CHAPTER 17 **Testicular Cancer**

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INTRODUCTION

The incidence of testicular cancer is increasing. In 2005, approximately 8,000 new cases were diagnosed in the United States (1). Although it constitutes less than 1% of all malignancies in males, testicular cancer is the most common cancer in men 20 to 34 years of age. Because of advances in therapy, overall survival rates are high—and the management of testicular cancer represents a paradigm for successful multimodality therapy.

Modifications in both surgical and radiation techniques, as well as improved methods of employing systemic chemotherapy, have substantially diminished the morbidity of therapy. Nonetheless, the sequelae of multimodality therapy are not insignificant and can have broad and far-reaching consequences with regard to general health, reproduction, and economic productivity.

The small numbers of cases, changing treatment practices coupled with an absence of standardized therapy, the young age of affected patients, and the relative paucity of these patients in databases makes evaluation of trends in the treatment of testicular cancer difficult. The diagnostic and procedure codes commonly associated with the disease are listed in Table 1. The narrow scope of these codes underscores the limitations to collecting and analyzing information. More comprehensive and detailed data are needed to provide a better understanding of the impact of this cancer on health and prosperity.

DEFINITIONS AND DIAGNOSIS

The term *testicular cancer* characteristically refers to seminomatous and non-seminomatous germ cell tumors, as these constitute nearly 98% of primary neoplasms of the testes (2). These malignancies arise in the germinal cells of the testes. Other tumors can arise from stromal, mesenchymal, and adnexal elements of the testes, but such tumors are exceedingly uncommon and generally carry an excellent prognosis. Metastatic spread of other cancers to the testes is rare, although involvement by lymphoma and leukemia may occur. Diagnosis is usually made on physical examination and confirmed with scrotal ultrasound.

Histologic Classification

Seminomas are the most common testicular germ cell tumor (3). They are derived from spermatogenic cells within the seminiferous tubules and recapitulate certain aspects of spermatogenesis. Although they are subclassified by histology, their management is relatively uniform across the subtypes. Nonseminomatous germ cell tumors (NSGCTs) are also derived from spermatogenic cells. They are progeny of pluripotent embryonic cells formed from germ cells through the process of parthenogenesis. They encompass a variety of histologic patterns and can develop as a single entity or as a combination of choriocarcinoma, embryonal, teratoma, and yolk sac elements. NSGCTs can occur in conjunction with seminomas. Each subtype behaves differently, and this has implications for prognosis, as well as for therapy. Diagnosis is based on tissue removed during

Table 1. Codes used in the diagnosis and management of testicular cancer -

	ies used in the diagnosis and management of testicular cancer
Males with o	one or more of the following:
ICD-9 d	iagnosis codes
186	Malignant neoplasm of testis
186.0	Malignant neoplasm of undescended testis
186.9	Malignant neoplasm of other and unspecified testis
CPT pro	ocedure codes
38564	Limited lymphadenectomy for staging (separate procedure); retroperitoneal (aortic and/or splenic)
38780	Retroperitoneal transabdominal lymphadenectomy, extensive, including pelvic, aortic, and renal nodes (separate procedure)

54530 Orchiectomy, radical, for tumor; inguinal approach

54535 Orchiectomy, radical, for tumor; with abdominal exploration radical orchiectomy or, rarely, when a metastatic site is biopsied.

Serum Tumor Markers

In addition to histopathologic classifications, some testicular tumors are associated with the production of α -fetoprotein (AFP) by trophoblastic cells and β -human chorionic gonadotrophin (HCG) by syncytiotrophoblasts (4). Serologic measurements of HCG and AFP are used in the diagnosis and management of testicular cancer. AFP is never produced by pure seminomas or choriocarcinomas. HCG production is seen in all choriocarcinomas, most embryonal tumors, and occasional seminomas. Both AFP and HCG can be used to monitor response to therapy when they are present at the time of initial diagnosis. In addition, measurements of the enzyme lactate dehydrogenase (LDH) are often used in prognostication, as LDH correlates with tumor burden.

Staging

Once a diagnosis of testicular cancer is made (almost always after radical orchiectomy: the removal of the testis and spermatic cord), treatment decisions are primarily based on clinical staging, which usually consists of computed tomography (CT) imaging of the chest, abdomen, and pelvis. Patterns of metastatic spread in testicular cancer are very predictable, and the reproducibility of expected "landing sites" aids

Table 2. Staging systems for testicular car	ncer	
Conventional Clinical Staging System		AJCC Staging System
	Primary tur	nor (T)
I: Confined to testis	pTX:	Primary tumor cannot be assessed
	pT0:	No evidence of primary tumor
	pTis:	Intratubular germ cell neoplasia (carcinoma in situ)
	pT1:	Tumor limited to the testis and epididymis without vascular/lymphatic invasion; tumor may invadeinto the tunica albuginea but not the tunica vaginalis
	pT2:	Tumor limited to the testis and epididymis with vascular/lymphatic invasion, or tumor extending through the tunica albuginea with involvement of the tunica vaginalis
	рТ3:	Tumor invades the spermatic cord with or without vascular/lymphatic invasion
	pT4:	Tumor invades the scrotum with or without vascular/lymphatic invasion
	Regional ly	mph nodes (N)
II: Retroperitoneal Spread	NX:	Regional lymph nodes cannot be assessed
IIa: positive nodes, no node > 2 cm	N0:	No regional lymph node metastasis
IIb: positive nodes between 2 and 5 cm	N1:	Metastasis with a lymph node mass 2 cm or less in greatest dimension; or
IIc: positive nodes > 5 cm		multiple lymph nodes, none more than 2 cm in greatest dimension
	N2:	Metastasis with a lymph node mass more than 2 cm but not more than 5 cm in greatest dimension; or multiple lymph nodes, any one mass greater than 2 cm but not more than 5 cm in greatest dimension
	N3:	Metastasis with a lymph node mass more than 5 cm in greatest dimension
	Distant me	tastasis (M)
III. Metastatic: supraclavicular or mediastinal	MX:	Distant metastasis cannot be assessed
	M0:	No distant metastasis
	M1:	Distant metastasis
	M1a:	Non-regional nodal or pulmonary metastasis
	M1b:	Distant metastasis other than to non-regional lymph nodes and lungs
	Serum Tun	nor Markers (S)
	SX:	Marker studies not available or not performed
	S0:	Marker study levels within normal limits
	S1:	LDH < 1.5 x normal; and hCG < 5,000 mlu/ml; and AFP < 1,000 ng/ml
	S2:	LDH 1.5–10 x normal; or hCG 5,000–50,000 mlu/ml; or AFP 1,000–10,000 ng/ml
	S3:	LDH > 10 x normal; or hCG > 50,000 mlu/ml; or AFP > 10,000 ng/ml

SOURCE: Testis. In: American Joint Committee on Cancer.: AJCC Cancer Staging Manual. 6th ed. New York, NY: Springer, Copyright 2002, 317–322.

	198	35–1986 ^b	199	00–1991°	19	95–1996ª
	Early	Advanced	Early	Advanced	Early	Advanced
Seminoma						
Surgery Alone						
Excision of testicle without LND	4.7	1.4	13.9	3.3	16.8	4.4
Excision of testicle with LND	3.7	1.4	1.0	1.4	0.6	0.7
Orchiectomy, NOS	3.5	2.7	3.6	0.7	3.5	1.7
Surgery, NOS	3.9	0	0.5	0	0.1	0.2
Surgery and radiation						
Excision of testicle without LND	25.3	18.9	57.4	15.6	61.2	20.4
Excision of testicle with LND	12.0	8.1	2.2	2.5	1.7	1.2
Orchiectomy, NOS	17.2	9.5	12.4	5.8	11.1	4.9
Surgery, NOS	20.9	6.8	1.1	1.1	0.1	0.7
Surgery and chemotherapy						
Excision of testicle without LND	0.5	10.8	1.7	30.1	1.2	38.4
Excision of testicle with LND	0.5	5.4	0.3	4.3	0.1	4.1
Orchiectomy, NOS	0.8	2.7	0.8	8.7	0.6	7.3
Surgery, NOS	0.5	6.8	0.1	2.9	0	1.7
Other treatment modalities	5.7	21.6	4.0	22.1	1.9	13.6
No treatment indicated	0.7	4.1	1.1	1.4	0.5	0.5
Total	100	100	100	100	100	100
Cases, n	593	74	2,393	276	3,391	411
Nonseminomatous Germ Cell Tumor						
Surgery Alone						
Excision of testicle without LND	18.3	2.7	35.2	2.6	45.3	5.9
Excision of testicle with LND	12.6	1.6	21.4	3.2	17.6	3.6
Orchiectomy, NOS	13.3	2.1	9.2	2.1	9.5	1.6
Surgery, NOS	25.6	5.3	2.4	1.3	2.4	0.8
Surgery and radiation						
Excision of testicle without LND	0.7	0	0.4	0.1	0.6	0.1
Excision of testicle with LND	0.3	0	0	0	0.1	0
Orchiectomy, NOS	0.3	0	0.1	0	0.1	0
Surgery, NOS	0.3	0	0	0.1	0	0
Surgery and chemotherapy						
Excision of testicle without LND	5.6	28.3	13.6	38.4	15.0	45.6
Excision of testicle with LND	5.6	14.4	6.5	20.6	2.8	17.9
Orchiectomy, NOS	4.3	13.9	5.2	10.4	3.9	12.1
Surgery, NOS	8.0	18.7	1.2	7.0	0.5	3.5
Other treatment modalities	1.7	11.8	4.0	13.4	1.8	8.5
No treatment indicated	3.3	1.1	0.9	0.8	0.5	0.4
Total	100	100	100	100	100	100
Cases, n	301	187	1,207	719	1,542	827
•	_			6	,	

Table 3. Percent distribution of treatment modalities by histologic type of disease and stage of testicular cancera

...data not available.

LND, lymph node dissection; NOS, not otherwise specified.

^aEarly stage: 1985–1986 American Joint Committee on Cancer (AJCC) Stage I and II; 1990–1991 AJCC Stage I, II, and III; 1995–1996 AJCC Stage I, II NI; Advanced Stage: 1985–1986 AJCC Stage III and IV; 1990–1991 AJCC Stage IV; 1995–1996 AJCC Stage II (N2 or higher) and III.

^bCases staged according to the American Joint Committee on Cancer *Manual for Staging of Cancer*, 2nd edition.

°Cases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 3rd edition.

^dCases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 4th edition.

SOURCE: Reprinted from Cancer, 86, Steele, GS, Richie, JP, Stewart AK, and Menck HR, The National Cancer Data Base report on patterns of care for testicular carcinoma, 1985–1996, 2,171–2,184, Copyright 1999, with permission from Wiley.

tremendously with staging and treatment decisionmaking (5). In the simplest form, staging is divided into localized (testis only), retroperitoneal (minimal or bulky), and extralymphatic/extraretroperitoneal metastatic disease. The staging data are combined with levels of serologic tumor markers to guide management. The prevalence of several different staging schemas, as well as the complexity of the chosen tumor, nodes, and metastases (TNM) system, has confounded data acquisition and outcome reporting. The staging systems for testicular cancer referenced in this chapter and accepted by most practicing urologists are summarized in Table 2. Accurate clinical staging is crucial, as it is the basis for management decision-making.

The onset of testicular cancer is insidious, and there are few symptoms (5). Testicular tumors are usually discovered after the patient or his sexual partner palpates an abnormal mass. Patients may experience sensations of testicular heaviness or aching or may develop a reactive hydrocele. Rarely, pain is a presenting symptom. Occasionally, a patient presents with signs or symptoms of metastatic disease, including back pain, hydroureteronephrosis, and constitutional symptoms. The formal diagnosis of testicular cancer is based on histopathologic evaluation of the testis after orchiectomy or, rarely, biopsy of a retroperitoneal mass or subclavian lymph node (if the patient presents with metastatic disease and palpably normal testes). Such extraprimary/extragonadal disease constitutes less than 5% of cases.

When an obvious mass is present, no further diagnostic imaging is necessary, and the patient is brought to surgery for an orchiectomy. If the mass is subtle, ultrasonography may be performed to confirm its presence. Imaging of the chest and retroperitoneum is paramount for clinical staging and management decision-making, although it offers little with regard to diagnosis. Serum tumor markers are drawn prior to orchiectomy to aid with management. Occasionally, they can assist with diagnosis, as AFP is never produced by pure seminomas or choriocarcinomas. Prior to initial treatment, many physicians classify patients into "good," "intermediate," or "poor" prognostic groups to aid with management and help predict the probability of cure. Given the potential morbidity of treatment, many patients are advised to bank sperm prior to initiating treatment.

Screening

Other than genital examinations during routine physical examination, no formal screening algorithm exists for testicular cancer. Routine ultrasound examinations and serologic testing are not costeffective. Moreover, most men of the ages at greatest risk for developing testicular cancer do not routinely see a physician because of their general good health and because of limited access to healthcare, since many are students and hence are more likely to be uninsured.

Many men are hesitant to see a physician for evaluation even after self-detection of a testicular mass, because of the obvious fear of undergoing orchiectomy. Hence, a delay in diagnosis is not uncommon. Fortunately, current multimodal treatment yields excellent survival rates for men with all stages of the disease. Despite advocacy by some in the urologic community for men to perform monthly self-examinations of the testes (analogous to monthly breast examinations by women) to enable early diagnosis, evidence does not support this recommendation (6).

RISK FACTORS

Risk factors postulated for testicular cancer include cryptorchidism, trauma, prenatal exposure to hormones, familial and genetic factors, and occupational exposure (7). Interestingly, while the incidence of testicular cancer is increasing, the rate of increase in incidence is slowing. The reason for this deceleration is unclear. No formal prevention programs exist for testicular cancer, and cryptorchidism rates have remained stable for the past two decades (8). It is also unlikely that the gene pool has changed dramatically enough to decrease malignant transformation. Hence, none of these factors is likely responsible.

However, programs directed at increasing awareness and limiting the risk of trauma during driving, sports, and other leisure activities that may result in testicular trauma have been instituted. Moreover, these programs are coincidentally aimed at men in the age groups most susceptible to testicular cancer. Awareness of the dangers of prenatal and occupational exposure to toxins and hormones has also been enhanced. These factors may explain the slowing of the rate of increase in testicular cancer incidence.

TREATMENT

Once a diagnosis of testicular cancer is made, clinical staging is performed (Table 2). Several factors, including histopathology, serologic biomarkers, stage of disease, and the preference (or bias) of the providing physician and patient, dictate how an individual testicular cancer is treated. Treatment can involve any or all of the following: surgery, radiotherapy, chemotherapy, and surveillance. A recent review and consensus statement by the European Germ Cell Cancer Consensus Group provides excellent background material (9).

Surgery

Within the lexicon of testicular cancer therapies, surgery can refer to either radical orchiectomy or retroperitoneal lymph node dissection (RPLND), which involves resection of retroperitoneal lymphatic tissue for both staging and therapeutic purposes. This can occur *de novo* or after chemotherapy to remove a residual mass. Although almost all testicular cancer patients undergo orchiectomy, a small minority have an RPLND, and the extent of RPLND has changed, as has the indication for adjuvant systemic therapy post-RPLND (10). This complicates data analysis, as many databases refer to surgical treatment without specifying the type and scope of procedure or separating single- from combination-modality therapy.

Radiotherapy

Radiotherapy is limited to the treatment of seminoma and is used in several situations (9). For clinically localized disease (no lymphadenopathy on CT scanning), post-orchiectomy adjuvant therapy currently involves 20Gy of radiation to the infradiaphragmatic para-aortal and para-caval lymphatics. Alternatives include surveillance (given a nearly 100% cure rate with salvage therapy) with administration of irradiation or chemotherapy in cases of relapse. Radiotherapy is also used for patients with clinical stage IIA/B seminoma, in which 30–36Gy is given to the infradiaphragmatic para-aortal and para-caval and ipsilateral iliac lymphatics.

The recommended dose and field of abdominal radiotherapy have changed frequently in recent decades.

Systemic Therapy

Cisplatin-based chemotherapy is highly effective for the treatment of testicular cancer and can be used in several settings (9). For NSGCT, chemotherapy is used whenever tumor markers remain elevated postorchiectomy. In addition, it can be used in clinical stage I tumor-marker-negative disease (with the exception of teratoma) as an alternative to surgery or surveillance, or for salvage therapy after post-surveillance relapse. Chemotherapy can also be utilized as an alternative to surgery in NSGCT for clinical stage IIA/B tumormarker-negative disease. Chemotherapy is standard for all clinical stage IIC or greater NSGCT. For seminomas, chemotherapy is standard for all clinical stage IIC or greater tumors. It can also be used as an alternative to radiotherapy for clinical stage IIA/B seminomas. Finally, chemotherapy can be used as an adjuvant to RPLND if positive nodes are found at the time of resection.

Observation

Given the success rates of salvage therapy, some patients with clinical stage I tumor-marker-negative disease undergo close surveillance as primary treatment. This places tremendous responsibility on the patient and the physician, as monthly scans and blood tests are initially required.

Changes in Treatment Approaches

The National Cancer Data Base (NCDB), a joint project of the American Cancer Society and the American College of Surgeons, includes information that can be used to monitor changes in treatment approaches for testicular cancer between 1985 and 1996 (11). These changes are summarized in Table 3 and illustrated graphically in Figures 1a and 1b.

The management of seminoma has remained relatively consistent over the past decade. Approximately 75% of patients in the NCDB underwent radiotherapy after radical orchiectomy (11). However, a growing proportion of patients with clinical stage I disease are being treated initially by surgery alone (an increase from 15.8% in 1985–1986 to 21% in 1995–1996) (3). The use of surgery and



Figure 1a. Changing patterns of seminoma treatment by early or advanced stage in patients diagnosed in 1985–1996.





SOURCE: Adapted from Cancer, 86, Steele, GS, Richie, JP, Stewart AK, and Menck HR, The National Cancer Data Base report on patterns of care for testicular carcinoma, 1985–1996, 2,171–2,184, Copyright 1999, with permission from Wiley.

radiation remained stable at 76% in 1985–1986 to 74% in 1995–1996 during the period studied. As expected, the use of lymphadanectomy in seminoma is rare, at 0.6%. Chemotherapy is becoming the standard treatment for advanced seminoma after orchiectomy; its rate of use increased from 25.7% in 1985–1986 to 51.5% in 1995–1996 (Table 3) (11). Consequently, the rate of use of radiotherapy in higher-stage disease

decreased from 43.3% in 1985–1986 to 27.2% in 1995–1996 (3).

For patients with early-stage NSGCT, the NCDB data revealed an increase in the use of surgery as a single-modality therapy (from 69.8% in 1985–1986 to 75% in 1995–1996) (Table 3). While the use of RPLND increased (from 12.6% in 1985–1986 to 17.6 in 1995–1996), so did orchiectomy as a single therapy (from 18.3% in 1985–1986 to 45% in 1995–1996), again

reflecting the use of surveillance as a primary treatment, followed by salvage therapy if necessary (3). The increase in RPLND is not surprising, since refinements in technique have diminished the morbidity of this operation. The rate of use of chemotherapy in early disease has remained relatively stable at 22–23%. However, its rate of use for advanced NSGCT increased from 75% in 1985–1986 to 79% in 1995–1996 (3). This primarily reflects improved methods of supporting patients that make chemotherapy more tolerable. Certain aspects of therapy are not covered in the NCDB, including the use of laparoscopy and changes in the dosing of chemotherapeutic agents. These are expected to have a profound effect on management in the next decade.

PREVALENCE AND INCIDENCE

Testicular cancer constitutes less than 1% of all male cancers (12). According to the Surveillance, Epidemiology, and End Results (SEER) database, the age-adjusted incidence rate of testicular cancer from 1997 to 2001 was estimated to be 5.5 per 100,000 population. The lifetime prevalence count of testicular cancer on January 1, 2001, was 157,349. These data, as well as the racial disparity in testicular cancer between Caucasian and African American men, are clearly delineated in Table 4.

The overall incidence of testicular cancer in the United States has been steadily increasing (12). SEER

Table 4. Incidence and pre cancer, by race/ethnicity,	evalence rate 1997–2001	sª for testio	cular
	All Races	White Males	Black Males
Age-adjusted incidence rate	S		
All ages	5.5	6.5	1.5
< 65	6.2	7.2	1.6
65+	0.9	1.1	*
Prevalence Counts of Testic Cancer on Jan 1, 2001	ular		
Δ1I	157 340	150 181	2 948

*Figure does not meet standard for reliability or precision. aRate per 100,000 men of the same stratum.

SOURCE: Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L., Mariotto A, Feuer EJ, Edwards BK (eds). SEER Cancer Statistics Review, 1975-2001, National Cancer Institute. Bethesda, MD. http://seer.cancer.gov/csr/1975_ 2001/, 2004.

Table 5. Incidence ratesª for testicular cancer, age- adjusted, by age					
	All	< 50	≥ 50		
Year of Diagnosis					
1975	3.7	4.2	2.4		
1976	3.4	4.2	1.5		
1977	4.3	5.1	2.2		
1978	3.6	4.2	1.8		
1979	3.9	4.5	2.2		
1980	4.4	5.3	1.9		
1981	4.2	5.1	1.8		
1982	4.4	5.2	2.3		
1983	4.6	5.5	2.2		
1984	4.4	5.3	1.9		
1985	4.5	5.5	1.8		
1986	4.8	5.8	2.2		
1987	5.0	6.3	1.8		
1988	4.6	5.8	1.5		
1989	5.5	6.7	2.2		
1990	5.1	6.3	2.1		
1991	5.1	6.2	2.1		
1992	5.2	6.4	1.9		
1993	5.1	6.4	1.6		
1994	5.5	6.7	2.1		
1995	4.6	5.6	1.8		
1996	5.2	6.6	1.7		
1997	5.4	6.5	2.4		
1998	5.6	7.1	1.6		
1999	5.4	6.8	1.8		
2000	5.7	7.1	2.0		
2001	5.4	6.7	2.0		
1997–2001	5.5	6.9	2.0		

^aSeer 9 areas. Rates are per 100,000 and are age-adjusted to the 2000 standard population by 5-year age groups.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

data show although that the overall incidence of testicular germ cell tumors rose 46% between 1975 and 2001, from 3.7 per 100,000 to 5.4 per 100,000 population, the absolute change is relatively small (Table 5). This corresponds to an annual change of 1.5% across all populations under study. However, the rate of increase in incidence appears to be leveling. Over the past three decades, it has decreased from 22% to 15% to 4%, respectively. No formal testicular cancer prevention programs exist, so there is no obvious explanation for this diminution. It is possible that the

decrease is the indirect result of changes in behavior that influence risk factors, most specifically, programs directed at preventing trauma and at awareness of the hazards of maternal hormone exposure, although this has never been definitively studied.

Age

Testicular cancer is being diagnosed at an earlier age. Among men under 50 years of age in the SEER database (Table 5), the incidence of testicular cancer increased from 4.2 per 100,000 to 6.7 per 100,000

Table 6. Incidence rates ^a for testicular cancer, 1997–2001, age-adjusted and age-specific rates, by race/ethnicity				
	All Males	White Males	Black Males	
Age-specific Rates				
Age at Diagnosis				
<1				
1–4				
5–9				
10–14				
15–19	3.3	3.9		
20–24	9.4	11.3		
25–29	11.9	14.4		
30–34	13.6	16.2		
35–39	12.2	14.3	4.0	
40–44	10.2	12.0		
45–49	6.8	7.8		
50–54	4.1	4.7		
55–59	2.1	2.3		
60–64	1.6	1.8		
65–69				
70–74				
75–79				
80–84				
85+				
Age-adjusted Rates				
Age at Diagnosis				
All ages	5.5	6.5	1.5	
< 65	6.2	7.2	1.6	
65+	0.9	1.1		
data not available				

^aSEER 9 areas. Rates are per 100,000 and are age-adjusted to the 2000 standard population by 5-year age groups.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973-2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

between 1975 and 2001. During the same period, the incidence in men older than 50 decreased from 2.4 per 100,000 to 2.0 per 100,000. This reflects a shift in

diagnosis to younger men and is demonstrated by the SEER data, in which testicular cancer incidence is stratified by age at diagnosis (Table 6). McKiernan et al. reviewed similar SEER data from 1973-1995 and found that birth cohort was strongly associated with the relative risk of testicular cancer (12). They also demonstrated that the peak age at diagnosis has decreased for each successive birth cohort (Figure 2). This shift may reflect improved physician education, a greater emphasis on making young and teenage boys more aware of their own health issues, and the dissemination of self-examination programs. As noted later in this chapter, however, the lack of stage migration at the time of diagnosis casts doubt on the success of self-examination programs.

Ethnicity

It has long been known that there is a disparity in the incidence and prevalence of testicular cancer between Caucasians and African Americans in the United States. SEER data indicate that the lifetime risk of being diagnosed with testicular cancer is four times greater for Caucasians than for African Americans (Table 7). The age-adjusted incidence in 1997-2001 for Caucasians was 6.2 per 100,000 population (7.0 for non-Hispanic Caucasians), while that for African Americans was 1.5 per 100,000 (Table 8). The ageadjusted incidence in Hispanic and Asian/Pacific Islander and North American Native populations fell between these rates. Prevalence of testicular cancer is displayed in Table 9.

Biggs and Schwartz examined relationships between race and ethnicity and testicular cancer, using 16,086 cases from the SEER database (13). The characteristics of the patients are summarized in Table 10. African American, North American Native, Hawaiian American, and Hispanic patients were more likely than Caucasians to be diagnosed with latestage testicular cancer. In addition, a slightly greater percentage of Native American, Hawaiian American, and Hispanic men had NSGCTs. It is unclear whether this represents a sampling bias or a true biological and genetic difference. In all populations, testicular cancer is more common in men under 40 years of age.

60

All Males > 10 years > 20 years > 30 years Eventually Current Age 0 0.30 0.12 0 0.35 0.02 0.12 0.24 0.36 10 20 0.10 0.22 0.29 0.34 30 0.12 0.19 0.22 0.24 0.08 0.11 0.12 0.12 40 50 0.03 0.04 0.05 0.05

0.02

0.02

0.02

Table 7. Percent of males diagnosed with testicular cancer after 10, 20, and 30 years and during remaining lifetime, given freedom from cancerat current age, by race/ethnicity

^{0.01} Lifetime Risk of Dying = Lifetime Risk of Being Diagnosed = 0.35% 0.02%

		White Male	S	
	> 10 years	> 20 years	> 30 years	Eventually
Current Age				
0	0	0.03	0.15	0.42
10	0.03	0.15	0.28	0.42
20	0.12	0.26	0.34	0.40
30	0.14	0.23	0.26	0.28
40	0.09	0.12	0.13	0.14
50	0.03	0.05	0.05	0.06
60	0.01	0.02	0.02	0.02
Lifetime Risk of Being Diagnosed =	0.42%	Lifetime Risk of Dying =	0.02%	
		Black Male	s	

		DIACK IVIA	lies	
	> 10 years	> 20 years	> 30 years	Eventually
Current Age				
0	0	0.01	0.03	0.10
10	0.01	0.03	0.06	0.10
20	0.02	0.05	0.07	0.09
30	0.03	0.05	0.06	0.07
40	0.02	0.03	0.04	0.04
50	0.01	0.02	0.02	0.02
60	0.01	0.01	0.01	0.01
Lifetime Risk of Being Diagnosed =	0.10%	Lifetime Risk of Dying =	0.01%	

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973-2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

Table 8. Age-adjusted incidence ^a rates for test	icular cancer, 1997–2001, by race/ethnicity	
	Rate per 100,000 persons	Annual Percent Change
	1997–2001	Trend 1992–2001
Race/ethnicity		
Total	5.2	1.2°
White	6.2	1.3°
White Hispanic ^b	3.7	1.0
White Non-Hispanic ^b	7.0	1.8°
Black	1.5	6.4°
Asian/Pacific Islander	2.1	2.3
N. American Native/Alaska Native	2.3	
Hispanic ^b	3.6	1.1

...data not available.

^aIncidence data are from the 12 SEER areas (San Francisco, Connecticut, Detroit, Hawaii, Iowa, New Mexico, Seattle, Utah, Atlanta, San Jose-Monterey, Los Angeles, and Alaska Native Registry.

^bHispanic and Non-Hispanic are not mutually exclusive from Whites, Blacks, Asian/Pacific Islanders, and American Indians/Alaska Natives. Incidence data for Hispanics and Non-Hispanics do not include cases from Detroit, Hawaii, and Alaska Native Registry. °The annual percent of change is significantly different from zero (p < 0.05)

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973-2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.







Between 1975 and 2001, the incidence of testicular cancer among Caucasian men rose 54%, from 4.1 per 100,000 to 6.3 per 100,000 (Table 11). As expected, this was most notable in men younger than 50 years of age, among whom the incidence increased from 4.7 per 100,000 to 7.9 per 100,000. In men older than 50, the incidence declined by 15%. Among African American men, the overall incidence of testicular cancer remained stable between 1973 and 1998 at about 1.0 per 100,000 (2). Data on the changes in incidence in other ethnic groups is limited, although Paltoo and Chu found that testicular cancer rates in American Indian and Native Alaskan men stabilized at 2.2 per 100,000 throughout the 1990s (8).

Histology

Changes in the incidence of testicular germ cell tumors among Caucasian and African American men stratified by histologic subtypes are illustrated in Figure 3. McGlynn et al. analyzed SEER data from 1973 to 1998 and found that seminoma and non-seminoma have distinguishable incidence patterns among Caucasian and African American racial groups (2). They demonstrated that for Caucasian men, only the incidence of seminoma was increasing. The incidence of NSGCT was decreasing. In addition, the ratio of seminoma to non-seminoma among Caucasian men changed from 50:50 in 1973-1978 to 60:40 in 1994–1998. In African American men, seminoma also showed continued increasing incidence, coupled with an overall decrease in NSGCT (despite a small upward surge in the final period under study). The seminoma to non-seminoma ratio in African American men increased from 60:40 to 70:30. The divergent trends in the incidence of seminoma and NSGCT may be the result of changes in underlying risk factors and etiologic causes, alterations in biology, refinements in histologic evaluation, or changes in diagnostic practices, including coding practices (i.e., the classification for mixed germ cell was introduced as an ICD-9 code in 1990).

Biggs and Schwartz evaluated the relationships between histology and ethnicity in their examination of 16,086 cases from the SEER database (Table 10) (13). Seminomas constituted an average of 56% of the cases under study, ranging from a low of 51% (Hispanic Americans) to a high of 70% (Japanese Americans). Among the NSGCT subtypes, mixed germ cell

Total ^b		36,6	54	31,755	56	9,060	21,69	*	15,57	5 74,	<u>666</u>	136,9	28	20,421	157,349
White ^b		34,4	66	30,101	28	3,019	20,92	~	15,000	3 70,	615	130,5	59 、	19,622	150,181
Black ^b		1,0	21	622		417	36	~	25(0 1,	723	2,7	47	201	2,948
Asian/Pacific Islande	ې د	2 2	62	494		:	:		:	. –	122		:	:	:
Hispanic ^d		3,2	36	2,389		:	:		:	2	066		:	:	:
data not available.															
^a US 2001 cancer preva 2000 and 2001 populat	lence count	s are base	ed on 20	01 cancer p	Census Pr	proportions evalence w	s from the S	EER re	gistries ar	Nd 1/1/2001 US Malignant Pri	S popula	Ition estim	ates base	d on the a	verage of
biology based on (b) SEER 9 /	Areas, (c)	SEER 1	1 Areas, an	d (d) SEER	t 11 Areas	excluding H	awaii ar	nd Detroit.						
•Maximum limited-dura	tion prevale	nce: 26 ye	ears for 1	975-2001	SEER 9 da	ta; 11 year	s for 1990–	2001 SI	EER 11 da	ita (used to ca	lculate p	revalence	e for Hispa	inics and A	sian/
Pacific Islanders.					:										
^{1g} (f)Cases diagnosed 2 obtained by summing t	26 years or 0 to < 26 ar	more ago nd ≥ 26.	were e	stimated us	sing the col	mpletenes	s index me	thod (C	apocacci	a et. al. 1997.	Merrill	et. al 2000	0); (g) Cor	nplete pre	valence is
SOURCE: Surveillance.	Epidemiolog	av. and En	d Results	(SEER) Pr	oaram (wwv	v.seer.canco	er.dov) SEE	R*Stat D	atabase:	ncidence-SEE	R 9 Rea	Public-U	se. (1973–2	2002). Nati	onal Can-
cer Institute, DCCPS, St	rveillance R	esearch Pr	rogram, C	Cancer Stati	stics Branch	n, released	April 2005, t	based or	the Nove	mber 2004 sub	mission.				5
Table 10. Characterist	tics of testi	cular can	cer pati	ents, by aç	je and race	a/ethnicity									
Characteristic	Non-Hisp. (n=13,9)	White 22)	Africal (n=3	n Am. 29)	Native Am (n=89)	G	inese =129)	Japar (n=1	lese 41)	Filipino (n=60)	Haw; (n=	aiian 94)	Hisp. Whi (n=1.322	(i	Total 1=16,086)
Age at Diagnosis										-					
15–19	598	(%)	13	(4%)	6 (7%)	-	(1%)	0	(1%)	1 (2%)	9	(%9)	107 (8%	(734 (5%)
20–29	4,572	(33%)	101	(31%)	43 (48%) 41	(32%)	39 (28%)	27 (45%)	42	(45%)	575 (44%	%) 5,	443 (34%)
30–39	5,185	(37%)	137	(42%)	25 (29%) 50	(39%)	09	43%)	19 (32%)	33	(35%)	426 (32%	%) 5,	935 (37%)
40-49	2,354	(17%)	55	(17%)	10 (11%) 25	(19%)	28 (20%)	8 (13%)	8	(%6)	150 (119	%) 2,	638 (16%)
50-59	767	(%9)	13	(4%)	3 (3%)	10	(%8)	9	(4%)	2 (3%)	ო	(%2)	41 (3%	(845 (5%)
+09	446	(3%)	10	(3%)	2 (2%)	7	(2%)	9	(4%)	3 (5%)	0	(2%)	20 (2%	(491 (3%)
Stage at Diagnosis															
Localized	9,084	(65%)	202	(61%)	51 (57%) 98	(%92)	100 (71%)	43 (72%)	47	(20%)	841 (64%	%) 10,	466 (65%)
Regional	2,896	(21%)	65	(20%)	13 (15%) 16	(12%)	25 (18%)	10 (17%)	22	(23%)	248 (19%	%) 3,	295 (21%)
Distant	1,640	(12%)	53	(16%)	24 (27%) 12	(%6)	15 (11%)	6 (10%)	25	(27%)	213 (16%	%) 1,	988 (12%)
Unstaged	302	(2%)	ດ	(3%)	1 (1%)	с,	(2%)	-	(1%)	1 (2%)	0		20 (2%	(337 (2%)
Histology															
Seminoma	7,779	(26%)	197	(%09)	47 (53%) 84	(65%)) 66	20%)	39 (65%)	49	(52%)	678 (51%	%) 8,	972 (56%)
NSGCT	5,995	(43%)	119	(36%)	40 (45%) 39	(%08)	42 (30%)	20 (33%)	4	(47%)	635 (48%	%) 6,	934 (43%)
Embryonical	2,318	(17%)	33	(10%)	12 (13%) 15	(12%)	12	(%6)	6 (10%)	16	(17%)	182 (14%	%) 2,	594 (16%)
Yolk Sac	118	(1%)	ო	(1%)	1 (1%)	0		0		0	2	(2%)	14 (1%	(238 (1%)
Teratoma	447	(3%)	Ø	(2%)	4 (4%)	e	(2%)	5	(4%)	0	5	(2%)	50 (4%	(533 (3%)
Choriocarcinoma	124	(1%)	2	(2%)	3 (3%)	e	(2%)	0		0	-	(1%)	11 (1%	(149 (1%)
Mixed Germ Cell	2,988	(21%)	68	(21%)	20 (22%) 18	(14%)	25 (18%)	14 (23%)	20	(21%)	378 (29%	%) 3,	531 (22%)
Non-germ cell	146	(1%)	13	(4%)	2 (2%)	9	(2%)	0		1 (2%)	1	(1%)	9 (1%	(180 (1%)
NSGCT, non-seminomé	atous germ (cell tumor.													
SOURCE: Reprinted froi	n Springer a	Ind Klurer /	Acadame	ic Publisher	s, Cancer C	auses and	Control, 15(5),2004, 201 Scio	437–444,	Differences in	testis ca	ncer survi	val by race	and ethnic	ity: a popu-
lation based study, 1975	-1888 (UO),	BIGUS IVIL,	OCIIWAIL	Z DIVI, TAUIE	ן, שונה אווט	permission	inide mont	ger ocie	nce and D	usiness meuia.					

Urologic Diseases in America

Complete⁹

≥ 26^ŕ

0 to < 26^e

0 to < 11^e

20 to < 25

15 to < 20

10 to < 15

5 to < 10

0 to < 5

Years Since Diagnosis

Race/ethnicity

Table 9. Estimated testicular cancer prevalence counts^a on January 1, 2001 in the United States, by race/ethnicity and years since diagnosis

		white males	
	All	< 50	50+
Year of Diagnosis			
1975	4.1	4.7	2.6
1976	3.8	4.5	1.7
1977	4.9	5.8	2.4
1978	3.9	4.6	2.1
1979	4.3	5.1	2.3
1980	4.9	6.0	2.1
1981	4.8	5.8	2.1
1982	5.0	6.0	2.4
1983	5.2	6.3	2.5
1984	5.0	6.4	2.1
1985	5.1	6.3	2.1
1986	5.6	6.8	2.3
1987	5.7	7.1	1.9
1988	5.4	6.9	1.7
1989	6.3	7.8	2.5
1990	6.0	7.4	2.3
1991	5.7	7.0	2.3
1992	6.1	7.6	2.1
1993	6.0	7.5	1.9
1994	6.3	7.8	2.3
1995	5.3	6.5	2.1
1996	6.2	7.8	1.9
1997	6.3	7.7	2.7
1998	6.6	8.4	1.8
1999	6.3	8.0	2.0
2000	6.7	8.4	2.3
2001	6.3	7.9	2.2
1997–2001	6.5	8.1	2.2

Table 11. Incidence rates^a for testicular cancer, among white males, age-adjusted, by age

^aSeer 9 areas. Rates are per 100,00 and are age-adjusted to the 2000 standard population by 5-year age groups.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

(mean 22%; range 14%–29%) was the most common, followed by embryonal (mean 16%; range 9%–17%), then teratoma (mean 3%; range 0%–5%), and finally choriocarcinoma and yolk sac (mean 1% each; ranges 0%–3% and 0%–2%, respectively). This order of histologic frequency (mixed, embryonal, teratoma, choriocarcinoma and yolk sac) was found across all ethnic groups. African American, Native American, Hawaiian American, and Hispanic testicular cancer patients were more likely than Caucasian patients to have more-aggressive NSGCTs. Steele et al. examined testicular cancer data from the NCDB collected between 1985 and 1996 (11). The incidence of testicular cancer divided by histologic subtype in this database is summarized in Table 12. The percentage of seminoma remained stable at approximately 55% over the ten years of collected data, while the percentage of embryonal tumors and choriocarcinomas decreased from 18.9% to 11.4% and 5.2 to 2.5%, respectively, and the percentage of teratomas increased from 16.4% to 22.3%. In addition, the percentage of non–germ-cell tumors increased from 5.2% to 7.2%. It is unclear whether this represents a true transformation in the percentage of histologic subtypes as opposed to changes in histopathologic





SOURCE: Adapted from Cancer, 97,McGlynn K, Devesa SS, Sigurdson AJ, Brown, LM, Tsao L, and Tarone RE, Trends in the incidence of testicular germ cell tumors in the United States, 63–70, Copyright 2003, with permission from Wiley.

Table 12. Testicular tumor characteristics

	1985–	1986ª	1990–	1991 ^b	1995–	1996°
	Cases	Percent	Cases	Percent	Cases	Percent
Anatomic site						
Undescended testis	39	1.7	138	2.4	160	2.1
Descended testis	56	2.5	278	4.9	2,187	29.3
Testis, NOS	2,185	95.8	5,261	92.7	5,105	68.5
Total	2,280	100	5,677	100	7,452	100
Histology						
Seminoma, NOS	1,219	53.5	3,029	53.4	4,171	56.0
Spermatocytic seminoma	14	0.6	31	0.5	40	0.5
Embryonal carcinoma	430	18.9	846	15.4	853	11.4
Malignant teratoma	373	16.4	1,203	21.2	1,659	22.3
Choriocarcinoma	118	5.2	210	3.7	184	2.5
Nongerm cell tumors	118	5.2	322	5.7	537	7.2
Unspecified	8.0	0.4	6.0	0.1	8.0	0.1
Total	2,280	100	5,677	100	7,452	100
AJCC stage						
I	779	64.2	3,141	65.0	4,800	73.4
II	156	12.9	295	6.1	1,107	16.9
III	108	8.9	324	6.7	633	9.7
IV	170	14.0	1,069	22.1		
Total	1,213	100	4,829	100	6,540	100
Unknown	1,067	46.8	848	14.9	912	12.2
Total	2,280	100	5,677	100	7,452	100

...data not available.

NOS = not otherwise specified; AJCC: American Joint Committee on Cancer.

^aCases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 2nd edition.

^bCases staged according to the American Joint Committee on Cancer *Manual for Staging of Cancer*, 3rd edition.

°Cases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 4th edition.

SOURCE: Reprinted from Cancer, 86, Steele, GS, Richie, JP, Stewart AK, and Menck HR, The National Cancer Data Base report on patterns of care for testicular carcinoma, 1985–1996, 2,171–2,183, Copyright 1999, with permission from Wiley.

Table 13	Percent distribution	of AJCC stage at	diagnosis b	v disease histology
Table 13.	r ercent uistribution	UI AJOO Slage al	ulagilusis, b	y uisease mistology

	1985–19	986ª	1990–19	991 ^b	1995–19	996°
	Seminoma	NSGCT	Seminoma	NSGCT	Seminoma	NSGCT
Stage of Diseased						
Early	88.9	61.7	89.7	62.7	89.2	65.1
Advanced	11.1	39.3	10.3	37.3	10.8	34.9
Total	100	100	100	100	100	100
Cases, n	667	488	2669	1926	3802	2369

...data not available.

NOS = not otherwise specified; AJCC = American Joint Committee on Cancer.

^aCases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 2nd edition.

^bCases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 3rd edition.

°Cases staged according to the American Joint Committee on Cancer Manual for Staging of Cancer, 4th edition.

^dEarly stage: 1985–1986 American Joint Committee on Cancer Stage I and II; 1990–1991 American Joint Committee on Cancer Stage I, II, and III; 1995–1996 American Joint Committee on Cancer Stage I and II N1. Advanced stage: 1985–1986 American Joint Committee on Cancer Stage IIIand IV; 1990–1991 American Joint Committee on Cancer Stage IV; 1995–1996 American Joint Committee on Cancer Stage II (N2 or higher) and III.

SOURCE: Reprinted from Cancer, 86, Steele, GS, Richie, JP, Stewart AK, and Menck HR, The National Cancer Data Base report on patterns of care for testicular carcinoma, 1985–1996, 2,171–2,183, Copyright 1999, with permission from Wiley.

		,			• • •				
		All Males		W	hite Males	;	Bla	ack Males	;
	All	< 50	≥ 50	All	< 50	≥ 50	All	< 50	≥ 50
All Stages (n)	4,148	3,827	321	3,822	3,521	301	121	113	8
Localized	70	71	67	71	71	68	63	65	38
Regional	18	18	17	18	18	17	20	20	13
Distant	10	10	13	10	10	13	16	13	50
Unstaged	1	1	2	1	1	2	2	2	

Table 14. Survival rates for testicular cancer, 1995–2000^a by race, stage, and age, percent

...data not available.

^aRates are from SEER 9 areas. Rates are per 100,000 and are age-adjusted to the 2000 standard population by 5-year age groups. They are based on data from population-based registries in Connecticut, Puerto Rico, Utah, Iowa, Hawaii, Atlanta, Detroit, Seattle-Puget Sound, and San Francisco-Oakland. Rates are based on follow up of patients into 2001.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

analysis, or whether it reflects the 3.25-fold increase in patients entered into the database between 1985 and 1996. It is also unclear why these data differ from the SEER data. It is possible that the NCDB and the SEER data were influenced differently by changes in coding practices during the period of data collection.

Stage

Distribution by stage at diagnosis in the NCDB is summarized in Table 12. From 1985 to 1991, the proportion of tumors presenting as stage I remained relatively stable at approximately 65%, whereas the percentage of stage II and III tumors decreased from 12.9% to 6.1% and 8.9% to 6.7%, respectively. Stage IV tumors increased from 14% to 22% during the same period. This is an unexpected finding. With increased physician and patient education and awareness, as well as self-examination programs, one would expect a stage migration, i.e., an increasing percentage of localized tumors (stage I) coupled with diminishing rates of disseminated disease (stages II-IV). Several factors may explain these findings. Nearly half of the patients in the NCDB had an unknown stage; this rate decreased to 12.2% by 1995-1996. In addition, considerable changes in staging practices occurred during the 11 years of data acquisition. However, when the NCDB data are further divided into "early" and "advanced" disease (Table 13), there still appears to be little change in stage distribution over time. These data do confirm that more seminomas than NSGCTs are discovered earlier in their course.

The stage distribution of all 4,148 men in the SEER database from 1995 to 2000 showed 70% localized, 18% with regional spread, 10% with distant spread, and 1% unstaged (Table 14). Caucasian men were more likely to present with localized disease than were African American men (71% vs 63%), who, conversely, were more likely to have metastatic disease (20% vs 18% and 16% vs 10% for regional and distant spread, respectively). Biggs and Schwartz evaluated the relationships between stage and ethnicity in the SEER database (Table 10) (13) and found that, on average, 65% of patients presented with localized disease, which is similar to findings in the NCDB data. However, African American, Native American, Hawaiian American, and Hispanic testicular cancer patients were more likely than Caucasians to be diagnosed with late-stage disease. Overall, 21% of the patients in the database examined by Biggs and Schwartz presented with regional metastases, 12% with distant metastases, and 2% without formal staging (Table 10). The differences between these findings and those of the NCDB could be the result of differences in nomenclature, biological differences in the tumors of the study populations, or disparity in other social factors, including healthcare access and usage. Interestingly, in the SEER analysis, men of Asian ancestry (China, Japan, and the Philippines) had the highest incidence of localized disease, whereas Hawaiians, who share some genetic heritage with this population, had the lowest. This may reflect access to

		All Males		Ŵ	hite Male	s	BI	ack Male	s
	All	< 50	≥ 50	All	< 50	≥ 50	All	< 50	≥ 50
5-Year Relative Survival Rates									
Year of Diagnosis									
1960–1963ª				63.0					
1970–1973ª				72.0					
1974–1976 ^b	78.7	78.1	82.9	78.8	78.2	83.3	75.9 ^d		
1977–1979 ^b	87.5	88.6	77.1	87.9	89.0	78.1	66.2 ^d		
1980–1982 ^b	91.9	91.9	91.8	92.1	92.0	92.7	89.7 ^d	89.2 ^d	
1983–1985 ^b	91.0	91.8	82.3	91.3	92.3	80.7	87.9 ^d	84.3 ^d	
1986–1988 ^b	95.2	95.3	93.5	95.7	95.7	94.4			
1989–1991 ^b	95.4	95.5	93.8	95.9	95.8	94.8	89.8 ^d	93.6 ^d	
1992–1994 ^b	95.4	95.7	90.4	95.6	95.9	60.1	85.2 ^d	84.5 ^d	
1995–2000 ^b	95.9°	96.4°	88.3	96.2°	96.7°	89.4	87.3	90.4	
1995-2000 ^b									
All Stages	95.9	96.4	88.3	96.2	96.7	89.4	87.3	90.4	
Localized	99.4	99.4	97.0	99.4	99.4	97.6	96.5 ^d	99.6	
Regional	95.9	96.4	89.9 ^d	96.1	96.5	90.6 ^d			
Distant	71.8	75.1	38.7 ^d	73.1	76.6	39.5₫			
Unstaged	89.1	91.6		90.2	93.0				
5-Year Relative Survival Rates,	1995–2000 ^b								
Age at Diagnosis									
< 45	96.5			96.7					
45–54	94.5			95.3					
55–64	87.2			87.9					
65–74	74.2 ^d			75.9 ^d					
75+									
< 65	96.1			96.4					
65+	73.9 ^d	<u></u>		77.6 ^d	<u></u>			<u></u>	<u></u>

Table 15. Survival rates^{a,b} for testicular cancer, by race/ethnicity, diagnosis year, stage, and age

...data not available.

^aRates are based on End Results data from a series of hospital registries and one population-based registry.

^bRates are from SEER 9 areas. They are based on data from population-based registries in Connecticut, Puerto Rico, Utah, Iowa, Hawaii, Atlanta, Detroit, Seattle-Puget Sound, and San Francisco-Oakland. Rates are based on follow up of patients into 2001. ^cThe difference in rates between 1974–1976 and 1995–2000 is statistically significant (p < 0.05).

^aThe standard error of the survival rate is between 5–10 percentage points.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

1					- B	2.22												
1976–1979		1980–1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
93.3		96.0	97.1	98.1	98.1	98.9	98.2	98.2	98.4	97.9	96.8	97.7	98.3	97.9	97.4	98.2	97.0	97.7
88.6		92.7	94.0	96.7	97.3	96.6	95.7	96.7	96.9	96.7	95.9	96.5	97.5	96.4	95.7	96.6	96.1	
86.5		92.1	93.4	96.5	96.3	95.8	95.0	96.3	95.8	95.6	95.2	95.9	96.4	96.0	95.2	96.2		
85.6		91.5	92.8	95.8	95.6	95.2	94.8	96.3	95.3	95.6	95.2	95.9	96.4	96.0	94.9			
85.1		91.2	92.5	95.5	95.3	94.8	94.7	96.3	95.3	95.3	95.2	95.7	96.3	96.0				
84.8		90.9	92.3	95.4	94.6	94.6	94.6	96.0	95.1	94.9	95.2	95.7	96.3					
84.7		90.8	92.3	95.4	94.5	94.4	94.3	95.9	95.1	94.8	95.2	95.6						
84.6		90.5	91.5	95.4	94.5	93.5	94.2	95.9	94.8	94.0	95.0							
84.5		0.06	91.5	95.2	94.5	93.5	96.8	95.9	94.6	94.0								
83.8		89.9	91.4	94.7	94.5	93.4	96.8	95.9	94.5									
83.3		89.9	90.6	94.7	94.4	93.3	96.8	95.9										
92.1		89.8	90.6	94.2	94.4	93.3	96.8											
92.8		89.7	89.9	94.2	93.7	93.3												
82.1		89.2	89.0	94.0	93.6													
81.9		88.7	88.9	94.0														
81.5		88.5	88.5															
80.9		88.0																
80.4		87.6																
79.9																		
79.7																		
SEER	9 ar	eas are rates e	xpresse	d as per	cents.													

Table 16. Survival rates^a for testicular cancer, by year of diagnosis

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

	Non-Hisp. White	African Am.	Native Am.	Chinese	Japanese	Filipino	Hawaiian	Hisp. White	Total
Characteristic	(n=13,922)	(n=329)	(n=89)	(n=129)	(n=141)	(n=60)	(n=94)	(n=1,322)	(n=16,086)
Completeness of follow-up	87%	80%	89%	88%	92%	85%	85%	86%	87%
Number of testicular cancer deaths	728	35	12	7	6	5	15	78	886
5-Year Survival									
1973–1989	0.93	0.87	0.85	0.94	0.90	0.94	0.77	0.91	0.92
1990–1999	0.97	0.90	0.90	0.99	0.99	0.92	0.92	0.95	0.97
1973–1999	0.95	0.89	0.89	0.97	0.95	0.93	0.84	0.94	0.95

Table 17. Completeness of follow-up, number of testicular cancer deaths, and Kaplan Meier estimates of 5-year cause-specific survival among testicular cancer patients by race/ethnicity, 12 SEER registries, 1973–1999

SOURCE: Reprinted from Springer and Klurer Acadameic Publishers, Cancer Causes and Control, 15(5),2004, 437–444, Differences in testis cancer survival by race and ethnicity: a population based study, 1973–1999 (US), Biggs ML, Schwartz SM, Table 2, with kind permission from Springer Science and Business Media.

healthcare on the Hawaiian Islands, as well as dietary and other environmental factors.

Survival

When considering the epidemiologic data, it is important to recognize the difference between mortality, the deaths in the general population due to the specific disease, and survival, which is limited to the patient cohort with the disease. Five-year relative survival rates by race, year of diagnosis, stage, and age from the SEER database are summarized in Table 15. From 1974 to 2000, five-year survivals rates increased successively, reaching the most recent level of 95.9%. This may be explained in part by a stage migration to earlier presentation of disease (14). African American men with testicular cancer experienced a decrease in survival rates between 1989–1991 and 1992–1994, from 89.8% to 85.2%. This was a temporary downturn, however, and in the 1995–2000 dataset survival of African American men increased to 87.3%. Possible explanations include differential access to medical care.

Table 15 also demonstrates that survival rates are best for patients who present with localized disease. When stratified by stage at presentation, men diagnosed between 1995 and 2000 with localized disease had survival rates of 99.4%, compared with 95.9% and 71.8% for regional and distant disease, respectively. Men diagnosed at a younger age also have better survival rates. In the 1995–2000 cohort, men younger than 50 years of age had five-year relative survival rates of 96.4%, compared with 88.3% for men older than 50. Finally, men diagnosed more recently had better survival rates. A man diagnosed in 1995 had a 95.9% chance of five-year survival, while the rate for a man diagnosed in 1974 was 78.7%.

Table 18. Multivariat	e adjusted ha	zard ratios fo	or association of	of race/ethnic	city with death	from testicu	ular cancer	
Adjustment	Non-Hisp. White (n=13,922)	African Am. (n=329)	Native Am. (n=89)	Chinese (n=129)	Japanese (n=141)	Filipino (n=60)	Hawaiian (n=94)	Hisp. White (n=1,322)
Histology, period of diagnosis	1.0	2.6	2.9	1.3	1.4	2.1	3.6	1.6
Histology, period of diagnosis, stage	1.0	2.3	2.1	1.6	1.1	3.6	2.4	1.4

SOURCE: Reprinted from Springer and Klurer Acadameic Publishers, Cancer Causes and Control, 15(5), 2004, 437–444, Differences in testis cancer survival by race and ethnicity: a population based study, 1973–1999 (US), Biggs ML, Schwartz SM Table 4, with kind permission from Springer Science and Business Media.



Figure 4a. Relative survival of seminoma patients by cancer stage for cases diagnosed in 1985–1991.

Stage I represents 1,796 patients; Stage II represents 158 patients; and Stage III represents 117 patients.



Figure 4b. Relative survival of nonseminoma patients by cancer stage for cases diagnosed in 1985–1991.

Stage I represents 801; Stage II represents 129 patients; and Stage III represents 202 patients.

SOURCE: Adapted from Cancer, 86, Steele GS, Richie JP, Stewart AK, and Menck HR, The National Cancer Data Base report on patterns of care for testicular carcinoma, 1985–1996, 2,171–2,183,. Copyright 1999, wiht permission from Wiley.

Survival rates by year of diagnosis from 1976 to 2000 are given in Table 16.

Biggs and Schwartz evaluated the relationship between survival and ethnicity in their examination of 16,086 cases from the SEER database between 1973 and 1999 (Table 17) (13). Survival rates after a diagnosis of testicular cancer were high, with only 886 deaths among 16,086 patients. After multivariate analysis was performed to control for stage, histology, and period of diagnosis (Table 18), African American,

		All Males	;		White Male	es		Black Male	s
	All	< 50	≥ 50	All	< 50	≥ 50	All	< 50	≥ 50
Year of Diagnosis									
1975	0.7	0.7	0.7	0.8	0.8	0.8			
1976	0.7	0.7	0.7	0.8	0.8	0.7	0.4		
1977	0.6	0.7	0.6	0.7	0.7	0.6			
1978	0.6	0.6	0.5	0.6	0.6	0.6	0.2		
1979	0.5	0.5	0.6	0.5	0.5	0.6	0.4		
1980	0.5	0.4	0.5	0.5	0.5	0.5			
1981	0.4	0.4	0.4	0.4	0.4	0.4			
1982	0.4	0.4	0.4	0.4	0.4	0.4			
1983	0.4	0.4	0.4	0.4	0.4	0.4			
1984	0.3	0.3	0.4	0.4	0.4	0.4			
1985	0.4	0.3	0.4	0.4	0.3	0.4			
1986	0.3	0.3	0.3	0.3	0.4	0.4			
1987	0.3	0.4	0.3	0.4	0.3	0.3			
1988	0.3	0.3	0.4	0.3	0.3	0.4			
1989	0.3	0.3	0.3	0.3	0.3	0.3	0.2		
1990	0.3	0.3	0.2	0.3	0.3	0.2			
1991	0.3	0.3	0.3	0.3	0.3	0.3			
1992	0.3	0.3	0.3	0.3	0.3	0.3			
1993	0.3	0.3	0.3	0.3	0.3	0.3			
1994	0.3	0.3	0.2	0.3	0.3	0.2			
1995	0.2	0.2	0.2	0.3	0.3	0.3			
1996	0.3	0.3	0.3	0.3	0.3	0.3			
1997	0.2	0.2	0.2	0.3	0.3	0.3			
1998	0.3	0.3	0.3	0.3	0.3	0.3			
1999	0.3	0.3	0.3	0.3	0.3	0.3			
2000	0.2	0.3	0.2	0.3	0.3	0.2	0.2		
2001	0.2	0.2	0.3	0.3	0.3	0.2			
1997–2001	0.3	0.3	0.3	0.3	0.3	0.3	0.2	0.1	0.2

Table 19. United States death rates^a for testicular cancer, age-adjusted, by race and age

...data not available.

^aNHS public use data file for the total US. Rates are per 100,000 and are age-adjusted to the International Agency for Researchon Cancer (IARC) world standard population.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

Native American, Filipino, and Hawaiian men were found to have a 2- to 3.5-fold greater risk of dying than non-Hispanic Caucasian men. The risk of dying was 40% higher for Hispanic men than for non-Hispanics. Still, because of the high survival rates, these differences translate into a small absolute number of deaths. The authors postulate that the observed disparities may reflect biological differences in the tumor, patient comorbidities, or differences for which race is a proxy, including social, economic, and health insurance status; treatment options and uptake; healthcare access and utilization; and environment, cultural, and lifestyle factors (13). Men with seminomas have better survival rates than do those with NSGCT. Survival data based on stage and divided between seminoma and NSGCT from the National Cancer Data Base (NCDB) are shown graphically in Figures 4a and 4b (11). The fiveyear survival rate for seminoma is 97.9%, and that for NSGCT is 96.5%. Although this may represent a difference in tumor biology and behavior between the two types of testicular cancer, it may also result from the finding that men with seminoma generally present at an earlier stage (Table 13).

	Rate per 100,000 persons	Annual Percent Change
Race/ethnicity	1997–2001	Trend 1992–2001
Total	0.3	-1.3
White	0.3	-1.4
White Hispanic ^b	0.2	-4.0
White Non-Hispanic ^b	0.3	-0.8
Black	0.2	2.3
Asian/Pacific Islander		
N. American Native/Alaska Native		
Hispanic ^b	0.2	-3.9

...data not available.

^aMortality data are analyzed from public use file provided by the National Center for Health Statistics (NCHS).

^bHispanic and Non-Hispanic are not mutually exclusive from Whites, Blacks, Asian/Pacific Islanders, and N. American Natives/Alaska Natives. Incidence data for Hispanics and Non-Hispanics do not include cases from Detroit, Hawaii, and Alaska Native Registry.

SOURCE: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence-SEER 9 Regs Public-Use, (1973–2002), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2005, based on the November 2004 submission.

Mortality

Testicular tumors are exceedingly curable, and mortality is very low. SEER data from 1997–2001 place the age-adjusted death rate from testicular cancer for American men at 0.3 per 100,000 (Table 19). The overall death rate from testicular germ cell tumors decreased by 71% between 1975 and 2001, from 0.7 per 100,000 to 0.2 per 100,000. During this period, the

death rate decreased from 0.8 per 100,000 to 0.3 per 100,000 for Caucasian men and from 0.4 per 100,000 to 0.2 per 100,000 for African American men. These findings indicate that Caucasian males have a higher lifetime risk of dying from testicular cancer than do African Americans males (0.02% vs 0.01%) (Table 7).

Age-adjusted mortality rates from SEER for 1997– 2001 for different ethnic groups are shown in Table



Figure 5. Incidence and death rates for testicular cancer, by race.

SOURCE: SEER 9 areas and NCHS public use data file for the total US. Rates are age-adjusted to the 2000 US standard million population by 5year age groups. Regression lines are calculated using the Joinpoint Regression Program Version 2.7, September 2003, National Cancer Institute.



Figure 6. Incidence and death rates for testicular cancer, by age and race.

Regression line could not be calculated for the black ages 50+ incidence rates due to years with a rate of zero.

SOURCE: SEER 9 areas and NCHS public use data file for the total US. Rates are age-adjusted to the 2000 US standard million population by 5year age groups. Regression lines are calculated using the Joinpoint Regression Program Version 2.7, September 2003, National Cancer Institute.

20. Caucasian men in the database had the highest mortality rates, at 0.3 per 100,000. However, from 1992 to 2001, the annual mortality rate for Caucasian men declined by 1.3%. While the annual mortality rate for African American men was lower than that for Caucasians, it increased by 2.3% between 1992 and 2001. No clear explanation for this divergence is apparent. It seems unlikely that the biology of testicular cancer in African American men has changed to make it more deadly. However, it is plausible that changes in epigenetic factors such as diet or environmental exposure could be worsening the prognosis. It is also possible that access to medical care or the treatment provided to African American men deteriorated over the decade under study. In fact, five-year relative survival rates for African American men declined between 1992 and 1994 (they have subsequently rebounded), as evidenced in Table 15.

SEER data for the incidence of testicular cancer and death rates among American men are presented in Figures 5 and 6. Incidence increased between 1975 and 2001, although the rate of increase slowed. The percentage increase was greater for Caucasians than for African Americans and was greatest in men under 50. During the same period, death rates fell. This decrease was greater for Caucasians than for African Americans, although overall mortality (much like incidence) is greater for American men of European descent. In the 1990s, there was a temporary increase in the annual percentage mortality rate for African American men, the only racial group to experience such a setback. Still, overall mortality from testicular cancer is quite low.

Table 21. Inpati	ent hospit	al stays for testi	icular cance	r listed as	orimary diagnos	sis, count, r	ateª (95% C	 age-adjusted 	rate ^b			
		1994			1996			1998			2000	
			Age- Adiusted			Age- Adiusted			Age- Adiusted			Age- Adiusted
	Count	Rate	Rate	Count	Rate	Rate	Count	Rate	Rate	Count	Rate	Rate
Total⁰	2,230	1.8 (1.6–2.0)	1.8	1,890	1.5 (1.3–1.7)	1.5	1,993	1.5 (0.9–2.2)	1.5	1,907	1.4 (1.2–1.6)	1.4
Age												
<18	*	*		*	*		*	*		*	*	
18–24	407	3.3 (2.4-4.2)		296	2.4 (1.7–3.1)		414	3.3 (1.5–5.1)		396	3.0 (2.2–3.8)	
25-34	951	4.7 (3.8–5.6)		771	3.9 (3.1–4.7)		732	3.8 (2.0–5.6)		647	3.5 (2.8-4.3)	
35-44	561	2.8 (2.3–3.4)		483	2.3 (1.8–2.8)		522	2.4 (1.4–3.4)		553	2.5 (2.0–3.1)	
4554	158	1.1 (0.7–1.6)		*	*		*	*		151	0.8 (0.5–1.2)	
55-64	*	*		*	*		*	*		*	*	
65-74	*	*		*	*		*	*		*	*	
75-84	*	*		*	*		*	*		*	*	
85+	*	*		*	*		*	*		*	*	
Race/ethnicity												
White	1,526	1.7 (1.4–1.9)	1.7	1,221	1.3 (1.1–1.5)	1.3	1,333	1.4 (0.6–2.2)	1.5	1,027	1.1 (0.9–1.3)	1.1
Hispanic	176	1.4 (0.8–1.8)	1.1	138	1.0 (0.5–1.4)	0.9	*	*	1.1	289	1.8 (1.0–2.5)	1.5
Region												
Midwest	489	1.7 (1.2–2.1)	1.7	477	1.6 (1.2–2.0)	1.6	349	1.1 (0.7–1.6)	1.2	392	1.3 (0.9–1.6)	1.3
Northeast	686	2.8 (2.1–3.5)	2.8	345	1.4 (0.9–1.9)	1.4	*	*	3.0	334	1.4 (1.0–1.8)	1.4
South	579	1.4 (1.1–1.7)	1.4	610	1.4 (1.1–1.7)	1.4	455	1.0 (0.8–1.2)	1.0	575	1.2 (0.9–1.6)	1.2
West	475	1.7 (1.3–2.1)	1.7	458	1.6 (1.0–2.1)	1.5	443	1.5 (1.0–2.0)	1.4	606	2.0 (1.4–2.6)	2.0
MSA												
Rural	188	0.6 (0.4–0.8)	0.6	187	0.6 (0.4–0.9)	0.7	*	*	*	194	0.7 (0.4–0.9)	0.7
Urban	2,034	2.2 (1.9–2.5)	2.2	1,695	1.7 (1.4–2.0)	1.7	1,863	1.8 (1.0–2.7)	1.8	1,713	1.6 (1.4–1.9)	1.6
*Figure does not	meet stand	dard for reliability	or precision									
MSA, metropoliti	an statistica	่า area.										
^a Rate per 100,00	0 is based	on 1994, 1996, ' IS male civilian r	1998, 2000 p	opulation es	stimates from Cu	rrent Popula	tion Survey	(CPS), CPS Utili	ties, Unicon	Research C	orporation, for rel	evant
^b Age-adjusted to	the US Ce	nsus-derived age	e distribution	of the vear	under analysis.							
^c Persons of othe	r races, mis	ssing or unavaila	ble race and	ethnicity, ar	Id missing MSA	are included	in the totals					
NOTE: Counts n	nay not sur	n to totals due to	rounding. roiact Nation	wide Innatio	nt Samula 1001	1006 1008						
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TRENDS IN HEALTHCARE RESOURCE UTILIZATION

Inpatient Care

Testicular require cancer patients may inpatient hospitalization for surgery (i.e., RPLND), chemotherapy, or any of the potential side effects of either. Currently, orchiectomy rarely requires hospitalization. According to the Healthcare Cost and Utilization Project (HCUP), the rate of national inpatient hospitalizations for testicular cancer as a primary diagnosis was 1.8 per 100,000 (2,230 admissions) in 1994 and 1.4 per 100,000 (1,907 admissions) in 2000 (Table 21). The age-adjusted hospitalization rate decreased slightly for Caucasian men and increased slightly for Hispanic men. No HCUP data are available for African American men with testicular cancer.

Hospitalization rates were highest in the 25- to 34-year-olds, followed by 18- to 24-year-olds, 35- to 44-year-olds, and 45- to 54-year-olds. This reflects the age distribution of men with testicular cancer. Little geographic variation exists, except in the Northeast, where hospitalization rates were nearly double those of all other regions in 1994. It is unclear whether this disparity results from financial considerations or differences in management practices by physicians in that geographical region. Admission rates were highest in urban areas, most likely reflecting the treatment of many testicular cancer patients in tertiary care centers of excellence for both complex surgery and chemotherapy.

The steady decline in hospitalizations in the HCUP data likely reflects (1) improved surgical technique, (2) trends among surgeons to shorten post-operative hospital stays, (3) outpatient orchiectomies, (4) decreases in the number of cycles of chemotherapy as primary treatment and the forgoing of some as adjuvant to RPLND, (5) greater reliance on outpatient chemotherapy, (6) improved management and support of patients receiving chemotherapy, and (7) increasing utilization of surveillance as a primary modality of treatment.

Outpatient Care

An individual with testicular cancer may be seen in the outpatient setting during diagnosis,

treatment, and follow-up. This includes initial work-up, before and after orchiectomy, before and after any secondary surgeries (i.e., RPLND), during radiation and chemotherapy, and during surveillance for recurrence. Emergency room visits are exceeding rare; consequently, there is insufficient information on which to base any conclusions.

Physician Office Visits

In the Medicare data for 1992, 1995, 1998, and 2001, physician office visit rates increased significantly from 1992 to 1998 and then remained stable for men younger than 65 years of age (Table 22). For men older than 65, the age-adjusted rate varied minimally from 1992 to 2001. Variability was seen across geographic regions and racial/ethnic strata. Greater reliance on outpatient care resulted, not surprisingly, in increased physician office visits (corresponding to the decrease in inpatient hospitalizations (Table 21)).

Data regarding physician office visits by African American and Hispanic men are difficult to interpret due to small sample size; low counts preclude drawing firm conclusions regarding trends. However, for African American men, the rates of physician office visits fell steadily from 1992 to 2001 (with one exception, in 1998), with an overall ultimate decrease of 50%. A similar trend was seen in Hispanic men, for whom the number of physician office visits nearly tripled from 1995 to 1998, then subsequently fell by 40%. These racial/ethnic differences are difficult to explain but may be tied to the decreased survival rates for African American men in the 1990s mentioned earlier (Table 15). We have already noted that non-Caucasian testicular cancer patients present with later-stage disease (Table 10). Perhaps, in addition, non-Caucasian men are now presenting with moreaggressive tumors that require greater amounts of inhospital care and are associated with worse survival outcomes.

Alternatively, the high rates of hospitalization and low rates of outpatient visits by non-Caucasian men with testicular cancer may reflect a reluctance of physicians to use surveillance or outpatient chemotherapy for minority populations. In addition, it is possible that the non-Caucasian men are more comfortable receiving more aggressive, definitive, and/or in-patient care and opt against outpatient

	Adjusted Adjusted Total ⁴ 4,360 29 (25-33) 6,0 Total < 65 1,840 59 (47-71) 2,4 Total < 65 1,840 59 (47-71) 2,4 Age 65-69 660 16 (11-22) 1,4 Age 65-69 660 16 (11-22) 1,1 Age 65-69 660 16 (11-22) 1,1 Age 65-69 660 16 (11-22) 1,1 Page 65-89 160 27 (8.2-46) 1,1 90-94 240 18 (51-186) 2,3 1,0 95-97 0 0 9 3,3 2,4 3,3 White 3,840 31 (26-35) 30 5,3 3,4 3,3 Asian	Count Rate 6,080 40 (35–44)	Ì		>>>-			- >>4	
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	Total*4,36029(25-33)6,0Total*651,84059 $(47-71)$ 2,4Total 65+2,52021 $(18-25)$ 233,6Age66016 $(11-22)$ 1,4 $67-74$ 52016 $(9.8-22)$ 1,6 $75-79$ 74033 $(22-43)$ 1,0 $80-84$ 20015 $(5.8-25)$ 22 $85-89$ 16027 $(8.2-46)$ 1 $90-94$ 240118 $(51-186)$ 11 $90-94$ 240118 $(51-186)$ 11 $90-94$ 240118 $(51-133)$ 243 $91-94$ 240118 $(51-133)$ 243 $91-94$ 28022 $(10-33)$ 243 $91-94$ 28022 $(10-33)$ 243 $91-94$ 28022 $(10-33)$ 243 $91-94$ $92-97$ 0 0 0 0 $91-94$ 280 22 $(10-33)$ 24 3 $91-94$ 31 $(26-35)$ 30 $5,3$ $91-94$ 31 $(26-35)$ 24 3 $91-94$ 280 22 $(10-33)$ 24 3 $91-94$ 31 $(26-35)$ 24 3 $92-94$ 31 $(26-32)$ 24 31 $92-94$ 31 $(26-32)$ 24 31 $92-94$ 92 92 $(10-33)$ 25 </th <th>6,080 40 (35-44)</th> <th>Age- Adjusted Rate</th> <th>Count</th> <th>Rate</th> <th>Age- Adjusted Rate</th> <th>Count</th> <th>Rat</th> <th>Age- Adjust</th>	6,080 40 (35-44)	Age- Adjusted Rate	Count	Rate	Age- Adjusted Rate	Count	Rat	Age- Adjust
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	Total 65+ 2,520 21 (18–25) 23 3,6 Age 65–69 660 16 (11–22) 1,4 70–74 520 16 (9.8–22) 1,0 75–79 740 33 (22–43) 1,0 85–89 160 27 (8.2–46) 1,0 90–94 200 15 (8.2–46) 1,1 90–94 240 118 (51–186) 1,1 95–97 0 0 9 1,1 95–97 0 0 18 1,1 1,1 95–97 0 0 13 (26–35) 30 5,3 White 3,840 31 (26–35) 30 5,3 30 5,3 Black 280 22 (10–33) 24 3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 5,3 30 <td>2,440 71 (58–83)</td> <td></td> <td>2,920</td> <td>85 (71–99)</td> <td></td> <td>3,180</td> <td>84 (71–9</td> <td></td>	2,440 71 (58–83)		2,920	85 (71–99)		3,180	84 (71–9	
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Region Region 1,340 25 1,700 44 35-53 43 1,440 39 30-48 40 1,340 35 27-44 37 Northeast 1,700 54 (42-65) 48 880 28 (19-36) 26 820 30 (20-39) 29 1,780 61 (48-74) 60 Northeast 1,700 54 (42-65) 48 880 28 (19-36) 26 820 30 (20-39) 29 1,780 61 (48-74) 60 South 1,360 26 (20-32) 27 1,980 36 (29-43) 34 2,480 46 (38-54) 49 2,540 44 (36-51) 42 West 440 18 (11-26) 19 1,520 66 (51-80) 75 1,140 51 (38-64) 43 26.540 44 (36-51) 42 «Unweighted counts auotavaiable.	Region 25 1,7 Midwest 860 23 (16–30) 25 1,7 Northeast 1,700 54 (42–65) 48 8 South 1,360 26 (20–32) 27 1,9 West 440 18 (11–26) 19 1,5	0	0	0	0	0	40	120 (0–28	5) 120
Midwest 860 23 (16–30) 25 1,700 44 35 27–44 37 Northeast 1,700 54 (42–65) 48 880 28 (19–36) 26 820 30 (20–39) 29 1,780 61 (48–74) 60 South 1,360 26 (20–32) 27 1,980 36 (29–43) 34 2,480 46 (38–54) 49 2,540 44 (36–51) 42 West 440 18 (11–26) 19 1,520 66 (51–80) 75 1,140 51 (38–64) 43 (36–51) 42 "Unweighted counts multiplied by 20 to arrive at values in the table. * 1,140 51 (38–64) 43 (36–32) 23 (15–32) 23 (15–32) 23 "Unweighted counts multiplied by 20 to arrive at values in the table. * * 1,140 51 (38–64) 43 (36–32) (15–32) 23 (15–32)	Midwest 860 23 (16–30) 25 1,7 Northeast 1,700 54 (42–65) 48 8 South 1,360 26 (20–32) 27 1,9 West 440 18 (11–26) 19 1,5								
Northeast 1,700 54 (42–65) 48 880 28 (19–36) 26 820 30 (20–39) 29 1,780 61 (48–74) 60 South 1,360 26 (20–32) 27 1,980 36 (29–43) 34 2,480 46 (38–54) 49 2,540 44 (36–51) 42 West 440 18 (11–26) 19 1,520 66 (51–80) 75 1,140 51 (38–64) 43 (36–51) 42 data not available.	Northeast 1,700 54 (42–65) 48 8 South 1,360 26 (20–32) 27 1,9 West 440 18 (11–26) 19 1,5 data not available. 10 1,5	1,700 44 (35–53)	43	1,440	39 (30–48)	40	1,340	35 (27-4	t) 37
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West 440 18 (11–26) 19 1,520 66 (51–80) 75 1,140 51 (38–64) 43 580 23 (15–32) 23 data not available. • Unweighted counts multiplied by 20 to arrive at values in the table. • Rate per 100,000 male Medicare beneficiaries in the same demographic stratum. • Anotarised to the 11S Creatic-drarived are distribution of the vear under analysis	West 440 18 (11–26) 19 1,5 data not available.	1,980 36 (29–43)	34	2,480	46 (38–54)	49	2,540	44 (36–5	1) 42
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ªUnweighted counts multiplied by 20 to arrive at values in the table. bRate per 100,000 male Medicare beneficiaries in the same demographic stratum. ⊙Ana-adimeted to the LIS. Census-derived and distribution of the vear under analysis.									
bRate per 100,000 male Medicare beneficiaries in the same demographic stratum. מאמ-במוווונדמיו to the LIS Census-derived and distribution of the vear under analysis	^a Unweighted counts multiplied by 20 to arrive at values in the ta	i the table.							
obre-adjuncted to the LIS. Censure-derived are distribution of the year under analysis	^b Rate per 100,000 male Medicare beneficiaries in the same der	ne demographic stratum.							
	°Are-adjinsted to the US Census-derived are distribution of the	of the vear under analysis							

^dPersons of other races, unknown race and ethnicity, and other region are included in the totals.

NOTE: Counts less than 600 should be interpreted with caution. SOURCE: Centers for Medicare and Medicaid Services, 5% Carrier and Outpatient Files, 1992, 1995, 1998, 2001.

Table 23. Hospiti	al outpat	ient visits by M 1992	ledicare ben	eticiaries	with	testicular c 1995	ancer listed	as prima	ry diagno. 195	sis, col 38	int ^a , rate ^o (9	15% CI), aç	ge-adjusted rate 2001	5
	Count	Rate	Age- Adjusted Rate	Count		Rate	Age- Adjusted Rate	Count	Rate		Age- Adjusted Rate	Count	Rate	Age- Adjusted Rate
Totald	1,800	12 (9.6–15)		820	5.4	(3.7-7.0)		1,060	7.3 (5.4-	-9.3)		460	3.0 (1.8-4.2)	
Total < 65	1,320	42 (32–52)		520	15	(9.3–21)		006	26 (19–	-34)		200	5.3 (2.0-8.5)	
Total 65+	480	4.1 (2.4–5.7)	4.2	300	2.5	(1.3–3.8)	2.4	160	1.4 (0.4-	-2.5)	1.4	260	2.2 (1.0–3.5)	2.2
Age														
65–69	100	2.5 (0.3-4.6)		180	4.7	(1.6–7.7)		80	2.4 (0.1-	-4.7)		120	3.4 (0.7–6.1)	
70–74	140	4.3 (1.1–7.5)		40	1.2	(0-2.8)		20	0.7 (0-1	(6.		60	1.9 (0-4.2)	
75-79	160	7.1 (2.2–12)		60	2.6	(0-2.6)		60	2.6 (0-5	(9.5		80	3.3 (0.1–6.4)	
80-84	80	6.1 (0.2–12)		20	1. 4.	(0-4.2)		0	0			0	0	
85-89	0	0		0	0			0	0			0	0	
90–94	0	0		0	0			0	0			0	0	
95–97	0	0		0	0			0	0			0	0	
98+	0	0		0	0			0	0			0	0	
Race/ethnicity														
White	1,660	13 (10–16)	13	740	5.7	(3.9–7.5)	5.5	006	7.4 (5.2-	-9.5)	7.4	380	2.9 (1.6–4.2)	2.9
Black	20	1.6 (0-4.6)	1.6	20	1. 4.	(0-4.3)	1.4	20	1.5 (0-4	(4)	1.5	60	4.1 (0–8.7)	4.1
Asian	:	:	:	0	0		0	20	15 (0-4	(2)	15	0	0	0
Hispanic	:	:	:	20	10	(0-30)	10	120	36 (7.2-	-64)	36	20	5.3 (0–16)	5.3
N. American								,					,	
Native	:	:	:	0	0		0	0	0		0	0	0	0
Region														
Midwest	680	18 (12–25)	19	440	7	(6.6–16)	10	180	4.9 (1.7-	-8.1)	4.9	120	3.2 (0.6–5.7)	3.2
Northeast	540	17 (11–23)	16	180	5.7	(1.9–9.4)	5.7	80	2.9 (0.1-	-5.7)	2.9	80	2.7 (0.1–5.4)	2.7
South	200	3.8 (1.5–6.2)	3.8	80	1.5	(0-2.9)	1.5	620	12 (7.5-	-16)	12	120	2.1 (0.4–3.7)	2.1
West	380	16 (8.7–23)	15	120	5.2	(1.0–9.3)	6.0	160	7.2 (2.2-	-12)	7.2	120	4.8 (1.0–8.7)	4.8

...data not available.

^aUnweighted counts multiplied by 20 to arrive at values in the table.

^bRate per 100,000 male Medicare beneficiaries in the same demographic stratum.

^cAge-adjusted to the US Census-derived age distribution of the year under analysis.

^dPersons of other races, unknown race and ethnicity, and other region are included in the totals.

NOTE: Counts less than 600 should be interpreted with caution.

SOURCE: Centers for Medicare and Medicaid Services, 5% Carrier and Outpatient Files, 1992, 1995, 1998, 2001.

		1994	4		199	6	
	Count	Rate	Rate per 100,000 visits for Testicular Cancer	Count	Rate	Rate per 100,000 visits for Testicular Cancer	
Total	2,230	1.8 (1.6–2.0)		1,890	1.5 (1.3–1.7)		
Infusion of Chemotherapy Performed	364	0.3 (0.2–0.4)	16,323 (11,883–20,807)	298	0.2 (0.2–0.3)	15,767 (11,376–20,159)	
	1998			2000			
	Count	Rate	Rate per 100,000 visits for Testicular Cancer	Count	Rate	Rate per 100,000 visits for Testicular Cancer	
Total	1,993	1.5 (0.9–2.2)		1,907	1.4 (1.2–1.6)		
Infusion of Chemotherapy Performed	336	0.3 (0.2–0.3)	16,859 (11,139–22,529)	295	0.2 (0.2–0.3)	15,469 (10,383–20,556)	
^a Rate per 100,000 Corporation, for rel	is based on evant demo	1994–2000 popula graphic categories	ation estimates from Current of US male civilian non-insti	Population Su tutionalized po	rvey (CPS), CPS	Utilities, Unicon Research	

Table 24. Use of chemotherapy during inpatient hospital stays for testicular cancer listed as primary diagnosis, count, rate^a (95% CI), rate per 100,000 visits^b (95% CI)

SOURCE: Healthcare Cost and Utilization Project Nationwide Inpatient Sample, 1994, 1996, 1998, 2000.

^bRate per 100,000 male visits testicular cancer in HCUP_NIS 1994–2000.

Table 25. Estimated annual expenditures of privately insured employees with and without a medical claim for testicular cancer in 2002 ^a
Annual Expenditures (per person)

		A	inuai Experioiture	es (per person)		
	Ма	les Age 18–54		Ма	les Age 18–54	
	without Testi	cular Cancer (N=2	85,095)	with Testi	cular Cancer (N=2	236)
	Medical	Rx Drugs	Total	Medical	Rx Drugs	Total
Total	\$2,682	\$1,035	\$3,717	\$8,816	\$1,137	\$9,953
Age						
18–34	\$1,288	\$654	\$1,942	\$6,905	\$875	\$7,780
35–44	\$2,149	\$875	\$3,024	\$6,443	\$1,193	\$7,636
45–54	\$3,067	\$1,211	\$4,278	\$9,680	\$1,941	\$11,621
Region						
Midwest	\$2,584	\$1,022	\$3,606	\$8,492	\$1,126	\$9,618
Northeast	\$2,611	\$1,122	\$3,733	\$8,580	\$1,232	\$9,812
South	\$2,747	\$969	\$3,716	\$9,029	\$1,057	\$10,086
West	\$2,920	\$1,058	\$3,978	\$9,596	\$1,174	\$10,770

Rx, Prescription.

^aThe sample consists of primary beneficiaries ages 18 to 64 having employer-provided insurance who were continuously enrolled in 2002. Estimated annual expenditures were derived from multivariate models that control for age, gender, work status (active/ retired), median household income (based on zip code), urban/rural residence, medical and drug plan characteristics (managed care, deductible, co-insurance/co-payments) and binary indicators for 28 chronic disease conditions. Predicted expenditures for males age 55 to 64 are omitted due to small sample size.

SOURCE: Ingenix, 2002.

treatment. Either way, this disparity requires further study.

Hospital Outpatient Visits

In the Medicare data for 1992, 1995, 1998, and 2001, age-adjusted outpatient hospital visit rates fell consistently from 1992 to 1998 before rebounding slightly in 2001, for an overall decrease of 48% (Table 23). The decrease was most notable in men younger than 65 years of age (an 88% decrease). Outpatient visits from 1992 to 2001 decreased by 83% in the Midwest and Northeast, 68% in the West, and 45% in the South. There is no clear explanation for this variation.

Caucasians experienced a drop in outpatient hospital visits of 78%. A decrease would be expected for men on surveillance and outpatient chemotherapy, as these treatments commonly taking place in physicians' offices. Table 24 confirms that inpatient chemotherapy is declining. From 1994 to 2000, the rate of inpatient chemotherapy infusions decreased by 33%.

An increase in hospital outpatient visits would also be expected if there were an increase in the number of men receiving radiotherapy. Hence, when the data presented in Table 23 are combined with the inpatient hospital and physician office visit data presented earlier, one can postulate that Caucasian testicular cancer patients are receiving increasing surveillance and in-office chemotherapy treatments, whereas non-Caucasians are receiving less surveillance and more primary therapy, including radiation and procedures that require hospitalization, such as surgery and highdose chemotherapy.

ECONOMIC IMPACT

According to data from the Ingenix dataset for 2002, the estimated annual expenditure for privately insured individuals between the ages of 18 and 54 with a diagnosis of testicular cancer was \$9,953 (Table 25). Of this, \$8,816 was for medical costs, and \$1,137 was for prescription medications. The annual expenditure for males aged 18 to 54 without testicular cancer was \$3,717. The difference of \$6,236 (after controlling for differences in age distribution, median household income, type of health insurance, and 28 comorbid conditions) may be attributed to expenditures either directly or indirectly related to testicular cancer.

Men 45 to 54 years of age had the highest annual expenditure (Table 25), although sample sizes were small. Moreover, this age group had an increase in medication costs, which were 70% greater than the mean medication costs for all age groups. This may reflect a greater use of chemotherapy in the older patient population and a greater reliance on surgery and/or observation in younger patients. When stratified by region, costs were fairly consistent and generally correlated with expenditures of men without testicular cancer (Table 25).

National estimates of annual medical expenditures place the total cost of treating testicular cancer at \$21.8 million in 2000 (exclusive of medications) (Table 26), an increase of 10% over the total in 1994. Between 1994 and 2000, the percentage of total costs attributed to hospital outpatient visits remained stable at 7.7% to 8.7%, the percentage of ambulatory surgery costs remained stable at 14.9% to 16.8%, and inpatient costs decreased slightly, from 77.4% to 74.6%. Again, this reflects the trends already discussed, with care being transferred to the office and outpatient settings.

|--|

Table 20. Experiultur	es ioi lesticulai	cancer, by	Sile Of Service (/	o or iolai)				
Service Type	1994		1996		1998		2000	
Hospital Outpatient	\$1,521,508	7.7%	\$1,638,654	8.7%	\$1,740,460	8.4%	\$1,885,498	8.7%
Physician Office		0.0%		0.0%		0.0%		0.0%
Ambulatory Surgery	\$2,941,777	14.9%	\$3,168,275	16.9%	\$3,365,113	16.2%	\$3,645,539	16.8%
Emergency Room		0.0%		0.0%		0.0%		0.0%
Inpatient	\$15,300,472	77.4%	\$13,966,091	74.4%	\$15,642,173	75.4%	\$16,214,464	74.6%
TOTAL	\$19,763,756		\$18,773,020		\$20,747,745		\$21,745,500	

SOURCE: National Ambulatory and Medical Care Survey; National Hospital and Ambulatory Medical Care Survey; Healthcare Cost and Utilization Project; Medical Expenditure Panel Survey, 1994, 1996, 1998, 2000.

		19	99		2000			2001		
	Count	N	lean Cost	Count	I	Mean Cost	Count	N	lean Cost	
Total	23	\$9,177	(6,282–12,073)	23	\$20,603	(10,141–31,066)	44	\$21,892	(8,574–35,210)	
Age										
0–2	2	\$3,955	(2,748–5,162)	5	\$18,629	(0-51,259)	9	\$5,663	(3,760-7,565)	
3–10	6	\$9,817	(639–18,995)	2	\$12,895	(0-66,586)	6	\$10,390	(3,009–17,771)	
11–17	14	\$9,737	(6,012-13,462)	15	\$23,445	(9,003-37,886)	26	\$22,850	(7,664–38,035)	
18–24	1	\$7,947		1	\$3,272		3	\$85,286	(0-397,727)	
Race/ethnicity										
White	19	\$9,907	(6,698–13,115)	12	\$14,138	(3,672-24,603)	28	\$15,030	(9,324–20,736)	
Black	1	\$11,700		6	\$32,129	(0-68,690)	5	\$46,139	(0–147,741)	
Asian	1	\$0		1	\$65,623		1	\$3,861		
Hispanic	0	\$0		2	\$7,270	(0-17,023)	7	\$39,275	(0–117,340)	
N. American						. ,			. ,	
Native	0	\$0		0	\$0		0	\$0		
Missing	1	\$0		1	\$22,515		3	\$10,975	(0-26,967)	
Other	1	\$11,151		1	\$8,774		0			
Region										
Midwest	7	\$8,484	(2,457–14,510)	9	\$23,815	(839–46,792)	14	\$11,991	(2,670–21,311)	
Northeast	2	\$8,752	(0-19,102)	1	\$3,272		3	\$82,816	(0-400,428)	
South	9	\$10,291	(4,837–15,746)	11	\$20,520	(5,235-35,804)	20	\$22,382	(2,877-41,887)	
West	4	\$10,391	(0-24,094)	2	\$15,276	(0–107,250)	7	\$14,184	(5,690-22,677)	
Missing	1	\$0		0	\$0		0	\$0		
data not availabl	0				0		0			

Table 27. Mean inpatient cost per child admitted with testicular cancer listed as primary diagnosis, count, mean cost^a (95% Cl)

...data not available.

^aCalculated using adjusted ratio of costs to charges, including variable and fixed cost among participating children's hospitals.

SOURCE: National Association of Children's Hospitals and Related Institutions, 1999–2001.

Testicular cancer is rare in pre-pubertal males. However, data from the National Association of Children's Hospitals and Related Institutions (NACHRI) database indicate that the mean inpatient cost per child with testicular cancer listed as a primary diagnosis was \$21,892 in 2001, a 2.3-fold increase over the cost in 1999 (Table 27). In summary data from 1999-2001, increases in costs correlated directly with increases in age: males 11 years of age and older with a primary diagnosis of testicular cancer had costs nearly three times greater than those for patients 10 and under. This may be due to the fact that older children admitted to inpatient facilities had a higher proportion of recurrent cancers involving moreintensive care, while younger patients were admitted for their initial cancer procedure.

Marketscan data from 1999 allow assessment of the impact of a diagnosis of testicular cancer on employment (Table 28). Most men with testicular cancer are in the age range where they would be either enrolled in school or employed. Market scan data indicate that 16% percent of men with testicular cancer missed work for treatment of the disease. An average of 0.7 hour of work was missed for inpatient hospitalization, and 7.7 hours were missed for outpatient visits. Hence, the average total hours of work missed was 8.4. This suggests that most of the men with testicular cancer were under surveillance or underwent primary treatment prior to 1999, either of which would result in only occasional follow-up visits to a physician's office. Overall, the impact of testicular cancer on the workplace seems limited.

CONCLUSIONS

Testicular cancer is relatively uncommon, constituting less than 1% of all male malignancies. Still, it is currently the most common cancer in men 20 to 34 years of age. Although the incidence of testicular cancer in the United States continues to rise, the rate of increase is slowing. The reasons for this are unknown, although there is speculation that an increase in environmental endocrine disruptions may play a role (15).

Fortunately, testicular tumors are exceedingly curable. Their successful treatment represents a

v				Average Work Absence (I	nrs)
	Number of Workers ^a	% Missing Work	Inpatient ^b		Total
Total	45	16%	0.7 (0–2.1)	7.7 (0–19.5)	8.4 (0–20.3)
Age					
18–29	5	0%	0	0	0
30–39	16	19%	2 (0-6.3)	0.8 (0-1.9)	2.8 (0-7.1)
40–49	18	17%	0	17.9 (0-48.6)	17.9 (0-48.6)
50-64	6	17%	0	1.8 (0-6.5)	1.8 (-0-6.5)
Region					
Northeast	4	0%	0	0	0
North Central	15	13%	0	1.3 (0–3.1)	1.3 (0–3.1)
South	18	17%	1.8 (0-5.5)	2.9 (0-8.5)	4.7 (0-11.2)
West	5	40%	0	54.7 (0-198.3)	54.7 (0-198.3)
Unknown	3	0%	0	0	0

Table 28. Average annual work loss of males treated for testicular cancer

^aIndividuals with an inpatient or outpatient claim for testicular cancer and for whom absence data were collected. Work loss based on reported absences contiguous to the admission or discharge dates of each hospitalization or the date of the outpatient visit. ^bInpatient and outpatient include absences that start or stop the day before or after a visit.

SOURCE: Marketscan Health and Productivity Management, 1999.

medical triumph and underscores the strength of multimodality therapy. Overall, the death rate from testicular cancer continues to decrease. However, African American men have experienced a slight decline in survival, possibly due to differences in access to care.

Modifications in surgical technique and radiotherapy, as well as improved methods of employing systemic chemotherapy, have substantially diminished the morbidity of therapy. More patients are being treated with surveillance for early-stage disease. In Caucasian men, who are the vast majority of testicular cancer patients, care has shifted to the outpatient setting. Because of these successes, however, the treatment paradigms for testicular cancer are changing. As a result, there is little standardization in treatment approaches. This, coupled with the relative rarity of testicular cancer and subsequent limited database information, makes evaluation for a project such as Urologic Diseases in America very difficult. There is a need to collect more-comprehensive, detailed information so that the burden of testicular cancer on patients and the economy can be better evaluated.

Several recent high-profile celebrity and athlete cases of testicular cancer and their attendant publicity have increased awareness of the disease. This may mitigate fear and embarrassment and encourage men to seek care.

RECOMMENDATIONS

The underlying limitations to this analysis are that testicular cancer is a relatively rare disease and it occurs in young men, a population that is not routinely captured in database studies. The following efforts could improve data collection and analysis.

Classification and Coding

- ICD and CPT codes should be more detailed, and greater attention should be given to the therapeutic management options most germane to testicular cancer: surveillance, specifying orchiectomy for testicular cancer and differentiating *de novo* and postchemotherapeutic RPLND, and denoting when radiotherapy and chemotherapy are given as a primary treatment or in the salvage setting.
- The underlying causes of infertility in men with testicular cancer (for example, innate, post-RPLND anejaculation, and/or chemotoxicity) should be detailed more thoroughly in database studies.
- Terminology and coding need to be standardized.

Data Collection

- Data should be collected with attention to accurate staging information.
- An objective and standardized staging system (see Table 2) should be used.
- Terms such as "early" and "late" disease are subjective and should be discouraged.
- Clinical and pathologic staging data should be separated and detailed individually.
- Data should be collected with attention to histology.
- Data should be collected with strict regard to risk stratification, which takes into consideration clinical, radiographic, serologic, and pathologic features.

Impact on Education

- Since many men with testicular cancer are enrolled in college or other educational institutions, the impact of a diagnosis on education should be evaluated. Relevant questions include:
 - How much school is missed? Is graduation delayed?
 - What is the financial impact of missing school after tuition has been paid?

Racial/Ethnic Data

Several disturbing and provocative findings with regard to racial disparities need to be addressed:

- Why did African American men experience a decline in survival rates in the 1990s?
- Are there genetic and biological differences in testicular cancer among different ethnic groups?
- Why do treatment patterns for testicular cancer appear to be different for Caucasian and non-Caucasian patients?
- Why is the treatment of minority children with testicular cancer more expensive than that of Caucasian children?
- Is there a racial disparity in the treatment of testicular cancer? If so, how do we rectify the situation?

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