Conclusion: Early post-prostatectomy salvage radiation before the PSA reaches 0.2 ng/mL results in superior bPFS compared to those treated later. This strongly suggests that a new definition of post-prostatectomy progression is needed.

Table 1: Multivariate Cox regression analysis for predictors of PFS

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Hazard Ratio</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>T stage</td>
<td>1.580</td>
<td>1.176-2.123</td>
<td>0.002</td>
</tr>
<tr>
<td>Positive Margins</td>
<td>0.592</td>
<td>0.356-0.985</td>
<td>0.043</td>
</tr>
<tr>
<td>Gleason Score</td>
<td>1.368</td>
<td>1.054-1.774</td>
<td>0.018</td>
</tr>
<tr>
<td>Neoadjuvant ADT</td>
<td>0.780</td>
<td>0.398-1.527</td>
<td>0.469</td>
</tr>
<tr>
<td>Whole Pelvis</td>
<td>0.662</td>
<td>0.367-1.955</td>
<td>0.171</td>
</tr>
<tr>
<td>Age</td>
<td>0.958</td>
<td>0.957-1.023</td>
<td>0.527</td>
</tr>
<tr>
<td>PSA &gt; 0.2 ng/mL</td>
<td>2.358</td>
<td>1.350-4.119</td>
<td>0.003</td>
</tr>
</tbody>
</table>

1 HR for T stage represents each increase in stage with T2a as the reference
2 HR for GS represents each increase in GS with 5 as the reference up to 10

Patient centric approach: myth or fact?

P Poortmans1
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I am honoured to have been invited to give this memorial lecture for which there are three main criteria: it is firstly to honour Emmanuel van der Schuren, one of the fathers of our society. Secondly it aims to recognise scientific work within the field of radiation oncology and thirdly a contribution to education through the ESTRO programmes, in which I have been privileged to participate for the last 30 years or so.

The first ESTRO annual conference was held in London in 1982 and was memorable with the preparations being agreed between Emmanuel and Mike Peckham, my boss at the Royal Marsden Hospital at the time. I also want to acknowledge how dependent we were on many others for support, particularly among others for Lea, of whom we are thinking with gratitude especially at this time.

Scientific breakthroughs usually build on work that others have done and there are many examples from within the field of radiation oncology which I have experienced particularly in my area of research into whole-body irradiation. We work with the unchanging laws of physics but technology advances all the time and new biological understanding and new agents impact on the way in which we practice oncology.

I will discuss some of the ways in which progress in radiotherapy may occur and consider the factors which determine the impact of clinical trials, with particular reference to the START trials run by John Yarnold and his team. Consensus guidance, such as that contained in the ICRU report 50, has changed practice but there is still much evaluation work to be done in some areas. In our activity currently, process sometimes seems to take precedence over everything else, without the evaluation which would validate it.

ESTRO’s contribution to education has been enormous and it has been exciting to be involved in the teaching courses and publications of ESTRO with its ever-changing and innovative approaches. It is good to note that a new era is starting for the School. Amongst all the changes in current practice the needs of individual patients must remain our priority.

Do we need randomised clinical data to justify the use of SABR for primary and oligometastatic cancer?

To be confirmed

Pre-clinical and clinical data on the radiobiological mechanism for the efficacy of SABR

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Because the results obtained with stereotactic radiosurgery (SRS) and stereotactic ablative radiotherapy (SABR) have been impressive they have raised the question of whether classic radiobiological modeling are appropriate for large doses per fraction. In addition to objections to the LQ model, the possibility of additional biological effects resulting from endothelial cell damage and/or enhanced tumor immunity, have been raised to account for the success of SRS and SABR. However, the preclinical data demonstrate the following:

1) Quantitative in vivo endpoints, including late responding damage to the rat spinal cord, acute damage to mouse skin and early and late damage to the murine small intestine, are consistent with the LQ model over a wide range of doses per fraction, including the data for single fractions of up to 20 Gy.

2) Data on the response of tumors to high single doses are consistent with cell killing at low doses. Thus the dose to control 50% of mouse tumors (the TCD50) can be predicted from cell survival curves at low doses and the number of clonogenic cells in the tumors.

3) The high local control of NSCLC and of brain metastases by SABR and SRS is the result of high radiation doses leading the high BED. In other words the high curability is predicted by current radiobiological modeling.

4) Because high doses are required in SABR it is not possible to use it in all circumstances (e.g. for tumors close to critical normal structures). But because these high doses are needed.