

Death in Patients With Recurrent Prostate Cancer After Radical Prostatectomy: Prostate-Specific Antigen Doubling Time Subgroups and Their Associated Contributions to All-Cause Mortality

Stephen J. Freedland, Elizabeth B. Humphreys, Leslie A. Mangold, Mario Eisenberger, Frederick J. Dorey, Patrick C. Walsh, and Alan W. Partin

From the Departments of Urology and Oncology, The James Buchanan Brady Urological Institute, Johns Hopkins Medicine, Baltimore, MD; Department of Surgery, Veterans Administration Medical Center Durham; Division of Urologic Surgery and Duke Prostate Center, Departments of Surgery and Pathology, Duke University School of Medicine Durham, NC; and The Biostatistics Core, University of Southern California, Keck School of Medicine at Childrens Hospital Los Angeles, Los Angeles, CA.

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Address reprint requests to Stephen Freedland, MD, DUMC Box 3850, Duke University Medical Center, Durham, NC 27710; e-mail: steve.freedland@duke.edu.

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ABSTRACT

Purpose

Among patients with biochemical recurrence after radical prostatectomy, we found previously that postoperative prostate-specific antigen doubling time (PSADT) was associated with risk of prostate cancer death. However, given the small number of patients in the highest risk PSADT subgroup, it is unclear which PSADT subgroups contribute the greatest to prostate cancer-specific death and how this influences all-cause mortality.

Patients and Methods

This study was a retrospective analysis of 379 patients treated with radical prostatectomy between 1982 and 2000 who had a biochemical recurrence and PSADT data available. Mean and median follow-up after surgery was 11.4 (standard deviation, 5.4) and 11.0 years, respectively (range, 1.6 to 23.0 years).

Results

Shorter PSADT was significantly associated with prostate cancer-specific and all-cause mortality ($P < .001$). Although patients with a PSADT less than 3 months were at the greatest risk of death, because of the limited number of patients in this group, they accounted for only 13% of prostate cancer deaths at 15 years after biochemical recurrence, whereas patients with an intermediate PSADT (3.0 to 8.9 months) accounted for 58% of all prostate cancer deaths. Among patients with a PSADT less than 15 months, prostate cancer accounted for 90% of all deaths. Only patients in the slowest PSADT subgroup (≥ 15 months) had a greater risk of competing-causes mortality compared with that from prostate cancer.

Conclusion

Among a select cohort of young, healthy patients with PSA recurrence after radical prostatectomy and a PSADT less than 15 months, prostate cancer accounted for an estimated 90% of all deaths by 15 years after recurrence. The majority of prostate cancer deaths occurred among patients with an intermediate PSADT (3.0 to 8.9 months).

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INTRODUCTION

Although radical prostatectomy (RP) offers excellent long-term cancer control, one in three patients experience a prostate-specific antigen (PSA) recurrence within 10 years.¹⁻⁴ Many patients have an indolent course with a median time from recurrence to prostate cancer death of 16 years.⁵ However, prostate cancer patients today are often younger⁶ and have an extended natural life expectancy with fewer competing mortality causes. Thus, a slowly progressive clinical course may result in prostate cancer death. Moreover, not all patients experience indo-

lent disease progression; some patients experience rapid progression and early death.

To stratify patients according to risk, we studied previously 379 patients with PSA recurrence after RP and identified three risk factors for prostate cancer-specific mortality: time to PSA recurrence, postrecurrence PSA doubling time (PSADT), and pathologic Gleason sum.⁵ Among these factors, PSADT was the strongest prognostic factor. Similar to results from other studies, we found that PSADT less than 3 months was strongly associated with prostate cancer-specific mortality.⁷ However, the number of patients in this high-risk category was small (6% in our cohort). Thus, if this group of men,

due to the limited numbers, constitutes only a small percentage of all prostate cancer deaths, then focusing aggressive secondary treatments and clinical trials solely on these men will have a limited impact on reducing overall prostate cancer mortality.

In this study, we sought to test the age-old adage that most patients die with their prostate cancer not as a result of it, by identifying the PSADT group that had the greatest contribution to prostate cancer-specific and all-cause mortality. To accomplish this, we used the same cohort of 379 patients with PSA recurrence we described previously, but with slightly longer follow-up.⁵ We focused on PSADT because in our prior analyses PSADT was the strongest risk factor for prostate cancer death⁵ and has been associated consistently with prostate cancer death in other series.⁷⁻⁹

PATIENTS AND METHODS

Patient Population

The institutional review board at Johns Hopkins University (Baltimore, MD) approved this study, and when required, written informed consent was obtained. We identified 5,100 patients with prostate adenocarcinoma treated by RP at the Johns Hopkins Hospital from April 1982 to December 2000 with follow-up data available. During a mean follow-up of 6.3 (standard deviation [SD], 4.5) and median follow-up of 5 years, 997 patients (20%) developed a biochemical recurrence (single postoperative PSA ≥ 0.2 ng/mL).¹⁰ Of these 997 patients, 411 patients had data available to calculate PSADT (\geq two PSA values separated by ≥ 3 months within 2 years after recurrence and no adjuvant radiation or hormonal therapy before recurrence). There were no significant differences between those with known or unknown PSADT data for time to recurrence (log-rank, $P = .53$) or time from recurrence to prostate cancer-specific (log-rank, $P = .41$) or all-cause mortality (log-rank, $P = .16$).

Of the 411 patients with PSA recurrence and known PSADT data, those who received preoperative radiation ($n = 2$) or hormonal therapy ($n = 10$) were excluded. Patients who received salvage radiation with a durable PSA response (> 2 years) were considered to have local-only recurrence and to have been cured by surgery plus radiation, and were excluded ($n = 20$). Patients who underwent salvage radiation but did not achieve a durable PSA response were considered to have distant failure and were included ($n = 22$). These exclusions resulted in a total of 379 patients.

In general, our postoperative surveillance was as follows: PSA determinations and rectal examinations every 3 months for year 1, semiannually for year 2, and yearly thereafter. After PSA recurrence, PSA was measured every 6 to 12 months. PSA values were obtained either at Johns Hopkins Hospital or at a local laboratory near the patient. In general, but not always, the same laboratory and assay were used to measure PSA values in a given patient.

Prostate cancer death was defined as death in any patient with metastasis that showed progression after hormonal therapy or death in any patient not treated with hormonal therapy but with widespread metastases without another obvious cause of death.

Determination of PSADT

PSADT was calculated by the natural log of 2 (0.693) divided by the slope of the linear regression line of natural log of all PSA values obtained within the first 2 years after biochemical recurrence.¹¹ Thus, PSADT was not calculated until after PSA recurrence and all PSA values used were ≥ 0.2 ng/mL. The mean and median number of PSA values used to calculate PSADT was 4.1 (SD, 1.7) and 4, respectively (range, two to 11). Patients ($n = 7$) with a negative or zero PSADT (no increase in PSA) were assigned a PSADT of 100 months. All PSA values were obtained before subsequent therapy (radiation or hormonal). In a subset ($n < 100$), frozen banked sera collected at the time of routine postoperative visits before the introduction of the PSA assay were used to measure PSA retrospectively.

Statistical Analysis

The distribution of clinicopathologic characteristics across PSADT categories was assessed using the χ^2 test for categorical variables and analysis of variance for continuous variables. Preoperative PSA (logarithmically transformed), age, and time to recurrence were considered as continuous variables. Clinical stage (T1, T2, and T3), Gleason sum (2 to 6, 7, and 8-10), race (white and nonwhite), and binary adverse pathologic features were considered as categorical variables.

The association between PSADT and time from recurrence to death was examined using Cox proportional hazards regression. For multivariable analysis, a forward-stepwise Cox proportional hazards model was used with $P < .15$ determining which variables to enter into the model. The variables considered for entry included preoperative PSA, clinical stage (T1 v T2/3), surgical margin status, extraprostatic extension, seminal vesicle invasion, lymph node metastasis, pathologic Gleason sum (≤ 7 v ≥ 8), age at recurrence (continuous), time from surgery to recurrence (≤ 3 years v > 3 years), and PSADT. The analyses were repeated using a backwards-stepwise selection to ensure that the results were insensitive to the statistical approach used. The possibility of a time effect was examined by comparing the Kaplan-Meier survivorship curves for patients treated before 1990 versus in 1990 or later. Given that these curves were almost identical and the Cox model estimates were also representative of the curves, we concluded that any time effect in these data was minimal. Moreover, year of surgery as a continuous variable was unrelated to risk of prostate cancer-specific ($P = .83$) or all-cause death ($P = .29$).

The proportional hazards assumption of the Cox model was tested through the graphical examination of the log-log plots of the variables used in the model. These plots formed approximate parallel straight lines as required. In addition, internal validation of the model was tested by comparing the Kaplan-Meier and Cox estimated values for several subsets that were defined using factors not included in the Cox model. In these cases, the estimated points at the death times appeared randomly scattered about the Kaplan-Meier curves. The predictive performances of PSADT and the entire multivariable model for risk of prostate cancer-specific and overall survival were assessed using the concordance index C .¹² Competing risks analysis¹³ and nonparametric CIs¹⁴ were used to assess the risk of prostate cancer-specific deaths and deaths not related to prostate cancer.

Given that in some men, the first documented elevated PSA value was more than 0.2 ng/mL, it was impossible to determine the earliest date at which the PSA was exactly 0.2 ng/mL. As such, the true recurrence date for some men was unknown. Given that all analyses used recurrence time as time zero, we evaluated whether this uncertainty regarding the true recurrence date affected our results. Specifically, we used the value of the first elevated PSA and the PSADT (assuming equivalently that the increase in PSA follows an exponential distribution) to estimate the time at which the PSA value would have been 0.2 ng/mL. Men in whom this estimated time was before surgery were assigned a recurrence date of 1 month. When using this estimated recurrence date, the 15-year survival estimates and hazard ratios all fell within the 95% CI of the values when the documented recurrence date was used (data not shown). Therefore, the documented recurrence date was used throughout. All statistical analyses were performed using STATA 9.1 (Stata Corp, College Station, TX).

RESULTS

Association Between PSADT and Clinicopathologic Characteristics

Patients with a shorter PSADT had earlier PSA recurrences ($P < .001$), higher biopsy ($P < .001$) and RP Gleason sums ($P < .001$), and were less likely to have positive surgical margins ($P = .06$) or extraprostatic extension ($P = .04$) but more likely to have seminal vesicle invasion ($P = .01$) or lymph node involvement ($P = .005$; Table 1). Short PSADT was associated significantly with younger age at PSA recurrence ($P < .001$) but not age at surgery ($P = .55$).

Table 1. Clinical and Pathologic Features of Patients Undergoing Radical Prostatectomy With a PSA Recurrence Segregated by Postrecurrence PSADT

Feature	PSADT (months)								P*
	< 3.0		3.0-8.9		9.0-14.9		≥ 15.0		
	No.	%	No.	%	No.	%	No.	%	
No. of patients	23	6	119	31	79	21	158	42	
Years to PSA recurrence									< .001
Median		0.7		1.1		2.8		3.2	
Mean		1.5		1.9		3.5		4.0	
SD		1.8		2.3		3.0		3.4	
Race									.61
White	23	100	112	94	74	94	151	96	
Nonwhite	0	0	7	6	5	6	7	4	
Age, years									
At surgery									.55†
Mean		59.4		59.1		59.9		60.2	
SD		7.7		5.6		6.0		6.5	
At PSA recurrence									< .001†
Mean		61.4		61.5		63.9		65.0	
SD		8.5		5.8		6.9		7.6	
Preoperative PSA, ng/mL									.65†
Median		8.5		10.8		11.2		10.6	
Mean		10.4		15.3		12.6		15.3	
SD		7.9		16.2		7.5		17.5	
Biopsy Gleason sum									< .001
2-6	8	36	41	35	42	53	82	53	
7	5	23	55	47	27	34	59	38	
8-10	9	41	21	18	10	13	13	8	
Clinical stage									.48
T1	7	32	22	19	16	21	28	18	
T2	13	59	86	74	59	76	120	78	
T3	2	9	9	8	3	4	6	4	
Pathologic Gleason sum									< .001
2-6	1	4	5	4	10	13	38	24	
7	4	17	63	53	45	57	83	53	
8-10	18	78	51	43	24	30	37	23	
Positive surgical margins	9	39	45	38	43	54	81	51	.06
Extraprostatic extension	18	78	102	86	76	96	141	89	.04
Seminal vesicle invasion	8	35	57	48	32	41	46	29	.01
Lymph node involvement	9	39	53	45	22	28	40	25	.005

Abbreviations: PSA, prostate-specific antigen; PSADT, PSA doubling time; SD, standard deviation.

*P value by χ^2 except where noted.

†P value by analysis of variance.

Prognostic Factors for Prostate Cancer–Specific and All-Cause Mortality

Mean and median time to recurrence was 3.1 (SD, 3.1 years) and 2.0 years, respectively. Mean and median follow-up after surgery was 11.4 (SD, 5.4 years) and 11.0 years, respectively (range, 1.6 to 23.0 years). Mean and median follow-up after recurrence was 8.2 (SD, 4.4 years) and 7.3 years, respectively. During this time, 79 (21%) patients died as a result of prostate cancer and 23 patients (6%) died as a result of other causes. The 5-, 10-, and 15-year overall all-cause survival rates were 94% (95% CI, 91% to 96%), 72% (95% CI, 66% to 78%), and 54% (95% CI, 45% to 61%), respectively (Fig 1A).

Shorter PSADT, earlier biochemical recurrence, and pathologic Gleason sum ≥ 8 were all associated with prostate cancer–specific and all-cause mortality (Table 2). Older age at recurrence was associated with all-cause but not prostate cancer–specific mortality. The concor-

dance index C of PSADT as a continuous variable to estimate time to prostate cancer–specific and all-cause death was 0.82 and 0.75, respectively, compared with 0.83 and 0.72 for the full multivariable model that included all significant risk factors.

We explored various PSADT cut points by dividing patients into groups based on 3-month increments in PSADT: less than 3.0, 3.0 to 5.9, 6.0 to 8.9, 9.0 to 11.9 months, and so on. The PSADT groups were then examined as a categoric variable in multivariable analysis and categories with similar hazard ratios for prostate cancer–specific death were combined. Analogous to our prior analysis,⁵ this resulted in PSADT being divided into the following groups: less than 3.0, 3.0 to 8.9, 9.0 to 14.9, ≥ 15.0 months (Table 2). The concordance index C of PSADT as a categoric variable to estimate time to prostate cancer–specific and all-cause death was 0.81 and 0.74, respectively, compared with 0.83 and 0.77 for the full multivariable model that included all significant risk factors.

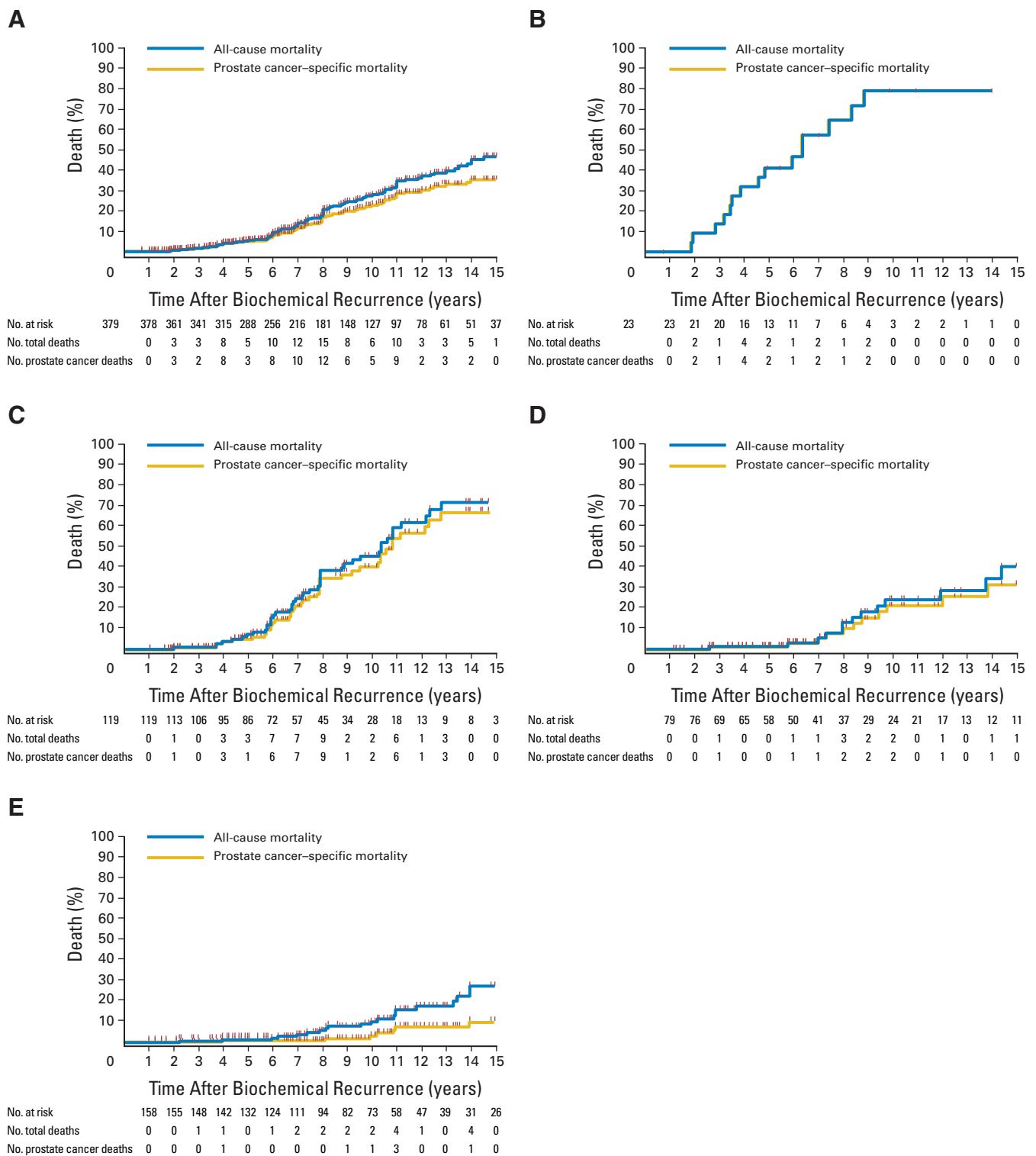


Fig 1. Fifteen-year actuarial Kaplan-Meier prostate cancer-specific and all-cause estimated risk of death among patients with a prostate-specific antigen (PSA) recurrence after radical prostatectomy. (A) All patients with PSA recurrence; (B) patients with a PSA doubling time (PSADT) less than 3 months; (C) patients with a PSADT 3.0 to 8.9 months; (D) patients with a PSADT less than 9.0 to 14.9 months; (E) patients with a PSADT \geq 15 months. Please note that in (B) the lines for prostate cancer-specific and all-cause mortality are superimposed.

Table 2. Cox Proportional Hazards Analysis of Factors Predicting Time From PSA Recurrence to Prostate Cancer–Specific and All-Cause Death

Factor	HR	95% CI	β	SE	P
PSADT as a continuous variable					
Time to prostate cancer–specific mortality					
PSA doubling time (in months)	0.92	0.89 to 0.95	−0.09	0.02	< .001
Pathologic Gleason sum (≥ 8 v < 8)	1.42	1.13 to 1.77	0.35	0.11	.002
Years from surgery to PSA recurrence (≤ 3 v > 3)	2.28	1.23 to 4.24	0.83	0.32	.01
Time to all-cause mortality					
PSA doubling time, months	0.98	0.97 to 0.99	−0.02	0.01	.01
Pathologic Gleason sum (≥ 8 v < 8)	1.33	1.09 to 1.62	0.29	0.10	.005
Older age at PSA recurrence	1.03	1.00 to 1.07	0.03	0.02	.04
Years from surgery to PSA recurrence (≤ 3 v > 3)	1.58	0.99 to 2.53	0.46	0.24	.06
PSADT as a categorical variable					
Time to prostate cancer–specific mortality					
PSA doubling time, months (relative to ≥ 15.0 months)					
< 3.0	24.89	10.56 to 58.65	3.21	0.42	< .001
3.0-8.9	8.01	3.90 to 16.44	2.08	0.37	< .001
9.0-14.9	3.09	1.36 to 7.02	1.13	0.44	.01
Pathologic Gleason sum (≥ 8 v < 8)	1.35	1.07 to 1.71	0.30	0.12	.01
Years from surgery to PSA recurrence (≤ 3 v > 3)	2.55	1.35 to 4.82	0.94	0.32	.004
Time to all-cause mortality					
PSA doubling time, months (relative to ≥ 15.0 months)					
< 3.0	11.46	5.69 to 23.09	2.44	0.36	< .001
3.0-8.9	4.35	2.57 to 7.35	1.47	0.27	< .001
9.0-14.9	1.53	0.80 to 2.91	0.42	0.33	.20
Pathologic Gleason sum (≥ 8 v < 8)	1.21	0.98 to 1.49	0.19	0.11	.07
Older age at PSA recurrence	1.05	1.01 to 1.09	0.05	0.02	.01
Years from surgery to PSA recurrence (≤ 3 v > 3)	1.65	1.00 to 2.71	0.50	0.25	.05

Abbreviations: PSA, prostate-specific antigen; HR, hazard ratio; PSADT, PSA doubling time; SE, standard error of the β .

PSADT and Risk of Prostate Cancer–Specific and All-Cause Death at 15 Years Postrecurrence

Using actuarial competing risk analysis, the 15-year postrecurrence estimated number of prostate cancer–specific and all-cause deaths among the 379 patients are shown in Table 3. When stratified by PSADT, prostate cancer was estimated to account for $\geq 92\%$ of deaths among patients with a PSADT less than 9 months (Fig 1B and 1C), 78% (Fig 1D) of deaths among men with a PSADT of 9.0 to 14.9 months, but only 35% of deaths among patients with a PSADT ≥ 15 months (Fig 1E).

Although patients with a PSADT less than 3 months were at the greatest risk of prostate cancer–specific death, because of the small numbers in this group (n = 23; 6%), these patients were estimated to

account for only 13% (95% CI, 7% to 20%) of prostate cancer–specific deaths. Men with a PSADT 3.0 to 8.9 months, because of the larger numbers in this group (n = 119; 31%) and the elevated risk of prostate cancer–specific death relative to longer PSADT groups, were estimated to account for 58% (95% CI, 38% to 89%) of prostate cancer–specific deaths. Similar patterns were observed when all-cause death was estimated, with patients with a PSADT 3.0 to 8.9 months accounting for 48% (95% CI, 33% to 68%) of deaths, whereas patients with a PSADT less than 3 months accounted for 10% (95% CI, 6% to 15%) of deaths.

Effect of Early Hormonal Therapy

Of the 379 patients, 54 received hormonal therapy before metastasis. There was no significant association between PSADT and early

Table 3. Fifteen-Year Actuarial Competing Risk Estimate of Number of Total Deaths, Prostate Cancer–Specific Deaths, and Deaths Not Caused by Prostate Cancer Stratified by PSADT Among Patients Who Experienced a Biochemical Recurrence After Radical Prostatectomy (N = 379)

Prostate Cancer Stratified by PSA at Timing Patients Who Experienced a Biochemical Recurrence After Radical Prostatectomy (N = 379)									
PSADT (months)	No. of Patients	Estimated Deaths						Portion of All-Cause Mortality Caused by Prostate Cancer	
		Total		Caused by Prostate Cancer		Not Caused by Prostate Cancer			
		No.	95% CI	No.	95% CI	No.	95% CI	%	95% CI
< 3.0	23	18	13 to 22	18	11 to 21	0	0 to 3	100	80 to 100
3.0-8.9	119	85	69 to 100	79	60 to 93	6	2 to 14	93	81 to 98
9.0-14.9	79	32	19 to 50	25	13 to 39	7	1 to 20	78	39 to 98
≥ 15.0	158	43	28 to 65	15	6 to 30	28	15 to 45	35	12 to 67
All patients	379	178	146 to 208	137	105 to 160	41	25 to 66	77	61 to 86

Abbreviation: PSADT, prostate-specific antigen doubling time.

hormonal therapy (χ^2 , $P = .47$). Analogous to when the entire cohort was examined, when patients treated with early hormonal therapy were excluded, patients with a PSADT of 3.0 to 8.9 months had the greatest contribution to prostate cancer–specific and all-cause mortality, and the 15-year actuarial estimates of all-cause and prostate cancer deaths closely mirrored the findings from the entire cohort.

DISCUSSION

We reported previously the natural history of recurrent prostate cancer can be indolent, with a 16-year median time to death as a result of prostate cancer.⁵ Although the current analyses confirm the often slow course of recurrent prostate cancer, they suggest that of patients who die during the first 15 years after recurrence, 77% of deaths are attributable to prostate cancer. Moreover, prostate cancer accounted for 90% of deaths among patients with a PSADT less than 15 months. Although patients with a PSADT less than 3 months were at the greatest risk of prostate cancer–specific and overall death, because of small numbers in this group, they accounted for only 13% of prostate cancer deaths. Only among patients with a PSADT ≥ 15 months was the risk of death as a result of prostate cancer low enough that competing causes of mortality were greater. These findings suggest that with continued follow-up, recurrent prostate cancer in young healthy patients is often a fatal disease and most patients in the current study with a PSA recurrence after RP died as a result of their cancer, rather than with their cancer.

PSADT is strongly associated with progression^{11,15,16} and prostate cancer–specific death.^{5,7-9} However, among our select cohort, only 6% of patients were in the highest risk PSADT group. In less-select cohorts (ie, military and community settings), 12% of patients with recurrence after surgery had PSADT less than 3 months.⁷ Thus, regardless of the practice setting, this highest risk group represents a minority. The high risk of death as a result of prostate cancer among this group translates into the fact that with short follow-up, most deaths occur among this group. However, with longer follow-up, patients with slower PSADT begin to die, and by 15 years after recurrence, the group with PSADT less than 3 months is estimated to account for only 13% of all deaths as a result of prostate cancer.

The age-old adage is that patients are more likely to die with rather than as a result of their prostate cancer. However, patients today are younger, with fewer competing causes of mortality. A study from Cancer of the Prostate Strategic Urologic Research Endeavor (CaPSURE), a community based prostate cancer registry, found that the average age at diagnosis was 65 years and 28% were younger than age 60 years.⁶ The average age at RP in contemporary series is 57 to 61 years, depending on the practice setting.¹⁷⁻²⁰ Thus, although the current historical study represents a select group of young, healthy patients, the similarity of age (59 years) to the age of contemporary patients suggests that the current data may be relevant to present patients.

Median life expectancy for a 60-year-old male is 20.3 years.²¹ Given this long natural life expectancy, events occurring 15 years or later after recurrence can influence overall survival. Indeed, among patients with a slow to intermediate PSADT (9.0 to 14.9 months), given a 15-year follow-up, 41% of patients were expected to die, with 78% of deaths attributable to prostate cancer. Alternatively, older patients face greater competing mortality risks and only those at the highest risk of early death as a result of prostate cancer are likely to die as a result of prostate cancer. The importance of age as a prognostic

factor was highlighted by the fact that age at recurrence was significantly related to all-cause mortality. In the future, if age at surgery continues to decline and life expectancy continues to improve, and in the absence of improved secondary therapies, it is possible that prostate cancer–specific mortality will have an even greater contribution to all-cause mortality among patients with PSA recurrences.

We focused our analyses on PSADT because it has been linked consistently with prostate cancer–specific death among patients with recurrence after RP.^{5,7-9} Indeed, PSADT alone had a concordance index C for estimating prostate cancer–specific and all-cause death of 0.81 and 0.74, respectively. Moreover, although on multivariable analysis time to recurrence and Gleason sum were also independent factors, their contribution to risk assessment was small, as evidenced by only modest improvements in the concordance index C when these factors were added to the model. It is important to recognize that all three prognostic factors are highly correlated. Thus, it is difficult to determine the true independent contribution of each factor.

In the current study, 54 patients received hormonal therapy before metastasis. Exclusion of these patients did not materially change our findings. The current study in which most patients did not receive early hormonal therapy allows a unique insight into the natural history of prostate cancer. Because hormonal therapy can delay metastasis,^{22,23} many patients today receive early hormonal therapy, although whether this affects prostate cancer–specific or overall survival remains controversial.

PSA and PSADT are time-dependent factors. We calculated PSADT assuming that PSA increased exponentially during the first 2 years after recurrence. Therefore, although the PSADT may not have been calculable in some patients until the end of the second year because no second PSA value was available until then, the calculated PSADT at that point was presumed to be the PSADT at the time of biochemical recurrence. However, it is plausible that in the long term, PSADT values may not be stable. Future studies are needed to address whether changes in PSADT over time reflect changing risk of death as a result of prostate cancer. The risk of non–prostate cancer death is highly dependent on patient age. As such, the fact that the patients in the current series were relatively young and healthy likely accounted for the low risk of non–prostate cancer death. Therefore, the estimates in the current study may not apply to older patients with greater risk of competing mortality. The overall numbers of patients and deaths in the current study are small. Consequently, the CIs for some subsets are high. In particular, because the Gleason sum, time to recurrence, and PSADT are highly related, the CIs generated in Table 2 must be viewed with caution. Finally, these results need to be confirmed in other studies. Until that time, these results should be viewed as preliminary.

In conclusion, among a select cohort of young, healthy patients treated with RP who experienced a biochemical recurrence, 15-year actuarial survival estimates suggest that death as a result of prostate cancer will account for 77% of all deaths and 90% of deaths among patients with a PSADT less than 15 months. Although patients with a PSADT less than 3 months were at the greatest risk of death as a result of prostate cancer, the majority of deaths as a result of prostate cancer occurred among patients with a PSADT of 3.0 to 8.9 months.

AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

Although all authors completed the disclosure declaration, the following author or immediate family members indicated a financial interest. No

conflict exists for drugs or devices used in a study if they are not being evaluated as part of the investigation. For a detailed description of the disclosure categories, or for more information about ASCO's conflict of interest policy, please refer to the Author Disclosure Declaration and the Disclosures of Potential Conflicts of Interest section in Information for Contributors.

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AUTHOR CONTRIBUTIONS

Conception and design: Stephen J. Freedland, Mario Eisenberger, Patrick C. Walsh, Alan W. Partin

Financial support: Alan W. Partin

Administrative support: Elizabeth B. Humphreys, Leslie A. Mangold

Provision of study materials or patients: Mario Eisenberger, Patrick C. Walsh, Alan W. Partin

Collection and assembly of data: Stephen J. Freedland, Elizabeth B. Humphreys, Leslie A. Mangold, Patrick C. Walsh, Alan W. Partin

Data analysis and interpretation: Stephen J. Freedland, Frederick J. Dorey

Manuscript writing: Stephen J. Freedland, Mario Eisenberger, Frederick J. Dorey, Patrick C. Walsh, Alan W. Partin

Final approval of manuscript: Stephen J. Freedland, Elizabeth B. Humphreys, Leslie A. Mangold, Mario Eisenberger, Frederick J. Dorey, Patrick C. Walsh, Alan W. Partin

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