NEUROGENIC BLADDER

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The literature since World War II is replete with articles about the various phases of the neurogenic bladder. Bors (1957) covers the entire field comprehensively and his paper can always be used as a basic standard source of information. The purpose of this paper is to relate some of my clinical observations made over the years at both the Veterans Administration Spinal Cord Centre at Long Beach, California, and my Charity Services.

Classification. Unfortunately, a great drawback has been that urologists do not always speak the same language concerning the neurogenic bladder. There are urologists who would classify their patients on cystoscopic or cystometric observations; Talbot (1963) states that '. . . classifications tend to become Procrustean; we fit cases to them instead of them to cases and run the risk of discovering one day we are treating categories rather than patients'. I cannot agree with this concept, and I take particular issue with the last quotation. If, for no reason than for teaching purposes, let alone treatment, medicine has always attempted to classify pathological entities, be it anaemia, tumour, disease, etc. Obviously, not every pathological condition will fall readily into a particular classification, but the astute physician will ascertain this, list this patient under 'others' or 'miscellaneous' and will treat such a condition individually dependent on his experience. I whole-heartedly endorse the classification as described by Bors (1957). It is interesting to note that many of the steadfast advocates of other classifications are more and more beginning to use Bors' terminology. The dysfunction of these bladders reflect the end result of neurological involvement. What is more logical than to use a neurologically accepted terminology?

Bors' Classification is as follows:

1. Sensory-Motor Neurone Lesions
   - (*a*) Upper Motor Neurone Lesions
   - (*b*) Lower Motor Neurone Lesions
   - (*c*) Mixed Lesions
2. Primary Sensory Neurone Lesions
3. Primary Motor Neurone Lesions.

To these basic diagnoses Bors adds the terms complete or incomplete which designate the completeness or incompleteness of a lesion in the central or peripheral nervous system. Furthermore, he introduced the term 'balance' of bladder function as part of the diagnosis. The latter is expressed as a percentage derived at by placing the amount of the residual urine over the amount of the capacity.

Comarr (1959) has stated that 'if residual urines exceed somewhat these percentages, yet the patient remains clinically well without a catheter, *i.e.* without

fevers and with good excretory urograms, he may be permitted to go on without a catheter if closely observed. On the other hand, if with the same residual urine the patient is not well or shows pyelographic deterioration of the kidneys, he is not ready to part with his catheter. A few patients may have excellent kidneys but become febrile if left without a catheter, in spite of a zero residual urine and no demonstrable vesico-ureteral reflux with cystograms. We permit this small group to retain their catheters.'

History and Diagnosis. The reader is referred to Bors' (1960) publication for methodology of a complete history and physical examination in neurological-urology.

The urologist must develop a sense of 'awareness' of the neurogenic bladder. Of utmost importance is a good history. Thorough questioning of the patient about the voiding habits from the period of childhood will often allow to make tentative diagnoses, confirmed by the examinations that follow. Many patients may relate abnormal voiding patterns which were overlooked by the parents in early life and which were accepted as normal as the child grew into adulthood; only when these voiding habits changed drastically or when retention resulted was a neurological diagnosis suspected. One should always re-examine a patient whose consultation request states 'essentially normal neurological findings'. Pathological signs may have been missed or they may have developed in the interval between examinations. It is not uncommon to see a relatively young man subjected to a transurethral resection with poor results. Upon questioning it becomes evident that a neurological examination was not performed; had it been done, unequivocal Babinski signs and/or hyper-reflexia would have been found. I have seen patients labelled as poliomyelitis for many years whose bladder dysfunction was consistent with upper rather than lower motor neurone findings. The diagnosis of a neurogenic bladder should be unequivocal in so far as the examiner is concerned. It is of great importance to have a patient return for repeated examinations if one cannot find neurological deficits. It is always advisable to work with a competent neurologist.

The examiner should not be misled by the patient with a psychogenic bladder described by Smith (1963). Psychogenic bladder dysfunction in females may be caused by such facts that the husband is unfaithful or that he spends his pay cheque at the local bars. These patients do not show neurological deficits upon repeated examinations. Once the physician establishes rapport he will learn more about this patient. One should ask for psychiatric consultation on such patients if indicated.

The following brief report describes the less frequent case of psychogenic bladder dysfunction in a male. Mr. J. W. B., a 31-year old white male, was admitted to our hospital with the following history: In 1951 he sustained a fracture of the 1st lumbar vertabra. He stated that he was temporarily paralysed but that he had no difficulty in voiding. A laminectomy was performed in October of 1962 because the patient complained of back pain since his injury in 1951. He was admitted to the orthopaedic service three months after the laminectomy, complaining of backache and the inability to void. A catheter was introduced. Repeated attempts at voiding were unsuccessful. The neurological examination performed on 29 January 1963 revealed only the following deficits: loss of pin-prick sensation from L2 to L5 and an absent tendon of achilles reflex on the left. Sensation of the skin of the penis, scrotal skin and saddle area was intact. Rectal examination

revealed a positive bulbocavernosus reflex; the patient was able to contract voluntarily the external sphincter around the examining digit. Introduction of the catheter for cystometry was painful; cystometry results were within normal limits. I felt that on the basis of these findings this patient should be able to void normally. Without suggesting my suspicions to the patient, I asked the technician to prepare the patient for cystoscopy. As anticipated, the patient made a plea to forgo cystoscopy. I arbitrated with the patient by stating that we would give him another chance to void on the ward by the following morning; if unsuccessful, a cystoscopy would follow. The following morning the nurse called, stating that the patient had voided three times from 350 mm. to 400 mm. with a zero residual urine. Now, 10 months later, the patient still voids normally without difficulty.

As urologists, it is not incumbent upon us to make a specific neurological diagnosis, such as multiple sclerosis, poliomyelitis, paralysis agitans, etc. However, we should be able to perform the simple neurological examination taught us as medical students and we certainly should be able to distinguish between an upper and lower motor neurone lesion.

Once this general neurological examination has been done, we perform those special neurological tests which determine the presence or absence of reflex activity of the sacral cord segments over the pelvic nerves and internal pudendal nerves; pelvic nerves—Bors' (1957) ice water test; internal pudendal nerves—Bors' (1959) bulbocavernosus test, external rectal sphincter test, and the anal reflex test. Correct categorisation into the Bors' (1957) classification is directly dependent on these tests. To aid in the diagnosis, Bors (1962) has formulated a chart showing the subjective changes of micturition, objective sensory mucosal changes of the bladder and urethra, and objective motor changes of the detrusor, smooth sphincter and striated sphincter as found in lesions of the peripheral and central nervous system.

Management. Management of the neurogenic bladder is based on the state of reflex activity in the sacral segments via the pelvic and the internal pudendal nerves. Once this has been ascertained by the tests previously mentioned, we can begin a rational method of therapy.

The experience of Bors (1957) and Comarr (1959, 1960) at our Centre over an 18-year period has shown (*a*) that 'time' is an all-important factor and (*b*) that every form of conservative therapy must be attempted before resorting to surgery. Patience both on the part of the physician and the patient is of utmost importance. The physician must take time to explain to the patient in simple terms the damage to the 'nerve tracts'.

Irrespective of the type of bladder, the insertion of a catheter depends on the bladder balance. In the United States with an acute traumatic cord lesion an intra-urethral catheter is usually inserted. However, Guttmann (1953, 1963) of England, with a vast experience of fresh injuries, has shown that he is able to keep the urine sterile either throughout or at least for many weeks by intermittent catheterisation, carried out by a physician with the non-touch technique; moreover, his technique does not produce peno-scrotal pathology such as fistulae. My enthusiasm for the use of intermittent catheterisation among fresh spinal cord injury patients has been amplified by Rossier (1962, 1964) of Switzerland and Wilmot (1964) of Ireland. I am now in the process of making arrangements to carry out intermittent catheterisation among fresh spinal cord injury patients on my Charity Services.

The patient with a non-traumatic lesion may require only supervision by a residual urine test. The first post-voiding cystogram following excretory urography may be adequate in cases of non-traumatic neurogenic bladders to check residual urine, depending on the symptomatology. Patients with precipitate micturition and negligible residual urines may not need a catheter. Such a patient may benefit from minimal dosages of banthine, but he must be closely observed to prevent bladder imbalance. He may even have to wear an external penile appliance. Urecholine may be helpful for patients with incomplete lesions and hypoactive bladders. Experience with urecholine among patients with complete lesions has failed to be of value, even with maximum oral dosage. Banthine, on the other hand, is undoubtedly the best drug for the treatment of hyperactive bladders in patients with complete or incomplete lesions. The various anaesthetic blocking procedures of Bors (1957, 1962) may be helpful to decrease the residual urine in patients with upper motor neurone bladders. Good bladder habits consisting of regular fluid intake and a regularly spaced voiding schedule will help markedly. The patient with tabes may aid his bladder balance by the use of strain and/or Credé on a regular schedule.

The major problems and challenges of the neurogenic bladder are found among those patients who require a catheter from onset of the neurological pathology. We have followed a conservative regimen as far as possible. Patients with reflex activity of the sacral segments are placed on bladder training in an attempt to 'condition the voiding reflex', realising that bladder training is not a panacea. It has no other purpose among patients with lower motor neurone bladders but a psychological; it serves as a method of a regular fluid intake. Suffice it to say that we do not use tidal drainage which was advocated by Munro (1947). The anaesthetic blocks of Bors are used concomitantly with bladder training among the patients with reflex activity of the conus medullaris.

We do not consider surgery of any kind before one year or more has passed among patients with spinal cord injury. Often the patient with a non-traumatic neurogenic bladder may be in the age group of prostatism. Comarr's (1962) experience with patients with traumatic lesions has shown that the surgeon need not turn to surgery too quickly in this older age-group with prostatism. This is proved by the fact that the prostate may atrophy subsequent to the neurological defect. Mr. A. T., aged 68, was injured on 2 February 1961 in an automobile accident wherein he sustained a fracture of the fourth thoracic vertebra. He was transferred to our Centre on 7 February 1961. Neurological examination at that time revealed his lesion was segmentally complete below T4. He had a bladder dysfunction consistent with an upper motor neurone lesion.

Routine cystoscopy performed during the first month following injury showed 'kissing of the prostatic lobes'. The usual conservative bladder rehabilitation procedures did not achieve a catheter-free state; therefore, on 9 February 1962 another cystoscopy was performed prior to contemplating a transurethral resection. Mr. A. T. requested that we postpone the operation a few months. Resectable tissue was noted, but, interestingly, the lateral lobes of the prostate had shrunken. The patient's request was granted since one year is normally only our minimal period of waiting before performing a transurethral resection. Conservative measures were continued and on 28 April 1962 the patient spontaneously developed a balanced bladder function. Had a transurethral resection been performed, the catheter-free state would have been attributed to the operation. One and one-half years later, at age 71, the patient still has a well-functioning reflex bladder.

We have applied the principles of treatment used for traumatic cord lesions to cases of demyelinating diseases of the cord, as well as to lesions of the cord caused by viral and bacterial infections.

Where indicated Bors (1957) and Comarr (1959) consider transurethral resections of the vesical neck, pudendal neurotomies, pelvic neurotomies, sacral neurotomies, sacral rhizotomies, subarachnoid (alcohol or phenol) blocks, or transurethral resection of the external sphincter after one year. It is of importance that the surgeon realises that patients with incomplete lesions may have so little deficit that they are nearly normal and that destructive operations of the central or peripheral nervous system, if not carefully selected, may do more harm than good, not only creating unwarranted losses to the patient, but also medical-legal losses to the physician. Our conservative philosophy becomes reinforced when we see iatrogenic pathology.

Many urologists are prone to overlook the many statistical reports of the world literature, Bors (1957), Comarr (1959, 1960), Guttmann (1953), Munro (1947), indicating that a majority of traumatic cord bladders will become catheter-free within the first year or even later. Bricker (1956) and Cordonnier (1955) have turned to substitute bladders, Lapides (1961) has turned to cutaneous vesicostomies within a few weeks after injury. Comarr (1963) recently moderated a panel concerning a new approach directed toward electric stimulation of the bladder.

We have yet to perform our first Bricker operation or cutaneous vesicostomy. We feel that based on our statistics there is certainly no justification to perform these operations within the first year of injury.

To date, if we are unable to create a balanced bladder within one year, we do not have a catheter-phobia (realising all the drawbacks of a catheter) and keep these patients on catheter drainage.

Once the first year has passed and, if one is still unable to create a catheter-free state, a substitute bladder or vesicostomy may, perhaps, be contemplated. However, only time will prove the justification of this attitude. We have seen renal calculosis as well as reflux among patients with ileal conduits. What, then, is accomplished with this surgery which makes it superior to catheter drainage?

Lapides (1961) has proposed that vesicostomies be performed within two weeks after injury if a neurogenic bladder has not reached a state of balance. Again, the statistical results of return of bladder function within one year are overlooked. The attitude is that, should function return, the 'vesicostomies can be undone'. Obviously, these advocates are not conscious of the fact that patients with cervical lesions, for example, with Schneider's (1954) central cord syndrome can even void normally after many months. This is illustrated by a typical history described by Comarr (1964) of Mr. S. W. B., who was injured in a car accident on 15 April 1962. The patient was 48 years of age at time of injury. The vertebral level was cervical four. Initially, he was paralysed in all four extremities, sphincter control of his bowel and bladder was lost, and he required an intra-urethral catheter. Six months after injury, Mr. B. was able to walk without the use of prosthetic devices. His bowel movements became normal, he could distinguish between gas and faecal matter, yet he was unable to empty his urinary bladder. He became depressed because of the difficulty with his bladder. However, eight months after injury, while still under conservative management, the patient was able to void; he had regained and since then retained normal bladder function. Should this patient have had a vesicostomy two weeks after injury because he could not void at that

time? Should he have had a vesicostomy only to have it 'undone' eight months later?

Recently I addressed a post-graduate group of occupational therapists. One of the participants told me that the urologist at her hospital had persuaded a paraplegic early to have a cutaneous vesicostomy because 'it would lengthen his life'. How, with a clear conscience, can any physician make such a statement to a patient when it will take a generation of patients with this particular operation to prove the point?

Electrical stimulation of the bladder is still in its infancy. Good results in the dog do not assure similar results in the human. However, it is too early to be able to assess what its value will be. Chou (1963) has stressed one important point, namely, that in chronic infections of the bladder, results are poor. Should those interested be able to perfect their apparatus for human use, are we then going to have to perform this procedure immediately after injury, before the introduction of a catheter and before laminectomy? We know that bacteria from the urethral catheter will be multiplying in the bladder within 48 to 72 hours.

Obviously, one cannot be dogmatic and state that the newer techniques will not have a place in the armamentarium of neurological urology. But, we can be dogmatic in requesting indications for the use of each procedure. There is nothing in medicine that does not require indications; there are hardly any panaceas that can be applied routinely to every patient without discrimination.

Mortality studies are a great aid in determining whether one treatment is better than another. The mortality rate at Guttmann's Spinal Cord Injury Centre in England according to Tribe (1963) covering 2000 patients was 13·2 per cent.; at our Centre, Nyquist (1960) showed that it is 14 per cent., of which 60 per cent. were uraemic deaths. Obviously, when the various investigators can present statistics with comparable numbers of patients and can show that their mortality rates are far better than ours, we will be the first to change our approach to the problem. But one must remember again that, only after the time span of a generation, will those advocates of substitute bladders, vesicostomies and bladder stimulation know what the final answer will be. At this moment we choose to follow the conservative approach.

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