	RT end	1 month	4 months	8 months	14 months	20 months	26 months	32 months	38 months
N of observed patients	350	214	255	212	146	91	53	22	7
No ADT [%]	42.6	64.8	72.7	78.1	85.7	84.4	96.2	100	100
GI 0 [%]	90.3	91.0	93.9	93.3	97.8	96.1	100	100	100
GI 1 [%]	9.1	6.6	4.9	6.2	2.2	3.9	-	-	-
GI 2 [%]	0.6	1.9	0.8	0.5	-	-	-	-	-
GI 3 [%]	-	0.5	0.4	-	-	-	-	-	-
GU 0 [%]	77.1	70.8	89.4	95.9	87.3	97.4	98.1	95.2	100
GU 1 [%]	16.3	25.0	8.2	3.6	9.7	2.6	1.9	4.8	-
GU 2 [%]	6.0	3.8	2.4	0.5	3.0	-	-	-	-
GU 3 [%]	0.6	0.4	-	-	-	-	-	-	-
PSA range [ng/ml]	0.008-20.4	0.003-16.3	0.002-8.2	0.0-6.4	0.002-3.5	0.04-2.2	0.0-3.3	0.02-3.8	0.003-0.6
PSA mean	3.7	1.9	1.1	0.7	0.5	0.4	0.4	0.5	0.3
PSA median	2.2	1.0	0.3	0.3	0.2	0.2	0.2	0.1	0.2

Conclusion: The results obtained permit us to form the conclusion that CK based radioablation of low and intermediate risk PC patients is an effective treatment modality enabling OTT shrinkage and giving a very low percentage of adverse effects.

PV-0090

Stereotactic body radiotherapy for localized prostate cancer: a 7-year experience

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Purpose or Objective: Recent understanding of radiobiology for prostate cancer suggested hypofractionation might achieve a higher therapeutic benefit. Stereotactic body radiation therapy (SBRT) is able to delivery high dose per fraction precisely. SBRT for prostate cancer might escalate biological effective doses while without increasing toxicity. Here, we reported our 7-year experience of SBRT for localized prostate cancer.

Material and Methods: Between November 2008 and Sep 2013, a total of 135 patients with clinically localized prostate were enrolled for analysis. Patients were low-risk (19%), intermediate-risk (37%), and high-risk (44%). Low- and intermediate-risk patients were treated with SBRT alone (37.5Gy in 5 fractions). High-risk patients were treated with whole pelvic irradiation (45Gy in 25 fractions) and SBRT boost (21Gy in 3 fractions). All of intermediate- and high-risk patients received hormone therapy with different duration. The toxicities of gastrointestinal (GI) and genitourinary (GU) tracts were scored by Common Toxicity Criteria Adverse Effect (CTCAE v3.0). Biochemical failure was defined as Phoenix definition.

Results: With a median follow-up of 52 months, there were seven patients with biochemical failure (one low-risk patient; one intermediate patient; five high-risk patients). The estimated 50-month biochemical failure-free survival (BFFS) was 95.8%, 96.4% and 81.5% for low-, intermediate, and high-risk patients, respectively. In the high-risk group, there were two late biochemical failures around 60 months. In the SBRT alone group, acute Grade 3 GU and GI toxicities were seen in 2.8% and 1.4% of the low/intermediate-risk patients, respectively; the incidence rate of late Grade 3 GU and GI toxicity were 3.5% and 0%. In the whole pelvic irradiation with SBRT boost group, acute Grade 2 GU and GI toxicity occurred in 31% and 21% of the high-risk patients, respectively; there was no grade 3 or higher late toxicity of GU and only one patient experienced grade 3 GI tract. Most of acute toxicity effects in the both groups resolved within three to six months of treatment completion.

Conclusion: SBRT with or without whole pelvic irradiation for localized prostate cancer is feasible with minimal toxicity and encouraging biochemical failure-free survival but should be aware of late failure in the high-risk group. Use of whole pelvic irradiation for high-risk patients was not associated with higher GU or GI toxicity. Continued accrual and follow-up would be necessary to confirm the biochemical control rate and the toxicity profiles.

PV-0091

Early salvage RT for PSA recurrence postprostatectomy improves biochemical progression free survival

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Purpose or Objective: The definition of biochemical recurrence following radical prostatectomy for prostate cancer remains controversial in the era of ultrasensitive PSA. The AUA definition of PSA > 0.2 ng/mL may not be valid when PSA can be detected as low as 0.01 ng/mL. Randomized trials have shown a benefit in terms of biochemical progression-free survival (bPFS) and metastasis free survival with adjuvant radiation compared to salvage but many patients enrolled as adjuvant actually had detectable PSA values. We compared patient outcomes with salvage radiotherapy based on pretreatment PSA in order to identify whether early salvage radiotherapy is more effective than treating later.

Material and Methods: We performed an institutional review board-approved retrospective analysis of patients treated at our institution with post-prostatectomy image guided radiotherapy from 2005 to 2013. Patients with positive lymph nodes, those with an undetectable PSA and those with metastatic disease were excluded from our analysis. Data were abstracted from each patient's electronic medical record including age, pathologic stage, Gleason score, margin status, androgen deprivation therapy, treatment to the pelvis, dose and PSA values. Patients were either treated with intensity modulated radiotherapy (IMRT) or volumetric arc therapy (VMAT) using daily image guidance. The use of ADT and the treatment of nodes was at the discretion of the treating physician. Radiation dose ranged from 6200-7400 cGy. Post-salvage bRFS was defined as PSA < 0.4 ng/mL. Kaplan-Meier survival analysis was used to compare patients with a pre-RT PSA value ≤ 0.2 ng/mL to those with a value > 0.2 ng/mL. Multivariate Cox regression analysis was used to evaluate significance of covariates on bPFS.

Results: 196 patients staged N0 or Nx were treated with salvage RT after prostatectomy during the study period. Median pre-treatment PSA was 0.29 ng/mL; 117 patients had a PSA > 0.2 ng/mL and 79 ≤ 0.2 ng/mL. Median follow up time was 36 months, determined by the reverse Kaplan-Meier method. Overall comparison of the two groups showed that patients treated with a PSA < 0.2 ng/mL had significantly improved bPFS (p=0.003) and increased 36 month bPFS (76% vs 56%, p=0.0074) compared to those treated with higher PSA values (Figure 1). In multivariate analysis a pre-RT PSA > 0.2 and increasing T stage and Gleason score were all significantly associated with worsening bPFS while positive margins were significant for improved bPFS (Table 1). Other covariates including treatment of nodes and use of ADT did not significantly influence bPFS following salvage.

Figure 1: Kaplan-Meier plot of biochemical progression free survival in post-prostatectomy patients stratified by pre-treatment PSA

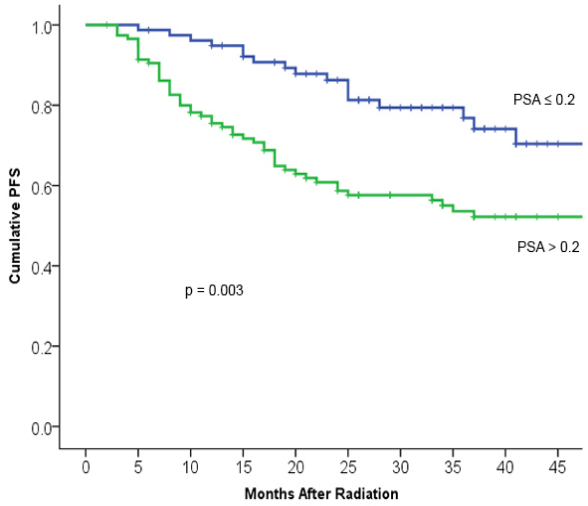

Table 1: Multivariate Cox regression analysis for predictors of PFS

Covariate	Hazard Ratio	95% CI	p-value
T stage†	1.580	1.176-2.123	0.002
Positive Margins	0.592	0.356-0.985	0.043
Gleason Score°	1.368	1.054-1.774	0.018
Neoadjuvant ADT	0.780	0.399-1.527	0.469
Whole Pelvis	0.662	0.367-1.195	0.171
Age	0.989	0.957-1.023	0.527
PSA > 0.2 ng/mL	2.358	1.350-4.119	0.003

† HR for T stage represents each increase in stage with T2a/b as the reference

° HR for GS represents each increase in GS with 6 as the reference up to 10

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.

Presidential Symposium:

SP-0092
Patient centric approach: myth or fact?
P Poortmans[1]
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Award Lecture: E. Van der Schueren Award

SP-0093
Did I do it right? What was the result? Process and outcomes in radiotherapy
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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 Scheuren, 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

Symposium with Proffered Papers: Hot topics in SABR: time for randomised clinical trials?

SP-0094
Do we need randomised clinical data to justify the use of SABR for primary and oligometastatic cancer?

To be confirmed

SP-0095
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.
Further the clinical data show:
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