EUROPEAN UROLOGY xxx (xxxx) xxx–xxx

available at www.sciencedirect.com
journal homepage: www.europeanurology.com

European Association of Urology

Guidelines

EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent

Philip Cornford[a,*], *Roderick C.N. van den Bergh*[b], *Erik Briers*[c], *Peter Chiu*[d], *Daniel Eberli*[e], *Adina Epure*[f,ee], *Andrea Farolfi*[g], *Valérie Fonteyne*[h], *Giorgio Gandaglia*[i], *Silke Gillessen*[j], *Nikolaos Grivas*[k], *Ann M. Henry*[l], *Geert J.L.H. van Leenders*[m], *Estefania Linares Espinos*[n], *Bo Matsen*[c], *Jan Oldenburg*[o,p], *Inge M. van Oort*[q], *Daniela E. Oprea-Lager*[r], *Matthew J. Roberts*[s,t], *Olivier Rouvière*[f], *Ashwin Sachdeva*[u], *Maria De Santis*[v], *Ivo G. Schoots*[w,x], *Emma J. Smith*[y], *Johan Stranne*[z,ff], *Thomas Wiegel*[aa], *Derya Tilki*[bb,cc,dd]

[a] *Department of Urology, Bon Secours Hospital, and University College Cork, Cork, Ireland;* [b] *Department of Urology, Erasmus University Medical Center, Rotterdam, The Netherlands;* [c] *Europa Uomo, Antwerp, Belgium;* [d] *S.H. Ho Urology Centre, Department of Surgery, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China;* [e] *Department of Urology, University Hospital Zurich, Zurich, Switzerland;* [f] *Department of Imaging, Hôpital Edouard Herriot, Hospices Civils de Lyon, Lyon, France;* [g] *Nuclear Medicine, IRCCS Azienda Ospedaliero-Universitaria di Bologna, Bologna, Italy;* [h] *Department of Radiation Oncology, Ghent University, Ghent, Belgium;* [i] *Division of Oncology/Unit of Urology, Soldera Prostate Cancer Laboratory, Urological Research Institute, IRCCS San Raffaele Scientific Institute, Milan, Italy; Vita-Salute San Raffaele University, Milan, Italy;* [j] *Oncology Institute of Southern Switzerland (IOSI), EOC, Bellinzona and Faculty of Biomedical Sciences, USI, Lugano, Switzerland;* [k] *Department of Urology, Lefkos Stavros-The Athens Clinic,Athens, Greece;* [l] *Leeds Cancer Centre, St. James's University Hospital and University of Leeds, Leeds, UK;* [m] *Department of Pathology, Erasmus University Medical Center, Rotterdam, The Netherlands;* [n] *Department of Urology, University Hospital La Paz, Madrid, Spain;* [o] *Department of Oncology, Akershus University Hospital (Ahus), Lørenskog, Norway;* [p] *Faculty of Medicine, University of Oslo, Oslo, Norway;* [q] *Department of Urology, University of Groningen, University Medical Center Groningen, Groningen, The Netherlands;* [r] *Department of Medical Imaging, Radboud University Medical Center, Nijmegen, The Netherlands;* [s] *Department of Urology, Royal Brisbane and Women's Hospital, Brisbane, Australia;* [t] *Faculty of Medicine, The University of Queensland Centre for Clinical Research, Herston, QLD, Australia;* [u] *Division of Cancer Sciences, University of Manchester, Manchester, UK;* [v] *Department of Urology, Universitätsmedizin Berlin, Berlin, Germany and Department of Urology, Medical University of Vienna, Vienna, Austria;* [w] *Department of Radiology and Nuclear Medicine, Erasmus University Medical Center, Rotterdam, The Netherlands;* [x] *Department of Radiology, Netherlands Cancer Institute, Amsterdam, The Netherlands;* [y] *European Association of Urology, Arnhem, The Netherlands;* [z] *Department of Urology, Institute of Clinical Sciences, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden;* [aa] *Department of Radiation Oncology, University Hospital Ulm, Ulm, Germany;* [bb] *Martini-Klinik Prostate Cancer Center, University Hospital Hamburg-Eppendorf, Hamburg, Germany;* [cc] *Department of Urology, University Hospital Hamburg-Eppendorf, Hamburg, Germany;* [dd] *Department of Urology, Koc University Hospital, Istanbul, Türkiye;* [ee] *Université de Lyon, Université Lyon 1, UFR Lyon-Est, Lyon France;* [ff] *Department of Urology, Sahlgrenska University Hospital-Västra Götaland, Gothenburg, Sweden*

Article info

Article history:
Accepted April 7, 2026

Abstract

Background and objective: To present a summary of the 2026 version of the European Association of Urology (EAU)–European Association of Nuclear Medicine (EANM)–European Society for Radiotherapy and Oncology (ESTRO)–European Society of

* Corresponding author. Prof Philip Cornford at the Department of Urology, Bon Secours Hospital, College Road, Cork Ireland.
E-mail address: prof.cornford@gmail.com (P. Cornford).

https://doi.org/10.1016/j.eururo.2026.04.013

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

Editor-in-Chief: Alberto Briganti

Keywords:
EAU guidelines
Prostate cancer
Risk stratification
Imaging
Prostate biopsy
Active surveillance
Radiotherapy
Hormonal therapy

Urogenital Radiology (ESUR)–International Society of Urological Pathology (ISUP)–International Society of Geriatric Oncology (SIOG) guidelines on screening, diagnosis, and treatment of clinically localised prostate cancer (PCa).

Methods: The Panel performed a literature review of all new data published in English, covering the time frame between May 2023 and 2025. The Guidelines were updated, and a strength rating for each recommendation was added based on a systematic review of the evidence.

Key findings: A risk-adapted strategy for identifying men who may develop PCa is advised, generally commencing at 50 yr of age and based on individualised life expectancy. The use of multiparametric magnetic resonance imaging to avoid unnecessary biopsies is recommended. When a biopsy is considered, a combination of targeted and regional biopsies should be performed. A new five-tier EAU classification has been introduced. Prostate-specific membrane antigen positron emission tomography imaging is the most sensitive technique for identifying metastatic spread. Active surveillance is the appropriate management for men with low-risk PCa, as well as for patients with selected favourable intermediate-risk ISUP grade group 2 lesions. Local therapies are addressed, as well as the management of persistent prostate-specific antigen after surgery. A recommendation to consider hypofractionated radiotherapy in intermediate-risk patients is provided. Patients with cN1 PCa should be offered radiotherapy to the primary tumour combined with long-term intensified hormonal treatment.

Conclusions and clinical implications: The evidence in the field of diagnosis, staging, and treatment of localised PCa is evolving rapidly. These PCa Guidelines reflect the multidisciplinary nature of PCa management.

1. Introduction

The prior summary of the European Association of Urology (EAU)–European Association of Nuclear Medicine (EANM)–European Society for Radiotherapy & Oncology (ESTRO)–European Society of Urogenital Radiology (ESUR)–International Society of Urological Pathology (ISUP)–International Society of Geriatric Oncology (SIOG) Guidelines on Prostate Cancer (PCa) was based on the 2024 edition of the Guidelines [1]. The present summary is based on the latest Guidelines published in March 2026 [2].

It must be emphasised that clinical guidelines present the best evidence available to experts, but following guideline recommendations will not necessarily result in the best outcome. Guidelines can never replace clinical expertise when making treatment decisions for individual patients, but rather help to focus decisions that also take the personal values, preferences, and individual circumstances of patients into account. Guidelines are not mandates and do not purport to be a legal standard of care (SOC).

2. Methods

For the 2026 update of the EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on PCa, new evidence was identified, collated, and appraised through a structured assessment of the literature. Databases searched included Medline, EMBASE, and the Cochrane Library. Detailed search strategies are available on the EAU website: https://uroweb.org/guidelines/prostate-cancer/publications-appendices.

Recommendations within the Guidelines were developed by the Panels to prioritise clinically important care decisions. The strength of each recommendation is determined by the balance between desirable and undesirable consequences of alternative management strategies, the quality of the evidence (including certainty of estimates), and the nature and variability of patient values and preferences. Strong recommendations are made when evidence quality is high and/or there is a favourable balance of benefits to harms and patient preferences. Weak recommendations are made when the evidence is of lower quality and/or benefits and patient preferences are less clear [3].

3. The Guidelines

3.1. Epidemiology and risk factors

In Europe, PCa is the most frequently diagnosed cancer in men and the third most common cancer-related cause of death in men [4]. Globally, incidence is greatly affected by the rate of prostate-specific antigen (PSA) testing and national recommendations on screening [5]. Age, African origin, and a family history of PCa [6] are well-established risk factors. However, it appears that these are related to multiple single-nucleotide polymorphisms (SNPs) [7] rather than gene defects. Nevertheless, individuals with gene alterations in *BRCA2* [8], *MSH2* and *MSH6* (Lynch syndrome) [9] are at increased risk and, to a lesser degree, those with *BRCA1* [10]. Only a small subpopulation of men have true hereditary disease ($\geq$ three cases in the same family, PCa in three successive generations, or $\geq$ two men diagnosed with PCa <55 yr), which is associated with an onset 6–7 yr earlier than nonhereditary cases but does not differ in other ways [6].

A wide variety of exogenous or environmental factors have been discussed as being associated with the risk of developing PCa or as being aetiologically important for the

progression from latent to clinical PCa. However, there are no known effective preventative dietary or pharmacological interventions. Men with hypogonadism receiving testosterone supplements do not have an increased risk of developing PCa [11]. Furthermore, although the evidence is limited, men with hypogonadism who undergo testosterone supplementation and are managed expectantly for PCa, or who have received radical curative therapy, do not have worse outcomes despite concerns regarding testosterone use and a higher risk of progression [12].

3.2. Classification and staging

The 2025 Tumour, Node, Metastasis (TNM) classification of the Union for International Cancer Control (UICC) 9th edition for staging of PCa should be used [13], while the ISUP 2005 Gleason score (GS), together with its 2014 and 2019 modifications, is the recommended PCa grading system [14,15]. The GS comprises the Gleason grade of the most extensive (primary) pattern, plus the second most common (secondary) pattern if two are present, and the most extensive and highest pattern if three are present at biopsy. In GS 7 biopsies, the Gleason pattern 4% should be reported, whereas the presence or absence of invasive cribriform and/or intraductal carcinoma (IDC) should be reported in all biopsies [15]. Results should be expressed using ISUP grade groups (GG) [15].

The EAU classification of risk groups has moved to a five-tier model based on separating intermediate-risk patients into favourable and unfavourable groups using factors previously shown to differentiate risk of PCa-related death despite local treatment [16]. The T stage was historically based on digital rectal examination (DRE) only; however, changes in the diagnostic pathway, particularly the introduction of pre-biopsy magnetic resonance imaging (MRI), mean that additional information is readily available, and MRI-based T stage (MRI-T) should be reported separately. Prostate-specific membrane antigen (PSMA) positron emission tomography (PET) imaging, where available, is the recommended staging test. The use of modern imaging is resulting in a shift in the risk-group distribution, and this should be considered when making treatment decisions. The extent of this prognosis shift remains to be assessed, as well as its impact.

3.3. Clinically significant prostate cancer

The descriptor ‘clinically significant’ is widely used, but definitions are variable and not supported by long-term follow-up or correlation with hard oncological outcomes. It is clear that many more men harbour a small focus of PCa than will ever develop metastatic disease or succumb to the disease [4,17], and overtreatment of insignificant PCa risks harmful side effects for patients. Epidemiological and autopsy data suggest that many ISUP GG 1, but also ISUP GG 2, PCa would remain undetectable during a man’s life [18] and therefore are clinically insignificant in most men. Combining MRI-targeted biopsy with systematic biopsy has improved diagnostic accuracy as compared to systematic biopsy alone [19], but has also led to stage inflation and grade inflation in targeted biopsies, further blurring the definition of clinically significant PCa (csPCa). It is imperative that clinicians explain to patients why immediate treatment is warranted.

3.4. Screening and early detection

The diagnostic pathway for PCa (Fig. 1) aims for timely detection of significant PCa, while leaving insignificant PCa undetected, balancing diagnostic accuracy with the burden on the individual and the health care provider. Patient-specific factors, such as ethnicity, family history, age, and comorbidity, should always be considered. Localised PCa is usually asymptomatic, although local progression may cause symptoms such as lower urinary tract symptoms (LUTS), erectile dysfunction (ED), retention, pain, or haematuria. Prior to PSA testing, most men diagnosed with PCa presented with pain from bone metastases or even spinal cord compression.

Population screening is defined as the ‘systematic examination of asymptomatic men to identify individuals at risk for a specific disease’. The coprimary objectives are a reduction in disease-specific mortality and a maintained quality of life (QoL). Screening is associated with an increased diagnosis of PCa (incidence ratio [IR]: 1.23; 95% confidence interval [CI]: 1.03–1.48), with detection of more localised disease (risk ratio (RR): 1.39; 95% CI: 1.09–1.79) and less advanced PCa (T3–4, N1, M1; RR: 0.85; 95% CI: 0.72–0.99) [20,21]. In the largest study, the population-based European Randomised Study of Screening for Prostate Cancer (ERSPC), which included >182 000 men, they also showed a significant reduction in PCa mortality in the screening arm (RR: 0.79; 95% CI: 0.69–0.91) [22]. With extended follow-up, the mortality reduction (21% and 29% after noncompliance adjustment) remains unchanged. However, the number needed to screen (NNS), to diagnose (NND), and to treat (NNT), 456 and 12, respectively, continue to fall [23]. In the Rotterdam section of the ERSPC with 21 yr of follow-up, the RR of death due to PCa was 0.73 in the screening group, with NNS and NND of 246 and 14, respectively, to prevent one death [24]. These numbers are 121 and seven to prevent one man from metastatic disease.

Individual requests for PSA testing may be considered following discussion of the rationale and risk of both the associated lead time and the possibility of identifying insignificant cancer. An abnormal DRE is an indication for further evaluation, but as an independent variable, PSA is a better predictor of cancer than DRE. Prostate-specific antigen is a continuous parameter, with higher levels indicating a greater likelihood of PCa, precluding an optimal PSA threshold for detecting nonpalpable but csPCa. A limited PSA elevation alone (3–10 ng/ml) should be confirmed after a few weeks under standardised conditions (ie, no ejaculation, manipulations, or urinary tract infections [UTIs]) in the same laboratory before considering further testing [25].

PSA density (PSA-D) (serum PSA divided by prostate volume) may also help predict the presence of csPCa, especially in smaller prostates, using a cut-off of 0.15 ng/ml/cc [26]. Patients with a PSA-D below 0.09 ng/ml/cc were found to be unlikely (4%) to be diagnosed with csPCa [27], and this may allow men to avoid the need for biopsy.

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

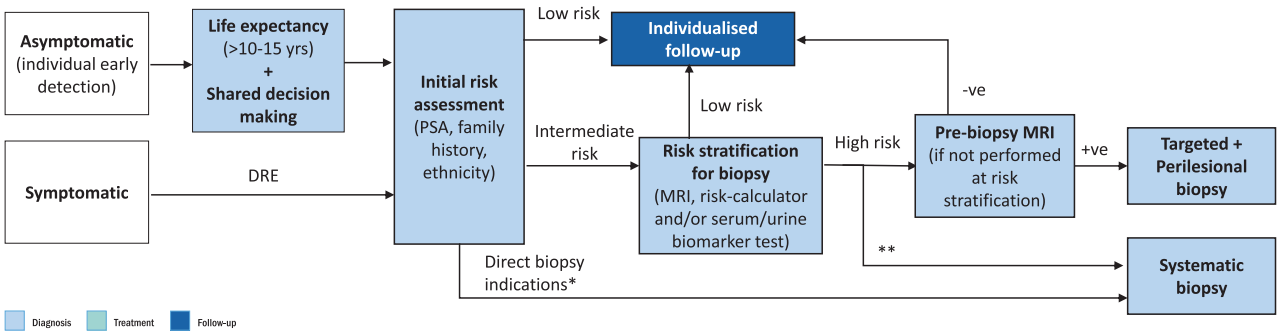

Fig. 1 – Early detection/suspicion prostate cancer biopsy indications. * PSA >50, cT3-4. ** If MRI not available / possible.

All of the screening studies presented were conducted in the pre-MRI era, with a PSA-based threshold for biopsy, and only systematic biopsies. In Fig. 1, MRI is initiated upon suspicion of PCa based on PSA and/or DRE. A systematic review concluded that integrating MRI in PCa screening pathways is associated with a reduction in the number of unnecessary biopsies and overdiagnosis of insignificant PCa, while maintaining csPCa detection as compared with PSA-only screening [28], in part because tumours visible on MRI are enriched in molecular hallmarks of aggressivity as compared to invisible lesions [29]. In younger (45–50 yr) men, MRI may be less effective [30]. Data suggest that using biparametic MRI may be noninferior to multiparametric MRI (mpMRI) if careful attention is paid to ensuring high-quality images with expert interpretation [31].

In addition to suggesting the presence of PCa, imaging also enables guidance in targeted prostate biopsy and provides staging information. In a Cochrane meta-analysis which compared mpMRI to template biopsies (≥20 cores) in biopsy-naïve and repeat-biopsy settings, MRI had a pooled sensitivity and specificity of 0.91 (95% CI: 0.83–0.95) and 0.37 (95% CI: 0.29–0.46) for ISUP GG ≥2 cancers, and 0.95 (95% CI: 0.87–0.99) and 0.35 (95% CI: 0.26–0.46] for ISUP GG ≥3 cancers, respectively [32].

In biopsy-naïve men, an MRI-based indication for biopsy after referral leads to lower rates of biopsy, lower rates of men diagnosed with PCa labelled as insignificant, and more men with PCa labelled as csPCa [33,34] as compared with systematic biopsy alone. This is also true in men with a prior negative biopsy [32,35]. Combining PSA-D and MRI may also be helpful to identify patients at risk among those with a negative (PI-RADS 1-2) or indeterminate (PI-RADS 3) MRI [36,37].

Optimal intervals for PSA testing are unknown. A risk-adapted strategy might be a consideration, based on the initial PSA level. Men with a baseline PSA <1ng/ml at 40 yr or < 2ng/ml at 60 yr are at decreased risk of PCa metastasis or death from PCa several decades later [38,39]. The retesting interval can therefore be every 2 yr for those initially at increased risk or postponed up to 8 yr for those at low risk [39,40].

The age at which to stop early diagnosis should be based on individual's life expectancy, where comorbidity is at least as important as age [41]. Men who have <15 yr of life expectancy are unlikely to benefit from any form of early diagnosis.

3.5. *Prostate biopsy*

Definitive diagnosis normally depends on histopathological verification in prostate biopsy cores. However, men with a high suspicion of malignancy (eg, malignant-feeling prostate, PSA >100 ng/ml) and a positive bone scan might avoid a biopsy, especially if pre-existing comorbidities would exclude second-line treatment.

Ultrasound (US)-guided prostate biopsy under local anaesthesia is the SOC [42]. Where MRI has shown a suspicious lesion, MR-targeted biopsy can be obtained through cognitive guidance, US/MR-fusion software, or direct in-bore guidance [43–45]. A minimum of three to five cores is required for proper sampling of an MRI-detected lesion [46]. In addition, several concordant studies showed that, in the case of a unilateral MRI lesion, contralateral systematic biopsy (ie, from the MRI-negative lobe) has little added value for diagnosing csPCa (0.3–4%). Paradoxically, the added value of ipsilateral systematic biopsy is higher (4.9–18.4%) and comes mostly from the systematic cores obtained in the sextant containing the MR lesion, or the sextant immediately adjacent [47,48]. Therefore, it may also be possible to avoid systematic biopsies entirely by including peri-lesional/regional biopsies. This will decrease the total number of cores taken (by avoiding systematic biopsies in MRI-negative lobes) and improve the detection of csPCa (by compensating for guiding imprecision). A meta-analysis of eight studies [49] showed a nonsignificant difference in the detection of ISUP GG ≥2 cancer in the MRI-directed targeted and regional biopsy approach, compared to the MRI-directed targeted plus systematic biopsy approach (RR: 0.95; 95% CI: 0.90–1.01; $p = 0.09$). However, the MRI-directed targeted and regional biopsy approach detected significantly more ISUP GG ≥2 cancers than MRI-targeted biopsy alone (RR: 1.18; 95% CI: 1.10–1.25; $p < 0.001$). In addition, the MRI-targeted and regional biopsy approach could avoid detecting 12–17% of the insignificant cancers (ISUP GG 1) detected by the classical combined approach [50,51]. A meta-analysis of 21 studies also supported the targeted and regional biopsy approach as a valuable strategy for the detection of ISUP GG ≥2 cancer in MRI-visible lesions while avoiding overdiagnosis of ISUP GG 1 cancer [52].

Prostate biopsy can be performed by either the transperineal approach or the transrectal approach. However, the only systematic review and meta-analysis comparing MRI-

targeted transrectal biopsy to MRI-targeted transperineal biopsy, analysing eight studies, showed a higher sensitivity for detection of csPCa when the transperineal approach was used (86% vs 73%; $p < 0.003$) [51]. This benefit was especially pronounced for anterior tumours. It is associated with increased discomfort for the patient [53], but evidence also suggests a reduced infection risk with the transperineal route [54,55], such that antibiotic prophylaxis might not be required. A systematic review and meta-analysis including 106 unique studies comparing transperineal biopsy with and without prophylactic antibiotics found no significant differences in UTI or sepsis rates [56]. Therefore, for patients without significant risk factors, such as diabetes or a history of urinary retention [57], antibiotic prophylaxis may be safely omitted for transperineal biopsy. For the transrectal route, the use of rectal povidone-iodine [58] and targeted prophylaxis based on rectal swab/stool culture [59] should be considered to minimise the risk of infection.

Each biopsy site should be reported individually, including its location, the GS, ISUP GG, and extent. If identified, lymphovascular invasion and extraprostatic extension (EPE) must each be reported, as well as IDC and invasive cribriform pattern, as these represent independent factors for metastasis [60] and cancer-specific survival (CSS) [61]. Clinicians and patients should note that MRI-targeted biopsy is more sensitive than systematic biopsy in detecting areas of high-grade cancer, and as a consequence, ISUP GG $\geq$2 cancers detected by MRI-targeted biopsy are, on average, of better prognosis than those detected by systematic biopsy alone [62]. When long-term follow-up of patients who underwent MRI-targeted biopsy is available, a revision of the risk-group definition will become necessary. In the meantime, results of MRI-targeted biopsy must be interpreted in the context of this potential grade shift.

3.6. Staging of prostate cancer

The decision to proceed with a further staging work-up is guided by which treatment options are available, considering the patient's preference and comorbidity.

3.6.1. T-category

The clinical (cT) category relies on DRE findings, but increasing use of MRI, and in particular T2-weighted imaging, is driving stage migration. In 552 men treated by RP at seven different Dutch centres, MRI showed significantly higher sensitivity (51% vs 12%; $p < 0.001$), and lower specificity (82% vs 97%; $p < 0.001$), than DRE for nonorgan-confined disease. All risk groups redefined using MRI findings rather than DRE findings showed better biochemical recurrence (BCR)-free survival due to improved discrimination and the associated stage shift [63]. In addition, MRI features such as the tumour PI-RADS score, diameter, apparent diffusion coefficient (ADC) value, or extracapsular extension (ECE)/seminal vesicle invasion (SVI) features, are strong predictors of BCR, alone or in combination with clinical data [64].

3.6.2. N category

Abdominal computed tomography (CT) and MRI indirectly assess nodal invasion by using lymph node (LN) diameter and morphology. Usually, LNs with a short axis of >8 mm in the pelvis and >10 mm outside the pelvis are suspicious for malignancy, with a sensitivity below 40% [65]. Since CT and MRI lack sensitivity for direct detection of positive LNs, nomograms combining clinical and biopsy findings have been used to estimate the risk of patients harbouring positive LNs [66]. PSMA PET/CT has a good specificity for LN involvement. In a meta-analysis including 37 articles, a subgroup analysis was performed in patients undergoing PSMA PET/CT for primary staging. On a per-patient-based analysis, the sensitivity and specificity of 68Ga-PSMA PET were 77% and 97%, respectively, after extended LN dissection (LND) at the time of RP. On a per-lesion-based analysis, sensitivity and specificity were 75% and 99%, respectively [67]. In summary, PSMA PET/CT is more sensitive in N-staging as compared to MRI, abdominal contrast-enhanced CT or, choline PET/CT. However, small LN metastases, under the spatial resolution of PET, may still be missed.

3.6.3. M category

Bone scintigraphy has been the most widely used method for evaluating bone metastases of PCa, with combined sensitivity and specificity of 79% (95% CI: 73–83%) and 82% (95% CI: 78–85%), respectively, at the patient level [68]. Diffusion-weighted whole-body and axial MRI are more sensitive than bone scan and targeted conventional radiography in detecting bone metastases in high-risk PCa [69]. Whole-body MRI is also more sensitive and specific than combined bone scan, targeted radiography, and abdominopelvic CT [70]. However, PSMA PET/CT appears to be the most accurate form of staging metastatic spread. In a prospective multicentre study in patients with high-risk PCa before curative surgery or RT (proPSMA), 302 patients were randomly assigned to conventional imaging or ^{68}Ga-PSMA-11 PET/CT [71]. The primary outcome focused on the accuracy of first-line imaging for the identification of pelvic LN or distant metastases. Accuracy of ^{68}Ga-PSMA PET/CT was 27% (95% CI: 23–31) higher than that of CT and bone scintigraphy (92% [95% CI: 88–95] vs 65% [95% CI: 60–69]; $p < 0.0001$). Conventional imaging had a lower sensitivity (38% [95% CI: 24–52] vs 85% [95% CI: 74–96]) and specificity (91% [95% CI: 85–97] vs 98% [95% CI: 95–100]) than PSMA PET/CT. Furthermore, ^{68}Ga-PSMA PET/CT scan prompted management change more frequently as compared to conventional imaging (41 [28%] men [95% CI: 21–36] vs 23 [15%] men [95% CI: 10–22]; $p = 0.08$), with less equivocal findings (7% [95% CI: 4–13] vs 23% [95% CI: 17–31]) and lower radiation exposure (8.4 mSv vs 19.2 mSv; $p < 0.001$) [71]. Consequently, replacing bone scan and abdominopelvic CT with more sensitive imaging modalities may be a consideration in patients with high-risk PCa undergoing initial staging. Initial results of a follow-up study of the surgical cohort in the multicentre prospective phase 3 imaging trial demonstrate that presurgical PSMA-PET is a strong prognostic biomarker, improving BCR-free survival risk assessment [72].

3.7. Evaluating life expectancy and health status

Evaluation of life expectancy and health status is important in clinical decision-making regarding screening, diagnosis,

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

and treatment of PCa. Country-specific life tables are available; however, survival must be individualised [73], based, for example, on gait speed [74] or using tools such as the Cumulative Illness Score Rating-Geriatrics (CISR-G) [75], the Charlson Comorbidity Index (CCI) [76], or the clinical frailty score [77]. To assess older adults' suitability for treatment, the Panel suggests a systematic evaluation of health status using the Geriatric 8 screening tool [41], as well as the Mini-Cog [78]. This may identify reversible health issues that, after treatment, would facilitate alternative treatment options.

In localised disease, >10 yr life expectancy is considered mandatory for any survival benefit from local treatment. Increasing comorbidity greatly increases the risk of dying from non-PCa-related causes. In an analysis of 19 639 patients aged >65 yr who were not given curative treatment, most men with a CCI score $\geq$2 had died from competing causes at 10-yr follow-up regardless of their age at the time of diagnosis. Tumour aggressiveness had little impact on overall survival (OS), suggesting that patients could have been spared biopsy and diagnosis of cancer. While patients with a CCI score $\leq$ 1 had a low risk of death at 10 yr, especially for well- or moderately differentiated lesions [79]. Patients with a life expectancy of <10 yr should undergo monitoring with initiation of androgen deprivation therapy to palliate symptoms (watchful waiting [WW]). Estimation of competing benefits of active versus conservative treatment and death from any cause at ten and 15 yr can be estimated using the PREDICT Prostate tool (available from https://prostate.predict.nhs.uk/).

3.8. Primary local treatment

Management decisions should be made after all options have been discussed with a multidisciplinary team (including urologists, radiation oncologists, medical oncologists, pathologists, and radiologists), and using a shared-care approach to balance the benefits and side effects of each therapeutic modality together with the patient's views and preferences.

3.8.1. Active surveillance

While all available radical treatment options are associated with significant unwanted side effects, mortality from untreated screen-detected PCa in patients with ISUP GG 1–3 has been recorded as just 7% after 15-yr follow-up [80]. Consequently, active surveillance (AS) strategies aim to reduce overtreatment in patients with localised ISUP GG 1 and possibly GG 2 PCa, without compromising opportunities for cure.

A systematic review summarised the available data on AS [81]. Protocols do not fully align regarding patient selection, follow-up policies, and criteria to switch to an active treatment, although there is significant overlap. The most often published criteria based upon standard systematic biopsies include ISUP GG 1, cT1c or cT2a, PSA <10 ng/ml (low-risk disease), and PSA-D <0.15 ng/ml/cc. [82]. The addition of MRI and additional targeted biopsies when indicated results in fewer failures of surveillance (19% vs 35%; p = 0.02) and in fewer patients progressing to ISUP GG $\geq$2 cancer (9.9% vs 23%; p = 0.048) at 2-yr follow-up [83]. Therefore, the men eligible for AS after combined systematic and MRI-targeted biopsy do not require a confirmatory biopsy [84]. However, it remains important to exclude sampling errors for patients selected based on systematic biopsy or MRI-targeted biopsy alone [32,33,85].

There is increasing recognition that AS can be extended to other groups. In the ProtecT randomised controlled trial (RCT), 1643 patients were randomised into one of three arms: active treatment with either RP or external beam RT (EBRT) or active monitoring (AM), with outcomes now reported at 15 yr [86]. Follow-up involved PSA monitoring and DRE alone, with relaxed criteria to define progression and no role for MRI or scheduled repeat biopsy. Categorisation at study entry was inaccurate, as demonstrated by the fact that in men undergoing RP within 12 mo of randomisation, histology showed that 50.5% were ISUP GG $\geq$2, while 28.5% had pT3 or pT4 disease. Despite this, AM was as effective as active treatment at 15 yr (CSS = 96.9% in the AM-group vs 97.8% in the RP-group and 97.1% in the EBRT-group; p = 0.53), but at a cost of increased metastatic progression risk (9.4% vs 4.7% and 5.0% respectively). It is, therefore, clear that men with favourable ISUP GG 2 cancer (PSA <10 ng/ml, < cT2b, and a small number of positive cores) could also be considered for deferred treatment [87]. Men harbouring intraductal or invasive cribriform adenocarcinoma [88], sarcomatoid carcinoma, small cell carcinoma, EPE, or lymphovascular invasion in needle biopsy [89] should not be considered.

The follow-up strategy is based on serial PSA (at least once every 6 mo), DRE, and MRI (at least annually), and includes standard repeat biopsy. However, several factors have been found to be associated with low reclassification rates and long progression-free survival (PFS): negative baseline or negative repeat MRI during AS [74,84], low PSA-D [84,90], low PSA velocity (PSAV) [91], or negative biopsy (ie, no cancer at all) at confirmatory or repeat biopsy during AS [92].

STRATCANS (STRATified CANcer Surveillance) stratifies patients into three groups based on the combination of Cambridge Prognostic Grouping (CPG; CPG 1 - ISUP GG 1 and PSA <10 and cT1-2, CPG 2 – ISUP GG 2 or PSA 10-20 and cT1-2), PSA-D (<0.15 vs $\geq$0.15), and MRI visibility (based on risk of progression) [93]. This may be used to individualise follow-up intensities (18–12–6 mo and MRI 36–18–12 mo with increasing risk, respectively, and no standard repeat biopsy in the lowest risk tier). This aligns with data showing that patients with stable disease (PRECISE 3) on repeat MRI during AS and a low PSA-D (<0.15) have a very low rate of progression, and repeat biopsy may therefore be omitted [94].

Men may remain on AS while they continue to consent, and have a life expectancy of >10 yr, and the disease remains indolent. Anxiety in patients about continuing AS is best managed with psychological support [95] before considering switching to an active treatment. A PSA change or MRI change should trigger further investigation, including re-biopsy, before considering active treatment [87,95]. The development of other comorbidities resulting in a life

expectancy falling below 10 yr should merit a new discussion with the patient and may result in a decision to transfer to a WW strategy.

3.8.2. Radical prostatectomy

The goal of RP is eradication of PCa while preserving continence and, whenever technically and oncologically feasible, potency. It is the only treatment for localised PCa to show a benefit for OS and CSS, compared with WW in an RCT [96]. Patients should not be denied this procedure on the grounds of age alone [41] provided that they have at least 10 yr of life expectancy and are aware that increasing age is linked to an increased incontinence risk. Data from SPCG-4 [96] and a pre-planned subgroup analysis (PIVOT) [97] highlight the benefit of RP compared with WW in men with intermediate-risk disease.

Provided that the tumour is not fixed and not invading the urethral sphincter, RP with or without extended pelvic LN dissection (ePLND) is a reasonable first step in patients with high-risk and locally-advanced PCa. This is particularly relevant as PSMA PET/CT imaging allows identification of many patients with metastatic disease [71]. Among the individual elements that make up high-risk disease, an ISUP GG 4/5 prostate-confined adenocarcinoma has a good prognosis after RP. In addition, frequent downgrading exists between the biopsy and the specimen GS [98]. At 10 yr of follow-up, the CSS is up to 88% [99]. A PSA value of >20 ng/ml is associated with CSS at 10 yr ranging between 83% and 91% [99]. Radical prostatectomy for cT3N0 PCa is associated with an increased risk of positive margins and pN+ and/or distant relapse. Retrospective case series demonstrated CSS at 10 yr between 85% and 92% [100]. The overall heterogeneity of this high-risk group was highlighted by a large retrospective multicentre cohort of 1360 high-risk patients treated with RP in a multimodal approach [100]. At 10 yr overall, 91.3% CSS was observed: 95% for those having only one risk factor (ie, ISUP GG >3, cT category higher than cT2, or PSA >20 ng/ml), 88% for those having a cT3–4 and PSA >20 ng/ml, and 79% if all three risk factors were present.

Incontinence is a concern for all patients undergoing surgery, and systematic reviews and meta-analyses found that every extra millimetre of membranous urethral length seen on MRI preoperatively improves early return to continence post-RP [101,102]. A greater membranous urethral length measured on preoperative MRI was an independent prognostic factor for return to urinary continence within 1 mo after RP and remained prognostic at 12 mo [102]. Therefore, it is likely that preservation of as much urethral length as possible during RP will maximise the chance of early return to continence. It may also be useful to measure urethral length preoperatively on MRI to facilitate counselling of patients on their relative likelihood of early postoperative continence [103].

Nerve-sparing (NS) RP can be performed safely in most men with localised PCa, and preserving parasympathetic nerve branches of the pelvic plexus might spare erectile function [104]. A systematic review of 19 studies analysing the parameters used for selection of NS found that individual clinical and radiological factors were poor at predicting EPE, and consequently, the appropriateness of NS. However, nomograms that incorporated mpMRI performed better [105]. Multiparametric MRI may be helpful for selecting a NS approach because it has good specificity (0.91; 95% CI: 0.88– 0.93) but low sensitivity (0.57; 95% CI: 0.49–0.64) for detecting pT3a stages [106].

Use of intraoperative surgical margin assessment, such as the frozen section examination (NeuroSAFE) technique, enables a systematic evaluation of surgical margins intraoperatively, allowing for adjustment if positive margins are detected, to reduce positive surgical margin rates (odds ratio (OR): 0.68; 95% CI: 0.51–0.91) [107]. The NeuroSAFE PROOF trial [108] assessed this technique in a randomised prospective manner and showed an improvement in continence at 3 mo but not at 6 mo, along with an improvement in ED scores from moderate (International Index of Erectile Function {IIEF-5} of 9.7) to mild to moderate (IIEF-5 score 12.7), but at the risk of worse oncological control (freedom from recurrence or treatment dropped from 93% in the control group to 86% in the NeuroSAFE group).

3.8.3. Pelvic lymph node dissection

Extended PLND, which includes the removal of the nodes overlying the external iliac artery and vein, the nodes within the obturator fossa located cranially and caudally to the obturator nerve, and the nodes medial and lateral to the internal iliac artery, provides accurate staging information [109] when identifying metastatic LN deposits, usually small volume, missed by modern imaging [110]. There has been significant interest in whether this would also improve clinical outcomes. However, a meta-analysis demonstrated that performing PLND during RP failed to improve oncological outcomes, including survival [111]. Moreover, two RCTs show no benefit of ePLND versus limited PLND in terms of freedom from BCR [112,113]. After additional follow-up, a secondary report from the largest RCT did demonstrate a lower rate of distant metastasis [112], while confirming the lack of effect of ePLND on the development of BCR or local pelvic nodal recurrence. This finding does not appear to be related to the number of LNs removed or the number of involved LNs removed, which were similar in the limited versus extended groups. On the other hand, ePLND is associated with an increased risk of complications; 8% of patients need drainage of a lymphocele [114,115], and there is an increased risk of thromboembolic disease [116]. Lymphoedema is also more likely after ePLND followed by postoperative pelvic RT than either treatment in isolation [117]. Therefore, before offering ePLND, clinicians should discuss the uncertainties regarding the benefits and possible advantages of detecting low-volume nodal disease via ePLND and how this may impact postoperative management against the risks and harms associated with ePLND.

3.8.4. Adjuvant treatment after radical prostatectomy

Extraprostatic extension and positive surgical margins are associated with an increased risk of recurrence. However, for patients with undetectable postoperative PSA, a meta-analysis suggests that adjuvant radiation treatment (ART) and early salvage radiotherapy (SRT) (before the PSA

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

>0.5 ng/ml) offer similar outcomes for event-free survival [118]. For patients with adverse pathology, that is, ISUP GG 4–5 and pT3/4 with or without positive margins [119], who were under-represented in the aforementioned meta-analysis, immediate adjuvant EBRT to the surgical bed after recovery of urinary function is still recommended.

3.8.5. *Definitive radiation treatment*

Dose-escalated intensity-modulated RT (IMRT) or volumetric modulated arc therapy (VMAT) are the gold standard for EBRT because they are associated with less toxicity than three-dimensional conformal RT (3D-CRT) techniques [120]. However, with such techniques, organ movement becomes a critical issue in terms of both tumour control and treatment toxicity. Therefore, they are performed with some form of image-guided RT (IGRT) (usually gold marker or cone-beam CT), in which organ movement can be visualised and corrected for in real time. Rates of severe late side effects (>grade 3) are 2–4% for the rectum and 2–6% for the genitourinary (GU) tract when this combination is used [121,122]. A systematic review and meta-analysis of observational studies comparing patients exposed or unexposed to RT in the course of treatment for PCa demonstrate an increased risk of developing second cancers for the bladder (OR: 1.39), colorectal (OR: 1.68), and rectum (OR: 1.62), with similar risks over lag times of 5 and 10 yr. Absolute risks over 10 yr are small (1–4%) but should be discussed with younger men in particular [113].

RCTs have shown that escalating the dose into the range of 74–80 Gy leads to a significant improvement in 10-yr BCR-free survival [123,124] and PCa-specific survival [125]. In men with intermediate- and high-risk PCa, there is evidence to support an OS benefit from a non-randomised but well-conducted propensity-matched retrospective analysis covering a total of 42 481 patients [126]. There may also be a benefit to adding a focal boost to the dominant lesion visible on MRI associated with good tolerability and improved biochemical PFS [127].

Fractionated RT utilises differences in the deoxyribonucleic acid repair capacity of normal and tumour tissue. Slowly proliferating cells are more sensitive to an increased dose per fraction, and PCa is considered to have a slow proliferation rate, so hypofractionation (HFX) has been explored.

A Cochrane review on moderate HFX, defined as RT with 2.5–3.4 Gy/fx for clinically localised PCa [128] included 11 studies (n = 8278) with a median follow-up of 72 mo showing little or no difference in PCa-specific survival (HR: 1.00) and similar toxicity, while dose-escalated moderate HFX failed to improve PFS but was associated with increased gastrointestinal (GI) toxicity [129] and is not recommended.

Ultra-HFX uses even larger doses per fraction and ideally requires stereotactic body RT (SBRT). In treating predominantly intermediate-risk localised disease, biochemical control is comparable to conventional fractionation. However, there are concerns about higher-grade GU toxicity, and ultra-HFX should be avoided in patients with severe pre-existing LUTS (International Prostate Symptom Score [IPSS] >19) and/or outflow obstruction with or without median lobe [130,131]. In the PACE-B trial, extreme HFX 36.25 Gy in five fraction SBRT was compared with conventional schedules of RT in patients with T1-2 disease, ISUP GG 1 (8%), ISUP GG 2 (92%), and PSA <20 ng/ml. Androgen deprivation was not permitted. Five-year Radiation Oncology/Toxicity grading (RTOG) rates were similar, although with a slight increase in grade 2 or worse GU toxicity (3.2% vs 5.5%). More interestingly, 5-yr biochemical control rates were equivalent [131].

Biodegradable spacer insertion involves using a liquid gel or balloon to increase the distance between the prostate and rectum and consequently reduce the amount of radiation reaching the rectum. No difference is observed in significant grade 3+ GI (acute or late) events, and there is no statistical difference in bowel-related QoL (risk difference = −0.16, 95% CI: −0.38 to 0.06, p = 0.15) [132]. Implantation is associated with a learning curve and should only be undertaken by teams with experience of transrectal ultrasound and transperineal procedures, with robust audit reporting in place.

The combination of androgen deprivation therapy (ADT) with various forms of RT has been extensively studied, with extremely strong evidence for the use of such combined modality therapy in several settings. An individual patient data meta-analysis included 12 trials with 10 853 patients. Median follow-up was over 11 yr. The use of ADT was clearly associated with significant improvements in BCR, metastatic recurrence, metastasis-free survival (MFS), and OS. The benefits of ADT were independent of RT dose, age, and risk groups [133]. For intermediate-risk disease, a short duration of 4–6 mo is optimal [134], while a longer duration of 1–3 yr is needed for high-risk patients [135]. In patients with localised high-risk PCa, dose-escalated IMRT, possibly including the pelvic lymphatics [136], and/or a brachytherapy (BT) boost is an option [137]. Long-term ADT, generally for 2–3 yr, is mandatory. The duration of ADT must consider performance status, comorbidities, and the number of poor prognostic factors.

3.8.6. *Brachytherapy*

Low-dose rate (LDR) BT uses permanent radioactive seeds implanted into the prostate and, as a monotherapy, is an option for those with favourable intermediate-risk disease (low-volume GS 3 + 4) and good urinary function, defined as an IPSS of <12 and a maximum flow rate of >15 ml/min [138]. Up to 85% relapse-free survival at 10 yr is demonstrated [139]. Patients who have had a previous transurethral resection of the prostate (TURP) can undergo BT without an increase in risk of urinary toxicity, with due attention to dose distribution. A minimal channel TURP is recommended, leaving at least a 1 cm rim of prostate tissue around the post-TURP urethral defect at the postero-lateral sides of the prostate, and there should be at least a 3-mo interval between TURP and BT to allow for adequate healing [140].

Although seen as a low-impact treatment modality, some patients experience significant urinary complications following implantation, such as urinary retention (1.5–22%), postimplantation TURP (8.7% of cases), and incontinence (0–19%) [141]. In about 40% of patients, ED develops after 3–5 yr.

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

As a boost with EBRT, LDR can be used as dose escalation in unfavourable intermediate- and high-risk patients combined with 12 mo of ADT. PSA PFS improved from 70 to 85% at 10 yr, although there was no impact on MFS or OS. It was associated with increased late grade 3+ GU toxicity (18% compared to 8%) and two treatment-related deaths [142,143]. Urinary toxicity was mainly related to the development of urethral strictures and incontinence.

High-dose rate (HDR) BT uses a radioactive source temporarily introduced into the prostate to deliver radiation. High-dose rate BT is combined with EBRT of at least 45 Gy as a method of dose escalation in intermediate- or high-risk PCa. QoL changes are similar to high-dose EBRT alone [144]. A systematic review of non-RCTs and data from population studies suggests outcomes with EBRT plus HDR BT are superior to EBRT alone [145].

3.8.7. *Alternative local treatment options*

New modalities have been developed as minimally invasive procedures with the aim of providing equivalent oncological safety, reduced toxicity, and improved functional outcomes. These modalities have largely been evaluated in cohorts of patients who we now recognise are unlikely to benefit from treatment in terms of oncological control.

3.8.7.1. Cryotherapy. Cryotherapy has been used for whole-gland treatment in PCa either as a primary or salvage treatment option. There is a lack of prospective comparative data regarding oncological outcomes of whole-gland cryosurgery as a curative treatment option for men with localised PCa, with most studies being noncomparative single-arm case series with short follow-up [146]. The main adverse effects of whole-gland cryosurgery are ED (18%), urinary incontinence (2–20%), urethral sloughing (0–38%), rectal pain and bleeding (3%), and recto-urethral fistula formation (0–6%) [146].

3.8.7.2. High-intensity focused ultrasound treatment. High-intensity focused US (HIFU) treatment consists of focused US waves emitted from a transducer that cause tissue damage by mechanical and thermal effects, as well as by cavitation. Data have shown poor long-term oncological outcomes for patients undergoing whole-gland treatment for high-risk localised disease [147], which prevents it from being considered as a reasonable alternative to the established curative treatment options. In addition, the expected improvements in functional outcome failed to materialise, with 12% of patients developing incontinence and 61% developing ED [148].

3.8.7.3. Focal therapy. PSA screening combined with MRI has led to the identification of smaller unifocal tumours. This, combined with the risks of overtreatment and consequences of whole-gland treatments, has led to the investigation of focal therapy aiming to ablate tumours while sparing the neuro-vascular bundles, sphincter, and urethra. The question remains which, if any, of these small unifocal tumours need treatment.

Prospective registry data confirm low treatment-related toxicity (4% decrease in pad-free continence; reduction in IIEF of 0.4 points after treatment) [149,150]. Oncologic outcomes are less clear with no randomised comparative data. The largest case series on oncologic outcomes following focal HIFU included 1379 patients with a median follow-up of 32 mo (65% of patients were D'Amico intermediate-risk and 28% high-risk) [151]. In this study, one repeated focal HIFU session was allowed and performed in 18% of all patients. MRI was performed if consecutive PSA rises were identified, and biopsies were offered if the MRI was suspicious. Eighty percent of patients had at least one follow-up MRI, and 44% had a follow-up biopsy. The primary outcome was failure-free survival (FFS), which was defined as evidence of cancer requiring whole-gland salvage treatment. At 7 yr, the FFS for intermediate- and high-risk cancers was 68% and 65%, respectively [151]. For now, focal therapy should be performed only within the context of a clinical trial setting or a well-designed prospective registry.

3.9. *Locally advanced prostate cancer: T3–4 N0, M0*

Data from retrospective case series of patients undergoing RP for T3 disease using conventional imaging demonstrated over 60% CSS at 15 yr and over 75% OS at 10 yr [152–154]. For cT3b–T4 disease, PCa cohort studies showed 10-yr CSS of over 87% and OS of 65%. For patients treated with RT, OS and disease-free survival were significantly better with additional adjuvant ADT for 3 yr [155]. Comparative data of surgery versus radiation strategies are still awaited.

3.10. *Persistent prostate-specific antigen after radical prostatectomy*

Between 5% and 20% of patients continue to have detectable PSA after RP (most often defined as post-RP PSA 0.1 ng/ml within 4–8 wk of surgery) [156,157]. It is often associated with poor prognosis: 74% experience further PSA progression [156] and an increased risk of metastases [157] and death [158]. As for PSA relapse, PSMA PET/CT is the most sensitive imaging modality to detect metastases [159].

The benefit of SRT in patients with persistent PSA remains unclear due to a lack of RCTs. Positive results have been suggested [159] after a 1:1 propensity score matching between SRT and no RT; OS rate after RP was 86.6% versus 72.6% in the entire cohort ($p < 0.01$ at 10 yr). Poor outcomes are driven by the level of pre-RT PSA, the presence of ISUP GG 4 in the specimen, and pT3b status [157,160,161]. The addition of ADT may improve PFS [160]. The available data suggest that patients with PSA persistence after RP may benefit from early aggressive multimodality treatment; however, the lack of prospective RCTs makes firm recommendations difficult.

3.11. *Treatment of cN1 prostate cancer*

In patients with pN+ PCa, early adjuvant ADT was shown to achieve a 10-yr CSS rate of 80% [162]. Pelvic RT combined with long-term ADT appeared to be beneficial in pN+ PCa patients treated with an ePLND, with at least a local control improvement and possibly survival [163,164].

The treatment of cN1 M0 PCa was evaluated in a systematic review [165] that included five studies. The findings suggested an advantage in both OS and CSS after local treat-

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

ment (RT or RP) combined with ADT, as compared with ADT alone. The intensification of systemic treatment with either abiraterone acetate or docetaxel for very high-risk localised or cN1 disease has also been assessed in analyses from the STAMPEDE multiarm RCT. Two RCTs from the study reported on the addition of abiraterone acetate to SOC in men with *de novo* high-risk/locally-advanced M0 disease, or relapse after primary curative therapy with high-risk features. On conventional imaging, 39% of patients (n = 774) were N1. Radiotherapy in addition to long-term ADT was administered in 71% of these patients. A meta-analysis involving 1974 patients [166] demonstrated an improvement in MFS (82% vs 69% at 6 yr) and OS (HR: 0.60; $p < 0.001$), suggesting combined abiraterone (for 2 yr) and ADT (for 3 yr) should be SOC in cN1 patients in addition to prostate- and whole-pelvic RT. A similar analysis assess-

Table 1 – Recommendations for the management of localized disease

Recommendations	Strength rating
Expectant management	
Offer watchful waiting in asymptomatic patients with life expectancy <10 years (based on comorbidities and age).	Strong
Low-risk disease	
Manage patients with a life expectancy >10 years and low-risk disease with active surveillance.	Strong
Favourable intermediate-risk disease	
Offer active surveillance (AS) to selected patients with ISUP GG 2 disease, e.g. < 10% pattern 4, PSA <10 ng/mL, ≤ cT2a, low disease extent on imaging and low extent of tumour in biopsies (≤ 3 positive cores with ISUP GG 2 and ≤ 50% cancer involvement/core) or another single element of intermediate-risk disease with low disease extent on imaging and low biopsy extent, accepting the potential increased risk of metastatic progression.	Weak
Reclassify patients with low-volume ISUP GG 2 disease included in AS protocols if repeat non-magnetic resonance imaging (MRI)-based systematic biopsies performed during monitoring reveal >3 positive cores or maximum cancer involvement >50%/core of ISUP GG 2 disease.	Weak
Offer RP to patients with a life expectancy of > ten years.	Strong
Offer nerve-sparing surgery to patients with a low risk of extracapsular disease on that side.	Strong
Offer low-dose rate (LDR) brachytherapy to patients with good urinary function and NCCN-favourable intermediate-risk disease.	Strong
Offer intensity-modulated radiotherapy (IMRT)/volumetric modulated arc therapy (VMAT) plus image-guided radiotherapy (IGRT), with a total dose of 76–78 Gy or moderate hypofractionation (60 Gy/20 fx in four weeks or 70 Gy/28 fx in six weeks), in combination with short-term androgen deprivation therapy (ADT) (four to six months).	Strong
Offer focal boosting to MRI-defined dominant intraprostatic tumour when using conventionally fractionated IMRT/IGRT (1.8–2.0 Gy per fraction), ensuring that Organ at Risk constraints are not exceeded.	Weak
Offer ultra-hypofractionated IMRT/IGRT or stereotactic body radiation therapy, using either 36.25 Gy (40 Gy to prostate) in 5 fx or 42.7 Gy in 7 fx delivered alternate days in patients with favourable intermediate risk considering urinary function.	Weak
Unfavourable intermediate-risk disease	
Patients with ISUP GG 3 disease should be excluded from AS protocols.	Strong
Offer intensity-modulated radiotherapy (IMRT)/volumetric modulated arc therapy (VMAT) plus image-guided radiotherapy (IGRT), with a total dose of 76–78 Gy or moderate hypofractionation (60 Gy/20 fx in four weeks or 70 Gy/28 fx in six weeks), in combination with short-term androgen deprivation therapy (ADT) (four to six months).	Strong
Offer focal boosting to MRI-defined dominant intraprostatic tumour when using conventionally fractionated IMRT/IGRT (1.8–2.0 Gy per fraction), ensuring that Organ at Risk constraints are not exceeded.	Weak
Offer LDR brachytherapy boost combined with IMRT/VMAT plus IGRT to patients with good urinary function and NCCN unfavourable intermediate-risk disease, in combination with short-term ADT (four to six months).	Weak
Offer high-dose rate brachytherapy boost combined with IMRT/VMAT plus IGRT to patients with good urinary function and NCCN unfavourable intermediate-risk disease in combination with short-term ADT (four to six months).	Weak
Other therapeutic options for intermediate-risk disease	
Only offer whole-gland ablative therapy (such as cryotherapy, high-intensity focused ultrasound, etc.) or focal ablative therapy within clinical trials or registries.	Strong
Do not offer ADT monotherapy to asymptomatic men not able to receive any local treatment.	Weak
High-risk disease	
Offer RP to selected patients.	Strong
In patients undergoing a lymph node dissection you should perform an ePLND.	Strong
Do not perform a frozen section of nodes during RP to decide whether to proceed with, or abandon, the procedure.	Strong
Offer intensity-modulated radiotherapy (IMRT)/volumetric modulated arc therapy (VMAT) plus image-guided radiotherapy (IGRT), with a total dose of 76–78 Gy or moderate hypofractionation (60 Gy/20 fx in four weeks or 70 Gy/28 fx in six weeks), in combination with long-term androgen deprivation therapy (ADT) (two to three years).	Strong
Offer focal boosting to magnetic resonance imaging (MRI)-defined dominant intraprostatic tumour when using normo-fractionated IMRT/IGRT (1.8–2.0 Gy per fraction) ensuring that Organ at Risk constraints are not exceeded.	Strong
Offer patients with good urinary function IMRT/VMAT plus IGRT with brachytherapy boost (either high-dose rate or low-dose rate) in combination with long-term ADT (two to three years).	Weak
Locally advanced disease	
Offer RP to selected patients with cN0 disease as part of multi-modal therapy.	Weak
In patients undergoing a lymph node dissection you should perform an ePLND.	Strong
Offer patients with cN0 disease intensity-modulated radiation therapy (IMRT)/volumetric modulated arc therapy (VMAT) plus image-guided radiation therapy in combination with long-term androgen deprivation therapy (ADT).	Strong
Offer patients with cN0 disease and good urinary function, IMRT/VMAT plus IGRT with brachytherapy boost (either high-dose rate or low-dose rate), in combination with long-term ADT.	Weak
Offer long-term ADT for at least two years.	Strong
Offer IMRT/VMAT plus IGRT to the prostate in combination with long-term ADT and two years of abiraterone to cN0M0 patients with ≥ 2 high-risk factors (cT3–4, Gleason ≥ 8 or prostate-specific antigen ≥ 40 ng/mL).	Strong
Offer IMRT/VMAT plus IGRT to the prostate plus pelvis in combination with long-term ADT and two years of abiraterone to cN1M0 patients.	Strong
Only offer ADT monotherapy to those patients unwilling or unable to receive any form of local treatment if they have a prostate-specific antigen (PSA)-doubling time <12 months, and either a PSA >50 ng/mL or a poorly differentiated tumour.	Strong

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

ing the role of docetaxel in this population failed to show a similar benefit.

4. Conclusions

The present text represents a summary of the 2026 EAU–EANM–ESTRO–ESUR–ISUP–SIOG PCa Guidelines. A summary of recommendations is presented in Table 1. For more detailed information and a full list of references, refer to the full-text version available at the EAU website (http://uroweb.org/guidelines/prostate-cancer/).

Author contributions: Philip Cornford had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Cornford, Tilki.
Acquisition of data: Tilki, van den Bergh, Briers, Chiu, Eberli, Epure, Farolfi, Fonteyne, Gandaglia, Gillessen, Grivas, Henry, van Leenders, Linares Espinos, Matsen, Oldenburg, van Oort, Oprea-Lager, Roberts, Rouvière, Sachdeva, Santis, Schoots, Smith, Stranne, Wiegel, Cornford.
Analysis and interpretation of data: Tilki, van den Bergh, Briers, Chiu, Eberli, Epure, Farolfi, Fonteyne, Gandaglia, Gillessen, Grivas, Henry, van Leenders, Linares Espinos, Matsen, Oldenburg, van Oort, Oprea-Lager, Roberts, Rouvière, Sachdeva, Santis, Schoots, Smith, Stranne, Wiegel, Cornford.
Drafting of the manuscript: Cornford.
Critical revision of the manuscript for important intellectual content: Tilki, van den Bergh, Briers, Chiu, Eberli, Epure, Farolfi, Fonteyne, Gandaglia, Gillessen, Grivas, Henry, van Leenders, Linares Espinos, Matsen, Oldenburg, van Oort, Oprea-Lager, Roberts, Rouvière, Sachdeva, Santis, Schoots, Smith, Stranne, Wiegel, Cornford.
Statistical analysis: None.
Obtaining funding: Smith.
Administrative, technical, or material support: Smith.
Supervision: Cornford, Tilki.
Other: None.

Financial disclosures: Philip Cornford certifies that all conflicts of interest, including specific financial interests and relationships and affiliations relevant to the subject matter or materials discussed in the manuscript (eg, employment/ affiliation, grants or funding, consultancies, honoraria, stock ownership or options, expert testimony, royalties, or patents filed, received, or pending), are the following:
Philip Cornford reported receiving company honoraria or consultation fees from Accord Healthcare, AstraZeneca and Bayer UK; receiving company speaker honoraria fees from Astellas Pharma a/s Danish Sales, AstraZeneca, Bayer UK, Ipsen Biopharm and Janssen Cilag; and serving on the board of trustees of Prostate Cancer Research.
Derya Tilki reported receiving company honoraria or consultation fees from A3P Biomedical, Amgen, Apogepha, Astellas, AstraZeneca, Bayer, Biontech, Ipsen, J&J, MSD, Novartis, Pfizer, Roche, Takeda, Veracyte.
Roderick C.N. van den Bergh reported receiving company honoraria or consultation fees from Astellas Pharma International, Ipsen Farmaceutica and Johnson & Johnson Innovative Medicine; participation in a company-sponsored speaker's bureau for Amgen; receiving fellowship or travel grants from Bayer; and receiving grants or research support from Astellas and Johnson & Johnson Innovative Medicine.
Daniel Eberli reported receiving company speaker honoraria fees from Astellas Pharma, Bayer (Schweiz), EDAP Germany and Janssen Cilag; serving as a company consultant to Astellas Pharma, Bayer (Schweiz), EDAP Germany and Iaculis; receiving grants or research support from Astellas Pharma, Bayer (Schweiz) and EDAP TMS; receiving fellowship or travel grants from Bayer (Schweiz); being a stock shareholder in MUVON Therapeutics and Ontrack Biomedical; serving as a director of the Department of Urology at University Hospital Zurich; holding an appointment as a Professor at University of Zurich; and being the owner of three patents.
Valérie Fonteyne reporting participation in the THUNDER (NCT06282588) and SAVE trials; received honoraria for lectures from Johnson & Johnson, Astellas, Bristol Myers Squibb and Issecam; travel support from Johnson & Johnson; and participation on advisory boards from Astellas.
Maria De Santis reported receiving company honoraria or consultation fees from Amgen, Astellas Pharma EMEA, De Santis, AAA, AbVie, Amgen, Astellas, AstraZeneca, Basilea, Bayer, BMS, EISAI, Ferring, Gilead, Immunomedics, Ipsen, Janssen, MSD, Merck, Novartis, Pfizer, Roche, Sandoz, Sanofi, SeaGen and Thermosome; and participation in clinical trials by Amgen, Astellas, AstraZeneca, Bayer, BMS, EISAI, Ferring, Ipsen, Janssen, MSD, Merck, Novartis, Pfizer, Roche, Sandoz, Sanofi, Amgen, Astellas, AstraZeneca, Bayer, BMS, EISAI, Ferring, Ipsen, Janssen, MSD, Merck, Novartis, Pfizer, Roche, Sandoz, Sanofi and SeaGen.
Giorgio Gandaglia reported receiving company speaker honorarium from Accord Healthcare and Ipsen; honoraria or consultation fees from Bayer S.p.A. and Janssen Cilag S.p.A; fellowship, travel grants from Bayer S.p.A. and Ipsen S.p.A; being a company consultant for Telix; and receiving grants/research supports from AIRC - Fondazione AIRC per la ricerca sul cancro.
Silke Gillessen reported receiving company honoraria or consultation fees from Amgen, Astellas Pharma, Boehringer Ingelheim Pharma, Daiichi Sankyo Schweiz, Ipsen Innovation, Novartis Pharma, PeerVoice, University of Applied Sciences and Arts of Southern Switzerland, UroPratica Group, EPG Health, Macrogenics, Avalere Health, InnoMedica Schweiz and Silvio Grasso Consulting; receiving company speaker honoraria fees from ASCO, ESMO, Orikata, PlayToKnow, Intellisphere, SGMO and AdMeTech Foundation; receiving fellowship or travel grants from Bayer (Schweiz), Johnson & Johnson Innovative Medicine, Intellisphere and Gilead; serving as a company consultant to Bayer (Schweiz), BMS, Merck - Pfizer Alliance, MSD, Pfizer and LinkinVax; participation in a company sponsored speaker's bureau for SAKK; being the owner of one patent; and attending a senior executive meeting at AstraZeneca.
Ann M. Henry reported receiving grants or research support from the University of Leeds and participation in clinical trials for Cancer Research UK. Jan Oldenburg reported receiving company honoraria or consultation fees from Astellas Pharma Norway, BMS, Janssen Cilag, MSD (Norge), Pfizer and Roche Accord; and receiving company speaker honoraria fees from Astellas Pharma Norway and Janssen Cilag.
Daniela E. Oprea-Lager reported receiving company honoraria or consultation fees from Novartis, Bayer, Curium, Ipsen, Astellas, Janssen and Telix. Inge van Oort reported receiving grants/research supports from Amgen, Astellas, Bayer B.V. and Novartis Pharma; receiving honoraria or consultation fees from Bayer B.V. and Novartis Pharma; and receiving company speaker honorarium from Recordati B.V. Matthew J. Roberts reported receiving company honoraria or consultation fees from Astellas Pharma Australia, AstraZeneca, Janssen and Cook Medical Australia; receiving fellowship or travel grants from Bayer Australia; receiving grants or research support from ANZUP Cancer Trials Group; serving as a company consultant to BXTAccelyon Australia; and participating in clinical trials by Janssen and AdvanCell Isotopes.
Olivier Rouvière reported receiving fellowship or travel grants from Philips France, and being the owner of one international patent. Johan Stranne reported being the chair of the Swedish national guidelines committee. Thomas Wiegel reported receiving company speaker honoraria fees from Bayer Vital GmbH, Ipsen Pharma GmbH, Janssen, Johnson &

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

Johnson Innovative Medicine, Recordati, Takeda Pharma Vertrieb GmbH & Co. KG; serving as a company consultant to Janssen; serving as a member of the steering committee of the German association of scientific medical societies S3 prostate guidelines.

Peter Chiu reported receiving speaker honoraria fees for a lecture.

Giorgio Gandaglia reported receiving company speaker honoraria fees from Accord Healthcare and Ipsen; receiving company honoraria or consultation fees from Bayer and Janssen Cilag; receiving fellowship or travel grants from Bayer and Ipsen; serving as a company consultant to Telix; and receiving grants or research support from AIRC.

Estefania Linares Espinós reported receiving company honoraria or consultation fees from Bayer, Astellas, Casen Recordati, MSD Spain and Pfizer Spain; receiving company speaker honoraria fees from Johnson & Johnson, Ipsen Pharma, Bayer, and Astellas; serving as a company consultant to Ipsen Pharma, Bayer and Johnson & Johnson; participation in clinical trials by Janssen Cilag; and receiving grants or research support from Astellas.

Ashwin Sachdeva reported receiving company speaker honoraria fees from Ipsen, Janssen Cilag; receiving company honoraria or consultation fees from Ipsen and Veracyte; receiving fellowship or travel grants from the Prostate Cancer Foundation; and participation in the STAMPEDE trial.

Geert J.L.H. van Leenders is President Elect of the International Society of Urological Pathology (ISUP) and co-chair of the European Society of Pathology Uropathology working group.

Ivo G. Schoots, Adina Epure, Erik Briers, Bo. Matsen, Andrea Farolfi, Emma J. Smith and Nikolaos Grivas have nothing to declare.

Funding/Support and role of the sponsor: Philip Cornford certifies that all funding, other financial support, and material support for this research and/or work are clearly identified in the manuscript. The names of the organization or organizations that had a role in sponsoring the data and material in the study are also listed below: European Association of Urology.

All funding or other financial support, and material support for this research and/or work, if any, are clearly identified hereunder: The specific role of the funding organization or sponsor is as follows: Design and conduct of the study, Collection of the data, Management of the data, Analysis, Interpretation of the data, Preparation, Review, and Approval of the manuscript.

References

[1] Cornford P, van den Bergh RCN, Briers E, et al. EAU-EANM-ESTRO-ESUR-ISUP-SIOG guidelines on prostate Cancer-2024 update. Part I: Screening, diagnosis, and local treatment with curative intent. Eur Urol 2024;86:148–63.

[2] Cornford P, Tilki D, van den Bergh RCN, et al. EAU-EANM-ESTRO-ESUR-ISUP-SIOG guidelines on prostate cancer. Edn presented at the 41st EAU Annual Congress London; 2026. https://uroweb.org/guidelines/prostate-cancer.

[3] Guyatt GH, Oxman AD, Kunz R, et al. Going from evidence to recommendations. BMJ 2008;336:1049–51.

[4] European Union. Prostate cancer burden in EU-27. European Commission; 2021. ecis.jrc.ec.europa.eu/sites/default/files/2023-12/prostate_cancer_En-Nov_2021.pdf.

[5] Bergengren O, Pekala KR, Matsoukas K, et al. 2022 Update on prostate cancer epidemiology and risk factors-A systematic review. Eur Urol 2023;84:191–206.

[6] Hemminki K. Familial risk and familial survival in prostate cancer. World J Urol 2012;30:143–8.

[7] Huynh-Le MP, Karunamuni R, Fan CC, et al. Common genetic and clinical risk factors: association with fatal prostate cancer in the Cohort of Swedish Men. Prostate Cancer Prostatic Dis 2021;24:845–51.

[8] Page EC, Bancroft EK, Brook MN, et al. Interim results from the Impact study: evidence for prostate-specific antigen screening in BRCA2 mutation carriers. Eur Urol 2019;76:831–42.

[9] Bancroft EK, Page EC, Brook MN, et al. A prospective prostate cancer screening programme for men with pathogenic variants in mismatch repair genes (Impact): initial results from an international prospective study. Lancet Oncol 2021;22:1618–31.

[10] Nyberg T, Tischkowitz M, Antoniou AC. BRCA1 and BRCA2 pathogenic variants and prostate cancer risk: systematic review and meta-analysis. Br J Cancer 2022;126:1067–81.

[11] Haider A, Zitzmann M, Doros G, Isbarn H, Hammerer P, Yassin A. Incidence of prostate cancer in hypogonadal men receiving testosterone therapy: observations from 5-year median followup of 3 registries. J Urol 2015;193:80–6.

[12] Golla V, Kaplan AL. Testosterone therapy on active surveillance and following definitive treatment for prostate cancer. Curr Urol Rep 2017;18:49.

[13] Brierley JD, Gospodarowicz MK, Wittekind C. TNM classification of malignant tumours, ed. 8. Oxford: Wiley-Blackwell; 2017.

[14] Epstein JI, Allsbrook WC, Jr, Amin MB, Egevad LL, ISUP Grading Committee. The 2005 International Society of Urological Pathology (ISUP) consensus conference on Gleason grading of prostatic carcinoma. Am J Surg Pathol 2005;29:1228–42.

[15] van Leenders GJLH, van der Kwast TH, Grignon DJ, et al. The 2019 International Society of Urological Pathology (ISUP) consensus conference on grading of prostatic carcinoma. Am J Surg Pathol 2020;44:e87–99.

[16] Gnanapragasam VJ, Lophatananon A, Wright KA, Muir KR, Gavin A, Greenberg DC. Improving clinical risk stratification at diagnosis in primary prostate cancer: A prognostic modelling study. PLoS Med 2016;13:e1002063.

[17] Bell KJL, Del Mar C, Wright G, Dickinson J, Glasziou P. Prevalence of incidental prostate cancer: A systematic review of autopsy studies. Int J Cancer 2015;137:1749–57.

[18] Van der Kwast TH, Roobol MJ. Defining the threshold for significant versus insignificant prostate cancer. Nat Rev Urol 2013;10:473–82.

[19] Goel S, Shoag JE, Gross MD, et al. Concordance between biopsy and radical prostatectomy pathology in the era of targeted biopsy: A systematic review and meta-analysis. Eur Urol Oncol 2020;3:10–20.

[20] Ilic D, Djulbegovic M, Jung JH, et al. Prostate cancer screening with prostate-specific antigen (PSA) test: a systematic review and meta-analysis. BMJ 2018;362:k3519.

[21] Hayes JH, Barry MJ. Screening for prostate cancer with the prostate-specific antigen test: a review of current evidence. JAMA 2014;311:1143–9.

[22] Hugosson J, Roobol MJ, Månsson M, et al. A 16-yr Follow-up of the European Randomized study of Screening for Prostate Cancer. Eur Urol 2019;76:43–51.

[23] Roobol MJ, de Vos II, Månsson M, et al. European study of prostate cancer screening - 23-year follow-up. N Engl J Med 2025;393:1669–80.

[24] de Vos II, Meertens A, Hogenhout R, Remmers S, Roobol MJ, ERSPC Rotterdam Study Group. A detailed evaluation of the effect of prostate-specific antigen-based screening on morbidity and mortality of prostate cancer: 21-year follow-up results of the Rotterdam section of the European randomised study of screening for prostate cancer. Eur Urol 2023;84:426–34.

[25] Nordström T, Adolfsson J, Grönberg H, Eklund M. Repeat prostate-specific antigen tests before prostate biopsy decisions. J Natl Cancer Inst 2016;108:djw165.

[26] Grey ADR, Scott R, Shah B, et al. Multiparametric ultrasound versus multiparametric MRI to diagnose prostate cancer (CADMUS): a prospective, multicentre, paired-cohort, confirmatory study. Lancet Oncol 2022;23:428–38.

[27] Yusim I, Krenawi M, Mazor E, Novack V, Mabjeesh NJ. The use of prostate specific antigen density to predict clinically significant prostate cancer. Sci Rep 2020;10:20015.

[28] Fazekas T, Shim SR, Basile G, et al. Magnetic resonance imaging in prostate cancer screening: a systematic review and meta-analysis. JAMA Oncol 2024;10:745–54.

[29] Houlahan KE, Salmasi A, Sadun TY, et al. Molecular hallmarks of multiparametric magnetic resonance imaging visibility in prostate cancer. Eur Urol 2019;76:18–23.

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

[30] Al-Monajjed R, Boschheidgen M, Lakes J, et al. Prostate cancer detection in younger men: A comparative analysis of systematic and magnetic resonance imaging-targeted biopsy in the PROBASE trial. Eur Urol 2025;88:240–4.
[31] Ng ABCD, Asif A, Agarwal R, et al. Biparametric vs multiparametric MRI for prostate cancer diagnosis: the PRIME diagnostic clinical trial. JAMA 2025;334:1170–9.
[32] Drost FH, Osses DF, Nieboer D, et al. Prostate MRI, with or without MRI-targeted biopsy, and systematic biopsy for detecting prostate cancer. Cochrane Database Syst Rev 2019;4:CD012663.
[33] Kasivisvanathan V, Rannikko AS, Borghi M, et al. MRI-targeted or standard biopsy for prostate-cancer diagnosis. N Engl J Med 2018;378:1767–77.
[34] Rouvière O, Puech P, Renard-Penna R, et al. Use of prostate systematic and targeted biopsy on the basis of multiparametric MRI in biopsy-naive patients (MRI-FIRST): a prospective, multicentre, paired diagnostic study. Lancet Oncol 2019;20:100–9.
[35] Exterkate L, Wegelin O, Barentsz JO, et al. Is there still a need for repeated systematic biopsies in patients with previous negative biopsies in the era of magnetic resonance imaging-targeted biopsies of the prostate? Eur Urol Oncol 2020;3:216–23.
[36] Schoots IG, Padhani AR. Risk-adapted biopsy decision based on prostate magnetic resonance imaging and prostate-specific antigen density for enhanced biopsy avoidance in first prostate cancer diagnostic evaluation. BJU Int 2021;127:175–8.
[37] Rajendran I, Lee KL, Thavaraja L, Barrett T. Risk stratification of prostate cancer with MRI and prostate-specific antigen density-based tool for personalized decision making. Br J Radiol 2024;97:113–9.
[38] Carlsson S, Assel M, Sjoberg D, et al. Influence of blood prostate specific antigen levels at age 60 on benefits and harms of prostate cancer screening: population based cohort study. BMJ 2014;348:g2296.
[39] Vickers AJ, Ulmert D, Sjoberg DD, et al. Strategy for detection of prostate cancer based on relation between prostate specific antigen at age 40–55 and long term risk of metastasis: case-control study. BMJ 2013;346:f2023.
[40] Gelfond J, Choate K, Ankerst DP, Hernandez J, Leach RJ, Thompson Jr IM. Intermediate-term risk of prostate cancer is directly related to baseline prostate specific antigen: implications for reducing the burden of prostate specific antigen screening. J Urol 2015;194:46–51.
[41] Boyle HJ, Alibhai S, Decoster L, et al. Updated recommendations of the International Society of Geriatric Oncology on prostate cancer management in older patients. Eur J Cancer 2019;116:116–36.
[42] Kanagarajah A, Hogan D, Yao HH, Dundee P, O'Connell HE. A systematic review on the outcomes of local anaesthetic transperineal prostate biopsy. BJU Int 2023;131:408–23.
[43] Watts KL, Frechette L, Muller B, et al. Systematic review and meta-analysis comparing cognitive vs. image-guided fusion prostate biopsy for the detection of prostate cancer. Urol Oncol 2020;38:734.e19–e25.
[44] Wegelin O, Exterkate L, van der Leest M, et al. The FUTURE trial: A multicenter randomised controlled trial on target biopsy techniques based on magnetic resonance imaging in the diagnosis of prostate cancer in patients with prior negative biopsies. Eur Urol 2019;75:582–90.
[45] Wegelin O, van Melick HHE, Hooft L, et al. Comparing Three Different Techniques for magnetic resonance imaging-targeted Prostate Biopsies: A Systematic Review of In-bore versus magnetic resonance imaging-transrectal ultrasound fusion versus Cognitive Registration. Is there a Preferred Technique? Eur Urol 2017;71:517–31.
[46] Barrett T, de Rooij M, Giganti F, Allen C, Barentsz JO, Padhani AR. Quality checkpoints in the MRI-directed prostate cancer diagnostic pathway. Nat Rev Urol 2023;20:9–22.
[47] Ruan M, Wang H, Li X, Song G. Novel sampling scheme with reduced cores in men with multiparametric MRI-visible lesions undergoing prostate biopsy. Abdom Radiol (NY) 2023;48:2139–47.
[48] Zambon A, Nguyen TA, Fourcade A, et al. Which protocol for prostate biopsies in patients with a positive MRI? Interest of systematic biopsies by sectors. Prostate Cancer Prostatic Dis 2024;27:500–6.
[49] Hagens MJ, Fernandez Salamanca M, Padhani AR, van Leeuwen PJ, van der Poel HG, Schoots IG. Diagnostic performance of a magnetic resonance imaging-directed targeted plus regional biopsy approach in prostate cancer diagnosis: a systematic review and meta-analysis. Eur Urol Open Sci 2022;40:95–103.
[50] Noujeim JP, Belahsen Y, Lefebvre Y, et al. Optimizing multiparametric magnetic resonance imaging-targeted biopsy and detection of clinically significant prostate cancer: the role of perilesional sampling. Prostate Cancer Prostatic Dis 2023;26:575–80.
[51] Tu X, Liu Z, Chang T, et al. Transperineal magnetic resonance imaging-targeted biopsy may perform better than transrectal route in the detection of clinically significant prostate cancer: systematic review and meta-analysis. Clin Genitourin Cancer 2019;17:e860–70.
[52] Jäderling F, Bergman M, Engel JC, et al. Tailoring biopsy strategy in the MRI-fusion prostate biopsy era: systematic, targeted or neither? BMC Urol 2024;24:168.
[53] Bass EJ, Donaldson IA, Freeman A, et al. Magnetic resonance imaging targeted transperineal prostate biopsy: a local anaesthetic approach. Prostate Cancer Prostatic Dis 2017;20:311–7.
[54] Roberts MJ, Bennett HY, Harris PN, et al. Prostate biopsy-related infection: a systematic review of risk factors, prevention strategies, and management approaches. Urology 2017;104:11–21.
[55] Pilatz A, Veeratterapillay R, Köves B, et al. Update on strategies to reduce infectious complications after prostate biopsy. Eur Urol Focus 2019;5:20–8.
[56] Basourakos SP, Alshak MN, Lewicki PJ, et al. Role of prophylactic antibiotics in transperineal prostate biopsy: a systematic review and meta-analysis. Eur Urol Open Sci 2022;37:53–63.
[57] Ding XF, Luan Y, Lu SM, et al. Risk factors for infection complications after transrectal ultrasound-guided transperineal prostate biopsy. World J Urol 2021;39:2463–7.
[58] Shaker HS, Badawy M, Emam A, Abdulbaki MS. Does the use of povidone iodine suppository decrease the infective complications of TRUS guided prostate biopsies? A randomized prospective study. QJM: Int J Med 2020;113.
[59] Pilatz A, Dimitropoulos K, Veeratterapillay R, et al. Antibiotic prophylaxis for the prevention of infectious complications following prostate biopsy: a systematic review and meta-analysis. J Urol 2020;204:224–30.
[60] Kweldam CF, Kümmerlin IP, Nieboer D, et al. Disease-specific survival of patients with invasive cribriform and intraductal prostate cancer at diagnostic biopsy. Mod Pathol 2016;29:630–6.
[61] Saeter T, Vlatkovic L, Waaler G, et al. Intraductal carcinoma of the prostate on diagnostic needle biopsy predicts prostate cancer mortality: a population-based study. Prostate 2017;77:859–65.
[62] Ploussard G, Manceau C, Beauval JB, et al. Decreased accuracy of the prostate cancer EAU risk group classification in the era of imaging-guided diagnostic pathway: proposal for a new classification based on MRI-targeted biopsies and early oncologic outcomes after surgery. World J Urol 2020;38:2493–500.
[63] Soeterik TFW, van Melick HHE, Dijksman LM, Biesma DH, Witjes JA, van Basten JA. Multiparametric magnetic resonance imaging should be preferred over digital rectal examination for prostate cancer local staging and disease risk classification. Urology 2021;147:205–12.
[64] Zaurito P, Stabile A, Montorsi F, Briganti A, Gandaglia G. The prognostic role of prostate MRI in prostate cancer patients. Curr Opin Urol 2025;35:418–25.
[65] Hövels AM, Heesakkers RA, Adang EM, et al. The diagnostic accuracy of CT and MRI in the staging of pelvic lymph nodes in patients with prostate cancer: a meta-analysis. Clin Radiol 2008;63:387–95.
[66] Di Pierro GB, Salciccia S, Frisenda M, et al. Comparison of four validated nomograms (Memorial Sloan Kettering Cancer Center, briganti 2012, 2017, and 2019) predicting lymph node invasion in patients with high-risk prostate cancer candidates for radical prostatectomy and extended pelvic lymph node dissection: clinical experience and review of the literature. Cancers 2023;15:1683.
[67] Perera M, Papa N, Roberts M, et al. Gallium-68 prostate-specific membrane antigen positron emission tomography in advanced prostate cancer-updated diagnostic utility, sensitivity, specificity, and distribution of prostate-specific membrane antigen-avid lesions: A systematic review and meta-analysis. Eur Urol 2020;77:403–17.

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

[68] Shen G, Deng H, Hu S, Jia Z. Comparison of choline-PET/CT, MRI, SPECT, and bone scintigraphy in the diagnosis of bone metastases in patients with prostate cancer: a meta-analysis. Skelet Radiol 2014;43:1503–13.
[69] Lecouvet FE, El Mouedden J, Collette L, et al. Can whole-body magnetic resonance imaging with diffusion-weighted imaging replace Tc 99m bone scanning and computed tomography for single-step detection of metastases in patients with high-risk prostate cancer? Eur Urol 2012;62:68–75.
[70] Pasoglou V, Larbi A, Collette L, et al. One-step TNM staging of high-risk prostate cancer using magnetic resonance imaging (MRI): toward an upfront simplified "all-in-one" imaging approach? Prostate 2014;74:469–77.
[71] Hofman MS, Lawrentschuk N, Francis RJ, et al. Prostate-specific membrane antigen PET-CT in patients with high-risk prostate cancer before curative-intent surgery or radiotherapy (proPSMA): a prospective, randomised, multicentre study. Lancet 2020;395:1208–16.
[72] Djaïleb L, Armstrong WR, Thompson D, et al. Presurgical 68Ga-PSMA-11 positron emission tomography for biochemical recurrence risk assessment: a follow-up analysis of a multicenter prospective Phase 3 imaging trial. Eur Urol 2023;84:588–96.
[73] Walter LC, Covinsky KE. Cancer screening in elderly patients: a framework for individualized decision making. JAMA 2001;285:2750–6.
[74] Studenski S, Perera S, Patel K, et al. Gait speed and survival in older adults. JAMA 2011;305:50–8.
[75] Groome PA, Rohland SL, Siemens DR, Brundage MD, Heaton J, Mackillop WJ. Assessing the impact of comorbid illnesses on death within 10 years in prostate cancer treatment candidates. Cancer 2011;117:3943–52.
[76] Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis 1987;40:373–83.
[77] Searle SD, Mitnitski A, Gahbauer EA, Gill TM, Rockwood K. A standard procedure for creating a frailty index. BMC Geriatr 2008;8:24.
[78] Borson S, Scanlan JM, Chen P, Ganguli M. The Mini-Cog as a screen for dementia: validation in a population-based sample. J Am Geriatr Soc 2003;51:1451–4.
[79] Albertsen PC, Moore DF, Shih W, Lin Y, Li H, Lu-Yao GL. Impact of comorbidity on survival among men with localized prostate cancer. J Clin Oncol 2011;29:1335–41.
[80] Albertsen PC. Observational studies and the natural history of screen-detected prostate cancer. Curr Opin Urol 2015;25:232–7.
[81] Thomsen FB, Brasso K, Klotz LH, Røder MA, Berg KD, Iversen P. Active surveillance for clinically localized prostate cancer–a systematic review. J Surg Oncol 2014;109:830–5.
[82] Bruinsma SM, Roobol MJ, Carroll PR, et al. Expert consensus document: semantics in active surveillance for men with localized prostate cancer - results of a modified Delphi consensus procedure. Nat Rev Urol 2017;14:312–22.
[83] Klotz L, Pond G, Loblaw A, et al. Randomized study of systematic biopsy versus magnetic resonance imaging and targeted and systematic biopsy in men on active surveillance (ASIST): 2-year postbiopsy follow-up. Eur Urol 2020;77:311–7.
[84] Amin A, Scheltema MJ, Shnier R, et al. The magnetic resonance imaging in active surveillance (MRIAS) trial: use of baseline multiparametric magnetic resonance imaging and saturation biopsy to reduce the frequency of surveillance prostate biopsies. J Urol 2020;203:910–7.
[85] van der Leest M, Cornel E, Israël B, et al. Head-to-head comparison of transrectal ultrasound-guided prostate biopsy versus multiparametric prostate resonance imaging with subsequent magnetic resonance-guided biopsy in biopsy-naive men with elevated prostate-specific antigen: a large prospective multicenter clinical study. Eur Urol 2019;75:570–8.
[86] Hamdy FC, Donovan JL, Lane JA, et al. Fifteen-year outcomes after monitoring, surgery, or radiotherapy for prostate cancer. N Engl J Med 2023;388:1547–58.
[87] Lam TBL, MacLennan S, Willemse PM, et al. EAU-EANM-ESTRO-ESUR-SIOG prostate cancer guideline panel consensus statements for deferred treatment with curative intent for localised prostate cancer from an international collaborative study (DETECTIVE study). Eur Urol 2019;76:790–813.
[88] Ericson KJ, Wu SS, Lundy SD, Thomas LJ, Klein EA, McKenney JK. Diagnostic accuracy of prostate biopsy for detecting cribriform Gleason Pattern 4 carcinoma and intraductal carcinoma in paired radical prostatectomy specimens: implications for active surveillance. J Urol 2020;203:311–9.
[89] Moreira DM, Fleshner NE, Freedland SJ. Baseline perineural invasion is associated with shorter time to progression in men with prostate cancer undergoing active surveillance: results from the REDEEM study. J Urol 2015;194:1258–63.
[90] Stavrinides V, Papageorgiou G, Danks D, et al. Mapping PSA density to outcome of MRI-based active surveillance for prostate cancer through joint longitudinal-survival models. Prostate Cancer Prostatic Dis 2021;24:1028–31.
[91] Olivier J, Kasivisvanathan V, Drumez E, et al. Low-risk prostate cancer selected for active surveillance with negative MRI at entry: can repeat biopsies at 1 year be avoided? A pilot study. World J Urol 2019;37:253–9.
[92] Chu CE, Cowan JE, Fasulo V, et al. The clinical significance of multiple negative surveillance prostate biopsies for men on active surveillance-does cancer vanish or simply hide? J Urol 2021;205:109–14.
[93] Gnanapragasam VJ, Keates A, Lophatananon A, Thankapannair V. The 5-year results of the Stratified Cancer Active Surveillance programme for men with prostate cancer. BJU Int 2025;135:851–9.
[94] O'Connor LP, Wang AZ, Yerram NK, et al. Changes in magnetic resonance imaging using the prostate cancer radiologic estimation of change in sequential evaluation criteria to detect prostate cancer progression for men on active surveillance. Eur Urol Oncol 2021;4:227–34.
[95] Moore CM, King LE, Withington J, et al. Best current practice and research priorities in active surveillance for prostate cancer-A report of a Movember international consensus meeting. Eur Urol Oncol 2023;6:160–82.
[96] Holmberg L, Garmo H, Andersson SO, et al. Radical prostatectomy or watchful waiting in early prostate cancer. N Engl J Med 2024;391:1362–4.
[97] Wilt TJ, Jones KM, Barry MJ, et al. Follow-up of prostatectomy versus observation for early prostate cancer. N Engl J Med 2017;377:132–42.
[98] Schreiber D, Wong AT, Rineer J, Weedon J, Schwartz D. Prostate biopsy concordance in a large population-based sample: a surveillance, epidemiology and end results study. J Clin Pathol 2015;68:453–7.
[99] Yossepowitch O, Eggener SE, Serio AM, et al. Secondary therapy, metastatic progression, and cancer-specific mortality in men with clinically high-risk prostate cancer treated with radical prostatectomy. Eur Urol 2008;53:950–9.
[100] Joniau S, Briganti A, Gontero P, et al. Stratification of high-risk prostate cancer into prognostic categories: a European multi-institutional study. Eur Urol 2015;67:157–64.
[101] Mungovan SF, Sandhu JS, Akin O, Smart NA, Graham PL, Patel MI. Preoperative membranous urethral length measurement and continence recovery following radical prostatectomy: a systematic review and meta-analysis. Eur Urol 2017;71:368–78.
[102] van Dijk-de Haan MC, Boellaard TN, Tissier R, et al. Value of different magnetic resonance imaging-based measurements of anatomical structures on preoperative prostate imaging in predicting urinary continence after radical prostatectomy in men with prostate cancer: a systematic review and meta-analysis. Eur Urol Focus 2022;8:1211–25.
[103] Veerman H, Hagens MJ, Hoeks CM, et al. A standardized method to measure the membranous urethral length (MUL) on MRI of the prostate with high inter- and intra-observer agreement. Eur Radiol 2023;33:3295–302.
[104] Walz J, Epstein JI, Ganzer R, et al. A critical analysis of the current knowledge of surgical anatomy of the prostate related to optimisation of cancer control and preservation of continence and erection in candidates for radical prostatectomy: an update. Eur Urol 2016;70:301–11.
[105] Vis AN, van den Bergh RCN, van der Poel HG, et al. Selection of patients for nerve sparing surgery in robot-assisted radical prostatectomy. BJUI Compass 2022;3:6–18.
[106] de Rooij M, Hamoen EHJ, Witjes JA, Barentsz JO, Rovers MM. Accuracy of magnetic resonance imaging for local staging of

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

prostate cancer: A diagnostic meta-analysis. Eur Urol 2016;70:233–45.
[107] Kroon LJ, van der Slot MA, van den Bergh RCN, Roobol MJ, van Leenders GJLH. Neurovascular structure-adjacent frozen-section examination (NeuroSAFE) during radical prostatectomy: a systematic review and meta-analysis. Eur Urol Oncol 2025;8:1365–74.
[108] Dinneen E, Almeida-Magana R, Al-Hammouri T, et al. Effect of NeuroSAFE-guided RARP versus standard RARP on erectile function and urinary continence in patients with localised prostate cancer (NeuroSAFE PROOF): a multicentre, patient-blinded, randomised, controlled phase 3 trial. Lancet Oncol 2025;26:447–58.
[109] Fossati N, Willemse PM, Van den Broeck T, et al. The benefits and harms of different extents of lymph node dissection during radical prostatectomy for prostate cancer: a systematic review. Eur Urol 2017;72:84–109.
[110] Hope TA, Eiber M, Armstrong WR, et al. Diagnostic accuracy of 68Ga-PSMA-11 PET for pelvic nodal metastasis detection prior to radical prostatectomy and pelvic lymph node dissection: a multicenter prospective Phase 3 imaging trial. JAMA Oncol 2021;7:1635–42.
[111] Ding G, Tang G, Wang T, Zou Q, Cui Y, Wu J. A comparative analysis of perioperative complications and biochemical recurrence between standard and extended pelvic lymph node dissection in prostate cancer patients undergoing radical prostatectomy: a systematic review and meta-analysis. Int J Surg 2024;110:1735–43.
[112] Touijer KA, Vertosick EA, Sjoberg DD, et al. Pelvic lymph node dissection in prostate cancer: update from a randomized clinical trial of limited versus extended dissection. Eur Urol 2025;87:253–60.
[113] Wallis CJD, Mahar AL, Choo R, et al. Second malignancies after radiotherapy for prostate cancer: systematic review and meta-analysis. BMJ 2016;352:i851.
[114] Cacciamani GE, Maas M, Nassiri N, et al. Impact of pelvic lymph node dissection and its extent on perioperative morbidity in patients undergoing radical prostatectomy for prostate cancer: a comprehensive systematic review and meta-analysis. Eur Urol Oncol 2021;4:134–49.
[115] Pose RM, Knipper S, Beyer B, et al. Impact of peritoneal bladder flap in robot-assisted radical prostatectomy patients on lymphoceles: a prospective randomised trial. World J Urol 2025;43:148.
[116] Wani M, Al-Mitwalli A, Mukherjee S, et al. Venous thromboembolism (VTE) in post-prostatectomy patients: systematic review and meta-analysis. J Clin Med 2023;12:3979.
[117] Clinckaert A, Callens K, Cooreman A, et al. The prevalence of lower limb and Genital lymphedema after prostate cancer treatment: a systematic review. Cancers 2022;14:5667.
[118] Vale CL, Fisher D, Kneebone A, et al. Adjuvant or early salvage radiotherapy for the treatment of localised and locally advanced prostate cancer: a prospectively planned systematic review and meta-analysis of aggregate data. Lancet 2020;396:1422–31.
[119] Tilki D, Chen MH, Wu J, et al. Adjuvant versus early salvage radiation therapy for men at high risk for recurrence following radical prostatectomy for prostate cancer and the risk of death. J Clin Oncol 2021;39:2284–93.
[120] Yu T, Zhang Q, Zheng T, et al. The effectiveness of intensity modulated radiation therapy versus three-dimensional radiation therapy in prostate cancer: a meta-analysis of the literatures. PLOS One 2016;11:e0154499.
[121] Viani GA, Stefano EJ, Afonso SL. Higher-than-conventional radiation doses in localized prostate cancer treatment: a meta-analysis of randomized, controlled trials. Int J Radiat Oncol Biol Phys 2009;74:1405–18.
[122] Kerkmeijer LGW, Groen VH, Pos FJ, et al. Focal boost to the intraprostatic tumor in external beam radiotherapy for patients with localized prostate cancer: results from the FLAME randomized Phase III trial. J Clin Oncol 2021;39:787–96.
[123] Heemsbergen WD, Al-Mamgani A, Slot A, Dielwart MFH, Lebesque JV. Long-term results of the Dutch randomized prostate cancer trial: impact of dose-escalation on local, biochemical, clinical failure, and survival. Radiother Oncol 2014;110:104–9.
[124] Michalski JM, Moughan J, Purdy J, et al. Effect of standard vs dose-escalated radiation therapy for patients with intermediate-risk prostate cancer: the NRG oncology RTOG 0126 randomized clinical trial. JAMA Oncol 2018;4:e180039.
[125] Pasalic D, Kuban DA, Allen PK, et al. Dose escalation for prostate adenocarcinoma: a long-term update on the outcomes of a Phase 3, single institution randomized clinical trial. Int J Radiat Oncol Biol Phys 2019;104:790–7.
[126] Kalbasi A, Li J, Berman A, et al. Dose-escalated irradiation and overall survival in men with nonmetastatic prostate cancer. JAMA Oncol 2015;1:897–906.
[127] Poon DMC, Yuan J, Yang B, et al. Magnetic resonance imaging-guided focal boost to intraprostatic lesions using external beam radiotherapy for localized prostate cancer: a systematic review and meta-analysis. Eur Urol Oncol 2023;6:116–27.
[128] Hickey BE, James ML, Daly T, Soh FY, Jeffery M. Hypofractionation for clinically localized prostate cancer. Cochrane Database Syst Rev 2019;9:CD011462.
[129] Kishan AU, Sun Y, Tree AC, et al. Hypofractionated radiotherapy for prostate cancer (Hydra): an individual patient data meta-analysis of randomised trials in the MARCAP consortium. Lancet Oncol 2025;26:459–69.
[130] Widmark A, Gunnlaugsson A, Beckman L, et al. Ultra-hypofractionated versus conventionally fractionated radiotherapy for prostate cancer: 5-year outcomes of the HYPO-RT-PC randomised, non-inferiority, phase 3 trial. Lancet 2019;394:385–95.
[131] van As N, Griffin C, Tree A, et al. Phase 3 trial of stereotactic body radiotherapy in localized prostate cancer. N Engl J Med 2024;391:1413–25.
[132] Wong CHM, Ko ICH, Leung DKW, et al. Does biodegradable perirectal spacer mitigate treatment toxicities in radiation therapy for localised prostate cancer-a systematic review and meta-analysis. Prostate Cancer Prostatic Dis 2025;28:927–37.
[133] Kishan AU, Sun Y, Hartman H, et al. Androgen deprivation therapy use and duration with definitive radiotherapy for localised prostate cancer: an individual patient data meta-analysis. Lancet Oncol 2022;23:304–16.
[134] Krauss DJ, Karrison T, Martinez AA, et al. Dose-escalated radiotherapy alone or in combination with short-term androgen deprivation for intermediate-risk prostate cancer: results of a Phase III multi-institutional trial. J Clin Oncol 2023;41:3203–16.
[135] Bolla M, de Reijke TM, Van Tienhoven G, et al. Duration of androgen suppression in the treatment of prostate cancer. N Engl J Med 2009;360:2516–27.
[136] Moris L, Cumberbatch MG, Van den Broeck T, et al. Benefits and risks of primary treatments for high-risk localized and locally advanced prostate cancer: an international multidisciplinary systematic review. Eur Urol 2020;77:614–27.
[137] Morris WJ, Tyldesley S, Rodda S, et al. Androgen suppression combined with elective nodal and dose escalated radiation therapy (the ASCENDE-RT Trial): an analysis of survival endpoints for a randomized trial comparing a low-dose-rate brachytherapy boost to a dose-escalated external beam boost for high- and intermediate-risk prostate cancer. Int J Radiat Oncol Biol Phys 2017;98:275–85.
[138] Martens C, Pond G, Webster D, McLean M, Gillan C, Crook J. Relationship of the international prostate symptom score with urinary flow studies, and catheterization rates following 125I prostate brachytherapy. Brachytherapy 2006;5:9–13.
[139] Grimm P, Billiet I, Bostwick D, et al. Comparative analysis of prostate-specific antigen free survival outcomes for patients with low, intermediate and high risk prostate cancer treatment by radical therapy. Results from the Prostate Cancer Results Study Group. BJU Int 2012;109(Suppl. 1):22–9.
[140] Salembier C, Henry A, Pieters BR, Hoskin P. A history of transurethral resection of the prostate should not be a contra-indication for low-dose-rate ^{125}I prostate brachytherapy: results of a prospective Uro-GEC phase-II trial. J Contemp Brachytherapy 2020;12:1–5.
[141] Budäus L, Bolla M, Bossi A, et al. Functional outcomes and complications following radiation therapy for prostate cancer: a critical analysis of the literature. Eur Urol 2012;61:112–27.
[142] Oh J, Tyldesley S, Pai H, et al. An updated analysis of the survival endpoints of ASCENDE-RT. Int J Radiat Oncol Biol Phys 2023;115:1061–70.

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013

[143] Rodda S, Tyldesley S, Morris WJ, et al. ASCENDE-RT: an analysis of treatment-related morbidity for a randomized trial comparing a low-dose-rate brachytherapy boost with a dose-escalated external beam boost for high- and intermediate-risk prostate cancer. Int J Radiat Oncol Biol Phys 2017;98:286–95.
[144] Vordermark D, Wulf J, Markert K, et al. 3-D conformal treatment of prostate cancer to 74 Gy vs. high-dose-rate brachytherapy boost: a cross-sectional quality-of-life survey. Acta Oncol 2006;45:708–16.
[145] Parry MG, Nossiter J, Sujenthiran A, et al. Impact of high-dose-rate and low-dose-rate brachytherapy boost on toxicity, functional and cancer outcomes in patients receiving external beam radiation therapy for prostate cancer: a national population-based study. Int J Radiat Oncol Biol Phys 2021;109:1219–29.
[146] Ramsay CR, Adewuyi TE, Gray J, et al. Ablative therapy for people with localised prostate cancer: a systematic review and economic evaluation. Health Technol Assess 2015;19:1–490.
[147] Bründl J, Osberghaus V, Zeman F, et al. Oncological long-term outcome after whole-gland high-intensity focused ultrasound for prostate Cancer-21-yr follow-up. Eur Urol Focus 2022;8:134–40.
[148] Dickinson L, Arya M, Afzal N, et al. Medium-term outcomes after whole-gland high-intensity focused ultrasound for the treatment of nonmetastatic prostate cancer from a multicentre registry cohort. Eur Urol 2016;70:668–74.
[149] Lovegrove CE, Peters M, Guillaumier S, et al. Evaluation of functional outcomes after a second focal high-intensity focused ultrasonography (HIFU) procedure in men with primary localized, non-metastatic prostate cancer: results from the HIFU Evaluation and Assessment of Treatment (HEAT) registry. BJU Int 2020;125:853–60.
[150] Hopstaken JS, Bomers JGR, Sedelaar MJP, Valerio M, Fütterer JJ, Rovers MM. An updated systematic review on focal therapy in localized prostate cancer: what has changed over the past 5 years? Eur Urol 2022;81:5–33.
[151] Reddy D, Peters M, Shah TT, et al. Cancer control outcomes following focal therapy using high-intensity focused ultrasound in 1379 men with nonmetastatic prostate cancer: a multi-institute 15-year experience. Eur Urol 2022;81:407–13.
[152] Walz J, Joniau S, Chun FK, et al. Pathological results and rates of treatment failure in high-risk prostate cancer patients after radical prostatectomy. BJU Int 2011;107:765–70.
[153] Spahn M, Joniau S, Gontero P, et al. Outcome predictors of radical prostatectomy in patients with prostate-specific antigen greater than 20 ng/ml: a European multi-institutional study of 712 patients. Eur Urol 2010;58:1–7, discussion 10–1.
[154] Ward JF, Slezak JM, Blute ML, Bergstralh EJ, Zincke H. Radical prostatectomy for clinically advanced (cT3) prostate cancer since the advent of prostate-specific antigen testing: 15-year outcome. BJU Int 2005;95:751–6.
[155] Bolla M, Van Tienhoven G, Warde P, et al. External irradiation with or without long-term androgen suppression for prostate cancer with high metastatic risk: 10-year results of an EORTC randomised study. Lancet Oncol 2010;11:1066–73.
[156] Ploussard G, Staerman F, Pierrevelcin J, et al. Predictive factors of oncologic outcomes in patients who do not achieve undetectable prostate specific antigen after radical prostatectomy. J Urol 2013;190:1750–6.
[157] Wiegel T, Bartkowiak D, Bottke D, et al. Prostate-specific antigen persistence after radical prostatectomy as a predictive factor of clinical relapse-free survival and overall survival: 10-year data of the ARO 96–02 trial. Int J Radiat Oncol Biol Phys 2015;91:288–94.
[158] Spratt DE, Dai DLY, Den RB, et al. Performance of a prostate cancer genomic classifier in predicting metastasis in men with prostate-specific antigen persistence postprostatectomy. Eur Urol 2018;74:107–14.
[159] Preisser F, Chun FKH, Pompe RS, et al. Persistent prostate-specific antigen after radical prostatectomy and its impact on oncologic outcomes. Eur Urol 2019;76:106–14.
[160] Choo R, Danjoux C, Gardner S, et al. Prospective study evaluating postoperative radiotherapy plus 2-year androgen suppression for post-radical prostatectomy patients with pathologic T3 disease and/or positive surgical margins. Int J Radiat Oncol Biol Phys 2009;75:407–12.
[161] Ploussard G, Staerman F, Pierrevelcin J, et al. Clinical outcomes after salvage radiotherapy without androgen deprivation therapy in patients with persistently detectable PSA after radical prostatectomy: results from a national multicentre study. World J Urol 2014;32:1331–8.
[162] Messing EM, Manola J, Yao J, et al. Immediate versus deferred androgen deprivation treatment in patients with node-positive prostate cancer after radical prostatectomy and pelvic lymphadenectomy. Lancet Oncol 2006;7:472–9.
[163] Abdollah F, Dalela D, Sood A, et al. Impact of adjuvant radiotherapy in node-positive prostate cancer patients: the importance of patient selection. Eur Urol 2018;74:253–6.
[164] Gupta M, Patel HD, Schwen ZR, Tran PT, Partin AW. Adjuvant radiation with androgen-deprivation therapy for men with lymph node metastases after radical prostatectomy: identifying men who benefit. BJU Int 2019;123:252–60.
[165] Ventimiglia E, Seisen T, Abdollah F, et al. A systematic review of the role of definitive local treatment in patients with clinically lymph node-positive prostate cancer. Eur Urol Oncol 2019;2:294–301.
[166] Attard G, Murphy L, Clarke NW, et al. Abiraterone acetate and prednisolone with or without enzalutamide for high-risk non-metastatic prostate cancer: a meta-analysis of primary results from two randomised controlled phase 3 trials of the STAMPEDE platform protocol. Lancet 2022;399:447–60.

Please cite this article as: P. Cornford, Roderick C.N. van den Bergh, E. Briers et al., EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent, Eur Urol (2026), https://doi.org/10.1016/j.eururo.2026.04.013