Cystometry as an adjunct in the evaluation of lumbar disc syndromes

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One hundred patients with the provisional diagnosis of nerve root compression in the lumbar spine were subjected to routine bladder evaluation by cystometry in addition to the standard somatic neurological examination. Eighty-three were found to have cystometrograms characteristic of bladder hypofunction. Residual urine was found in 20% of the 100 patients. By contrast, the examination of the somatic nervous system disclosed sensory deficits in 36%, weakness in 59%, reflex alterations in 66%, and positive sciatic-stretch tests in 69%. Almost all cystometric studies (96%) became normal after successful surgical or conservative therapy. It is concluded that occult bladder dysfunction is a major manifestation of lumbar nerve root compression. The neurophysiological mechanisms involved are discussed in detail.

In the evaluation of patients with symptoms of nerve root compression in the lumbar spine, the standard neurological examination consists of sensory, motor, and reflex testing of the somatic lumbosacral representation in the legs and perineum. The lumbar segments have a relatively large distribution peripherally compared to that of the sacral segments, the latter being important as the spinal source of parasympathetic innervation to the abdominal and pelvic viscera. Neither are the lumbar and thoracic segments entirely somatic in function, for they provide the sympathetic innervation of the same viscera. Theoretically, therefore, a complete evaluation of neural activity should include an examination of both somatic and visceral components.

In 1963, Rosomoff and associates reported a study in which the standard evaluation of 50 consecutive patients with the provisional diagnosis of lumbar root compression was supplemented by cystometry to test autonomic innervation of a readily accessible viscus, the bladder. This investigation indicated that, despite a relative paucity of symptoms, bladder hypofunction was one of the most consistent and characteristic signs of this syndrome. The observation was confirmed by Kontturi, et al., but neither group had obtained data to determine the effect of treatment on the ultimate fate of the bladder. The present report answers this question based on the study of 100 patients who, following evaluation by both neurological examination and cystometry, went on to myelography, surgery, and verification of the pathological lesion, and who were then ex-
Ained repeatedly over a period not less than 2 years or until they had become lost to observation.

Methods

A patient qualified for study if the routine history and physical examination resulted in a provisional diagnosis of lumbar nerve root compression. After lumbosacral roentgenographic studies, a cystometrogram was done in the following manner. The patient was asked to void, a catheter was inserted into the bladder, and the residual urine was measured. The catheter was connected to a recording cystometer adjusted to the level of the symphysis pubis with the patient in the supine position. Sterile water, at room temperature in most, but iced in some, was continuously dripped into the bladder at rates up to 50 ml/min. The patient was asked to report the first urge to void, the feeling of fullness, the maximum desire to urinate, and the sensations of cramping or pain. If discomfort did not occur before a total inflow of 500 ml, the test was continued until it became obvious that normal bladder capacity had been exceeded or that bladder sensation was impaired. The patient was asked to void volitionally while still in the supine position. The resulting pressure was measured, and whether or not the catheter was expelled was noted. Most of the patients who had abnormal bladder activity were not able to void or expel the catheter when in the supine position, including those who had ice water cystometrograms. The patient, therefore, was allowed to sit or stand so that voiding could be accomplished. The length of time necessary to initiate micturition was observed, and the presence or absence of accessory abdominal straining was recorded in the description of the urinary stream. The study was completed by testing the anal and bulbocavernosus reflexes.

It is to be emphasized that a patient became a candidate for myelography and surgical treatment only when pain or neurological deficits persisted after a trial of bed rest, conservative management with muscle relaxants, and physiotherapy. The indications for operative intervention were the standard time-honored criteria of intractable pain and/or unremitting mechanical or neurological abnormalities.

Rosomoff, et al.

Results

Sex and Age

Among the total of 100 patients, there were 96 males and four females. The predominance of the male was disproportionate since the majority of the patients came from the neurosurgical service of a Veterans Administration Hospital. Eighty-three of the patients were between the ages of 30 and 50 years, four were less than 30 years old, and 13 were more than 50.

Symptoms and Signs

Eighty-eight of the 100 patients complained of low-back pain with a radiating component extending into one or both buttocks or legs. Four reported leg pain only, and eight described only back pain. Eighty-one had no sensory or motor symptoms. Ten complained of numbness or paresthesias in the feet or legs without motor symptoms. Two described weakness only, and seven had combinations of sensory and motor symptoms.

Eighty-seven of the patients denied any bladder symptoms. Twelve described dysfunction, consisting of frequency of urination in six, incomplete emptying of the bladder in three, and difficulty with starting the urinary stream in three (Table 1). One patient reported impotence.

Eighty-nine of the 100 patients manifested abnormal motor, sensory, or reflex signs alone or in combination. Sixty-six of the 100 were found to have reflex alterations with or without motor or sensory deficits. Fifty-nine exhibited motor weakness alone or in combination with sensory or reflex changes. Thirty-six had sensory deficits with or without motor or reflex abnormality. There were 11 patients in whom no sensory, motor, or reflex abnormality could be demonstrated.

<p>| TABLE 1 |
| Urogenital symptoms in 100 patients |</p>
<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>urinary frequency</td>
<td>6</td>
</tr>
<tr>
<td>incomplete emptying</td>
<td>3</td>
</tr>
<tr>
<td>difficulty starting</td>
<td>3</td>
</tr>
<tr>
<td>impotence</td>
<td>1</td>
</tr>
<tr>
<td>none</td>
<td>87</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

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Sciatic stretch tests in the form of straight-leg raising or the Lasègue maneuver were positive in 69 of the 100 patients. Therefore, there was no single sign present in more than 69% of the cases by conventional methods of testing (Table 2).

Cystometric Findings

On the other hand, the cystometrogram was abnormal in 83 of the 100 patients (Table 3). Twenty patients had significant amounts of residual urine, ranging between 15 and 400 ml, although only 12 patients reported bladder symptoms. The abnormality in the cystometrogram was interpreted as that seen with a hypotonic bladder and included the following characteristics. The tonus limb of the cystometrogram was shifted to the right resulting in bladder capacities of 500 to 1200 ml. With voluntary attempts to void, vesical pressure could not be raised sufficiently high to elicit the micturition reflex, nor could the reflex be evoked by further distortion of the bladder or by the instillation of cold water. Sensation was impaired, the perception of fullness with the desire to void being delayed and painful urgency being felt only at the extreme of filling. Control was faulty, requiring the summation of voluntarily applied extravesical pressure to residual intrinsic contractibility to produce emptying.

Roentgenographic Studies

The lumbosacral roentgenograms of 28 patients were interpreted as being normal. Narrowed spaces were demonstrated in another 51, and congenital bone defects were found in 14 patients. Four fractures were

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**TABLE 3**

Cystometric studies before treatment in 100 patients

<table>
<thead>
<tr>
<th>Residual Urine</th>
<th>Cystometrogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount (ml)</td>
<td>No. of Cases</td>
</tr>
<tr>
<td>0</td>
<td>80</td>
</tr>
<tr>
<td>15-29</td>
<td>4</td>
</tr>
<tr>
<td>30-59</td>
<td>4</td>
</tr>
<tr>
<td>60-90</td>
<td>4</td>
</tr>
<tr>
<td>90+</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

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seen, and the remaining three patients had unrelated miscellaneous findings.

Myelography was performed in all 100 patients. Nine myelograms were interpreted as being normal. There were 47 patients with defects at the level of the fourth lumbar interspace, 36 with defects at the fifth lumbar interspace, and seven had abnormalities at both of these spaces.

Cerebrospinal Fluid

Routine cytological and chemical determinations were made on the cerebrospinal fluid removed at the time of myelography. The only significant disturbance was in the protein content, which was found to be abnormally high in 46 of 100 fluids studied. The cerebrospinal fluid protein level was 50 to 70 mg% in 25 patients and greater than 70 mg% in 21.

Surgical Pathology

Each of the 100 patients was operated upon on the basis of either clinical localization or myelographic defect. The findings were: 48 extrusions (free fragments) of the nucleus pulposus, 29 herniations of the nucleus pulposus, 14 degenerated intervertebral discs, five adhesions, two with spondylosis, one spondylolysis, and one tumor (Table 4).

**TABLE 2**

Neurological signs in 100 patients

<table>
<thead>
<tr>
<th>Sign</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>straight-leg raising</td>
<td>69</td>
</tr>
<tr>
<td>motor-sensory reflex</td>
<td>15</td>
</tr>
<tr>
<td>motor-sensory</td>
<td>6</td>
</tr>
<tr>
<td>motor-reflex</td>
<td>25</td>
</tr>
<tr>
<td>motor</td>
<td>13</td>
</tr>
<tr>
<td>sensory-reflex</td>
<td>11</td>
</tr>
<tr>
<td>sensory</td>
<td>4</td>
</tr>
<tr>
<td>reflex</td>
<td>15</td>
</tr>
<tr>
<td>none</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

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Postoperative Bladder Function and Cystometry

Sixty-eight of the 83 patients with hypotonic bladders were followed with serial neurological and cystometric examinations for 2 years or until reversion of the bladder to normal (Table 5). One month following sur-
surgery, 15% of the patients with hypotonic bladders had reverted to normal. At the end of 6 months, 52% were normal; at 12 months, 68%; at 18 months, 81%; and, at the end of 2 years, 96%. Patients with abnormal function after 2 years were considered to have irreversible changes.

It is of further interest that patients with the same cystometric findings who were treated conservatively without surgery showed a comparable return of a normal cystometric pattern once their neurological symptoms had remitted.

**Analysis of Patients Without Neurologic Deficits**

Eleven of the 100 patients were hospitalized with unremitting back and leg pain of 2 months to 4 years' duration (Table 6). All showed abnormal mechanical signs such as lumbar spasm, pelvic tilt, limitation of movement, or a positive sciatic-stretch test. Yet none had motor, sensory, or reflex changes. Cystometry demonstrated dysfunction in nine of these 11 patients, with abnormal residual urines in three. Only one had reported difficulty with micturition. When conservative management in the hospital failed to improve their status, myelography was carried out and showed defects in all 11. Surgery produced an asymptomatic state in 10 of the 11, and the bladder reverted to normal in 8 of the 9 abnormal cases during a period of 6 weeks to 21 months.

**Table 4**

<table>
<thead>
<tr>
<th>Operative Findings</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>extruded nucleus pulposus</td>
<td>48</td>
</tr>
<tr>
<td>herniated nucleus pulposus</td>
<td>29</td>
</tr>
<tr>
<td>degenerated nucleus pulposus</td>
<td>14</td>
</tr>
<tr>
<td>adhesions</td>
<td>5</td>
</tr>
<tr>
<td>spondylosis</td>
<td>2</td>
</tr>
<tr>
<td>spondylolysis</td>
<td>1</td>
</tr>
<tr>
<td>tumor</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

**Discussion**

Cystometry provides information about both the muscular and neural components of bladder function. As the cystometrogram is carried out, the physical state of the bladder musculature is assessed first, as determined from the level of pressure measured in the flat part or tonus limb of the tracing (Fig. 1). Normally, this pressure is 10 to 20 mm of mercury with a bladder capacity of 350 to 450 ml. The expulsive force of the bladder is estimated by having the patient attempt to void voluntarily when the bladder has been filled sufficiently to produce a desire to empty but not enough to activate the micturition reflex. Detrusor activity alone produces a pressure elevation of approximately 40 mm of mercury, and when this is accompanied by contraction of the thoracic diaphragm and abdominal musculature, the pressure rises to 60 to 80 mm of mercury. With continued filling, a terminal sharp rise of pressure is seen accompanied by voiding. This is the micturition reflex; it is neurogenic in origin and is a stretch reflex.

Neural sensory function is tested by asking the patient to report the first desire to void. When room temperature water is used, this occurs usually after 150 ml of water have been instilled. Maximal desire occurs at the limits of capacity, 350 to 450 ml, and in the normal patient painful urgency develops if this point is exceeded. However, if there is a question of lack of cooperation from the patient or demonstrable bladder hypotonia with an abnormal micturition reflex, the cystometrogram may be repeated using ice water in the manner described by Bors. Under this circumstance, a powerful micturition

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**Table 5**

<table>
<thead>
<tr>
<th>Mos. Post Treatment</th>
<th>Normal Bladder</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>%</td>
</tr>
<tr>
<td>1</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>41</td>
</tr>
<tr>
<td>6</td>
<td>35</td>
<td>52</td>
</tr>
<tr>
<td>9</td>
<td>40</td>
<td>59</td>
</tr>
<tr>
<td>12</td>
<td>46</td>
<td>68</td>
</tr>
<tr>
<td>15</td>
<td>51</td>
<td>75</td>
</tr>
<tr>
<td>18</td>
<td>57</td>
<td>81</td>
</tr>
<tr>
<td>21</td>
<td>63</td>
<td>93</td>
</tr>
<tr>
<td>24</td>
<td>65</td>
<td>96</td>
</tr>
</tbody>
</table>

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TABLE 6
Analysis of 11 patients without neurological deficits

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Symptoms*</th>
<th>X-rays</th>
<th>CSF Protein (mg %)</th>
<th>Myelogram</th>
<th>Surgery†</th>
<th>Preop. Cystometrogram</th>
<th>Postop. Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>31</td>
<td>M</td>
<td>pain, back, 9 mos</td>
<td>normal</td>
<td>37</td>
<td>defect L4-5</td>
<td>HNP</td>
<td>hypotonic, residual: 0</td>
<td>cysto. norm. 6 mos, asymptomatic</td>
</tr>
<tr>
<td>40</td>
<td>M</td>
<td>pain, back, 5 yr; LLE, 2 mos</td>
<td>lumbarized L5-S1</td>
<td>---</td>
<td>defect L5-S1</td>
<td>HNP</td>
<td>hypotonic, residual: 50 ml</td>
<td>cysto. norm. 21 mos, asymptomatic</td>
</tr>
<tr>
<td>34</td>
<td>M</td>
<td>pain, back, LLE, 2 mos; paresthesia 1st big toe</td>
<td>normal</td>
<td>80</td>
<td>defect L5-S1</td>
<td>HNP</td>
<td>hypotonic, residual: 15</td>
<td>cysto. norm. 6 mos, asymptomatic</td>
</tr>
<tr>
<td>53</td>
<td>M</td>
<td>pain, back, LLE, 8 mos</td>
<td>normal</td>
<td>56</td>
<td>defect L5-S1</td>
<td>HNP</td>
<td>hypotonic, capacity 1000 ml, residual: 0</td>
<td>cysto. abnormal 2 yr, asymptomatic</td>
</tr>
<tr>
<td>56</td>
<td>F</td>
<td>pain, back, rt hip, 2 mos</td>
<td>narrow L4-5</td>
<td>---</td>
<td>defect L4-5</td>
<td>HNP L4-5, deg. L5-S1</td>
<td>hypotonic, residual: 5</td>
<td>cysto. norm. 18 mos, asymptomatic</td>
</tr>
<tr>
<td>44</td>
<td>M</td>
<td>pain, back, 1 yr; RLE, 7 wks</td>
<td>normal</td>
<td>52</td>
<td>defect L4-5</td>
<td>HNP</td>
<td>hypotonic, residual: 0</td>
<td>cysto. norm. 3 mos, asymptomatic</td>
</tr>
<tr>
<td>36</td>
<td>M</td>
<td>pain, back 4 yr; LLE, 2 mos</td>
<td>narrow L4-5, L5-S1</td>
<td>29</td>
<td>defect L4-5 adhesions L4-5, L5-S1</td>
<td>hypotonic, residual: 0</td>
<td>cysto. norm. 6 mos, sl. back pain, rheumatoid spondylitis</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>M</td>
<td>pain, back, RLE, 3 mos</td>
<td>narrow L4-5, L5-S1</td>
<td>44</td>
<td>defect L4-5</td>
<td>HNP L4-5, L5-S1</td>
<td>normal</td>
<td>normal, asymptomatic</td>
</tr>
<tr>
<td>52</td>
<td>M</td>
<td>pain, back, LLE, 3 yr</td>
<td>normal</td>
<td>40</td>
<td>defect L5-S1 spondylosis L5-S1</td>
<td>normal</td>
<td>normal, asymptomatic</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>M</td>
<td>pain, back, LLE, 1 yr</td>
<td>narrow L5-S1</td>
<td>40</td>
<td>defect L5-S1</td>
<td>HNP</td>
<td>hypotonic, residual: 20 ml</td>
<td>cysto. norm. 6 wks, asymptomatic</td>
</tr>
<tr>
<td>38</td>
<td>M</td>
<td>pain, back, 2 yr; LLE, 1 wks; difficulty initiating micturition</td>
<td>narrow L4-5, L5-S1</td>
<td>66</td>
<td>defect L4-5</td>
<td>HNP</td>
<td>hypotonic, residual: 50 ml</td>
<td>cysto. norm. 6 mos, asymptomatic</td>
</tr>
</tbody>
</table>

* LLE = pain on left leg raising; RLE = pain on right leg raising.  
† HNP = herniated nucleus pulposus.

reflex is evoked in the normal bladder. In the presence of an intact motor supply, lack of response to this stimulus is positive proof of sensory impairment. The neurological evaluation is complete when the corresponding somatic components are examined by testing the anal and bulbocavernous reflexes which are supplied through the pudendal nerve.

The bladder muscle was considered to be abnormal when the pressure in the tonus limb of the cystometrogram was less than the normal 10 ml of mercury. According to Ruch,\(^7\) the tonus limb depends on the state of the bladder muscle, not its motor innervation; therefore, a low pressure was interpreted as being indicative of decompensated musculature due to episodic overdistention, rather than flaccidity of denervation. Rose,\(^7\) however, believes that a low pressure does indicate denervation, if the cystometrogram is repeated immediately and progressive lowering of the pressure in the tonus limb is demonstrated with each successive filling. Successive tracings, therefore, were carried out in several of the patients with low pressures, and the tonus limb was found at the same level each time, confirming that his abnormality was from a muscular deficiency and not from motor denervation. If a normal pressure was found, it was interpreted as meaning that muscle decompensation had not occurred.

Neural abnormality was inferred from the following observations. The tonus limb was elongated or shifted to the right, resulting in bladder capacities of 500 to 1200 ml (Fig. 2). Voluntary attempts to void failed to raise the vesical pressure to the heights seen in the normal bladder during emptying, and the micturition reflex was not evoked even with distention to 1000 ml. The first desire to
void was not perceived until the bladder was filled past the normal range of 150 to 250 ml. With further instillation, there was no urgency or cramping, although with extreme distention there was a dull suprapubic pain. Cold water could not be distinguished from warm water, and substitution of ice for warm water did not produce voiding, indicating that sensory innervation was impaired.

When the catheter was removed, the patients could void volitionally but usually not in the supine position. It was necessary for most to sit or stand, utilizing the weight of the abdominal viscera while applying further pressure by bearing down to reach the pressure necessary to empty the bladder. Even so, 20% failed to empty completely, and residual retentions of urine of 15 to 400 ml were found. The anal and bulbocavernosus reflexes were found to be intact, indicating preferential impairment of the visceral innervation with relative sparing of the somatic contribution from the same spinal cord segment. A bladder with the aforementioned

Fig. 1. A normal cystometrogram.

Fig. 2. An abnormal cystometrogram demonstrating a hypotonic neurogenic bladder.
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cystometric characteristics was given the
designation, “hypotonic neurogenic blad-
der.”

The mechanisms by which abnormal blad-
der function is produced in patients with
nerve root compression in the lumbar spine
is no clearer now than when described by us
in 1963.6 It is not reasonable to invoke
cauda equina involvement by an intraspinal
space-occupying lesion as the explanation.
This may have been possible in the few pa-
tients who demonstrated complete blockage
of the spinal canal, contributory in some
who had large extrusions or herniations of
the nucleus pulposus, but unlikely in others
with smaller lateral herniations or spondylo-
ysis.

It is possible that bladder sensation was
impaired as the result of “pressure anesthe-
sia.” That is, in the early phases of the com-
pressive syndrome when pain is a prominent
feature, the patient may have voluntarily in-
hibited micturition to avoid increasing his
discomfort during the bearing-down period
of voiding. This effort may have led to over-
distention and “pressure anesthesia,” which
is easily induced and slow to disappear.

It is more likely, however, that a reflex
neural mechanism is responsible, with irrita-
tion of the offended nerve roots evoking cen-
tripetal impulses that inhibit the micturition
reflex at the level of the spinal cord. The
micturition reflex is the longest sustained
phasic spinal reflex.9 This fact may be due to
a peripheral arrangement of receptors or to a
diffuse central reflex mechanism. In the lat-
ter instance, interneurons are intercalated
between afferent fibers and efferent neurons
so as to spread neural activity over a larger
field longitudinally in the spinal cord. Thus,
temporal dispersion of impulses returning to
the segmental preganglionic cells is provided.
It is conceivable, therefore, that the spinal
reflex center for micturition is not restricted
to the sacral segments but may also include
cephalad extension via the intermediate in-
terneuronal zone through the lumbar cord. In
fact, the nucleus sympathetic medialis,
whose axons appear in the ventral roots as
the sacral autonomic elements of the para-
sympathetic nervous system, actually begins
at the third lumbar level, is largest in the
fifth lumbar, and continues to the caudal end
of the cord. Therefore, circumscribed reflex
activity with direct synapsis of afferent col-
laterals with efferent neurons is possible in
the lumbar area, with the efferent axons ap-
ppearing in the sacral roots. It would appear
possible that the afferent fibers in question
travel with the thoracolumbar nerves from
the bladder neck and fundus, giving greater
functional importance to the nerves than had
heretofore been accorded.

Physiologically, the rate of discharge in
single afferent fibers of the pelvic nerve is
unusually low. During passive distention
with filling of the bladder, Iggro3 observed an
initial peak frequency of 20 to 30 per sec,
which after 1 to 2 sec fell to 1 per sec. With
active contraction, a peak of 30 to 40 per sec
was reached, but rhythmic contractions were
seen with a frequency of only 4 to 6 per min.
Therefore, the micturition reflex requires
heavy facilitation at the spinal cord level in
order to discharge, or, conversely, it is easily
inhibited centrally at the internuncial pool or
peripherally by blocking the bladder recep-
tors or afferent nerves.

Peripherally, afferent reception is clearly
impaired so that reflex activity may be defi-
cient by virtue of sensory deprivation alone.
Centrally, several spinal cord mechanisms
have been demonstrated by which inhibition
may occur. Each depends on stimulation of
afferent units which, for the purpose of this
discussion, is assumed to be mechanical tra-
ction or irritation of nerve roots by disc herni-
ation or extrusion. Jefferson and Schlapp4
reported depression of reflex activity in seg-
ments below that of an afferent nerve root
which was being stimulated repetitively. Sa-
cral inhibition was found with lower lumbar
stimulation, a similar circumstance to the
present clinical problem. Granit and Ström5
shown that, with an increase in muscle ten-
sion as a conditioning reflex, the excitability
of the spinal motoneurons fell to zero, and
they were rendered incapable of activity.
Similarly, Laporte6 found a disynaptic reflex
linkage between muscles of the hind limb. In
this linkage, conditioning potentials, after
one internuncial relay, led to inhibition of
muscles not related in either synergism or
antagonism. Thus, bladder would be in-
hibited, not being a member of a hind limb
myotatic unit. Wilson and Lloyd10 also dem-
strated a monosynaptic inhibitory path-
way in the caudal spinal cord, for which they
were unable to identify the peripheral origin of the involved fibers.

Preliminary experiments in our laboratory confirm the presence of a peripheral mechanism causing central inhibition. In these studies, dogs were catheterized, and isometry of the bladder was produced by filling the bladder with water to a sustained constant pressure. Exposure and manipulation of a lumbar nerve root produced an immediate and prolonged relaxation of the bladder, resulting in an increased capacity and marked lowering of the contained pressure.

It is interesting that only a few patients had overt bladder symptoms despite a greater than 20% incidence of residual urine. Micturition probably did not become a problem because emptying could be accomplished by raising the extravesical pressure through bearing down. In these relatively young people, without obstructive urinary outflow disease, this action was sufficient to raise vesical pressure to the necessary level for voiding. It may now be clear why some postoperative disc patients have urinary retention requiring catheterization, despite what appears to have been a relatively atraumatic surgical procedure. These patients had occult hypotonic bladders preoperatively, and postoperatively are unable to bear down because of incisional pain; thus the bladder abnormality becomes overt. In the older male with coincidental urethral obstructive uropathy, it is likely that bladder decompensation will be manifested earlier, more frequently, and to a greater degree.

Conclusions

We concluded that cystometry is an important adjunct in the evaluation of nerve root compression in the lumbar spine, as it appears to demonstrate one of the most consistent signs of this syndrome, namely, bladder hypofunction. We suggest that occult bladder dysfunction is a major manifestation of lumbar nerve-root compression, with a prevalence heretofore unsuspected and not observed.

References


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