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Continence and Micturition: An Anatomical Basis.

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ABSTRACT

Background: Urinary incontinence remains an important clinical problem worldwide, having a significant socio-economic, psychological and medical burden. Maintaining urinary continence and coordinating micturition are complex processes relying on interaction between somatic and visceral elements, moderated by learned behavior. Urinary viscera and pelvic floor must interact with higher centers to ensure a functionally competent system. This article aims to describe the relevant anatomy and neuronal pathways involved in the maintenance of urinary continence and micturition.

Methods: Review of relevant literature was carried out focusing on the anatomy of the pelvic floor and urinary sphincters, and on the neuroanatomy of urinary continence and micturition. Data obtained from both live and cadaveric human studies are included.

Results: The stretch during bladder filling is believed to cause release of urothelial chemical mediators, which in turn activates afferent nerves and myofibroblasts in the muscosal and submucosal layers respectively, thereby relaying sensation of bladder fullness. The internal urethral sphincter is

continuous with the detrusor muscle, but its arrangement is variable. The external urethral sphincter blends with fibers of the levator ani muscle. Executive decisions about micturition in humans rely on a complex mechanism involving communication between several cerebral centers and primitive sacral spinal reflexes. The pudendal nerve is most commonly damaged in females at the level of the sacrospinous ligament.

Conclusion: We describe the pelvic anatomy and relevant neuroanatomy involved in maintaining urinary continence and in allowing micturition, subsequently highlighting the anatomical basis of urinary incontinence. Comprehensive anatomical understanding is important for appropriate medical and surgical management of affected patients, and helps guide development of future therapies.

KEY WORDS: levator ani; pudendal nerve; urothelium; micturition; urinary incontinence

INTRODUCTION

The maintenance of continence and the process of micturition in humans are complex mechanisms that rely on normal functioning anatomy and learned behavior, and a neurally orchestrated interplay between both (Fowler, 2006). Failure of any of these systems, for example through pathology, trauma or iatrogenic injury, may result in dramatic lifestyle changes due to its potential social, psychological and medical consequences (Minassian et al., 2003; Molinuevo and Batista-Miranda, 2012). Urinary incontinence (UI) is a worldwide problem that has a higher prevalence in females, particularly in individuals above 65 years of age (Perry et al., 2000; Minassian et al., 2003; Thirugnanasothy, 2010). In the UK, prevalence of UI in those over 65 years of age varies from 5% to 20% in women and 3% to 10% among men (Royal College of Physicians, 1995). Appreciation of the anatomy of the pelvic floor and its relations to the pelvic components of the urinary system are integral to understanding the pathophysiology of UI. This in turn enables provision of better operative and non-operative therapies for patients suffering from UI.

ANATOMY

Endopelvic fascia, the pelvic floor and the perineal body

Condensations of visceral fascia surrounding the pelvic organs form a dense, fibrous connective tissue layer called the endopelvic fascia (also referred to as visceral endopelvic fascia) (Raychaudhuri and Cahill, 2008). The endopelvic fascia is continuous with the transversalis fascia of the abdomen and the parietal pelvic fascia investing the obturator internus, piriformis, levator ani and coccygeus muscles (Raychaudhuri and Cahill, 2008). The endopelvic fascia attaches to each arcus tendineus fascia pelvis laterally. The arcus tendineus fascia pelvis can be seen running from the pubic bone ventrally to the ischial spine dorsally. The fascia helps to suspend the urinary bladder neck and urethra on the anterior vaginal wall – the basis of the hammock hypothesis (DeLancey, 1994a). Additionally, the fascia stabilizes the organs in position above the levator ani.

The levator ani and coccygeus (also called ischiococcygeus) muscles form the lower limit of the true pelvis, referred to as the pelvic diaphragm. Three groups

of muscles form the levator ani, the main muscle of the pelvic floor: (i) iliococcygeus, (ii) pubococcygeus , which is considered to have three divisions as outlined in Table 1, (iii) puborectalis (Ashton-Miller and DeLancey, 2007). Collectively, peripheral attachments of the levator ani muscle include the body of the pubic bone, the ischial spine and arcus tendineus fascia pelvis (Bharucha, 2006).

Table 1: Differences in nomenclature between males and females of the three divisions of pubococcygeus muscle (Ashton-Miller and DeLancey, 2007; Yavagal et al., 2011; Molinuevo et al., 2012).

Within the anterior portion of the levator ani is an opening called the urogenital hiatus through which only the urethra passes in the male, whereas both the urethra and vagina pass in the female. The urogenital hiatus is bounded ventrally by the pubic bone and levator ani, and dorsally by the perineal body (Ashton-Miller and DeLancey, 2007). The perineal body is a connective structure into which the levator ani, superficial transverse perineal

muscles and perineal membrane attach (Yavagal et al., 2011). Clinically important functions of the perineal body are summarized in Box 1 (Woodman and Graney, 2002).

Compression of the urethra against the pubic bone and compression of the distal vagina against the posterior wall of the urethra is achieved by closure of the urogenital hiatus as a result of the normal baseline activity of the levator ani muscle. Further compression of the mid-urethra, distal vagina and rectum can be achieved by maximal voluntary contraction of puborectalis and pubococcygeal muscles (Ashton-Miller and DeLancey, 2007).

Box 1: Summary of the clinically important functions of the perineal body.

Bladder

The urinary bladder is a reservoir, which when empty lies entirely within the true pelvis and adopts a tetrahedral shape. Upon filling, the bladder rises

anterosuperiorly into the abdominal cavity towards its apex, from where the median umbilical ligament (urachus) arises. The base of the bladder is located posteroinferiorly and is triangular in shape. Each ureter forms the superolateral angle of the triangular base, while the internal urethral orifice forms the anteroinferior angle. This triangle, within the base of the bladder, is referred to as the trigone and can be seen on cystoscopy. Its smooth appearance is attributed to the lack of trabeculations, seen in the bladder mucosa elsewhere (Standring, 2008).

Fat in the retropubic space of Retzius separates the anterior surface of the bladder from the pubic symphysis. The superior surface of the bladder is covered by peritoneum which extends slightly on to the base and into the rectovesical pouch in males. However, in females, the peritoneum covers the superior surface of the bladder but is then reflected posteriorly onto the uterus to form the vesicouterine pouch anteriorly and rectouterine pouch of Douglas posteriorly. Each of the two inferolateral surfaces of the bladder is related anteriorly to the pubis and puboprostatic ligaments in males and to the pubis and pubovesical ligaments in females (Standring, 2008).

Histologically, the bladder wall is composed of the mucosal layer, the muscularis propria and the adventitia or serosa. Urinary bladder mucosa consists of the urothelium, a basement membrane and the lamina propria. Urothelium is a stratified epithelium lining the urinary tract between the renal calices and the urinary bladder, including the upper urethra and glandular ducts of the prostate (Birder, 2013). The urothelium is composed of at least three layers – a basal cell layer, an intermediate layer and an apical layer of 'umbrella' cells. The urothelium has several functions : (i) as a barrier to infections and molecules, (ii) in the release of signaling molecules (signaling role) and, (iii) in activating sensory neurons in response to physiological and chemical stimuli (transducer role) (Birder and de Groat, 2007).

The lamina propria lies between the urothelial basement membrane and the more peripheral detrusor muscle. It is composed of several types of cells including the fibroblasts, sensory nerve endings and myofibroblasts (also referred to as interstitial cells) (Birder, 2013). Based on their location within

the bladder wall, several subgroups of interstitial cells have been identified (McCloskey, 2013). Interstitial cells of the lamina propria are stellate in shape and have been found to be linked extensively by gap junctions (Sui et al., 2002). Detrusor interstitial cells are elongated non-networked cells arranged in circular, longitudinal and oblique orientation, on the boundary of smooth muscle bundles (McCloskey, 2013). Interbundle stellate-shaped interstitial cells, within the interstitial spaces between the detrusor bundles, form regions of interconnected cells close to nerves. Finally, perivascular interstitial cells have been identified on the periphery of small mucosal vessels in the bladder wall (McCloskey, 2013).

It is thought that interstitial cells play an amplification role in the sensory response to bladder-wall stretch, as occurs during bladder filling (Fry et al., 2007). A dense nexus of sensory nerves lies in close proximity to the suburothelial layer of interstitial cells (Gosling and Dixon, 1974). Two major subtypes of afferent nerve fibers are Aδ (myelinated) and C (unmyelinated) fibers. The Aδ fibers are distributed mainly within the detrusor smooth muscle and are responsive to detrusor stretch during bladder filling. The C fibers are

more widespread and are distributed within the lamina propria in close proximity to the urothelium (Fowler et al., 2008). C fibers are believed to have a higher threshold for activation (de Groat and Yoshimura, 2010) and are thought to be involved in sensing noxious stimuli (Habler et al., 1990).

Urothelial cells are specialized to detect both physical and chemical stimuli. This transducer role of the urothelium is enhanced by the close proximity to the urothelium of afferent and autonomic neurons (Jen et al., 1995; Grol et al., 2008). Studies have shown the secretion of transmitters or mediators such as ATP (Ferguson et al., 1997; Wang et al., 2005), acetylcholine (Kullmann et al., 2008), prostaglandins (Downie and Karmazyn, 1984), nitric oxide (Birder et al., 1998) and cytokines (Wood et al., 2012) from the urothelium. The mechanism underlying the release of these chemical mediators remains unclear. Additionally, it is still not known whether different layers of the urothelium are responsible for secreting different mediators (Birder, 2011). The variety of transmitters and mediators, released in part by the urothelium, can activate the nerve plexi (Birder et al., 2010). The suburothelial nerve plexus is particularly prominent at the bladder neck but is relatively sparse at the dome

of the bladder (Gabella and Davis, 1998). This pattern of distribution is thought to be important in the non-painful sensation of bladder fullness and emptying, and in pain sensation.

It is thus believed that the stretching associated with bladder filling causes a release of chemical mediators from the urothelium, which in turn activates afferent nerves and myofibroblasts in the muscosal and submucosal layers respectively, thereby relaying the sensation of bladder fullness (Fry et al., 2004).

URETHRAL SPHINCTERS

Internal urethral sphincter (IUS)

At the level of the bladder neck, the IUS surrounds the proximal urethra and is seen as a continuation of the detrusor smooth muscle, thereby favouring proximal urethral closure by constricting its lumen (Ashton-Miller and DeLancey, 2007). Smooth muscle fibers within the IUS are orientated in a

horse-shoe shaped arrangement, but Wallner et al., (2009) described the superior part of the urethra to have a completely circular arrangement of smooth muscle. Layers of striated muscle, arranged in a circular configuration and thought to be derived from levator ani, surround the smooth muscle layer of the IUS in the mid-portion of the urethra (Ashton-Miller and DeLancey, 2007; Jung et al., 2012). The IUS is innervated by the sympathetic nervous system, and is therefore under involuntary control.

External urethral sphincter (EUS)

Skeletal muscle, derived from the inner fibers of the levator ani muscle, surrounds the urethra as it traverses the deep perineal pouch thus forming the EUS. In males, the EUS covers the inferior aspect of the prostate and is located at the level of the membranous urethra (Jung et al., 2012) where fibers are oriented in a horse-shoe shape and without anatomical fixation to the levator ani muscle. This implies that voluntary closure of the urethra in males is executed by the EUS alone, without any involvement of the levator ani muscle

(Yucel and Baskin, 2004). The EUS is under voluntary control via the pudendal nerve.

In females, the EUS begins at the inferior end of the bladder and includes (i) the sphincter urethrae muscle, (ii) the compressor urethrae muscle, and (iii) the urethrovaginal sphincter (Macura and Genadry, 2008; Jung et al., 2012). Dorsolateral extensions of the inferior portion of the sphincter urethrae muscle are continuous with compressor urethrae muscle, whose contraction causes compression of the ventral part of urethra. The urethrovaginal sphincter is a thin, broad and flat muscle. As the inferior portion of EUS, the urethrovaginal sphincter encircles both the anterolateral parts of urethra and lateral aspect of vagina (Jung et al., 2012). Based on their findings from fetal pelvises, Wallner et al., (2009) proposed the following urethral closure mechanism in females: (i) the contraction of the levator ani muscle compresses the vagina against the posterior urethra above the level of EUS, (ii) the simultaneous contraction of EUS and levator ani muscle induces an anteriorly convex bend in the midurethra, (iii) the contraction of the inferior part of the EUS induces a posteroinferor force on the urethra as a result of a tendinous connection between the inferior part of EUS and the puborectalis portion of

levator ani (Wallner et al., 2009). Histological (Sebe et al., 2005) and magnetic resonance imaging (Macura and Genadry, 2008) studies have demonstrated the smooth muscle component of the IUS and the striated muscle component of the EUS to be maximally thick in the middle third of the urethra, therefore forming the true annular sphincter surrounding the urethra.

NEURONAL INNERVATION

Autonomic

Sympathetic innervation of the bladder and IUS originate as preganglionic neurons from the thoracolumbar segments T10 – L2. They traverse the paravertebral sympathetic chain bilaterally to join the pre-aortic plexuses. These preganglionic neurons ultimately converge on the superior hypogastric plexus, either via the aortic plexus in the case of the least splanchnic nerve from T12, or the inferior mesenteric plexus in the case of the lumbar splanchnic nerves from L1-L2. The superior hypogastric plexus is located in the midline at the level of the bifurcation of the aorta and above the sacral promontory. Along their course, the majority of these neurons will have

synapsed with their postganglionic sympathetic counterparts, which descend from the superior hypogastric plexus to the right and left inferior hypogastric plexuses via their respective hypogastric nerve. Preganglionic parasympathetic fibers from the pelvic splanchnic nerves, coursing from the ventral rami of S2-S4, join company with the sympathetic nerve fibers to form the inferior hypogastric plexuses. The inferior hypogastric plexuses are located posterolateral to the urinary bladder, and give rise to vesical, prostatic, uterovaginal and rectal plexuses that innervate the bladder, prostate, uterus, vagina and rectum respectively (Bharucha, 2006; Standring, 2008).

Visceral afferent fibers from the bladder are carried within the hypogastric and pelvic nerves to the dorsal root ganglia of the corresponding lumbosacral segments (Standring, 2008). Afferent nerve pathways provide input into the reflex circuits that control bladder filling and emptying. Additionally, afferent nerves are the source of non-painful sensations of bladder fullness (de Groat, 2006; Birder, 2013).

Somatic

Cholinergic motor innervation to the striated muscle fibers of the EUS is derived mainly from the pudendal nerve, whose cell bodies are located within spinal segments S2-S4. Cell bodies of the pudendal nerve motor neurons are located in Onuf's nucleus – first identified as nucleus X located anteromedial to the anterolateral nucleus and extending between the distal part of S1 and the proximal part of S3 (Pullen et al., 1997). The ventral rami of sacral spinal nerves S2 – S4 give rise to the pudendal nerve, which is formed at the upper border of the sacrotuberous ligament. The pudendal nerve leaves the pelvis via the greater sciatic foramen to enter the gluteal region. It crosses the sacrospinous ligament close to its attachment with the ischial spine and then courses posterior to the ischial spine to enter the gluteal region. From here on, the pudendal nerve is susceptible to compression, descent and stretch during vaginal childbirth as it continues to course anteriorly within the pudendal canal (of Alcock). The inferior rectal, perineal and posterior scrotal nerves are branches of the pudendal nerve. Apart from somatic motor innervation to the EUS, the perineal nerve also supplies sensory and motor input to the following muscles within the pelvic floor and deep perineal pouch: transverse perinei,

bulbospongiosus, ischiocavernosus, anterior part of external anal sphincter and levator ani (Bharucha, 2006). The inferior rectal nerve is the main motor supply to the external anal sphincter (Shafik, 2000).

Figure 1: Neuronal innervation of the urinary bladder.

MAINTENANCE OF CONTINENCE

Continence can only be maintained if mechanisms that cause the intra-urethral closure pressure to exceed urinary bladder (intravesical) pressure are functioning normally, both at rest and during times of raised intra-abdominal pressure (Allen and Keane, 2005). This involves integration of complex neuronal input (outlined below) into the anatomical components described above.

MICTURITION

The elimination of urine after forming at the renal collecting ducts involves two phases; the storage phase – where the urinary bladder acts a reservoir for the collection of urine – followed by the voiding phase, which is initiated once a bladder threshold volume of urine is reached. Both phases are controlled by reflex mechanisms within the autonomic and somatic nervous systems. The process of micturition is also influenced by supraspinal central nervous system input mechanisms, which are discussed below (Drake et al., 2010).

During bladder filling, stretch-sensitive mechanoreceptors in the bladder wall are activated. First-order visceral afferent neurons convey sensory information, via the pelvic nerves, to a cell group in the lateral dorsal horn and lateral part of the intermediate zone within the sacral spinal cord termed Gert's nucleus (Holstege, 2005; Holstege, 2010). Through complex interneuron circuitry within the spinal cord, parasympathetic innervation of the detrusor is inhibited (Fig. 2). Supraspinal input ensures that the voiding reflex remains under voluntary control as the decision to void is based on a combination of

emotional, social and visceral sensation factors (Fowler et al., 2008). Therefore, to maintain continence, simultaneous stimulation of the pudendal nerve to the EUS and sympathetic activity to the bladder neck and IUS via the hypogastric nerve occurs (Fig. 2). The process of maintaining continence throughout bladder filling is called the guarding reflex (Park et al., 1997; Fowler, 2006; Fowler et al., 2008; Drake et al., 2010). The net effect of the guarding reflex is caused by closure of both the IUS and EUS and prevention of bladder contraction.

From Gert's nucleus, second-order afferent fibers ascend within the fasciculus gracilis to relay sensory information pertaining to bladder filling to the midbrain periaqueductal gray (PAG) matter (Kavia et al., 2005; Griffiths and Tadic, 2008), where third-order neurons originate. Higher centers such as the insula, thalamus, anterior cingulate gyrus (ACG) and prefrontal cortices have multiple connections with the PAG. Together, these higher centers determine the temporal and social appropriateness for micturition to occur (Fowler, 2006). Bladder afferents received by PAG are relayed onto the insula – often referred to as the sensory cortex of the autonomic nervous system (Drake et

al., 2010). The insula and ACG have shown increased signal activation on brain functional imaging, particularly during bladder filling rather than voiding (Kavia et al., 2005).

Figure 2: Neuronal pathways involved in the guarding reflex, thereby maintaining continence during bladder filling.

The PAG acts as an interface between the afferent and efferent limbs of bladder control circuits. It has main control of the pontine micturition centre (PMC), also known as the M-region or Barrington's nucleus. The PMC is located in the dorsal part of the caudal pontine tegmentum, adjacent to the locus coerulus (Fowler et al., 2008; Holstege, 2010). The PAG informs the PMC about the degree of bladder fullness and mediates higher influences on the PMC such that higher centers ensure maintenance of voluntary control of the voiding reflex. From the PMC, long fibers descend to the parasympathetic sacral bladder motor neurons and to the inhibitory interneurons to Onuf's nucleus (Holstege, 2010). When a critical level of bladder distension is reached,

maximal bladder afferent activity within the PAG results in stimulation of the PMC (Fig. 3). As a result of this spinobulbospinal reflex, voiding occurs. The PMC is therefore regarded as the final efferent nucleus of the micturition pathways that co-ordinates inhibition of the sphincters and initiation of detrusor contraction. Hence, activity of the PMC needs to be inhibited during the bladder filling and storage phases. If the spinobulbospinal reflex were to act alone without any input from higher centers, for example in suprapontine cerebral lesions or thoracolumbar cord lesions, involuntary voiding would take place whenever the bladder volume reached a critical level. It is thought that the pre-frontal cortex of the frontal lobe is the seat of planning cognitive behaviors, expression of personality and appropriate social behavior (Fowler, 2006). During functional brain imaging, the pre-frontal cortex was found to be activated during both the urine-withholding and voiding phases (Kavia et al., 2005). It therefore plays a major executive role in deciding whether or not micturition occurs, and if so, the appropriate time and place.

Figure 3: Neuronal pathways involved in initiation of micturition.

(+) denotes stimulatory effect, (-) denotes inhibitory effect

UNDERSTANDING INCONTINENCE

Incontinence is defined by the International Continence Society as the involuntary loss of urine that is a social or hygienic problem. In females, genuine stress incontinence (GSI) remains the major cause of UI. GSI is defined as the involuntary loss of urine when the intra-vesical pressure exceeds the maximal urethral pressure in the absence of detrusor activity (Abrams et al., 2002). One possible explanation for GSI is the pressure transmission theory, where hypermobility of the bladder neck and urethra as a result of inadequate supporting structures causes them to lie below the general level of the pelvic floor. Therefore increases in intra-abdominal pressure result in GSI due to failure of counteractive pelvic floor and pelvic fascia pressure (Allen and Keane, 2005). One group has shown that the medial pubovisceral muscle (pubococcygeus) undergoes the largest stretch of any levator ani muscle during vaginal childbirth (DeLancey et al., 2003; Lien et al., 2004). However, studies from other groups have shown stretch-related muscular defects in the

puborectalis muscle resulting from vaginal birth (Hoyte et al., 2008; Svabik et al., 2009) and caesarean delivery (Novellas et al., 2010). Damage to the branches of the pudendal nerves or the pudendal nerve itself close to the ischial spine results in levator ani muscle atrophy. The endopelvic fascia and suspensory ligaments take over responsibility for pelvic organ support, but with time these connective tissue structures stretch leading to pelvic organ prolapse (DeLancey and Ashton-Miller, 2004; Dietz and Lanzarone, 2005; Ashton-Miller and DeLancey, 2007).

Detrusor overactivity (DO) is defined by the International Continence Society as 'a bladder shown to contract, spontaneously or on provocation, during urodynamic bladder filling while the patient is attempting to inhibit micturition' (Abrams et al., 2002). As described earlier, higher centers inhibit the PMC and therefore maintain voluntary control of the voiding reflex (Fig. 3). However, an intact spinobulbospinal reflex in the absence of higher centre control causes involuntary voiding during bladder filling. Thus, suprapontine lesions as a result of vascular, degenerative or neoplastic etiology affecting the anterior (frontal) brain or degeneration of the dopaminergic neurons as in

Parkinson's disease result in removal of the tonic inhibitory control over the PMC, and thereby DO. However, suprapontine lesions are characterized by an intact micturition reflex and therefore symptoms in these patients range from UI, urinary retention and DO. Incontinence occurs early in multiple system atrophy (MSA), a neurodegenerative disorder characterized by prominent cell loss in the pons, descending sympathetic pathways, in the interomediolateral cell column and in Onuf's nucleus, resulting in incomplete bladder emptying, open bladder neck (i.e. a patent internal urethral orifice resulting from decreased IUS tone) and weakness of the striated urethral sphincter (Fowler, 2006; Fowler et al., 2008).

In spinal cord injuries occurring rostral to the lumbosacral cord level, voluntary and supraspinal control of voiding are blocked. Clinically, complete urinary retention is initially noted as a result of an areflexic bladder. This is followed by a slow development of automatic micturition and neurogenic DO that is mediated by development of spinal reflex pathways (de Groat and Yoshimura, 2006), eventually resulting in detrusor sphincter dyssynergia (DSD) and a low compliance bladder – a urinary bladder that demonstrates large increases in

detrusor pressure when filled with a small volume as a result of fibrosis and decreased elasticity within the bladder wall (Sand and Ostergard, 1995). The International Continence Society defines DSD as ‘a detrusor contraction concurrent with an involuntary contraction of the urethral and/or periurethral striated muscle.’ (Abrams et al., 2002; Bacsu et al., 2012). Autonomic dysreflexia may be a significant problem in patients with lesions above vertebral level T6. The urinary bladder is subject to high pressure that can lead to damage to the upper urinary tract. Briefly, autonomic dysreflexia results from noxious stimuli below the level of spinal cord injury, for example from bladder distension. Sensory input from noxious stimuli is carried to the spinal cord below the level of the injury, which results in an unopposed sympathetic response manifesting with cardiovascular symptoms (Milligan et al., 2012). Conus/cauda equine lesions lead to a lower motor neuron-type injury characterized by an areflexic, acontractile bladder with urethral sphincter weakness, thus leading to stress and overflow UI. The urinary bladder tends to be low pressure and therefore no upper urinary tract dilatation is seen.

UI in men is a potential complication of radical prostatectomy. Damage to the horse-shoe shaped EUS during dissection at the bladder neck may be responsible for post-operative UI. Furthermore, the neurovascular structures that innervate the IUS and EUS tend to be located posterolaterally and are symmetrical on either side of the prostate. These are susceptible to injury during radical prostatectomy, despite maximal efforts to carry out nerve-sparing radical procedures (Stolzenburg et al, 2007; Raychaudhuri and Cahill, 2008). The maximum urethral closure pressure (MUCP) and functional urethral length in men are lower post-radical prostatectomy. Nerve sparing radical prostatectomy produces better continence rates, longer functional urethral length and improved MUCP.

CONCLUSION

The levator ani muscle and the endopelvic fascia play an important role in supporting the pelvic organs. Damage to these structures, most commonly in females during pregnancy and childbirth, may result in pelvic organ prolapse and GSI. The neural control of micturition is a complex mechanism, with

primitive sacral spinal reflexes communicating with higher centers in the brain, allowing humans to make executive decisions about micturition. Pathology involving the higher centers can result in UI, while spinal cord injury rostral to the lumbosacral cord can result in neurogenic DO. Understanding the anatomical basis of continence and micturition enables the development of therapies to treat, non-operatively and operatively, pelvic organ prolapse and UI resulting from pelvic floor trauma. Furthermore, clinicians can minimize the risk of pelvic floor injuries that may occur during childbirth. Most importantly, an understanding of the anatomy can aid clinicians in improving significantly the quality of life of patients suffering from UI.

REFERENCES

Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, van Kerrebroeck P, Victor A, Wein A, Standardisation Sub-committee of the International Continence Society. 2002. The standardisation of terminology of lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. Neurourol Urodyn 21:167-78.

Allen C, Keane D. 2005. Pathophysiology of urinary incontinence. Rev Gynaecol Pract 5:65-70.

Apodaca G, Balestreire E, Birder LA. 2007. The uroepithelial-associated sensory web. Kidney Int 72:1057-64.

Ashton-Miller JA, DeLancey JO. 2007. Functional anatomy of the female pelvic floor. Ann N Y Acad Sci 1101:266-96.

Bacsu CD, Chan L, Tse V. 2012. Diagnosing detrusor sphincter dyssynergia in the neurological patient. BJU Int 109:31-4.

Bharucha AE. 2006. Pelvic floor: anatomy and function. Neurogastroenterol Motil 18:507-19.

Birder LA, Apodaca G, de Groat WC, Kanai AJ. 1998. Adrenergic- and capsaicin-evoked nitric oxide release from urothelium and afferent nerves in urinary bladder. Am J Physiol 275:F226-9.

Birder LA. 2006. Urinary bladder urothelium: molecular sensors of chemical/thermal/mechanical stimuli. Vascul Pharmacol 45:221-6.

Birder LA, de Groat WC. 2007. Mechanisms of disease: involvement of the urothelium in bladder dysfunction. Nat Clin Pract Urol 4:46-54.

Birder LA, Kanai AJ, Cruz F, Moore K, Fry CH. 2010. Is the urothelium intelligent? Neurourol Urodyn 29:598-602.

Birder LA. 2011. Urothelial signaling. Handb Exp Pharmacol 202:207-31.

Birder LA. 2013. Nervous network for lower urinary tract function. Int J Urol 20:4-12.

Birder L, Andersson KE. 2013. Urothelial signaling. Physiol Rev 93:653-80.

Burnstock G. 2013. Purinergic signalling in the lower urinary tract. Acta Physiol (Oxf) 207:40-52.

de Groat WC. 2006. Integrative control of the lower urinary tract: preclinical perspective. Br J Pharmacol 147:S25-40.

de Groat WC, Yoshimura N. 2006. Mechanisms underlying the recovery of lower urinary tract function following spinal cord injury. Prog Brain Res 152:59-84.

de Groat WC, Yoshimura N. 2010. Changes in afferent activity after spinal cord injury. Neurourol Urodyn 29:63-76.

de Groat W C, Yoshimura N. 2012. Plasticity in reflex pathways to the lower urinary tract following spinal cord injury. Exp Neurol 235:123-32.

DeLancey JO. 1994a. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol 170:1713-20.

DeLancey JO. 1994b. The anatomy of the pelvic floor. Curr Opin Obstet Gynecol 6:313-6.

DeLancey JO, Kearney R, Chou Q, Speights S, Binno S. 2003. The appearance of levator ani muscle abnormalities in magnetic resonance images after vaginal delivery. Obstet Gynecol 101:46-53.

Delancey JO, Ashton-Miller JA. 2004. Pathophysiology of adult urinary

incontinence. Gastroenterology 126:S23-32.

Dietz HP, Lanzarone V. 2005. Levator trauma after vaginal delivery. Obstet Gynecol 106:707-12.

Downie JW, Karmazyn M. 1984. Mechanical trauma to bladder epithelium liberates prostanoids which modulate neurotransmission in rabbit detrusor muscle. J Pharmacol Exp Ther 230:445-9.

Drake MJ, Fowler CJ, Griffiths D, Mayer E, Paton JF, Birder L. 2010. Neural control of the lower urinary and gastrointestinal tracts: supraspinal CNS mechanisms. Neurourol Urodyn 29:119-27.

Ferguson DR, Kennedy I, Burton TJ. 1997. ATP is released from rabbit urinary bladder epithelial cells by hydrostatic pressure changes--a possible sensory mechanism? J Physiol 505:503-11.

Fowler CJ. 2006. Integrated control of lower urinary tract--clinical perspective. Br J Pharmacol 147:S14-24.

Fowler CJ, Griffiths D, de Groat WC. 2008. The neural control of micturition. Nat Rev Neurosci 9:453-66.

Fry CH, Ikeda Y, Harvey R, Wu C, Sui GP. 2004. Control of bladder function by peripheral nerves: avenues for novel drug targets. Urology 63:24-31.

Fry CH, Sui GP, Kanai AJ, Wu C. 2007. The function of suburothelial myofibroblasts in the bladder. Neurourol Urodyn 26:914-9.

Gabella G, Davis C. 1998. Distribution of afferent axons in the bladder of rats. J

Neurocytol 27:141-55.

Gosling JA, Dixon JS. 1974. Sensory nerves in the mammalian urinary tract. An evaluation using light and electron microscopy. J Anat 117:133-44.

Griffiths D, Tadic SD. 2008. Bladder control, urgency, and urge incontinence: evidence from functional brain imaging. Neurourol Urodyn 27:466-74.

Grol S, van Koeveringe GA, de Vente J, van Kerrebroeck PE, Gillespie JI. 2008. Regional differences in sensory innervation and suburothelial interstitial cells in the bladder neck and urethra. BJU Int 102:870-7.

Habler HJ, Janig W, Koltzenburg M. 1990. Activation of unmyelinated afferent fibres by mechanical stimuli and inflammation of the urinary bladder in the cat. J Physiol 425:545-62.

Holstege G. 2005. Micturition and the soul. J Comp Neurol 493:15-20.

Holstege G. 2010. The emotional motor system and micturition control. Neurourol Urodyn 29:42-8.

Hoyte L, Damaser MS, Warfield SK, Chukkapalli G, Majumdar A, Choi DJ, Trivedi A, Krysl P. 2008. Quantity and distribution of levator ani stretch during simulated vaginal childbirth. Am J Obstet Gynecol 199:198.e1-5.

Jen PY, Dixon JS, Gosling JA. 1995. Immunohistochemical localization of neuromarkers and neuropeptides in human fetal and neonatal urinary bladder. Br J Urol 75:230-5.

Jung J, Ahn HK, Huh Y. 2012. Clinical and functional anatomy of the urethral

sphincter. Int Neurourol J 16:102-6.

Kavia RB, Dasgupta R, Fowler CJ. 2005. Functional imaging and the central control of the bladder. J Comp Neurol 493:27-32.

Kearney R, Sawhney R, DeLancey JO. 2004. Levator ani muscle anatomy evaluated by origin-insertion pairs. Obstet Gynecol 104:168-73.

Kullmann FA, Artim D, Beckel J, Barrick S, de Groat WC, Birder LA. 2008. Heterogeneity of muscarinic receptor-mediated Ca2+ responses in cultured urothelial cells from rat. Am J Physiol Renal Physiol 294:F971-81.

Lien KC, Mooney B, DeLancey JO, Ashton-Miller JA. 2004. Levator ani muscle stretch induced by simulated vaginal birth. Obstet Gynecol 103:31-40.

Macura KJ, Genadry RR. 2008. Female urinary incontinence: pathophysiology, methods of evaluation and role of MR imaging. Abdom Imaging 33:371-80.

McCloskey KD. 2010. Interstitial cells in the urinary bladder--localization and function. Neurourol Urodyn 29:82-7.

McCloskey KD. 2013. Bladder interstitial cells: an updated review of current knowledge. Acta Physiol (Oxf) 207:7-15.

Milligan J, Lee J, McMillan C, Klassen H. 2012. Autonomic dysreflexia: recognizing a common serious condition in patients with spinal cord injury. Can Fam Physician 58:831-5.

Minassian VA, Drutz HP, Al-Badr A. 2003. Urinary incontinence as a worldwide problem. Int J Gynaecol Obstet 82:327-38.

Molinuevo B, Batista-Miranda JE. 2012. Under the tip of the iceberg: psychological factors in incontinence. Neurourol Urodyn 31:669-71.

Namasivayam S, Eardley I, Morrison JF. 1999. Purinergic sensory neurotransmission in the urinary bladder: an in vitro study in the rat. BJU Int 84:854-60.

Novellas S, Chassang M, Verger S, Bafghi A, Bongain A, Chevallier P. 2010. MR features of the levator ani muscle in the immediate postpartum following cesarean delivery. Int Urogynecol J 21:563-8.

Oberwalder M, Thaler K, Baig MK, Dinnewitzer A, Efron J, Weiss EG, Vernava AM, Nogueras JJ, Wexner SD. 2004. Anal ultrasound and endosonographic measurement of perineal body thickness: a new evaluation for fecal incontinence in females. Surg Endosc 18:650-4.

Oswald J, Heidegger I, Steiner E, Brenner E, Ladurner Rennau M, Pichler R, Becker T, Loidl W, Horninger W, Fritsch H. 2013. Gender-related Fetal Development of the Internal Urethral Sphincter. Urology 82:1410-5.

Park JM, Bloom DA, McGuire EJ. 1997. The guarding reflex revisited. Br J Urol 80:940-5.

Perry S, Shaw C, Assassa P, Dallosso H, Williams K, Brittain KR, Mensah F, Smith N, Clarke M, Jagger C, Mayne C, Castleden CM, Jones J, McGrother C.

2000. An epidemiological study to establish the prevalence of urinary symptoms and felt need in the community: the Leicestershire MRC Incontinence Study. Leicestershire MRC Incontinence Study Team. J Public Health Med 22:427-34.

Pullen AH, Tucker D, Martin JE. 1997. Morphological and morphometric characterisation of Onuf's nucleus in the spinal cord in man. J Anat 191:201-13.

Raychaudhuri B, Cahill D. 2008. Pelvic fasciae in urology. Ann R Coll Surg Engl 90:633-7.

Royal College of Physicians. 1995. Incontinence: causes, management and provision of services.

Sand PK, Ostergard DR. 1995. The Low Compliance Bladder. In Urodynamics and the Evaluation of Female Incontinence, 86-87. Springer London.

Sebe P, Fritsch H, Oswald J, Schwentner C, Lunacek A, Bartsch G, Radmayr C. 2005. Fetal development of the female external urinary sphincter complex: an anatomical and histological study. J Urol 173:173----------*8-42.

Shafik A. 2000. Neuronal innervation of urethral and anal sphincters: surgical anatomy and clinical implications. Curr Opin Obstet Gynecol 12:387-98.

Shobeiri SA, Leclaire E, Nihira MA, Quiroz LH, O'Donoghue D. 2009. Appearance of the levator ani muscle subdivisions in endovaginal three-

dimensional ultrasonography. Obstet Gynecol 114:66-72.

Standring S, ed. 2008. ******. Edited by S Standring. 40th ed. Spain: Churchill Livingstone.

Stolzenburg JU, Schwalenberg T, Horn LC, Neuhaus J, Constantinides C, Liatsikos EN. 2007. Anatomical landmarks of radical prostatecomy. Eur Urol 51:629-39.

Sui GP, Rothery S, Dupont E, Fry CH, Severs NJ. 2002. Gap junctions and connexin expression in human suburothelial interstitial cells. BJU Int 90:118-29.

Svabik K, Shek KL, Dietz HP. 2009. How much does the levator hiatus have to stretch during childbirth? Bjog 116:1657-62.

Takenaka A, Hara R, Soga H, Murakami G, Fujisawa M. 2005. A novel technique for approaching the endopelvic fascia in retropubic radical prostatectomy, based on an anatomical study of fixed and fresh cadavers. BJU Int 95:766-71.

Thirugnanasothy S. 2010. Managing urinary incontinence in older people. BMJ 341:c3835.

Wallner C, Dabhoiwala NF, DeRuiter MC, Lamers WH. 2009. The anatomical components of urinary continence. Eur Urol 55:932-43.

Wang EC, Lee JM, Ruiz WG, Balestreire EM, von Bodungen M, Barrick S, Cockayne DA, Birder LA, Apodaca G. 2005. ATP and purinergic receptor-

dependent membrane traffic in bladder umbrella cells. J Clin Invest 115:2412-22.

Wood MW, Breitschwerdt EB, Nordone SK, Linder KE, Gookin JL. 2012. Uropathogenic E. coli promote a paracellular urothelial barrier defect characterized by altered tight junction integrity, epithelial cell sloughing and cytokine release. J Comp Pathol 147:11-9.

Woodman PJ, Graney DO. 2002. Anatomy and physiology of the female perineal body with relevance to obstetrical injury and repair. Clin Anat 15:321-34.

Yavagal S, de Farias TF, Medina CA, Takacs P. 2011. Normal vulvovaginal, perineal, and pelvic anatomy with reconstructive considerations. Semin Plast Surg 25:121-9.

Yucel S, Baskin LS. 2004. An anatomical description of the male and female urethral sphincter complex. J Urol 171:1890-7.

LEGENDS

Table 1: Differences in nomenclature between males and females of the three divisions of pubococcygeus muscle (Ashton-Miller and DeLancey, 2007; Yavagal et al., 2011; Molinuevo and Batista-Miranda, 2012).

Box 1: Summary of the clinically important functions of the perineal body.

Figure 1: Neuronal innervation of the urinary bladder.

Figure 2: Neuronal pathways involved in the guarding reflex, thereby maintaining continence during bladder filling.

Figure 3: Neuronal pathways involved in initiation of micturition.

(+) denotes stimulatory effect, (-) denotes inhibitory effect