

**MEditorial March 2011**

**“Shrinking the Prostate”**

**The medication Proscar, now generic as finasteride was initially felt to be a wonder drug. The word with this, as well as the more recently developed Avodart (dutasteride), is that it would reduce the “surgical business” of urologists by reducing greatly the number of operations, especially transurethral resection of the prostate (TURP) for benign enlargement (BPH).**

**These drugs are engineered specifically to block an enzyme found in “male” cells (abundantly in the prostate) which converts testosterone to dihydrotestosterone (DHT). DHT is far stronger than testosterone in promoting prostate growth. By not effecting blood testosterone levels, these so called 5-ARI drugs have a relatively low incidence of causing impotence (although some men taking these do still complain of reduced libido and sexual function) or other hormonal abnormalities, such as breast swelling/tenderness.**

Studies do reveal these drugs modestly reduce the size of the prostate over many months; it can take 6 or more months to detect a clinical difference. Avodart, by blocking two different “types” of the 5-alpha reductase converting enzyme, may “shrink” the prostate faster.

Unfortunately, it is difficult to assess the degree of shrinkage unless the man is in a study whereby prostate gland size is measured ‘before” and “after” using, e.g., transrectal ultrasound. The urologist’s finger turns out to be a quite a crude measuring stick; and it is rare to see dramatic changes by simply palpating the gland on rectal exam from year to year. More often, I see a softening of the gland which may mean reduced size or at least less compression of the 1<sup>st</sup> part of the urethra. More relevant is that reduction in prostate size does not always equate with improvement in voiding, which, after all, is the real goal here. How many men will be happy with chronic use of a drug which may measurably shrink the prostate but not improve flow or reduce frequency/nocturnal urinating/urgency, etc.? There are many theories as to this discrepancy, but I suspect the most likely

explanation is muscular damage to the chronically blocked bladder; permanent loss of strength of the bladder may not be significant helped, let's say, by a 10-25% reduction in prostate size. Also, there are different types of benign tissue comprising what we call BPH and response to these "shrinking" drugs may depend on one's mix (glandular versus stromal) Excess nerve innervation of the prostate (even in those glands with unimpressive size) may make it more ideal in some cases to avoid these drugs and rely on alpha-blockers (see below) .

Further studies by the drug companies (or in urology clinical trials, sometimes sponsored by the pharmaceutical manufacturers!) point to a lower incidence of acute retention (blockage necessitating at least a temporary catheter) and reduced need, in one's future, to have a TURP. When one reads between the statistical lines, it is apparent these risk reductions are relatively minor ( perhaps 5-10% of an already low number--since a minority of men with BPH symptoms

either go into retention or need/have surgery anyways) , and may not warrant the personal and societal costs of these drugs and the occasionally bothersome side effects.

I see a fair number of men placed on these drugs as 1<sup>st</sup> line treatment for BPH, even on the basis of sheer enlargement as opposed to symptomatic enlargement. There are many men, prescribed 5 ARI drugs, who have minimally enlarged glands and are far less likely than those with large glands to have a response. The rate of drug discontinuance in these patients is high. I would avoid prescribing these just because a prostate gland feels large to me.

In my practice, I usually use alpha-blockers (e.g., tamsulosin) as 1<sup>st</sup> line treatment for enlarged prostate symptoms, emphasizing dealing with that which bothers the patient as opposed to the size issue, per se.

Alpha-blockers do not shrink the gland--but block nerve impulses and in so doing, relax the pressure of the prostate on that part of the urethra within it. Since I do not like starting two new drugs at once (?)

which one caused the side effects) , I will wait to see the response to alpha-blockers for at least a month before deciding which patient may benefit, in the long run, from addition of a 5ARI drug. Not as often as I would like, some men in 3-6 months can come off the alpha-blocker and rely solely on the finasteride or Avodart.

As to risk reduction for prostate cancer, studies have been done and are still underway with both of the current 5ARI drugs. As regards Proscar, it did reduce the incidence of prostate cancer by over 15% over a 10-year period of observation; but it seemed to “select out” for patients with more high grade and aggressive prostate tumors. A higher percentage of these tumors, more likely to spread and cause harm, were seen in the finasteride group as opposed to the control/placebo arm of the study. Anything that treats the hormonal component of prostate cancer can, over time, effect “competitive advantage” and growth to those prostate cancer cells not responsive to hormonal manipulation. Since the less aggressive prostate cancers

**are easier to treat and can take decades to cause death, I myself am  
NOT advising patients, as of now, to use 5ARI drugs as a form of  
cancer prevention.**

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