Conclusion

We find that duplex Doppler evaluation of post prostatectomy patients reveals a low prevalence of cavernous arterial asymmetry in individuals with vasculogenic impotence. This finding, in conjunction with the high prevalence of cavernous asymmetry seen in individuals with normal vascular responses, does not support the theory that accessory pudendal sacrifice leads to post-RRP vascular impotence.

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Editorial Comment

This study was undertaken to determine whether sacrifice of the accessory pudendal artery (APA) at the time of radical retropubic prostatectomy (RRP) is an important determinant of post-operative erectile dysfunction. The authors hypothesize that if this artery is unilaterally destroyed that postoperative differences in peak flow velocity of the two cavernosal arteries should be demonstrable. The data do not show a high incidence of cavernous artery asymmetry in patients with vascular impotence following RRP, and the authors conclude that APA sacrifice does not lead to vascular impotence.

This is a well-constructed study which answers the question about as well as it can be currently answered. On the other hand, it underscores our lack of ability to precisely measure what we want to measure. What does a $>10\,\mathrm{cm/s}$ flow velocity difference between the two cavernosal arteries really mean? Do the doses of PGE₁ utilized in this study (or any study) appropriately answer the questions posed? Finally, if the APA is important in erectile physiology, what is its innervation?

G Benson

Editorial Comment

These authors evaluated penile inflows in 103 previously potent patients who underwent nerve sparing radical retropubic prostatectomy for the treatment for prostate cancer. They evaluated whether identification or sparing of the accessory pudendal artery (APA) was of any use. They tested the hypothesis that the presence of the APA in a significant number of men and its ligation would contribute significantly to post operative impotence. Should this be true there would be an increased incidence of asymmetry between right and left cavernous arterial flows among these post-operative patients with vascular impotence.

They found that 26% of patients postoperatively developed excellent rigidity consistent with isolated neurogenic impotence, 23% had adequate vascular responses and 52% had inadequate rigidity consistent with vascular insufficiency. In testing the hypothesis 48% of the patients with excellent erectile response exhibited cavernosal artery flow asymmetry suggesting that sacrifice of a unilateral accessory pudendal artery *did not* play a role in postoperative impotence. The authors properly emphasized considerable variability in the anatomy of the APA. In addition the variations of the entire penile vascular system supply should be reemphasized. The authors demonstrate a null-hypothesis

which agrees with opinion of Walsh's group that routine sparing of the APA might not be productive and might actually impair the nerve sparing dissection.

It is apparent that a further follow up to this study would be useful if the same cavernosal asymmetry was sought pre- and post-operatively and a more meticulous comparison with actual operative findings were made. Carrying this one step further, angiography might also be helpful but this would be a great deal to put a patient through. Overall 49% of their patients had adequate penile vascular responses suitable for intercourse; and this is a most commendable result. The lesson to be learned from this study is that the classical technique of nerve sparing prostatic dissection should not be altered.

RG DePalma