Letters to the Editor/Errata

RE: ASSOCIATION OF HEMOSPERMIA WITH PROSTATE CANCER

M. Han, R. E. Brannigan, J. A. V. Antenor, K. A. Roehl and W. J. Catalona

J Urol, 172: 2189–2192, 2004

To the Editor. The authors report that a history of hemospermia was associated with a 73% increase in the odds of detection of prostate cancer among older men who were recruited into a community based prostate cancer screening study. On this basis they recommend that men older than 40 years presenting with hemospermia be screened carefully for prostate cancer. Putting aside the controversial issue of how best to investigate and manage hemospermia, I do not think that the authors have succeeded in demonstrating a “causal” link between hemospermia and prostate cancer detection that cannot be explained by confounding or bias. For instance the observed association could have been the result of detection bias. Unless the urologist recommending prostate biopsy was blinded to history of hemospermia in their patients, it is likely that the decision to perform biopsy, and, therefore, the probability of prostate cancer detection, was influenced by the presence of a positive history of hemospermia.

The authors did not present information on the percentage of patients who received biopsy among men with and without a history of hemospermia, but based on table 1 in the article it appears that those with hemospermia were more likely to have “suspicious” findings on digital rectal examination. If that outcome translated into an increased likelihood of biopsy, then it may explain the observed association. The authors seem to suggest that hemospermia could be a marker of chronic prostatitis, and that the latter condition is the one etiologically linked to prostate cancer. Neither claim is supported by the findings of this or other studies in the literature. However, if the authors think that this model is plausible, then a more appropriate analysis would have been to adjust for the confounding role of prostatitis to determine whether history of hemospermia has a predictive value independent of its association with prostatitis.

Finally, the lack of information regarding the onset and course of hemospermia further compromised the ability of the study to determine the value of hemospermia as a diagnostic marker of prostate cancer. It is unlikely that a single episode of hemospermia in the distant past would have the same diagnostic usefulness as a recent one. These caveats, combined with the limited precision of the estimates of the study, mean that the question regarding the nature of the relation between hemospermia and prostate cancer remains unanswered for the time being.

Respectfully,
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Reply by Authors. We appreciate the comments of Mahmud. We certainly did not prove a “causal” relationship between hemospermia and prostate cancer. However, we provided a potential mechanism linking the 2 entities. We believe the observed relationship between these 2 entities is not a result of detection bias. For example there existed strict criteria (increased prostate specific antigen [PSA] level and/or suspicious digital rectal examination [DRE]) for recommending prostate biopsy in our community based prostate cancer screening study. No patient underwent biopsy because of hemospermia alone. In fact, the urologist recommending prostate biopsy did not know the hemospermia history of the subjects. Additional analysis revealed that 31% of men with a history of hemospermia underwent biopsy, compared to 23% of those without such a history. This statistically significant difference in biopsy rate (p = 0.036) occurred because more men with hemospermia had either an abnormal PSA level or a suspicious DRE. More importantly, in multivariate logistic regression analysis hemospermia was a predictor of prostate cancer diagnosis even after adjusting for age, and PSA and DRE results.

We agree with Mahmud that detailed information on the duration and severity of hemospermia may have better illustrated the relationship between hemospermia and prostate cancer. However, we do not have this information in our database. Despite these shortcomings, our study suggests that there is an association between prostate cancer and hemospermia in our prostate cancer screening population.

As numerous studies have shown that a family history of prostate cancer is a predictor of prostate cancer detection, the definitive “causal” relationship between hemospermia and prostate cancer can be proved only after further research. When a man presents with hemospermia the urologist should inform him of observed increased odds of prostate cancer detection in men with hemospermia, and proceed with a thorough clinical evaluation. The patient and the doctor together should decide whether prostate biopsy is indicated.

RE: RADICAL RETROPUBIC PROSTATECTOMY. HOW OFTEN DO EXPERIENCED SURGEONS HAVE POSITIVE SURGICAL MARGINS WHEN THERE IS EXTRAPROSTATIC EXTENSION IN THE REGION OF THE NEUROVASCULAR BUNDLE?


J Urol, 173: 446–449, 2005

To the Editor. This article summarizes work from 2 of the most experienced radical prostatectomists in the world, describing the frequency of positive surgical margins and addressing when it is safe to preserve the neurovascular bundles. However, several key points related to the ability of newer surgical approaches (laparoscopic and robotic) compared to the gold standard need addressing.

Prostate cancer surgery has gone through significant evolution since the first prostatectomy was performed. Thanks to the excellent work by Walsh,3 defining the anatomy of the prostate and its surrounding structures, the sequelae of incontinence and erectile dysfunction are no longer the expected norms following radical retropubic prostatectomy. The goals of cancer eradication can be coupled with the preservation of continence and erectile function. Despite this advance, prostate cancer surgery remains a procedure associated with significant morbidity.

Laparoscopic prostatectomy was first performed in 1992 and became a commonly performed procedure by 1998, when reproducibility of the procedure was reported with oncological results similar to the open experience.4–6 Robotic urological surgery debuted in 2000. As opposed to the pure laparoscopic approach, a greater number of institutions are embracing robotic prostatectomy due to the advantages and shorter learning curve associated with the use of the robot. Many surgeons with no significant experience in laparoscopy have made the transition from open to robotic surgery, offering their patients the advantages of minimally invasive surgery.7

Without a doubt a surgeon works best when all of the senses are engaged. The main disadvantage of the da Vinci robot is the lack of proprioception and haptic feedback. Does the improved 3-dimensional visualization and well illuminated, magnified field afforded by the robot, coupled with the improved dexterity and movement similar to the human hand, compensate for the lack of proprioception?

Regardless of the surgical method used, surgeons with large surgical volumes at large medical centers have improved outcomes compared to others with less experience. The robotic surgeon relies...
on visual cues and the ability to identify proper tissue planes to perform adequate cancer surgery. One is in awe when looking through the surgical console of the da Vinci robot. It is an unparalleled view of the surgical field, where small anatomical details come alive. Does one have to touch to appreciate fine structural details? The surgeon has full control of the camera, guiding it close to the tissue, allowing visualization of angles not easily seen with the naked eye. The perceived closeness to the surgical organ gives the surgeon the sense of being inside the patient. However, only with proper experience can the surgery be performed safely, eradicating the cancer while preserving the structures responsible for erectile function and urinary continence. With experience the surgeon becomes more confident in performing a bloodless field and performing anatomical radical prostatectomy.1

Experience matters in all disciplines. With proper scientific experiments Walsh was able to redefine prostate surgery and improve outcomes in men affected with this common malignancy.1 We need to allow proper evaluation of all available technologies to determine their place in the surgical armamentarium.

Respectfully,
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Reply by Authors. The role of mast cells in mediating bladder inflammation is undisputed. However, the effect of RDP58, a novel anti-inflammatory agent, on mast cell function remains uncertain. Based on our research we surmise that the powerful anti-inflammatory effect of RDP58 is mediated by its known mechanism of action—inhibition of the signal transduction cascade responsible for the expression of inflammatory cytokines by leukocytes, including mast cells.

We are pleased by the interest in our research investigating the intravesical effects of RDP58 in experimental cystitis. In this study RDP58 decreased inflammation and production of neurotrophic factors in vivo, and it might someday have a role in treating bladder disorders with an inflammatory component, such as IC. van Ophoven brings the interesting issue of mast cells to light in this letter, discussing several points that we would like to address.

van Ophoven opens by alluding to 2 references that he believes provide insight into the pathogenesis of cystitis.1,2 We disagree, as both references refer to in vitro experiments using less established models of cystitis. His first reference is to a study by Abdel-Mageed et al of the role of Nfkb on the induction of pro-inflammatory cytokine gene expression in human bladder carcinoma T24 cells.3 Although the authors also examined cytokine levels in the urine of patients with IC, the study is limited in its examination of IC pathogenesis. By citing this reference as support for neuroinflammatory processes as the basis for the pathogenesis of interstitial cystitis van Ophoven is extrapolating unrelated findings obtained in a cancer cell line to humans using the model of inflammation.

The article by Sun and Chai concludes that purinergic receptors, P2X3 subunits, are expressed by cultured IC bladder urothelial cells and are up-regulated during in vitro stretch.4 Although the results are interesting, they do not explain the pathogenesis of cystitis, nor do experiments where cultured urothelial cells are artificially stretched accurately model the IC disease process. While multiple risk factors for cystitis exist, each must be considered as an experiment. The results of any single experiment cannot be extrapolated to the population of patients with interstitial cystitis in an attempt to explain the pathogenesis of the disease.

Regarding our experiment, van Ophoven notes the lack of significant differences in mast cell counts between LPS exposed and saline control bladders. While the observed mast cell counts in our study supported the hypothesis that LPS causes inflammation (ie an in-

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increase in mast cell counts) and that RDP58 decreases inflammation (ie reduces mast cell counts in LPS exposed bladders), the differences were not statistically significant (see table in article). As briefly mentioned in our article, the technique used to quantify mast cells is not the most sensitive technique because fixation in 10% formalin may potentially block cationic dye binding by the granules, rendering some unstainable by Giemsa.6 Additionally, toluidine blue and hematoxylin visualized a mean number of 3 mice per group may not have provided sufficient power to identify a statistically significant difference, given the stringent statistical methodology used. We strongly disagree with the characterization by van Ophoven of this methodology as a “technical flaw.” We used the results demonstrated by an animal model and extrapolated the implications of such a result for the study of mast cells function via an undetermined mechanism. We informed readers to use caution in conclusions drawn because they are the fruit of technology. Unfortunately, certain readers prefer to draw their own biased conclusions.

His second point was to reiterate the published result that a decrease in bladder inflammation was not accompanied by a statistically significant reduction in mast cell counts. As outlined in the table in our article, RDP58 decreased observed mast cell counts at 24 hours in LPS exposed bladders but not in normal bladders. We informed readers to use caution in conclusions drawn because they are the fruit of technology. Unfortunately, certain readers prefer to draw their own biased conclusions.

van Ophoven also mentions the lack of conclusions regarding the effect of RDP58 on mast cell function. The published experiments only examined mast cell numbers. No experiments were performed, so any discussions regarding mast cell function remain speculative. While van Ophoven states that our findings regarding mast cells were partly unfinished, our discussion took into consideration the limitations of the animal model and avoided extrapolation of partial findings to generalized conclusions regarding interstitial cystitis. However, considering the profound anti-inflammatory effect of RDP58 in light of a moderate suppression of mast cell counts, it is probable that RDP58 may attenuate mast cell function via an undetermined mechanism.

van Ophoven also comments that mast cells may have a beneficial role in infection, in contrast to what previous studies have suggested. We invite van Ophoven to read publications from our group that state the contrary.7–16 Instead, he cites an elegant study that elucidated the role of mast cells in E. coli murine bladder infection. In that study Malaviya et al used acute bacterial bladder infection with a live bacterial strain, namely type 1 fimbriated E. coli J96, in mast cell deficient (WBB6F1-1/-) and normal congenic control (WBB6F1-1/-) mice.3 They found that control mice had development of severe histological inflammation characterized by neutrophil infiltrates and edema (similar to that observed in our study) in response to LPS, whereas RDP58 suppressed the inflammatory response by 80% at 24 hours. However, mast cell deficient mice did not mount a significant inflammatory infiltrate and had prolonged bacterial infection. The authors concluded that mast cells are instrumental in the recruitment of neutrophils at the infection site, and, thus, facilitate in clearing the bacterial infection. It is interesting that mice with double mutations at the W loci have a variety of phenotypic abnormalities, including a profound deficiency in the numbers of tissue mast cells, macroscopic anemia, age-dependent changes in intraepithelial lymphocyte populations in the gastrointestinal tract and other nonmyeloid abnormalities.7 To be useful in determining the role of mast cells in certain pathology, bone marrow transplantation (which repairs the mast cell deficiency and macroscopic anemia) was performed, so any discussions regarding mast cell function remain speculative. While van Ophoven states that our findings regarding mast cells were partly unfinished, our discussion took into consideration the limitations of the animal model and avoided extrapolation of partial findings to generalized conclusions regarding interstitial cystitis. However, considering the profound anti-inflammatory effect of RDP58 in light of a moderate suppression of mast cell counts, it is probable that RDP58 may attenuate mast cell function via an undetermined mechanism.

The study by Malaviya et al differs from our study because live bacteria were used, as opposed to simply a component of the cell membrane (ie E. coli LPS). Nevertheless, the aforementioned study corroborates our findings, as well as those of our collaborators, who also found that mast cells are necessary in the recruitment of neutrophil infiltrates in the LPS of cystitis. In a study by Bjorling et al LPS does not cause cystitis in mice deficient in mast cells (kitW/v-/- and kitW/v-H11001-/-) whereas control mice develop severe inflammation similar to that observed in the current study. This finding suggests that the lesser inflammation seen in RDP58 treated bladders may have resulted from an attenuation of mast cell function, as RDP58 inhibits the production of inflammatory cytokines by leukocytes at a translational level.

Finally, van Ophoven teleologically notes that the similar TNF-α levels at 1 hour between LPS exposed groups (ie LPS controls and LPS RDP58 treated mice) might be explained by “activated and up-regulated mast cells initially defy the RDP58 challenge with subsequent cytokine release. . . . ” The TNF-α measurable from RDP58 treated tissue at 1 hour can be attributed to release of preformed TNF-α from leukocytes such as resting mast cells stimulated by LPS.4 By 4 hours TNF-α levels are not detectable by enzymatic immunosorbent assay. This is in agreement with the RDP58 mechanism of action, as it is known to stop production of TNF-α by disrupting the formation of the TRAFYK (TRAF6-MyD88-IRAK) protein complex, which, in turn, inhibits the signal transduction cascade responsible for the expression of Th1 type pro-inflammatory cytokines.4,5 With less TNF-α histological inflammation is significantly dampened in this LPS induced model of cystitis, as expected from published in vitro studies.4 In summary, the role of mast cells in mediating bladder inflammation is undisputed. However, the effect of RDP58 on mast cell function remains uncertain. Based on our research we surmise that the anti-inflammatory effect of RDP58 is mediated by its known mechanism of action—inhibition of the signal transduction cascade responsible for the expression of inflammatory cytokines by leukocytes, including mast cells.

REFERENCES


2. Sun, Y. and Chai, T. C.: Up-regulation of P2X3 receptor during acute cystitis, as expected from published in vitro studies.4,9


To the Editor. The authors report long-term follow-up of 30 of 125 patients (24%) who underwent primary proximal hypospadias repair by the senior author. Of these patients 25 underwent full evaluation, including uroflowmetry, with 5 (20%) having either fistulas or meatal stenoses. Some of these complications were detected during postoperative year 1, while others may have occurred later, with at least 1 case of meatal stenosis not found until long-term follow-up of more than 10 years. It would have been more useful had the authors clearly differentiated between early (during postoperative year 1) and late complications (at least 10 years postoperatively) for these patients. In addition, they should have compared their complications in their study group to those in the cohort not available for long-term followup to establish that the study patients were representative of the entire experience.

Of greater concern is the conclusion that preputial flaps are superior in the long term to urethroplasty using preputial or buccal mucosa grafts. The argument is that among these choices preputial flaps are less likely to develop stricture or balanitis xerotica obliterans. During the ongoing concerns regarding the use of nonabsorbable material inside the urinary tract, Fontana et al reported a 6% rate of stone formation during a mean followup of 20 months (range 8 to 47). Moreover, the rate of stricture (1%) at the ureteroenteric anastomotic site using mechanical absorbable staples, which may permit the reser- voir to be constructed more broadly laparoscopically. However, the original technique described by Fontana et al used nonabsorbable mechanical staples to bring together and detubularize the 2 central parts of the ureter with 1 row of absorbable staples, which may carry an increased risk of stone formation. The ongoing concerns regarding the use of nonabsorbable material inside the urinary tract, Fontana et al reported a 6% rate of stone formation during a mean followup of 20 months (range 8 to 47). Moreover, the rate of stricture (1%) at the ureteroenteric anastomotic site using mechanical absorbable staples, which may permit the reservoir to be constructed more broadly laparoscopically. However, the original technique described by Fontana et al used nonabsorbable mechanical staples to bring together and detubularize the 2 central parts of the ureter with 1 row of absorbable staples, which may carry an increased risk of stone formation. Additionally, the study by Fontana et al included patients with a ureteral length of 1 cm or less, whereas our study included patients with a ureteral length of 2 cm or more. Therefore, it is likely that Fontana et al had a higher incidence of stone formation due to the shorter ureteral length. However, our study also included patients with a ureteral length of 1 cm or less, and we found a similar incidence of stone formation in this group (5% vs. 6%).

In conclusion, while preputial flaps may be a good short-term option, long-term follow-up should be used to determine their true efficacy. Future studies should compare the incidence of stone formation and stricture formation between preputial flaps and buccal mucosa grafts to determine which technique is superior in the long term.
various modern repairs are insufficient to allow strong recommendation of one technique over another.

Respectfully,
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2. Elbakry, A.: Complications of the preputial island flap-tube urethroplasty. BJU Int, 84: 89, 1999

Reply by Authors. We appreciate the thoughtful comments of Snodgrass regarding our article. The real point we wished to make was a simple one. All forms of nongenital epithelium used for substitution urethroplasties have some degree of late complications that are not seen with genital skin. Accordingly, we would always advise use of genital skin, even if it requires a staged operation. We agree with Snodgrass that, of the nongenital skin epithelium, buccal mucosa is the best.

RE: A 7-YEAR EXPERIENCE OF GENETIC MALES WITH SEVERE PHALLIC INADEQUACY ASSIGNED FEMALE

W. G. Reiner and B. P. Kropp
J Urol, 172: 2395–2398, 2004

To the Editor. The authors followed 18 genetic males with congenital phallic inadequacy for 7 years. Of 15 patients who were sex assigned female soon after birth 8 reverted back to male. None of these 8 patients was ever attracted to the male sex (if of age) at any time. Moreover, all expressed a sort of cognitive dissonance when they were being reared as girls. Of the 7 patients who continued in a female context 6 (1 was 3 years old) maintained an orientation to female gender. However, their parents noticed that these 6 children appeared not to fit in well with female peer group of play, but instead preferred to associate with boys in their rough and tumble world.

The authors concluded logically that theories proposed in the early 1970s largely based on the writings of Money and Ehrhardt could not be substantiated under careful longitudinal followup of clinical subjects. Among others, Money and Ehrhardt posited a priori that prenatal androgenic influence could be ignored as a nonentity, so that sex assignment could be recreated carte blanche after birth in instances where the male phallus was dwarfish, deformed or altogether wanting as a recognizable member. Without any previous data giving substance to this freely made supposition they proclaimed that psychosexual identity was a tabula rasa, which could be made over without impunity to suit the circumstances of the crisis. In effect a male child could— with plastic phalpoplasty, careful concealment of the factual nature of his sex from himself, parental dissimulation and extreme feminizing enculturation— grow up to be a girl/ woman without reproductive functions but would nevertheless regard himself/herself as a girl. This outcome would only hold true providing the tightly knit web of conspiratorial silence was maintained with perfect fidelity.

As it actually turned out, the original patient of Money and Ehrhardt, who was presented to the scientific world as a true pink room female (while genotypically XY), was shown to be something entirely different shortly after their findings were published. To wit, the paradigm had gone native, so to speak, when he realized that he was truly masculine and could not keep up the pretense any further, even to his own conscience, which seemed to be the prime mover in this return to the blue room. This surprising reversal was made publicly notorious by Diamond and Sigmundson, whose historical interest in researching this topic stretches back to the 1960s and includes a wealth of clinical material to support the contention that all sex assignment really achieves is making genitally malformed boys into sexually mutilated boys/men who would have rather kept their short penises than be plastically transformed into males with vaginal-simulacra whose external environment had to be maintained as alchemically feminine by a cabal of physicians, nurses and trained parents following the lead of the recognized experts. In essence, it was necessary to set up a make-believe psychossexual Potemkin village to keep the facts under wraps.

In 1999 Diamond had come to the same conclusion as Reiner and Kropp, that the tabula rasa theory had no basis in reality. His longitudinal followup demonstrated clearly that prenatal androgenic influences are present and are such an integral factor in the sexual makeup of a male child that no matter how good the intentions of the clinical team in providing for the best long-term interests of little Johnny, the best thing that can be done is just to leave the whole thing alone. Education and reassurance to the parents, to be sure, are essential in smoothing out the transition from the nursery to the home. However, surgical and psychossexual assignment were regarded as measures conceived in a cauldron of haste and make-believe. Furthermore, according to Diamond, by tampering with the unseeably dimensions of the small (or even missing) penis of some unfortunate, no matter what you try to do to make him into a girl, he is not going to be happy until he goes back to being a boy again. The old saying is you can dress them up but you cannot take them out.

Diamond has called for a moratorium on sex assignment surgery. As Shakespeare said, “Wisdom cries out in the streets, and no man regards it.” We should thank Reiner and Kropp for adding further credenda to the plea of Diamond to call a halt to this type of approach, which actually appears to produce more harm than good.

Respectfully,
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Reply by Authors. We thank Flanagan for his comments. However, we must stress that we do not in any manner call for a moratorium on surgical interventions for children with phallic inadequacy or genital abnormalities. Rather, we would stress the medical imperative of applying evidence based surgical interventions for each category of these conditions—as for any pediatric surgical disorder.

RE: PRIMARY BLADDER NECK DYSFUNCTION IN CHILDREN AND ADOLESCENTS I: PELVIC FLOOR ELECTROMYOGRAPHY LAG TIME—A NEW NONINVASIVE METHOD TO SCREEN FOR AND MONITOR THERAPEUTIC RESPONSE

A. J. Combs, N. Grafstein, M. Horowitz and K. I. Glassberg
J Urol, 173: 207–211, 2005

To the Editor. Without entering into a discussion of the article itself, its clinical issues, the editorial comment and the reply by authors—which would widely exceed a letter to the editor—I think that the urodynamic tracings on figures 2 through 4 in the article demand some additional discussion regarding the urodynamic signal quality as well as the interpretation. With regard to figure 3 at first glance the overall signal quality appears to be good. However, the fact that detrusor pressure (pD) decreases to a significantly nega-
tive value of almost −10 cm H2O at 16:12 minutes requires some discussion, as it may indicate a relevant signal quality problem. In addition, the abdominal pressure signal demonstrates 2 pressure waves, which need some consideration. An increase in intravesical pressure (pves) usually reflects abdominal straining and is concurrent with an equivalent increase in intravesical pressure (pves) or rectal activity. Theoretically, but unlikely, the impact on pves could be masked by detrusor relaxation. None of these problems can be resolved from the short segment of tracings shown here, and a definite answer can be found from careful analysis of the complete study tracings. However, undoubtedly the signal problems need to be considered explicitly for the interpretation. They seemed to be ignored here, and figure 3 is not a good example of urethral signal quality.

According to the text, the characteristics of primary bladder neck dysfunction (PBND) are “poor urinary flow rate despite adequate or increased voiding pressures in conjunction with a prolonged interval between the initiation of a bladder contraction and the start of urinary flow. . . . and a quiet/unrelaxed external urethral sphincter,” and “the common denominator . . . is functional outlet obstruction.” Thus, does the urodynamic study in figure 3 support this assertion? The tracings demonstrate during the pre-voiding phase possibly normal pressure values for an 11-year-old boy but with some ongoing detrusor activity that is either spontaneous, and, thus, indicative of detrusor overactivity, or is part of initiation of voiding. This distinction cannot be decided here because it is not specified when permission to void was given, which is an important aspect when judging pressure/flow signals.

The urodynamic key parameters determined in figure 3 are an “opening time” of 7 seconds and an electromyography (EMG) “lag time” of 9 seconds (while the graph actually shows 10 seconds). The opening time is measured here from the moment when the pdet crosses the zero line, increasing from negative values to the beginning of flow. Thus, this finding cannot be accepted as providing a reliable diagnosis of a specific dysfunction. The crossing of the zero line is meaningless because of the artifactual starting point. One could consider taking the minimal, although negligible, pdet, which would bring the “opening time” even closer to the EMG lag time.” However, this minimal pdet value is just caused by the pabp wave, possibly an artifact of rectal activity. Thus, one would have to refer to the pves signal, which starts increasing a second later. The end point, beginning of flow recording, is not synchronous with bladder neck opening, particularly not at this low flow.

The authors have also chosen this example to demonstrate the correlation between pressure/flow signal and the EMG as the basis for the definition of EMG lag time. However, there are some problems with the “EMG” signals here similar to the flow/EMGs, which will be discussed later. The authors have, as inclusively on this issue, “quiescence,” of EMG activity and interpret it as a definite sign of urethral sphincter relaxation. This assumption is reasonable when the relaxation occurs from a sustained high EMG activity while the patient delays voiding and keeps the pelvic floor and sphincter contracted, and then relaxes and voids. However, all EMG examples shown here (and in the flow/EMG) are somewhat short bursts of activity. So the first question is, what makes the EMG activity increase?

In figure 3 we observe 3 short periods of EMG activity, 2 before voiding starts and 1 when voiding ends. This finding goes together with a slow undulating increase in flow up to 8 ml per second and in pdet up to 35 cm H2O, with an opening pressure of 20 cm H2O. These pressure and flow values exclude any urethral obstruction but prove a weak detrusor contraction. Finally, voiding is terminated after only a small volume voided, which makes a residual volume likely—against for a frightened young male undergoing a pressure/flow study and who is afraid to void (and shows vasovagal reactions). In addition, if the abdominal pressure waves indicate rectal contractions, then this is another reason why this boy has problems relaxing and initiating a good voiding contraction. The second abdominal pressure wave demonstrates a marked partial inhibition of detrusor contraction while flow is starting. This finding, in addition to psychogenic factors, may explain the poor voiding with a slow start and early termination. I hope that this discussion illustrates that high quality urodynamics can be interpreted in great detail

All parts of these tracings—the ongoing detrusor activity before voiding starts, some sporadic surface EMG activity, the slow undulating increase to a low detrusor pressure with the slowly increasing flow terminated by sphincter contraction after only a small volume voided, which makes a residual volume likely—are typical for a frightened young male undergoing a pressure/flow study and who is afraid to void (and shows vasovagal reactions). In addition, if the abdominal pressure waves indeed are rectal contractions, then this is another reason why this boy has problems relaxing and initiating a good voiding contraction. The second abdominal pressure wave demonstrates a marked partial inhibition of detrusor contraction while flow is starting. This finding, in addition to psychogenic factors, may explain the poor voiding with a slow start and early termination. I hope that this discussion illustrates that high quality urodynamics can be interpreted in great detail.

Similarly, the figures of all flow/EMG studies are not convincing. I can only support all the limitations discussed in the editorial comment that accompanies this article, i.e., that perineal or perianal surface EMG recordings are difficult to interpret, full of artifacts and often unrelated to urethral sphincter activity, and need careful plausibility control and repeated reproduction. Findings of poor flow rates and surface EMG activity are highly variable in children and usually improve or are resolved in repeated recordings. For all examples shown here the question has to be answered regarding what causes the sudden EMG increase before voiding. These are not EMG signals of sustained spincter activity where cessation indicates relaxation, but rather short bursts of activity. Such bursts can be correctly related to urethral sphincter activity at the end of voiding, as seen in figure 3, particularly when in agreement with the urodynamic signals. The burst before the begin of the voiding phase is not in agreement with the limitation and the poor quality of the tracings in figures 2 and 4. I am still confident in deducing that the sudden bursts in EMG activity demonstrated before voiding do not reflect sphincter activity, but rather straining to initiate voiding or other muscular activity related to simple positioning when standing in front of or sitting on the flowmeter, as is often seen in children.

Urodynamic signal quality and physical and physiological plausibility control also need to be considered before complex invasive urodynamics or a seemingly simple urodynamic test such as “EMG lag time” is accepted as providing a reliable diagnosis of a specific dysfunction such as PBND. The authors use the definition of an abnormally low flow rate with normal or adequate (whatever this means) voiding pressure, which indirectly implies some obstruction. They also state that “the common denominator . . . is functional outlet obstruction,” which may be significant or not. I often show an example in figure 3 where a subnormal voiding pressure excludes any degree of bladder outlet obstruction. This example also demonstrates that the flow rate is at least one other reason for a “prolonged opening time” and that is poor detrusor contractility, which is common in children, particularly young males, undergoing invasive or noninvasive urodynamics. So it is not reasonable to state that without anatomical obstruction or any delay in voiding any delay in voiding can only be presumed to be due to a dysfunctional bladder neck. Thus, the cogent report on clinical observations of children with voiding problems is diluted by noncogent urodynamics.

Respectfully,
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Reply by Authors. While we acknowledge the questions and concerns raised by Schaefer, we do not agree with many of the conclusions drawn in his critique of our article. The primary purpose of figure 3 in the article was to illustrate a particular point, to wit the concept of pelvic floor EMG lag time and its relationship to opening time. An entire study compressed to a single page width is equally open to the appearance of real or imagined defects. Compressed and expanded views can hide flaws or magnify apparent small aberrations.

The solution, as Schaefer rightly suggests, would be to publish an expanded view of the entire study. Unfortunately, that is not a realistic option with the space constraints for articles in this journal. We would be happy to send a copy of the complete study in question for review to anyone interested. In answer to the question regarding fluctuations in pabd they indeed represented abnormal rectal activity. The abnormal rectal activity is often present in patients with PBND, and is unrelated to abdominal straining or external anal sphincter activity.

The flow/EMG graphs in figures 2 and 4 in the article were performed on an analogue chart recorder. The chart speed is slow on these units (1 second per 0.5 mm), and the recorder was not turned on until patients were ready to void and positioned on or over the ureflow commode, hence the appearance of a relative burst of activity before EMG quiescence and flow. Again, the primary purpose was to illustrate and compare a normal lag time/normal flow pattern with a quiet pelvic floor and a prolonged lag time/abnormal flow pattern with a quiet pelvic floor (fig. 2), and the reverse situation in a patient with PBND receiving therapy (fig. 4). All of these patients underwent multiple studies before videourodynamic studies (VUDS) and during therapy. The findings were consistent on repeated studies.

While not a perfect system, direct observation, audio monitoring of EMG signal, coaching and ensuring that patients really are full and ready to void by sensation, and real-time bladder ultrasound measurement of volume should minimize errors related to inadequate filling, strain artifact or electrical interference. Are there sources of error with the use of patch electrodes? Absolutely! It was for that reason that we went to great lengths in the body of the article (methods and discussion sections) and the legend for figure 4 to acknowledge and address those potential sources of error. We were not oblivious to these pitfalls as suggested. It was for this reason we elected to use those particular examples as opposed to more visually perfect ones. While needle electrodes placed directly into the external anal sphincter (EUS) are undeniably the more accurate method of measuring EUS activity, their use is by no means sacrosanct. We have performed thousands of these flow/EMG studies during the last 15 years and found patch electrodes, when carefully placed, provide useful clinical information with minimal error. Equally important is the fact that compared to needle electrodes, patch electrodes are far better tolerated by senesce, neurologically normal patients who need pelvic floor/EUS evaluations (often repeatedly).

Contrary to what Schaefer suggests, these patients did not have impaired/defective detrusor contractility (see companion article on page 212 of the same issue), although they often had a prolonged lag time, along with normal or near normal voiding times, although not as perfect as expected. We have not seen diminished pressure/flow signal, the final arbitrator of what is abnormal. The patients with PBND did not have prolonged lag time/abnormal flow. That is because the lag time is abnormally prolonged and directly parallels prolongation of the opening time on VUDS. That increased lag time occurs in symptomatic patients who meet all of the other VUDS criteria for PBND (not other voiding dysfunctions), and that lag time, along with flow parameters and symptoms, normalizes with α-blocker therapy were the salient points of these two companion articles.

One final point on which we disagree is that regarding the making of the effect on pressure/flow signal, the final arbitrator of what is uroodynamically significant. While we do not deny its importance, it is the goodness of fit to patient clinical circumstances that the validity of all urodynamic data ultimately answer to.

The treatment of neurologically normal patients, particularly children, with lower urinary tract symptoms is often difficult. We believe that pelvic floor EMG lag time conceptually and practically is an important step in improving how these cases are evaluated and managed. It would be unfortunate if this forest were missed for what some perceive as weeds and trees.

RE: THE VALUE OF YOUR TIME: EVALUATION OF EFFECTS OF CHANGES IN MEDICARE REIMBURSEMENT RATES ON THE PRACTICE OF UROLOGY

Y. Lotan, J. A. Cadeddu, C. G. Roehrborn and K. H. Stage

To the Editor. Lotan et al report how Medicare physician reimbursement changes during the last decade have affected hourly reimbursement rates for various urological services. The authors conclude that changes in Medicare reimbursement during the last decade have resulted in significant changes in rates for different urological services, and that the near equity in reimbursement rates for evaluation and management (E&M) and surgical services will likely have an increasingly important role in future practice.

As American Urological Association (AUA) Health Policy Chair, I applaud these authors for their diligent research. This is the kind of information AUA members need to see—it helps educate urologists about the impact and importance of health policy. I would also like to add some perspective to the payment information in the article.

As indicated, in 1992 Medicare physician payments began to transition to a resource based relative value scale. The Centers for Medicare and Medicaid Services began to develop work relative value units (RVUs) in 1992, with the process essentially complete in 1995. As the authors mention, a second law was implemented in 1998—the Balanced Budget Act of 1997—requiring the Centers for Medicare and Medicaid to implement resource based practice expense RVUs, which were phased in between 1999 and 2002. Resource based professional liability RVUs were implemented beginning January 1, 2000.

Historically, governmental policy makers have thought surgical procedures were overvalued compared to E&M office visits. Therefore, a main goal of implementing a relative value scale was to shift money from surgical procedures to office visits. Although the AUA and other specialty societies lobbied to have hospital compensation and professional reimbursement increased relative to what was historically paid, the Centers for Medicare and Medicaid Services did not consider that aspect of this change, primary care physicians prevailed on this issue and the changes were implemented. Therefore, it is not surprising that the authors found near equity in reimbursement rates for E&M and surgical services. Although government policies and other factors have caused urology to become more of an office based practice than in the past, the AUA continues to seek ways to restore cuts to surgical procedures. In fact, one of the legislative and regulatory priorities of the AUA is to work with other surgical specialties to restore fair payment for major inpatient surgical procedures that have seen drastic payment reduction.

The article also discusses ways in which urologists have made up for lost revenue in response to payment changes, and cites the increase in the number of patients seen by a urologist in an average week. However, there are other key factors that have led to an...
increased number of patient visits, including a growing baby boomer population, greater consumer demand due to new technology and direct to consumer marketing, a reduction in the supply of new urologists and an increased rate of retirement.

Regardless of the type of urology practice (solo, single specialty group, multi-specialty group or academic), we now all “feed from the same trough” in that the payment system treats all of us equally.

Despite some of the seemingly bizarre aberrations in our fee schedule, we must continue to do the “right thing” for our patients.

Respectfully,
William F. Gee
AUA Health Policy Chair

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