WRAMC US TOO, Inc.

A PROSTATE CANCER SUPPORT GROUP
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NEWSLETTER

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♦ 16 YEARS WITH PROSTATE CANCER AND COUNTING ♦

Colonel Charles E. Preble, Jr., USA (Retired)

I was first diagnosed with prostate cancer (PCa) in 1987 at age 64. Sixteen years later, I still have my prostate and my most recent PSA blood test was <0.002 ng/ml. But believe me, it wasn't easy! Let me start at the beginning. I had been seeing a miltary urologist for years for a neuropathic bladder condition and I was long overdue (four years overdue!) for a follow-up appointment. So with some trepidation I went to Walter Reed Army Medical Center in July 1987 for a routine appointment with my crusty urologist who was likely to upbraid me for my delinquency. While performing the digital rectal examination (DRE), he uttered a mild expletive. He had detected a nodule on the left lobe. Nodules that can be felt by DRE strongly suggest the presence of PCa. I immediately agreed to a transrectal needle biopsy of the prostate. The pathology report came back negative, giving me a false sense of security. Five months later I decided to to get another opinion at a local hospital, requiring another biopsy. It was also negative, but a related cystoscopy revealed that my urethral channel was constricted, so I had a transurethral resection of the prostate (TURP). Ninety-seven prostate chips were sent to pathology and the report came back "2 of 97 chips show evidence of infiltrating carcinoma" with a Gleason grade of 5 (2+3). It was March 1988, and I now knew that the nodule on my prostate was really PCa. **(Continued on page 7)**

♦ New US TOO! International Logo and Web Site ♦

US TOO! International recently unveiled a new logo intended to communicate more immediately to the viewer the mission and purposes of the organization, as well as a sense of support and unity. The logo incorporates an arch, the prostate cancer blue ribbon, and the tag line "Prostate Cancer Education and Support." You can see the new logo on page 16 of this newsletter. John Page, president and CEO, also announced the availability of an enhanced, user-friendly web site with new and expanded content. Be sure to visit it at **www.ustoo.org.**

♦ *INSIDE THIS ISSUE* ♦

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♦ FROM THE EDITOR'S DESK ♦

Another Personal Account

We have another personal account in this issue. Charles Preble provides us with his remarkable odyssey in combatting prostate cancer. You get the benefit of his experience with the various treatment options, as well his "if I had it to do over again" opinion. Keep those personal accounts coming!

We are very grateful to Dr. Aaron L. Stack, Department of Nuclear Medicine, WRAMC, for being the guest lecturer at our November meeting on short notice when the scheduled speaker had to postpone his appearance. His topic, *Prostate Cancer Diagnosis and Evaluation*, presented insights into the role of radiation and nuclear medicine in the diagnostic process. A summary of Dr. Stack's presentation begins at page 10.

♦ PROGRAM FOR FEBRUARY 4, 2004 ♦

WRAMC US TOO meets next at 7 PM on **Wednesday, February 4, 2004,** at Joel Auditorium at WRAMC. Our speaker is Dr. Arnold M. Kwart, Chairman, Department of Urology, Washington Hospital Center. Dr. Kwart was our scheduled November speaker, but had an unavoidable schedule conflict. We are pleased that he is able to join us on February 4. A graduate of Duke University, Dr. Kwart served his residency in general surgery at Bellevue Medical Center, New York University, and his residency in urology at Johns Hopkins University Hospital. His topic will be "Watchful Waiting--Who is it for? When is it Appropriate?" Plan now to attend and bring your spouse or a friend. They are always welcome.

♦ **Positive Biopsy Rate From Prostate's Affected Side Predicts Cancer Recurrence.**
Freedland, et al, at UCLA studied radical prostatectomy patients to compare outcomes according to the total percentages of cancer-positive biopsy cores from the dominant (the more involved side of the prostate) and the nondominant side. The researchers combined PSA level, Gleason score, and percentage of cores positive from the dominant side in a model that provides a high degree of prediction for 2-year recurrence risk. The research confirms the belief of many urologists that men with multiple positive biopsy cores on the same side of the prostate have a decreased chance of cure, and that nerve-sparing may not be possible for those men. The researchers conclude that it is not acceptable to look at a prostate biopsy and simply say whether there is cancer or not. Instead, the urologist must know the number of positive cores, and more specifically, the number of positive cores on each side of the prostate. This increases the ability to predict whether the cancer will be cured by surgery. (Source: *Cancer,* December 1, 2003 via Reuters Health Information, October 20, 2003)

♦ **Brachytherapy Selected More Often.**
Lee, et al, Wake Forest University, report that men with early prostate cancer are increasingly selecting brachytherapy as their primary treatment option. In a study of more than 36,000 men between 1994-1999, the percentage of US men with early prostate cancer who were treated with brachytherapy increased from less than 5% to 36%. However, brachytherapy was combined with external beam radiotherapy (EBRT) in about half the cases. Brachytherapy-only patients were significantly younger, had lower pretreatment PSAs, and had more favorable prognostic groupings compared to men who had EBRT as a monotherapy. The researchers said it will be interesting to observe whether this dramatic shift to brachytherapy will result in improved outcomes for patients. (Source: *Cancer*, 2003; 98: 1987-1994 via Reuters Health, November 14, 2003)

♦ **Aren't They Spraying in the Wrong Place?** Palatin Technology says its experimental anti-impotence nasal spray achieved its primary goals during a recent clinical trial. The trial involved 271 men with mild to severe erectile dysfunction who had responded well to Viagra. The company reported statistically significant improvements in erections at all four dosage levels. About 12% of the patients had adverse side effects, mainly gastrointestinal in nature, associated with the higher dosage levels. A company spokesman sees a clear path to eventual FDA approval, but not before 2008. (Source: Reuters Health, November 3, 2003)

♦ **New Prostate Cancer Drug Approved.**
The FDA recently approved a new injectable drug intended for men with advanced prostate cancer who have no other treatment options. The drug Plenaxis, made by Praecis Pharmaceuticals, works by lowering testosterone levels. The company agreed to certain marketing restrictions because the drug raises the risk of potentially life-threatening allergic reactions. Plenaxis, a gonadotropin-releasing hormone agonist, may help men with advanced prostate cancer who cannot tolerate other hormone therapies and who have refused surgical castration. Its distribution will be restricted to doctors and hospital pharmacies enrolled in a special safety program. The drug is injected into the buttocks every two weeks for the first month, then every four weeks thereafter. Common side effects include hot flashes, sleep disturbance, breast enlargement or pain, and constipation. The manufacturer plans to make initial shipments of the drug in early 2004. (Source: Reuters Health Informa-

tion, November 26, 2003)

♦ **Fracture Risk and Orchiectomy.** Androgen deprivation therapy is associated with bone loss in men being treated for prostate cancer. Melton, et al, were interested in determining if there was increased fracture risk for prostate cancer patients following bilateral orchiectomy (surgical removal of the testicles). They followed 429 men who underwent bilateral orchiectomy, primarily for prostate cancer. Actual incidence of fractures within the study population were compared to expected incidence rates. The study showed that androgen deprivation resulting from bilateral orchiectomy places patients at a significantly increased risk for osteoporotic fractures. (Source: *J Urology*, 2003; 169: 1747-1750 via Medscape, October 2, 2003)

♦ **Screening Older Men for PCa.** Yao, et al, Cancer Institute of New Jersey and the Institute for Clinical Evaluation Sciences, Toronto, question the need for prostate cancer screening for men older than 75. Their study of 7,889 men over 75, extrapolated to the population at large, estimates that 1.5 million older men get PSA testing annually at a cost of $38 million to Medicare. The researchers maintain that most patients with an elevated PSA do not have prostate cancer, and those men over 75 who do are likely to die of some other disorder. They also express surprise that fewer older men are screened for colon cancer using the fecal occult test that has a long-established health benefit compared to the PSA test. In a rejoinder, Dr. Richard Middleton, University of Utah Medical School, says the Yao study is simplistic. He cites circumstances when the PSA test is helpful in monitoring elderly men, particularly those with a history of prostate problems or a previous history of elevated PSAs. (Source: *J Natl Cancer Inst,* 21000;95: 1792-1797 via AOL News, December 2, 2003) (**Editor's Note**: See the next item for a related topic.)

♦ **Prostate Cancer Groups Respond.** A recent study funded by the National Cancer Institute discourages PSA screening for men over 75. Advocacy groups in the United States and Canada expressed surprise and disappointment with the study conclusions. Spokesman for the National Prostate Cancer Coalition and the Canadian Prostate Cancer Network suggest that the study is more concerned with the expense to government-funded health care programs, such as Medicare and Medicaid, than about men's health. (Source: Canada Newswire, December 4, 2003)

♦ **Prostate Cancer Deaths Decline in US and UK.** A noteworthy decline in prostate cancer mortality between 1990 and 2000 in the US and UK has been attributed to earlier detection and hormonal therapy. For twenty years prior to 1990, there had been an upward trend in prostate cancer mortality. During the decade under study, deaths from the disease fell by 33% among US men between the ages of 50 and 74. The UK experienced a 20% decline in mortality during the same period. Information provided at the recent European Cancer Conference indicates that developments in hormonal therapies, particularly luteinizing hormone-releasing hormone (LHRH) therapy, have probably had the biggest impact. An analysis of about 5,000 men treated with hormone therapies shows that early resort to treatment has a significant effect on 10-year mortality. There was a 74% survival rate in men given early hormonal treatments compared to 62% in those receiving delayed treatment. (Source: Reuters Health Information, September 23, 2003)

♦ **Obesity, Race, and Prostate Cancer Aggressiveness and Recurrence.** Two recent studies indicate that obesity increases the risk of higher grade prostate cancer and higher recurrence rates after radical prostatectomy (RP). They also indicate that obesity may at least partially explain the racial disparity in prostate cancer outcomes. Amling, et al, Naval Medical Center, San Diego, evaluated data for 3,162 men who had a RP between 1987 and

2002, as documented by the DOD Center for Prostate Disease Research (CPDR). Nineteen of the cohort was obese, defined as a body mass index (BMI) of 30 kg/m or above. Compared to the non-obese members of the cohort, obese patients had a higher median PSA, higher Gleason score, higher incidence of positive margins, and a higher biochemical failure rate. Compared to white men, black men developed prostate cancer at a younger age with higher grade tumors and stage. They were also significantly more obese. In a related study by Freedland, et al, Johns Hopkins School of Medicine, the researchers evaluated 1,752 patients treated with RP between 1988-2002. They also found that black men had a significantly higher mean BMI than white men. Obese patients were younger and had higher biopsy and pathologic Gleason scores. The risk of PSA failure was greater for men whose BMI of 35 kg/m or higher. They conclude that programs targeted to control obesity in the black community may be warranted. In commenting on these studies, Neugut, et al, Columbia University, note that obesity, while not a consistent risk factor for prostate cancer incidence, is consistently associated with prostate cancer mortality. (Source: *J Clin Oncol* 2004; 22 via Reuters Health Information December 22, 2003)

♦ **Lycopene Again.** Some previous studies have shown that men consuming tomato products have a lower risk of prostate cancer, leading scientists to credit lycopene–the compound that makes tomatoes red. Supplements based on tomato extracts may not work to prevent prostate cancer the way the whole fruit does. Recent research on laboratory rats suggests that men seeking health benefit from tomatoes should consume tomato sauce, tomato paste or the whole fruit instead of popping a pill. The studies at the University of Illinois and Ohio State University note that

many men are consuming lycopene supplements in the hope of preventing prostate cancer or enhancing the treatment of the disease. The researchers do not say lycopene is useless, but instead emphasize that if men seek health benefits from tomatoes, they should eat tomatoes and tomato products, rather than relying on supplements. (*Wall Street Journal*, Section D, page 2, November 5, 2003)

♦ **Elderly Men and Radiation Therapy.** A ten-year study at the University of Pittsburgh Medical Center found that elderly men with prostate cancer can tolerate and benefit from external beam radiation therapy (EBRT). Deutsch, et al, studied 33 men, aged 80 or older, most of whom had advanced, aggressive forms of the disease. All were treated by EBRT at the same radiation levels as younger patients in their 50s and 60s. The patients had a five-year survival rate of 61.6 percent and had no unusual or prolonged treatment interruption due to illness from the radiation therapy. The researchers note that the 61 percent survival rate is better than the five-year survival rate for lung cancer patients. Elderly prostate cancer patients who are otherwise severely ill are not good candidates for EBRT. Some doctors believe the effort and cost of radiation therapy is not beneficial to elderly prostate cancer patients, but the researchers believe this study shows that elderly patients can tolerate and benefit from such treatment. The study was presented at the December 3, 2003, annual meeting of the Radiological Society of North America. (Source: Yahoo! News, December 3, 2003)

THIS WRAMC US TOO NEWSLETTER IS AVAILABLE ON THE WEB SITE OF THE DEPARTMENT OF DEFENSE CENTER FOR PROSTATE DISEASE RESEARCH AT WWW.CPDR.ORG. BACK ISSUES ARE ALSO AVAILABLE.

"THE DOCTOR IS IN"

Colonel Judd W. Moul, MD

(Editor's Note: Readers should not act on the responses without prior consultation with their own physicians.)

QUESTION. I have read that the complexed PSA test is more accurate than the total PSA test. Please explain the difference between the two tests and their clinical implications.

ANSWER. As we all know, "PSA" stands for "prostate-specific antigen." It is a protein that is produced only by the prostate gland and prostate tissue. It enters the bloodstream in varying amounts when diseases of the prostate gland are present. So PSA is a "tumor marker." PSA is "prostate specific", but not "prostate cancer specific." Hence, when a man has an elevated PSA it means something is wrong with the prostate, but it may not necessarily be cancer. PSA is a very useful tumor marker, but it is not perfect. Therefore, other forms of PSA have been discovered and used to try to improve on the accuracy of PSA as a marker of prostate cancer.

We now know that PSA protein exists in various forms in the bloodstream that can be measured separately. There are three forms that I will discuss here: (1) Total PSA; (2) Free PSA; and (3) Complexed PSA. These are abbreviated as "tPSA," "fPSA,", and "cPSA" respectively.

Total PSA (tPSA) is the standard PSA that has been around since the mid-1980s. This was the first one discovered and it remains the "gold standard" marker that is critically important. tPSA measures the entire amount of free plus complexed PSA in the bloodstream. Free PSA (fPSA) is the portion of the PSA protein that is "free" in the bloodstream, i.e., it is not attached to other proteins. It has been available as a lab test for more than 5 years. The greater the amount of fPSA a man has in his bloodstream, the lower the probability of cancer. This is usually measured as "% free PSA" compared to total PSA. A low % free PSA (10% or less) is associated with a 50-60% risk of a man having prostate cancer. On the other hand, a high % free PSA (greater than 25%) is associated with a low (approximately 10%) risk of prostate cancer. However, just like total PSA, % free PSA is not perfect. All men do not fit within these guidelines.

Complexed PSA (cPSA) measures just the amount of the PSA protein that is attached to other proteins. It has been available for about two years as a tool to help screen for prostate cancer. Some experts think it is somewhat better than fPSA, while others think it is about the same as fPSA in its accuracy. The protein test for cPSA may be somewhat more reproducible since cPSA is more stable in blood at room temperature whereas fPSA is less stable and the blood needs to be kept on ice for stability. In this sense, cPSA may be more practical in "real world" settings.

Overall, total PSA and free PSA and/or complexed PSA are useful in prostate cancer screening—none are perfect, but they do a pretty good job of risk assessment for the possibility of prostate cancer. Walter Reed has total and free PSA capability "in house" and cPSA as a special situation test that is sent to an outside lab if a doctor feels it is needed. In monitoring the status of prostate cancer in a man who has already been treated, the total PSA is the only one that is clinically useful. There is no major value to fPSA or cPSA in the follow-up monitoring of the disease.

QUESTION. First there was Viagra, now Levitra, and soon Cialis. Is there a dime's worth of clinical difference among them, other than the football?

ANSWER: This is a great question, and I wish I knew the answer! To my knowledge, there have been no "head-to-head" comparisons among the three oral drugs for erectile dysfunction (ED). All three of the pills are in the class called "PDE inhibitors", or "phosphodiesterase inhibitors". They work in the nitric oxide pathway in the penile tissues to improve blood flow for erection. None will spontaneously cause erection, but they improve the natural response to stimulation. Viagra was the first one available, and in my mind, remains the "gold standard." Urologists have a lot of experience with it. It is safe and effective and has been a wonder drug to countless men and their partners. I do not have any personal experience with Levitra or Cialis and neither is yet available in the military health care system. My understanding and reading suggests that Levitra may be very similar to Viagra in its efficacy. Cialis, which was just recently FDA-approved, has been touted as a "weekend Viagra" due to its longer half-life in the body. However, any side effects would also likely last longer, too. Eventually, the military hospitals will likely carry the most cost-effective one. Overall, it is great to see multiple products now available for the devastating problem of ED.

(Sixteen Years with Prostate Cancer and Counting–Continued from page 1)

In August 1988 my first PSA was 3.7 ng/ml. About that time Dr. Patrick Walsh, Chairman of Urology at Johns Hopkins, visited Walter Reed for consultations. My case was among those reviewed by Dr. Walsh and the Walter Reed urology staff. Their consensus was that "watchful waiting" was a reasonable modality for me at that time. By May 1989 my PSA had risen to 5.3 ng/ml. I continued "watchful waiting" until my PSA reached 7.5 ng/ml in February 1990. At this point, my urologist said in his direct way, "why don't we take that sucker out?" Frankly, I felt that my neuropathic bladder condition would leave me incontinent after surgery. Still, I should have replied "let's do it." Instead I said, "I've read about a new procedure called palladium 103 (Pd103) seed implant being done in Atlanta and I plan to go to Atlanta next month." He said, "you know that is not the recommended course for you."

In hindsight, he was absolutely right. But I thought that if the Pd103 implant failed, my prostate could then be removed. This was a naïve, uninformed expectation that I will explain later.

I went to the Georgia Prostate Center at Marietta, Georgia, on March 1, 1990. Sixty-two Pd103 seeds were implanted in my prostate under local anesthesia in an out-patient procedure. Pd103 seeds generate short-range, high-intensity radiation and have a half-life of 17 days, which means they are inert after about three months. The procedure is known as brachytherapy. The surgeon places a probe in the rectum, and using ultrasound, guides long needles containing the seeds into the prostate. My post-op KUB x-ray showed uniform distribution of the seeds throughout the prostate. A flow cytometry (DNA) analysis of my tumor slides showed that my cancer was a diploid. A diploid is the most "favorable" form of prostate cancer. The cells are slow growing and very similar to normal cells. An aneuploid and tetraploid are more aggressive prostate cancer tumors.

By August 1990 my PSA had dropped to 2.6, then gradually rose until it reached 5.3 in September 1991. I was in trouble. A transrectal ultrasound scan of the prostate (TRUSP) biopsy was done and 2 of 4 fragments were positive with a Gleason grade of 6 (3+3). By December 1991 my PSA was 5.8. At that point I asked my WRAMC urologist to remove my prostate. He referred to the procedure as a "salvage radical prostatectomy" because radiation from the Pd103 seeds usually causes fibrosis and scar tissue to form around the prostate, making subsequent surgery very difficult and risky. If I had known that failed brachytherapy might foreclose a salvage RP, I probably would have foregone the seed implant in favor of a radical prostatectomy. Nevertheless, we agreed to proceed with the salvage RP.

On January 9, 1992, I was taken to the OR at Walter Reed where my surgeon commenced the salvage procedure. He soon discovered my prostate had adhered to the neck of the bladder on one end and to the rectum on the other end. To successfully remove the prostate with the entire tumor, he would have to remove my bladder and resect a portion of the rectum. I would wake up with two bags on my abdomen for evacuation. He very wisely said "this guy is 69; I'm not going to leave him with that quality of life." After performing a bilateral lymphadenectomy (which were negative for PCa), he sewed me up. It was a wise professional decision for which I shall be forever grateful. (A side note: My wife kissed me before they wheeled me into the OR for the three hour operation. Thirty minutes later she heard herself paged on the hospital PA system: "Mrs. Preble, please report to the Urology Clinic." As she ran to the clinic, she thought, "Oh my God, he died in the OR." My surgeon was there to reassure her saying, "I've known your husband for 20 years and I just couldn't leave that fine gentleman with that quality of life.")

I was still not out of the woods. My PSA continued to rise and by December 1992 it was 10.3 and my PAP was 1.44. The PAP or prostate acid phosphatase blood test is another useful "marker" for monitoring the progression of PCa. About this time I read of pioneering work being done at Allegheny General Hospital in Pittsburgh, PA. The research using dogs demonstrated that cryoablation of the prostate was effective in destroying cancer cells. I went to Pittsburgh in March 1993 for an evaluation for this new procedure. My pre-op PSA was 18.2 when the cryosurgical ablation procedure was performed in June 1993. The procedure is similar to brachytherapy, but instead of seeds, the surgeon implants into the prostate five probes the temperatures of which are brought down to a sub-freezing level. The location of the probes is monitored with ultrasound to ensure that the ice balls do not freeze vital tissue. A warming catheter is inserted in the urethra and the entire prostate and seminal vesicles are frozen twice during the one hour procedure. I tolerated it well. Three months later I had a 12-point TRUSP biopsy. Two of the 12 cores came back positive and my PSA had reached 20.7. Now I had two strikes against me – first the failed brachytherapy and now the failed cryoablation.

The last remaining treatment modality for me was hormone therapy (HT), also known as androgen ablation. It is chemical castration. Its purpose is to shut down the entire production of testosterone since PCa cells need testosterone in order to grow. Back at Walter Reed in October 1993, I began HT with a Lupron shot (an LHRH agonist) every 28 days and two flutamide tabs (an antiandrogen) every eight hours. The LHRH blocks 95% of the testosterone which comes from the testicles and the antiandrogen blocks the other 5% coming from the adrenal glands. Finally some good news! My PSA went from 20.7 to <0.1 and my PAP went from 2.2 to 0.7 within 60 days. I

stayed on hormones for 48 months and my PSA remained essentially undetectable.

At this point in my story, I would like to mention my resort to alternative medicine in the form of dietary supplements. I know the jury is still out on supplements while rigorous clinical trials are being conducted. In the meantime, reputable medical sources have reasonable evidence that supplements such as selenium, vitamin E and lycopene have some efficacy in the treatment of PCa. So when my PSA blipped up to 0.3 after 42 months on HT, I started taking 200 mcg of selenium, 400 IU of vitamin E, and 20 mg of lycopene on a daily basis. My PSA almost immediately fell back to <0.1 and stayed there. As the saying goes–"Whatever works!"

After 48 months on hormones (October 1997), my favorite urologist said, "we have a new modality called intermittent hormone therapy (IHT) – you come off everything and your PSA may stay down for a year, and if and when it rises above 2.5, you go back on hormones." I welcomed the opportunity. IHT is working for me! I have been off all hormones for 73 months as of November 2003, and my most recent PSA is <0.002 and my PAP is 0.6.

Let me make another comment about the efficacy of dietary supplements. After 47 months into the IHT off cycle, my PSA blipped up from <0.002 to 0.008, an increase by a factor of 4! At that point I doubled the selenium to 400 mcg, doubled the vitamin E to 800 IU, and increased the lycopene from 20 mg to 30 mg daily. My PSA immediately fell back to <0.002 and it has remained there.

I have been through the prostate cancer mill, so to speak. Over the course of sixteen years, I've had two biopsies; a TURP; watchful waiting; radiotherapy in the form of brachytherapy; cryosurgery; hormonal therapy, and its variant, intermittent hormonal therapy, not to mention more PSA tests than I can count! Given my experience with salvage radical prostatectomy, don't you agree I should get at least half-credit for an RP too?

The only explanation I have for the good fortune of my PCa staying in remission for so many years is (1) many prayers and my faith in God, (2) a relatively low Gleason Score of 5 (3) a diploid tumor, and (4) there may have been a synergistic effect from the extended period on hormone therapy in combination with the brachytherapy and cryoablation.

I am often asked what I would do differently if I could relive my experiences with prostate cancer. I have no doubt about what I would do. Upon diagnosis, all the primary therapies were available to me. I would opt for an RP very early when there is organ-confined disease. The RP is the gold standard for treating prostate cancer when it is organ-confined. There are two good reasons for this: (1) with an RP you get a post-op pathology report, and if it says "all margins are free of tumor" and your post-op PSA is 0.05 ng/ml or less, there is a very high probability (but not absolute) that you are cured; and (2) if the RP fails down the road, you have resort to other treatment modalities such as radiotherapy and hormonal therapy. If radiation is your first choice, you more or less foreclose on the RP option because of the fibrosis problem mentioned above.

PERSONAL EXPERIENCE WITH PROSTATE CANCER

Our first person stories about coping with prostate cancer have been favorably received. Do you have a story to tell that would help others? If so, contact the editor (see page 2) to express your interest in participating.

Prostate Cancer Diagnosis and Evaluation

Major Aaron L. Stack, MC, USA
Department of Nuclear Medicine, WRAMC

(A summary of a presentation to WRAMC US TOO on November 5, 2003.)

(Editor's Note: Dr. Stack illustrated his remarks by a series of slides that are referred to, but not included in this summary.)

Introduction

Let me say at the outset that Walter Reed is a great place to practice medicine. The Army's commitment to research and the education of its medical staff is second to none. So I'm very proud and honored to be a part of this organization.

Modern medicine can be time-consuming and expensive. As military service members, we are somewhat isolated from the treatment costs associated with high technology medical science. This reminds me of a story! A lady goes to her veterinarian carrying her pet duck that she suspects is dead. She presents it to the vet and says, "Doc, please tell me, is my duck dead?" The vet carefully examines the duck and says, "Yes, your duck is dead." She asks if there is anything else he can do to be sure. The vet whistles and out from the back office trots a black Labrador retriever. The vet points to the duck and the dog slowly circles it, sniffing all the while. Then the Labrador looks up at the vet and nods its head to confirm the duck is dead. The vet says to the lady, "Yes, your duck is dead." The lady says, "Well, I thought I saw the duck move while I was driving here. Is there anything else you can do to be sure?" The vet whistles again and out from the back office slinks an orange tabby cat. The vet points to the duck and the cat slowly circles it, sniffing all the while. Then the cat looks up at the vet and nods its head to confirm the duck is dead. The now-exasperated vet says emphatically, "**Lady, your duck is dead**." She replies, "OK, OK, I believe you. What do I owe you?" The vet says, "That will be $600." The shocked lady says, "What?--$600 just to tell me my duck is dead?" To which the vet replies, "Well, if you had trusted me in the first place, I wouldn't have ordered the lab test and the cat scan!"

That is an off-beat introduction to my topic tonight--prostate cancer diagnosis and evaluation using the Prostascint procedure. There is a lot of buzz in the prostate cancer community about the Prostascint evaluation, and that will be the main thrust of my presentation. As you will see, it is a good diagnostic test when it is used the right way—just like any medical test. Some patients want everything done right up front, but sometimes immediately ordering a whole battery of tests isn't the right answer.

Most of you are familiar with the epidemiology of prostate cancer--the number of new cases and projected deaths annually; the disease as the second most common malignancy among men; the special risks for African Americans; one out of every eleven men will develop clinically evident prostate cancer; etc. Unfortunately, if we men lived long enough, all of us would eventually get prostate cancer. It is noteworthy that, on autopsy, evidence of prostate cancer will be found in 35% of men over the age of 45.

The Anatomy

In my discussion of anatomy, we are going to talk in general terms. This is what is called a sagittal section. It's as if I split myself down the center and then you are looking at me from the side. The prostate gland is here, right at the base of the bladder; and the urethra comes right through the middle of the prostate gland; and of course, the tube that connects the testes into the urethra also travels through the prostate. This has important

implications when you start talking about surgical interventions. Now, you will notice the close proximity of the prostate gland to the rectum. That's why a digital rectal exam (DRE) is able to detect palpable abnormalities in the prostate gland. The peripheral zone is usually the one we can feel during the DRE. Fortunately, the majority of cancers occur in the peripheral zone of the prostate—about 70% of them. So, the DRE is a good screening tool.

The Diagnosis

There are three techniques used in the diagnosis of prostate cancer. I have already mentioned the DRE. The second is the test for prostate specific antigen (PSA). All of us in this room are familiar with the PSA test. If the PSA test shows a value above 4 ng/ml, there is an increased chance of prostate cancer, and below that level, we think there is much less risk. Of course, there is a significant number of men with a PSA level higher than 4 ng/ml who are normal. By the same token, there are men with a PSA level lower than 4 ng/ml who have prostate cancer. The PSA is not a perfect screening tool, but when combined with the DRE, it's a good place to start. Biopsy is not a screening tool. It is performed in cases where there is a strong suspicion, based on the DRE and PSA, that cancer is present in the prostate gland.

Imaging

I am an imager. My job is to take and analyze images of one sort or another. I provide my analysis to the referring physician, telling what I think is going on and how best to proceed. The CT scan, the transrectal ultrasound, the MRI, the bone scan, and the Prostascint are my tools. Prostascint and bone scan are two imaging tools that I use a great deal because I specialize in nuclear medicine. I spend about 80% of my time in Nuclear Medicine and about 20% in Radiology. The Prostascint and bone scan help me evaluate prostate cancer and determine whether or not the disease has spread outside of the prostate gland. Of course, detection of prostate cancer may be an incidental finding on a study done for another reason. For example, a man coming to the emergency room with belly pain might get a CT scan of his abdomen and pelvis. We could discover something abnormal in his prostate as an incidental finding to some of these studies.

When we do a bone scan, we look at all the bones in the body. This is the initial bone scan on a patient who had been diagnosed with prostate cancer. At first glance, it looks pretty normal. There are a couple of abnormalities—a little focus down here in the low pelvis in the bone we call the ischium. Here you see some activity that is just urinary contamination. What about the activity seen here; is this just another spot of urinary contamination? It is hard to say. Then there is some activity in the left shoulder, compared to the right shoulder. I'd be concerned that this patient may have some early spread of his prostate cancer. At this point, I typically recommend the patient get some plain film x-rays and see if there is another reason for the apparent abnormality, such as severe arthritis. If the plain film x-rays look normal, this is an indication that the prostate cancer has probably spread to these bones.

Six months later this same patient returned for another bone scan. Here is the image. You clearly can see the abnormalities. Unfortunately, this was one of those cases in which the prostate cancer was very aggressive. When I see a bone scan like this, I don't need other images. I already know the prostate cancer has spread far beyond the prostate, diffusely involving the bones. This outcome points the urologist or oncologist in a completely different direction because now we know that local surgery or focal radiation therapy are unlikely to help in this case. A systemic approach like hormonal therapy is needed to slow the growth of the disease.

If you have a bone scan done, don't be alarmed if the doctor says you need to come back for a follow-up study. Here is why. Take a look at these scans. On the far left is a bone scan of a patient at initial presentation who definitely had cancer that had spread to bone. We see the skull is involved,

as well as the shoulder, the arm, the ribs, the pelvis, the femurs, even the tibia. The patient went on systemic hormone therapy and came back after five months of therapy. Look at the scan on the right. You may be saying, "Well, now the cancer has spread and gotten worse." That's not necessarily the case. Bone scans don't actually image the cancer itself. They image the bone's reaction to cancer. Even if we're carrying a cancer, the bone around it is going to rebuild itself and get stronger. In this case, it can make it look like the cancer is spreading, but actually what you see is healthy bone trying to rebuild and normalize the bone. When this patient returned eighteen months later, his skull had improved significantly and much of the other activity had diminished. So this patient was actually responding to therapy, even though on quick glance, it may look as though his condition had worsened. We refer to this as a "flare phenomenon," where the bone scan looks as though it's flaring, but actually the patient is responding to treatment.

Ultrasound is another way to look at the prostate gland. Here is an example. You can see here an area that looks different from the rest of the surrounding prostate tissue. This is a classic example of what we would see on ultrasound for prostate cancer. It tends to be hypoechoic, i.e., less echo-causing than the rest of the prostate tissue. It is in the peripheral zone of the prostate, just as we would expect. Also, we can look at this area in different planes to help confirm its location, and we can use what is called "Doppler flow" to look at the blood flow in the area. This helps to make sure the hypoechoic area isn't a simple cyst, but that it is, in fact, a solid tumor. As you can see, there is blood flow within this area. If this were a cyst in the prostate gland, there would be no blood flow. So ultrasound can be a good tool to help us evaluate the prostate gland.

(**Editor's Note:** Dr. Stack presented a summary explanation of PSA levels, the Gleason scoring system, and tumor staging systems incidental to his discussion of the Prostascint.) In the worst case of metastasis, there is cancer spread to lymph nodes, bones, or other organs in the body. Prostate cancer can spread not only to the lymph nodes in the pelvis, but farther up into the lymph nodes in the abdomen or even into the chest. It can go to a lung, the liver and many other places. So the challenge to the imager is to detect any abnormalities that might be present

Anatomic imaging such as the CT scan is useful. It helps evaluate any anatomic abnormalities, but it does not definitively tell us whether the cancer is present. Prostascint is a physiologic image and is a good way of looking at whether or not an anatomic abnormality is actually involved with cancer. With a Prostascint, even if something looks anatomically normal on a CT scan, I can tell whether or not there is cancer here because of the way the study is done.

The earlier we detect tumor spread, certainly the better. That explains why there is such a widespread screening program in this country where we follow PSA levels in adult males who are over 45, and we regularly do digital rectal exams during physical examinations. The older we get, the more desirable it is to have a yearly exam so that prostate cancer is detected at its very earliest stage, thereby offering the best chance for a complete cure.

You might hear a man say, "I had a T1, N0, M0," which means that the cancer was detected at its earliest stage-- no lymph nodes involved and no evidence of metastatic disease outside the prostate. That classification corresponds to a Stage A in the other major classification system. Both of these systems are used. Within the oncology field there are movements, not just with prostate cancer, but with other cancers, to get one standard staging system. Unfortunately, some staging systems have been used for so long that it is difficult to get international agreement to use a common staging system.

When you have a prostatectomy and there is suspicion of cancer spread outside the prostate gland, you may undergo what is called a "pelvic lymph node dissection." This is much more

extensive surgery than just removing the prostate. It samples all the lymph nodes in the pelvis up to about the level where the aorta divides to feed our legs with blood. This procedure helps evaluate whether or not there is lymph node involvement. Surgeons do this by looking or feeling for abnormal lymph nodes. If they can't detect any abnormal lymph nodes, they sample the areas where they know the highest incidence of cancer is likely to occur. Obviously, this is not a perfect system. We have thousands of lymph nodes in our body, and only a few of these can be sampled.

Anatomical imaging, such the CT scan and ultrasound, help us determine if there is local disease, but the CT scan may only be 50-55% accurate, and ultrasound only 40-50% accurate in diagnosing the disease. Of course, in advanced disease, their accuracy is higher, but we don't want to wait that late to detect it. We want to try and detect it as early as possible. For that we need a more accurate tool. This leads me to a discussion of Prostascint. The Prostascint may be employed in patients presenting for initial diagnosis with a very high PSA, but it is more typically done in patients who had a radical prostatectomy and who then experience a rising in PSA. The question for the urologist is, "Does this mean that the prostate cancer has come back?"

The technical name for Prostascint is ^{111}In-CAPROMAB PENDETIDE. It is FDA-approved for the detection of soft tissue metastases in prostate cancer patients at high risk for metastatic disease. It is an antibody directed against prostate specific membrane antigen (PSMA) that is expressed by prostate tissue. There is a receptor we can actually image with nuclear medicine studies. The antibodies have binding sites which attach to prostate tissues. We connect the antibodies to a tracer that emits a signal our cameras can detect.

Not every hospital is going to be able to do a Prostascint exam. Hospitals are recognized by the manufacturer of Prostascint as centers of excellence because they have proven themselves to be very accurate readers of prostascint exams. Walter Reed is one of these centers of excellence. So you can be guaranteed that if you come to Walter Reed for this study, we have a highly qualified staff.

This is the equipment we use. The "gamma cameras" detect the gamma rays from the radioactive substance injected into the patient. These gamma rays are localized so we can tell where those gamma rays are coming from. The cameras spin all around the patient, creating three-dimensional images that help us locate the abnormality.

Prostascint requires a fair amount of commitment on your part. It takes four days as a minimum. You don't have to come to the hospital all of those four days, but you come twice, and it requires a certain preparation that I will describe. We actually image two things during a Prostascint. We image the Prostascint that we've injected into you—the antibody that localizes the prostate tissue—and we also take some of your blood and label it with a separate radioactive substance and reinject it to see where your blood vessels and arteries are located. We do this because the lymph nodes live along blood vessels. At this point, I have probably told you more than you wanted to know! But you should know that our equipment at Walter Reed is second to none. We are very fortunate. We have state of the art equipment throughout our entire clinic to give us the best diagnostic capability. Look at this slide. This is what is called the "whole body image" of a Prostascint study. It is a normal exam which shows the imager both anterior views and posterior views. Here you see the liver, the spleen, spine, pelvis and the bone marrow. You also can see the blood, the iliac arteries, and a touch of the aorta. This is what a normal study would look like if we looked at the whole body.

The injected antibody is made from a purified mouse antibody line derived from healthy, disease-free laboratory mice bred to produce it. A small percentage of people will develop antibodies against the mouse antibodies causing what is called a "human anti-mouse antibody

response (HAMA).” When this occurs, it causes the liver to “grab” the mouse antibodies from the bloodstream, and all we end up seeing on the image is the liver. The Prostascint will not be useful to that person because the HAMA response will always be there.

Is there any risk to a Prostascint study? Very little. The HAMA response I just described can affect subsequent PSA testing for that patient, but this can be overcome by techniques available to laboratories. The laboratory must be aware that a patient has had a HAMA response in order to select the proper PSA evaluation tool. Adverse reactions are very mild and minor. About 4% of patients injected have mild abnormalities. Blood pressure fluctuation was one of the most common, but it was not severe and required no treatment. There was a transient change in the liver function in only a very small percentage of patients. This is expected because the liver processes the prostascint. This transient change in liver function is not severe, and it does not pose any threat of liver problems later. Also, some patients develop an almost imperceptible reaction at the injection site. We've never had a problem with that at Walter Reed.

Prostascint can be given more than once, but you need to be aware that the Human Anti-Mouse Antibody (HAMA) response may have a slight increase in incidence in patients who have more than one Prostascint. Those with adverse reactions had about the same amount on repeat injection as people who had only one injection. Here at Walter Reed, we've actually had an extremely low incidence of that.

Image interpretation is no easy task as you can see by looking at the slides I have presented. Considerable experience is required. Here's what we look at. This half of the screen is the Prostascint; this half is the bloodpool. I look at all these images, and I compare them, trying to decide, "Is there anything abnormal here?" The prostate bed itself is difficult to evaluate. Can I tell you whether or not that is definitely cancer in the prostate bed? I can't, because prostate tissue can remain in the prostate bed even after surgery, and it is going to pick up the radio tracer whether it is cancerous or normal tissue. But if I see this radio tracer in lymph nodes outside the prostate bed—yes—I can tell you then that the cancer has gone to that lymph node, because lymph nodes do not have prostate tissue.

As already noted, anatomic abnormalities don't necessarily mean cancer. The CT is a good screening tool, but just because we see an abnormality, such as a lymph node here adjacent to the spine or a lymph node over here next to the aorta, doesn't necessarily mean cancer has gone to that lymph node. But prostascint is a great way of telling us that the lymph node is or isn't involved with prostate cancer. This is an image of a patient who had fairly significant spread of disease that you could see on what we call "the whole body images." There was also some activity in the bowel. That can be a problem. When the liver picks up a tracer or a substance of any sort, it pushes it into the bile; the bile then goes into your bowel. I would say that 15 to 20% of patients who come through Walter Reed need to come back on a second imaging day so that we can see whether certain activity is actually in the bowel or in lymph nodes.

Let me quickly cover how a Prostascint study is done, and then I'll take questions. At Walter Reed, you would come in on a Thursday and we would inject the antibody, the Prostascint. We'll watch you for about fifteen minutes after the injection to make sure everything is fine. Then you would go home. On Sunday you would eat a light breakfast and light lunch, and then you would not eat any solid food the rest of the day. On Sunday evening you would drink a laxative, probably a bottle of Phosphomax. We usually recommend taking it at six o'clock. It should have done the job by midnight. When you return on Monday, the first thing we do is to label your red blood cells, as I said earlier, so that we can see
where the red blood cells live. Then we put you on the camera and image your whole body under the window—the Indium window—to look at where the Prostascint is. We actually have the

camera look for two different substances, your red blood cell substance and the substance attached to the Prostascint so that these images are perfectly matched. This permits us to look at them together so we can compare them confidently. If there is sufficient diagnostic information, you're done. If there is any uncertainty, say, bowel activity, you may return on Tuesday for a second set of images of the Prostascint only. You wouldn't need to have your blood relabeled. If you do return Tuesday, guess what you're going to do Monday night again! You're going to take another bottle of that laxative to cleanse the bowel. It's not pleasant, but it gives us the best chance to provide you with reasonably accurate information from the examination.

In summary, how good is Prostascint? Let's discuss it in terms of sensitivity, specificity, positive predictive value, and negative predictive value. Sensitivity means it can detect an abnormality when the abnormality is actually present. The sensitivity of the Prostascint is in the range of 60-80%. Specificity means it can tell that you don't have an abnormality when you actually don't have one. In this case the range is in the 70-80's%. Positive predictive value and negative predictive value tell you how accurate the Prostascint study is overall in a large population. The positive predictive value is between 60 and 80%, and the negative predictive value is between 70 and 80%. Obviously, the test is not perfect. But recall that the CT scan and MRI are in the 50% range as diagnostic tools. So Prostascint is certainly the best test we have available. At Walter Reed we do these tests on an advanced camera that most hospitals don't have. This allows us to do what is called "fusion imaging." We have a CT scanner connected to our gamma camera. We not only get the images that you saw during the presentation, but we get a CT scan matched to our Prostascint images, giving us even more information. I think more hospitals will be using these fusion imaging cameras. Another development is the Positron Emission Tomography (PET) scan which uses a sugar analogue as the radio tracer. At this point, we don't think PET scans are that helpful in prostate cancer diagnosis. Studies have been pro and con. There are certain tracers being investigated with PET scanners that may in the future be helpful to evaluate prostate cancer, but right now, Prostascint is the best imaging tool we have. Walter Reed does have a PET scanner, in fact, it's a PET-CT scanner. As medical science progresses, we will continue be in the forefront utilizing the best available technology to evaluate the spread of prostate cancer.

This has been a whirlwind tour of how I look at prostate cancer from an imager's perspective. I trust it has been educational and helpful. Are there any questions?

Q: Do you do fusion imaging on all the Prostascints performed at Walter Reed?

A: Yes, we do fusion imaging on all our Prostascints. It is part of our standard protocol.

Q: How many patients per day can you accommodate for Prostascint?

A: It is a time-consuming test. Starting with the injection on a Thursday, then the imaging session on Monday and possibly Tuesday, we must dedicate a camera for basically three hours each morning on both days. We can't schedule anything else in those blocks of time, so we only do one or two of these a week. Of course, it's not a test that is indicated for everyone.

Q: When I was diagnosed, my urologist said a bone scan and Prostascint were not necessary because of my relatively low PSA and Gleason score. I selected surgery, which was successful, but I would like to have had as much information as possible in making my therapy decision.

A: We rely on referrals from the attending urologist. Given your low PSA and Gleason, your urologist apparently felt that additional testing for cancer spread was not necessary. Also, we know that the CT scan, MRI, and Prostascint are not

perfect tools, and undoubtedly, some physicians have not fully embraced the technology. Don't overlook the economic aspect. For example, a bone scan costs about $400 and a Prostascint about $1,800. So typically upon referral, we would do a bone scan first. It doesn't make sense to do an $1,800 test if the $400 test will provide the answer. As far as a Prostascint is concerned, it is generally reserved for a patient whose PSA level continues to rise despite prior definitive treatment.

Q: How often can the Prostascint be repeated?

A: It can be done in an interval ranging from six months to a year. Let's take an example. If a Prostascint is done and disease is detected, the patient likely would be put on hormonal therapy for six months to a year. Then we would repeat the Prostascint to see if any change had occurred. Much depends on clinical indicators such as the PSA trend. It can be can repeated several times. There is no real limit. Obviously, the HAMA response would be the only thing that would stop us from doing another serial follow-up.

Q: When a man is diagnosed with prostate cancer, why not do a Prostascint at the outset?

A: This is related to a previous question. The Prostascint is not too effective when employed in this manner. It looks for prostate tissue, so the prostate will appear as "hot." It may be utilized prior to surgery if there is a high Gleason score. Also, when lymph node involvement is suspected, we may do one before surgery to help localize lymph nodes that may be affected. Again, because of the way medicine is practiced, we can't screen everyone with a CT scan, MRI or Prostascint. We just can't do it. The best screening tests for prostate cancer are the digital rectal exam (DRE) and the PSA. If a biopsy is indicated, an ultrasound is performed. The combination of DRE, PSA test, and ultrasound-assisted biopsy will detect the vast majority of disease incidence.

I enjoyed being with you tonight. I must get right back to the office. It is my turn to walk our Labrador retriever and feed our orange tabby cat!

NEW US TOO! INTERNATIONAL LOGO

(NOTE: LOGO ADDED SEPARATELY)

THIS NEWSLETTER IS MADE POSSIBLE BY AN EDUCATIONAL GRANT FROM ASTRAZENECA, MAKER OF CASODEX AND ZOLADEX.

Screening for Prostate Cancer - An Opinion

Stanley Klein

(**Editor's Note**: Stanley Klein is a prominent prostate cancer activist in the Boston area and a frequent contributor to this newsletter. Mr. Klein is commenting on recent research that suggests performing biopsies on men at a lower PSA level (below 4.0 ng/ml) may be associated with better clinical outcomes.)

There has been considerable discussion in the medical literature and the popular press about the desirability of lowering the PSA standard from 4.1 ng/ml to 2.6 mg/ml. I want to give you my personal perspective as a prostate cancer survivor and counselor based on my own experience in a large prostate cancer support group. My concern is that the medical profession may start performing biopsies on 35-45 year old men who have a PSA level of 2.6 ng/ml.

The current PSA test is so sensitive that it has been detecting prostate cancer years before there are any clinical indications. A PSA level over 4.0 ng/ml has been the threshold for considering a biopsy. Lowering this standard will undoubtedly detect prostate cancers at an earlier stage, but at what cost? Based upon discussions within the support group that I facilitate, newly diagnosed younger men would likely opt for Watchful Waiting, hoping that improved treatment options will eventually become available. These men want a normal sex life. These younger men (47-54 years of age) come month after month to our meetings and are typically confused about what therapy to select. Naturally, they are very concerned about post-therapy quality of life, especially the likelihood of impotence and incontinence. Lowering the threshold for PSA-indicated biopsy will add large numbers of men to this state of concern, and for very little benefit. It would be hard to exaggerate the concerns of these men. They are real and pervasive.

The studies do not say that PSA testing should start at an earlier age, but the implication is there. If a 50 year old man has a PSA of 2.6 ng/ml, would it not be worse if he were 40 with the same PSA, and even of more concern if he were 35? Is this younger man to be biopsied, then treated with the potential for impotence or incontinence at age 35 or 40? The biopsy would not be recommended because the man is 50, 45, or 35, but because his PSA is 2.6 ng/ml.

If the studies' recommendations are adopted, younger men will be detected with prostate cancer. Will these younger men then pursue treatment? I hope not. Why would they risk the side effects of impotence and incontinence. After all, the cancer is not likely to kill them, and if their PSA levels continue to rise they then can be treated in accordance with existing practices. I cannot visualize properly informed 35-45 year old men rushing to be treated because their PSA levels are 2.6 ng/ml. There are tens of thousands of men whose PSAs never reach 4.1 ng/ml, and who are never treated for prostate cancer. But if the alarm is sounded at a PSA level of 2.6, the aggravation begins. They and their families face years of worry about something they cannot control.

Another consideration is whether the biopsy of younger men will detect the prostate cancer. Biopsies performed at lower PSA levels will likely have less chance of detecting the smaller cancer. This could result in frequent biopsies with the

attendant apprehension, pain, and medical expense.

In short, I am very concerned that lowering the PSA level at which biopsy is indicated will have undesirable consequences. The social, economic, and medical costs will be high without any redeeming benefit. More definitive research is required before the medical community adopts the reduced PSA standard of 2.6 ng/ml.

Opportunities to Contribute

In past issues we announced opportunities to help in the fight against prostate cancer. Some of you responded to the announcements. Two of them are being repeated here because volunteers are still needed. Read them and ask yourself whether you can help.

Attention African-American Readers

You know the facts. Prostate cancer affects African-Americans more than any other group of men. African-American men have a 60% higher incidence of the disease, and their mortality is twice as high as that of Caucasian men. They are also less likely to seek help with symptoms and problems following their treatment for prostate cancer. The Duke University Medical Center wants to do something about this situation, and it needs your help. It is conducting a study to assist African-American prostate cancer survivors to cope with, and communicate about, the post-therapy issues of incontinence, erectile dysfunction, and pain. Study participants are being sought nationwide from within the diverse African-American community, considering such criteria as medical history, socioeconomic status, and access to health care. The study objective is to help health care providers better assist African-American men and their spouses and partners to deal effectively with the aftereffects of prostate cancer therapy. The entire study is telephone-based, so men located anywhere throughout the United States are able to participate. OK, no excuses!

CALL (919) 681-3090 TO EXPRESS YOUR INTEREST IN PARTICIPATING

Newly Diagnosed? Help Others While You Help Yourself!

Researchers at the Georgetown University Hospital and the Lombardi Cancer Center invite men who have recently been diagnosed with early-stage prostate cancer and not yet made a treatment decision to participate in a study to evaluate a computer-based health education program. The educational tool is a computer disk that contains up-to-date information on prostate anatomy, diagnostic tests, and treatment options. It is designed to help men make informed decisions about treatment for prostate cancer. The study involves participation in four telephone interviews and using the disk to obtain treatment information about prostate cancer. All contacts are by telephone or mail, and participants need not receive their treatment at Georgetown University Hospital. A modest stipend is paid to participants. If you are interested or have any questions, **contact Tara Lamond**, the project coordinator, at **(202) 687-0435**, **or Kathryn Taylor**, the principal investigator, at **(202) 687-0649**.

CPDR STUDIES

Don't forget! Our own Center for Prostate Disease Research (CPDR) at WRAMC has multiple prostate studies that need volunteers. **Contact Stephanie Shaar at (202) 782-4000** for more information.

WRAMC US TOO COUNSELORS (AS FEBRUARY 1, 2004)

(These persons are willing to share their experiences with you. Feel free to call them.)

SURGERY			
Jack Barnes	Oakton, VA	(703) 620-2818	
Jack Beaver	Falls Church, VA	(703) 533-0274	
Jerry Bussing	Laurel, MD	(301) 490-8512	
Gil Cohen	Baltimore, MD	(410) 367-9141	
Edward G. Courey	Silver Spring, MD	(301) 589-4092	
Tony French	Annandale, VA	(703) 750-9447	
Robert Gerard	Carlisle, PA	(717) 243-3331	
Harry B. Harris	Silver Spring, MD	(301) 384-5260	
Monroe Hatch	Clifton, VA	(703) 323-1038	
Bill Johnston	Berryville, VA	(540) 955-4169	
Dennis Kern	Reston, VA	(703) 391-9418	
James Padgett	Silver Spring, MD	(301) 622-0869	
George Savitske	Alexandria, VA	(703) 671-5469	
Don Williford	Laurel, MD	(301) 317-6212	
RADIATION			
John Barnes	Springfield, VA	(703) 354-0134	(Intensity-Modulated Radiation Therapy)
Leroy Beimel	Glen Burnie, MD	(410) 761-4476	(External Beam Radiation)
Philip Brach	Washington, DC	(202) 966-8924	(External Beam Radiation)
Ron Gabriel	Bethesda, MD	(301) 654-7155	(Brachytherapy)
Irv Hylton	Woodstock, VA	(540) 459-5561	(Brachytherapy)
Harvey Kramer	Silver Spring, MD	(301) 585-8080	(Brachytherapy)
Bill Melton	Rockville, MD	(301) 460-4677	(External Beam Radiation)
Oliver E. Vroom	Crofton, MD	(410) 721-2728	(Proton Radiation)
John Waller	Yorktown, VA	(757) 865-8732	(Brachytherapy)
Barry Walrath	McLean, VA	(703) 676-6405	(Brachytherapy)
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Ray Walsh	Annandale, VA	(703) 425-1474	
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"Mac" Showers	Arlington, VA	(703) 524-4857	
Tony Bicknell	Springfield, VA	(703) 451-7517	
SPOUSE SUPPORT			
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Catherine Williams	Brandywine, MD	(301) 372-8650	
Frances Porter	Bowie, MD	(301) 464-8721	
MULTIPLE THERAPIES			
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Arthur E. Clough	Kerryville, TX	(210) 896-8826	(Surgery and Radiation)
S.L. Guille	Sumerduck, VA	(540) 439-8066	(Surgery, Radiation, Hormonal)
Joseph C. Kiefe	Reston, VA	(703) 860-3697	(Surgery, Radiation, Hormonal)
Hank Lohmann	Kensington, MD	(301) 933-3678	(Surgery and Radiation)
Charles Preble	Annandale, VA	(703) 560-8852	(Cryosurgery, Hormonal, Intermittent Hormonal)
Emerson Price	Absecon, NJ	(609) 652-7315	(Hormonal, Radiation, Cryosurgery)

S.L. Ross	Alexandria, VA	(703) 360-3310	(Brachytherapy, Radiation, Hormonal)
Ken Simmons	Alexandria, VA	(703) 823-9378	(Radiation and Hormonal)
Bill Stierman	Vienna, VA	(703) 573-0705	(Surgery and Hormonal)
David C. Williams	Brandywine, MD	(301) 372-8650	(Surgery, Radiation, and Hormonal)

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♦ MEETING ANNOUNCEMENT ♦

WEDNESDAY, FEBRUARY 4, 2004
7 PM

JOEL AUDITORIUM (SECOND FLOOR)
WALTER REED ARMY MEDICAL CENTER

♦ SPEAKER ♦

DR. ARNOLD M. KWART
CHAIRMAN, DEPARTMENT OF UROLOGY
WASHINGTON HOSPITAL CENTER

♦ TOPIC ♦

"WATCHFUL WAITING--WHO IS IT FOR? WHEN IS IT APPROPRIATE?"