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Updated Nomogram to Predict Pathologic Stage of Prostate Cancer Given Prostate-Specific Antigen Level, Clinical Stage, and Biopsy Gleason Score (Partin Tables) Based on Cases from 2000 to 2005

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Abstract

OBJECTIVES—To update the 2001 "Partin tables" with a contemporary patient cohort and revised variable categorization, correcting for the effects of stage migration.

METHODS—We analyzed 5730 men treated with prostatectomy (without neoadjuvant therapy) between 2000 and 2005 at the Johns Hopkins Hospital. Average age was 57 years. Multivariable logistic regression was used to estimate the probability of organ-confined disease, extraprostatic extension, seminal vesicle involvement, or lymph node involvement. Predictor variables included preoperative prostate-specific antigen (PSA) level (0 to 2.5, 2.6 to 4.0, 4.1 to 6.0, 6.1 to 10.0, and greater than 10.0 ng/mL), clinical stage (T1c, T2a, and T2b/T2c), and biopsy Gleason score (5 to 6, 3 + 4 = 7, 4 + 3 = 7, or 8 to 10). Bootstrap resampling was used to generate 95% confidence intervals for predicted probabilities.

RESULTS—Seventy-seven percent of patients had T1c, 76% had Gleason score 5 to 6, 80% had a PSA level between 2.5 and 10.0 ng/mL, and 73% had organ-confined disease. Nomograms were developed for the predicted probability of pathologically organ-confined disease, extraprostatic extension, seminal vesicle invasion, or lymph node involvement. The risk of non-organ-confined disease increased with increases in any individual prognostic factor. The dramatic decrease in clinical stage T2c compared with the patient series used in the previous models resulted in T2b and T2c being combined as a single predictor in the nomogram.

CONCLUSIONS—These updated "Partin tables" were generated to reflect trends in presentation and pathologic stage for men diagnosed with clinically localized prostate cancer at our institution. Clinicians and patients can use these nomograms to help make important decisions regarding management of prostate cancer.

Radical prostatectomy (RP) offers the best chance for curing prostate cancer (CaP) when disease is localized.[1,2] Accurate prediction of pathologic stage is fundamental to determining which patients benefit from RP.[3] Our group and others have published nomograms predicting organ-confined CaP (OC).[4-12] We combine the most commonly available preoperative variables—serum prostate-specific antigen (PSA) level, clinical (TNM) stage, and biopsy Gleason score (GS)—into a model, the "Partin tables," using data from a large series of patients to predict pathologic stage at RP.[4-6]

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The American Cancer Society reports a continued decrease in CaP death rates: 90% of newly diagnosed patients present with local or regional disease.[13] This trend ("stage migration"), whether the result of earlier detection or changes in disease biology, has created a dramatic shift in the clinical stage of newly diagnosed CaP patients.[14]

Data for the original Partin tables were collected from men treated from 1982 to 1991.[4] A multi-institutional update was performed 4 years later, based on data from 1982 to 1996.[5] A further update accounting for continuing stage migration was made in 2001, examining patients from 1994 to 2000.[6] This model has been validated in various clinical settings, from academic medical centers in the United States,[15] Canada,[16] and Turkey[17] to cohorts of community patients.[18]

Over the past 6 years, increasing numbers of men have presented with PSA levels at 2.5 to 10.0 ng/mL, GS 6 or less, nonpalpable lesions on digital rectal examination (clinical stage T1c), and localized OC. Previously determined staging algorithms are based on cohorts different from contemporary CaP patients. Here we update the Partin tables with a contemporary cohort of men treated since 2000 with revised GS and clinical stage categories.

MATERIAL AND METHODS

Patients

The institutional review board at Johns Hopkins approved this study and, when required, written informed consent was obtained from study participants. From 2000 to 2005, we identified 5988 men with clinically localized CaP who underwent RP and staging pelvic lymphadenectomy at the Johns Hopkins Hospital by any of 22 attending surgeons.

Inclusion Criteria

Men enrolled in this cohort had (a) preoperative monoclonal serum PSA level assessed on an ambulatory basis before RP, either before or at least 4 weeks after prostate biopsy, (b) biopsy histologic grade (GS) determined at our institution, and (c) clinical stage assigned by the attending physician (American Joint Committee on Cancer TNM staging system, 1992/2002) of T1c or T2a/b/c. Men were excluded because they lacked this information (n = 29) or received preoperative neoadjuvant hormonal therapy (107). Men were also excluded for preoperative treatment with 5-alpha reductase inhibitors (71), chemo-therapy (5), or androgenic/estrogenic herbal therapies (1) because of potential influence on PSA. Men with pathologic diagnoses other than adenocarcinoma of the prostate (7), absence of cancer on pathology (4), or missing pathologic information (33) (including 30 men with tumor extending to an inked surgical margin, unevaluable for OC versus extraprostatic extension [EPE]) were excluded. One man with biopsy GS 2 + 2 = 4 was excluded,[19] leaving a cohort of 5730 patients.

Pathologic Examination

All pelvic lymph nodes removed at surgery were sectioned and examined for the presence of cancer. The surgical specimen, the prostate and seminal vesicles, was analyzed and the pathologic stage determined as OC if all cancer was confined within the prostate, EPE if cancer was evident outside the prostate and the seminal vesicles and the pelvic lymph nodes were free of disease, positive seminal vesicle involvement (SV+) if tumor invaded the muscular wall of the seminal vesicle without lymph node involvement, and lymph node involvement (LN+) if the pelvic lymph nodes demonstrated CaP.

Statistical Analysis

Multivariable logistic regression analysis was performed to develop a predictive model for the four non-ordered pathologic stage categories: OC, EPE, SV+, or LN+. As in our previous

nomograms,[4-6] the predictor variables were preoperative PSA level, categorized as 0 to 2.5, 2.6 to 4.0, 4.1 to 6.0, 6.1 to 8, 8.1 to 10, and greater than 10.0 ng/mL; clinical stage as T1c, T2a, T2b, and T2c; and GS categorized as 5 to 6, 3 + 4 = 7, 4 + 3 = 7, and 8 to 10. Gleason score was grouped as 5 to 6 and 8 to 10 on the basis of previous work demonstrating that biochemical progression-free survival was equivalent within these groupings.[20] Previous data also demonstrated that GS 3 + 4 = 7 and 4 + 3 = 7 behave differently and warrant substratification.[21,22] Biopsy GS less than 5 were excluded, as recommended by Epstein.[19] The likelihood ratio (LR) chi-square test was used as a measure of goodness of fit. A bootstrap resampling approach with 1000 replications was used to generate 95% confidence intervals for the predicted probabilities of each pathologic stage. Predictive performance of the models was evaluated using the concordance index (c), calculated separately for each non-OC pathologic stage versus OC.[23] The nomogram was calibrated graphically, comparing the predicted probabilities for each covariate pattern with the actual proportion of patients with each pathologic stage; a Loess curve was fit to the data and compared with an ideal fit. Analyses were performed with SAS 9.1 (SAS Institute, Cary, NC), Stata 9.0 (Stata Corporation, College Station, Tex), and PRISM 4 (Graphpad Software, San Diego, Calif).

RESULTS

A total of 5730 men, average age (± standard deviation) 57.4 ± 6.4 years (range, 34 to 75 years), meeting the eligibility criteria were consecutively enrolled (Table 1). Of these, 89% were white and 7% African American. Prostate-specific antigen levels, GS, and clinical stage groupings are described in Table 1. The final pathologic stage demonstrated that 73%, 22%, 3%, and 1% had OC, EPE, SV+, or LN+, respectively. Comparisons with previous Partin nomograms are shown in Table 2.

Year of RP significantly predicted pathologic stage ($P = 0.001$). It was not included in the nomogram model because it is not clinically feasible to include past years of surgery as an input to a nomogram predicting future pathologic stage, and adding year into the model did not materially improve predictive performance (EPE, SV+, and LN+,c = 0.697, 0.832, and 0.906, respectively, with year in the model, and c = 0.695, 0.830, and 0.888 without). Consistent with stage migration, the number of men with clinical stage T2c was dramatically smaller (n = 34, 0.6%) than before.[4-6,10] Models separating T2b and T2c versus those combining them showed no significant improvement (LR chi-square with 3 degrees of freedom [df] = 3.37, $P = 0.338$). Thus, we combined T2b/T2c as a single predictor category. We also examined whether PSA categories should be separated as 6.1 to 8.0 and 8.1 to 10 ng/mL or combined as 6.1 to 10 ng/mL, because clinical decisions rarely differ in this range. Separate categories showed statistically significant improvement in the model (LR chi-square [3 df] = 9.88, $P = 0.020$). However, predictive performance using either grouping was very similar (EPE, SV+, and LN+, c = 0.695, 0.830, and 0.888, respectively, with combined PSA 6.1 to 10, and c = 0.696, 0.830, and 0.894 when 6.1 to 8.0 and 8.1 to 10.0 ng/mL were separately modeled). Therefore, we combined the categories, producing a nomogram more consistent with clinical practice.

As in our previous nomograms,[4-6] PSA level, GS, and clinical stage contributed significantly to prediction of pathologic stage according to multivariable logistic regression. The combination of three preoperative variables predicted pathologic stage better than any single variable individually. The predicted probabilities from the multivariable logistic regression analysis and bootstrapped 95% confidence intervals are presented in Table 3. The numbers within each cell of the nomogram represent the predicted probability of a given pathologic stage based on regression from three preoperative variables. For example, a man with a preoperative PSA level of 2.7 ng/mL, GS 3 + 3 = 6, and nonpalpable results (T1c) on digital rectal examination has predicted probabilities of 88% OC, 11% EPE, 1% SV+, and a negligible

risk of LN+. A man with a PSA level of 11.4 ng/mL and GS 8 with a large palpable tumor (T2c) has predicted probabilities of only 12% OC, 33% EPE, 28% SV+, and 26% LN+.

Risk of EPE, SV+, and LN+ each increased significantly with successively higher PSA level, clinical stage, or GS (P <0.001). An exception occurred when pathologic stage was compared between GS 4 + 3 and GS 8 to 10. Regardless of clinical stage or PSA level, men with GS 8 to 10 had a slightly higher predicted probability of OC and similar or slightly lower probability of LN+, although these trends were not statistically significant (P = 0.90 and 0.77, respectively). Of 164 patients with GS 8 to 10, 3 (2%) underwent diagnostic pelvic lymph node dissection, 120 (73%) underwent bone scan or another modality assessing skeletal metastasis, and 86 (52%) underwent computed tomography or another modality assessing lymph node or visceral metastasis. Of this group with high-risk, high-grade cancer, 123 (75%) had meta-static workup; all these evaluations were negative.

Another exception was a lack of significant difference in risk of SV+ or LN+ between PSA level 0 to 2.5 ng/mL and 2.6 to 4.0 ng/mL (P = 0.507). This may reflect the small numbers of these advanced pathologic stages among men with a low PSA level (n = 19). A similar lack of association between SV+ or LN+ has been observed between PSA level 2.6 to 4.0 ng/mL and 4.1 to 6.0 ng/mL in men undergoing RP.[24]

COMMENT

As a result of either changed CaP biology or improved detection, men presenting with CaP today are increasingly likely to have OC.[20] This stage migration must be accounted for in models predicting the behavior of CaP for contemporary patients. We have used data from 5730 patients treated between 2000 and 2005 to develop an updated nomogram (Partin tables), using preoperative PSA level, clinical stage, and GS to provide the estimated probability of various final pathologic stages at RP. Because this stage migration resulted in few surgical candidates (0.6%) with clinical stage T2c, we found that combining T2b and T2c into a single group generated a model that may have greater validity for use in other populations. These updates and improvements should make this model more useful to clinicians and patients.

Two GS changes appear on this version of the Partin tables. First is absence of GS 2 to 4, reflecting current consensus among many pathologists interpreting prostate biopsies.[19] Gleason pattern 2 is commonly found anteri-orly, in the transition zone and not the peripheral zone, and most GS 2 to 4 is upstaged upon examination of the entire gland, suggesting that biopsy GS 2 to 4 represents sampling error. Gleason score 6 or less, 7, and 8 to 10 maintain their relative frequencies over time. Gleason score 6 or less is present in 76%, 72%, 80%, and 77% of men from 1982 to 1991, 1982 to 1996, 1994 to 2000, and 2000 to 2005, respectively. Although GS 7 disease has been subdivided into 3 + 4 and 4 + 3, its relative fraction has remained stable, as has the fraction of 3 + 4 and 4 + 3 recently. Some investigators have reported an overall increase in the determination of high GS.[25] We, apart from the discontinuation of GS 2 to 4, see stability in the reporting of high GS in men undergoing RP at our institution.

The second change is a previously undescribed phenomenon: a seemingly paradoxical improvement of pathologic stage predictions in men with GS 8 to 10 versus GS 4 + 3. For GS 8 to 10, in every combination of PSA level and clinical stage, the predicted probability of OC was somewhat higher than in GS 4 + 3. A calibration plot for the model demonstrated that the modeled predicted probabilities of OC were very close to observed values (data not shown). In counseling patients, it is important to relay that patients with GS 8 to 10 in this series were carefully selected; 75% of them had been cleared for surgery with negative results on metastatic workup. The predictions from this nomogram do not apply to all patients with high-grade cancer. The Partin tables and other nomograms relating to men with GS 8 to 10 who have

undergone RP represent best-case scenarios of a select few, carefully screened patients with limited, high-grade cancer on biopsy and no evidence of metastasis on radiographic imaging.

Preoperative PSA levels have changed substantially through time. From 1982 to 1991, 40% of patients had a PSA level less than 4.0 ng/mL, perhaps as a result of increased CaP screening via DRE after the development of anatomic RP and before the acceptance of PSA. In a multi-institutional study with more patients treated from 1982 to 1996, only 23% undergoing RP were in the low PSA category.[5] This fraction declined to 17% when our data from 1994 to 2000 were examined[5] but rose again to 25% in the current 2000 to 2005 sample. This recent rise is likely the result of recently established decreased PSA biopsy thresholds.[26-28] As PSA screening detects patients with CaP earlier in the course of their disease, there has been a decline in patients presenting with a PSA level greater than 10 ng/mL. The fraction of men between 4.1 and 10.0 ng/mL has remained stable in our cohort since the advent of PSA screening in the early 1990s.

As expected with the popularization of PSA screening, the fraction of clinical stage T1c increased from 33% during 1982 to 1996[5] to 63% in the 1994 to 2000 cohort,[6] to 77% presently. Because few patients are diagnosed with cancer by transurethral resection of the prostate, these have been excluded from our two most recent nomograms. As expected, there was a decline in palpable disease from 86%[4] to 63%[5] to 36%[6] to 23% across the timeline of published nomograms (Table 2). Our data for stage at presentation are consistent with other publications describing the effects of CaP screening on clinical stage at presentation.[29]

Changes in clinical stage are mirrored by changes in pathologic stage. As previously reported,[13,14,29] an increasing number of men present with OC disease. In our published nomograms, 54%,[4] 48%,[5] 64%,[6] and finally 73% of men undergoing RP were OC (Table 2). Concomitantly, the fractions of LN+ and SV+ patients have declined to 3% and 1%, respectively.

There is an overall increase in the predicted probability of OC from the 2001 nomogram[6] to the present one (Table 3). This trend is most apparent among patients with GS 8 to 10 and clinical stage T2b/T2c. Despite inclusion of T2c, the combined T2b/T2c group from this cohort predicts more OC than the 2001 nomogram predicts for T2b. The predicted probabilities of OC in clinical stage T2b from 2001[6] for GS 8 to 10 and increasing PSA level are 34%, 23%, 18%, 13%, and 6%. The corresponding values from Table 3 for T2b/T2c and GS 8 to 10 are 49%, 39%, 27%, 24%, and 12%. These data suggest a trend toward improved pathologic stage in these carefully selected patients with high clinical stage. Such a difference may suggest an expanded role for surgery in advanced clinical stage, advanced GS CaP. This also possibly represents a “Will Rogers” phenomenon,[25,30] whereby biopsies that would formerly have been classified GS 4 + 3 are now more carefully graded (or better detected due to an increased number of biopsy cores) and classified as GS 8 to 10. Further validation of our results is necessary before changes in clinical practice can be suggested.

CONCLUSIONS

We have updated the “Partin tables” on the basis of data from men treated after 2000, to reflect current trends in presentation and pathologic stage in men newly diagnosed with CaP. Clinicians can use these nomograms, based on the large cohort of men with CaP treated at our institution, to counsel individual patients and help them make important management decisions.

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Table 1

Demographics of men undergoing radical prostatectomy.

Patients (n)	5730
Age (yr), mean ± SD	57.4 ± 6.4
Race (%)	
White	5081 (89)
African American	372 (7)
Hispanic	56 (1)
Asian	58 (1)
Other	158 (3)
PSA groups (ng/mL)	
0–2.5	452 (8)
2.6–4.0	946 (17)
4.1–6.0	1994 (35)
6.1–8.0	1093 (19)
8.1–10.0	578 (10)
>10.0	667 (12)
Biopsy Gleason score	
5–6	4402 (77)
3 + 4 = 7	816 (14)
4 + 3 = 7	348 (6)
≥8	164 (3)
Clinical stage (AJCC-TNM 2002)	
T1c	4419 (77)
T2a	998 (17)
T2b	279 (5)
T2c	34 (1)
Prostatectomy Gleason score	
5–6	3693 (64)
3 + 4	1304 (23)
4 + 3	425 (7)
8–10	308 (5)
Organ confined	4204 (73)
Extraprostatic extension (SV−, LN−)	1276 (22)
Seminal vesicle involvement (LN−)	180 (3)
Lymph node involvement	70 (1)

SD = standard deviation; PSA = prostate-specific antigen; AJCC-TNM = American Joint Committee on Cancer-Tumor Node Metastasis; SV = seminal vesicle; LN = lymph node.

Values are number (percentage), unless otherwise noted.

Table 2

Demographic information of men included in previous versions of the Partin tables

Characteristic	Year of Publication			
	1993	1997	2001	2007
Years of treatment	1982 to 1991	1982 to 1996	1994 to 2000	2000 to 2005
Patients (n)	703	4133	5079	5730
Age (yr), mean ± SD			57.9 ± 6	57.4 ± 6.4
Race				
White			4571 (90)	5081 (89)
African-American			305 (6)	372 (7)
Hispanic				56 (1)
Asian				58 (1)
Other				158 (3)
PSA groups (ng/mL)				
0–2.5	*	*	356 (7)	452 (8)
2.6–4.0	284 (40)	943 (23)	508 (10)	946 (17)
4.1–6.0	†	†	1371 (27)	1994 (35)
6.1–10.0	246 (35)	2006 (48)	1778 (35)	1671 (29)
>10.0	173 (25)	1184 (29)	1067 (21)	667 (12)
Biopsy Gleason score				
2–4	64 (9)	222 (5)	30 (0.6)	‡
5–6	471 (67)	2783 (67)	4012 (79)	4402 (77)
3 + 4 = 7	§	§	660 (13)	816 (14)
4 + 3 = 7	130 (19)	906 (22)	223 (4.4)	348 (6)
≥8	38 (5)	222 (5)	152 (3)	164 (3)
Clinical stage (AJCC-TNM 1992/2002)				
T1a	31 (4)	74 (1)	‡	‡
T1b	71 (10)	149 (3)	‡	‡
T1c	NA	1358 (33)	3250 (64)	4419 (77)
T2a	326 (46)	1186 (29)	1524 (30)	998 (17)
T2b	179 (26)	852 (21)	203 (4)	279 (5)
T2c	60 (9)	398 (10)	102 (2)	34 (1)
T3a	36 (5)	116 (3)	‡	‡
Pathologic stage				
Organ confined	382 (54)	1957 (48)	3251 (64)	4204 (73)
Extraprostatic extension (SV−, LN−)	203 (29)	1661 (40)	1524 (30)	1276 (22)
Seminal vesicle involvement (LN−)	44 (6)	303 (7)	203 (4)	180 (3)
Lymph node involvement	74 (11)	212 (5)	1016 (2)	70 (1)

NA = not applicable. Other abbreviations as in Table 1.

Values are number (percentage), unless otherwise noted.

* 0–4 combined.

† 4–10 combined.

‡ Excluded from analysis.

§ All 7s combined.

Table 3

Nomograms predicting pathologic stage of prostate cancer according to clinical stage (TNM), PSA level, and Gleason score

PSA Range (ng/mL)	Pathologic Stage	Biopsy Gleason Score 5–6	3 + 4 = 7	4 + 3 = 7	8–10
Clinical Stage T1c (nonpalpable, PSA elevated) (n = 4419)					
0–2.5	Organ confined (n = 226)	**93 (91–95)**	**82 (76–87)**	**73 (64–80)**	**77 (65–85)**
	Extraprostatic extension (n = 19)	**6 (5–8)**	**14 (10–18)**	**20 (14–28)**	**16 (11–24)**
	Seminal vesicle (+) (n = 1)	0 (0–1)	2 (0–5)	2 (0–5)	3 (0–8)
	Lymph node (+) (n = 3)	0 (0–1)	2 (0–6)	4 (1–12)	3 (1–12)
2.6–4.0	Organ confined (n = 619)	**88 (86–90)**	**72 (67–76)**	**61 (54–68)**	**66 (57–74)**
	Extraprostatic extension (n = 92)	**11 (10–13)**	**23 (19–27)**	**33 (27–39)**	**26 (19–34)**
	Seminal vesicle (+) (n = 8)	1 (0–1)	4 (2–7)	5 (2–8)	7 (3–13)
	Lymph node (+) (n = 1)	0 (0–0)	1 (0–1)	1 (0–3)	1 (0–3)
4.1–6.0	Organ confined (n = 1266)	**83 (81–85)**	**63 (59–67)**	**51 (45–56)**	**55 (46–64)**
	Extraprostatic extension (n = 297)	**16 (14–17)**	**30 (26–33)**	**40 (34–45)**	**32 (25–40)**
	Seminal vesicle (+) (n = 37)	1 (1–1)	6 (4–8)	7 (4–10)	10 (6–15)
	Lymph node (+) (n = 12)	0 (0–0)	2 (1–3)	3 (1–6)	3 (1–6)
6.1–10.0	Organ confined (n = 989)	**81 (79–83)**	**59 (54–64)**	**47 (41–53)**	**51 (41–59)**
	Extraprostatic extension (n = 281)	**18 (16–19)**	**32 (27–36)**	**42 (36–47)**	**34 (26–42)**
	Seminal vesicle (+) (n = 36)	1 (1–2)	8 (6–11)	8 (5–12)	12 (8–19)
	Lymph node (+) (n = 5)	0 (0–0)	1 (1–3)	3 (1–5)	3 (1–5)
>10.0	Organ confined (n = 324)	**70 (66–74)**	**42 (37–48)**	**30 (25–36)**	**34 (26–42)**
	Extraprostatic extension (n = 165)	**27 (23–30)**	**40 (35–45)**	**48 (40–55)**	**39 (31–48)**
	Seminal vesicle (+) (n = 25)	2 (2–3)	12 (8–16)	11 (7–17)	17 (10–25)
	Lymph node (+) (n = 13)	1 (0–1)	6 (3–9)	10 (5–17)	9 (4–17)
Clinical Stage T2a (palpable <½ of one lobe) (n = 998)					
0–2.5	Organ confined (n = 156)	**88 (84–90)**	**70 (63–77)**	**58 (48–67)**	**63 (51–74)**
	Extraprostatic extension (n = 18)	**12 (9–15)**	**24 (18–30)**	**32 (24–41)**	**26 (18–36)**
	Seminal vesicle (+) (n = 2)	0 (0–1)	2 (0–6)	3 (0–7)	4 (0–10)
	Lymph node (+) (n = 1)	0 (0–1)	3 (1–9)	7 (1–17)	6 (1–16)
2.6–4.0	Organ confined (n = 124)	**79 (75–82)**	**57 (51–63)**	**45 (38–52)**	**50 (40–59)**
	Extraprostatic extension (n = 49)	**20 (17–24)**	**37 (31–42)**	**48 (40–55)**	**40 (30–50)**
	Seminal vesicle (+) (n = 5)	1 (0–1)	5 (3–9)	5 (3–10)	8 (4–15)
	Lymph node (+) (n = 0)	0 (0–0)	1 (0–2)	2 (0–5)	2 (0–4)
4.1–6.0	Organ confined (n = 171)	**71 (67–75)**	**47 (41–52)**	**34 (28–41)**	**39 (31–48)**
	Extraprostatic extension (n = 101)	**27 (23–31)**	**44 (39–49)**	**54 (47–60)**	**46 (37–54)**
	Seminal vesicle (+) (n = 10)	1 (1–2)	7 (4–10)	7 (4–11)	11 (6–17)
	Lymph node (+) (n = 3)	0 (0–1)	2 (1–4)	5 (2–8)	4 (2–9)
6.1–10.0	Organ confined (n = 142)	**68 (64–72)**	**43 (38–48)**	**31 (26–37)**	**36 (27–44)**
	Extraprostatic extension (n = 99)	**29 (26–33)**	**46 (41–51)**	**56 (49–62)**	**47 (37–56)**
	Seminal vesicle (+) (n = 12)	2 (1–3)	9 (6–13)	9 (5–14)	13 (8–20)
	Lymph node (+) (n = 6)	0 (0–1)	2 (1–4)	4 (2–8)	4 (1–8)
>10.0	Organ confined (n = 36)	**54 (49–60)**	**28 (23–33)**	**18 (14–23)**	**21 (15–28)**
	Extraprostatic extension (n = 47)	**41 (35–46)**	**52 (46–59)**	**57 (48–66)**	**49 (39–59)**
	Seminal vesicle (+) (n = 9)	3 (2–5)	12 (7–18)	11 (6–17)	17 (9–25)
	Lymph node (+) (n = 7)	1 (0–3)	7 (3–14)	13 (6–24)	12 (5–22)
Clinical Stage T2b (palpable ≥½ of lobe) or T2c (palpable both lobes) (n = 313)					
0–2.5	Organ confined (n = 16)	**84 (78–89)**	**59 (47–70)**	**44 (31–58)**	**49 (32–65)**
	Extraprostatic extension (n = 10)	**14 (9–19)**	**24 (16–33)**	**29 (19–42)**	**24 (14–36)**
	Seminal vesicle (+) (n = 0)	1 (0–3)	6 (0–14)	6 (0–14)	8 (0–21)
	Lymph node (+) (n = 0)	1 (0–3)	10 (2–25)	19 (4–40)	17 (3–42)
2.6–4.0	Organ confined (n = 28)	**74 (68–80)**	**47 (39–56)**	**36 (27–45)**	**39 (28–50)**
	Extraprostatic extension (n = 15)	**23 (18–29)**	**37 (28–45)**	**46 (36–55)**	**37 (27–48)**
	Seminal vesicle (+) (n = 3)	2 (1–5)	13 (7–21)	13 (7–22)	19 (9–32)

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PSA Range (ng/mL)	Pathologic Stage	Biopsy Gleason Score 5–6	3 + 4 = 7	4 + 3 = 7	8–10
	Lymph node (+) (n = 2)	0 (0–1)	3 (0–7)	5 (0–14)	4 (0–13)
4.1–6.0	Organ confined (n = 46)	**66 (59–72)**	**36 (29–43)**	**25 (19–32)**	**27 (19–37)**
	Extraprostatic extension (n = 40)	**30 (24–36)**	**41 (33–47)**	**47 (38–55)**	**38 (28–48)**
	Seminal vesicle (+) (n = 7)	4 (2–6)	16 (10–23)	15 (9–23)	22 (13–33)
	Lymph node (+) (n = 4)	1 (0–2)	7 (3–12)	13 (6–21)	11 (4–23)
6.1–10.0	Organ confined (n = 53)	**62 (55–68)**	**32 (26–38)**	**22 (17–29)**	**24 (17–33)**
	Extraprostatic extension (n = 28)	**32 (26–38)**	**41 (33–49)**	**47 (38–56)**	**38 (29–48)**
	Seminal vesicle (+) (n = 15)	5 (3–8)	20 (13–28)	19 (11–28)	27 (16–39)
	Lymph node (+) (n = 5)	1 (0–2)	6 (3–11)	11 (5–19)	10 (3–20)
>10.0	Organ confined (n = 8)	**46 (39–53)**	**18 (13–24)**	**11 (7–15)**	**12 (7–18)**
	Extraprostatic extension (n = 15)	**41 (34–50)**	**40 (31–51)**	**40 (30–52)**	**33 (22–46)**
	Seminal vesicle (+) (n = 10)	7 (4–12)	23 (15–33)	19 (10–29)	28 (16–42)
	Lymph node (+) (n = 8)	5 (2–8)	18 (9–30)	29 (15–44)	26 (12–44)

Abbreviations as in Table 1.

Values are percent probability (95% confidence interval) of a given pathologic stage.