ileum or ileocecal region) and supratrigonal bladder substitution (ileum without/with calcitonin administration) on the bone metabolism of male Wistar rats. After 6 months measurement of femur length (microradiography) and volume, bone mineral density, bone mineral content (dual photon absorptiometry), histomorphometry and conventional histology were performed. All animals in the ammonium chloride loading group showed compensated metabolic acidosis but normal bone analyses (no difference than controls). Although none of the animals in the bladder substitution group showed metabolic acidosis or impaired kidney function, bone analyses disclosed overall increased endocortical bone degradation with osteoclastic resorption lacunae and deficits of appositional periosteal bone growth as seen in osteoporosis or osteopenia. These osteopathic changes were not observed in the group with calcitonin administration, implying increased osteoclastic activity in the group without calcitonin. In contrast to this study by Hochstetler et al (no histology/histomorphometry), the results of Roth et al suggest the existence of a pathological mechanism independent of metabolic acidosis, which is at least partly confirmed by the only relevant clinical study on growth following enterocystoplasty. Wagstaff et al reported that the majority of patients with impaired growth had a normal acid base balance.[3] The incidence of borderline and overt metabolic acidosis was higher in patients with normal growth.

Although I agree that hyperchloremic metabolic acidosis may contribute to growth failure and impairment of bone metabolism following enterocystoplasty, I believe that the complexity of these phenomena deserves further investigation.

Respectfully,
Elmar W. Gerharz
The Institute of Urology
University College London Medical School
48 Riding House Street
London W1P 7PN
United Kingdom

1. Roth, S. and Gasser, J.: The risk of alterations of bone metabolism following urinary intestinal diversion: an animal model. Akt. Urol., **24:** 114, 1993.
2. Roth, S., Gasser, J., Catheline, M., Cipolla, B., Moulinoux, J. -P., Bernard Lobel, R. and Hertle, L.: Urinary diversion and bone metabolism: effects of metabolic acidosis and resection of different intestinal segments. J. Urol., part 2, **149:** 373A, abstract 643, 1993.
3. Wagstaff, K. E., Woodhouse, C. R., Duffy, P. G. and Ransley, P. G.: Delayed linear growth in children with enterocystoplasties. Brit. J. Urol., **69:** 314, 1992.

RE: INTERMEDIATE TERM ASSESSMENT OF THE RELIABILITY, FUNCTION AND PATIENT SATISFACTION WITH THE AMS700 ULTREX PENILE PROSTHESIS

F. B. Holloway and R. N. Farah

J. Urol., **157:** 1687–1691, 1997

To the Editor. This article is confusing. In 1988 Montague issued a plea in an editorial that all subsequent penile prosthesis articles should be formatted to reflect Kaplan-Meier survival curves.[1] Only in this way can we actually compare prosthesis to prosthesis and operator skill to operator skill. This article omits Kaplan-Meier survival curves and seems to have an inordinately high rate of primary implant complication. There is also a lack of information concerning infection, since 6 erosions but only 2 infections are noted. Most authorities consider erosion a sign of infection. The outcome data with regard to patient satisfaction are admirable but it would have been more informative if the authors had provided a more meaningful discussion of the freedom from revision survival rate with respect to mechanical failure, iatrogenic reason, patient infection or patient dissatisfaction.

Respectfully,
Steven K. Wilson
Wilson Urology Associates
2010 Chestnut
Van Buren, Arizona 72956

1. Montague, D. K.: Penile prostheses. J. Urol., **139:** 1031, 1988.

RE: QUANTITATIVE POLYMERASE CHAIN REACTION DOES NOT IMPROVE PREOPERATIVE PROSTATE CANCER STAGING: A CLINICOPATHOLOGICAL MOLECULAR ANALYSIS OF 121 PATIENTS

M. H. Sokoloff, C.-L. Tso, R. Kaboo, S. Nelson, J. Ko, F. Dorey, R. A. Figlin, S. Pang, J. deKernion and A. Belldegrun

J. Urol., **156:** 1560–1566, 1997

To the Editor. There is considerable interest in molecular markers as an aid in prostate cancer diagnosis. Prostate specific antigen (PSA) was the first of these molecular markers. When an elevation of PSA is found in the serum, prostate cancer is likely.[1]

Recently, 2 other markers, prostate specific membrane antigen and free PSA, have been introduced. The ratio of free-to-total PSA can be used to diagnose prostate cancer more accurately in men whose PSA is elevated.[2–4] If the free-to-total PSA ratio is less than 7%, the likelihood is that the patient has cancer. If the free-to-total PSA is more than 25%, no cancer is probably present. Prostate specific membrane antigen is of prognostic significance with elevation correlating well with more advanced cancer.[5] However, even when these molecular markers are used to evaluate men with possible prostate cancer, biopsy is still necessary for a definitive diagnosis. Thus, additional markers are actively being sought.

This study suggests that the reverse transcriptase (RT) polymerase chain reaction (PCR) for PSA may be a valuable molecular marker for prostate cancer even if it offers no benefit for preoperative prostate cancer staging. A PSA messenger ribonucleic acid (mRNA) signal was detected in only 1 of 19 patients with benign prostatic hypertrophy. In contrast, a PSA signal was detected in 59% of the patients with stages pT1 and pT2 prostate cancer. Overall, the assay was 88% sensitive and 94% specific. The results of the assay could be combined with PSA, free-to-total PSA, age and race to produce a nomogram giving the probability before biopsy that prostate cancer was present. Partin et al have already derived such a nomogram for the prediction of pathological stage in prostate cancer.[6]

Respectfully,
Steven Lehrer
Department of Radiation Oncology
Mount Sinai School of Medicine
New York, New York 10029-6574

1. Murphy, G. P., Barren, R. J., Erickson, S. J., Bowes, V. A., Wolfert, R. L., Bartsch, G., Klocker, H., Pointner, J., Reissigl, A., McLeod, D. G., Douglas, T., Morgan, T., Kenny, G. M., Ragde, H., Boynton, A. L. and Holmes, E. H.: Evaluation and comparison of two new prostate carcinoma markers. Free-prostate specific antigen and prostate specific membrane antigen. Cancer, **78:** 809, 1996.
2. Chen, Y. T., Luderer, A. A., Thiel, R. P., Carlson, G., Cuny, C. L. and Soriano, T. F.: Using proportions of free to total prostate-specific antigen, age, and total prostate-specific antigen to predict the probability of prostate cancer. Urology, **47:** 518, 1996.
3. Luderer, A. A., Chen, Y. T., Soriano, T. F., Kramp, W. J., Carlson, G., Cuny, C., Sharp, T., Smith, W., Petteway, J. and Brawer, M. K.: Measurement of the proportion of free to total prostate-specific antigen improves diagnostic performance of prostate-specific antigen in the diagnostic gray zone of total prostate-specific antigen. Urology, **46:** 187, 1995.
4. Catalona, W. J., Smith, D. S., Wolfert, R. L., Wang, T. J., Rittenhouse, H. G., Ratliff, T. L. and Nadler, R. B.: Evaluation of percentage of free serum prostate-specific antigen to improve specificity of prostate cancer screening. J.A.M.A., **274:** 1214, 1995.
5. Murphy, G., Ragde, H., Kenny, G., Barren, R., Erickson, S., Tjoa, B., Boynton, A., Holmes, E., Gilbaugh, J. and Douglas, T.: Comparison of prostate specific membrane antigen, and prostate specific antigen levels in prostatic cancer patients. Anticancer Res., **15:** 1473, 1995.
6. Partin, A. W., Yoo, J., Carter, H. B., Pearson, J. D., Chan, D. W., Epstein, J. I. and Walsh, P. C.: The use of prostate specific antigen, clinical stage, and Gleason score to predict pathological stage in men with localized prostate cancer. J. Urol., **150:** 110, 1993.

To the Editor. We read with interest this article in which the application of a quantitative RT-PCR of PSA mRNA and prostate specific membrane antigen mRNA, as representative of prostate cancer cells in the peripheral circulation, was addressed. The purpose of this prospective study was to explore the potential of this assay to