HYPERTROPHIC SPINAL PACHYMENINGITIS
WITH SPECIAL REFERENCE TO APPROPRIATE SURGICAL TREATMENT*

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CHARCOT and his pupil Joffroy studied and delineated the clinical picture of hypertrophic pachymeningitis of the cervical spinal canal so accurately that little has been added to our knowledge since then. In 1869 they described a case in the Archives de Physiologie and in 1871 Pierret reported another example from the service of Charcot. In 1873 Joffroy published his classical thesis on the subject under Charcot's direction. They were able to find only 4 cases of this condition that had previously been recognized and reported. The first example was recorded by Abercrombie and the second by Ollivier d'Angers. William Gull in 1858 published the third case, with illustrations of the appearance of the cross-section of the thickened dura mater and the compressed cervical spinal cord. They credited Köhler with a fourth case but review of Köhler's original description reveals that it was the pia and arachnoid that were grown together forming a thick, leather-like covering to the spinal cord and that the dura mater was not involved in this process.

Charcot and Joffroy pointed out that the symptomatology is divisible into three periods. (1) The painful period. The onset is with pain in the neck and back of the head, which soon begins to radiate into the upper extremities. These pains are caused by involvement of the cervical meninges and, in turn, of the posterior cervical spinal roots. At first the pain is remittent but after 2 to 5 months it becomes continuous. It is often associated with stiffness of the neck and is aggravated by movement of the neck. Finally, tingling, like needles and pins, develops in the hands and fingers, and sensibility in the upper extremities is gradually impaired. (2) Atrophic paralysis. The painful period and that of atrophic paralysis in the upper extremities may be combined or clearly separated. In any event the pain occurs first and after a variable interval atrophy and weakness appear in the upper extremities. The distribution of the atrophy is variable. It may appear in one or both upper extremities. The small muscles of the hands are usually involved, but the muscles of the arms are by no means spared. (3) Spastic paralysis. As the disease progresses a spastic paralysis of the lower extremities, with the usual changes in reflexes, appears. Respiratory movements may be interfered with. The bladder and bowels are usually affected and sensory

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changes of variable nature and extent develop. Eventually a severe paraplegia supervenes and the patient becomes bedridden with paroxysmal flexor-defense reflexes.

Charcot and Joffroy recognized that the dura mater posterior to the spinal cord was most severely involved and that the disease usually occurred in the cervical region. They were also aware that it might occur in other parts of the spinal canal and might even involve the intracranial dura mater, particularly that in the posterior fossa. In the latter location the disease might give rise to paralysis of various cranial nerves. They described the thickened dura mater as composed of concentric layers of fibrous connective tissue, similar to the cornea, the vessels increased in number and their walls thickened. They believed that the disease initially is confined to the dura mater but that gradually the spinal roots become inflamed and compressed, and that the involvement tends to extend gradually to the leptomeninges and even to the spinal cord. They noted that the leptomeninges often become thickened and that the dura mater may become adherent to them and to the spinal cord. In addition to evidence of inflammation the spinal cord may develop areas of softening and cavitation secondary to the prolonged and increasing compression.

They clearly distinguished between hypertrophic pachymeningitis and various other diseases of the dura mater. They were well aware of the difference between this condition and hemorrhagic pachymeningitis interna (subdural hematoma), which they frequently found intracranially but seldom intraspinally; between it and pachymeningitis externa which was seen particularly in association with Pott's tuberculosis of the vertebrae; and between it and purulent affections of the internal surface of the dura mater. They also stated clearly that they had been unable to find the cause for hypertrophic pachymeningitis, and that no specific or satisfactory form of treatment had been found.

Since the 1870's little progress has been made and much confusion has been added. The distinct entity that Charcot and Joffroy so clearly described has been largely lost sight of in a welter of confusion with epidural and subdural disorders, particularly epidural granulomas and intradural inflammatory processes secondary to the acute meningitides. Probably the most serious misunderstanding has come from the now nearly universally accepted belief that the disease is almost invariably of luetic origin. This is simply not true. The disease has also occasionally been erroneously attributed to tuberculosis. It would not be possible to state that these infections had never given rise to hypertrophic spinal pachymeningitis but when one reviews the cases so diagnosed he finds that they either occurred before the introduction of the Wassermann reaction (1906) and the discovery of the Treponema pallidum (1905) or rested solely upon the microscopic evidence of a chronic inflammatory process in the dura mater without any positive evidence as to the etiology. In fact in many cases the disease has been attributed to syphilis in spite of negative serological tests on the blood and
spinal fluid and in the absence of any other evidence of syphilis. Joffroy stated very frankly that the etiology was unknown and that the long list of possible causes that had been suggested, including arthritic diathesis and living in a cold damp place, had not been substantiated. It is important to recognize that we are still ignorant of the causative agent and that valuable time should not be lost in administering antiluetic treatment in the absence of positive evidence of syphilis. Nor should surgery be unduly delayed in those occasional cases with evidence of syphilis as even here antiluetic therapy may prove ineffective while proper surgical treatment may bring about improvement. It must be borne in mind that with progression of the disease, the dura mater may become densely adherent to the leptomeninges and the spinal cord making satisfactory separation impossible or very difficult and that eventually changes within the spinal cord, softenings and cystic degeneration, may become irremediable.

The first operation for this disease, according to Kment and Salus, was performed by Scheide in 1901 (reported by Schultze). Since then a number of patients with this condition have been operated upon. In 1931, Kment and Salus reported 3 cases of their own and 16 others that they had been able to find in the literature. Of these 19 patients, 12 were improved or cured. However, as some had been observed for only a short time after operation, often only a few months, no final conclusion as to the results can be drawn from these cases. Furthermore, in some of the cases the disorders were not hypertrophic pachymeningitis of the type described by Joffroy and Charcot, but were instances of slight thickening of the dura mater (2 or 3 mm.) in reaction to an extradural or intradural inflammatory process (e.g. Case 1 of Kment and Salus), or, as in the case of Odin and Runström, were examples of chronic leptomeningitis, not pachymeningitis.

The operations that have been performed have varied from mere incision of the dura mater, to separation of the dura mater from the leptomeninges and spinal cord, and finally to actual extirpation of the involved dura mater. This latter procedure was apparently first recommended but not done by Borchardt in 1912. This extensive procedure is the most logical and has given the best results, although on occasion mere incision of the dura mater has been sufficient to result in marked improvement (Hohlbaum, Case 1; Ricard, Dechaume and Croizat; García Díaz and Buylla).

For over 8 years we have followed the 2 patients we operated upon for this condition. In both of these that portion of the greatly thickened dura mater that lay posterior to the emergence of the spinal roots was removed and the complete recovery that followed the operations has been maintained.

Case 1. #85165. Female, aged 53 years.

Summary. Sharp shooting pains in shoulders and arms, 3 months. Paralysis of right leg, 10 days. Inability to urinate, 3 days. Diminution of sensation below T3; weakness of arms and left leg; total paralysis of right lower extremity; atrophy of muscles of both hands; hyperactive tendon reflexes throughout; absent abdominal
reflexes; bilateral Babinski sign. Negative Wassermann and Kahn tests on blood and CSF. No cells in spinal fluid; total protein 1330 mg. per cent. Colloidal gold curve 0013455555. Obstruction of spinal canal to pantopaque above T2. Laminectomy C3-T2. Grossly thickened dura mater excised. Dramatic postoperative recovery. Continued good health.

History. A.P., a housewife 53 years of age, was referred by Dr. Marie Ortmayer of Chicago and admitted to The Chicago Memorial Hospital on Feb. 23, 1944. For several months she had noted numbness and tingling in both arms to a minor degree but since Dec. 15, 1943 she had suffered from sharp shooting pains which began in the right shoulder and radiated down the right arm. These pains soon spread to the left shoulder and arm. They were very severe and frequently incapacitated her completely. Ten days prior to admission the right leg became weak and she soon became unable to use it. During the last 3 days she had had no urge to urinate and was unable to do so. She had been catheterized repeatedly.

Examination. The cranial nerves were intact. There was diminution of perception of pin prick and light touch below the 3rd thoracic segment, more marked on the right side. Vibratory sense was intact but there was slight diminution in position sense in the toes of both feet. The localization of points stimulated was not accurate on the abdomen or the right lower extremity and the perception of figures written on the skin was totally inaccurate on the legs but good on the palms. There was moderate atrophy of the small muscles of both hands. Both arms were weak, particularly the right. The left leg was fairly strong but the right leg was totally paralyzed. No fibrillations were seen. The tendon reflexes were hyperactive in the arms and legs. The abdominal reflexes were absent. Babinski's sign and all of the supporting signs were positive bilaterally. The neck was not stiff.

Urinalysis was negative. Examination of the blood revealed an anemia of 3,890,000 RBC/c.mm., 12.6 gm. per cent Hb., 7,100 WBC/c.mm., and negative Wassermann and Kahn tests. The spinal fluid contained no WBC; total protein was 1330 mg. per cent; Wassermann's test was negative and colloidal gold curve was 0013455555.

Pantopaque was injected into the spinal canal and its course observed under the fluoroscope as the patient was tilted head downward. Its flow was completely obstructed at the level of the interspace between the 1st and 2nd thoracic vertebrae (Fig. 1). Roentgenograms of the upper thoracic spine showed a slight scoliosis with the concavity toward the right side. Except for some osteo-arthritis changes of the

Fig. 1. Case 1. Pantopaque myelogram showing complete obstruction at the 1st thoracic vertebra.
5th, 6th and 7th cervical vertebrae the bony structure appeared normal. The intervertebral disc between the 6th and 7th cervical vertebrae was narrowed. There was no abnormality of any of the pedicles.

Operation. On Feb. 25, 1944, a laminectomy from the 3rd cervical through the 2nd thoracic vertebra was done. The dura mater was pinkish-grey in color, gelatinous in appearance and grossly thickened. The thickening of the dura mater extended from the upper border of the 3rd cervical to the upper border of the 2nd thoracic vertebra (Fig. 2). The dura mater was excised in two strips measuring 9 cm. in length, and 2 cm. in width; they were 1 cm. thick at the greatest point. The excision extended laterally and forward to the point of emergence of the spinal roots from the dural sac. The greatest thickening lay posterior to the spinal cord but the portion of the dura mater that extended anterior to the spinal nerves was also somewhat involved. The spinal cord appeared to be flattened and moderately indented at several points. The dura mater was not adherent to it. There was considerable troublesome oozing of blood from the cut edge of the dura mater. This was finally controlled and the wound was closed tightly.

Bacteriological Studies. Cultures of the tissue removed proved sterile and tissue injected into guinea pigs gave negative results.

Microscopic Examination. Sections of the abnormal dura mater (Fig. 3) were
HYPERTROPHIC SPINAL PACHYMENINGITIS

composed of dense fibrous tissue heavily infiltrated with lymphocytes and plasma cells intermingled with numerous giant cells of foreign body type. These giant cells varied widely in size but usually contained numerous hyperchromatic nuclei situated at the periphery of the cytoplasm (Fig. 4). There were also fairly well circumscribed accumulations of lymphocytes (Fig. 5) with centrally located giant cells. No necrosis was seen. Although a special search was made, after staining sections with various methods, no organisms of any kind could be found. Sections of the bone of the spinous processes and laminae revealed a few more plasma cells than are usually seen in the bone marrow but no definite abnormality. Both Dr. Otto Saphir and Dr. Percival Bailey regarded this as a granulomatous hypertrophic pachymeningitis of unknown etiology.

Postoperative Course. The patient improved rapidly and was discharged from the hospital on March 18, 1944. She was able to walk and showed more strength in her hands. However, she continued to complain of pains in her neck and shoulders although they were not so severe or incapacitating as they had been before operation. In view of the apparently favorable effect of X-ray therapy in Case 2 (reported below) she was given similar treatment to the cervical area and received 3,250 r., divided into 26 treatments given between April 27 and June 8, 1944. The pains soon subsided. She has been followed at frequent intervals since and was last seen in the

Fig. 3. Case 1. Photomicrograph of hypertrophic dura mater. There are scattered accumulations of inflammatory cells among the fibers of the dura mater. Hematoxylin and eosin, X45.

FIG. 5. Case 1. In one region a dense infiltration of lymphocytes was found. ×340.
Spring of 1951. She is perfectly well. The atrophy of the small muscles of the hands has completely disappeared. There is no weakness anywhere nor are there any sensory changes.

Comment. This is a typical example of hypertrophic pachymeningitis of the dura mater in the cervical region. It presents all of the clinical findings that Charcot and Joffroy described. However, the course was shorter than in their cases or in our Case 2. The prompt recovery of the right leg from a complete paralysis could hardly have been more dramatic.

Whether the subsidence of the radicular pains was aided by the radiation therapy is of course debatable and cannot be solved by either of our 2 cases. Such a possibility is strongly suggested.

Case 2 closely resembles Case 1 except that there was a longer history of pain and discomfort in the area supplied by the involved posterior roots and hypertrophy of the dura mater was in the upper thoracic region.

Case 2. #83629. Male, aged 52 years.

Summary. Intermittent pain in upper back and shoulders, 3 years. Shock-like pain down spine on coughing 1 year. Numbness of trunk and legs, difficulty with bowel and bladder and with walking, 2 to 3 weeks. Sensory loss below T5; spastic paraparesis. Negative Wassermann and Kahn tests on blood and CSF. Complete spinal block; xanthochromic CSF; protein 720 mg. per cent. Laminectomy C7-T5. Hypertrophic granulomatous pachymeningitis; thickened dura mater excised. Practically complete recovery.

History. A.E., a hod carrier 52 years of age, was referred by Dr. M. L. Weinstein of Chicago and was admitted to The Chicago Memorial Hospital on Nov. 14, 1943. The patient had been born in Italy.

For 3 years he had suffered from burning pains and a sense of constriction in his upper back and shoulders. At times he had been free of pain. For 1 year coughing had caused an electric-shock-like pain to radiate down his spine from his upper back. For the last 2 weeks there had been numbness of his body from the upper chest downward, and urinary frequency with trouble in voiding. He had had difficulty in defecating and fecal incontinence for 4 days. During the 3 weeks prior to admission the pain had been constant and severe. His legs had become weak, with increasing difficulty in walking. Finally, he was confined to bed.

A benign chondroma had been removed from the right thumb in 1941. He had had gonorrhea in 1918, influenza in 1920, and a peritonsillar abscess in 1930. He consumed a moderate amount of alcohol and became intoxicated about once a month. He was married. His wife was living and well, and they had four normal children. There had been no miscarriages.

Examination. General physical findings were negative except that the rectal sphincter was completely relaxed, the prostate gland was moderately enlarged and there was a hard immovable mass, 1X2 cm., on his right thumb under an old scar.

Neurological examination disclosed an analgesia and hypalgesia below the level of the 5th thoracic segment, and absence of sense of position in the toes and of vibratory sensibility from the anterior superior iliac spines downward. The cranial nerves were intact. Strength in the arms was normal but there was marked weakness in the legs, greater in the left. There was an increase of resistance to passive manipula-
tion of the legs but not of the arms. The tendon reflexes in the arms were normally active while the knee and ankle jerks were hyperactive. Sustained clonus was present at both ankles. Babinski’s sign and all of the supporting signs were positive. There was no stiffness of the neck and no tenderness of the spine. His gait was hesitant and “bouncing”, and he had marked difficulty in walking.

Urinalysis and blood count were normal. Wassermann and Kahn tests on blood were negative. Total protein of blood serum was 6.7 gm. per cent with 3.7 gm. albumen and 3.1 gm. globulin. Gastric analysis revealed no free hydrochloric acid.

X-ray examination of the cervical and upper dorsal spine revealed no change except that the right pedicles of the 4th and 5th thoracic vertebrae were not as sharply defined as the others. Roentgenograms of the chest showed only pleural thickening at the base on the left side and a marked convexity of the right border of the ascending aorta.

On lumbar puncture the initial pressure was 165 mm. of fluid. This pressure was not altered by compression of the veins in the neck, but abdominal compression caused a rise to 210 mm. The fluid was very xanthochromic; it contained no cells; Pandy’s test was very strongly positive; protein was 720 mg. per cent; Wassermann and Kahn tests were negative. The colloidal gold curve was 0.0000003331.

Operation. On Nov. 18, 1943 a laminectomy of the 7th cervical through the 5th thoracic vertebra was made. The ligamenta flava were directly adherent to the dura

Fig. 6. Case 2. Photomicrograph of hypertrophic dura mater. Clusters of inflammatory cells are scattered among the fibers of collagen of the dura mater. Hematoxylin and eosin, X45.
HYPERTROPHIC SPINAL PACHYMENINGITIS


There was no epidural fat present. The dura mater was greatly thickened and filled the spinal canal. On its posterior surface could be seen indentations where the laminae had pressed against it, and it had bulged posteriorly between them. The greatest thickening was at the level of the 3rd and 4th thoracic vertebrae. The dura mater was first incised in the midline but was too thick and stiff to be reflected laterally to expose the spinal cord. It therefore was excised in two pieces. On each side the excision extended from the midline to the point of exit of the spinal nerves from the dural theca. The dura mater was not adherent to the leptomeningeal structures. There was little if any thickening of the dura mater above the 7th cervical vertebra or below the 5th thoracic, or anterior to the points of emergence of the spinal nerves. The tissue removed measured 1.2 cm. in thickness, 2.0 cm. in width and 7.5 cm. in length in greatest dimensions. Its inner surface was smooth, glistening and greyish in color with numerous vessels visible in it. The spinal cord and nerve roots were obviously compressed but not otherwise abnormal. The wound was closed tightly.

Microscopic Examination. Sections of the material removed (Fig. 6) consisted of much hyalinized connective tissue richly infiltrated with lymphocytes, endothelial leucocytes and plasma cells as well as fibroblastic cells (Fig. 7). In addition to this diffuse infiltration there were more or less well localized groups of similar cells, mostly lymphocytes or plasma cells (Fig. 8). In these circumscribed areas were a number of giant cells some of which appeared to be of the Langhans type while others had centrally placed overlapping nuclei. In none of these foci was there any predominance of endothelial leucocytes. A careful search with special stains for infectious organisms, inclusion bodies or yeast-like structures yielded negative results. The
blood vessels showed thickened walls but no endarteritic or periarteritic changes. There was no necrosis.

In the opinion of our pathologist, Dr. Otto Saphir, these sections gave no evidence of leukemic infiltration, neoplasm, syphilis or tuberculosis. He referred to this as a granulomatous hypertrophic pachymeningitis of unknown etiology.

Postoperative Course. The patient’s recovery was rather slow but progressed steadily, so that he was discharged on Dec. 13, 1943. For a time after leaving the hospital he complained of pains in his shoulders and back. A course of X-ray therapy of unknown amount (1 treatment a week for 9 months) was administered by the referring physician, Dr. M. L. Weinstein, and shortly thereafter these pains subsided. He gradually recovered full control of his legs, bowel and bladder.

![Fig. 8. Case 2. A rather large lymphocytic nodule within the thickened dura mater. ×125.](image)

When he was last examined on Oct. 24, 1952, over 8 years after operation, he was working as a laborer in a steel mill. For the past several years he had been very active and quite well. He had no motor difficulties with his arms and complained only of slight weakness of his right ankle. He suffered little pain except for a mild burning discomfort below the left elbow and along the inner aspect of the left thigh and leg. He no longer had any difficulties with his bowel or bladder. Examination revealed no involvement of cranial nerves, no sensory change of any type, and no muscular weakness or atrophy. All tendon reflexes in both upper and lower extremities were definitely hyperactive. Those in the arms were equal whereas those in the right lower extremity were a little more active than those in the left. Babinski’s sign was elicited on the right side. As he walked he tended to favor his right leg slightly and was not able to hop well on either foot alone. To all intents however, he had made a complete recovery from his previously completely incapacitating disability.
Comment. This seems to be a typical example of hypertrophic pachymeningitis of undetermined etiology. It differs from the usual cervical variety only in that it was located in the upper thoracic region. Clinically it presented the typical first stage of posterior-root irritation with radicular pain, followed after some 3 years by signs of compression of the spinal cord. The evidence of involvement of the anterior motor roots, localized atrophic muscular atrophy, which is such a common manifestation of the cervical form of this disease, was overlooked if it was present in this case. This is probably accounted for by such changes being much less apparent with involvement of the upper thoracic anterior roots.

DISCUSSION

Etiology. It is noteworthy that neither of these 2 patients had any evidence of a syphilitic infection, nor were we able to establish the nature of the infectious agent although the condition appeared to be the result of some low-grade chronic inflammatory process. A number of observers have reported the occurrence of an abnormal colloidal gold curve with hypertrophic pachymeningitis, whether intracranial or spinal in location, even when there were no other evidences of syphilis (Hassin and Zeitlin\textsuperscript{10}—5543211000 and 4555543210; Naffziger and Stern\textsuperscript{20}—0012311000; Lewis, Hall and Bernstein\textsuperscript{17}—555554421). This, of course, is not surprising in view of the gross abnormality of the protein content of the cerebrospinal fluid that is so commonly present. However, in our Case 2 the curve was only slightly abnormal (0000002331) whereas in Case 1 it was grossly abnormal (0013455555). In these two instances the greatest change was in the right side of the curve. This is not typical and has not been the case in any considerable number of other reports.

The cases reported here are typical in their lack of evidence of syphilis. The idea that this condition is always or even commonly the result of syphilis is undoubtedly erroneous and has led to the extensive administration of useless antiluetic therapy and unfortunate delays in appropriate surgical treatment. On the other hand, it appears likely that on rare occasion hypertrophic pachymeningitis does arise as the result of syphilis.\textsuperscript{25}

Pathology. The thickening, or hypertrophy, of the dura mater that occurs in these cases is characterized by a marked proliferation of collagenous fibers which are arranged in parallel rows and infiltrated with cells characteristic of a chronic inflammatory reaction. These are principally lymphocytes and plasma cells. There are also a variable number of gigantic polymorphonuclear cells. Some of these giant cells have their nuclei centrally placed and overlapping each other; others are of the foreign body type with the nuclei forming a ring at the periphery of the cytoplasm. Both types may be present in the same case. There are, in addition, foci in which there are accumulations of these various cells. There are, however, no areas of necrosis and no epithelioid cells. No infectious organisms of any kind can be demonstrated. There is usually an increase in the number of small blood vessels and a thickening of their walls. There is little or no tendency toward a perivascu-
lar arrangement of the inflammatory cells. Other authors have reported changes in the leptomeninges, the nerve roots and the central nervous system but there was no opportunity to make any observations on those tissues in our cases.

Localization. Our Case 1 is characteristic of the cervical form of this disease as originally described by Charcot and Joffroy. Undoubtedly this is the most common single locus for this unusual condition. However, in their original papers Charcot and Joffroy recognized that the same type of pathological change might be found elsewhere in the spinal canal and even in the intracranial cavity, particularly in the posterior fossa.

Case 2 is an instance of the same process occurring in the upper thoracic region. Although such a localization is uncommon a number of authors have recorded similar cases. As one descends the spinal canal the incidence of this disease becomes less, but Odys and Rendu have each recorded instances of hypertrophic pachymeningitis at the thoracolumbar junction and Cassirer found 1 case in which the disease involved the cauda equina. Involvement of the intracranial dura mater has been even less common than spinal involvement. Hassin studied 2 cases with predominantly frontoparietal involvement, and Hassin and Zeitlin made a detailed pathological study of a case in which the dura mater of the posterior fossa and above the tentorium as well as the tentorium itself was involved. Naffziger and Stern reported a case in which the principal involvement was in the posterior fossa but the disorder did extend downward into the cervical spinal canal.

Symptomatology. There is little that one can add to the original description of Charcot and Joffroy. The disease is characterized by radicular pains referable to the site of the disease, muscular weakness and atrophy similarly localized, and muscular weakness and spasticity below the level of the compression of the spinal cord. The bowel and bladder are commonly involved. Changes in sensibility are also the rule. However, the changes are by no means characteristic of this disorder in contradistinction to any other compression of the spinal cord. Spinal (lumbar) puncture usually reveals the presence of an obstruction of the spinal subarachnoid space on Quackenstedt’s maneuver. Where it has been used iodized oil has also demonstrated such an obstruction. It is to be anticipated that in an early case, perhaps during the period of radicular pain, an obstruction of the spinal subarachnoid space need not be present. The protein content of the spinal fluid is usually increased, often to rather high levels, with xanthochromia and, at times, coagulation of the fluid as Babonneix and Voisin have pointed out. Furthermore, the colloidal gold curve is often grossly abnormal but it is in no way typical or diagnostic of the condition.

Treatment. As with intraspinal neoplasms there would seem to be only one form of treatment to be recommended, that is an extirpation of the growth that is compressing the spinal cord. As is shown by the cases presented, that form of treatment is highly satisfactory and not difficult to
accomplish. Naffziger and Stern\textsuperscript{20} have shown the advisability of doing the same thing with similar intracranial lesions. It is not surprising, however, that even though the great bulk of the involved dura mater is excised the patient might continue to suffer from radicular pains. That was true in both of our cases. The spinal nerve roots at their points of emergence from the dural sheath are compressed by the hypertrophic dura mater. Furthermore, they are often involved by the inflammatory process. In both of our cases these pains gradually subsided and have largely or entirely disappeared. Whether the X-ray therapy which was administered postoperatively was influential in ameliorating these pains or whether they would gradually have subsided anyway is something we do not know. Nonetheless, we are convinced that with the severe compression of the spinal cord that is present in these cases, and with the ever-present danger of extension of the inflammatory process to the leptomeninx and to the spinal cord and of ultimate softening and necrosis of the spinal cord, it would be foolhardy to treat these patients only with the X-rays without first decompressing the spinal cord by removing as much of the involved dura mater as possible. It is also obvious that mere incision of the dura mater, with or without freeing it from the underlying leptomeninx and spinal cord, is inadequate surgical treatment.

SUMMARY

Two cases of hypertrophic spinal pachymeningitis, one in the cervical and one in the upper thoracic region, have been reported. Both patients were operated upon, and all of the grossly thickened dura mater lying posterior to the points of emergence of the spinal nerve roots was removed. Both made dramatic recoveries from severe spastic paraplegias. The recovery from the atrophic weakness of the upper extremities in the patient with a cervical lesion was also dramatic. In both instances the recoveries have been maintained for many years.

A study of these cases and of the literature reveals a rather constant clinical picture and the fact that there is no known etiological factor for this condition in the majority of cases. Specifically, this condition is seldom of syphilitic origin. There is no known form of useful treatment except the surgical excision of the hypertrophic dura mater that is compressing the nervous system.

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