

**WRAMC US TOO, Inc.**  
A PROSTATE CANCER SUPPORT GROUP  
SPONSORED BY  
WALTER REED ARMY MEDICAL CENTER  
**NEWSLETTER**

VOLUME 12

NUMBER 4

NOVEMBER 2003

◆ **Making a Treatment Decision --A Personal Experience** ◆

**Regina M. Faure**

The time for my husband's "annual physical" had arrived at last, fifteen years behind schedule! Why so late? The usual reasons -- he had had no serious illness, claimed to be feeling fine, and didn't particularly care for strangers poking and prodding him or sticking needles into his body. Spousal prodding was another story, so he cheerfully, if reluctantly, went off to his scheduled appointment.

When I arrived at home that night Bob smugly informed me that he passed his physical with flying colors -- chest x-ray good, EKG great, some minor arthritis in two fingers and the left knee, and the digital rectal exam (DRE) showed a slightly enlarged prostate. A slightly enlarged prostate is not necessarily an abnormal condition for men his age. More importantly, the DRE found no detectable hard spots or bumps. He claimed the experience wasn't so bad after all -- he might even have another "annual physical" in five years or so! (CONTINUED ON PAGE 7)

◆ **PACE Race in Washington, DC Area - A Success!** ◆

Over 400 enthusiastic prostate cancer survivors, their family members, friends, and supporters ran or walked among the historic monuments of Washington, DC, on September 7, 2003. The first-ever such local event, the Prostate Awareness & Cancer Education (PACE) Race (5K) was sponsored by the Prostate Cancer Education Council and the Georgetown University Hospital. The participants' spirits were buoyed by glorious weather and the enthusiastic participation of Sister Maria, a diminutive, 79 year-old Catholic nun who lent encouragement to participants throughout the route. She even won a medal in her event category! Dr. John Lynch, Chief of Urology at the Georgetown University Hospital and the local organizer of the event, said that planning for a similar event in 2004 was already underway.

◆ **INSIDE THIS ISSUE** ◆

*A Personal Experience* . . . . . Page 1  
*November Meeting* . . . . . Page 2  
*Prostate-Specific Issues* . . . . . Page 3

*"The Doctor Is In* . . . . . Page 6  
*The Face of Prostate Cancer* . . . . . Page 11  
*Counselors Listing* . . . . . Page 19

**WRAMC US TOO  
NEWSLETTER EDITOR**

**Write or Call  
Vincent P. McDonald  
8661 Chase Glen Circle  
Fairfax Station, VA 22039  
Telephone: (703) 643-2658  
FAX: (703) 643-2658  
E-Mail: vpmjam@aol.com**

**MEDICAL ADVISORY STAFF**

**Colonel David G. McLeod, MC,  
USA  
Colonel Judd W. Moul, MC, USA  
Thomas A. Esther, PA-C  
Barbara Haralson, RN  
Jane Hudak, RN, DNSc  
Editha Orozco, RN  
Kimberly Peay, RN, NP  
Grace Rondeau, RN**

**BOARD OF DIRECTORS**

**Colonel David G. McLeod  
(Chairman)  
Colonel Judd W. Moul  
(President)  
Vin McDonald  
( Vice President)  
Edward T. Watling  
(Secretary)  
Ken Simmons  
(Treasurer)  
Jack Barnes  
Philip Brach  
Jack Hessman  
Jim Padgett  
George Savitske  
Don Williford  
Chester L. Wilson**

**◆ FROM THE EDITOR'S DESK ◆**

**Personal Accounts**

**W**e have appealed on several occasions for readers to submit their personal experiences in dealing with prostate cancer--without much success. We are fortunate in this issue to have the story of Regina and Bob Faure who give our readers the benefit of their experience in Bob's confronting prostate cancer. I hope it motivates other readers to do the same in future editions of the newsletter. How about you, have you got a story to tell?

**O**ur own Dr. Judd W. Moul, Director of the DoD Center for Prostate Disease Research (CPDR), was our August speaker. His topic, *The Changing Face of Prostate Cancer - 2003*, presented the latest research from the CPDR database, as well as the "hot topics" from the recent annual meeting of the American Urological Association. A summary of Dr. Moul's presentation begins at page 11.

**◆ PROGRAM FOR NOVEMBER 5, 2003 ◆**

**W**RAMC US TOO meets next at 7 PM on **Wednesday, November 5, 2003**, at Joel Auditorium at WRAMC. Our speaker is Dr. Arnold M. Kwart, Chairman, Department of Urology, Washington Hospital Center. 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.

**DISCLAIMER: The materials contained in this newsletter are solely the individual opinions of the authors. They do not represent the views of any Department of Defense agencies. This newsletter is for informational purposes only, and should not be construed as providing health care recommendations for the individual reader. Consult with your physician before adopting any information contained herein for your personal health plan.**

◆ **Taking Vitamin Supplements? Read This.**

The United States Preventive Services Task Force says there is little evidence that any type of vitamin supplement reduces the risk of cancer, and some may actually increase the risk. After reviewing studies of such antioxidants as vitamins A, C, and E, as well as multivitamins, the task force concluded there was insufficient evidence to recommend for or against supplement use to prevent cancer. The panel did recommend against the use of beta carotene supplements, saying they might actually increase the risk of lung cancer among heavy smokers. The studies needed to determine if specific vitamins are effective are expensive, difficult to perform, and take years to complete. The bottom line is that vitamin supplements are no substitute for a healthy diet. The researchers noted that vitamin supplements might have value for people with unmet nutritional needs, and with the exception of beta carotene, there is little danger in taking vitamin supplements in doses considered to be safe. But the fact remains that there is no solid evidence that any vitamin or other supplement can reduce the risk of developing cancer. (Source: *Annals of Internal Medicine*, Vol. 139, No. 1: 51-55 via Yahoo Health, July 30, 2003)

◆ **Outcomes for Familial Prostate Cancer.**

The characteristics of prostate cancer are not affected by whether the cancer is inherited or otherwise occurs. Researchers in the Royal Hallamshire Hospital in Sheffield, UK, compared the postoperative outcomes of 35 patients who had familial prostate cancer with 85 patients whose cancer had otherwise occurred. They were unable to identify any clinical or biologic features that distinguished familial cancer patients from other patients. The comparative relapse rates and disease-free survival rates were unremarkable. The researchers concluded that familial prostate cancer does not

appear to be more aggressive than prostate cancer in other men. Prostate cancer appears to develop along the same path regardless of any familial connection. (Source: *Urology*, 2003; 61:1193-1197 via Reuters Health, July 18, 2003) **(Medical Editor's Note:** Multiple studies have had similar results, including a prior publication from the CPDR.)

◆ **PSA Testing Disparities for African American Men.**

African American men diagnosed with prostate cancer are about half as likely to receive annual PSA testing as their white counterparts. Eltzioni, et al, at the Fred Hutchison Cancer Center, Seattle, followed 658 men diagnosed with biopsy-proven prostate cancer for a median of 6.6 years. They found that African American men were half as likely as white men to have annual testing. The researchers cautiously suggest that this may be one additional factor contributing to the unexplained gap in prostate cancer mortality rates which are nearly twice as high among African American men compared to Caucasian men. (Source: *Cancer*, 2003; 98: 496-503 via OncoLink, August 5, 2003)

◆ **Detecting Prostate Cancer Tumors.**

A new scanning technology may reduce the need for prostate cancer patients to have surgery. Tiny iron oxide particles are introduced into the patient's bloodstream, then viewed on MRI equipment, enabling doctors to determine more effectively whether the cancer has spread to the lymph nodes. Healthy lymph nodes absorb the particles, but diseased ones do not, thereby producing more definitive images. The new technology could reduce the need for surgical removal of the lymph nodes for biopsy and allow more informed decisions regarding a primary therapy. The new agent called Combidex was developed by Advanced Magnetics and studied at Massachusetts General Hospital and the

University Medical Center, Nijmegen, The Netherlands. The study involved 80 patients using the new scanning technique and the traditional MRI technique. The new method was 100% successful in identifying cancer spread in the lymph node region, while the old technique was successful only 67% of the time. Combidex may also be useful to detect the spread of other cancers, such as breast, kidney, and testicular. The company is working with the Food and Drug Administration to obtain approval. (Source: The Wall Street Journal, June 19, 2003))

◆ **New Brachytherapy Report.** Researchers at St. Vincent's Hospital, Sydney, Australia, say that men treated with high-dose brachytherapy had no major bowel damage and were no more likely to develop impotence than men who had conventional radiation treatment. The procedure involves the precise placement of twenty catheters to deliver intense radiation to the cancerous tissue. The study involved men whose prostate cancer was classified as either intermediate or high-risk. After five years, 50% of the men reported impotence, a rate similar to men treated with conventional radiotherapy. The researchers noted that, in experienced hands, the procedure did not cause incontinence, while surgery causes incontinence in 1.5% of cases. They also said that 98% of patients could expect to be alive ten years after treatment. (Source: news.com.au, June 23, 2003)

◆ **So, Are You Getting with the Kegel Program?** A team at the Kaiser Permanente Center in Los Angeles studied 38 men whose cancerous prostate glands were removed by radical prostatectomy, which can cause at least temporary incontinence in many patients. Half the men got instructions on how to do the Kegel exercises and were advised to do them twice a day. The other half got no instructions. Overall, 66% of the patients were continent after sixteen weeks, but the Kegel group regained control earlier. After a year, 82% of patients had regained control whether they did Kegels or not. The moral is: If you recently had a radical prostatectomy and are still temporarily

incontinent, you should consider doing Kegel exercises regularly. Some men who have persistent incontinence after surgery also state that Kegels help to moderate their condition. (Source: Yahoo! News, June 27, 2003)

(**Medical Editor's Note:** The CPDR at Ward 56, WRAMC, offers a formal Kegel exercise program conducted by Thomas Esther, PAC.)

◆ **Kegels - A Second Opinion!** Dr. Charles E. Myers, the well-known prostate cancer specialist, has a contrary opinion about the efficacy of Kegel exercises as a method to overcome temporary incontinence associated with radical prostatectomy (RP). He notes that Kegel exercises have been used successfully since the late 1940's to help women cope with stress incontinence. He discounts study claims that Kegels help post-RP men, citing flaws in research design. Even better designed studies have failed to show a significant advantage of Kegels versus no Kegels. He notes that the recent Kaiser Permanente study (see previous item) involved only 38 patients and showed only a minimal benefit. Since most men regain continence within a year, he says that Kegel exercises may be a waste of time. Dr. Myers also advises against spending money on equipment designed to train pelvic muscles. He recommends simply waiting out the normal recovery period, seeking medical or surgical interventions if the post-RP incontinence problem becomes unbearable. (Source: *Prostate Forum*, Vol. 7, No. 7: July 2003, page 5)

◆ **Smoking and Aggressive Prostate Cancer.** Investigators at the Fred Hutchison Cancer Research Center, Seattle, found that smoking is an independent risk factor for prostate cancer, particularly the more aggressive type. In a study of 753 men that focused on men less than age 65, current smokers had a odds ratio of 1.4 for prostate cancer, compared to non-smokers. Early smoking cessation seems to reduce the increased risk over time. For those who smoked more than forty packs a year, the risk of prostate cancer was increased by sixty per cent. Their risk was even higher for distant stage disease and for tumors

with a Gleason score of 8 to 10. Smoking was associated with higher levels of testosterone and sex-hormone-binding globulin. Cigarettes contain significant levels of cadmium and there is evidence that cadmium may interact with androgen receptors to cause higher androgen activity. (Source: *Cancer Epidemiological Biomarkers Prev* 2003; 12:12:604-609 via Reuters Health Information, July 14, 2003)

◆ **Increased Reliance on Androgen Deprivation Therapy Questioned.** Researchers at the University of California, San Francisco, have expressed concern about the dramatic increase in the number of patients with localized prostate cancer who are treated with androgen deprivation therapy (ADT). Granting the role of ADT in the treatment of advanced prostate cancer, they say there is no good evidence to support its use in localized disease, except where ADT is given prior to radiation for high-risk tumors. An analysis of 3,439 patients treated between 1989 and 2001 showed continuous increases in the use of ADT for localized prostate cancer, both alone and in combination with surgery and radiation therapy. Increases in ADT use rates were across the various risk levels, and the largest proportional increases occurred among patients with the lowest risk disease. The researchers are concerned that the increasing use of ADT in localized prostate cancer represents potential overtreatment in many cases, noting that ADT has significant impact on quality of life and that it is expensive. They think that widespread use of ADT for men with low-risk, localized tumors should be delayed until its efficacy has been proven in clinical trials. (Source: *J Natl Cancer Inst* 2003; 95: 930-931, 981-989 via Reuters Health Information, July 9, 2003)

◆ **Agent Orange Requests Increasing.** The Agent Orange Registry reports a substantial increase in participants in the past two years. Previously, about 5,000-8,000 veterans per year had been requesting examinations. The number

of examination participants in 2001 and 2002 jumped to more than 23,000 each year. In contrast, fewer than 20,000 were added during the three previous years combined. A VA spokesperson said that more than 314,000 veterans have participated in the program since it began in 1978. She said the recent large increase is probably attributable to: the recognition of type 2 diabetes as Agent Orange-related; the initial decision to open the Agent Orange Registry first to Vietnam veterans who had served in Korea, and then to all other veterans exposed to Agent Orange; the aging of the Vietnam-era veterans; and better publicity. Vietnam veterans with questions or concerns about Agent Orange should contact the VA's Gulf War/Agent Orange Helpline at **800-749-8387**. More information also is available on the VA's web site at **www.va.gov/agentorange**. (Source: Veterans Administration: *Agent Orange Review*, Vol. 19, No. 2 July, 2003, pages 5, 13)

◆ **Research Information Source.** Now you can access a database and find hundreds of cancer research projects, find out who is doing them, how much money is involved, and read an abstract of the research--all this on a user-friendly web site. The database is the International Cancer Research Portfolio (ICRP) created by a consortium of international cancer research funding organizations. It has obvious benefits for cancer patients and their families and scientists. This project is led in the United States by the National Cancer Institute and the Department of Defense Office of Congressionally Mandated Medical Research Programs. The ICRP currently holds about 13,000 records. The ICRP web site is at **www.cancerportfolio.org**. Go to it and look for research projects that interest you. (Source: News Release: Department of Defense Office of Congressionally Directed Medical Research; July 9, 2003)

## “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 am 84 years old. After surgery, my PSA remained down for years, but began to rise a couple of years ago. It is now 76 ng/ml. I have no symptoms, and my physician says not to be overly concerned about my PSA level, saying it is just a number.

**ANSWER.** It is true your PSA is just a number. The concern about PSA is this. Right now the FDA will not allow pharmaceutical companies to use PSA as an end point to approve new drugs because it is just a number and it may not correlate to actual patient events like metastasis and death from prostate cancer. On the other hand, a CPDR research paper is going to be published in *The Journal of The National Cancer Institute*. It's been through many reviews by many national experts, and the data is real. If your PSA doubling time is less than three months, you will die of prostate cancer, unless you get hit by a bus in the meantime! So, the conventional wisdom will change when this paper is published. We say that PSA doubling time is important, and in general, if it's less than

three months, it's really bad news. Even if your PSA is doubling in less than ten or twelve months, that's probably almost as bad. The problem is that we can't guarantee, even if you start hormone therapy, that you will live longer. We hope that you will, and we can extrapolate from other studies in advanced prostate cancer that you may. For example, men who had cancer in their lymph nodes definitely lived longer when they got hormones earlier rather than later. While I agree to some extent that PSA is just a number, you also must recognize that PSA doubling time is definitely emerging as an important factor in dealing with PSA recurrence. I would certainly consider some type of hormone treatment if your PSA is doubling at a rate of less than 12 months.

**QUESTION.** I see very little in the literature about cryotherapy. Is it considered a primary therapy? What clinical indicators would lead a newly diagnosed man to consider cryotherapy as a treatment option?

**ANSWER.** Cryotherapy for clinically localized or locally advanced prostate cancer has been around for more than 40 years, but reemerged in its current form about a decade ago. The procedure was reborn as transrectal ultrasound-guided percutaneous cryosurgical ablation of the prostate gland. Metal probes were placed into the prostate gland through the perineum and guided using ultrasound. The perineum is the skin between the scrotum and the anus and the ultrasound probe is placed into the rectum. About 1990, the technology involved

approximately 5 probes placed simultaneously into the prostate gland. The freezing was done using liquid nitrogen pumped into the probes from a machine, almost the size of a kitchen stove. Ice balls formed around the five probes to completely encompass the prostate gland and this was the basis for the operation. It was a bit cumbersome and it was associated with a high rate of impotence. Furthermore, in men who had prior radiation and who were getting cryotherapy as a second local treatment, the procedure was associated with severe incontinence in over half

of the men. We did this at Walter Reed in a dozen or so men, but gave up the operation due to these side effects and the concern that the effectiveness of cancer control was suboptimal.

In the last two years, the procedure of cryotherapy has been reinvented yet again. In this third generation prostate cryotherapy, the probes are much smaller and use gas instead of liquid nitrogen for cooling. Due the smaller size, more than 5 probes can be placed in the prostate, resulting in more precise iceball formation and better control of the freezing. In fact, the probes now are small enough to fit through the same perineal template that doctors use for seed brachytherapy. This allows for a more familiar technique, similar to prostate brachytherapy, that lessens the learning curve for urologists. This advancement came as a result of Medicare reimbursement for the procedure in 2001. Since there would be payment, medical companies were more apt to invest to improve the therapy, and doctors were more likely to learn and embrace the technology knowing they would be compensated.

Most recently, Han, et al, reported in the Journal of Urology (Volume 170; pages 1126-1130;

October 2003) on a multicenter experience with 106 men treated by the new technique and followed for a minimum of one year. The most striking news was that the incontinence rates were much lower than in the previous generation cryotherapy - even for men who had prior radiation, the rate of leakage requiring pads was 11%. However, the biggest problem was that 87% of men who were previously potent were rendered impotent from the procedure. The freezing iceball goes beyond the prostate capsule and freezes the nerves that control erections. Over 80% of the treated men had good initial PSA response - their PSAs dropped to 0.4 ng/ml or less at 3 month follow-up.

Overall, I am keeping an open mind on this therapy. In fact, we are offering this new technique again at Walter Reed with the help of Dr. Mohan Verghese, a urologic surgeon from Washington Hospital Center, who is a consultant. For older men who are considering radiation, this may be a reasonable alternative. However, at the current stage cryotherapy would not appear to be a substitute to nerve-sparing radical prostatectomy due to the high rate of sexual dysfunction.

---

## **(Making A Treatment Decision -- A Personal Experience -- Continued from page 1)**

Whoops! A few days later a blinking light on the telephone indicated that a recorded message waited to be answered. The message was from the medical clinic. Could Robert call the clinic to schedule another blood test? His recent test indicated that he had a slightly elevated PSA reading. When informed of the message, he shrugged and said he would call them in a day or two. More spousal prodding -- what's a PSA? Let's check it out.

A quick Internet search on the old Compaq via AOL quickly got our attention. PSA was prostate specific antigen. The PSA is produced

by normal and cancerous prostate cells. The normal range for PSA is 0 to 4.0 nanograms per milliliter (ng/ml). If the PSA is 4.0 ng/ml or higher, a biopsy is normally recommended by the physician. My husband was in the mid-range with a reading of 6.68 ng/ml. An elevated PSA can be caused by other than cancer. For example, benign prostatic hyperplasia (noncancerous prostate enlargement) and prostatitis (inflammation of the prostate) can also effect PSA levels.

The second blood test resulted in a similar PSA score, so the doctor recommended further testing.

The options were more blood tests to determine percent free-PSA ratios, density and velocity or a prostate biopsy. More blood tests would only provide additional indicators. The only way to know for sure was a biopsy. Bob chose to skip the blood testing and go straight for the biopsy.

His physician used a transrectal ultrasound probe to assist with the biopsy. It generates sound waves to produce a picture giving accurate measurements of the prostate size and shape. While looking at the ultrasound picture, he selected ten locations in which to insert a needle and extract a portion of the prostate for analysis. The ultrasound technology enabled the doctor to identify areas that looked potentially abnormal and to select sites that would provide a broad coverage of the prostate. The biopsy procedure took only 15 - 20 minutes. Bob said the discomfort was minimal. Then he had to wait around until he was able to provide two post-biopsy urine samples to ensure that any biopsy-induced bleeding was under control.

Within the week the results were back -- Bob was positive for prostate cancer at age 58. We immediately made a medical appointment to discuss treatment options. In the meantime, it was back to the Internet. We got literally thousands of hits by using the word "cancer" as a search term. Using "prostate cancer" reduced the number of hits to more manageable proportions, but clearly a more selective process was necessary. As we scanned the Internet, we became more confident by reading reputable articles associated with major hospitals, universities and health organizations. We also turned to others for advice. My sister-in-law formerly headed the Radiology School at Hahnemann Medical College. We sought her help, but we did not abandon the Internet. Our education had begun in earnest.

We began to notice certain consistencies among the articles. Grading of prostate cancer is primarily accomplished using the Gleason score system. The appearance of the cancer is given a numerical value from 1 to 5. A grade 1 means the tumor is not likely to be fast-growing. Grade

5 indicates the tumor is fairly irregular and very different from normal prostate cells -- the higher the number the faster the tumor growth. All biopsy samples that have a positive cancer finding are evaluated individually. The Gleason score assigned is the highest combined number found in any of the positive samples. For example, the finding of a 3-4 would result in a Gleason score of 7 on a range of 1 to 10. Scores on the higher end (7-10) indicate a more serious prognosis.

We also learned that tumor stage is another major indicator of the cancer's progress. Tumor stage is a description of the size or quantity of the cancer and the extent of its spread from its original site. It is always much better to have a low-volume, low-grade tumor rather than a larger, more advanced, high-grade tumor. The smaller the tumor, the less likely it will be able to spread. The TNM grading system is used to stage prostate cancer. "T" describes the cancer itself, with different numbers explaining how large the cancer is. "N" stands for nodes and tells us if the cancer has spread to the lymph nodes. "M" tells if the cancer has spread, or become metastatic.

When Bob's biopsy report came back, the results were not what we wanted to hear. His stage was T1c, a cancer that is detected only because of an elevated PSA. His Gleason score was 7. Four of the ten biopsies were positive with Gleason scores of 3-3, 3-3, 3-3, and 3-4. All were on the right side of his prostate. The results were in. Now we faced the critical task of selecting a therapy to deal with the prostate cancer.

Bob's next question was: why me? There was no ready answer because the cause of prostate cancer is still unknown. Genetic factors seem to increase the risk, but no one in his family had been diagnosed with prostate cancer. There was one clue. The number of Vietnam veterans with prostate cancer exceeds the expected rate in the male population. Scientific research indicated that exposure to Agent Orange, a defoliant used during the Vietnam War, might be a factor. The Veterans Administration (VA) subsequently

ruled that exposure to Agent Orange was the presumptive cause of prostate cancer in those veterans exposed to it while serving in Vietnam. My husband filed his claim with the VA.

Now we faced the challenge of selecting the primary therapy appropriate to Bob's situation. Given his PSA level and tumor stage, he had a range of options to consider. They included surgery, cryosurgery, radiation, hormonal therapy (androgen deprivation) and observation (watchful waiting). We soon learned that treatment recommendations frequently depend on whom you are talking to. Surgeons tend to favor surgery; radiologists favor radiation, and so on. Bob and I decided to research his options and see what the advantages and disadvantages were for each choice.

We quickly eliminated observation as an option. The doctors told us Bob's cancer was treatable. We felt observation was an option for a more elderly man, or for someone with other health complications.

A radical prostatectomy was the next option to consider. It is a major operation requiring a hospital stay of 4-5 days. A bladder catheter would have to be in place for two or three weeks, and some degree of temporary incontinence was likely. It's principal advantage is that it is potentially curative. It also permits detailed pathologic evaluation of the excised prostate to determine whether the disease was indeed contained within the prostatic capsule. Of course, we considered the potential side effects of impotence and incontinence.

We looked carefully at external beam radiation. It would require daily treatment (Monday to Friday) for 6 to 8 weeks. It is potentially curative for cancer outside the prostate capsule and it would avoid the risks associated with major surgery. We noted the possible complications from radiation to surrounding tissues, possible complication to future pelvic surgery, and long term risk for impotence and rectal irritation.

Brachytherapy (seed implants) was next. It involves the precise placement of radioactive pellets within the prostate. It is a "same day" procedure, although several prior visits are required for work-up. It is potentially curative for cancer contained in the prostate capsule. It also should reduce the risk of radiation damage to surrounding tissue. But it cannot affect cancer that has spread beyond the prostate capsule, and the radiation side effects are more intense compared to external beam radiation. Important for us was the fact that there is less long term data available. Also, there is a major long-term risk of impotence and injury to the bladder, urethra and rectum.

We learned that hormonal therapy (androgen deprivation) can be used as a main treatment for some men with prostate cancer or in combination with another primary therapy to improve overall effectiveness. The idea is to eliminate the male hormones, in particular testosterone, from the body. Hormonal therapy entails monthly, quarterly, or yearly hormonal injections and hormonal pills taken daily. Its advantage is that it shrinks the size of the tumor and stops or slows growth by lowering testosterone. Unfortunately, it does not cure the cancer, and there are important quality of life considerations, such as the risk for impotence, loss of sexual desire, hot flashes, and breast tenderness or enlargement. Of course, orchiectomy (surgical removal of the testicles) results in rapid and effective decrease in testosterone level without having to be concerned about regular injections and daily pills, but Bob wasn't ready for that.

We had done our homework. Now it was time to act. Bob never considered hormonal therapy as an option because it was not curative. He also felt that the risk of complications from radiation were greater than those associated with surgery. Surgery had the additional advantage of a complete post-operative biopsy of the prostate. This was important to us because we would know with some certainty whether the cancer had been contained in the prostatic capsule. Radiation would require a constant concern about PSA readings and trends. Bob is a Type A

guy -- faced with a problem, he gathers information, makes decisions, then moves on. He selected surgery.

The treatment decision made, the next questions were where to have the surgery and who would perform it. We turned again to Bob's sister. She said that the "Who" was more important than the "Where." Research indicated that experience counts. Men considering a radical prostatectomy should seek a hospital that performs at least 60 radical prostatectomies a year. More importantly, a surgeon should perform at least 40 such procedures a year to maintain proficiency. As a retired Army officer, Bob was a beneficiary of the military health care system and he was familiar with the reputation of the Walter Reed Army Medical Center (WRAMC). In addition, there were other advantages. Walter Reed was located close to home; it had a prestigious prostate disease research center; and it had skilled surgeons who performed over 200 prostatectomies per year. Now we had the "Where" and the "Who." Bob scheduled an appointment with the Center for Prostate Disease Research (CPDR) at WRAMC.

The people at the CPDR were outstanding. Their forte is patient education and counseling. They thoroughly and patiently explained Bob's options to us. All the research we had done paid off because we were able to participate fully in the dialogue. They also referred us to radiation specialists in order to give us the benefit of a second opinion. We also were impressed by their clarification of Bob's entitlement to use of civilian facilities in locations from Boston to California. We also learned about the several facilities that had the capability of providing the latest proton beam computer-controlled treatments. In the end Bob reaffirmed his first decision that surgery was the best way for him to

go. Bob elected to have his surgery at WRAMC, and he was fortunate to have it performed by one of its most capable and experienced surgeons. He was admitted for surgery in the morning on July 8, 2003. He recuperated rapidly. By the afternoon of his surgery, he was standing up and taking steps, albeit tentatively. The next day he was walking around the hospital ward, and two days after surgery he was walking all over the hospital, even outdoors. They sent him home on the third day after surgery. He says only two things about the experience really bothered him - that "damn catheter" and waiting for the final biopsy results. Well, I warned you he was a Type A guy!

**(Editor's Notes:** (1) Sequel - Bob Faure's postoperative pathology report said his Gleason score was 8, and he had six tumors, one of which was outside the prostate. He considered radiation as a salvage therapy, but decided against it. Instead, he enrolled in a two-year, Phase 3 clinical trial that will assign him to either a hormonal therapy regimen or a combined hormonal-chemotherapy regimen. (2) Regina Faure, the author, is an Army lieutenant colonel serving in the Washington, DC, area.) **(Medical Editor's Note:** The trial just described is supported by the National Cancer Institute and conducted by the Cancer and Leukemia Group B (CALGB) at Walter Reed. It is a critically important study. Men are randomized to two years of hormonal therapy with a LH-RH agonist and an oral antiandrogen, or to the hormonal treatment plus an initial course of chemotherapy. The chemotherapy is a drug called "mitoxantrone" plus a drug called "prednisone" which is a steroid.)

**THIS NEWSLETTER AND BACK ISSUES ARE AVAILABLE ON THE WEB SITE OF THE  
DEPARTMENT OF DEFENSE CENTER FOR PROSTATE DISEASE RESEARCH AT  
WWW.CPDR.ORG.**

# **The Changing Face of Prostate Cancer - 2003: Insights from the DoD CPDR Multi-Center Prostate Disease Research Registry Database (and Other "Hot News!")**

**Judd W. Moul, MD, FACS  
Colonel, MC, USA**

**Director, Department of Defense Center for Prostate Disease Research**

*(A summary of a presentation to WRAMC US TOO, August 6, 2003)*

## **Introduction**

Tonight I want to present some of the latest developments in the battle against prostate cancer. Before I do, let me take a moment to make sure that everyone has an understanding about the Department of Defense Center for Prostate Disease Research (CPDR). Established and funded by Congress since 1991, the CPDR is a program to study prostate disease within the military health care system.

We have three key programs. The first is the Tri-Service Multi-Center Prostate Cancer Research Database. Many of you here tonight are in that database. In a very real sense, you are part of the CPDR team because your data is essential to our research effort. The second program is our molecular genetic scientific program at our laboratory in Rockville, Maryland, where we are investigating molecular biology to detect genes that may be involved in prostate cancer. Third, there is the clinical trial center here at WRAMC whose basic goal is to provide enhanced patient care, education and clinical trials for men with prostate disease.

## **The Changing Face of Prostate Cancer - 2003**

A whopping 221,000 new cases of prostate cancer are expected in 2003, up from 189,000 cases last year. And 28,000 men will die from the disease during 2003. The lifetime probability is still that one of every six men will develop prostate cancer. We are not sure of the reason for the surge in new cases. It may be due to the baby boomers reaching the age where they are more likely to develop prostate cancer. It may also be due to family

doctors doing the PSA test more often and diagnosing the disease more. The bottom line is that more men will be diagnosed and they will need your help. The men in this room who compose our support group are in the unique position to offer advice and support to newly diagnosed men. Your colleagues, friends, neighbors, and family members will no doubt be turning to you for help in coping with prostate cancer.

The DoD CPDR database has about 17,000 men enrolled. We note that 43% had undergone a radical prostatectomy, 25% had primary radiation therapy, 12% had primary hormone treatment, 4% had undergone seed radiation, and about 16% had at least started on watchful waiting. Now I want to show you how the face of prostate cancer has changed with the advent of increased reliance on the PSA test. We note four phenomenon during the period 1990-2002. We are finding that more patients are being diagnosed at a younger age (Age Migration). We also demonstrated that clinical stage T1c (prostate cancer that is non-palpable upon the digital rectal examination) is now the most common stage, whereas the incidence of more advanced stages is going down (Stage Migration). This translates into very good news for men within the military health system. Of the military men who were newly diagnosed back in the early PSA era, about 12% had metastatic prostate cancer. That is now down to less than 4%—too many, but still a notable improvement. Next, the PSA blood test is the marker for prostate cancer. In general, the lower the PSA at diagnosis, the smaller the prostate cancer, and the higher the PSA at diagnosis, the larger the prostate cancer. Now we are seeing fewer men with high PSAs

(either between 10 and 20 or greater than 20, and we are seeing more men with PSAs either less than 4 or between 4 and 10 (PSA Level Migration). In general, anything under 10 is considered to have a very low likelihood of having spread. So that's good news. Finally, the number of biopsy cores with cancer is a surrogate of how much cancer the patient probably has in his prostate. Again, we are seeing men with smaller tumors being diagnosed as we moved through the PSA era. All of this is good news, suggesting that we may get better outcomes in the future. This is the first inkling of better long-term results.

More encouraging news! Let's look at the percentage of men actually dying of prostate cancer. In 1991, 40% of the men enrolled in this military database and diagnosed with prostate cancer were dying of prostate cancer, not something else. By 2000, that was down to about 10%. Granted, we have a longer follow-up for the 1991 group than the 2000 group, but the general trend is such that we are curing more men of their prostate cancer so that they eventually die of something else. We know we are all going to die of something! As a urologist— a guy who deals with prostate cancer— I joke that I “win” if you die of something else, because it means that the cause of death is some other doctor's problem! Seriously, I will say that it is always better to die of something like a sudden heart attack or a sudden stroke than to die of prostate cancer. It is better to prevent the death of people from diseases with a prolonged suffering period and painful bone metastasis.

I hope you all are being good advocates for prostate cancer screening. You may hear men say, "Well,, who cares about prostate cancer? It's that disease of old men. I'm going to die of something!" The argument should be, "Sure, we're all going to die of something. But why do we want to put men through the agony of death from potential painful bone metastasis, when there are certainly better ways that they can die through natural causes in old age?"

### **Recent Developments**

**The PCPT Report.** Let's move on to some specific hot topics. You may have seen a recent headline to this effect: "Baldness drug found to cut risk of prostate cancer." It referred to the Prostate Cancer Prevention Trial (PCPT). Walter Reed participated in this national study which enrolled 18,882 men. At the time of enrollment, the participants had to be at least 55 years of age, and have a PSA level of 3.0 ng/ml or less. They were randomized to a placebo or a drug called finasteride which already had FDA approval for treatment of baldness and enlargement of the prostate. Finasteride changes the way the body responds to the male hormone testosterone, which is felt to be the fertilizer for prostate disease. All the men agreed to have a prostate biopsy after seven years regardless of their PSA levels at that time.

These volunteers certainly made an immense contribution to science by participating in the PCPT. During the course of the trial, it became clear that finasteride was effective in reducing the incidence of prostate cancer. The men taking finasteride had an 18.4% rate of prostate cancer, compared with a rate of 24.4 % for men assigned to the placebo. This translated into a 24.8% reduction in prostate cancer prevalence. These positive results were so significant that the study was terminated early, and the results were published in *The New England Journal of Medicine*. But there is a potential downside. The potentially high-grade, aggressive cancers (Gleason sum 7-10) were more common in the finasteride arm than in the placebo arm. So, the message is mixed. Even though finasteride prevents prostate cancer, the men in the finasteride arm who got prostate cancer tended to have a more potentially aggressive disease.

The PCPT report created a firestorm. Here's why. One school of pathologists thinks it was inappropriate to grade these tumors on the grounds that finasteride changes the way the cancer looks under the microscope, but has nothing to do with the way the cancer behaves. Another school accepts the PCPT findings and says that patients should at least be made aware of the potential for

more aggressive cancer. My personal opinion is that finasteride is probably a useful drug in preventing prostate cancer. As noted earlier, it is already FDA-approved for treating men with enlarged prostates. Men with enlarged prostates and suffering from urinary symptoms can kill two birds with one stone, so to speak. That is, they can treat their enlarged prostate and reduce their prostate cancer risk. So it is kind of a no-brainer for men in those circumstances. But other experts say, "Well, be careful. It may not be that great a preventer of prostate cancer, and there is always the risk of high-grade tumors." Tonight I simply wanted to make sure that everyone here is aware of the PCPT report in case other men who look to you for guidance ask you about it.

**Testosterone and Prostate Cancer Stage.** The DoD Center for Prostate Disease Research presented a paper that received a lot of attention at the recent convention of the American Urological Association. Our study showed there was a correlation between pretreatment testosterone levels and pathologic stage. But more than that, lower testosterone correlated with **worse or higher** cancer stage. This is contrary to the conventional wisdom, and it has implications for testosterone replacement therapy for both healthy men and men with a history of prostate cancer. One of the hot topics right now is the concept of andropause which was featured recently on the covers of *Newsweek* and *Time*. You likely are aware of the controversy about hormone replacement therapy for women in menopause. Now there is a school of thought that sees testosterone replacement as a way to slow the aging process in men, e.g., loss of energy level, reduced interest in sex, and weight gain. The problem is that we also know prostate cancer is fueled by more testosterone. A man with a history of prostate cancer who may be completely cured complains about lethargy, and his doctor tells him his testosterone level is low. But the doctor will be reluctant to prescribe testosterone therapy for fear it will affect the man's prostate cancer. Our CPDR study looked at pretreatment testosterone levels pretreatment in 879 men who had undergone radical prostatectomy. Interestingly,

what we found was the men who had lower testosterone levels had worse cancer. These findings generated considerable debate within the national media. Does this mean that a low testosterone level is bad and causes prostate cancer and contributes to its progression? Should we strive to maintain normal testosterone levels in men? Should we strive to maintain normal testosterone levels in men with a history of prostate cancer? We don't have the final answer. You will be hearing more about testosterone replacement in the future. It is not inconceivable that conventional thinking about testosterone for men with a history of prostate cancer may change in the near future.

**Quantitative Histology.** This is a medical term referring to the amount of cancer that is detected in the biopsy. For example, if a man had a prostate biopsy and the results showed four of the twelve individual biopsy needles contained cancer, then the percentage of cancer is 33%. That percentage is felt to be a prognostic factor, but is it of clinical value? The CPDR published a paper which basically says that counting the percentage of the biopsy cores containing cancer is an important factor in predicting whether the disease is still contained in the patient's prostate. Many of you are familiar with the Parton Tables developed at Johns Hopkins. The CPDR tables are similar in many respects. Knowing a patient's PSA, his Gleason score, and the percent of biopsies cores containing cancer, you can consult the CPDR table to find the probability that the cancer will be either organ-confined, outside the capsular prostate, in the seminal vesicles, or in the lymph nodes. It is not an end-all answer, but it can help doctors and the patients decide on which primary therapy is appropriate under the circumstances.

**Watchful Waiting.** The concept of watchful waiting was another hot topic at the AUA. Is there anybody in the room who is on watchful waiting? I see several hands. Men in this situation have been diagnosed with prostate cancer and elected to take a wait-and-see approach. That is

certainly appropriate for some men who may have very early-stage prostate cancer, who are older, who have other health considerations, or who simply are able to live with it because they believe that they are likely to die of something else before the prostate cancer becomes a factor.

The CPDR database provided the data for our presentation at the AUA in Chicago. Entitled "Watchful Waiting and Predicting Factors for Secondary Treatment of Prostate Cancer," it involved 1,158 men who started on watchful waiting. Watchful waiting was defined as "no active treatment for at least nine months after the date of diagnosis." After five years, it was about 50-50 whether a man stayed on watchful waiting or decided to have surgery, radiation, hormones, brachytherapy, etc. We then did a study of this same concept of watchful waiting in a selected group of 313 men with lower PSA levels, lower stage and grade of disease, and younger age. All of them had relatively good clinical profiles. Only about a quarter of those men stayed on watchful waiting for four years - a dropout rate of approximately 75%. The "take home message" could take two forms: "We did not know how to do watchful waiting because most patients dropped out"...or... "We delayed treatment so patients could avoid side effects and maintain quality of life longer." We really don't know the answer. We need to explore it further. Why is it that so many men who start on watchful waiting drop out?

I think there are a couple of messages. First, we truly don't know how to select men for watchful waiting. Since we had so many dropouts, there must be other factors to help us do a better job of selection. On the other hand, maybe the dropout rate is appropriate. Maybe this is just the right way to do it, because these men eventually get other treatments. One of the key research questions remains unanswered. If a man started on watchful waiting and then went on to surgery or radiation, was he harmed by losing the window of opportunity for successful therapy? We don't know that. We're trying to explore this question in the CPDR database.

**Surgery versus Radiation.** This is always a controversial issue. The CPDR was fortunate to be part of a large collaborative study on this topic. The study addressed cancer-specific mortality after surgery or radiation for patients with clinically localized prostate cancer. Anthony D'Amico, the well-known radiation oncologist at Harvard Medical School and Peter Carroll, Chief of Urology at the University of San Francisco were the other study participants. We combined data to create a group of over 7,000 patients treated at 44 institutions. These patients represented almost 5,000 surgery patients and almost 2,500 radiation patients. We risk-stratified these men, that is, we used their PSA levels, biopsy Gleason scores, and clinical stages to place them into groups (low risk, intermediate risk, high risk) that provide a probability of how likely it is that the cancer is completely contained or spread. This helps to determine how a patient is treated, whether he needs additional treatments beyond surgery or radiation, etc.

In general, we found that at ten years, low-risk men treated by radical prostatectomy, regardless of age, had a higher probability of dying of something other than prostate cancer compared to men treated by radiation. A urologist looking at this might say, "This is great! Surgery is better." The radiation oncologist would respond, "The study is not a randomized trial. Radiation was conventional for the most part, and current radiation technology is much improved." So you see dilemma here. It does appear that surgery is somewhat better for the low risk category, but it was not a randomized trial. It is impossible to do a randomized trial because no one would agree to flip a coin to determine his therapy. At the very least, the study provides another patient education and counseling tool to help newly diagnosed men make their treatment decisions.

**Rising PSA.** The interest in the meaning of a rising PSA after primary therapy is unabated. As virtually everyone in this room knows, if you've gone through surgery, radiation, brachytherapy, or cryotherapy, your PSA should go very low and

stay there. If it rises again, it generally means the cancer is back. So a rising PSA is an indicator of recurrent prostate cancer. But is it localized cancer or advanced cancer? That's the big question. A rising PSA poses a difficult challenge, and I know that some of you here tonight face it.

The CPDR database provides some insights. We looked at 4,966 men who had undergone a radical prostatectomy between 1987-2002. Of these men, 1,753 (35%) had a PSA recurrence -- a recurrence being defined as a PSA going back above 0.2 at some point after the surgery. We had a lot of them. The more interesting issue for the patient is that at a follow-up after about 6-7 years, only 170 of the 4,966 patients (3.4%) have progressed to metastatic prostate cancer that we can see on a bone scan or CAT scan. The key message is what I call the "disconnect factor." Even though there are a lot of men who have a rising PSA, it does not necessarily translate into something bad in the short or intermediate term. They reasonably can anticipate a life without metastasis.

As another illustration, let me show you what is called a "survival curve." It indicates that over time, more than 90% of these men who had surgery in the military health care system are doing pretty well. Their life goes on. That's the other message for the counselors in this room. When you are counseling men who have a PSA recurrence, in light of this data it is critically important to make sure they understand it—not just to make them feel good, but the reality is that in the large majority of cases, a rising PSA does not translate into anything devastating for prostate cancer. Life will likely go on without metastasis.

Some of these men went on hormone treatments and some did not. The point is that they don't have metastasis. They are still living a relatively normal life. In a related matter, we looked at the effect of early versus late hormone therapy after PSA recurrence in the incidence of developing clinical metastasis. Basically, the bottom line is that if we take high risk men who have the worst cancer features—higher Gleason scores, higher

PSA levels, higher PSA doubling times during recurrence—we do see that applying hormone treatment early versus late does seem to prevent a man from developing bone metastasis. So there is some evidence that applying hormone treatment earlier may be better for certain subgroups of patients.

**PSA Doubling Time.** One of the last concepts I want to mention is PSA doubling time. If a man is experiencing a rising PSA, then typically he and the doctor regularly will monitor the PSA. Many of you have done this. You've gone back every six, eight, or twelve weeks to get another PSA and follow its rise. The key factor is how quickly that PSA doubles. You should not want to start treatment too early. You would follow your PSA for a while to see how quickly it is rising before prescribing hormones. In another study with Harvard and the CAPSURE database, we evaluated the PSA doubling time after primary therapy to determine if PSA doubling time did indeed predict death from prostate cancer. We had almost 6,000 surgery patients and over 2,500 radiation patients with the same data set in the other study. The bottom line is this: If a man gets a PSA doubling time of three months or less, that is a marker for death from prostate cancer. If the PSA doubling time is greater than three months, we are more optimistic. Whether we use three months in every case is uncertain. In general, the concept is if a patient has a PSA doubling time of 12 months or less, the doctor will be inclined to recommend some type of hormone treatment.

### Summary

Let me summarize and then take questions. We have shown the changes in the epidemiology of prostate cancer in the PSA era: lower stage, lower PSA levels, and better outcomes. This portends well for recommending PSA testing because we seem to be making a difference in these large groups of military men by reducing their death rates. We've shown that low pretreatment testosterone is associated with the worst post-operative stage, and there will be a lot of interest in the whole concept of andropause, i.e., what the

testosterone levels should be in older men with or without prostate cancer. We've shown that biopsy quantification is important, i.e., counting the number of biopsy cores that contain cancer. It is a significant prognostic factor, not only to predict the stage of the cancer, but also to predict how well a man is going to do after treatment. A combination of PSA level, Gleason sum, and clinical stage allows the categorization of patients into risk groups—low, intermediate, and high risk—which then can be used to predict a patient's ten-year chance of dying from prostate cancer or dying of some other cause. Finally, PSA doubling time seems to be very important in determining how to proceed if a patient is experiencing a PSA recurrence. With that I'll stop to take any questions or comments. Thank you very much for being with us tonight.

### Questions and Answers

**Q:** How many PSA readings are needed to establish that the cancer is recurring? I have had five PSA tests, but they were up and down.

**A:** In our study, we had a median of about six PSAs. In other words, the data that we generated was based on an average of about a half dozen PSAs. Common sense would suggest you probably ought to have four to six PSAs and that they should have a consistent pattern. If you saw fluctuation - three go up and then one go down—then you probably should be reluctant to rely on that compared to having a clear upward pattern. The other concern is how to measure PSA doubling time. There are a couple of ways reported in the literature. We don't know the exact answer, but generally, the clinical answer is, you must work with your doctor, look at these PSAs, plot them, and talk about it. If you and he conclude that your PSA is doubling fairly quickly, then it makes sense to consider doing something other than watchful waiting.

**Q:** Can you tell us more about when PSA doubling time becomes a factor?

**A:** Yes. It is important to know that PSA doubling time only comes into play when the patients meet the definition of recurrence. Men treated by radiation have to have three consecutive rises after reaching their PSA nadir (low point) to define recurrence. At that point (after three consecutive rises), we start counting doubling time. For surgery patients, the cut point for defining recurrence has to be 0.4 ng/ml. We don't generally start counting doubling time until your PSA gets to 0.4 ng/ml because below that level there is too much "noise" in PSA, meaning too much variability—it can bounce up and down. That's an important point. You should not start counting those rises until your PSA gets to 0.4 ng/ml. If you are plotting it yourself, you should draw a line at 0.4 ng/ml and start counting from there. Then get three or four values over a period of three to six months to see what your PSA does.

**Q:** What about combining hormones with radiation?

**A:** I'm glad you brought that up. There are now reliable studies that suggest in general that men who get radiation and combined hormone therapy for anywhere from four months to three years have a better long-term survival than men who get radiation alone. Let me add something here. In our study comparing surgery to radiation, the survival rates for radiation were generally in men who had radiation alone, before radiation combined with hormones was in vogue. So the combination of radiation and hormone therapy may change the survival curves I presented.

**Q:** What about having external beam radiation and then salvage brachytherapy?

**A:** It's an interesting concept. It is being done at some medical centers., but it should be done only by someone who has performed it multiple times. It's a technically demanding procedure. The tissues of the prostate gland, which sits close to the rectum, have already gotten a large dose of radiation, so neither the prostate tissues nor the

rectum can tolerate much more radiation. If too many seeds are placed in the prostate close to the rectum, there is a risk that the tissues will break down, and a fistula will develop. A fistula is a connection between the urinary tract and the bowel - you don't want that! The message to men considering this combined therapy should be: Consult only with a major medical center of excellence where the staff has considerable experience in the procedure.

**Q:** In men who have been through primary cancer treatment, what dietary procedures should they follow to help prevent a recurrence?

**A:** This is another hot topic. Unfortunately, we don't have a definitive answer. Dr. Neal Barnard spoke to this group recently. Many of you were here, and his remarks were summarized in the August issue of the WRAMC US TOO newsletter. He is an advocate of diet in general, and I thought he did a great job. I liked some of his concepts. Honestly, the jury is still out. Dean Ornish has a study going on to see if aggressive dietary intervention can prevent recurrences. Common sense says that a diet lower in saturated fats would be good for general health—reducing our weight, lowering our cholesterol levels, etc. Some of the popular diet recommendations probably help; they probably don't do any harm. But I can't say whether they are going to make a difference in prostate disease recurrence. Much depends on the cancer itself. For example, it is probably naive to think that dietary intervention for a man with a high Gleason score, a higher stage disease, and a higher PSA will have much impact. On the other hand, it may help the patient who has a really low level recurrence. I'm just speculating here. You can probably sense my frustration. People ask me about dietary intervention all the time, and I just don't know what answer to give them.

**Q:** What is the role of the bone scan in detecting disease recurrence?

**A:** Bone scans used to be done routinely for men with prostate cancer before the PSA era. Prostate cancer tends to spread to the skeletal system, so the doctors did periodic bone scans to look for cancer spread. Monitoring the PSA has made it less necessary to do bone scans. What we do in our practice—what most doctors do—is to order a bone scan only if the PSA has changed significantly, as indicated by doubling time, for instance. Or a patient may report a newly developed back, rib, or leg pain. As we get older, we all develop aches and pains. Men with prostate cancer may think, "The cancer's back—got it in my bones!" That's a natural tendency. A bone scan helps to relieve the anxiety. Believe me, people are delighted when they are told the problem is arthritis, not metastasis. The point is that we don't do bone scans as often. Remember this, if you have prostate cancer and you develop a new, persistent ache or pain that worsens over four to six weeks, then you should consult with your physician. This is particularly important if you are on hormones, even if your PSA has not gone up much. If you get a come-and-go ache or pain that lasts a couple of days and then goes away, it's probably not related to cancer.

**Q:** We read so much about vitamins and herbal supplements regarding prostate cancer. How do you feel about them?

**A:** This is another controversial area. Men are spending a lot of money on supplements in the hope of preventing cancer or preventing its recurrence. As far as prevention is concerned, the only thing that we can hang our hat on is Proscar, the FDA-approved drug that does prevent prostate cancer. It's been proven in a randomized trial. No nutritional supplement is likely to top Proscar's results. The National Cancer Institute has its SELECT study which is looking at selenium and vitamin E to see if those two supplements prevent prostate cancer. There also is interest in lycopene, soy, saw palmetto, and the like. Supplements may help, and they likely do no harm, but they should not be taken willy-nilly. We know from the vitamin A studies done in Europe that people

thought beta carotene was harmless, when actually it was detrimental to persons with lung cancer. Many people think a supplement is just an over-the-counter product at a health food store - "let's try it." It is possible that a particular supplement could do harm to some people. I take a

multivitamin every day, a broad spectrum type. I also take a baby aspirin every day. Most people can safely do that. I think this is reasonable. Just be sure your doctor is aware of what supplements you are taking.

---

### **Election to the Board of Directors, WRAMC US TOO**

WRAMC US TOO is governed by a twelve-member board of directors. Board members serve three-year terms. In accordance with the by-laws, an election to fill vacancies is conducted at the November meeting of the organization. There are two vacancies for the term 2004-2007. The Nominating Committee recommends that Vin McDonald and Ray Walsh be elected to fill the vacancies. Both are active in WRAMC US TOO. Vin currently is our vice president and the editor of our newsletter. Ray is a regular volunteer in our group and he is involved in other prostate cancer activities in Northern Virginia. He is also a leader within the Virginia Prostate Cancer Coalition.

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

**WRAMC US TOO COUNSELORS**

**(AS OF NOVEMBER 1, 2003)**

**(These persons are willing to share their experience 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 Beigel	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)

## **INCONTINENCE**

Larry Schindler	Silver Spring, MD	(301) 649-5946
Ray Walsh	Annandale, VA	(703) 425-1474

## **HORMONAL**

"Mac" Showers	Arlington, VA	(703) 524-4857
Tony Bicknell	Springfield, VA	(703) 451-7517

## **SPOUSE SUPPORT**

Faye Lohmann	Kensington, MD	(301) 933-3678
Catherine Williams	Brandywine, MD	(301) 372-8650
Frances Porter	Bowie, MD	(301) 464-8721

## **MULTIPLE THERAPIES**

Howard Bubel	Fairfax, VA	(703) 280-5765	(Cryosurgery, Hormonal, Sexual Function)
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)
Joe Porter	Bowie, MD	(301) 464-8721	(Surgery, Radiation, Hormonal)
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)

## **WRAMC US TOO, Inc., NEWSLETTER**

**c/o CPDR CLINICAL CENTER, WARD 56**

WALTER REED ARMY MEDICAL CENTER  
WASHINGTON, DC 20307-5001

**FIRST CLASS MAIL**  
**MAIL**

**FIRST CLASS**

◆ MEETING ANNOUNCEMENT ◆

WEDNESDAY, NOVEMBER 5, 2003  
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?”

