



Informed Prostate Cancer Support Group Inc.

"A 501 C 3 CORPORATION ID # 54-2141691"



February 2019 NEWSLETTER

P.O. Box 420142 San Diego, CA 92142

Phone: 619-890-8447 Web: <http://ipcs.org>

We Meet Every Third Saturday (except December)



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President

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Next Meeting

Feb 16, 2019

10:00AM to Noon

Meeting at
Sanford-Burnham-
Prebys Auditorium
10905 Road to the
Cure, San Diego CA
92121

SEE MAP PAGE 10

**PROSTATE
CANCER
2 WORDS, NOT A
SENTENCE**

Monday, February 04, 2019

Volume 12 Issue 02

What We Are About

Our Group offers the complete spectrum of information on prevention and treatment. We provide a forum where you can get all your questions answered in one place by men that have lived through the experience. Prostate cancer is very personal. Our goal is to make you more aware of your options before you begin a treatment that has serious side effects that were not properly explained. Impotence, incontinence, and a high rate of recurrence are very common side effects and may be for life. Men who are newly diagnosed with PCa are often overwhelmed by the frightening magnitude of their condition. Networking with our members will help identify what options are best suited for your life style.

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Editor: Stephen Pendergast

WE ARE SEEKING REPLACEMENT FOR SOME OF OUR IPCSG TEAM

Serving in this team can be rewarding and is a way to pay it forward to the group. To offer your services and/or ask questions about functions, Contact any of the individuals at their listed phone number.

FUNCTIONS NEEDED:

1. **President:** IPCSG public relations, research and advice. Lyle LaRosh has performed for 18 years. 619-892-3888
2. **Vice President:** Support all team members, assist in monthly planning and speaker acquisition. *currently vacant* Gene Van Vleet has performed Functions 2, 4, 5 for 11 years. 619-890-8447.
3. **Meeting facilitator:** Monthly planning and speaker acquisition. George Johnson has performed for 8 years. 858-456-2492
4. **Treasurer/Secretary:** Handle banking, accounting, government reporting (see 2)
5. **Hot Line:** Communicate directly with newcomers and handle phone inquiries. (see 2)

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Video DVD's

DVD's of our meetings are available in our library for \$10ea. Refer to the index available in the library. They can also be purchased through our website: <http://ipcs.org> Click on the 'Purchase DVDs' tab.

The DVD of each meeting is available by the next meeting date.

IPCSG is a 501c3 non-profit organization and all positions are performed gratis.

January 2019 Meeting Summary by Bill Lewis

Dr. Arno J. Mundt, Professor & Chair of the Dept. of Radiation Oncology, and Sr. Deputy Director, Moores Cancer Center at the University of California San Diego.

There is a long history of the use of radiation therapy for prostate cancer, both as a definitive treatment (without surgery) and as an adjuvant treatment following surgery. Two main types of radiation are used: External beam radiation therapy (EBRT) using photons (X-rays) or protons (particles), normally delivered over several weeks; and Brachytherapy. The latter is internal radiation therapy, in which radioactive pellets are introduced into the prostate, either permanently (low dose rate) or temporarily (high dose rate). Deciding which approach to use depends on multiple factors, of which the most important is the risk group the patient is in. The risk group may be low, medium or high and depends on the PSA enzyme blood level (whether <10 , moderate, or >20), the Gleason score (6, 7, or ≥ 8), and the "T Stage" (originally based on rectal exam results as to whether nodules are present, and now also whether the seminal vesicles are involved, or whether other organs have been invaded). The intermediate risk group is subdivided into "favorable" and "unfavorable" groups, mainly depending on how much higher-Gleason-grade disease is present.

Low risk disease is now usually monitored with "Active Surveillance," with close attention to the PSA levels, rectal exams and symptoms. PSA tests are usually performed every three months, with a repeat biopsy every 1-2 years. These patients used to be commonly treated with Brachytherapy, but not often any more. No treatment is given until test results change significantly, or MRI shows definite growth within the prostate.

Favorable intermediate risk disease is not usually given Active Surveillance, but is treated with surgery or radiation, as discussed below. The "good news" is that ADT (hormone suppression with Lupron or the like, with many side effects commonly experienced) is usually not considered to be needed.

Which treatment is best? Both surgery and radiation have improved tremendously over the past 30 years. Both have equal cure rates, but toxicity risks (side effects) are very different. No one treatment is best. One treatment may be good for one patient and bad for another. It's a very personal decision – it's your body. Be open-minded and become involved in the decision. Don't make snap decisions based on incomplete information or emotion.

Photons vs. Protons: Intensity Modulated Radiation Therapy (IMRT) uses sophisticated computers to "shrink wrap" the X-ray dose around the prostate, reducing the dose to normal tissue and thus the risk of toxicity. Protons, unlike X-rays, stop at a predetermined point in the body, so are even better at sparing surrounding tissue, and can deliver very focused treatment. UCSD is affiliated with the California Proton Center. (Refer to our DVD 2018 09 Dr. Rossi available to purchase for more proton information)

External beam vs. Brachytherapy: Both are equally effective. There is a different time commitment for the patient: EBRT requires 4-5 weeks (though this is getting shortened). Brachytherapy requires 1-4 treatments, depending on the dose rate. EBRT has less bladder toxicity, but can have more rectal bleeding. Brachytherapy may have less sexual dysfunction.

Unfavorable intermediate risk disease is usually treated EBRT with or without Brachytherapy, along with 6 months of ADT. Clinical trials suggest that adding a Brachytherapy "boost" leads to better outcomes, and allows a shorter course of EBRT. This is becoming common at UCSD. Surgery is considered less desirable, given the high likelihood of needing radiation therapy following surgery.

High risk disease (PSA >20 , Gleason 8 or more, and T3 or T4 stage) may be treated with EBRT (X-ray or protons) + Brachytherapy (as above, increasingly common) + 18 months of ADT. Brachytherapy is not likely to be used if the patient already has poor urinary function, has had a prior TURP (reaming out

(Continued on page 3)

of the urethra), a large prostate, or disease spread beyond the prostate. Dr. Mundt does not recommend surgery for high risk patients, because they will almost certainly need radiation as well, and then would have the negative side effects of both procedures.

Postoperative radiation therapy is commonly, but not always, needed following prostatectomy (i.e., surgery to remove the prostate). The radiation (either protons or X-rays) can be given soon after surgery, or can be delayed until the PSA rises. Prompt administration of radiation is called for in men who have regained urinary continence (i.e., have healed from the surgery) and who have positive margins (tumor left behind by the surgery), seminal vesicle invasion, or disease found outside the prostate. If the PSA rises from zero after surgery, radiation should be considered. It is most effective if the disease is only in the prostate “bed,” which may include margins and seminal vesicles. Imaging studies can be critical in determining where the disease is, so that the radiation can be effectively targeted. Brachytherapy is not used after surgery. If the PSA is greater than 0.2, 6 months of ADT is usually added.

Another traditional use of radiation therapy is in preventing or reducing painful gynecomastia (breast growth) in men on prolonged ADT. It only requires 3 treatments to each breast. It’s not routinely used, but may be requested.

A new indication for radiation therapy is for oligo-metastatic disease. It is traditionally used in patients with painful metastases, such as in the bones or spine, and is very helpful to avoid the need for narcotics. It also can help prevent fractures, which could require surgery and prolonged bed rest (which is actually very unhealthy in old patients!).

If there are only a few metastases, instead of being considered incurable and only being treated with ADT, now it is considered appropriate to treat such patients for cure with a combination of ADT and/or chemotherapy plus very focused radiation therapy. The advent of new imaging techniques such as C-II and Axumin has made possible the numbering and localization of these oligo-metastases. It often takes only 1 – 5 radiation sessions to “zap” the spot or spots, which means a very low risk of side effects.

Two recently-published clinical trials (SABR-COMET trial in Europe/Canada and STOMP trial in Europe) strongly support the use of SBRT (stereotactic body radiation therapy, which is super-focused radiation) in patients with oligo-metastatic disease. In the SABR-COMET trial, remission time and lifespan after treatment were both doubled. In the STOMP trial, the need for ADT was delayed almost two years, with no significant side effects. At UCSD, this approach for oligo-metastatic disease is now standard.

New Imaging Research – presentation by Tyler Seibert, MD, PhD, of the UCSD faculty.

Standard imaging (bone, CT) scans miss some tumors and have false positives or ambiguous results. Newer PET/CT scans such as C-II or PSMA are much better, but are not widely available, are usually not reimbursed by insurance, and the PSMA scans are not yet FDA approved.

MRI may become an attractive option. It is available at every modern hospital in the U.S. It is very good at detecting cancer in the prostate, and studies in the U.S. and Europe have shown promise for metastases too. Scientists at UCSD have developed an advanced MRI technique called Restriction Spectrum Imaging (RSI) that is now the clinical standard at UCSD for targeting biopsies, and for looking for cancer spread outside the prostate.

An ongoing clinical trial at UCSD is studying the use of whole-body RSI for a) Men with metastatic prostate cancer and b) Men at high risk of metastases (newly diagnosed with high- or very-high-risk prostate cancer, newly diagnosed with disease in lymph nodes, or men with a rising PSA after treatment by radiation or surgery). There is no cost to the patient or to his insurance company!

The imaging takes about an hour, and includes standard MRI and RSI from head to knees. Results

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will be compared to other imaging the patient gets. At least one such scan needs to be within 3 months of the MRI-RSI scan. The study will also look at treatment response after radiation, ADT, chemotherapy or other treatment (Keytruda, Xofigo, etc.).

Whole-body MRI-RSI could become a widely available way to look at bone metastases. Currently, there is not very good imaging for evaluating bone lesions for response to therapy. MRI-RSI could be used for precision radiation therapy (SBRT / Cyberknife), could make it easier for doctors to decide if their treatment is working or not, and would facilitate clinical trials of new therapies for metastatic disease. Note that there is no ionizing radiation with MRI, and no IV contrast is needed.

Questions:

If the PSA is rising after surgery, how high does it need to be, to qualify for the whole-body MRI-RSI study? No lower limit.

Any hope for patients with a pacemaker to have an MRI scan? Yes, but “down the road.”

Can patients who are Kaiser Permanente clients join the trial? Yes, since there is no cost to them (or to the patient), but they do need to approve your going to UCSD.

How long will the trial last? At least another 1 – 2 years.

How does the MRI-RSI compare with PSMA scanning? Both show similar results. The study is intended to give a more detailed answer as to “which is better when.”

Can a PET / CT scan (such as a C-11 acetate scan at the Mayo Clinic) be used for radiation treatment planning? Yes.

Whole body MRI scans were offered a year ago at UCSD as part of a research study, but the results weren't available to the patient. Has this changed? Yes, somewhat, and working through Dr. Mundt or Dr. Seibert would help.

After Cyberknife treatment for a rising PSA, the PSA has been at zero. What to do? Be happy, until the PSA rises. It's not a guarantee that no cancer is growing, but it's a very good indication.

Can prostate cancer affect markers for other cancers? Not likely, especially if the PSA is not rising. Look for another explanation.

How do UCSD radiologists and Kaiser interact? Good working relationship for 9 years already. UCSD can make recommendations, and a Kaiser group in L.A. decides which treatment to approve. Contact any of the UCSD radiologists for an appointment, or go through your Kaiser doctor – they all know each other.

The video of this presentation, including the PowerPoint slides, will be available via the website shortly before the next meeting, or at that meeting, which will be held on February 16th, 2019.

Anyone interested in the RSI trial discussed by Dr. Seibert during our Jan. 19th meeting may contact:

Katie O'Neil, Clinical Research Manager

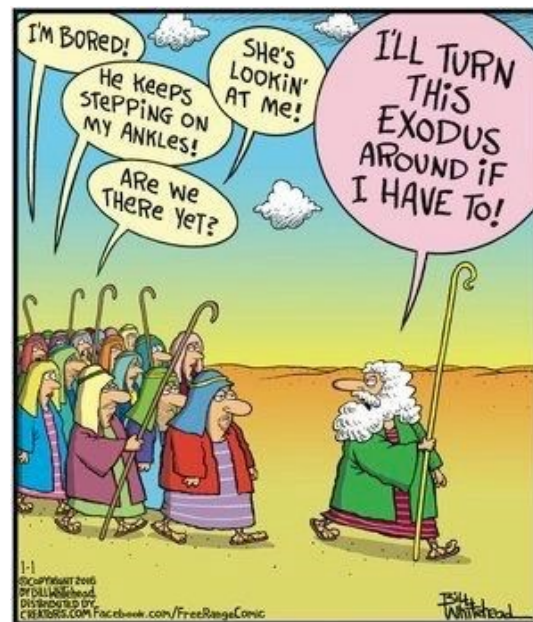
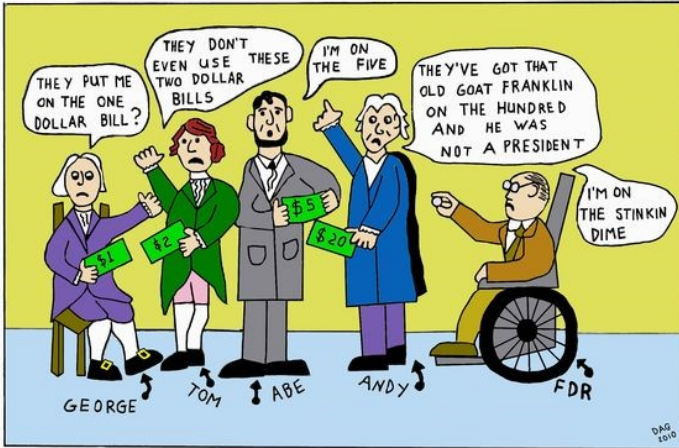
UC San Diego Health, 1200 Garden View Suite 200, Encinitas, CA 92024-0871

Office: (760) 479-5376 Fax: (858) 246-0501 croneil@ucsd.edu

FUTURE MEETINGS

- February 16, 2019 Member Panel
- **For further reading:** <https://spendergast.blogspot.com/2019/02/prostatecancernews-for-february-2019.html>
- **For Comments, Ideas and Questions,** email to Newsletter@ipcs.org

ON THE LIGHTER SIDE



Notable Articles

Gene Responsible for Spread of Prostate Cancer Identified

Fri, 01/18/2019 - 4:30pm

by Rutgers University

A Rutgers study has found that a specific gene in cancerous prostate tumors indicates when patients are at high-risk for the cancer to spread, suggesting that targeting this gene can help patients live longer.

The study, which was published in the journal Nature Communications, identified the NSD2 gene through a computer algorithm developed to determine which cancer genes that spread in a mouse model were most relevant to humans. The researchers were able to turn off the gene in the mice tumor cells, which significantly decreased the cancer's spread.

Deeper Insights

Exploring DNA Damage/Genotoxicity Using a 7-Plex DNA Damage/Genotoxicity Kit

"Currently, when a patient is diagnosed with prostate cancer, physicians can determine how advanced a tumor is but not whether the patients' cancer will spread," said lead author Antonina Mitrofanova, an assistant professor at Rutgers School of Health Professions and a research member of Rutgers Cancer Institute of New Jersey. "If we can determine whether a patient's cancer is likely to spread at the time of diagnosis, we can start them on a targeted treatment plan as soon as possible to decrease the likelihood of their cancer spreading."

Mitrofanova and collaborators are researching a potential drug to target NSD2, but she encourages doctors to begin incorporating NSD2 screening so they can start high-risk patients on anti-metastatic treatment as soon as possible.

While the algorithm used in the study focused on prostate cancer, Mitrofanova said it can be applied more broadly to study other cancers to better understand what findings can be translated to people.

According to the American Cancer Society, prostate cancer is the second most common cancer in American men and the second leading cause of cancer deaths.

Cancer Research

Radical Prostatectomy or Watchful Waiting in Prostate Cancer — 29-Year Follow-up | NEJM:

Original Article from The New England Journal of Medicine —

Abstract

Background

Radical prostatectomy reduces mortality among men with clinically detected localized prostate cancer, but evidence from randomized trials with long-term follow-up is sparse.

Methods

We randomly assigned 695 men with localized prostate cancer to watchful waiting or radical prostatectomy from October 1989 through February 1999 and collected follow-up data through 2017. Cumulative incidence and relative risks with 95% confidence intervals for death from any cause, death from prostate cancer, and metastasis were estimated in intention-to-treat and per-protocol analyses, and numbers of years of life gained were estimated. We evaluated the prognostic value of histopathological measures with a Cox proportional-hazards model.

Results

By December 31, 2017, a total of 261 of the 347 men in the radical-prostatectomy group and 292 of the 348 men in the watchful-waiting group had died; 71 deaths in the radical-prostatectomy group and

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110 in the watchful-waiting group were due to prostate cancer (relative risk, 0.55; 95% confidence interval [CI], 0.41 to 0.74; $P < 0.001$; absolute difference in risk, 11.7 percentage points; 95% CI, 5.2 to 18.2). The number needed to treat to avert one death from any cause was 8.4. At 23 years, a mean of 2.9 extra years of life were gained with radical prostatectomy. Among the men who underwent radical prostatectomy, extracapsular extension was associated with a risk of death from prostate cancer that was 5 times as high as that among men without extracapsular extension, and a Gleason score higher than 7 was associated with a risk that was 10 times as high as that with a score of 6 or lower (scores range from 2 to 10, with higher scores indicating more aggressive cancer).

Conclusions

Men with clinically detected, localized prostate cancer and a long life expectancy benefited from radical prostatectomy, with a mean of 2.9 years of life gained. A high Gleason score and the presence of extracapsular extension in the radical prostatectomy specimens were highly predictive of death from prostate cancer. (Funded by the Swedish Cancer Society and others.)

www.medscape.com

Does 'Castrate' in Prostate Cancer Need Revision? Pilot Dataset Questions the Current Standard

Nick Mulcahy

January 24, 2019

The level of testosterone (T) in the blood of men with riskier, localized prostate cancers who undergo hormone therapy should be suppressed to a level lower than currently accepted to achieve optimal outcomes, suggests a new retrospective study.

Androgen deprivation therapy (ADT) is a standard treatment for some men with intermediate- and high-risk prostate cancer that has not yet spread outside of the gland, point out the study authors, led by Brent Rose, MD, a radiation oncologist at the University of California, San Diego.

The goal of ADT is to reduce T levels to a "castrate" level, which is historically defined as < 50 ng/dL, and thereby slow the hormonal stimulation of the cancer.

However, other research has suggested that, in the setting of metastatic disease, patients with "nadir" T levels from 20 - 50 ng/dL have poorer outcomes compared to men who achieve even lower levels of T, namely, < 20 ng/dL. In short, the lower the testosterone level, the better the outcome tends to be.

So Rose and colleagues decided to study this issue in men with earlier-stage disease and add to the small body of related literature.

Their study was published online December 10, 2018, in the International Journal of Radiation Oncology - Biology - Physics.

Using a Veteran's Administration database, the investigators identified 764 men with intermediate- or high-risk localized prostate cancer who underwent treatment with ADT and definitive radiotherapy from 2000 to 2015.

The men were divided into two groups on the basis of their minimum T level during continuous gonadotropin-releasing hormone agonist therapy: < 20 ng/dL, and 20 - 49 ng/dL.

Rose and colleagues report that for men with T levels from 20 - 49 ng/dL, 10-year biochemical recurrence rates were higher (28.1% vs 18.3%), as were metastasis rates (12.9% vs 7.8%), compared to the patients with T levels < 20 ng/dL.

The difference in rates persisted when the investigator performed multivariable analyses.

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Also, shorter-term measures favored the group with lower T levels.

Specifically, the T nadir of 20 - 49 ng/dL was associated with higher 3-month postradiotherapy prostate-specific antigen (PSA) levels compared to a T nadir <20 ng/dL ($\beta = 0.16$, $P = .001$) and higher 2-year PSA nadir ($\beta = 0.12$, $P = .005$).

Higher PSA levels are undesirable and are a sign of more active disease.

There was also a trend toward inferior prostate cancer-specific mortality for the 20 - 49 ng/dL group.

"Our study suggests that there are clinically significant differences in early PSA response and long-term clinical outcomes among patients who achieve differing levels of testosterone suppression below the historical 50 ng/dL castrate level," summarize the authors.

Inadequate Testosterone Suppression?

The new results raise questions about current practice and castrate levels of serum T, suggested David Wise, MD, PhD, a medical oncologist at New York University (NYU) Langone Health in New York City.

"The NCCN [National Comprehensive Cancer Network] guidelines recommend <50 ng/dL, and this is an accepted standard," he told Medscape Medical News in an email.

"This study provides an intriguing pilot dataset that calls into question the reliance on this standard <50 ng/dL threshold," he added.

The new findings need further validation in an independent cohort, Wise advised.

The NYU physician also said that it is standard to check the serum T level after initiation of androgen suppression to ensure appropriate suppression. "Inadequate testosterone suppression can happen and is a preventable cause of poor outcome," he reminded.

Wise said the new findings are consistent with previous studies that suggest a link between the depth of testosterone suppression and clinical outcome.

According to the study authors, the new results also prompt the question, what do you do clinically when T levels cannot be satisfactorily suppressed?

"Newer therapies such as abiraterone [multiple brands] and enzalutamide [Xtandi, Astellas] may suppress serum testosterone more effectively in many patients and thus hold promise as adjunct therapies in patients with insufficient testosterone suppression," they propose.

Again, Wise said use of these agents needed confirmation: "Further prospective studies will be needed to validate this approach for this highly curable disease."

The study was supported by the National Institutes of Health. The authors and Dr Wise have disclosed no relevant financial relationships.

Int J Radiat Oncol Biol Phys. Published online December 10, 2018. Abstract

Follow Medscape journalist Nick Mulcahy on Twitter. For more from Medscape Oncology, follow us on Twitter.

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Home » Harvard Health Blog » Hormonal therapy for aggressive prostate cancer: How long is enough? - Harvard Health Blog

Hormonal therapy for aggressive prostate cancer: How long is enough?

Posted January 28, 2019, 3:57 pm

Charlie Schmidt

Editor, Harvard Medical School Annual Report on Prostate Diseases

Men weighing treatment options for intermediate- or high-risk cancer that is still localized to the prostate can face a tricky question. A standard approach in these cases is to give radiation to the prostate along with drugs that block testosterone, a hormone that makes the cancer cells grow faster. For how long should this hormone therapy last? That's not entirely clear. The drugs have side effects, such as fatigue, impotence, and a loss of muscle mass. But radiation doesn't control prostate cancer effectively without them. Doctors therefore aim to give hormone therapy only for as long as it takes to help their patients, without causing any undue harm.

Now, newly published results from a phase 3 clinical trial are providing some needed guidance.

How the study was performed

During the study, scientists randomized 1,071 men with intermediate- or high-risk localized prostate cancer into four groups. One group received radiation and six months of an anti-testosterone drug called leuporelin, and the second group received radiation plus 18 months of leuporelin therapy. Two other groups were treated with the same regimens of either radiation plus six or 18 months of leuporelin therapy, along with another drug called zoledronic acid, which helps to limit skeletal pain and related complications should cancer spread to the bones. Study enrollment occurred between 2003 and 2007 at 23 treatment centers across New Zealand and Australia.

Here's what the results showed

After a median follow-up of just over 10 years, 9.7% of men who were treated with radiation and leuporelin for 18 months had died from prostate cancer, compared to 13.3% of the men treated with radiation and leuporelin for six months. Adding zoledronic acid made no difference in either case.

The authors concluded that hormonal therapy is more effective at preventing prostate cancer death when it's given for 18 months rather than six. And similar benefits were noted for other endpoints as well. For instance, prostate tumors were less likely to metastasize, or spread, among men in the longer duration treatment group, and it took longer for their cancers to become resistant to hormone therapy if it was reinitiated later.

In earlier clinical research, scientists discovered that hormonal therapy given for three years protects against prostate cancer death more effectively than a six-month treatment regimen. But three years of hormone therapy isn't easily tolerated, and evidence so far shows that 10-year survival rates after either 18 months or three years of hormonal therapy are similar, the authors of the new study claim.

"This study reaffirms what many clinicians have put into practice: longer duration hormonal therapy in appropriately selected patient populations provides a greater benefit," said Dr. Marc Garnick, the Gorman Brothers Professor of Medicine at Harvard Medical School and Beth Israel Deaconess Medical Center, and editor in chief of HarvardProstateKnowledge.org. "Prior studies using three years of hormonal therapy have also shown this, but it is important to recognize that some men may have significantly delayed return of the body's testosterone upon completion of the therapy — a fact that needs to be discussed when contemplating longer-term treatment programs."

NETWORKING

Please help us in our outreach efforts. Our speakers bureau consisting of Lyle LaRosh, Gene Van Vleet and George Johnson are available to speak to organizations of which you might be a member. Contact Gene 619-890-8447 or gene@ipcsg.org to coordinate.

Member and Director, John Tassi is the webmaster of our website and welcomes any suggestions to make our website simple and easy to navigate. Check out the Personal Experiences page and send us your story. Go to: <https://ipcsg.org/personal-experience>

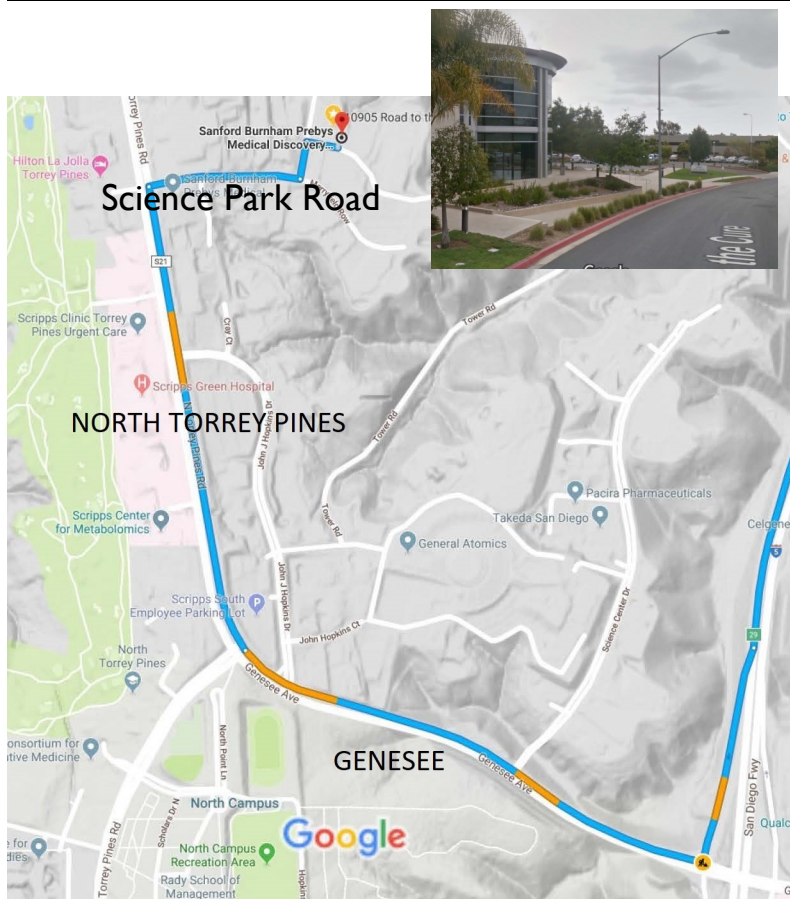
Our brochure provides the group philosophy and explains our goals. Copies may be obtained at our meetings. Please pass them along to friends and contacts.

FINANCES

We want to thank those of you who have made special donations to IPCSG. Remember that your gifts are tax deductible because we are a 501(c)(3) non-profit organization.

We again are reminding our members and friends to consider giving a large financial contribution to the IPCSG. This can include estate giving as well as giving in memory of a loved one. You can also have a distribution from your IRA made to our account. We need your support. We will, in turn, make contributions from our group to Prostate Cancer researchers and other groups as appropriate for a non-profit organization. Our group ID number is 54-2141691. Corporate donors are welcome!

If you have the internet you can contribute easily by going to our website, <http://ipcsg.org> and clicking on "Donate" Follow the instructions on that page. OR just mail a check to: IPCSG, P. O. Box 4201042, San Diego CA 92142



Directions to Sanford-Burnham-Prebys Auditorium 10905 Road to the Cure, San Diego, CA 92121

Take I-5 (north or south) to the Genesee exit (west).

Follow Genesee up the hill, staying right.

Genesee rounds right onto North Torrey Pines Road.

Do not turn into the Sanford-Burnham-Prebys Medical Discovery Institute or Fishman Auditorium

Turn right on Science Park Road.

Watch for our sign here.

Turn Left on Torreyana Road. Watch for our sign here.

Turn Right on Road to the Cure (formerly Altman Row). Watch for our sign here.