

KEYPOINTS:

- Although the pelvic organs are innervated peripherally by the autonomic nervous system, an essential feature of their physiological behavior is that they are under voluntary control. Cortical input is therefore critical.
- Lesions in the plane of the genu of the corpus callosum involving some of the white matter anterior to the anterior horns of the lateral ventricle cause a permanent disorder of control of micturition. The mechanism was thought to be that disconnection of the frontal or anterior cingulate regions from the septal and hypothalamic areas allowed micturition to proceed automatically and involuntarily.
- Studies of patients' bladder function following CVAs have examined the relationship in two different ways: there have been studies of urodynamic changes following CVAs in small groups of patients with correlation of the site of the lesion with the urodynamic findings; and other studies which have examined incontinence following stroke.

CHAPTER 9

BRAIN DISEASES

INTRODUCTION

Although the pelvic organs are innervated peripherally by the autonomic nervous system, an essential feature of their physiological behavior is that they are under voluntary control. Cortical input is therefore critical. Whereas some clinical information currently exists in relation to the cortical control of the bladder, much less is known about cerebral determinants of bowel and sexual function. This chapter focuses on how brain disease can cause disorders of bladder, bowel and sexual function.

CEREBRAL CONTROL OF MICTURITION

Cortex. The most authoritative work in this area was the study by Andrew and Nathan in 1964, which described 38 patients with disturbances of micturition as a result of lesions in the anterior frontal lobe [1]. The infrequency with which such patients are encountered is highlighted by the fact that these authors had been collecting cases separately over a period of 24 years and only later learnt of each other's interest and prepared a joint paper. There were 10 patients with intracranial tumors, two with anterior frontal lobe damage following rupture of an aneurysm, and four who had penetrating brain wounds. There were also 22 patients who had undergone leukotomy and these patients' lesions proved to be the most valuable in terms of localization of important brain structures.

The conclusion was that lesions in the plane of the genu of the corpus callosum involving some of the white matter anterior to the anterior horns of the lateral ventricle cause a permanent disorder of control of micturition. The mechanism was thought to be that disconnection of the frontal or anterior cingulate regions from the septal and hypothalamic areas allowed micturition to proceed automatically and involuntarily. A typical case of incontinence due to frontal lobe impairment described in this paper was of a patient with severe urgency and frequency

of micturition and urge incontinence without dementia, socially aware and embarrassed by their incontinence. Micturition was normally coordinated, indicating that the disturbance was in the higher control of this process.

This paper was followed by the report of a series of 50 consecutive cases of frontal lobe tumors, seven of which were found to exhibit the syndrome previously described [2]. It was not found in 100 consecutive nonfrontal intracranial tumors, indicating the localizing value of the syndrome.

Although the paper of Andrew and Nathan has been the most influential in the study of frontal lobe control of the bladder, it was not the first. There had been a much earlier study from Germany, and in 1960, Ueki, a Japanese neurosurgeon, analyzed the urinary symptoms of 462 patients with brain tumors who had undergone surgery, 34 cases of frontal lobectomy and 16 cases of bilateral anterior cingulectomy [3]. His conclusions were illustrated with a diagram of the brain showing a strong positive influence on micturition of an area in the pons and an inhibitory input from the frontal lobe.

With the advent of computerized tomography (CT) scanning and magnetic resonance imaging (MRI), several other studies have examined the role of focal brain lesions causing urinary dysfunction [4].

Urinary retention may also occur as a result of a cortical lesion. In the series by Andrew and Nathan, two patients were in urinary retention at some stage. More recently, there have been single-case studies of patients with reversible right frontal lesions who presented with urinary retention [4]. Their voiding disorder resolved with successful treatment of the underlying neurological pathology.

Changes in bladder control following cerebrovascular accidents (CVAs). Studies of patients' bladder function following CVAs have examined the relationship in two different ways: there have been studies of urodynamic changes following CVAs in small groups

of patients with correlation of the site of the lesion with the urodynamic findings; and other studies which have examined incontinence following stroke.

Urodynamics studies following CVAs. A study by Khan et al. [5] examined 20 patients who had presented at a urology department with urinary incontinence 3 or 4 more months after a CVA. CT scanning was performed to localize the area of brain injury. In four, the basal ganglia had been affected; in eight, the frontoparietal region; in one, the frontal region; in one, the parietal region; in four, there was diffuse bilateral ischemic damage; and in two, CT scans were normal. The most common cystometric finding in this disparate group was of detrusor hyperreflexia which was found in all but one patient who had a stable bladder, but was unable to communicate due to aphasia and had poor mobility. These authors followed up their first report with a further study of a similar design in which there were 33 patients with voiding problems following a CVA [6]. Again, the predominant finding was of involuntary contractions of the bladder which was present in 26, all of whom had normal co-ordinated voiding. The majority of patients with cerebral cortex and/or internal capsular lesions had uninhibited relaxation of the sphincter during involuntary bladder contractions, while all of the patients with lesions only in the basal ganglia or thalamus had normal sphincter function.

Similar findings were reported by Tsuchida et al. [7] who looked at 39 hemiplegic patients using urodynamic studies and CT scanning. Again the most common finding was of urinary urge incontinence, although 13 patients complained of difficulty with micturition. Ten of the 11 patients who had frontal and internal capsular lesions showed bladder hyperreflexia, as did nine of the 10 patients who had lesions of the putamen. Normal sphincter relaxation was coordinated in all these patients. In the remaining patients, no correlation was found between urodynamic dysfunction and type of brain injury.

Most recently, Sakakibara et al. [8] reported on the bladder symptoms of 72 patients who had been admitted with an acute hemispheric stroke. When assessed at 3 months, 53% were found to have significant urinary complaints. The commonest problem was of nocturnal

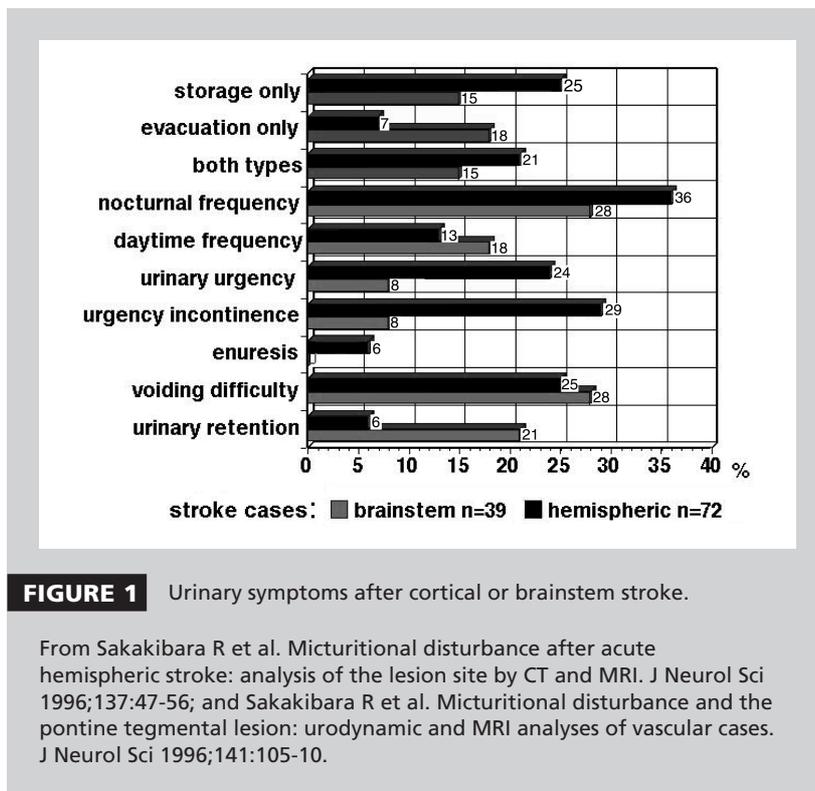


FIGURE 1 Urinary symptoms after cortical or brainstem stroke.

From Sakakibara R et al. Micturitional disturbance after acute hemispheric stroke: analysis of the lesion site by CT and MRI. *J Neurol Sci* 1996;137:47-56; and Sakakibara R et al. Micturitional disturbance and the pontine tegmental lesion: urodynamic and MRI analyses of vascular cases. *J Neurol Sci* 1996;141:105-10.

urinary frequency which affected 36%, while urge incontinence affected 29% and a difficulty in voiding affected 25% (Figure 1).

Urodynamic studies of 22 symptomatic patients showed detrusor hyperreflexia in 68%, detrusor-sphincter dyssynergia in 14% and uninhibited sphincter relaxation in 36% (Figure 2). The phenomenon of uninhibited sphincter relaxation appears to be quite specific for frontal lesions and is not seen with basal ganglia lesions causing hyperreflexia and never seen following spinal cord injury. The patients complain of first sensation and maximum desire to void simultaneously and a strong detrusor hyperreflexia occurs accompanied by sphincter relaxation (Figure 3). If the patient lacks a sensation of urgency, this is called "reflex incontinence".

There was some indication that lesion size was related to the occurrence of urinary symptoms: large fronto-temporo-parietal lesions caused a higher incidence of urinary problems than frontal lesions alone, and whereas large lesions of the putamen or thalamus caused voiding difficulty, small lesions did not. Lacunar infarcts (smaller than 10 mm in diameter) of the internal capsule, however, also caused

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- The phenomenon of uninhibited sphincter relaxation appears to be quite specific for frontal lesions and is not seen with basal ganglia lesions causing hyperreflexia and never seen following spinal cord injury. The patients complain of first sensation and maximum desire to void simultaneously and a strong detrusor hyperreflexia occurs accompanied by sphincter relaxation.

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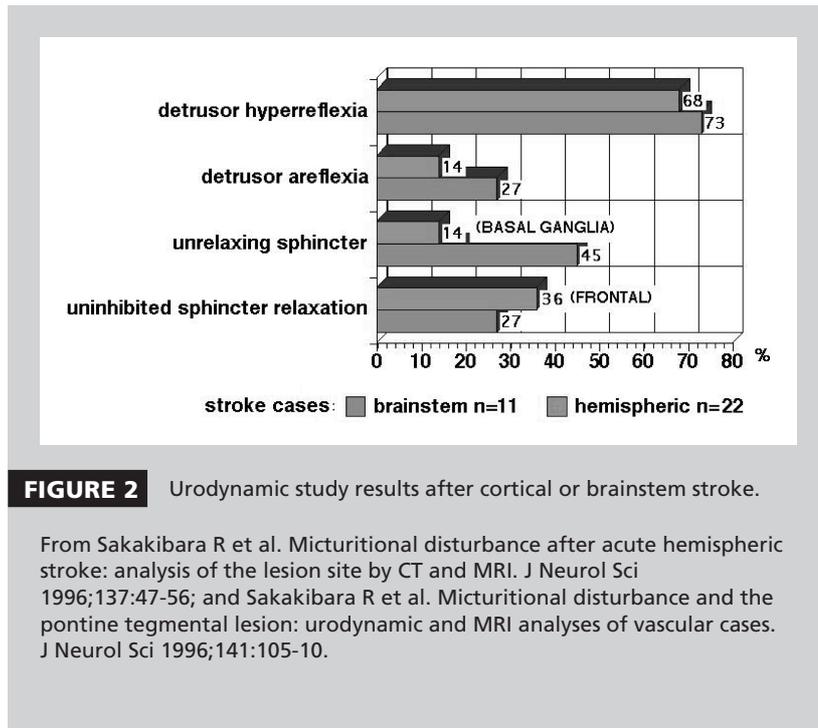


FIGURE 2 Urodynamic study results after cortical or brainstem stroke.

From Sakakibara R et al. Micturitional disturbance after acute hemispheric stroke: analysis of the lesion site by CT and MRI. *J Neurol Sci* 1996;137:47-56; and Sakakibara R et al. Micturitional disturbance and the pontine tegmental lesion: urodynamic and MRI analyses of vascular cases. *J Neurol Sci* 1996;141:105-10.

KEYPOINTS:

- Epidemiological studies of urinary incontinence following CVA indicate that urinary incontinence following a stroke is a specific indicator of poor prognosis.

urinary incontinence. From this, it seems lesion size is only a partial determinant of urinary dysfunction. In this study, no preponderance of laterality was demonstrated, whereas other studies have suggested urinary symptoms are more common in patients with right-sided lesions.

No statistically significant correlation could be demonstrated between any particular lesion site and urodynamic findings, but a significant correlation was found between the occurrence of a urinary disturbance and hemiparesis ($P < 0.05$) and a negative correlation with hemianopia ($P < 0.05$). Brain imaging techniques confirmed a more anterior location of brain lesions in the former group (Figure 4). These findings further supported the idea that lesions of the anteromedial frontal lobe and its descending pathway and the basal ganglia are mainly responsible for micturitional dysfunction in stroke patients.

Urinary retention was seen in the acute phase of illness in 6% and these patients initially had detrusor areflexia and a nonrelaxing sphincter, but subsequently developed detrusor hyperreflexia. Detrusor areflexia soon after stroke can be regarded as similar to the "shock phase" (Cases 1 and 2) which occurs after acute spinal injury.

Epidemiological studies of incontinence following CVA. Epidemiological studies of urinary incontinence following CVA indicate that urinary incontinence following a stroke is a specific indicator of poor prognosis. In acute brain disease, the occurrence of a voiding disorder may depend on the timing in relation to the stroke, and such symptoms tend to improve together with neurological signs.

Wade and Langton Hewer [9] analyzed the symptoms of 532 patients seen within 7 days of their stroke and found that the presence of urinary incontinence appeared to be a more powerful prognostic indicator for poor

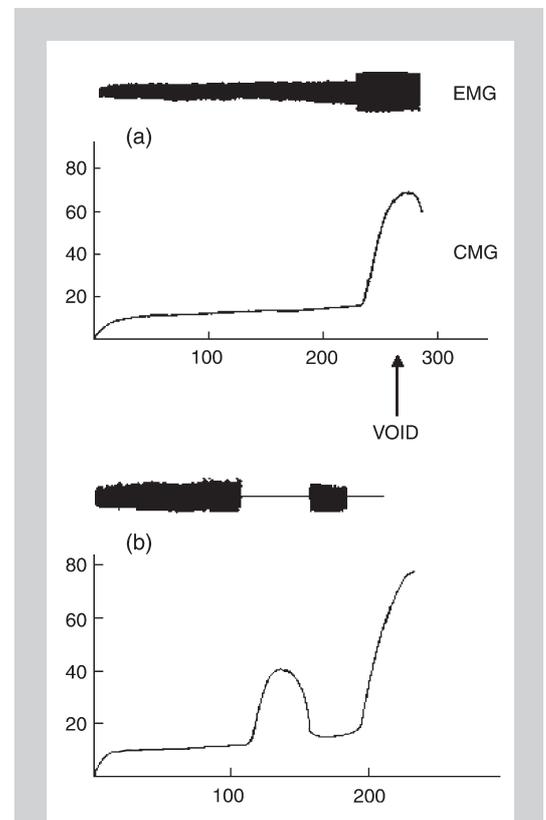


FIGURE 3 (a) Simultaneous sphincter EMG with cystometry demonstrating detrusor sphincter dyssynergia. The EMG and CMG traces are labeled. Volume infused (in ml) is recorded on the x-axis and subtracted detrusor pressure (in cm of water) is on the y-axis. (b) Simultaneous sphincter EMG with cystometry in a patient with involuntary detrusor contractions secondary to detrusor hyperreflexia demonstrating uninhibited sphincter relaxation.

KEYPOINTS:

- The role of diffuse brain disease in causing incontinence is not clear, although this is a problem of immense socio-economic importance because of the cost of caring for demented incontinent patients.



FIGURE 4 (a) Lesions on brain CT or MRI in patients with micturitional disturbance. Most of the patients had lesions on the anterior and medial surface of the frontal lobe, anterior edge of the paraventricular white matter, genu of the internal capsule, large lesion of the putamen and large lesion of the thalamus adjacent to or including the genu of the internal capsule. (b) Lesions on brain CT or MRI in patients without micturitional disturbance. Most of the patients had lesions of the occipital, temporal or parietal lobe, posterior lateral surface of the frontal lobe, crus posterius of the internal capsule and small lesion of the putamen or the thalamus.

From Sakakibara R et al. Micturitional disturbance after acute hemispheric stroke: analysis of the lesion site by CT and MRI. *J Neurol Sci* 1996;137:47-56.

survival and eventual functional dependence than was a depressed level of consciousness. These authors stress that they did not attempt to investigate the cause of incontinence and pointed out that many patients were immobile or aphasic. They suggested either incontinence was the result of a severe, general deficit rather than specific loss of function or that those who were incontinent may have been less motivated to remain continent and to recover loss of function. Other large studies have confirmed this observation. It seems likely that incontinence is a strong predicting factor for poor prognosis for a number of reasons: the same lesion might cause neurogenic bladder dysfunction and motor or cognitive

impairment; urinary incontinence may cause psychological problems or urinary continence may be an important factor in rehabilitation, gaining independence and quality of life.

Diffuse brain disease. The role of diffuse brain disease in causing incontinence is not clear, although this is a problem of immense socio-economic importance because of the cost of caring for demented incontinent patients. The onset of incontinence has been shown to bear a close relationship to the duration and stage of illness in patients with Alzheimer's disease. Urinary incontinence usually appears 3-8 years after onset of dementia, indicating no direct relation between these two abnormalities [10]. Nevertheless, in established

NEUROLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION

CASE 1

History: A 68-year-old, previously healthy woman was admitted to our hospital because of headache and acute-onset weakness of her left body.

Examination: Her blood pressure was high (180/105 mm Hg). Neurological examination showed that she was obtunded. She had left hemiplegia with decreased sensation for pin prick on the left side. She was also incontinent of urine. An indwelling catheter was inserted to facilitate bed-rest and to monitor urinary volume. Emergency CT and MRI scan revealed hemorrhage in the right putamen.

Management: She was administered an anti-hypertensive drug to reduce her blood pressure, and 600 ml of glycerol to reduce brain swelling. After 2 weeks, she regained consciousness and her left hemiplegia gradually recovered. The catheter was then removed. However, she was still in a state of urinary retention. Urodynamic studies revealed detrusor areflexia and nonrelaxing sphincter on voiding. Nursing staff performed intermittent catheterization to avoid bladder retention, and after 3 weeks, she gradually regained voluntary voiding without large residual amounts.

Comment: One to 4 weeks after acute stroke, either infarction or hemorrhage, detrusor areflexia with nonrelaxing sphincter can occur. This is regarded as the "shock phase" and is similar to that which occurs after acute spinal cord injury.

KEYPOINTS:

- The incidence of urinary dysfunction depends on the nature and extent of the brain disease, but has probably been underestimated.

Alzheimer's disease, both detrusor hyperreflexia and functional incontinence due to cognitive decline are not uncommon [11]. On the other hand, urinary urgency and urge incontinence may be the earliest and even sole initial symptom in vascular dementia [12]. Detrusor hyperreflexia and uninhibited sphincter relaxation are the main features in these patients. In diffuse Lewy body disease, urinary incontinence tends to appear with an intermediate period (2-4 years after onset of dementia) in between multiple cerebral infarction and Alzheimer's disease [10].

Efforts should be made to treat urinary incontinence in patients with diffuse brain disease, particularly in the early stages.

Conclusion. The incidence of urinary dysfunction depends on the nature and extent of the brain disease, but has probably been underestimated. There are a number of possible reasons for this: brain disease is likely to cause conditions of critical illness, such as disturbance of consciousness and other major neurological deficits. In addition, a bladder disorder may not be so apparent when it is of

gradual onset due to the slow progression of a causative lesion. Careful observation of urinary symptoms should therefore be made in these situations and a cortical cause of bladder dysfunction should be considered in a patient with urinary dysfunction of unknown etiology [13].

Brainstem. The brainstem tegmentum contains areas critical for micturition, the so-called "pontine micturition center" (PMC). This was first demonstrated by the experimental studies of Barrington in the cat which showed that the mechanisms for co-ordinating bladder and sphincter activity reside in the brainstem. Subsequent experimental studies have defined the brainstem activity involved in bladder storage and voiding in animals in great detail and shown that separate centers control voiding and storage. The recent positron emission tomography (PET) imaging studies [14] have confirmed that the neurophysiology of bladder control is essentially similar in humans.

Studies of patients with brainstem lesions. That a posterior fossa tumor can cause voiding dysfunction has long been known. However, little attention has been paid to this because acute brainstem lesions commonly cause a serious neurological state characterized by disturbed consciousness or respiratory arrest. Ueki found that in 152 patients with posterior fossa tumors, voiding difficulty occurred in 46 (30%) and urinary incontinence in three (1.9%) [3]. Looked at in greater detail, voiding difficulty occurred in 77.3% with lesions of pons, 66.7% with fourth ventricle, 40.9% with the midline of cerebellum, 24.2% with cerebellar hemisphere and 9.1% with cerebellopontine angle in posterior fossa tumors. Urinary retention may be an early feature in children with pontine gliomas.

An analysis of urinary symptoms of 39 patients who had had brainstem strokes showed dorsally situated lesions resulted in disturbance of micturition (Figure 5) [15]. Forty-nine percent of all the patients had urinary symptoms. Nocturnal urinary frequency and voiding difficulty occurred in 28%, urinary retention in 21% and urinary incontinence in 8% (Figure 1). The problems were more common in patients following hemorrhage, possibly because the damage was usually bilateral. MR scanning showed that the responsible lesions were in the pontine reticu-

lar nucleus and the reticular formation, adjacent to the medial parabrachial nucleus and the locus ceruleus. A correlation was found with urinary symptoms and sensory disturbance, abnormal eye movement and with inco-ordination. Urodynamics in 11 symptomatic patients showed detrusor hyperreflexia in eight (73%), low compliance bladder in one (9%), detrusor areflexia in three (27%) (3 months, 6 months and 3 years after the occurrence), nonrelaxing sphincter on voiding in five (45%) and uninhibited sphincter relaxation in three (27%). Three asymptomatic patients had normal findings.

Further single-case histories have reported patients with various pontine lesions and urinary retention due to detrusor areflexia [4]. The proximity of the medial longitudinal fasciculus in the dorsal pons to the presumed pontine micturition center means that a disorder of eye movements, such as an internuclear ophthalmoplegia, is highly likely in patients with pontine pathology causing a voiding disorder (Case 3).

CEREBRAL CONTROL OF DEFECATION

Very much less has been written about cerebral control of the bowel than of the bladder. Andrew and Nathan stated that, in general, defecation was affected much less often than micturition [1]. Three out of the 10 patients with brain tumors studied by them had fecal frequency, incontinence and constipation. One of two patients after aneurysm surgery had fecal incontinence on occasion with diarrhea. Two of four brain-injured cases had fecal incontinence without warning and one reported not to feel feces passing, while the other patient had fecal incontinence only when asleep. Rectal examination showed that these patients had full voluntary control of their levator ani muscles and of the external anal sphincter. Some

patients following leukotomy were incontinent of feces and, in such cases, this was always accompanied by urinary dysfunction. Other papers which describe disorders of bladder function in frontal lobe disease mostly do not mention the patients' bowel symptoms.

Weber et al. described the results of similar recordings in seven patients with frontal lobe injury [16]. Anorectal manometric recordings and urodynamic investigations were carried out in seven patients who had either right or left or bilateral frontal lobe injury. Two patients lacked sensation of bladder filling and

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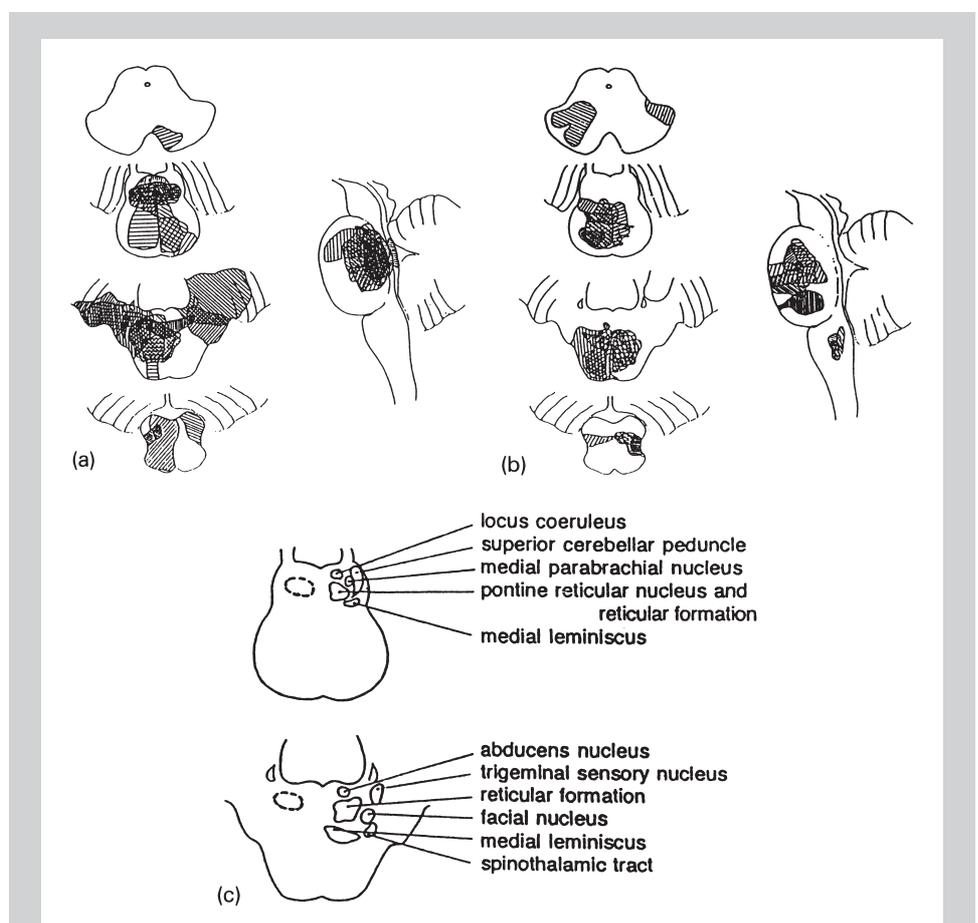


FIGURE 5 (a) Lesions on brain MRI in patients with micturitional disturbance. Most of the patients had lesions of the pontine tegmentum and the dorsal medulla. (b) Lesions on brain MRI in patients without micturitional disturbance. Most of the patients had lesions of the pontine basis and the lateral medulla. (c) Responsible sites of lesions for micturitional disturbance. The regions seemed to be located in the dorsolateral pons, including the pontine reticular nucleus and the reticular formation, adjacent to the medial parabrachial nucleus and the locus ceruleus.

From Sakakibara R et al. Micturitional disturbance and the pontine tegmental lesion: urodynamic and MRI analyses of vascular cases. *J Neurol Sci* 1996;141:105-10.

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KEYPOINTS:

- The authors concluded that the results favor the pons as the possible level of supraspinal control of colonic and anorectal motility.

two other patients had increased perception threshold of rectal distension, five cases had uninhibited detrusor hyperreflexia or spontaneous rectal contractions and one patient had lost reflex micturition and the rectoanal inhibitory reflex. The authors concluded that the medial prefrontal area and the anterior cingulate gyrus were involved in neurological control of anorectal motility as for bladder function, but the lack of correlation between urinary and anorectal abnormality in individual cases suggests that these functions depend on distinct areas of the frontal lobes.

A study using PET examined the areas of brain activation after rectal distension in both healthy control subjects and patients with irritable bowel syndrome [17]. A balloon catheter was inserted into the rectum, and different degrees of distension were produced at timed intervals. Differences in response were found between the control subjects and patients both on maximal filling and in anticipation of filling, but in healthy subjects, activation of the anterior cingulate cortex could be demonstrated on filling only to pressures that were perceived as painful.

Weber et al. [18] described the results of measuring transit times of radio-opaque markers, anorectal manometric recordings and urodynamic investigations in three patients with pontine vascular lesions. All three patients had severe constipation and distended abdomens. One patient had right colonic inertia and lost the rectoanal inhibitory reflex. The other two had prolonged transit times of the left colon. The authors concluded that the results favor the pons as the possible level of supraspinal control of colonic and anorectal motility. Uro-

dynamic studies showed large postmicturition residuals of 100-400 ml in two patients, detrusor hyperreflexia in two patients and detrusor-sphincter dyssynergia in two patients.

Experimental studies have suggested that Barrington's nucleus (PMC) also plays an important role in colonic motility and defecation (the pontine defecation center) [19]. Other sites related to defecation include the hypothalamo-thalamic area, the limbic structures, including the medial frontal lobe. Chemical stimulation of Barrington's nucleus in experimental animals results in colonic motility. These areas may also account for the clinical fecal dysfunction in some cases.

CEREBRAL CONTROL OF SEXUAL FUNCTION

In experimental animals, the deep anterior midline structures which form the limbic system have been shown to be important in determining sexual responses and the medial preoptic-anterior hypothalamic area has been shown to be an integrating area (see Chapter 4). Electrical stimulation of the hypothalamic and limbic pathways in experimental animals results in erection, although a review of reports of brain stimulation in awake man during stereotaxic neurosurgery concluded there had been no reliable instances of erection. However, damage to the dorsolateral hypothalamic nuclei during stereotaxic surgery for the relief of myoclonus, may cause loss of libido and erectile dysfunction, whereas hypersexuality occurs as a consequence of septal injury.

Little is known about the contribution of the cortex to sexual function, although it is thought that cerebral processing determines

CASE 2

History: A 62-year-old woman presented with vomiting and vertigo and was admitted to hospital. She had a 7-year history of hypertension, but her blood pressure was well controlled by medication.

Examination: Neurological examination revealed she was alert and cooperative. She had counter-clockwise rotatory nystagmus, mild right deafness and arm deviation to the right side. In addition, she also had mild ataxia of her right arm, very mild right facial paresis, mild right abducent nerve palsy and voiding difficulty. MRI scan revealed a lacunar infarction in the right dorsolateral pons.

Management: After a week her neurological signs started to improve. Urodynamic studies revealed detrusor areflexia on attempts to void, which also gradually ameliorated.

Comment: This patient had a vascular lesion in the right dorsolateral pons, which probably included locus, the pontine micturition center (PMC).

libido and desire: the ability to effect a sexual response is determined by spinal, autonomic reflexes. Libido is hormone-dependent with a major hypothalamic component probably mediated by D1 receptors in the medial preoptic area. Prolactin acts centrally on the dopaminergic pathway and loss of libido may be the earliest symptom of a prolactin-producing pituitary tumor [20].

A study using SPET scanning, showed an increase in activity in the right prefrontal cortex during ejaculation in healthy male subjects [21]. The frontotemporal regions, particularly the right hemisphere, are important in sexual activity and these regions of the brain can be involved by pathology which causes epilepsy, by trauma, tumors, cerebrovascular disease, or encephalitis.

Temporal lobe epilepsy may cause sexual apathy of which patients rarely complain [22], although various sexual perversions and occasionally hypersexuality have also been described. Studies comparing sexual dysfunction between groups of patients with generalized epilepsy and those with focal temporal lobe epilepsy, provide evidence that the deficit is a function of the specific area of brain involvement rather than a consequence of epilepsy, psychosocial factors or anti-epileptic medication. Some investigators have suggested that the hyposexuality of temporal lobe epilepsy is due to a subclinical hypothalamic-pituitary dysfunction.

Erectile dysfunction with preserved libido of which a patient therefore complains, can also occur in men with temporal lobe damage and epilepsy and may be characterized by loss of nocturnal penile tumescence. Surgery for epilepsy rarely restores function, although a survey of operated patients showed a higher level of satisfaction with sexual function amongst those who were seizure-free.

Sexual “problems” are not uncommon following head injury, particularly if there has been cognitive damage or a personality change and appear to contribute significantly to the long-term failure of relationships which predated the injury. A study of people who had been admitted to hospital for a minimum of 24 hours after a closed head injury found significant sexual dysfunction in 50% over a 15-year period. The most common complaint was of infrequency [23] and there is some evidence that hypogonadotropic hypogonadism may be a significant factor. Sexually demanding behavior combined with a loss of empathic sensitivity can also occur after head injury.

TREATMENT

The approach to treatment is no different to that of other neurological causes of these symptoms. Such treatments are discussed in Chapter 2 (urinary incontinence and retention); Chapter 3 (constipation and fecal incontinence) and Chapter 4 (male and female sexual dysfunction).

KEYPOINTS:

- Little is known about the contribution of the cortex to sexual function although it is thought that cerebral processing determines libido and desire: the ability to effect a sexual response is determined by spinal, autonomic reflexes.
- Temporal lobe epilepsy may cause sexual apathy of which patients rarely complain.

CASE-ORIENTED MULTIPLE CHOICE QUESTIONS

- ▶ A 68-year-old woman presented with an acute onset of left hemiplegia and an ipsilateral decrease in sharp sensation. She had an indwelling catheter inserted. A CT scan identified a hemorrhage in the right putamen. After 2 weeks the catheter was removed, but the patient remained in urinary retention.

What was the likely cause of this patient’s urinary retention?

- A. Detrusor areflexia and detrusor-sphincter dyssynergia.
- B. Uninhibited sphincter relaxation.
- C. Urethral stricture.
- D. Severe constipation.
- E. Urinary tract infection.

Multiple choice questions
continued ▶

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Multiple choice questions
continued ►

The answer is A. Up to 1 month following an acute stroke, detrusor areflexia with nonrelaxing sphincter can occur. This time period is similar to the “shock phase” which follows acute spinal injury.

- A 50-year-old man had a right thalamic hemorrhage. He was initially in urinary retention with detrusor areflexia and DSD on EMG cystometry. He was taught clean intermittent self-catheterization (CISC). 2 months later, he developed urge urinary incontinence.

What is the likely cause of his new bladder symptoms?

- A. Detrusor areflexia.
- B. Detrusor hyperreflexia.
- C. Low compliance.
- D. Genuine stress incontinence.
- E. Detrusor instability.

The answer is B. After the acute illness (shock phase) in brain lesions, the patient’s bladder symptoms could either resolve or develop into detrusor hyperreflexia.

- A 71-year-old, previously healthy woman had a new onset of nocturia and urge urinary incontinence. On examination, she had a mild short-stepping, festinating gait. She also complained of mild difficulty on drinking water and had an absent soft palate reflex. Brain MRI identified moderate leukoaraiosis.

What would be the expected cystometric finding?

- A. Detrusor areflexia.
- B. Detrusor hyperreflexia.
- C. Low compliance.
- D. Genuine stress incontinence.
- E. Absent sensation of filling.

The answer is B. Leukoaraiosis is a cause of detrusor hyperreflexia.

- A 38-year-old woman presented with an acute onset of headache, vertigo, diplopia, difficulty using her right arm and voiding dysfunction. She was diagnosed with right MLF (medial longitudinal fasciculus) syndrome and right-sided cerebellar ataxia. An MRI scan identified a hemorrhage from a cavernous angioma of the pontine tegmentum.

What was the basis of her voiding dysfunction?

- A. Detrusor areflexia.
- B. Low compliance.
- C. Detrusor hyperreflexia.
- D. Detrusor instability.
- E. Detrusor-sphincter dyssynergia.

The answer is A.

- What is the likely location of the lesion in the patient described above?

- A. Pyramidal tract.
- B. Spinothalamic tract.

- C. Middle cerebellar peduncle.
- D. Medial pontine micturition center.
- E. Paraventricular nucleus.

The answer is D. The medial pontine micturition center is crucial for micturition particularly of voiding phase.

- ▶ A 45-year-old man presented with an acute onset of confusion and left hemiparesis predominantly affecting the leg. Following recovery, he had forced use of his right hand (align hand sign) that his left hand tended to restrain. In addition, he had urge urinary and fecal incontinence.

Which site of lesion is expected on brain MRI scan?

- A. Medial frontal cortex.
- B. Lateral frontal cortex.
- C. Parietal cortex.
- D. Occipital cortex.
- E. Lateral pontine micturition center.

The answer is A. Lesions in the medial frontal cortex result in various neurological and neuropsychiatric signs, including the align hand sign. The medial frontal cortex also contains the frontal micturition center, and lesions in this area can present with detrusor hyperreflexia and/or detrusor areflexia.

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