Causes of Urinary Incontinence After Acute Hemispheric Stroke

David A. Gelber, MD; David C. Good, MD; Laurie J. Laven, MD; and Steven J. Verhulst, PhD

Background and Purpose: We prospectively studied bladder function in stroke patients to determine the mechanisms responsible for poststroke urinary incontinence.

Methods: Fifty-one patients with recent unilateral ischemic hemispheric stroke admitted to a neurorehabilitation unit were enrolled. The presence of urinary incontinence was correlated with infarct location, neurological deficits, and functional status. Urodynamic studies were performed on all incontinent patients.

Results: Nineteen patients (37%) were incontinent. Incontinence was associated with large infarcts, aphasia, cognitive impairment, and functional disability (<0.05) but not with age, sex, side of stroke, or time from stroke to entry in the study. Urodynamic studies, performed on all 19 incontinent patients, revealed bladder hyperreflexia in 37%, normal studies in 37%, bladder hyporeflexia in 21%, and detrusor–sphincter dyssynergia in 5%. All of the patients with normal urodynamic studies were aphasic, demented, or severely functionally impaired. All of the patients with hyperreflexic bladders had underlying diabetes or were taking anticholinergic medications. Forty-six percent of incontinent patients treated with scheduled voiding alone were continent at discharge compared with 17% of patients treated pharmacologically.

Conclusions: There are three major mechanisms responsible for poststroke urinary incontinence: 1) disruption of the neuromicturition pathways, resulting in bladder hyperreflexia and urgency incontinence; 2) incontinence due to stroke-related cognitive and language deficits, with normal bladder function; and 3) concurrent neuropathy or medication use, resulting in bladder hyporeflexia and overflow incontinence. Urodynamic studies are of benefit in establishing the cause of incontinence. Scheduled voiding is a useful first-line treatment in many cases of incontinence. (Stroke 1993;24:378–382)

Key Words • cerebrovascular disorders • urinary incontinence • urodynamics

Urinary incontinence is a common sequela of acute stroke, with the incidence ranging from 38% to 60% in the early recovery period.1–3 Previous investigators have suggested that poststroke urinary incontinence is most often due to disruption of the neuromicturition pathways, which results in bladder hyperreflexia.4–6 Although urodynamic studies have shown that many patients do not have hyperreflexic bladders, the causes of urinary incontinence in these patients have not been investigated. Identifying other potential causes of incontinence is extremely important from a clinical standpoint because treatment strategies may differ depending on the etiology.

Previous studies have shown an association between the development of urinary incontinence and the presence of certain neurological deficits, including moderate or severe motor deficits,2 aphasia,1,2 and the combination of hemiplegia, proprioceptive deficits, and visual neglect.1 However, the studies have not specifically determined whether these deficits might be directly responsible for incontinence, independent of bladder dysfunction. We hypothesized that in some stroke patients urinary incontinence may be due directly to stroke-related cognitive or language deficits rather than to selective dysfunction of the neuromicturition pathways.

In addition, previous investigators have noted bladder hyperreflexia in 17–25% of poststroke patients. This raises the possibility of overflow incontinence as another cause of urinary incontinence in stroke patients.1,7–9

We prospectively evaluated a series of patients admitted to a rehabilitation unit after acute unilateral hemispheric stroke. Ours is the first study to critically evaluate the various mechanisms responsible for the development of poststroke urinary incontinence and to propose treatment strategies based on these mechanisms.

Subjects and Methods

Fifty-one consecutive patients with unilateral ischemic hemispheric stroke admitted to the neurorehabilitation service at our institution were enrolled. Patients with nonischemic stroke (intracerebral or subarachnoid hemorrhage), history of previous stroke, evidence of brain stem or bilateral signs on examination, history of urinary incontinence, or previous urologic surgery were excluded. Written consent was obtained from all patients.

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A detailed physical and neurological examination was performed on all patients. A Mini-Mental State (MMS) examination was obtained for all nonaphasics patients (total possible score, 30). Functional status on admission and discharge from the rehabilitation service was assessed by Barthel Index (BI) score, a valid and reliable outcome measure for stroke. The score is calculated based on the patient’s ability to perform 10 different mobility tasks and activities of daily living, with a maximum score of 100. Because the BI includes an assessment of bladder function, a modified Barthel Index (MBI) score was also calculated on admission and discharge; the MBI excluded the measure of bladder function, leaving a maximum score of 90.

A head computed tomographic (CT) scan or magnetic resonance imaging (MRI) was obtained for all patients. Infarctions were classified by side of lesion (left or right hemisphere) and localization (pure cortical, pure subcortical, or combination cortical plus subcortical) on the basis of neuroimaging.

To eliminate the possibility of bladder irritability due to urinary tract infection, a urine culture was performed for all patients. If the culture was positive, a 7-day course of antibiotics was given. Initiation of the study protocol began only after a repeat urine culture was negative.

The frequency of episodes of incontinence was carefully recorded by staff nurses daily throughout the hospitalization. Patients incontinent at any time during the hospitalization underwent a urodynamic study, including cystometry and urethral sphincter electromyography. Studies were performed using a Browne Profile 6 (six-channel) urodynamic machine, with carbon dioxide distillation at 50 cc/minute and simultaneous recording of bladder and urethral sphincter pressures. Urodynamic study results were classified as follows: 1) normal study, sensation of bladder filling at volumes below 300 cc, absence of detrusor contractions greater than 15 cm water during filling, and relaxation of the urethral sphincter during voiding; 2) detrusor hyperreflexia, presence of spontaneous uninhibited bladder contractions greater than 15 cm water at bladder volumes less than 300 cc; 3) detrusor hypo-reflexia, absence of detrusor contractions greater than 15 cm water at bladder volumes greater than 450 cc; 4) detrusor–sphincter dyssynergia, contraction of the urethral sphincter simultaneously with detrusor contraction.

No specific treatment protocol was followed. Treatment of each incontinent patient was based on the recommendations of the consulting urologist and rehabilitation team. Patients with severe bladder hyperreflexia were often treated with anticholinergic medications (oxybutynin, imipramine) or antispasmodics (flavoxate). α-Adrenergic blockers (prazosin, terazosin) were used if there was evidence of internal sphincter hypertonus. Incontinent patients with normal urodynamic studies or mild bladder hyperreflexia were generally treated nonpharmacologically, with only a scheduled voiding program: this nursing strategy involved placing patients on the commode or offering them a urinal or bedpan every 2–4 hours. Patients with bladder hypo-reflexia were managed with scheduled voiding, intermittent straight catheterization, or an external collection device (condom catheter).

### Table 1. Admission Demographics in Incontinent and Continent Patient Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Incontinent</th>
<th>Continent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>19</td>
<td>32</td>
</tr>
<tr>
<td>Time from stroke to entry*</td>
<td>21.4±1.8</td>
<td>20.6±1.9</td>
</tr>
<tr>
<td>Age* (years)</td>
<td>69.2±2.2</td>
<td>70.4±2.0</td>
</tr>
<tr>
<td>Sex†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Women</td>
<td>6</td>
<td>17</td>
</tr>
</tbody>
</table>

Data are mean±SEM.  
*p>0.05 difference between incontinent and continent groups by Student’s t test.  
†p>0.05 difference between incontinent and continent groups by 2×2 χ² analysis.

Comparisons between groups of patients were performed with an independent t test. Relations between the variables were evaluated by Pearson correlations and logistic regression. Categorical variables were analyzed with the χ² test. All continuous variables are presented as mean±SEM. Values of p<0.05 were considered significant.

### Results

Nineteen of the 51 patients enrolled (37%) were incontinent. Incontinent and continent patients did not differ with respect to sex, age, or time from stroke to entry in the study (p>0.05; Table 1).

There was no correlation between side of stroke and the development of urinary incontinence. However, incontinence was significantly associated with large infarctions; 17 of 24 patients with combined cortical plus subcortical strokes developed urinary incontinence, whereas only two of 15 patients with cortical infarcts and none of 12 patients with subcortical infarcts were incontinent (p<0.001; Table 2). The odds ratio derived from logistic regression analysis showed that patients with large combined cortical plus subcortical strokes were 5.3 times more likely to be incontinent than patients with pure cortical or pure subcortical strokes.

The presence of aphasia strongly correlated with the development of urinary incontinence (p=0.003). Of the 19 incontinent patients, 14 (74%) were aphasic, whereas

### Table 2. Stroke Localization in Incontinent and Continent Patient Groups

<table>
<thead>
<tr>
<th>Side of lesion*</th>
<th>Incontinent</th>
<th>Continent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>Right</td>
<td>6</td>
<td>16</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Site of lesion</th>
<th>Incontinent</th>
<th>Continent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortical</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>Subcortical</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Cortical plus subcortical</td>
<td>17†</td>
<td>7</td>
</tr>
</tbody>
</table>

*p>0.05 difference between incontinent and continent groups by 2×2 χ² analysis.  
†p<0.01 for association of incontinence with large cortical plus subcortical infarcts by 2×3 χ² analysis.
TABLE 3. Neurological Deficits and Functional Status Scores in Incontinent and Continent Patient Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Incontinent</th>
<th>Continent</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasia</td>
<td>0.003*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>14</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>5</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Mini-Mental State score</td>
<td>24.0±1.2</td>
<td>26.7±0.6</td>
<td>0.041†</td>
</tr>
<tr>
<td>Barthel Index score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission</td>
<td>21.8±2.1</td>
<td>44.2±3.1</td>
<td>0.0001†</td>
</tr>
<tr>
<td>Discharge</td>
<td>42.9±4.1</td>
<td>73.0±3.4</td>
<td>0.0001†</td>
</tr>
<tr>
<td>Modified Barthel Index score</td>
<td>20.8±1.7</td>
<td>38.0±2.6</td>
<td>0.0001†</td>
</tr>
<tr>
<td>Discharge</td>
<td>38.7±3.4</td>
<td>63.4±3.1</td>
<td>0.0001†</td>
</tr>
</tbody>
</table>

Data are mean±SEM.
*Association of aphasia with incontinence by 2×2 χ² analysis.
†By Student's t test.

only 10 of 32 continent patients (31%) were aphasic. Aphasic patients were 2.3 times more likely to be incontinent than nonaphasic patients. In the nonaphasic patients, incontinence was significantly associated with cognitive impairment as measured by the MMS examination; the mean MMS score was 26.7 for continent patients and 24.0 for incontinent patients (p=0.041; Table 3). There was poor correlation between large combined cortical plus subcortical infarcts and the presence of either aphasia or cognitive impairment (r=0.08 and r=0.09, respectively).

Incontinence was significantly associated with poorer overall functional status as measured by the BI and MBI on both admission to and discharge from the rehabilitation unit (Table 3). Mean BI scores were 44.2 for continent patients and 21.8 for incontinent patients on admission and 73.0 and 42.9, respectively, at discharge (p=0.0001). Modified BI scores were 38.0 for continent patients and 20.8 for incontinent patients on admission and 63.4 and 38.7, respectively, at discharge (p=0.0001).

Urodynamic studies were performed on all 19 incontinent patients (Figure 1). Seven patients (37%) had normal studies, seven (37%) had detrusor hyperreflexia, one (5%) had detrusor–sphincter dyssynergia, and four (21%) had detrusor hyporeflexia. In the hyporeflexic group, three patients had diabetic polyneuropathy, and one was taking medication with anticholinergic side effects. Of the subgroup with normal urodynamic studies (n=7), five patients were aphasic, one was cognitively impaired (MMS score of 23), and the other severely functionally impaired (BI score of 25).

Of the 19 incontinent patients, 13 were treated non-pharmacologically (with a scheduled voiding program alone), and six were treated with medications. The patients treated only with scheduled voiding had a mean frequency of 4.7 incontinent episodes per day on admission and 1.5 incontinent episodes per day on discharge from the rehabilitation unit. The patients treated with medications had a mean frequency of 3.6 incontinent episodes per day on admission and 3.2 on discharge. Six of 13 patients (46%) treated with scheduled voiding alone were continent at discharge, whereas only one of six (17%) of the patients treated pharmacologically were continent. Of the subgroup of patients with bladder hyperreflexia, three of five (60%) treated with scheduled voiding alone showed improvement in the frequency of incontinent episodes, whereas neither of the two patients treated pharmacologically showed improvement. Similarly, in the subgroup of patients with normal urodynamic studies, two of three (67%) treated only with scheduled voiding improved, whereas only one of four (25%) treated medically improved.

FIGURE 1. Graph showing results of urodynamic studies performed on all of the stroke patients with urinary incontinence (n=19).

Discussion

Urinary incontinence is a common sequela of acute stroke. In this prospective study of patients with acute ischemic hemispheric stroke admitted to an acute rehabilitation service, 37% exhibited urinary incontinence. This figure is in relative agreement with other studies that have reported the incidence of poststroke urinary incontinence to be 38–60%. The prevalence of urinary incontinence decreases with time elapsed from the acute stroke. In a study by Borrie et al., the prevalence of urinary incontinence was 60% at 1 week, 42% at 4 weeks, and 29% at 12 weeks after stroke. Our patients were studied an average of 21 days after the acute stroke; it is therefore likely that our incontinence figure would have been even higher if these patients had been studied earlier in the poststroke period.

Our data suggest that there are three major causes of poststroke urinary incontinence. The first mechanism is direct injury to the neuromicturition pathways caused by infarction. Cerebral hemispheric lesions most commonly cause detrusor hyperreflexia, with sphincters that relax in a coordinated fashion during voiding; as expected, this is the urodynamic abnormality noted most commonly in stroke patients. In previously reported series of poststroke patients in rehabilitation units, bladder hyperreflexia has been documented in 50–82%.

In our series, bladder hyperreflexia was documented in only 37% of continent patients. Our figure may have been lower than in these other series for several reasons. We selected patients only with unilateral ischemic hemispheric stroke and were careful to exclude patients with any past history of urinary symptoms. The time from stroke to study entry may also have been a factor because there is evidence that bladder hyperreflexia may actually increase with time after acute stroke. Feder et al. studied stroke patients with serial urodynamic studies. Forty-seven percent had hyperreflexic bladders within the first 2 months of their stroke,
whereas 65% did when studied up to 5 months after stroke. Since our patients were studied at only 3 weeks after stroke, it is possible that we would have found a higher incidence of bladder hyperreflexia had our patients been studied further into the recovery period.

A second proposed mechanism is that poststroke urinary incontinence may result from stroke-related cognitive or language deficits. Previous studies have shown an association between incontinence and the presence of certain neurological deficits but have not definitively concluded that these deficits might be directly responsible for incontinence. Reding et al. found an association between incontinence and the presence of aphasia, or the combination of hemiplegia, visual neglect and proprioceptive loss, but did not find an association between incontinence and cognitive impairment. On the other hand, Borrie et al. did find an association between incontinence and cognitive impairment, as well as moderate-to-severe motor deficits and impaired mobility. In our series there was a significant correlation between the development of incontinence and the presence of aphasia, cognitive impairment, and poor overall functional status as measured by the BI, even when the measure of bladder function was excluded from the scale. Of the seven incontinent patients with normal urodynamic studies, all were aphasic, dem- ented, or markedly impaired functionally. These patients were not incontinent before the stroke and had no other explainable cause for their incontinence. This suggests that stroke-related neurological deficits may be directly responsible for urinary incontinence, especially in patients with normal bladder function demonstrated by urodynamic studies deficits. Aphasic or cognitively impaired patients may not be able to communicate the need to void. Patients with impaired motor or perceptual skills may not be able to handle a urinal or maneuver safely to a commode to adequately maintain continence.

A third proposed cause of urinary incontinence is bladder hyporeflexia, with resultant overflow incontinence. In our series, 21% of incontinent patients were found to have detrusor hyporeflexia; this finding is in agreement with the 17–25% figures noted in other series. Although bladder hyporeflexia typically results from injury to the sacral spinal cord, sacral nerve roots, or peripheral nerves, some have postulated that hyporeflexia might occur in the early phase of acute hemispheric stroke, with gradual increase in bladder tone over time. In Maru’s series, seven of 31 patients studied within the first 48 hours of stroke had areflexic bladders; subsequent urodynamic studies showed “normalization” of bladder function in all. Feder et al. noted resolution of bladder hyporeflexia in one of four patients when studied with serial urodynamic studies. However, these studies did not evaluate patients for other possible causes of bladder hyporeflexia. In all of our patients with bladder hyporeflexia, there were causes other than the stroke itself to explain this urodynamic pattern (either diabetic polyneuropathy or use of anticholinergic medications).

One patient had evidence of detrusor–sphincter dyssynergia on urodynamic study. This abnormality is most commonly associated with lesions between the pons and sacral spinal cord and cannot be explained by unilateral hemispheric stroke. This patient may have had concurrent spinal cord disease that accounted for this urodynamic abnormality; however, this was not investigated.

Information regarding the location of hemispheric lesions associated with urinary incontinence is lacking. Although animal studies have identified various cortical and subcortical structures that influence voiding, the location of specific lesions that cause detrusor hyperreflexia in humans remains speculative. Studies by Khan et al. and Tsuchida et al. noted bladder hyperreflexia in strokes involving the cerebral cortex, internal capsule, and basal ganglia. Unfortunately, these studies were retrospective, included patients with strokes of varying age, and evaluated only symptomatic stroke patients referred to a urolgy. In recent prospective studies of urinary incontinence after acute stroke, neither Reding et al. nor Linsenmeyer et al. were able to correlate lesion localization with the development of incontinence. Although our study did not specifically evaluate infarct localization, there was no correlation between the side of stroke and the development of incontinence. Further studies using more definitive CT/MRI localization are needed to more convincingly localize the brain “bladder centers” in humans.

The relation of infarct size to the development of urinary incontinence also remains controversial. Reding et al. found no correlation between lesion size and the development of poststroke incontinence. Feder et al., on the other hand, found a significant association between infarct size (lesions greater than 40 mm in diameter) and the presence of bladder hyperreflexia with urinary incontinence. Although our study did not volumetrically measure infarct size, we found a significant correlation between the development of incontinence and the presence of combined cortical plus subcortical infarctions, most of which were due to large infarcts in the distribution of the middle cerebral artery. It could be anticipated that large infarcts would be more likely to lead to urinary incontinence on the basis of two of the mechanisms described above: large infarcts would be more likely to affect the neuromicturition pathways and also cause significant cognitive or language impairments that might have an impact on bladder function. In our study, however, there was no correlation between the presence of large combined cortical plus subcortical strokes and either aphasia or cognitive impairment, suggesting that these are independent variables. This further supports our hypothesis that incontinence in some stroke patients may be due to specific neurological impairments independent of stroke size and location.

The results of this study support the role of urodynamic studies in evaluating bladder function in the stroke patient. These data show that incontinent patients have a number of different urodynamic patterns, each of which warrants a different treatment strategy. Unfortunately, in clinical practice anticholinergic and antispasmodic medications are often used empirically in the treatment of incontinent poststroke patients, based on the presumption that bladder hyperreflexia exists. This study clearly shows that a majority of poststroke incontinent patients, studied early in the recovery period, do not have bladder hyperreflexia; use of anticholinergic or antispasmodic medications may not improve incontinence in these patients, and it subjects them to the risk of unnecessary
side effects. Even our patients with bladder hyperreflexia often responded favorably to treatment with a scheduled voiding program alone. Because this study was not designed to specifically compare treatments or evaluate treatment outcomes, these data should be interpreted cautiously; they do suggest, however, that simple nursing strategies may be the most useful first-line treatment, with anticholinergic medications reserved for refractory cases of bladder hyperreflexia. Incontinent patients with normal urodynamic studies often respond well to simple nursing strategies, such as a scheduled voiding program, and should not be treated pharmacologically. Treatment for patients with hyperreflexic bladders and overflow incontinence, in the absence of bladder outlet obstruction, includes intermittent straight catheterization, or use of cholinergic medications. Further detailed studies are needed to prospectively evaluate these treatment strategies in the poststroke patient with urinary incontinence.

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References

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