

# Examining the association between real-world extended vs. standard pelvic lymph node dissection and early and late oncologic outcomes in men undergoing radical prostatectomy

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Cite as: MacNevin W, Kim SSY, Spooner JTR, et al. Examining the association between real-world extended vs. standard pelvic lymph node dissection and early and late oncologic outcomes in men undergoing radical prostatectomy. *Can Urol Assoc J* 2025;19(12):379-86. <http://dx.doi.org/10.5489/auaj.9213>

Published online August 28, 2025

## ABSTRACT

**INTRODUCTION:** In patients with prostate cancer (PCa), the impact of extended pelvic lymph node dissection (E-PLND) during radical prostatectomy (RP) on oncologic outcomes remains controversial. This study examined the association between extended vs. standard PLND (S-PLND) and biochemical recurrence (BCR), an early outcome, as well as metastatic PCa (mPCa), and castration-resistant PCa (CRPC) development, late outcomes, in a multi-institutional cohort.

**METHODS:** High-risk post-RP patients from a Canadian PCa database were analyzed from January 1, 2005, to December 31, 2016. The association between PLND and BCR, mPCa, and CRPC development and complication rate was examined using regression and correlation analysis.

**RESULTS:** Data were collected from patients who underwent S-PLND (n=494) and E-PLND (n=107). The median followup was 40.1 months, and time to BCR, mPCa, and CRPC development was 9.8, 46.0, and 52.1 months, respectively. The median (interquartile range) number of lymph nodes extirpated was 7 (7) and 14 (11) for the S-PLND and E-PLND groups, respectively. E-PLND was associated with increased intraoperative blood loss and higher postoperative complication rate. There were no differences in BCR-free survival based on PLND approach, with 67.1% of S-PLND cases and 71.1% of E-PLND cases reaching BCR-free survival at the end of the followup period (hazard ratio [HR] 0.784 [0.506, 1.215], p=0.28). PLND extent was not a predictor for mPCa progression (p=0.963). Similarly, there were no differences in CRPC-free survival based on dissection type (S-PLND 90.9% vs. E-PLND 89.1%, p=0.561). Lymph node positivity was predictive of BCR, mPCa, and CRPC progression.

**CONCLUSIONS:** E-PLND did not show significant differences in the rates of BCR, mPCa, or CRPC progression when compared to S-PLND. E-PLND was associated with higher complication rates. This study adds to the data exploring the association between PLND and PCa oncologic outcomes.

## INTRODUCTION

For patients with prostate cancer (PCa) undergoing radical prostatectomy (RP), pelvic lymph node dissection is commonly performed at the time of the procedure. Based on the probability of finding lymph nodes (LN) containing malignancy, urologists may opt to perform an extended pelvic lymph node dissection (E-PLND) or a standard (S-PLND) dissection.<sup>1</sup>

The role of E-PLND vs. S-PLND remains controversial, as the therapeutic benefit remains unclear.<sup>1-5</sup> Randomized controlled trials (RCTs) comparing E-PLND vs. S-PLND in intermediate- and high-risk PCa have demonstrated that E-PLND provides better pathologic staging but no improvements in biochemical recurrence (BCR)-free survival or metastatic-free survival.<sup>4,5</sup> Despite this, high-quality systematic reviews and meta-analyses have demonstrated improved BCR-free survival with E-PLND.<sup>1</sup> Due to this controversy, E-PLND is often done at the surgeon's discretion for those with extensive local disease, enlarged nodes on imaging, high-risk preoperative clinical and/or pathologic features, and for those perceived to benefit the most from accurate nodal staging.<sup>1</sup>

Most studies examining the outcomes of E-PLND vs. S-PLND often examine very late oncologic endpoints, which tend to occur years later, resulting in missing data on the natural course of PCa after RP due to loss of patient followup.<sup>3,5-7</sup>

## KEY MESSAGES

- Controversy exists surrounding the role of extended pelvic lymph node dissection (E-PLND) vs. standard pelvic lymph node dissection (S-PLND) during radical prostatectomy (RP) and its impact on oncologic outcomes.
- E-PLND was associated with a higher complication rate and increased intraoperative blood loss when compared to S-PLND, with no statistically significant differences in biochemical recurrence, metastatic progression, or development of castration-resistant prostate cancer.
- Although E-PLND was not associated with clear oncologic benefits compared to S-PLND, E-PLND may offer more accurate staging in select high-risk patients who may benefit from further treatment.

Few studies exist examining the role of PLND type on both early oncologic outcomes, such as BCR, and late oncologic outcomes, such as the development of metastatic disease and castration-resistant prostate cancer (CRPC).<sup>4</sup> Furthermore, a better understanding of the utility of E-PLND vs. S-PLND can help inform the value of less invasive approaches to PLND.<sup>8,9</sup>

Herein, we examined the association between E-PLND and S-PLND on the oncologic outcomes of BCR, progression to metastatic PCa (mPC), and the development of CRPC.

## METHODS

### Study and population

This study used a multicenter, retrospective database created from seven high-volume Canadian centers. This database contains demographic, pathologic, surgical, and postoperative data on patients undergoing RP at each site. Data was collected from January 1, 2005, to December 31, 2016, and was retrospectively reviewed. A total of 26 surgeons were included in this group. Choice of E-PLND vs. S-PLND was non-standardized and decided upon by each surgeon individually. E-PLND (in general) was defined as PLND from the obturator nerve, external iliac artery, and internal

iliac artery, whereas S-PLND was defined as PLND from the obturator nerve and external iliac artery.<sup>5</sup> Classification of S-PLND vs. E-PLND was done through review of operative notes and the surgical boundaries used for PLND.

BCR was defined as an outcome determined when a patient's serum prostate-specific antigen (PSA) level reached  $\geq 0.2$  ng/mL, with a second confirmatory level. Metastatic disease development was defined as any volume of new metastatic disease detected radiologically. Development of CRPC was defined as biochemical or clinical progression despite achieving castrate-level testosterone levels  $< 1.7$  nmol/L.

### Data collection

Data was collected from the study population through review of patient electronic medical records (EMR) at each site, forming a database of patient information. Demographic information, preoperative laboratory values, clinic visit information, operative reports, perioperative and postoperative bloodwork, use of adjuvant or salvage therapy, and followup clinical reports and bloodwork were reviewed for clinical information, and anonymized data were stored in a central database. Institutional research ethics approval was obtained prior to analysis.

### Statistical analysis

Demographic, surgical factors, and outcomes were expressed using frequencies and percentages and were analyzed using Chi-squared and Mann-Whitney U tests. Spearman correlation analysis was used to analyze the correlation between E-PLND and S-PLND with patient and surgical factors. Cox regression analysis was used to model BCR, mPC development, and CRPC progression. Factors included for regression analysis were determined by review of the literature and by the strength of association between predictor and outcome variables after controlling for collinearity. Variables in the regression analysis included: pathologic stage, preoperative PSA, pathologic Gleason grade group, adjuvant systemic therapy, LN yield, LN positivity, margin status, and prostate volume. Statistical significance was set at  $p=0.05$  with a 95% confidence interval.

## RESULTS

### Patient population

Six hundred and one patients were included in the study, with 494 undergoing S-PLND and 107 undergoing E-PLND. Patients in the E-PLND group were

younger (mean ± standard deviation: 61.8±7.18 years vs. 63.8±5.98 years, p=0.008) and had slightly higher body mass index (BMI) (28.9±4.3 vs. 28.3±4.04, p=0.05) (Table 1). Most patients had Gleason grade ≥8 disease (S-PLND: n=384/494, 77.7%; E-PLND: n=79/107, 73.8%; p=0.01). Salvage radiation was administered to 34.8% (n=172/494) of S-PLND patients and 48.6% (n=52/107) in the E-PLND group (p=0.007).

There was no difference between groups regarding the use of adjuvant androgen deprivation therapy (S-PLND: n=49/494, 9.9%; E-PLND: n=9/107, 8.4%; p=0.632). There were no statistically significant differences in preoperative PSA (S-PLND: 8.97 ng/mL [0.10, 250.0]; E-PLND: 9.90 ng/mL [0.47, 300.0]; p=0.347), pathologic T stage (p=0.738), or positive margin status (S-PLND: n=184/494, 37.2%; E-PLND: n=45/107, 42.1%).

**Lymph node dissection**

The median [interquartile range] number of lymph nodes dissected was 7 [7] and 14 [11] in S-PLND and E-PLND, respectively (p=0.002). Positive LN involvement was more common with E-PLND (n=25/107, 23.4%) compared to S-PLND (n=60/489, 12.3%) (p=0.003). There were no significant increases in operative time noted between dissection type (p=0.184). E-PLND was associated with increased intraoperative blood loss (p=0.252, p<0.001; E-PLND: 500 [100, 4000] mL; S-PLND: 300 [10, 3000] mL; p<0.001).

**Complications**

Patients undergoing E-PLND (n=18/107, 16.8%) had higher complication rates compared to S-PLND (n=56/494, 11.3%) (p=0.01) (Table 2). Of patients who experienced postoperative complications, patients recovering from E-PLND were subjected to higher Clavien-Dindo grade complications compared to S-PLND patients, with nine patients in the E-PLND group (50.0%) compared to seven (12.5%) patients in the S-PLND group experiencing a Clavien-Dindo grade ≥3 complication (p=0.01). The most common postoperative complications for E-PLND were anastomotic leaks (n=6/107, 5.61%) and symptomatic lymphocele development (n=5/107, 4.67%), while S-PLND patients most commonly experienced wound infections/abscess development (n=16/494, 3.24%) and anastomotic leaks (n=14/494, 2.83%).

**Oncologic outcomes**

The median followup was 40.1 [0.03, 137] months, with a median time from operation to BCR, mPC progres-

**Table 1. Demographic factors**

Patient demographics	Standard (n=494) Frequency (%)	Extended (n=107)	p
<b>Clinical T stage</b>			0.90
T1			
T1a	1 (0.2)	1 (0.9)	
T1b	114 (23.1)	13 (12.1)	
T1c	208 (42.1)	44 (44.1)	
T2			
T2a	56 (11.3)	18 (16.8)	
T2b	43 (8.7)	9 (8.4)	
T2c	44 (8.9)	14 (13.1)	
T3			
T3a	8 (1.6)	4 (3.7)	
T3b	–	–	
Missing	19 (3.8)	4 (3.7)	
<b>Pathologic T stage</b>			0.738
T1			
T1a	–	–	
T1b	–	–	
T1c	–	–	
T2			
T2a	35 (7.1)	6 (5.6)	
T2b	10 (2.0)	2 (1.9)	
T2c	95 (19.2)	16 (15.0)	
T3			
T3a	216 (43.7)	53 (49.5)	
T3b	131 (26.5)	30 (28.0)	
T4	4 (0.8)	–	
Missing	3 (0.6)	–	
<b>Pathologic N stage</b>			0.004
N0	429 (86.8)	81 (75.7)	
N1	53 (11.3)	25 (23.4)	
Missing	9 (1.8)	1 (0.9)	

PSA: prostate-specific antigen.

**Table 1 (cont'd). Demographic factors**

	Standard (n=494)	Extended (n=107)	p
<b>Patient demographics</b>	<b>Frequency (%)</b>		
<b>Gleason grade (preoperative)</b>			0.012
Gleason 6	21 (4.3)	3 (2.8)	
Gleason 7 (3+4)	43 (8.7)	9 (8.4)	
Gleason 7 (4+3)	41 (8.3)	16 (15.0)	
Gleason 8	265 (53.6)	41 (38.3)	
Gleason 9/10	119 (24.1)	38 (35.3)	
Missing	5 (1.0)	—	
<b>Gleason grade (postoperative)</b>			0.03
Gleason 6	4 (0.8)	—	
Gleason 7 (3+4)	104 (21.1)	14 (13.1)	
Gleason 7 (4+3)	178 (36.0)	32 (29.9)	
Gleason 8	77 (15.6)	17 (15.9)	
Gleason 9/10	122 (24.7)	41 (38.3)	
Missing	9 (1.8)	3 (2.8)	
<b>Extraprostatic extension</b>			0.107
Yes	344 (69.6)	83 (77.6)	
No	149 (30.2)	24 (22.4)	
Missing	1 (0.2)	—	
<b>Lymphovascular invasion</b>			0.010
Yes	118 (23.9)	39 (36.4)	
No	367 (74.3)	68 (63.6)	
Missing	9 (1.8)	—	
<b>Active surveillance</b>			0.656
Yes	45 (9.1)	11 (10.5)	
No	436 (88.3)	91 (85.0)	
Missing	13 (2.6)	5 (4.7)	
<b>Neoadjuvant chemotherapy</b>			0.222
Yes	—	1 (0.9)	
No	471 (95.3)	89 (83.2)	
Missing	23 (4.7)	17 (15.9)	
<b>Adjuvant androgen deprivation therapy</b>			0.632
Yes	49 (9.9)	9 (8.4)	
No	445 (90.1)	98 (91.6)	

PSA: prostate-specific antigen.

**Table 1 (cont'd). Demographic factors**

	Standard (n=494)	Extended (n=107)	p
<b>Patient demographics</b>	<b>Frequency (%)</b>		
<b>Surgical approach</b>			0.001
Open	211 (42.7)	79 (73.8)	
Robotic-assisted laparoscopic	261 (52.8)	26 (24.3)	
Missing	22 (4.5)	2 (1.9)	
<b>Positive lymph node involvement</b>			0.003
Yes	60 (12.2)	25 (23.4)	
No	429 (86.8)	82 (76.6)	
Missing	5 (1.0)	—	
<b>Margin status</b>			0.361
Positive	184 (37.2)	45 (42.1)	
Negative	309 (62.6)	62 (57.9)	
Missing	1 (0.2)	—	
<b>*Chi-squared test</b>			
	<b>Mean (standard deviation)/ Median [minimum, maximum]</b>		
Age	63.8 (5.98)	61.8 (7.18)	0.008
Body mass index	28.3 (4.04)	28.9 (4.3)	0.049
Preoperative PSA	8.97 (0.10, 250.0)	9.90 (0.47, 300.0)	0.347
Prostate volume	31.0 (10.0, 150.0)	37.5 (15.0, 180.0)	0.137
Operating room time	153.0 (61.0, 534.0)	153.0 (66.0, 445.0)	0.184
Number of lymph nodes dissected	7.00 (1, 29)	14.0 (1, 48)	0.0001
Number of positive lymph nodes dissected	0.19 (0.67)	0.58 (1.17)	0.002
<b>*Mann-Whitney U test</b>			
PSA: prostate-specific antigen.			

sion, and CRPC development of 9.8 [0.6, 121.4], 46.0 [1.2, 120.3], and 52.1 [12.0, 122.0] months, respectively. In the S-PLND group, 24.9% (n=123/494) developed BCR, with a median time of 9.3 [0.95, 94.75] months for BCR. In the E-PLND group, 22.4% (n=24/107) of patients developed BCR, with a median time to BCR of 11.84 [0.62, 67.46] months. Time to mPC progression was 58.8 [1.3, 111.5] and 44.5 [1.2, 120.3] months for S-PLND and E-PLND, respectively (p=0.328). When examining CRPC development, 7.66% (n=21/274) of

Table 2. Postoperative complications and complication rate		
Complication	Standard (n=494) n (%)	Extended (n=107) n (%)
Anastomatic urine leak	14 (2.83)	6 (5.61)
Bladder neck dehiscence	1 (0.20)	1 (0.93)
Wound infection/abscess	16 (3.24)	2 (1.87)
Lymphocele	8 (1.62)	5 (4.67)
Surgical bleed	8 (1.62)	2 (1.87)
Hernia	3 (0.61)	0 (0)
Hematuria requiring intervention	5 (1.01)	0 (0)
Venous thromboembolism	1 (0.20)	2 (1.87)
Complication rate	S-PLND: 11.3%	E-PLND: 16.8%
Highest Clavien-Dindo		
Grade 1	18 (32.1)	7 (38.9)
Grade 2	10 (17.9)	2 (11.1)
Grade 3	7 (12.5)	8 (44.4)
Grade 4	—	1 (5.56)

E: extended; PLND: pelvic lymph node dissection; S: standard.

patients who underwent S-PLND experienced CRPC progression, with a median time to CRPC of 9.27 [0.95, 94.75] months. In the E-PLND group, 9.80% (n=5/51) of patients progressed to CRPC, with a median time to CRPC progression being 8.55 [0.62, 67.46] months.

In multivariable Cox regression analysis, there were no differences in BCR-free survival based on PLND extent (E-PLND: hazard ratio [HR] 0.784 [0.506, 1.215], p=0.28) (Figure 1). LN positivity was found to be a predictive factor for BCR in S-PLND (HR 1.89 [1.77, 24.47], p=0.005) and E-PLND (HR 1.26 [1.12, 11.11], p=0.03). For patients with positive LNs, BCR-free survival was 48.9% at five years compared to 68.9% for patients with negative LNs (X<sup>2</sup>=33.3, p=0.001)

Metastatic disease progression was seen in 12.1% (n=13/107) of patients in the E-PLND group and in 9.1% (n=45/494) who underwent S-PLND (p=0.229). Metastasis-free survival was 91.2% at five years for patients who underwent S-PLND compared to 88.2% at five years for the E-PLND group (X<sup>2</sup>=0.956, p=0.328) (Figure 2). On multivariable analysis, PLND extent was not found to be a predictor for progression to metastatic disease (p=0.963). LN positivity (HR 5.23 [1.19, 22.9], p=0.028) was strongly predictive of metastatic disease progression, while older age at

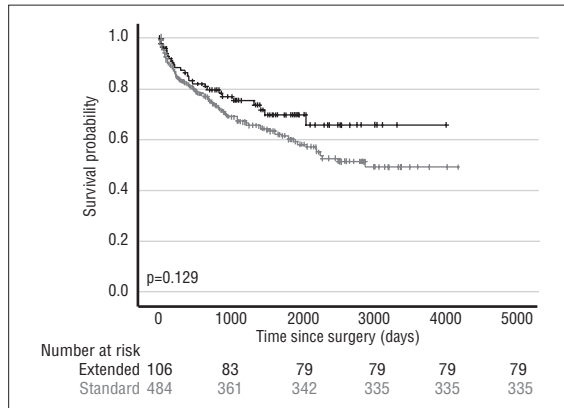


Figure 1. Biochemical recurrence-free (BCR) survival based on pelvic lymph node dissection (PLND) type.

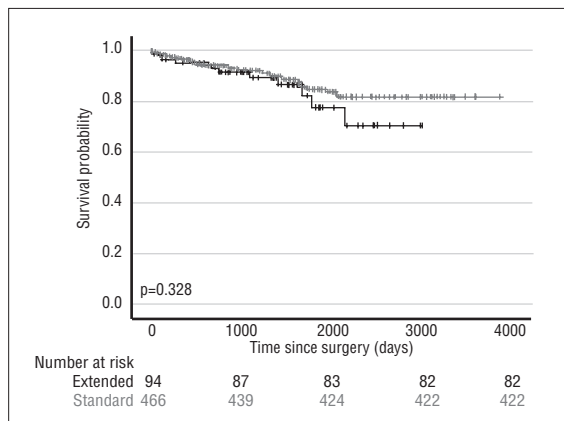


Figure 2. Metastasis-free survival based on pelvic lymph node dissection (PLND) type.

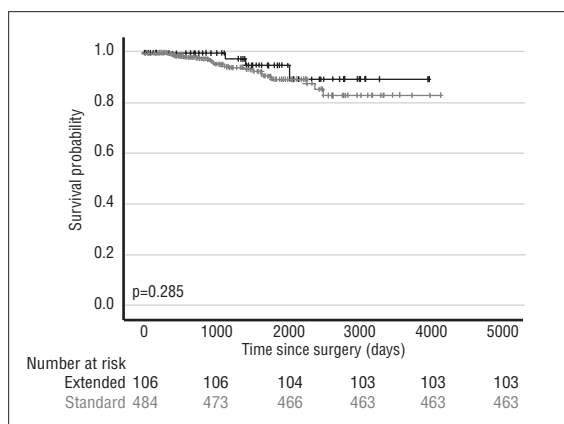


Figure 3. Castrate-resistant prostate cancer (CRPC)-free survival based on pelvic lymph node dissection (PLND) type.

time of surgery (HR 0.881 [0.777, 1.00], p=0.049) was protective.

Similarly, in multivariable Cox regression analysis, no differences in CRPC-free survival were seen based

on PLND extent (E-PLND: HR 1.34 [0.502, 3.567],  $p=0.561$ ) (Figure 3). Positive LN disease was a factor influencing CRPC-free survival. Of patients with positive LNs, CRPC-free survival was found to be 68.3% at five years compared to 90.9% for those with negative LNs ( $\chi^2=11.2$ ,  $p=0.001$ ). Therefore, patients with positive LN disease had a reduction in CRPC-free survival of approximately 22.6% at five years after surgery.

## DISCUSSION

The role of E-PLND vs. S-PLND during RP remains controversial, with limited evidence on the benefits of more extensive resection.<sup>1-3,5</sup> Previous comparative studies have shown no significant improvements in metastasis-free survival with more extensive dissection, but the impact of E-PLND on earlier and later oncologic outcomes remains understudied.<sup>4</sup> To address this knowledge gap, this study used a retrospective, multicenter approach to examine BCR, mPC progression, and CRPC development based on PLND type.

In our study, patients in the E-PLND group were slightly younger and had greater BMI compared to the S-PLND group, but were otherwise similar in demographic and disease characteristics, such as clinical and pathologic T stage and preoperative PSA. This similarity exemplifies that urologists do not have a unified approach or consensus on which patients should undergo E-PLND, but the decision is often influenced by Gleason grade, as well as tumor and nodal characteristics.<sup>1</sup>

E-PLND demonstrated double the LN yield compared to S-PLND, with an increased positive LN yield. This finding supports the current literature, which suggests that E-PLND has higher staging accuracy, which may influence treatment options and allow for improved risk stratification.<sup>4,10</sup> This improved staging accuracy has to be considered alongside the potential increased complication risk associated with E-PLND.<sup>11</sup> E-PLND has been shown to have higher complication risks, which was supported by our findings of E-PLND having increased intraoperative blood loss and complication rate.<sup>11,12</sup> Of note, patients in our study undergoing E-PLND not only had higher complication rates, but also higher average Clavien-Dindo complication grades.

When analyzing PLND type on BCR, there was no statistically significant reduction in BCR in the E-PLND group compared to S-PLND. Some studies have suggested that E-PLND may be associated with improved BCR-free survival compared to S-PLND, although this evidence is not consistent.<sup>13-18</sup> When analyzing predictors for the development of BCR, in both patients undergoing S-PLND and E-PLND, LN positivity was a

significant predictive factor. This is consistent with the current literature, citing Gleason score  $>7$ , preoperative PSA, pathologic T stage, positive surgical margins, and LN positivity being predictors of five-year BCR-free survival.<sup>5,19,20</sup> LN positivity demonstrated a reduction in BCR-free survival by 20% at five years, which is consistent with timelines in the current literature.<sup>19</sup>

Metastasis-free survival was worse for patients in the E-PLND group, with higher rates and earlier progression when compared to S-PLND, although this was not statistically significant. Furthermore, metastasis-free survival was significantly worse for patients with positive LN disease, which likely reflects the more aggressive disease characteristics of PCa that has invaded regional nodes.

Although E-PLND was mildly associated with increased positive LN yield compared to S-PLND, this did not translate to statistical significance when analyzing BCR-free survival, metastasis-free survival, or CRPC-free survival. Similar to BCR, limited data exist on the utility of E-PLND in improving CRPC-free survival.<sup>21</sup> Theoretically, E-PLND may confer improved CRPC-free survival, as potential cancer that has spread to the LNs will be fully resected, and staging is superior, which may allow for additional — and earlier — treatment for node-positive disease.

Additionally, patients in the E-PLND group were more likely to undergo salvage radiation treatment compared to the S-PLND group, which may have influenced oncologic outcomes despite controlling for salvage treatment in the multivariate analyses.

With respect to node positivity, in our study, patients with positive LNs had reduced CRPC-free survival compared to patients with negative node status by approximately 23%. Positive LN disease was associated with worse BCR-free survival, metastasis-free survival, and CRPC-free survival, highlighting that positive LN disease is a poor prognostic factor in PCa.<sup>22,23</sup> Despite this finding with node-positive disease, dissection type was not a significant predictor. Furthermore, although more accurate staging of disease may be accomplished with E-PLND when compared to S-PLND, E-PLND may aid simply in identifying patients at higher risk for disease progression rather than offer a direct therapeutic effect.<sup>17,22,23</sup>

E-PLND has consistently shown inconclusive evidence from a BCR-free survival, metastasis-free survival, and CRPC-free survival perspective, but provides increased nodal staging accuracy.<sup>17</sup> Therefore, for select patient groups with intermediate- and high-risk PCa who may benefit from the improved nodal staging,

E-PLND may be considered. This decision must be patient-specific and made in the context of increased risk for intraoperative complications and blood loss.

Additionally, with the recent advent and adoption of prostate-specific membrane antigen-positron emission tomography (PSMA-PET), high-risk patients may be further assessed for nodal involvement in a non-invasive manner rather than receive upfront E-PLND.<sup>24-26</sup> With the potential option of non-invasive nodal staging through PSMA-PET, the benefits of E-PLND over S-PLND may be further diminished, favoring S-PLND or node-sparing approaches.<sup>25,26</sup>

Another promising alternative to E-PLND is location-based sentinel node dissection using magnetic-fluorescent hybrid tracers.<sup>8,9</sup> Through preoperative injection of these tracers, surgeons can identify involved lymph nodes for extirpation to avoid extensive PLND.<sup>8,9</sup> This approach has shown early success and serves as a potential way of extending PLND beyond S-PLND templates to resect suspicious LNs while minimizing the increased morbidity of extended dissection.<sup>8,9</sup> Preliminary studies have shown comparable BCR-free survival rates in patients who received sentinel node dissection of LNs outside of the standard template with patients who underwent E-PLND — further minimizing the benefits of E-PLND.<sup>8</sup>

### Limitations

It is important to acknowledge the limitations of this study. This study is retrospective in nature and the decision to pursue E-PLND was based on surgeon preference, which may impact the strength of the findings. Despite this, the proportion of patients undergoing E-PLND vs. S-PLND is consistent with practice patterns.<sup>11</sup>

As well, a standardized template-based definition of E-PLND or S-PLND was not used. Although this reflects a real-world practical example of PLND, it is possible that surgeons performing E-PLND may be under-resecting and diminishing any potential advantages of E-PLND. For categorization of PLND in this study, E-PLND boundaries were defined based on large-scale studies with similar patient populations and outcomes.<sup>4,5</sup> Variations in the extent of PLND and templates used must be considered when interpreting results among similar studies, which may have differing boundaries.

Additionally, whether dissected LNs are sent for pathologic evaluation in a cluster vs. individually may impact LN yield and outcomes in this study and understate the benefits of increased nodal extirpation. Of important consideration, although data were collected

from a Canadian database featuring 26 surgeons, certain participating centers collected and reported more cases, which may bias the results of this study to align more with practices and outcomes from certain participating surgeons/centers.

The sample size examined was large but unbalanced, with more patients undergoing S-PLND than E-PLND. Although measures were taken to account for this through analysis, the ability of statistical analysis to detect group differences is more limited. Additionally, patients in the E-PLND group were more likely to undergo salvage radiation treatment compared to the S-PLND group, which may have influenced oncologic outcomes despite controlling for salvage treatment in multivariate analyses.

There were also instances where care deviated from the standard of care, with 9.9% of S-PLND and 8.4% of E-PLND patients receiving adjuvant hormonal therapy, and 0.9% of E-PLND patients receiving neoadjuvant chemotherapy, which is not standard of care for localized PCa. This may bias outcomes. Nonetheless, the lack of benefit with regard to BCR, mPC progression, and CRPC development is consistent with similar studies.<sup>1,4</sup>

Furthermore, limitations exist due to the nature of this study. A formal RCT or a large, volume-matched analysis study would improve the generalizability of results and provide stronger recommendations on the benefits of more extensive PLND. Although this study provides a meaningful perspective on real-world PLND for PCa, generalizability to larger populations may be limited. As this study used a database approach, there is a chance that patient interventions and data collected may not reflect current practices, which may further bias results.

### CONCLUSIONS

In our study comparing E-PLND and S-PLND in patients undergoing RP, there were no significant differences in BCR, metastatic development, or CRPC progression between patient groups. Despite this, E-PLND was shown to have increased LN yield and positivity, which may provide benefit to patient groups who benefit most from more accurate nodal staging. Further research is needed to refine patient selection criteria for patients who may benefit from E-PLND.

**COMPETING INTERESTS:** Dr. Rendon has been an advisory board and speakers bureau member for and received honoraria from Abbvie, Amgen, Astellas, AstraZeneca, Bayer, BMS, EMD Serono, Ferring, Janssen, Mckesson, Pfizer, TerSera, and Tolmar; has participated in clinical trials supported by AA/Novartis, Astellas, AstraZeneca, Bayer, Ferring, Janssen, Myovant, Pfizer, and Point Biopharma; and has leadership roles in the Canadian Uro-

Oncology Group and Nova Scotia Cancer Care Program. Dr. Saad has been an advisory board member for and has received payment/honoraria from Amgen, Astellas, AstraZeneca, Bayer, Janssen, Knight, Myovant, Novartis, Pfizer, Sanofi, and Tolmar; and has participated in clinical trials supported by Amgen, Astellas, AstraZeneca, Bayer, Janssen, Novartis, Pfizer, and Sanofi. Dr. Shayegan has been an advisory board member for AbbVie, Astellas, Bayer, Ferring, Janssen, Knight, Merck, Pfizer, and TerSera; and has participated in clinical trials supported by Ipsen, Janssen, Merck, Myovant, and Pfizer. Dr. Mason has been an advisory board member for Abbvie, Bayer, Ferring, Sanofi, Sumitomo, Tercera, and Verity; has received payment from Abbvie, Astellas, Bayer, Janssen, Tercera, and Verity; has received honoraria from Bladder Cancer Canada/CUOG; is involved in multiple clinical trials; and is a specialty committee member for the RCPSC. The remaining authors do not report any competing personal or financial interests related to this work.

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