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Chapter 1 : What is Proton Therapy | MD Anderson Cancer Center

Proton therapy is a type of radiation therapy â€" a treatment that uses high-energy beams to treat tumors. Radiation therapy using X-rays has long been used to treat cancers and noncancerous (benign) tumors. Proton therapy is an established type of radiation therapy that uses energy from positively.

Description[edit] In a typical treatment plan for proton therapy, the spread out Bragg peak SOBP, dashed blue line is the therapeutic radiation distribution. The depth-dose plot of an X-ray beam red line is provided for comparison. The pink area represents additional doses of X-ray radiotherapyâ€"which can damage normal tissues and cause secondary cancers, especially of the skin. In proton therapy, medical personnel use a particle accelerator to target a tumor with a beam of protons. Cancerous cells are particularly vulnerable to attacks on DNA because of their high rate of division and their reduced abilities to repair DNA damage. Some cancers with specific defects in DNA repair may be more sensitive to proton radiation. All protons of a given energy have a certain penetration range ; very few protons penetrate beyond that distance. Accelerators used for proton therapy typically produce protons with energies in the range of 70 to MeV. Adjusting proton energy during the treatment maximizes the cell damage the proton beam causes within the tumor. Tissue closer to the surface of the body than the tumor receives reduced radiation, and therefore reduced damage. Tissues deeper in the body receive very few protons, so the dosage becomes immeasurably small. These Bragg peaks are shown as thin blue lines in the figure to the right. The total radiation dosage of the protons is called the spread-out Bragg peak SOBP , shown as a heavy dashed blue line in figure to the right. It is important to understand that, while tissues behind or deeper than the tumor receive almost no radiation from proton therapy, the tissues in front of shallower than the tumor receive radiation dosage based on the SOBP. Equipment[edit] Most installed proton therapy systems utilise isochronous cyclotrons. Over the next 41 years, this program refined and expanded these techniques while treating 9, patients [14] before the cyclotron was shut down in By these facilities were joined by an additional seven regional hospital-based proton therapy centers in the United States alone, and many more worldwide. Disease sites that respond well to higher doses of radiation, i. In some instances, dose escalation has demonstrated a higher probability of "cure" i. In all these cases proton therapy achieves significant improvements in the probability of local control over conventional radiotherapy. In these cases, the tumor dose is the same as in conventional therapy, so there is no expectation of an increased probability of curing the disease. Instead, the emphasis is on reducing the integral dose to normal tissue, thus reducing unwanted effects. Radiation-induced secondary malignancy is another very serious adverse effect that has been reported. As there is no exit dose when using proton radiation therapy, the dose to surrounding normal tissues can be significantly limited, reducing the acute toxicity which positively impacts the risk for these long-term side effects. Cancers requiring craniospinal irradiation, for example, benefit from the absence of exit dose with proton therapy: Some published studies found a reduction in long term rectal and genito-urinary damage when treating with protons rather than photons meaning X-ray or gamma ray therapy. Others showed a small difference, limited to cases where the prostate is particularly close to certain anatomical structures. Owing to this low energy requirement, some particle therapy centers only treat ocular tumors. Head and neck tumors[edit] Proton particles do not deposit exit dose, which allows proton therapy to spare normal tissues distal to the tumor target. This is particularly useful for treating head and neck tumors because of the anatomic constraints encountered in nearly all cancers in this region. The dosimetric advantage unique to proton therapy translates into toxicity reduction. For recurrent head and neck cancer requiring reirradiation, proton therapy is able to maximize a focused dose of radiation to the tumor while minimizing dose to surrounding tissues which results in a minimal acute toxicity profile, even in patients who have received multiple prior courses of radiotherapy. Unfortunately, treatment-related toxicities caused by chemotherapy agents and radiation exposure to healthy tissues are major concerns for lymphoma survivors. Advanced radiation therapy technologies such as proton therapy may offer significant and clinically relevant advantages such as sparing important organs at risk and decreasing the risk for late normal tissue damage while still achieving the primary goal of disease control. This is especially important for lymphoma

patients who are being treated with curative intent and have long life expectancies following therapy. The possibility of decreasing radiation dose to organs at risk may also help facilitate chemotherapy dose escalation or allow for new chemotherapy combinations. Proton therapy will play a decisive role in the context of ongoing intensified combined modality treatments for GI cancers. The following review presents the benefits of proton therapy in treating hepatocellular carcinoma, pancreatic cancer and esophageal cancer. Some overseas clinics providing proton beam therapy heavily market their services to parents who are understandably desperate to get treatment for their children. Proton beam therapy can be very costly and it is not clear whether all children treated privately abroad are treated appropriately. Megavoltage X-ray therapy has less "skin scarring potential" than proton therapy: X-ray radiation at the skin, and at very small depths, is lower than for proton therapy. The differences between the two methods depends on the: Width of the SOBP Depth of the tumor Number of beams that treat the tumor The X-ray advantage of reduced damage to skin at the entrance is partially counteracted by damage to skin at the exit point. Since X-ray treatments are usually done with multiple exposures from opposite sides, each section of skin is exposed to both entering and exiting X-rays. In proton therapy, skin exposure at the entrance point is higher, but tissues on the opposite side of the body to the tumor receive no radiation. Thus, X-ray therapy causes slightly less damage to the skin and surface tissues, and proton therapy causes less damage to deeper tissues in front of and beyond the target. Spot scanning can adjust the width of the SOBP on a spot-by-spot basis, which reduces the volume of normal healthy tissue inside the high dose region. Also, spot scanning allows for intensity modulated proton therapy IMPT , which determines individual spot intensities using an optimization algorithm that lets the user balance the competing goals of irradiating tumors while sparing normal tissue. Spot scanning availability depends on the machine and the institution. Surgery[edit] Physicians base the decision to use surgery or proton therapy or any radiation therapy on the tumor type, stage, and location. In some instances, surgery is superior such as cutaneous melanoma , in some instances radiation is superior such as skull base chondrosarcoma , and in some instances they are comparable for example, prostate cancer. In some instances, they are used together e. The benefit of external beam proton radiation lies in the dosimetric difference from external beam X-ray radiation and brachytherapy in cases where the use of radiation therapy is already indicated, rather than as a direct competition with surgery. Indeed, the largest study to date showed that IMRT compared with proton therapy was associated with less gastrointestinal morbidity. However the dose outside of the treatment region can be significantly less for deep-tissue tumors than X-ray therapy, because proton therapy takes full advantage of the Bragg peak. Proton therapy has been in use for over 40 years, and is a mature treatment technology. However, as with all medical knowledge, understanding of the interaction of radiation proton, X-ray, etc. An analysis published in determined the relative cost of proton therapy is approximately 2. An analysis published in determined that the cost of proton therapy is not unrealistic and should not be the reason for denying patients access to the technology. As of the end of more than , patients had been treated. Several industrial teams are working on development of comparatively small accelerator systems to deliver the proton therapy to patients.

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Chapter 2 : Bibliography - Book Chapters

The advantage of proton therapy (also called proton beam therapy) is that the physician can control where the proton releases the bulk of its cancer-fighting energy. As the protons move through the body, they slow down and interact with electrons, and release energy.

It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications. The literature through has been reviewed thoroughly for information relevant to air quality criteria, although the document is not intended as a complete and detailed review of all literature pertaining to lead. An attempt has been made to identify the major discrepancies in our current knowledge and understanding of the effects of these pollutants. Although this document is principally concerned with the health and welfare effects of lead, other scientific data are presented and evaluated in order to provide a better understanding of this pollutant in the environment. To this end, the document includes chapters that discuss the chemistry and physics of the pollutant; analytical techniques; sources, and types of emissions; environmental concentrations and exposure levels; atmospheric chemistry and dispersion modeling; effects on vegetation; and respiratory, physiological, toxicological, clinical, and epidemiological aspects of human exposure. Biological Effects of Lead Exposure Chapter Features of Lead Nephropathy Table Page Expected and observed deaths for malignant neoplasms Jan. Intramuscular method of injection i. Intraperitoneally method of injection i-v. Box University, AL Dr. The evaluations and conclusions contained herein, however, are not necessarily those of the reviewers. Lee Annest Division of Health Examin. Louis, MO Dr. Harry Roels Unite de Toxicologie Industrie! Risk Assessment Principal Authors Dr. Early chapters of this document Chapters discuss: Chapter 8 evaluates the projected impact of lead on ecosystems. Chapters , immediately proceeding this one, discuss: This chapter assesses information regarding biological effects of lead exposure, with emphasis on 1 the qualitative characterization of various lead-induced effects and 2 the delineation of dose-effect relationships for key effects most likely of health concern at ambient exposure levels presently encountered by the general population of the United States. In discussing biological effects of lead, one should note at the outset that, to date, lead has not been demonstrated to have any beneficial biological effect in humans. Some in- vestigators have hypothesized that lead may serve as an essential element in certain mammalian species e. However, a critical evaluation of these studies presented in Appendix A of this chapter raises serious questions regarding interpretation of the reported findings; and the subject studies are currently undergoing intensive evaluation by an expert committee con- vened by EPA. Therefore, pending the final report from that expert committee, the present chapter does not address the issue of potential essentiality of lead. It is clear from the evidence evaluated in this chapter that there exists a continuum of biological effects associated with lead across a broad range of exposure. At rather low levels of lead exposure, biochemical changes, e. With increasing lead exposure, there are sequentially more pronounced effects on heme synthesis and a broadening-of lead effects to additional biochemical and physiological mechanisms in various tissues, such that increasingly more severe disruption of the normal functioning of many dif- ferent organ systems becomes apparent. In addition to impairment of heme biosynthesis, signs of disruption of normal functioning of the erythropoietic and nervous systems are among the earliest effects observed in response to increasing lead exposure. At sufficiently high levels of exposure, the damage to the nervous system and other effects can be severe enough to result in death or, in some cases of non-fatal lead poisoning, long-lasting sequelae such as permanent mental retardation. The etiologies of many of the different types of functional disruption of various mamma- lian organ systems derive at least in their earliest stages from lead effects on certain subcellular organelles, which result in biochemical derangements e. Some major effects of lead on subcellular organelles common to numerous organ systems in mammalian spe- cies are discussed below in Section Subsequent sections of the chapter discuss biological effects of lead in terms of various organ systems affected by that element and its compounds except for Section Additional cellular and subcellular aspects of the biological effects of lead are discussed within respective sections on particular organ systems. Major emphasis is placed first on discusssion of the three

systems classically considered to be most sensitive to lead i. The next sections then discuss the effects of lead on reproduction and development in view of the impact of lead on the fetus and pregnant women , as well as gametotoxic effects of lead; Genotoxic effects of lead and evidence for possible carcinogenic effects of lead are then reviewed, followed by discussion of lead effects on the immune system and, lastly, other organ systems. This chapter is subdivided mainly according to organ systems to facilitate presentation of information. It must be noted, however, that, in reality, all systems function in delicate concert to preserve the physiological integrity of the whole organism and all systems are in- terdependent. Thus, not only do effects in a critical organ often exert impacts on other organ systems, but low-level effects that might be construed as unimportant in a single speci- fic system may be of concern in terms of their cumulative or aggregate impact. Special emphasis is placed on the discussion of lead exposure effects in children. They are particularly at risk due to sources of exposure, mode of entry, rate of absorption and re- tention, and partitioning of lead in soft and hard tissues. The greater sensitivity of children to lead toxicity, their inability to recognize symptoms, and their dependence on par- ents and health care professionals all make them an especially vulnerable population requiring special consideration in developing criteria and standards for lead. The relationship of this basis for lead toxicity to organ- and organelle-specific effects is modulated by: Given complexities introduced by factors 2 and 3, it is not surprising that no single, unifying mechanism of lead toxicity has been demonstrated to apply across all tissues and organ systems. In the Air Quality Criteria Document for Lead, cellular and subcellular effects of lead were discussed, including effects on various classes of enzymes. Much of the literature detailing the effects of lead on enzymes per se has entailed study of relatively pure enzymes vn vitro in the presence of added lead. This was the case for data discussed in the earlier document and such information continues to appear in the literature. Much of this material is of questionable relevance for effects of lead iji vivo. On the other hand, lead effects on certain enzymes or enzyme systems are recognized as integral mechanisms of action underlying the impact of lead on tissues HI vivo and are logically discussed in later sections below on effects at the organ system level. This subsection is mainly concerned with organellar effects of lead, especially those that provide some rationale for lead toxicity at higher levels of biological organization. While a common mechanism at the subcellular level that would account for all aspects of lead toxicity has not been identified, one fairly common cellular response to lead is the impair- ment of mitochondrial structure and function; thus, mitochondrion effects are accorded major attention here. Lead effects on other organelles have not been as extensively studied as mitochondrion effects; and, in some cases, it is not clear how the available information, e. Given early re- cognition of this sensitivity, it is not surprising that an extensive body of jjn vivo and jn vitro data has accumulated, which can be characterized as evidence of: Earlier studies have been reviewed by Goyer and Rhyne , followed by later updates by Fowler and Bull Chronic oral exposure of adult rats to lead 1 percent lead acetate in diet results in structural changes in renal tubule mitochondria, including swelling with distortion or loss of cristae Goyer, Such changes have also been documented in renal biopsy tissue of lead workers Wedeen et al. While it appears that relatively high level lead exposures are necessary to detect struc- tural changes in mitochondria in some" animal models Goyer, ; Hoffman et al. Also, in the study of Fowler et al. Taken collectively, these differences point out relative tissue sensitivity, dosing protocol, and the possible effect of developmental status Fowler et al. Both jji vivo and jjn vitro studies dealing with such effects of lead as the impact on energy metabolism, intermediary metabolism, and intracellular ion transport have been carried out in various experimental animal models. Of particular interest for this section are such effects of lead in the developing versus the adult animal, given the greater sensitivity of the young organism to lead. Uncoupled energy metabolism, inhibited cellular respiration using succinate and NAD-linked substrates, and altered kinetics of intracellular calcium have all been documented for animals exposed to lead jn vivo, as reviewed by Bull Succinate-mediated respiration in these animals, however, was not different from controls. In contrast, Fowler et al. This may have been due to longer exposure to lead or a differential effect of lead exposure during early development. This is consistent with the observation of Bouldin et al. Numerous studies have evaluated relative effects of lead on mitochonodria of developing vs. Holtzman and Shen Hsu exposed rat pups at 14 days of age to lead via milk of mothers fed lead in the diet 4 percent lead carbonate with exposure lasting for 14 days. A 40 percent increase in state 4

respiratory rate of cerebellar mitochonodria was seen within one day of treatment and was lost at the end of the exposure period. However, at this later time 28 days of age , a substantial inhibi- tion of state 3 respiration was observed. This early effect of lead on uncoupling oxidative phosphorylation is consistent with the results of Bull et al. In these investigations, female rats received lead in drinking water ppm from 14 days before breeding through weaning of the pups. At 15 days of age, cerebral cortical slices showed alteration of potassium-stimulated respiratory response and glucose uptake. Cerebellar mitochondria showed a very early loss by 2 days of exposure of respiratory control in the pups with inhibition of phosphorylation-coupled respiration for NAD-linked substrates but not for succinate. Such changes were less pronounced in mitochondria of the cerebrum and were not seen for either brain region in the adult rat. This regional and age dependency of mitochondrial impairment parallels features of lead encephalopathy. In a second study addressing this issue, Holtzman et al. These were compared to adult animals exposed in like fashion. Changes in cerebral cytochromes, in contrast, were marginal. This study indicates that the most vulnerable period for lead effects on developing brain oxidative metabolism is the same period where a major burst in such activity begins. Related to effects of lead on energy metabolism in the developing animal mitochondrion is the effect on brain development. In the study of Bull et al. Delays in synaptic development in these pups also occurred, as indexed by synaptic counts taken in the parietal cortex. As the authors con- cluded, uncoupling of energy metabolism appears to be causally related to delays in cerebral cortical development. Consistent with the effects of lead on mitochondria! Sabbioni and Marafante as well as Murakami and Hurosawa also found that lead is selectively lodged in mito- chondria. Of interest in regard to the effects of lead on brain mitochondria are the data of Moore et al. While the possibility of translocation of lead during subcellular fractionation can be raised, the distribution pattern seen in the reports of Barltrop et al. In vitro studies of both the response of mitochondrial function to lead and its accumulation by the organelle have been reported, using both isolated mito- chondria and tissues. Bull reviewed such data published up to Significant reductions in mitochondrial respiration, using both NAD-linked and succinate substrates have been reported for isolated heart and brain mitochondria. The lowest levels of lead associated with such effects appear to be 5 uM or, in some cases, less. If substrate specificity is compared, e. As Bull noted, tissue- specific effects of lead on cellular energetics may be one bases for differences in toxicity across organs. Similarly, lead uptake into brain mitochondria is also energy dependent Holtzman et al. The recent elegant studies of Pounds and coworkers Pounds et al. In the presence of graded amounts of lead 10, 50, or uM , the kinetic analysis of desaturation curves of calcium label showed a lead dose-dependent increase in the size of all three calcium compartments within the hepatocyte, particularly that deep com- partment associated with the mitochondrion Pounds et al. Such changes were seen to be relatively independent of serum calcium or endogenous regulators of systemic calcium meta- bolism. Similarly, the use of lead label and analogous kinetic analysis Pounds et al.

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Chapter 3 : Vander's Human Physiology - The Mechanisms of Body Function | Shafinewaz RPh - calendrie

Proton therapy is a type of external beam radiotherapy, and shares risks and side effects of other forms of radiation therapy. However the dose outside of the treatment region can be significantly less for deep-tissue tumors than X-ray therapy, because proton therapy takes full advantage of the Bragg peak.

Request an Appointment at Mayo Clinic Risks Proton therapy can cause side effects as the cancer cells die or when the energy from the proton beam damages healthy tissue. Because doctors can better control where proton therapy releases its highest concentration of energy, proton therapy is believed to affect less healthy tissue and have fewer side effects than traditional radiation therapy. Still, proton therapy does release some of its energy in healthy tissue. What side effects you experience will depend on what part of your body is being treated and the dose of proton therapy you receive. In general, common side effects of proton therapy include: Determining the best position for you during treatment. During radiation simulation, your radiation therapy team works to find a comfortable position for you during treatment. Cushions and restraints are used to place you in the correct position and to help you hold still. Your radiation therapy team will mark the area of your body that will receive the radiation. Depending on your situation, you may receive temporary marking with a marker or you may receive permanent tattoos. Planning the path of the protons with imaging tests. Your radiation therapy team may have you undergo magnetic resonance imaging MRI or computerized tomography CT scans to determine the area of your body to be treated and how best to reach it with the proton beams. Consider the cost Proton therapy is a newer form of radiation therapy that may be more expensive. Not all insurance policies cover proton therapy. What you can expect During proton therapy You typically undergo proton therapy five days a week for several weeks. However, in some cases, you may undergo only one or only a few treatments, depending on your condition. The actual proton therapy treatment may take only a minute or so, but expect to spend 30 to 45 minutes preparing before each treatment session. You may also undergo weekly CT verification scans to see if the dose you receive needs to be recalculated based on changes in weight, or tumor size and shape, depending on your situation. Cushions and restraints will be used to hold your body still. Your radiation therapy team will then leave the room and go to an area where they can monitor you. They can still see and hear you. What you experience next depends on the type of proton therapy machine your treatment team uses: A proton therapy machine that rotates around you. The machine rotates around you to direct proton beams at precise points on your body. The movement of your table during treatment is controlled remotely by your radiation therapy team. How often your table moves during treatment depends on your situation. After proton therapy Once your treatment session is complete, you can go about your day. Side effects of radiation usually develop over time. You may experience few side effects at first. But after several treatments you may experience fatigue, which can make it feel like your usual activities take more energy or that you have little energy for everyday tasks. You may also notice a sunburn-like skin redness in the area where the proton beams are directed. Results Your doctor may recommend periodic imaging tests during and after your proton therapy to determine whether your cancer is responding to the treatments. Clinical trials Explore Mayo Clinic studies testing new treatments, interventions and tests as a means to prevent, detect, treat or manage this disease. See the stories of satisfied Mayo Clinic patients.

Chapter 4 : Proton therapy - Mayo Clinic

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Contact Us Proton Therapy Clinical Trials Clinical trials are research studies in which patients may volunteer to take part. Doctors use treatment trials to learn more about how to fight cancer. Clinical trials are part of a long, careful process, which may take many years. First, doctors study a new treatment in the lab. Then they often study the treatment in animals. If a new treatment shows promise, doctors then test the treatment in people. Doctors do this in three to four steps, or phases. Your doctor may offer you a clinical trial as a treatment option. The current Proton Therapy Center clinical trials are listed below. For more information on clinical trials and how to join one, call toll-free: Brain tumors NCT - Pilot Trial of Dose-Volume Constraints for Reirradiation of Recurrent Brain TumorsThe goal of this clinical research study is to test the safety of repeat radiation for brain tumors that came back after the first course of radiation. Researchers also want to learn how repeat radiation affects your quality of life. Radiation therapy uses high-energy x-rays and other types of radiation to kill tumor cells and shrink tumors. Specialized radiation therapy that delivers a high dose of radiation directly to the tumor may kill more tumor cells and cause less damage to normal tissue. Drugs, such as temozolomide, may make tumor cells more sensitive to radiation therapy. It is not yet known whether dose-escalated photon IMRT or proton beam radiation therapy is more effective than standard-dose radiation therapy with temozolomide in treating glioblastoma. Both groups will receive usual chemotherapy, temozolomide. The higher radiotherapy dose could shrink your cancer, but it could also cause side effects. This study will allow the researchers to know whether this higher dose is better, the same, or worse than the usual approach. To be better, the study should increase life by six months or more compared to the usual approach. Two methods of giving radiation therapy will also be compared. They are proton beam radiation and intensity-modulated radiation. Proton beam radiation therapy uses tiny charged particles to deliver radiation directly to the tumor and may cause less damage to normal tissue. Intensity-modulated or photon beam radiation therapy uses high-energy x-ray beams shaped to treat the tumor and may also cause less damage to normal tissue. Patients will be more likely to be randomized to proton beam radiation therapy. It is not yet known if proton beam radiation therapy is more effective than photon-based beam intensity-modulated radiation therapy in treating patients with glioma. Participants will receive chemotherapy along with radiation therapy. IMPT is designed to use beams of proton particles to send radiation to the tumor. IMRT is designed to use beams of photon therapy to send radiation to the tumor. Both of these types of radiation treatment may give a full dose of radiation treatment to the tumor while not damaging as much of the healthy tissue around it. PBT and IMRT are both forms of radiation therapy that are designed to treat a specific area of the body while affecting as little of the surrounding normal tissue as possible. PBT is a newer technology that is designed to further reduce the amount of radiation that affects the surrounding normal tissue. However, this is still being studied. Radiation therapy, such as photon therapy, uses high energy x-rays to send the radiation inside the body to the tumor while proton therapy uses a beam of proton particles. Proton therapy can stop shortly after penetrating through the tumor and may cause less damage to the surrounding healthy organs and result in better survival in patients with liver cancer. Participants will also receive standard chemotherapy. IMPT therapy and IMRT therapy are types of radiation therapy that are designed to use a beam of proton or photon particles similar to getting an x-ray to send radiation inside the body to the tumor. Specialized radiation therapy that delivers a high dose of radiation directly to the tumor, such as photon or proton beam radiation therapy, may kill more tumor cells and cause less damage to normal tissue. Drugs used in chemotherapy, such as paclitaxel, carboplatin, etoposide, and cisplatin, work in different ways to stop the growth of tumor cells, either by killing the cells or by stopping them from dividing. It is not yet known whether proton chemoradiotherapy is more effective than photon chemoradiotherapy in treating non-small cell lung cancer. Pediatric cancer NCT - Registry for Pedi Patients Treated With Proton RT In previous studies, Proton Beam Radiation Therapy PBRT has been found to show better results in treating patients with cancer, both because

there is better control of where in the body the radiation is directed and because it is associated with less severe long term side effects. However, there is limited published data demonstrating these results. The goal of the Pediatric Proton Consortium Registry PPCR is to enroll children treated with proton radiation in the United States in order to describe the population that currently receives protons and better evaluate its benefits over other therapies. The data collected from this study will help facilitate research on proton beam radiation therapy and allow for collaborative research. The PPCR will collect demographic and clinical data that many centers that deliver proton radiation therapy already collect in routine operations. Prostate cancer NCT - Hypofractionated Proton Beam Therapy for Localized Prostate Cancer The goal of this clinical research study is to learn if delivering proton therapy in higher doses per treatment may help control prostate cancer just as well as the standard of care treatment. The safety of this treatment will also be studied.

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Chapter 5 : Proton therapy - Wikipedia

Proton therapy, also called proton beam therapy, is a type of radiation therapy. It uses protons rather than x-rays to treat cancer.A proton is a positively charged particle. At high energy, protons can destroy cancer cells.

The field of radiation oncology has made significant strides since practitioners began using photon radiation therapy around the turn of the 20th century. Not until the s, though, did researchers begin to recognize the full potential of isolating protons for the treatment of medical conditions. Advanced theoretical understanding of particle acceleration, proton beams, and their applied use in radiation treatment has improved outcomes for patients of many types of cancers. This initial research was focused not on the treatment of human malignancies, but on the discovery and exploration of particles and nuclear physics. In , a Harvard particle physicist, Robert R. Wilson, proposed using proton beams for medical application. In the years following the initial human treatments, more cyclotrons were built to establish proton therapy programs across the United States â€" Harvard University in , the University of California, Davis in , and the Los Alamos National Laboratory in The research from these facilities, coupled with the technological advances in photon radiation therapy and its delivery methods, have advanced proton therapy to its current position in the field of cancer treatment today. Proton therapy received U. The first hospital-based proton therapy treatment center opened in California in â€" operating a modified, more efficient accelerator, called a synchrotron. By , it is estimated that between , and , patients will have received proton therapy treatment. In the United States, there are 28 active proton centers and 11 centers under construction or in the initial development stages. Some hospitals and oncology treatment centers have struggled to fund the cost of sophisticated particle accelerators, while others have grappled with how to find or construct the space required to house them. Fortunately, a more sophisticated approach to building proton therapy treatment centers has emerged. And facilities considering the incorporation of proton therapy into their treatment options are finding real benefit from pursuing flexible, more affordable synchrotrons. Traditional barriers to providing patient access to proton therapies â€" cost and space â€" are both addressed by modern methods of modular construction and affordable treatment delivery systems. Today, more than half of all cancer patients receive radiation therapy as part of their treatment plan. As stakeholders continue to refine and improve the delivery and administration of proton therapies, it is set to emerge as a primary radiation treatment option, particularly for inoperable, pediatric, and well-defined cancers. Further, as vanguard treatment systems address historic barriers to entry, more hospitals, and medical facilities will make strategic investments in proton therapies. Learn more about proton therapy. Download our free white paper.

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Chapter 6 : Proton Therapy Clinical Trials | MD Anderson Cancer Center

Proton therapy is an advanced form of radiation therapy that uses a single beam of high-energy protons to treat various forms of cancer. Just as with conventional radiation therapy, proton therapy treats tumors by directing radiation into the tumor site where doses of radiation destroy cancerous cells.

Standard radiation therapy has evolved and improved over the years and is effective in controlling many cancers. However, because X-ray beams are composed of primary photons and secondary electrons, they deposit their energy along the path of the beam, to the targeted tumor and beyond, and deliver radiation to healthy tissues before and after the tumor site. What is Proton Therapy? See full-size video The advantage of proton therapy also called proton beam therapy is that the physician can control where the proton releases the bulk of its cancer-fighting energy. As the protons move through the body, they slow down and interact with electrons, and release energy. A proton beam conforms to the shape and depth of a tumor, while sparing healthy tissues and organs. How does it work? The best way to understand how proton therapy works is to take a look at the physics and engineering inside the proton accelerator, or the synchrotron, and the beam delivery system. The proton begins its journey at the ion source. Within fractions of a second, hydrogen atoms are separated into negatively charged electrons and positively charged protons. After leaving the synchrotron, the protons move through a beam-transport system comprised of a series of magnets that shape, focus and direct the proton beam to the appropriate treatment room. To ensure that each patient receives the prescribed treatment safely and efficiently, the facility is controlled by a network of computers and safety systems. The gantry can revolve degrees, allowing the beam to be delivered at any angle. As protons come through the nozzle, a custom-made device the aperture shapes the beam of protons, and another custom-made device the compensator shapes the protons into three dimensions, delivering them to the depth of the tumor. At maximum energy, a proton beam travels , miles per second, which is equivalent to the two-thirds the speed of light. From the hydrogen canister to the patient, a proton typically travels , miles. Pencil beam and intensity modulated proton therapy The team at MD Anderson Proton Therapy Center continues to expand ways to use proton therapy to benefit patients. We are one of the few centers worldwide offering these types of proton therapy to our patients. Pencil beam technology and IMPT build on the benefits of proton therapy. Pencil beam is very effective in treating the most complex tumors, like those in the prostate, brain, eye, and cancers in children, while leaving healthy tissue and other critical areas unharmed. IMPT is best used to deliver a potent and precise dose of protons to complex or concave-shaped tumors that may be adjacent to the spinal cord or embedded head and neck or skull base, including nasal and sinus cavities; oral cavity; salivary gland; tongue; tonsils; and larynx. Proton therapy benefits patients whose tumors are solid with defined borders, meaning the cancer has not spread to other parts of the body. It does not require surgery, making it ideal for inoperable tumors.

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Chapter 7 : Air Quality Criteria for Lead: Volume IV of IV (Review Draft)

Proton therapy has become the most precise and advanced form of radiation treatment. The field of radiation oncology has made significant strides since practitioners began using photon radiation therapy around the turn of the 20th century.

Neuroendocrine Tumors of the GI Tract: Bronchopulmonary Carcinoid Tumors in Oberndorfer Centennial. Modlin IM, Oberg K, eds p Overview of therapeutic options for Gastrointestinal carcinoids. The History of Endoscopy: Thieme, Stuttgart, Germany Blackwell, Berlin, Germany, Publisher, Ontario Canada, Blackwell, Berlin, Germany Activation of muscarinic receptors on the rat pancreatic acinar cell is associated with cell injury. Perspectives on Stem Cells and Gut Growth: Follow-up for Cancer 4th Ed. Fischer, Lippincott - Raven, New York ; pp Five Years of Laparoscopic Cholecystectomy: Basel, Karger, , vol. A Practical Approach to Cloning Strategies. Clinical Gene Analysis and Manipulation: From Prout to the Proton Pump. Schnetztor-Verlag GmbH, Konstanz, Eds Frontiers of Gastrointestinal Research Series. Endocrine Tumors of the Pancreas Current State of the Art. Clinical Gastroenterology ed H. Spiro, McGraw-Hill, Supp 1; pp , Approaches to Pancreatic Disease. In Current Practice of Surgery. Esophageal, gastric and small intestine editorial overview. Current Opinion in General Surgery Clinical Applications of Gastrointestinal Hormones. Overview of gastric mucosal injury and inflammation: Rothenberger DA Ed Lippincott ; 9 4: Yale J Biol and Med ; 65 5: Cellular mechanisms of acute pancreatitis. Perspectives in General Surgery. Pathobiology of enterochromaffin-like cell tumor induction in Mastomys. Clinical and research significance: Karger, Basel ; Hormones, neurotransmitters and other humoral markers of endocrine tumors. In Friesen Sr ed: JP Lippincott ; Tumor of the Endocrine Glands [Carcinoid Tumors]. Current Guidelines for the Management of Cancer. Khafif, Sameer Rafla, Samuel Kopel eds. Peptic ulcer syndromes - hyper and hyposecretory states: In Surg Care II. Alfonso A and Gardner B eds. Appleton Century-Crofts, New York, Endocrine Society of America. The systemic release and pharmacokinetics of VIP. Churchill Livingstone, ; Gastrointestinal Hormones and Pathology of the Digestive System. Grossman and Speranza V eds Plenum Press ;

Chapter 8 : Carbonic anhydrase | Revolvy

Proton therapy is an ideal head and neck cancer treatment because, just as with brain cancers, it is important to only treat the cancerous areas and avoid secondhand radiation to healthy organs.

The gamma class of CAs come from methanogens , methane-producing bacteria that grow in hot springs. The distinction of this class of CA has recently[12] come into question, however. Thus, the two forms may be distantly related, even though the underlying amino acid sequence has since diverged considerably. The primary function of the enzyme in animals is to interconvert carbon dioxide and bicarbonate to maintain acid-base balance in blood and other tissues, and to help transport carbon dioxide out of tissues. There are at least 14 different isoforms in mammals. In plants, carbonic anhydrase helps raise the concentration of CO within the chloroplast in order to increase the carboxylation rate of the enzyme RuBisCO. This is the reaction that integrates CO into organic carbon sugars during photosynthesis , and can use only the CO form of carbon, not carbonic acid or bicarbonate. However, this species of phytoplankton appears to have adapted to the low levels of zinc in the ocean by using cadmium when there is not enough zinc. This type of carbonic anhydrase is therefore cambialistic, meaning it can interchange the metal in its active site with other metals namely, zinc and cadmium. CDCA also has a three-dimensional folding structure that is unlike any other carbonic anhydrase, and its amino acid sequence is dissimilar to the other carbonic anhydrases. The active site of CDCA is essentially "gated" by a chain of nine amino acids with glycine residues at positions 1 and 9. Normally, this gate remains closed and the cadmium ion is trapped inside. However, due to the flexibility and position of the glycine residues, this gate can be opened in order to remove the cadmium ion. A zinc ion can then be put in its place and the gate will close behind it. The metal in the active site can be switched between zinc and cadmium depending on which one is more abundant at the time. Studies have shown that T. The toxicity of the metal is reduced by the transcription and translation of phytochelatin , which are proteins that can bind and transport cadmium. Once bound by phytochelatin, cadmium is no longer toxic, and it can be safely transported to the CDCA enzyme. In all species tested, CDCA-like proteins showed high levels of expression even in high concentrations of zinc and in the absence of cadmium. The biggest challenge engineers face implementing carbonic anhydrase for this use is the harsh conditions of the flue exhaust streams. CA was placed in a N-methyldiethanolamine MDEA solution where it served to increase the concentration difference driving force of CO between the flue stream of the power plant and liquid phase in a liquid-gas contactor. Carbonic acid has a pK of around 6.

Chapter 9 : How Proton Treatment Works

In recent years the use of proton beam therapy (PBT) has expanded to include a variety of conditions including a number of cancer types, noncancerous brain tumors and cancerous conditions afflicting the central nervous system as well as eyes, lungs, liver, prostate, spine, and pelvis.

If given in sufficient doses, x-ray radiation techniques will control many cancers. Consequently, a less-than-desired dose is frequently used to reduce damage to healthy tissues and avoid unwanted side effects. The power of protons is that higher doses of radiation can be used to control and manage cancer while significantly reducing damage to healthy tissue and vital organs. Understanding how protons work provides patients and physicians with an insight into this mainstream treatment modality. Essentially, protons are a superior form of radiation therapy. Fundamentally, all tissues are made up of molecules with atoms as their building blocks. In the center of every atom is the nucleus. Orbiting the nucleus of the atom are negatively charged electrons. When energized charged particles, such as protons or other forms of radiation, pass near orbiting electrons, the positive charge of the protons attracts the negatively charged electrons, pulling them out of their orbits. This is called ionization; it changes the characteristics of the atom and consequentially the character of the molecule within which the atom resides. This crucial change is the basis for the beneficial aspects of all forms of radiation therapy. Because of ionization, the radiation damages molecules within the cells, especially the DNA or genetic material. Damaging the DNA destroys specific cell functions, particularly the ability to divide or proliferate. Enzymes develop with the cells and attempt to rebuild the injured areas of the DNA; however, if damage from the radiation is too extensive, the enzymes fail to adequately repair the injury. As a result, cancer cells sustain more permanent damage and subsequent cell death than occurs in the normal cell population. This permits selective destruction of bad cells growing among good cells. Both standard x-ray therapy and proton beams work on the principle of selective cell destruction. The major advantage of proton treatment over conventional radiation, however, is that the energy distribution of protons can be directed and deposited in tissue volumes designated by the physicians-in a three-dimensional pattern from each beam used. This capability provides greater control and precision and, therefore, superior management of treatment. Radiation therapy requires that conventional x-rays be delivered into the body in total doses sufficient to assure that enough ionization events occur to damage all the cancer cells. This undesirable pattern of energy placement can result in unnecessary damage to healthy tissues, often preventing physicians from using sufficient radiation to control the cancer. Protons, on the other hand, are energized to specific velocities. These energies determine how deeply in the body protons will deposit their maximum energy. As the protons move through the body, they slow down, causing increased interaction with orbiting electrons. Maximum interaction with electrons occurs as the protons approach their targeted stopping point. Thus, maximum energy is released within the designated cancer volume. The surrounding healthy cells receive significantly less injury than the cells in the designated volume. This allows the dose to be increased beyond that which less-conformal radiation will allow. The overall affects lead to the potential for fewer harmful side effects, more direct impact on the tumor, and increased tumor control. The minimized normal-tissue injury results in the potential for fewer effects following treatment, such as nausea, vomiting, or diarrhea. The patients experiences a better quality of life during and after proton treatment.