

Michele Pavone-Macaluso

Institute of Urology, University of Palermo,
Italy

Summary

It is my pleasurable task to summarize the presentations that were given as part of this stimulating symposium.

The first lecture, given by Prof. Altwein, looked at ways of improving the efficacy of radical prostatectomy in locally advanced prostate cancer. There is now a trend, very obvious in Germany and other European countries, to be more aggressive and try to operate on some early-stage T3 tumors. This is in contrast to the prevailing attitude, particularly seen in the United States, that radical prostatectomy should be limited to the very early stages of prostate cancer. Dr. Altwein noted that technical improvements, such as providing a wider resection margin with a very careful apical dissection and the use of the magnifying lens, might improve our surgical performance. However, once the pathological report shows the presence of positive margins what do we do? Shall we immediately treat these patients with adjuvant radiotherapy, use adjuvant hormonal treatment or wait until there is progression and then start treatment? In my view this is not a question that has been solved and there is a place for controlled randomized studies to investigate this further. Some clinicians prefer to wait until there is an increase in PSA. If such an increase is relatively rapid, perhaps within 3 months from surgery, then disseminated disease may be more likely and systemic therapy should be initiated. If a rise in PSA occurs later, maybe a year after surgery, this is more likely to denote a local recurrence. In this situation radiotherapy may be the more appropriate option. We all await results from ongoing randomized studies to help clarify this.

The second presentation was given by Prof. Jocham who gave a very convincing demonstration showing that the 3-month depot form of leuporelin is equivalent to the monthly injection in terms of results and in terms of lowering of plasma testosterone. This is a significant advance, reducing costs and enabling patients to have the often preferred option of visiting their doctor once every 3 months rather than monthly.

Another controversial point is the value, or perhaps the need, for neoadjuvant hormonal treatment before radical prostatectomy. I think there is no doubt that most hormonally treated patients will respond with a lowering of serum PSA. Also there will be a significant difference in positive-margin rates between hormonally pretreated patients versus patients who are treated immediately by radical pros-

tatectomy. The problem remains that we do not know what this signifies. Is this evidence of real downstaging? Is this modality going to have an impact on patient survival? Again, I think we do not yet know. Some people believe strongly in this approach, others deny it completely. With proponents on both sides of this debate fierce in their belief, I think it is important that we step back and try to assess the research as objectively as possible. Again, we must wait a few more years before we have some meaningful results on survival.

Prof. Costa provided us with some very elegant information about what is going on in France regarding incontinence following radical prostatectomy. While the incidence of incontinence in this series was 10%, it is likely to be much higher in centers where the surgeons are less experienced, and as we extend the indications to the T3 patients, it is likely that the number of incontinent patients will increase further. This is a disabling complication for the patient, and certainly affects their quality of life in a negative way. Therefore, the excellent results achieved with the artificial sphincter appear very promising for improving the quality of life of patients inflicted with this complication.

The problem of familial and hereditary prostate cancer has been undervalued for many years. Over the last 5–8 years it has reentered the spotlight, and the practical presentation by Dr. Mangin was very useful in that it offered suggestions for differentiating between sporadic cases, familial and hereditary ones. It will of course be even more useful if we could detect a specific chromosomal abnormality so that we can distinguish hereditary from familial cases, particularly if it is true that such genetic abnormalities may account for as much as 20–25% of cases. Incidental carcinoma rates diagnosed at autopsy are very similar around the world, while there are tremendous differences in clinical cancer rates. It would therefore be interesting to conduct an epidemiological survey to find out whether the prevalence of familial and hereditary cases differs between countries.

I would like to thank all of the speakers for their excellent and stimulating presentations. The issues presented cover a wide range of controversial and emerging areas of interest. As such, they provided the participants with a wonderful chance to hear some of the most expert researchers present their latest findings.