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COMPRESSION PARAGLEGIA IN POTT'S DISEASE OF THE SPINE.

Based Upon an Analysis of Seventy-Four Cases.

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ON the third of December, 1877, a little overing to this society a paper on the Paralysis nineteen years ago, I had the honor of presenting of Pott's Disease, Based Upon a Study of Fifty-eight Cases. The paper was published in the Journal of Nervous and Mental Diseases for April, 1878. In the present communication I have endeavored to tabulate cases subsequent to 1877. Since the publication of the first paper I have presented to the Section on Neurology of the New York Academy of Medicine, October 9th, 1895, a paper on Compression Myelitis of Pott's Disease Treated by Large Doses of Potassium Iodide. Within the last few years a decided impetus has been given to the interest in the subject by the publication of a number of cases in which laminectomy has been performed for the relief of the more obstinate paraplegias, but I have left the analysis of these cases to gentlemen who will follow me this evening, and shall endeavor to give you as accurately as possible the facts which I have been enabled to secure by a record of final results obtained without the assistance of operative procedures.

To one who is accustomed to statistical work and to the search for final results, the study of cases operated upon is far from satisfactory. The mere mention of a certain number of cases cured by this operation or that prompts one to look closely into the detailed history of the cases themselves. The observations are not always accurately recorded, the reasons for operation are not sufficiently explicit, and the results themselves correspond very closely with the results obtained by non-operative procedures. I make these statements, not to forestall any discussion of the subject or prejudice any one against the attempts at relief in what are known as desperate cases. All authors who have written upon this subject agree in relegating to laminectomy patients who have failed to get relief from the ordinary methods of treatment, and who seem to be hopelessly incurable. In looking over the literature, I find cases of incomplete paraplegia operated upon, cases that seemed to be already recovering; but there are a certain number where the operation was done with extraordinarily good results. The President of the Neurological Society has seemed desirous of obtaining all the information of a practical nature that is possible, and the standing of the gentlemen who are to follow is a guarantee that the work assigned to them will be well done. We ought, therefore, to know at the close of this discussion just what cases are suitable for operation, in what cases an operation is imperatively demanded, and in what cases an operation will be of no benefit whatever.

The question is often asked whether I still believe in the great value of large doses of potassium iodide, and I shall answer, not by statistics, but in a general way. In the first place, I am not wedded to any one form of treatment. I believe that the potash is an excellent adjuvant, and that it is much more efficient when perfect immobilization of the column is secured. I am also convinced that a case will recover more promptly if these two elements of treatment are combined. As a matter, therefore, of routine practice, I employ the drug in large doses up to the point of tolerance. I employ the Taylor brace in some instances, more frequently, however, the plaster-of-paris corset, with a head spring or chin rest. I sometimes resort to the wire cuirass in very young children, to the Cabot, or Bradford frame at times, and in a great many instances

I employ the Paquelin cautery. The management of an individual case depends largely upon the conditions present. If one can secure rest in bed, on a frame, with traction by the head, the patient, I am convinced, will progress more rapidly. I am still opposed to the use of electricity in any form, and can see no good reason for its employment.

In studying the cases for analysis on this occasion, the temptation to present many by way of illustration is difficult to resist. Of a fair number I have secured pathological information, which, however, is but confirmatory of the work done by many of my predecessors. The consensus of opinion seems to be against the employment of the term compression myelitis, and in favor simply of pressure paralysis. Yet there are instances where a myelitis does exist where cord changes remain permanent and where any form of treatment is necessarily of little avail. If we accept the pressure theory as the best working theory, it is easy to understand how the enlargement of the lumen of the canal by removal of the posterior arches is a commendable operation. The advances made in cerebral and spinal localization enable us to tell pretty accurately just what parts of the column are involved, just what tracts are encroached upon, and it is often possible to determine without an exploratory incision how extensively the cord is diseased.

One of the patients, a girl, twelve years of age, died April 15th, 1896, after a pretty complete paraplegia of six months' standing. She died of tubercular meningitis, and I was fortunate in securing an autopsy conducted by Dr. Henry S. Stearns, who made this report: "The cord just above the curvature shows a sclerosis of all areas of white matter, least in the posterior columns and greatest in anterior columns and direct cerebellar and Gowers' tracts. Moderate in the crossed pyramidal, antero-lateral and mixed lateral tracts. Below the curvature there is much less sclerosis generally, but the posterior columns and the descending antero-lateral tract are almost completely destitute of nerve fibres, while all of the anterior and remainder of lateral columns present only very moderate evidences of sclerosis. These central lesions are decidedly confused as to their distribution, which is partly, at least, accounted for by a considerable amount of productive in-

flammation under the dura mater for about two inches in the region of the curvature. There are also some small patches of new formation, sub-meningeal, above and below the curvature. These latter are isolated, and, of course, would give rise to some of the otherwise unexplainable areas of degeneration and sclerosis."

I am well aware that statistics, as a rule, are uninteresting; that is, for the reader of a paper, but I am sure that their value is appreciated by men who are writing books or cyclopædia articles, and hence I offer no further apology for presenting the data I have secured in statistical form. I have about ten tables, which will be represented in the customary order:

TABLE I.—SEX.

	MALE.	FEMALE.	WHOLE NO.
From recent statistics.....	33.....	41.....	74
From former paper.....	30.....	28.....	58
Total.....	63.....	69.....	132

It will be seen, therefore, that sex offers no predisposition.

In the following table I have endeavored to give the location as nearly as possible, and while the regions are mentioned, it must be remembered that the deformity frequently extends from one region to the other. The location, then, means the region in which the greatest deformity appears.

TABLE II.
Location of Bone Disease.

Cervical	5
Upper dorsal.....	28
Dorsal	41
Lumbar	0
Total.....	74

This table corresponds very closely with some observations I made in 1877 and published in the paper to which reference has already been made. I take the liberty of incorporating the following quotation: "From the notes of 295 cases of Potts' disease, I find sixty-two producing paralysis more or less complete. The number wherein the disease was situated above the mid-dorsal region was 189, and in this group the sixty-two paralytics are included, only three or four being associated with disease involving

the lumber vertebræ, about one-half of the patients that have been affected with caries in the cervical or upper dorsal regions were at one time paralyzed, while nearly one-fifth of the whole number, irrespective of locality, were thus affected."

In citing the cases for the present paper I found a few recorded as paraplegic, and I found a few cases of lumber Potts recorded as paraplegic, but on noting them closer I was able to eliminate the paraplegia altogether. Last notes as to these, "unable to stand," "unable to walk," were found, but the reason for this inability to stand or walk was discovered to be the faulty position of the limbs, by reason of psoas and iliac abscess or psoas contraction, involving one or both limbs. I have carefully tried, therefore, to incorporate those only where the paraplegia was indubitable, as shown by the exalted reflex spasm and inability to move the limbs, even when the patient was sitting or lying down. I am confident that a compression affecting the lumber cord will not produce a genuine paraplegia.

TABLE III.

The Degree of Deformity.

Much deformity.....	49
Little deformity.....	25

This gives one a general idea of the nature of the paraplegia, and helps to disabuse one's mind that the angular shape of the bosse is the prevailing factor in the production of the paralysis. A small number of my cases had no deformity whatever, and, indeed, cases are not at all rare where no deformity whatever existed, and yet the compression paraplegia existed. These have been demonstrated by autopsy.

One of the most aggravated cases that has ever come under my observation was one of this kind. It was in a boy who was admitted to the hospital the 28th of March, 1890. He had been under observation in Out-Patient Department for more than a year prior to this date, suffering from a tuberculous osteitis of the hip. The disease was under very good control. The deformity was very slight, and we had every reason to suppose that the resolution was the legitimate outcome of the treatment. A month prior to his admission he complained of a little pain in his

back, and on examination there was found to be tenderness on concussion of the upper part of the spine, recognized by striking the top of the head as he stood. There was also reflex spasm on moving the head and pain in the occipital nerves. A diagnosis was made of cervical Potts and admission was advised. A day or two after admission it was found that he had a little exaltation in the reflexes, both at knee and ankle, in the sound limb. We at once suspected incipient paraplegia and put him to bed with traction to his head. Sand bags were also applied on either side of the head, so as to give as much rest and protection to the parts as possible. He, nevertheless, developed complete paraplegia. The case progressed from bad to worse, and it became an exceedingly difficult problem how to prevent deformity at the diseased hip and also deformity in the cervico-dorsal region. Reflex spasms became intense, and for two or three years we were obliged to keep him, a greater part of the time, in plaster-of-paris from the axillæ to the balls of the feet. When he was not in plaster, traction was made on the limbs by weight and pulley, and counter-traction at the head, by the same means. It was fully two years, therefore, before any amelioration in his paraplegic symptoms was noted. During this time he suffered from incontinence of urine; occasionally, incontinence of fœces and sacral decubitus. It was next to impossible to apply plaster-of-paris without getting excoriation and finally a wire cuirass was employed, but the spasm was so great that pressure sores were induced. A recumbent posture was maintained all the while, even for one or two years longer, so that his treatment extended over a period of four years. The potassium iodide was employed in large doses. The pressure ulcers would heal under special dressings, and, finally, his paralysis began to recede. Then we had to resort to tenotomy and myotomy and various stretchings under ether or gas in order to get the limbs into good position. It was necessary to perform circumcision on account of a very severe form of ballanitis. After all, I am able to count this case as one among the improved, indeed, I might count it as one among the cured but for the difficulty he had in getting about by reason of the deformity of hip. It is interesting to note that after this prolonged siege there is no deformity in the cervical dorsal region. In other words, he has passed through

all the stages of Potts' disease, we may say, without deformity.

As further illustration of the little influence that deformity has upon the paraplegia, let me cite another case in which recovery occurred promptly without any recession of the deformity:

A girl, eight and a half years of age, was admitted to the hospital the second of May, 1883. She had suffered from Potts' disease of the spine for four years. The bosse included nearly all of the dorsal vertebræ and was very conspicuous. Shortly before her admission she became paraplegic. The treatment employed was a simple Knight spinal brace, which made no claim whatever to posterior pressure, but was a mild form of fixation. She began to improve within a few weeks after admission, and at the end of eight months was entirely restored. Letters received for a few years subsequent to her discharge confirmed the permanency of her cure.

TABLE IV.

Interval between recognition of Bone Disease and appearance of Paraplegia.

Simultaneously	14
Within 3 months.....	7
Within 6 months.....	15
Within 12 months.....	18
Within 2 months.....	9
Within 5 months.....	8
Within 7 months.....	2
Within 14 months.....	1
Total.....	<hr/> 74

In many instances the symptoms of bone disease are not recognized until the deformity appears. The symptoms may often exist, to a typical degree, and yet the proper interpretation is not given. Very good practitioners fail to recognize the disease until the deformity has arisen. They are so in the habit of looking for "malaria" and "rheumatism" and "neuralgia" and "growing pains," that symptoms which ought not to be overlooked, and which ought to be correctly interpreted, pass unnoticed, until the mother herself calls attention to the Potts' disease.

I take this opportunity, therefore, of making another appeal for the early diagnosis of Potts' disease of the

spine, merely suggesting that an examination of the child naked will assist materially in making such diagnosis.

TABLE V.

Completeness or Incompleteness of the Paraplegia.

Complete	65
Incomplete	9

Among the cases of complete paraplegia, there were several instances of dorsal and sacral decubitus, and eight cases where the incontinence of urine was a most annoying feature. In a few instances incontinence of fœces existed. The treatment was varied. The majority of the patients wore, at one time or another, a plaster-of-paris jacket. A certain number wore a spinal brace, sometimes a Knight brace, sometimes a Taylor posterior spinal assistant. A few were confined to bed on a frame, with traction. In a certain percentage the potassium iodide was employed in large doses. The cautery was employed in adolescence, in addition to the protection and the potassium iodide. I have not employed the cautery to any extent in children, for the reason that the thought of being burned is so repellent that I have contented myself with other means. It is unnecessary to give an elaborate display of the different methods of treatment, because I am not prepared to state definite results therefrom. I thought it best to make two general classifications; one giving cases treated by potassium iodide and an immobilization, and another class treated by some form of immobilization not always complete. In separating these two, I find that I can present a table which will give approximate results:

TABLE VI.

Immobilization only.

Cured	18	%64.28
Improved	2	% 7.14
Unimproved	4	%14.29
Died	4	%16.29
Total.....	28		

For comparison, let me present another table:

TABLE VII.

Immobilization and Large Doses of Potassium Iodide.

Cured	27	%58.69
Improved	10	%21.71
Unimproved.....	4	% 8.70
Died	5	%10.87
Total.....	46		

Before proceeding further with the final results, I deem it best to present a table which will give the duration of treatment and also one that will give the interval between the discharge of the patient from the hospital and the last recorded note. This may be further supplemented by a table giving the duration of the paralysis.

TABLE VIII.

Duration of Treatment.

Under 1 month.....	8
Under 3 months.....	8
Under 6 months.....	14
Under 12 months.....	20
Under 2 years.....	9
Under 3 years.....	5
Under 5 years.....	5
Under 5 years.....	2
Under 6 years.....	3

TABLE IX.

Interval between Discharge from Hospital and Last Note.

No interval.....	32
From 1 to 6 months.....	10
From 6 to 12 months.....	6
2 years.....	4
3 years.....	3
4 years.....	4
5 years.....	3
6 years.....	3
8 years.....	1
9 years.....	3
10 years.....	2
12 years.....	1
13 years.....	2

With regard to the number of cases in which there was no interval, I would like to state that a number of these were heard of afterwards, but I was unable to fix a date and was unable to find any record in the histories. It is fair to presume that a case once cured and so recorded will not relapse under a moderate degree of protective treatment. We have found that when the cases do relapse, there is some very good reason therefore, such as the premature removal of the apparatus, extra strain or impaired health.

TABLE X.

Duration of the Paralysis.

Three months and under.....	5
From 3 to 6 months, inclusive.....	12
From 6 to 9 months.....	8
From 9 months to 1 year.....	9
From 1 year to 1¼ years.....	4
From 1¼ years to 1½ years.....	6
From 1½ to 2 years.....	11
From 2 to 2½ years.....	6
From 2½ to 3 years.....	4
From 3 to 3½ years.....	1
From 3½ to 4 years.....	1
From 4 to 5 years.....	2
From 5 to 6 years.....	3
From 6 to 7 years.....	1
10 years.....	1
Total.....	74

I have not included in this list any cases that are at present under treatment, but have purposely omitted such. The case in which the paralysis existed for six years is worthy of note, as it was a most remarkable one, by reason of the extraordinary spasm, prior to and during the early part of the active treatment.

This was in a boy ten and a half years of age, who came under my observation Oct. 22d, 1891. He was referred to me by Dr. William H. Thompson, of this city. His Potts' disease dated from an attack of the grip in February, 1890. Ten months later, while wearing a plaster-of-paris jacket with head support, he manifested weakness in the upper and lower limbs. A month later he was unable to walk. The treatment employed at this time was traction and counter-traction, in the recumbent posture, and was carried out for two months most successfully. During this period the paralysis became profound. The upper extremities became paralyzed first. The reflex spasm was confined to the thigh flexors. In July, 1891, he was removed from bed and subjected to baths at the Hot Springs, Virginia, for seven weeks without any amelioration. At the time of my first observation his was a frail-looking body, chest sunken, muscles considerably wasted, bony prominence marked. He could not turn from side to side, but lay on his back and toward the side with the left thigh flexed strongly on the pelvis at an angle of 90°, the leg being flexed sharply on the thigh. The spasm was so great that the heel rested against the buttocks. The right

limb was held in moderate spasm, but he could, with difficulty, overcome the spasm himself. When he got the thigh and leg down to about 135° the whole limb would become suddenly extended to 180° , with a distinct snap like the trigger of a gun. At this time the thigh muscles were all tense; leg muscles the same; foot fully extended. The bladder sphincter was very weak, but he had control over his rectal sphincter. There was no reflex spasm in the upper extremities. The right hand grasp was very weak, but it was a little stronger in the left. The deformity of his spinal column was not very marked. Disease was located in the cervico-dorsal region. The treatment adopted was the cautery, a frame and potassium iodide, supplemented by the potassium bromide at night.

By the 13th of November there was a little relief in the spasm, but it was still very annoying at times, requiring hypodermics of morphia to secure sleep. A note made Nov. 30th states that he had reached ninety-five grains of potassium iodide three times a day. He rested better at night. The exacerbations were not so frequent and not so severe. He had very good control of the sphincter of his bladder. On Dec. 5th I gave him ether and fully extended the left limb. Weight and pulley was employed to maintain the extension. At this time he was taking one hundred grains of potassium three times a day. The improvement continued with very little interruption, and by the 21st of Dec. I gave him smaller doses of the potassium, employed as an adjuvant the hot water douche at a temperature of 112° . It was not until the 28th of March that any eruption from the iodide appeared. On the 4th of June, 1893, he was discharged from the hospital and removed to his home in a Western city. He was taken home in a wire cuirass, in which the limbs were easily held in full extension, the muscular spasm being very infrequent and very slight. I received letters from time to time, and on the 14th of November, 1892, when I saw him at his home, I found that he had gained flesh perceptibly, that the spasm was very slight, and that his pains were trifling. He slept well for three or four hours at a time, was taking no opiates or narcotics, but there was no appreciable power in the lower limbs. I saw him again a year later, when there was still no return of power. He was very comfortable and practically well but for the paraplegia. This condi-

tion continued, and on the 20th of Nov., 1896, he died from some intercurrent affection. There was no autopsy.

In estimating the results I am able to present the following table:

TABLE XI.

Final Results.

Cured	45	\$60.80
Improved	12	\$16.22
Unimproved	8	\$10.82
Died	9	\$12.16
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Total.....	74	

I am thoroughly convinced that the improved cases will recover, or have already recovered, but I am unable to get at the facts in the cases. I have simply put them under the heading "improved." In tracing out the causes of death, I find that in the nine patients who died during the course of the disease the causes are shown in the following table:

TABLE XII.

Cause of Death.

Myelitis	3
Tuberculosis	4
Capillary bronchitis	1
Unknown sudden death.....	1

A further table may be added which will show what became of the nine cured cases; that is, nine cases cured of the paralysis and so recorded. These died later of the following diseases:

TABLE XIII.

Cause of death in Patients subsequent to the Cure of the Paralysis.

Unknown (two dying suddenly).....	4
Tuberculosis	2
Pneumonia	1
Alcoholism	1
Pott's disease with suppuration.....	1

Before concluding this paper I desire to place on record an extraordinary case of rotary lateral curvature of the spine, involving the upper dorsal region. I am prompted to do this because of a patient at present under observation with lateral curvature of the spine, right side, and a marked degree of ataxia. One often wonders why we do

not get paralysis in cases of high degree of curvature, why the cord itself must not necessarily be compressed by the sharpness of the curve.

The case I report is in a boy who was fifteen years of age at the time of the development of the paraplegia symptoms. He first came under my care on Oct. 21st, 1891, and was referred by Dr. G. H. Fox, of Rutland, Vt. His curvature had been observed six months prior to the above date. He had no pain or inconvenience at the time and was in very good general condition. At the time I first saw him his thorax was in moderate pigeon breast; the lateral curvature was not great, but still noticeable. There was associated with the lateral curvature an antero-posterior curvature, which was less than an inch in height. The actual lateral deviation was less than one-half an inch, but the rotation was rather sharp, while the curve was proportionately small. I thought his a good case for gymnastic exercises, and taught him a number of movements which I fancied would be of assistance in correcting the deformity. He seemed interested in the work and on Jan. 12th, 1892, I made a note that he had been quite faithful. There was, however, no improvement that I could detect. I was forced to note a little increase in the rotation.

June 3d, 1892, I noticed that he was not exercising with any degree of energy and that it was a task for him to exercise. A few days later I fitted him with a plaster-of-paris corset and provided a swing for him to use night and morning. Oct. 20th, 1892, it was noticed that he was gaining in height and that the deformity was not increasing.

March 15th, 1893, because of sluggishness in gait, I called Dr. M. Allen Starr in consultation, and he found diminution in all the skin reflexes, exaggeration of the patella tendon reflexes, slight ankle clonus, no anæsthesia, no apparent paralysis, no rigidity. With the eyes closed the boy swayed a little. The mechanical irritability of the muscles was normal. There was some atrophy of the trunk muscles, especially the pectorals, which was out of proportion to that of the arms and legs. Dr. Starr made a diagnosis at that time of compression of the upper dorsal cord, probably below the second dorsal segment, and above the seventh, as the epigastric reflex was normal and the ab-

dominal reflex was exaggerated. He approved of apparatus which would fix and support spine and suggested a hot and cold douche to the back at night. I applied a spinal brace with a chin piece for the head, and ordered a continuance of traction by means of the swing. June 10th, 1893, he presented a marked exaltation of the reflexes, was much more sluggish in his movements and seemed to take very little interest in the treatment. There was no paralysis at this time. Oct. 19th, 1893, he was carefully measured and his limbs were found equal in length, equal in size. When he closed the eyes he swayed a little from side to side, both in standing and walking. He had been taking, since the 12th of June by my orders, potassium iodide, and when he reached thirty grains three times a day he had a troublesome diarrhoea, which was controlled after a discontinuance of the potash. April 28th, 1894, his deformity was greater. The chest walls were flattened on the right side in front, and the ribs formed a sharp ridge over the projecting side. The plane of the scapula on the right side formed an angle of 110° with the verticle axis of the body. The limbs were weak, but he was walking about. I made an attempt to fit him with a steel apparatus, but failing in this I applied in May a slight plaster-of-paris jacket.

I heard from him occasionally, but did not see him until Oct. 20th, 1894, when he was decidedly worse. His gait was shambling and unsteady. He used a cane. Drs. Starr and Shaffer on my suggestion were called in consultation at the time and each examined him very carefully. Dr. Starr found increase of the reflexes below the thighs, but no cremaster reflex. There was increased loss of power in the limbs. He made out a lateral sclerosis, but was unable to assign any cause, thinking it probably idiopathic. He had reluctantly abandoned the compression, because of the excellent support which the boy had had and because of the traction to which his spine had been subjected. Dr. Schaffer ruled out Potts' disease and was unable to explain the increasing loss of power or the lateral sclerosis as due to the curvature itself. He decided that all braces be dispensed with for the present, that the boy go to bed for three months and make no attempt to walk during that time. After a little further consultation we agreed to urge this treatment, adding thereto, on Dr. Starr's sug-

gestion, the use of farradism to the back muscles and belladonna and ergot internally. The prognosis was gloomy enough. The treatment, however, was carried out most successfully under the immediate direction of Dr. Thompson, of Rutland.

Jan. 27th, 1895, I visited the boy and found him very much improved in appearance, in general health and in muscular development of the upper extremities. The spinal deformity was certainly much less marked. He had some tenderness over the spinous processes in the upper dorsal and cervical regions. The thorax in front was in excellent shape. The patellar tendon reflexes and the ankle clonus were much aggravated. In fact, he had at this time all the symptoms of compression myelitis with paraplegia. He could move the feet about in bed, but this act seemed to bring on spasm. The sensation was good and the muscles were well developed. There was no localized atrophy. It was reported to me that during the first month of his bed treatment the feet were quite cold, subjectively and objectively, but latterly they had become warm, although the circulation in the toes seemed poor. He had borne the confinement so well that I ordered him to remain in bed three months longer. On June 29, 1896, the father reported that he was walking about with a little assistance, that his limbs seemed to be growing stronger every day. Was having massage twice a week.

He came to the city on the 17th of December, 1896, and I found him walking without support for at least a month. During the two weeks prior to his visit, the improvement had been most rapid. He could go up and down stairs and had lost that sense of weakness in his back. The deformity of his spine was no worse. The muscles about the chest and back were better developed. There was a little tenderness in two or three points over the spinal column. He could move his limbs in all directions and all the muscles were in good condition. He could stand and jump on his heels without pain. There was still a little exaltation of the patellar tendon reflexes and a little foot clonus, especially on the left side. I advised a continuance of the treatment, discouraged the use of a brace of any kind and gave a good prognosis. Indeed, this was quite unnecessary, as the progress of the case itself made this a foregone conclusion.