SURGICAL EXPERIENCES WITH ARTERIOVENOUS ANOMALIES OF THE BRAIN*

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It is a matter of record that there is no more formidable lesion dealt with by the neurologic surgeon than the arteriovenous anomalies encountered in the brain. In the first comprehensive work on this subject, Cushing and Bailey\(^1\) were extremely dubious as to whether or not these lesions would ever be amenable to direct surgical therapy. However, as better methods of hemostasis were developed through the evolution of the electro-surgical unit, reports appeared demonstrating the feasibility of therapeutic attack. Dandy,\(^2\) Norlén,\(^3\) Olivecrona,\(^4\) Pilcher,\(^5\) Ray,\(^6\) and Trupp and Sachs\(^7\) have made available their experience with the angiomas.

These abnormal remnants of the rapidly changing embryonic circulation of the developing brain usually come to the attention of the clinician during the late second and third decades of life although they do occasionally become symptomatic before that time. The usual story is one of the development of focal fits which gradually increase in frequency and severity, becoming generalized and often uncontrolled with more than adequate medical therapy. The development of headaches of increasing frequency and severity and, in our experience, repetitive episodes of subarachnoid hemorrhage are the next most common symptoms and signs in that order. Hemorrhage may also be intracerebral and in such an instance usually produces irreversible damage.

The greatest factor facilitating surgical treatment of these lesions has been the introduction and development of angiographic techniques. Precise localization and, most important of all, demonstration of major afferent arterial components are usually clearly defined. This is particularly important when the angioma is situated in the dominant hemisphere or in the depths of the brain in a position not favoring direct attack upon the lesion. Routine roentgenograms of the skull may frequently demonstrate an associated anomalous blood supply to the skull itself associated with the underlying vascular anomaly of the cortex (Fig. 1). These lesions also have a characteristic defect demonstrable by pneumography. Olivecrona\(^4\) has pointed out the importance of focal atrophy demonstrated by air study in the absence of other roentgen findings, particularly when the evidence for atrophy is associated with focal neurological signs compatible with such a lesion. In this group of cases air study in most instances was undertaken without the true nature of the lesion being suspected. Aside from the atrophy

as mentioned, these lesions present a typical bizarre type of deformity (Figs. 4, 5, 10, and 13).

Our experience with the arteriovenous anomalies of the brain is represented by the following 18 cases.

REPORT OF CASES

Case 1. C.P. was a 30-year-old white male admitted to the University Hospital on April 25, 1939 with the complaints of headache increasing in frequency and severity and Jacksonian fits involving the left hand and leg, the latter having begun 1 year previously, increasing in frequency and severity. Pertinent findings were bruit over the right parietal eminence and low-grade papilledema. A right parietal flap was turned down revealing an extensive angiomatous deformity on the surface of the right parietal lobe. Multiple surface ligations were done with silk and silver clips. Electrocoagulation of the available vessels was carried out. No attempt was made to dissect out the anomaly. A left hemiparesis was present postoperatively; however, the bruit was entirely gone and was still absent 2 months later. The hemiparesis completely disappeared and has not recurred. He continues to have an occasional seizure.

Case 2. B.C. was an 8-year-old white female admitted on Oct. 6, 1939 with a story of persistent headaches and vomiting since infancy. There was a bluish discoloration over the left frontal bossa. There had been numerous nosebleeds. One year previously the patient had had a subarachnoid hemorrhage. Two weeks prior to admission she had onset of severe, almost constant, right Jacksonian fits, recurrence of severe nosebleeds, and gradual lowering of consciousness and development of a right hemiparesis. There was a definite tumescence of the left frontal bossa, the eyelids, and the left side of the nose. A tangle of abnormal vessels was demonstrable in the retinal circulation on the left. There was a right hemiparesis. On Oct. 26, 1939 ligation of the left common and external carotid arteries was done and on Nov. 13, 1939 the left middle cerebral artery was partially occluded with silver clips. Her symptoms remained more or less unabated. She became progressively worse. Her hemiparesis became a total hemiplegia, headaches increased and fits persisted. She had become more or less vegetative as far as her mentality was concerned. She was last seen on Dec. 16, 1946, a helpless invalid.

Case 3. C.W. was a 37-year-old white male admitted on Oct. 29, 1941 with the story that he had had headaches as long as he could remember and during the 3 preceding years had had onset of left Jacksonian attacks involving both the arm and leg and visual disturbances in the left homonymous field. The fits could not be controlled with medication. Pertinent findings were a mild left hemiparesis, a definite bruit over the right occipital parietal region, and a left homonymous hemianopia. Routine x-rays of the skull demonstrated a typical calcified lesion (Fig. 1). On Oct. 30, 1941, a total ligation of the right internal carotid artery was done. He was last seen on Dec. 6, 1941. There was a marked diminution of bruit; hemianopia and hemiparesis were still present. Headaches were absent and the fits were better controlled on medication. Attempts at follow-up have been unsuccessful.

Case 4. W.W. was a 30-year-old white male admitted on Dec. 17, 1942, having
had Jacksonian fits involving the arm and leg on the right, and headache, increasing in frequency and severity, for 6 years. Seizures were uncontrolled by medication. There was a bruit over the left carotid artery behind the angle of the jaw. An arteriogram on Dec. 24, 1943 demonstrated the lesion seen in Fig. 3, and on Jan. 14, 1943 ligature of the common, external and internal carotid arteries was done. There were no complications to this procedure. He was last seen on May 24, 1943. He had had only 4 fits in the interim and was very well controlled on medication. Follow-up to date has been unsuccessful.

**Fig. 1. Case 3.** Routine lateral exposure showing excessive vascularization of skull and calcification at right parieto-temporal-occipital confluence.

**Case 5.** O.M. was a 12-year-old white male admitted on Dec. 19, 1944. One week previously he had sudden onset of coma and a right hemiparesis; both gradually improved with the exception that the foot and lower leg were still paretic. Subarachnoid and intracortical hemorrhage was diagnosed. Routine x-rays of the skull were normal. An arteriogram demonstrated an anomaly of the left anterior cerebral circulation (Fig. 3). On Jan. 2, 1945 a left pituitary flap was turned down and the left anterior cerebral artery was isolated by silver clips, above the anterior communicating artery. He has been followed intermittently since that time and has no neurologic residuals.

**Case 6.** W.B. was a 50-year-old white male admitted on Aug. 2, 1945. For 6 months he had had progressive headache and dizziness. The week before admission he had marked increase in his symptoms, had become aphasic and drowsy, and had undergone marked personality change. On examination an expressive aphasia was demonstrated. There was marked weakness of grip in the right hand and he was
quite stuporous. Routine x-rays of the skull were normal. A ventriculogram demonstrated a left posterior frontal space-taking lesion. At operation on Aug. 20, 1945 there was marked xanthochromic staining of the cortex. A walnut-sized subcortical hemorrhage was found associated with an angiomatous deformity of surface vessels. All feeding and draining vessels were ligated and the lesion was totally excised. Postoperative epilepsy ensued but was easily controlled by medication. He has been followed intermittently since that time and has no neurologic residuals.

Fig. 3. Case 5. Anterior and lateral angiograms of left frontal anomaly of the anterior cerebral system. Drainage is through the vein of Galen.
Case 7. C.P. was a 41-year-old white male admitted on Jan. 4, 1946 with an 18 months' history of petit mal fits gradually becoming more severe. One week prior to admission he had a grand mal seizure and had remained confused. On examination the patient was drowsy, complained of photophobia and had marked stiffness of the neck. There was low-grade papilledema. X-ray of the skull revealed calcification in the right posterior inferior frontal region. Ventriculogram demonstrated a space-taking lesion in this region (Fig. 4). A right frontal flap was turned down on Jan. 15, 1946 in such a way as to expose the medial surface of the frontal lobe, where a large arteriovenous angioma was encountered. Several large aneurysmal dilatations were ligated and the entire anomaly was removed following multiple ligations and coagulation. He has no neurologic deficit and has had no more fits although he was given medication for only 2 years postoperatively.

Case 8. R.K. was a 29-year-old white female admitted on July 9, 1946. Three months previously she had had a subarachnoid hemorrhage associated with left hemiparesis which had been complete. As her acute signs subsided the hemiplegia completely disappeared. She had uncontrolled epilepsy on a program of adequate medication. Arteriogram demonstrated a right frontal polar angioma. On July 11, 1946 this lesion was removed in toto. The patient expired suddenly on the 3rd postoperative day. It was believed that the cause of death was massive cerebral edema resulting from injudicious use of parenteral fluids.

Case 9. E.A. was an 18-year-old white male admitted on Oct. 5, 1946. His story was one of increasingly severe and uncontrollable Jacksonian fits involving primarily the left arm and present since age 16. He also had headaches of increasing frequency and severity. Upon admission he was stuporous and had a left hemiparesis. Brain tumor was suspected and ventriculogram demonstrated an anterior parasagittal space-taking lesion (Fig. 5). A bifrontal flap was turned down on Oct. 23, 1946 exposing a large angioma situated on the medial surface of the frontal lobe, running on to the corpus callosum. The right anterior cerebral artery was ligated, producing almost total collapse of the lesion which was then completely removed.
He was last seen on Jan. 12, 1948. He had been on anticonvulsant medication and although there was no neurological deficit, he was having a "light" fit about once a month.

Case 10. G.L. was a 34-year-old white woman admitted on Mar. 18, 1947 with the history of having had Jacksonian fits for several years. One week previously she had become comatose, remaining so for 2 or 3 hours. The coma had been preceded by severe pain behind the right eye. Arteriogram demonstrated the lesion seen in Fig. 6. On Apr. 14, 1947 a left parietal flap was turned down and the lesion was removed in toto. Except for a minor defect in stereognostic sensibility of the right hand, she has no neurological deficit 3 years later. Her seizures are well controlled on a regimen of anticonvulsant medication.

Case 11. L.J. was a 32-year-old white male admitted on Apr. 18, 1948. Two years before, he had had sudden nocturnal onset of intracerebral and subarachnoid
hemorrhage, following which he was totally aphasic and had a right hemiplegia. The hemiplegia cleared completely within 6 weeks; however, the aphasia remained profound although improvement was still gradually occurring at the time of admission. Grand mal epilepsy developed, the fits beginning as a right Jacksonian march involving the face, hand and leg before becoming generalized. They were not controlled on anticonvulsant medication. Angiogram demonstrated a large angioma situated in the left Sylvian fissure (Fig. 7). On Apr. 20, 1948, a left-sided pituitary flap was turned down and by arachnoidal dissection the major afferent trunks to this angioma arising from the left middle cerebral artery were isolated and ligated. The patient has been seen at frequent intervals since discharge. He is still partially aphasic, although speech continues to improve with training. His seizures are now easily controlled with medication.

Case 12. L.B. was a 25-year-old white female admitted on Sept. 2, 1946. She had had 3 episodes of subarachnoid hemorrhage in the preceding 6 years. A right arteriogram demonstrated a midline anomaly situated in the corpus callosum. The patient was having headache at that time but since this could be controlled with mild anodynes no further therapy was advised. She returned 2 years later on Apr. 19, 1948, with the story that her headaches were unbearable and almost constant. She had had 2 episodes of numbness involving the right arm and leg. A left-sided arteriogram was then performed demonstrating again the midline callosal lesion (Fig. 8) with its major afferent components arising from each anterior cerebral artery. On May 3, 1948 a 6-hole flap straddling the midline over the vertex was lifted and the afferent trunks from the anterior cerebral vessels on both sides were ligated. What could be seen of the angioma shrank considerably following ligation of the last of the afferent components. This structure was then further thrombosed with the electrosurgical unit. The patient has been seen frequently since discharge and has had no more

Fig. 7. Case 11. Frontal and lateral angiograms showing huge anomaly of the left Sylvian circulation. Major afferent components arise from the middle cerebral artery. Major drainage is superficial to the superior longitudinal sinus and deep through the vein of Galen.
headache. She had 1 major seizure following surgery. She has been on a minimal program of anticonvulsant medication since without attacks.

Case 13. B.B. was a 40-year-old white male admitted on Sept. 12, 1948. Twelve hours before, he had had sudden onset of right-sided headache associated with temporary loss of consciousness. Upon awakening he had a profound left hemiplegia. He also had bloody CSF, papilledema and a left homonymous hemianopia. X-ray films showed a pineal shift of 2 mm. to the left. An arteriogram demonstrated a
large, deep Sylvian vascular anomaly (Fig. 9), which was approached on Nov. 9, 1948 by turning down a right-sided pituitary flap. By arachnoidal dissection the major afferent components arising from the right middle cerebral artery were exposed and ligated. A good bit of the mass could be seen over the anteromedial portion of the temporal and parietal lobes and was thrombosed and shrunk down with the electrosurgical unit. A spastic left hemiplegia persists. He has, however, had no fits on a program of anticonvulsant medication.

Fig. 10. *Case 14.* Ventriculogram showing bizarre defect created by anomaly in the median fissure on surface of right parietal lobe.

Case 14. E.A. was a 49-year-old white female admitted on Nov. 8, 1948. She had an 18-year history of severe, left-sided headaches and Jacksonian epilepsy manifested primarily by twitching of the right side of the face. Six years after onset her fits became generalized and gradually increased in frequency and severity. Severe pain in the left side of the face had been treated elsewhere by alcohol blocks in the 2nd and 3rd divisions of the trigeminal nerve. Previous to admission her epilepsy had become uncontrolled and the headache constant and severe. On examination she was quite disoriented in all spheres; however, she had no other focal signs. Ventriculography on Nov. 17, 1948 revealed a right parietal parasagittal, space-taking lesion (Fig. 10). On Nov. 18, 1948 a midline parietal, frontal flap was turned down demonstrating a large arteriovenous anomaly of the medial portion of the parietal lobe. All of the large afferent vessels were ligated with silk and silver clips and coagulated. The patient expired on the 33rd postoperative day, never having reacted. Autopsy was refused.

Case 15. B.A. was a 44-year-old Mexican male admitted on Nov. 11, 1948 with
the history of having suffered a subarachnoid hemorrhage 2 months before. He continued to have residual severe, generalized headache, marked blurring of vision, photophobia, and was aphasic. On examination he presented a right homonymous hemianopia and aphasia without other focal signs. Routine x-rays showed increased vascularity over the left side of the skull. There was a pineal shift to the right of about 5 mm. An arteriogram demonstrated an anomaly (Fig. 11). On Nov. 22, 1948 a left parietal flap was turned down exposing a large arteriovenous anomaly. All of the visible afferent components were ligated and block excision of the lesion was carried out. He has been seen many times since. His aphasia has cleared completely, according to Spanish interpreters. His only residual is a persistent right homonymous hemianopic field defect.

**Case 16.** G.W. was a 40-year-old white female admitted on July 19, 1949. She had had headaches for several years, and during the past year they had been increasing in duration, frequency and severity. In the last 6 months she had had 3 subarachnoid hemorrhages with no residuals. An arteriogram (Fig. 12) demonstrated a callosal defect similar to that encountered in Case 12 (Fig. 8). On Aug 2, 1949, a 6-hole flap was elevated from the vertex and the major afferent components from the anterior cerebral vessels were ligated. With the ligation of the last large vessel there was a marked decrease in the volume of the lesion, which was then further thrombosed and shrunk down by the electrosurgical unit. She is without signs or symptoms to date.

**Case 17.** P.T. was a 21-year-old white female admitted on July 28, 1949. She had been seen frequently as an outpatient for 18 months. She had right Jacksonian fits when first seen in October, 1948, which became increasingly more difficult to control with medication. These had come on following onset of severe headaches in January 1946. There were no focal neurological signs. A pneumoencephalogram revealed a bizarre, space-occupying, midline, frontoparietal lesion (Fig. 13). An arteriogram
then demonstrated the arteriovenous anomaly (Fig. 14). On Aug. 9, 1949 a bifrontal flap was elevated exposing a large angioma involving the posterior portion of the left frontal lobe. One or two large abnormal vessels coursing over the superior and medial surfaces of the right hemisphere were ligated with silver clips and thrombosed with the electrosurgical unit. The main lesion involving the left posterior frontal lobe was attacked by individual ligation of all the demonstrable afferent components. Total removal was begun by rolling the lesion toward the midline with
Fig. 14. Case 17. Left lateral angiogram showing large posterior frontal anomaly (indistinct due to rapidity of flow in channels so large) with afferent contribution from anterior and middle cerebral systems. Drainage was entirely through the superior longitudinal sinus.

its connections to the superior sagittal sinus acting as a hinge. Extremely brisk bleeding was encountered during this procedure and for this reason, with the lesion more than half isolated, the wound was packed and closed. On Aug. 18, 1949 the procedure was completed. Bleeding was controlled by silver clips, muscle grafts, and electrothrombosis. In her immediate postoperative period she had profound

Fig. 15. Case 18. Frontal and oblique angiograms showing large left occipital polar anomaly with major feeders arising from both the anterior and middle cerebral system.
hemiplegia and was totally aphasic. During the 1st month her aphasia and hemiplegia disappeared except for a foot drop on the right. Her fits were easily controlled on medication and she returned to work as a secretary. During the last 6 months to date her foot drop has become minimal.

*Case 18.* J.D. was 52-year-old Mexican male admitted on Jan. 11, 1950 with the complaint of headache which was diminishing. In October, 1949 he had had a buzzing in his left ear. On Oct. 31, 1949, he had sudden onset of coma persisting for 15 to 20 days. CSF had been bloody and there was a bruit. Upon awakening he had a complete 6th nerve palsy on the left and severe headache. The bruit was less distinguishable. Upon admission no bruit could be heard. There were no objective findings except suggestive tortuosity of retinal vessels. B.P. was 200/120 and x-ray of the chest revealed bilateral apical tuberculosis of questionable activity. An angiogram demonstrated a large left occipital parietal angioma (Fig. 15). In view of the patient’s age, recent onset and paucity of symptoms, questionably active tuberculosis and hypertension, expectant treatment was felt advisable and the patient was discharged. He returned Mar. 13, 1950, asymptomatic and without further objective signs. He will be followed at 6-month intervals.

**TREATMENT**

Treatment of these lesions is expectant and surgical.

Expectant management is well illustrated by Cases 12 and 18. In each the lesion was demonstrated. However, since objective findings of progressive neurologic deficit, uncontrolled epilepsy, intolerable pain and recurrent bleeding were not present, active surgical attack had and has been deferred.

Subarachnoid bleeding does not appear to have been common in other cases in the literature with the exception of those reported by Wechsler and Gross. Most of our cases have had this complication although in no instance was the effect as devastating as that from the subarachnoid hemorrhage accompanying rupture of an aneurysm. Intracerebral bleeding, however, is crippling. Eleven patients have been subjected to one or more experiences with subarachnoid or intracerebral hemorrhage. They all had headaches of increasing frequency and severity and they all had fits which increased in frequency and severity in spite of adequate medical measures for their control. With the progressive development of any or all of these undesirable signs and symptoms, we believe that surgical treatment is mandatory if irreversible brain damage is to be prevented.

We do not believe that irradiation has a place in the treatment of these lesions since, in order to bring about change in the abnormal vessels, irradiation of sufficient degree to destroy the normal blood supply as well as to bring about changes within brain tissue would be necessary.

The location of the angioma as well as the location of its primary afferent components from the arterial side of the circulation as demonstrated in the arteriogram determines the method of attack.

Basal avertin and intratracheal nitrous oxide, oxygen, and ether is the anesthesia of choice in our experience.
Exposure is facilitated by drainage of cerebrospinal fluid through a lumbar puncture needle and tubing inserted just before positioning on the table. With complete drainage at the time of dural opening, marked increase in brain mobility is obtained.

The polar lesions may be easily isolated, particularly in the frontal and temporal poles, since their major feeders from either the anterior or middle cerebral arteries are easily approached. Following ligation of the main feeder vessels, the efferent elements communicating with the major dural sinuses may be ligated and the lesion dissected completely free by gentle blunt teasing.

Those lesions situated within the midpolar portions of the brain and penetrating deep within the parenchyma present a different problem. Usually their main afferent components arise from the middle cerebral artery and these structures can therefore be approached and ligated by arachnoidal dissection of this vessel, utilizing the pituitary approach to the carotid exposing the middle cerebral artery at its source. If situated within the midportion of the dominant hemisphere or deep in the basal ganglia, no further attempt at removal can be made with discretion other than to coagulate the accessible portions of the angioma with low voltage cutting current thus inducing widespread thrombosis.

The low voltage cutting current has a damped spark which is not hot enough to tear or liquefy the vessel walls or cause them to stick and tear when the instrument is removed. The abnormal vessels can then be smoothly and gently shrunken and totally thrombosed by stroking with the ball tip. Deeply situated lesions can be approached, their afferent components ligated, and the accessible portion of the anomaly thrombosed in this manner.

Lesions occurring on the surface away from the motor strip can be dissected free by careful teasing and individual step-by-step ligation of the afferent arterial components.

It is important to remember that ligation of any but afferent elements to the anomaly in the initial steps of the dissection will result in serious hemorrhage which may almost certainly result in brain damage if not in rapid exsanguination of the patient.

It has been our experience that these lesions can be most effectively dealt with by finger compression technique. If massive hemorrhage occurs the lesion can be milked dry by gentle finger compression and gradually rolled out of the brain bed as rapid ligation of feeder vessels with silver clips is utilized, the efferent venous components being occluded last. In some instances it may be necessary to do the removal in two stages, particularly if hemorrhage has been brisk and voluminous, as in Case 17, in which it was necessary to control the bleeding with packs which were removed on the 9th day and 2nd-stage total excision was done.

Simple ligation of the carotid artery in our experience is worthless since the collateral circulation present in these anomalous vascular structures
is excessive. Case 17 had total ligation of the common, internal, and external carotid vessels with no abatement of the hemorrhage during the 1st stage of the procedure. Common carotid ligation alone can be of no value.

Emphasis upon the use of transfusions of whole blood is important. It is not enough in attacking such formidable lesions as these to have blood available in a bank or in the operating room to be utilized only when signs for its need are too obvious. The primary principle of treatment for shock induced by blood loss is anticipatory prevention. Therefore, transfusion should be instituted before the skull is opened and the rate of administration determined by minute-to-minute evaluation of changes in the vital signs dependent upon peripheral vascular stability.

POSTOPERATIVE MORBIDITY AND MORTALITY

All of these patients operated upon demonstrated an associated postoperative neurologic deficit peculiar to the locus of the lesion. However, with the exception of Case 11 and Case 13 these signs cleared rapidly within 10 days to 6 weeks following operation. It is possible that this deficit represents not so much a sequel to trauma to the brain as it does the result of residual low-grade thrombosis on the venous side of the abnormal circulation with concomitant edema which gradually subsides as resolution of thrombosis occurs.

None of the survivors who could be followed was made worse by operation. Those who had headaches are now free; those who had uncontrolled fits now have none or a negligible number and they are more readily controlled on the usual dosages of anticonvulsant medication. Of more real danger to permanent brain crippling than operation itself is the risk of intracerebral hemorrhage, which had occurred in Cases 11 and 13 preoperatively.

There were 2 deaths in this series. That of Case 8 we felt was avoidable and induced by injudicious use of parenteral fluids in the acute postoperative state. Removal of such a large reservoir and shunt from the circulatory system and the temporary low-grade thrombosis resulting on the venous side of the system probably account for the ease of production of cerebral edema. Case 14 had been moribund for several days preceding admission. Clinically it was felt that death was due to patchy atelectasis with pneumonia and renal failure. Since autopsy was refused it was impossible to determine the exact role of the brain lesion in her demise.

SUMMARY

Experiences with the management of 18 cases of arteriovenous anomaly of the brain have been presented. It is felt that direct surgical attack is the treatment of choice when symptomatology becomes progressive and unmanageable.

The technique of attack is determined by the situation of the lesion with reference to depth, accessibility and its major arterial supply—points demonstrated by the angiogram.
REFERENCES


