Regression in size of arteriovenous anomaly of the cerebrum is rare. We have found only six such instances in the literature. Höök and Johanson described a patient with an arteriovenous anomaly in the left frontoparietal region demonstrated by angiography in 1934. Hemiplegia began suddenly in 1941, but gradually cleared. Between 1941 and 1945 the patient experienced repeated attacks of unconsciousness. Angiography in 1955 showed no evidence of the lesion previously demonstrated. Vascularization was sparse in this region, but the major vessels were well demonstrated.

Norlén described a patient in whom angiography displayed a small arteriovenous anomaly. At operation a considerably larger lesion was removed. Practically the entire arteriovenous anomaly was thrombosed, indicating that this lesion might have been cured spontaneously. The patient previously had sustained intracranial hemorrhage.

Paterson and McKissock reported two cases in which a portion of the arteriovenous anomaly was partially occluded by clot. In one, partial obliteration was demonstrated by serial angiography carried out at an interval of 2 years. In the other case, angiography revealed an occipital arteriovenous anomaly. At operation a large aneurysmal sac filled with clot lay adjacent to the lesion demonstrated by angiography. Both these patients gave histories of previous intracranial hemorrhage.

Krayenbühl described two cases in which the arteriovenous anomaly either disappeared or was greatly reduced after carotid ligation. In the case in which the lesion disappeared the repeat angiogram was made a year after carotid ligation. In the second case carotid ligation was combined with roentgen-ray therapy, and the second angiogram was made 9 years after carotid ligation.

We wish to add to the foregoing cases a case in which regression in the size of an arteriovenous anomaly took place.

REPORT OF CASE

A 46-year-old married woman registered at the Mayo Clinic on Oct. 20, 1950, complaining of headaches. The familial history was not contributory except for the fact that her mother and one sister had had migraine headaches for many years. The patient had undergone several operations: hysterectomy in 1945, removal of a lump from the right breast in 1948, and cholecystectomy in 1949. She had nine living children. There were no other significant aspects of the history.

The patient said she had had frontal headaches for most of her life. Sixteen years before her admission she had first noticed a protrusion above the left eye, in the eyelid. On March 21, 1950, the patient was involved in an automobile accident in which the occipital region of the head struck the back of the car seat. She did not become unconscious, and roentgenograms of the skull showed no fracture. In April, 1950, headaches became more frequent and severe.

* The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.
Between May and September she spent an average of 2 days a week in bed. On Oct. 6, 1950, the patient was suddenly seized by an episode in which her eyes began to stare and turn upward, and she vomited. This episode lasted for only a few seconds. From this date onward she experienced constant headache which involved the occipital portion of the head and extended to the frontal regions bilaterally. She had vomited almost every day since this episode.

General physical findings were essentially normal. Blood pressure was 138 systolic and 74 diastolic, expressed in millimeters of mercury. Results of laboratory studies of the urine and blood were within normal limits. Neurologic examination disclosed nothing significant except a slightly stiff neck. A soft mass was seen in the left upper eyelid; this lesion appeared to be a varicosity. The ocular fundi were normal. Exophthalmometer readings were 15 mm. on the right and 19 mm. on the left. The visual fields were normal. The cerebrospinal fluid was slightly xanthochromic. The value for total proteins was 30 mg. per 100 cc. of fluid and there were 62 lymphocytes and 2000 erythrocytes per c. mm. of fluid. Roentgenograms of the skull disclosed a shadow of calcification about 3 by 5 mm. in the region of the left internal carotid artery. Left carotid arteriography revealed a large arteriovenous anomaly arising at the bifurcation of the anterior and middle cerebral vessels. Interspersed with the tangle of arteries were large venous channels in this region, and one of these venous channels extended forward to the frontal region and filled the varicosity which could be palpated through the upper eyelid on the left (Fig. 1). Surgical treatment was not considered because of the extensiveness of the lesion and because of its presence in the dominant hemisphere. Roentgen-ray therapy was advised as an alternative and was accepted.

The patient was seen for numerous re-examinations. She came to the clinic a second time on Jan. 16, 1951. She said that since she had returned home she had been doing well; she complained only of severe headache on coughing or sneezing. The headaches lasted only several minutes. She said that the headaches were not nearly so severe as they had been prior to her first admission. There were no new neurologic signs. Another course of roentgen-ray treatment was prescribed and carried out.

This patient visited the clinic for the third time on June 7, 1951. She said she had been doing well and that she had had only one episode of severe headache in the interim. There were no new neurologic signs or findings.

The patient visited the clinic for the fourth time on Jan. 28, 1952. She said the severity of the headaches had increased during the few months prior to this registration. However, the headaches were not so severe as they had been prior to her first admission. No new findings were made at this examination.

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**Fig. 1.** (a) Lateral view, left carotid arteriogram, made on Oct. 28, 1950, at the time of the initial visit of the patient. (b) Anteroposterior view made at the same time.
The patient visited the clinic for the fifth time on April 23, 1952. She said she had occasional severe headaches. Examination showed that the dilated vein previously noted in the left upper eyelid was not present. Another course of roentgen-ray therapy was carried out on this visit.

The patient came to the clinic for the sixth time on June 22, 1953. She had experienced attacks of severe headache for some weeks prior to this visit, but there were no new findings.

The seventh visit of the patient took place on Dec. 18, 1953. The headaches had continued, as at the time of her last admission.

She sought medical counsel for the eighth time on Aug. 13, 1954, at which period she was doing well. During the interim she had experienced fewer distressing headaches.

The ninth admission of this patient was on Oct. 17, 1954, at which time she was taken to a hospital in an emergency condition. Her husband said that 10 days prior to this admission

the patient suddenly had been seized with nausea and vomiting and had become unconscious within a few minutes. She was taken to a local hospital, where she remained semiconscious for 3 days. She then improved rapidly. She did not regain consciousness completely until Oct. 10, 1954. On that date she seemed to be well oriented, reporting that she had no headache. However, on the afternoon of that day she again experienced sudden headache, and became drowsy and confused. Her condition remained about the same from that time until her admission to the clinic.

Examination revealed a lethargic but cooperative patient. Tendon reflexes were increased on the left side and Babinski's sign was elicited on the left. Hemiparesis was not present. Spinal puncture was done: the cerebrospinal fluid was clear and colorless. On October 20 a left carotid angiogram was made (Fig. 2). This study showed that many of the vessels noted in the previous angiogram no longer were present. Moreover, the caliber of many of the vessels was smaller and the size and number of arteriovenous sinusoids had markedly decreased. The anterior cerebral artery was shifted slightly to the right. A working diagnosis of intracerebral hematoma was made. The patient gradually improved and was discharged on Nov. 4, 1954. She said she felt well. Neurologic findings at that time were essentially normal.

The patient made a tenth visit for re-examination on Oct. 8, 1958, at which time she said

Fig. 2. (a) Lateral view, left carotid arteriogram, made on Oct. 20, 1954, showing marked regression of arteriovenous anomaly. (b) Anteroposterior view made at the same time.
she had not experienced any of the previous severe headaches since the last visit in October 
and November of 1954. She had no neurologic complaints and the neurologic examination 
disclosed no abnormality. The dilated vein in the left eyelid, noted at the time of the first 
admission and disappearing subsequently, was not evident.

The patient received irradiation therapy on four occasions. Two left lateral fields and a 
left frontal field converging on the lesion were used. At the time of this visit, computed from 
the time of the patient’s first registration at the clinic, she had received a total dose of 3700 
roentgens directed to the vascular lesion.

COMMENT

The foregoing case and similar cases in the literature suggest that the abnormal 
vessels in arteriovenous anomalies may have a propensity for thrombosis which is 
greater than that of adjacent normal vessels. In our case the relief of the severe 
headaches which constituted the presenting symptom abated after what we believe 
was intracerebral bleeding. Of some pertinence to this observation is the fact that 
in four of the six similar cases reported in the literature intracranial bleeding had 
ocurred at one time or another. Thus, it is possible that intracranial bleeding, and 
particularly intracerebral bleeding, is the factor resulting in thrombosis of these ab-
normal vessels.

In Krayenbühl’s cases, in which the arteriovenous anomaly was completely or 
partially obliterated after ligation of the carotid artery, there was evidently no intra-
cranial bleeding between the making of the arteriogram that demonstrated the 
lesion and the arteriogram that demonstrated no abnormal vessels. He assumed 
that the remarkable decrease in blood pressure in the lesion which follows carotid 
ligation (as determined for studies of retinal arterial pressure) may be sufficient to 
produce thrombosis of a large number of vessels of the malformation.

Finally, the possible role of roentgen therapy cannot be denied in our case and in 
one of Krayenbühl’s cases. Our patient noted that the dilated vein present in her 
left eyelid, which was shown to fill with dye at the time the first arteriogram was 
made, disappeared after two of the four roentgen-ray treatments. Whether the 
thrombosis of the superficial vein in the eyelid was accompanied by thrombosis of 
the intracranial lesion cannot, of course, be determined.

REFERENCES

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