ORCHIECTOMY? (Surgical Removal of the Testicles)

Considering Orchiectomy? Be aware that this surgical procedure does NOT stop all production of testosterone.

An alternative to Clinical Castration is Surgical Castration, the removing of the testicles, thus another method of preventing testicular production of testosterone. Since some men are bothered by the absence of the testicles, prosthetic testicles can be inserted before the surgical incision is closed so that an outward appearance of the scrotum, as well as the personal "feel," looks the same as pre-orchiectomy.

Surgical Castration/Orchiectomy, removes the testicles, thus preventing testicular production - the primary production of likely over 90% of testosterone - however an orchiectomy does NOT stop all production of testosterone. The adrenal glands are another area of metabolized testosterone production, albeit to a much lower extent than testicular, but with the reduction of testosterone from clinical or surgical castration, the adrenal glands react with more production of testosterone than usual.

In view of adrenal gland testosterone, the testosterone level should still occasionally be checked to make sure that level remains at "castrate" levels near or below 20ng/dl..
Many physicians as well as patients assume that with the patient having an orchiectomy his production of testosterone is halted. As noted above, not so. As Medical Oncologist Stephen B. Strum, M.D., FACP, notes "The therapy the physician selects to deprive the tumor cell population of androgen may have consequential effects on the course of PC. For example, work by Sciarra et al has shown that 37% of men undergoing orchiectomy have a reflex increase in the production of the adrenal androgen precursor androstenedione. Androstenedione is metabolized within the prostate cell (both benign and malignant prostate cells) into testosterone (see Insights July 1999, pp 3-4 and October 2000, page 4). If the physician assumes that orchiectomy has resulted in a castrate testosterone (< 20ng/dl) and does not monitor the serum testosterone, almost 40% of these patients face a significant risk of disease progression. If progressive PC occurs, it would likely be assumed to be a reflection of androgen independent PC. In fact, it may be due to the reflex stimulation of the pituitary-adrenal axis due to the lack of testosterone — the production of androstenedione — and the subsequent conversion of this androgen precursor to testosterone within the prostate cell. The

body tries to maintain balance or homeostasis in regard to testosterone and in doing so uses its backup systems.”