

## Original Article

# A Systematic Review of Estrogens for Recurrent Urinary Tract Infections: Third Report of the Hormones and Urogenital Therapy (HUT) Committee

L. Cardozo<sup>1</sup>, G. Lose<sup>2</sup>, D. McClish<sup>3</sup>, E. Versi<sup>4</sup> and H. de Koning Gans<sup>5</sup>

<sup>1</sup>King's College School of Medicine and Dentistry, London, UK; <sup>2</sup>Glostrup County Hospital, University of Copenhagen, Denmark; <sup>3</sup>Medical College of Virginia, Richmond, Virginia; <sup>4</sup>Brigham & Women's Hospital, Boston; <sup>5</sup>Pharmacia & Upjohn, Kalamazoo, Michigan, USA

**Abstract:** Our objective was to apply a meta-analysis to the available data to evaluate the effect of estrogen supplementation in the prevention of recurrent urinary tract infections in postmenopausal women. The literature review incorporated articles based on a search of *Excerpta Medica*, *Medline*, *Science Citation Index* and a manual search of commonly read journals in the fields of urology, gynecology, gerontology and primary healthcare, from January 1969 to December 1998. The search was not limited to English-language publications. Inclusion criteria were peer-reviewed articles containing original data with a primary outcome of symptomatic urinary tract infections and an estrogen-treated group. Articles were categorized into randomized controlled trials, case-control studies and self-controlled series. Of the articles reviewed, five were randomized controlled trials, two were case-control studies and three were self-control series. Meta-analysis of data from 334 subjects revealed a significant benefit from estrogen over placebo (odds ratio = 2.51, 95% confidence interval = 1.48–4.25). The most convincing results were obtained using the vaginal route of administration. A variety of different estrogen preparations have been employed in the few published reports, making comparison of the data difficult. However, vaginal administration seems to be effective in the prevention of recurrent urinary tract infections in postmenopausal women.

**Keywords:** Hormones; Postmenopausal; Recurrent urinary tract infections; Systematic review estrogens

---

## Introduction

Urinary tract infections (UTIs) are common in women of all ages and the prevalence has been shown to increase with increasing age [1]. Among postmenopausal women it has been estimated that as many as 15% of those seeking advice from their primary care doctor in the UK suffer from urinary tract infection [2], and the condition is thought to account for about 6 million doctor office visits per year in the USA, at an estimated cost in excess of \$1 billion per year [3]. However, there is no real evidence to suggest that estrogen deficiency per se has an effect, and it may be age-related changes or a combination of both that causes this increase in the prevalence of UTIs [4]. The reduction in circulating estrogen levels in women following the menopause leads to an increase in vaginal pH, in association with a change in vaginal flora [5–7]. There is a reduction in lactobacillus numbers and an increase in colonization by fecal uropathogens [8]. In view of this, estrogens have been prescribed empirically based on the clinical impression that they reduce the recurrence rate of urinary tract infections in postmenopausal women. It is thought that the mechanism for this involves an increased glycogen production in the vagina, leading to colonization by Doderlein's bacilli and a reduction in ambient pH [9]. The latter predisposes to changes in vaginal flora towards the premenopausal state.

Not all women who suffer from irritative bladder symptoms have an acute urinary tract infection, and estrogens may be effective in alleviating these symptoms in other ways. For example, estrogens have been shown to be an effective treatment for urogenital atrophy [10].

Exogenous estrogens can be administered in many ways. Different types can be prescribed by various routes of administration at different doses, and therapy may be short or long term. Given these variables and the lack of published data, there is no scientific basis on which to plan the optimal treatment strategy.

The few reports published on estrogen therapy for recurrent urinary tract infections have shown conflicting results. We therefore undertook a systematic review of the literature and a meta-analysis of the data to try to clarify this issue.

## Methods

A computer literature search was undertaken, including *Medline*, *Excerpta Medica* and *Science Citation Index*, and this was followed by a manual search of commonly read journals in the fields of urology, gynecology, gerontology and primary care medicine, from January 1969 to December 1998. The search was not limited to English-language articles. To be considered for inclusion in the analysis, papers had to contain original data; be peer reviewed; authors had to have reported symptomatic urinary tract infections as the primary outcome measure; and there had to be an estrogen-treated group. Abstracts were initially reviewed by the authors and articles discarded if they were reviews, surveys, editorials or letters, or did not address the variables of interest. Suitable articles were grouped as follows: (a) randomized controlled trials (RCTs), of which there were five [11–15]: four were placebo-controlled studies, but one [12] had an untreated control group only; (b) self-controlled studies, of which there were three [16–18]; and (c) case-control studies, of which there were two [19,20]. The data were pooled appropriately in order to produce a meta-analysis.

### Statistical Analysis

The probability of developing at least one UTI was analyzed as this variable was common to all five of the randomized controlled trials. Adequate information was published in four of the studies; we contacted an author of the fifth [14]. Effect size was expressed as the odds ratio (OR). An odds ratio less than 1 indicates that the control group was at less risk of a UTI than the estrogen group, whereas a value greater than 1 indicates that the control group was at greater risk. The average or common odds ratio was calculated by first determining the weighted average of the logarithm of the odds ratios for each study. The weights were the inverse of the variance of the log odds ratio. The average odds ratio was then calculated as the exponential (antilog) of the

average log odds ratio. By comparing the log odds ratio to its standard error, treatment effect was evaluated. The test statistic has a standard normal distribution. Ninety five percent confidence intervals (95% CI) were constructed for the log odds ratio, and then converted to confidence intervals for the odds ratio using the exponential.

Similar methods were used for the case-control studies. Although these studies were matched, the data were analyzed as if unmatched, because the manuscripts did not contain sufficient information for matched analyses. Orlander et al. [20] stated that the analyses for unmatched and matched data were similar.

The number of UTIs per 12 person-months was computed. In order to obtain sufficient data for this analysis, authors of two of the manuscripts [11,14] were contacted. Raz and Stamm [15] provided a *P* value for a comparison between estrogen and placebo (given as  $P < 0.001$  two-sided, assumed to be equal to 0.0005 for one-sided analysis). Kirkengen et al. [13] provided sufficient data to calculate the number of UTIs per 12 person-months for weeks 0–12. Comparison of estrogen and placebo groups could only be performed with Wilcoxon's rank sum test on data for either weeks 0–4 or weeks 5–12. We chose the latter. Data received from the authors of the Kjaergaard article [14] were also sufficient to calculate UTIs per 12 person-months and to perform a Wilcoxon rank sum test. The statistical review for the article by Cardozo et al. [11] provided data to compute the number of UTIs per 12 person-months, but statistical comparison was not possible. The Eriksen study [15] determined the number of UTIs per 12 person-months for the estrogen group but not for the control group, because the control subjects reached the study endpoint at the time of their first UTI.

A combined effect size for the number of UTIs per 12 person-months was determined as a weighted average of the individual results, with the weights equal to the number of months of observation. The method of Stouffer et al. [21] was used to establish the statistical summary, which involved summing the *Z* values of the individual tests and dividing the result by the square root of the number of tests.

## Results

A description of the five RCTs is shown in Table 1. Two of the studies used oral estriol; in one it was 3 mg/day [11] and the other used 3 mg/day for the first 4 weeks, followed by 1 mg/day for 8 weeks [13]. The other three studies used vaginal estrogens; Raz and Stamm [15] employed estriol cream (0.5 mg/day for 2 weeks, followed by 0.5 mg twice weekly for 8 months); Kjaergaard et al. [14] used estradiol tablets (25 µg/day for 1 week, followed by 25 µg twice weekly for 5 months); and Eriksen [12] used an estradiol-releasing vaginal ring (Estring) for 36 weeks. In total, data from 169 women receiving estrogen, 110 women receiving placebo and 55 untreated controls [12] were analyzed.

**Table 1.** Summary characteristics of randomized controlled trials

Reference	Estrogen	Route of administration	Study duration (months)	Sample size	
				Active	Control
Cardozo [11]	Estriol	Oral	6	36	36
Eriksen [12]	Estradiol	Vaginal ring	8.5	53	55
Kirkengen [13]	Estriol	Oral	3	20	20
Kjaergaard [14]	Estradiol	Vaginal tablet	5	10	11
Raz [15]	Estriol	Vaginal cream	8	50	43

**Table 2.** Odds ratio for UTI for the placebo group versus the estrogen Group in RCTs

Reference	Odds ratio	95% CI
Cardozo [11]	0.78	(0.29–2.15)
Eriksen [12]	3.85	(1.64–9.03)
Kirkengen [13]*	2.00	(0.51–7.72)
Kjaergaard [14]	0.80	(0.14–4.53)
Raz [15] <sup>†</sup>	44.3	(8.55–229.95)
Combined	2.51	(1.48–4.25)

\* Odds ratio calculated for months 2–3 of study.

<sup>†</sup> Odds ratio calculated at 4 months.

Table 2 has the odds ratios for the individual RCTs. The reports by Raz and Stamm [15], Kirkengen et al. [13] and Eriksen [12] found odds ratios greater than 1.0 (OR = 44.3, 2.0 and 3.85, respectively). Raz and Stamm [15] showed a significant reduction in UTIs for estrogen versus placebo, and Eriksen [12] showed a significant reduction in UTIs for estrogen versus controls. Cardozo et al. [11] and Kjaergaard et al. [14] had results favoring placebo which were not statistically significant. Combining the results of the five studies, the mean odds ratio was 2.51, with a 95% confidence interval of 1.48–4.25. This is significantly greater than 1.0, implying that estrogen decreases the risk of a UTI.

The impact of route of administration was studied by comparing mean odds ratio from the three reports using the vaginal route [12,14,15] with the mean odds ratio from the two reports employing oral administration [11,13]. The odds ratio for the vaginal route was 4.62 (95% CI 2.31–9.25), whereas that for the oral route was

1.10 (95% CI 0.49–2.46). This difference was statistically significant ( $P=0.008$ ).

The effect of the type of estrogen was studied by comparing data from the three studies using estriol [11,13,15] with the two studies using estradiol [12,14]. This yielded an odds ratio of 2.25 (95% CI 1.09–4.65) for the estriol group and 2.84 (95% CI 1.32–6.10) for the estradiol group. The difference was not statistically significant.

There were 1029 person-months of observation in the estrogen group and 528 in the placebo group. The number of UTIs per 12 person-months ranged from 0.46 to 3.6 in the estrogen group, and from 1.91 to 5.92 in the placebo group and (Table 3). The weighted average of the number of UTIs per 12 person-months was 1.54 for the estrogen group and 4.09 for the placebo group. Considering only the three studies [13–15] which allowed a comparison between estrogen and placebo groups, the average number of UTIs was significantly less for estrogen than for placebo ( $P=0.01$ ).

Examining the subgroups, the average number of UTIs per 12 person-months in the three studies using vaginal estrogen [13–15] was 1.34, compared to 2.24 for the two studies using oral estrogen [11,13]. The weighted average for the numbers of UTIs per 12 person-months in the three studies using estriol [11,13,15] was 1.21, versus 1.89 for the two studies using estradiol [12,14]. No statistical analysis was possible to compare these values as there were only three studies available for evaluation.

Two additional relevant RCTs were identified. Molander et al. [6] carried out a placebo-controlled RCT on 35 women with urogenital deficiency syndrome.

**Table 3.** Number of UTIs per 12 person-months in RCTs

Reference	Estrogen		Placebo	
	Number of person-months	Number of UTIs per 12 person-months	Number of person-months	Number of UTIs per 12 person-months
Cardozo [11]	165	2.11	188	1.91
Eriksen [12]	444	1.70	*	*
Kirkengen [13]	60	2.60	60	4.40
Kjaergaard [14]	50	3.60	55	3.71
Raz [15]	310	0.46	225	5.92
Combined	1029	1.54	528	4.09

\* Value could not be computed as follow-up was discontinued after first UTI.

Women in the treatment group took oral estriol 3 mg/day for 4 weeks, then 2 mg/day for another 6 weeks. At the end of the 10 weeks of treatment it was noted that 2 women in the estrogen group and 4 in the placebo group had a positive culture ( $P=0.33$ ). The second study, by Felding et al. [22], looked at 45 postmenopausal women scheduled for vaginal repair surgery. Subjects were randomized to 25  $\mu\text{g}$  estradiol or placebo tablet daily for 3 weeks prior to surgery. During a gynecologic examination 4 weeks postoperatively 3 women in the estrogen group and 10 in the placebo group had suffered from at least one UTI (OR = 4.88, 95% CI 1.12–21.2), resulting in 1.36 UTIs per 12 person-months in the estrogen group and 4.7 in the placebo group. These two studies differ from the five previously described RCTs. The women in these studies [6,22] did not necessarily present with recurrent UTIs prior to entry. In addition, the treatment phase for the women in the study by Felding et al. [22] ended at the time of surgery, so that estrogen treatment ceased 4 weeks prior to evaluation. These two studies were therefore not included in the meta-analysis.

A description of the three self-controlled trials is shown in Table 4. The number of subjects ranged from 5 to 40. Brandberg et al. [16] used oral estriol (3 mg/day for 1 month and 1 mg/day thereafter); Parsons and Schmidt [17] used conjugated estrogen as a vaginal cream (0.3 mg/day in the first week, every 2 days for the second week, and then every third day until normal flora was achieved, followed by a low maintenance dose for the duration of the study); Privette et al. [18] used conjugated oral estrogens (0.625 mg/day for 21 days each month), but 3 older women were subsequently converted to vaginal cream. In total, subjects were on estrogen for 626 person-months. The number of UTIs per 12 person-months ranged from 4.14 to at least 12 when the subjects were not receiving estrogen, and decreased to 0.17–0.38 when they were receiving estrogen.

Table 5 describes the two case–control studies, both of which matched 5 controls to each case. Cases were matched on age and practice. The study by Orlander et al. [20] presented data separately for women who had undergone a hysterectomy and those who had not. Oliveria et al. [19] compared estrogen users of varying durations to non-users. For the current analysis, those women who were on estrogen for at least 6 months prior to the index UTI were selected. Orlander et al. [20] found a significant increase in the frequency of UTIs in estrogen users with a uterus (OR = 0.48, 95% CI: 0.38–0.62) but not for women who had had a hysterectomy (OR = 0.92; 95% CI: 0.67–1.27). Oliveria et al. [19] found no relationship between estrogen use and frequency of UTIs (OR = 0.79; 95% CI: 0.56–1.12). When the information from these studies was combined, the mean odds ratio was 0.65 (95% CI 0.55–0.78), which implies an increased risk of UTI with estrogen supplementation.

## Discussion

Early self-controlled trials reported a beneficial effect of estrogen in the management of recurrent UTIs in postmenopausal women. In contrast, subsequent case–control studies showed an overall increased risk of UTIs in estrogen-supplemented women. It is generally accepted that RCTs are the gold standard for resolving these therapeutic dilemmas.

Our systematic review of the literature showed an overall significant beneficial effect of estrogen supplementation in this population. However, these findings should be considered cautiously in the light of the following. Our analysis was based on five RCTs, one of which used untreated controls rather than placebo. There was a total of only 334 subjects (169 active, 110 placebo, 55 untreated controls). These women received treatment for a variable length of time, with different estrogens, routes of administration and dose regimens.

**Table 4.** Summary characteristics of self-controlled trials

Reference	Estrogen	Route of administration	Study duration	Sample size
Brandberg [16]	Estriol	Oral	9 months	40
Parsons [17]	Conjugated estrogen	Vaginal	10–15 months	5
Privette [18]	Conjugated estrogen	Oral for most	2–10 yrs	12

**Table 5.** Summary characteristics of case–control studies

Reference	Subgroup	Number of cases	Number of controls	Estrogen exposure
Oliveria [19]	Exposed at least 6 months	236	1176	At least 6 months prior to index UTI
Orlander [20]	Intact uteri	2879	16614	At least 1 year prior to index UTI
Orlander [20]	Hysterectomized	737	2584	At least 1 year prior to index UTI

The two largest studies with the longest observation periods both used the vaginal route of administration and showed a clear benefit. The fact that they used different types of estrogen (estriol and estradiol) suggests that the route of administration may be more important than the compound itself. For example, as currently prescribed, vaginal estriol cream appears to be more effective than its oral counterpart on the frequency of recurrent UTIs [11,15].

None of the studies identified the onset of recurrent urinary tract infections in the subjects included in their trials. Thus the symptoms in question may have predated the menopause and could have been due to a variety of other underlying pathologies, for example cystocele (leading to a persistent urinary residual), calculus, constipation etc. Some of the women may have suffered from lifelong urinary tract infections, for example in association with sexual intercourse. Symptoms could have been due to other underlying disorders causing inflammation of the bladder, such as interstitial rather than infective cystitis. These possibilities have not been reported anywhere in the published series.

The most important indications for long-term use of hormone replacement therapy are the prevention of osteoporosis and cardiovascular disease. For these indications systemic (high)-dose estrogen therapy is required, which normally necessitates the additional use of a progestogen to protect the endometrium in women with a uterus. None of the studies quoted here addressed this issue, and all but one employed low-dose estrogen therapy, for which a progestogen is considered unnecessary. This raises two important questions. First, would the use of high-dose systemic (oral, transdermal, subcutaneous or percutaneous) estrogens have had a beneficial effect on the rate of recurrent UTIs, and secondly, would the addition of a progestogen in such situations modify this estrogen effect?

Meta-analytical techniques are susceptible to publication (selection) bias [23], as it is possible that many similar studies with negative results would not have been published, and this potentially skews the data presented here. All the studies analysed used a different time interval, which further detracts from the validity of the overall result. We tried to mitigate this by examining the infection rate per 12 person-months. This transformation also aids the detection of any decrease in infection rates among women who were not cured.

It is interesting to note that the only study that looked at the incidence of postoperative UTIs showed reduced rates in women receiving preoperative estrogens, despite the fact that estrogen therapy had ceased 4 weeks prior to assessment. This study [22] also utilized the vaginal route of administration.

It is possible that estrogens may be beneficial in postmenopausal women as an adjunct to other prophylactic interventions, for example in combination with cranberry juice or its derivatives, agents that lower urinary pH, or long-term antibiotic therapy.

The available data indicate that the vaginal administration of estrogens reduces the frequency of recurrent

UTIs in postmenopausal women. However, there are still many unanswered questions. None of the RCTs utilized high-dose systemic estrogen replacement therapy, which may be effective. It remains unclear which is the best type of estrogen. Alternative forms of estrogens, such as phytoestrogens or selective estrogen receptor modulators (SERMs), have not been studied. There are no data regarding progestogen supplementation. These questions can only be answered by clinical trials. The optimal design would be a randomized placebo-controlled trial with sufficient power, although this may be difficult to institute given that systemic estrogen replacement has become standard climacteric therapy in much of the developed world.

*Acknowledgements.* The work of the HUT Committee was supported by an educational grant from Pharmacia and Upjohn. We are grateful to Jarl Ekberg for administrative help and facilitating communications.

## References

1. Johansson C, Molander U, Milsom I, Ekelund P. Association between urinary incontinence and urinary tract infections, and fractures in postmenopausal women. *Maturitas* 1996;23:265–271
2. Brocklehurst JC, Griffiths LL, Kalton J. Dysuria in old age. *J Am Geriatr Soc* 1971;19:582–590
3. National Center for Health Statistics. Ambulatory medical care rendered in physicians' offices. United States 1975. *ADV Data* 1977;12:1–8
4. Molander U, Milsom I, Ekelund P, Mellstrom D. An epidemiological study of urinary incontinence and related urogenital symptoms in elderly women. *Maturitas* 1990;12:51–60
5. Stamey TA, Timothy MM. Studies of the introital colonization in women with recurrent urinary infections. The role of vaginal pH. *J Urol* 1975;114:261–263
6. Molander U, Milsom I, Ekelund P, Mellstrom D, Eriksson O. Effect of oral oestriol on vaginal flora and cytology and urogenital symptoms in the post-menopause. *Maturitas* 1990;12:113–120
7. Milsom I, Arvidsson L, Ekelund P, Molander U, Eriksson O. Factors influencing vaginal cytology, pH and bacterial flora in elderly women. *Acta Obstet Gynecol Scand* 1993;72:286–291
8. Stamey TA, Timothy MM. Studies of the introital colonization in women with recurrent urinary infections. II A comparison of growth in normal vaginal fluid of common versus uncommon serogroups of *Escherichia coli*. *J Urol* 1975;114:264–267
9. Stamey TA, Timothy MM. Studies of the introital colonization in women with recurrent urinary infections. III Vaginal glycogen concentration. *J Urol* 1975;114:268–270
10. Cardozo L, Bachmann G, McClish D, Fonda D, Birgerson L. Meta-analysis of estrogen therapy in the management of urogenital atrophy in postmenopausal women: second report of the Hormones and Urogenital Therapy Committee. *Obstet Gynecol* 1998;92:722–727
11. Cardozo L, Benness C, Abbott D. Low dose oestrogen prophylaxis for recurrent urinary tract infections in elderly women. *Br J Obstet Gynecol* 1998;105:403–407
12. Eriksen B. A randomized open, parallel-group study on the preventative effect of an estradiol-releasing vaginal ring (Estring<sup>®</sup>) on recurrent urinary tract infections in postmenopausal women. *Am J Obstet Gynecol* 1999; (in press)
13. Kirkengen AL, Anderson P, Gjersoe E, Johannessen GR, Johnsen N, Bodd E. Oestriol in the prophylactic treatment of recurrent urinary tract infections in postmenopausal women. *Scand J Prim Health Care* 1992;10:139–142
14. Kjaergaard B, Walter S, Knudsen A, Johansen B, Barlbeo H. Treatment with low-dose vaginal estradiol in post-menopausal

- women. A double-blind controlled trial. *Ugeskr Laeger* 1990;152:658–659
15. Raz R, Stamm WE. A controlled trial of intravaginal estriol in postmenopausal women with recurrent urinary tract infections. *N Engl J Med* 1993;329:753–756
  16. Brandberg A, Mellstrom D, Samsioe G. Low dose oral estriol treatment in elderly women with urogenital infections. *Acta Obstet Gynecol Scand* 1987;40(Suppl):33–38
  17. Parsons CL, Schmidt JD. Control of recurrent lower urinary tract infections in the postmenopausal woman. *J Urol* 1982;128:1224–1226
  18. Privette M, Cade R, Peterson J, Mars D. Prevention of recurrent urinary tract infections in postmenopausal women. *Nephron* 1988;50:24–27
  19. Oliviera SA, Klein RA, Reed JJ, Cirillo PA, Christor PJ, Walker AM. Estrogen replacement therapy and urinary tract infections in postmenopausal women aged 45–59. *Menopause* 1998;5:4–8
  20. Orlander JD, Jick SS, Dean AD, Jick H. Urinary tract infections and estrogen use in older women. *J Am Geriatr Soc* 1992;40:817–820
  21. Stouffer SA, Suchman EA, DeVinney LC, Star SA, Williams RM. The American soldier. Adjustments during army life. Volume I. Princeton, New Jersey: Princeton University Press, 1949
  22. Felding C, Mikkelsen AL, Clausen HV, Loft A, Grupe Larsen L. Preoperative treatment with oestradiol in women scheduled for vaginal operation for genital prolapse. A randomized double blind trial. *Maturitas* 1992;15:241–249
  23. Naylor CD. Meta-analysis and the meta-epidemiology of clinical research. *Br Med J* 1997;315:617–719

**EDITORIAL COMMENT: Cardozo et al. have done an excellent job reviewing the world's literature and providing us with a metaanalysis of the role of estrogens in the treatment of recurrent urinary tract infections in menopausal women.**

**It appears from the literature that estrogens do play a large role in treating recurrent urinary tract infections and the vaginal administration appears the most efficacious route of delivery. The type of estrogen was not important as long as it was administered through a vaginal route.**

**Also, it was interesting to see that there are very few studies regarding this condition and its treatment despite the very high prevalence of bladder infections in elderly menopausal females. This review points out that there is very little data in this area but that the available data suggest that estrogens play a large role in treating recurrent urinary tract infections in menopausal women. This is something we should all keep in mind when we see these patients.**

## Review of Current Literature

### The Supine Stress Test: A Simple Method to Detect Intrinsic Urethral Sphincter

Hsu THS, Rackley RR, Appell RA

Department of Urology, Section of Voiding Dysfunction and Female Urology, Cleveland Clinic Foundation, Cleveland, Ohio, USA  
*J Urol* 1999;162:460–463

The study assessed a non-urodynamic test to predict intrinsic urethral sphincter dysfunction (ISD) in women with stress incontinence. Forty-one women with symptoms of stress incontinence had a history and physical examination, followed by a supine stress test. This was performed in the lithotomy position, with 200 ml of fluid placed into the empty bladder; the patient was then asked to cough and strain (Valsalva). The loss of any fluid coincident with coughing or straining was seen as positive. An abdominal leak-point pressure (LPP) determination was made at a separate visit in the upright sitting position with 200 ml of dilute contrast material placed into the bladder through a 6 Fr lumen catheter. The patient was asked to Valsalva and/or cough, and fluoroscopy was used to detect leakage. Intrinsic sphincter dysfunction was diagnosed if LPP was less than 100 cmH<sub>2</sub>O. In 31 it was less than 100 cmH<sub>2</sub>O, 29 of whom had a positive stress test and 2 had a negative test. The LPP was greater than 100 cmH<sub>2</sub>O or negative in

9, of whom 1 had a positive stress test. The positive predictive value of the supine stress test was 96.7%, negative predictive value 81.8%, with a sensitivity of 93.5% and specificity of 90%.

#### Comment

The number of patients was very small in making this comparison: 100 or 200 patients would have made it more convincing. The percentage of patients overall with a low LPP was 30/41, or 73.2%. Considering the original description of McGuire in assessing surgical failures related to urethral function, only 13% of non-operated individuals were considered to have this condition. The current finding of over 70% with ISD is excessive. Most patients with stress incontinence having LPP will have a determination of less than 100 cmH<sub>2</sub>O. The LPP value to diagnose sphincter dysfunction is set too high, and it seems more rational to set a value less than 60 as ISD, 60–100 as indeterminate, and over 100 as normal. In this manner those with a value of 60–100 may be offered surgery, depending on other factors, which may lead to some having a retropubic suspension and others having a sling procedure. If one performs sling surgery for all patients there is no point in carrying out the test. The excellent long-term results for Burch colposuspension in indicated cases would appear to make the testing procedures valuable and recommended. It would be interesting to know how the testing correlated with patient management and outcome.