

## Flutamide Plus Castration in Patients with Previously Untreated Prostate Cancer

TO THE EDITOR:

The publication by Noldus *et al.*<sup>1</sup> has important implications for the most efficient hormonal therapy of localized prostate cancer. Although it is well demonstrated that the androgens acting in the prostate are of both testicular and adrenal origins,<sup>2</sup> quantitative clinical data such as those described by Noldus *et al.* are particularly timely since the majority of patients are now diagnosed at the clinically localized stage and androgen ablation is successfully used in association with the standard curative therapies, namely radical prostatectomy<sup>3</sup> and radiotherapy.<sup>4</sup>

Noldus *et al.* show that combination therapy using flutamide in association with surgical or medical (luteinizing hormone-releasing hormone [LHRH] agonist) castration causes an average 47.4% decrease in prostate size at 6 months of treatment while monotherapy with flutamide requires an average 38.7 months of treatment to reach the same degree of inhibition, namely 47.9%. At 6 months, the mean decrease in prostate size achieved with flutamide alone was 30.9%, a value similar to the inhibition obtained with castration achieved with LHRH agonists, estrogens, or orchiectomy.<sup>5-8</sup>

Another interesting finding is that the decrease in prostate size, although not reaching statistical significance, continued after 6 months of treatment in both groups. The inhibition went from 30.9% to 47.9% at an average 38.7 months of treatment in the monotherapy arm while the decrease in prostate size went from 47.4% to 56.5% at an average 29 months of treatment in the combination therapy arm. A third important observation is that monotherapy never reached the degree of inhibition already achieved at 6 months by combination therapy, even at slightly more than 3 years of treatment. In fact, after slightly more than 3 years of treatment (38.7 months), the percent decrease in prostate size was 47.9% with monotherapy while combination therapy could achieve the same result after only 6 months of treatment.

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## Enuresis

TO THE EDITOR:

I congratulate Dr. Husmann on his fine review of the subject of enuresis.<sup>1</sup> There are, however, three things that require comment:

1. He concludes that ". . . nocturnal enuresis is not related to sleep pattern, depth of sleep, or sleep arousal patterns,"

2. He states that children with attention deficit disorder "become distracted with their diurnal activities and fail to remember to void,"

3. The review does not address the role of urethral obstruction in children who suffer with persistent daytime urinary symptoms with or without bedwetting.

In 1968, Arnold<sup>2</sup> compared parental reports of sleep patterns among 450 children with persistent daytime urinary symptoms, 300 of whom wet the bed and 150 of whom did not. Parents of the bed-wetters described 90% of them as deep sleepers, whereas parents of the non-wetters described 80% of them as "light sleepers"—most of the non-wetters awakened at night to urinate. In 1994, Wille,<sup>3</sup> testing the ability of parents to awaken enuretics in contrast to non-enuretics found: "It was almost impossible to wake the children [en-