DISORDERS OF MICTURITION IN POLIOMYELITIS
Some Observations on Pathology and Treatment

BY

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Most textbooks give the impression that the bladder is seldom paralysed in poliomyelitis (Strumpell, 1931; Osler's Medicine, 1944; Brain, 1947). In the case of children this is probably so, but such an event is by no means rare in adults.

There are references to the occurrence of urinary symptoms in the earliest epidemics reported in American literature. Caverly (Infantile Paralysis in Vermont, 1924) mentioned them in connexion with three of the epidemics in Vermont. In 1894, there were 110 paralytic cases of which ten had retention of urine and two incontinence. In 1910, there were 69 cases of which 17 had retention, 4 incontinence and one frequency. In the small outbreak of the following year, 13 of the 27 paralytic cases had urinary symptoms. In 1913, a case was also reported in France (Schreiber and d'Allaines).

Bugbee (1925) claimed to be the first to report paralysis of the bladder as an initial manifestation of poliomyelitis. Eight years later, Toomey (1933) published details of 60 cases with urinary symptoms out of a total of 386 cases seen over a period of ten years, and noted that in 42 such symptoms preceded the onset of somatic paralysis.

The largest series of cases with bladder involvement seen in one epidemic was reported by Wright (1936), though his handling of the figures makes it impossible to tell the exact incidence. In the Los Angeles epidemic of 1934, 1,792 cases of poliomyelitis were diagnosed and he selected 420 for analysis "chosen at random, except that half were under 15 and half over 15 . . . Of the 210 under 15, 20 per cent had disturbed urinary functions during the acute stage, varying from slowness in starting to void to complete retention. In the younger children palliative measures were nearly always sufficient to relieve the symptoms, but in the older children the complaints were both more frequent and more difficult to relieve . . . Of the 210 adults, 65 per cent had disturbed urinary function." Apart from the high incidence of urinary symptoms, the other feature of note in this epidemic was the frequency of peripheral neuritis.

Poliomyelitis became a cause of considerable concern to the Army Command in India during the 1939-45 war. Although the actual number of cases was small, the high mortality made it the leading cause of death in many parts of the country. In Bombay, where the author spent the greater part of his service overseas between 1944 and 1947, poliomyelitis was responsible for
more than twice as many deaths as any other disease and actually accounted for almost half the deaths of officers during this period.

The clinical picture was very varied. The ordinary spinal type was that most commonly seen, though it tended to be particularly severe, with extensive paralysis and a high incidence of urinary symptoms. The ascending form was also frequently observed and accounted for most of the fatalities (see Table I).

<table>
<thead>
<tr>
<th>TABLE I. TYPE OF CASE</th>
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<tr>
<td>(Deolali, 1946, and Bangalore, 1946, are included for comparison)</td>
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<tr>
<td>Non-paralytic</td>
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<td>Bombay, 1944</td>
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<td>Bombay, 1946</td>
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The author saw some 45 adult cases of acute poliomyelitis in India and Burma. This communication is based on 26 of these which were under his observation for a sufficient length of time for careful study to be undertaken. In 22, the disease was contracted in Bombay: 20 were patients in the British Military Hospital, and the other two were seen on board ship between Bombay and Aden. Of the rest, 3 were patients in the British Military Hospital, Deolali and one in the Combined Military Hospital, Bangalore. All these cases were British servicemen, aged between 20 and 30, who had previously been quite fit.

Autopsies were performed on most of the fatal cases, but investigation was limited by the limited local facilities for histology and virus study. The characteristic naked-eye appearance consisted of meningeal hyperaemia and congestion of the spinal cord with occasional points of petechial bleeding.

Urinary Symptoms.—Of the 26 cases, 20 had symptoms of disturbed bladder function and, in 12, urinary symptoms preceded or appeared simultaneously with the onset of somatic paralysis. Seventeen cases had retention of urine lasting twenty-four hours or more and the other 3 complained of difficulty in emptying the bladder. In one patient, frequency of micturition and dysuria preceded retention. The bladder was palpable in 16 cases and 15 required catheterization.

Loss of bladder function was generally short-lived and normal micturition returned (provided the illness was not fatal) in from one to four days. Only one case failed to recover in this period. As there was still no sign of returning function after three weeks, the surgical specialist advised supra-pubic cystostomy and the bladder was still paralysed two months later when he was evacuated to the United Kingdom.

In 3 cases with retention of urine the effect of a subcutaneous injection of carbachol 0·25 mg. was tried. In Case K—a mild case with paresis of the abdomen and bilateral involvement of the hip girdle—there was an immediate response, one and a half pints of urine being voided. The bladder subse-
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quenty refilled, however, and a second injection had no effect. Normal function returned after thirty-six hours. Case O was also mild, with paresis affecting the abdomen, the left upper limb and the right iliopsoas. There was a good response to the injection and the patient passed urine normally thenceforward. The third case, P, was a severe one with involvement of both upper limbs, abdomen and chest, who was being given atropin 0.6 mg. four-hourly to keep the respiratory tract dry. Carbachol failed, probably on account of the antagonistic action of the atropin.

Other Clinical Features.—The clinical features of these cases have been reported in detail elsewhere (Short, 1948) so a brief summary only will be given here. The distribution of the paralysis in the individual cases is shown in Table II and the spinal segmental distribution in fig. 1. Two points call for special notice. The first is the frequent involvement of the upper limb which was paralysed, either alone or in combination with a lower limb, in 15 of the cases. The second concerns the peculiar distribution of the paralysis in the lower limb, where the fourth lumbar segment was more severely affected than the fifth lumbar and first sacral segments. The muscles most commonly and severely paralysed were the iliopsoas, the abductors and adductors of the thigh and the quadriceps femoris. The glutei were affected less than half as often as the iliopsoas, while the tibialis anterior and the peronei, usually regarded as the most vulnerable of the lower limb muscles in poliomyelitis, were rarely paralysed unless (or until) the lower limb was extensively involved. Eleven cases had a paralysis limited to the hip girdle whereas only two developed foot-drop without any paralysis above the knee. In a large proportion of the cases

Fig. 1.—Spinal segmental distribution of paralysis in 26 Bombay Service cases, showing two peaks, one in the cervical and the other in the lumbar region. Greatest incidence on the fourth lumbar segment.
**Table II. Distribution of Paralysis in 26 Cases**

| Muscle Group      | Case | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O | P | Q | R | S | T | U | V | W | X | Y | Z | Total |
| Cranial           |      | ++|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 2 |
| Trebezius         |      |   | 0 | ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| Deltoid           |      |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 8 |
| Spinatí           |      |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 15 |
| Pectorales        |      |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 12 |
| Biceps            |      |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 11 |
| Triceps           |      |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 8 |
| Forearm and hand  |      |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   | 19 |
| Intercostals      |      | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10 |
| Erector Spinae    |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 |
| Abdominals        |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 16 |
| Bladder           |      | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 20 |
| Iliopsoas         |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 29 |
| Glutei            |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 14 |
| Abductors         |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 20 |
| Adductores        |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 22 |
| Quadriceps        |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 21 |
| Hamstrings         |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 15 |
| Gastrocnemius     |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| Tibialis ant.     |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 14 |
| Peronei           |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 |
| Foot              |      | ++| ++| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5 |

**Note 1—Cases**

A—T: B.M.H., Bombay, Dec., 1945—June, 1947
U, V: At sea, June, 1947
W, X, Y: B.M.H., Deolali, June, 1946—April, 1947
Z: C.M.H., Bangalore, March, 1947

**Note 2—+ = paresis 0 = paralysis**
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inability to lift one lower limb off the bed was the first sign of somatic paralysis. At this stage paresis could usually also be demonstrated in one or more of the following groups: the adductors of the thigh, the abductors, or the muscles of the abdominal wall. Two severe and rapidly fatal cases developed a quadriplegia; in these the proximal and distal muscles were involved equally.

The abdominal muscles were paralysed in 16 cases, the erector spinae in 11 and the intercostals in 10. A triad frequently observed was paralysis of the abdominal wall and the iliopsoas (on one or both sides) together with retention of urine.

Three severe non-fatal cases were seen in which retention of urine was associated with an extensor plantar response. (In view of the frequency with which the abdominal muscles were paralysed in this series, absence of the abdominal reflexes was without value as an indication of a pyramidal lesion.)

Case E, an officer, aged 21, was admitted to the C.M.H., Kalyan (in April) with two days history of severe girdle pains around the lower part of the chest and slight difficulty in starting micturition. Apart from a band of hyperesthesia across the epigastrium, there were no abnormal physical signs at this stage. On the day following admission, the patient complained of backache, and definite neck rigidity was present. All the deep deflexes were exaggerated but both plantar reflexes were still flexor. Next day he developed paresis of both legs with retention of urine. During the succeeding days the paralysis extended, until both lower limbs, the abdominal wall and the left arms were paralysed. The left plantar reflex was now extensor. Suprapubic cystostomy was performed twenty-four days after admission. Two months later, on evacuation to the United Kingdom, the bladder was still paralysed and the left Babinski reflex positive.

Case K, an officer, aged 22, was admitted to the B.M.H., Bombay with a history of two days headache. Frequency of micturition and dysuria had been noted on the morning of admission, and when the patient tried to stand he found his legs were weak. Retention of urine developed and was at first relieved by carbachol, 0.25 mg., though subsequently catheterization became necessary for forty-eight hours. There was marked paresis of the abdomen and hip girdles and both plantar reflexes were extensor. After forty-eight hours the left plantar reflex returned to normal but the right was still extensor fifteen days after when the patient was last examined prior to his transfer to an orthopaedic centre.

Case T, was admitted to the B.M.H., Bombay, with paralysis of the abdomen, erector spinae and the left lower limb, paresis of the right lower limb and retention of urine. An extensor plantar response was obtained on the left side. The author did not have an opportunity of following this case further so the duration of pyramidal involvement is unknown.

DISCUSSION

That the symptoms of poliomyelitis are more severe in adults, the paralysis more extensive and the mortality higher has frequently been observed (Limper et al., 1931; Nissen, 1947; Short, 1948). Parallel with this greater severity goes a higher incidence of urinary symptoms. Wright's observation (1936) that paralysis of the bladder was more frequent in the higher age groups has repeatedly been confirmed in subsequent epidemics involving the adult population. Urinary symptoms have figured prominently in the reports on outbreaks of poliomyelitis among service personnel during the 1939–45 war. In the 1942–3 Malta epidemic, bladder involvement was observed in over 20 per cent of the Service cases (Bernstein et al., 1945); whereas of the 426 civilian cases,
98.5 per cent of whom were under 10 years of age, only one required catheterization (Agius et al., 1945). Van Rooyen and Morgan (1943) mentioned retention in two and incontinence in one of their seven brief case-histories of soldiers who died of poliomyelitis in Egypt.

Poliomyelitis is by no means rare among the native population of India though, owing to the absence of large epidemics, it has as yet received little attention. The disease is virtually confined to infants and young children. In Bombay, urinary symptoms are said to be uncommon though, interestingly enough, the only adult Indian known to have contracted the infection in the past ten years suffered from retention of urine.

Pathogenesis of Urinary Symptoms.—The site of the lesion responsible for urinary symptoms in poliomyelitis has hitherto received little attention, probably on account of the belief that such symptoms are uncommon. Hence it has been assumed either that the vesical centre in the spinal cord is affected or that there is some damage to the nerves supplying the bladder.

The physiology of micturition is complex. At least six reflexes have been described and four centres have been postulated. The primary reflex is a contraction of the detrusor evoked by distension of the bladder. The pelvic nerves form both the afferent and efferent limbs of this reflex arc which has its centre in the hind-brain. Some of the subsidiary reflexes have their centre in the sacral cord, probably in the second, third and fourth sacral segments. There is believed to be a third centre in the mid-brain, and the presence of voluntary control presupposes the existence of a fourth centre at the cortical level. The connexions between these four centres are as yet imperfectly understood. Reflex contraction of the detrusor is reinforced by voluntary contraction of the abdominal wall, diaphragm and muscles of the pelvic floor.

With such an intricate system of nervous control, micturition may be upset by lesions at many different levels. Thus paralysis of the bladder may result from such varied causes as injury to the cauda equina or sacral cord, transverse myelitis of the lumbar or dorsal segments and cerebral thrombosis.

In view of the known affinity of the virus of poliomyelitis for the motor horn cells, it is reasonable to consider first the possibility of a lesion of the sacral centre. There are two strong arguments against this conception of the pathology in the cases seen in Bombay. In the first place, if the lateral grey columns were damaged it would be reasonable to expect that the anterior grey columns of the same segments would also be damaged. No such association was in fact observed; voluntary muscles innervated by the second sacral segment being rarely involved and those innervated by the third segment never. On the other hand, 7 cases were observed (Cases B, G, J, N, O, V, Z) with retention of urine in which there was no evidence of cord involvement below the fourth lumbar segment. It is hard to believe that in these cases the lateral columns of the sacral cord were picked out and the anterior columns spared. The muscles most frequently paralysed in patients with retention of urine were the abdominals and the hip flexors—muscles deriving their nerve supply from the lumbar and lower dorsal segments. The other argument against the theory of damage to the sacral centre is the fact that bladder symptoms were
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almost invariably transient. In only one case (Case E) did retention of urine persist beyond four days. In all the other patients, normal micturition was established long before any return of power could be detected in the voluntary muscles affected. In Case E, paralysis was still complete after two months and in this single instance damage to the neurones in the sacral cord may reasonably be assumed. It is noteworthy that this patient also had paralysis of voluntary muscles innervated by the sacral cord.

Toomey (1933), impressed by the observation that paralysis of the detrusor was rarely accompanied by paralysis of somatic muscles supplied by the same spinal segment, postulated a peripheral lesion. In the following year there occurred the Los Angeles epidemic with the highest incidence of urinary symptoms yet recorded. This epidemic was peculiar, however, in that frequency of micturition and attacks of acute vesical distress were common and kept recurring over a period of eighteen months. Many of the cases also had signs of peripheral neuritis. Wright (1936) concluded that the bladder symptoms were due to a peripheral neuritis. This was manifestly an unusual epidemic and it would be unwise to generalize from these conclusions.

Neither of the above theories offers a satisfactory explanation of the transitory retention of urine which was observed in so many of the Bombay cases. The possibility of a lesion interrupting the pathway between the centre in the sacral cord and that in the hind-brain must therefore be considered. Retention of urine is a characteristic symptom of a transverse myelitis involving the lumbar or dorsal cord, and this may result from a great variety of pathological processes including infections, toxicoses and toxi-degenerations. Among the infective agents, the virus of poliomyelitis is a rare cause (Wilson, 1940)—the inflammatory reaction in the anterior grey columns being so intense that it overflows into the white matter. Less rarely, poliomyelitis gives rise to a minor degree of the condition with evidence that one or more of the long tracts is involved—particularly the pyramidal and spino-thalamics. There were three cases with extensor plantar responses in the present series all of whom suffered from retention of urine. It is suggested that in these three cases the urinary symptoms had the same pathological basis as the pyramidal lesion. If this is so, is it not conceivable that in other cases the tracts subserviving bladder control might be compressed and interrupted without the oedema being extensive enough to interrupt the pyramidal tracts sufficiently to give rise to an extensor plantar response? Such an explanation appears to fit the facts better than any other. It has its basis in established pathology, for oedema and swelling of the cord in those segments where there is extensive damage to the anterior grey columns has frequently been observed. Since the site of the disturbance may be at any level above the sacral cord, it accounts for the association of urinary symptoms with somatic paralysis of a lumbar and lower dorsal distribution. Furthermore, the brief duration of the bladder paralysis is readily understood if the oedema of the cord is regarded as a short-lived accompaniment of the phase of neuronal destruction.

There may be some cases in which retention of urine is due simply to paralysis of the abdominal muscles without any defect in the reflex mechanism
controlling micturition. The detrusor is used to relying in some measure upon a voluntary raising of the intra-abdominal pressure. If this support is withdrawn and particularly if, in addition, micturition has to be attempted in an unaccustomed position—in bed, for instance—retention may result. After a few days the detrusor readjusts itself; sometimes also the patient finds a particular position in which the act can be performed. This explanation cannot be held to account for more than a small proportion of the 20 cases with urinary symptoms described in this paper for almost half of them developed retention of urine before paresis of the abdomen, 3 cases showed no abdominal weakness at any time, and in one frequency and dysuria preceded retention.

The Use of Carbachol.—It seems so obvious to attempt to relieve retention of urine in poliomyelitis by the administration of carbachol that it is remarkable that none of the neurological textbooks mention the drug. Indeed, the author has been unable to trace any mention of its use in the literature, though this is so voluminous that some reference may well have escaped his notice. The omission is the more remarkable in view of the fact that its close cousin, prostigmine, is undergoing extensive trial in the U.S.A. as a stimulant of paralysed muscles in the early convalescent stage (Kabat and Knapp, 1943; Boines, 1944; Eveleth and Ryan, 1944). Moreover, carbachol is of proved value in the neurogenic retention of urine occurring in the course of tabes dorsalis (Langworthy, 1936).

In the three cases in this series in which it was tried, there was one complete and one partial success. It is not clear why in the latter there was no response to a second injection. The failure to obtain a response in Case P may well have been due to the fact that he was receiving atropin 0·6 mg. four-hourly in an attempt to keep the respiratory tract dry. It has been shown that such a dose of atropin completely blocks the normal effects of carbachol 0·3 mg. 20 minutes later (Goodman and Gilman, 1941).

Besides the two cases noted above, the author is able to report two other successes. An adult male civilian in Bombay developed poliomyelitis with extensive paralysis below the waist and retention of urine. By the use of "Moryl" an evacuation of the bladder was obtained twice daily for four days at the end of which period normal function returned. The other case was a man aged 49 with severe paralysis of both legs and the abdomen. When first seen, he had already been catheterized. Twelve hours later, when the bladder was again distended he was given carbachol 0·25 mg., and as there was no response a second injection of 0·5 mg. was given. There was still no response so twelve-hourly catheterization was instituted. After three weeks, by which time the question of cystostomy was being seriously considered, an injection of carbachol 0·25 mg. was tried again. This time it was successful and the patient was able to pass urine normally thenceforward.

SUMMARY

Urinary symptoms, though infrequent in infants and young children are not uncommonly seen in adult cases of poliomyelitis.

Of 26 cases of poliomyelitis in Service personnel in India, 20 had urinary symptoms and 15 required catheterization.
Paralysis of the bladder was generally transitory and in all but one case normal function had returned by the end of the fourth day.

The site of the lesion responsible for the urinary symptoms is discussed. The view is advanced that in the majority of these cases the essential pathology was an inflammatory œdema around the damaged anterior grey columns of the lumbar or dorsal cord interrupting the tracts linking the bladder centre in the sacral cord with that in the hind-brain.

Carbachol is sometimes successful in producing an evacuation of the bladder and should always be tried before resorting to catheterization.

My thanks to Col. F. M. Lipscomb for much helpful criticism.

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