

Urinary retention after stroke

Urinary retention after stroke is common and the mechanism is multifactorial. The management is similar to its that of any other cause. In the majority of patients, it resolves with time and the overall prognosis seems favourable.

Dr Walee Sayed* Consultant Geriatrician, Department of Geriatric Medicine, Wrexham Maelor Hospital, Wrexham, Clwyd LL13 7TD, UK.

Dr Vedamurthy Adhiyaman Consultant Geriatrician, Department of Geriatric Medicine, Glan Clwyd District Hospital, Rhyl, Denbighshire LL18 5UJ, UK.

Dr Indrajit Chattopadhyay Consultant Geriatrician, Department of Geriatric Medicine, Glan Clwyd District Hospital, Rhyl, Denbighshire, LL18 5UJ, UK.

*email Walee.sayed@new-trust.wales.nhs.uk

Urinary incontinence after stroke is common and well recognised. The incidence varies from 38 to 69% in the early period after stroke.¹⁻⁴ However, urinary retention following stroke is less well recognised and investigated. It can occur both immediately after stroke (the first 72 hours) and in the recovery period, and it can affect both sexes. The aim of this article is to review the existing literature on urinary retention after stroke.

Neuroanatomy of micturition

A complex neural network involving the cortex, brainstem, spinal cord, and peripheral nerves control the function of the urogenital tract.⁵⁻⁶ (figure 1) Three sets of peripheral nerves innervate the urogenital system. They include the sacral parasympathetic (pelvic nerves) outflow, which takes the excitatory input to the bladder; the thoracolumbar sympathetic outflow (hypogastric nerves and sympathetic chain), carrying the inhibitory signal; and the sacral somatic nerves (pudendal nerve) that innervate the external urethral sphincter.

The afferent pathways from the urogenital tract project to the dorsal horn of the spinal cord. From here, the spinal interneurons either ascend up the spinal cord and terminate in the brain stem, or make local connections with the sacral parasympathetic nucleus forming the sacral spinal reflex arc.

A variety of neurones that control the lower urinary tract have been identified in the brainstem in animal models. The most important ones include the collection of neurones in the pons called the pontine-micturition centre. Stimulation of this centre induces firing of the sacral preganglionic neurones, initiating bladder contractions leading to release of urine, and bilateral lesions here abolish micturition in animals.⁷ Other neurones include the medullary raphe nuclei, locus coeruleus, and periaqueductal grey.

Organisation of the cortical centres (otherwise called suprapontine centres) for micturition is less well defined. The prefrontal cortex (paracentral lobule) and the anterior cingulate gyrus are the most important sites. The neurones from these areas inhibit the pontine-micturition centre either directly or through the anterior hypothalamus. Positron emission tomography in humans has shown increased activity in these areas during voiding.⁸ Lesions in the prefrontal cortex are known to result in incontinence by abolishing inhibitory control to the pontine-micturition centre.

The hypothalamus has a substantial mediating role in control of micturition. Neurones from the anterior hypothalamus provide an excitatory input to the pontine-micturition centre, and electrical stimulation of this region induces bladder contractions. Neurones from the medial and posterior hypothalamus directly inhibit the sacral parasympathetic nucleus and the sphincter motor nucleus, and stimulation of this area inhibits bladder emptying.

Mechanism of retention

The mechanism of urinary retention after stroke is multifactorial. Acute urinary retention occurring immediately after stroke is caused by detrusor areflexia. The neurophysiological explanation for this phenomenon is unclear, but has been referred to as cerebral shock.⁹ Non-neurological causes precipitating retention, such as benign prostatic hyperplasia, diabetes mellitus, and anticholinergic drugs, have been reported.¹⁰⁻¹¹ Ineffective voiding demonstrated by post-voidal residual volumes is common in community-dwelling elderly patients.¹² In these patients, the onset of post-stroke detrusor hyporeflexia could precipitate retention.

Even though strong associations between urinary retention and poor functional status, cognitive impairment, and aphasia have been noted, retention is instead possibly due to difficulties in communication and poor mobility.¹⁰

One study showed an increased association between urinary retention and haemorrhagic and cerebellar strokes. But its importance remains unclear because the numbers were too small.¹¹ So far no correlation has been found between the site and nature of the stroke and occurrence of urinary retention. Urinary-tract infection is frequently associated with retention, but whether infection contributes to retention is debatable.

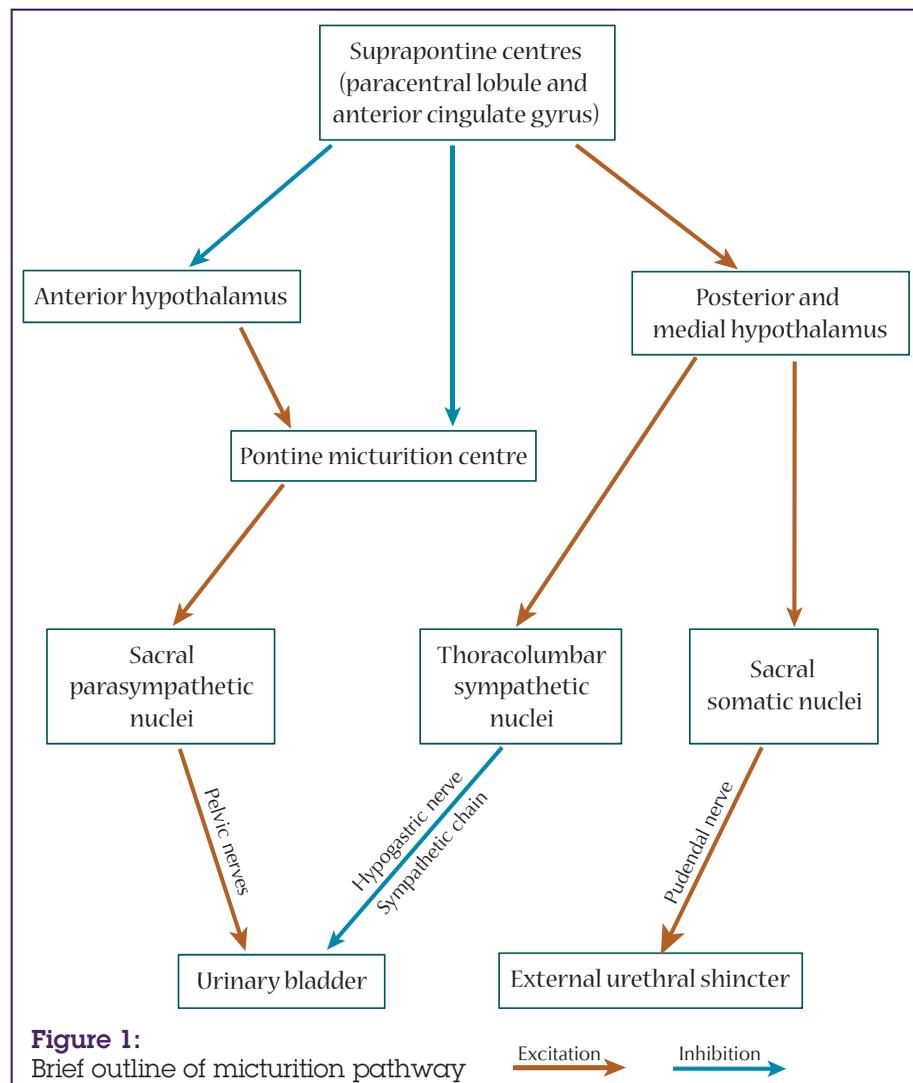
Influence of gender on retention

Data for sexual differences in urinary retention is very limited and too incomplete to draw any meaningful conclusion. A study of 80 patients (48 men, 32 women) with ischaemic stroke revealed that 23 had urinary retention, of which, 18 (80%) were men and 5 were women.¹⁰ Four patients had diabetes with peripheral

neuropathy (two men and two women), and the rest had no history of urinary incontinence or urological surgery. The slightly higher proportion of men in the study could not alone explain the higher incidence in men. Since the mean age of the cohort was 65.4 years, some of the men might have had mild or even asymptomatic prostatic hyperplasia and the occurrence of post-stroke hyporeflexia could have precipitated retention.

In another group of 39 patients, 5 had retention (3 men and 2 women), but this study had a very large variation in the interval between stroke and assessment (11 days to 13 years) rendering further analysis impossible.¹¹ From the existing literature, a possible conclusion is that retention can occur in both sexes, in the absence of any underlying urological disorder, but is more common in men.

Incidence of urinary retention



22–47% of patients had retention on urodynamic studies done within 72 hours of acute stroke. The majority of these patients (75%) showed detrusor areflexia and the rest had detrusor hyper-reflexia, but retention was due to either detrusor-sphincter dyssynergy or to prostatic hyperplasia.^{11,13,14}

The incidence of retention noted in studies during the first few weeks after stroke varies (29–56%), depending on the study group, the interval after stroke, and the criteria used to define retention. In a group of 80 patients with ischaemic stroke, 29% had urinary retention (post-voidal urine of more than 100 ml) at a mean interval of 14 days post-stroke.¹⁰ 80% of patients were men, and a third were asymptomatic. Cognitive impairment, aphasia, diabetes mellitus, and poor functional state were strongly associated with urinary retention. No significant association was found between the use of anticholinergic medication and retention. The incidence was higher (56%) in another study, which used a lower post-voidal residual urine (>50 ml) to define retention.¹⁵

Many patients with retention regain normal bladder function within weeks to months, and few data for incidence of urinary retention in the long-term are available. In a small study of 31 patients, all seven patients who had detrusor areflexia within 48 hours after stroke regained normal bladder function by discharge.¹⁴ Another study found that of 23 patients with detrusor areflexia, 19 had resolution by discharge after an average hospital stay of 46 days. The remaining four had diabetes mellitus with evidence of peripheral neuropathy. Three patients improved after 2 months and detrusor areflexia persisted in one.¹⁰ Therefore, the incidence of retention without incontinence in the long-term is probably very low.

Retention as a cause of incontinence

Many factors could contribute to urinary incontinence after stroke. These include: lesion to the neuromicturition pathway, leading to detrusor abnormalities; motor disability; altered consciousness; sensory loss; ataxia; depression; and aphasia. Detrusor hyporeflexia is the underlying cause in 17–25% of patients with urinary incontinence.^{16–17} In these patients, exclusion of this condition is important (post-voidal residual urine on bladder scan is a very good indicator) before prescribing anticholinergic drugs.

Diagnosis

The diagnosis of retention could be difficult in patients with aphasia and altered sensorium, especially if it is associated with overflow. The basic investigations of urinary symptoms include urine analysis, biochemical examinations (glucose, electrolytes, urea, creatinine, prostate-specific antigen in patients with enlarged prostate), maintenance of a frequency-volume chart, and bladder scan. Bladder scan is non-invasive, very simple to use, and can give valuable information such as post-voidal residual volume. When the cause of retention remains obscure after initial evaluation, urodynamic studies should be reviewed, and specialist referral is indicated.

Management

The management of urinary retention after stroke is similar to that for any other cause. The condition is transitional in most patients, occurring immediately after stroke. Intermittent clean catheterisation is the best option in the acute period for up to 6 weeks after stroke. Indwelling catheters should be avoided because

of the risks of urethral trauma and infection. Other causes of retention must be investigated and managed appropriately. Detrusor hyporeflexia must be borne in mind before treating urinary incontinence with anticholinergic agents.

Long-term prognosis

In the majority of patients, urinary retention resolves with time and the overall prognosis seems favourable. Retention persists only in patients with other contributing diseases, such as diabetes and prostatic hyperplasia. Some patients with retention could develop incontinence during recovery because of the alteration of detrusor function from areflexia to hyper-reflexia with time.¹⁸

We have no conflict of interest.

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