CHAPTER 3

Bladder Function in Normal Micturition

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Introduction

In spite of much detailed research, micturition is extremely poorly understood, and working hypotheses are usually the most that can be offered as explanations of common phenomena. Teleologically, the basic function of the bladder may be thought of as to discharge urine clear of the skin. This is achieved by a water-tight build-up, ending in a forceful discharge directed away from the body by the adoption of a micturition posture. In man, performance is complicated, as discharge of urine must be in a socially acceptable site—which has to be reached before the onset of micturition —and, in addition, micturition posturing has to include avoiding wetting the clothes. To allow for preparation for micturition, a period of voluntary postponement is therefore essential, during which considerable conscious cortical activity and somatic abilities are required.

In summary, sophisticated mature bladder function can be represented as a cycle (Fig. 1), made up of a series of phases: *filling*, *desire to void*, *postponement*, *initiation of 'sphincter' relaxation and bladder contraction*, *maintenance of both these until the bladder is empty*, *filling*, *etc.* To make use of the postponement phase, three abilities are required: to appreciate what is a socially acceptable micturition site, to get there, and to adopt a micturition posture. Each phase, and each requirement in the postponement phase, has its own disorders which may occur alone or in various combinations.

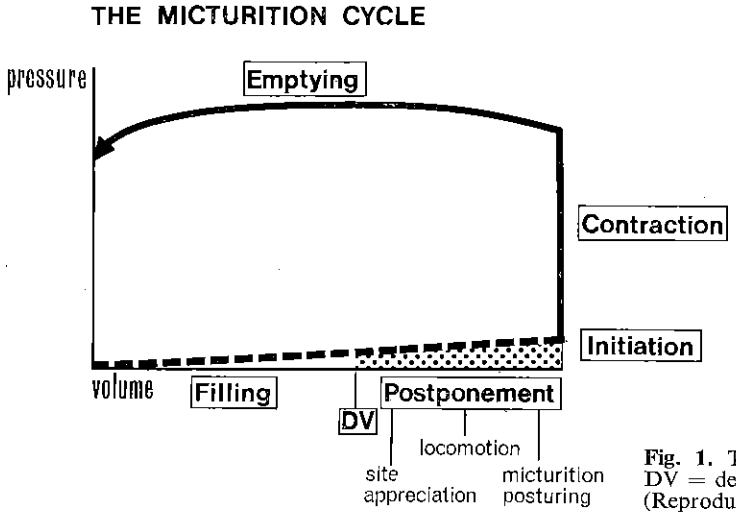

Fig. 1. The micturition cycle. DV = desire to void. (Reproduced from Yeates 1972.)

The Filling Phase

When the bladder fills at a physiological rate, there is only a very slight rise in intravesical pressure during filling, the pressure gradient being about 1 in 50. This gradient is taken to indicate bladder tone. The bladder tone is not under the direct control of the nervous system, being governed by the physical properties of the bladder wall itself; spinal anaesthesia eliminates only the major contractions (Tang and Ruch 1955). Increases in bladder tone occur in conditions where the physical state of the bladder wall is altered, *e.g.* when the bladder has been kept empty. Although changes in bladder tone are not primarily neurogenic, the tone may be secondarily affected by neurological conditions, *e.g.* (i) by sensory lesions, which are associated with bladder over-distension and decreased tone, or (ii) by lesions which increase urethral resistance and are associated with the production of bladder hypertrophy and increased tone.

Continence During Filling

During the filling phase, continence must be preserved in all positions and during all activities, by the resistance of the bladder outlet always being kept greater than the intravesical pressure. In quiet recumbency, the involuntary muscle and elastic tissue of the bladder neck and posterior urethra provide adequate resistance to prevent leakage (Lapides 1961). Rises in the intravesical pressure on standing (due largely to the pressure of the abdominal contents on the pelvic organs), or on raising the general intra-abdominal pressure by straining, are neutralised by a stretch response of the pelvic floor muscles. This muscular response raises the bladder neck, elongates and narrows the urethra, and increases the urethral resistance (Woodburne 1961), possibly by a spiral arrangement of the involuntary fibres (Fig. 2).

Bladder neck resection does not result in incontinence, even if combined with pudendal anaesthesia which paralyses the external sphincter, because the levator ani is still active. On the other hand, a combination of bladder neck resection and resection of posterior urethral valves commonly results in imperfect control, due to previous over-stretching of the urethral component of the pelvic floor-urethral complex. For incontinence to occur, there must be weakness both of the bladder neck and of the pelvic floor musculature-urethral complex. (This—'passive' or 'stress'—incontinence will not be considered further as it is not relevant to the incontinence in enuresis which is manifestly 'active', *i.e.* due to bladder contractions.)

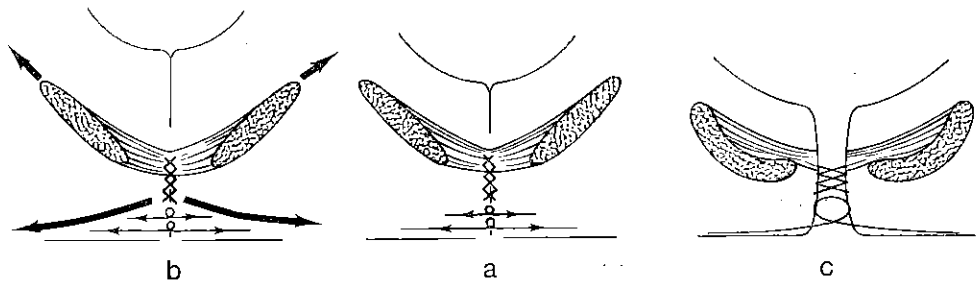

Fig. 2. Bladder outlet resistance: (a) at rest; (b) on raising intra-abdominal pressure; and (c) during micturition.

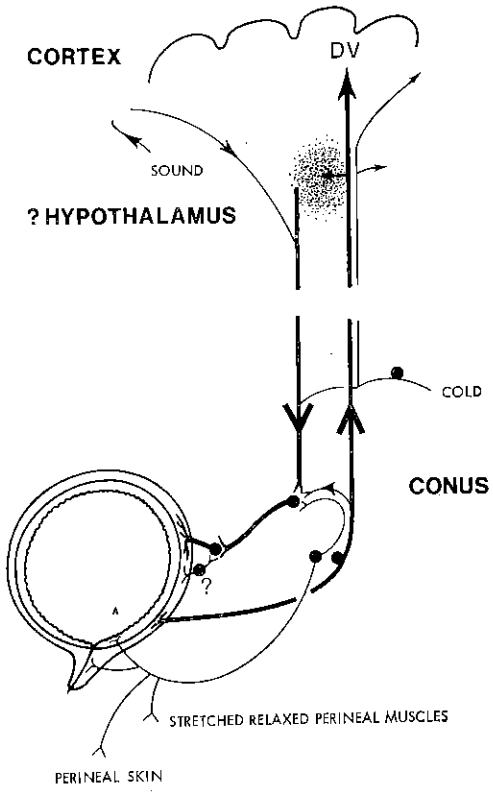

Fig. 3. Sensory components concerned in the desire to micturate. DV = desire to void. (Reproduced from Yeates 1972.)

The Desire to Void

The sensation of attainment of a moderate degree of bladder filling obviously arises essentially in the bladder itself, as a consequence of stretching of its wall, the impulses passing via the sacral segments 2, 3, and 4. This sensation of the 'desire to void' is not the same in different individuals. There are at least three normal patterns of sensation in the later stages of bladder filling: (i) perineal, becoming anterior urethral; (ii) retropubic becoming urethral; or (iii) urethral, becoming retropubic. However, in each type of prolonged postponement of micturition, the final sensation is suprapubic discomfort of colicy type (Yeates 1950). In subjects with an apparently normal nervous system, the sensation of the desire to void may be produced by stimulation of the *bladder muscle*, the *bladder or urethral mucosa*, the *perineal skin*, and probably by *stretching of the muscles of the pelvic floor* (Fig. 3).

Impulses Arising from the Bladder Muscle

(1) Talaat (1937) and Iggo (1955) showed in the dog and cat bladder, respectively, how afferent impulses in a single nerve fibre are set off only slightly by slow filling, more by very rapid filling, and most of all by bladder contractions.

(2) The desire to void can be produced by bladder distension without the presence of a contraction wave during ganglion blockade (Nesbit *et al.* 1947), and sudden

pressure on a moderately distended bladder similarly produces an immediate desire to void, without necessarily initiating a contraction wave.

(3) Contraction waves detectable on cystometry correspond to the desire to void (Plum 1960).

(4) Rapid filling at cystometry or by diuresis results in an earlier, more rapid contraction wave and an associated increased desire to void.

(5) Induction of increased bladder tone by keeping the bladder empty reduces the volume at which contraction waves and the desire to void arise, *i.e.* the micturition threshold is lowered.

(6) Cholinergic drugs produce an urgent desire to void, associated with a contraction wave.

(7) Although the small rhythmic contraction waves during filling are not the result of stimulation by the nervous system, and persist in spite of the use of anti-cholinergic agents (Tang and Ruch 1955, Plum 1960), larger contraction waves and the desire to void are abolished or delayed by these drugs.

(8) The development of bladder hypertrophy in response to bladder outlet obstruction is associated with an increase in bladder tone and the onset of more powerful bladder contraction waves at lower volumes, giving the desire to void the quality of urgency. The urgency is presumably due to hypertonia of the bladder muscle when constantly acting against increased resistance, since, after removal of the obstruction, the urgency is relieved long before the bladder hypertrophy disappears.

(9) Atrophy of the bladder muscle as a result of chronic over-stretching is associated with a decrease or absence of the desire to void.

In summary, the primary mechanism in the production of the desire to void seems to be the rate of increase in bladder wall tension; in particular, it is increased by bladder contraction in response to bladder distension. The effects of anti-cholinergic drugs in decreasing the desire to void suggest that the waves normally concerned with an urgent desire to void are of neurological reflex origin.

Other Afferent Stimuli Concerned With the Desire to Void

(1) *Afferents from the bladder mucosa, urethra and perineal skin.* Nathan (1964) has shown that anaesthetization of the perineal skin increases bladder capacity. This also occurs following local anaesthetization of the urethra or bladder mucosa. In contrast, stimulation of these areas, by bacterial or chemical irritation or the application of cold water, produces a reflex contraction wave, with an accompanying sense of urgency, at a lower level of bladder distension than was necessary previously.

(2) *The vertical posture.* The vertical posture may initiate or greatly increase the desire to void. This is probably primarily because assuming the vertical posture causes the abdominal contents to distort the bladder surface, and so stretch the bladder muscle. In addition, however, since elevation of the perineum (and the bladder neck) has suppressing effects on bladder activity (Vincent 1966), and electrical stimulation of the pelvic floor muscles relieves many cases of urgency of micturition (Alexander and Rowan 1968), it appears that either stimuli arising from passive stretching of these muscles, or the associated descent of the bladder neck, at least facilitate the production of the desire to void when the erect posture is assumed.

(3) *The sound of running water*. The sound of running water undoubtedly facilitates the micturition reflex, its effect being most marked when combined with an increase in other—sometimes subliminal—stimuli concerned with the desire to void.

(4) *Anxiety*. Anxiety can suppress (Plum 1960) or exaggerate (Straub *et al.* 1950) bladder contraction waves. The second effect is known to examination candidates and aircraft pilots on military missions, who may have frequency, urgency and even incontinence.

Summary of Factors which Produce the Desire to Void

The desire to micturate—and the symptom of urgency—is produced by an increase in tension in the bladder wall, accentuated by bladder contractions. These sensations are essentially initiated by bladder distension, but other impulses via sacral segments 2, 3, and 4 (from the bladder, bladder neck, urethral mucosa, pelvic floor muscles, and perineal skin) summate with those arising in the tensed bladder wall muscle. The contraction waves and resulting sensations are easily affected in either direction by cortical activities.

Transmission of the Sensory Stimuli

According to Nathan and Smith (1958), the afferent pathway in the spinal cord lies near the central canal on the equatorial plane.

Appreciation of the Sensory Stimuli

Reception, perception and analysis of the stimuli, and subsequent appreciation of the desire to void, obviously depend on cortical function. Here the sensation presumably competes for recognition and response with other sources of input (including preoccupation with other interests and activities).

Desire to Void During Sleep

Manifestly, should the usual stimuli be produced during sleep, the normal individual is awakened and becomes aware of the desire to void.

Suppression of Bladder Activity

It would seem reasonable to assume that the stimuli which give rise to the desire to void are the same as those which, through the central nervous system at various levels, tend to set off reflex bladder contractions. In man with an intact nervous system, all contraction waves are suppressed during filling (Higgins *et al.* 1951, Hodgkinson *et al.* 1963). The effects of lesions in the central nervous system—both clinical and experimental—on bladder excitability clearly show that reflex bladder contractions can be mediated by the conus medullaris. Following recovery from spinal shock, the isolated conus is associated with extreme bladder irritability, there being manifestly no inhibition controlling the activity of the reflex arc.

Clinical and, particularly, experimental work have shown almost alternating facilitatory and inhibitory effects on bladder activity at successive levels up to as high as above the hypothalamus. For example, in the cat (Tang 1955) the anterior pons is excitatory, the mid-brain inhibitory, the posterior hypothalamus excitatory, and the

cerebrum inhibitory to the bladder reflexes. Exclusion of inhibitory levels releases the reflex below, the effect being illustrated by the early onset of bladder contraction waves ('uninhibited' contractions), which, if powerful enough, may produce micturition. Less powerful contractions are easily detectable on cystometry, and usually produce the desire to micturate. Where this desire does not occur, there is, presumably, a defect of sensory conduction or of cortical appreciation.

Correlation of the effects of experimental lesions and diseases of the nervous system on bladder function is usually difficult or impossible, owing partly to species variation and partly to the indefinite location of pathological states. Murnaghan (1961) investigated 29 patients (11 female, 18 male) with Parkinsonism, of whom 25 per cent presented uninhibited types of neurogenic bladder disturbance. The effects of stereotaxic surgery on the thalamus, the posterior limb of the internal capsule, and the globus pallidus were investigated. These effects were variable, but clearly showed that the basal ganglia are concerned in unconscious suppression of bladder activity. The anticipated uninhibited contraction waves which sometimes result from thalamotomy produced urgency, but they were suppressable by reassurance, presumably by cortical activity.

Andrew and Nathan (1965) showed that disorders of the anterior part of the frontal lobe near the mid-line, the anterior part of the cingulate gyrus, and the rostral

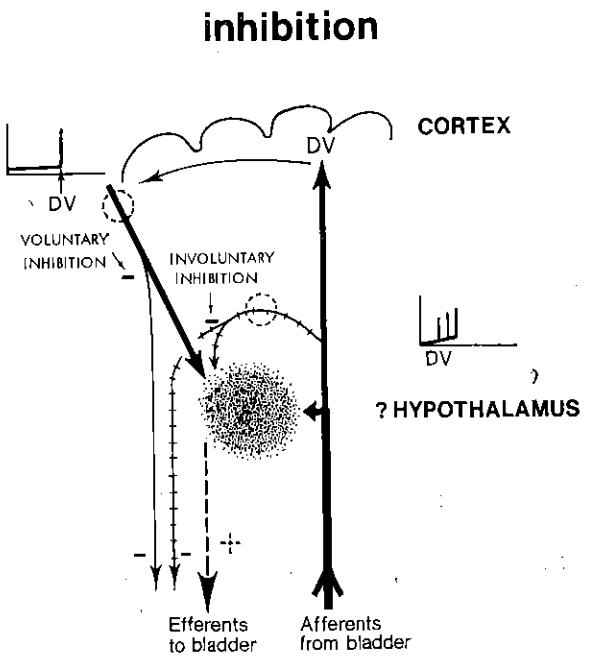

Fig. 4. Working concept of control of the micturition 'centre'. The dotted circles indicate sites of deficiency in inhibition, with corresponding diagrammatic cystometrograms. The two left-hand tracts inhibit lower reflex arcs. The main motor descending tract (+) is shown inactive (– – – –) due to inhibition. DV = desire to void. (Reproduced from Yeates 1972.)

part of the hypothalamus resulted in urgency and impairment of the ability to suppress reflex detrusor contractions. Brocklehurst and Dillane (1966) further illustrated the effects of cerebral lesions on bladder irritability. Lesions of the dominant hemisphere particularly resulted in frequency, urgency and a decrease in bladder capacity.

Summary (Fig. 4)

There is unconscious inhibition of bladder activity up to subcortical levels, the cortex normally being able to reinforce unconscious inhibition, and so suppress bladder contractions.

High or low bladder responsiveness therefore depends on the relation of the *sum of appropriate input impulses via sacral segments* 2, 3, *and* 4 to the *sum of unconscious and conscious inhibition of central origin.*

Micturition

It is usually thought that micturition is normally initiated by voluntary removal of its inhibition. Micturition then results from inhibition of the tone of the striated pelvic floor muscles (Fig. 2*c*) (the electromyogram of the latter becoming silent), followed immediately by descent of the bladder neck and by a sustained bladder contraction with opening of the bladder outlet. It seems that co-ordinated micturition requires mechanisms originating at least as high as the hypothalamus. This view is supported by many observations, including the report of Murnaghan (1961) that lesions of the basal ganglia were not associated with spasm of the external sphincter, unlike, as is well known, lesions at lower levels in the neural axis. But even when there is little bladder distension and hence few afferent stimuli, an evacuating bladder contraction can be actively induced by will (Lapides *et al.* 1957) or by psychological stress.

Relaxation of the pelvic floor is certainly essential to reduce urethral resistance during micturition, but it is not agreed whether this—with the resultant descent of the bladder neck—is the usual mechanism initiating it. The work of Vincent (1966) referred to above certainly supports this idea. During emptying there is a rise in intravesical pressure which remains fairly constant until the bladder is empty, following which there is usually an after-contraction of the bladder muscle, the significance of which is unknown.

Development of Bladder Control

For about the first six months of life, the bladder responds to distension by immediate evacuation; the stream is forceful and emptying complete—the appearances are those of a normal co-ordinated act of micturition, suggesting that the mechanism operates through centres at about the level of the hypothalamus. During the following two years, there is a general progressive lessening of bladder responsiveness, presumably due to the development of unconscious inhibition. Cystometry at increasing ages demonstrates contraction waves being progressively suppressed (Higgins *et al.* 1951).

Some time between 18 and 30 months, the infant becomes able to convey to its mother that it is about to micturate (indicating the development of perception of

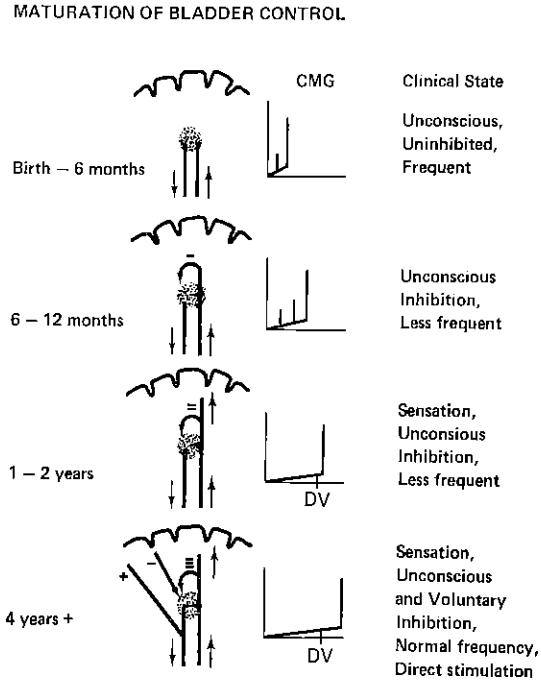

Fig. 5. Diagrammatic representation of progressive development of cerebral functions concerned in bladder control. Inhibitory tracts to lower reflex arcs are omitted, and lowest diagram shows direct voluntary initiation of bladder contraction—otherwise tracts correspond to Fig 4.

bladder sensation), and somewhat later it can consciously inhibit micturition for increasing lengths of time (Apley and Mac Keith 1962).

Maturation of the mechanisms necessary for the control of micturition (Fig. 5) therefore consists in:

(1) reduction of activity of the micturition reflex;

(2) the development of consciousness of bladder distension;

(3) the development of conscious ability to postpone or to initiate micturition as required (Yeates 1965).

During this normal development of the control of micturition, there is, before control is reliable, a phase in which incontinence may be precipitated by any condition which either increases the sensory input (*e.g.* severe cystitis), or reduces inhibition (*e.g.* psychological stresses), or reduces appreciation of bladder sensation (*e.g.* pre-occupation with other matters, or deep sleep after exhaustion).

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