the possibility of the coexistence nf mnlarial and enteric fever, nccount in vurious ways for the chills and sweating in esses presenting a similar clinical picture to those above reported, they not hnving found the mnlnrial plasmodium in the hlood.

Thus, Bouveret[1] reports four such cases in which he helieved the modified symptoms were due to a slow and irregular elimination of toxic materinl, a large volume nf which is sometimes thrown into the hlood at once; and Fränkel[2] repnrts a similar case in which he thought the symptoms might be due to a thromhus in a mesenteric vessel, with suppuration nnd possihly miliary ahscesses in the liver.

Professor Osler, in a recent number nf the *Johns Hopkins Hospital Reports*, vol. iv. No. 1, mentions three interesting cases in which mnlarial fever was present with the plasmndium within a few months of the occurrence of enteric fever and once within n few dnys, but as the result of his experience he says: "There was no case with the chnracters of the two diseases so hlended that it seemed a compound or hyhrid malndy, nor was there an instnnce in which the manifestations of the two diseases were concurrent."

It is not well to draw hasty conclusions from a very limited number of cases, but there is a kind of evidence whicb does not need to be multiplied to be convincing; and it seems to me, from the observations of the few cases herewith presented, that while it is unwise to necept the term typho-malarial fever as indicating a third form of disease, which is neither typhoid fever nor malarinl fever, it cannot he denied that the two lntter diseases inny coexist.

Case I. certninly proves that they mny do so, and this in n part of the country in which this form of douhle infection is most unlooked for, nnd the other cases, although perhnps less striking, are at least corroborutive.

A STUDY OF ONE HUNDRED AND THIRTY-EIGHT CASES OF POTT'S PARALYSIS.

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The mntter of this pnper has been gleaned from nll the printed sources nvailnhle, nnd from nn nriginnl study nf nne hundred nnd thirty-eight cases, one hundred nnd five of them heing collected from the literature of the subject, the rest, thirty-three in number, heiug nll the cases treated nt the Boston Children's Hospital hefore 1891, of which a fairly full nccount could he ohtnined. Fnr permission to use this last materinl I

[1] Lyon Médical, June 5, 12, 1892. [2] Lyon Médical, June 19, 1892.

am indebted to Drs. E. H. Bradford and H. L. Burrell, of the surgical staff of the hospital.

PATHOLOGY.—The work of Michaud,[1] Erb,[2] Courjon,[3] Elliot,[4] Gowers,[5] and others, has made the usual course of the process clear.

In the vast majority of cases the caseous osteitis of the bodies of the vertebræ extends back, involves first the posterior common ligament, which becomes ulcerated, and then the external surface of the dura, making a pachymeningitis externa. The tumor so formed projects into the vertebral canal, presses on the cord, sometimes forcing the cord against the laminæ, and thus sets up an interstitial myelitis of greater or less extent, from which ascending and descending degenerations may take their origin.

Macroscopically, the dura is seen to be thickened, now in one or two spots, again like a ring, again generally. Regularly, however, it is the outer surface which shows a change; the inner surface remains unaffected. The pia and arachnoid appear normal. The nerve roots are frequently found compressed by masses of exudation in the inter-vertebral foramina. The cord in early paralysis may show on gross examination absolutely no change in size, color or consistency, or it may look pale or red at the point of compression, and show some softening; later it may show hardening instead. The shape may be irregular, and the size may be reduced even to one-fifth of the normal calibre. Changes above and below the point of compression are not usually to be seen with the naked eye, except by the help of staining fluids.

Microscopically, one sees in the dura outside the normal inner layer a fibrous layer containing many leucocytes; the outer edge of this merges into a mass of caseous pus. At a late stage the dura may be seen to be made up of a mass of fibro-cartilaginous plaques. If compression has lasted some time the cord shows at the site of the lesion a considerable increase and thickening of interstitial connective tissue, a thickening and fatty degeneration of the intima of the vessels, and throughout the cross section many granular corpuscles, globules of myelin, and corpora amylacea; the gray matter may be distinguishable from the white only after staining, and then with difficulty; the axis cylinders are swollen or broken down; in the central gray matter the ganglion cells are swollen, vacuoles are formed, and there is here and there pigment deposited. As the seat of the caries is in the bodies of the vertebræ, and as the anterior portions of the cord are usually held close to the posterior surfaces of the vertebræ, while the posterior portion is five or six lines from that face of the canal, the meningitis is ordinarily anterior, and the myelitis first appears in that part of the cord. The myelitis may, however, be more or less unilateral, and may vary in extent from a slight infiltration to a complete destruction of the cord. There may be no changes except at the point of compression, or, above and below,

destructive processes may go on, and these, though they may affect the gray matter to a variable exteat in either direction, are especially apt to cnuse alterations in the white matter. Above, degeneration[6] usually follows the posterior columns—the direct cerebellar tract when the lesion is above the lumbar enlargement, the ascending antero-lnteral tract of Gowers, and high up the fasciculi graciles, even to the medulla, while below, degeneration goes on in the pyramidal tracts, direct and lateral, even to the cauda equina. In these consecutive lesions the microscope shows, in the gray matter, the same changes, though less advanced, that are seen at the point of compression, and in the white matter interstitial proliferation of connective tissue and degeneration of nerve fibres. This degencration is not generally uniform on the two sides, and is, to a certain extent, variable in the pnth followed. Such is the customary pathological process, hut there may also rarely he pressure paralysis in which tbe pressure is exerted by hone, as hy nn acute angle of hone, by n sudden dislocation of one vertebra on nnother, hy nn ohtuse angle of hone over which the cord is tighly stretched, or by a sequestrum[7] detnched within the caaal; or, agaia, the pressure may he caused by an ahscess[8,9] eacroaching on the canal.

Moreover, the paralysis may he due, as Elliott[4] has pointed out, to an obstruction of the hlood supply, in which case there may he no myelitis, oaly aa acute œdema, aad recovery may he very rapid.

Such obstruction is easy, as the vessels nre small, do aot anastomose freely, are exposed oa the surface of the cord, nnd enter through foramina easily hlocked hy exudation.

Fiaally, it is possible, though most rarely does it happea, for inflammation to spread to the cord, either hy contiguity through dura and pia or along the nerve roots, and there to set up a myelitis by extension.

In contrast to the class of cases showing paralysis witbout lesion of the cord, there are those cases which show compression nnd atrophy of the cord without parnlysis,[10] and complementary to these last are the recoveries from paralysis; for in these, examination at times finds well-marked sclerosis existing, while the functional activity of the cord is not lessened. The closeness of the cord to the anterior wall of the cannl, and the facts that the disease of the hoae is usually in the bodies of the vertebræ and that the tracts conducting motor impulses are more superficial than those transmitting sensory impulses, are reasons why the paralysis is usually motor hefore it is sensory. In some cases, however, there is relatively great destruction of the cord in the posterior columns, due to pressure on the laminæ, and in these there may he recovery of motion and sensation but permanent ntaxia.

Showing the proportion of the causes of paralysis, Poore[11] says that autopsies in 66 cases show a cord compressed 53 times hy thickened meninges; 4 times hy dislocation; once each hy enlargement of odontoid,

hy an acute angle of rapid formation, by dead bone, by a hlunt angle over which the cord was stretched, hy disensed hone which had given way, and three times hy an abscess which had hurst into the spinal canal.

Of the 35 deaths in the cases studied, records of autopsies have been found in only 22, and of these only 8 were complete, that is, included a microscopical study of the cord. In those examinations, mnde hy various ohservers, there was reported a pachymeningitis in 5; signs of myelitis in 4; softening with hemorrhagic infiltration in 1; sclerosis in 3; secondary degeneration, ascending and descending, in 5, and neuritis in 2 cases.

In every case the cord was found affected, and in most cases the meninges.

In tnhulating the 14 macroscopic examinations, it is found tnat an alteration of the shape or size of the cord, a flattening or n lessening, is the commonest lesion, occurring 8 times; softening is reported 6 times, while sclerosis was evident only once, a pachymeningitis was present in 5 cases, and it is clearly stated in two instances tbat the cord looked normnl.

Symptomatology.—(*a.*) This cannot be prefaced better than by the classical clinical history of Pott:[17]

Ths pntient "nt first complnins of being very soon tired, is lunguid, listless, and unwilling to movc much or at all hriskly; iu no great length of time after this he may bs ohserved frequently to trip nnd stumble, nlthough there hs no impediment in his way, and whenever hs nttempts to movs hriskly he finds tbnt his legs involuntarily cross each other, hy which he is frequently thrown down, nnd that without stumhling; upon endeavoring to stand still and erect without support, even for a few moments, his knees give way and hend forward; when the distemper is a little further advanced it will he found that he cannot, without much difficulty and deliheration, direct either of his feet precisely to any exact point, and very soon after this hoth thighs and legs lose n great deal of their natural sensihility, and hecome perfectly useless for all the purposes of locomotion.

"Some are rendered totally and absolutely incapnhle not only of wnlkiug, hut of using their legs in any mnnner, others can mnke shift to get ahout with the help of crutches, or hy grasping their thighs just ahove the knees with hoth hands; some can sit in an armed chair without much trouhle or fatigue, others cannot sit up with any help; some retain such a degree of power of using their legs, as to hs ahle to shift their posture when in hed; others have no such power, nnd nre ohliged to he moved on all occasions.

"The first return of the power of motion in the limhs is rather disngreeahle, the motions heing involuntary and of the spasmodic kind, principally in the night, and generally nttended with a sense of pnin in all the muscles concerned.

"In the milder kind of case, the power of volnntary motion generally soon follows the involuntary.

"The knees and ankles hy degrees lose their stiffness, and the relaxation of the latter enahles the patient to set his feet flat upon the ground; the certain mark that the power of walking will soon follnw."

(*b.*) *Prodromal period.* There may or may not be a prodromal period characterized hy sensory disturhances.

There may he pain, varying much in degree; it may he girdle-like, or

there may be persistent neuralgias at any spot, usually of abdomen or legs. Possible also are paræsthesiæ, as tinglings or sensations of heat or cold, hyperæsthesia and anæsthesia; this last is usually in spots. There are said to be sometimes herpetic or bullous eruptions over areas supplied by nerves from affected regions.[2]

(*c.*) *Paralysis of motion.* The paralysis which is characteristic of myelitis varies from a slight weakness of a few muscles, not to be noted without close observation, to a complete loss of power of most of the muscles of the body, so that the patient lies prostrate.

Though usually a paraplegia, the paralysis may involve all parts of the body supplied by spinal nerves, or it may be a monoplegia,[18] or a hemiplegia;[19] either, too, may develop after a time into a paraplegia; there may be a brachial paraplegia, and later this may extend and involve the legs.[20]

In severe cases the abdominal and respiratory muscles may be affected. The legs are very often affected unequally.*

Of 59 cases reported by Gibney,[21] 24 (40.6 per cent.) had complete paralysis, and of these 4 had also incomplete cervical paraplegia. Of 59 cases analyzed by Taylor and Lovett,[22] 32 (54.2 per cent.) had complete paralysis; 3 had paralysis of the arms. Of Myers'[23] 218 cases, paralysis affected the arms in 7. Of the 138 cases studied for this paper, 61 (44 per cent.) had complete paralysis, and 26 had more than a simple paraplegia. Of the 256 cases thus available for comparison, 117 (45 per cent.) had complete paralysis of the limbs affected.

In some cases the arms are first paralyzed, while the legs are free. This may be due to the fact that the lesion is at the level of the cervical enlargement, and in this place first attacks the anterior roots for upper extremities; in this form of palsy muscular atrophy and absence of reflex actions form a characteristic feature;[24,25] or if the lesion is in upper part of cervical cord, the motor paths in the antero-lateral columns which belong to the upper extremities may be attacked first; in this case reflex actions would be present. In a few cases, the compression being limited to one-half the cord has caused a hemiplegia with crossed anæsthesia, a typical unilateral lesion.[19]

(*d.*) *Paralysis of sensation.* "Some impairment of sensation is found in all Pott's paralyses, but it is usually so slight as only to be recognized by the most delicate tests, and for a short time."[26]

Very frequently, however, sensation in paralyzed parts is decidedly dulled, usually some time after the muscular weakness is noted, and there may be almost complete anæsthesia of all the body below the site of compression. This is significant of a very extensive transverse myelitis.

* There have been cases where paralysis of arms followed paralysis of legs, when the lesion was in the dorsal spine. This may be due to an ascending myelitis, which is not rare, or to ascending degeneration of the lateral columns, which is quite rare.

There may be, too, hemi-anæsthesia, or areas of anæsthesia; De Jonge[17] describes a case with increased sensibility in the paralyzed parts.

There may be, though usually there is not, pain of a constant, heavy, aching character all over the paralyzed limbs. There may be also paræsthesia and hyperæsthesia, all of these sensory symptoms being due to pressure or myelitis of the posterior columns.

Generally tactile sense disappears first, then the sense of temperature, and last the sense of pain. Michaud[1] quotes a case in which there was paralysis of sensation and not of motion, and there are several cases in which the sensation was found dull before muscular weakness was noted.

Taylor and Lovett[22] found out of 59 cases 15 (25.4 per cent.) with incomplete sensory paralysis.

Of the 138 cases, 71 (51.4 per cent.) had some notable impairment of sensation.

(*e.*) *Spasms and contractures.* In the prodromal period there may be twitchings of various muscles, due to irritation of the anterior nerve-roots; but generally at first the paralyzed muscles are completely relaxed, quiet, and soft; the joints also are relaxed and make no opposition to passive motion. In the later stages, with the advent of secondary degeneration in the cord, the flaccid muscles may become tense and rigid; usually at first the legs are extended stiffly and the feet are held in slight equino-varus; later a change comes on, slowly or suddenly, sometimes while the patient is asleep; the thighs become flexed on the pelvis, adducted, the legs flexed on the thighs, and the feet frequently take the position of calcaneo-valgus. If the limbs are extended again they frequently flex in a jerk without any apparent cause.

Sometimes there is present what is called "clasp knife" rigidity (for example, if, when the knee is flexed, the leg is gradually straightened, it moves smoothly and easily, till full extension is nearly reached, and then it suddenly becomes rigidly extended, and a good deal of force is required to bend the knee again).

At first abnormal positions can be readily passively corrected, but later contractions set in, and the legs become fixed in the position adopted.

Of the 138 cases 19 are noted as having rigid muscles.

(*f.*) *Bladder and rectum.* In severe cases either the bladder or the rectum, or both, may be affected; usually at first there is some difficulty in micturition—slowness in starting the stream—but later there is true incontinence.

The anal sphincter may be paralyzed and the contents of the bladder and rectum may be involuntarily evacuated consciously or unconsciously. Incontinence may result some months after symptoms of complete paraplegia have existed, apparently from downward extension in the cord, and this incontinence may last for years, and yet the patient finally improve and obtain entire volitional control over the bladder again.

In other cases the resulting cystitis is an important factor in causing death.

Taylor and Lovett's 59 cases furnish 8 examples of paralysis of bladder or rectum, and the 138 cases show 63 examples.

(*g.*) *Reflexes.* The condition of the deep reflexes is important in diagnosis.[28] [29] They are regularly exaggerated unless the seat of the lesion be the lumbar enlargement, in which case they are absent. The slightest tap on the patellar tendon, the lightest pressure on the sole of the foot, even a tap on the bone or fascia, or belly of muscle, will in many cases call forth a vigorous contraction of all the muscles of the leg, and sometimes be followed by a similar contraction of the muscles of the other leg. Even the evacuation of the bladder or rectum is said sometimes to be sufficient to call forth a clonus of the limbs.

The cutaneous reflexes are sometimes exaggerated, so that pinching the skin will cause a violent reflex clonus; again, are not to be found; and still again, are unaltered.

Gibney[15] quotes cases in which pressure over the inner side of the lower third of the thigh would cause sudden flexion of the thigh on the pelvis and of the leg on the thigh, followed in a few minutes by the same motion on the opposite side.

Bastian[28] says that cases show that "deep reflexes are abolished if there is a complete transverse lesion, *i. e.*, when there is a complete loss of sensibility as well as of voluntary power."

(*h.*) *Trophic and vasomotor changes.* Trophic changes are said to be uncommon unless the cervical or lumbar enlargement is involved, which may occur from secondary degeneration; but if the paralysis has existed any considerable time one frequently finds a notable degree of atrophy of the legs, together with some coldness, occasional lividity, and generally a dry, inelastic, scaly skin. There may be localized œdemas, sweatings, alterations of temperature,[18] and sometimes lesions of the joints secondary to lesions of the cord.

Roser[30] has seen three cases of spontaneous dislocation of the hip brought about by the reflex motions of Pott's paralysis; the luxation was behind, gradual and painless. In severe cases bedsores are frequent. Of the 138 tabulated cases, 20 had marked wasting of the limbs and 2 had profuse sweatings noted.[31]

(*i.*) *Medullary symptoms.* When the upper cervical cord is compressed Charcot has noted paralytic myosis or spastic mydriasis from paralysis or irritation of the corresponding paths in the cervical medulla.

Respiratory and gastric disturbances, vomiting, difficult swallowing, persistent hiccough, and retardation of the pulse have been met and are attributed to implication of the vagus, spinal accessory, or phrenic nerve; according to Micbaud,[1] general convulsions may occur from compression of the upper cord.

(*j.*) *Sexual disability.* Pott speaks of a characteristic loss of the power of erection. In the series under examination priapism and loss of sexual power are mentioned, the former occurring three times and the latter twice.

(*k.*) *Time in Pott's disease at which paralysis appears.* The paralysis may come on as a diagnostic symptom of Pott's disease before the latter has been otherwise made manifest, or it may come on when spinal disease has existed for years and when to all appearances the process has subsided into quiescence.

Taylor and Lovett say paralysis occurs on an average about two years after the beginning of the disease. Out of 40 cases it came promptly after a fall in 4 cases; in 8 cases it came within one year from the beginning of the disease; in 16 cases within two years; in 4 cases within three years; in 3 cases within four years; in 1 case in five years; 1 in nine years; 1 in eleven years; 4 in fifteen years; and 1 in twenty-eight years.

Gibney says the average duration of spinal trouble before paralysis is about three years, though the time has been as short as four and a half months and as long as eleven years. Myers puts the time at from a year to a year and a half.

The writer has found the time in question in 130 cases as follows: Under one year, 62; one to two years, 18; two to three years, 14; three to four years, 10; four to ten years, 12; ten years or over, 14; longest time, twenty-one years.

From this it is apparent that in nearly 48 per cent. the time is less than a year, and that in more than 72 per cent. of the cases the time is less than three years.

(*l.*) *Duration of increase of paralysis.* From the first note of weakness to the maximum of paralysis attained there may be less than a day or there may be several months, but perhaps oftenest a few weeks.

In 44 cases the time was noted with the following result:

Under one month, 19 cases; one to two months, 12; two to three months, 4; over three months, 9.

(*m.*) *Duration of paralysis.* The duration of the attack may be a few weeks or it may be a lifetime; usually it varies from a few months to a few years. Pott[17] put the time at from two months to two years. Gibney[15] in 13 cases of paresis found a duration ranging from four weeks to twenty-two months. From a study of 218 patients, Myers[23] gives an average duration in cervical cases of twelve months, in upper dorsal of nine and a half months, in lower dorsal of six months, and in lumbar cases of eight months. He also finds that the duration is several months less when the paralysis begins in a patient under treatment.

Taylor and Lovett[22] give as the average duration twelve months, but when paralysis comes on while the patient is under treatment an average

duration of only seven months. In their 36 cases the durations were as follows: One case, six years; 2, three years; 5, two years; 7, one year; 4, six to twelve months; 8, six months: 9, two to four months.

The duration to recovery or death of 108 cases was as follows: Under six months, 37; six months to one year, 28; one to two years, 25; two to three years, 11; three to four years, 4; four or more years, 3. Twelve of these cases were under treatment before paralysis began. Of these 12 the duration was under six months in 6; six to twelve months in 5; and from 1 to two years in 1 case. The shortest paralysis with recovery noted by the writer is seven weeks and the longest four years.

(*n.*) *Recurrences.* A few cases have more than one attack, and there are instances of three, and even of four attacks.

Taylor and Lovett found in 59 cases recurrences in 6; Myers in 218 cases, recurrences in 18; Gibney in 58 cases, recurrences in 8. The intervals between these recurrences varied from a few weeks to several years.

Of the 125 patients studied 9 had two attacks, 1 three attacks, and 1 four attacks. The intervals between these attacks varied from a few weeks to fifteen years.

(*o.*) *Termination.* There are three terminations—recovery, death, and permanent paralysis.

1. Recovery is usually gradual, and frequently lasts months. After some time of absolutely no change in the patient's symptoms it is found that a few motions can be executed by the paralyzed parts at times, then there is a permanent increase of power in those parts; the sensibility improves and generally becomes normal before entire power over the muscles is established; control of the bladder is regained; and, last of all, usually months after the patient believes himself well, the tendon reflexes become normal.

2. Death may result from progressive myelitis with its attendant cystitis, bedsores, diarrhœa, pyæmic fever, and exhaustion, or the patient may yield to tuberculosis or be cut off by some intercurrent disease.

Gibney in 13 deaths found 6 from myelitis, 2 from tuberculosis, and 5 from other diseases.

Taylor and Lovett in 59 cases found 5 deaths—2 pneumonias, 1 phthisis, 1 opium habit, and 1 acute cerebral meningitis.

The list here studied furnishes 27 deaths with known causes; 8 were the direct result of myelitis, 8 were the result of tuberculosis or amyloid disease, 4 the result of operation for the relief of the paralysis, and the remaining 7 came from acute affections not bearing any relation to the spinal disease.

3. A permanent paralysis may be slight in amount—simply noticed as a weakness—or it may be a paralysis with contractures, and the patient get about, if at all, with great difficulty. In the last case, how-

ever, the general health may be excellent and the condition without danger to life.

FREQUENCY.—The proportion of cases of spinal caries that become paralyzed varies greatly with the clinic of the one making the estimate.

The 2657 cases of Pott's disease from the following table give 404 cases of paralysis, or about 15 per cent.

Authority.	Cases of spinal caries.	Cases of paralysis.	Per cent.
Gibney[15]	295	62	21
Taylor and Lovett[22]	445	59	13
Mohr[32]	72	5	7
Drachmann[33]	166	22	13·6
Sayre[34]	109	38	35
Shaffer[23]	1570	218	13.9

In Gibney's cases, in 189 cases of cervical or upper dorsal disease, paralysis occurred 59 times, and in 106 of lower dorsal and lumbar there was paralysis only 3 times. Taylor and Lovett's 59 cases are distributed thus: 1 cervical, 7 cervico-dorsal, 37 dorsal, 7 dorso-lumbar, 4 lumbar, and 3 undesignated. Of Snyre's 38 cases, 2 were cervical, 1 dorso-cervical, 32 dorsal, 1 dorso-lumbar, and 2 lumbar. Shaffer's 218 cases are 16 cervical, 12 cervico-dorsal, 145 dorsal, 19 dorso-lumbar, 18 lumbar, and 1 unspecified.

In the present list the cases, so far as known, are apportioned as follows: Cervical, 15; cervico-dorsal, 10; dorsal, 85; dorso-lumbar, 6; and lumbar, 3. It is evident that nearly all the cases of paralysis result from lesions in the cervical or dorsal vertebræ, and that that locality of caries is, as Gibney says, the great predisposing cause of paralysis.

DIAGNOSIS.—Recognition of the beginning of paralysis is usually possible from the fact that it is generally preceded by an increase of pain in the abdomen or legs and by an increase in the deep reflexes of the knee and ankle. The diagnosis is made by establishing the existence of Pott's disease.

Trauma of the spine and pressure on the cord from other causes may give the same symptoms. Such other causes are cancer and sarcoma of the spine, small exostoses of the vertebræ, meningeal tumors, hypertrophic cervical pachymeningitis, aneurism, gumma, hydatid cyst, hemorrhage or abscess in canal, tumors of the cord, and spontaneous transverse myelitis.

When Pott's disease exists, the attendant asthenia or a double psoas contraction may be confounded with paralysis, but the latter has increased reflexes, and the former not.

PROGNOSIS.—In a broad way the prognosis of Pott's paralysis is good, *i. e.*, it tends to recover entirely. Taylor and Lovett in 47 cases, the results of which are known, find 39 recoveries (83 per cent.). Gibney

finds in 58 cases 29 recoveries (50 per cent.), nnd Myers reports 55 per cent. recovered in 218 cases.

Counting those 12 cases as "result not known," in which the paralysis has heen in existence less than n year nnd still continues, the list here made gives 126 "result known" cases, of which 78 are recoveries (62 per cent.).

The Children's Hospital cases, considered separately, give out of 28 cases 18 recoveries (64 per cent.).

In making the prognosis in nny particular case certain factors enter which may he considered in detail.

1. *Treatment of patient before paralysis.* If the patient has been under treatment, the prognosis is somewhnt hetter. Taylor and Lovett give 100 per cent. of recoveries in such cases, hut in the present list nre 15 such cases, with only 10 recoveries (67 per cent.).

2. *Duration of paralysis.* The mnximum time with recovery is, as hefore stated, four years, aud of the 14 cases whose paralysis lasted more than three years only 4 recovered. In the cases whose paralysis lasted less than six months, there were 18 recoveries; 21 recovered in from six months to one year; 20 in from one to two years; and 11 in from two to three years. From this one may infer that a patient's recovery is more apt to come in the second six months than in the first, that it is nearly as likely to take place during the second year as during the last half of the first, hut that the prognosis grows rapidly worse during the third year, and that after three years there is comparatively little hope.

3. *Time from beginning of spinal disease to beginning of paralysis.* Of 62 cases whose parnlysis hegan within n year, 27, or 43.5 per cent., recovered, while of 68 whose paralysis hegan more than a year after the spinal disease, 44, or 64.7 per cent., recovered. In other words, if the process is acute nnd advnncing rnpidly enough to hring on a paralysis early, the prognosis is worse thnn if slower changes compress the cord at n later stage.

4. *Age of patient.* Of 15 cases over forty years old when parnlysis hegan, only 5, or 33 per cent., recovered (the oldest cured was nt beginning of the paralysis fifty-four years), while of 76 patients under fifteen years, 48, or 63 per cent., recovered. (The youngest cured was at the heginuing of the paralysis twenty-two months old.)

Of 47 hetween fifteen and forty years, 24, or 51 per cent., recovered. Other things heing equnl, therefore, the prognosis hetters with the youth of the patient.

5. *Severity of the paralysis.* Of the cases with simple paresis of the nffected muscles, 65.6 per cent. recovered, and 15 per cent. died, while in those with complete paralysis of the affected parts, 36 per cent. recovered and 46 per cent. died; 38 per cent. of those with marked

sensory affection got well and 38 per cent. died; 41 per cent. of those with pnralysis of sphincters recovered nnd 32 per cent. died.

Clearly, then, the lighter the paralysis the better the prognosis, hut with complete paralysis the nddition of sensory or vesical pnlsy does not mnke the chnnce of recovery mnterinlly less.

6. *Location of lesion.* Cases other than cervical nnd dorsnl are too few to form n hasis for estimate. The percentage of recovery in pnralysis of cervical origin is 25, while in thnt of dorsal origin it is 55.

7. *Rigidity or contractures of limbs.* Of the 19 cases in which rigidity was mentioned as a prominent aymptom only 5 recovered.

8. *Marked wasting of limbs.* Of 20 cases affected hy considerahle atrophy, 8 recovered.

9. *Character of spinal deformity.* The kyphosis, as was noted hy Pott, is nppnrently not of grent importance in prognosis, hut n lnrge gradual curve, as the writer found in thirty cases exnmined, claims the largest per cent. of recoveries.

10. *Sex.* As to the linhility of paralysis in Pott's disease, it is interestiag to note that of the 125 patieats, 77 are males and oaly 48 females. The per cent. of recoveries among the mea is 52 and among the women 61.

11. *Part of body involved.* When there is more than a paraplegia preseat, the prognosis is made correspoadiagly worse—7 recoveries out of 26 cases; while in simple paraplegia there were 71 recoveries in 112 cases.

12. *Recurrences.* The cases studied give no reason for thiakiag the progaosis ia a second attack any worse thaa for a first attack.

13. *Intercurrent disease.* Bradford has noted n case of recovery from paralysis during nn attack of measles. The writer has seen two cases of intercurrent typhoid; in one, as the fever progressed, the paralysis grew worse till the pntient was powerless, and then lightened with convnlescence; the other, a fnirly severe case of paralysis, was quite unaffected hy the fever. He has seen also one case of nmyloid disease which died paretic; in this there was marked improvement in the parnlysis during the two months immediately preceding death.

Proportion of deaths. Taylor nnd Lovett fiad in 59 cases 5 deaths, 3 still paretic. Gihney finds in 58 cases 13 deaths, 14 still pnretic. In the 138 cases there were 34 deaths, 26 still paretic.

General summary of prognosis of paralysis. There is not a symptom characteristic of the paralysis that is of fatal import. Recovery has taken place even from threatening asphyxia.[35]

There is hardly a condition of the paralysis so severe as to deny the possihility of recovery." Recovery has occurred from paralysis of arms and legs, anæsthesia, and incontinence.

A child less than ten years old, with dorsal caries and paresis of legs for less than one year, will prohably recover.

An adult paralyzed more than four years will probably not recover.

The three most important elements in prognosis, in order of their importance, are (*a*) the duration nf the paralysis; (*b*) the severity and extent of the paralysis; (*c*) the age of the patient. The real index to the severity and extent of the paralysis is the completeness of the paralysis of the muscles of the affected limbs.

TREATMENT.—All nuthorities agree that the back should be put at rest; therefore confinement in bed, with n spinal support, either jacket or hrace, must he the first measure adopted.

In certain cases this enforced rest, perhaps aided hy extension nnd counter-extension, is sufficient for prompt cure, hut in more cases cure is n matter of gradual improvement extending over months. In all cases rest should be persisted in till the return of the use of the limbs is well established. Relief in a few days or weeks has been given hy opening an abscess connected with the spine, nnd operation is to he recommended if there is an ahscess present. Subsequent treatment is expectant: the number of drugs given outside of tonics is large enough to prove that no one is eminently satisfactory.

Gibney[36] is confident that potassic iodide in doses to the limit of toleration reduces the average duration of the paralysis hy months. He says: "Begin with gr. x, t. i. d. p. c. in mineral water, best Vichy; increase dose daily hy gr. x till stomach is intolerant." "Keep up as big a dose as the stomach will hear."

Bromide, ergot, silver nitrate, strychnine, physostigma, belladonna, have all had earnest advocates, the last especially, for removing neuralgias which precede paresis. Outside of drugs, electricity is recommended especially hy the neurologists, nnd Erb[2] says that n moderately strong galvnnic current applied in n stabile way to the point of lesion has been of service, and that he has never seen it do harm. Gibney,[36] however, quotes cases where electrical treatment has, he says, caused relapse or delay in cure. The cautery is a fnvorite with some, hut has objections for private practice. Althaus[37] says that this treatment is good when only the membranes are nffected, useless if the cord is involved.

Poore[35] gives directions hnw to cauterize without causing notahle suffering even in children, and cases are quoted in which a single cauterization has brought nhout an nlmost immediate cure.

In the same line of treatment are mentioned hlisters and local applications of hot water and ice.

Mitchell[29] urges the claims of süspension, saying: "It lets the patient get out of bed; it distinctly altera the pnthnlogical curve of the spine; it gives ease and relief to aches; it seems to nct with more speed than other plans, and when these have failed it gives n new resource."

Medicated baths are mentioned hy nne writer with the remark that they may he dangerous. Tenotomy[1] has been done to relieve the con-

tractures of paralysis. With reference to all the methods of treatment beyond rest, it is to be noted that as the paralysis tends to n cure which sometimes comes suddenly and unexpectedly, n careful estimate of the relative value of different remedies is difficult, nnd certainly from this series no conclusion can he drawn decidedly favorable to nny mode of trentment beyond rest. Some cases, however, do not get well, nnd even grow worse very rapidly; for such there has been since 1882 nnother treatment proposed, resection of the vertebral laminæ at the site of the lesion nnd relief of presure. The operation has as yet been done only n few times, but there hnve been some remarkable successes nnd severnl fntal results; improvement when noted has frequently proved temporary. At present Bradford[40] says: "The status of operation is that it offers hope to some cases that would otherwise be hopeless, while it has no plnce in treatment till conservative measures bnve been faithfully tried."

Bibliography.

1. *Michaud:* Sur le Méningite et la Myélite dans le Mal vertebml. Paris, 1871.
2. *Erb:* Ziemssen's Cyclopædia, vol. xiii.
3. *Courjon:* Paralysis of Pott's disease. Paris, 1879.
4. *Elliott:* Trans. IX. Internat. Med. Cong., 1887, iii. 363.
5. *Gowers:* Diseases of the Nervous System.
6. *Meige:* Amer. Journ. Med. Sci., 1890, N. s., c., 2, 128.
7. *Geffrier:* Soc. de Biologie, 1880, t. xxx.
8. *Ogle:* Trans. Path. Soc. London, 1867, xix. 16.
9. *Beck:* Illustrated Med. News, London, 1889, v. 193.
10. *Proust et Ballet:* Rev. mensuelle de Méd. et de Chir., v. 4. 425.
11. *Poore:* New York Med. Journ., 1878, xxvii. 140.
12. London Lancet, 1890, i. 402.
13. *Knecht:* Archiv f. Psychiatrie, Berlin, 1881, xii. 489.
14. *Frommann:* Virchow's Archiv, Bd. liv. 42.
15. *Gibney:* Illustrated Quarterly Med. and Surg. New York, 1882, i. 43.
16. *Dercum and White:* Annals of Surgery, ix. 421.
17. *Pott:* Remarks on that kind of Palsy affecting the Lower Limbs which is frequently found to accompany a curvature of the spine, and is supposed to be caused by it. London, 1779.
18. *Raymond:* Soc. de Biologie, 1882, 7. s., iii. 236.
19. *Troisier:* Archiv de Neurologie, Paris, 1882, iii. 322.
20. *Hun:* Med. News, Philadelphia, 1883, liii. 90.
21. *Gibney:* Journ. Nervous and Mental Diseases, New York, 1878, v. 259.
22. *Taylor and Lovett:* Med. Record, New York, 1886, xxix. 699.
23 *Myers:* Trans. Amer. Orthop. Assoc., iii.
24. *Jaccoud:* Gazette des Hôpitaux, Paris, 1884, lvii. 1145.
25. *Proust et Ballet:* Revue mensuelle de Méd. et de Chir., v. 4, 425.
26. *Bradford and Lovett:* Treatise on Orthopædics.
27. *DeJonge:* Deutsche med. Woch., Berlin, 1881, vii. 477.
28. *Bastian:* British Med. Journ., 1890, i. 480, 629.
29. *Kaderer:* Archiv d. Heilkunde, xvii. 6, 451.
30. *Roser:* Centralblatt für Chirurgie, 1885, xii. 569.
31. *Gowers:* Med. Times and Gazette, London, 1876, ii. 517.
32. *Mohr:* Schmidt's Jahrbuch, Bd. clxxi. 65.
33. *Drachman:* Schmidt's Jahrbuch, Bd. clxxi. 62.
34. *Sayre:* Trans. Amer. Med. Assoc., 1879. xxx. 657.
35. *Savory;* St. Bartholomew's Hospital Reports, 1869, v. 45.
36. *Gibney:* Med. Record, New York, 1885, xxviii. 452.
37. *Althaus:* Boston Med. and Surg. Journ., 1886, cxv. 405.
38. *Poore:* New York Med Journ., 1878, xxvii. 140.
39. *Mitchell:* Amer. Journ. Med. Sci., 1889, N. s. xcvii. 439.
40. *Bradford:* Boston Med. and Surg. Journ., 1889, 405.

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