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EDITORIAL

Prostate cancer: are we under the lee?

K. H. Kurth

Prostate cancer grows extremely slowly, and a high proportion of elderly men have a latent, small tumor which does not cause symptoms. The dilemma as to how active one should be in diagnosing prostate cancer has intensified as a result of the increasing use of prostate-specific antigen (PSA) for early diagnosis. Physicians in Denmark have questioned the benefit of diagnosing tumors that do not cause local or general symptoms. As a consequence, the incidence of prostate cancer in the Scandinavian countries varies from 48.9 per 100 000 person-years (Denmark) to 61.8 (Finland), 71.8 (Norway), and 81.6 (Sweden) for the years 1983–1987. Since PSA screening became generally available, the incidence increased in the years 1990–1992 by between 12% in Sweden and 21% in Finland, but not in Denmark. Interestingly, the mortality from prostate cancer is similar in all the Scandinavian countries [1]. Age-adjusted incidence rates increased even more dramatically in the US by between 61% among whites and 65% among African Americans from 1989 through 1992 [2]. It is hypothesized that prostate cancer has a long natural history prior to clinical detection and that PSA screening results in an estimated lead time of at least 5 years [3]. Obviously, the question whether screening for prostate cancer lowers mortality can only be answered in a large-scale randomized study, as discussed by Kirkel et al. in this issue of *Urological Research*. Although as many as half of the radical prostatectomies done in the US are either unnecessary because the cancer is too small or ineffective because the cancer has already spread beyond the prostate [4], the highest cure rates are achieved with surgery of early stage prostate cancer. Whether more can be gained with neoadjuvant hormonal therapy than just down-sizing is a matter of controversy (U. Tunn, this issue); and so long-term follow-up of prospective randomized studies is needed to appraise the value of neoadjuvant hormonal therapy. In principle, the approach to treatment of metastatic prostatic carcinoma has not changed in the last 50 years; however, since our insight into the biology of this tumor has increased with time, tailored therapy considering individual requests (temporary preservation of potency, suppressing hot flushes) can be offered. Medical cas-

tration, although more costly than surgical castration but equally as effective, may also be applied intermittently and might in this way prolong the escape of the tumor to an androgen-independent state (T. De Reijke, this issue). It was not until 1993 that withdrawal responses following the selective discontinuation of flutamide in patients with progressive disease were first reported (M. Wirth, this issue). More recently, it has been recognized that withdrawal responses represent a more generalized phenomenon, since objective benefit has been observed following the discontinuation of a number of agents that act via steroid hormone receptors. We learned through additional hormonal maneuvers that a tumor can become androgen independent but remain hormone sensitive or when sequential hormonal manipulation is ineffective that it can become androgen independent and hormone insensitive [5]. Second-line treatment of hormone-refractory prostate cancer does not prolong survival (D. Newling, this issue), but it can improve quality of life [6]. Quality of life domains in patients treated for prostate cancer have only rarely been scrutinized quantitatively (G. Van Andel, this issue), but reasonably predictive estimates of quality of life after various treatment options are valuable in helping the patient to make difficult choices. New options for treating metastatic prostatic cancer probably will come from the experimental laboratory. On the basis of promising results in animal studies, a team of investigators at Baylor College of Medicine in Houston, Tex., USA, is investigating gene therapy for prostate cancer. The team plans a clinical trial of a “suicide gene” (known as the herpes simplex virus-thymidine kinase gene) for men with prostate cancer. Whether new findings in prostate cancer biology (D. Schamhart and A. Kooistra, this issue) will lead to a better tailored treatment of prostate cancer or just provide us with a better understanding of all the factors playing a role in the development of the disease has to be awaited. I hope both.

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