Stroke *Special Report*

# Charles Foix— The First Modern Stroke Neurologist

Louis R. Caplan, MD

Charles Foix was born in Salies-de-Béarn, a small village in the southern part of France, in 1882. He was the son of a physician from Béarn. Foix came to Paris to study medicine and spent his entire career within the hospital systems of Paris (Hotel-Dieu, Necker, Bicétre, Salpêtrière). He was named hospital intern in 1906, and during his fourth year of training he won the Gold Medal Competition, a very coveted and prestigious award. Then came World War I, and Foix fought in France and on the Eastern Front. After returning to Paris he was named hospital physician in 1919 and Agrégé to the faculty in 1923, a position he held until his untimely death in 1927 at age 45. In France, the examination for the position of interne hôpitaux (a 1–4-year appointment with duties similar to those of residents in the United States) was very competitive. To become Agrégé, appointees had to pass another competitive examination and present a thesis (thèse d'Agrégation).

Foix had an aristocratic demeanor (Figure 1) and a deep, resonant voice. Roussy[1] described him as frank and straightforward with remarkable charm, goodness, and modesty. Foix was extremely popular with students, peers, and the senior faculty alike, and his teaching conferences were very well attended.[1–3] For years, he regularly gave courses at Guillain's clinic at the Salpêtrière. As a compulsive and effective diagnostician Foix had few peers. "In his hospital ward when he examined a patient, how many times would he forget the time and not leave the consultation room until the diagnosis had been made or the sign searched for was detected."[1] Of interest, this same description would be equally valid for his modern-day successor, Charles Miller Fisher. Well rounded, Foix loved the arts and literature, played the lyre, published several volumes of poetry, and was a dedicated family man.[4]

Like Charcot, Foix's first research studies were in the field of general medicine. He studied and reported the leukocyte-activating powers of body fluids and hemolysis. He was a student and disciple of Pierre Marie "from whom he learned a strict method of observation and a strong scientific discipline."[3] His first neurologic papers were with Marie and concerned abnormalities of motor and reflex function such as medullary automatisms and synkineses, flexor spinal reflexes, and synkinetic movements in hemiplegics. Foix then studied tonus and contractures. The anatomy, functions, and diseases of the extrapyramidal system were life-long interests of his and led to the publication of a book with Nicolesco on the basal ganglia and suboptic-mesencephalic region.[5] Foix also wrote extensively on higher cortical function abnormalities, especially aphasia and apraxia, and on cerebellar degenerations and syndromes. Two of his best-known nonvascular contributions were recognition with Sicard of albuminocytologic dissociation in the cerebrospinal fluid in patients with spinal cord compression[6] and the description of the syndrome of subacute necrotizing myelitis (Foix-Alajouanine syndrome).[7]

Though his interests, collaborations, studies, and contributions were extremely broad, Foix will undoubtedly be remembered best for his studies on cerebrovascular disease, and I will limit my analysis of his contribution to this subject. Hillemand, one of his major collaborators, cited an initial single observation as the stimulus for Foix's cerebrovascular studies.[8] In the neuropathology laboratory, he noted the frequent coexistence of thalamic infarction and softening of the inferior parts of the occipital and temporal lobes. At that time the thalamic syndrome was well known, having been described a decade previously by Dejerine and Roussy.[9] Those authors, however, made no comment on the vascular supply or the vascular lesions involved. Foix was intrigued by the coincidence of thalamic and cerebral hemisphere lesions and, meticulously dissecting the arterial supply, found that the calcarine cortex was not irrigated by the middle cerebral artery (MCA) but that the posterior cerebral artery (PCA) supplied the visual cortex, the splenium of the corpus callosum, and the inferior temporal lobe, as well as the thalamus. Now, Foix understood the association. All the lesions were due to obliteration of the same arterial vessel, and the thalamic syndrome fell into the framework of a more extensive vascular syndrome, that of the PCA.[10] Hillemand comments, "from that point an unex-

From the Tufts University School of Medicine, Boston, Massachusetts.

Address for reprints: Louis R. Caplan, MD, New England Medical Center, Department of Neurology, 750 Washington Street, Boston, MA 02111.

Received June 27, 1989; accepted August 29, 1989.

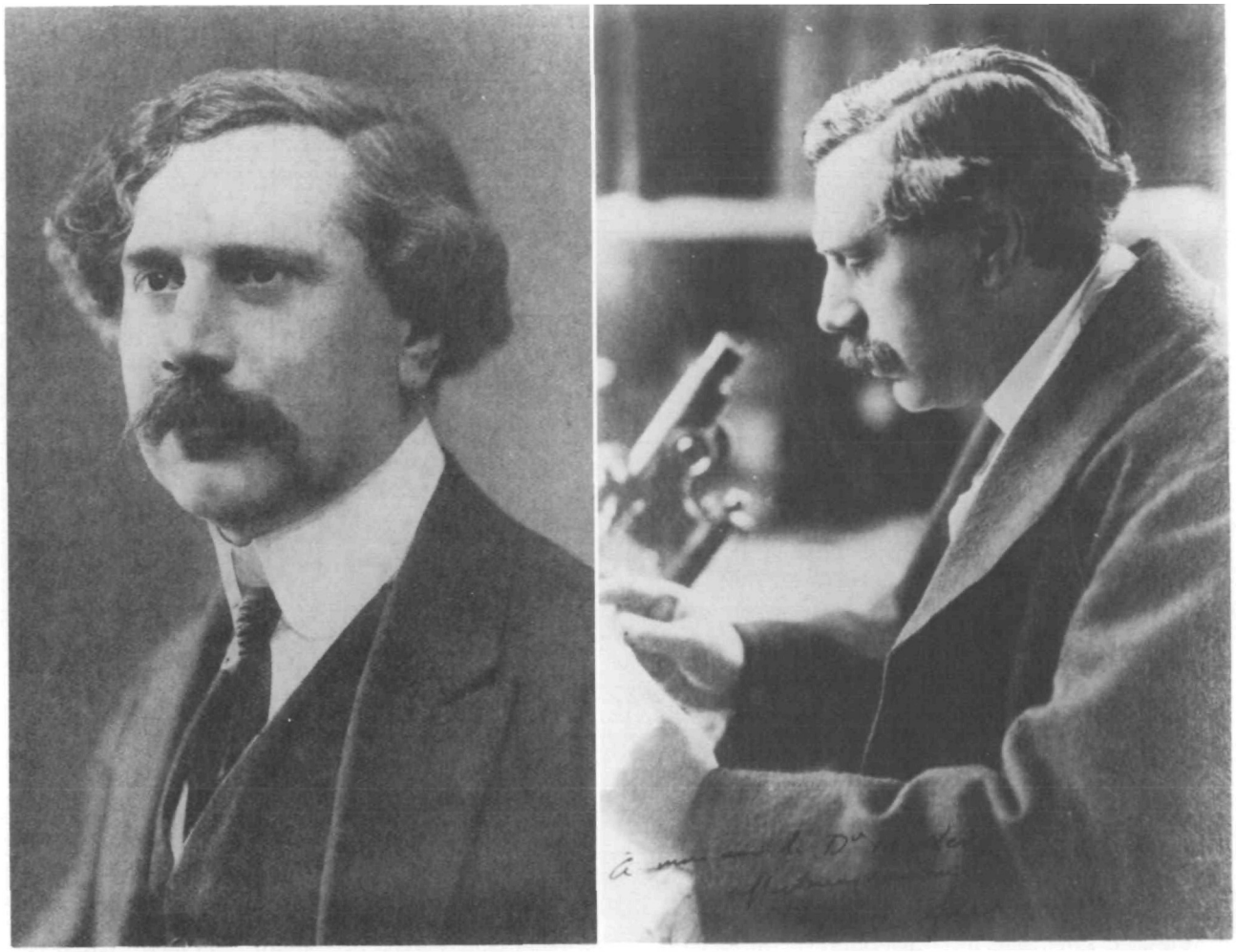

FIGURE 1. Left: *Youthful Charles Foix from Reference 4, p 241.* Right: *More mature Foix with his microscope in neuropathology laboratory.*

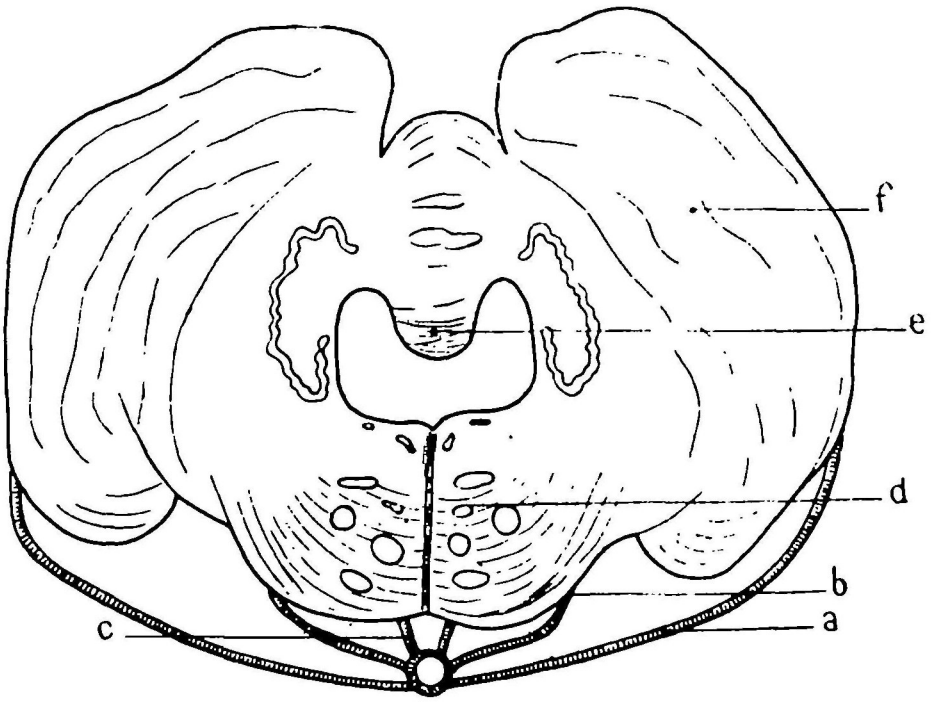


FIGURE 2. *Schema of brainstem blood supply from Reference 17, p 709. a, long circumferential artery; b, short circumferential artery; c, paramedian artery; d, pons ("protuberance"); e, cerebellar vermis; f, lateral lobe of cerebellum.*

plored avenue opened up before him the study of the extensive vascular syndromes . . . which would fill the most fruitful years of his career."[8]

Foix's major focus was on the anatomy and supply of the major cerebral arteries, the distribution of softenings (ramollissements), and the clinical signs related to the lesions. His tools were primarily anatomic, pathologic, and clinical, and he labored hard in his anatomicopathologic laboratory at the Salpêtrière, at the bedside, and in the outpatient clinic. Very early Foix recognized that previous treatises on anatomy often gave erroneous descriptions of the brain vascular supply and that the descriptions did not agree with the figures used as illustrations. Almost everything had to be reviewed and studied from the beginning. For months Foix meticulously dissected the intracranial arteries and their branches, subjecting both dye and opaque injections to x-rays.[8] First, he analyzed the distribution of brainstem arteries with their three systems of paramedian and short and long circumferential arteries (Figure 2), and then he found this same general schematic arrangement in the major cerebral arteries, the anterior cerebral artery (ACA), the MCA, and the PCA.

Foix's reports were detailed and very systematic, and most followed a uniform system and schema. First came a description of the anatomy of the artery(ies) being considered and their deep and superficial branches, then a description of the anatomic regions and nuclear structures supplied by each branch, and then an analysis of the distribution of softenings and their accompanying neurologic signs. This was the era of anatomicoclinicopathologic correlation. Foix paid little attention to the pace of illness, the accompanying risk factors, or the mechanism of infarction, and until the very end he gave little attention to the vascular pathology. His papers were nicely illustrated with diagrams that he drew himself and with pictures of gross and stained pathologic specimens. At the time of Foix, duplication was commonplace in the medical literature, and many of his diagrams and verbatim descriptions appeared in multiple publications on the same general topic. He seldom enumerated in his vascular papers the number of patients or specimens studied or quantified the relative frequency of various findings and signs.

### Posterior Circulation Studies and Reports

Foix's first important stroke paper concerned the syndrome of the PCA and was written with Masson.[10] The paper began with descriptions and drawings of the branching of the PCA (Figure 3) and the brain regions supplied by its branches (Figure 4). The major components of the clinical findings (hemianopsia, hemisensory loss, and alexia) in patients with complete PCA-territory infarcts were noted. Then the authors described partial syndromes, for example, an "anterior syndrome affecting the proximal PCA and cerebral peduncle with cerebellar findings and sensory loss, a thalamic syndrome, and a posterior syndrome affecting the occipital lobe with hemianopia and alexia the major findings." Abundant photographs of gross pathologic specimens illustrated the verbal descriptions. Foix always showed an interest in abnormalities of higher cortical functions; he later wrote an article with Hillemand on the role of the splenium of the corpus callosum in alexia without agraphia,[11] a PCA syndrome that had been described and explained by one of his mentors, Jules Dejerine.[12]

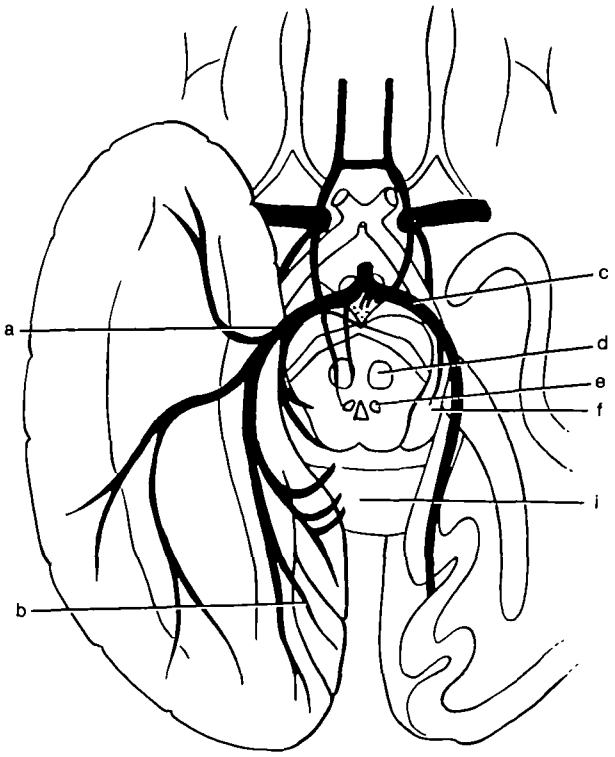


FIGURE 3. *Artist's redrawing of original figure from Reference 10, p 361, posterior cerebral artery and its branches. a, posterior cerebral artery; b, cuneus and calcarine fissure; c, posterior cerebral artery; d, red nucleus; e, oculomotor nucleus; f, optic tract and lateral geniculate body; i, splenium of corpus callosum.*

Foix and his colleagues must have studied both the rostral and the caudal intracranial portions of the posterior circulation concurrently because during the next 2 years he and his colleagues Hillemand and Schalit published reports on the blood supply of the pons,[13,14] the thalamic syndromes,[15] and the lateral medullary syndrome[16] and authored a major work on the arteries of the brainstem including the diencephalon.[17] The paper on the thalamic syndromes[13] contained detailed schematic drawings of the blood supply (Figure 5) and the thalamic regions supplied; the remainder of the paper analyzed the clinical findings in patients with infarcts in selected thalamic regions, for example, within the thalamoperforating artery territory. A drawing illustrating the combination of thalamic and temporal lobe infarctions (Figure 6) was included. In the paper that reviewed the arterial supply of the brainstem, Foix and Hillemand[17] included quite detailed drawings of the blood supply

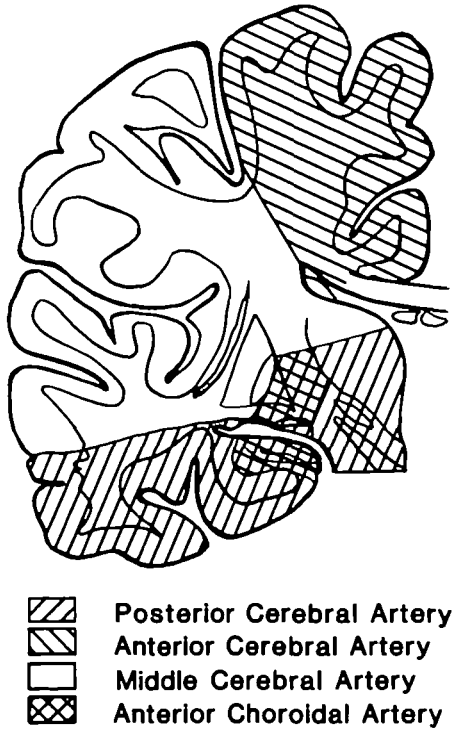


FIGURE 4. *Artist's redrawing of original figure from Reference 10, p 361, coronal brain section through thalamus with territories of major cerebral arteries.*

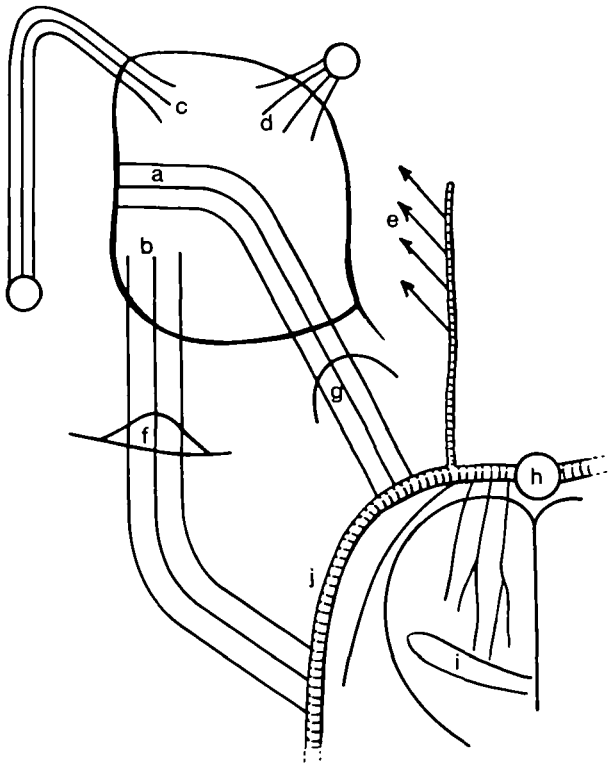


FIGURE 5. *Artist's redrawing of original figure from Reference 15, p 113, schema of diverse pedicles and their penetration into thalamus. a, thalamoperforating pedicle; b, thalamogeniculate pedicle; c, thalamo-optic pedicle; d, choroidal pedicle; e, thalamotuberal pedicle; f, geniculate body; g, red nucleus; h, basilar artery trunk; i, substantia nigra; j, posterior cerebral artery.*

(Figures 7 and 8); even the angulation of the arteries was illustrated (Figure 9). Then, the authors successively reviewed the arterial supply of the pons, medulla, midbrain, and diencephalon and the clinical syndromes at each level. For example, the syndromes of the pontine arteries were characterized as follows[14,17]: syndrome of the paramedian territory – the clinical "table" is very simple, the illness presents as a banal hemiplegia; syndrome of a short circumferential artery – cerebellar signs on the side of the lesion and abnormalities of pyramidal and sensory function on the opposite side, softenings of the pontine tegmentum were "numerous and particularly complex."[14]

The one major error Foix and his colleagues made was overemphasis on a circumferential artery that originated from the basilar artery and that they thought regularly supplied the superior portion of the lateral medulla (Figure 10).[16] This vessel, called "l'artère de la fossette latérale du bulbe," was the subject of a full paper in *Revue Neurologique* in 1925.[16] Foix, Hillemand, and Schalit[16] illustrated in detail the course of this artery and the portion of the medulla and its nuclear content supplied. They also reviewed all previous reports on the lateral medullary (Wallenberg) syndrome. A later detailed study of the pathology of the lateral medullary syndrome[18] did not corroborate Foix's analysis of the key role of this basilar artery branch.[16]

### Anterior Circulation

Though better known for studies on the vertebral basilar circulation, the reports of Foix and his colleagues on the major anterior circulation arteries (ACA,[19] MCA,[20] and anterior choroidal artery [AChA][21]) were equally detailed and complete. These studies were also published during 1925 to 1927, concurrent with the posterior circulation reports. Foix and his colleagues must have been studying the two circulations at the same time, extensively examining each pathologic specimen and each patient irrespective of the location of the infarct.

Following the same schematic outline used in the posterior circulation papers, in discussing the ACA, Foix and Hillemand[19] first noted the normal distribution of the artery and its branches and the brain regions supplied by each branch, and then they described the clinical findings in each partial syndrome. The three most common presentations of ACA-territory infarction were enumerated as 1) a simple crural monoplegia, 2) a hemiplegia with major crural predominance, and 3) a crural monoplegia (or hemiplegia with crural predominance) and a unilateral left ideomotor apraxia. Foix's recognition of the left apraxia, regardless of the side of the infarction, long antedated the discussions of Geschwind and colleagues on the anterior disconnection syndrome.[22,23]

Foix's analyses of the MCA syndrome were perhaps the most detailed of all his vascular studies.[20]

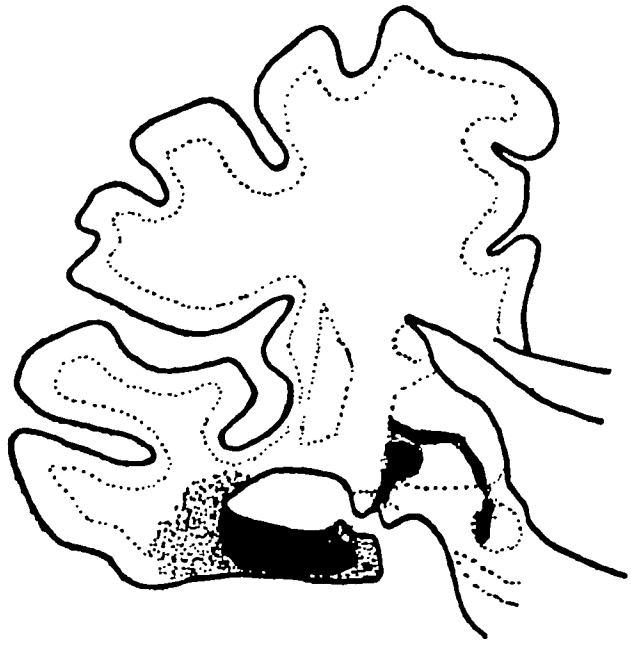

FIGURE 6. *From Reference 15, p 115, lesion of thalamoperforating and thalamogeniculate pedicles associated with softening of inferior aspect of temporal lobe. Infarct schematically in black.*

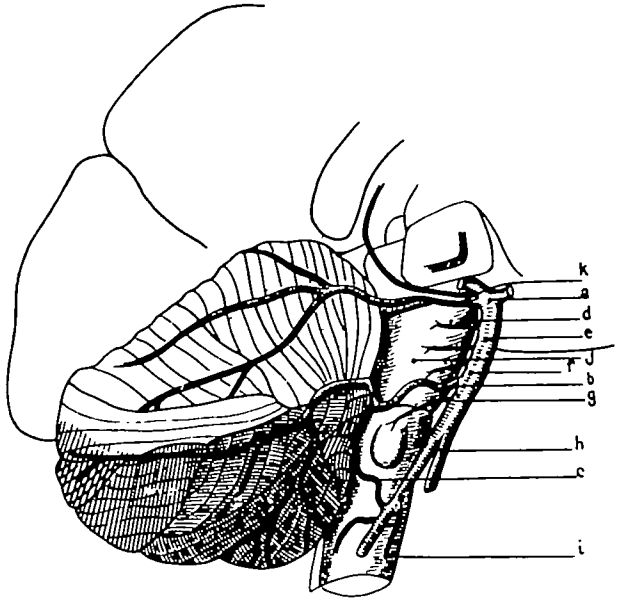


FIGURE 8. *Lateral aspect of brainstem arteries and supply from Reference 17, p 708. a, posterior cerebral artery; b, middle cerebellar artery (AICA); c, posterior inferior cerebellar artery (PICA); d, short circumferential pontine artery; e, basilar artery trunk and behind it, paramedian arteries; f, basilar trunk; g, lateral artery of bulb; h, vertebral artery; i, medulla oblongata; j, pons; k, superior cerebellar artery.*

This meticulously detailed study of sylvian infarcts is now probably the least often cited and least remembered among his vascular works. Yet, for the modern-day student or clinician, in my opinion, it is the single most worthwhile report to review because it illustrates fully the methodology of Foix and his colleagues. With Lévy, Foix[20] described the normal MCA anatomy (Figure 11), the deep and superficial territories of the MCA (Figure 12), and the MCA branches and accompanying clinical syndromes. Rules favoring a deep MCA lenticulostriate infarct were enumerated (Table 1) and are equally applica-

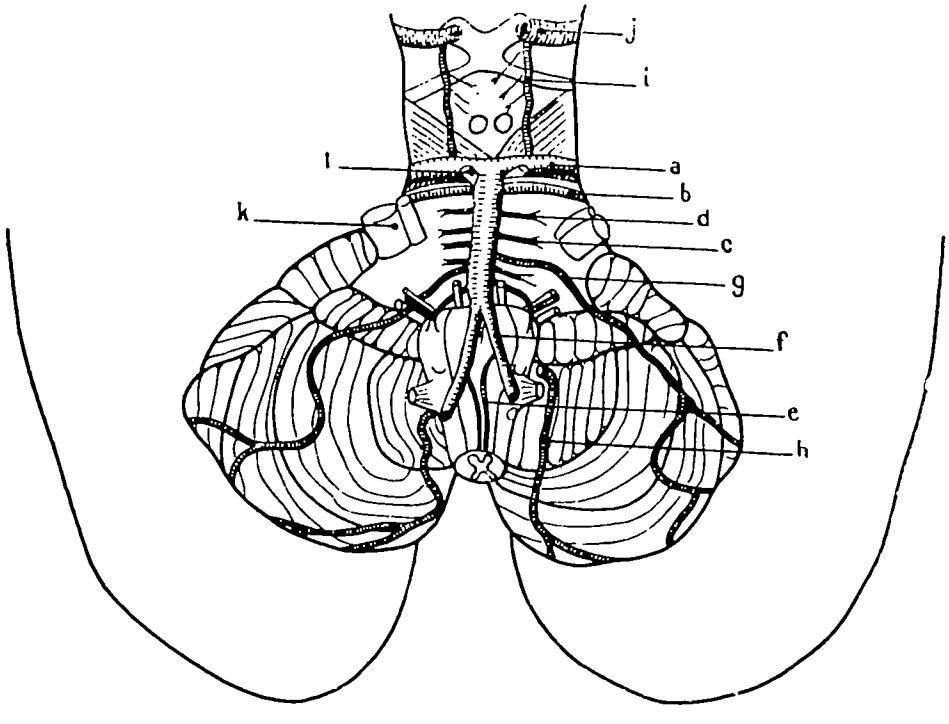


FIGURE 7. *Schematic drawing of brainstem arteries from Reference 17, p 707. a, posterior cerebral artery; b, superior cerebellar artery; c, basilar artery trunk; d, short circumferential pontine artery; e, anterior spinal artery; f, vertebral artery; g, middle cerebellar artery (AICA); h, inferior cerebellar artery (PICA); i, posterior communicating artery; j, carotid artery trunk; k, trigeminal nerve; l, oculomotor nerve.*

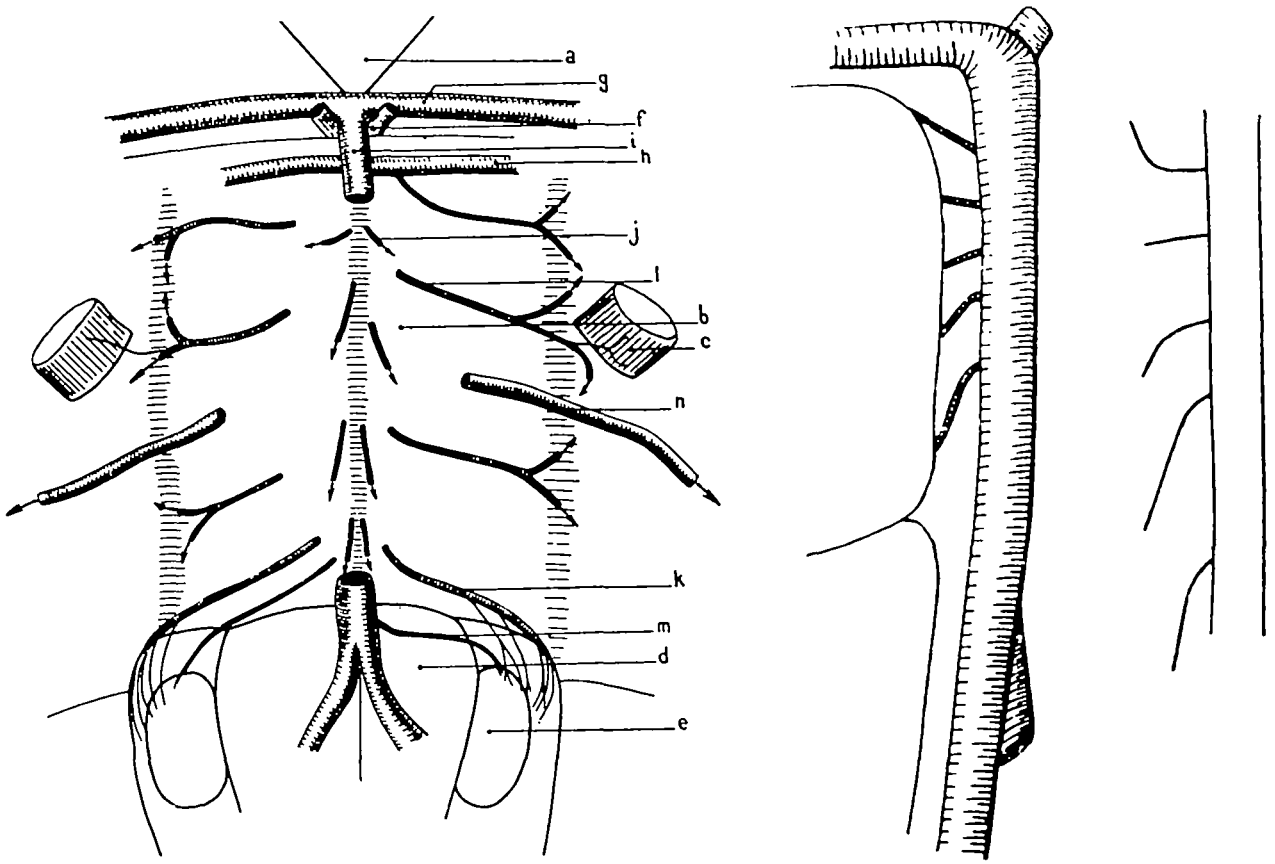


FIGURE 9. Left: *Disposition of brainstem arteries with basilar trunk removed, from Reference 17, p 711. a, posterior perforated space; b, pons; c, trigeminal nerve; d, pyramid; e, olive; f, oculomotor nerve; g, posterior cerebral artery; h, superior cerebellar artery; i, trunk of basilar artery; j, paramedian artery; k, lateral artery of bulb; l, short circumferential artery; m, lateral accessory artery; n, middle cerebellar artery (AICA).* Center *and* right: *Disposition and angulation of paramedian arteries of brainstem, from Reference 17, p 709.*

ble today. Multiple subtypes were analyzed (Table 2), and many pathologic pictures and diagrams served to illustrate the anatomy and pathology.

The report on the AChA syndrome was very brief and was delivered to the French Society of Ophthalmology.[21] This paper is now very difficult to find, but it may be the most often cited of Foix's works, probably because of the dearth of reports of AChA infarction in the succeeding 5 decades.[24] This report also probably gives a misleading interpretation of the clinical findings in AChA infarction, citing the invariable coexistence of hemiplegia, hemianesthesia, and hemianopsia and the absence of prominent cortical function abnormalities.[21] We now know that contralateral visual, sensory, and motor loss of function are uncommon after occlusion of the AChA, that only rarely are all three deficits present together, and that cortical function abnormalities are common, at least transiently.[24]

### Mechanism of Infarction

In his studies on the cerebral and brainstem circulation cited so far, Foix can be viewed as a product of his times, though a brilliant and productive one. In the 1920s, the emphasis was clearly on anatomico-clinicopathologic correlation and clinical phenomenology. None of Foix's vascular papers showed an interest in the tempo or pace of the symptoms. Onset was invariably abrupt, and transient ischemic attacks (TIAs) were not mentioned. Foix and his colleagues also showed no interest in pathology within the arteries; they emphasized only the distribution of the softenings and the clinical signs that accompanied infarction. However, several weeks before his death, Foix and his colleagues Hillemand and Ley delivered a report at a meeting of the Medical Society of the Hospitals of Paris on a study of vascular pathology in arteries supplying regions of brain softening. An abstract of this report was published in *Revue Neurologique,*[25] but as far as I know a full article on the subject never appeared.

In this report, Foix and colleagues analyzed 56 cases of brain softening. Some patients had hemorrhagic infarcts. Among these cases the artery supplying the infarct was totally occluded in 12 and subtotally occluded in 14, but in 30 cases the arteries were essentially open or had relatively unimportant stenosis. The authors stepped beyond their colleagues of

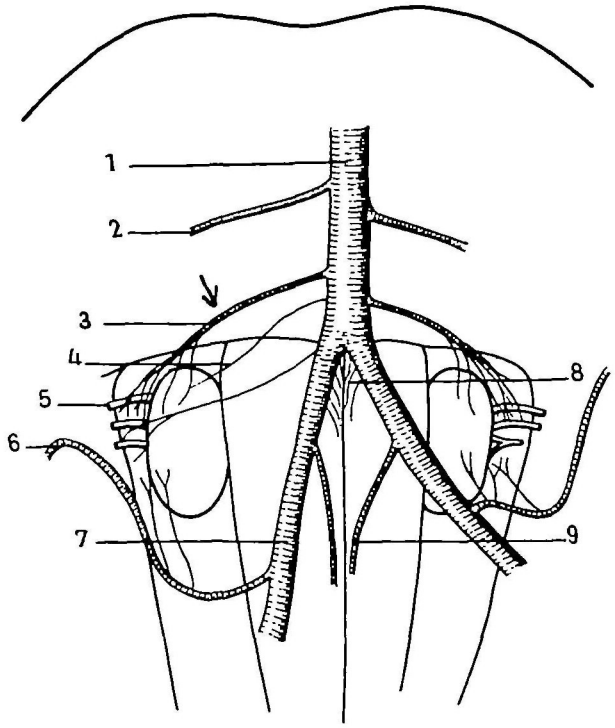


FIGURE 10. *Brainstem arteries from Reference 16, p 171. Artère de la fossette latérale du bulbe (numbered 3, arrow). 1, basilar artery trunk; 2, middle cerebellar artery (AICA); 4, accessory artery; 5, mixed nerves; 6, inferior cerebellar artery (PICA); 7, vertebral artery; 8, pedicle of spinal artery of median groove (?); 9, anterior spinal artery.*

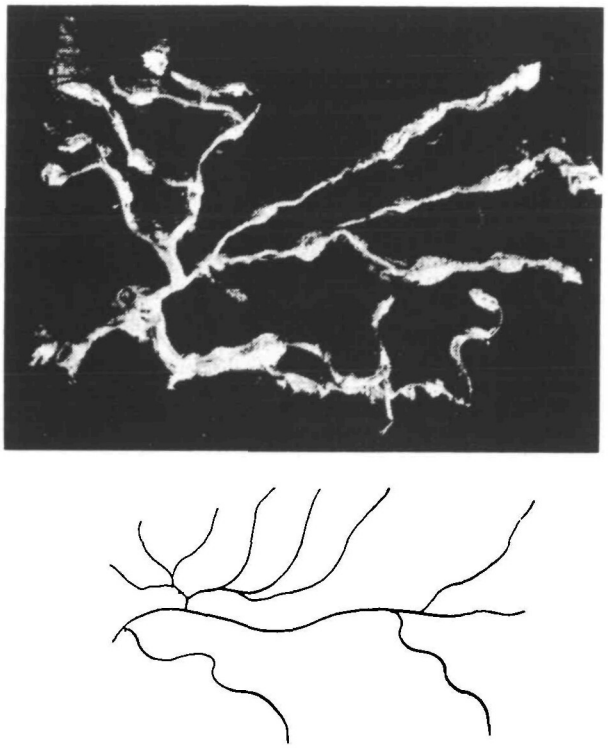

FIGURE 11. *From Reference 20, p 3.* Top: *Photograph of dissected sylvian artery and its branches.* Bottom: *Schema of arteries.*

the era in speculating on possible explanations for the frequent lack of arterial occlusion. Four possibilities were cited: 1) occlusion followed softening and would have developed later, 2) embolism with passage of material by the time of autopsy, 3) insufficiency ("l'insuffisance cardio-artérielle"), that is, more proximally located circulatory failure, and 4) vasospasm ("spasme artérielle"). Clearly, with these last two suggestions Foix was decades ahead of his time.

## Death and Placing the Contribution of Foix in Historical Perspective

Foix died in 1927 at the age of 45. His terminal illness was acute and accompanied by abdominal pain and fever. He died quickly after surgery, never recovering postoperatively. I could find no diagnosis proffered, but appendicitis with rupture and peritonitis sounds most likely. Struck down at the height of his career and productivity, we will never know how the history of stroke might have differed had Foix lived another two or three decades.

Foix surely owed his disciplined approach to cerebrovascular disease to Charcot and to the students and followers of Charcot who were his teachers and collaborators. Charcot and Marie were clear thinkers whose contributions depended on careful clinical examination and observation, on recognition, description, and analysis of clinical phenomenology, and above all else on correlation of the clinical findings with the anatomic and pathologic findings in the nervous system at necropsy. Often, the process started (as in multiple sclerosis or motor neuron disease) with the necropsy findings, and later the corresponding clinical picture was recognized. Charcot and his followers emphasized degenerative system disorders and multiple sclerosis. It was natural

**TABLE 1. Rules for Distinguishing Deep From Superficial Middle Cerebral Artery–Territory Lesions**

| Favoring a deep lesion are: |
|---|
| 1. Absence of hemianopsia |
| 2. Relative absence of sensory abnormalities |
| 3. Relatively proportional character of hemiplegia (face=arm=leg) |
| 4. Simultaneous involvement of arm and leg |
| 5. Distal emphasis of paralysis |

**TABLE 2. Subtypes of MCA Syndrome**

| |
|---|
| 1. Softening of entire sylvian (MCA) territory |
| 2. Large softening of deep territory |
| 3. Partial softening of deep territory |
| 4. Large softening of superficial territory |
| 5. Softening of posterior portion of MCA |
| 6. Partial softening of posterior territory |
| 7. Partial anterior and rolandic softenings |
| 8. Multiple softenings, bilateral, and associated |

MCA, middle cerebral artery.

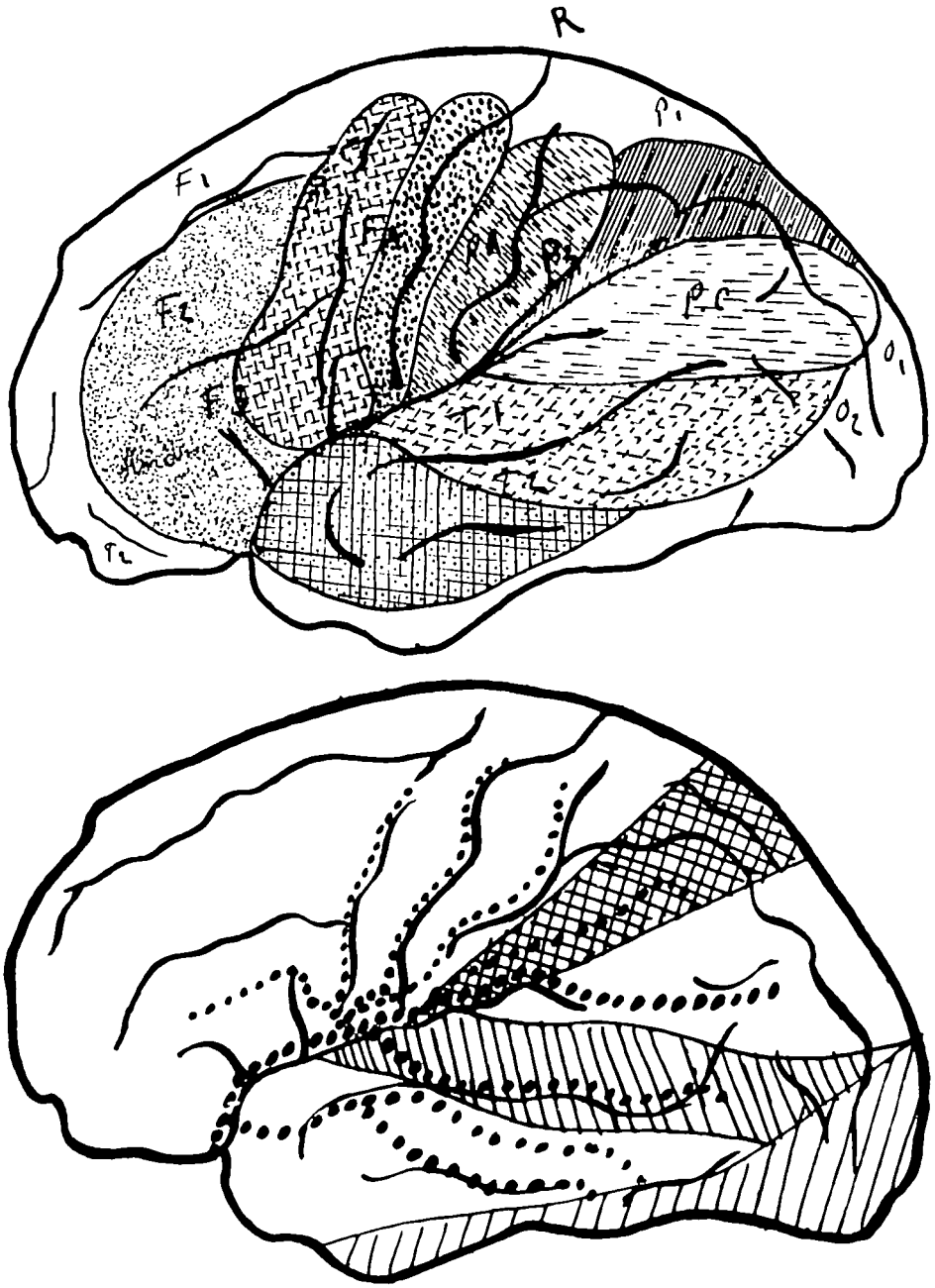


FIGURE 12. Top: *Schema of territory supplied by each sylvian branch, from Reference 20, p 33. F1, F2, and F3 refer to frontal gyri; P, parietal gyri; T, temporal gyri; O, occipital gyri.* Bottom: *Territories (hatched) of supply (from superior to inferior) are posterior parietal, superior temporal, and posterior temporal, from Reference 20, p 26.*

for Foix to apply the methods of his teachers to brain ischemia. In Foix's case, the early findings were clearly pathologic and anatomic. Clinical correlation was stimulated from the laboratory findings. From then on, clinical, anatomic, and pathologic works were pursued concurrently and much time was spent in the clinic and in the laboratory. Foix, in all of his work regarding higher cortical function and abnormalities of posture, muscle tone, gait, and reflex function, emphasized clinical phenomena. The clinical findings in stroke patients naturally flowed from his emphasis on clinical phenomenology.

Knowledge of vascular anatomy was relatively rudimentary in the early part of the 20th century, and the subject had little clinical relevance except for stroke. Foix knew the work of Henry Duret,[26,27] who had written on the anatomy of the cerebral arteries, primarily in relation to head trauma and brainstem hemorrhage. Stopford[28] in England had also analyzed the brain vascular supply, but Foix did not refer to this work. By 1920, Osler and Gowers had written about the location, pathology, and clinical findings in patients with large intracranial hemorrhages. Foix wrote very little about cerebral hemorrhage and did not seem very interested in this subject. Before the time of Foix, there were many reports of the clinical findings in patients with focal softenings, especially in the brainstem. Weber's, Benedict's, Wallenberg's, Raymond's, and other syndromes were described during the late 19th and early 20th centuries from

anatomicoclinical correlation of single cases or small series.[28] Many of the classic brainstem syndromes were not caused by vascular lesions,[29] and no one had systematically studied the findings in diseases of single arteries. The interest at the time was in anatomicoclinical correlation, not brain infarction or the nature of vascular disease. At that time there was, of course, no way to visualize the vessels during life and no treatment for patients with brain ischemia.

The major legacy of Foix was his methodology: meticulous anatomic and pathologic examination of blood vessels and brain lesions, detailed clinical descriptions, and correlation of anatomy, pathology, and clinical signs. The pathology laboratory at the Salpêtrière was dedicated to Charles Foix. In my opinion, Foix was the first real stroke neurologist and, in fact, he really has no competition for this designation. During the half century since his untimely death, a good many clinicians and pathologists have followed his methodology. Some examples in France and Europe include R. Escourolle, P. Castaigne, J.C. Gautier, J. Hauw, J. Bogousslovsky, and R. Ross-Russell, while in the United States C.M. Fisher, R. Adams, J. Moosy, J.P. Mohr, L. Caplan, C. Kase, M. Pessin, J.P. Kistler, L.D. DeWitt, C. Helgason, P. Gorelick, D. Hier, and J. Masdeu have followed the methodology of Foix. More recently, clinicoradiologic and radiologic–pathologic correlations have partially replaced and enriched our knowledge, but these have been gained by the same systematic approach. I believe that history will judge the emphasis of the last 25 years on the determination of the prognosis and the management of stroke patients solely in relation to the tempo of illness (TIA, reversible ischemic neurologic deficit, cerebral stroke, etc.) as a sorry detour away from the light that Foix and his followers had seen. Information about prognosis will follow when we all return to careful clinicoanatomicopathological–radiologic correlation of disorders and when treatment is aimed at the pathology and pathogenesis of disease.

As the century draws to a close and the number of physicians and researchers interested in stroke and the technology related to stroke grows and the stroke literature expands almost exponentially, we should acknowledge the contribution of Foix in starting it all. There is still much to learn from reexamining the findings and especially the methods of Charles Foix.



## Acknowledgments

I would like to thank Dr. Orrin Devinsky and the National Institutes of Health Library for making available the photographs and original articles. I also thank Prof. Jean-Jacques Hauw, MD, for his suggestions.

## References

1. Roussy G: Charles Foix (1882–1927). *Rev Neurol* (Paris) 1927;43:441–446
2. Lévy-Valensi: Charles Foix 1882–1927. *Sem Hop Paris* 1927; 3:185–189
3. Guillain G: L'oeuvre Neurologique de Charles Foix, in *Etudes Neurologiques.* Paris, Masson et Cie, 1933, ser 5, pp 439–458
4. Vinchon J: L'oeuvre poétique de Charles Foix. *Aesculape* 1927;17:241–251
5. Foix C, Nicolesco J: *Les noyaux gris centraux et la région mésencéphalo-sous-optique.* Paris, Masson et Cie, 1925
6. Sicard JA, Foix C: L'albumino réaction du liquide céphalorachidien; dissociation albumino-cytologique au cours des compressions rachidiennes. *Presse Med* 1912;20:1013–1014
7. Foix C, Alajournine T: La myélite nécrotique subaiguë. *Rev Neurol* (Paris) 1926;42:1–42
8. Hillemand P: Charles Foix (1882–1927) anatomical studies. *Ann Anat Pathol* (Paris) 1927;4:530–532
9. Dejerine J, Roussy G: Le syndrome thalamique. *Rev Neurol* (Paris) 1906;14:521–532
10. Foix C, Masson A: Le syndrome de l'artère cérébrale postérieure. *Presse Med* 1923;31:361–365
11. Foix C, Hillemand P: Role vraisemblable du splenium dans la pathogénie de l'alexie pure par lésion de la cérébrale postérieure. *Bull Mem Soc Med Hopitaux Paris* 1925;49:393–395
12. Dejerine J: Contribution à l'étude anatomo-pathologique et clinique des différentes variétés de cécité verbale. *C R Soc Biol* (Paris) 1892;4:61–90
13. Foix C, Hillemand P: Irrigation de la protubérance. *C R Soc Biol* (Paris) 1925;92:35–36
14. Foix C, Hillemand P: Contribution à l'étude des ramollissements protubérantiels. *Rev Med* 1926;43:287–305
15. Foix C, Hillemand P: Les syndromes de la région thalamique. *Presse Med* 1925;33:113–117
16. Foix C, Hillemand P, Schalit I: Sur le syndrome latéral du bulbe et l'irrigation du bulbe supérieur. *Rev Neurol* (Paris) 1925;41:160–179
17. Foix C, Hillemand P: Les artères de l'axe encéphalique jusqu'au diencéphale inclusivement. *Rev Neurol* (Paris) 1925; 41:705–739
18. Fisher CM, Karnes W, Kubick C: Lateral medullary infarction: The pattern of vascular occlusion. *J Neuropathol Exp Neurol* 1961;20:323–379
19. Foix C, Hillemand P: Les syndromes de l'artère cérébrale antérieure. *Encephale* 1925;20:209–232
20. Foix C, Lévy M: Les ramollissements sylviens. *Rev Neurol* (Paris) 1927;43:1–51
21. Foix C, Chavany H, Hillemand P, Schiff-Wertheimer M: Oblitération de l'artère choroidienne antérieure: Ramollissement de son territoire cérébral. Hemiplégie, hemianesthésie et hémianopsie. *Bull Soc Ophtalmol Fr* 1925;27:221–223
22. Geschwind N, Kaplan E: A human cerebral disconnection syndrome. A preliminary report. *Neurology* 1962;12:675
23. Geschwind N: Disconnection syndrome in animals and man. *Brain* 1965;88:237–294, 585–644
24. Helgason C, Caplan LR, Goodwin J, Hedges T: Anterior choroidal artery territory infarction: Case report and review. *Arch Neurol* 1986;43:681–686
25. Foix C, Hillemand P, Ley J: Relativement au ramollissement cérébral à sa fréquence et à son siège, et à l'importance relative des oblitérations artérielles, complètes ou incomplètes dans sa pathogénie. *Rev Neurol* (Paris) 1927;43:217–218
26. Duret H: Sur la distribution des artères nourricières du bulbe rachidien. *Arch Physiol Norm Pathol* 1873;2:97–113
27. Duret HY: Recherches anatomiques sur la circulation de l'encéphale. *Arch Physiol Norm Pathol* 1874:60–91, 316–353, 664–693, 919–957
28. Wolf JK: *The Classical Brainstem Syndromes.* Springfield, Ill, Charles C Thomas Publishing, 1971
29. Stopford JS: The arteries of the pons and medulla oblongata. *J Anat Physiol* 1928;50:225–280

KEY WORDS • cerebrovascular disorders • pathology