Lower urinary tract dysfunction in children with central nervous system tumours

D Soler, M Borzyskowski

Abstract
The findings in 10 children with neuro-pathic vesicourethral dysfunction after the onset of a central nervous system tumour are presented. Eight had a spinal tumour and two a brainstem tumour. Bladder dysfunction occurred late in most children except in those with neoplastic infiltration of the conus and cauda equina. Moreover, tumour recurrence was often heralded by loss of bladder control before other neurological signs became obvious. Videourodynamic (VUD) showed various combinations of “filling” and “voiding” dysfunction in tumours extending from the pons to the cauda equina, whereas an isolated “filling” dysfunction was evident in the patient with a suprapontine tumour. Urinary incontinence and recurrent urinary infection can be immensely distressing to children and their families, particularly when they have had to cope with the stress of diagnosis and treatment of the underlying tumour. Different management strategies, based on VUD findings, are discussed highlighting the impact these have on the children's quality of life. (Arch Dis Child 1998;79:344–347)

Keywords: urinary tract dysfunction; central nervous system tumours; oncology; videourodynamic

Tumours along the neural axis can interrupt the pathways modulating the storage or voiding mechanisms of the bladder and urethra. As a result, various degrees of bladder dysfunction can occur in children with central nervous system (CNS) tumours. This may range from transient loss of control, resolving after surgical treatment of the tumour, to persistent dysfunction.13 The psychological impact and physical distress caused by bladder symptoms may be immensely stressful, even devastating, to children and families. We have reviewed children referred to us with chronic neuropathic vesicourethral dysfunction secondary to a CNS tumour. We discuss the results and management of investigations highlighting the impact of a CNS tumour on the quality of life for these children.

Methods
We identified 10 children with bladder disturbance after discovery of a CNS tumour from within a population referred for neuourological evaluation to Guy's Hospital, London between 1980 and 1996. No child had urinary symptoms before tumour diagnosis. We reviewed medical notes, radiological investiga-

Tumours along the neural axis can interrupt the pathways modulating the storage or voiding mechanisms of the bladder and urethra. As a result, various degrees of bladder dysfunction can occur in children with central nervous system (CNS) tumours. This may range from transient loss of control, resolving after surgical treatment of the tumour, to persistent dysfunction.13 The psychological impact and physical distress caused by bladder symptoms may be immensely stressful, even devastating, to children and families. We have reviewed children referred to us with chronic neuropathic vesicourethral dysfunction secondary to a CNS tumour. We discuss the results and management of investigations highlighting the impact of a CNS tumour on the quality of life for these children.

Results
There were five boys and five girls with a mean age at diagnosis of 4.8 years (9 months to 10 years). Two children presented with a brain tumour, one arising within the brainstem at the level of the pons and another within the cerebellum with secondary midbrain extension. Eight children presented with a spinal cord tumour, six of which were intramedullary and two were extradural. Three had an astrocytoma of the spinal cord, two an astrocytoma of the brain, and there was one each of spinal ependymoma, ganglioglioma, rhabdoid tumour, ganglioneuroblastoma, and neuroblastoma. All tumours were debulked, four children requiring repeated debulking procedures. Postoperatively, five children had radiotherapy, one received chemotherapy, and one had radiotherapy and chemotherapy. In all, urinary
Table 1  Urinary symptoms, site, and histology of the tumours

<table>
<thead>
<tr>
<th>Case</th>
<th>Age at diagnosis (years)</th>
<th>Urinary symptoms</th>
<th>Time from initial symptoms to bladder dysfunction (years)</th>
<th>Site of tumour (cord segments)</th>
<th>Histological diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>Dribbling stream, urge incontinence, nocturnal enuresis, recurrent UTI</td>
<td>1.2</td>
<td>Brainstem (pons)</td>
<td>Pilocytic astrocytoma (grade 1)</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>Frequency, urgency, diurnal enuresis</td>
<td>2.3</td>
<td>Cerebellar + midbrain recurrence</td>
<td>Pilocytic astrocytoma (grade 1)</td>
</tr>
<tr>
<td>3*</td>
<td>10</td>
<td>Dribbling stream, stress incontinence, recurrent UTI</td>
<td>1.2</td>
<td>Cervicoomedullary (medulla–C6)</td>
<td>Astrocytoma (grade 2)</td>
</tr>
<tr>
<td>4*</td>
<td>10</td>
<td>Nocturia, urgency, hesitancy</td>
<td>1</td>
<td>Cervical to upper thoracic (C1–T5)</td>
<td>Ependymoma (grade 2)</td>
</tr>
<tr>
<td>5</td>
<td>2.5</td>
<td>Urgency, frequency, nocturia</td>
<td>4.6</td>
<td>Cervicothoracic (C7–T3)</td>
<td>Ganglieneuroblastoma</td>
</tr>
<tr>
<td>6*</td>
<td>0.8</td>
<td>Dribbling, retention</td>
<td>0 (at presentation)</td>
<td>Cervicothoracic (diffuse)</td>
<td>Rhabdoid tumour</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>Urge incontinence, frequency, nocturnal enuresis</td>
<td>6.7</td>
<td>Lower thoracic (T9–T12)</td>
<td>Fibrillary astrocytoma (grade 2)</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>Retention, recurrent UTI</td>
<td>0.8</td>
<td>Lower thoracic to conus (T5–conus)</td>
<td>Ganglioglioma (grade 1)</td>
</tr>
<tr>
<td>9</td>
<td>1.4</td>
<td>Dribbling, retention</td>
<td>0.4</td>
<td>Conus and cauda equina</td>
<td>Neuroblastoma</td>
</tr>
<tr>
<td>10</td>
<td>2.5</td>
<td>Delay in getting dry</td>
<td>0 (at presentation)</td>
<td>Conus</td>
<td>Fibrillary astrocytoma (grade 2)</td>
</tr>
</tbody>
</table>

*Died. UTI, urinary tract infection.

Table 2  Urodynamic findings

<table>
<thead>
<tr>
<th>Site of tumour (cord segments)</th>
<th>Case</th>
<th>Capacity</th>
<th>Detrusor hyperreflexia</th>
<th>Bladder compliance</th>
<th>Bladder neck</th>
<th>Voidsing pressure</th>
<th>Distal sphincter</th>
<th>VUR</th>
<th>Voidsing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper brainstem (midbrain)</td>
<td>2</td>
<td>D</td>
<td>+</td>
<td>N</td>
<td>Open</td>
<td>I</td>
<td>N</td>
<td>Nil</td>
<td>Complete</td>
</tr>
<tr>
<td>Lower brainstem (pons)</td>
<td>1</td>
<td>I</td>
<td>+</td>
<td>N</td>
<td>Open</td>
<td>I</td>
<td>DSD</td>
<td>Nil</td>
<td>Incomplete</td>
</tr>
<tr>
<td>Cervicothoracic</td>
<td>5</td>
<td>D</td>
<td>+</td>
<td>N</td>
<td>Open</td>
<td>N</td>
<td>N</td>
<td>Nil</td>
<td>Complete</td>
</tr>
<tr>
<td>Lower thoracic</td>
<td>7</td>
<td>D</td>
<td>+</td>
<td>N</td>
<td>Closed</td>
<td>N</td>
<td>N</td>
<td>Nil</td>
<td>Complete</td>
</tr>
<tr>
<td>Lower thoracic to conus</td>
<td>8</td>
<td>I</td>
<td>+</td>
<td>N</td>
<td>Closed</td>
<td>N</td>
<td>DSD</td>
<td>Nil</td>
<td>Incomplete</td>
</tr>
<tr>
<td>Conus+cauda equina</td>
<td>9, 10</td>
<td>D, N</td>
<td>–</td>
<td>D</td>
<td>Open</td>
<td>Fixed</td>
<td>Nil</td>
<td>Incomplete</td>
<td></td>
</tr>
<tr>
<td>Conus</td>
<td>10</td>
<td>N</td>
<td>+</td>
<td>N</td>
<td>Open</td>
<td>N</td>
<td>N</td>
<td>Nil</td>
<td>Complete</td>
</tr>
<tr>
<td>Micturating cystourethrogram</td>
<td>4</td>
<td>I</td>
<td>Open</td>
<td>Fixed</td>
<td>Nil</td>
<td>Incomplete</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervicothoracic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No urodynamics in case 3 (cervicoomedullary) and case 6 (cervicothoracic) tumours. An ultrasound scan taken before and after micturition showed large incompletely emptying bladders in both cases. D6, distal sphincter; DSD, detrusor sphincter dyssynergia; VUR, vesicoureteric reflux, D, decreased; I, increased; N, normal; +, present; −, absent.
**Table 3** Treatment and duration of bladder dysfunction

<table>
<thead>
<tr>
<th>Case</th>
<th>Treatment regimen</th>
<th>Duration (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>CIC, oxybutinin, and antibiotics</td>
<td>3.5</td>
</tr>
<tr>
<td>2</td>
<td>Oxybutinin</td>
<td>1.5</td>
</tr>
<tr>
<td>3</td>
<td>CIC and antibiotics</td>
<td>0.6*</td>
</tr>
<tr>
<td>4</td>
<td>CIC</td>
<td>3*</td>
</tr>
<tr>
<td>5</td>
<td>Oxybutinin</td>
<td>0.3</td>
</tr>
<tr>
<td>6</td>
<td>CIC and oxybutinin</td>
<td>0.6*</td>
</tr>
<tr>
<td>7</td>
<td>Nil (regular voiding)</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>CIC and antibiotics</td>
<td>1.7</td>
</tr>
<tr>
<td>9</td>
<td>CIC, ephedrine, and antibiotics</td>
<td>16</td>
</tr>
<tr>
<td>10</td>
<td>None</td>
<td>0.25</td>
</tr>
</tbody>
</table>

*D, CIC, clean intermittent catheterisation.

These findings confirm that various combinations of storage and voiding dysfunction can occur with tumours along the neural axis from the pons to the cauda equina. An isolated storage dysfunction was seen, however, in the child with the suprapontine lesion.

**Management**

All children were treated conservatively, and table 3 shows the treatment regimen and duration of treatment. Selective treatment was based on results of VUD studies and consisted of a combination of clean intermittent catheterisation, oxybutinin, ephedrine, and prophylactic antibiotics. The six children with voiding difficulties and significant residual urine volumes were started on clean intermittent catheterisation alone or together with prophylactic antibiotics in those with recurrent urinary tract infections. Children with detrusor hyperreflexia on VUD were treated with oxybutinin, whereas one child with mild bladder neck incompetence was treated with ephedrine. All achieved continence. The children have been followed up for a mean of three years (3 months to 16 years). Two children died within eight months of onset of the bladder dysfunction, but treating their bladder symptoms helped ease the discomfort and the inconvenience associated with urine infections and incontinence. Medication was unnecessary in two cases (7 and 10) as incontinence was acceptable in one child because of young age and a regular voiding regimen achieved continence in the other.

**Discussion**

CNS tumours are a rare cause of bladder dysfunction in children, as reflected in one series by Blavias in which spinal and brain tumours accounted for 0.06% of 336 patients with neurological conditions investigated for vesicourethral dysfunction.7 We have shown that various combinations of storage and voiding dysfunction can occur with tumours along the neural axis from the pons to the cauda equina. This is what we would expect given the complexity of central neural control of micturition, which requires coordinated actions of the autonomic and somatic nervous system. An area in the dorsal tegmentum of the pons is thought to act on a spino-bulbo-spinal pathway and “switch” between the storage and the voiding phases of micturition.7 The central function of the pons in the normal micturition reflex was first recognised by Barrington in animal studies in 1925.8 This was later supported by electrophysiological studies in which stimulation of bladder afferents produced field potentials in the pontine micturition centre and by positron emission tomography of the human brain during micturition, which showed an increased metabolic activity in the pontine area.9 Neural pathways that modulate bladder function traverse the length of the spinal cord between the pons and the sacral spinal cord. Interruption of these pathways may result in detrusor hyperreflexia and/or loss of the coordinated action of the detrusor and the external striated urethral sphincter known as detrusor sphincter dyssynergia (DSD).

Few clinical reports in adults and children with pontine tumours document voiding dysfunction.10 Ueki reported urinary symptoms in 22 patients with pontine tumours,11 and Renier and Gabreels in 1980 observed urinary retention and difficulty in voiding in 71% of children with pontine tumours.12 One of our patients had a brainstem astrocytoma arising at the level of the left facial colliculus and developed DSD. The contribution of suprapontine centres to neuropathic bladder dysfunction is poorly defined with the exception of the frontal lobes.13 Our patient with midbrain involvement secondary to a recurrence of a cerebellar astrocytoma showed a storage dysfunction in the form of hyperreflexia, with no dyssynergia. This may imply that the micturition centre modulating filling extends to the midbrain or that this area in the brain mediates the inhibitory influences from higher centres. This child had an internuclear ophthalmoplegia presumably owing to the proximity of the median longitudinal fasciculus to the area involved in micturition.

**INCIDENCE OF BLADDER DYSFUNCTION IN SPINAL AND BRAIN TUMOURS**

Urinary dysfunction is a frequent symptom in children with spinal tumours. Dincer et al in a retrospective review reported urinary dysfunction in 40.8% of children with spinal tumours.14 Bladder dysfunction is, however, rare in children with brain tumours and this is reflected in the few clinical reviews reporting this association. In a series of 462 patients with brain tumours, Ueki reports an incidence of 18% of patients with bladder dysfunction with the highest incidence seen in those with pontine tumours.15

**ONSET AND DEGREE OF BLADDER DYSFUNCTION IN SPINAL TUMOURS**

The location, size, underlying histology, and any delay in diagnosis of a spinal cord tumour combine to determine the clinical course of bladder dysfunction—that is, the time of onset and severity. Bladder dysfunction is often a late finding unless there is neoplastic infiltration of the conus medullaris.21 Bladder dysfunction observed in our series was related to the tumour encroaching on the spinal cord, whereas urinary symptoms were the first to appear in one child with a conus and cauda equina tumour. The biological behaviour of the...
Lower urinary tract dysfunction in children with CNS tumours

347

infections are also distressing and can be

functional incontinence and recurrent urinary tract

tumours and normal bladder function presenting over the same time interval. The main feature differentiating the two groups was tumour histology. In the children with normal bladder function most tumours were of low grade malignancy with predominantly cystic components.

OUTCOME

Advances in microsurgical techniques, radiotherapy, and, possibly, chemotherapy have improved survival in children with CNS tumours. Because of this the goals of treatment have broadened to include an improved quality of life; urological complications then become important determinants of the quality of life.

A VUD study remains the definitive and most informative investigation in children with neuropathic vesicourethral dysfunction. The simultaneous pressure measurements superimposed on the fluoroscopic appearance of the bladder and urethra provide valuable information on the behaviour of the bladder and urethra during filling and voiding. We were able to diagnose two types of voiding dysfunction in terms of distal sphincter behaviour: DSD and a fixed distal sphincter. The diagnosis of poor compliance, open bladder neck during filling, hyperreflexia, high pressure voiding, and vesicoureteric reflux can be made quickly and easily with this method. Correct interpretation of the urodynamic findings is essential for rational treatment, and these findings may also have prognostic significance. In their study on VUD evaluation of neuropathic vesicourethral dysfunction in children, Mundy et al showed that DSD was the main determinant of voiding efficiency and the most important cause for renal impairment; the state of the bladder neck during filling was the main determinant of bladder capacity; and poor compliance was the main ominous prognostic factor for continence. An ultrasound scan before and after micturition is a useful screening test that reliably identifies cases with significant residual volumes, thickening of the bladder wall, and upper renal tract dilatation—all indicators of altered lower urinary tract function.

The diagnosis and treatment of CNS tumours are distressing in themselves, additional incontinence and recurrent urinary tract infections are also distressing and can be demoralising for children and families. Clean intermittent catheterisation is an effective method of bladder emptying and can be used alone or in combination with anticholinergic and ß adrenergic agents to achieve satisfactory continence.

CONCLUSIONS

Urinary symptoms, although frequent in children with spinal cord tumours, are often underplayed. Our series shows that although bladder dysfunction occurs late in the course of most spinal cord tumours, loss of bladder control in established cases can be the earliest indication of tumour recurrence. Bladder dysfunction causes significant morbidity in surviving children. In these cases it is important to assess fully bladder and urethral dysfunction. VUD remains the definitive and most informative investigation in children with neuropathic vesicourethral dysfunction. However, if this is not possible because of the medical condition of the child, useful information can be obtained from an ultrasound scan before and after micturition.

Lower urinary tract dysfunction in children with central nervous system tumours

D Soler and M Borzyskowski

Arch Dis Child 1998 79: 344-347
doi: 10.1136/adc.79.4.344

Updated information and services can be found at:
http://adc.bmj.com/content/79/4/344

These include:

References
This article cites 19 articles, 3 of which you can access for free at:
http://adc.bmj.com/content/79/4/344#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic Collections
Articles on similar topics can be found in the following collections

- Oncology (778)
- Stroke (227)
- Neurooncology (62)
- Neuromuscular disease (166)
- Rheumatology (522)
- Urology (446)

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/