The Pelvic Floor

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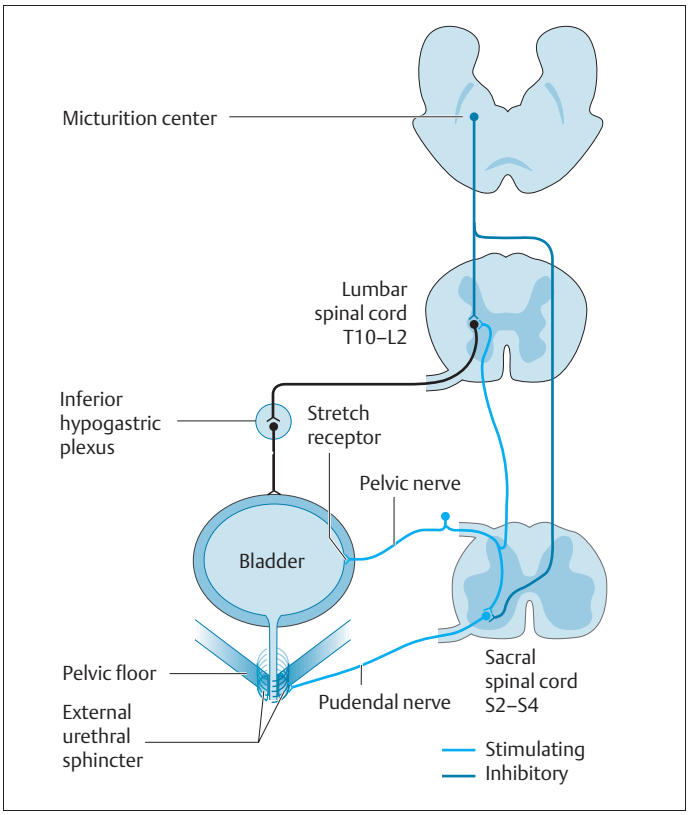

Fig. 1.**17** The nerves of the urogenital complex.

traction of the smooth muscle of the detrusor. Micturition is initiated by opening of the bladder neck, and is continued into dilation of the urethra, allowing urine to flow unimpeded. In the male, the bladder neck is also subject to adrenergic control, closing the bladder during ejaculation.

The lower urinary tract is therefore regulated by a relatively simple reflex arc, which, however, is under the influence of the central nervous system (pontine micturition center, sacral spinal cord) [Merkle 1997].

From a physiotherapeutic point of view, it should be noted that the musculature of the pelvic floor takes part in the physiologic control of urinary outflow. The relaxation of the muscles of the pelvic floor needed for micturition is facilitated by certain body postures, such as sitting [Wennergren and Oberg 1995]. On the other hand, in other postures muscular activity increases and so supports continence. These two examples are linked to interactions of the pelvic floor muscles with central motor programming and synergic patterns [Shumway-Cook and Woollacott 2001].

The Anorectal Complex

The anal hiatus lies in the posterior part of the fibrous arch of the levator. The anal canal, surrounded by the external anal sphincter, passes through it to its external opening. As in the urogenital hiatus, striated and smooth muscles meet in this hiatus, where they are important factors in maintaining fecal continence. When either somatic or autonomic minor motor problems occur, the plasticity of the CNS should accommodate to maintain function. That adaptation or plasticity will occur more easily following motor learning principles. (See the section on motor learning and motor control, pp. 21–35, for additional information.)

The striated musculature in this area consists of parts of the external anal sphincter, which receives its somatic innervation through the pudendal nerve (S2–S4) (Fig. 1.**18**). Superiorly, the sling of the puborectalis is continuous with the striated external anal sphincter. Its somatic innervation is mixed, with afferents from both the pudendal nerve and the sacral plexus com-

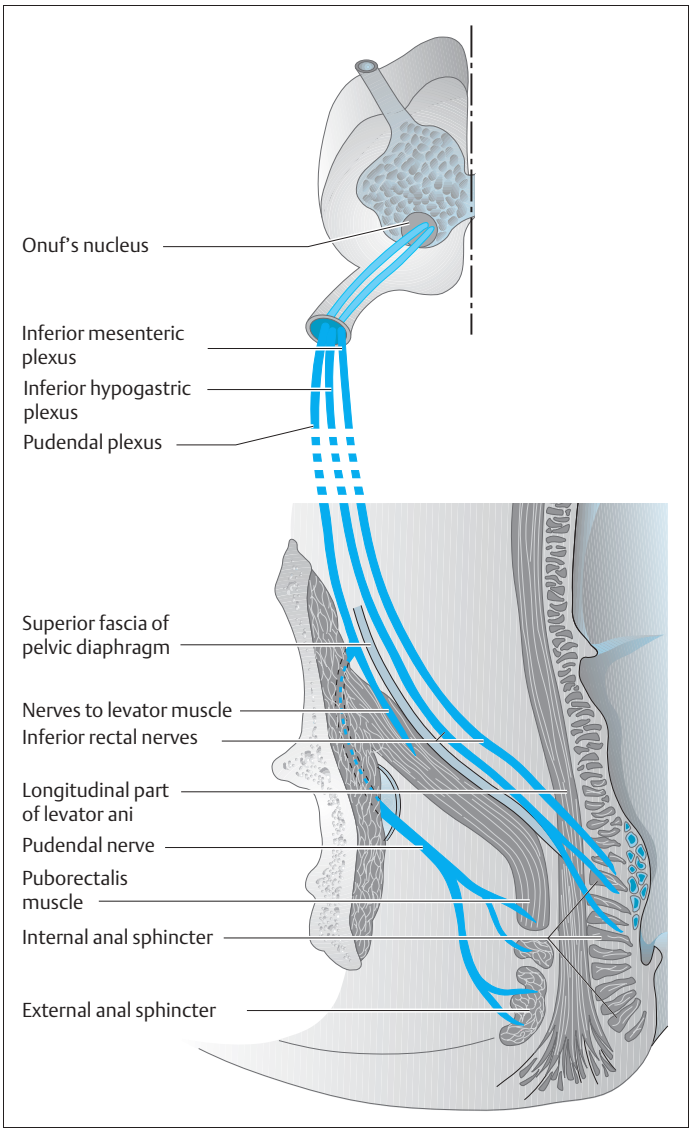

Fig. 1.**18** The nerves of the anorectal complex.

municating sensory information to the CNS [Roberts et al. 1988].

The motoneurons for the striated sphincters originate lateral to the anterior horn cells of the respective segments of the spinal cord (Fig. 1.**18**). This collection of cells is known as Onuf's nucleus [Schröder 1981].

The smooth muscles of the internal anal sphincter and the outer longitudinal layer are innervated by the autonomic nervous system. However, the mechanisms are less well known than those of the smooth muscle of the urinary system. What is accepted is that this region receives sympathetic efferents from the inferior thoracic cord and the lumbar cord (T6–L2), as well as parasympathetic efferents from S2–S4 (pelvic splanchnic nerves). Yamamoto et al. [1978] determined the presence of nerve cell collections in Onuf's nucleus supplying the smooth muscle of the internal anal sphincter, while demonstrating that the nuclei for the striated and smooth parts of the sphincter are difficult to separate. The autonomic nerves reach the rectum and anal canal by way of the inferior hypogastric

plexus. Apart from this extramural autonomic nerve supply, both of these organs possess an intramural nervous network [Wedel et al. 1999] similar to that of the colon segments above them. The structure and function of this network have not yet been adequately defined.

Tonic contraction of both sphincters closes the anal canal, with the internal anal sphincter contributing about 80% and the external anal sphincter about 20% [Stelzner 1998, Schäfer and Enck 1997]. The closure of the anal canal is also supported by the puborectalis muscle, which contributes to a sharp angling of the anorectal angle, which in turn prevents passage of the stool from the rectum into the anal canal. Schumann [1986] was able to show that the physiologic strength of the puborectalis muscle is distinctly greater in the male than in the female, and this is in agreement with morphologic investigations of the structure of the sphincter apparatus [Fritsch et al. 2002]. According to this work, the anal canal in the female is closed voluntarily by perineal contraction occurring below the level of the urethral and vaginal openings. The external closure of the intestinal tube by muscular contraction is further enhanced internally by vascular engorgement of the corpus cavernosum recti (arteriovenous plexus).

Like micturition, defecation is regulated by a nervous reflex arc, the details of which, however, have not yet been completely clarified. Initially, the wall of the rectal ampulla is distended by an increase in fecal mass. The anatomic location of the corresponding stretch receptors has not been defined. It has been established, however, that the response to an increase in tension in the wall of the rectal ampulla is contraction of the sphincters. When the rectum empties, the sphincters are relaxed, although the striated sphincter can "intervene" at any time and postpone defecation for a time by contracting voluntarily.

Normal defecation is aided by contraction of the anterior abdominal muscles, especially the transversus abdominis. This increases intra-abdominal pressure, and this—in contrast to the brief rise in intra-abdominal pressure accompanying coughing and sneezing—is accompanied by relaxation of the pelvic floor, including the puborectalis muscle. Practicing this motor control as a small child leads to the capacity of the CNS for motor learning and thus rectal control. As an individual over time practices the interaction of muscle groups during defecation and micturition, these patterns become motor programs. Following injury that causes loss of function in sensory, motor, or both neurons, the patient may experience an imbalance in function or response in the learned motor programs. Strengthening weak muscles in isolation from the motor programs will not guarantee that the established power will automatically transform into adequate motor programs. Linking motor learning with motor control and the interactions of sensory input and motor pattern responses will lead to the most efficacious interventions. For example, a child initially has no control over emptying the rectum. The elimination is automatic/reflexive and a response to tension within the rectum. The child learns through repetition and practice to sense afferent stimuli coming from the increased tension within the rectum. Simultaneously, the child learns to inhibit reflexive relaxation of the striated sphincter and thus learns bowel control. The child learns and thus practices releasing fecal matter in a seated position or squatting, depending on the local culture. In both functional positions gravity and the abdominal muscles are used to assist in elimination. Thus, sitting and squatting become the environmentally specific functional positions that a patient must practice prior to being functionally independent in both retention and elimination. Another spatial position might initially be incorporated, such as supine, with the legs over a gymnastic ball. This position takes away the stretch to the pelvic muscles and postural stability of the pelvic floor and back muscles needed for sitting. The patient can practice pelvic tilts while contracting and relaxing the anal region. The therapist should be looking for anal contraction during both anterior and posterior tilts, as well as relaxation during those movements. As the patient is moving the ball toward and away from the hips, during tilts and squeeze/release movements he or she will be practicing a variety of motor programs. The therapist can then move the patient to the sitting position and continue with the same exercises. This upright position requires integration of postural programs of the back and their interaction with abdominal muscles for stabilization of the trunk in vertical. Progressing first to a hard base, which eliminates the organ stretch to the pelvic floor, should be easier than a toilet surface that increases the pull of gravity on the organs and the need for sphincter contraction to retain either feces or urine. Treatment has progressed toward using a natural environment that will lead to control of both elimination and retention. By understanding the in-

teractions between the striated and smooth-muscle anatomy with motor learning and control, the therapist should be able to individualize the intervention while maintaining a highly effective treatment environment.

Levator Ani–Gluteus Maximus Complex

The levator ani is without a doubt the key structure in the female pelvic floor. Its function is supported by the fat body of the ischioanal fossa and the gluteus maximus, both of which form a functional unit with the levator ani. Anatomically, the two muscles are connected by strong connective-tissue septa that cross the ischioanal fossa (Fig. 1.**19**). In a functional magnetic resonance image, the fat body of the ischioanal fossa can be observed to shift cranially when the gluteus maximus contracts. This shift is accompanied by a synchronous movement of the levator ani [Janssen et al. 2001]. Thus, the levator ani, ischioanal fossa, and gluteus maximus form an anatomically and functionally interlocking complex (the LFG complex) that is of great importance for the functional integrity of the posterior compartment of the pelvic floor and for learned motor programs.

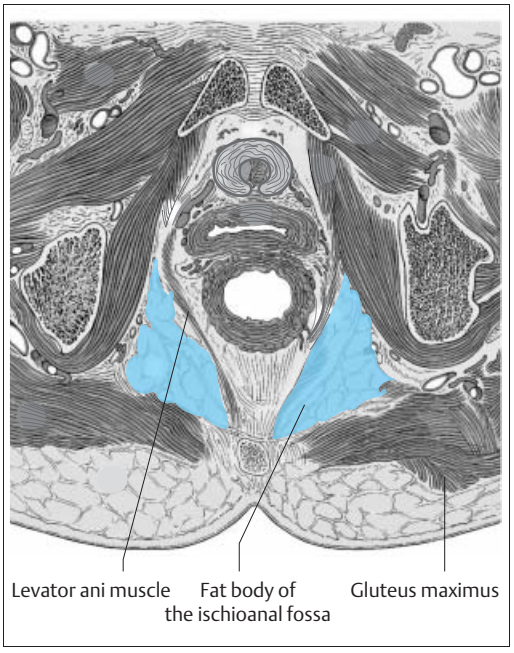

Fig. 1.**19** The levator ani–gluteus maximus complex.

Aging of Pelvic Floor Muscles

It is generally accepted that the pelvic floor is subject to the physiological process of aging. However, there have been few reliable investigations of this process.

Functional changes in the lower urinary tract occur in the process of aging in both sexes. These are expressed specifically in a diminished bladder capacity, weaker urinary flow and increased nocturia—i. e., essentially tied to the smooth-muscle structures. A tendency to weaker detrusor contractions has been demonstrated in postmenopausal women (Malone-Lee and Wahedna 1993, Sommer et al. 1990]. With weakness within the detrusor, abdominal contraction could be used to compensate. Teaching the CNS when to contract the abdominal muscles with relaxation of the external sphincter for micturition, versus when to tighten the abdominal muscles when standing up from a chair or sneezing, while tightening the external sphincter to avoid micturition, requires two motor programs and thus repetitive practice in both patterns.

The functional changes of aging in the anorectal region can be attributed to both the smooth-muscle internal anal sphincter and the striated external anal sphincter. While resting pressure in the internal anal sphincter decreases with age and the muscle undergoes wasting, voluntary activity of the external anal sphincter diminishes. This has been discussed in connection with alterations in the composition of fibers of these muscles [Schäfer and Enck 1997]. The plasticity of the CNS and its adaptive behavior in overcoming these changes and maintaining function can be optimized by linking voluntary anal sphincter control with motor programs that increase intra-abdominal pressure in order to practice functional movement control of the external sphincter.

In women, old injuries to the pelvic floor sustained during labor may impair the function of the organs involved in continence and of the levator ani. The connective tissue within the pelvic floor, as well as the muscle fibers themselves, have often been stretched during the latter phases of pregnancy and during delivery. If pelvic floor reconditioning is not made an integral part of the normal functional movement, these fibers can remain stretched. With age, this motor control problem often becomes exacerbated, and the ability of the CNS to adapt is limited. Practicing pelvic patterns that encourage tightening during functional activities has the greatest potential

for pelvic floor control—for example, sitting on a compliant surface (e. g., a ball or a dome rocker). Preventing or discouraging patterns that would stretch the floor, such as prolonged sitting on a commode, can be recommended.

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References

Burnett AL, Wesselmann U. History of the neurobiology of the pelvis. Urology 1999; 53:1082–9.

DeGroat WC, Booth AM. Synaptic transmission in the pelvic ganglia. In: Maggi CA, ed. Nervous control of the urogenital system. Chur, Switzerland: Harwood Academic Publishers, 1993: 291–347.

Elbadawi A, Schenk EA. A new theory of the innervation of bladder musculature, 2: innervation of the vesicourethral junction and external urethral sphincter. J Urol 1974; 111:613–5.

Federative Committee on Anatomical Terminology, International Federation of Associations of Anatomists. Terminologia anatomica: international anatomical terminology. Stuttgart: Thieme, 1998.

Fritsch H. Topography of the pelvic autonomic nerves in human fetuses between 21–29 weeks of gestation. Anat Embryol 1989; 180:57–64.

Fritsch H. [Development of the rectal fascia; in German.] Anat Anz 1990; 170:273–80.

Fritsch H. Topography and subdivision of the pelvic connective tissue in human fetuses and in the adult. Surg Radiol Anat 1994; 16:259–65.

Fritsch H, Fröhlich B. Development of the levator ani muscle in human fetuses. Early Hum Dev 1994; 37:15–25.

Fritsch H, Kühnel W, Stelzner F. [Development and clinical anatomy of the rectal adventitia: significance for radical operation of rectal carcinoma; in German.] Langenbecks Arch Chir 1996; 381:237–43.

Fritsch H, Brenner E, Lienemann A, Ludwikowski B. Anal sphincter complex: reinterpreted morphology and its clinical relevance. Dis Colon Rectum 2002; 45:188–94.

Fritsch H, Lienemann A, Brenner E, Ludwikowski B. Clinical anatomy of the pelvic floor. Adv Anat Embryol Cell Biol 2004; 175:iii–ix, 1–64.

Gasparri F, Brizzi E. Significato anatomo-chirurgico delle formazioni connetivali del piccolo bacino. Arch Ital Anat Embryol 1951; 66:151–169.

Janssen U, Lienemann A, Fritsch H. Die Bedeutung des M. levator ani-Fossa ischioanalis-Glutaeus maximus (LFG)-Komplexes für den weiblichen Beckenboden. Ann Anat 2001; 183(Suppl):11.

Juenemann KP, Lue TF, Schmidt RA, Tanagko RA. Clinical significance of sacral and pudendal nerve anatomy. J Urol 1988; 139:74–80.

Kandel ER, Schwartz JH, Jessel TM. Principles of neural science, 4th ed. New York: McGraw-Hill, 2000.

Kuo AD. The relative roles of feedforward and feedback in the control of rhythmic movements. Motor Control 2002; 6:129–145

Lepor H, Gregerman M, Crosby R, Mostofi FK, Walsh PC. Precise localization of the autonomic nerves from the pelvic plexus to the corpora cavernosa: a detailed anatomical study of the adult male pelvis. J Urol 1985; 133:207–12.

Malone-Lee J, Wahedna I. Characterisation of detrusor contractile function in relation to old age. Br J Urol 1993; 72:873–80.

Merkle W. Physiologie und Pathophysiologie der Miktion. In: Jost WH, ed. Neurologie des Beckenbodens. Weinheim, Germany: Chapman & Hall, 1997: 39–45.

Oelrich TM. The striated urogenital sphincter muscle in the female. Anat Rec 1983; 205:223–32.

Pernkopf E. Pernkopf anatomy: atlas of topographic and applied human anatomy, ed. Platzer W, trans. Monsen H. 3rd ed. Baltimore: Urban and Schwarzenberg, 1989.

Roberts WH, Harrison CW, Mitchell DA, Fischer HF. The levator ani muscle and the nerve supply of its puborectalis component. Clin Anat 1988; 1:267–283.

Sahrmann SA. Diagnosis and treatment of movement impairment syndromes. St. Louis: Mosby, 2002: 19–23.

Sapolsky RM. Stress and plasticity in the limbic system. Neurochem Res 2003; 28:1735–42.

Schäfer R, Enck P. Physiologie und Pathophysiologie der Defäkation. In: Jost WH, ed. Neurologie des Beckenbodens. Weinheim, Germany: Chapman & Hall, 1997: 15–22.

Schröder H. Onuf's nucleus X: a morphological study of a human spinal nucleus. Anat Embryol (Berl) 1981; 162:443–53.

Schumann J. Physiologische Grundlagen kontinenzerhaltender Eingriffe am Rektum und Anus. Stuttgart: Thieme, 1986.

Shumway-Cook A, Woollacott MH. Motor control theory and practical application, 2nd ed. Baltimore: Lippincott, Williams & Wilkins, 2001.

Sommer P, Bauer T, Nielsen KK, Kristensen ES, Hermann GG, Steven K, et al. Voiding patterns and prevalence of incontinence in women: a questionnaire survey. Br J Urol 1990; 66:12–5.

Stelzner F. Die anorektale Kontinenz. In: Stelzner F, ed. Chirurgie an den viszeralen Abschlusssystemen. Stuttgart: Thieme, 1998: 121–125.

Stelzner F, Fritsch H, Fleischhauer K. Die chirurgische Anatomie der Genitalnerven des Mannes und ihre Schonung bei der Exzision des Rektums. J Chirurg 1989; 60: 228–234.

Umphred DA. The limbic system: influence over motor control and learning. In: Umphred DA, ed. Neurological rehabilitation, 4th ed. St. Louis: Mosby, 2001: 148–177.

Walsh PC, Donker PJ. Impotence following radical prostatectomy: insight into etiology and prevention. J Urol 1982; 128:492–7.

Wedel T, Roblick U, Gleiss J, Schiedeck T, Bruch HP, Kühnel W, et al. Organization of the enteric nervous system in the human colon demonstrated by wholemount immunohistochemistry with special reference to the submucous plexus. Ann Anat 1999; 181: 327–37.

Wennergren J, Oberg B. Pelvic floor exercises for children: a method of treating dysfunctional voiding. Br J Urol 1995; 76:9–15.

Yamamoto T, Samoti H, Ise H. Sacral spinal innervations of the rectal and vesical smooth muscle and sphincter striated muscles. Neuroscience 1978; 7:47.

1.2 The Nervous System and Motor Learning

Darcy Umphred

Introduction

Therapists throughout the world are constantly asked to examine, evaluate, and intervene with individuals who present large variations in movement problems that affect their everyday activities and quality of life. These functional movement limitations generally arise from musculoskeletal, neuromuscular, cardiopulmonary, and integumentary problems, although organ, digestive, endocrine, and a variety of other systems may also interact and cause motor problems. Each system presents the therapist with different challenges, and many problems within each system can lead to pelvic floor dysfunction. No matter the disease or pathology that has led to the dysfunction, a large component of the movement itself is controlled and modified by the central nervous system (CNS) and its interaction with the way in which the muscles function within the pelvic floor. Conceptual models of the regulation and control of movement by the CNS have changed dramatically over the last few decades. In the 1960s, therapists were taught that movement control within the nervous system was developmental and hierarchically based, hard-wired, and functioned by feedback. Today, students learn that the motor output from the nervous system is based on a dynamic systems model in which there is distributed function, consensus, and a feedforward program that is soft-wired and constantly changing depending on practice, environmental demands, and internal restraints within the individual. Concepts and current research relevant to the regulation of movement by the central nervous system affect the way therapists analyze clinical problems, establishing the scientific rationale for selecting appropriate interventions; each physician therefore needs to ensure continuing education. Discussion of the CNS principles of motor learning, motor control, neuroplasticity, and the neural substrates underlying the way in which these concepts and principles apply to pelvic floor dysfunction is just one piece of a much larger puzzle relating to human performance and movement. The goal of this section is to provide an opportunity to identify these CNS variables and analyze how and why they may affect the outcome of an intervention.

Motor Control and Motor Learning

Before discussing the specific concepts behind motor control and motor learning, the basis for the general concepts used needs to be clearly identified. Motor control is the way in which the CNS controls motor performance, and the study of it developed from the sciences of neurophysiology and neuropsychology. Motor control measurements are made in units of milliseconds, and these continue to provide basic research data leading to a better understanding of the way in which the CNS controls behavior [Kandel et al. 2000, Shumway-Cook and Woollacott 2001]. Motor learning is not defined in millisecond responses, but rather as the ability of the CNS to retain movement programs and then transfer those programs into new environments.