The Pelvic Floor: Neurocontrol and Functional Concepts

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Learning Objectives

- The pelvic floor is a musculo-elastic structure that, in women, includes the vagina as a central elastic structure.
- Impaired pelvic floor elasticity prevents normal muscle anchoring, causing muscle weakness and functional disorder, such as pain, urgency, and incontinence of urine or faeces.
- The motor and sensory innervation of the pelvic floor is critically important for continence and evacuation, through its connexions to spinal cord and brainstem neural control systems.
- In healthy adults higher-level control systems override these reflex systems to establish voluntary evacuation.
- Pelvic floor dysfunction in women is often initiated by obstetric damage to the pelvic floor muscles and ligaments. In extreme cases, direct anal sphincter tears are a marker of pelvic floor damage. When the pelvic floor is incompetent, its motor and sensory innervation is often progressively damaged by recurrent stretching during straining at stool.
- Pelvic floor repair procedures should be designed to improve functional ligamentous elasticity: this can be effective even when there is established muscle and nerve damage.

4.1 Introduction

The pelvic floor is a holistic functional entity, concerned with urinary and faecal continence and voiding, and sexual function. In women there are specialised features allowing childbirth, always a natural process but nonetheless a function that is associated with risk for damage to both the anterior and posterior components of the pelvic floor [1–4], including its nerve supply [5]. Sometimes childbirth leads to urinary or faecal incontinence and organ prolapse as delayed, chronic problems [6]. In everyday life urinary and faecal storage (continence) and voiding (micturition and defaecation) are under voluntary control, although subject to stimulus in relation to sensory input to the central nervous system. The central nervous system (CNS) control systems for these functions mature during childhood in relation to the imposed norms of society that require voiding at appropriate times and places [7]. It is therefore necessary for the bladder and rectum to act as storage receptacles until voiding is appropriate and possible. Consequently, for most of the time, the pelvic floor maintains bladder and bowel in a continent, storage mode [7]. As in most neurocontrol systems, there are several levels of control circuits, following a Jacksonian system of progressively higher levels of awareness and control. All of these levels in the control system are modulated by sensory afferent and descending motor neural command systems [8]. The systems controlling bowel and bladder are analogous to each other although separately ‘wired’.

4.2 The Urinary and Recto-Anal Systems

The bladder wall consists of smooth muscle innervated by parasympathetic motor nerve fibres. This parasympathetic innervation of the detrusor muscle is cholinergic, dependent on muscarinic motor endings on smooth muscle fibres [9]. These parasympathetic nerve fibres are postganglionic, derived from pelvic parasympathetic ganglia that are themselves innervated by preganglionic fibres originating

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in the intermediolateral columns of the lumbosacral cord. Muscle fibres in the bladder base and in the internal urethral sphincter are innervated by sympathetic nerve fibres, also originating in the intermediolateral cell columns of the spinal cord, but using norepinephrine as transmitter [6]. Relaxation of the internal urethral sphincter is dependent, in part, on sympathetic innervation of the muscle, utilising beta-adrenergic parasympathetic efferents that release nitric oxide as transmitter. Although it is widely believed that urinary continence is achieved by low resting tone in the bladder detrusor muscle, and tonic contraction of the internal urethral sphincter, this concept is an oversimplification. Circumferential contraction around a narrow tube, such as the urethra, is not, in general, an effective sphincteric mechanism. A more effective mechanism utilises kinking of the urethra, due to a backward muscular vector pulling the urethra posteriorly and causing a kink, thus obstructing any urine flow. This is achieved by contraction of the posterior muscle vectors in a backward/downward direction, i.e. by the levator plate and the conjoint longitudinal muscle of the anus (Fig. 4.1), a muscle grouping that has been shown to be in a state of continuous mild tonic contractile activity in the default storage mode [10]. The pubococcygeus muscle provides an opposing force, centered on the urethra. Mild resting contraction of the puborectalis muscle is a major factor in the maintenance of faecal continence, generating tension and kinking of the recto-anal junction by opposing muscle forces [11].

In addition to sympathetic motor innervation of the base of the bladder and internal urinary sphincter muscle, there is an extensive sensory innervation of the bladder wall and mucosa [6]. This consists of mechanoreceptors, sensitive to stretch and distension, and other receptors that signal inflammatory change, including pain. The receptors responsible for these sensations are complex and interdependent. The main sensitive area in the bladder is at the bladder base in the trigone region and in the immediately adjacent proximal urethra, in the urinary sphincter region. There are similar anatomical specialisations in the anal canal [12]. Slight relaxation of the internal anal sphincter allows fractional extrusion of faecal content into the proximal anal canal, inducing the 'sampling reflex' [13], that acts as a potent stimulus which, unless voluntarily suppressed, leads to the initiation of defaecation. Similarly, the increasing sense of urinary urgency felt when the bladder is full, particularly in response to a change of posture, such as standing, or coughing, reflects the passage of small quantities of urine into the proximal urethra within the urinary sphincter pressure zone, initiating an urgent voiding response. The external urinary sphincter is innervated by somatic efferent motor fibres from the S2/S3 spinal segments. This voluntary striated sphincter muscle does not totally encircle the urethra

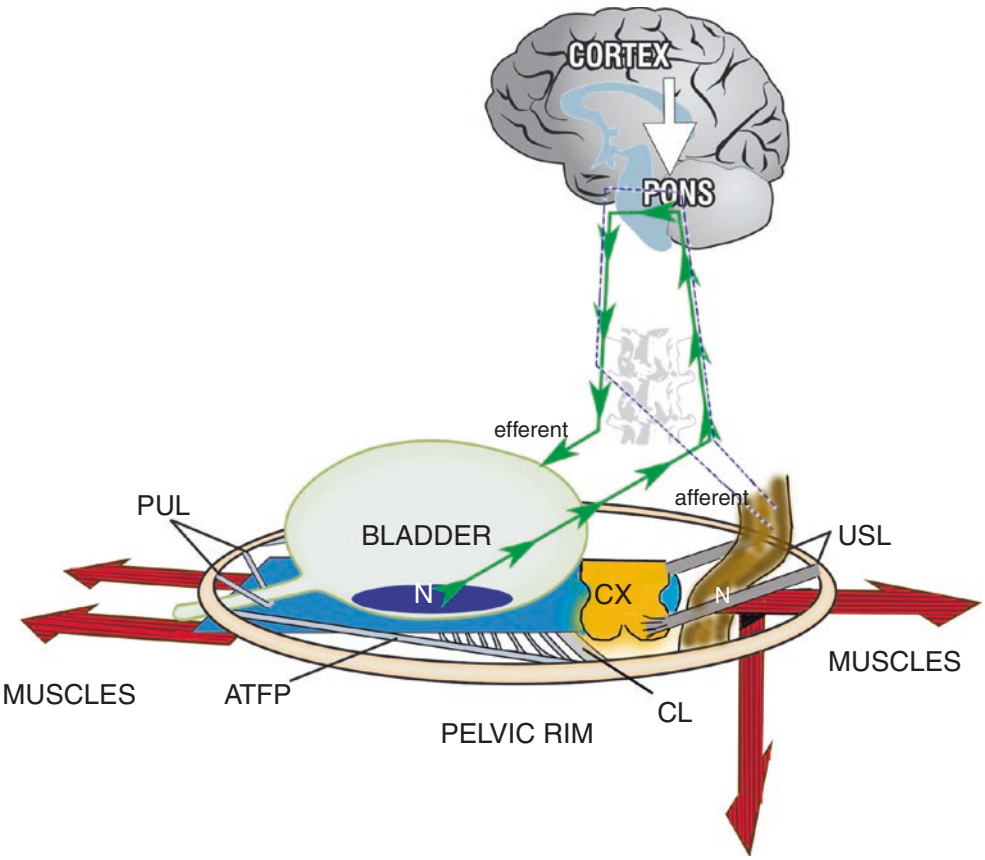

Fig. 4.1 Central and peripheral control of bladder and bowel. Schematic 3D sagittal view. System in default storage mode. Three directional muscle forces PCM (pubococcygeus), LP (levator plate), and conjoint longitudinal muscle of the anus (LMA) tense the organs bidirectionally against the suspensory ligaments PUL (pubourethral), CL (cardinal), and USL (uterosacral) to prevent activation of micturition and defaecation responses. Green arrows, neurological pathways; white arrow, central cortical control via the closure reflex. *ATFP* arcus tendineus fascia pelvis, *CX* cervix. N, sensory receptors in bladder trigone

and so is incapable of completely occluding it when contracted [14]. Like the external anal sphincter in faecal continence, the striated external urinary sphincter, or its smooth muscle counterpart, is not the major muscle of urinary continence.

4.3 Urinary and Faecal Storage and Voiding

Voluntary urinary voiding occurs in response to increasing pressure and volume within the bladder, causing excitation of afferents from bladder trigone sensory receptors, and also in response to subtle passage of urine into the upper urethra—the equivalent to the sampling reflex within the anal canal [15]. In babies and young children, or in spinal cord injury above the sacral level, the response to this afferent information, signalling the immediate need to void urine, is a switch in equilibrium from storage to voiding.

4.3.1 Bladder Equilibrium

At the most caudal, and unconscious, level, the change in command from default storage to voiding occurs at the Onuf urinary and faecal sphincter spinal nucleus at S2/S3 leading to bladder detrusor contraction, internal and external urethral sphincter relaxation, and relaxation of the tonic pubococcygeus contraction [16]. Urine can then flow through the urethra. In health, however, this switch between storage and voiding is itself controlled by more rostral circuits. These are the storage and voiding components of the pontine micturition centre, located in the periaqueductal grey matter of the pons [6, 16]. This pontine system is itself managed by higher-level thalamic and cortical mechanisms (see below and Fig. 4.1). The pressure within the urethra during voiding varies, non-linearly, according to the urethral resistance. Urethral resistance is inversely proportional to the fourth power of the radius of the urethra, as described by Poiseuille's Law for flow of a liquid in a narrow tube [17] (see below). Thus, a widely open urethra requires much less detrusor pressure to void urine than does a less widely open urethra, and a focal narrow urethral diameter, as in prostatic enlargement, may be so resistant to urine flow as to cause difficulty voiding [18].

4.3.2 The Lumbosacral Loop

In the normal human, the simple spinal mechanism limited to the sacral spinal cord for urinary voiding and defaecation described above is subject to modulation from higher CNS centres. After spinal transection, for example, due to spinal injury, the reflex relationship between the Onuf nucleus and the bladder and bowel is intact, and urinary voiding and defaecation are therefore then under strictly lower-level reflex control, without the possibility of voluntary modulation. Voluntary control of these functions requires supraspinal mechanisms [16].

4.3.3 The Pontine Loop

The first of these supraspinal systems depends on another 'switch mechanism' involving the periaqueductal grey (PAG) matter of the midbrain and the pontine micturition centre (PMC), first described by Barrington in 1933 [19]. Two groups of neurons have been recognised in this region, subserving storage (PAG activity) and voiding (PMC activity), respectively [12]. Sensory information from the bladder projects from the Onuf system in the sacral cord to this cluster of neurons adjacent to the periaqueductal grey matter in the midbrain [16].

4.3.4 The Cortical System

The pontine storage and voiding systems also receive descending input from higher centres including frontal lobe, insular cortex, and hypothalamus [12, 16]. Neural output from the pontine PAG projects caudally, initiating motor commands to the Onuf nucleus and modulating bladder detrusor contraction in the coordinated response necessary for voiding. The equilibrium between these two functions is determined by the balance between sensory input and descending activity from the brain itself, reflecting information regarding timing, and determinations based on learned social and behavioural rules, thus requiring predominantly frontal cortical control of the peripheral opening mechanism (see Figs. 4.1 and 4.2) for both defaecation and micturition. These concepts have been investigated in some detail using functional MRI and PET scanning [8]. This work has also shown activity in a lateral pontine region (the L region) adjacent to the PMC that has been found to be to be active during suppression of the voiding response and therefore in the maintenance of storage (continence) until a suitable opportunity arises.

4.3.5 Central Representation of Afferent Information from Bladder and Bowel

Processing of afferent information from the bladder and bowel therefore involves relaying this information, most of it carried in autonomic afferents, to the thalamus, and thence to the insular cortex, a region of the brain concerned with infor-

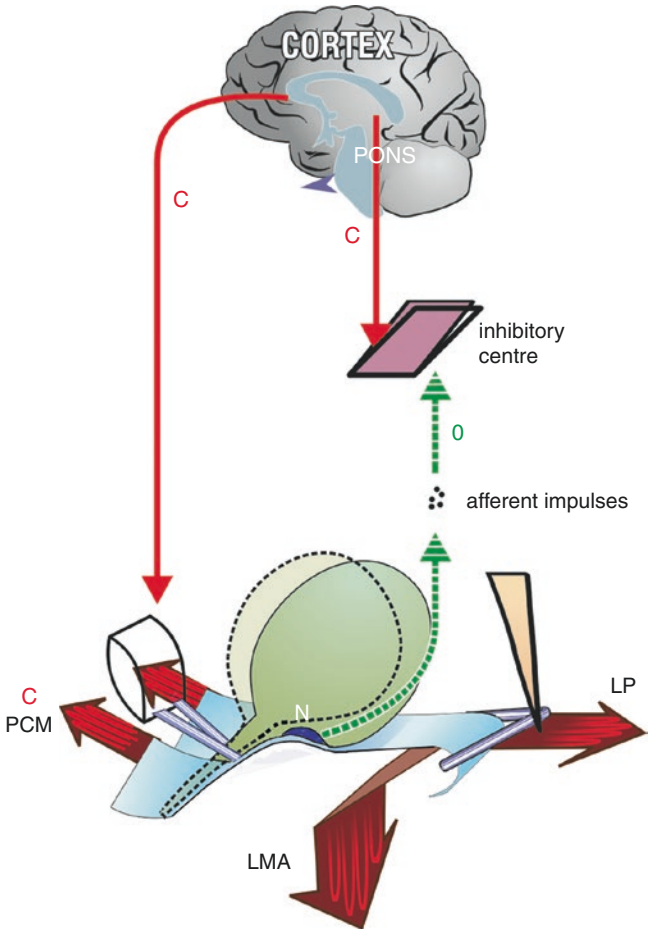

Fig. 4.2 Storage is dominant (C). Micturition is suppressed (green broken lines). The closed trapdoor represents inactive inhibitory centres. 'N', stretch receptors. Arrows, the three-directional vectors (PCM, LP, LMA). *O* open (voiding) response (inhibited in this sequence). *LMA* levator muscle ani, *LP* levator plate, *PCM* pubococcygeus muscle

mation management from internal organs, and therefore in homeostasis [8, 12, 16]. This region is linked to affective and social context-driven aspects of brain function in concert with the anterior cingulate cortex. Prefrontal cortical connexions to the limbic system, including the suprachiasmatic nucleus, important in timing cyclic tasks, are dominant in normal humans in managing both continence and voiding. During voiding in normal subjects, there is activation of the medial prefrontal area [20], suggesting that this region is important in conscious decision-making about voiding. Lesions in prefrontal cortex or its deep white matter are classically associated with uncontrolled micturition in socially inappropriate circumstances [21, 22]. Such behaviour is characteristically a feature of prefrontal traumatic brain injury, deep white matter anterior cerebral infarcts, gliomas, and certain neurodegenerative conditions, especially frontal dementia syndromes.

4.3.6 Universal Organisation of CNS Control Systems

This successive layering of control systems built on a simple lower-level reflex system is a characteristic common to the organisation of all mammalian motor systems [23]. Such a system allows modulation of various control points within the layered systems. Thus the afferent-efferent system located in the sacral spinal cord at the Onuf nucleus is modulated by the ponto-mesencephalic storage and voiding neurons, which intercede in the balanced storage equilibrium that maintains urinary and faecal continence. This system is itself modulated by time-dependent neuronal systems, probably mainly of hypothalamic origin, based on the suprachiasmatic nucleus, which subserves time modulatory functions in many functional domains, and is also influenced by prefrontal cortical 'decision-making' neuronal circuits and by emotional input from insular and callosal neuronal systems. Voluntary control of sympathetic and parasympathetic neuronal systems, in the context of bladder and recto-anal and bowel detrusor and sphincteric muscular systems, is perhaps more precise and overt than control systems involving other autonomic neuronal pathways, but it is not unique; for example, smooth muscle peristalsis in the oesophagus links seamlessly to swallowing itself and to striated muscle activity in the upper oesophagus as well as control of the cardiac sphincter at the lower end of the oesophagus.

Like other striated muscles in the body, the local muscular systems in the pelvic floor contain muscle spindles and other sensory endings [24], including tendon organs at the points of insertion of muscles into tendon, and smaller myelinated and unmyelinated fibres responsible for the sensation of pain and other nociceptive functions. In addition Pacinian corpuscles, which signal pressure, are distributed in fascial planes within the muscles and in the tissue, especially peritoneum, surrounding the bladder and bowel. The system thus receives signals not only from the smooth muscle systems of the bladder and rectum but also from the mucosa of these organs and from the muscles of the pelvic floor. The signals include pressure, tension, stretch, and rate of change of stretch, the letter two types of sensation representing spindle activity. There is a complex sensory system intrinsic to receptors on epithelial sensory cells in the bladder and bowel walls, conferring spatial and temporal information about bladder and bowel contents, respectively [6]. Clearly, not all these sensations are perceived in consciousness, but all are important afferent systems that integrate with central neural pathways in storage and voiding of urine and faeces.

4.4 The Pelvic Floor and Its Innervation

The basic structure of the pelvic floor consists of muscular and fascial planes positioned in relation to the anorectal, vaginal, and urethral openings. The perineal musculature within the pelvic floor functions as a whole during urinary and faecal storage, which should be regarded as the default mode, but the anterior and posterior components are capable of distinct, separate function during micturition and defaecation, respectively. These functions are under separate but analogous control within the central nervous system, as described above. The innervation of the pelvic floor musculature is largely from somatic efferent and afferent nerve fibres travelling within the lumbosacral plexus, and therefore entering these muscles superiorly, but the external anal sphincter and external urethral sphincter muscles, and the puborectalis and pubococcygeus muscles, are innervated by branches of the pudendal nerve, the external urethral and inferior rectal nerves, that supply these muscles from a caudal aspect. All these nerves are derived from sacral spinal segments via the lumbosacral plexus [25].

4.5 Pelvic Floor Dysfunction in Incontinence

When there is damage to the pelvic floor, for example, from stretch injury to ligaments, muscles, and pelvic nerves during a difficult childbirth, or in multipara, the pelvic floor musculature is functionally at a mechanical disadvantage [26]. If a muscle tendon is stretched and has lost its normal elasticity, the force applied during contraction of its muscle will be reduced, and it will reach its maximum more slowly as the lax ligaments are tightened more slowly than normal [27]. Since it is likely that damage to pelvic floor ligaments during a difficult childbirth will not be equally distributed across all the pelvic ligaments (see illustrations) within the pelvic floor, the muscle force vectors resulting from contraction of the pelvic floor muscles will then be distributed in an abnormal pattern. This results in voiding dysfunction and difficulty in maintaining urinary and/or faecal storage. There may also be associated pelvic pain, and, since the anatomy is distorted, there will be a degree of visible perineal descent on coughing or straining and, in the extreme case, organ prolapse [14]. These functional and structural abnormalities usually slowly progress over time [4]. Very difficult deliveries, especially those requiring forceps assistance, are frequently associated with more severe abnormalities in the pelvic floor postpartum, sometimes with anal sphincter tears. The latter are particularly likely to be associated with ligamentous damage and with damage to the innervation of the pelvic floor musculature, a combination of abnormality especially associated with disorders of continence. In the presence of weakness of the pelvic floor due to lax ligaments, tearing of muscle fibres, and damage to the somatic innervation of pelvic floor muscles and sphincters, the central nervous system will adapt and modify descending motor commands, insofar as it is capable of doing so, to prevent urinary or faecal leakage. Similarly, adaptations in CNS function occur in relation to other pelvic floor disorders, for example, urgency. These natural adaptations form the functional basis for the use of conditioning therapy, or other neuromodulation procedures, usually based on methods to facilitate increased sensory awareness [28]. These management strategies reinforce the process of maintaining storage at rest but are unlikely to have any biological effect on the already damaged system, particularly regarding its multifactorial nature. When the innervation of some of the muscles of the pelvic floor is damaged, there is less capacity for resetting of control systems.

4.6 Investigation of the Pelvic Floor

The clinical features of pelvic floor disorders and the use of investigations such as video cystometry and anorectal manometry are described elsewhere in this book. Video cystometrograms provide information about pressure/flow relationships within the urinary system, together with bladder volumes before and after voiding. This information is essentially descriptive and does not, of itself, provide insight into the underlying pathophysiology of pelvic floor dysfunction. Anorectal manometry, similarly, is essentially a descriptive account of pressures generated within the anal canal, the anal sphincter region, and the rectum at rest and during attempted straining at stool. Voluntary straining at stool is not the same process as normal defaecation. These investigative data must therefore be interpreted in relation to the historical pathogenesis of the pelvic floor dysfunction and the results of quantitative clinical assessment of pelvic floor function made by careful clinical examination.

Neurophysiological investigation of the pelvic floor musculature and its innervation has been important in defining clinically relevant abnormalities, but it does not necessarily guide the surgeon or physician in designing therapy. For example, pudendal and perineal nerve terminal motor latency determinations showed that there was damage to the pelvic floor nerves in women with pelvic floor prolapse, people with urinary incontinence, and even those with a history of difficult childbirth without overt pelvic floor symptoms [2, 24, 29]. However, best management of these disorders, when

necessary, requires supporting the lax ligaments that accompany damage to the nerve supply of the pelvic floor, thus at least partially alleviating muscular weakness and resolving the clinical disorder.

4.7 Urinary Storage: The Default Mode

During urinary storage [14] in women (Figs. 4.2 and 4.3), three muscle vectors stretch the vagina, like a membrane, in opposite directions. The voiding response is quiescent, and afferent sensory activity is reduced (green broken lines). Tonic activity in the pubococcygeus muscle (PCM) has stretched the suburethral vaginal hammock forwards against the pubourethral ligament (PUL) to close the distal urethra. The levator plate (LP) stretches the proximal vagina and bladder base backwards against the PUL, further tensioning the vagina. The longitudinal muscle of the anus (LMA) contracts against the uterosacral ligament (USL) to rotate the bladder and kink the urethral tube at the bladder neck, maintaining continence (see Fig. 4.4).

4.8 Urethral Opening: Voiding

The bladder is a highly distensile receptacle which can hold large amounts of urine, even in excess of 1000 ml. Voiding via the urethra follows activation of the micturition response [5, 6] (Fig. 4.5). For urine evacuation to occur, storage must be suppressed and voiding activated. Normal voiding is defined as the controlled emptying of the bladder on demand and rapid recovery to the closed state on completion. Voiding is activated by both stretch and surface receptors at the bladder base that vary in sensitivity from person to person. Voiding is a neurological feedback system, requiring coordination by the central nervous system. There are four main components (Fig. 4.5):

1. The hydrostatic pressure of a full bladder activates bladder stretch and mucosal surface receptors (N) that signal to the cortex via the spinal cord and brainstem (see above).
2. The anterior striated PCM muscles relax.
3. The posterior striated muscles (LP/LMA) stretch open the outflow tract (Figs. 4.5 and 4.6) reducing internal urethral resistance to flow.
4. Parasympathetic activation causes bladder detrusor muscle contraction. The bladder contracts as a whole due to electrical transmission fibre to fibre [30] to expel urine.

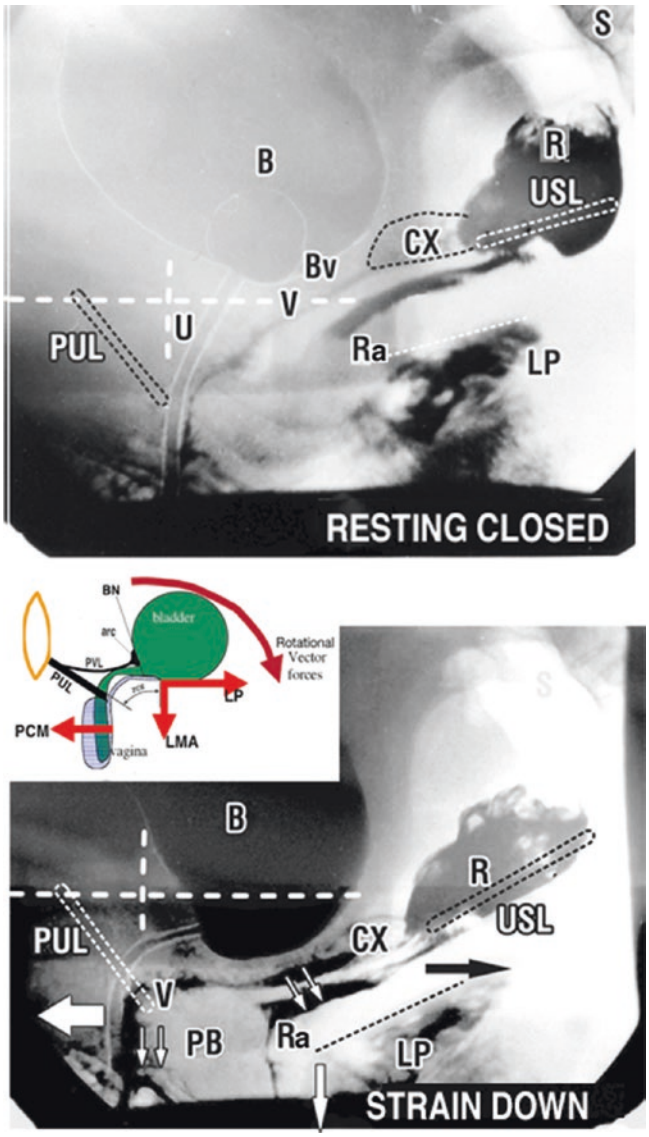

Fig. 4.3 Changes in bladder and anorectum when storage is dominant. Note the change in vaginal shape (V) and the positions of bladder (B), rectum (R), and levator plate (LP) relative to the vertical and horizontal bony coordinates (white broken lines) during straining. Vaginal elasticity is critical for transmission of muscle forces by directional vectors (arrows). *Upper XR*. *Resting closed (storage) phase PUL* pubourethral ligament, *USL* uterosacral ligament. *CX* cervix, *U* urethra, *Ra* anorectal angle, *Bv* ligamentous attachment of bladder base to upper vagina. *Lower XR*. *On effort* (*straining down*) Directional vector forces (arrows) stretch the distal vagina and urethra forwards against PUL. The bladder base and rectum are tensed backwards against PUL by the backward vector LP (backward arrow). The downward vector (white arrow) pulls bladder base and rectum downwards against USL to close the bladder neck (BN) and Ra. Adequate elasticity is required in the anterior vaginal wall 'ZCE', a zone of critical elasticity (see insert), for this to occur. *Arc* precervical arc of Gilvernet, *PB* perineal body, *PCM* pubococcygeus ligaments, *PVL* pubovesical ligament, *S* sacrum

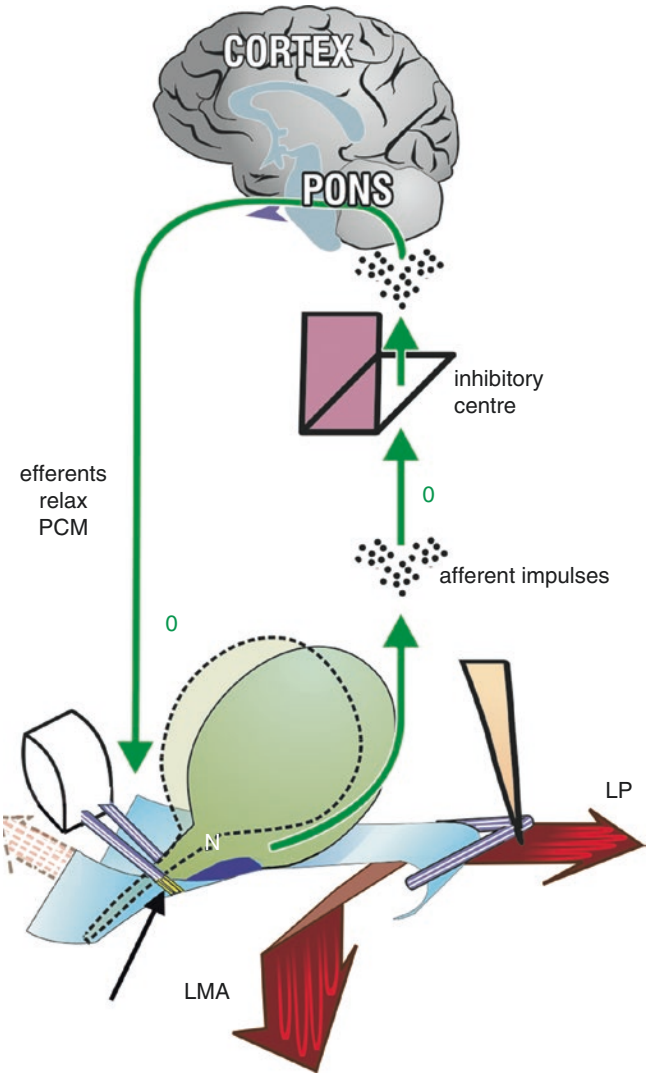

Fig. 4.4 Opening (micturition) reflex is dominant. The brain is in "voiding" (micturition) mode 'O'. The storage mode is suppressed and does not appear in the figure. Afferent impulses are upregulated (black dots). Relaxation of PCM (forward arrow) allows LP/LMA to stretch the vagina (blue) backwards. This stretches open (funnels) the posterior urethral wall decreasing resistance to urine flow and reducing the detrusor pressure required to void urine. The small black arrow indicates a slight downward excursion of PUL to facilitate backward stretching of the vagina. *LMA* longitudinal muscle ani, *LP* levator plate, *PCM* pubococcygeus muscle, *PUL* pubourethral ligament

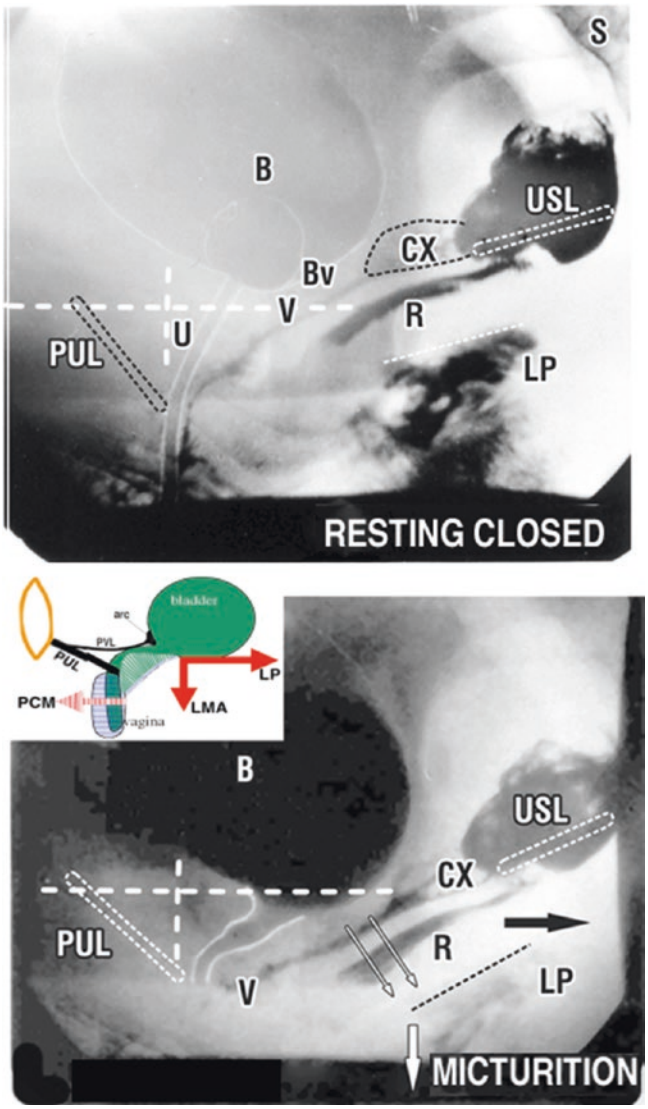

Fig. 4.5 Anatomical changes in bladder and anorectum when voiding is active. *Upper XR Resting storage mode.* Tonic muscle contraction (forward vector) maintains urethral closure distally and at bladder neck. *Lower XR Voiding*. The forward vector (insert) relaxes. Backward vectors stretch the vagina backwards and downwards against USL to open out the posterior urethral wall. The pubovesical ligament attachment to the arc of Gilvernet (PVL, insert figure) prevents the anterior bladder wall prolapsing into the outflow tract. *B* bladder, *CX* cervix, *LMA* longitudinal muscle ani, *LP* levator plate, *PCM* pubococcygeus muscle, *PUL* pubourethral ligament, *R* rectum, *S* sacrum, *USL* uterosacral ligament, *V* vagina

In Fig. 4.4, the lower urinary tract is in voiding mode (O) due to afferent input to the central nervous system. The inhibitory pontine L centre is inactivated (open trapdoor in Fig. 4.4); the forward vector PCM relaxes (faint broken lines); LP/LMA vectors stretch the vagina and open the urethra; this action decreases the urethral resistance to flow (Poiseuille's Law, see Fig. 4.10). Urine entering the proximal urethra further enhances afferent sensory impulses [7], and the posterior urethral wall is opened out by flattening the trigone, accelerating micturition (Video 1).

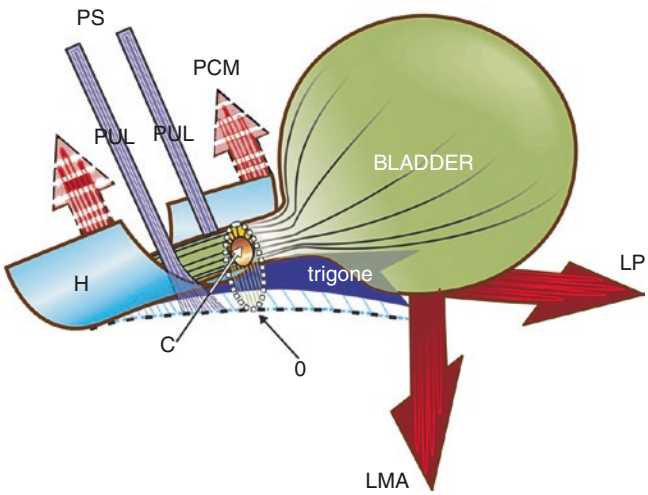

Fig. 4.6 The trigone. The trigone extends to the external urethral meatus. Pubococcygeus muscle (PCM) relaxation is indicated by a broken line pink arrow. When the upward and forward contracting PCM relaxes, levator plate (LP) stretches the vagina and trigone backwards into a semi-rigid structure. Longitudinal mascle ani (LMA) pulls down the trigone. *C* closed diameter of urethra (storage reflex dominant), *O* open diameter of urethra (voiding reflex dominant). *H* hammock, *PS* pubic symphysis, *PUL* pubourethral ligaments

4.9 The Bladder Trigone During Micturition

The trigone (Fig. 4.6) extends from the bladder base to the tip of the external meatus. Its intrinsic stiffness resembles that of the anterior vaginal wall. The posterior muscular vector, the levator plate muscle, stretches the trigone posteriorly. This reduces urethral resistance to urine flow because the posterior urethral wall becomes semi-rigid, creating a funnel, as seen in the micturition X-ray.

4.10 Neurological Feedback Control of Anorectal Function

A system similar to that for bladder control operates for anorectal closure and evacuation (Fig. 4.1) (see also Video 2). As the rectum fills, the stretch and surface receptors (N) send signals to the cord that are relayed to the frontal cortex. These can be suppressed voluntarily by the storage response (white arrow), which activates contraction of the opposed directional musculoligamentous forces (arrows) that support the anorectum from below. A further temporary control mechanism for both bladder and rectum is voluntary upward contraction of the puborectalis muscle. This counteracts the downward mechanical pressure of the bladder or faecal contents and diminishes sensory activation (green arrows).

4.11 When Things Go Wrong: Urge, Frequency, and Nocturia

Urgency to micturate or defecate represents inappropriate activation of the micturition or defaecation reflexes. PUL or USL ligaments (Fig. 4.1) are the effective insertion points of the three-directional muscle vectors (arrows). If PUL or USL is lax, their attached muscles (arrows) lose contractile efficacy. The weakened PCM and LP/LMA muscles then cannot tense the vagina or anorectum sufficiently to modulate sensory input. Micturition or defaecation responses may be then activated leading to urge urinary or faecal incontinence.

4.12 Overactive Bladder Syndrome (OAB)

In 1993, it was shown that prematurely activated, but otherwise normal micturition [31, 32], usually termed urge incontinence but also known as ‘detrusor instability’ or more recently ‘detrusor overactivity’ (DO), followed the same activation sequence as normal micturition (Fig. 4.4): first, a feeling of urgency, then a decrease in urethral pressure (X), followed detrusor contraction causing increased detrusor pressure, and urine flow (Fig. 4.7). At the onset of the syndrome, there is a strong afferent phase of urethral sensory activation, due to urine entering the upper urethra, as the trigone opens out slightly. Conflict between a natural desire to inhibit this unwanted signal for micturition and the urge to void produces physiological uncertainty between the storage (C) and voiding responses, which can be recognised urodynamically (Fig. 4.8).

4.13 How Does Detrusor Overactivity Relate to Feedback Control?

Detrusor overactivity represents a prematurely activated micturition sequence. Close examination of the urodynamic patterns in Fig. 4.7 indicates that the micturition reflex has been activated and bladder (B) and urethra (U) show identical wave patterns. When the micturition response is activated, excitation of smooth muscle fibres by parasympathetic nerve endings in the detrusor muscle causes the bladder to contract as a whole [30], as seen in the video X-ray voiding study (see Video 1). However, the detrusor muscle spasms. It does not relax and contracts like a striated muscle. What the bladder pressure transducer is measuring is the repeated striated muscle contractions of the rhabdosphincter as it tries to close the urethra at the base of the bladder.

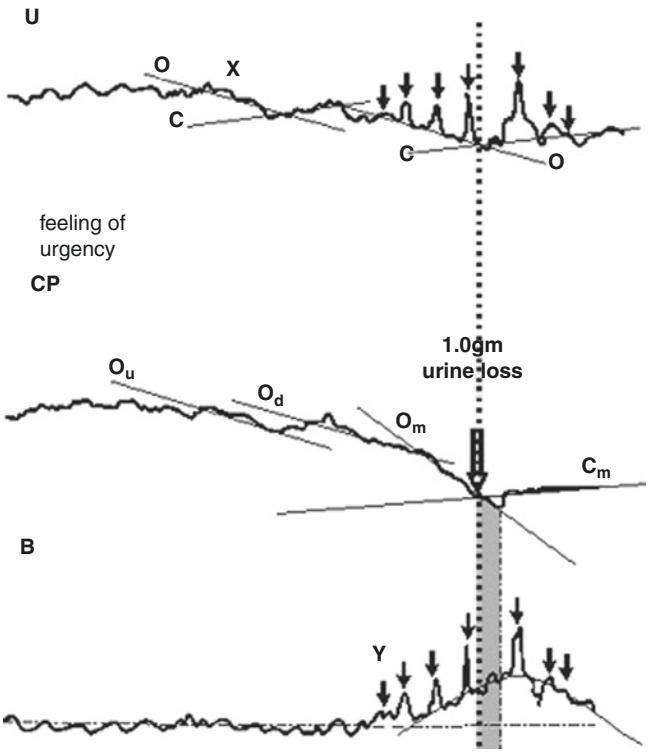

Fig. 4.7 Detrusor overactivity 'DO'—premature activation of a normal micturition reflex. Urodynamic graph of a patient with a full bladder undergoing a handwashing test. The binary control system becomes unstable. 'C' indicates the storage mode is dominant—the striated muscles are acting to close the urethra and trigone, causing a rise in urethral pressure (UP). 'O' indicates the muscles are relaxing, causing a fall in urethral pressure and leading to voiding. The sequence of events in a patient with urge incontinence and DO is: (1) A feeling of urgency, (2) A fall in urethral pressure at *X* (graph 'U'), (3) A rise in bladder pressure at *Y* (graph 'B'), (4) 1.0 g urine loss arrow, (graph 'CP'). *U* urethral pressure graph, *B* bladder pressure graph, *CP* closure pressure graph (*U* − *B*), *C* closure (continence) reflex, *O* opening (micturition) reflex, with its components being: *Ou* urethral relaxation, *Od* detrusor contraction, *Om* opening out of the outflow tract by the posterior muscle forces before voiding

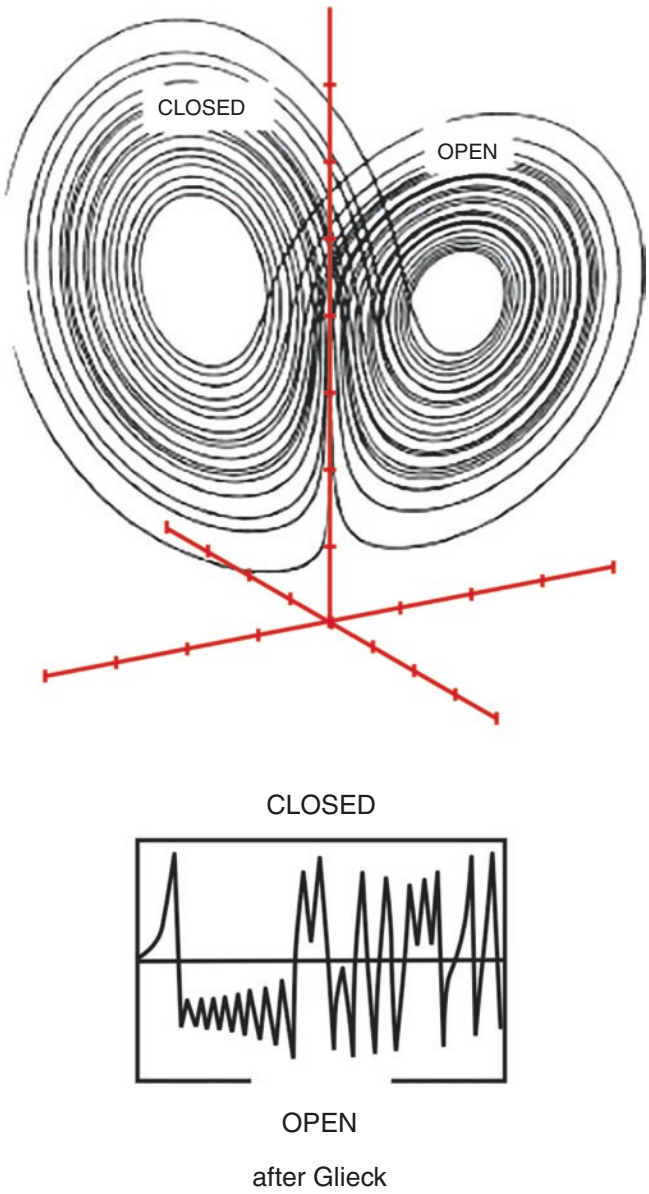

Fig. 4.8 Bladder instability. Bladder control swings between storage and voiding. Lax ligaments prevent maintenance of the closed storage position; the bladder now oscillates between storage (continence) and voiding phases. Because there is a short-time delay in switching between these two modes, the pressure curves are sinusoidal. This is clearly illustrated in both 'U' and 'B' in Fig. 4.7. The process resembles the feedback systems described in Chaos Theory (see text)

4.14 Events Occurring in Detrusor Overactivity and Overactive Bladder Syndrome

These are both manifestations of abnormal neurocontrol of the voiding response. When voiding is activated, there is a sensation of urgency associated with bladder contraction, beginning with slow waves of contraction, before the bladder contracts as a whole during voiding. If it is inconvenient to pass urine, the urethral closure response intervenes so that urine storage continues. Urethral closure increases the pressure in the urethra as well as in the bladder (see 'C', Fig. 4.9). However, if there is sufficient sensory input from bladder and trigone and from the urinary sampling response in the proximal urethra, urgency intensifies and micturition resurges. The forward vector PCM relaxes, and the urethral pressure falls (see 'O', Fig. 4.9). Urodynamically (Fig. 4.8), the switch between storage and micturition is manifested as a wave pattern (Figs. 4.8 and 4.9). When the closure response 'C' is dominant, the urethra narrows, and the urethral pressure rises. When the opening

(voiding) response 'O' is dominant, the striated muscles acting on the urethra relax and the urethral pressure falls. Because there is a brief delay between the afferent signals from the bladder reaching brainstem and higher centres causing this switch to the voiding response via the pontine micturition centre, the system may 'hunt' between the two states of storage and voiding, thus creating a 'wave pattern' (Fig. 4.9).

4.15 Non-linear Flow Mechanics Enhance the Storage and Voiding Responses

Bladder and rectum are elastic and expansile receptacles. The urethral and anal diameters are narrowed during storage and widened during voiding or evacuation by externally acting striated muscle forces (Fig. 4.10). This external mechanism causes a non-linear change in resistance to flow that is inversely proportional to the fourth power of the radius (Poiseuille's Law; see Fig. 4.10) [17, 18]. Therefore, in the urinary system, the flow response to narrowing or opening the urethral diameter is very rapid. For example, young people empty their bladders in just a few seconds. This concept of internal resistance to flow within the urethra is key to understanding normal storage (continence) and voiding (micturition) and abnormal bladder states, e.g. incontinence, 'obstructive' micturition, and obstructive defaecation. This interpretation is also important in understanding data from urodynamics, especially its wide variance, and the results of corrective surgery.

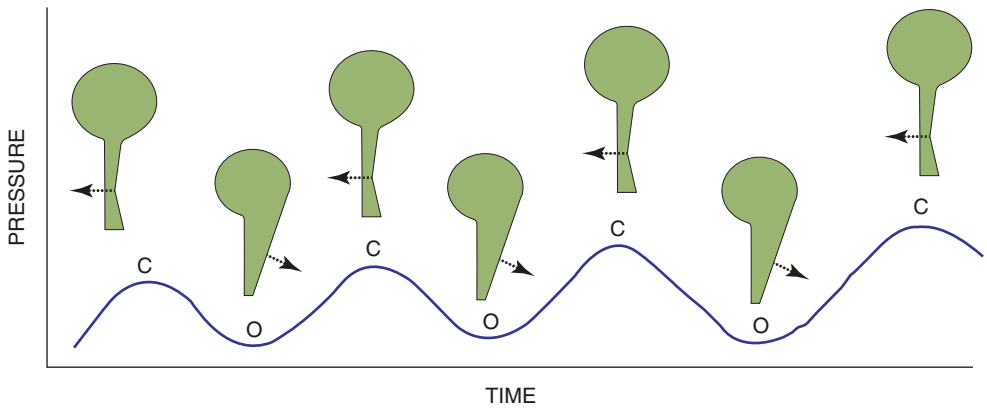

Fig. 4.9 Unstable phasic urodynamic bladder pattern 'DO'. The micturition reflex has been activated. The bladder swings between 'open' and 'closed' attractors (see Fig. 4.8). Intravesical pressure rises, while the 'storage' mode 'C' is dominant; the pressure falls, while the 'voiding' mode 'O' is dominant. Delay in switching from closure mode to open mode produces the phasic pattern

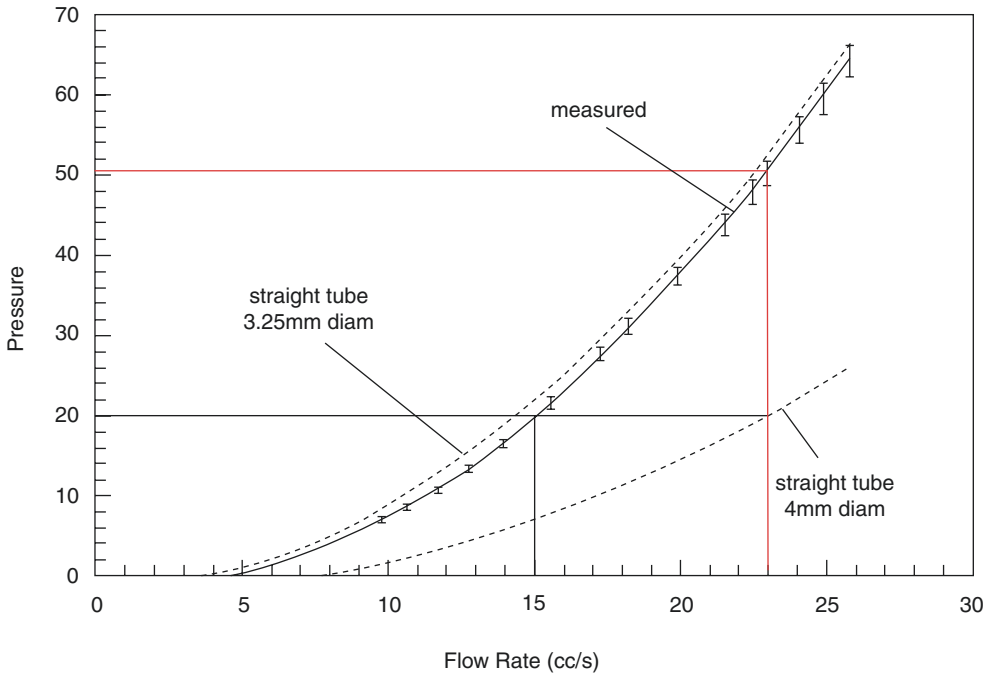

Fig. 4.10 The pressure/flow relationship as measured during bench experiments (unbroken lines) and by computer simulation (broken lines). Note that a 0.75 mm decrease in tube diameter from 4 mm to 3.25 mm (a 19% decrease) increases the expulsion pressure by 250% (red lines), consistent with Poiseuille's Law. After Bush et al. [17, 18]

4.16 Why Urodynamic Urethral Pressure Measurements Correlate Poorly with Clinical States

During passage of urine, including incontinent leakage, urine flows from the bladder to the exterior. Bench testing [17] and mathematical modelling [18] indicate that the key factor controlling the rate of urine outflow is the diameter of the urethra (Figs. 4.9, 4.10, and 4.11) which, in turn, regulates the resistance to flow against the pressure (*P*) of detrusor contraction. Whether urine leaks or not depends on the resistance to flow within the urethra (see above). Urodynamic studies are limited to measurement of intravesical and intraurethral pressures '*P*' and flow rates. Pressure is derived

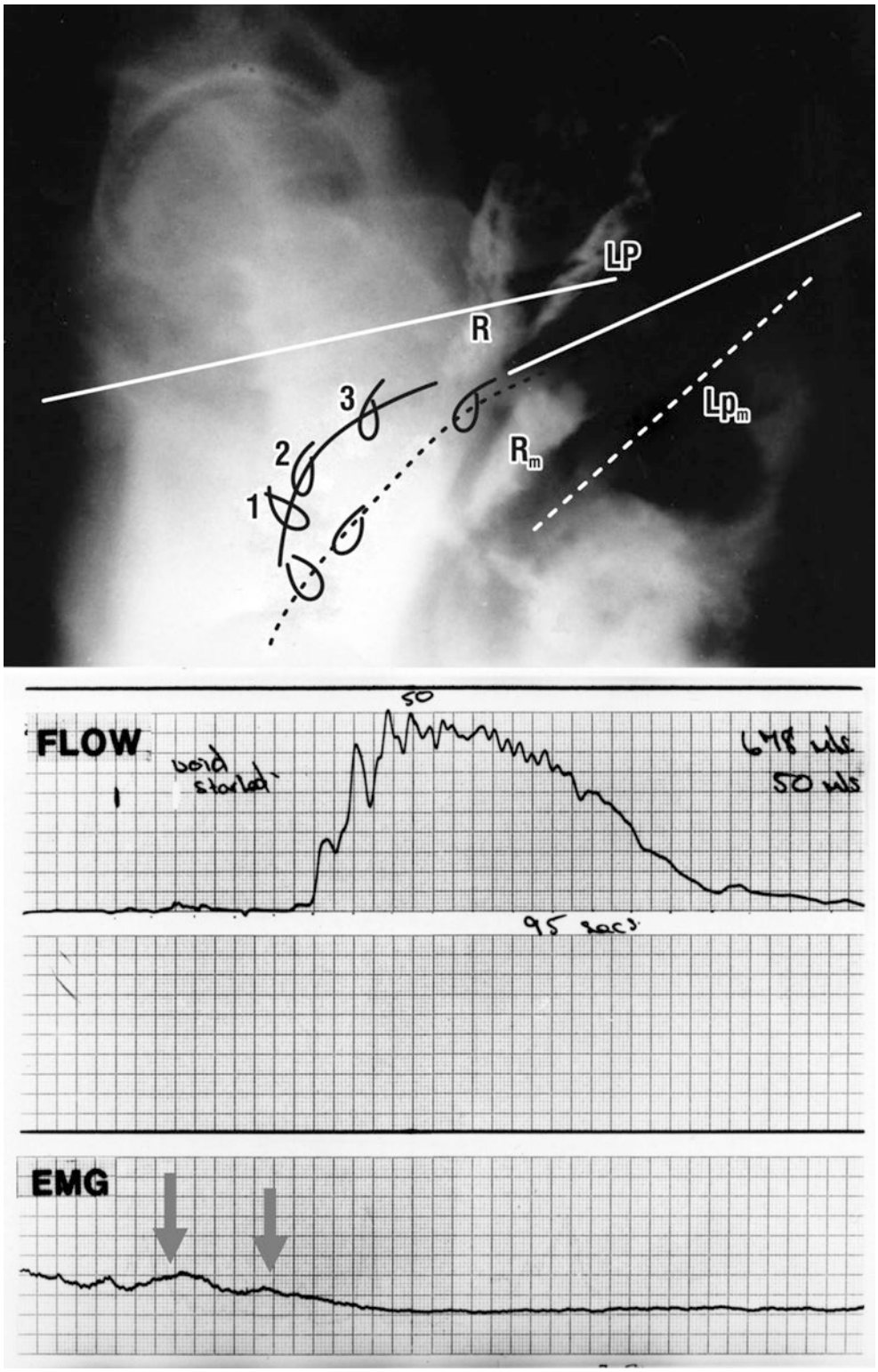

Fig. 4.11 Normal micturition in the female. *Upper image:* Voiding X-ray (broken lines, subscript m) superimposed on resting X-ray. Clips have been applied to the midurethra '1', bladder neck '2', and bladder base '3'. Note downward/backward displacement of the clips indicating stretching open of the posterior urethral wall. *Lower image* Note electromyography (EMG) activity (arrows) mainly at the start of micturition—the urine occupying the urethral tube is incompressible and helps to hold the urethra open as long as it is flowing. *LP* levator plate

Fig. 4.12 'Outflow obstruction' in the female—Lax ligament insertions for the LP/LMA opening vectors cannot open the urethra sufficiently against its elastic forces. Even with constant activation of the pelvic muscles, the stream is slow and prolonged with electromyography (EMG) activity seen throughout the micturition cycle

from the relation between applied force and transverse urethral area (πr^2). Intra-urethral pressure 'P', being proportionate to r^2, is not what prevents urine loss. Pressure at any point on the urethral diameter is not a sufficiently sensitive measurement index of incontinence, because it is the resistance to urine flow, which is proportionate to r^2, which prevents urine loss, not external urethral pressure.

Conversely, for urine to flow easily, there must be adequate external opening of the urethra, by opening out its posterior wall, as shown in the micturition X-ray (Figs. 4.5 and 4.11). If the urethra cannot be opened out adequately, there is a sensation of obstruction to flow. In Fig. 4.11, the urethra has been rapidly opened out by prior external muscle contraction, so micturition is rapid and effective. Continued external striated muscle contraction is not required as urine is incompressible and itself maintains urethral opening. In contrast, in Fig. 4.12, the urethra cannot be adequately opened. The posterior muscles LP and LMA (Fig. 4.6) have to contract repeatedly, and urine flow is slow.

4.17 How Repeatable Are Urine Flow Measurements in an Individual?

Urine flow studies measure the volume of urine flowing through the urethra at a defined diameter expressed as ml/second. Urine flow is determined by the internal resistance to flow [17, 18] as discussed above. Individual repeat flow measurements [18] are remarkably variable, because the opening mechanism assisting micturition is activated by pelvic floor striated muscle vectors (see EMGs in Fig. 4.12) and detrusor contractile power may vary from void to void. A very minor change in urethral diameter causes a profound change in flow rate.

4.18 Detrusor Underactivity

Detrusor underactivity (DU) is an ill-defined entity. Osman et al. [33] noted that:

> DU is present in 9–48% of men and 12–45% of older women undergoing urodynamic evaluation for non-neurogenic lower urinary tract symptoms (LUTS). Multiple aetiologies are implicated, affecting myogenic function and neural control mechanisms, as well as the efferent and afferent innervations. Diagnostic criteria are based on urodynamic approximations relating to bladder contractility such as maximum flow rate and detrusor pressure at maximum flow. Other estimates rely on mathematical formulas to calculate isovolumetric contractility indexes or urodynamic "stop tests." Most methods have major disadvantages or are as yet poorly validated. Contraction strength is only one aspect of bladder voiding function. The others are the speed and persistence of the contraction.

The 'myogenic function and neural control mechanisms' mentioned by Osman et al. [33] are relevant but undefined concepts, as they are at present impossible to quantify. We present here an alternative mechanism based on research by Bush et al. [17] which is consistent with Osman's conceptual analysis [33]. If the urethra can be opened by the external muscle forces to 4 mm diameter (Fig. 4.11), urine could flow at a rate of 7 ml/second *with no recordable detrusor pressure*. This does not mean that the bladder is 'underactive'. The bladder smooth muscle will contract sufficiently that the bladder can empty fully. Demonstrable urinary flow indicates that the urethra has been opened sufficiently to lower the internal resistance to a point at which the detrusor force enables flow to occur. For example, at a flow rate of 20 ml/s (Fig. 4.10), a 50 cm head of pressure from the detrusor is needed to overcome the internal resistance to flow in a 3.25 mm tube, but only a 20 cm head of pressure is required for a 4 mm tube. The graph (Fig. 4.10) illustrates the difference in voiding patterns in Fig. 4.11 (rapid flow) and Fig. 4.12 (slow flow).

4.19 Low Bladder Compliance

In a study of detrusor instability (now termed detrusor overactivity) and low compliance [34], the low compliance data could only be reconciled by considering 'low compliance' as a partially activated micturition reflex. Bladder 'stiffness' is a consequence of smooth muscle contraction by an activated but modulated micturition reflex.

4.20 Clinical Variations in Bladder Symptoms Are Consistent with the Chaos Theory Feedback Equation

One of the mysteries of clinical urology is the marked variability of symptoms such as urgency and nocturia. This variability is consistent with the classic Chaos Theory [35] feedback equation $X_{next} = X_c\ (1 - X)$. In Fig. 4.13 the feedback control system [34] was tested for compatibility with the Chaos Theory graph derived from the Chaos Theory feedback equation $X_{next} = X_c\ (1 - X)$. Calculations were made (see below) in three functional modes: *low afferent activity* (normal mode), *increased afferent activity* from a micturition reflex activated but controlled (low compliance mode), and *excessive afferent activity* exceeding the ability of the closure reflex to inhibit the afferent impulses 'overactive bladder' mode (see Fig. 4.1). An excess of afferent signals brings the system into the 'chaotic zone' in which equilibrium oscillates between voiding (open) and storage (closed): in this zone the closure reflex is in unstable competition with the micturition reflex (see Figs. 4.8 and 4.9). These three functional modes conform with classic Chaos Theory feedback calculations. As long as the closure reflex mechanisms, both central and peripheral, can control the increasing sensory input 'X' (X = no of afferent impulses), the patient's bladder remains in 'stable closed' mode, and the patient is dry. At the peak of the curve, the reflex closure mechanisms are overcome by the excessive number of afferent impulses (X) arriving at the pontine micturition centre and cortex; the micturition response begins to be activated. The system becomes unstable and oscillates between 'open' and 'closed' (Figs. 4.8 and 4.9). Patients may therefore report complete dryness on some days, yet they may wet four to five times on other days.

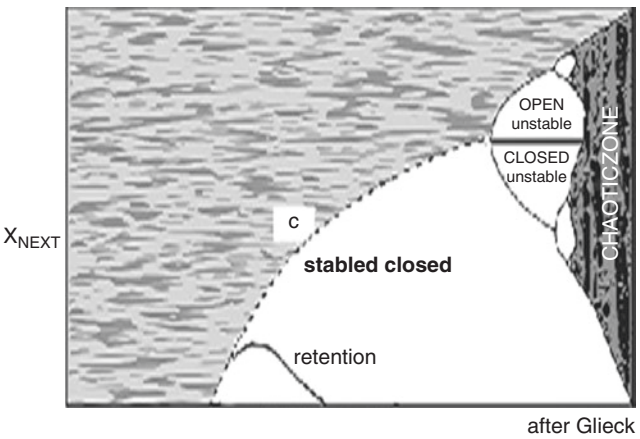

Fig. 4.13 (after Gleick 1987). The graph is that of a classic Chaos Theory feedback equation $X_{next} = X_c\ (1 - X)$ applied to normal bladder, low compliance bladder, and unstable bladder (see text). The line 'c' represents the sum of cortical and peripheral inhibition via the musculoelastic system (two variables). X_{next} can be equated to the number of afferent impulses. x axis, time; y axis, X_{next}. If the control mechanisms are all working properly, the system is in storage mode. Along line 'c', the voiding response is activated. However, while the peripheral and central mechanisms remain sufficiently supportive, the feedback system maintains the patient dry. Further along 'c', there is a bifurcation. The quantum of afferents is now excessive and exceeds the ability of the cortex to retain the closed phase; the bladder begins to swing between the 'storage' and 'void' attractors. This is 'detrusor instability' (DI) 'overactivity'. The system in the Chaotic Zone is very finely balanced. Any minor factor, for example, perimenstrual relaxation of the cervix, will loosen USLs, and the muscle vectors cannot maintain equilibrium. Afferent activity X_{NEXT} increases, and the system may enter the Chaotic Zone. The patient is then sometimes dry, sometimes wet

4.21 Concluding Remarks

The neurocontrol systems responsible for appropriate voiding of urine and faeces are linked to the default position of continent storage. The control system is organised on a layered sequence of neural systems that consist of local pelvic floor and bladder and anorectal structures—the end-organs—and spinal, brainstem, and higher-level cortical circuits. These circuits are integrated and responsive to each other. When the pelvic floor and its sphincteric systems are damaged, for example, by a difficult childbirth, or prostatic obstruction, a measure of adaptation in these neural systems is possible, although little is known about their adaptive capacity. It is essential that any corrective pelvic floor surgical procedures should retain the inherent musculo-elasticity of this system, since this determines the musculotendinous function necessary for normal function of the pelvic floor and its sphincteric systems.

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