| **Required/ Recommended** | **Element name** | **Values** | **Commentary** | **Implementation notes** |
| --- | --- | --- | --- | --- |
| Recommended | CLINICAL INFORMATION | Not providedORMulti selection value list (select all that apply):•Previous history of prostate cancer (including the Gleason grade and score of previous specimens if known)• Previous biopsy, specify date and where performed• Previous therapy, specify • Other, specify | It is the responsibility of the clinician requesting the pathological examination of a specimen to provide information that will have an impact on the diagnostic process or affect its interpretation. The use of a standard pathology requisition/request form including a checklist of important clinical information is strongly encouraged to help ensure that important clinical data is provided by the clinicians with the specimen. Information about prior biopsies or treatment aids interpretation of the microscopic findings and accurate pathological diagnosis. Radiation and/or endocrine therapy for prostate cancer have a profound effect on the morphology of both the cancer and the benign prostatic tissue. For this reason, information about any previous therapy is important for the accurate assessment of radical prostatectomy specimens. Following irradiation, benign acinar epithelium shows nuclear enlargement and nucleolar prominence,1 while basal cells may show cytological atypia, nuclear enlargement and nuclear smudging.2 There may also be increased stromal fibrosis, which may resemble tumour-induced desmoplasia. These changes may persist for a considerable period, having been reported up to 72 months after treatment, and are more pronounced in patients who have undergone brachytherapy compared to those who have received external beam radiation therapy.2,3 It is important to document any previous radiotherapy to help the pathologist to interpret changes accurately. Radiation may be associated with apparent upgrading of prostate cancer in prostatectomy specimens.4 Likewise, neoadjuvant androgen deprivation therapy (ADT) may induce morphological changes in both prostate cancer and benign tissue. Androgen blockade induces basal cell hyperplasia and cytoplasmic vacuolation in benign prostatic tissue, although this is unlikely to be confused with malignancy.5 More significantly from a diagnostic point of view, neoadjuvant ADT may increase the risk of overlooking acinar adenocarcinoma on low power microscopic examination due to collapse of glandular lumina, cytoplasmic pallor and shrinking of nuclei.6-8 The effect of androgen blockage on prostate cancer is variable and an apparent upgrading of the cancer has been reported in a number of studies.4,5 Hence, it has been suggested that in prostate glands resected following either radiotherapy or androgen deprivation therapy, tumours that show significant treatment effect should not be graded.9 The Gleason grade and score of prostate cancer in any previously submitted specimen should also be provided by the clinician as this allows assessment of any progression of the tumour towards a higher grade/more undifferentiated state, which itself may be of prognostic significance. References1 Cheng L, Cheville JC and Bostwick DG (1999). Diagnosis of prostate cancer in needle biopsies after radiation therapy. Am J Surg Pathol 23(10):1173–1183.2 Magi-Galluzzi C, Sanderson HBS and Epstein JI (2003). Atypia in non-neoplastic prostate glands after radiotherapy for prostate cancer: duration of atypia and relation to type of radiotherapy. Am J Surg Pathol 27:206–212.3 Herr HW and Whitmore WF, Jr (1982). Significance of prostatic biopsies after radiation therapy for carcinoma of the prostate. Prostate 3(4):339–350.4 Grignon DJ and Sakr WA (1995). Histologic effects of radiation therapy and total androgen blockage on prostate cancer. cancer 75:1837–1841.5 Vailancourt L, Ttu B, Fradet Y, Dupont A, Gomez J, Cusan L, Suburu ER, Diamond P, Candas B and Labrie F (1996). Effect of neoadjuvant endocrine therapy (combined androgen blockade) on normal prostate and prostatic carcinoma. A randomized study. Am J Surg Pathol 20(1):86-93.6 Montironi R, Magi-Galluzzi C, Muzzonigro G, Prete E, Polito M and Fabris G (1994). Effects of combination endocrine treatment on normal prostate, prostatic intraepithelial neoplasia, and prostatic adenocarcinoma. J Clin Pathol 47(10):906-913.7 Civantos F, Marcial MA, Banks ER, Ho CK, Speights VO, Drew PA, Murphy WM and Soloway MS (1995). Pathology of androgen deprivation therapy in prostate carcinoma. A comparative study of 173 patients. Cancer 75(7):1634-1641.8 Bostwick DG and Meiers I (2007). Diagnosis of prostatic carcinoma after therapy. Arch Pathol Lab Med 131(3):360-371.9 Epstein JI and Yang XJ (2002). Benign and malignant prostate following treatment. In: Prostate Biopsy Interpretation, Lippincott Williams and Wilkins, Philadelphia, Pennsylvania, 209–225. |   |
| Recommended | PRE-BIOPSY SERUM PSA | Numeric:• \_\_\_ ng/mL | The clinician requesting the pathological examination should provide information on the pre-biopsy serum prostatespecificantigen (PSA) level. The use of a standard pathology requisition/request form including a checklist of important clinical information is strongly encouraged to help ensure that important clinical data is provided by the clinicians with the specimen. Despite criticisms about the utility of PSA-based prostate cancer screening, most prostate cancers are detected in asymptomatic men on the basis of PSA testing. Although PSA levels provide some indication of the likelihood of discovering cancer within a biopsy of the prostate, a diagnosis of malignancy should be based on histological findings and should not be influenced by PSA levels. Pre-biopsy serum PSA is a key parameter in some nomograms widely used to estimate the risk of recurrence postoperatively and guide clinical decision making on adjuvant therapy.1-3 If the patient is on 5-alpha-reductase inhibitor medications, such as finasteride or dutasteride, this should be recorded as it may lower serum PSA levels and affect interpretation of serum PSA values for detecting prostate cancer.4-7References1 Kattan MW, Wheeler TM and Scardino PT (1999). Postoperative nomogram for disease recurrence after radical prostatectomy for prostate cancer. J Clin Oncol 17(5):1499-1507.2 Partin AW, Piantadosi S, Sanda MG, Epstein JI, Marshall FF, Mohler JL, Brendler CB, Walsh PC and Simons JW (1995). Selection of men at high risk for disease recurrence for experimental adjuvant therapy following radical prostatectomy. Urology 45(5):831-838.3 Han M, Partin AW, Zahurak M, Piantadosi S, Epstein JI and Walsh PC (2003). Biochemical (prostate specific antigen) recurrence probablity following radical prostatectomy for clinically localised prostate cancer. J Urol 169:517-523.4 Guess HA, Gormley GJ, Stoner E and Oesterling JE (1996). The effect of finasteride on prostate specific antigen: review of available data. J Urol 155(1):3-9.5 Oesterling JE, Roy J, Agha A, Shown T, Krarup T, Johansen T, Lagerkvist M, Gormley G, Bach M and Waldstreicher J (1997). Biologic variability of prostate-specific antigen and its usefulness as a marker for prostate cancer: effects of finasteride. The Finasteride PSA Study Group. Urology 50(1):13-18.6 Marberger M, Freedland SJ, Andriole GL, Emberton M, Pettaway C, Montorsi F, Teloken C, Rittmaster RS, Somerville MC and Castro R (2012). Usefulness of prostate-specific antigen (PSA) rise as a marker of prostate cancer in men treated with dutasteride: lessons from the REDUCE study. BJU Int 109(8):1162-1169.7 Andriole GL, Humphrey P, Ray P, Gleave ME, Trachtenberg J, Thomas LN, Lazier CB and Rittmaster RS (2004).Effect of the dual 5alpha-reductase inhibitor dutasteride on markers of tumor regression in prostate cancer. J Urol 172(3):915-919. |   |
| Required | SPECIMEN WEIGHT | Numeric:• \_\_\_ g | The prostate gland should be weighed without the seminal vesicles since the seminal vesicles can vary markedly in size; hence, if only a combined weight is recorded, this will introduce error into the measurement of the prostate gland weight and distort comparisons with the radiologically estimated weight. Given this, a working group at the 2009 International Society of Urological Pathology (ISUP) Consensus Conference in Boston recommended that the prostate should beweighed following removal of the seminal vesicles.1References1 Samaratunga H, Montironi R, True L, Epstein JI, Griffiths DF, Humphrey PA, van der Kwast T, Wheeler TM, Srigley JR, Delahunt B, Egevad L and The ISUP Prostate Cancer Group (2011). International Society of Urological Pathology (ISUP) consensus conference on handling and staging of radical prostatectomy specimens. Working group 1: specimen handling. Mod Pathol 24:6-15. | Weight of the prostate gland without the seminal vesicles. |
| Recommended | SPECIMEN DIMENSIONS (of the prostate gland) | Numeric:• length \_\_\_ mm x width \_\_\_ mm x depth \_\_\_mm | Although the shape of the prostate changes somewhat once removed from the pelvis, measurements of specimen size are generally considered part of a standard pathology report. In addition, measurements for apex to base, right to left and anterior to posterior enable comparison with clinical and imaging estimates of volume. |  |
| Required | SEMINAL VESICLES | Single selection value list: • Absent• Present (partially or completely resected) | A record of all organs/tissues received is typically a standard item in gross/macroscopic pathologyreports. |  |
| Required andRecommended | LYMPH NODES | Single selection value list: • Absent• Present (partially or completely resected)  Laterality (Recommended) o Left  o Right  o Bilateral  o Other  | A record of all organs/tissues received is typically a standard item in gross/macroscopic pathology reports. If present, the laterality of the lymph nodes submitted may be recorded as left, right or bilateral. |  |
| Recommended | BLOCK IDENTIFICATION KEY | Text | The origin/designation of all tissue blocks should be recorded and it is preferable to document this information in the final pathology report. This information greatly assists review of the case findings by another pathologist. If this information is not included in the final pathology report, it should be available on the laboratory computer system and relayed to the reviewing pathologist.1 Recording the origin/designation of tissue blocks also facilitates retrieval of blocks, for example for further immunohistochemical or molecular analysis, research studies or clinical trials.References1 ICCR (International Collaboration on Cancer Reporting) (2017). Guidelines for thedevelopment of ICCR datasets. Available from: http://www.iccr-cancer.org/datasets/dataset-development (Accessed 1st March 2017).  | List overleaf or separately with an indication of the nature and origin of all tissue blocks. |
| Required | HISTOLOGICAL TUMOUR TYPE | Multi selection value list (select all that apply):• Adenocarcinoma (Acinar, usual type)• Other, specify | The vast majority (>95%) of prostate cancers are acinar adenocarcinomas.1 Other types of carcinoma are rarer but must be recorded if present, since some variants, such as ductal adenocarcinoma, small cell carcinoma, sarcomatoid carcinoma and urothelial-type adenocarcinoma, have a significantly poorer prognosis.1-7 The tumour type should be assigned in line with the 2016 World Health Organisation (WHO) classification and mixtures of different types should be indicated.1Subtypes of prostate carcinoma are often identified in combination with acinar type and in such cases the tumour type should be classified according to the subtype.**WHO classification of tumours of the prostatea1**Descriptor / ICD-O codes**Epithelial tumours***Glandular neoplasms*Acinar adenocarcinoma 8140/3 Atrophic Pseudohyperplastic Microcystic Foamy gland Mucinous (colloid) 8480/3 Signet ring-like cell 8490/3 Pleomorphic giant cell Sarcomatoid 8572/3Prostatic intraepithelial neoplasia, high-grade 8148/2Intraductal carcinoma 8500/2Ductal adenocarcinoma 8500/3 Cribiform 8201/3 Papillary 8260/3 Solid 8230/3Urothelial carcinoma 8120/3*Squamous neoplasms* Adenosquamous carcinoma 8560/3 Squamous cell carcinoma 8070/3Basal cell carcinoma 8147/3**Neuroendocrine tumours**Adenocarcinoma with neuroendocrine differentiation 8574/3Well-differentiated neuroendocrine tumour 8240/3Small cell neuroendocrine carcinoma 8041/3Large cell neuroendocrine carcinoma 8013/3a The morphology codes are from the International Classification of Diseases for Oncology (ICD-O). Behaviour is coded /0 for benign tumours; /1 for unspecified, borderline, or uncertain behaviour; /2 for carcinoma in situ and grade III intraepithelial neoplasia; and /3 for malignant tumours.© WHO/International Agency for Research on Cancer (IARC). Reproduced with permission Urothelial carcinomas arising in the bladder or urethra are dealt with in separate datasets; however, those rare urothelial carcinomas arising within the prostate are included in this dataset.References1 World Health Organization (2016). World Health Organization (WHO) Classification of tumours. Pathology and genetics of the urinary system and male genital organs. Humphrey PA, Moch H, Reuter VE, Ulbright TM, editors. IARC Press, Lyon, France.2 Christensen WN, Steinberg G, Walsh PC and Epstein JI (1991). Prostatic duct adenocarcinoma. Findings at radical prostatectomy. Cancer 67(8):2118-2124.3 Rubenstein JH, Katin MJ, Mangano MM, Dauphin J, Salenius SA, Dosoretz DE and Blitzer PH (1997). Small cell anaplastic carcinoma of the prostate: seven new cases, review of the literature, and discussion of a therapeutic strategy. Am J Clin Oncol 20:376-380.4 Dundore PA, Cheville JC, Nascimento AG, Farrow GM and Bostwick DG (1995). Carcinosarcoma of the prostate. Report of 21 cases. Cancer 76:1035-1042.5 Hansel DE and Epstein JI (2006). Sarcomatoid carcinoma of the prostate. A study of 42 cases. Am J Surg Pathol 30:1316-1321.6 Osunkoya AO and Epstein JI (2007). Primary mucin-producing urothelial-type adenocarcinoma of prostate: report of 15 cases. Am J Surg Pathol 31:1323-1329.7 Curtis MW, Evans AJ and Srigley J (2005). Mucin-producing urothelial-type adenocarcinoma of prostate: report of two cases of a rare and diagnostically challenging entity. Mod Pathol 18:585-590. | Note that permission to publish the WHO Classification of Tumours may be needed in your implementation. It is advisable to check with the International Agency for Research on Cancer (IARC). |
| Required and Recommended | HISTOLOGICAL GRADE | Numeric: **Gleason score:** Indicate how Gleason score is being reported:\_\_ Largest tumour nodule present\_\_ Highest score tumour (if it is smaller than the largest)\_\_ Composite (global) score Single selection value list: Primary pattern/grade:• 1 • 2 • 3 • 4 • 5Secondary pattern/grade:• 1• 2• 3• 4•5Tertiary pattern/grade (if present and higher than primary and secondary grade): • 3• 4• 5•Not applicableo Indeterminate, specify reason**International Society of Urological Pathology (ISUP) Grade (Grade Group)** Indicate how ISUP grade is being reported:Numeric: \_\_ Largest tumour nodule present\_\_ Highest grade tumour (if it is smaller than the largest)\_\_ Composite (global) grade Single selection value list: •ISUP Grade (Grade Group) 1 (Gleason score ≤6)• ISUP Grade (Grade Group) 2 (Gleason score 3+4=7)• ISUP Grade (Grade Group) 3 (Gleason score 4+3=7)• ISUP Grade (Grade Group) 4 (Gleason score 8)• ISUP Grade (Group Group) 5 (Gleason score 9-10)o Indeterminate, specify reasonRecommended: Numeric: **Percentage Gleason pattern 4/5 (applicable for Gleason scores ≥7)**Indicate how Gleason pattern 4/5 is being reported:\_\_ Largest tumour nodule present\_\_ Highest score tumour (if it is smaller than the largest)\_\_ Carcinoma as a whole \_\_% OR  • Not identified | The Gleason score of radical prostatectomy specimens is usually obtained by adding the two predominant Gleason patterns/grades or doubling the pattern in cases with uniform grade. In the 2005 International Society of Urological Pathology (ISUP) revision it was recommended that this is done for each dominant tumour nodule(s). 1 The rationale was that additional separate tumours of lower grade (e.g. transition zone cancers) would not be expected to mitigate the prognostic impact of the main tumour and, thus, their grades should not be included in the overall Gleason score. Reporting of separate tumours may, however, be difficult in practice, if the prostatectomy specimen is not totally embedded and multifocal tumour nodules may merge into a single large tumour mass. The ISUP 2005 Gleason grading modified the definitions for Gleason scoring of needle biopsies to always include the highest grade, regardless of its amount. It was recommended that minor (<5%) secondary or tertiary patterns of higher grade be included in the Gleason scores of biopsy specimens where there are 2 or 3 different patterns, respectively. The rationale behind this recommendation was that biopsies only sample a minor fraction of the tumour and reporting of small components of higher grade would indicate to the clinician that there might be more extensive involvement of highgrade disease elsewhere in the tumour. The issue of how to deal with a minor (<5%) secondary pattern of higher grade in radical prostatectomy specimens was not specifically addressed in the 2005 consensus conference. However, it was agreed that in radical prostatectomy specimens, where the Gleason score was composed of two most predominant grades, a minor (<5%) tertiary grade should be mentioned separately in the report. The grading practices for radical prostatectomy specimens currently vary and some pathologists would include a tertiary component of Gleason pattern 5 in the Gleason score, at least if more than 5%. At the 2014 ISUP expert consultation meeting on Gleason grading, a grouping of the Gleason scores into 5 grade categories was proposed. Over the past decades Gleason scores below 6 have become less commonly used, especially on needle biopsies. There is also an understanding that Gleason score 7 tumours have a worse outcome if there is a predominant pattern 4 (4+3) than if pattern 3 dominates (3+4). In line with this, a recommendation has been issued to report the percentage of Gleason pattern 4 in cases with a Gleason score of 7 (ISUP grades 2 or 3). Some pathologists also report the percentage of Gleason pattern 4/5. The grade groups and associated definitions are outlined in Table 1. Both the Gleason score and the ISUP grade (Grade group) should always be reported for the sake of clarity. At the 2014 ISUP expert consultation meeting it was not decided how tertiary patterns of higher grade be reported in radical prostatectomy specimens when applying the ISUP grading. By also reporting the Gleason score and tertiary Gleason patterns of higher grade this information is included. **Table 1: ISUP grading system, radical prostatectomy specimens** ISUP grade (Grade group) / Gleason score / Definition Grade 1 / 2-6 / Only individual discrete well-formed glands Grade 2 / 3+4=7 / Predominantly well-formed glands with lesser component (\*) of poorly- formed/fused/cribriform glands Grade 3 / 4+3=7 / Predominantly poorly-formed/fused/cribriform glands with lesser component (\*\*) of well-formed glands Grade 4 / 4+4=8 / Only poorly-formed/fused/cribriform glands  3+5=8 / Predominantly well-formed glands and lesser component (\*) lacking glands 5+3=8 / Predominantly lacking glands and lesser component (\*\*) of well-formed glands Grade 5 / 9-10 / Lack gland formation (or with necrosis) with or without poorly formed/fused/cribriform glands\* A high-grade pattern is included in the grade only if it is at least 5%. If less than 5%, it should be mentioned separately in the report. \*\* The low-grade pattern is included in the grade only if it is at least 5%.References 1 Epstein JI, Allsbrook WCJ, Amin MB and Egevad LL (2005). The 2005 International Society of Urological Pathology (ISUP) Consensus Conference on Gleason Grading of Prostatic Carcinoma. Am J Surg Pathol 29(9):1228–1242. |  |
| Recommended | INTRAGLANDULAR EXTENT | Single selection value list: • Tumour identified  Numeric:  \_\_%  \_\_ mm  \_\_mL(cc)  \_\_Other units (specify)• No tumour identified | Some measurement of the size or extent of the tumour is typically given in histopathology reports for most sites and this parameter forms part of the generic International Collaboration on Cancer Reporting (ICCR) dataset for all tumour types. However in prostate, while cancer volume is a prognostic factor on univariate analysis, it is significantly correlated with other clinicopathological features, including Gleason score, extraprostatic extension (EPE), surgical margin status andpathological TNM stage, and the majority of studies have not demonstrated independent prognostic significance on multivariate analysis.1-6 Hence, the ICCR expert panel regarded this factor as a recommended (non-core) rather than a required item. The irregular distribution and often multifocal nature of prostate cancer makes accurate calculationof tumour volume challenging for the pathologist in routine diagnostic practice; a situation where precise methods, such as computerised planimetry or image analysis, are too time and labour intensive to be practical. However, there was consensus at the 2009 International Society of Urological Pathology (ISUP) Conference that some quantitative measure of the extent of the tumour in a prostatectomy specimen should be recorded. This can be done either as a visual estimate ofintraglandular percentage of cancer7,8 or by measuring the maximum size of dominant tumour nodule.9,10 The latter has been shown to correlate with tumour volume and has also been recommended as a readily assessed surrogate for tumour volume in some studies and protocols. 6,9,10References1 Wheeler TM, Dillioglugil O, Kattan MW, Arakawa A, Soh S, Suyama K, Ohori M and Scardino PT (1998). Clinical and pathological significance of the level and extent of capsular invasion in clinical stage T1-2 prostate cancer. Hum Pathol 29(8):856–862.2 Epstein JI, Carmichael M, Partin AW and Walsh PC (1993). Is tumor volume an independent predictor of progression following radical prostatectomy? A multivariate analysis of 185 clinical stage B adenocarcinomas of the prostate with 5 years of followup. J Urol 149(6):1478–1481.3 Kikuchi E, Scardino PT, Wheeler TM, Slawin KM and Ohori M (2004). Is tumor volume an independent prognostic factor in clinically localized prostate cancer? J Urol 172:508-511.4 Van Oort IM, Witjes JA, Kok DE, Kiemeney LALM and Hulsbergen-vandeKaa CA (2008). Maximum tumor diameter is not an independent prognostic factor in high-risk localised prostate cancer. World J Urol 26:237-241.5 Wolters T, Roobol MJ, van Leeuwen PJ, van den Bergh RC, Hoedemaeker RF, van Leenders GJ, Schröder FH and van der Kwast TH (2010). Should pathologist routinely report prostate tumor volume? The prognostic value of tumor volume in prostate cancer. Eur Urol 57(5):735-920.6 Dvorak T, Chen MH, Renshaw AA, Loffredo M, Richie JP and D’Amico AV (2005). Maximal tumor diameter and the risk of PSA failure in men with specimen-confined prostate cancer. Urology 66:1024-1028.7 Epstein JI, Oesterling JE and Walsh PC (1988). Tumor volume versus percentage of specimen involved by tumor correlated with progression in stage A prostatic cancer. J Urol 139:980- 984.8 Partin AW, Epstein JI, Cho KR, Gittelsohn AM and Walsh PC (1989). Morphometric measurement of tumor volume and per cent of gland involvement as predictors of pathological stage in clinical stage B prostate cancer. J Urol 141:341-345.9 Wise AM, Stamey TA, McNeal JE and Clayton JL (2002). Morphologic and clinical significance of multifocal prostate cancers in radical prostatectomy specimens. Urology 60(2):264–269.10 Renshaw AA, Richie JP, Loughlin KR, Jiroutek M, Chung A and D'Amico AV (1999). Maximum diameter of prostatic carcinoma is a simple, inexpensive, and independent predictor of prostate-specific antigen failure in radical prostatectomy specimens. Validation in a cohort of 434 patients. Am J Clin Pathol 111(5):641–644. |  |
| Required and Recommended | EXTRAPROSTATIC EXTENSION | Single selection value list: • Not identified • Present  o Recommended:\_\_ Location(s) o Required: Extent.  Single selection value:  • Focal • Non-focal • Indeterminate | Extraprostatic extension (EPE), defined as the extension of tumour beyond the confines of the gland into the periprostatic soft tissue, is a required (core) element of the generic International Collaboration on Cancer Reporting (ICCR) dataset as it is a significant predictor of recurrence in node negative patients.1,2 EPE replaced earlier, less clearly defined terms, such capsular penetration, perforation or invasion, following a 1996 Consensus Conference. 3 The assessment of EPE can bedifficult, as the prostate is not surrounded by a discrete, well defined fibrous capsule, 4 but rather by a band of concentrically placed fibromuscular tissue that is an inseparable component of the prostatic stroma.5 EPE can be recognised in several different settings: (1) the presence of neoplastic glands abutting on or within periprostatic fat or beyond the adjacent fat plane in situations where no fat is present in the immediate area of interest (most useful at the lateral, posterolateral and posterior aspects of the prostate); (2) neoplastic glands surrounding nerves in the neurovascularbundle (posterolaterally) beyond the boundary of the normal prostatic glandular tissue; (3) the presence of a nodular extension of tumour bulging beyond the periphery of the prostate or beyond the compressed fibromuscular prostatic stroma at the outer edge of the gland—since there is often a desmoplastic reaction in the vicinity of EPE and the neoplastic extraprostatic glands may then be seen in fibrous tissue, rather than in fat.5,6 Extraprostatic tumour in fibrous tissue is best identified initially at low power magnification, but should be then confirmed by high power magnificationexamination verifying that the neoplastic glands are in stroma that is fibrous and beyond the condensed smooth muscle of the prostate.2,6 The presence of cancer within fibrous stroma that is in the same tissue plane as adipose tissue on either side is a helpful indicator of EPE. The boundary of the prostate gland cannot be readily identified anteriorly and at the base or apex of the prostate. Moreover, at the apex benign glands are frequently admixed with skeletal muscle andthe presence of neoplastic glands within skeletal muscle does not necessarily constitute EPE. Hence, in this region it is more important to accurately assess the completeness of surgical resection. Similarly, the assessment of EPE at the anterior aspect of the prostate may be difficult as the prostatic stroma blends in with extraprostatic fibromuscular tissue, but in this location EPE can be diagnosed (in the manner described in the previous paragraph) when the carcinoma appears to bulge beyond the boundary of the normal prostate gland.6,7 Extent of EPE Categorisation of the extent of EPE as focal or non-focal (also referred to as ‘extensive’ or ‘established’) is a required (core) item in the ICCR dataset. Focal EPE was originally defined no more than ‘a few’ neoplastic glands just outside the prostate, then subsequently, in a more semiquantified manner, as extraprostatic glands which occupy no more than one high power field in no more than two sections, with extensive EPE representing anything more than this.2 More rigorous quantification of the extent of EPE by measuring the maximum distance that the tumour bulges beyond the outer edge of the fibromuscular prostatic stroma radially has been proposed by some investigators.8 However, the practical value of such parameters is limited by the difficulty in precisely defining the outer limit of the prostate gland, especially when the tumour is associated with adesmoplastic reaction. The identification of any EPE is important, as both focal and non-focal EPE are associated with a significantly higher risk of recurrence at both 5 and 10 years.1,2 Following radical prostatectomy, the progression-free probability for node negative patients with uninvolved seminal vesicles at 10 years for organ confined disease is 85–89%, falling to 67–69% for focal EPE and to 36– 58% for extensive EPE. 1,2 Location of EPE Since it was considered a generic element forming part of a comprehensive pathology report, the location of any EPE present has been included in the recommended (non-core) dataset, despite the lack of published evidence for its influence on staging, prognosis or treatment.6 It provides potentially useful information to the urologist, enabling correlation with clinical findings and anypre-operative imaging studies performed.References1 Epstein JI, Partin AW, Sauvageot J and Walsh PC (1996). Prediction of progression following radical prostatectomy. A multivariate analysis of 721 men with long-term follow-up. Am J Surg Pathol 20(3):286–292.2 Wheeler TM, Dillioglugil O, Kattan MW, Arakawa A, Soh S, Suyama K, Ohori M and Scardino PT (1998). Clinical and pathological significance of the level and extent of capsular invasion in clinical stage T1-2 prostate cancer. Hum Pathol 29(8):856–862.3 Sakr WA, Wheeler TM, Blute M, Bodo M, Calle-Rodrigue R, Henson DE, Mostofi FK, Seiffert J, Wojno K and Zincke H (1996). Staging and reporting of prostate cancer-sampling of the radical prostatectomy specimen. Cancer 78(2):366–368.4 Ayala AG, Ro JY, Babaian R, Troncoso P and Grignon DJ (1989). The prostatic capsule: does it exist? Its importance in the staging and treatment of prostatic carcinoma. Am J Surg Pathol 13:21-27.5 Chuang AY and Epstein JI (2008). Positive surgical margins in areas of capsular incision in otherwise organ-confined disease at radical prostatectomy: histologic features and pitfalls. Am J Surg Pathol 32(8):1201–1206.6 Magi-Galluzzi C, Evans AJ, Delahunt B, Epstein JI, Griffiths DF, van der Kwast TH, Montironi R, Wheeler TM, Srigley JR, Egevad LL, Humphrey PA and ISUP Prostate Cancer Group (2011). International Society of Urological Pathology (ISUP) consensus conference on handling and staging of radical prostatectomy specimens. Working group 3: extraprostatic extension, lymphovascular invasion and locally advanced disease. Mod Pathol 24:26-38.7 Epstein JI, Amin M, Boccon-Gibod L, Egevad L, Humphrey PA, Mikuz G, Newling D, Nilsson S, Sakr W, Srigley JR, Wheeler TM and Montironi R (2005). Prognostic factors and reporting of prostate carcinoma in radical prostatectomy and pelvic lymphadenectomy specimens. Scand J Urol Nephrol Suppl 216:34–63.8 Sung MT, Lin H, Koch MO, Davidson DD and Cheng L (2007). Radial distance of extraprostatic extension measured by ocular micrometer is an independent predictor of prostate specific antigen recurrence: a new protocol for the substaging of pT3a prostate cancer. Am J Surg Pathol 31:311-318. |  |
| Required | SEMINAL VESICLE INVASION | Single selection value list: • Not identified • Present • Not applicable (Refers to rare cases where seminal vesicles are not included in specimen.) | The expert panel included seminal vesicle invasion (SVI) as a required (core) element of the International Collaboration on Cancer Reporting (ICCR) dataset as SVI is a well-established, independent, adverse prognostic factor1-3 and an integral component of the commonly used nomograms and tables that predict risk of post prostatectomy cancer recurrence.4-6 The finding of SVI at the time of radical prostatectomy is associated with a significantly increased risk of prostatespecific antigen (PSA) recurrence2,3,7 and the presence of SVI and a positive surgical margin may also influence the response to adjuvant radiotherapy.8,9 Bilaterality and extent of extraprostatic SVI are not independently predictive of prognosis and were not included as required or recommended items in the ICCR dataset.10 Different definitions of seminal vesicle invasion have been used over the years complicating comparison of the published survival analyses.8,11 Older definitions including involvement of the adipose tissue or adventitia around the seminal vesicle are problematic with regard to distinction from EPE; while in other studies a distinction between intraprostatic and extraprostatic seminal vesicle invasion has not always been made, impeding comparisons between series.12,13 At the 2009 Society of Urological Pathology (ISUP) meeting, the proposal that SVI should be defined as carcinomatous invasion of the muscular wall of the seminal vesicle exterior to the prostate was endorsed.11 Only extraprostatic seminal vesicle is included in this definition of SVI, since it is difficult differentiating between intraprostatic seminal vesicle and ejaculatory duct invasion as these structures merge without a clear histological cut off.14 It was concluded that older definitions that include invasion of the adipose tissue around the seminal vesicle are imprecise and should be discarded.8,11 References 1 Epstein JI, Amin M, Boccon-Gibod L, Egevad L, Humphrey PA, Mikuz G, Newling D, Nilsson S, Sakr W, Srigley JR, Wheeler TM and Montironi R (2005). 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| Required | URINARY BLADDER NECK INVASION | Single selection value list: • Not identified • Present • Not applicable (Refers to rare cases where seminal vesicles are not included in specimen) | Microscopically, invasion of the urinary bladder neck can be identified when there are neoplastic glands within the thick smooth muscle bundles of the bladder neck in sections from the base of the prostate in the absence of associated benign prostatic glandular tissue.1 Microscopic bladder neck involvement is a significant predictor of prostate-specific antigen (PSA)-recurrence in univariate analysis, although not in multivariate modelling in most studies.2-4 Neoplastic glands intermixed with benign prostatic glands at the bladder neck margin is equivalent to capsular incision rather than true bladder neck invasion.2,5,6 In the 7th and 8th Editions of the American Joint Committee on Cancer (AJCC)/Union for International Cancer Control (UICC) Cancer Staging Manual microscopic bladder neck invasion is classified as stage pT3a disease since it has a similar biochemical recurrence free survival and cancer specific survival to patients with seminal vesicle invasion or extraprostatic extension. 1,7-10 References 1 Pierorazio PM, Epstein JI, Humphreys E, Han M, Walsh PC and Partin AW (2010). The significance of a positive bladder neck margin after radical prostatectomy: the American Joint Committee on Cancer Pathological Stage T4 designation is not warranted. J Urol 183:151-157.2 Zhou M, Reuther AM, Levin HS, Falzarano SM, Kodjoe E, Myles J, Klein E and Magi-Galluzzi C (2009). Microscopic bladder neck involvement by prostate carcinoma in radical prostatectomy specimens is not a significant independent prognostic factor. Mod Pathol 22(3):385–392. 3 Dash A, Sanda MG, Yu M, Taylor JM, Fecko A and Rubin MA (2002). Prostate cancer involving the bladder neck: recurrence-free survival and implications for AJCC staging modification. American Joint Committee on Cancer. Urology 60(2):276–280. 4 Yossepowitch O, Engelstein D, Konichezky M, Sella A, Livne PM and Baniel J (2000). Bladder neck involvement at radical prostatectomy: positive margins or advanced T4 disease? Urology 56(3):448–452. 5 Poulos CK, Koch MO, Eble JN, Daggy JK and Cheng L (2004). Bladder neck invasion is an independent predictor of prostate-specific antigen recurrence. Cancer 101(7):1563–1568. 6 Rodriguez-Covarrubias F, Larre S, Dahan M, De La Taille A, Allory Y, Yiou R, Vordos D, Hoznek A, Abbou CC and Salomon L (2009). Prognostic significance of microscopic bladder neck invasion in prostate cancer. BJU Int 103(6):758–761. 7 Edge SE, Byrd DR, Compton CC, Fritz AG, Greene FL and Trotti A (eds) (2010). AJCC Cancer Staging Manual 7th ed., New York, NY.: Springer.8 Amin M.B., Edge, S., Greene, F.L., Byrd, D.R., Brookland, R.K., Washington, M.K., Gershenwald, J.E., Compton, C.C., Hess, K.R., Sullivan, D.C., Jessup, J.M., Brierley, J.D., Gaspar, L.E., Schilsky, R.L., Balch, C.M., Winchester, D.P., Asare, E.A., Madera, M., Gress, D.M., Meyer, L.R. (Eds.) (2017). AJCC Cancer Staging Manual 8th ed. Springer, New York.9 Brierley JD, Gospodarowicz MK, Wittekind C (eds) (2016). UICC TNM Classification of Malignant Tumours, 8th Edition. Wiley-Blackwell. 10 International Union against Cancer (UICC) (2009). TNM Classification of Malignant Tumours (7th edition). Sobin L, Gospodarowicz M and Wittekind C (Eds). Wiley-Blackwell, Chichester, UK and Hoboken, New Jersey. |  |
| Recommended | INTRADUCTAL CARCINOMA OF PROSTATE | Single selection value list: • Not identified • Present  | Intraductal carcinoma of the prostate (IDC-P) is found in approximately 17% of radical prostatectomy specimens and is usually associated with invasive prostate cancer.1 However, occasionally isolated IDC-P is found without invasive carcinoma; this latter situation is very rare and beyond the scope of this dataset. IDC-P has been well characterised at the histological and molecular levels over the past decade and its clinical significance is now also better understood.2 The diagnosis of IDC-P is based on morphology and the key criteria include: 1) large calibre glands that are more than twice the diameter of normal non-neoplastic peripheral glands; 2) preserved (at least focally) basal cells identified on H&E staining (or with basal cell markers, such as p63, keratin 34βE12 and keratin 5/6, however, the use of immunohistochemistry to identify basal cells is optional, rather than mandatory, for the diagnosis of IDC-P); 3) significant nuclear atypia including enlargement and anisonucleosis; and 4) comedonecrosis, which is often but not always present.3,4 It is important to distinguish IDC-P from high grade prostatic intraepithelial neoplasia (HGPIN): compared to IDC-P, HGPIN has less architectural and cytological atypia, and cribriform HGPIN is rare. When present in combination with invasive carcinoma in radical prostatectomy specimens, IDC-P is strongly associated with high volume, high grade and stage (extraprostatic extension (EPE) or seminal vesicle invasion (SVI) positive)) carcinoma.5 Moreover the presence of IDC-P is independently associated with biochemical recurrence, regional lymph node metastasis and cancer specific survival.1,6,7 Hence, in radical prostatectomy specimens, the presence of IDC-P in association with invasive carcinoma should be recorded. There was a strong consensus (82%) at the recent International Society of Urological Pathology (ISUP) consensus meeting (Chicago 2014) that IDC-P should not be assigned an ISUP or Gleason grade.8 It is also unnecessary to measure the extent of the IDC-P. References1 Miyai K, Divatia MK, Shen SS, Miles BJ, Ayala AG and Ro JY (2014). Heterogeneous clinicopathological features of intraductal carcinoma of the prostate: a comparison between "precursor-like" and "regular type" lesions. Int J Clin Exp Pathol 7(5):2518-2526.2 Zhou M (2013). Intraductal carcinoma of the prostate: the whole story. Pathology 45(6):533- 539. 3 Cohen RJ, Wheeler TM, Bonkhoff H and Rubin MA (2007). A proposal on the identification, histologic reporting, and implications of intraductal prostatic carcinoma. Arch Pathol Lab Med 131(7):1103-1109.4 Guo CC and Epstein JI (2006). Intraductal carcinoma of the prostate on needle biopsy: Histologic features and clinical significance. Mod Pathol 19(12):1528-1535. 5 McNeal JE and Yemoto CE (1996). Spread of adenocarcinoma within prostatic ducts and acini. Morphologic and clinical correlations. Am J Surg Pathol 20(7):802-814. 6 Kimura K, Tsuzuki T, Kato M, Saito AM, Sassa N, Ishida R, Hirabayashi H, Yoshino Y, Hattori R and Gotoh M (2014). Prognostic value of intraductal carcinoma of the prostate in radical prostatectomy specimens. Prostate 74(6):680-687. 7 Kryvenko ON, Gupta NS, Virani N, Schultz D, Gomez J, Amin A, Lane Z and Epstein JI (2013). Gleason score 7 adenocarcinoma of the prostate with lymph node metastases: analysis of 184 radical prostatectomy specimens. Arch Pathol Lab Med 137(5):610-617. 8 Epstein JI, Egevad L, Amin MB, Delahunt B, Srigley JR and Humphrey PA (2015). The 2014 International Society of Urological Pathology (ISUP) Consensus Conference on Gleason Grading of Prostatic Carcinoma: Definition of Grading Patterns and Proposal for a New Grading System. Am J Surg Pathol 40(2): 244-52. |  |
| Required and Recommended | MARGIN STATUS | Single selection value list: • Not involved • Involved  o Location of positive margin(s)• Indeterminate Recommended: **Type of margin positivity**• IndeterminateORMulti selection value list (select all that apply):• Extraprostatic (EPE)• Intraprostatic (capsular incision)Single selection value list: Recommended: Single selection value list: **Extent of margin positivity.** If more than 1 positive margin, the extent should reflect the cumulative length.• <3 mm linear extent• ≥3 mm linear extentRecommended: Single selection value list: **Gleason pattern of tumour present at positive margin**. If more than 1 pattern at margin select highest.• Gleason pattern 3• Gleason pattern 4/5 | A positive surgical margin (PSM) significantly reduces the likelihood of progression-free survival, including prostate-specific antigen (PSA) recurrence-free survival, local recurrence-free survival and development of metastases after radical prostatectomy in multivariate analysis.1-6 Moreover, positive margins are associated with a 2.6-fold increased risk of prostate cancer specific mortality.7 Careful inking of the outer surface of the radical prostatectomy specimen before macroscopic dissection (grossing) greatly facilitates the determination of margin status. A PSM can then be defined as cancer extending to the inked surface of the specimen, representing a site where the urologist has cut through cancer. 1,8 PSMs are reported in between 10–48% of patients treated by radical prostatectomy for both organ confined and non-organ confined prostate cancer with the rates in the lower range typically found in more modern cohorts.6,9-11 The presence of prostate carcinoma close to, but not touching the inked margin should not be labelled as a PSM as this finding has been shown to have little, if any, prognostic significance.12-14 Close surgical margins are most commonly seen posterolaterally in cases where neurovascular bundle preservation leaves virtually no extraprostatic tissue. Studies on such nerve sparing cases have shown that additional tissue removed from these sites did not contain any carcinoma and a close margin was not associated with a worse prognosis.12,14 Stating the location of the PSM is useful information for the urologist who can then modify future operations to avoid iatrogenic margin positivity and increase the likelihood of curative surgery. The site of the PSM and the number of positive margins have been shown to influence biochemical recurrence and risk of progression. For instance, a margin involving the bladder neck or the posterolateral surface of the prostate has a more significant adverse impact on prognosis than an involved apical or anterior margin.11,15 **Type of margin positivity** Intraprostatic margin involvement or capsular incision (CI) occurs when the urologist inadvertently develops the resection margin within the plane of the prostate rather than outside the capsule. CI with a positive surgical margin is diagnosed when malignant glands are cut across adjacent to benign prostatic glands.16 In these cases, the edge of the prostate in this region is left in the patient. Data on the prognostic significance of CI vary among studies.17-19 According to the largest series published, a significantly higher recurrence rate is found in patients with CI/intraprostatic margin involvement than in patients with organ confined disease with negative margins, or focal extraprostatic extension (EPE) with negative margins, although CI has a significantly better outcome than that associated with non-focal EPE and positive margins.20 Margin involvement associated with EPE is diagnosed when malignant glands in extraprostatic tissue are transected by the resection margin. This can be difficult to distinguish from capsular incision in some cases, particularly posteriorly and posterolaterally if there is a desmoplastic reaction. Cancer extending to a margin which is beyond the normal contour of the prostate gland, or beyond the compressed fibromuscular prostatic stroma at the outer edge of the prostate, can be diagnosed as a positive surgical margin with EPE, similarly to margin involvement when there is cancer in adipose tissue.18 At the apex, the histological boundaries of the prostate gland can be difficult to define and again EPE with a positive margin can be difficult to differentiate from CI/intraprostatic margin involvement. Hence, if carcinoma extends to an inked margin at the apex where benign glands are not transected, this is considered a positive margin in an area of EPE by some authors.1,18 In contrast, other authors, and the majority of survey participants at the 2009 International Society of Urological Pathology (ISUP) Consensus Conference, believe there is no reliable method to diagnose EPE in sections from the prostatic apex. 21 **Extent (total) of margin involvement** Although a positive surgical marginal (PSM) has a significant adverse impact on the overall likelihood of progression-free survival, in most published series only about a third of individual patients with a PSM will experience biochemical recurrence.2,3,9,22 The expert panel considered that there is sufficient evidence to include measurement of the length of margin involved by carcinoma as an element in the International on Cancer Reporting (ICCR) dataset.12,14,20,22-26 In particular, the 5 year prostate-specific antigen (PSA) recurrence risk appears to be significantly greater when the length of the involved margin is 3 mm or more, (53% versus 14%).20,23,27-29 However, in one series, Cao et al25 found that the linear length of a positive margin was an independent prognostic factor for organ confined tumours only, i.e. pT2 not pT3, while, another investigation found that the impact of a positive surgical margin after radical prostatectomy was greater in intermediate and high risk groups (based on Gleason score and pre-biopsy PSA) than in low risk patients.5 Further studies of such factors potentially affecting the impact of PSMs are required before there is sufficient evidence justifying their inclusion as required (core) data elements. The optimal method of assessing the extent of margin involvement when multiple positive margins are present is currently uncertain, but, until more evidence is available, it is suggested that extent is measured as the linear cumulative length of all positive margins. 30**Gleason pattern at the margin** Four recently published papers have found that Gleason pattern/grade or score of the tumour at the positive surgical margin is an independent predictor of biochemical recurrence and may aid optimal selection of patients for adjuvant therapy.22,31-33 In one of these studies patients with Gleason pattern 4 or 5 carcinoma (Gleason score 3+4, 4+3, 4+4 or 4+5) at a PSM had double the risk of PSA relapse compared to those with only Gleason grade 3 (score 3+3) at the margin. Moreover, men with Gleason pattern/grade 3 at the PSM had a similar 5-year biochemical relapse-free survival rate to those with negative margins.22 Another study, restricted to men with dominant nodule Gleason score 7 and non-focal EPE, also found that the grade of cancer at the site of a PSM was associated with biochemical recurrence.31 The largest series, including 405 cases with a PSM, confirmed that a lower Gleason score at the margin was independently associated with a decreased risk of early biochemical recurrence.33 In each of the published studies, the potential problem of cautery/thermal artefact was considered - each group noted that in slides where the cancer at the margin was distorted by cautery/thermal or crush artifact and could not be reliably assessed, the margin pattern, or score, was designated as that of the closest, well preserved carcinoma in direct continuity with the distorted neoplastic glands.22,31-33 Limiting assessment to only the highest pattern present at the PSM may simplify measurement of this parameter, however, it should be noted that in most of the published studies Gleason score could be reported.31-33 In the event there are multiple positive margins with differently scored cancers present, the highest pattern or score should be recorded.References 1 Epstein JI, Amin M, Boccon-Gibod L, Egevad L, Humphrey PA, Mikuz G, Newling D, Nilsson S, Sakr W, Srigley JR, Wheeler TM and Montironi R (2005). 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| Recommended | LYMPHOVASCULAR INVASION | Single selection value list: • Not identified • Present • Indeterminate | Lymphovascular invasion (LVI) is defined as the unequivocal presence of tumour cells within endothelial-lined spaces with no or only thin underlying muscular walls.1,2 Lymphatic and venous invasion should be assessed together due to the difficulties in distinguishing between the two by routine light microscopy and it is important that artefacts, such as retraction or mechanical displacement of tumour cells into vessels, are excluded. Immunohistochemistry for endothelial markers, e.g. CD31, CD34 or D2-40, may aid in the assessment of equivocal cases but is not recommended for routine use at present. LVI has been reported to be associated with decreased time to biochemical progression, distant metastases and overall survival after radical prostatectomy.1-6 Multivariate analysis, controlling for other pathological variables known to affect clinical outcome, showed that LVI is an independent predictor of disease recurrence in some studies.1,2,4,6,7 However, the independent prognostic value of LVI is uncertain as definitions of LVI have varied between studies and most included a substantial number of patients with lymph node metastases or seminal vesicle invasion, failing to stratify patients into clinical meaningful categories. Further well designed studies with standardised definitions are necessary to confirm the independent prognostic significance of LVI. References 1 Herman CM, Wilcox GE, Kattan MW, Scardino PT and Wheeler TM (2000). Lymphovascular invasion as a predictor of disease progression in prostate cancer. Am J Surg Pathol 24(6):859-863.2 Cheng L, Jones TD, Lin H, Eble JN, Zeng G, Carr MD and Koch MO (2005). Lymphovascular invasion is an independent prognostic factor in prostatic adenocarcinoma. J Urol 174(6):2181–2185.3 van den Ouden D, Hop WCJ, Kranse R and Schroder FH (1997). Tumour control according to pathological variables in patients treated by radical prostatectomy for clinically localized carcinoma of the prostate. Brit J Urol 79:203-211. 4 Van den Ouden D, Kranse R, Hop WC, van der Kwast TH and Schroder FH (1998). Microvascular invasion in prostate cancer: prognostic significance in patients treated by radical prostatectomy for clinically localized carcinoma. Urol Int 60:17-24. 5 Loeb S, Roehl KA, Yu X, Antenor JA, Han M, Gashti SN, Yang XJ and Catalona WJ (2006). Lymphovascular invasion in radical prostatectomy specimens: prediction of adverse prognostic features and biochemical progression. Urology 68:99-103.6 Yee DS, Shariat SF, Lowrance WT, Maschino AC, Savage CJ, Cronin AM, Scardino PT and Eastham JA (2011). Prognostic significance of lymphovascular invasion in radical prostatectomy specimens. BJU Int 108:502-507. 7 May M, Kaufmann O, Hammermann F and Siegsmund M (2007). Prognostic impact of lymphovascular invasion in radical prostatectomy specimens. BJU Int 99:539-544. |  |
| Required and Recommended | LYMPH NODE STATUS | Numeric: \_\_ Number of lymph nodes examined\_\_ Number of involved nodes  Recommended: Laterality  o Left  o Right  o Bilateral  o Other Recommended: Numeric: Maximum dimension of largest deposit \_\_ mm  | A record of all organs/tissues received is typically a standard item in gross/macroscopic pathology reports. If present, the laterality of the lymph nodes submitted may be recorded as left, right or bilateral. |  |
| Required | PATHOLOGICAL STAGING (AJCC TNM 8th edition) TNM descriptors | Choose if applicable:• m - multiple primary tumours • r - recurrent • y - post neoadjuvant therapy | The pathological primary tumour (T), regional lymph node (N) and distant metastasis (M) categories are considered as generic required (core) elements for all International Collaboration on Cancer Reporting (ICCR) cancer datasets. Staging data should be assessed according to the most recent edition of the American Joint Committee on Cancer (AJCC) Staging Manual (8th Edition). 1,2 However, it should be noted that the implementation of AJCC TNM 8th edition has been deferred until January 2018 in some jurisdictions. Union for International Cancer Control (UICC) or AJCC 7 th editions may be used in the interim. If TNM 7th edition is used pT2 subcategorization should be considered optional in line with ISUP recommendations as it lacks additional prognostic significance.3 It should also be noted that that the UICC 8th Edition Stage Grouping differs from the AJCC Prognostic Stage Groups.2,4 The reference document TNM Supplement: A commentary on uniform use, 4th Edition (C. Wittekind editor) may be of assistance when staging.5 References 1 Edge SE, Byrd DR, Compton CC, Fritz AG, Greene FL and Trotti A (eds) (2010). AJCC Cancer Staging Manual 7th ed., New York, NY.: Springer. 2 Amin M.B., Edge, S., Greene, F.L., Byrd, D.R., Brookland, R.K., Washington, M.K., Gershenwald, J.E., Compton, C.C., Hess, K.R., Sullivan, D.C., Jessup, J.M., Brierley, J.D., Gaspar, L.E., Schilsky, R.L., Balch, C.M., Winchester, D.P., Asare, E.A., Madera, M., Gress, D.M., Meyer, L.R. (Eds.) (2017). AJCC Cancer Staging Manual 8th ed. Springer, New York.3 Van der Kwast TH, Amin MB, Billis A, Epstein JI, Griffiths D, Humphrey PA, Montironi R, Wheeler TM, Srigley JR, Egevad L, Delahunt B and ISUP Prostate Cancer Group (2011). International Society of Urological Pathology (ISUP) consensus conference on handling and staging of radical prostatectomy specimens. Working group 2: T2 substaging and prostate cancer volume. Mod Pathol 24:16-25. 4 Brierley JD, Gospodarowicz MK, Wittekind C (eds) (2016). UICC TNM Classification of Malignant Tumours, 8th Edition. Wiley-Blackwell. 5 Wittekind C (ed) (2012). TNM Supplement : A Commentary on Uniform Use, The Union for International Cancer Control (UICC), Wiley-Blackwell. | Note that permission to publish the TNM cancer staging tables may be needed in your implementation. It is advisable to check.Used with the permission of the American College of Surgeons, Chicago, Illinois. The original source for this information is the AJCC Cancer Staging Manual, Eighth Edition (2016) published by Springer Science+Business Media. |
| Required  | Primary tumour (pT) | Single selection value list: • TX Primary tumour cannot be assessed• T0 No evidence of primary tumour• T2 Organ confined• T3 Extraprostatic extension• T3a Extracapsular extension (unilateral or bilateral) or microscopic invasion of bladder neck• T3b Tumour invades seminal vesicle(s)• T4 Tumour is fixed or invades adjacent structures other than seminal vesicles such as external sphincter, rectum, levator muscles, and/or pelvic wall |  |  |
| Required | Regional lymph nodes (pN) | Single selection value list: • NX Regional nodes were not assessed• N0 No positive regional nodes• N1 Metastases in regional node(s) |  |  |
| Required and Recommended | Distant metastasis (pM) | Single selection value list: • Not applicable• M1 Distant metastasis Recommended:• M1a Non-regional lymph node(s)• M1b Bone(s)• M1c Other site(s) with or without bone disease |  | When more than 1 site of metastasis is present, the most advanced category is used. pM1c is the most advanced category. |