# TREATMENT OF BLADDER DYSFUNCTION AFTER NEUROLOGIC TRAUMA[1]

LLOYD G. LEWIS, Lt. Colonel, M.C., A.U.S.[2]

*From the Walter Reed General Hospital, Washington, D. C.*

## ANATOMY

The great muscle of the bladder, the detrusor, is made up of interlacing fibers of smooth muscle derived from the cloacal membrane. Henle and Kohlrausch and Rosenthal in 1856 described circular and longitudinal detrusor fibers which invested themselves around the vesical orifice so that the longitudinal fibers passed through the circular fibers at the outlet. Young and Wesson showed that the fibers intermingled in no set pattern over the fundus. Wesson has beautifully demonstrated the orderly arrangement of fibers at the orifice. No true internal sphincter has ever been described. Griffiths has shown that in normal individuals no thickening of musculature exists at the vesical orifice.

Within the bladder the trigonal muscle is situated between the ureteral orifices and the prostatic urethra. The trigonal fibers and the muscles of Bell pass through the detrusor fibers of the orifice. This mechanical arrangement, the hypertrophy of the trigonal muscle in cases of median prostatic obstruction, and cystoscopic observation, prompted Young to assign to the trigone the function of opening the vesical orifice for micturition.

## INNERVATION

The detrusor is innervated by the autonomic nervous system by fibers from the 2d, 3d and 4th sacral roots which pass by way of the hypogastric plexus on the lateral surfaces of the rectum. Sensation of stretch or distention is elicited from nerve endings on the muscle bundles passing through nerves traversing the 2d, 3d and 4th dorsal sacral roots. Dees has produced a tabetic type of bladder by experimentally dividing these roots bilaterally. Sensations of pain and temperature from the bladder are recorded through nerves which pass through the hypogastric plexus by way of the presacral nerve to enter the thoracico-lumbar cord. Sympathetic fibers from the lumbar segments innervate the blood vessels of the bladder wall but it is improbable that the sympathetic fibers have any direct influence on the detrusor muscle. The trigone as well as the musculature of the prostate, seminal vesicles and ejaculatory ducts, all derived from the wolffian duct, are innervated by the sympathetic nervous system.

## PHYSIOLOGY OF MICTURITION

The desire to urinate is recorded from muscle tension irrespective of bladder volume (Langworthy, Kolb and Lewis). We have all experienced strong desire to urinate on small as well as on large bladder capacity. Through reflex arcs

[1] Read at a meeting of the Chicago Urological Society in January 1945.
[2] On leave, Brady Urological Institute, The Johns Hopkins Hospital, Baltimore, Md.

in the sacral cord the detrusor is activated by stretch. Since the vascular bed of the corpora cavernosa is also innervated by parasympathetic fibers from the 1st, 2d and 3d sacral segments, the phenomenon of morning erections in the uninhibited phase is understandable. Voiding is brought about by uninhibited detrusor stimulus from pressure in excess of 12 mm Hg by reflex contraction. Urination in the newborn is uninhibited and automatic, but after conditioned reflexes have been acquired, urination may be deferred willfully and the intravesical pressure voluntarily lowered. If circumstances are not auspicious, urination can be inhibited by contraction of the voluntary sphincter which in turn sets up reflex relaxation of the detrusor, as described by Barrington.

Urine is normally retained at the vesical orifice, as described by Voelcker and Lichtenberg, Leedham-Green, Barringer and McKee, and by Uhle and Mackinney, in beautiful cystographic studies. The most important factor in urinary control is the ability of the detrusor to contract around the vesical orifice. Patients with the external sphincter shot away can retain urine and void normally except that urination cannot be suddenly voluntarily interrupted. Patients immediately after perineal prostatectomy may void at intervals through the perineal prostatic urethral fistula. Patients after radical perineal prostatectomy retain urine only if the vesical orifice is free to contract uninhibited by scar. After transurethral or suprapubic prostatectomy, urinary control becomes normal as soon as the vesical orifice contracts freely. The voluntary sphincter is not adapted anatomically for retention of urine. Striated musculature has not the endurance for continuous maintenance of tone which is the great attribute of smooth muscle. Operations for cure of incontinence should primarily be directed to relief of obstruction at the vesical orifice. Incontinence following transurethral resection may be relieved by complete removal of prostatic tissue at the orifice. Otherwise operations for urinary control must be directed toward formation of a kink or constriction of the urethra externally as by gracilis transplant.

Since the detrusor and the trigonal muscles are antagonistically innervated, it was difficult for those of us who have observed, as Young has described, the contraction of the trigone at the onset of urination through the cystoscope, to correlate the function of the trigone in micturition. Apparently, trigonal action is not essential in the normal individual, but it is of great assistance to patients with median prostatic obstruction. Patients urinate normally after division of the sympathetic innervation by presacral neurectomy. The action of the trigone can only be correlated if we assume overflow innervation or direct stimulation of the trigonal muscle by stretching as the detrusor contracts and the base of the bladder widens at the onset of voiding. It seems improbable that trigonal contracture would open the vesical orifice unless contraction of the detrusor and fixation of the trigonal muscle occurs simultaneously. On the other hand, the sympathetic innervation of prostate, ejaculatory ducts, seminal vesicles and the trigone might suggest a sexual function for the trigonal muscle. After presacral neurectomy, ejaculation is impossible because of paralysis of the ejaculatory and prostatic musculature. After prostatectomy, ejaculation might be considered incomplete because of a weakened vesical orifice allowing sperm to enter the

bladder. However, Langworthy has shown that dogs do not ejaculate normally after transvesical section of the trigonal muscle. Contracting without the support of the detrusor, the trigone will thicken in its midportion at the vesical orifice, thus effectively preventing passage of semen into the bladder.

In the light of this physiologic concept, how can one justify presacral neurectomy in the treatment of the neurogenic bladder? Certainly section of the sympathetics will have no effect on a spastic vesical orifice. There being no true internal sphincter and no proven sympathetic innervation of the detrusor or vesical orifice musculature, we must look for another explanation of the effect produced by this operation. When peripheral circulation is impaired as in frost bite or by peripheral vascular disease, increase in vascularity can be accomplished by sympathectomy. We believe the same effect on vascularity of the bladder wall accounts for the increase in bladder tone following this operative procedure.

## THE NEUROGENIC BLADDER

Since the bladder is controlled by reflex arcs through the brain and spinal cord, any interruption of either the motor or sensory sides of the reflex arc will make the bladder inefficient either for storage or evacuation of urine. Such a concept calls for differentiation of various types of dysfunction in correlation with the neurological defect. The urologist and the neurologist must cooperate in establishing the correct diagnosis before any logical form of treatment can be instituted.

## THE TABETIC BLADDER

After division or injury to the dorsal sacral roots by trauma or disease (syphilis), sensation of bladder fullness is partially or completely lost. The patient with tabes dorsalis seldom has total loss of sensation of distention. After extensive injury or pelvic surgery, part or all of the sacral innervation may be lost. The patient has no desire and does not void. The bladder becomes distended and secondary atrophic changes occur. Because the detrusor is atrophic, dilatation of the vesical orifice is regularly noted unless prostatic obstruction complicates the picture. When the parasympathetic fibers are divided, detrusor contracture cannot occur. In either instance the reflex arcs are divided in the lower segment and no automaticity can develop.

## LESIONS OF THE MOTOR PATHWAYS

When the motor pathways in the spinal cord are interrupted, voluntary urination with urgency and frequency occurs as in disseminated sclerosis or in cerebral arteriosclerosis. This clinical picture was described by Langworthy, Dees and Lewis, in 1935. The predominant motor innervation is from the left cerebral cortex. Patients with right hemiplegia, left brain lesion, are frequently incontinent. Patients with left hemiplegia have fewer bladder symptoms.

## TRANSVERSE MYELITIS

When the spinal cord is *completely* divided above the level of L-I, one or more reflex arcs remain intact. The efficiency of the uninhibited automatic bladder

developed therefrom depends upon the number of reflex arcs intact. Patients with lesions from C-VII to D-VII have fairly efficient involuntary voiding. Patients with lesions from D-VII to D-XII may have considerable automaticity but it is not very satisfactory in most instances and considerable residuum remains. Lesions at L-I, around the conus medullaris and in the cauda equina do not allow development of automatic bladders. These patients have retention and can only develop overflow incontinence.

### ACUTE CEREBROSPINAL INJURY

The immediate effect of severe injury to the brain or spinal cord is urinary retention. This may result from either temporary or permanent injury to the spinal pathways. Hemorrhage, edema, or compression of the fibers without actual division of them results in spinal shock. Bladder innervation is interrupted for a period of a few hours to periods up to 6 to 8 months. Simple spinal shock is normally relieved within 48 hours. However, compression of the cord can exist until either postural or surgical correction of the impingement restores circulation and the undivided tracts regain their function of transmission of impulses. Although it has been the common teaching that cord fibers compressed for more than 48 hours are permanently damaged, we shall show that patients have regained bladder function weeks and even months after surgical decompression of the cord.

Spinal cord injury may result in either complete or partial permanent disability, partial, or complete temporary disability. Vesical dysfunction occurs in each category. At the time of injury it is not possible to determine to what extent the cord is damaged. Neither is it possible to determine the type nor to estimate the duration of bladder dysfunction. Treatment in this phase must be considered in the light of three factors: (1) Prevention of ascending urinary tract infection; (2) protection of paralyzed muscle; (3) restoration of function. Three types of treatment have been advocated. Each method emphasizes the importance of one of the three factors.

Young has advocated "no instrumentation" to prevent infection. This is the ideal method if spinal shock clears and normal voiding or automaticity develops within 48 hours. However, spinal shock does not always clear within 48 hours; the overstretched muscle does not respond to normal stimulus and overflow incontinence rather than automatic voiding occurs. We are all familiar with the possibility of infection of residual urine.

Many neurosurgeons and urologists demand drainage of the bladder after spinal injury, to prevent overstretching of the paralyzed muscle. Either continuous catheter or suprapubic drainage had been instituted in all of our cases transferred from other Army hospitals. Obviously cystitis results and prevention of ascending infection depends upon the well functioning tube. Some of our patients had catheter drainage initially and this was replaced by cystostomy on transfer to another hospital. Several patients had suprapubic drains removed in other hospitals to which they were transferred, to be replaced by the urethral catheter with tidal drainage. We are convinced that suprapubic drainage is not

the ideal method and should not be used routinely. Although much has been written about placing the tube high at the vault of the bladder, common practice is to place the tube close to the symphysis pubis. Stab wound cystostomy is not adequate in these cases.

Monroe and Hahn have advocated tidal drainage. Some urologists have gained the impression that all that is necessary in case of spinal injury is the insertion of a retention catheter with tidal irrigation; that ascending infection is prevented, urethritis reduced to a minimum, and restoration of function follows. Monroe has stated that tidal drainage is not indicated in all cases. Our experience with various modifications of the Monroe apparatus is that the bladder is not always emptied after the syphonage, either due to the change of position of the patient or due to air trapping. Irrigation of the bladder must be used as an adjunct.

Certainly continuous bladder drainage with or without tidal irrigation is not indicated after simple spinal shock. We would therefore practice "no instrumentation" for at least 24 hours after injury. The bladder does not become palpably distended unless large quantities of intravenous or subcutaneous fluid have been given. If the patient has not passed any urine and the bladder is palpably distended, we would catheterize using the strictest aseptic technique. If, following this decompression, the patient cannot void, and a second catheterization is necessary, we would leave the tube in place for continuous drainage. Irrigation pressures must not exceed 12 mm Hg whether tidal or intermittent irrigation is used. Mercurial drugs must not be used for continuous or massive irrigations. We prefer acid solutions such as boric acid, Suby's solution, or buffered citrate. The catheter must be changed at least every fifth day. We have usually used a No. 20 Foley bag retention catheter. It is our impression that more incrustation occurs on these latex catheters than on the old red rubber ones. However, this difficulty can be overcome by frequent change of the tube, and the advantage of the Foley catheter outweighs its disadvantage. Meatotomy must be performed if narrowing exists. The catheter must be of adequate size but not too large. Paralyzed tissues will not tolerate pressure. For this reason bed sores are frequent and urethritis and periurethral slough is common. Certainly great care in the passage of catheters and maintenance of adequate catheter drainage is of utmost importance.

After long periods of urethral catheter drainage, eight periurethral abscesses have occurred in our cases. All have been located at the penoscrotal juncture. When not contraindicated, we have done perineal urethrostomy. This is accomplished without anesthesia, by making a one-inch incision in the bulb over a sound, bulging the urethra toward the perineum. All tissue down to the sound is divided in one stroke. The urethral edges are grasped with Allis clamps while a large de Pezzer or a Malecot tube is passed through the external sphincter and prostatic urethra into the bladder. After the initial dressing none is required. The tube is very easy to change. When drainage is discontinued the catheter may be removed, and the patient voids through the urethra at intervals. We recognize there are contraindications to this method, such as the occasion of

prostatitis and prostatic abscess, but we have been well pleased with the procedure. These patients are able to walk with support or crutches, without urinary leakage by clamping the tube or allowing it to drain into an urinal strapped to the leg.

The cases described in table 1 are used to illustrate the types of lesion and results of treatment in neurologic bladder dysfunction.

Ten cases of complete transverse lesions of the spinal cord are outlined. In 2 instances the nutrient arteries of the cord were severed, resulting in degeneration below the site of injury. The 1 patient with a lesion at C-VII died. The other, with transverse section by compression at D-V, is alive but there has been no return of reflex activity 2 years after injury. Five had complete transverse myelitis at the levels of D-IV, D-V, D-IX and D-X. All of these patients developed automatic voiding from 4 to 6 months after injury. Three had complete severance of the cord at the level of L-I. None of these developed any reflex bladder activity other than inefficient myoneural contractures.

Five cases of incomplete lesion of the cord are outlined. Two of these patients had complete severance of bladder innervation at levels of D-I and D-V. Both developed efficient automatic bladders 1 and 2 months after laminectomy and decompression of the cord. One patient had a tabetic type of bladder lesion following compression at D-VII. There has been no return of bladder sensation and no reflex activity one year after injury. Two patients had incomplete lesions of the cord at L-I. Both developed uncontrolled voiding within one month after decompression of the cord. Urination is urgent in both cases and cannot be interrupted, due to loss of external sphincter innervation. Neither has any residual urine.

The value of early decompression of the cord is illustrated by a patient who suffered complete loss of sensation and reflex activity below L-I, produced by compression fracture of L-I. Laminectomy and decompression of the cord 6 days after injury allowed return of reflex activity 21 days after operation and normal voiding 47 days after injury.

Four types of incontinence must be differentiated. Complete total incontinence results from complete loss of urethral and sphincteric resistance. Paradoxical or overflow incontinence is caused by division of the sensory pathways which transmit stretch sensation, as in tabes. The incontinence of urgency and frequency, with or without loss of external sphincter innervation, is described. Involuntary or automatic voiding occurs after complete section of the bladder innervation above the level of L-I.

The following principles are recommended in the urological care of patients following spinal injury:

1. No instrumentation for 24 hours.
2. Absolute aseptic catheterization at 24 hours if necessary.
3. The use of urethral retention catheter if 2d catheterization is necessary.
4. The use of intermittent or tidal irrigation of the bladder with acid solutions to prevent incrustation cystitis.
5. Perineal urethrostomy in selected cases.

TABLE 1

| | TYPE OF INJURY | INITIAL TREATMENT | SUBSEQUENT TREATMENT | ADMISSION W.R.G.H. | TREATMENT | RESULT |
|---|---|---|---|---|---|---|
| 1 | Complete transverse myelitis C-VII, 1/13/43. Truck accident. | Catheterized 24 hours. Overflow incontinence to 1/29/43. Head traction. | Retention catheter drainage 1/29/43. | 2/21/43 gross pyuria, decubitus severe, catheter obstructed. Cystometry showed no reflex response. | Tidal drainage. Striker frame. Decubitus healed. | Died 7/14/43; pyelonephritis, complete cord degeneration below C-VI. |
| 2 | Complete transverse myelitis D-IV, 4/25/43. Shell fragment. | Catheterized daily 4 days, cystostomy 4/29/43. | Cystostomy tube removed, retention catheter placed. 5/2/43 Tidal drainage (Laminectomy 5/6/43). | 6/16/43 gross pyuria. Cystometry 7/4/43 showed slight stretch response. No voiding. | Tidal drainage 8/9/43. R.U.-400 cc. | Automatic voiding 6 months after injury. Voiding Q 1–2 hours. |
| 3 | Complete transverse myelitis D-IV, 3/21/43. Bullet wound. | No catheter; overflow voiding. | Cystostomy 3/27/43. | 6/15/43 gross pyuria. Cystometry showed some stretch reaction. | Tube removed 7/11/43; voiding; cystometry 7/15/43 showed good stretch response. | Efficient automatic bladder 4 months after injury. |
| 4 | Complete transverse myelitis D-V, 11/12/42. Bullet wound. | Retention catheter 24 hours after injury. | Cystostomy 12/29/42. | 4/27/43 gross pyuria, decubitus severe, chills and fever. Cystostomy leaking. | Tube replaced. Striker frame. B.C.-100 cc. | Automatic voiding 6 months after injury. Small capacity due to fixation of bladder. Decubitus healed. |

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| | Complete transverse myelitis D-V, compound compression fracture D-V, VI, VII, VIII. Shell fragments 1/3/44. | Urethrostomy 1/4/44. (Laminectomy 1/27/44, macerated cord.) | Urethrostomy discontinued. Cystostomy because of chills and fever. 1/27/44. | 4/27/44 cystostomy satisfactory. Cystometry shows no reflex activity. | Continued cystostomy drainage. | No evidence of automaticity. Probable degeneration of cord below site of injury. Prognosis poor. |
| 6 | Complete transverse section D-X. Fracture D-IX, 12/2/42. | Retention catheter drainage. | Continued. | 1/23/43 gross pyuria, decubitus severe. | Periurethral abscess. 3/12/43, urethrostomy. Striker frame. Cystometry shows good reflex action 3/20/43. | Urethrostomy discontinued. 3/20/43. Automatic voiding. Decubitus healed. |
| 7 | Complete transverse section D-XI, 8/25/42. Bullet wound. | Retention catheter. (Suture diaphragm, splenectomy, perirenal drainage 8/25/42.) | Continued. | 9/17/42 gross pyuria, decubitus severe. | Tidal drainage. Cystometry 9/25/42 showed slight response to stretch; normal reaction 12/21/42. | Automatic voiding 12/22/42. 4 months, decubitus healed. |
| 8 | Complete transverse myelitis L-I, 10/12/42. Truck accident. | Retention catheter drainage; acute urethritis. | Pyelonephritis. R/x sulfathiazole. | 11/23/42 gross pyuria. | Tidal drainage. Cystometry using water manometer showed myo-neural reflexes. No voiding. Urethrostomy 2/15/43. Litholapaxy 3/28/43. | No voiding. Transferred to Veterans Facility. Prognosis poor. |

TABLE 1—*Concluded*

| | TYPE OF INJURY | INITIAL TREATMENT | SUBSEQUENT TREATMENT | ADMISSION W.R.G.H. | TREATMENT | RESULT |
|---|---|---|---|---|---|---|
| 9 | Complete transverse myelitis L-I, 2/4/44. Compression fracture L-I. | Catheterization daily. | Cystostomy 2/20/44. | 5/18/44 gross pyuria, decubitus. Heals. Cystostomy satisfactory. | Continued cystostomy drainage. | Cystometry shows no sensation, no reflex action. Prognosis poor. |
| 10 | Complete transverse section L-I. 10/20/42. Crushing injury. | Retention catheter drainage. | Continued. | 2/14/43 gross pyuria. | Tidal drainage. Urethrostomy 4/13/43. Cystometry 2/23/43 showed slight response to stretch. No voiding. | Cystometry 6/2/43 showed no sense of fullness, no voiding reflex. Transferred to Veterans Facility. |
| 11 | Incomplete penetration. Bullet D-I 11/10/43. | Retention catheter. | Continued. (11/10/43 Laminectomy, decompression.) | 11/28/43 tidal drainage. Cystometry 11/29 showed no reflex activity. | Tidal drainage continued. Return of sensation and reflex activity 12/24/43. | Automatic voiding 12/25/43, Q-III, IV h, voluntary use of right leg. |
| 12 | Incomplete penetration, hemisection. Bullet D-V 3/28/43. | Retention catheter for 8 days. | Involuntary voiding 9th day. Could move right leg (Laminectomy 4/22/43). | 6/5/43 involuntary voiding. | Urinary antiseptics and bladder irrigation. | Efficient automatic voiding R.U., 0. |
| 13 | Compression of cord at D-VII 3/5/43. Compression fracture D-VII, VIII, by falling limb. | Retention catheter. | Drainage cont., post opr. return of skin sensation and plantar reflexes. (Laminectomy, decompression 3/14/43.) | 4/16/43 pyelonephritis. Chills and fever. | Tidal drainage, chills and fever. R/x sulfathiazole. Cystometry showed no reflex activity; urethrostomy 8/10/43. | Cystometry shows no return of reflex activity; tabetic type of bladder lesion. Prognosis poor. |

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| 14 | Compression of L-I by displacement of L-I on L-II. Land mine 12/8/42. | Catheterized 4 days. | Hyperextension cast applied 12/31/42. Voiding followed decompression. Sensation in legs and perineum return. | 1/27/43 urgency and frequency without control. | Bladder irrigations | Normal voiding with urgency and frequency. No control of external sphincter. Patient walking 5/5/43. |
| 15 | Compression of L-I. Compression fracture L-I 3/21/43. | Cystostomy. | Cystostomy closed. 4/4/43 urethral catheter placed. Overflow incontinence following operation. (Laminectomy, decompression 4/4/43) | 5/15/43 involuntary voiding R.U.-25 cc. Acute epididymoorchitis. Orchidectomy. | Cystometry showed normal reflex action. Urgency noted. | Normal voiding; urgency and frequency. No control of external sphincter. Patient walking 6/15/43. |
| 16 | Compression of L-I. Compression fracture of L-I 11/7/43. | No catheterization. | | 11/7/43 bladder not distended. Paralysis below L-I level. | Retention catheter 11/9/43.<br>Laminectomy and decompression 11/13/43. | Cystometry 12/4/43 showed good stretch response. Normal voiding 12/24/43. |

6. The use of a well placed suprapubic tube when definitely indicated for long or permanent bladder drainage. This is indicated in complete transverse lesions below L-I. Six weeks is about the limit of tolerance of the urethra to catheter drainage.

We have no quarrel with those who would adequately drain neurological bladders with large amounts of residual urine. Certainly this is not an emergency procedure and should not be done at the front nor in evacuation hospitals unless traumatic injury complicates the picture. We would not do cystostomy until neurological examination and/or surgery shows that function will not be restored for a long period of time. A well placed suprapubic tube will do no harm.

The question of when to discontinue drainage is not so easily answered. One hesitates to remove the tube and allow the patient to suffer in a wet bed for days until the wound heals. We have found cystometry of great value in determining the return of function or reflex activity. We do not remove the drain until adequate reflex responses to pressures of 12 mm Hg indicate return of function.

Permanent drainage may be indicated for patients with complete transverse lesions at or below the level of L-I. Some patients with automaticity with large amounts of infected residual urine may require drainage.

We do not believe that operations to remove tissue from the vesical orifice are indicated unless actual obstruction exists. Certainly transurethral resection is not indicated in the tabetic type of bladder with relaxation of the orifice unless fibrous contraction or prostatism is present. When obstruction is present every possible reason for resection exists (Emmett).

The indications for presacral neurectomy are not clear-cut. When the detrusor tone is poor, residual urine is present and the ejaculatory function is of no concern to the patient, we would consider the procedure as experimental. Increased tone of the detrusor, produced by increased blood supply, results from the operation of sympathectomy. Some excellent results have been obtained but not for the reasons brought out by Learmonth and others. Transurethral resection, not presacral neurectomy, is indicated for relief of obstruction or contracture of the vesical orifice.

In our opinion patients would be more comfortable with an adequate cystostomy tube than with total incontinence as after resection to eliminate urethral resistance and sphincteric action.

The care of patients after severe cerebrospinal injury requires close co-operation between the neurosurgeon and the urologist. From our experience the prognosis is most uncertain for several months after injury. Restoration of function may follow neurosurgical or postural correction of compression factors. Surgical exploration of the cord is indicated when function fails to return within 48 hours after injury and blockage of the spinal canal is demonstrated by lumbar puncture and the Queckenstadt test. When improvement is noted, surgery may be delayed. Surgery is indicated for relief of compression on the cord. Surgical exploration is the only means of differentiating complete transverse section of

the cord from compression myelitis. The operation done by a qualified neurosurgeon is safe, can do no harm and certainly offers the patient hope of return of function unless division of the fibers is proven.

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