ARTERIAL ANOMALIES OF THE SPINAL CORD*

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Vascular anomalies of the central nervous system include both arterial and venous malformations. Although found most frequently in the brain, they have also been encountered in the spinal cord and retina. While the venous and arteriovenous abnormalities have received much attention, those of an arterial nature have rarely been described. A review of such lesions in the spinal cord has disclosed 3 reports. Only in the case of Brasch\(^4\) were satisfactory histopathological studies available. He employed the term, angioma arteriale racemosum, to describe his findings. A designation other than “arterial anomaly” as suggested by Wyburn-Mason\(^14\) is unjustified because the inadequate number of postmortem examinations makes it impossible to define the exact nature of the lesion. Names selected because of a gross resemblance to lesions of the brain variously called angioma arteriale racemosum, cirrroid aneurysm, angioma plexiforme and varix arteriale or aneurysmaticus are lacking in specificity and their use only results in error and confusion.

The paucity of reports is surprising in view of the number of these lesions found at operation for spinal cord neoplasm at The Mount Sinai Hospital. Since 1931, 77 intradural, extramedullary cord lesions were exposed. Of this group, 6 could be classified as arterial anomalies. Five cases have been found in the literature after eliminating those of a venous or arteriovenous character. It is felt that similar lesions almost surely have been observed elsewhere but, unfortunately, have never been described. This presentation may act as a stimulus to help establish the arterial anomalies as a nosological entity separating them from the other vascular malformations.

CASE REPORTS

Case 1. A 40-year-old housewife was admitted June 1946 with a 7-year history of sensations of “electricity” in the left leg, occasionally associated with mild low back pain. During the last 4 years intermittent “cramp-like spasms” of increasing severity radiated from the back to the groin. They were aggravated by walking long distances but unaffected by straining. Her pain was worse during menstruation.

Examination. No gross physical abnormalities were disclosed. There was a tendency to drag the right foot. Deep tendon reflexes were increased in the right lower extremity, where Babinski and Chaddock signs were found. Superficial abdominal reflexes were absent on the right. There was a questionable band of hyperalgesia at

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the level of T12 bilaterally and over L2 on the left. Flexion of lumbar spine was limited. There was minimal tenderness on percussion of lumbosacral spine.

Lumbar puncture revealed an initial pressure of 110 mm. of cerebrospinal fluid; manometrics normal; no cells seen; total protein 38 mg. per cent. Roentgenograms showed only slight narrowing of the lumbosacral interspace. Pantopaque myelography disclosed an obstruction at level of upper border of 1st lumbar vertebra. Manometrics repeated the next day again showed no evidence of block. A myelo-

![Image](image-url)

**Fig. 1.** Myelogram (Case 1) showing curvilinear shadows in the lower dorsal region outlining the anomalous vessels. A. Cisternal myelogram; B. Endolumbar myelogram.

gram, repeated through the cisternal route, showed pantopaque dispersed in a scattered, irregular pattern at the level of lower 3 dorsal vertebrae and 1st lumbar vertebra, suggestive of an arachnoiditis. She was discharged with this diagnosis.

**Course.** The patient's symptoms persisted and she was readmitted July 1947. Attacks of pain in the low back and left thigh occurred more often and were of greater intensity. There were no sphincteric disturbances. Neurological findings were similar to those noted at previous admission.

Manometric studies indicated a partial block. On being repeated 3 days later, this finding was confirmed. Spinal fluid studies were normal.

Pantopaque myelography revealed an almost complete block at the space between the 1st lumbar and 12th dorsal vertebrae. Here, the oil dispersed and trickled down the left side. Oil injected through the cisternal route was delayed temporarily at the 10th interspace where the column dispersed, forming globular and curvilinear shadows (Fig. 1). While the findings at this time were suggestive of an arachnoiditis, varices or a hemangioma of the cord could not be excluded.
Operation. Laminectomy (Dr. Sidney W. Gross). The spines and laminae of the 10th, 11th, and 12th thoracic vertebrae were removed. The dura appeared normal and was opened, disclosing an extensive vascular malformation on the posterior aspect of the cord extending to the right (Fig. 2). Tortuous coils and loops of vessels, measuring up to 3 mm. in diameter and filled with bright red blood, entwined themselves on the surface and among the nerve roots. The spinal cord seen between the vessels appeared normal. In order to provide an adequate decompression, the dura was left open and a flap of fascia and fatty tissue was sutured over the defect.

Course. The patient was almost completely relieved of pain. Six days later the ankle and knee jerks were equal and of normal intensity. Although the Babinski sign was equivocal on the right, the Rossolimo sign was markedly positive. There were no sensory changes. It was not considered advisable to give radiotherapy and she was discharged 11 days postoperative. When seen in April, 1948, she was symptom free except for the appearance of pain during menstrual periods and during heavy exertion. A band of hyperalgesia over L1 bilaterally was the only positive finding.

Case 2.* A 48-year-old Chinese man was admitted October 1947 with a 4-month history of persistent numbness and impaired appreciation of temperature over the entire left lower extremity. Soreness and a “dull pain” between the shoulder blades, of greater intensity on the right, were noted 2 months later. These symptoms were aggravated by activity, and were most severe when the patient was recumbent. About 1 1/2 months before admission, he became severely constipated and could defecate only after enemas. Weakness of the left leg was noted at this time; 2 weeks before admission it spread to the right leg. During the last week, he had great difficulty in voiding, urinating only after prolonged periods and with much stress.

Past history was non-contributory. There were no traumatic incidents.

Examination. There was a soft, non-tender subcutaneous mass, measuring 8 by 6 by 3 cm., in the dorsal region lateral to the spinous process of T5 on the right. It was not fixed to the skin and underlying tissues.

Gait was slightly unsteady, with weakness of right lower extremity. Deep tendon reflexes were increased in both lower extremities, more so on the right. Babinski signs were elicited bilaterally, more actively on the right. Abdominal and cremasteric reflexes were absent. There were no abnormal findings in upper extremities.

* We would like to thank Dr. I. Freiman for permission to present this case.
Sensory examination revealed inconstant, poorly defined levels. There was loss of pain and temperature appreciation from approximately T9 down on the left side including the saddle area.

Spinal fluid pressure was normal; manometrics revealed evidence of a partial block; total protein 50 mg. per cent; no cells.

X-rays of dorsal spine revealed no evidence of intrinsic disease. There was slight hypertrophic spondylitis of lower dorsal vertebrae and scoliosis of upper dorsal spine to the left.

A cisternal myelogram, using 3 cc. of pantopaque, revealed moderate delay in passage of the column of oil at level of 5th cervical interspace. Here it broke up and slowly trickled downwards scattering throughout the lower levels, remaining suspended in particular and curvilinear fashion in upper dorsal regions (Fig. 3). The findings were suggestive of an arachnoiditis or a vascular anomaly.

The suggestion of a Brown-Séquard syndrome persisted with variation in the upper levels of impaired sensation on the left. During the 9 days that preceded operation, the level varied from T2 to T12, being most persistent in upper dorsal regions. Sphincter disturbances were consistently out of proportion to the slight motor deficiency.

Operation. Laminectomy (Dr. Ira Cohen). Removal of spines and laminae of C6, C7, and T1. The region was not unusually vascular. The dura appeared normal. On opening the dura, numerous coiled and intertwined vessels filled with bright red blood were exposed on the surface of the cord, extending laterally between the nerve roots and varying in size from 1 to 3 mm. (Fig. 4). They appeared to be suspended in the subarachnoid space and did not pulsate. There were no venous dilatations. The upper and lower limits of the arterial anomaly extended beyond the operative exposure. The dura was closed and wound sutured in usual manner.

Course was uneventful and patient was discharged 10 days after operation. Neurological findings had persisted with a sensory level at T3 on the right. There were no reflex changes from those noted at admission. He began to void spontaneously on 7th postoperative day and required no further catheterization.

He was given a course of deep x-ray therapy. After a brief period of relief, his symptoms and signs returned and have subsequently been progressive. When seen
6 months after discharge, there was marked increase in weakness of the lower extremities, a sensory level at T2, and sphincteric disturbances were prominent.

Case 3. A 40-year-old shipping clerk was admitted with a 3-month history of progressive weakness of his lower extremities, at first involving the left and later the right. For the past 2 months, he complained of difficulty in starting urination and of constipation of increasing severity. Coldness and burning sensations were present in both feet. There were no other sensory abnormalities. Past history was not contributory.

Examination. There was marked tenderness on percussion over the spines of 4th and 5th lumbar vertebrae. There was weakness of both legs, the right more than the left. Gait was broad based and the patient fatigued rapidly. Knee jerks were hyperactive, more so on the right, and ankle jerks could not be elicited. Babinski sign was present on the right but equivocal on the left. Superficial abdominal reflexes were absent. Sensory findings were inconsistent and varied considerably on repeated examinations. There was a vague level of hypalgesia and hypesthesia bilaterally from approximately T9-T10 down, most evident over the distal portions of the extremities. Vibratory and position sense were undisturbed.

Lumbar puncture revealed normal pressure and dynamics; fluid contained 5 cells/c.mm.; total protein 59 mg. per cent. Roentgenograms of lumbosacral spine revealed no significant findings. Cisternal myelography disclosed a rapid passage through the dorsal canal. Following endolumbar injection, there was temporary delay in flow at level of lower border of 4th lumbar vertebra. When this was overcome, there was no definite deformity in the canal.

Within a 2-week period he developed an almost complete paraplegia. Deep tendon reflexes in lower extremities were no longer elicited and plantar responses were inactive. Sensory examination continued to yield varying results. Vibratory and position sense were found to be impaired by some observers, and a poorly demarcated level of analgesia was defined at level of 1st lumbar segment.

Operation. Laminectomy (Dr. Ira Cohen), on 14th hospital day. Removal of spines and laminae of 11th and 12th dorsal vertebrae. On opening the dura tortuous, dilated adventitious vessels, arterial in nature, were exposed overlying the left lateral surface of the spinal cord and extending caudally over conus, epiconus and roots of the cauda equina. A few similar vascular collections were seen on the right.

Fig. 4. Drawing made from operative sketch of the lesion found in Case 2.
side. A catheter was passed upward and downward for distances of 8–10 cm. without encountering any obstruction. The dura was closed and wound approximated in usual manner.

**Course.** Operation was followed by a complete flaccid paraplegia, cord bladder and rectal incontinence. Sensory levels were still not definite. There was loss of appreciation of pin-prick up to the 11th thoracic dermatomes. Touch, position sense and pallesthesia were markedly impaired. The patient left the hospital 6 weeks after operation, at which time sacral decubitus ulcers and bladder infection were serious problems. There was no further return of function.

He was transferred to the Montefiore Hospital where he died 7 years later. An autopsy was performed and the histopathological nature of the lesion confirmed.

**Case 4.** A 54-year-old man was first admitted in November, 1931. During the preceding 8 years, he had two attacks of “lumbago” which subsided spontaneously. Three months before admission he began to complain of numbness, coldness and progressive weakness in both lower extremities, occasionally associated with sensations of “pins and needles.” Disagreeable “drawing sensations” involving muscles of the posterior aspect of legs and thighs were also present. Three weeks before admission he noted difficulty in starting the urinary stream and 2 weeks later, involuntary contractions of right lower extremity.

**Examination.** There was an area of impaired appreciation of heat and cold with hyperpathia below the level of L3 bilaterally. Plantar responses, abdominal and cremasteric reflexes could not be elicited. Deep tendon reflexes were hyperactive in both lower extremities.

Manometrics revealed no block; spinal fluid protein 49 mg. per cent.

**Course.** The diagnosis of intramedullary cord disease was made and the patient was given non-specific protein therapy with resulting marked recession of subjective and objective sensory changes. The hyperreflexia, however, persisted as did the sphincteric disturbances. He was discharged slightly improved.

Relief was temporary and his symptoms recurred the following month. Occasional episodes of incontinence alternated with the usual picture of urinary retention. Radiotherapy had no beneficial effect. On Mar. 28, 1932 he had an episode of acute retention requiring catheterization. He was unable to completely empty his bladder thereafter. Because of these difficulties, myelography was performed. It revealed partial obstruction at level of 11th thoracic vertebral body.

**Operation.** Laminctomy (Dr. Jefferson Browder) was performed at another institution on May 5, 1932. The report was as follows: “There was found a very extensive angioblastic malformation with and about the conus extending downward over the upper aspect of the cauda equina. This aneurysmal-like formation of blood vessels was made up for the most part of arteries woven into a snarl-like, pulsating mass. Some of these vessels were as large as the radial artery. They seemed to practically fill the intradural space. Because of the extensive involvement, no attempt was made to coagulate them. The dura was left open.”

**Course.** Following operation, there was fecal and urinary retention. Numbness, paresthesias and inability to walk were associated with involuntary contractions of right lower extremity. After a course of radiotherapy in November 1932, involuntary contractions disappeared and motor power improved. In January 1933, a relapse occurred leaving patient completely unable to walk.

He was readmitted to The Mount Sinai Hospital in March 1933, at which time there was paraparesis with involuntary contractions in right lower extremity, hyperesthesia below 3rd lumbar segment bilaterally and anesthesia in perianal region.
Knee jerks were hyperactive. Ankle jerks could not be elicited. Rectal sphincter was relaxed. Urinary retention with a large residua was persistent. He was discharged in June 1933. There was no improvement during this period. No further information was obtainable.

**Case 5.** A 62-year-old woman was admitted March 1941 with a 13-month history of dull pain over the lower spine, accompanied by nocturia and urgency of urination. Constipation was a major problem. One week before admission she was awakened by excruciating pain deep in the right buttock which radiated down the thigh. To relieve her pain, she doused herself. In doing this, she discovered that the perineal region was numb and that there was no sensation in the vagina. By the next morning there was involuntary dribbling of urine and inability to stand on her legs, which were numb and weak.

**Examination.** There was weakness of both lower extremities, more marked in biceps femoris and in dorsiflexors of right foot. Ankle jerks were absent bilaterally. Knee jerks were hyperactive. Neutral plantar responses and active abdominal reflexes were elicited. Vibratory sense was absent up to lower lumbar spine. Position sense was intact. Appreciation of touch, pain and temperature in S3, S4 and S5 dermatomes was absent bilaterally. There was tenderness over lower lumbar spine. Overflow incontinence was present and rectal sphincter was patent.

Clinical diagnosis: Tumor of cauda equina, probably metastatic.

Manometric study disclosed a partial block; no cells in cerebrospinal fluid; total protein 110 mg. per cent. X-rays of dorsolumbosacral spine showed marked osteoarthritis. Myelography disclosed no abnormalities.

**Operation.** Laminectomy (Dr. Ira Cohen). Removal of lamina of T12 and L1. When the arachnoid was opened, many abnormal vessels were seen on the dorsal aspect of the lower end of the cord and the conus. Those overlying the conus were bunched to form a small rosette. A diagnosis of arterial anomaly was made, and dura and wound closed.

**Postoperative course** was smooth but without improvement. Deep radiotherapy was given without any change in her condition.

The patient was seen 10 months later at which time her chief complaints were incontinence of urine and pain in the rectum. She was still alive when last heard from 6½ years after discharge.

**Case 6.** A 56-year-old man was admitted with a 5-month history of feelings of coldness and heaviness in both feet gradually spreading up to the hips, more marked on the left. During this period he experienced weakness in both legs, the left greater than the right. He also noticed that he did not know the position of his legs and had more difficulty in walking at night than in the day.

**Examination** disclosed moderate weakness in both lower extremities with slight atrophy of right calf. There were hyperactive knee jerks and absent ankle jerks. Babinski and Chaddock signs were elicited bilaterally. Fibrillations were present in both calves. Absent vibratory and markedly diminished position sense were found in all toes. Hypalgesia was present over L4, L5, S1 and S2 segments. The saddle area was not affected. The clinical impressions included amyotrophic lateral sclerosis, meningovascular lues, combined sclerosis, and intramedullary neoplasm. Manometric study revealed a partial block. Initial pressure was 150 mm.; final pressure after removal of 10 cc. of clear cerebrospinal fluid was 0; 6 cells; total protein 48 mg. per cent.

X-rays of dorsolumbosacral spine showed moderate hypertrophic spondylitis.
Myelogram showed a disturbance in passage of the iodized oil at level of T11 with dispersal of the stream of opaque material.

Operation. Laminec{}tomy (Dr. Ira Cohen). Removal of spines and lamina of T10 and T11. "Immediately on opening the dura there were seen lying on the posterior surface of the cord, opposite T11, two large tufts of abnormal vessels 1 cm. in diameter in addition to many single strands of supernumerary vessels. It was obvious that we were dealing with an angiomatous anomaly. The dura and wound were closed."

Course. Patient was given deep x-ray therapy. When discharged 2 1/2 weeks later there was marked improvement of power in both lower extremities, otherwise neurological status was unchanged.

He was observed in the follow-up clinic for 22 months. During this time there was improvement in motor power; however, sensory abnormalities persisted. He still complained of pain in the back radiating down both legs.

The following 5 cases taken from the literature are abstracted in brief.

The patient of Brasch was a 61-year-old man with a history of progressive weakness of lower extremities for 2 years. He later became incontinent and complained of pain in gluteal region and legs. There was weakness and absence of deep tendon reflexes in lower limbs. Hypalgesia was found over the thighs, and anesthesia more distally. He died 10 days after admission. Postmortem examination disclosed thickened, tortuous arteries coiled over dorsal and ventral surfaces of cord from D5 to L1 and extending into its substance.

The patient (age not recorded) presented by Balck and Harman had an episode of pain, weakness and sensory changes in lower extremities 4 years before admission. Two years later, a similar episode was followed by a brief remission and sudden relapse, this time associated with urinary retention and pain in cervical region as well as in thighs. His temperature rose rapidly and he became unconscious, losing control of his sphincters. Death followed development of nuchal rigidity and small pupils. Postmortem examination showed evidence of subarachnoid hemorrhage extending into cervical region with destruction of a portion of the lumbar spinal cord. An arterial anomaly was present in this region.

The 3 patients of Wyburn-Mason were 55, 39, and 35 years of age. They were men and all showed symptoms of a progressive or intermittent nature with subsequent evidence of transverse lesions in dorsal region of spinal cord. Symptoms had been present for 8 months, 2 years and 3 years respectively. Manometric study performed in 2 cases disclosed evidence of a partial block in 1; spinal fluid protein was elevated in 2. Roentgenograms of spine were essentially normal. Myelography showed a block at D6 in the 1st case and no obstruction in the 2nd. The lesion was demonstrated at operation in each case, in the 1st and 2nd at middorsal levels and in upper dorsal region in the 3rd.

Of the entire group, 9 were men and 2 were women. The youngest was 35, the oldest 62 and the average age was 54. The occurrence of arterial anomalies about 4 times more frequently in males than in females is similar to that seen in the venous abnormalities and is unlike that of the arteriovenous type in which the incidence is almost equal.14

The symptoms were progressive in nature without remission in 7 cases and were intermittent with gradually developing signs of progression in 4.
In the first group, the duration of the illness was comparatively short, varying from periods of 1 to 8 months to a maximum of 2 years. In the second group, the duration was from 3 to 8 years. In 1 case, progressive symptoms of 1 month's duration were climaxed by a sudden hemorrhage into the subarachnoid space with signs of an acute transverse myelopathy.

Noteworthy was the absence of cutaneous naevi. This is in contrast with the numerous reports of skin hemangiomata in the dermatome distribution of segments of the spinal cord involved by a venous or arteriovenous anomaly. Congenital malformations were nowhere in evidence. A subcutaneous lipoma was found in 1 case. In general, the physical findings have not been significant. In no case was the blood pressure abnormal. Percussion tenderness of the spine overlying the lesion was noted in 2 cases. The neurological examination was characterized by sensory levels that shifted during the same and on subsequent days. Findings of progressive cord damage with final evidence of a complete transverse myelopathy were the presenting features or followed in the wake of recurrent attacks. There was a suggestion of a Brown-Séquard syndrome accompanying the lesion of the cervical cord. Five patients showed signs of a tumor of the cauda equina. The others manifested evidence of intramedullary and extramedullary cord disease at the dorsal levels. Varying signs of root irritation were present. These findings resulted in numerous diagnoses such as tabes, amyotrophic lateral sclerosis, combined sclerosis and, most frequently, neoplasm.

The serology was negative in every case of the present series. Study of the cerebrospinal fluid revealed slight to moderate elevation in the total protein. In the 6 patients of our series, the protein was above normal, reaching a maximum of 110 mg. per cent. Manometric tests disclosed a partial block in 5 cases with no evidence of obstruction in 2. In 4 cases, no information was available. Myelography showed no evidence of block in 2 cases, a partial block in 5 and a complete block in 1. In the 2 cases of our series in which pantopaque was used through both the cisternal and endolumbar routes, fluoroscopy was performed by Dr. W. Newman. It disclosed an interruption in the column of oil at the upper level of the lesions with a subsequent dispersal into small rivulets and droplets whose outlines formed a pattern defining the course of tortuous, convoluted vessels, at the same time vaguely suggesting arachnoid adhesions. Roentgenograms showed with clarity the defects suggested during fluoroscopy. The findings were considered to be pathognomonic of a vascular malformation. This is the only means by which a preoperative diagnosis may be ventured with assurance.

Laminectomy was performed in 9 of the 11 cases. The subcutaneous tissues and muscles were not unusually vascular. The epidural fat was present in normal amounts and no extradural abnormalities were encountered. The dura was of normal color and under no unusual tension. Nothing abnormal could be discerned through it. On opening the dura, the lesion was exposed. It consisted of convoluted, intertwined vessels filled with bright red blood and measuring up to 3 mm. in diameter. The vessels coiled around and be-
tween the nerve roots extending laterally and ventrally, at times filling the entire space between cord and dura. Pulsations were seen in 1 case. Neither the upper nor the lower limits were exposed. The spinal cord seen between the vessels appeared to be of normal size and color and no evidence of cord compression or swelling was observed. No vessels filled with venous blood contributed to the malformation.

The dura was left open in 3 cases and was closed in 4, no record being available in the remaining cases. Two in the present group showed improvement, 1 followed for a period of 2 years after operation, the 2nd for 10 months. In the former, the dura was closed and radiotherapy was instituted. In the latter, the dura was left open and a fat-fascia pellicle graft inserted into the defect. No radiotherapy was given. This patient was relieved postoperatively of the excruciating pain which formerly was her chief complaint.

The lesion occurred with equal frequency in the lumbosacral and dorsal segments of the cord, 1 being found in the cervicodorsal region.

Unfortunately, postmortem examination was performed in but 3 of the entire series. An adequate histopathological study is available only in the case reported by Brasch1 in which the arterial malformation extended from T5 to L1. A greatly enlarged vessel from which the malformation had its origin pierced the dura at T5 and formed numerous coils and loops on the dorsal surface of the cord. On cross section, the enlarged surface vessels showed a concentric medial hypertrophy with preservation of a single intimal layer and a normal adventitia. The tunica elastica was interrupted and fragmented. Numerous small arteries penetrated the cord at its dorsal surface in the region of the posterior roots. These also showed the same medial hypertrophy with occasional obliteration of the lumen by concentric thickening of the tunica muscularis. A well preserved internal elastic membrane normally absent in arteries of this size was prominent.

Multiple small extravasations were present in the lumbar segments of the cord. Specific stains disclosed degenerative changes in the cord substance and included both the grey and white matter, mainly involving the dorsal columns.

In the case described by Balck2 evidence of fresh hemorrhage was discovered on opening the dura. The right half of the cord in the lumbar region was destroyed. On the dorsal surface of the cord, an "angiomatous malformation" was found which extended laterally. The vessels were large and thick-walled. Rupture had occurred with a resultant massive hemorrhage into the subarachnoid space extending up to the cervical region. There was no evidence of malignancy. In the only case of the present series in which autopsy was performed, 7 years after the patient was transferred to the Montefiore Hospital, the arterial nature of the lesion was confirmed.

On a symptomatic basis and by examination, it is impossible to differentiate the arterial anomalies from the venous and arteriovenous malformations. The same fluctuation in symptoms, the varying sensory levels and the evidence of intramedullary and extramedullary cord disease with root
compression may exist in all. The absence of cutaneous vascular naevi in the present group can be of little assistance in the differentiation of the arterial malformations from the others, although its occurrence in association with the venous, the arteriovenous lesions and hemangiomas is quite frequent.\textsuperscript{5,6,7,9,10,14} The presence of a lipoma is also of no aid in differentiation as it may occur in all three vascular anomalies.\textsuperscript{3,7,9}

The evidence of a partial block on a manometric study, the pathognomonic findings on pantopaque myelography and the normal roentgenogram of the spine are shared by all. In conjunction with each other, these findings serve to differentiate this group from cord neoplasm, disseminated sclerosis, inflammatory lesions of the cord, thrombosis of the anterior spinal artery and syringomyelia. The narrow curvilinear shadows in the myelogram distinguish these vascular lesions from spinal arachnoiditis in which droplets and ragged linear collections are suspended in the subarachnoid space.

It is of significance that certain veins of the spinal cord may attain huge varicose proportions and produce symptoms similar to those of the other arterial and venous malformations. They may be distinguished by their unusual size and resultant displacement of iodized oil over a broad serpentine course.\textsuperscript{8}

**DISCUSSION**

The malformations of the spinal cord have been compared to similar lesions found on the surface of the brain. Although a distinctive group has been classified as predominantly arterial, rarely has the histological structure been adequately confirmed. Cushing and Bailey\textsuperscript{6} described a vascular malformation which they preferred to call angioma arteriale. Although the vessels possessed a thickened intima, and a definite internal elastic lamina, they were thin-walled and lacked the muscular layer so prominent in the lesions of the spinal cord. Therefore, it is impossible to consider them to be identical. Because both arteries and veins entered into the primary composition of the lesion in the brain, the name angioma arteriale et venosum as suggested by the same authors would be more exact.

An arterial angioma described by Asenjo and Uiberal\textsuperscript{1} at the base of the brain resembles more closely the anomalous vascular formation of the cord. The presence of arterial and at times of undifferentiated vessels forming both efferent and afferent trunks justifies the name arterial angioma. However, medial thickening, which is the outstanding feature of the cord lesion, is lacking, thereby making it difficult to establish identity. After reviewing the so-called arterial angiomas of the brain, in no case has a malformation with all of the characteristics of the lesion in the spinal cord been found.

In 1 case of an aneurysm of a retinal artery described by Theodore and Bonser,\textsuperscript{13} the optic disc was obscured by a convoluted, serpentine dilatation of the inferior temporal vessel which was definitely arterial in structure and did not pulsate. Nothing further could be determined as the lesion was asymptomatic.

Evidence of hemorrhage is not unusual in cases of arteriovenous anomalies
of the brain and spinal cord.\textsuperscript{3,6,11,12,14} Such hemorrhages undoubtedly contribute to the clinical picture, especially where evidence of massive injury has occurred. The constriction of small vessels by the concentric thickening of the media with eventual occlusion is the probable cause of the fluctuating symptomatology and the persistence of signs of cord injury. It is difficult to evaluate the variation in the signs of root compression following changes in position. A change in the vascular dynamics on standing with probable relief of tension on the nerve roots entwined with the vessels may explain this phenomenon.

Of surgical importance in the exposure of an arterial malformation is the relatively normal state of the subcutaneous tissues and bone overlying the lesion. The dura is neither distended nor discolored. In the venous and arteriovenous malformations, the overlying structures are often excessively vascular and the dura unduly tense transmitting the bluish discoloration of the underlying veins.

Radiotherapy has little effect on the course. Because of the possibility of further occlusion of the small nutrient vessels, it is not considered advisable. Any operative procedure other than decompression would be disastrous in view of the character of the lesion.

**SUMMARY**

Six cases of arterial anomalies of the spinal cord have been presented with a review of the 5 cases already reported. The distinguishing clinical, roentgenographic and histopathological characteristics are discussed and evaluated, due consideration being given to similar vascular malformations in the brain and retina.

**REFERENCES**