women likely to have some abnormality of the endometrium.[2] On the other hand, when the endometrial thickness is <5 mm, one would expect to find an atrophic endometrium. We have not done enough studies in patients to find a correlation. We are doing ultrasonographic examination before endometrial biopsy in several clinical protocols in women who are receiving estrogen and progestins.

How good is ultrasonography in assessing the entire endometrial cavity? I am a neophyte in this area, and I feel very inadequate, thinking that I might miss something, if I look at a vaginal sonogram that I have obtained in terms of endometrial thickness. This goes back to the same point that adenocarcinoma can be focal, and we may be missing something with these biopsies. The use of vaginal ultrasonography is interesting in the evaluation of the endometrium, but I am not sure of its clinical validity.

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Assessment of Kegel pelvic muscle exercise performance after brief verbal instruction

Richard C. Bump, MD, W. Glenn Hurt, MD, J. Andrew Fantl, MD, and Jean F. Wyman, PhD
Richmond, Virginia

Forty-seven women had urethral pressure profile determinations performed at rest and during a Kegel pelvic muscle contraction, after brief standardized verbal instruction. Twenty-three (49%) had an ideal Kegel effort—a significant increase in the force of urethral closure without an appreciable Valsalva effort. Twelve subjects (25%) displayed a Kegel technique that could potentially promote incontinence. Age, parity, weight, estrogen deprivation, prior continence surgery or hysterectomy, and passive urethral function did not predict a successful effort. We concluded that simple verbal or written instruction does not represent adequate preparation for a patient who is about to start a Kegel exercise program. (Am J Obstet Gynecol 1991;165:322-9.)

Key words: Kegel exercises, pelvic muscle exercises, urinary incontinence

In the late 1940s, the gynecologist Arnold Kegel[1] proposed the use of pelvic muscle exercises to improve the function and tone of the pelvic floor after childbirth, to correct mild anatomic defects such as early cystocele and rectocele, and to treat stress urinary incontinence. Kegel exercises represent the voluntary contraction and relaxation of the levator ani muscle (principally the pubococcygeus and puborectalis portions), which supports the vagina, bladder, and urethra and which contributes to the skeletal muscle component of the urethral sphincteric mechanism. The goal of Kegel exercises in the treatment of urinary incontinence is to increase the strength and endurance of these muscles, thereby enhancing the force of urethral closure under certain conditions, such as with a sudden increase in abdominal pressure. Implicit to our understanding of the physiologic basis of Kegel exercises is that a properly performed contraction should increase the force of urethral closure.

Some women seem unable to contract the proper muscles when given verbal instruction about Kegel exercises; they often perform a Valsalva maneuver or contract the gluteal and thigh muscles exclusively or in combination with contraction of the levators. Kegel noted that many women are unaware of their pelvic

From the Department of Obstetrics and Gynecology, Medical College of Virginia, Virginia Commonwealth University.
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Reprint requests: Dr. Richard C. Bump, Department of Obstetrics and Gynecology, Box 34 MCV Station, Richmond, VA 23298.
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muscle function and require some method of performance feedback to successfully contract the proper muscles. To assist patients by providing such biofeedback, he developed the perineometer, a pneumatic device consisting of a vaginal probe connected to a manometer. While vaginal pressure measurements, electromyographic activity, and digital palpation of pubococcygeus muscle tone have all been advocated as biofeedback techniques to facilitate the initiation of a Kegel exercise program, most patients start (and finish) such a program with only brief written or oral instructions. The American College of Obstetricians and Gynecologists' patient education pamphlet on urinary incontinence instructs patients to "stop the flow of urine in midstream without tightening or tensing your leg or stomach muscles." The aim of this study was to assess the effect of such a simple brief verbal instruction on urethral sphincteric function during a Kegel contraction. A secondary aim was to attempt to identify clinical or urodynamic characteristics that might predict an effective Kegel contraction effort.

Subjects and methods

The study population included 47 consecutive subjects who presented to the Gynecologic Urodynamic Laboratory at the Medical College of Virginia Hospitals for evaluation. Their mean age was 53.6 years (range 23 to 83 years) and their mean parity was 2.5 (range 0 to 8). Twenty-eight subjects (60%) had urinary incontinence as the major complaint. Of those who were incontinent, 14 had pure genuine stress incontinence, four had pure detrusor instability, six had mixed incontinence (genuine stress incontinence plus detrusor instability), and four had pure urethral instability. Nineteen subjects (40%) did not complain of incontinence. Five of these had vaginal vault prolapse, four had emptying phase defects, five had sensory urgency without incontinence or motor instability, and five were being evaluated 6 months after successful continence surgery. Eighteen subjects (38%) had prior continence surgery, 27 (60%) had a prior hysterectomy, 7 (15%) had severe pelvic organ prolapse (2 of whom also had detrusor instability), and 7 (15%) were hypoestrogenic.

All subjects underwent a standardized urodynamic evaluation that included uroflowmetry with postvoid residual urine volume measurement, retrograde provocative multichannel water urethrocystometry with the use of microtip transducer catheters, passive and dynamic urethral pressure profiles, resting and stressed urethral axis determinations, and direct visualization testing for fluid loss. Specific details of the urodynamic methods have been described in detail previously.[2-4] All subjects had a negative bacterial urine culture (<100 CFU/ml) before being scheduled for urodynamic testing and none had pyuria at the time of the testing.

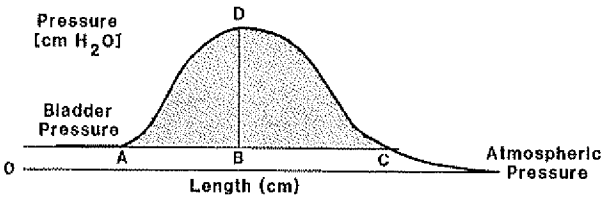

Fig. 1. Measurements derived from the urethral pressure profile. *Line A to C* is total functional urethral length; *line B to D* is maximum urethral closure pressure. *Shaded area* represents total urethral pressure profile area. Mean urethral closure pressure is calculated by dividing profile area by functional urethral length.

After each subject had completed the standard passive and dynamic urethral pressure profile (at a bladder volume of 300 ml and a transducer withdrawal speed of 0.5 mm/sec), a passive urethral pressure profile was repeated at a catheter speed of 5.0 mm/sec. Each subject was then asked to "contract the muscles you would use if you were trying to keep from losing your urine or if you were trying to stop your stream after you had started to urinate." Once the subject had acknowledged that she understood the instructions and had practiced a contraction, she was allowed to relax for a short time. It was then explained that she would be asked to contract the same muscles on command and to hold the contraction until she was told she could relax. Another urethral pressure profile was then performed, with the patient having been asked to start a contraction just before catheter withdrawal was started and being allowed to relax as soon as the urethral closure pressure became negative. This profile was designated the Kegel urethral pressure profile. The catheter withdrawal speed of 5.0 mm/sec allowed all Kegel profiles to be completed in <8 seconds, a period of time for which most patients could sustain a single contraction effort.

Pressures recorded with a physiologic recorder during the profiles included the vesical, urethral, abdominal (vaginal), detrusor, and urethral closure pressures. Parameters measured and analyzed from the urethral pressure profiles included the functional urethral length, maximum urethral closure pressure, total profile area (determined with a polar compensation planimeter), and mean urethral closure pressure (profile area/functional urethral length). These measurements are illustrated in Fig. 1. Urodynamic techniques and measurements, terms, and diagnostic criteria conform to the recommendations of the International Continence Society.[5]

A subject was considered to have had an effective Kegel effort if the Kegel urethral pressure profile area was ≥120% of the passive urethral pressure profile area or if both the Kegel maximum urethral closure pressure and the mean urethral closure pressure were

Table I. Comparison of changes from passive to Kegel urethral pressure profile according to Kegel effort group assignment

Change	*Effective effort (n = 28)*	*Ineffective effort (n = 19)*	*p Value*
Functional urethral length			0.04*
Absolute (cm)			
Mean ± SE	0 ± 0.9	−2.0 ± 0.7	
Range	−11-14	−8-6	
Percentage			
Mean	+1	−9	
Range	−41-58	−28-18	
Maximum urethral closure pressure			0.0000003*
Absolute (cm H_2O)			
Mean ± SE	16.5 ± 2.4	−1.5 ± 1.4	
Range	−4-56	−16-6	
Percentage			
Mean	+70	−6	
Range	−6-450	−53-33	
Area			0.00000009*
Absolute (mm × cm)			
Mean ± SE	223.3 ± 35.3	−53.3 ± 19.8	
Range	−38-888	−222-102	
Percentage			
Mean	+82	−17	
Range	−14-779	−70-18	
Mean urethral closure pressure			0.00000005*
Absolute (cm H_2O)			
Mean ± SE	9.4 ± 1.3	−1.0 ± 0.7	
Range	1-36.1	−7.4-6.1	
Percentage			
Mean	+80	−10	
Range	8-633	−60-28	

*Kruskal-Wallis one-way analysis by ranks.

≥120% of the passive maximum and mean urethral closure pressure. Subjects who did not fulfill either of these criteria were considered to have had an ineffective Kegel effort. The degree of Valsalva effort was assessed by measuring the changes in vesical and vaginal pressures. The Valsalva effort was considered excessive if the rise in vesical pressure exceeded 15 cm H_2O during the Kegel urethral pressure profile.

Categoric data were analyzed for significant differences with the χ^2 test with Yates' correction. Quantitative data were analyzed with analysis of variance and the Kruskal-Wallis one-way analysis by ranks for between-group comparisons, the paired *t* test for passive and Kegel urethral pressure profile comparisons, and simple linear regression analysis for vesical and vaginal pressure correlations.

Results

Twenty-eight subjects (60%) had an effective Kegel effort and 19 (40%) had an ineffective effort. With a Kegel contraction the former group increased the maximum urethral closure pressure (mean ± SE) from 35.0 ± 3.2 to 51.5 ± 3.7 cm H_2O ($p = 0.000001$), the total profile area from 449 ± 54 to 671 ± 68 mm × cm ($p = 0.0000008$), and the mean urethral closure pressure from 18.7 ± 2.0 to 28.1 ± 2.4 cm H_2O ($p = 0.0000004$). In contrast, the group with an ineffective effort had no significant increases in any of these parameters with maximum urethral closure pressure values of 32.2 ± 3.5 and 30.7 ± 3.6, total profile areas of 440 ± 53 and 386 ± 58, and mean urethral closure pressure values of 17.4 ± 1.7 and 16.4 ± 2.0 for passive and Kegel profiles, respectively. There were no significant changes in functional urethral length in either the effective or ineffective group. Table I compares the changes between the passive and Kegel profiles for the two groups. As expected, there were highly significant differences between the two groups for the changes in all profile measurements.

As demonstrated in Table II, there were no significant differences between the effective and ineffective Kegel effort groups with respect to age, parity, weight, or any of the passive profile measurements. Likewise, there were no significant differences between the groups with respect to the prevalence of prior continence surgery, prior hysterectomy, severe pelvic organ prolapse, hypoestrogenism, or genuine stress incontinence. When the ability to perform an effective Kegel contraction was compared to urodynamic diagnosis, no clear pattern was seen; 57% of patients with pure gen-

uine stress incontinence (8/14), 67% with mixed incontinence (4/6), 75% with pure unstable urethra (3/4), 33% with pure detrusor instability (1/3), 60% with vaginal vault prolapse (3/5), 100% with emptying phase dysfunction (4/4), and 50% with no abnormal diagnosis (5/10) were able to generate an effective Kegel effort.

Twelve subjects (26%) had increases in vesical pressure >15 cm H_2O during the Kegel contraction, 5 in the effective effort group and 7 in the ineffective effort group (difference not statistically significant). The change in vesical pressure (mean ± SE) for those with excessive Valsalva effort was 39.8 ± 12.5 cm H_2O in the effective Kegel group and 21.6 ± 4.6 cm H_2O in the ineffective Kegel group. The respective changes for those without excessive Valsalva effort were 5.9 ± 2.1 and 6.5 ± 1.2 cm H_2O. Four of the five subjects with improvement in urethral measurements after Valsalva effort had either prior successful continence surgery or severe pelvic organ prolapse, conditions in which Valsalva stress is known to enhance urethral closure because of physical obstruction.[6,7] As anticipated, there was a highly significant correlation between the changes in vesical and vaginal pressures during the Kegel effort ($r = 0.8306$, $p < 0.00001$).

Table II. Clinical and passive urethral pressure profile comparisons between effective and ineffective Kegel effort groups

	Effective effort (n = 28)	*Ineffective effort (n = 19)*
Age (yr)		
Mean ± SE	52.3 ± 2.8	55.7 ± 3.5
Range	23-74	27-83
Parity		
Mean ± SE	2.4 ± 0.3	2.7 ± 0.4
Range	0-6	0-8
Weight (kg)		
Mean ± SE	80.4 ± 6.2	66.3 ± 3.1
Range	55-200	46-104
Functional urethral length (mm)		
Mean ± SE	23.5 ± 0.8	23.5 ± 1.5
Range	16-32	7-35
Maximum urethral closure pressure (cm H_2O)		
Mean ± SE	35 ± 3.2	32 ± 3.5
Range	11-68	4-68
Area (mm × cm)		
Mean ± SE	449 ± 55	440 ± 53
Range	114-1220	14-1014
Mean urethral closure pressure (cm H_2O)		
Mean	18.7	17.4
Range	5.3-45.0	2.0 to 32.3
Continence surgery (No.)	10 (37%)	8 (42%)
Hysterectomy (No.)	15 (56%)	12 (63%)
Severe prolapse (No.)	3 (11%)	4 (21%)
Hypoestrogenic (No.)	3 (11%)	4 (21%)
Genuine stress incontinence (No.)	12 (43%)	8 (42%)

Comment

This study has demonstrated that 60% of the 47 subjects were able to generate an effective increase in the force of urethral closure after brief standardized verbal instruction. However, 5 of 28 subjects with an effective Kegel effort achieved this with an appreciable increase in abdominal and vaginal pressure. Thus only 23 of 47 subjects (49%) had an ideal Kegel effort, that is, a significant increase in urethral pressure without a concurrent increase in vesical and abdominal pressure. Moreover, we were unable to identify any historic or urodynamic parameter that reliably identified subjects who were likely to have either an effective or an ineffective Kegel effort. These included age, parity, weight, prior continence surgery, prior hysterectomy, severe pelvic organ prolapse, any parameter on a passive urethral pressure profile, and any urodynamic diagnostic category.

It is recognized that our criteria for an effective Kegel contraction were arbitrary, but this was necessary because no criteria have been previously established. In defense of our criteria, it should be emphasized that very few subjects in either group came close to our cutoff values. For example, only one subject in the ineffective group increased the total profile area by >8% and 13 of 19 subjects in this group actually decreased the profile area with the Kegel effort. In contrast, only 4 subjects in the effective group had <20% increase in total profile area; they had increases in maximum and mean urethral closure pressures that averaged 31% and 46%, respectively. The lack of change in profile area in these subjects was due to a decrease in functional urethral length that averaged 28%. We thought this resulted from urethrovesical junction movement (elevation) away from the urethral transducer during the Kegel profile, the net effect of which would be an increase in the speed of withdrawal of the urethral transducer relative to the paper speed of the recorder. We attempted to have the Kegel contraction established before catheter withdrawal to obviate this technical problem; however, this goal obviously was not realized in these four subjects.

We also recognize that the small number of subjects in some of our groups limits the power of any conclusion regarding the lack of effect of certain factors on the ability to perform an effective Kegel contraction. However, it is clear that none of these factors guarantees either success or failure. Fig. 2 contains tracings from three subjects that illustrate these points vividly. Part *2A* is a passive and Kegel urethral pressure profile from a 62-year-old, para 5 woman with recurrent mixed incontinence, a prior hysterectomy, and a Marshall-Marchetti-Krantz procedure. She was post-

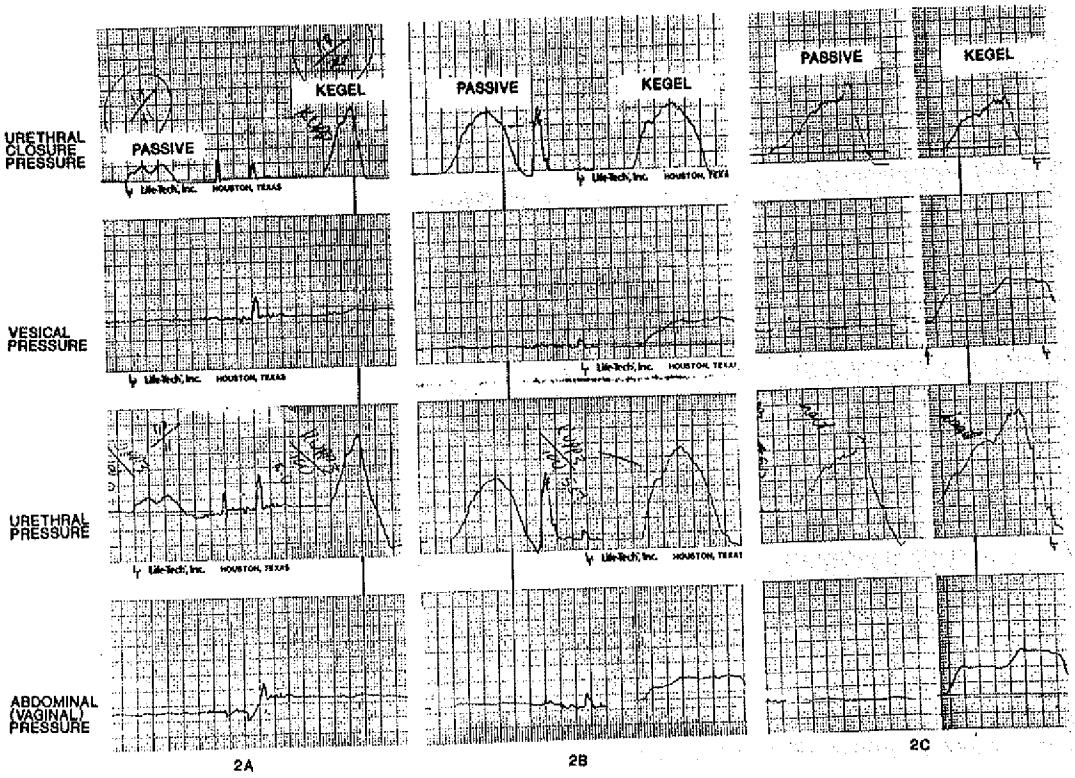

Fig. 2. Tracings of passive and Kegel urethral pressure profile of three subjects. See Comment section for details. **2A,** 62-year-old, multiparous subject with effective Kegel effort; **2B,** 27-year-old nulliparous subject with ineffective Kegel effort and significant Valsalva effort; **2C,** 52-year-old multiparous subject with genuine stress incontinence whose Valsalva effort with Kegel attempt resulted in decrease in force of urethral closure.

menopausal for 14 years but had never received estrogen replacement therapy. The passive profile demonstrated a markedly hypotonic urethra with a functional urethral length of 17 mm and a maximum urethral closure pressure of 11 cm H_2O. In spite of a variety of factors that led us to intuitively believe that she would be unable to display an effective Kegel contraction, she was able to increase the total profile area, maximum urethral closure pressure, and mean urethral closure pressure by 186%, 318%, and 170%, respectively. In contrast, part *2B* is a tracing from a 27-year-old nulliparous woman with sensory urgency, normal ovarian function, and no prior urogynecologic survey. In spite of characteristics that we believed would predict an effective Kegel effort, the only effect of her Kegel effort was a 15 cm H_2O increase in vaginal and vesical pressure with no appreciable change in urethral closure pressure.

Part *2C* illustrates a potential danger of incorrectly performed Kegel exercises. This tracing is from a 52-year-old, gravida 2 woman with primary pure genuine stress incontinence. Her Kegel effort was accompanied by a significant Valsalva effort with an increase in vaginal and vesical pressure of nearly 30 cm H_2O. This increase in abdominal pressure was transmitted only partially to the urethra with the net effect being a decrease in total profile area, maximum urethral closure pressure, and mean urethral closure pressure of 32%, 28%, and 11%, respectively. Thus, instead of improving the force of urethral closure, the Kegel technique actually had the opposite effect. Further, a structured program of regular repetition of this technique might actually lead to progression of the anatomic and functional defect that is the cause of incontinence in this woman.

This study offers no insight into the predictive value of the Kegel urethral pressure profile. We do not know if an effective Kegel effort by a woman predicts successful control of incontinence if she pursues a structured Kegel exercise program, nor do we know if ineffective Kegel efforts can be made effective through reeducation and the use of biofeedback. Finally, we do not know if an improvement in the force of urethral closure with a Kegel contraction will correlate positively with an improvement in urinary control. These are all topics of ongoing inquiry in our unit.

What does seem obvious from this study is that simple verbal or written instruction does not represent ade-

quate preparation for a patient who is about to pursue a Kegel exercise program. While approximately half of women are able to perform an effective contraction, half are not and as many as one fourth may adopt a technique that could potentially promote the defect responsible for their incontinence. Some form of initial assessment, instruction, and feedback such as pubococcygeal and abdominal muscle palpation with verbal feedback, the use of differential (upper vagina reflecting abdominal pressure and lower vagina reflecting levator tone) vaginal pressure biofeedback, or the use of electromyographic biofeedback seems desirable. Saving such techniques only for Kegel failures needlessly postpones potentially effective therapy for a significant number of women who suffer from incontinence. In addition, many women who feel that they have failed their initial trial with a Kegel exercise program will refuse to accept any additional physiotherapy.[8]

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Discussion

DR. A. CULLEN RICHARDSON, Atlanta, Georgia. I congratulate the authors on a very carefully done study with carefully recorded exact data. It is encouraging to see attention returned to the striated muscle in the pelvis. We need to understand more about its role in pelvic support in general, as well as in urinary continence.

My principal criticism of this article is that at this point I consider it to be an incomplete study. This is an excellent first step in a much-needed investigation of Kegel exercises, but the data contained in this study, although excellent, are not sufficient to permit significant conclusions.

The study lacks a control group of perfectly normal nulliparous and parous patients. Patients presenting to a urodynamics clinic are hardly a representative patient population. These results are not applicable to the general population.

The authors concluded that simple verbal instruction does not represent adequate preparation for a patient who is about to start a Kegel exercise program. However, they did not report how many of those who had no change in urethral closure pressure were eventually taught to sustain good levator contractions.

There were variables for which there were no corrections.

At the beginning the authors made an assumption: "Implicit to our understanding of the physiologic basis of Kegel exercises is that a properly performed contraction should increase the force of urethral closure." This would be true only in patients with perfectly normal anatomy, which few of their patients had.

Potentially, there are two anatomic variables that must be controlled for that were not addressed in this article. As this is essentially a test of isolated striated muscles, normal function would require, first, structural integrity of the muscle itself and its attachment and, second, intact neurologic innervation of the particular muscle being studied.

The effect on urethral pressure with voluntary contraction of the pelvic floor muscle is exerted through three groups of striated muscle fibers. First and most prominent is the pubovaginal portion of the pubococcygeus muscle (fibers of Luschka). Second, there are the very thin, roughly circular striated periurethral fibers that terminate in the musculus transversus perinei profundus of the urogenital diaphragm. Third are the fibers in the musculus transversus perinei profundus that go over the top of the urethra just as it passes under the symphysis (so-called compressor urethra muscle). In the case of the pubovaginal fibers of Luschka, the force is not on the urethra itself but is transmitted by way of the pubocervical fascia within which the urethra is embedded.

If these striated muscle fibers are detached from the vagina or the paraurethral fascia is broken, then even with good pubococcygeus contraction there would be no effect on urethral closure pressure. As the authors continue their study, there should be a control for pubococcygeus contractions, with fine-wire electromyography, to identify those patients who in spite of pubococcygeal contraction show no change in urethral closure pressure. I am sure these patients exist.

Even in patients whose muscles are intact, there can be a problem with neurologic innervation. This of course can extend from the cerebral cortex down through the cord and then through the peripheral nerves to the myoneural junction. The assumption in the study seemed to be that all of the problems were at the level of the cerebral cortex—failure to understand the instructions.

There is increasing evidence that many patients with genital support problems with or without bladder dys-

function manifest weakness in the pelvic floor striated muscle as a result of deficient or damaged neurologic innervation—a problem not particularly affected by exercise.

Gilpin et al.[1] and Smith et al.[2, 3] in Manchester, England, Snooks et al.[4] in London, and Constantinou and Govan[5] in this country have studied and are studying this in depth. They have found some women whose problem is in the peripheral nerves at the spine as a result of bony deformities such as are seen with spondylitis, many in the pudendal nerves as a result of stretch injury during parturition, and some with stretch injury within the muscle itself.

This becomes even more complicated when one considers that the pelvic floor striated muscle contains both slow- and fast-twitch fibers. The slow-twitch fibers, which are quite prominent in the pelvic floor muscles, maintain some constant involuntary tone, much as is the case with the external anal sphincter. Undoubtedly the resting tone of these slow-twitch fibers in the levator group is the important factor in keeping the levator hiatus essentially closed in the patient with normal support.

Normal pelvic support is always a delicate interplay between the fibrous connective-type tissues and striated muscle tone—involuntary, reflex, and voluntary. Mechanically, the striated muscles not only serve as a backup mechanism to protect the overlying connective-type tissues (the fascias and ligaments) but are actually the mediators of certain functions, such as aiding in the closure of the bladder neck, as well as the anal sphincter. As yet we have no medical or good surgical treatments for inadequate striated muscle function.

We all tend to recommend Kegel exercises rather routinely. These would be ineffective, however, if the problem is one of neurologic change. Further, the authors have apparently identified a subset of patients in whom the exercises are not only ineffective but potentially deleterious.

Again I congratulate the authors on carefully recording these preliminary observations. My hope is that they will continue to study the pelvic floor striated muscle with normal controls, electromyographic monitoring of the pubococcygeus, and a careful record of the anatomic findings. Clearly their preliminary results confirm the need for much better understanding. Further, their study makes it clear that we need a better method of instructing patients in the "how to" of Kegel exercises.

I would ask them several questions at this time:

1. Do you have data that you did not include that would answer the questions posed?

2. What percent of patients were able to effect good levator contractions after more detailed instructions and/or some biofeedback?

3. At present, what have you found to be the best "how to" in instructing patients for Kegel exercises?

It is hoped that these authors, who possess the expertise to do such careful and exact studies, will continue their efforts and give us the much-needed additional data to clarify the preliminary findings reported here.

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Dr. Bump (Closing). With respect to the appropriateness of our study group, I would defend our population as exactly representative of the types of patients who are started on a Kegel exercise program. Therefore the information is directly applicable to the portion of the population that might start such a program. Kegel advocated his exercises for patients who needed to improve the function and tone of the pelvic floor, that is, after childbirth, with early cystocele and rectocele, and with incontinence. I should also mention that our incidence of ineffective Kegel effort corresponds with that of several other authors in the physiotherapy literature.

I don't doubt that some of our ineffective contractors were ineffective not because they did not understand which muscles to contract but because the muscles or the connective tissue attachments were rendered incapable of responding. However, our findings do not support this as the primary reason for failure, because subjects who were most likely to have such neuromuscular and anatomic compromise were able to perform just as well as subjects who had no evidence of such injury. Further, a significant proportion of subjects were not simply ineffective; nearly one quarter had efforts that were counterproductive.

With respect to Dr. Richardson's question of clandestine data that may address the questions as to the type and location of striated muscle involved in the continence mechanism, we are in the process of completing a study that confirms and expands on the findings in the studies of the groups he mentioned. One of our major research priorities is to develop techniques to study denervation injury and recovery. By way of drastic oversimplification I would simply state that there is no question in my mind that there are critical neuromuscular components to stress incontinence in women and that simple anatomic alterations do not explain the condition.

As to our eventual ability to teach ineffective contractors to become effective and the best way of pro-

viding instruction for Kegel exercises, I hope that our groups' efforts, under the primary direction of Dr. Jean Wyman, will allow us to answer that question, but the answer is 4 to 5 years away.

I think the worst way to instruct patients is to ask them to interrupt the urinary stream repeatedly during micturition. I have three primary aversions to this time-honored technique. First, many women, and most incontinent women, cannot interrupt the stream, and asking them to do what they are unable to do simply accentuates their feelings of helplessness and hopelessness. Second, I don't think that the way you improve the bladder's storage ability is by interfering with its emptying function. A contracting sphincter in the midst of normal micturition is a pathologic condition known as vesicosphincter dyssynergia, and I don't think we should encourage patients to practice this technique. Remember, we can entrain our bladders to adopt bad habits in addition to good ones. Finally, when the completely normal woman interrupts the urine stream, the urethral closure pressure becomes positive and flow stops several seconds before the detrusor muscle contraction is suppressed. This detrusor contraction against a voluntary outlet obstruction several times during every voiding episode may result in reflux and eventually upper tract injury.

Vulvar squamous cell carcinoma and papillomaviruses: Two separate entities?

Willie A. Andersen, MD,[a] Douglas W. Franquemont, MD,[b] John Williams,[b] Peyton T. Taylor, MD,[c] and Christopher P. Crum, MD[a, b, c]

Charlottesville, Virginia

Vulvar squamous precancers (vulvar intraepithelial neoplasia) are associated with sexual factors, cigarette smoking, and human papillomaviruses. However, epidemiologic studies of invasive carcinoma of the vulva have produced conflicting evidence for these associations, in part because of a strong association with vulvar inflammatory disease (dystrophies) in older women. We analyzed a series of 42 vulvar invasive carcinomas for papillomavirus nucleic acids by deoxyribonucleic acid–deoxyribonucleic acid in situ hybridization and correlated their presence with age, smoking history, and morphologic type. The carcinomas were divided into well-differentiated, moderately and poorly differentiated, and intraepithelial-like growth patterns, the latter composed of nests of invasive neoplastic epithelium with preserved cell polarity, similar to intraepithelial disease. Of the lesions studied, 28% were human papillomavirus deoxyribonucleic acid–positive. Intraepithelial-like neoplasms segregated in women with a younger mean age (64 versus 73 years) than that of women with conventional squamous cell carcinoma and they more frequently had a history of cigarette smoking (88% versus 28%). Moreover, intraepithelial-like lesions contained human papillomavirus nucleic acids more frequently (67% versus 13%) when analyzed by in situ hybridization. These observations confirm the diverse nature of vulvar squamous cell carcinoma and may explain in part why conflicting results are obtained from studies investigating the role of sexual and viral factors in the genesis of vulvar cancer. They suggest that many invasive vulvar cancers may not be linked to papillomaviruses. (Am J Obstet Gynecol 1991;165:329-36.)

Key words: Vulvar carcinoma, human papillomavirus, vulvar intraepithelial neoplasia

From the Departments of Pathology,[b] Microbiology,[c] Obstetrics and Gynecology,[a] University of Virginia Health Sciences Center.
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Reprint requests: Christopher P. Crum, MD, Women's and Perinatal Pathology Division, Brigham and Women's Hospital, 75 Francis St., Boston, MA 02115.
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Invasive carcinoma of the vulva is a rare disease with an incidence rate approximately one tenth that of its counterpart in the cervix.[1, 2] Traditionally the disease has predominated in women in their seventh and eighth decades and is rarely observed in women younger than age 30 years.[2] This pattern of disease has changed slightly in recent years, and investigators have focused on three components of this disease spectrum that may shed light on the increasingly younger age of women seen with vulvar neoplasia. The first is the oc-