The Manitoba Prostate Cancer Support Group NEWSLETTER

Prostate Cancer Canada Network Winnipeg

Vol. 248 – FEBRUARY 2012

NEXT MEETING: Thursday February 16, 2012
Heather Wiens, B.Sc., R.N., B.N., M.Sc.
Coordinator, Patient Services,
"Patient Services at CancerCare Manitoba"
Location: Seven Oaks General Hospital
Main Floor Auditorium - Leila & McPhillips
Time: 7:00 p.m. - 9:00 p.m.

Medical Advisors

Paul Daeninck M.D.
Pain Management

Darryl Drachenberg
M.D. Urologist

Graham Glezerson
M.D. Urologist

Ross MacMahon
M.D. Urologist

John Milner
M.D. Urologist

Jeff Sisler M.D.
Family Practitioner

Thanks!

The Manitoba Prostate Cancer Support Group encourages wives, loved ones, and friends to attend all meetings.

Feel free to ask basic or personal questions without fear of embarrassment. You need not give out your name or other personal information.

The Manitoba Prostate Cancer Support Group does not recommend treatment modalities, medications, or physicians.

All information is however freely shared.

PROSTATE AWARENESS PRESENTATIONS

Over the years PCCN – WINNIPEG has made public presentations to a variety of groups. Tom Boomer, others and I have traveled within the city and outside the city to make the presentations to employer/employee session, English Second Language students and church groups. The presentations are about 45 minutes long and we allow additional time for questions and answers. Those interested in a presentation may contact any board member to make arrangements for us to attend.

Len Bueckert

Thought for the Day

"It is common sense to take a method and try it. If it fails, admit it frankly and try another. But above all, try something."
Franklin D. Roosevelt

The Manitoba Prostate Cancer Support Group has been providing services for 20 years:
Newsletter – Website - Monthly Meetings - Hospital visits - Presentations
Your **DONATIONS** make it all possible. **We Thank You.**

Donor's Name: _____
Address: _____ Postal code: _____
This gift is in memory/honour of_____ Please send notification to:
Name: _____
Address: _____ Postal code: _____
$25 $50 $75 $100 $250 other_____ Make payment to:
Manitoba Prostate Cancer Support Group 315 – 971 Corydon Ave. Winnipeg, MB R3M 3S7
*A tax deductible receipt will be issued. Charity number: 88907 1882 RR001

Where to Draw the Line on the Definition of Prostate Cancer

By: Gary Schwitzer January 13, 2012

A recent commentary in the British Journal of Urology International (BJUI) by authors from urology departments in Canada and the UK asks, "Should we really consider Gleason 6 prostate cancer?" (subscription required for full access).
The National Cancer Institute defines the Gleason score as:
A system of grading prostate cancer tissue based on how it looks under a microscope. Gleason scores range from 2 to 10 and indicate how likely it is that a tumor will spread. A low Gleason score means the cancer tissue is similar to normal prostate tissue and the tumor is less likely to spread; a high Gleason score means the cancer tissue is very different from normal and the tumor is more likely to spread.

But on that spectrum of 2 to 10, different doctors draw different cut-off points for decision-making. The BJUI commentary stated:

"There is no doubt that prostate cancer kills, but only a minority of men who are given this diagnosis, die from prostate cancer. In the developed world we are now overdiagnosing and, more importantly, overtreating prostate cancer, a fact for which we will be criticized in generations to come. As well-intentioned urologists, we should have no trouble in justifying our radical therapy for pathologically moderate to high grade, Gleason 7 – 10 cancers. Despite the opinions of some urological luddites, careful active surveillance is slowly becoming the standard for Gleason 6, particularly for those with low volume disease associated with low serum PSA values, however, many patients with Gleason 6 still receive radical treatment. We (and others) would like to hypothesize, at least for the sake of discussion, that Gleason 6 pattern prostate pathology is not in itself a lethal prostate cancer, but rather can be associated with a higher risk of potentially lethal prostate cancer (e.g. Gleason 7 or higher) or, alternatively, is a precursor to such prostate cancer. This change in thinking would mean that patients with Gleason 6 scores would not be labelled with a ' lethal ' cancer diagnosis and would be less anxious about the appropriate treatment plan of active surveillance. Many patients drop out of active surveillance and pursue radical treatment, not because of rising PSA levels, biopsy results or other forms of disease progression, but because of anxiety. There may be less morbidity (and cost) if patients were not given the 'cancer-label ' until they had Gleason 7 disease.

Whether Gleason 6 is really a cancer or not is a mute point, one that can only be debated, at this time. We continue to over-diagnose and subsequently over-treat unfortunate men who are labelled with a 'lethal' cancer, when in fact they will probably never die from it. It is a fact, however, that some men continue to die from prostate cancer, so we must try and direct our therapies to those men, a task that will only be possible through enlightened discussion coupled with basic and clinical research. We need to change our paradigm when dealing with Gleason 6 pattern diagnosis, whether it is a low-risk cancer, a benign disease associated with a high risk of developing real potentially lethal cancer, or a true prostate cancer precursor. Let 's find a way to treat only those men who are destined to die from this serious cancer and relieve some of the psychological burden and significant morbidity from those men who should never have been labelled as having a lethal cancer in the first place. Let us make the case and put in the effort to develop improved prostate cancer screening for the higher grade prostate cancers, while at the same time relegating low volume Gleason 6 to the status of no more than a significant risk factor. Let us decide as a profession to stop the push for inappropriate, expensive, inopportune and perhaps even unethical radical therapies for a condition that by itself does not kill our patients."
What to call certain abnormal cellular findings is increasingly becoming an issue for doctors.

In breast cancer, there's been some discussion of re-naming ductal carcinoma in situ and removing the "carcinoma" from the diagnosis.

In cervical cancer, there are ASCUS cells – or "atypical squamous cells of unknown significance."

Gleason 6 cells in the prostate cancer field have been called "adenosis." They've been called IDLE – indolent lesions of epithelial origin.

Whatever these cells are called, one practical goal for now is to educate men about the harms of overdiagnosis and overtreatment, and offer active surveillance as a treatment option.

• • •

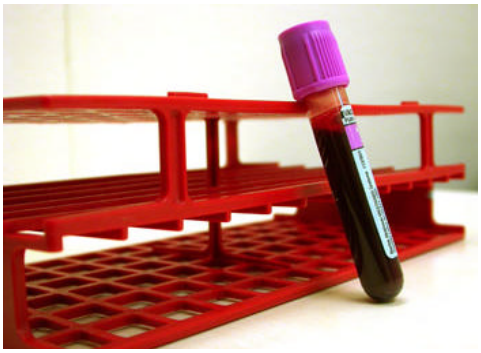
Early Detection Vital In Prostate Cancer:

A guest column by Dr. J. Christian Winters

Published: Saturday, November 05, 2011, 10:17 AM

Urologists have long recognized the wide differences of prostate cancer biology. These differences are largely responsible for the variability in outcome.

Patients with aggressive cancers usually progress and are likely to need treatment. However, patients with low-grade prostate cancers may not progress and may not need treatment. In these cancers, a significant number of men receive treatment for a cancer that probably doesn't need to be treated.



In response, the recent United States Preventative Services Task Force report has determined that the use of routine PSA (Prostate Specific Antigen blood test) testing is unnecessary. This dangerous report may lead to future denials of the use of PSA testing in men, which will be a substantial impediment in providing high-quality care.

In contrast to this report, urologists have already taken steps to improve detection of prostate cancer at its earlier stages and identify men who may not need treatment.

Prior to PSA, cancers were detected by physical findings or lower urinary tract symptoms. Unfortunately for many, this is too late for cure. PSA is the most sensitive indicator to detect prostate cancer. PSA screening has resulted in a decrease in advanced prostate cancer at presentation and allowed more men to undergo potentially curative procedures for earlier stage disease.

Thus, widespread screening programs to detect men with smaller, localized prostate cancer followed. More cancers were detected in earlier stages and resulted in more procedures for treatment. With more men undergoing treatment, complications did increase. In addition, the concerns of treating men with potentially insignificant low-grade cancers were recognized.

Urologists have already addressed these very important issues by changing methods in the diagnosis and management of prostate cancer. The American Urological Association has published Clinical Guidelines that specifically recommend limiting the use of PSA screening to at-risk populations and educating all men regarding the risks and benefits of PSA screening (including overtreatment).

Thus, we are currently recommending selective use of PSA screening. Additionally, urologists are increasingly using active surveillance as a viable treatment strategy for low-risk localized prostate cancer. This consists of closely following serum PSA blood tests and repeating biopsies, reserving aggressive therapies such as surgery or radiation for patients who truly need it.

These are contemporary best practices of prostate cancer, which is not the focus of the task force report. The report focuses on PSA screening. As clinicians, our focus is on outcomes. We use PSA as an integral part in the diagnosis and management of prostate cancer. This is not reflected in the task force report and is the major deficiency in the recommendations.

In an era where we can actually localize cancers and apply targeted therapies to treat prostate cancer, we continue the quest of decreasing complications from the treatment of prostate cancer. To abandon the use of PSA testing is relegating us back to the 1960s and digital rectal examinations. By then, it's just too late.

Here lies the powerful issue of patient choice. Can we really tell a 62-year-old man not to worry at all about his low-grade prostate cancer and that treatment is unnecessary? No, we cannot. The truth is, we can try to predict his outcome but not with absolute certainty.

In 2011, I should be able to choose a PSA test if I want and make an informed decision with my doctor to treat my cancer. That's not unnecessary treatment. It's patient-focused care, and that should be the standard. Breast cancer advocates clearly acknowledge that early detection and treatment choice is much better than silent, incurable progression. Again, the task force report misses this point.

I hope many men are as concerned about a report based on technology and treatments as old and outdated as the diagnostic methods we may be relegated to. Gentlemen, we should follow the lead of women who rallied with outrage when the task force made similar reckless recommendations about screening mammography.

Dr. J. Christian Winters is professor and chair of Urology at LSU Health Sciences Center New Orleans School of Medicine.

• • •

Penile Rehabilitation: What's Up?

Note: The following article on penile rehabilitation was written by Winnipeg Pharmacist, Greg Harochaw. He is a recognized expert in pain management, palliative care, erectile dysfunction and the art of compounding medicines. Mr. Harochaw has given many lectures and educational seminars on these topics. The MPCSG Board wishes to thank him for this article and his continued support.

In a modern era of early prostate cancer detection, penile rehabilitation is something that you hear about more and more. This is a definition I found on the internet: "Penile rehabilitation is a term most often used to describe treatment men may receive after having surgery on their prostate to treat prostate cancer. These surgeries often lead to erectile dysfunction, and penile rehabilitation involves a variety of treatments designed to allow a man to have erectile functioning to facilitate the sex life he would like. Broadly speaking penile rehabilitation simply refers to an organized way of resolving penile and erectile difficulties after some sort of trauma to the penis has occurred."

At one time we thought if people had a nerve sparing operation with a radical prostatectomy and then waited to heal, then within 2 years one would start returning to normal erectile function. Mulhall and colleagues found that after a nerve-sparing radical prostatectomy, that men ranged from 14% venous leakage at 4 months to more than 50% at 12 months (significant venous leakage prevents the penis from maintaining or forming an erection satisfactory for penetration). As well only 9% of men with evidence of venous leakage had erections sufficient for intercourse, compared with 47% of men with normal hemodynamics.

People with known injury to the neurovascular bundles (nerves) likely proceed through a continued cycle of smooth muscle cell death, leading to irreversible veno-occlusive disease. Similarly, people with preserved neurovascular bundles might demonstrate progressive fibrosis of the cavernosal tissue during the period of neuropaxia (death of a nerve), leading to the same endpoint of venous leakage. In this latter group, these people can be targeted with penile rehabilitation to decrease fibrotic changes. So having functioning nerves are important to start the whole erectile process but, there is more to the story of erectile function returning than just sparing the nerves from harm.

I like the ending of the above definition "simply refers to an organized way of resolving penile and erectile difficulties". In trying to offer penile rehabilitation one first needs to understand a normal erectile process, the trauma that happens with a treatment one receives for prostate cancer and what happens with prolonged states of not having an erection after treatment. Once this is understood then one can offer an organized way of resolving penile and erectile difficulties.

The average man prior to surgery will experience 3 – 6 erections every night of his life (approximately 70% rigidity and 10 – 15 minutes in duration). It is believed that these nocturnal erections are to protect erectile tissue during periods of sexual abstinence. During the erection process the partial pressure of oxygen (PO2) measures 30 to 40mm of Hg during the flaccid (non-erect state) and 90 – 100mm Hg during the erection. So when the mm Hg falls below 90 – 100mm, then it is quite common men have trouble achieving an erection.

Arteries are blood vessels which leave the heart and this blood is rich in oxygen (O2). As this arterial blood circulates through the body, O2 is exchanged for carbon dioxide (CO2) formed in our tissues. Veins are responsible for returning this non-oxygenated blood to the heart where this CO2 is transported to our lungs and the CO2 is then exchanged for O2 and the whole process starts over again. So when a man receives regular erections he is exchanging blood and oxygen to feed the tissues in the corpus cavernosum (erectile chambers).

So what happens if the erectile chambers are not being feed a constant supply of oxygen and blood? TFG-ß1 and prostaglandin E1 (PGE1) are messengers found in the body which respond to PO2. Moreland and colleagues demonstrated that cells subjected to low PO2 showed a 2- to 3-fold increase of TFG-ß1. TFG-ß1 showed an increase in collagen synthesis in cultured cavernosal smooth muscle tissue. With the addition of PGE1, this suppressed the effect of TFG-ß1 on collagen synthesis. During low oxygen tension in the erectile chamber, there is an increased expression of TGF-ß1 and during high levels of oxygen tension there is an increased expression of PGE1.

So what is collagen synthesis? Collagen synthesis is the development of scar tissue which can lead to actual death of smooth muscle cells in the erectile chamber. If the erectile

(Continued on page 5)

(Continued from page 4)

tissue is damaged, then even with complete nerve recovery post-prostatectomy the erectile tissue will be unresponsive to these nerves and the patient will have permanent erectile dysfunction. The exact etiology of the fibrotic changes that occurs in the erectile chamber after radical prostatectomy remains a topic of discussion but it seems that hypoxia (low oxygen) with a prolonged flaccid state after nerve sparing radical prostatectomy can lead to cavernosal changes.

So for the natural erectile process we need functioning nerves and healthy erectile tissue. Erectile dysfunction can result from damage to the erectile nerve and the absence of O2 from increased regular blood flow to the erectile chambers.

Many studies have used PGE1 injections (alprostadil), vacuum devices and phosphodiesterase type 5 inhibitors (PDE5i) [Viagra®, Levitra® & Cialis®]. Raina and coworkers evaluated the daily use of vacuum devices beginning 2 months after surgery. After 9 months of treatment, 17% of patients using the device had a return to natural erections sufficient for intercourse compared with 11% of patients in the non-treatment group. This is not surprising since the erectile chamber will fill with 50% arteriole blood and 50% venous blood when using a vacuum device. Remember the arteriole blood is rich in O2 and the venous blood is not. However what they did find was 23% of patients in the treatment group reported a decrease in penile length and circumference, compared with 60% in the non-treatment group. I know many men I talk to on how to use an intracavernosal injection to produce an erection quite often comment "I'll need to find it first" because of the amount of penile shrinkage that happened after the operation.

Montorsi and colleagues evaluated the use of alprostadil injections starting at 1 month after bilateral nerve-sparing radical prostatectomy. The investigators reported a higher rate of recovery of spontaneous erections after 6 months compared with no treatment. Specifically, 67% of men in the study group had return of spontaneous erections sufficient for intercourse at 6 months. Montorosi through the use of colour Dopler sonography found that 53% of patients who did not receive treatment demonstrated venous leakage, compared with 17% of patients receiving injection therapy. These results are consistent with what Mulhall and colleagues reported above in regards to venous leakage.

Mulhall and coworkers followed 132 patients through an 18-month period after they were placed in "rehabilitation" or "no rehabilitation" groups after radical prostatectomy. Patients undergoing rehabilitation agreed to take sildenafil (Viagra®) or alprostadil injections to induce erections 3 times weekly starting within the first 4 weeks after surgery. 52% of men in the rehabilitation protocol group reported spontaneous functional erections, compared with 19% of the men in the no-rehabilitation group.

PED5i might cause an improvement in endothelial cell function through a mechanism independent of the neural-induced nitric oxide pathway, which is the normal pathway in which these medications work. Sommer and Schulze evaluated men with erectile dysfunction of multiple etiologies with the daily use of sildenafil over a 12-month period and a second group who took sildenafil on an "as needed basis". He found that 59% of men who took sildenafil daily for 1 year responded to quality erections vs. 9% of men who took sildenafil on "as needed basis".

Mulhall finds that it is very uncommon to have venous leakage before 4 months of surgery and feels one should start some form of rehabilitation within that time frame. At 8 months after surgery, 30% of men will experience venous leakage and 50% at 1 year. When I spoke to Greenspan, he said he starts his rehabilitation after 1 month of treatment using vacuum devices.

Mulhall's experience is that about 15% of patients respond to PDE5i in the early stages after surgery. He recommends a daily night time dose of with either Viagra® 25 – 50mg or Levitra® 5 – 10mg. If one breaks the stronger strengths, Viagra® 100mg or Levitra® 20mg into quarters or halves, then the price can be reduced dramatically but these may be difficult to do so as these tablets were designed not to be split. If after a month there are no results then he moves onto a combination of one of these two medications and intracavernosal injections.

Despite that hypoxia is the inciting factor in these fibrotic changes; the exact etiology is still unknown. Many studies are very small in number and have not had an adequate placebo group (a group that uses no treatment) to make fair comparisons.

So here is what I believe is an organized way to penile rehabilitation. One should use a combination of regular intracavernosal injections combined with vacuum devices starting between 1 – 3 months after surgery (the earlier the

(Continued on page 6)

(Continued from page 5)

better). The intracavernosal injections to be used 3 times a week regularly to allow for the exchange of oxygenated blood to reduce the chance of cell death leading to a lesser chance of venous leakage. This combined with using an erection device on the off injections days will help with the reduction of penile size men commonly see after an operation.

Just a note on vacuum devices, I get asked sometimes how come our vacuum devices cost more money than ones people find in "Love Shops". The answer is the ones we sell are regulated by the FDA and they have pressure pop-off valves to prevent penile injury. Devices not FDA approved may not have these pop-off valves and can expose the penis to pressures in excess of what is deemed safe and can possibly cause damage to the penis.

With the use of intracavernosal injections we make a lot of different combinations and this would be a discussion on its own. A very common one we make is one called Trimix which I feel is the most cost effective product available. Caverject® is a commercial product available and can be purchased with a prescription from any pharmacy. The manufacturer states that after reconstitution, the solution of Caverject® should be discarded within 24 hours. However studies done by Uebel showed stability of this product if kept in a fridge at a temperature of 5°C will retain its potency for up to 5 weeks. A side note on this is that one needs to ensure they have good sterile technique, otherwise the product may be contaminated and increase the chance of an infection when used.

References

1. Dall'Era JE, Mills JN, Koul HK, Meacham RB. Penile Rehabilition After Prostatectomy: Important Therapy or Wishful Thinking?. Urology 2006;Fall;8(4):209-215 [PubMed].

2. Mulhall, John P. Saving Your Sex Life: A Guide For Men With Prostate Cancer. Hilton Publishing Company. 2008 Print.

• • •

Factors That Influence Patient Enrollment In Active Surveillance For Low-Risk Prostate Cancer - Abstract

Department of Urology, University of Miami Miller School of Medicine, Miami, Florida.

To learn from patients their rationale for enrollment in active surveillance (AS) for low-risk prostate cancer as an alternative to primary treatment.

A rank-order survey was designed to assess the relative influence of factors that contributed to the decision to elect AS. The survey was mailed to 185 patients enrolled in AS at our university-based urologic oncology practice. Participants were also asked whether they had been offered AS as an alternative to primary treatment by the urologist who had initially diagnosed their cancer.

The survey was returned by 105 (57%) of 185 patients. AS was offered to 38 (36%) of 105 patients by the physician who had made the initial diagnosis. Patients most frequently reported physician influence as the greatest contributor to their decision to elect AS (73%). Patients also cited concerns regarding the potential side effects of incontinence (48%) and erectile dysfunction (44%) associated with therapy as reasons for choosing AS.

The results of the present study have shown that patients are heavily influenced by physicians in their decision to elect AS. Notably, the majority of our sampled patients were not offered AS at diagnosis. Evidence has indicated that AS is an appropriate approach for low-risk prostate cancer and should be discussed with patients in this risk category.

Written by: Gorin MA, Soloway CT, Eldefrawy A, Soloway MS.

• • •

Study:
Trends In The Prevalence Of Cancer

1997 to 2008

Five-year cancer prevalence rates for most cancers increased from January 1, 1997, to January 1, 2008. Increases were relatively large for liver and thyroid cancer, while rates declined for cancers of the larynx and cervix. The biggest disparity between the sexes was for cancer of the lung, for which rates declined slightly among men, but continued to increase among women.

For all cancers combined, the five-year prevalence rate at the beginning of 2008 was 1,490 cases per 100,000 population. The most prevalent was prostate cancer (610 cases per 100,000). In comparison, the corresponding prevalence rates for thyroid (53.1), cervical (32.5), laryngeal (10.0) and liver cancer (6.2), were considerably lower.

In general, cancer care services required within the first five years after diagnosis include primary treatment and supportive care, followed by close clinical assessment for recurrence.

Several factors may lead to increases in prevalence rates, including the aging of the population, improved detection of disease through advancements in screening, more extensive use of screening, increases in underlying risk factors for disease and improved rates of survival for people with cancer.

For all cancers combined, roughly half of the reported average annual rates of increase for five-year prevalence were attributable to aging. However, for individual cancers, the role of aging varied considerably. For example, about 20% of the increase in prevalence for liver cancer was a result of aging.

Rates of change

The five-year prevalence rate for all cancers combined rose 2.1% per year from 1997 to 2008.

The average annual increases in five-year prevalence rates for liver and thyroid cancer were more than double the increase for any other cancer.

The five-year liver cancer prevalence rate increased 8.3% per year. For thyroid cancer, the average annual increase was 7.9%: 3.7% from 1997 to the beginning of 2000, and 9.5% from 2000 to 2008. Increases were higher in men for liver cancer, but higher in women for thyroid cancer.

Note to readers

This study is the first detailed report of trends in cancer prevalence in Canada. "Prevalence" is used here to refer to all cancers diagnosed within a given period among people alive on a specified date. It should not be confused with "incidence," which refers to newly occurring cases.

Five-year prevalence at the beginning of 2008 was estimated by counting the number of cancers diagnosed from January 1, 2003, to December 31, 2007, among people alive at the beginning of 2008.

Changes in cancer prevalence rates result from changes in the incidence of and survival from the disease. Several factors, the importance of which varies by cancer type, may account for changes in incidence and survival.

Prevalence was calculated using cancer incidence data from the January 2011 version of the Canadian Cancer Registry (CCR), a population-based database maintained by Statistics Canada. The CCR contains information on cases diagnosed from 1992 onward, compiled from reports from every provincial and territorial cancer registry.

Mortality data, also used in prevalence calculations, come from the Canadian Vital Statistics Death Database, also maintained by Statistics Canada. Data on deaths are based on information provided by the vital statistics registrars in each province and territory.

For data comparability reasons, the analysis excludes data from Quebec.

Among the cancers considered in this study, declines in prevalence rates occurred only for cancers of the larynx and cervix. For example, the annual average rate of decrease in five-year prevalence for laryngeal cancer was 1.9% and for cervical cancer, 1.5%.

Leading Cancers

Prevalence rates for prostate cancer, the most common cancer in Canada, rose substantially, primarily because of

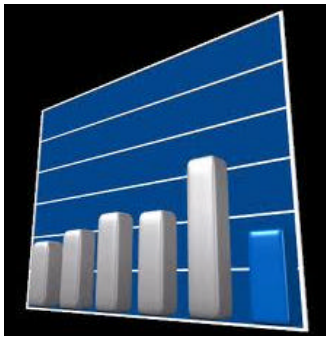
(Continued on page 8)

Return undeliverable Canadian addresses to
Manitoba Prostate Cancer Support Group
315-971 Corydon Ave.,
Winnipeg R3M 3S7

Publications Agreement
40037332

(Continued from page 7)

the aging of the population over the study period. For example, the five-year prevalence rate for this cancer increased 3.0% per year from 1997 to 2008.



Substantial increases in prostate cancer prevalence rates occurred among men in all age groups younger than 70 years. Average annual rate increases were highest at ages 40 to 49. The size of the increase fell in each successively older age group.

Increases in the prevalence of breast cancer, the second most common cancer and the most common in women, were more moderate. The annual rate of increase in five-year breast cancer prevalence was about three times higher before the beginning of 2001 (+2.3%) than afterward (+0.7%). Over the entire period, the five-year prevalence rate rose by an average of 1.3% per year.

Colorectal and lung cancer were the third and fourth most common cancers, respectively. The average increase in five-year colorectal cancer prevalence from 1997 to 2008 was 2.3% per year: 2.5% per year to the beginning of 2003 and 1.9% per year afterward. Increases for colorectal cancer were highest for people aged 20 to 39.

For lung cancer, the five-year prevalence rate increased 2.6% per year since the beginning of 2005, up from a rate of less than 1% a year before this period. Over the whole period, the rate increased by an average of 1.3% per year.

Between the sexes, changes in the prevalence rate of lung cancer diverged. In men, the rate declined slightly, 0.3% per year, but in women it increased 3.0% per year. This discrepancy was the result of sharper decreases in smoking prevalence among men since the mid-1960s.

Source: Statistics Canada January 18, 2012

• • •

Email - manpros@mts.net

Answering Machine - (204) 989-3433

SPEAKERS:

February 16, 2012
Heather Wiens, B.Sc., R.N., B.N., M.Sc.
Coordinator, Patient Services
"Patient Services at CancerCare Manitoba"

March 15, 2012
Dr. Dara Morden, Naturopathic Doctor
“The Impact of Adrenal Fatigue for both Patient and Caregiver”

April 19, 2012
Dr. Darrel Drachenberg, Urologist -
"New Prostate Cancer Therapeutic"

All meetings are held at
Seven Oaks General Hospital Auditorium
7-9 p.m.
Everyone welcome

M.P.C.S.G. Board

Brian Sprott - Chair	668-6160
Joseph Courchaine - Treasurer	257-2602
Len Bueckert - Newsletter	782-4086
June Sprott - Secretary	668-6160
Darlene Hay - Membership	837-6742
Kirby Hay - Information Kits	837-6742
Liz & Pat Feschuk - Special Projects	654-3898
Jim Leddy - Outreach	326-1477
Laurie Courchaine - Member at Large	257-2602