traversed the gracilis and adductor magnus muscles, it perforated the most dorsal portion of the adductor brevis muscle in 7 of the 10 passages dissected (Results section). Indeed, in 3 passages the tape did not traverse this adductor muscle. This finding again emphasizes the fact that the posterior branch of the obturator nerve is well away from the trajectory of the tape. It is absolutely true that the obturator nerve divides into 2 branches before or, more usually, directly at the level of the obturator canal.

In figure 3 in our article it must be understood that the adductor brevis muscle was sectioned at its origin and reflected, while the 2 branches of the nerve were spared by this dissection. These 2 branches (numbered 10 and 14) appear bound together after their exit through the obturator canal simply because they are supported by a lace. They do not correspond to the posterior branch with a corollary. Figure 4 in our article provides another example of such dissection, without any "upward retraction" of the nerve. It clearly indicates the direction of the helical passer and the evident divergent direction of the obturator bundle.

With regard to figure 6, *B*, a careful look reveals visualization of the location of the tape. The ends of the tape can be identified without difficulty—one end is close to the Foley catheter and the other is at the upper right corner of the photograph. Arrow 25 represents, as detailed in figure 1, the dorsal nerve to the clitoris and not the tape. Therefore, readers can clearly observe that the tape and the dorsal nerve to the clitoris are separated by the perineal membrane. Again, this finding was also observed by Raders et al in their series of 20 cadaver dissections.[2] Indeed, they found that "the tape's passage is deep to or above the perineal membrane under which the terminal branches of the perineal nerve course rendering them unsusceptible to injury."[2]

Furthermore, Achtari et al measured the distances between the dorsal nerve and the clitoris using various tension-free tapes, including TVT, SPARC®, Monarc™ subfascial hammock and TVT-O.[3] In their anatomical study, which was reported at the most recent International Continence Society meeting in Paris, the mean distances were similar among all devices assessed (ranging from 14 to 16 mm, with a minimum of 1 cm for the Monarc and TVT-O devices). Again, these findings are in contrast to the observations of Spinosa and Dubuisson. Many textbooks of anatomy clearly describe the course of the dorsal nerve of the clitoris—in its most anterior course (at the level where the TVT-O is inserted) it is demonstrated to be under the perineal membrane. Therefore, we question how Spinosa and Dubuisson performed their dissections.

Finally, our quotation of the anatomical studies of Delmas is correct. Delmas et al studied "10 female anatomical subjects preserved without formol, aged between 74 and 89 years."[4] They report that "the Uratape sling passed above the perineal membrane and crossed the levator ani muscle at the level of its puborectal part. It passed through the tendinous arch of the pelvic fascia and into the muscular and fascial attachments of the vagina."[4] In addition, in the article documenting the first known bladder perforation with TOT it is stated that the TOT traverses the levator ani muscle and the tendinous arch of the pelvic fascia,[5] with a reference to the original anatomical works of Delmas. Similar data were also presented at the annual meeting of the French Urology Association in 2002.[6] More recently, Delmas again observed in a series of 10 fresh female anatomical subjects 74 to 89 years old that "the tape passes above the internal pudendal pedicle and then through the levator ani muscle, crosses the tendinous arch of the pelvic fascia and continues in the middle third of the urethrovaginal septum."[7] It is highly intriguing that, depending on the abstract or article selected, Delmas finds a different trajectory of the TOT, ie under or through the levator ani muscle. What is the truth? This discrepancy is extremely confusing to the readers. For the sake of clarification full abstracts and reports of all studies quoted in our reply have been sent to the Editor.

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RE: IS BENIGN PROSTATIC HYPERPLASIA A RISK FACTOR FOR CHRONIC RENAL FAILURE?

A. D. Rule, M. M. Lieber and S. J. Jacobsen

J Urol, **173:** 691–696, 2005

To the Editor. We read with interest the article by Rule et al regarding the often neglected aspect of renal impairment in benign prostatic hyperplasia (BPH). As they have correctly indicated, the prevalence of renal impairment due to BPH is often underestimated in the general community.[1] We would like to draw the attention of the readers to recent developments taking place in terms of endogenous markers of glomerular filtration rate (GFR).

GFR is generally considered the best measure of renal function. Brown and O'Reilly report that the accurate measurement of GFR in urological practice has been neglected, leading to delayed recognition of renal impairment.[1] Unfortunately, serum creatinine (SCr) is still used as a screening tool for renal impairment in BPH. Other than the fact that SCr increases only after a decrease of 50% in GFR, it is fraught with analytical problems. In addition, interindividual variation can account for 93% and intraindividual variation for 7% of serum creatinine biological variation. Therefore, to lie outside the assay reference interval, some subjects may have to exceed 13 SD from the usual mean value, whereas in others a change of only 2 SD would be sufficient.[2]

SCr has also been shown to be an inadequate marker of renal function in the elderly population, where BPH is a significant problem.[3] Late referral due to failure to interpret mildly increased SCr by physicians also has an impact on morbidity, mortality and resource utilization. Mendelssohn et al, studying the referral pattern in Canada, reported that 84.3% of general practitioners would not refer their patients with an SCr of 120 to 150 [micron]mol/l, and almost 30% would not even refer patients with an SCr of 151 to 300 [micron]mol/l.[4] A similar attitude among general practitioners has been observed in the United Kingdom.[5]

Munshi et al, who studied the outcome of renal replacement therapy in the elderly, reported that obstructive uropathy is the second leading cause of end stage renal disease, accounting for nearly 23% of cases.[6] Early recognition and appropriate intervention in patients with mild to moderate renal impairment in the so-called "creatinine blind area" has been increasingly recognized as an important opportunity to delay the progression of renal disease and modify the risk factors for co-morbid diseases.[7,8]

Recent developments in new endogenous markers of GFR such as cystatin C have not made an impact among the urological community. Cystatin C is a low molecular weight glycoprotein, produced at a constant rate by all nucleated cells, unaffected by age, sex, race, muscle mass, dehydration or inflammation.[9] Cystatin C has been demonstrated to be a better marker than SCr for measuring GFR, and has been used increasingly by nephrologists in treating patients with renal transplants and diabetic nephropathy, and those receiving chemotherapy.[10–12] However, there is a paucity of studies using cystatin C as a marker of GFR in urology.[13] In future trials of interventions for BPH cystatin C may be used as a marker of renal

function to identify men at risk for early renal failure, much earlier than that detected by measuring SCr.

Respectfully,
Amrith Raj Rao, Roger O. Plail, Hanif G. Motiwala

and

Omer M. A. Karim
Conquest Hospital
The Ridge
St. Leonards-on-Sea
East Sussex TN37 7RD
United Kingdom

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Reply by Authors. We appreciate the thoughtful comments of Rao et al regarding the association between BPH and renal failure. Underscoring this association, we recently published a cross-sectional study in which we found that men with signs and symptoms of bladder outlet obstruction were about 3 times more likely to have an increased serum creatinine (age and comorbidity adjusted) than men from the general community.[1] While we agree that early recognition and treatment of renal failure are needed and that cystatin C holds promise as a more sensitive marker of renal failure, it may also be a marker of inflammation.[2] When interpreting cystatin C or serum creatinine levels it is important to recognize that the relationship between these analytes and GFR can vary in different populations.[3, 4] In the general population cystatin C levels appear to be affected by age, sex, C-reactive protein and cigarette smoking, independent of GFR.[3] With the expense and inconvenience of measuring GFR (eg inulin clearance) these serum analytes may be useful but we need to recognize that biological factors other than GFR may lead to increased levels.

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ERRATA

DYSFUNCTIONAL ELIMINATION SYMPTOMS

Volume 174, Number 4, Part 2, Page 1628: The author of the first Editorial Comment is Piet Hoebeke from the Department of Pediatric Urology and Urogenital Reconstruction, Ghent University Hospital, Ghent, Belgium, and not Stephen A. Koff.

RECEPTORS IN HUMAN FETAL TESTIS AND EPIDIDYMIS

Volume 174, Part 2, Number 4, Page 1698; Dr. Mario Maggi, Department of Clinical Physiopathology, Andrology Unit, University of Florence, Florence, Italy is a coauthor of the Editorial Comment.