Traumatic intracranial aneurysms

PAUL S. LARSON, M.D., ANDREW REISNER, M.D., DANTE J. MORASSUTTI, M.D.,
BASSAM ABDULHADI, M.D., AND JOHN E. HARPRING, M.D.

Department of Neurological Surgery, University of Louisville, Louisville, Kentucky

Traumatic intracranial aneurysms are rare, occurring in fewer than 1% of patients with cerebral aneurysms. They can occur following blunt or penetrating head trauma and are more common in the pediatric population. Traumatic aneurysms can be categorized histologically as true, false, or mixed, with false aneurysms being the most common. These aneurysms can present in a variety of ways, but are typically associated with an acute episode of delayed intracranial hemorrhage with an average time from initial trauma to aneurysm hemorrhage of approximately 21 days. The mortality rate for patients harboring these aneurysms may be as high as 50%. Prompt diagnosis based on arteriography and aggressive surgical management are associated with better outcome than conservative treatment. The authors describe a classification scheme for traumatic aneurysms based on their anatomical location and conclude that 1) post-traumatic aneurysm must be considered in patients with acute neurological deterioration following closed head injury; 2) they can occur following mild closed head injury; 3) they occur more commonly in children than in adults; and 4) surgical clipping and/or endovascular occlusion is the definitive treatment.

KEY WORDS • aneurysm • blunt trauma • intracranial hemorrhage • subarachnoid hemorrhage • traumatic injury

Intracranial aneurysms that develop following closed head injuries present the clinician with both diagnostic challenges and surgical difficulties. Traumatic intracranial aneurysms are rare, comprising 1% or less of all cerebral aneurysms.^{10,27,60,63} They can occur after even mild or seemingly trivial head trauma, and are associated with significant morbidity and a mortality rate as high as 50%.^{14,19,27,44,61} Although found in patients of all ages, intracranial aneurysms are more common in the pediatric population^{12,21,32,38,42,59} and may occur as the result of either blunt or penetrating trauma.^{1,12,27,28,30} Although they occur infrequently, these lesions are well described in the literature.^{1,5,6,8,12,15,17,18,28,49,59}

REVIEW OF TRAUMATIC INTRACRANIAL ANEURYSMS

Causes of Traumatic Intracranial Aneurysms

Traumatic intracranial aneurysms may result from varied causes. They have been reported in association with both blunt and penetrating trauma, with the former being more commonly described.^{12,30,63} Of the penetrating injuries, stab wounds appear to have the highest probability of producing traumatic aneurysms;^{27,28} in one series in-

volving patients with 109 stab wounds to the head, in 11 (14.9%) of the 74 patients who underwent angiography developed posttraumatic aneurysms.²⁸ In contrast, in a series of 223 patients who suffered high-velocity missile injuries and underwent angiography, there were only eight (3.6%) traumatic aneurysms.¹ Some authors have described iatrogenic traumatic aneurysms that developed after a variety of procedures, including endoscopic ventriculostomy, intranasal procedures, intracranial surgery, and repeated subdural taps.^{15,34}

Histological Types

Histologically, traumatic aneurysms can be categorized as true, false, or mixed. True aneurysms involve disruption of the intima and variable involvement of the internal elastic layer and media, which leads to localized weakening of the vessel wall and aneurysm formation with the adventitia of the native vessel intact.^{27,29,60} This phenomenon is presumably secondary to flow dynamics against the weakened vessel wall. False aneurysms are considered to be the most common histological type.^{12,29} These lesions result from disruption of all three layers of the vessel wall with formation of a contained hematoma outside the vessel. A false lumen then develops, creating an aneurysmal dilation.^{6,12,27} These are presumably the histological type associated with penetrating injuries.^{6,25,62} The third histological type, the mixed aneurysm, is initially a true aneurysm that subsequently undergoes a contained rupture forming hematoma and false lumen.^{1,6,12,29,60} Some authors have used the term "mixed aneurysm" to describe saccular aneurysms that occur in association with dissection of

Abbreviations used in this paper: ACA = anterior cerebral artery; CT = computerized tomography; MR = magnetic resonance.

Fig. 1. *Left:* A CT scan revealing acute intracranial hemorrhage and a spherical mass just above the circle of Willis on the left. *Right:* An arteriogram revealing a dissection of the internal carotid artery ending in a large saccular aneurysm of the supraclinoid segment.

the parent vessel (Fig. 1).^{41,45,48,63} The relative incidence of these histological types is not known, as most case reports contain little or no histological data. The histological type is not particularly relevant in terms of clinical management because intervention is required regardless of the type or mechanism.^{12,64}

CLASSIFICATION OF TRAUMATIC INTRACRANIAL ANEURYSMS

Traumatic aneurysms can be classified into 1) those that involve the vessels proximal to the circle of Willis, and 2) those that occur distal to the Circle of Willis (Table 1).

This classification is based on both the anatomy and mechanism of traumatic aneurysm formation. The majority of aneurysms occur in the supraclinoid segment of the carotid artery and along the anterior cerebral artery and its branches, particularly the pericallosal and callosomarginal arteries (Figs. 2 and 3).^{2,8,17,19,32,44,47,50}

Mechanism of Injury

Several mechanisms have been proposed in the formation of traumatic aneurysms, all of which involve either direct injury to the vessel or stretching of the vessel by adjacent forces. The mechanism of injury is closely related to the anatomical location of the involved artery. Intraclinoid carotid and basilar artery aneurysms are commonly associated with basilar skull fractures, which is not surprising given the intimacy of these vessels with the skull base.^{9,33,37,39,46,53,54} In the supraclinoid segment, the carotid artery transitions from a relatively fixed structure in the skull base and cavernous sinus to a relatively mobile structure as it ascends in the cisternal spaces. It is believed that either movement of the supraclinoid segment against the anterior clinoid process or stretching of the carotid artery at this transition zone leads to the formation of an aneurysm.^{49,60,64}

Distal subcortical aneurysms occur predominantly along the anterior cerebral artery and its branches. The

proximity of these vessels along much of their length to the falx cerebri has led some authors to the hypothesis that traumatic movement of the brain and vessels against the relatively fixed falx cerebri can lead to aneurysm formation.^{12,20,40,63} Likewise, posterior cerebral artery aneurysms are thought to be the result of trauma of the vessel against the tentorium.³⁸ Distal cortical aneurysms occur in association with linear or depressed skull fractures and dural lacerations, commonly involving the middle cerebral artery or ACA.^{2,8,12,19,44,63} It is believed that momentary or prolonged herniation of the cortical vessel up into the fracture defect leads to direct injury to the vessel wall.^{22,52}

Clinical Presentation

Traumatic aneurysms are more common in children; in one review the author estimate that 30% of all traumatic aneurysms occur in patients younger 20 years of age.¹² In addition, there appears to be a consistent male predominance, with reported male/female ratios ranging from just over 1:1 to as high as 12:1.^{4,31,36,38,58} Most authors have concluded that this discrepancy reflects a higher likelihood that behavior leads to blunt trauma among males in this age group.³²

Traumatic aneurysms have varied clinical presentations (Table 2). The most common symptoms include an acutely decreased level of consciousness, seizure, or focal neurological deficit. Computerized tomography scanning usually

TABLE 1
CLASSIFICATION OF TRAUMATIC INTRACRANIAL ANEURYSMS

proximal to the circle of Willis
intraclinoid carotid artery
supraclinoid carotid artery
vertebrobasilar
distal to the circle of Willis
subcortical
cortical

Fig. 2. *Left:* A CT scan revealing a large intraparenchymal hemorrhage in the right frontal lobe. *Right:* An arteriogram obtained in same patient, demonstrating an aneurysm on a branch of the ACA.

demonstrates acute intracranial hemorrhage, which may be subarachnoid, intraparenchymal, intraventricular, or subdural.^{15,17,43,47,52} The average time from initial trauma to aneurysmal hemorrhage is approximately 21 days and is associated with a mortality rate as high as 50%.^{14,19,27,44,60} Patients with infraclinoid carotid artery aneurysms can present with cranial nerve deficits, diabetes insipidus, recurrent or massive epistaxis, unilateral blindness, or symptoms of a cavernous-carotid fistula.^{7,11,12,23,24,37,46,49,53,57} Patients with supraclinoid carotid artery lesions can present with headache, memory disturbance, and progressive visual loss prior to rupture; such symptoms have been reported to occur for as long as 7 years prior to diagnosis.^{10,16} At least three cases have presented with hydrocephalus, two involving pericallosal aneurysms and the third with a distal cortical lesion.^{18,51,58}

Distal cortical aneurysms have unique properties that

may make them more likely to be diagnosed prior to the occurrence of hemorrhage. These aneurysms can lead to the development of a growing skull fracture that becomes physically palpable months to years after the injury.^{3,18} Buckingham and colleagues¹² found 11 reported cases of distal cortical aneurysms associated with blunt trauma; seven of these patients (63.6%) presented without hemorrhage and were diagnosed primarily with either on routine radiographic follow up or by evaluation of growing skull fractures. However, only 20.5% (of 44) of blunt traumatic aneurysms were diagnosed prior to hemorrhage in more proximal locations.

Clinicians in the early part of the century did not have the benefit of neuroimaging modalities such as CT and MR imaging. The definitive diagnosis of a traumatic aneurysm could only be made in the angiography suite, in the operating room, or at autopsy.⁵⁶ In the modern era of neuroimag-

Fig. 3. *Left:* A CT scan revealing acute intraparenchymal hemorrhage in the medial left frontal lobe. *Right:* A arteriogram demonstrating a saccular aneurysm of the left callosomarginal artery.

TABLE 2
CLINICAL PRESENTATION OF TRAUMATIC
INTRACRANIAL ANEURYSMS

alteration of consciousness
headache
seizure
focal motor/sensory deficit
cranial nerve deficit
unilateral blindness
proptosis
chemosis
retroorbital pain
epistaxis
diabetes insipidus
palpable growing skull fracture

ing, the use of emergency CT scanning has allowed clinicians to diagnose delayed intracranial hemorrhage due to traumatic aneurysmal rupture more rapidly and more frequently than previously possible. Table 3 represents an overview of the more recent literature regarding traumatic aneurysms following blunt trauma since 1970. Earlier definitive diagnosis in this era seems to be associated with a trend toward more aggressive surgical treatment, and better outcomes are reported than in earlier cases.

Diagnosis

Traumatic aneurysms should be suspected in the setting of acute neurological deterioration following any type of closed head injury. Patients suffering closed head injury should undergo immediate CT scanning, and angiography should be undertaken as soon as possible.^{26,27,29} Patients with history of trauma and recurrent epistaxis, visual loss, progressive cranial nerve palsy or an enlarging skull fracture should also be evaluated with MR imaging/MR angiography, and if a suspicious lesion is found, the patient should be immediately undergo arteriography.^{29,37} Several cases have been described in which patients with normal arteriograms subsequently present with delayed hemorrhage and have aneurysms that are then revealed on repeated studies.^{1,13,28,55} Penetrating injuries, particularly stab wounds, require special consideration, and the authors of several large series recommend routine angiography 2 weeks after the injury to rule out the delayed formation of aneurysm.^{6,28,29}

Treatment Options

The goal of treatment is to exclude the aneurysm from the circulation by surgical or endovascular methods. In 1975, Fleischer and coworkers¹⁹ reported a 41% mortality

TABLE 3
SUMMARY OF CASE REPORTS OF TRAUMATIC INTRACRANIAL ANEURYSMS AFTER BLUNT TRAUMA SINCE 1970*

Authors & Year	Age, Sex	Mechanism of Injury	Presentation	Skull Fracture	Time to Rupture	Location	Management	Outcome
Rumbaugh, et al., 1970	18 yrs, F	MVA	delayed SDH	parietal	7 days	distal MCA	clipped	good
Pathak, 1972	16 yrs, M	MVA	epistaxis	basilar	5 mos	cavernous carotid	observed	died
Benoit & Wortzman, 1973	9 yrs, M	CHI	visual loss	no	7 yrs	supraclinoid carotid	?	?
Thompson, et al., 1973	4 mos, M	CHI	hydrocephalus	no	2 mos	pericallosal	?	?
Amacher & Drake, 1979	10 yrs, M	fall	ICH	no	4 wks	pericallosal	clipped	died
Dharker, et al., 1975	17 yrs, M	MVA	MS change, visual loss, hemiparesis	basilar	5 yrs	supraclinoid carotid	observed	died
Fleischer, et al., 1975	4 yrs, F	MVA	IVH	yes	4 wks	distal ACA	clipped	good
Almeida, et al., 1977	6 mos, M	blunt	growing skull fx	parietal	14 days	distal MCA	clipped	good
Asari, et al., 1977	4 yrs, F	fall	ICH	yes	9 wks	pericallosal	clipped	fair
	6 yrs, F	MVA	asymptomatic	frontal	?	distal ACA	excised	good
Nakamura, et al., 1977	1 yr, F	fall	SAH	no	21 days	cavernous carotid	trapped	poor
Laun, 1979	7 yrs, F	MVA	SAH	yes	12 days	calloso-marginal	wrapped	poor
	4 yrs, F	MVA	SAH	parietal	5 days	distal MCA	excised	good
Endo, et al., 1980	5 mos, M	fall	HCP, growing skull fx	parietal	5 mos	distal MCA	excised	poor
Parkinson & West, 1980	11 yrs, M	MVA	CHI	basilar	none	supraclinoid carotid	none	died
Paul, et al., 1980	8 yrs, M	fall	SAH, IVH	no	18 days	vertebral artery	observed	died
Shallat, et al., 1981	17 yrs, M	MVA	epistaxis, DI, SAH	basilar	21 days	cavernous carotid	carotid ligation	good
Pozzati, et al., 1982	17 yrs, M	MVA	VI nerve palsy, hemiparesis	no	?	supraclinoid carotid	carotid ligation	good
	16 yrs, M	MVA	visual loss, DI	no	26 days	supraclinoid carotid	carotid ligation	good
Nov & Cromwell, 1984	14 yrs, M	MVA	SAH, ICH	no	26 days	pericallosal	observed	died
	4 yrs, F	MVA	SAH	no	14 days	pericallosal	excised	good
Amagasa, et al., 1986	4 yrs, M	ped/MVA	SAH	no	17 days	calloso-marginal	trapped	good
	2 yrs, M	ped/MVA	SAH, ICH, IVH	basilar	20 days	calloso-marginal	wrapped	fair

TABLE 3, CONTINUED
SUMMARY OF CASE REPORTS OF TRAUMATIC INTRACRANIAL ANEURYSMS AFTER BLUNT TRAUMA SINCE 1970*

Authors & Year	Age, Sex	Mechanism of Injury	Presentation	Skull Fracture	Time to Rupture	Location	Management	Outcome
Buckingham, et al., 1988	4 yrs, M 18 yrs, F	fall MVA	headache VI nerve palsy	parietal basilar	3 yrs 9 wks	distal MCA cavernous	excised endo-vascular trapping clipped	good good
Holmes & Harbaugh, 1993	25 yrs, M	MVA	MS change	no	15 days	pericallosal	clipped	good
O'Brien, et al., 1993	23 yrs, M	fall	IVH, seizure, hemiparesis	no	46 days	pericallosal	clipped	good
Lin, 1995	33 yrs, M	MVA	MS change	basilar	32 days	cavernous carotid	trapped	fair
Yazbak, et al., 1995	12 yrs, M 14 yrs, M 2 yrs, M 9 yrs, M 1 yr, F 5 yrs, F	assault MVA MVA blunt assault MVA	ICH ICH ICH MS change growing skull fx SAH	? ? ? ? yes ?	2 yrs 3 mos 6 wks 4 mos 1 yr 2 wks	distal MCA pericallosal distal MCA distal ACA distal MCA pericallosal	excised clipped excised excised excised wrapping	good good ? good good fair
Quintana, et al., 1996	27 yrs, M	assault	MS change, hemiparesis, ICH	clival	none	basilar artery	GDC coiling X 2	good
Gallari, et al., 1997	3 yrs, M	fall	seizure, ICH, IVH	orbital roof	4 wks	pericallosal	clipped	good
Voelker & Ortiz, 1997	14 yrs, M	MVA	resp arrest, ICH, IVH	?	20 days	pericallosal	clipped	fair

* CHI = closed head injury; DI = diabetes insipidus; fx = fracture; GDC = Guglielmi detachable coil; ICH = intracerebral hemorrhage; IVH = intraventricular hemorrhage; MCA = middle cerebral artery; MS = mental status; MVA = motor vehicle accident; ped/MVA = pedestrian injured by motor vehicle; RA = respiratory arrest; SAH = subarachnoid hemorrhage; SDH = subdural hematoma; ? = unknown.

rate in patients treated conservatively as compared with an 18% mortality rate in surgically treated patients. In subsequent case reports the authors have also described poor outcomes in patients who underwent conservative treatment.¹² We agree that aggressive surgical management is the most appropriate treatment.^{6,12,15,27,32} Prompt craniotomy with clipping, resection, or trapping of the aneurysm is considered to be the definitive management.

Endovascular techniques such as trapping, in which detachable balloons or embolization with detachable coils are used, have also been successfully performed in the treatment of traumatic aneurysms.^{12,23,50} Surgical clipping has advantages in that it provides definitive isolation of the aneurysm, allows for the reconstruction of the parent artery if needed, and facilitates removal of mass effect via evacuation of intracranial hematoma and deflation of the aneurysm itself. On the other hand, the use of endovascular therapy avoids prolonged anesthesia, minimizes manipulation of adjacent vessels and structures, and allows diagnostic angiography to be performed throughout the case.³⁵ The wide variation in aneurysm geometry, anatomical location, and comorbid conditions that may be encountered mandates individual consideration as to which technique is most appropriate on a case-by-case basis.

CONCLUSIONS

We conclude that: 1) posttraumatic aneurysms must be considered in patients with acute neurological deterioration after closed head injury; 2) they can occur after mild closed head injury; 3) they occur more commonly in children than adults; and 4) surgical clipping and/or endovascular occlusion are the definitive treatments.

References

- Aarabi B: Traumatic aneurysms of brain due to high velocity missile head wounds. *Neurosurgery* **22**:1056–1063, 1988
- Acosta C, Williams PE Jr, Clark K: Traumatic aneurysms of the cerebral vessels. *J Neurosurg* **36**:531–536, 1972
- Almeida GM, Pindaro J, Plese P, et al: Intracranial arterial aneurysms in infancy and childhood. *Childs Brain* **3**:193–199, 1977
- Amacher AL, Drake CG: The results of operating upon cerebral aneurysms and angiomas in children and adolescents. I. Cerebral aneurysms. *Childs Brain* **5**:151–165, 1979
- Amagasa M, Onuma T, Suzuki J, et al: [Traumatic anterior cerebral artery aneurysms—experiences in 4 cases and review of the literature.] *No To Shinkei* **14**:1584–1592, 1986 (Jpn)
- Amirjamshidi A, Rahmat H, Abbassioun K: Traumatic aneurysms and arteriovenous fistulas of intracranial vessels associated with penetrating head injuries occurring during war: principles and pitfalls in diagnosis and management. A survey of 31 cases and review of the literature. *J Neurosurg* **84**:769–780, 1996
- Araki C, Handa H, Handa J, et al: Traumatic aneurysm of the intracranial extradural portion of the internal carotid artery. Report of a case. *J Neurosurg* **23**:64–76, 1965
- Asari S, Nakamura S, Yamada O, et al: Traumatic aneurysm of peripheral cerebral arteries. Report of two cases. *J Neurosurg* **46**:795–803, 1977
- Bank WO, Nelson PB, Drayer BP, et al: Traumatic aneurysm of the basilar artery. *AJR* **130**:975–977, 1978
- Benoit BG, Wortzman G: Traumatic cerebral aneurysms. Clinical features and natural history. *J Neurol Neurosurg Psychiatry* **36**:127–138, 1973
- Birley JL, Trotter W: Traumatic aneurysm of the intracranial portion of the internal carotid artery. *Brain* **51**:184–208, 1928
- Buckingham MJ, Crone KR, Ball WS, et al: Traumatic intracranial aneurysms in childhood: two cases and a review of the literature. *Neurosurgery* **22**:398–408, 1988
- Burton C, Velasco F, Dorman J: Traumatic aneurysm of a peripheral cerebral artery. Review and case report. *J Neurosurg* **28**:468–474, 1968
- Crowell RM, Ogilvy CS: Traumatic intracranial aneurysms, in Ojemann RG, Ogilvy CS, Crowell RM, et al (eds): *Surgical Management of Neurovascular Disease*. Baltimore: Williams & Wilkins, 1995, pp 377–384
- Dario A, Dorizzi A, Scamoni C, et al: Iatrogenic intracranial

- aneurysm: case report of review of the literature. **J Neurosurg Sci** 41:195–202, 1997
16. Dharker SR, Chandy MJ, Chandy SM, et al: Giant traumatic aneurysm of the intracranial carotid artery (case report). **Neurol India** 23:207–209, 1975
 17. Dial DL, Maurer GB: Intracranial aneurysms: report of 13 cases. **Am J Surg** 35:2–21, 1937
 18. Endo S, Takaku A, Aihara H, et al: Traumatic cerebral aneurysm associated with widening skull fracture. Report of two infancy cases. **Childs Brain** 6:131–139, 1980
 19. Fleischer AS, Patton JM, Tindall GT: Cerebral aneurysms of traumatic origin. **Surg Neurol** 4:233–239, 1975
 20. Gallari G, Chibbaro S, Perra G: Traumatic aneurysms of the pericallosal artery in children. Case report. **J Neurosurg Sci** 41:189–193, 1997
 21. Gerosa M, Licata C, Fiore DL, et al: Intracranial aneurysms of childhood. **Childs Brain** 6:295–302, 1980
 22. Gurdjian ES, Webster JC, Lissner HR: The mechanism of skull fracture. **J Neurosurg** 7:106–114, 1950
 23. Han MH, Sung MW, Chang KH, et al: Traumatic pseudoaneurysm of the intracavernous ICA presenting with massive epistaxis: imaging diagnosis and endovascular treatment. **Laryngoscope** 104:370–377, 1994
 24. Handa J, Handa H: Severe epistaxis caused by traumatic aneurysm of cavernous carotid artery. **Surg Neurol** 5:241–243, 1976
 25. Harvey FH, Downer JL: Traumatic production of an intracranial berry-like aneurysm in a monkey. **Acta Neuropathol** 31:263–266, 1975
 26. Herman JM, Spetzler RF: MR imaging in carotid artery dissection. **J Neurosurg** 72:987–988, 1990 (Letter)
 27. Holmes B, Harbaugh RE: Traumatic intracranial aneurysms: a contemporary review. **J Trauma** 35:855–860, 1993
 28. Kieck CF, de Villiers JC: Vascular lesions due to transcranial stab wounds. **J Neurosurg** 60:42–46, 1984
 29. Kumar M, Kitchen ND: Infective and traumatic aneurysms. **Neurosurg Clin North Am** 9:577–586, 1998
 30. Laun A: Traumatic aneurysms—survey, in Pia HW, Langmaid C, Sierski J (eds): **Cerebral Aneurysms: Advances in Diagnosis and Therapy**. Berlin: Springer-Verlag, 1979, pp 364–375
 31. Locksley HB: Report on the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage. Section V, Part 1: Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations based on 6368 cases in the Cooperative study. **J Neurosurg** 25:219–239, 1966
 32. Loevner LA, Ting TY, Hurst RW, et al: Spontaneous thrombosis of a basilar artery traumatic aneurysm in a child. **AJNR** 19:386–388, 1998
 33. Loop JW, White LE Jr, Shaw CM: Traumatic occlusion of the basilar artery within a clivus fracture. **Radiology** 83:36–40, 1964
 34. McLaughlin MR, Wahlig JB, Kaufmann AM, et al: Traumatic basilar aneurysm after endoscopic third ventriculostomy: case report. **Neurosurgery** 41:1400–1404, 1997
 35. Martin NA: The combination of endovascular and surgical techniques for the treatment of intracranial aneurysms. **Neurosurg Clin North Am** 9:897–916, 1998
 36. Matson DD: Intracranial arterial aneurysms in childhood. **J Neurosurg** 23:578–583, 1965
 37. Maurer JJ, Mills M, German WJ: Triad of unilateral blindness, orbital fractures and massive epistaxis after head injury. **J Neurosurg** 18:837–849, 1961
 38. Meyer FB, Sundt TM Jr, Fode NC, et al: Cerebral aneurysms in childhood and adolescence. **J Neurosurg** 70:420–425, 1989
 39. Nakamura T, Matsuoka Y, Shuro N: [Traumatic cerebral aneurysm showing remarkable extravasation into the lateral ventricle—report of a case in an infant.] **No To Shinkei** 5:371–378, 1977 (Jpn)
 40. Nakstad P, Nornes H, Huage HN: Traumatic aneurysms of the pericallosal arteries. **Neuroradiology** 28:335–338, 1986
 41. Nass R, Hays A, Chutorian A: Intracranial dissecting aneurysms in childhood. **Stroke** 13:204–207, 1982
 42. Norris JS, Wallace MC: Pediatric intracranial aneurysms. **Neurosurg Clin North Am** 9:557–563, 1998
 43. Nov AA, Cromwell LD: Traumatic pericallosal artery aneurysm. **J Neuroradiol** 11:3–8, 1984
 44. Parkinson D, West M: Traumatic intracranial aneurysms. **J Neurosurg** 52:11–20, 1980
 45. Patel H, Smith RR, Garg BP: Spontaneous extracranial carotid artery dissection in children. **Pediatr Neurol** 13:55–60, 1995
 46. Pathak PN: Epistaxis due to ruptured aneurysm of the internal carotid artery. **J Laryngol Otol** 86:395–397, 1972
 47. Paul GA, Shaw CM, Wray LM: True traumatic aneurysm of the vertebral artery. Case report. **J Neurosurg** 53:101–105, 1980
 48. Pica RA Jr, Rockwell BH, Raji MR, et al: Traumatic internal carotid artery dissection presenting as delayed hemilingual paresis. **AJNR** 17:86–88, 1996
 49. Pozzati E, Gaist G, Servadei F: Traumatic aneurysms of the supraclinoid internal carotid artery. Report of two cases. **J Neurosurg** 57:418–422, 1982
 50. Quintana F, Diez C, Gutierrez A, et al: Traumatic aneurysm of the basilar artery. **AJNR** 17:283–285, 1996
 51. Raimondi AJ, Yashon D, Reyes C, et al: Intracranial false aneurysms. **Neurochirurgia** 11:219–233, 1968
 52. Rumbaugh CL, Bergeron RT, Talalla A, et al: Traumatic aneurysms of the cortical cerebral arteries: radiographic aspects. **Radiology** 96:49–54, 1970
 53. Shallat RF, Taekman MS, Nagle RC: Delayed complications of craniocerebral trauma: case report. **Neurosurgery** 8:569–573, 1981
 54. Shaw CM, Alvord EC Jr: Injury of the basilar artery associated with closed head trauma. **J Neurol Neurosurg Psychiatry** 35:247–257, 1972
 55. Smith DR, Kempe LG: Cerebral false aneurysm formation in closed head trauma. Case report. **J Neurosurg** 32:357–359, 1970
 56. Smith KR, Bardenheier JA III: Aneurysm of the pericallosal artery caused by closed cranial trauma. Case report. **J Neurosurg** 29:551–554, 1968
 57. Spurling RG: **Practical Neurological Diagnosis**. Springfield, IL: Charles C. Thomas, 1960, p 42
 58. Thompson JR, Harwood-Nash DC, Fitz CR: Cerebral aneurysms in children. **AJR** 118:163–175, 1973
 59. Ventureyra EC, Higgins MJ: Traumatic intracranial aneurysms in childhood and adolescence. Case reports and review of the literature. **Childs Nerv Syst** 10:361–379, 1994
 60. Voelker JL, Ortiz O: Delayed deterioration after head trauma due to traumatic aneurysm. **W V Med J** 93:317–320, 1997
 61. Watridge CB, Muhlbaier MS, Lowery RD: Traumatic carotid artery dissection: diagnosis and treatment. **J Neurosurg** 71:854–857, 1989
 62. White JC, Sayre GP, Whisnant JP: Experimental destruction of the media for the production of intracranial arterial aneurysms. **J Neurosurg** 18:741–745, 1961
 63. Yazbak PA, McComb JG, Raffel C: Pediatric traumatic intracranial aneurysms. **Pediatr Neurosurg** 22:15–19, 1995
 64. Yonas H, Dujovny M: “True” traumatic aneurysms of the intracranial internal carotid artery: case report. **Neurosurgery** 7:499–502, 1980

Manuscript recieved October 18, 1999.

Accepted in final form December 10, 1999.

Address reprint requests to: Paul S. Larson, M.D., Department of Neurological Surgery, 210 East Gray Street, Suite 1102, Louisville, Kentucky 40202.