PARAPLEGIA AS A COMPLICATION OF SYMPATHECTOMY FOR HYPERTENSION*

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SIX YEARS AGO we encountered paraplegia as a postoperative complication in a patient who had undergone thoraco-lumbar sympathectomy for hypertension. Such a phenomenon was unique in our experience. After a search of the literature and a number of informal inquiries among our colleagues, we were surprised to find that such an occurrence is not as unusual as we had believed. Bassett, in 1948,[1] reporting on his experience with sympathectomy in the treatment of hypertension, stated: "We have had four cases of thrombosis of the anterior spinal artery with resultant permanent residual ischemic myelitis." Poppen, in a personal communication,[5] has stated that, although this complication has not occurred in his own experience, three cases have been brought to his attention in which paraplegia followed thoraco-lumbar sympathectomy for hypertension. Therefore, we have knowledge of eight cases in which such a catastrophe followed an elective operation which has enjoyed wide usage during the past decade.

Dr. Bassett has kindly permitted us to report the clinical notes of two of his cases, and these, along with our own case, make up the body of this communication.

CASE REPORTS

Case 1. J. McC., 54-year-old white male, was admitted to the Mercy Hospital on September 5, 1946, with the complaints of headache, dizziness, and blurred vision in the right eye of 6 months' duration. Five years before he had been told that he had low blood pressure. Ophthalmoscopic examination showed grade 3 retinopathy. Blood pressure readings, taken every hour for 24 hours, showed a range from 180 to 220 systolic, and 90 to 128 diastolic. The electrocardiogram was normal.

On October 10, 1946, left-sided thoraco-lumbar sympathectomy was done, resecting as far cranially as the ninth dorsal ganglion and as far caudal as the first lumbar ganglion. There were several large retroperitoneal lymph glands and the retroperitoneal lymphatics seemed large and thickened, suggestive of an old chronic inflammatory reaction. At no time was any abnormal bleeding encountered, and at the termination of the procedure the blood pressure was 152/100. Postoperative course was entirely uneventful, and on October 17, 1946, the same procedure was carried out on the right side. The greater splanchnic nerve appeared larger than normal. In the course of mobilizing the sympathetic chain, it was noted that there appeared to be a ganglion on the trunk with communicating fibers to the prevertebral plexus. Blood pressure remained normal throughout, and the fall in blood pressure frequently encountered during the second stage of a thoraco-lumbar sympathectomy was not observed in this instance. A kidney biopsy, taken at the second operation, showed benign nephrosclerosis.

His postoperative course was complicated by a wound infection, and he was disoriented for several days after operation. Twelve days after operation blood pressure in sitting position was 160/110, and he complained of dizziness and faintness when standing. On November 3rd, he had a sudden elevation of temperature, was found to have pneumonia, and was placed on penicillin and sulfadiazine. Five days later, redness, swelling, numbness and increased warmth of the hands, and to a lesser extent, the feet, developed. Chemotherapy was discontinued. On the following day symmetrical round and oval shaped areas of derma-

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titis, with an elevated base, appeared on the flexor surfaces of both forearms, associated with some puffiness. The feet were similarly, but less severely, involved. These lesions were thought to be manifestations of penicillin sensitivity and disappeared rapidly on Benadryl therapy after penicillin was discontinued, although swelling of hands and feet persisted for several days.

Iodine-starch sweating test on November 4th showed absence of sweating over the D-9 to L-1 dermatomes.

On November 13th, he was confused and disoriented, and the next day complained of numbness in hands and feet, with weakness in grasping and raising the feet. Examination showed muscle weakness in the extensors of wrists, fingers, and feet, being more marked on right side, with moderate wrist drop. The deep tendon reflexes were diminished in both upper limbs and absent in both lower limbs. Tactile sense was absent over the dorsum of both feet; temperature sense was everywhere reduced, and vibration sense was absent in fingers and toes and diminished in both ankles. It was thought that he had a toxic peripheral neuritis and psychosis. Three days later myocardial failure was noted, along with ecchymoses on trunk, arms and hard palate. The latter were interpreted as a toxic purpura by a medical consultant. On November 18th, a papulo-pustular folliculitis was noted unlike the purpura present elsewhere on the body. The eruption stopped abruptly at the limits of the sympathectomy, and the skin of the sympathectomized area was entirely free of the folliculitis. He remained restless, irrational, confused and incoherent.

On the evening of November 21st, the patient suddenly developed a transverse myelitis with paraplegia and complete loss of all forms of sensation below the 12th dorsal dermatome. The following morning his intercostal muscles were not functioning; he had labored, gasping respirations, and was comatose. Despite digitalization and the administration of cardiac and respiratory stimulants, respiratory failure was progressive and he expired on the evening of November 22, 1946.

Autopsy. The liver weighed 1750 Gm.; heart, 525 Gm.; spleen, 155 Gm.; kidneys, 500 Gm.; right lung, 725 Gm., and left lung, 825 Gm. The bases of both lungs were atelectatic with bilateral hydrothorax. There was a scarred and shrunken kidney on the left, with compensatory hypertrophy of the right kidney and an old infarct. The only other findings outside the nervous system were widespread atherosclerosis and marked hypertrophy of the left ventricle of the heart.

Sections of the spinal cord were stained by Weil and H & E six years after the death of the patient. Abnormal findings were limited to two sections of the upper lumbar cord. The architecture of the cord was not well preserved. The demarcation between gray and white matter was not pronounced and the cord parenchyma tended to fall away from the meninges and the intra-medullary blood vessels. There was displacement and distortion of the anterior horn cells, with suggestive neuron swelling on the H & E sections. No nuclear changes could be demonstrated on the H & E sections. Except for enlarged perivascular spaces in the cord, no blood vessel changes were observed. Sections showed poor affinity of the myelin for Weil's stain. The nerve fibers showed early fragmentation but gitter cell activity was not apparent.

It was thought that the histological changes in the spinal cord were compatible with early acute myelomalacia as seen with vascular thrombosis.

Case 2. E. S., University of Michigan Hospital number 444482, was a 44-year-old white female. She was first seen on May 2, 1944, with an 8-year history of hypertension, marked fatigue, shortness of breath, and severe and persistent headache. In December, 1943, she had had a transient episode of aphasia and transient right homonymous hemianopsia. She had become progressively emotionally unstable and depressed.

Ophthalmoscopic examination showed grade 4 retinopathy with papilledema, hemorrhages and exudates. There was minimal cardiac enlargement, and at bed rest the blood pressure varied between 250/160 and 260/180 mm. Hg. On May 18, 1944, bilateral supradiaphragmatic splanchnicectomy and lower dorsal sympathetic ganglionectomy, D-8 through D-12, was performed bilaterally. The procedure went without incident and was well tolerated.

Upon reacting from the anesthetic, the patient complained of numbness of both lower extremities and inability to move her legs. Examination revealed a level at D-12 with a complete sensory loss except for superficial tactile sensation. Pain and temperature perception were absent below D-12. There was a flaccid paralysis of both lower extremities, and none of the deep tendon reflexes were obtainable in the lower limbs. She was incontinent of urine and feces. Spinal puncture performed at this time revealed normal dynamics, protein, and cell count.

This deficit persisted for approximately 6 months, and then began to regress. Nine months postoperatively the level for loss of pain and temperature sensation had diminished to mid-calf bilaterally. She had regained control of bowel and bladder; knee jerks were present, although diminished, and the ankle jerks were normal. Although

there remained a generalized weakness of all muscle groups of both lower limbs, she was improving rapidly and had learned to walk with the aid of a walker. Blood pressure at that time was 160/100. She has not been seen since that date.

Case 3. M. V. H., 35-year-old white female, University of Michigan Hospital number 541465, was admitted to the University Hospital on April 23, 1944. Hypertension had begun at the age of 24, with her first pregnancy, and blood pressure levels during this 11-year period averaged about 165 to 170 systolic. During December, 1943, her systolic pressure was found to vary between 240 and 250, and the diastolic between 130 and 140. She had been having severe, almost constant, suboccipital headaches for 10 years, but during early 1944 they became constant. She also complained of shortness of breath and intermittent ankle edema.

On examination at the time of admission, blood pressure fluctuated between 240/140 and 260/150. There were no other clinical findings of significance except for grade 3 hypertensive retinopathy with pronounced angiospasm.

On April 23, 1944, bilateral supra-diaphragmatic lower dorsal sympathectomy and splanchnicectomy was carried out, the ganglia being removed from D-8 through D-12. Upon reacting from the anesthetic, the patient complained of inability to move her legs, and on examination, there was loss of pain and temperature sensation up to the umbilicus. Tactile sensation was intact, but there was flacid paralysis of both lower extremities and retention of urine. Spinal puncture at this time revealed normal dynamics, protein and cell count.

The patient was treated symptomatically until May 12, 1944, at which time, at the insistence of the family, exploratory dorsal laminectomy from D-4 to D-8 was done. The spinal cord at the level of the 8th dorsal segment and below was dead white in appearance, with little if any vascularity visible. During the 24 hours preceding laminectomy the patient had loss of superficial tactile sensation to the level of D-12. She remained in the hospital on frame care with catheter bladder drainage, until January 8, 1945, at which time she was discharged to her home. The patient has been heard from intermittently, most recently on January 12, 1951, and is still completely paraplegic to the level of D-12.

DISCUSSION

It is difficult to envision mechanisms whereby paraplegia becomes a complication of thoraco-lumbar sympathectomy. We do not have adequate clinical data on the three cases which came to our attention through Dr. Poppen,[5] to hazard a guess as to the underlying pathological process. In Case 1 of this report, clinical evidence suggested an inflammatory etiology of the spinal symptoms; but the postmortem studies, of limited value because the spinal cord was not sectioned until six years after death, suggested a vascular lesion. Cases 2 and 3, as well as Bassett's other two cases,[1] may be considered as instances of thrombosis of the anterior spinal artery. Such evidence as is available then suggest a vascular mechanism.

In addition to the anterior spinal and posterior spinal arteries arising from the vertebral arteries, the human spinal cord is supplied by from two to 17 anterior root arteries, and by a slightly greater number of posterior root arteries. Segmental arteries supplying the spinal cord arise from: vertebral, ascending cervical, deep cervical, intercostal, lumbar, iliolumbar, lowest lumbar, and lateral sacral arteries.[12] One of the anterior radicular arteries, the *arteria radicularis anterior magna,* is considerably larger than any of the other segmental arteries which supply the spinal ganglia and the spinal cord. This vessel is consistently found in the upper lumbar or lower thoracic region, and when it is occluded in a monkey,[12] there occurs paraplegia with loss of pain perception below the level of the artery. Although some of the anterior radicular vessels may be occluded without event, occlusion of the great anterior radicular artery in humans is not without hazard. There has been reported a case in which the latter vessel was obstructed by metastatic carcinoma with resulting severe myelomalacia.[12] This vessel originates from either the intercostal or lumbar arteries between the eighth thoracic and fourth lumbar segments,[6] and accordingly damage to this artery could occur in

either thoracic or abdominal surgery. In each of the three cases in which clinical details are available, the sensory level was at D-12.

Whether or not the *arteria radicularis anterior magna* is the vessel occluded in these circumstances, it seems reasonable to assume that paraplegia noted immediately upon reacting from the anesthesia for sympathectomy has a vascular etiology. Among the factors which might contribute to vascular insufficiency of the spinal cord in these circumstances are hypotension (as a result of blood loss or chemically induced), arterial spasm, and vascular occlusion due to thrombosis, embolism, or ligature. One might postulate that retrograde thrombosis may lead to spinal cord vascular insufficiency following ligature of an intercostal or lumbar artery. We have no direct knowledge that this occurred in any of the cases under consideration. It seems reasonable that occlusion of one or more lumbar or intercostal arteries will sensitize the spinal cord to vascular insufficiency from one of the above causes. It may be that manipulation of an intercostal or lumbar artery is followed by spasm of that vessel or adjacent vessels. The effect of manipulation of sympathetic nervous tissue on the calibre of nearby blood vessels is not known to us.

Spinal anesthesia was not employed in any of the five cases on which we have clinical details. Case 1 received nitrous oxide, oxygen and ether. Case 2 and 3, as well as Dr. Bassett's other two cases, were anesthetized with Avertin given rectally in doses of 110 mg. per Kg. of body weight, supplemented with nitrous oxide and oxygen inhalation. In the latter four cases, blood pressures were maintained at levels no lower than 120 mm. Hg. systolic by intravenous injections of Ephedrine. It is perhaps significant though these four patients, ages varying between 30 and 40, paraplegic immediately following sympathectomy for hypertension, all were anesthetized with Avertin, a drug which may induce a pronounced fall in blood pressure in hypertensive patients. Possibly significant also is the pressor effect of the intravenous injections of Ephedrine and of the nitrous oxide if its administration were coupled with periods of hypoxia.

In circumstances such as aortic compression and dissecting aortic aneurysm where several of the anterior radicular arteries are affected, spinal cord dysfunction is a very likely sequel. The association of spinal cord symptomatology with dissecting abdominal aortic aneurysm has prompted a number of clinical reports in recent years. In an experimental attempt to reproduce in dogs the spinal cord changes brought about by a dissecting aneurysm of the abdominal aorta, Wagner and Alvarez[10] ligated various combinations of the lumbar arteries bilaterally, as well as the upper abdominal aorta. After encountering no postoperative spinal cord changes in these animals, Wagner and Alvarez[10] then placed their animals in shock by blood-letting immediately prior to ligation of the lumbar arteries or upper abdominal aorta, and under the latter circumstances either a temporary or permanent paraplegia ensued postoperatively.

Lam and Aram[3] reported the case of a 56-year-old male in whom they resected the thoracic aorta because of an aneurysm, and replaced the resected area with an homograft. On the first day following operation there was marked weakness in the lower extremities and hypo-esthesia to pinprick below the level of L-1. In addition to their dramatic case report, these authors described experimental operations on ten dogs in which they ligated and divided all of the intercostal arteries in an endeavor to discover the cause of spinal cord ischemia. One of these ten dogs showed paralysis of the hind limbs, which persisted until the animal was sacrificed two days later, and one other dog showed a very transient paralysis. They then concluded that their

patient's temporary partial paraplegia was due to excessively long periods during which the aorta was clamped. It is interesting though to note that during the time of occlusion of the aorta, their patient's systolic blood pressure had dropped to 70 mm. Hg. despite blood replacement, and when the last aortic clamp was removed, the systolic blood pressure dropped precipitously from 140 mm. Hg. to 40 mm. Hg.

The consideration of a relationship between neurologic deficit following ligation of a vessel and blood pressure at time of occlusion of that vessel was first suggested by the experimental studies of Watts in 1934[11] on the anterior cerebral artery of the monkey. Although pathologic unconsciousness had been the rule following ligation of the left anterior cerebral artery in humans, Poppen[4] was able to avoid this complication in nine cases by maintaining normal blood pressure during ligation of the vessel. Thompson and Rhodes[8] described a similar phenomenon several years later in monkeys; and more recently Thompson and Smith[9] have found that the same circumstances prevail with regard to ligation of the middle cerebral artery. In the above-mentioned experiments, hypotension was induced by letting of blood. Preliminary studies of Thompson and Mosberg[7] suggest that major cerebral vessels may be ligated in animals without severe neurologic defict in the presence of chemically induced hypotension.

Although postoperative folliculitis sparing the denervated areas (Case 1) presents ground for interesting physiologic and dermatologic speculation, we do not plan to develop that theme at this time. Apparently, though, the distribution of the folliculitis attests to the extent of the sympathectomy.

SUMMARY

In at least eight instances, paraplegia has followed sympathectomy for hypertension. Clinical details on three of these cases are here reported. This complication may follow either the Peet or Smithwick operation, and may occur in young people as well as old. The phenomenon may be at least partially reversible or paraplegia may be permanent.

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