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Special Contribution

The Politics of Prolapse: A Revisionist Approach to Disorders of the Pelvic Floor in Women*

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> *'And this, he said, is the reason why the cure of many diseases is unknown to the physicians of Hellas, because they disregard the whole, which ought to be studied also; for the part can never be well unless the whole is well.' Socrates, in Plato's* Charmides [1]

Pelvic floor dysfunction, particularly as manifested by genital prolapse and urinary or fecal incontinence, remains one the the largest unaddressed issues in women's health care today. These problems result in substantial social embarrassment, emotional distress and physical discomfort, and are the cause of tens of thousands of surgical operations each year. However, many women with these afflictions continue to bear them stoically in resigned silence, regarding them as normal and inevitable parts of aging – which they are not. The economic costs of these problems are also immense and similarly unappreciated. At at consensus development conference held in October 1988, for example, the National Institutes of Health estimated the total direct and indirect costs of managing adult urinary incontinence alone at $10.3 billion per year – far more than the current costs of the AIDS epidemic [2]. While the public at large has remained oblivious to these facts, the paper products industry has launched a multi-million-dollar campaign to promote the sales of a vast array of absorbent pads, panty-liners, and undergarments in hope of opening up this gigantic source of potential profits. Despite the enormity of these problems and our longstanding clinical experience in treating them, however, prolapse recurring after an attempt at surgical repair remains a significant clinical problem, and the approach to uterine prolapse by gynecologic surgeons appears to have changed little in 60 years.

Why? What has led to such an impasse? Why has our thinking about these problems remained so narrow and so unfruitful? We propose that this is largely due to the compartmentalization of the pelvic floor into unnatural spheres of influence by competing medical specialities, with resultant neglect of the interrelationships among the pelvic organ systems.

Over the past 2000 years western medicine has dramatically narrowed its focus and changed its preoccupations. Greek medicine, which dominated medical thought in many ways until the 17th and 18th centuries, viewed illness largely as a disruption of *generalized* bodily processes, an imbalance among four humors whose interrelationships constituted the foundations of human pathology. The rise of empiric and experimental science gradually replaced this conception of illness with one which saw it as arising from *specific* disease processes in a *local* group of tissues or organs. This reorganization of medicine around the 'anatomic idea' led to the development of specialties dealing with disorders of specific organ systems: ophthalmology, cardiology, gastroenterology, urology, gynecology etc. [3–5]. In the 20th century this process has been hastened by the development of techniques which permit specialized examination of discrete organ systems – and which also allow large professional fees to be collected by the specialists capable of performing these procedures [6]. This financial factor has created a 'territorial impera-

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tive' in medical practice, as specialties fight to control their own turf – as any gynecologist who has gotten into a fight over the use of a sigmoidoscope or a cytoscope can attest. The organ systems have become narrow medical kingdoms with their own little boundaries and specialized sets of data, and woe betide the unwary practitioner who traverses a border without the proper stamp on his passport.

The 'Hole' Pelvis and the 'Whole' Pelvis

Nowhere is this fragementation more evident in the pelvic floor, where gynecologists, colorectal surgeons, gastroenterologists and urologists all practice within millimetres of one another and yet have little interaction with each other concerning the disorders which they treat.

Examination of a female patient immediately reveals that there are three 'holes' passing through the pelvic floor, each of which has its own doctor and its own medical speciality. The urethra and bladder belong to the urologist; the vagina and female genital organs belong to the gynecologist; the colon and rectum belong to the gastroenterologist and the colorectal surgeon. Each specialty has its own narrow view and its own vested scientific and economic interests. Each specialty fights to keep others from trespassing in its own 'hole' (Fig. 1). We feel that the time has come for a fresh look at this situation and its effect on our treatment of pelvic floor disorders in women.

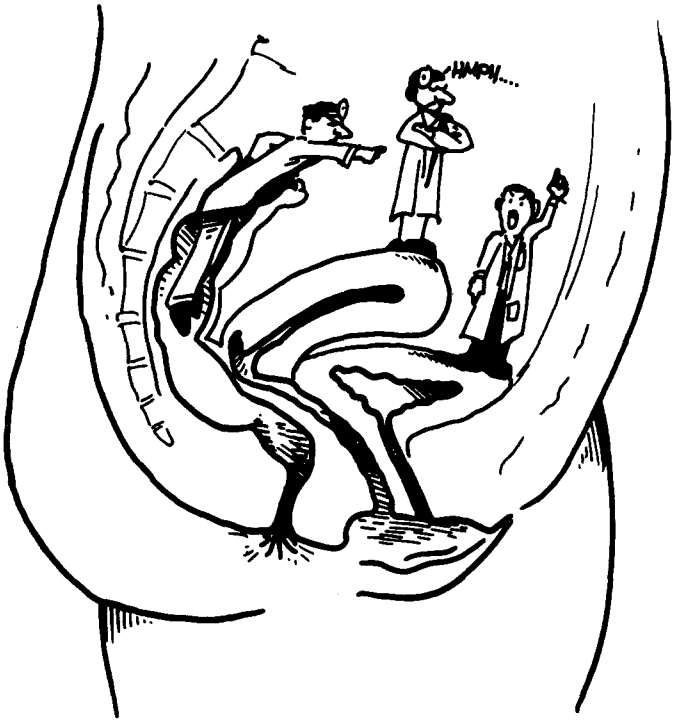

Fig. 1. Territorial Imperatives at work on the pelvic floor. A gynecologist, a urologist, and a colorectal surgeon quarrel with each other while ignoring the common ground on which they all stand.

While urologists rarely venture into the rectum, and colorectal surgeons rarely trespass in the urinary tract, the central location held by gynecologists should place them in an ideal position to see how often disorders of the other two passageways adversely affect the female patient. In spite of this fact, however, the most recent attempts to examine the pelvic floor as a whole have originated with Michael Swash, a neurologist, and the late Sir Alan Parks, a colorectal surgeon. This has reawakened our conviction that the problem of pelvic support lies not in the hollow viscera, which plummet downward as their support fades, but rather in the tissue which surround them.

The three organ systems upon which the several specialties are based would fall to the ground were it not for the connective tissue and muscle that surround them. The pelvic cavity is not half empty with three cavernous holes in it: it is half full of abundant muscle and connective tissue. Perhaps our inability to solve some of the difficult problems of pelvic floor dysfunction and pelvic floor support lies in our overemphasis on organ systems, and a nosology of dysfunction which has been built up piecemeal by three separate clinical specialties looking at isolated sections of the problem through their own sets of 'organ system' blinders. The gynecologist primarily speaks of fascia, the colorectal surgeon of muscle, and the urologist, because of his experience with spinal cord injuries, appreciates many neurological aspects of pelvic organ dysfunction overlooked by the other two. The progress we seek to make in the treatment of pelvic floor dysfunction in women will come most rapidly if we develop a unified, cross-disciplinary approach to disorders of pelvic support.

The Problem: What You See is *Not* What You Get

The history of gynecologic observations concerning genital support has been summarized in two excellent books [7,8] and need not be recapitulated here. It is clear, however, that although a number of authors [9–11] have drawn attention to the role of muscular damage in pelvic floor dysfunction, most gynecologists consider prolapse to be the result of damage to ligaments. Why do gynecologists have so little understanding of the musculature of the pelvic floor? After all, as practicing clinicians they encounter it thousands of times each year. They palpate it during pelvic examination, put stitches into it during posterior colporrhaphy, watch it rotate the fetal head, which then stretches (and sometimes tears) it during labor and delivery. Despite the extensive opportunities for observing it, comprehension of this muscular complex still lies out of reach, even as it did 100 years ago when Dickinson wrote 'There is no considerable muscle in the body the form and function of which are more difficult to understand than those of the levator ani, and about which such nebulous impressions prevail' [12].

In part this is because the pelvic floor is the environ-

ment in which the events which really capture our attention take place, and not the actor at center stage. It surrounds and directs the fetus during parturition. It is pushed aside during vaginal hysterectomy. In spite of its overwhelming importance for normal pelvic function in women, few clinicians have actually seen the levator ani muscles or could describe their anatomy with any precision. They remain deep and mysterious in both form and function.

To some extent this is because what is found on clinical examination is not what we have been led to expect. Since this is not a muscle which is readily seen, we tend to think of it in terms of diagrams and drawings from anatomy books half-remembered from the first year of medical school. Unfortunately, the pelvic muscles of the living bear little resemblance to those of the dead, from whence our artistic conceptions arise [11,13]. The drawings in anatomy books often come from cachectic and aged individuals with little muscle left. Because the levator ani maintains its position in the pelvis of a living woman through continuous activity [14], the permanent and irreversible loss of contractility brought on by death causes the levator to lose its normal position. When embalming takes place, the preservative fluid which is pumped into the cadaveric arterial system causes the bowel and abdominal viscera to swell, distending the abdomen and driving the levator plate downwards into a grossly distorted position. While these muscles form a horizontal shelf in the living, in the embalmed cadaver they exist as the sloping sides of a deep and unnatural crater.

The difficulty in coming to grips with the muscles of the pelvic floor is compounded by several other factors. While other muscles originate from or insert onto adjacent bones connected by a joint, the muscles of the levator ani have a complex shape and function which makes them different. They do not fit our notion of what muscles 'ought' to do. Even anatomists still disagree about the proper nomenclature of this muscular complex. Finally, neuromuscular physiology and biomechanics have not been areas of interest to obstetrician–gynecologists.

Towards a Unified Pelvic Myology

Our past abilities to investigate these muscle groups have been crude indeed. We have tried such indirect and cumbersome techniques are taking X-rays of the pelvic floor after direct injection of radiographic medium into the levator plate [10] or coating the vagina with contrast material [15], estimating crude muscular force with a perineometer [16], and attempting to make wax molds of the vagina and pelvic floor [12]. Newer technologies such as magnetic resonance imaging are likely to give us a more accurate look at the pelvic floor, but the most exciting work has come from the application of modern electrophysiologic techniques to the muscles of pelvic support. These studies, which examine the neuromuscular unit itself, began in investigations of anorectal incontinence but have profound implications for obstetrics, gynecology, and all other clinical disciplines which touch on pelvic floor disorders.

An increasing body of scientific data suggests that aspects of many problems in all three pelvic compartments are interrelated, connected by a common neuromuscular threat. In evaluating muscle biopsies from 25 patients with anorectal incontinence in 1977, Parks, Swash and Urich found extensive histologic evidence of denervation of the external anal sphincter compared to controls [17]. They concluded that idiopathic anorectal incontinence was related to denervation of the muscular components of the anorectal sling and anal sphincter mechanism.

Since women were found to be eight times more likely than men to develop idiopathic anorectal incontinence [17], a group of colorectal surgeons began to investigate the role of obstetric trauma in producing this problem, even though this was not part of their territory. Electrophysiologic studies performed on 71 women by Snooks and co-workers 48–72 hours after delivery, and again 8 weeks later, produced some interesting revelations [18]. They found that damage to the pelvic floor was a common but unrecognized result of vaginal delivery. Immediate postpartum abnormalities were most marked in multiparae, in patients with a prolonged second stage of labor, and in women undergoing forceps delivery. While primiparae showed substantial recovery from nerve damage at follow-up 8 weeks after delivery, recovery from denervation injury was less complete among multiparae. This probably represents the effect of cumulative damage to the pelvic floor following multiple childbirths. Since their early observations, a large body of evidence documenting neurologic damage in various forms of pelvic floor dysfunction has been amassed. This evidence the demonstration of delayed pudendal nerve conduction in patients with urinary and fecal incontinence, and markedly increased fiber density in the pelvic muscles of such patients demonstrated by single-fiber electromyography [19–28].

Stimulated by such observations, gynecologist David Warrell and his group in Manchester, England, have begun to tie these findings together across the entire pelvic floor in women. Biopsies from the pubococcygeus muscles of symptomatic women with prolapse and/or stress incontinence show evidence of significant denervation of the posterior pelvic floor compared to asymptomatic women [29]. Electrophysiological studies done by the same researchers using single-fiber EMG techniques show that partial denervation and subsequent reinnervation of the pelvic floor is part of normal aging, and is increased with childbirth [30]. However, women with stress incontinence of urine, or genitourinary prolapse, or both, show a significant increase in pelvic floor denervation compared to asymptomatic women. In women with genuine stress incontinence, pudendal nerve conduction studies show markedly prolonged conduction times to both the striated periurethral muscle and the pelvic floor when compared to women with prolapse but normal urinary control. The

latter group of women had significantly delayed conduction times to the pelvic floor, but not to the urethra. In the stress incontinent women, delayed conduction time to the periurethral striated muscle was associated with decreased maximum urethral closure pressure [31]. Thus, patterns of denervation injury across the pelvic floor appear to be correlated with deficits of support and function in specific areas.

These data show us a way of integrating many different aspects of pelvic floor dysfunction. They indicate that, rather than being problems located in isolated compartments, disorders of the pelvic floor have complex interrelations linked by denervation injury, which is present in pelvic and perineal prolapse as well as fecal and stress urinary incontinence. It may be that denervation-associated loss of pelvic muscle tone leads to decrease support, and to the poor transmission of pressure across the pelvic floor, which is associated with the genesis of stress incontinence of urine [30,31]. Both perineal descent and genital tract prolapse cause increased mechanical strain on the fascial supports of the pelvic viscera, which may further weaken smooth muscle, blood vessels and connective tissue, leading to further neurological damage. The problem of the pelvic floor is therefore a problem with the structural integrity and neurological 'wiring' of the whole building, not just an isolated defect in some specialist's private 'apartment'.

The notion that pelvic floor dysfunction in women may be a neurological problem will no doubt seem foreign to many practitioners. Rather than rejecting this idea out of hand, however, thoughtful consideration will let us place this in its proper perspective. To argue over whether prolapse results from fascial damage *or* neuromuscular injury is to miss the central question, which is how do these two elements interact in the supportive function of the pelvic floor as a whole?

Basic biomechanical principles can help us understand the interactions of muscles and ligaments and may give us a clue as to how these elements are related in the pelvis. A structural analysis of the knee provides evidence that muscles and ligamentous tissues have complementary roles. Contrary to our usual beliefs, ligaments are poorly suited to sustain constant loads. This can be seen from the orthopedic sequelae of patients suffering from poliomyelitis [32]. Post-polio patients who have lost the function of their quadriceps and hamstring muscles must rely upon locking their knee, thereby depending on the ligaments to maintain joint stability when they stand or walk. Over time, these ligaments elongate and allow the knee to bend back, creating the deformity of genu recurvatum. This occurs due to collagen polymer lengthening [33] and decreased vascularity, which leads to microdegeneration within the collagenous matrix [34]. The reason that this does not happen under normal conditions is that the muscles maintain joint stability, something which they cannot do in the patient with polio. Unlike ligamentous support, which is not suited to sustaining constant loads, muscular support is both renewable and self-regulating. The muscles around a joint respond to sensory feedback and adjust themselves to maintain stability over a wide range of motion [35,36]. Because of their ability to elongate and shorten, the muscles act as self-adjusting shock absorbers, which allow the joint to move but do not allow the ligaments to become permanently stretched. During brief episodes when the joint is forced out of the normal range of motion which the muscle controls, the ligaments act as a check to prevent the joint from exceeding the bounds of motion which the muscles can modulate. In this way the muscle constantly adjusts to maintain stability under frequent and prolonged stress, while the ligaments stabilize the joint temporarily when it exceeds the bounds in which this occurs.

Application of this analogy to the pelvic floor suggests that muscular tissues are responsible for support during everyday activities, and provide resilient and renewable support. The endopelvic fasciae ('pelvic ligaments') come into play when the muscle is relaxed or damaged. In this system, *both* ligamentous and muscular support have important and complementary roles to play.

For a patient with genital prolapse, the question of whether neuromuscular or ligamentous damage is responsible for the problem is unanswerable at present. Neuromuscular injury at parturition may make muscular support ineffective; this would mean that the pelvic ligaments would have to bear the entire burden of support. If strong, they may succeed; if weak, they fail. Alternatively, the ligaments and fascia may break during a difficult birth. Muscular denervation occurring at the same time, but healing subsequently, could then be an associated phenomenon but not causal in the genesis of the ligamentous damage.

Whatever the precise contributions of the muscular or ligamentous elements, their complex interactions are crucial to understanding what constitutes a healthy environment for the normal functioning of the pelvic viscera. To function properly they must be maintained in a useful, well-defined relationship to each other. To date, however, we have concentrated more on individual organ systems, their attendant medical specialities, and the territoriality of each 'hole' than on the system of mutually supportive relationships among them which exists in the pelvic floor. Normal function of the pelvic viscera requires a balanced system of interrelationships – a 'pelvic ecosystem' if you wish. The pelvic floor is the environment in which the interrelated parts of this pelvic ecosystem exist. A change in one aspect of this environment can lead to a disruption of the normal relationships just as much as a derangement of an individual viscus can upset the balance. Thus, it is well established that a high suspension of the vesical neck, although eliminating the complaint of stress urinary incontinence, can disrupt the normal homoeostasis of pelvic support and result in uterine prolapse, enterocele formation or vaginal vault inversion [37,38].

The New Study of Pelvic Floor Dysfunction

Where does this all lead? In the first place, it points out the need for more work on *all* aspects of pelvic floor dysfunction. Research in basic anatomy and histology, studies of the histopathology of pelvic muscle and connective tissue, and detailed electrophysiological mapping of the pelvic floor all need to be undertaken. Specific studies to examine the integrity of the muscles and fascial structures independently in a large number of patients with genital prolapse and related problems will be necessary to determine the relative contributions of each system to the genesis of prolapse and pelvic floor dysfunction. In addition, increased work must be done on the interrelationships of physiological processes throughout the pelvic viscera. Whorwell et al. [39], for example, have shown a tenfold increase in unstable activity of the detrusor muscle of the bladder in patients with irritable bowel syndrome, suggesting that some of these patients may have a generalized disorder of smooth muscle and *not* just a problem in one or other 'hole'.

Each pelvic organ specialist must become familiar with the disorders of neighbouring pelvic structures and the techniques that exist for evaluating them. They must begin looking for concomitant pathology outside their own territories in patients referred to them for evaluation and treatment if we are ever to understand how these organ systems interact with one another in the pelvis. We suggest that the time is right for the creation of a new approach that cuts across previously entrenched specialties organized solely on the basis of organ systems. For want of a better term, this new discipline might be called pelvic egerterology, from the Greek *egerterios* meaning 'lift up or support', from which the Greek term for the levator complex is derived, *egerterios mous*. This new science should be devoted to understanding the nature, origins, and treatment of pelvic floor dysfunction in women. As a new field for unified research, it must clearly draw from many established specialties. Because the female genital organs are centrally located, and also because so much of the problem of pelvic floor dysfunction appears to stem from the cumulative effects of obstetrical trauma, obstetrics and gynecology seems the logical place for this process to begin. This is the only branch of medicine whose exclusive interest is the health care of women from conception through senescence: its practitioners care for women throughout their lives as these problems evolve, from their origins in pregnancy and childbirth to the development of distressing and sometimes disabling genitourinary prolapse. This is the clinical foundation on which such a new science must be built, but it must take advantage of the special skills and insights available from other technical disciplines, most notably urology, gastroenterology, colorectal surgery and neurology.

Such multidisciplinary collaboration is not easy, for it means head-on collision with firmly entrenched prejudices and the removal of the blinders created by medical specialty boards and our current nosology of disease processes. Practical considerations dictate that this new study must originate in multidisciplinary clinics run by open-minded and compatible colleagues. We believe that the time has come for a major redirection in our approach to the pelvic floor. We urge tertiary care medical centers to begin collaborative research and clinical treatment programs in this area.

References

1. Jowett B. (Trans) The Dialogues of Plato, 4th edn. Vol. 1. Oxford: Clarendon Press, 1953
2. Rowe JW, Besdine RW, Ford AB et al. Urinary incontinence in adults – National Institutes of Health Consensus Development Conference. *JAMA* 1989;261:2685–2690
3. Hudson RD. Disease and its control: the shaping of modern thought. Westport, Conn.: Greenwood, 1983
4. Rosen G. The specialization of medicine, with particular reference to ophthalmology. New York: Froben Press, 1944
5. Faber K. Nosography: the evolution of clinical medicine in modern times, 2nd edn. New York: Hoeber, 1930
6. Reiser SJ. Medicine and the reign of technology. London: Cambridge University Press, 1978
7. Nichols DH, Randall CL. Vaginal surgery, 3rd end. Baltimore: Williams and Wilkins, 1989
8. Zacharin RF. Pelvic floor anatomy and the surgery of pulsion enterocele. New York: Springer-Verlag, 1988
9. Berglas B, Rubin IC. Study of the supportive structures of the uterus by levator myography. *Surg Gynecol Obstet* 1953;97:677–692
10. Halban J, Tandler J. Anatomie und Atiologie der Genital prolapse biem Weibe. Vienna: Wilhelm Braunmuller, 1907
11. Nichols DH, Milley PS, Randall CL. Significance of restoration of normal vaginal depth and axis. *Obstet Gynecol* 1970;36:251–256
12. Dickinson RL. Studies of the levator ani muscle. *Am J Obstet Dis Women* 1889;22:897–917
13. Richter K. Lebendige anatomie der vagina. *Geburtschilfe Frauenheilk* 1966;26:1213
14. Parks AG, Porter NH, Melzack J. Experimental study of the reflex mechanism controlling the muscles of the pelvic floor. *Dis Colon Rectum* 1962;5:407–414
15. DeMarsovsky PJ, Nichols DN, Randall CL. Urethrocolpography. *Arch Gynäkol* 1973;215:351–358
16. Kegel AH. Physiologic therapy for urinary stress incontinence. *JAMA* 1951;146:915–917
17. Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut* 1977;18:656–665
18. Snooks SJ, Sitchill M, Swash M, Henry MM. Injury to innervation of the pelvic floor sphincter musculature in childbirth. *Lancet* 1984;2546–550
19. Beersick F, Parks AG, Swash M. Pathogenesis of anorectal incontinence: a histometric study of the anal sphincter musculature. *J Neurol Sci* 1979;42:111–127
20. Neill ME, Swash M. Increased motor unit fibre density in the external anal sphincter muscule in anorectal incontinence: a single-fibre EMG study. *J Neurol Neurosurg Psychiatr* 1980;43:343–347
21. Kiff ES, Swash M. Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. *Br J Surg* 1984;71:614–616
22. Neill ME, Parks AG, Swash M. Physiological studies of the anal sphincter musculature in faecal incontinence and rectal prolapse. *Br J Surg* 1981;68:531–536
23. Henry MM, Parks AG, Swash M. The pelvic floor musculature in the descending perineum syndrome. *Br J Surg* 1982;69:470–472
24. Kiff ES, Barnes PRH, Swash M. Evidence of pudendal neuropathy in patients with perineal descent and chronic straining at stool. *Gut* 1984;25:1279–1282
25. Snooks SJ, Barnes PRH, Swash M. Damage to the innervation of

the voluntary anal and periurethral sphincter musculature in incontinence: an electrophysiological study. *J Neurol Neurosurg Psychiatr* 1984;47:1269–1273
26. Snooks SJ, Swash M. Abnormalities of the innervation of the urethral striated sphincter in incontinence. *Br J Urol* 1984;56:401–406
27. Snooks SJ, Badenoch DF, Tipstaft RC, Swash M. Perineal nerve damage in genuine stress urinary incontinence: an electrophysiological study. *Br J Urol* 1985;57:422–426
28. Anderson RS. A neurogenic element to urinary stress incontinence. *Br J Obstet Gynaecol* 1984;91:41–45
29. Gilpin SA, Gosling JA, Smith ARB, Warrell DW. The pathogenesis of genitourinary prolapse and stress incontinence of urine. A histological and histochemical study. *Br J Obstet Gynaecol* 1989;96:15–23
30. Smith ARB, Hosker GL, Warrell DW. The role of partial denervation of the pelvic floor in the aetiology of genitourinary prolapse and stress incontinence of urine. A neurophysiological study. *Br J Obstet Gynaecol* 1989;96:24–28
31. Smith ARB, Hosket Gl, Warrell DW. The role of pudendal nerve damage in the aetiology of genuine stress incontinence of urine. *Br J Obstet Gynaecol* 1989;96:29–32
32. Perry J. Orthopedic management of post-polio sequelae. In: Halstead LS, Wiechers DO (eds) Late effects of poliomyelitis. Miami: Symposia Foundation, 1985, pp. 193–206
33. Nordin M, Frankel VH. Biomechanics of collagenous tissues. In: Nordin M, Frankel VH (eds) Basic biomechanics of the musculoskeletal system. Philadelphia: Lea and Febiger, 1980, pp. 87–110
34. Rathbun JB, Macnab I. The microvascular pattern of the rotator cuff. *J Bone Joint Surg* 1970;52B:540–553
35. Abbott LC, Saunders JBdeCM, Bost FC, Anderson CE. Injuries to the ligaments of the knee. *J Bone Joint Surg* 1944;26:503–521
36. Grana WA, Muse G. The effect of exercise on laxity in the anterior cruciate ligament deficient knee. *Am J Sports Med* 1988;16:586–588
37. Burch JG. Cooper's ligaments urethrovesical suspension for stress incontinence. Nine years' experience – results, complications, technique. *Am J Obstet Gynecol* 1968;100:764–774
38. Addison WA, Livengood CH III, Sutton GP, Parker RT. Abdominal sacral colpopexy with Mersilene mesh in the retroperitoneal position in the management of posthysterectomy vaginal vault prolapse and enterocele. *Am J Obstet Gynecol* 1985;153:140–146
39. Whorwell PJ, Lupton EW, Erduran D, Wilson K. Bladder smooth muscle dysfunction in patients with irritable bowel syndrome. *Gut* 1986;27:1014–1017

Reviews of Current Literature

Obesity and Lower Urinary Tract Function in Women: Effect of Surgically Induced Weight Loss

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Am J Obstet Gynecol 1992;167:392–399

Thirteen morbidly obsese women underwent gastric bypass surgery. They were evaluated thoroughly before and many months after surgery to assess lower urinary tract function. Twelve subjects had troublesome urinary incontinence prior to surgery, versus 3 after wards. It was concluded that weight reduction was desirable for women with urinary incontinence.

Comment

Prior studies have not shown the benefit of weight loss to lower urinary tract disorders of continence. This is the one article that states that beneficial effects do occur from marked weight loss. It may therefore be useful to have this article handy for reference as a way to encourage obsese patients to lose weight. Further study of less morbidly obese patients would be required in order to be able to apply the information to our usual patient population.

Intravesical Lidocaine: Topical Anesthesia for Bladder Mucosal Biopsies

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J Urol 1992;148:795–796

Forty patients (35 men and 5 women) had mucosal bladder biopsies following intravesical instillation of lidocaine. First, 10 ml of 2% lidocaine gel was placed into the urethra. A catheter was then inserted to drain the bladder, and 50 ml of 1.5% lidocaine hydrochloride solution was instilled. After 5 minutes, multiple biopsies were obtained. Biopsy sites were not cauterized. Measurement of lidocaine blood levels at 5, 10, and 30 minutes were clinically insignificant. Patients tolerated the procedures with minimal discomfort.

Comment

Another technique for female patients is to place a pillar block by injecting 5 ml of 2% lidocaine at each bladder pillar. The lidocaine gel is placed into the urethra, and lidocaine instillation can be a 4% solution. After 5 minutes the procedure can be completed with minimal discomfort.