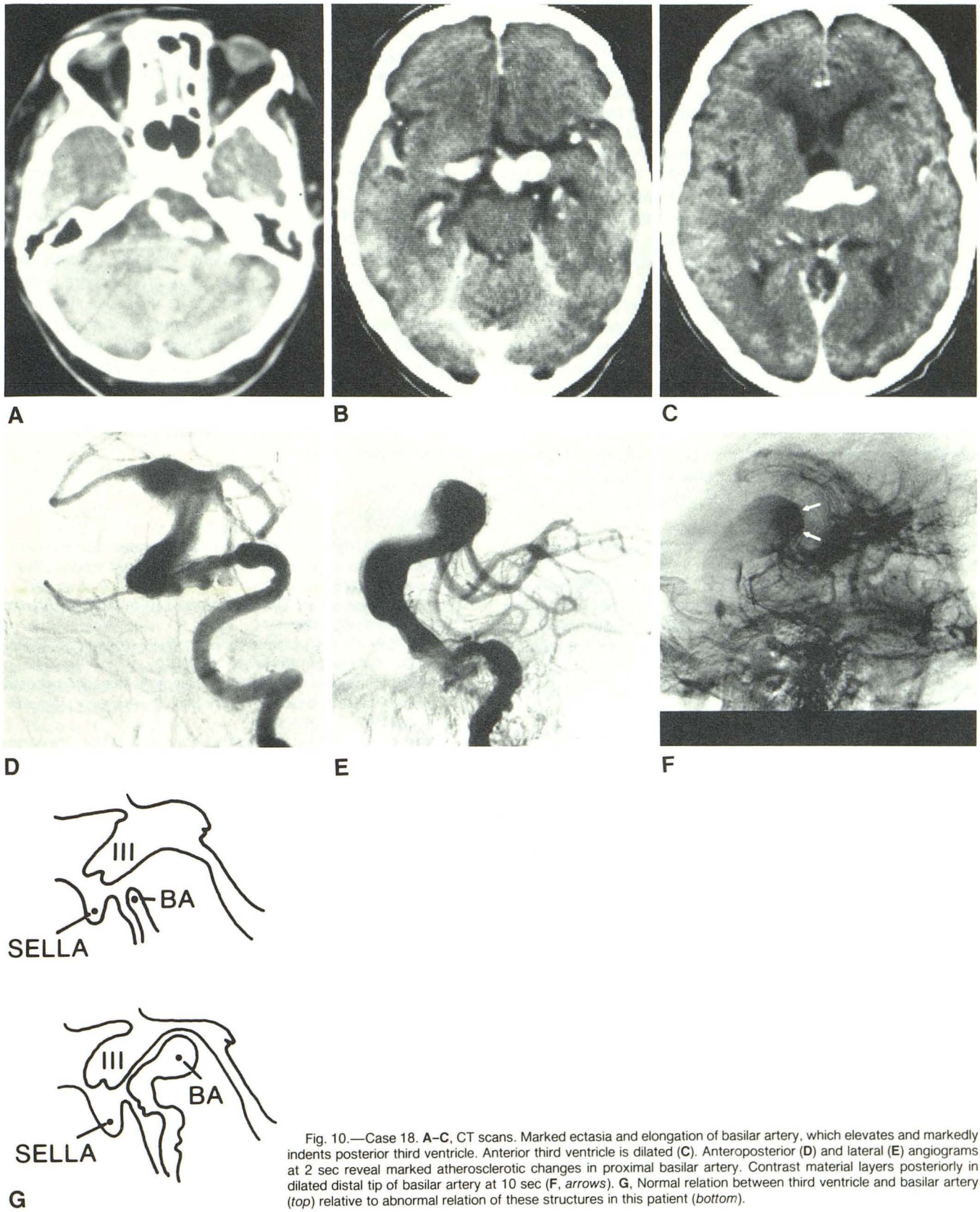


Fig. 10.—Case 18. **A–C**, CT scans. Marked ectasia and elongation of basilar artery, which elevates and markedly indents posterior third ventricle. Anterior third ventricle is dilated (**C**). Anteroposterior (**D**) and lateral (**E**) angiograms at 2 sec reveal marked atherosclerotic changes in proximal basilar artery. Contrast material layers posteriorly in dilated distal tip of basilar artery at 10 sec (**F**, *arrows*). **G**, Normal relation between third ventricle and basilar artery (*top*) relative to abnormal relation of these structures in this patient (*bottom*).

The Role of Angiography

Nine of our patients underwent angiographic evaluation. Seven had conventional angiography and two had intraarterial DSA. Two of the seven patients who underwent conventional angiography experienced severe brainstem ischemic symptoms, reversible in one (case 18), but causing infarction in the other (case 12), probably due to stasis of contrast material (figs. 8G and 10F). It is noteworthy that both patients had basilar artery diameters three to four times normal (table 1). Our only patient with a diameter larger than these (case 13) did not undergo angiographic examination. Pribram et al. [39] reported an angiographic complication resulting in death of a patient with a fusiform aneurysm.

With high-resolution CT and the currently noncorrectable nature of VBD, we see no justification for conventional angiography. We believe that basilar artery diameters greater than 1 cm on CT represent a contraindication to conventional angiography. Although some authors (prior to the advent of DSA) maintained that angiography is necessary for the evaluation of this entity [1, 40, 43], most believe that CT alone is sufficient to establish the diagnosis [7, 14, 15, 22, 25, 26, 48, 63]. If an angiographic study is necessary, intravenous DSA or nonselective intraarterial DSA is recommended.

A Plea for the Term Vertebrobasilar Dolichoectasia (VBD)

The finding of cerebral arterial ectasia in a 2-year-old child has led some to speculate on a congenital origin [17]. A case of TGN and dementia secondary to VBD in a patient with the ectodermal dysplastic EEC syndrome (ectodermal dysplasia, ectrodactyly, and cleft lip-palate) has also been reported [16]. However, most VBD patients do not fall into such categories. It is possible that deficiencies of the internal elastic lamina encountered in cases of VBD are present at birth. The actual dilatation and elongation, however, are not usually encountered until much later in life, most often in the presence of long-standing hypertension. An acquired component would seem to be necessary. For this reason, we believe references to VBD using the term *malformation* (a congenital deformity) or *anomaly* ("marked deviation from the normal standard, especially as a result of congenital or hereditary defects" [70]) should be discontinued (aneurysmal malformation, megadolichovascular malformation, megadolichobasilar anomaly, etc.).

Cases of VBD have often been reported under the headings of "fusiform" and "cirsoid" aneurysms. The basilar artery is frequently not fusiform (L. "fusus," a spindle), since the entire artery is often involved and the vessel is not "spindle-shaped" at all. The term *cirsoid aneurysm*, however, is quite appropriate (G. "kirsos" varix, + "eidos," appearance), defined as dilatation and tortuosity of an artery resembling varices in a vein. The term has not been in common usage for some time, and, paired with the term *aneurysm* (a blood-containing tumor), its use should not be reestablished.

Our cases clearly demonstrate symptomatology referable to elongation and tortuosity of vertebrobasilar vessels without associated ectasia. The same observation has been made by others [42, 43, 71]. The correct term would be dolichovertebrobasilar artery, as distinguished from dolichoectatic vertebrobasilar artery. Since, however, clear-cut differences in signs and symptoms between these conditions are not demonstrable, we prefer and encourage the use of the more inclusive term *vertebrobasilar dolichoectasia*.

Summary

Clinical signs and symptoms in 20 patients with symptomatic VBD were analyzed in relation to size and positional abnormalities of the basilar artery. Patients with elongated and tortuous, but normal-sized, basilar arteries tend to have isolated cranial nerve involvement, whereas patients with dilatation (ectasia) of the basilar artery are far more likely to suffer from multiple neurologic deficits. Because the basilar artery parameters and neurologic symptoms present as a spectrum, the most appropriate descriptive term for this entity is *vertebrobasilar dolichoectasia*. Conventional angiography poses an increased risk of complications in patients with severe VBD. If vascular studies are necessary, intravenous DSA or nonselective intraarterial DSA examinations should be performed.

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