CNE Objectives and Evaluation Form appear on page 264.

CNE SERIES

An Overview of Prostate Cancer: Diagnosis and Treatment

Dawn Mielke Strief

This review of the state of the science for prostate cancer includes a description of current screening, diagnosis, and treatment options for localized prostate cancer. Educational resources appropriate for at-risk patients are outlined.

— Linda H. Yoder, PhD, MBA, RN, AOCN®, FAAN

Prostate cancer is the most commonly diagnosed cancer and the second leading cause of cancer-related death among American males (Terris & Rhee, 2006). The incidence of this disease is highest among Caucasian and African-American males, with reported incidences of 161/100,000 and 256/100,000, respectively. The estimated overall incidence of prostate cancer in the United States for 2008 is 186,320, with projected mortality of 28,660 (Jemal et al., 2008). Despite the high incidence of the disease, positive outcomes can be achieved through early, effective, and appropriate treatment. Many health professionals will care for individuals with prostate cancer, or know individuals at risk for developing the disease. All nurses should be familiar with the guidelines for screening, treating, and managing early or localized prostate cancer.

Stages of Prostate Cancer

Localized prostate cancer has been defined as a cancer confined to the prostate (Bott, Birtle, Taylor, & Kirby, 2003). Research to date has failed to link prostate cancer to any specific modifiable lifestyle choices, but it has identified non-modifiable risk factors. These include increasing age, a family history of the disease, and ethnicity. Higher incidences of localized prostate cancer are reported among men over age 60, especially in African Americans. Caucasian men have the second highest incidence of prostate cancer, and rates of the disease are lowest among men of Asian-American and Hispanic-American decent (U.S. Department of Health and Human Services, 2006). The American Cancer Society (ACS) (2007) estimates that one in six men will be diagnosed with prostate cancer during their life-

Dawn Mielke Strief, MS, RN, ANP, GNP, is a Nurse Practitioner, VA Medical Center, Minneapolis, MN.

Editor's Note: This article is reprinted with permission from *Urologic Nursing, 27*(6), 475-479. Certain facts and figures have been updated and modified for *MEDSURG Nursing* readers by "Cancer: Caring and Conquering" Column Editor Linda H. Yoder, PhD, MBA, RN, AOCN®, FAAN.

Notes: This column is made possible through an educational grant from C-Change, a 501(3)c (not-for-profit) organization. The purpose of the "Cancer: Caring and Conquering" column is to strengthen the cancer knowledge, skills, and confidence of medical-surgical nurses who care for patients at risk for or living with cancer.

C-Change is a not-for-profit organization whose mission is to eliminate cancer as a public health problem, at the earliest possible time, by leveraging the expertise and resources of our members. C-Change is the *only* organization that assembles cancer leaders from the three sectors – private, public, and not-for-profit – from across the cancer continuum – prevention, early detection, treatment, and quality of life. C-Change invests in the resolution of problems that cannot be solved by one organization or one sector alone. For more information about C-Change, visit www.c-changetogether.org.

The author and all *MEDSURG Nursing* Editorial Board members reported no actual or potential conflict of interest in relation to this continuing nursing education article.

time, but only 1 in 35 will die of this disease.

The identification of family history as a major risk factor for prostate cancer has resulted in a great deal of research to isolate a genetic component (Lessick & Katz, 2006). The first prostate cancer susceptibility gene, HPC1 (Hereditary prostate cancer 1), was discovered in 1996 (Smith et al., 1996) and mapped to the long arm of chromosome 1. Since then, several other prostate cancer susceptibility genes have been linked to various regions on other chromosomes (Ostrander, Markianos, & Stanford, 2004; Verhage & Kiemeney, 2003).

In addition to the discovery of the HPC1 gene, several other theories have been developed in an attempt to explain the genetic basis of prostate cancer. The Mendelian autosomal dominant inheritance theory is thought to best explain familial clustering of prostate cancer among men with early-onset disease. Approximately 43%-65% of prostate cancer cases diagnosed in patients before age 56 have been linked to the presence of a rare autosomal dominant, high-risk susceptibility gene (Verhage & Kiemeney, 2003). A multifactorial model has been developed which describes how prostate cancer may occur when several susceptible genes interact with environmental factors (Gong et al., 2002).

A virus was identified recently which may be an environmental influence on the development of prostate cancer. The HPC1 gene has been implicated in viral defense, and scientists have found that men with mutations in their HPC1 gene harbor this virus 30 times more than men without the genetic mutation. The HPC1 gene encodes an antiviral protein which is activated by viral infection. Any impairment in this gene has been proposed as a susceptibility factor in the development of prostate cancer (Hampton, 2006). There has been speculation that the virus interacts with the prostate and the tissue surrounding it to cause prostate cancer. Further research in this area is targeting development of a vaccine to prevent prostate cancer (Simard et al., 2003).

Prostate cancer frequently offers no specific clinical symptoms. Lower urinary tract symptoms may be present, but these are neither specific nor sensitive enough to diagnose prostate cancer. Lower urinary tract symptoms are more specific to another condition known as benign prostatic hyperplasia (BPH) and should not be correlated directly to the presence of prostate cancer. However, if prostate cancer is present, lower urinary tract symptoms also may be present, especially if the prostate enlarges or intrudes into the urethral space. These symptoms may include urgency, hesitancy, frequency, dysuria, weak stream, and urine leakage. In a review article, Hamilton and Sharp (2004) determined that lower urinary tract symptoms are more prevalent in the presence of prostate cancer, yet the high prevalence of these symptoms among the general population decreases their predictive value. No evidence thus suggests that lower urinary tract symptoms are associated with localized prostate cancer, and their presence is not sensitive or specific enough to aid in the diagnosis of prostate cancer.

At present, no symptoms are specific to the diagnosis of prostate cancer. Rather than rely on symptoms, patients should have routine prostate cancer screening, which includes a digital rectal examination (DRE) and obtaining a blood sample to determine the presence of the prostate-specific antigen (PSA). If one or both of these screening examinations are abnormal, further investigation should occur. Intervention at this stage allows prostate cancer to be detected early, when it may be localized and treatment may cure or control.

Diagnostic Work-Up

Screening. PSA is an enzyme created only by prostate cells (Stutzman, 2003); it is a normal product of the prostate gland and normal component of the seminal fluid. Minute amounts of this protein leach into the blood and are detectable quantitatively by several chemical assays (Linn, Ball, & Maradiegue, 2007). Catalona and colleagues (1991) first reported the use of PSA serum levels as a first-line screening tool to detect prostate cancer. They found that a single PSA determination was more accurate than a DRE, previously the standard and only method available to detect prostate cancer and to identify early prostate cancer. Elevated levels of PSA have been correlated positively with prostate cancer, but are not specific for prostate cancer. Elevated levels of PSA also are seen in the presence of BPH, prostatitis, prostate abscess, manipulation of the prostate, prostatic infarction, and ejaculation within the previous 48 hours (Stutzman, 2003). Thus, screening only by PSA results has been met with some controversy.

The variability of PSA results within an individual further complicates testing and contributes to the continuing controversial nature of the test. Soletormos and associates (2005) determined that biological variations of total prostate-specific antigen (tPSA) exist. These researchers determined that while tPSA is increasingly used for screening, diagnosis, and monitoring of prostate cancer, serial measurements of individual tPSA levels varied by more amplitude than could be accounted for by the analytical variation of the test. This variation was believed to be caused by intra-individual variation. Further research must be performed to determine if the variation of tPSA

is greater in men with known prostate cancer when compared to those who do not have the disease. It appears that the biological variation of tPSA is 20% and is influenced by age. Thus, using this laboratory value to screen or follow treatment is not a certainty. See Table 1 for age-specific reference ranges for serum PSA.

In addition to the biological variation associated with tPSA levels, the accuracy of the PSA test should be explored. The Physicians' Health Study, performed by Gann and colleagues in 1995 (Harris & Lohr, 2002), used longitudinal follow-up data rather than biopsy to determine the sensitivity and specificity of the PSA test. Analysis of these data determined that the sensitivity for PSA in detecting prostate cancer within 2 years was 73.2%. The specificity of the PSA test was 85.4% among men who did receive a diagnosis of prostate cancer within 2 years.

These data also determined that the specificity of the PSA level is lower among men with large prostate glands, a population which includes men with BPH. The researchers concluded that prostate cancer screening using the PSA test is not appropriate among men with BPH because the PSA level would be decreased falsely. The results of this study have led experts to suggest that PSA levels be adjusted for age, with the abnormal PSA level decreased from 4.0 ng/mL to 2.6 ng/mL (Gretzer & Partin, 2003).

Digital rectal examinations also are used as a screening mechanism for prostate cancer. In a metaanalysis, Harris and Lohr (2002) determined that the sensitivity of this test was 59% and its specificity was undeterminable. Thus, while this procedure may be useful in detecting prostate cancer among those with a low PSA, the limited reproducibility of the examination limits its clinical significance.

Prostate cancer screening may result in a false-positive response that can lead to unnecessary and invasive testing. The net benefit of prostate screening activities thus remains controversial. However, even in light of these facts, the American Urological Association and ACS recommend offering a screening PSA along with a DRE for men age 50 and above who are expected to live longer than 10 years. Men at higher risk, such as African Americans and men with a family history of PCA, should begin testing at age 40-45, or 5 years before the age of onset of disease of the affected family member. The DRE is considered an integral part of the screening protocol and is used in conjunction with the PSA. The sensitivity of diagnosing tumors is improved markedly with the use of both modalities (Coley, Barry, Fleming, & Mulley, 1997). See Table 2 for a variety of current screening recommendations for prostate cancer. Clearly, further research needs to occur with respect to appropriate screening activities, the meaning of the results, and necessity and timing of further diagnostics and treatment.

Table 1.
Age-Specific Reference Ranges for Serum PSA

Age Range	Asians	African Americans	Whites
40-49 years	0-2.0 ng/mL	0-2.0 ng/mL	0-2.5 ng/mL
50-59 years	0-3.0 ng/mL	0-4.0 ng/mL	0-3.5 ng/mL
60-69 years	0-4.0 ng/mL	0-4.5 ng/mL	0-4.5 ng/mL
70-79 years	0-5.0 ng/mL	0-5.5 ng/mL	0-6.5 ng/mL

Source: Richardson & Oesterling, 1997.

Diagnostic examinations. Men with prostate cancer usually have a PSA result higher than 10 ng/mL, while men with BPH rarely have such an elevation. Men who have a PSA serial increase in 1 year of 0.75 ng or more over baseline PSA should undergo further screening. This rate of change is known as *PSA velocity*, and has been associated with and specific for the diagnosis of localized prostate cancer (Berger et al., 2007). If the patient is asymptomatic, an elevated PSA should be correlated with a DRE. If these results create a suspicion for prostate cancer, a referral and prostate biopsy should follow. If results are positive, the cancer should be staged using the Gleason Scoring System, the Tumor, Nodes, Metastasis (TNM) classification, or the Whitmore-Jewett staging system (Stutzman, 2003).

Gleason Scoring System. The Gleason Scoring System separates the cancer diagnosis into five different histologic grades. Grade 1 tissue is well-differentiated and provides the best prognosis. Treatment for individuals with a Grade 1 tumor may result in a cure. Poorly differentiated cancers are classified as Grade 5 and carry a poor prognosis. Affected individuals require extensive treatment with an appropriate goal being tumor control. Tissue samples are obtained from two different sites, with the grade for each tissue sample defined separately and the two scores added. The highest Gleason score possible is 10, if each site obtains a grade of 5. Prostatic intraepithelial neoplasia (PIN) also may be identified in the biopsy tissue. PIN is known to be a premalignant lesion for prostate cancer and these individuals have an increased risk for prostate cancer in subsequent biopsies (Stutzman, 2003).

Table 2.
Recommendations for Screening

Organization	Published Recommendations for Screening
American Urologic Association	The prostate-specific antigen (PSA) test and the digital rectal examination (DRE) should be offered annually, beginning at age 50, to men who have a life expectancy of at least 10 years. Men at high risk should begin testing at age 45.
American Cancer Society	Annual screening: The American Cancer Society believes that doctors should offer the PSA blood test and DRE (digital rectal exam) yearly, beginning at age 50 to men who do not have any major medical problems and can be expected to live at least 10 more years. Men at high risk should begin testing at age 45. Men at high risk include African Americans and men who have a close relative (father, brother, or son) who had prostate cancer before age 65. Men at even higher risk (because they have several close relatives with prostate cancer at an early age) could begin testing at age 40. Depending on the results of the first tests, they might not need more testing until age 45.
United States Preventative Services Task Force	There is insufficient evidence to support prostate cancer screening.
National Cancer Institute	No published standards or guidelines. The National Cancer Institute posts information: "What is PSA?" "Why is PSA performed?" "For whom is it recommended?" http://www.cancer.gov/cancertopics/factsheet/Detection/PSA
American College of Physicians	There is insufficient evidence to support prostate cancer screening.
American College of Family Physicians	No published standards or guidelines.
Centers for Disease Control	Although there is good evidence that PSA screening can detect early-stage prostate cancer, evidence is mixed and inconclusive about whether early detection improves health outcomes. Additionally, prostate cancer screening is associated with important harms, which include anxiety and followup procedures based on test results that sometimes are false positive, as well as the complications that may result from treating prostate cancers that, if left untreated, might not have affected the man's health.
U.S. Department of Veterans Affairs	There is insufficient evidence to support prostate cancer screening.
National Comprehensive Cancer Network	Practice Guidelines in Oncology suggest "talking points" to discuss with patients. The pros and cons of screening are listed within this guideline. Men should make an informed decision about having a PSA screening test.

Tumor staging classifications. Stage A (T1, N0, M0) prostate cancer is disease that cannot be palpated during DRE of the prostate. This stage is further divided into T1a and T1b. T1a is well-differentiated and involves less than 5% of the prostate gland. T1b also is well differentiated, yet involves more than 5% of the prostate gland. This determination often is made during a surgical procedure for symptoms of BPH. Stage T1c indicates that the prostate gland was biopsied as a result of an elevated PSA level (Stutzman, 2003).

Stage B (T2, N0, M0) is limited to the prostate gland and usually is found by DRE in which a nodule or hardness may be palpated. Stage T2a is defined as the presence of a palpable node which involves up to one-half of one lobe. Stage T2b is defined as a palpable lesion which involves more than half of one lobe, but not both lobes. Stage T2c describes a palpable node, present in both lobes (Stutzman, 2003).

Stage C (T3 and T4, N0, M0) is local-extensive prostate cancer. This stage is marked by presence of a palpable node and unilateral capsular penetration. Stage T3b describes the presence of a palpable node which has bilaterally extracapsular extension. Stage T3c involves a palpable node which has invaded the seminal vesicles (Stutzman, 2003).

Stage D (T4, M+) is a cancer which has invaded adjacent structures, such as the bladder neck, sphincter, rectum, or pelvic wall. The inclusion of N or M in the staging describes a cancer that has metastasized to the lymph nodes or has distant metastases (such as

bone), respectively (Stutzman, 2003).

Whitmore-Jewett Staging. The Whitmore-Jewett Staging classification is similar to the TNM classification. Stage A is a clinically undetectable tumor, usually found incidentally. Stage A1 is focal and well-differentiated. Stage A2 is diffuse or poorly differentiated. Stage B is limited to the prostate upon rectal examination. Stage B1 implies one solitary nodule, less than 1.5 centimeters, and involves only one lobe. Stage B2 involves one whole lobe or both lobes. Stage C extends locally outside the prostatic capsule or into the seminal vesicles. Stage D implies metastatic disease; Stage D1 involves pelvic lymph node metastases, while Stage D2 involves distant metastases (Stutzman, 2003).

Treatment Options

Watchful waiting. Watchful waiting, or active surveillance, involves routine observation of a patient's prostate cancer clinically with routine PSA testing. According to Bott and associates (2003), a link exists between Gleason scores and PSA levels at time of diagnosis to rate of progression. Higher grades of prostate cancer increase the risk of metastases and death. The risk of having a known prostate cancer progress, other health care concerns, and co-morbidities, along with other demographic data that may place this person at risk for disease progression, should be included when treatment is selected.

Radical prostatectomy. This surgical procedure requires removal of the entire prostate gland, along with the seminal vesicles and usually the obturator lymph nodes. These nodes are obtained for cancer staging purposes. Surgical approaches can include a transperineal route, a laparoscopic approach, or a transurethral resection of the prostate. Each approach has obtained similar outcomes, but the transperineal approach may be accomplished with less blood loss, no abdominal incision, and decreased pain. This approach has two potential disadvantages. First, because lymph node sampling is not possible, an additional procedure will be required. In addition, this approach cuts the prostate during removal, which may pose a risk of tumor seeding.

Potential side effects or complications associated with a radical prostatectomy include temporary urinary incontinence and impotence. Urinary dysfunction subsides within the first year after surgery, and potency is regained for approximately 80% of patients (Bott et al., 2003). Return of these physical functions is dependent on patient age, the disease stage, and if nerve bundles are intact postoperatively.

Many men who do not regain potency can benefit from an oral phosphodiesterase type 5 inhibitor such as sildenafil (Viagra®) (Bott et al., 2003). Sildenafil has the best results for patients less than age 60 who had a bilateral nerve-spare procedure and had some spontaneous return of erectile function after surgery. The effect of sildenafil increased over time, generally requiring 12-24 months following surgery for maximum results; 35%-75% of patients regained potency after a nerve-sparing surgery, compared to 0-15% of those who underwent non-nerve-sparing surgery (Briganti et al., 2007).

Robotic prostatectomy. Robotic prostatectomy is a laparoscopic prostatectomy aided by a surgeon-assisted robot. This procedure increases visualization of delicate structures that surround the prostate, adds dexterity to the surgical removal, and eliminates surgeon-dependent hand movements. Data on the effectiveness of robotic prostatectomy are reported to be comparable to open and laparoscopic prostatectomy in terms of efficacy and clinical outcomes (El-Hakim & Tewari, 2004).

Robotic prostatectomy appears to be slightly more efficient at reducing complications related to impotency, urinary incontinence, and recovery time (Starnes & Sims, 2006). Robotic prostatectomy is slightly more expensive than other surgical procedures and may not be available at all treatment centers.

External beam radiotherapy. External beam radiotherapy treatment (EBRT) has similar efficacy to surgery with some differing complications. EBRT usually is performed every weekday for 4-6 weeks. This procedure affects normal tissue, resulting in temporary adverse effects that include diarrhea, tenesmus, proctitis, dysuria, frequency, and lethargy. Long-term effects of EBRT include impotence in 10%-30% of men. In addition, chronic proctitis may lead to bleeding and fibrosis. Incontinence usually is not a side effect from this treatment. Disturbances in stool frequency are reported among fewer than 20% of men (Bott et al., 2003).

Brachytherapy. Brachytherapy involves the insertion of a radioactive source directly into the prostate gland. This procedure provides a high dose of radiation delivered locally, which spares surrounding normal tissue. Brachytherapy may be performed using either iodine-125 seeds or iridium-192 rods. Iodine-125 seeds are placed permanently and recommended for patients with a life expectancy of at least 10 years, a Gleason score of 6 or less, a prostate volume of less than 50 milliliters, and no previous prostate surgery. Potential complications associated with this procedure include irritation to the urethra, which results in incontinence and impotence.

Iridium-192 rods are placed temporarily and recommended for patients with PSA results greater than 10 or a Gleason score of 8-10.

Along with iridium-192 rod placement, EBRT is delivered over 4 weeks. Potential complications associated with this procedure include incontinence and impotence.

Radiation damage to the urethra can result in both irritating and obstructive urinary symptoms. These symptoms may last up to 60 days, which is the half-life of the iodine rods. Approximately 10%-15% of men who undergo brachytherapy experience erectile dysfunction. Use of phosphodiesterase type 5 inhibitors may help them regain sexual function (Bott et al., 2003).

Hormonal manipulation. Patients with a Gleason score of 8-10 can benefit from at least 2 years of adjuvant luteinizing hormone-releasing hormone agonist therapy following EBRT, according to a review by Bott and associates (2003). Significant increases have been documented in disease-specific survival and overall survival with a median follow up of over 5 years. The use of adjuvant hormonal therapy in conjunction with standard treatments is being researched. The results at this time seem promising, but further research is needed to determine the long-term effects on bone density and endocrine metabolism. The disease-specific survival figures also must be known before an approach such as this can be considered standard.

Follow-up care. Serum PSA is synthesized almost exclusively by the prostate gland, making its assessment an excellent method to determine treatment response. The PSA level should be decreased after treatment, and be close to zero after surgery. Routine screening using the PSA should continue to occur after treatment. A rising or elevated PSA level may indicate either residual prostate cancer or recurrence and should be further investigated (Naito, 2005). Radiographic and nuclear medicine examinations can aid in detecting recurrent prostate cancer. If cancer recurs, additional treatment may be recommended.

Patient and Family Education

Education, knowledge, and understanding are critical factors in making an informed decision with respect to treatment decision making (Lepore, Helgeson, Eton, & Schulz, 2003). Education should include accurate information, pathophysiology, grading and staging, treatment options, and potential short-term and long-term side effects and adverse effects (Held-Warmkessel, 2002). Fagerlin and associates (2004) performed a critical review of existing patient education materials for localized prostate cancer. They concluded that few materials were available which provided assistance in making treatment decisions. Most of the reviewed documents failed to describe all treatment options, did not contain complete information with respect to potential adverse effects of treatment, and required a high school reading ability to comprehend. Regarding Internet sources for health information, patients and their families should be encouraged to visit nationally recognized sites to receive accurate information. The five highest-rated patient education Web sites are the ACS (www.cancer.org), AstraZeneca (www.prostateinfo.com), Memorial Sloan-Kettering Cancer Center (www.mskcc.org), Cancer Care, Inc. (www.cancercare.org), and University of Toronto (www.prostatecentre.ca) (Fagerlin et al., 2004). These sites may provide education, guide treatment decision making, and assist diagnosed patients to cope with their disease and treatment. ■

References

American Cancer Society (ACS). (2007). *Detailed guide: Prostate cancer. What are the key statistics about prostate cancer?* Retrieved June 18, 2008, from http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_are_the_key_statistics_for_prostate_canc er_36.asp?sitearea

Berger, A., Deibl, M., Strasak, A., Bektic, J., Pelzer, A., Klocker, H., et al. (2007). Large-scale study of clinical impact of PSA velocity: Long-term PSA kinetics as method of differentiating men with from those without prostate cancer. *Urology, 69*(1), 134-138.

Bott, S., Birtle, A., Taylor, C., & Kirby, R. (2003). Prostate cancer management: An update on localized disease. *Postgraduate Medical Journal, 79*, 575-580.

Briganti, A., Salonia, A., Gallina, A., Chun, F., Karakiewicz, P., Graefen, M., et al. (2007). Management of erectile dysfunction after radical prostatectomy in 2007. *World Journal of Urology, 25*(2), 143-148.

Catalona, W., Smith, D., Ratliff, T., Dodds, K., Coplen, D., Yaun, J., et al. (1991). Measurement of prostate specific antigen in serum as a screening test for prostate cancer. *New England Journal of Medicine, 324*, 1156-1161.

Coley, C., Barry, M., Fleming, C., & Mulley, A. (1997). Early detection of prostate cancer: Part 1: Prior probability and effectiveness of tests. *Annals of Internal Medicine, 126*, 394-406.

El-Hakim, A., & Tewari, A. (2004). Robotic prostatectomy – A review. *Medscape General Medicine, 6*(4), 20.

Fagerlin, A., Rovner, D., Stableford, S., Jentoft, C., Wei, J., & Holmes-Rovner, M. (2004). Patient education materials about the treatment of early-stage prostate cancer: A critical review. *Annals of Internal Medicine, 140*, 721-728.

Gong, G., Oakley-Girvan, I., Wu, A., Kolonel, L., John, E., West, D., et al. (2002). Segregation analysis of prostate cancer in 1,719 white, African-American, and Asian-American families in the United States and Canada. *Cancer Causes and Control, 13*, 471-482.

Gretzer, M., & Partin, A. (2003). *Campbell's urology updates: PSA and PSA molecular derivatives, 1*(2), 1-12.

Hamilton, W., & Sharp, D. (2004). Symptomatic diagnosis of prostate cancer in primary care: A structured review. *British Journal of General Practice, 54,* 617-621.

Hampton, T. (2006). New virus linked to prostate cancer. *JAMA, 295*(13), 1503.

Harris, R., & Lohr, K. (2002). Screening for prostate cancer: An update of the evidence for the U.S. Preventive Services Task Force. *Annals of Internal Medicine, 137*, 917-929.

Held-Warmkessel, J. (2002). What your patient needs to know about prostate cancer. *Nursing2002, 32*(12), 36-42.

Jemal, A., Siegel, R., Ward, E., Haoe, Y., Xu, J., Murray, T., et al. (2008). Cancer statistics, 2008. *CA: A Cancer Journal for Clinicians, 58*(2), 71-96.

continued on page 269

Cancer: Caring and Conquering

continued from page 263

Lepore, S., Helgeson, V., Eton, D., & Schulz, R. (2003). Improving quality of life in men with prostate cancer: A randomized controlled trial of group education interventions. *Health Psychology, 22*(5), 443-452.

Lessick, M., & Katz, A. (2006). A genetics perspective on prostate cancer. *Urologic Nursing, 26*(6), 454-460.

Linn, M., Ball, R., & Maradiegue, A. (2007). Prostate-specific antigen screenings: Friend or foe? *Urologic Nursing, 27*(6), 481-490.

Naito, S. (2005). Evaluation and management of prostate-specific antigen recurrence after radical prostatectomy for localized prostate cancer. *Japan Journal of Clinical Oncology, 35*(7), 365-374.

Ostrander, E., Markianos, K., & Stanford, J. (2004). Find prostate cancer susceptibility genes. *Annual Review of Genomics and Human Genetics, 5,* 151-175.

Richardson, T., & Oesterling, J. (1997). Age-specific reference ranges for serum prostate-specific antigen. *Urology Clinics of North America, 24,* 339-351.

Simard, J., Dumont, M., Labuda, D., Sinnett, D., Meloche, C., El-Alfy, M., et al. (2003). Prostate cancer susceptibility genes: Lessons learned and challenges posed. *Endocrine-Related Cancer, 10*, 225-259.

Smith, J.R., Freije, D., Carpten, J., Gronberg, H., Xu, J., Isaacs, S., et al., (1996). Major susceptibility locus for prostate cancer on chromosome 1 suggested by a genome-wide search. *Science, 274*, 1371-1374.

Soletormos, G., Semjonow, A., Sibley, P., Lamerz, R., Petersen, P., Albrecht, W., et al. (2005). Biological variation of total prostate-specific antigen: A survey of published estimates and consequences for clinical practice. *Clinical Chemistry, 51*(8), 1342-1351.

Starnes, D., & Sims, T. (2006). Care of the patient undergoing robotic-assisted prostatectomy. *Urologic Nursing, 26*(2), 129-136.

Stutzman, R. (2003). Bladder outlet obstruction. In L. Barker, J. Burton, & P. Zieve (Eds.), *Principles of ambulatory medicine* (6th ed., pp. 725-736). Philadelphia: Lippincott, Williams & Wilkins.

Terris, M., & Rhee, A. (2006). *Prostate cancer: Metastatic and advanced disease.* Retrieved May 22, 2007, from http://www.emedicine.com/med/topic3197.htm

U.S. Department of Health and Human Services. (2006). *United States cancer statistics: 2003 incidence and mortality.* Retrieved May 22, 2007, from http://www.cdc.gov/cancer/npcr/npcrpdfs/US_Cancer_Statistics_2003_Incidence_and_Mortality.pdf

Verhage, B., & Kiemeney, L. (2003). Genetic susceptibility to prostate cancer: A review. *Familial Cancer, 2,* 57-67.

MSN J0812

Answer/Evaluation Form:
An Overview of Prostate Cancer: Diagnosis and Treatment

COMPLETE THE FOLLOWING

This test may be copied for use by others.

Name: _____

Address: _____

City:_____State: _____ Zip: _____

Preferred telephone: (Home)_____ (Work) _____

Registration fee: **Complimentary CNE provided as an educational service by C-Change (www.c-changetogether.org).**

ANSWER FORM

1. If you applied what you have learned from this activity into your practice, what would be different?

_____

_____

_____

_____

Evaluation	Strongly disagree				Strongly agree
2. By completing this activity, I was able to meet the following objectives:					
a. Describe the stages of prostate cancer.	1	2	3	4	5
b. Discuss the diagnostic work up for prostate cancer.	1	2	3	4	5
c. List the treatment options for prostate cancer.	1	2	3	4	5
d. Explain the nurse's role in patient and family education related to prostate cancer.	1	2	3	4	5
3. The content was current and relevant.	1	2	3	4	5
4. The objectives could be achieved using the content provided.	1	2	3	4	5
5. This was an effective method to learn this content.	1	2	3	4	5
6. I am more confident in my abilities since completing this material.	1	2	3	4	5

7. The material was (check one) ___new ___review for me
8. Time required to complete the reading assignment: _____minutes

I verify that I have completed this activity: _____

Comments

_____

_____

OBJECTIVES

This continuing nursing educational (CNE) activity is designed for nurses and other health care professionals who care for and educate patients and their families regarding prostate cancer. For those wishing to obtain CNE credit, an evaluation follows. After studying the information presented in this article, the nurse will be able to:

1. Describe the stages of prostate cancer.
2. Discuss the diagnostic work up for prostate cancer.
3. List the treatment options for prostate cancer.
4. Explain the nurse's role in patient and family education related to prostate cancer.

CNE Instructions

1. To receive continuing nursing education credit for individual study after reading the article, complete the answer/evaluation form to the left.
2. Photocopy and send the answer/evaluation form to *MEDSURG Nursing*, CNE Series, East Holly Avenue Box 56, Pitman, NJ 08071–0056.
3. Test returns must be postmarked by August 31, 2010. Upon completion of the answer/evaluation form, a certificate for 1.4 contact hour(s) will be awarded and sent to you.
4. CNE forms can also be completed online at www.medsurgnursing.net.

This independent study activity is co-provided by **AMSN** and **Anthony J. Jannetti, Inc. (AJJ)**.

AJJ is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation (ANCC-COA).

Anthony J. Jannetti, Inc. is a provider approved by the California Board of Registered Nursing, Provider Number, CEP 5387.

This article was reviewed and formatted for contact hour credit by Dottie Roberts, MSN, MACI, RN, CMSRN, OCNS-C, ***MEDSURG Nursing*** Editor; and Sally S. Russell, MN, CMSRN, AMSN Education Director.