The Management of Chronic Interstitial Cystitis by Differential Sacral Neurotomy*

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In 1915, Hunner introduced his observations on ulcer formation in the summit of the bladder in the presence of interstitial cystitis with these words:

"I wish to present for your consideration this evening a group of cases in which the lesion differs from the so-called Fenwick ulcer which has been the type heretofore described as simple ulcer. This group presents a strikingly uniform picture, an appreciation of the features of which by our branch of the profession will save the patient from much suffering, both from errors in diagnosis and consequent futile operations on other organs, and from the unnecessary prolongation of ordinary cystitis treatment when more radical measures are demanded... There is nothing absolutely characteristic in the cystoscopic picture. Perhaps the most characteristic thing is the insignificance of the lesion as compared with the long duration and intensity of the patient's suffering."¹,⁴

The pathological entity which Hunner described that evening has become known to us as a Hunner ulcer.

Intractable pain and urinary frequency are the most incapacitating features of chronic interstitial cystitis. Hypertonicity, urgency, incontinence, and concomitant reduction of bladder capacity characterize this chronic and disabling disorder. Far-advanced cases show a bladder capacity ranging from 30 to 80 cu cm.

Reduction of parasympathetic outflow to the detrusor and reduction of the sacral somatic outflow result in decrease of bladder hypertonicity and, secondarily, an appreciable increase of bladder capacity. This has been demonstrated experimentally;¹³ it has also been demonstrated in the clinical management of the neurogenic bladder.⁶-¹⁰ Based on this principle, differential sacral neurotomy was employed first in 1954 in the treatment of interstitial cystitis.¹² In utilizing the same principle, Bohm and his associates have performed posterior sacral rhizotomy in cases of interstitial cystitis with resulting reduction of urinary frequency, reduction of pain, but without appreciable effect on bladder capacity.² It appears then that anterior and posterior rhizotomy, or sacral neurotomy, rather than posterior sacral root section are required to achieve the necessary decrease in bladder tone with the concomitant increase in bladder capacity.

The etiology of the disease remains obscure. Bohm and his associates³ have described histologic changes in sacral nerves in the presence of interstitial cystitis, consisting of endoneural and perineural fibrosis, myelin sheath degeneration, round cell infiltration, and the appearance of granulation tissue. On the basis of clinical studies, they have suggested that sacral rhizopathy may represent the etiology of a variety of pathologic entities such as urethrotrigonitis, prostatitis, interstitial cystitis, and coccygodynia.

Investigations have been carried out in dogs and monkeys.⁷ Sacral rhizopathy has been produced by application of aluminum gel or of lycopodium spores. The series of studies carried out in dogs was inconclusive. Studies carried out on monkeys were suggestive of an etiologic relationship between sacral rhizopathy and interstitial cystitis. Eight monkeys were subjected to lumbosacral laminectomy and application of lycopodium spores or aluminum gel to the sacral nerve roots. Five monkeys were used as controls. Preliminary cystoscopy was carried out on all 13 monkeys. The period of observation averaged 14 months. Four of the eight animals with sacral rhizopathy developed changes in the bladder, consisting of thicken-
ing of the lamina propria due to edema, round cell infiltration, and submucosal fibrosis. There was no frank ulceration. None of the bladders of the control group showed any histologic changes.

There is, then, to date no conclusive answer to the question whether sacral neurotomy, or rhizotomy, has a beneficial effect on the signs and symptoms produced by interstitial cystitis in man because of the resulting increase in bladder capacity or because of elimination of diseased nerve roots. In our own cases of interstitial cystitis, we have not found any histologic changes in the sectioned sacral nerves. Observations made by Bohm and his associates, and results of studies on monkeys, are sufficiently interesting to warrant further investigations of the etiologic role which sacral rhizopathy may play in the production of Hunner ulcer.

Clinical observations have established that differential sacral neurotomy, or anterior and posterior sacral rhizotomy, have a beneficial effect on the symptoms produced by interstitial cystitis in man.°11,12,14 The question remains whether this effect is the result of reduction of parasympathetic and sacral somatic outflow, or is due to elimination of impulses to the bladder travelling by way of pathologically altered nerve roots.

**Clinical Management**

It is essential to bring patients with Hunner ulcer to neurosurgical attention prior to development of bladder wall fibrosis which would prohibit any appreciable increase in bladder capacity. Preliminary studies include determination of bladder capacity, urinary output with each micturition, continence, and the amount of residual urine.

The potential of bladder capacity can best be determined under spinal anesthesia with a level caudad to L-1. Patients with a sufficient degree of bladder wall fibrosis, prohibiting any appreciable increase in bladder capacity, should be excluded from consideration for differential block studies and sacral neurotomy. While a bilateral block of S-3 yields the best results in the majority of the cases, test block of S-2 and possibly S-4 should be carried out. In some instances a combination block of S-2 and S-3 may have to be done. In isolated cases, bilateral blocking of one pair of nerve roots, and unilateral blocking of an additional root may be indicated.

When first doing sacral block studies some 20 years ago, we depended on measurements relating the position of the sacral foramina to the posterior superior iliac spine. Since then it has become increasingly clear that some of our failures then were due to technical errors. Dr. Joseph H. Allen has worked out a fluoroscopic technique for the localization of posterior sacral foramina which has made it possible to perform differential sacral block studies with dependability.1 After frontal and lateral sacral views have been taken, lead arrows are placed under fluoroscopic control at the lateral margin of the lateral sacral crest overlying each posterior sacral foramen. Accurate position of the markers is verified by another set of frontal and lateral films. Number 20 gauge spinal needles are then introduced into the posterior foramina. The presence of branches of the lateral sacral arteries in the posterior foramina necessitates aspiration prior to injection. So as to prevent flooding, not more than 1 cu cm of 1% xylocaine is injected into each foramen. The needle is then withdrawn. Bladder studies are carried out within 45 minutes of the onset of the block.

Cases developing urinary incontinence while the block is effective should be eliminated from consideration for differential sacral neurotomy. The residual urine should not exceed 60 cu cm. In making final selection of cases suitable for neurosurgical intervention, it is well to remember that the block is never quite as effective as the actual neurotomy. Nevertheless, the block is a dependable indicator of the potential improvement that may be achieved with differential sacral neurotomy. Whenever the bladder capacity increases under the block by 100 to 200 cu cm, one may safely assume an even greater increase following neurotomy.

Operation is carried out by midline incision permitting exposure of the sacral foramina by use of self-retaining retractors. The foramen is enlarged with a Kerrison punch. Each sacral nerve is picked up with two blunt hooks and divided. Three characteristic cases are described below.

**Case 1.** This patient, first reported in 1956,12 began to develop increasing urinary
frequency and intractable pain in 1950. The diagnosis of chronic interstitial cystitis was established. Conservative urological treatment failed to alleviate symptoms. Bladder capacity ranged from 30 to 60 cu cm. Under spinal anesthesia, bladder capacity was increased to 215 cu cm suggesting that there was no clinically significant degree of bladder wall fibrosis. A xylocaine block of the third pair of sacral nerves resulted in a capacity of 130 cu cm, voiding without incontinence, and without residual urine. A differential sacral neurotomy of S-3 bilaterally was performed in 1954. Following operation, the patient's bladder capacity was 300 cu cm. She was able to void without pain, without incontinence and without residual urine. During the following year she experienced occasional episodes of frequency and mild pain. She became asymptomatic 1 year following surgery and has remained so to this date, voiding an average of 400 to 500 cu cm of urine.

Case 2. A 31-year-old married woman had suffered from chronic interstitial cystitis, marked frequency and intractable pain for a period of 5 years. For over a year, she had been unable to engage in any social activities because of the ever-increasing frequency, accompanied by intractable pain. Day and night, she was unable to hold her urine longer than 45 minutes to 1 ½ hours. Blocking of the S-3 root bilaterally with 1% procaine on June 7, 1954, resulted in a capacity of 180 cu cm. The patient was relieved of all pain and was able to empty her bladder without residual and without incontinence. Pain and frequency recurred 36 hours after the block. On June 11, 1954, the S-3 block was repeated and a similar result obtained. Sacral neurotomy of S-3 bilaterally was performed on June 15, 1954. Following operation the patient was able to void without straining, without residual and without incontinence. Her bladder capacity was 600 cu cm and she had no bladder pain. She has remained asymptomatic and has an average capacity of 400 to 500 cu cm.

Case 3. A 73-year-old white woman had a history of urgency, frequency, nocturia, and dysuria of 6 years' duration. There had been a gradual progression of symptoms with decreasing bladder capacity and increasing bladder pain. Bladder capacity ranged between 30 and 60 cu cm. Painful micturition had become so severe and so frequent as to prevent her from participating in any social activities and from getting adequate rest at night. Cystoscopy, as described by Dr. Charles E. Haines, revealed a Hunner ulcer located in the superior portion of the bladder. Bladder capacity under spinal anesthesia increased from 30 to 210 cu cm, indicating a mild degree of fibrosis of the bladder wall. Bilateral block of the third sacral nerves with 1% xylocaine resulted in a bladder capacity of 125 cu cm. A repeat block produced a capacity of 150 cu cm. Under the block, the patient was able to void without pain and without incontinence. There was no appreciable amount of residual urine.

Bilateral neurotomy of S-3 was performed on July 14, 1967. Histological examination of the sectioned sacral nerves showed no pathologic changes. At the time of discharge from the hospital on July 26, 1967, the patient was voiding an average amount of 180 cu cm without pain, without incontinence and without residual. Since then she has reported an average amount of 275 cu cm of urine with each voiding without pain and without incontinence. She has remained asymptomatic until this date.

Summary

The rationale for the employment of differential sacral neurotomy in the management of dysuria and intractable pain due to chronic interstitial cystitis has been discussed. Differential sacral block studies as an aid in the selection of patients for sacral neurotomy have been described. The characteristic case histories of two patients who had a differential sacral neurotomy 14 years ago have been reported; a third case, illustrating current methods of management, has also been described.

References

1. Allen, J. H. (Unpublished data.)