INTERMEDIATE PHASE OF TREATMENT pdf

1: Meniscus Tear Treatment and Rehabilitation â€” Healing Through Movement

The early phase of damage usually occurs within 24 h of injury and is directly related to tissue damage and deregulated physiological functions, the intermediate phase takes place in the days following TBI and entails neuroinflammation, and the late phase is primarily associated with seizures and epileptogenesis and arises days to weeks after TBI.

Treatment of most patients with acute myeloid leukemia AML is typically divided into 2 chemotherapy chemo phases: Remission induction often just called induction Consolidation post-remission therapy The acute promyelocytic leukemia APL subtype of AML is treated differently. Treatment for AML usually needs to start as quickly as possible after it is diagnosed because it can progress very quickly. Sometimes another type of treatment needs to be started even before the chemo has had a chance to work. Treating leukostasis Some people with AML have very high numbers of leukemia cells in their blood when they are first diagnosed, which can cause problems with normal blood circulation. This is called leukostasis. Chemo can take a few days to lower the number of leukemia cells in the blood. In the meantime, leukapheresis sometimes just called pheresis might be used before chemo. Two intravenous IV lines are required â€” the blood is removed through one IV, goes through the machine, and then is returned to the patient through the other IV. Sometimes, a single large catheter is placed in a vein in the neck or under the collar bone for the pheresis, instead of using IV lines in both arms. This type of catheter is called a central venous catheter CVC or central line and has both IVs built in. This treatment lowers blood counts right away. The effect is only for a short time, but it may help until the chemo has a chance to work. Induction This first phase of treatment is aimed at quickly getting rid of as many leukemia cells as possible. Doctors often give the most intensive chemo to people under the age of 60, but some older patients in good health may benefit from similar or slightly less intensive treatment. People who are much older or are in poor health might not do well with intensive chemo. Treatment for these patients is discussed below. Age, health, and other factors clearly need to be taken into account when considering treatment options. For example, people whose leukemia cells have certain gene or chromosome changes are more likely to benefit from certain types of treatment. In younger patients, such as those under 60, induction often involves treatment with 2 chemo drugs: In some situations, a third drug might be added as well to try to improve the chances of remission: For patients whose leukemia cells have an FLT3 gene mutation, the targeted therapy drug midostaurin Rydapt might be given along with chemo. This drug is taken twice daily as a pill. For patients whose leukemia cells have the CD33 protein, the targeted drug gemtuzumab ozogamicin Mylotarg might be added to chemo. Adding the chemo drug cladribine might be another option for some people. Patients with poor heart function might not be able to be treated with anthracyclines, so they may be treated with another chemo drug, such as fludarabine Fludara or etoposide. In rare cases where the leukemia has spread to the brain or spinal cord, chemo may also be given into the cerebrospinal fluid CSF. Radiation therapy might be used as well. Patients typically need to stay in the hospital during induction and possibly for some time afterward. Induction destroys most of the normal bone marrow cells as well as the leukemia cells, so most patients develop dangerously low blood counts, and may be very ill. Most patients need antibiotics and blood product transfusions. Drugs to raise white blood cell counts called growth factors may also be used. Blood counts tend to stay low for a few weeks. About a week after chemo is done, the doctor will do a bone marrow biopsy. Most people with leukemia go into remission after the first round of chemo. But if the biopsy shows that there are still leukemia cells in the bone marrow, another round of chemo may be given, either with the same drugs or with another regimen. Sometimes a stem cell transplant is recommended at this point. Over the next few weeks, normal bone marrow cells will return and start making new blood cells. The doctor may do other bone marrow biopsies during this time. When the blood cell counts recover, the doctor will again check cells in a bone marrow sample to see if the leukemia is in remission. Remission induction usually does not destroy all the leukemia cells, and a small number often remain. Without post-remission therapy consolidation , the leukemia is likely to return within several months. Consolidation post-remission therapy Induction is considered successful if remission is achieved. Further treatment called consolidation is then given to try to destroy any remaining leukemia cells and help prevent a relapse. Consolidation for younger

patients For younger patients typically those under 60 , the main options for consolidation therapy are: Several cycles of chemo with high-dose cytarabine ara-C sometimes known as HiDAC Allogeneic donor stem cell transplant Autologous stem cell transplant The best option for each person depends on the risk of the leukemia coming back after treatment, as well as other factors. For HiDAC, cytarabine is given at very high doses, typically over 5 days. This is repeated about every 4 weeks, usually for a total of 3 or 4 cycles. For people who got the targeted drug midostaurin Rydapt during induction, this is typically continued during consolidation. Again, each round of treatment is typically given in the hospital because of the risk of serious side effects. For patients who got chemo plus the targeted drug gemtuzumab ozogamicin Mylotarg for their induction therapy, a similar regimen might be used for consolidation. Stem cell transplants have been found to reduce the risk of leukemia coming back more than standard chemo, but they are also more likely to have serious complications, including an increased risk of death from treatment. Consolidation for patients who are older or have other health problems Older patients or those in poor health may not be able to tolerate intensive consolidation treatment. Often, giving them more intensive therapy raises the risk of serious side effects including treatment-related death without providing much more of a benefit. These patients may be treated with: Higher-dose cytarabine usually not quite as high as in younger patients Standard-dose cytarabine, possibly along with idarubicin, daunorubicin, or mitoxantrone For people who got the targeted drug midostaurin Rydapt during induction, this is typically continued during consolidation as well. Each has pros and cons. Doctors look at several factors when recommending what type of therapy a patient should get. How many courses cycles of chemo it took to bring about a remission. If it took more than one, some doctors recommend that the patient get a more intensive program, which might include a stem cell transplant. If a close enough tissue match is found, an allogeneic donor stem cell transplant may be an option, especially for younger patients. The possibility of collecting leukemia-free bone marrow cells from the patient. Stem cells collected from the patient would be purged treated in the lab to try to remove or kill any remaining leukemia cells to lower the chances of relapse. The presence of one or more adverse prognostic factors , such as certain gene or chromosome changes, a very high initial white blood cell count, AML that develops from a previous blood disorder or after treatment for an earlier cancer, or spread of AML to the central nervous system. These factors might lead doctors to recommend more aggressive therapy, such as a stem cell transplant. On the other hand, for people with good prognostic factors, such as favorable gene or chromosome changes, many doctors might advise holding off on a stem cell transplant unless the disease recurs. Older patients or othose with other health problems might not be able to tolerate some of the severe side effects that can occur with high-dose chemo or stem cell transplants. An important issue is the higher chance of death from high-dose chemo or a stem cell transplant. This and other issues must be discussed between the patient and the doctor. Stem cell transplants are intensive treatments with real risks of serious complications, including death, and their exact role in treating AML is not always clear. Some doctors feel that if the patient is healthy enough to withstand an allogeneic transplant and a compatible donor is available, this option offers the best chance for long-term survival. Others feel that studies have not yet shown this conclusively, and that in some cases a transplant should be reserved in case the leukemia comes back after standard treatment. Still others feel that stem cell transplants should be given if the leukemia is likely to come back based on certain gene or chromosome changes. Research in this area continues to study which AML patients get the most benefit from stem cell transplant and which type oftransplant is best in each situation. Treating frail, older adults Treatment of AML in people under 60 is fairly standard. It involves cycles of intensive chemo, sometimes along with a stem cell transplant as discussed above. Many patients older than 60 are healthy enough to be treated in the same way, although sometimes the chemo may be less intense. People who are much older or are in poor health may not be able to tolerate this intense treatment. In fact, intense chemo could actually shorten their lives. Treatment of these patients is often not divided into induction and consolidation phases, but it may be given every so often as long as it seems helpful. In some cases, doctors may recommend low-intensity chemo with a low dose of cytarabine given in cycles. Sometimes, these patients may be treated with other chemo drugs like azacitidine Vidaza or decitabine Dacogen. In some cases, this may induce remission. In others, it may control the leukemia for a time. Another option might be the targeted drug gemtuzumab ozogamicin Mylotarg. Some

people might decide against chemo and other drugs and instead choose supportive care. This focuses on treating any symptoms or complications that arise and keeping the person as comfortable as possible.

INTERMEDIATE PHASE OF TREATMENT pdf

2: Intermediate-Risk Prostate Cancer Varies in Prognosis - Renal and Urology News

In my last post, Ankle Sprains - Acute Phase (Part I of III), I addressed how to handle the initial acute phase of an ankle sprain. I will continue to guide you through the treatment plan on how to rehabilitate your ankle in this three part series by addressing the progression from the acute phase into the intermediate phase.

July 29, Welcome to this article on meniscus tear. Meniscus Tear Treatment and Rehabilitation There are certain factors that should be considered when creating your meniscus treatment plan. Your physician will base your treatment and rehabilitation plan on the type of meniscal tear that you have, size of the tear, area of the meniscus involved, your age, your level of activity, and other related injuries. Degree of healing is highly dependent on the location of your meniscus tear. Recall that not all areas of a meniscus receive adequate supply of blood, which carries the nutrients required for tissue healing. The outer one-third of the meniscus is sufficiently supplied with blood. In contrast, the inner two-thirds of the meniscus is poorly supplied with blood. A tear in this part of the meniscus may not be treated conservatively. A surgical procedure may be recommended. Conservative treatment If your meniscus tear is small and found on the outer region of the meniscus, a conservative treatment is most likely to be recommended by your physician. The RICE protocol aims to minimize the swelling, reduce the pain, normalize the gait, encourage normal range of motion of the knee, prevent muscle wasting, and maintain proprioception Baker, Adherence to this treatment plan is a must to ensure adequate healing of the damaged meniscus: The pain has a function. It informs your body to rest and stop performing the activities that may aggravate the condition. Cold constricts the blood vessels, leading to reduced swelling and redness. Ice may also numb the nerves around the area of the injury, decreasing the pain. It is recommended to apply ice 15 to 20 minutes at a time, several times a day, during the first 24 to 48 hours after the injury. Wrap the ice in a towel to decrease the risk of causing contact burns on the skin. Application of heat is only recommended 48 hours after the injury. Heat may exacerbate the condition by increasing joint swelling and pain. Using the correct compression technique reduces swelling and provides support and protection for the injured knee. Medication for a Meniscus Tear Intake of certain medications can also help reduce pain during the recovery stage. You may take analgesics like acetaminophen and non-steroidal anti-inflammatory drugs, such as ibuprofen, naproxen, diclofenac, and celecoxib, to control minimal to moderate pain. Your physician may prescribe oral or intramuscular narcotic analgesics if the pain becomes intolerable or if you fail to respond to analgesics and NSAIDs. These drugs are only used sparingly during the conservative treatment period. Surgical Procedures for a Meniscus Tear If the signs and symptoms of meniscus injury are not resolved by conservative management, your physician may recommend a surgical procedure to manage the injury. A surgical intervention may also be recommended if the injury causes locked knee. The meniscus has essential functions, and its removal is not the first choice when surgery is being considered. Not all tears respond to surgery. Tears with good chances of healing with surgery are repaired through arthroscopy. Arthroscopy, a minimally invasive procedure, is the standard of surgical care. Compared to partial removal of the meniscus, arthroscopy offers better long-term outcomes. Partial meniscectomy may be recommended if your tear occurs in the region of the meniscus where the supply of blood is not adequate. This procedure is also recommended if your tear is not amenable to repair through arthroscopy. Exercises for Meniscus Injuries Exercises are vital when treating, recovering, and preventing meniscal injuries. It is highly recommended that you have an effective rehabilitation program that will help you regain and maintain strength and flexibility of your knee, like Meniscus Tear Solution. Before exercising, it is important to use the right footwear and clothes. It is also essential to perform warm up exercises before the exercise session and cool down exercises after the exercises to prevent further injuries or aggravating the condition. Exercises During the Acute Meniscus Tear Stage During the acute stage or initial phase of rehabilitation, complete immobilization of the knee is discouraged. Ankle pumps and gentle range of motion exercises of the knee are first recommended to facilitate adequate flow of blood, which facilitates healing. These simple and non-stressful exercises may also prevent complications, such as stiffening of the knee. After 48 to 72 hours of the injury or surgery, you may perform quadriceps sets, straight leg raises, and hamstring sets. These isometric

or static exercises help prevent muscle wasting and loss of strength and function of the affected knee. Overall, the initial stage includes exercises that aim to overcome limitations to range of motion. Flexibility exercises for the lower extremities, including the quadriceps, hamstrings, hip flexors, hip adductors, and calf muscles, must be initiated. Passive flexion ROM exercises, such as wall slides, are also included. These exercises are also recommended as you progress: Isotonic strengthening exercises of the hamstring muscles Hip abduction strengthening Toe raises Stationary bicycling Keep in mind to limit your activities during the acute stage. Avoid, limit, or modify squatting, kneeling, heavy lifting, climbing, and running. Exercises During Intermediate Meniscus Tear Phase Exercises recommended during the intermediate phase are only initiated if you have achieved full or near full range of motion. Flexibility and strengthening exercises performed during the initial phase are continued with added resistance, as tolerated. It is also during this stage where you can start performing isokinetic strength exercises and endurance training. Closed kinetic chain exercises may be initiated as soon as the quadriceps is strong enough to resist the load. Running in place on a trampoline is gradually introduced into the program. Jogging for 10 to 15 minutes can be started, once the knee demonstrates absence of swelling and pain. Exercises During Advanced Meniscus Tear Phase As you make your progress, your physical therapist may order the initiation of strength-training exercises. Sport-related activities can be started. Exercises for the Prevention of a Meniscus Injury Exercises that build strength and flexibility of your knee and leg are effective in preventing future injuries. After achieving recovery, the exercises discussed previously must be continued to avoid or delay degeneration of the knee. If you have a meniscus injury or were recently diagnosed with this condition, twisting of the knee and walking on an uneven train is discouraged. Wear the right footwear and learn the right techniques in cutting, slowing down, pivoting, and landing from a jump. Thank you for reading. If you would like to see the exercise program that I use for meniscus tears, you can check it out here:

INTERMEDIATE PHASE OF TREATMENT pdf

3: Neuroblastoma - Wikipedia

The vapor optimization time is shortened to several seconds, and the intermediate phase forms on the surface layer of PbI2 films. We achieve porous PbI2 surface with smaller grains through dimethyl sulfoxide vapor treatment, which promotes the migration and reaction rate between CH3NH3I vapor and PbI2 layer.

Share Tweet Share print email Ankle sprains are one of the most common and prevalent musculoskeletal injuries. Although more likely to occur in children, ankle sprains can happen to anyone anytime. I will continue to guide you through the treatment plan on how to rehabilitate your ankle in this three part series by addressing the progression from the acute phase into the intermediate phase. A Grade I sprain is the most common. A Grade II sprain is a partial tear to the ligament and is usually associated with some laxity hypermobility. Good muscle strength and proprioception of the lower foot is important to limit future sprains. In Grade III sprains, a full tear of the ligament occurred. One typically consults with an orthopaedic surgeon for possible repair. After surgery, a guided physical therapy program is recommended. For discussion purposes, I will only address a Grade I sprain. Initially, one may wear an air splint, ACE wrap, or some other lace-up or slip-on style brace to help with stability, inflammation, and pain control of the ankle. In most cases, a person will want to transition from wearing the brace as soon as the initial pain subsides. At this point in your recovery, you are likely three to seven days since the initial injury. This phase of rehabilitation can last from seven days to several weeks before progressing into the final phase of rehabilitation and ultimately, back to full function. Progression out of the intermediate phase is always symptom dependent. You should be able to stand with equal weight on your feet and not experience an increase in ankle pain. The ankle is likely stiff at this time, but it is time to start walking, progress range of motion ROM , and start gentle resistive exercises. Walking â€" If you have been using a crutch to unweight the foot, then start the progression to weight bearing during walking. If you have been walking, then increase the amount of weight you have been putting on the ankle and foot. At this time, the focus will be to normalize your walking pattern. This means having a good heel strike, rolling onto the foot into full weight bearing on the leg, and then propelling forward with a good toe off. You will continue to use the crutch as long as needed until you can walk nearly normal without limping. Until then, utilize the crutch to unweight the leg and foot as much as necessary to perform a nearly normal walk or gait sequence. Initially, work to progress the plantarflexion and dorsiflexion movement the forward and backward movement of the ankle. As pain subsides, progress the side to side motion as well as all other motions. Ankle Pumps â€" A very easy exercise. Just pump your ankle forward and backward into plantarflexion and dorsiflexion movement. Perform repetitions several times a day on both feet Ankle Alphabet â€" Move the foot and ankle only by pretending your big toe is a pen, and draw the alphabet using capital letters. Perform times a day. Calf Stretching â€" Hold each stretch for at least 30 seconds, three times on each leg, times a day. Perform plantarflexion and dorsiflexion movement by initially using an exercise band. I recommend using a Thera-Band Exercise Band http: As pain and range of motion improve, progress to inversion and eversion with the exercise band. Stop if you experience more than a mild increase in pain levels. Initial Balance and Proprioception Exercises. Standing on one foot. Initially, you may need to use your hand or a finger on a counter top for added support. As the pain subsides and your balance improves, you may need to increase the difficulty level. As you progress, balance will become of greater importance to be addressed in Part III. Toward the end of the intermediate phase, you should be walking fairly normally. There will likely be some swelling. It is typical for some amount of swelling to come and go. It will be directly related to how long you are on your feet and your general lower extremity circulation. I highly recommend you continue to wear compression stockings during this time. You may also continue to experience soreness and painâ€"particularly after a long day or a lot of upright activity. Continue to utilize a regular icing protocol as needed for pain and swelling. Also, continue to supplement with Capra Flex by Mt. The final stage of rehabilitation includes a full return to daily activities and eventually, all sport or athletic activities. I will address the specifics of the final stage of rehabilitation in Part III. If you have a question that you would like featured in an upcoming blog post, please e-mail contact thephysicaltherapyadvisor. The Physical Therapy Advisor blog is for general

informational purposes only and does not constitute the practice of medicine or other professional health care services, including the giving of medical advice. The use of information on this blog or materials linked from this blog is at your own risk. The content of this blog is not intended to be a substitute for professional medical advice, diagnosis, or treatment. Do not disregard, or delay in obtaining, medical advice for any medical condition you may have. Please seek the assistance of your health care professionals for any such conditions.

INTERMEDIATE PHASE OF TREATMENT pdf

4: Typical Treatment of Acute Myeloid Leukemia (Except APL)

Intermediate Phase of Interpersonal Psychotherapy (8 sessions) Content Objectives Strategies Pathologic Facilitate the grieving Review symptoms of depression (in each session).

Fatigue , loss of appetite , fever , and joint pain are common. Symptoms depend on primary tumor locations and metastases if present: A tumor pressing on the spinal cord may cause weakness , thus an inability to stand, crawl, or walk. Bone lesions in the legs and hips may cause pain and limping. A tumor in the bones around the eyes or orbits may cause distinct bruising and swelling. Infiltration of the bone marrow may cause pallor from anemia. Neuroblastoma can also develop anywhere along the sympathetic nervous system chain from the neck to the pelvis. Frequencies in different locations include: In rare cases, no primary tumor can be discerned. The great majority of cases are sporadic and nonfamilial. Familial neuroblastoma in some cases is caused by rare germline mutations in the anaplastic lymphoma kinase ALK gene. Neuroblastoma is also a feature of neurofibromatosis type 1 and the Beckwith-Wiedemann syndrome. MYCN oncogene amplification within the tumor is a common finding in neuroblastoma. The degree of amplification shows a bimodal distribution: The presence of this mutation is highly correlated to advanced stages of disease. Due to characteristic early onset, many studies have focused on parental factors around conception and during gestation. Factors investigated have included occupation i. It may arise from any neural crest element of the sympathetic nervous system SNS. Esthesioneuroblastoma , also known as olfactory neuroblastoma, is believed to arise from the olfactory epithelium and its classification remains controversial. However, since it is not a sympathetic nervous system malignancy, esthesioneuroblastoma is a distinct clinical entity and is not to be confused with neuroblastoma. When it is radio-iodinated with I or I radioactive iodine isotopes , it is a very good radiopharmaceutical for diagnosis and monitoring of response to treatment for this disease. With a half-life of 13 hours, I is the preferred isotope for imaging sensitivity and quality. I has a half-life of 8 days and at higher doses is an effective therapy as targeted radiation against relapsed and refractory neuroblastoma. Homer Wright rosettes are tumor cells around the neuropil , not to be confused with pseudorosettes, which are tumor cells around a blood vessel. This distinction in the pre-treatment tumor pathology is an important prognostic factor, along with age and mitosis - karyorrhexis index MKI. This pathology classification system the Shimada system describes "favorable" and "unfavorable" tumors by the International Neuroblastoma Pathology Committee INPC which was established in and revised in Localized tumor confined to the area of origin. Unilateral tumor with incomplete gross resection; identifiable ipsilateral and contralateral lymph node negative for tumor. Unilateral tumor with complete or incomplete gross resection; with ipsilateral lymph node positive for tumor; identifiable contralateral lymph node negative for tumor. Tumor infiltrating across midline with or without regional lymph node involvement; or unilateral tumor with contralateral lymph node involvement; or midline tumor with bilateral lymph node involvement. Dissemination of tumor to distant lymph nodes, bone marrow, bone, liver, or other organs except as defined by Stage 4S. Although international agreement on staging INSS has been used, the need for an international consensus on risk assignment has also been recognized in order to compare similar cohorts in results of studies. Beginning in , representatives of the major pediatric oncology cooperative groups have met to review data for 8, neuroblastoma patients treated in Europe, Japan, USA, Canada, and Australia between and Retrospective studies revealed the high survival rate of 12â€”18 month old age group, previously categorized as high-risk, and prompted the decision to reclassify 12â€”18 month old children without N-myc also commonly referred to as MYCN amplification to intermediate risk category. Localized disease without image-defined risk factors. Localized disease with image-defined risk factors. Metastatic disease "special" where MS is equivalent to stage 4S. The new risk stratification will be based on the new INRGSS staging system, age dichotomized at 18 months , tumor grade, N-myc amplification, unbalanced 11q aberration, and ploidy into four pre-treatment risk groups: Screening asymptomatic infants at three weeks, six months, and one year has been performed in Japan, Canada, Austria and Germany since the s. Screening was halted in after studies in Canada and Germany showed no reduction in deaths due to neuroblastoma, but rather caused an increase in diagnoses that would have disappeared

without treatment, subjecting those infants to unnecessary surgery and chemotherapy. However, long-term survival for children with advanced disease older than 18 months of age is poor despite aggressive multimodal therapy intensive chemotherapy , surgery , radiation therapy , stem cell transplant , differentiation agent isotretinoin also called cis-retinoic acid, and frequently immunotherapy [39] with anti- GD2 monoclonal antibody therapy. Biologic and genetic characteristics have been identified, which, when added to classic clinical staging, has allowed patient assignment to risk groups for planning treatment intensity. Low-risk disease can frequently be observed without any treatment at all or cured with surgery alone. The randomization was stopped so all patients enrolling on the trial will receive the antibody therapy. Agents commonly used in induction and for stem cell transplant conditioning are platinum compounds cisplatin , carboplatin , alkylating agents cyclophosphamide , ifosfamide , melphalan , topoisomerase II inhibitor etoposide , anthracycline antibiotics doxorubicin and vinca alkaloids vincristine. Some newer regimens include topoisomerase I inhibitors topotecan and irinotecan in induction which have been found to be effective against recurrent disease. Further treatment is available in phase I and phase II clinical trials that test new agents and combinations of agents against neuroblastoma, but the outcome remains very poor for relapsed high-risk disease. The majority of survivors have long-term effects from the treatment. Survivors of intermediate and high-risk treatment often experience hearing loss. Growth reduction, thyroid function disorders, learning difficulties, and greater risk of secondary cancers affect survivors of high-risk disease. Tumors presenting with any kind of segmental chromosome copy number changes were associated with a high risk of relapse. Within tumors showing segmental alterations, additional independent predictors of decreased overall survival were N-myc amplification, 1p and 11q deletions, and 1q gain. Earlier publications categorized neuroblastomas into three major subtypes based on cytogenetic profiles: Subtypes 2A and 2B: Virtual karyotyping can be performed on fresh or paraffin-embedded tumors to assess copy number at these loci. SNP array virtual karyotyping is preferred for tumor samples, including neuroblastomas, because they can detect copy neutral loss of heterozygosity acquired uniparental disomy. Copy neutral LOH can be biologically equivalent to a deletion and has been detected at key loci in neuroblastoma. The annual mortality rate is 10 per million children in the 0- to 4-year-old age group, and 4 per million in the 4- to 9-year old age group. The characteristics of tumors from the sympathetic nervous system and the adrenal medulla were then noted in by German pathologist Felix Marchand. In James Homer Wright understood the tumor to originate from primitive neural cells, and named it neuroblastoma. He also noted the circular clumps of cells in bone marrow samples which are now termed "Homer Wright rosettes". Of note, "Homer-Wright" with a hyphen is grammatically incorrect, as the eponym refers to just Dr. The measure was signed into law in July by U. Edwards was inspired in the endeavor by the illness and subsequent death of Erin Channing Buenger â€” of Bryan , daughter of one of his constituents, Walter L.

INTERMEDIATE PHASE OF TREATMENT pdf

5: Understand rehabilitation to facilitate a return to competition

Interpersonal Psychotherapy: An overview similar to how the initial phase of treatment was structured. The IPT problem areas Within the four problem areas.

Patients with AML undergo extensive evaluation including: Medical history and physical examination Blood tests Bone marrow aspiration with biopsy Immunophenotyping of the marrow to define protein expression in leukemia cells Doctors review this information to confirm the AML subtype. Treatment begins as soon as possible and varies by age, general condition and cytogenetics results. For most patients age 69 or younger, the goal is a cure. Typical therapy includes three phases: First phase â€" Induction chemotherapy Second phase â€" Consolidation chemotherapy, which may include stem cell transplantation Third phase â€" In some cases, blood or marrow transplant BMT One AML subtype, called acute promyelocytic leukemia APL , has a unique biology and is treated differently. This type of leukemia is treated with a vitamin pill called all trans-retinoic acid ATRA and arsenic together with chemotherapy. Most people undergo multiple cycles of this therapy. Bone marrow transplantation is rarely needed. Induction Chemotherapy The goals of induction chemotherapy are to eliminate leukemia cells from the blood and bone marrow and to induce a remission. A complete remission is defined as having no visible leukemia cells in the blood or bone marrow and having normal blood counts without the need of transfusions. Induction therapy for patients under the age of 70 typically includes ARA-C, daunorubicin and occasionally etoposide chemotherapy. The medications are given over a six- to seven-day period. Although side effects are expected and can be severe, our team is very skilled at caring for patients undergoing induction therapy for AML, administering all needed supportive care measures, such as transfusions, antibiotics and medications. During most of the hospital stay, patients receive intensive supportive care to protect them during their period of very low blood counts, including transfusions of red blood cells and platelets to correct anemia and to prevent bleeding. Antibiotics are used both preventatively and to treat bacteria and fungal infections. Additional antibiotics may be needed if other infections occur. The chemotherapy agents also are associated with complete but temporary hair loss, sores in the mouth, and throat and skin rashes. Other support care measures include anti-nausea drugs to prevent or decrease nausea, anti-diarrheals to decrease diarrhea, eye drops to prevent irritation and nutrient-rich beverages to improve nutrition. Patients are expected to be hospitalized for four to five weeks. Once the blood counts have returned to normal and the bowels function normally, patients may be discharged from the hospital. At the end of the induction therapy, a bone marrow biopsy is performed to see if a complete remission has been achieved. Approximately 70 percent to 80 percent of patients are expected to enter complete remission. If a complete remission is achieved, patients are then given a one-month break to prepare for the second consolidation treatment cycle. Consolidation Chemotherapy Once a patient achieves a complete remission more chemotherapy is needed to destroy any residual leukemia in the body. Consolidation chemotherapy can consist of the following: Additional cycles of intensive chemotherapy. A bone marrow transplant using blood or marrow from a donor, such as a brother or sister or unrelated person. This is called an allogeneic transplant. The type of consolidation therapy offered to any particular patient depends on the subtype of leukemia, the initial chromosome analysis and the expertise of the leukemia doctor and center. This second phase of treatment will likely include similar chemotherapy medications including the drug ARA-C cytarabine , sometimes with etoposide. The actual number of chemotherapy cycles required during consolidation as well as the need for a stem cell transplant varies from case to case. In general, consolidation treatment has similar toxicities as induction therapy and also requires intensive supportive care. At some institutions, the consolidation treatments can be done as an outpatient, but most patients are treated in the hospital and require a four- to five-week hospitalization. Overall, approximately 30 percent to 40 percent of patients receiving consolidation chemotherapy are cured of their AML. If a patient is going to have an autologous bone marrow transplant, his or her stem cells are collected after the consolidation chemotherapy stage is complete and once his or her blood counts recover. A process called "mobilization" enables the collection of stem cells from the blood. This catheter is connected to an "apheresis" machine that separates the

blood into individual components allowing the collection of only the white blood cells. All the other cells, including red blood cells and platelets, are given back to the patient. Each apheresis procedure takes four hours, and two to three procedures are usually necessary to collect enough stem cells. Once the stem cells are collected, and all other toxicities have resolved, the patients may be discharged from the hospital. The stem cells are frozen and saved for future use. Patients are then given a one-month break to prepare for high-dose chemotherapy and the re-infusion of the stem cells. This stage of treatment is both the most important and most dangerous. This therapy requires a one-month hospitalization. The goal of this therapy is to utilize much higher doses of chemotherapy to completely destroy any residual leukemia cells. The side effects are rather severe and the chance of dying from complications associated with high-dose chemotherapy and autologous stem cell transplant is 1 to 2 percent. The chemotherapy medications include Busulfan and etoposide, which are administered over five days. After two days of chemotherapy, the frozen stem cells will be re-infused into an IV catheter. The rest of the hospitalization consists of waiting for the new stem cells to grow, and during this time supportive care is administered as needed. Once the blood counts have improved and the other side effects have resolved, patients may be discharged. Patients are followed closely in the outpatient clinic for approximately three months. Most patients are able to return to work within three to six months of transplantation. The preferred treatment for patients with good or intermediate-risk chromosome test results is autologous stem cell transplantation, while allogeneic transplantation is favored for those with high-risk chromosome test results. Allogeneic transplantation using stem cells or bone marrow from a tissue-matched brother or sister or unrelated donor produces cure rates of approximately 50 percent to 60 percent in patients with intermediate-risk AML. However, allogeneic transplantation is significantly more difficult and dangerous than either chemotherapy or autologous transplantation, and 15 percent to 30 percent of patients do not survive the first year of treatment. For this reason, it is often reserved for more difficult cases. Poor-risk AML patients are best treated with allogeneic transplant despite its difficulties and dangers. Cure rates of 20 percent to 25 percent may be achieved. For patients with poor-risk leukemia who do not have a matched sibling donor, an unrelated donor may be sought through the National Marrow Donor Program NMDP. Investigational Therapies UCSF is dedicated to improving outcomes for AML patients through the use of investigational therapies and clinical research trials. Recent efforts focus on increasing the busulfan dose during autologous transplant, treating older patients with chemotherapy packaged in balls of fat liposomes to decrease side effects and possibly increase remission rates, and treating patients with AML derived from myelodysplastic syndromes or from therapy for other cancers with experimental chemotherapy agents thay may be more likely to achieve remission.

INTERMEDIATE PHASE OF TREATMENT pdf

6: Myelofibrosis - Wikipedia

However, some critical infrastructure/key resources or lifesaving missions may arise in the intermediate phase, where these guidelines would apply. Emergency personnel may be exposed to increased radiation during the unique catastrophic event of an IND detonation resulting in firestorm and widespread destruction of structures.

Increased susceptibility to infection, such as pneumonia Pallor and shortness of breath due to anemia In rarer cases, a raised red blood cell volume Cutaneous myelofibrosis is a rare skin condition characterized by dermal and subcutaneous nodules. These mutations are not specific to myelofibrosis, and are linked to other myeloproliferative neoplasms, specifically polycythemia vera and essential thrombocythemia. Janus kinases JAKs are non-receptor tyrosine kinases essential for the activation of signaling that is mediated by cytokine receptors lacking catalytic activity. These include receptors for erythropoietin , thrombopoietin , most interleukins and interferon. The VF mutation appears to make hematopoietic cells more sensitive to growth factors that need JAK2 for signal transduction , which include erythropoietin and thrombopoietin. A mutation in that gene, known as a W mutation, leads to the production of an abnormal thrombopoietin receptor protein, which results in the overproduction of abnormal megakaryocytes. It is one of the myeloproliferative disorders , diseases of the bone marrow in which excess cells are produced at some stage. Production of cytokines such as fibroblast growth factor by the abnormal hematopoietic cell clone particularly by megakaryocytes [10] leads to replacement of the hematopoietic tissue of the bone marrow by connective tissue via collagen fibrosis. However, the proliferation of fibroblasts and deposition of collagen is a secondary phenomenon, and the fibroblasts themselves are not part of the abnormal cell clone. In primary myelofibrosis, progressive scarring, or fibrosis , of the bone marrow occurs, for the reasons outlined above. The result is extramedullary hematopoiesis , i. This causes an enlargement of these organs. In the liver, the abnormal size is called hepatomegaly. Enlargement of the spleen is called splenomegaly , which also contributes to causing pancytopenia, particularly thrombocytopenia and anemia. Another complication of extramedullary hematopoiesis is poikilocytosis , or the presence of abnormally shaped red blood cells. Myelofibrosis can be a late complication of other myeloproliferative disorders, such as polycythemia vera , and less commonly, essential thrombocythaemia. In these cases, myelofibrosis occurs as a result of somatic evolution of the abnormal hematopoietic stem cell clone that caused the original disorder. In some cases, the development of myelofibrosis following these disorders may be accelerated by the oral chemotherapy drug hydroxyurea. Sites of hematopoiesis[edit] The principal site of extramedullary hematopoiesis in myelofibrosis is the spleen , which is usually markedly enlarged, sometimes weighing as much as g. As a result of massive enlargement of the spleen , multiple subcapsular infarcts often occur in the spleen, meaning that due to interrupted oxygen supply to the spleen partial or complete tissue death happens. On the cellular level , the spleen contains red blood cell precursors, granulocyte precursors and megakaryocytes , with the megakaryocytes prominent in their number and in their bizarre shapes. Megakaryocytes are believed to be involved in causing the secondary fibrosis seen in this condition, as discussed under "Mechanism" above. Sometimes unusual activity of the red blood cells , white blood cells , or platelets is seen. The liver is often moderately enlarged, with foci of extramedullary hematopoiesis. Microscopically, lymph nodes also contain foci of hematopoiesis, but these are insufficient to cause enlargement. There are also reports of hematopoiesis taking place in the lungs. These cases are associated with hypertension in the pulmonary arteries. Both early and late in disease, megakaryocytes are often prominent and are usually dysplastic. Diagnosis[edit] Epidemiologically , the disorder usually develops slowly and is mainly observed in people over the age of Diagnosis of myelofibrosis is made on the basis of bone marrow biopsy. Primary myelofibrosis can begin with a blood picture similar to that found in polycythemia vera or chronic myeloid leukemia. Most people with myelofibrosis have moderate to severe anemia. Eventually thrombocytopenia , a decrease of blood platelets develops. When viewed through a microscope, a blood smear will appear markedly abnormal, with presentation of pancytopenia , which is a reduction in the number of all blood cell types: Red blood cells may show abnormalities including bizarre shapes , such as teardrop-shaped cells , and nucleated red blood cell precursors may appear in the blood smear

leukoerythroblastic reaction. Normally, mature red blood cells in adults do not have a cell nucleus, and the presence of nucleated red blood cells suggests that immature cells are being released into the bloodstream in response to a very high demand for the bone marrow to produce new red blood cells. Immature white cells and platelets large megakaryocytes are also seen in blood samples, and basophil counts are increased. When late in the disease progression an attempt is made to take a sample of bone marrow by aspiration , it may result in a dry tap, meaning that where the needle can normally suck out a sample of semi-liquid bone marrow, it produces no sample because the marrow has been replaced with collagen fibers. A bone marrow biopsy will reveal collagen fibrosis , replacing the marrow that would normally occupy the space. Treatment[edit] The one known curative treatment is allogeneic stem cell transplantation , but this approach involves significant risks. These data showed that the treatment significantly reduced spleen volume, improved symptoms of myelofibrosis, and was associated with much improved overall survival rates compared to placebo. Chiropractic is also considered as an alternative pain control option for patients with myelofibrosis. The World Health Organization utilized the name "chronic idiopathic myelofibrosis", while the International Working Group on Myelofibrosis Research and Treatment calls the disease "primary myelofibrosis". In WHO has adopted the name of "primary myelofibrosis.

INTERMEDIATE PHASE OF TREATMENT pdf

7: Ankle Sprains â€“ Intermediate Phase (Part II of III) | The Physical Therapy Advisor

A quasi-chemical treatment of the superlattice formation model is applied to intermediate phases appearing in nonstoichiometric compounds. Two kinds of interaction energy are introduced and both the intermediate phase and two-phase separation are described in a single formula.

This article has been cited by other articles in PMC. While precise pathological mechanisms are lacking, the growing base of knowledge concerning TBI has put increased emphasis on its understanding and treatment. Most treatments of TBI are aimed at ameliorating secondary insults arising from the injury; these insults can be characterized with respect to time post-injury, including early, intermediate, and late pathological changes. Early pathological responses are due to energy depletion and cell death secondary to excitotoxicity, the intermediate phase is characterized by neuroinflammation and the late stage by increased susceptibility to seizures and epilepsy. Current treatments of TBI have been tailored to these distinct pathological stages with some overlap. Many prophylactic, pharmacologic, and surgical treatments are used post-TBI to halt the progression of these pathologic reactions. In the present review, we discuss the mechanisms of the pathological hallmarks of TBI and both current and novel treatments which target the respective pathways. Introduction Individuals of all ages, background, and health status are susceptible to traumatic brain injury TBI. Every year in the United States 1. While the numbers suggest a grim state concerning TBI treatment there have been improvements in its management. Evidence-based guidelines for TBI management were introduced in because of varied treatment approaches but in the years following there have still been lapses in consistent implementation [3 , 4]. One problem in the development of reliable guidelines for treatment of TBI is the varied pathophysiology of injury. TBI may be penetrating or non-penetrating, diffuse or focal, vary in severity, location, and patient characteristics, just to name a few. Additionally, since TBI is often accident-related, there are limited primary prophylactic measures. Much of the resultant acute and chronic harm from TBI is related to secondary generation of tissue damage and inflammation. In the present review, we will attempt to describe the pathophysiology of three distinct yet over-lapping states post-injury, the early, immediate, and late phases. The early phase of damage usually occurs within 24 h of injury and is directly related to tissue damage and deregulated physiological functions, the intermediate phase takes place in the days following TBI and entails neuroinflammation, and the late phase is primarily associated with seizures and epileptogenesis and arises days to weeks after TBI. Following each phase we will describe current and novel treatments and interventions that directly target the pathophysiology of each phase. There is a wealth of TBI data with countless views on injury mechanisms and treatment modalities; thus, this review provides a detailed but limited glimpse into components of the literature. Early Phase Different forms of mechanical insult ensue depending on the type of TBI, including acceleration-deceleration shearing and penetrating injury. Regardless, early damage following TBI often stems from the ischemic cascade. The sequential ischemic cascade begins with interruption of normal blood flow and numerous experimental studies demonstrate this effect. PBBI also decreases brain tissue oxygenation tension and causes spreading cortical depolarizations shortly after injury [5]. As mentioned earlier, there is overlap between pathological phases. For instance, clinical evidence has demonstrated chronic CBF reduction in particular brain regions of TBI patients which cause a lasting effect to normal functioning [7]. Overall, the literature suggests CBF disturbance is one of the first pathological steps and the effect varies with age and time. Those anemic patients with compromised brain oxygen tension were over six times more likely to suffer unfavorable outcomes, regardless of the injury severity; suggesting proper oxygenation minimizes damage due to injury [10]. The data may be extrapolated into the clinical realm where they become especially relevant. Yet, Eriksson et al. Reduced blood flow and oxygen metabolism in the brain promotes a metabolic switch from the usual aerobic process to an anaerobic program. Lactate is a marker of anaerobic respiration and builds up in tissue deprived of oxygen. Many studies have used measures of glucose consumption or oxygen levels prove there is a reduction in normal cerebral metabolism [13 , 14]. Even if other vital measures are controlled, metabolic deregulation still occurs. Not only might cerebral blood flow and oxygen affect metabolic functioning but also the ability for glucose to enter the brain. One study

using positron emission tomography PET with radioactively tagged glucose demonstrated diminished uptake of glucose into both cerebral hemispheres after FPI; further, glial activation and axonal damage seemed to persist in regions deprived of glucose uptake [16]. In later phases of TBI pathophysiology, large variations in glucose levels have been associated with worse long-term outcomes, suggesting a more complicated metabolic relationship [17]. Interestingly, glucose administration after controlled-cortical impact CCI is neuroprotective in the hippocampus and cortex, suggesting exogenous glucose supplementation is beneficial post-TBI [18]. The variation in the literature paint a confusing metabolic landscape which likely varies based on the heterogeneity of TBI and time period of analysis. Deregulated cerebral metabolism and the favored breakdown of lactate rather than glucose necessarily lead to a deficit in cerebral energy production [21]. Ischemia, reduced CBF, and altered metabolic function ultimately lead to excitotoxicity-mediated cell death, including both apoptosis and necrosis [23 , 24]. Glutamate is the prime excitatory amino acid and is released via pre-synaptic vesicles or leaks out of damaged membranes after TBI. Such glutamate release also correlates with age, being elevated in microdialysates of elderly TBI patients compared to their younger TBI counterparts; in the same study, other measures such as some cytokines had no quantitative change [27]. Studies in vitro confirmed elevated glutamate activity leads to hyperexcitability and neuronal death in a dose-response relationship [28]. These findings are in agreement with other research where MK, an NMDA receptor antagonist, decreased neuronal caspase-3 expression, neuronal nitric oxide synthase nNOS positive neurons, and mitochondria degeneration [31]. NO is a direct component of the neuroinflammatory cascade, intriguingly glutamate indirectly promotes inflammatory processes as well. Overall, it is well accepted that glutamate opens the proverbial flood gates of the cell which produce significant cellular harm. Structurally, mitochondria exhibit swelling due to a mitochondrial permeability transition pore that compromises their function. Experimental findings demonstrated mitochondrial pathology precedes neuronal loss and can be seen as early as 30 min post-TBI in CCI rats [38]. Of clinical relevance, both cytochrome c and caspase have been identified in the CSF of patients with severe TBI [39]. Clinical evidence corroborates the importance of mitochondrial pathology since N-acetylaspartate, a surrogate of mitochondrial function, is correlated with TBI patient outcomes [40]. Overall, immediate physical and structural damage from TBI interrupts blood flow and oxygenation to the brain which are both tightly regulated variables. Mechanical stress and ischemia help advance the excitotoxic cascade and deregulate cerebral metabolism, producing the earliest pathological indications of TBI. Prophylactic Hypothermia and Hyperbaric Oxygen Therapy HBOT Initial management of the TBI patient is generally centered on prophylaxis and supportive measures, including blood pressure and oxygenation monitoring, infection and deep vein thrombosis prophylaxis, analgesia, and setting thresholds on vital values including ICP and CPP. Deregulation of cerebral metabolism, blood flow, and lost perfusion are early changes post-TBI. Prophylactic hypothermia is one option that directly combats the problematic nature of early TBI pathology. Hypothermia lowers cerebral metabolic rates and slows damage occurring post-TBI. Hypothermia also dampens the innate immune response post-TBI in experimental models, also demonstrating the overlap with inflammatory phase which is yet to be discussed [42]. Simultaneously, the BTF reports preliminary evidence which suggests a decrease in mortality upon maintaining target temperatures for 48 h and that patients receiving prophylactic hypothermia had higher Glasgow Outcome Scale GOS scores compared to normothermic patients [43]. Prophylactic hypothermia has received mixed results in the literature because of multiple variables involved in its successful implementation; these include temperature at time of injury, initial onset of cooling, rate of cooling, final temperature sought, and mechanism of cooling. Early research corroborated such variability by finding spontaneous hypothermia upon time of admission to be associated with poorer prognosis [44 â€” 46]. Thus, it may be said that hypothermia administration is as heterogeneous as the TBI pathology itself. Since brain temperature cannot be predicted with high confidence from body temperatures separate monitoring is recommended [47]. However, recent retrospective analysis of pooled neurotrauma data revealed patients receiving hypothermia treatment had significantly higher favorable outcomes compared to normothermic patients and those with no temperature management; it is important to note that hypothermic patients were, on average, significantly younger [49]. Indeed, other studies have replicated these findings. One such study demonstrated hypothermia of The same study also identified

hyperglycemia to be an independent risk for poor outcome and that hypothermic patients had reduced glucose concentrations compared to normothermic counterparts. Such a finding suggests hypothermia may promote favorable outcomes by decreased glucose levels. A meta-analysis by Fox et al. Besides strategic design, another key variable influencing prophylactic hypothermia studies is re-warming strategy. Another prospective study cooled patients to Importantly, that study found hypothermic patients to have significantly improved GOS compared to the normothermic group [50]. Undoubtedly then, re-warming strategies may be just as significant as cooling ones in optimizing patient outcomes. Table 1 summarizes results of evidence regarding the effect of prophylactic hypothermia on various outcomes. The selected results contain mixed results regarding the effectiveness of TBI.

INTERMEDIATE PHASE OF TREATMENT pdf

8: Acute Myeloid Leukemia Treatment | Conditions & Treatments | UCSF Medical Center

This second phase of treatment will likely include similar chemotherapy medications including the drug ARA-C (cytarabine), sometimes with etoposide. The actual number of chemotherapy cycles required during consolidation as well as the need for a stem cell transplant varies from case to case.

The process of returning to competition following injury involves healing of the injured tissues, preparation of these tissues for the return to function, and use of proper techniques to maximize rehabilitation and reconditioning. While the goal is a rapid resumption of activity, it is important to remember that each athlete responds differently to injury and thus progresses uniquely during rehabilitation. Goals of Rehabilitation and Reconditioning As a preface to discussion of the goals of treatment during injury rehabilitation, two points must be made. First, healing tissue must not be overstressed 44, It should be obvious that when one is choosing the load, it is necessary to consider the phase of healing and athlete type. For example, a stress that underloads a tissue during remodeling probably overloads it during inflammation. Further, a stress that underloads a professional basketball center probably overloads an amateur cross-country runner. The plane of movement is another necessary consideration. As an example, the medial collateral ligament of the knee is most loaded in the frontal plane during terminal knee extension. Therefore, frontal plane movements should be avoided during early healing phases. However, those frontal plane movements should probably be included in some form during the later phases. Second, the athlete must meet specific objectives to progress from one phase of healing to the next 44, These objectives may depend on range of motion, strength, or activity. Treatment Goal The goal for treatment during the inflammatory phase is to prevent disruption of new tissue. A healthy environment for new tissue regeneration and formation is essential for preventing prolonged inflammation and disruption of new blood vessel and collagen production. To achieve these goals, relative rest, ice, compression, and elevation are the primary treatment options. Passive modalities that help reduce inflammation e. The athletic trainer provides the majority of passive treatment for the athlete during this acute phase. It is also important to realize that a quick return to function relies on the health of other body tissues. Therefore, the power, strength, and endurance of the musculoskeletal tissues and the function of the cardiorespiratory system must be maintained. The strength and conditioning professional can provide significant knowledge and expertise in this area. To accomplish these tasks, the strength and conditioning professional should consult with the athletic trainer to determine which types of exercises are indicated and contraindicated for the specific injury. Maximal protection of the injured structures is the primary goal during this phase. Assuming that this requirement is fulfilled, exercises may include general aerobic and anaerobic training and resistance training of the uninjured extremities. If movement of the injured limb is not contraindicated, isolated exercises that target areas proximal and distal to the injured area may also be permissible provided that they do not stress the injured area. Examples include hip abduction and rotation exercises following knee injury 22, 24, 31 or scapula stabilizing exercises following glenohumeral joint injury 25, Exercise Strategies Although a rapid return to competition is crucial, rest is necessary to protect the damaged tissue from additional injury. Therefore, exercise involving the injured area is not recommended during this phase. Repair Phase After the inflammatory phase, the body begins to repair the damaged tissue with similar tissue, but the resiliency of the new tissue is low. Repair of the weakened injury site can take up to eight weeks if the proper amount of restorative stress is applied, or longer if too much or too little stress is applied. Treatment Goal The treatment goal during the repair phase is to prevent excessive muscle atrophy and joint deterioration of the injured area. In addition, a precarious balance must be maintained in which disruption of the newly formed collagen fibers is avoided but low-load stresses are gradually introduced to allow increased collagen synthesis and prevent loss of joint motion. To protect the new, relatively weak collagen fibers, the athlete should avoid active resistive exercise involving the damaged tissue. Too little activity, though, can also have a deleterious effect, as newly formed fibers will not optimally align and may form adhesions, thereby preventing full motion. Early protected motion hastens the optimal alignment of collagen fibers and promotes improved tissue mobility. As in the inflammatory phase, therapeutic modalities are

permissible, but their goal during repair is to promote collagen synthesis. Ultrasound, electrical stimulation, and ice are continued in order to support and hasten new tissue formation 5, 23, Again, the maintenance of muscular and cardiorespiratory function remains essential for the uninjured areas of the body. The strength and conditioning professional has considerable expertise to offer the other members of the sports medicine team regarding selection of the appropriate activities. Possible exercise forms during the repair phase include strengthening of the uninjured extremities and areas proximal and distal to the injury, aerobic and anaerobic exercise, and improving strength and neuromuscular control of the involved areas. Exercise Strategies The following exercises should be used during the repair phase only after consultation with the team physician, athletic trainer, or physical therapist. Isometric exercise may be performed provided that it is pain free and otherwise indicated. Submaximal isometric exercise allows the athlete to maintain neuromuscular function and improve strength with movements performed at an intensity low enough that the newly formed collagen fibers are not disrupted. Unfortunately, isometric strengthening is joint angle specific; that is, strength gains occur only at the angles used Therefore, if indicated, it may be appropriate for the athlete to perform isometric exercises at multiple angles Resistance training is velocity specific 26 ; therefore, isokinetic exercise can be an important aspect of strengthening following injury. Isokinetic exercise uses equipment that provides resistance to movement at a given speed e. Because no sport is performed at one speed, however, isokinetic exercise is somewhat limited in its real-world application. Furthermore, most isokinetic equipment allows single-joint exercise only, which permits concentration on a specific muscle or joint but is not always the most functional method of strengthening. While isotonic exercise involves movements with constant external resistance, the amount of force required to move the resistance varies, depending primarily on joint angle and the length of each agonist muscle. Isotonic exercise uses several different forms of resistance, including gravity i. The speed at which the movement occurs is controlled by the athlete; movement speed can be a program design variable, with more acute injuries calling for slower movement and the later phases of healing amenable to faster, more sport-specific movement. Proprioception is an afferent response to stimulation of sensory receptors in skin, muscles, tendons, ligaments, and the joint capsule. Proprioception contributes to the conscious and unconscious control of posture, balance, stability, and sense of position Neuromuscular control, on the other hand, is the ability of muscle to respond to afferent proprioceptive information to maintain joint stability For example, when running on an uneven surface, cross-country runners require their lower extremities-especially their ankles-to adjust to the ground to prevent falls and injuries; that ability to adjust is neuromuscular control. After an injury, neuromuscular control, like strength and flexibility, is usually impaired Specific types of exercises exist to improve neuromuscular control following injury and can be manipulated through alterations in surface stability, vision, and speed. Mini-trampolines, balance boards, and stability balls can be used to create unstable surfaces for upper and lower extremity training. Athletes can perform common activities such as squats and push-ups on uneven surfaces to improve neuromuscular control. Exercises may also be performed with eyes closed, thus removing visual input, to further challenge balance. Finally, increasing the speed at which exercises are performed provides additional challenges to the system. Specifically controlling these variables within a controlled environment will allow the athlete to progress to more challenging exercises in the next stage of healing. Remodeling Phase The outcome of the repair phase is the replacement of damaged tissue with collagen fibers. After those fibers are laid down, the body can begin to remodel and strengthen the new tissue, allowing the athlete to gradually return to full activity. Treatment Goal Optimizing tissue function is the primary goal during the final phase of healing. Athletes improve function by continuing and progressing the exercises performed during the repair phase and by adding more advanced, sport-specific exercises that allow progressive stresses to be applied to the injured tissue. The athlete can be tempted to do "too much too soon," which may further damage the injured tissues. It is important to remember that, while there may be less pain with activity at this point, the injured tissues have not fully healed and require further attention to achieve complete recovery figure Progressive tissue loading allows improved collagen fiber alignment and fiber hypertrophy. Exercise Strategies Ultimately, rehabilitation and reconditioning exercises must be functional to facilitate a return to competition. Examples of functional training include joint angle-specific strengthening, velocity-specific muscle activity, closed kinetic chain

exercises, and exercises designed to further enhance neuromuscular control. Strengthening should transition from general exercises to sport-specific exercises designed to replicate movements common in given sports. For example, for a basketball guard who has rotator cuff tendinitis, rotator cuff strengthening may progress from a specific rotator cuff exercise to lateral dumbbell raises to machine seated shoulder presses to push-press exercises figure Specificity of movement speed is another important program design variable. Strengthening exercises are velocity specific; that is, the speed at which an athlete trains is directly related to the speed at which strength increases. Consider a sprinter with a hamstring muscle strain. Exercise selection for a sprinter with an improving hamstring muscle strain might progress from hamstring flexibility to eccentric strength to concentric strength to dynamic stretching and finally to rapid isotonic strengthening. Examples of velocity-specific exercise include isokinetic, plyometric, and speed training. Please refer to chapters 16 and 17 for a thorough discussion of plyometric and speed training, respectively. The kinetic chain is the collective effort or involvement of two or more sequential joints to create movement A closed kinetic chain exercise is one in which the terminal joint meets with considerable resistance that prohibits or restrains its free motion 38 ; that is, the distal joint segment is stationary. Lower extremity closed kinetic chain exercises have often been classified as a more functional form of exercise compared with open kinetic chain exercises 7, 21, 41 because most sport-related activities are performed with the feet "fixed" to the surface. For example, during the closed kinetic chain squat exercise, the feet are "fixed" to the floor and essentially do not move, providing a base upon which movement occurs figure Closed kinetic chain exercises have several advantages, including increased joint stability and functional movement patterns; during sport activity, joints are not typically used in isolation but rather work in concert with the adjacent joints and surrounding musculature. Although closed kinetic chain exercises are commonly viewed as lower extremity exercises, closed chain upper extremity exercises exist as well figure Get the latest news, special offers, and updates on authors and products.

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