

REPORT TO PHYSICIANS

JULY/AUGUST 2007 VOL. 52, NO. 7-8

Oncology

Harnessing the Power of Protons

The precision of proton beam radiation allows more effective targeting of tumor cells with fewer side effects.

by **Dianne C. Witter**

Protons accelerate to nearly light speed as they whip through a vacuum chamber, guided by powerful magnets. Near the end of the journey, the beam delivery system shapes the proton beam so that it delivers its radiation precisely to the dimensions of the patient's tumor. These subatomic particles then slow down and stop in their target, depositing their energy within the malignant tissue and leaving adjoining tissues unharmed.

The patient feels nothing, and yet in 20 minutes, a positron emission tomography scan will allow the physician to begin to see the cancer cells die.



The appeal of proton therapy, it turns out, is not in the type of radiation it delivers, but in its precise delivery.

Consider the size of the “C” at the beginning of this sentence. That is the size of the specificity with which proton beams can be directed at a target. When you're talking about a malignant tumor nestled among critical nerves and sensitive human tissues,
(Continued on next page)

In the treatment room, a massive wheel-like gantry can be rotated around the patient to precisely direct the proton beam.

THE UNIVERSITY OF TEXAS
**MD ANDERSON
CANCER CENTER**

Harnessing the Power of Protons

(Continued from page 1)

that pinpoint aim takes on enormous significance.

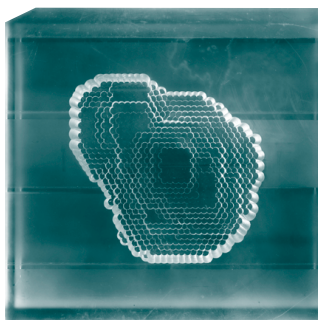
“Proton beams can be conformed to the exact dimensions of a tumor and delivered directly to it without damaging the surrounding organs or tissues,” explained James Cox, M.D., head of the Division of Radiation Oncology and medical director of the Proton Therapy Center at The University of Texas M. D. Anderson Cancer Center. Because the radiation doesn’t stray to healthy tissues, patients experience fewer side effects and are less likely to have long-term complications.

The radiation from protons has the same effect on tumor tissue as the X-rays traditionally used in radiation therapy, but with a more advanced delivery system, explained Dr. Cox. “While X-ray beams go all the way through the body, affecting any tissues in their path, proton beams have a very low entrance dose and no exit dose—the protons stop when they reach the defined target and release the radiation,” he said.

While delivering radiation with a proton beam is an advanced technology available at only a few hospital-based proton therapy centers in the country, Dr. Cox notes that proton therapy has actually been in use for some time.

“People often assume proton therapy is experimental,” said Dr. Cox. “They want to know, ‘Is it safe?’ and ‘Is it effective?’ They’re usually surprised to learn that proton therapy has been in use for medical purposes for over 50 years and that it has been approved by the U.S. Food and Drug Administration for cancer treatment since 1988.” More recently, advances in imaging technology have significantly expanded the potential applications for proton therapy.

Cancer treatment has long been a hazardous balancing act of determining how to deliver enough toxic chemicals or radiation to effectively kill a tumor without causing substantial harm, or even death, to the patient. Like molecularly targeted therapies, proton therapy represents an important leap forward in our ability to kill the cancer while sparing the patient.



A clear Lucite compensator forms the shape of the proton beam to match the back of the tumor, allowing the radiation to conform to the target.

Dr. Cox cautions that proton therapy is more appropriate for some tumor types than others, and careful selection of candidates is essential to the treatment’s success. In some cancers, the broader penetration of X-rays is preferable—in breast cancer, for instance, it’s advantageous for the radiation to go all the way through the breast to kill any stray cancer cells, he noted. Proton therapy also is not needed if tumors are especially sensitive to radiation or if high doses of

radiation are not required. Conventional radiation therapy remains a proven and important cancer treatment and will often be the treatment of choice, especially given the limited availability of proton therapy around the country.

Proton therapy is optimal for certain localized solid tumors with well-defined borders, including those of the prostate, eye, lung, brain, head, and neck (see box at right). Prime candidates for proton therapy are patients who need a higher dose of radiation than would be safe with X-rays or whose malignancy is next to critical structures.

The question of exactly which patients will ultimately benefit most from proton therapy is still being addressed in clinical trials at the Proton Therapy Center, which currently has 6 clinical trials under way and another 25 or so in the works. The trials capture data about effectiveness and toxicity and look at ways to increase the effectiveness of proton therapy, such as by giving it in combination with other treatments.

Clinical Trials of Proton Therapy

- Phase II Randomized Trial of 70 Gy Versus 78 Gy Proton Beam Therapy for Skull Base Chordoma (2005-0038). Principal Investigator (PI): Eric L. Chang. This trial will compare the effectiveness and late side effects of two different doses, 70 Gy and 78 Gy, of proton therapy in the treatment of skull base chordoma after debulking surgery.
- Prospective Evaluation of Quality of Life after Proton Therapy for Prostate Cancer (2005-0956). PI: Andrew K. Lee. This trial will collect information on the incidence of acute and late side effects of proton therapy given for the treatment of biopsy-proven, previously untreated prostate cancer. The effect of proton therapy on patients’ quality of life will also be evaluated.
- Phase II Concurrent Proton and Chemotherapy in Locally Advanced Stage IIIA/B Non–Small Cell Lung Cancer (NSCLC) (2004-0976).

PI: Joe Y. Chang. This trial will investigate the ability of a combination of proton therapy and standard chemotherapy (paclitaxel and carboplatin) to control locally advanced disease in patients who have inoperable stage IIIA/B NSCLC without malignant pleural effusion.

- Phase II Escalated/Accelerated Proton Radiotherapy for Inoperable Stage I (T1–T2, N0, M0) Non–Small Cell Lung Cancer (NSCLC) (2004-0977). PI: Joe Y. Chang. This trial will examine whether escalated/accelerated proton therapy can improve the survival rate and decrease early and late side effects in patients with medically inoperable stage IA/B NSCLC not involving the main bronchus.

For more information on these and other clinical trials, visit www.clinicaltrials.org or call 1-877-632-6789.

Who Benefits from Proton Therapy?

Pediatric patients: Their rapidly growing—and thus easily damaged—cells make children more sensitive than adults to the adverse effects of radiation. Because it minimizes the amount of healthy tissues exposed to radiation, proton therapy is the ideal form of radiation for many children.

Lung cancer patients: Particularly in patients with very little lung function, saving as much normal lung tissue as possible is a major challenge for radiation oncologists. Studies under way now suggest that proton therapy may be a more effective alternative.

Patients with cancers of the head, neck, and brain: Treatment of head, neck, and brain tumors almost always includes radiation therapy. With proton therapy, high doses can be given with-

out increasing toxicity to important structures nearby. For example, for intracranial tumors near the brain stem, a pencil-beam scanning nozzle will deliver an extremely narrow beam of protons, sparing the delicate brain stem tissue.

Prostate cancer patients: About 65% of all prostate cancer patients can be treated with high-dose proton therapy, which spares the rectum and bladder and leaves both urological and sexual functions intact.

Patients with cancers of the eye: Proton therapy's efficacy in cancer treatment was first demonstrated in ocular melanoma. The control of the proton beam enables physicians to successfully treat cancer of the eye while preserving the patient's eye and vision.

Dr. Cox described a quality-of-life study under way in prostate cancer patients who undergo proton therapy. "It's too early to know if the proton therapy will improve survival, but the acute effects typically seen with X-rays have been amazingly absent," he said. "Maintaining functions like bladder control and erectile capability after treatment can have a major effect on a man's quality of life."

Dr. Cox believes proton therapy has particular promise in lung cancer. In an analysis of patients treated for non-small cell lung cancer, published in the July 2006 issue of the *International Journal of Radiation Oncology, Biology, Physics*, Dr. Cox and colleagues found that, even with dose escalation, proton treatment significantly reduced radiation to healthy lung tissue, the esophagus, the spinal cord, and the heart, as compared to standard-dose X-ray beams. Based on these findings, the group hypothesized that proton therapy with dose escalation could result in longer survival without increased toxicity. Studies are currently under way in the Proton Therapy Center

to look at this and other questions.

"One of the key areas of proton therapy research right now is combining chemotherapy or other agents with proton therapy for a synergistic effect," Dr. Cox said. "It's a huge, relatively unexplored area with a lot of potential. We're looking at this in clinical trials right now—the decrease in toxicity from giving radiation with proton beams allows us to give higher doses of both radiation and chemotherapy than is typically possible with chemoradiation.

"So far—the studies are still under way—we've delivered proton radiation at doses 15% higher than is possible with X-ray/chemotherapy combinations; and the acute side effects, particularly esophageal effects, have been much lower or non-existent," Dr. Cox noted. "This is an exciting area because, potentially, remissions and survival rates will increase as well.

"This, for me, is the real promise of proton therapy." ●

For more information on proton therapy or to refer a patient, call 1-866-632-4782, or visit www.mdanderson.org/protontherapy.

Lymphocyte Count Predicts Outcomes in Young Patients with Acute Leukemias

Researchers from the Children's Cancer Hospital at M. D. Anderson have found that absolute lymphocyte counts (ALCs) measured 15 days after chemotherapy are a simple but powerful predictor of relapse and survival in children and young adults with acute myelogenous leukemia (AML) or acute lymphocytic leukemia (ALL).

ALC, a measure of normal immune cells, is found on routine complete blood count (CBC) reports but is not currently used to guide therapy.

"This is a simple measurement from the CBC, and what it may provide is information that is critical to early and accurate risk stratification," said Patrick Zweidler-McKay, M.D., Ph.D., assistant professor of pediatrics at M. D. Anderson.

Improvements in risk stratification based on the ALC information would allow physicians to alter treatment and potentially improve remission durability and cure rates, said Guillermo De Angulo, M.D., a Children's Cancer Hospital researcher and fellow. "The ALC test can help us identify patients who might need more or could benefit from less chemotherapy very early in their treatment," he said.

The researchers' findings were presented at the American Society of Pediatric Hematology/Oncology's annual meeting, held in May. The study, which involved multiple analyses of patient outcomes, ALCs, and other potential indicators, included 171 patients with AML or ALL, age 21 years or younger, who had begun treatment at M. D. Anderson between 1995 and 2005.

"An important aspect of what we found is that ALC measurements from different days can be used to stratify high- and low-risk groups," said Dr. Zweidler-McKay, the study's senior author. For example, in patients with AML, the patients

(Continued on page 6)

Gene-Environment Interaction Studies Answer the Question, “Why Did I Get Cancer?”

by Vickie J. Williams
and Dawn Chalaire

Every patient with cancer will at some point ask the question: “Why me? Why did I get cancer?” Some patients blame their lifestyle choices; others attribute their disease to fate or bad luck. As it turns out, the answer to the question could be said to lie somewhere in between.

It has long been known that both heredity and environment play a role in cancer susceptibility. All cancers have a genetic component because they arise from the faulty genetic control of cell growth, and about 75% of the cancers diagnosed in the United States can be attributed in part to environmental factors such as tobacco use, diet, infectious diseases, excessive sunlight exposure, industrial chemicals, and ionizing radiation. For many years, genetic and environmental causes of cancer were considered and studied separately.

Then research began to show that these factors work in concert, as co-determinants of cancer susceptibility. This realization that most cancers are caused by an interaction between genes and the environment represented a new paradigm in cancer risk assessment.

“Although an element of chance is likely to play a role in the complex, multi-step process leading to cancer development, there is mounting evidence that genetic factors influence susceptibility to cancer-causing exposures,” said Margaret Spitz, M.D., professor in and chair of the Department of Epidemiology at The University of Texas M. D. Anderson Cancer Center.

Genetic markers of cancer risk

The Human Genome Project, which identified and sequenced the approximately 30,000 genes in the human body, provided researchers with a blueprint for studying the biologic components of diseases such as cancer and has propelled the science of molecular epidemiology.

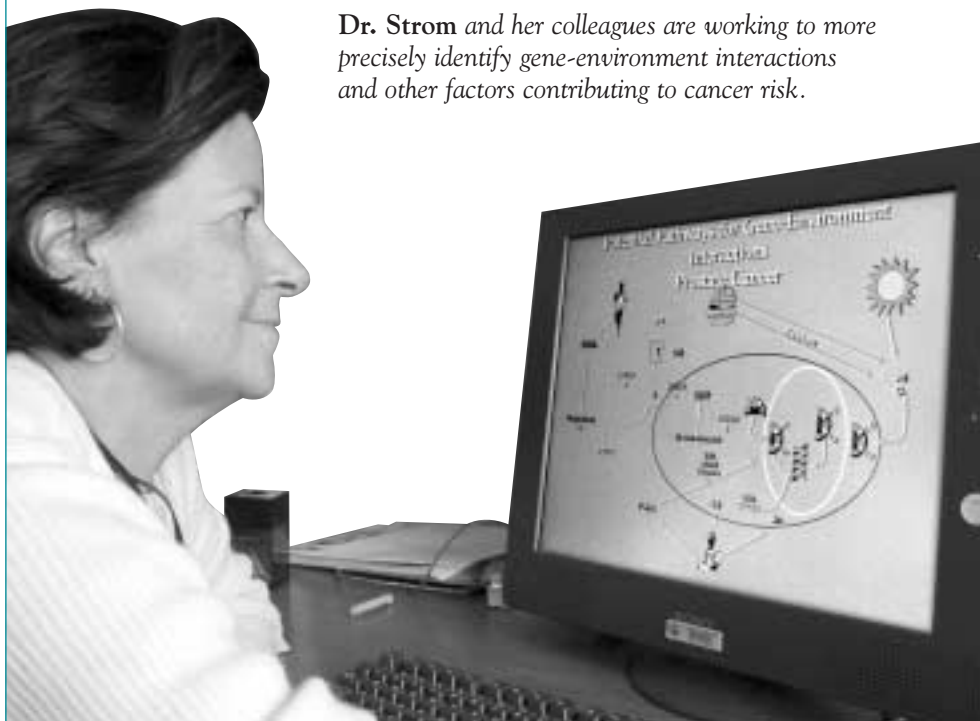
“Scientists now have a clearer picture of the composition of human DNA, which will facilitate epidemiologic studies of which genes contribute to cancer susceptibility,” said Sara Strom,

Ph.D., associate professor in the Department of Epidemiology.

Researchers with the Cancer Genome Project, a massive undertaking aimed at mapping the genetic mutations linked to cancer, are using the Human Genome Project’s gene-sequencing and high-throughput mutation detection techniques to identify the gene sequence variants and mutations critical to the development of human cancers.

The interplay between genetic markers of cancer risk and environmental factors is also the focus of extensive research at M. D. Anderson. Here, clinicians, basic scientists, and epidemiologists collaborate to identify molecular biomarkers of individual risk by studying commonly occurring variations in genes related to carcinogen metabolism, DNA repair, cell cycle control, stress responses, and immunity. “Our objective is to identify markers of genetic susceptibility for evaluation in case-control studies,” said Dr. Strom. Their studies, emphasizing genetically determined differences in people’s responses to environmental agents, or interindividual variations in cancer risk, are being conducted in tobacco-related cancers, leukemia, colon cancer, melanoma, and other cancers.

Dr. Strom and her colleagues are working to more precisely identify gene-environment interactions and other factors contributing to cancer risk.



Gene-environment interactions in smoking-related cancers

The most recognized disease outcome linked to gene-environment interactions is lung cancer. Epidemiologic and clinical studies clearly document that smoking is the leading cause of lung cancer, accounting for nearly 90% of these tumors. However, it is also well documented that only a fraction (about 15%) of long-term tobacco smokers will get lung cancer. These patients are genetically susceptible to the carcinogenic effects of tobacco.

“The diversity of human beings is remarkable,” Dr. Spitz said. “The fact that some smokers develop lung cancer while others don’t suggests that there are differences among smokers in susceptibility to the cancer-causing compounds in cigarettes.”

To date, no specific lung cancer gene has been identified; however, researchers, including Christopher Amos, Ph.D., professor in the Department of Epidemiology, have isolated a narrow region of about 50 genes on a segment of chromosome 6. This region was found in 52 families with strong family histories of cancers of the lung, throat, and larynx. The next step is to determine the exact gene or genes in this region that are associated with lung cancer.

Individuals whose cells are unable to properly repair damaged DNA also may be at higher risk for lung cancer. DNA repair systems are designed to maintain the integrity of the genome by preventing the accumulation of DNA damage that can lead to cancer. Nucleotide excision repair is one pathway responsible for repair of genes damaged by tobacco carcinogens. Unfortunately, the same nucleotide excision repair pathway is involved in repairing the damage to cancer cells caused by treatment with the common chemotherapy drugs cisplatin and carboplatin. Researchers in the Department of Epidemiology hope that by studying the genes involved in DNA repair, they might be able to construct genetic profiles that could be used to individualize therapy and to better understand treatment response in lung cancer patients.

Research is also under way in other smoking-related cancers. A study published earlier this year by Xifeng Wu, M.D., Ph.D., professor in the Department of Epidemiology, and her team analyzed the relationship between eight genetic variations that affect DNA repair, smoking, and bladder cancer. The researchers found that smoking had the greatest effect on bladder cancer risk among the factors studied. However, three of the gene variants could predict bladder cancer risk with high consistency. These results support the hypothesis that gene-gene and gene-environment interactions contribute to bladder cancer risk.

Dr. Strom is co-principal investigator of another study that is examining how genetic predisposition and environmen-

tal exposures interact to determine susceptibility to acute myelogenous leukemia. "Inhalation of benzene, which is present in gasoline, polluted air, and cigarette smoke, can induce changes in the expression of some genes, and these changes cause leukemia in some people. However, certain genetic traits must be present for a person to be affected by these exposures," said Dr. Strom. "This study will help determine how the presence of genetic markers combined with environmental exposures and cytogenetic factors can help us identify which individuals are at greatest risk for acute myelogenous leukemia."

Gene-environment interactions in other cancers

Colon cancer includes among its variants one of the most common inherited cancer syndromes known, hereditary nonpolyposis colorectal cancer (HNPCC). People with mutations of *MSH2* and *MSH6*, both on chromosome 2, and *MLH1*, on chromosome 3, are at increased risk of HNPCC. These may interact with environmental predictors of colon cancer, including obesity, low levels of physical activity, smoking, and alcohol consumption. Gene-environment studies that would confirm such interactions in colon cancer are sparse.

Marsha Frazier, Ph.D., professor in the Department of Epidemiology, is studying modifier genes in a unique cohort of patients with documented HNPCC. Dr. Frazier and her colleagues have found that variants in the insulin-like growth factor-1 (*IGF-1*) gene are involved in colorectal carcinogenesis. Their study, published this year in the *Journal of the National Cancer Institute*, was the first to report that these vari-

The fact that some smokers develop lung cancer while others don't suggests that there are differences among smokers in susceptibility to the cancer-causing compounds in cigarettes."

— Dr. Spitz

ants, which are thought to increase the production of *IGF-1*, modify the risk of HNPCC and, for that matter, any hereditary form of cancer. The findings are consistent with studies from other groups showing that high levels of *IGF-1* in the blood are associated with a higher risk of sporadic (non-hereditary) colorectal cancer. Combining what is learned about these *IGF-1* variants with information about other genetic and environmental risk factors may improve

risk prediction and allow earlier identification of individuals who are genetically susceptible to developing colon cancer.

In melanoma, evidence for a gene-environment intersection is very strong. This disease has been shown to run in families, and a mutation in the *CDKN2* gene on chromosome 9 is common in these families. The mutation, coupled with exposure to ultraviolet (UV) radiation and photosensitizing chemicals, substantially increases the risk for this cancer. However, how UV exposure from sunlight leads to the development of melanoma is not yet clear. The first large case-control study reporting an important role for faulty DNA repair in UV-induced DNA damage was led by Qingyi Wei, M.D., Ph.D., professor in the Department of Epidemiology. In a study of more than 300 patients with melanoma and matching cancer-free controls, Dr. Wei and his colleagues showed that inefficient repair of UV-damaged DNA is a risk factor for melanoma. These findings help explain the variations in susceptibility to sunlight-induced melanoma.

In another case-control study, Dr. Wei's group also compared in

(Continued on page 6)

Gene-Environment Interaction Studies

(Continued from page 5)

vitro chromosomal damage induced by UVB radiation in the lymphocytes of patients with nonmelanoma skin cancer, patients with melanoma, and cancer-free controls. Compared with controls, the lymphocytes of patients with nonmelanoma skin cancer, but not those with melanoma, were much more likely to have UVB-induced chromosomal damage.

“Both of these studies were the largest of their kinds,” Dr. Wei said. “The first study showed that UV radiation damages DNA and increases the risk of melanoma. The second study showed that the mechanism of action was not at the chromosome level. This doesn’t mean that UV exposure isn’t important, it just means that UV

radiation doesn’t cause damage at the chromosome level in melanoma. The bottom line is avoiding UV exposure is the best strategy for skin cancer prevention.”

Looking to the future

Research in the Department of Epidemiology will continue to focus on elucidating gene-environment interactions in cancer etiology and developing individualized risk prediction profiles.

“The long-term goal is to more precisely answer the question of who gets cancer and recommend personalized prevention and treatment interventions,” Dr. Strom said.

Identifying individuals or groups who are most susceptible to cancer may

make it possible to recommend careful surveillance and early detection, behavior modification strategies, or chemoprevention interventions. In cases where the disease is already present, oncologists may be able to tailor treatments according to individual genetic profiles.

“We may one day be able to answer the ‘why me’ question—‘why did I get cancer?’—and perhaps we might be able to prevent cancer from occurring at all,” agreed Dr. Spitz. “It won’t happen overnight, or even in my lifetime, but we’re definitely moving in the right direction.” ●

For more information, visit www.mdanderson.org/departments/epidemiology/.

In Brief

(Continued from page 3)

with an ALC lower than 350 cells/ μ L on day 28 of treatment had a dismal five-year overall survival rate of 10%; in contrast, the five-year overall survival rate was 85% when the ALC was 350 cells/ μ L or higher on day 15.

The findings were similar for patients with ALL, the most common form of childhood leukemia, with six-year overall survival rates of 55% versus 87% for low and high ALCs—a smaller difference but highly significant. “ALL therapy already benefits from a successful risk-stratification approach; however, currently at least half of patients who do not survive are thought to be standard risk, and this is where ALC may help,” Dr. Zweidler-McKay said.

In addition to acute leukemias, the M. D. Anderson team has found that ALC is associated with survival in pediatric patients with non-Hodgkin’s lymphoma and Ewing’s sarcoma, and other investigators have demonstrated that ALC predicts outcome in adults with AML and patients who have undergone stem cell transplants for various malignancies.

“All these findings from different sources seem to imply that a generalized post-therapy ALC phenomenon exists,” Dr. Zweidler-McKay said. “If so, it would be relevant to the way we treat a wide range of malignancies and ages, and the fact that a CBC is a universal,

inexpensive test is key. It means that physicians all over the world, even in developing countries, could use ALCs to help determine what treatments their patients really need, very early on in treatment.” ●

Gefitinib Under Study for Squamous Cell Carcinoma of the Skin

Although most cases of squamous cell carcinoma of the skin are successfully treated by aggressive surgery, sometimes followed by radiation, researchers at M. D. Anderson have identified certain factors that predict a poor outcome for squamous cell carcinoma patients. Among these factors are large tumor size (4 cm or more in diameter), lymph node metastasis, perineural invasion, and deep invasion into underlying tissue. According to Randal Weber, M.D., professor in and chair of the Department of Head and Neck Surgery, “Up to 40% of patients with these factors will die of their disease, so it’s not just an innocuous skin cancer that’s easily managed. It has a significant mortality rate.”

Squamous cell carcinoma cells overexpress the epidermal growth factor receptor. Activation of that receptor promotes cell proliferation, invasion, angiogenesis, and cell motility. However, these tumor cell actions

can be diminished if the receptor is blocked. Dr. Weber and colleagues are hoping that gefitinib (ZD1839), a small-molecule tyrosine kinase inhibitor that binds to the epidermal growth factor receptor, will block the receptor’s activation and thus inhibit or even reverse tumor growth.

So far, 15 squamous cell carcinoma patients have been treated in an ongoing phase II clinical study of gefitinib being conducted at M. D. Anderson (protocol 2004-0204). “In three patients, we’ve seen a complete disappearance of the tumor on clinical exam. In one of those patients, after we excised the tumor site, there was no residual cancer. In several other patients, their disease either regressed partially or showed no growth,” said Dr. Weber. “These preliminary results are pretty remarkable.”

Gefitinib is given daily for two periods of 30 days each (60 days total) before patients undergo surgery. Some patients, but not all, also undergo radiation therapy, depending on tumor size and other factors.

The ongoing trial is for patients who have locally advanced or recurrent squamous cell carcinoma of the skin. Tumors must be at least 2 cm in diameter or involve muscle, bone, lymph node, or perineural tissue. For more information, contact Dr. Weber at 713-745-0497 or ask MDAnderson at 1-877-632-6789 (or www.mdanderson.org). ●



Parents: Be Hopeful and Honest When Telling Kids about Your Serious Illness

Being diagnosed with a serious illness like cancer can be overwhelming in itself. But parents in this situation face the added challenge of talking about it with their children. The following guidelines can make this difficult process more manageable.

Don't keep secrets

Adults may think they are protecting their children by not discussing a serious illness. But keeping secrets is the wrong approach, experts say.

"Children have a right to know what's going on—they are part of the family," says Martha Aschenbrenner, manager of the KNIT (Kids Need Information Too) program at the Children's Cancer Hospital at M. D. Anderson.

Kids usually know when people are keeping something from them. This breeds distrust and teaches them that it's okay to not talk about problems. Moreover, they'll be deeply hurt—and you'll be stuck playing catch-up—when the truth finally comes out.

Instead, Ms. Aschenbrenner suggests talking openly to your children in an age-appropriate way. No one knows them better than you, so rely on your judgment. The important thing is to be hopeful and honest about the situation.

Take a few days to work through your own emotions, educate yourself about your illness, and think about what you want to say to your children. Starting the conversation is the hardest part, says Ms. Aschenbrenner, a cancer survivor who was diagnosed when her son was 4 years old.

Tots, tweens, and teens need different information

When dealing with a younger child, up to about 5 or 6 years old, it's not wise to simply explain that you are "sick." Then the child might start to

think that even getting the sniffles will mean a trip to the hospital.

Instead, tell the younger child that you have to go to the doctor, and identify the illness by name. Doing this won't cause fear—to kids at these ages, a word like "cancer" is just a word. Unless you have a communicable disease, they also need to know that they can't catch it.

Talking with younger children about the possibility of dying can be difficult, and parents should allow children to ask questions as they come up. Even without the parent's illness, children will eventually experience someone's death, so it is useful to help even young children understand what "dying" means. Separate this from the illness talk; explain that when something dies, the body stops working. Faith and religious beliefs can be discussed at this point.

"And later, if the parent's death is imminent, you can refer back to that earlier conversation about what it means to die," Ms. Aschenbrenner says.

Older children probably have heard broadly about illnesses like "cancer" or "heart disease." So it's important to explain your specific type of disease and prognosis. You don't want a child thinking you are going to die if your chances for recovery are excellent.

It is, however, normal for children between the ages of about 7 and 11 years to ask whether a parent with a serious illness will die. Do not promise that won't happen. Rather, Ms. Aschenbrenner suggests answering like this: "I hope not. I'm in the best hospital, and the doctors are working hard to get me better. If anything changes, I'll tell you." Then, when something does change, follow through.

Teenagers are likely to have many more questions. Again, answer these openly and honestly until their need for information is satisfied. Be forthcoming with older children should the disease worsen, and explain how that changes your prognosis.

But don't be alarmed if teens don't seem to have much to say. "Teenagers

More Ways to Help Kids Cope

- Tell your child's school about your illness.
- Keep a notebook in the house so children can write down questions they may not want to ask out loud.
- Post a master calendar for household activities and your doctor's visits.
- Hold family meetings after seeing the doctor.
- Seek out a support group for your child or family.
- Take children to the hospital.
- Find a book on this topic, such as *How to Help Children Through a Parent's Serious Illness* by Kathleen McCue (St. Martin's Griffin, 1994).

don't talk to their parents," Ms. Aschenbrenner says. "That's normal developmental behavior. The important thing is to make sure they're talking to someone."

Manage the situation for your child's sake

Children of all ages need assurances that no matter what happens, they will be cared for. Explain who will pick them up from school, for example, should you have to go to the doctor. Such conversations help kids relax and maintain their routines. ●

For more information, talk to your physician, or visit:

- the American Cancer Society Web site (www.cancer.org) and search for "support groups for children"
- the CancerCare Web site at www.cancercare.org/get_help/special_progs/cc_for_kids.php
- www.mdanderson.org.

July/August 2007

J. LeBas

©2007 The University of Texas
M. D. Anderson Cancer Center

Helping Physicians Help Patients

Some 7,000 physicians from around the world refer more than 13,000 patients to M. D. Anderson Cancer Center every year. With each of those referrals, important new relationships are created—and not only between the patient and the institution.

Just as important is the referring physician relationship, and it's one to which M. D. Anderson has committed an entire department: the Office of Physician Relations. The office's 32-member staff is dedicated to helping community physicians tap into the institution's resources, navigate the referral process, and stay in touch with their patients' progress.

"The majority of patients come to M. D. Anderson because their physicians referred them or told them to come," said Lyle D. Green, associate vice president for Physician Relations. "We're here to help those physicians in any way we can and to tell them about the treatments and programs that can benefit their patients."

One of the department's newest initiatives is *myMDAnderson*—a Web-based patient care tool that is rapidly gaining popularity. Using *myMDAnderson*, community physicians can make detailed referrals online, sending a patient's clinical and insurance information to the institution over a secure Internet connection. After a referral, *myMDAnderson* allows physicians to view patients' appointment schedules and reports and send messages to the M. D. Anderson clinical staff.

"Community physicians tell me that *myMDAnderson* is very useful, especially for making patient information available promptly. Now, most documents related to patient care can be accessed through the Web site," said Lewis Foxhall, M.D.,

vice president for health policy and co-medical director for Physician Relations. "We want to keep referring physicians informed about what's happening with their patients while they are treated here."

About 1,400 referring physicians are registered users of *myMDAnderson* (<https://my.mdanderson.org>). And while the hope is that this number will grow, the Web portal is not meant to replace person-to-person interaction. That's why Physician Relations has a team of specialists ready to answer phone calls and e-mails about how to make referrals, use *myMDAnderson*, and keep up with patients after the referral.

Perhaps you want to know about one of the more than 1,000 clinical trials at M. D. Anderson. Or maybe you need information about the institution's latest treatment facilities and initiatives or want to schedule a tour. Physician Relations can help with all of these.

Not all of the department's activities are performed in Houston. A team of six nurses makes 3,500 annual visits to physicians, bringing them information about M. D. Anderson programs. The staff also arranges off-site continuing medical education, guest lectures from M. D. Anderson faculty, and conference exhibits on cancer research, treatment, and patient care.

For more information about these and other resources:

- Call Physician Relations at 713-792-2202 or 1-800-252-0502
- E-mail physicianrelations@mdanderson.org
- Visit <http://www.mdanderson.org/departments/physrelations/>.

OncoLog

The University of Texas
M. D. Anderson Cancer Center

President

John Mendelsohn, M.D.

Provost and Executive Vice President

Raymond DuBois, M.D., Ph.D.

Senior Vice President for Academic Affairs

Stephen P. Tomasovic, Ph.D.

Director, Department of Scientific Publications

Walter J. Pagel

Contributing Editors

Melissa G. Burkett
Dawn Chalaire
Manny Gonzales
John LeBas
Lionel Santibañez
Angelique L. Siy
Ann M. Sutton
Vickie J. Williams
Dianne C. Witter

Design

The Very Idea®

Photography

Jim Lemoine

Editorial Board

Michael Fisch, M.D., Chair
Lyle Green, Vice Chair
Therese Bevers, M.D.
Elihu Estey, M.D.
Robert Gagel, M.D.
Beverly Handy, M.D.
Patrick Hwu, M.D.
Maurie Markman, M.D.
Shreyaskumar Patel, M.D.
David Schwartz, M.D.
Rena Sellin, M.D.
Randal Weber, M.D.
Christopher Wood, M.D.

OncoLog, M. D. Anderson Cancer Center's report to physicians, is a monthly newsletter sent to more than 30,000 physicians throughout the nation and the world. Published by the Department of Scientific Publications, *OncoLog* reports on innovative developments in research and treatment at M. D. Anderson. Current and previous issues are available online in English and Spanish at www2.mdanderson.org/depts/oncolog. For editorial information, call 713-792-3305 or email scientificpublications@mdanderson.org. To refer a patient or request information, call 1-877-632-6789 or 713-792-6161, or visit www.mdanderson.org.

Made possible in part by a gift from the late Mrs. Harry C. Wiess.