

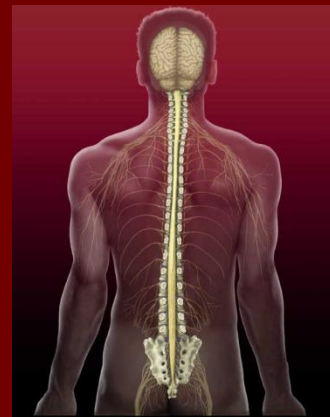
An anatomical illustration of the human nervous system, showing the brain, spinal cord, and peripheral nerves. The illustration is centered on a human figure, with the brain at the top and the spinal cord running down the back. The nerves are shown branching out from the spinal cord to the rest of the body. The background is a gradient of red and purple.

Complications of SCI

Denise C. Hill
University of Calgary
SCI Education Day
April 9, 2026

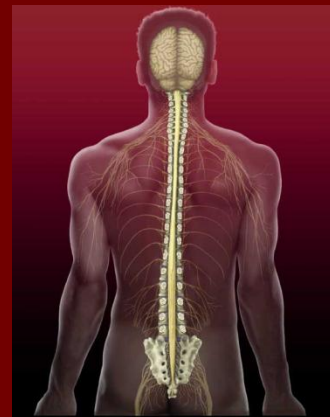
Outline

- Venous Thromboembolism
- Syringomyelia
- Heterotopic Ossification



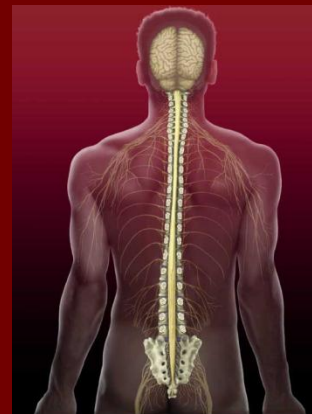
Venous thromboembolism Objectives

- Scope of the problem
- Patient risk factors
- Diagnosis
- Preventing DVT



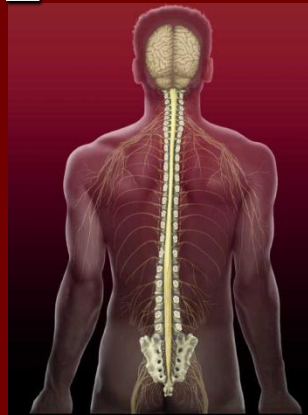
Case: Pulmonary Embolism

- Healthy 32 year old man on unit 58 for rehab
- SCI Dx: incomplete paraplegia
- Bedside round on Monday: new pleuritic chest discomfort and cough over w/e
- CT PE:
- Treatment initiated



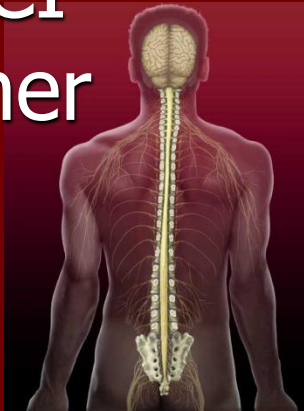
Venous Thromboembolism: overview

- Incidence: if not on anticoagulation: 40 – 70%
 - On anticoagulation: incidence is still @ 7%
- Morbidity and Mortality : ranges fr small asymptomatic thrombi to massive fatal PE
 - 35 % of acute deaths in SCI are due to VTE
- Risk of death due to PE is declining. Now 6th cause of death in first year after SCI (was 2nd - 3rd cause)



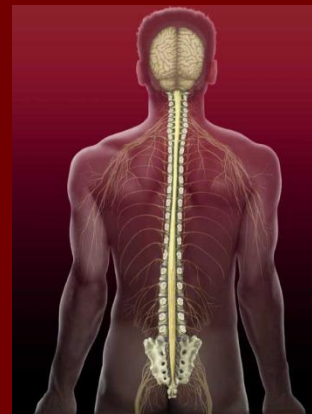
Chronic VTE

- Chronic PE may be as prevalent as 18% - is a cause of pulmonary htn
- For those with acute SCI who survive the first year after injury, the risk of death due to PE is 210 times greater than that of a similar healthy population
- Case of younger female w/ chronic SCI – fluctuating dyspnea “Call to Dr. Rimmer and restart anticoagulation”



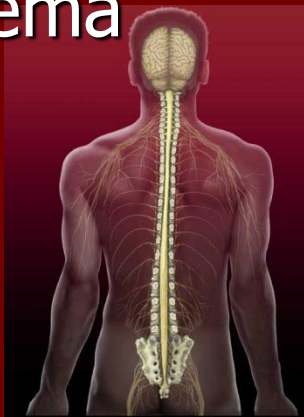
Patient Risk Factors

- Patient risk factors:
 - Paraplegia
 - complete injuries,
 - skeletal fractures,
 - older age,
 - sooner after injury,
 - prior DVT,
 - absent or delayed thromboprophylaxis



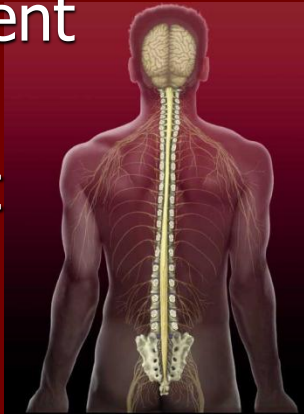
Diagnosis

- Clinical presentation (both can be silent):
 - **DVT**: new swelling, pain, erythema of calf, tenderness, fever
 - **PE**: dyspnea, pleuritic chest pain, decreased O2 sats, fever, tachycardia, code blue
- Investigations:
 - routine US discouraged.
 - US for patients with a clear, new leg edema or to confirm source of VTE in a patient already diagnosed with a PE
 - Blood gas, ECG, and CTPE for PE



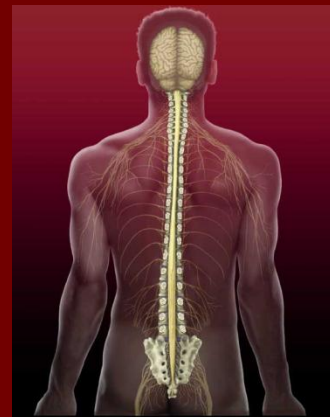
VTE Prophylaxis

- Consortium for SCI: “We recommend that mechanical thrombo-prophylaxis with intermittent pneumatic compression devices (PCDs) with or without graduated compression stockings (GCSs) be applied as soon as feasible after acute spinal cord injury (SCI) when not contraindicated by lower-extremity injury. [1C]”
- Heparin choice: must be Low molecular weight; Enoxaparin and Dalteparin are similarly effective
- Duration: 8 weeks or until discharged from inpatient rehab
- Reinitiate prophylaxis in the rehospitalized patient



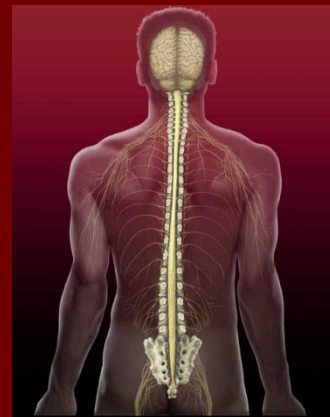
Syringomyelia: Objectives

- Be able to define syringomyelia
- Review presenting symptoms and signs of post traumatic syringomyelia
- Provide an overview of the pathophysiology
- Discuss treatment



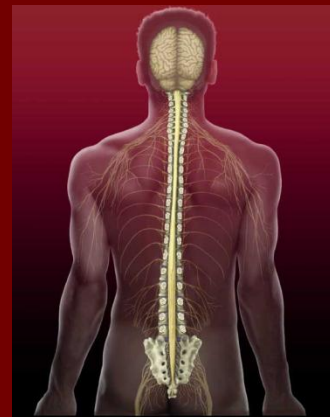
Syringomyelia

The post-traumatic development of a cerebrospinal fluid-filled cavity or cyst within the spinal cord or central canal



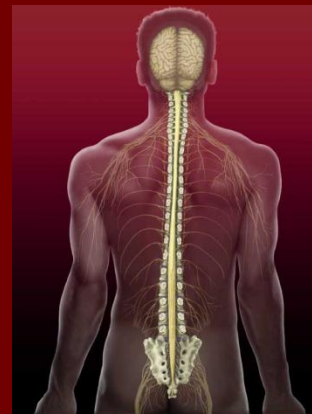
Syringomyelia Case

- Mrs. S.K. 61 y.o. lady
- High thoracic spinal cord tumor resected in India in 1999
- Seen in SCI clinic 8 mo after immigration in 2005
- Initial issues:
 - severe neuropathic pain
 - neurogenic bowel and bladder
 - sacral pressure ulcer
 - osteoporosis

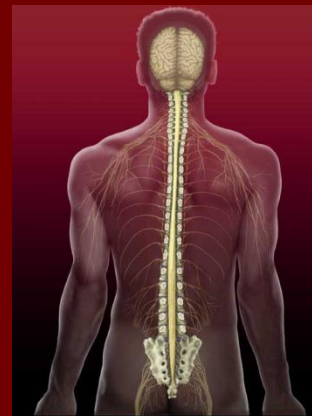
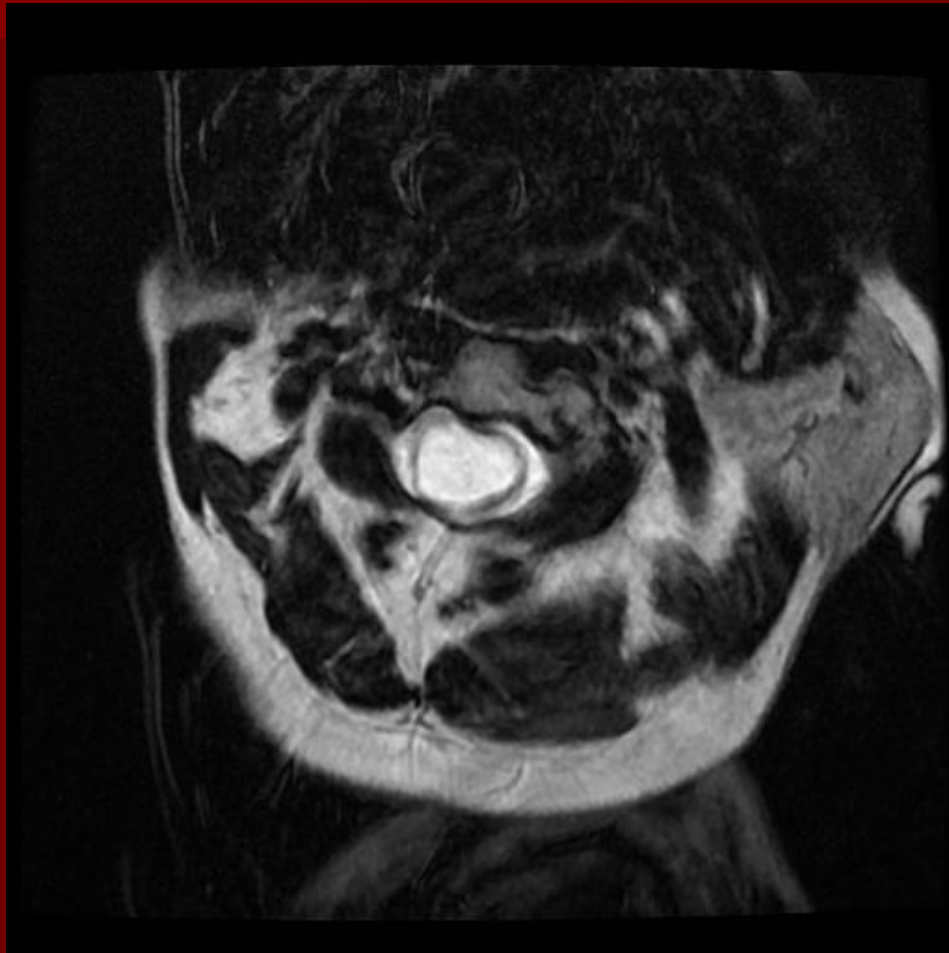


Mrs. S.K.

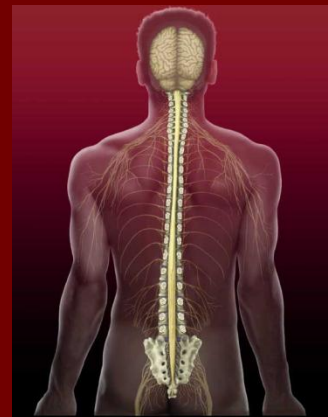
- Initial Physical Exam:
 - Sensory level was T2
 - Strength: Normal in ASIA muscles in UE
 - + anal sensation
- Diagnosis: ASIA B T2 paraplegia
- Pain persisted: Chest, axillae, genitalia
- Imaging ordered
 - ? Tumor vs syrinx?



MRI

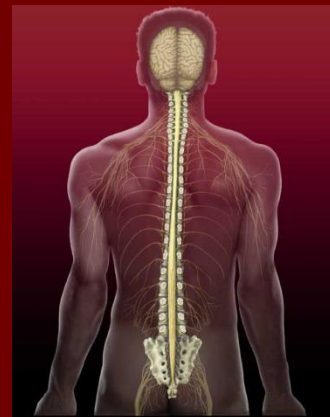


MRI



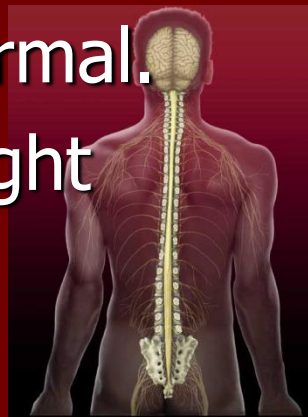
Follow-up

- Ongoing severe pain, especially at level
- Physical Exam
 - Tone in UE mildly increased
 - Deltoid 4/5 bilaterally (pain described)
 - No other weakness identified in UE's
 - Sensory level unchanged
 - UE DTR's mildly brisk, L > R, 3+



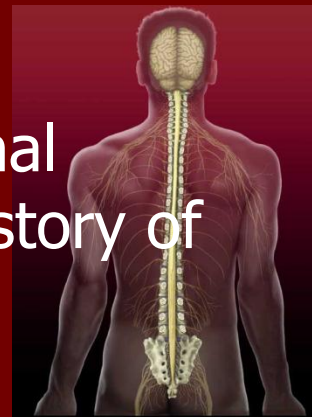
Clinical follow-up

- Neurosurgery opinion: Obtained a one year f/u MRI which was stable; advised to follow symptomatically
- MRI films from 1999 compared to 2010: similar degree of syrinx
- Pain persisted:
 - UE strength and sensation remained normal.
 - Ongoing slight increase in DTR's and slight increase in UE tone



Facts in Syringomyelia

- Symptomatic syringomyelia occurs in up to 10% of patients (long term prev 30%)
- Can occur anytime after SCI: 1 mo – 45 years, median 9 -15 years
- More common in complete injuries, male, fairly = in thoracic and cervical lesions, if no immediate decompression done, or if #/dislocation
 - Increased incidence if there is associated spinal canal stenosis, posttraumatic kyphosis, or a history of prior spinal surgeries



Symptoms of Syringomyelia

■ Classic:

- suspended sensory loss, segmental weakness and burning pain

■ Nonspecific

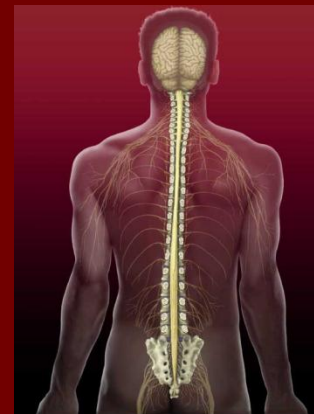
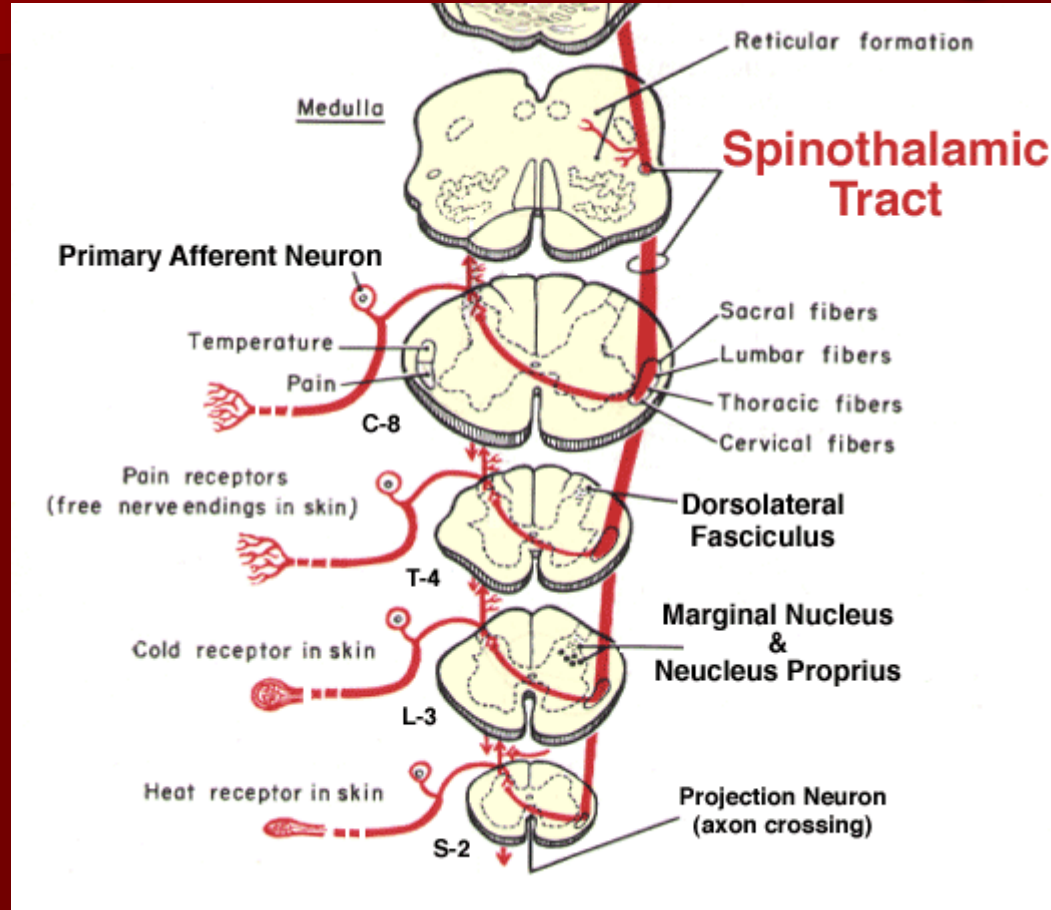
- Worsening spasticity
- Worsening/change in b/b function
- New unexplained AD
- Worsening Neuropathic pain

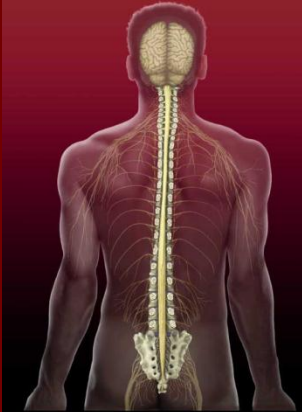
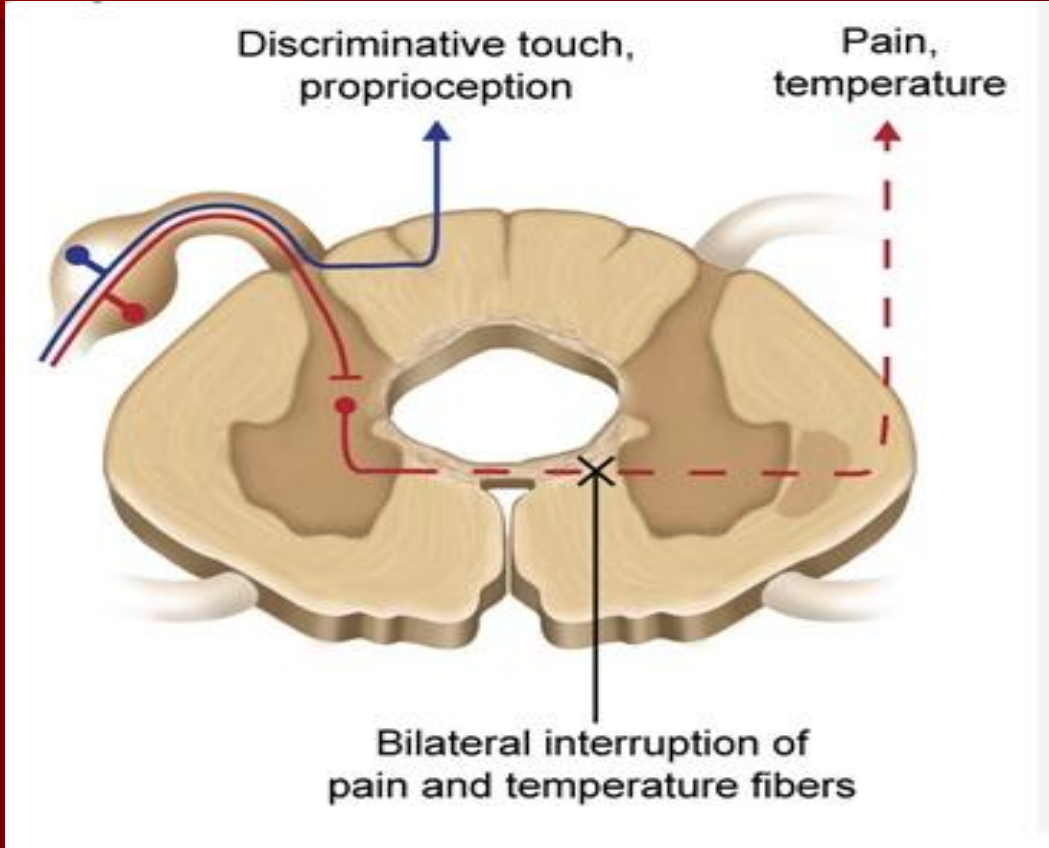
■ More specific

- Pain with coughing/straining, Radicular pain, new ascending sensory symptoms, excessive sweating above injury

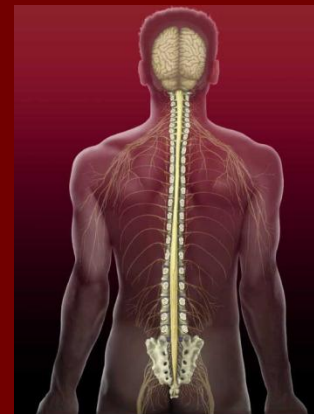
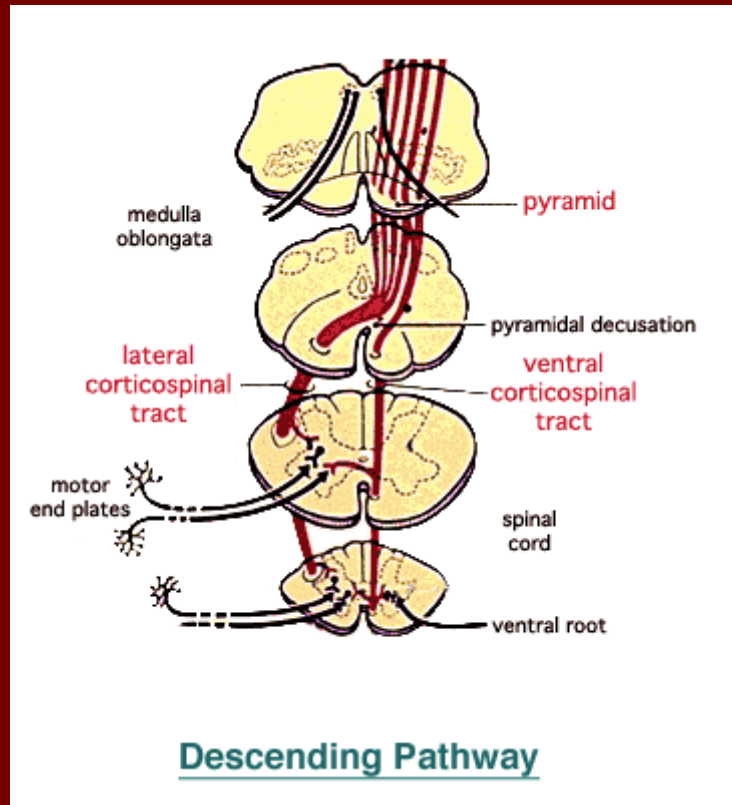


Loss of pain and temperature is an earlier finding



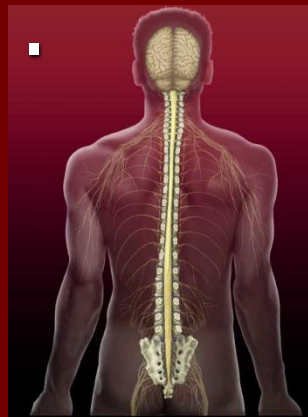


Weakness is a later finding



Less common presentations

- Brainstem symptoms: Occurs if syrinx extends proximally into medulla: Dysphagia, tongue weakness/atrophy, facial pain/sensory loss.
- MSK issues: Charcot joint Aka neurogenic arthropathies may be seen in the upper limbs (shoulder, elbow, wrist, spine).
Progressive neurogenic scoliosis

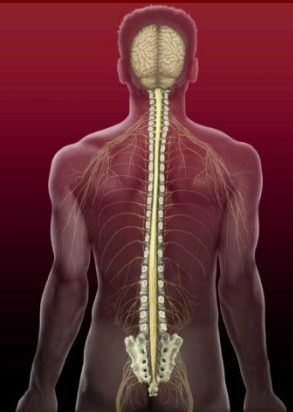
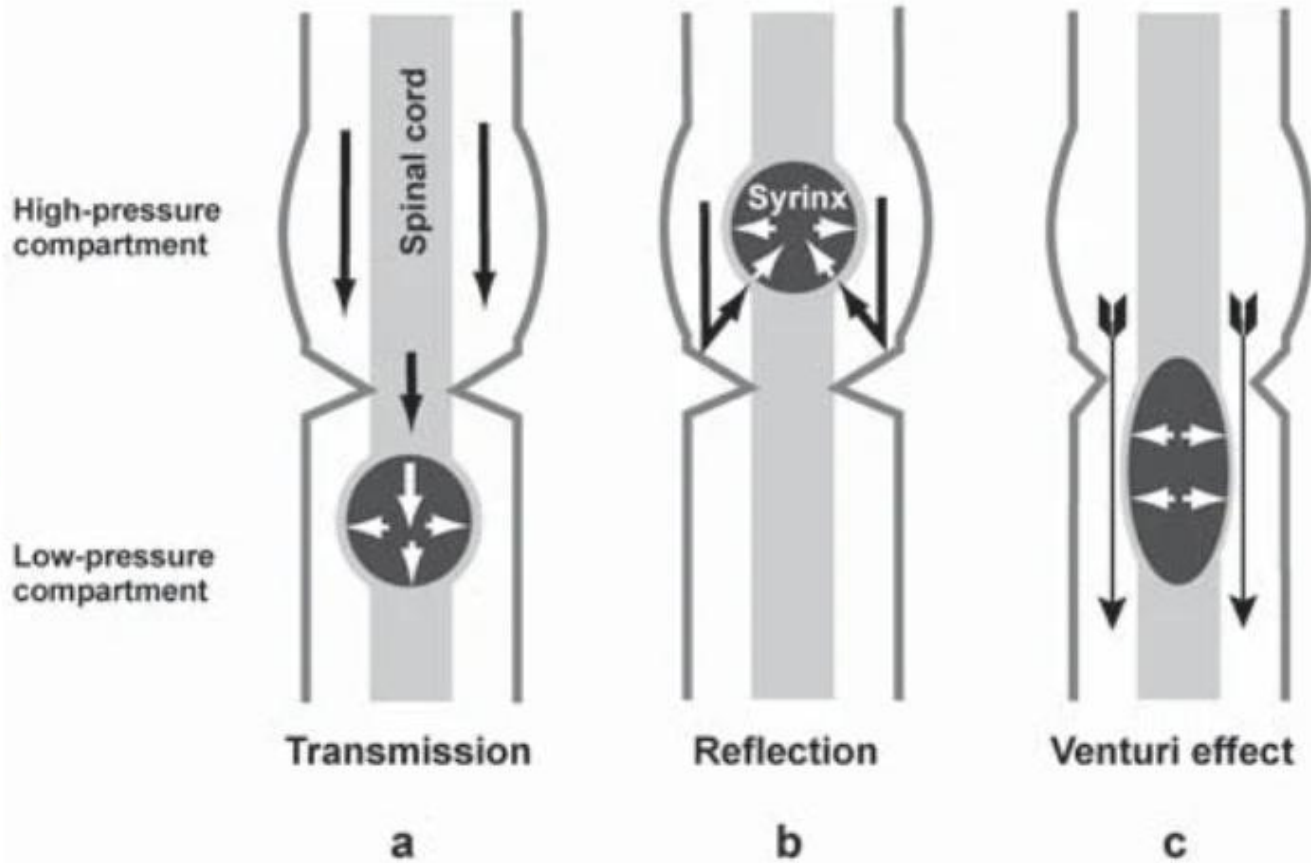


Pathophysiology

- CSF pulsates synchronously with pulse and respiration
- When CSF pulsations encounter partial obstruction it becomes easier for CSF to enter into the SC than to travel along its surface
- Transparenchymal migration of CSF into the cord becomes easier if the cord tissue has broken down due to focal injury in the cord tissue eg myelomalacia/scar

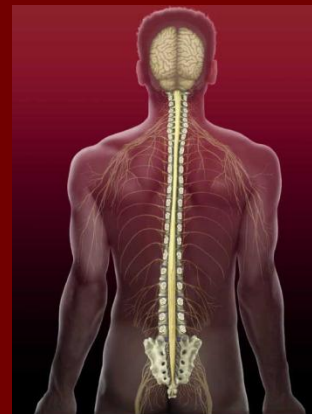


Pathophysiology



Issues in Syringomyelia

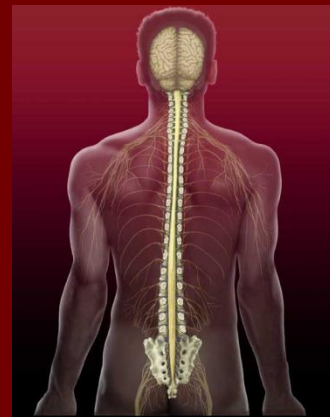
- Natural History:
 - Most progress, a few resolve.
 - 50% + can be stable for 10 years
- Conservative advice
- Medication:
- Surgical indications
- Surgical options:
 - Resection of scar
 - Shunt – from syrinx, or from SAS



Heterotopic ossification

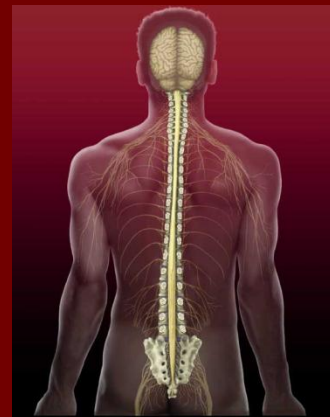
Objectives

- At the completion of this talk, the participant should be able to:
 - Define HO
 - Discuss the pathophysiology of HO
 - Recognize the clinical manifestations of HO
 - Have an approach to the investigation and management of HO



HO Case

- 39 yr old male C7 Quadriplegia January 2012,
- Stiffness hip noted during rehab
- Alk phos 290 Apr 9; CK 238 (can be in the 1000's)
- Xray and bone scan +
- Started bisphosphonates for 6 months daily (PO etidronate). Stopped bisphosphonate
- 3 months later: more hip stiffness
- Second course of Etidronate prescribed





LAO PELVIS



RAO PELVIS

- April 2012 (3 mo post SCI)

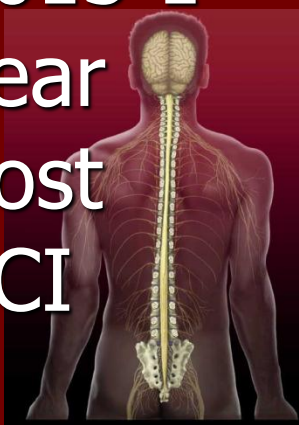


RAO



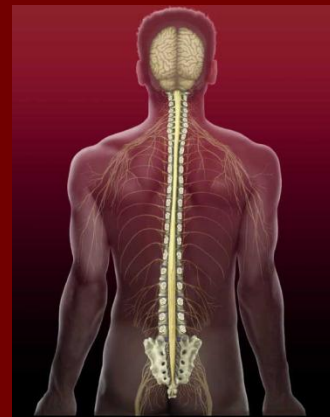
LAO

- January 2013 1 year post SCI



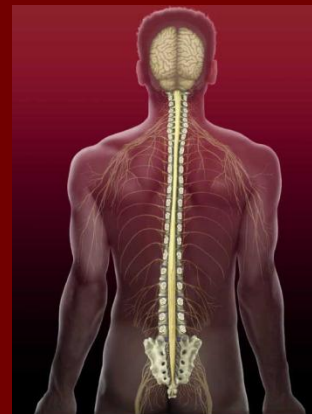
Definition

- HO is the formation of mature lamellar peri-articular bone in soft tissue sites where it is not normally present



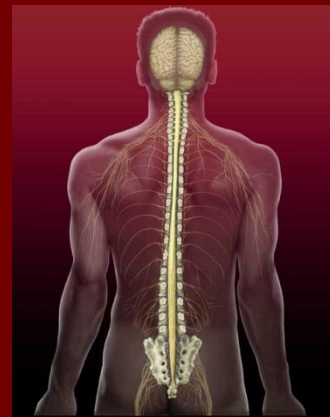
Predisposing Diagnoses

- Spinal cord injury (traumatic and non-traumatic)
- Traumatic brain injury
- Post total hip arthroplasty (and other arthroplasties)
- Stroke
- Burns
- Long bone fractures



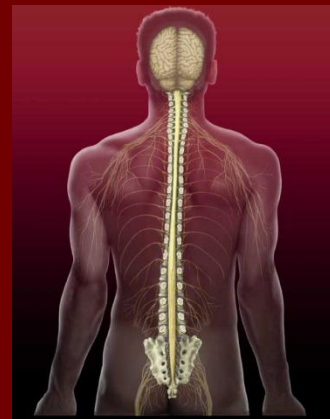
Epidemiology

- Rates from 16-53% occur in the literature, with many people quoting $\sim 50\%$
- In the TBI literature rates vary from 11-76%
- The rate of *clinically significant* HO is in the range of 10-20%
- THA – incidence is $\sim 20\%$
- Burns – incidence is $\sim 1-3\%$



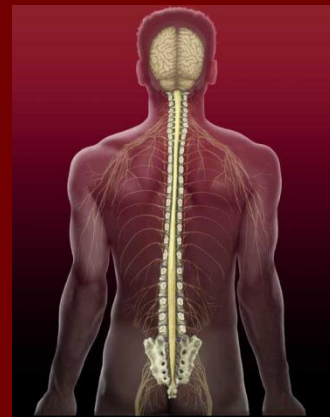
Time Course

- Most SCI related HO appears 1-3 months post injury, with the earliest reports being 20 days and the latest >1 year post-injury
- Final maturation of bone formation usually occurs about 12 - 18 months after it first appears



Risk Factors: SCI

- Higher levels of spasticity
- Complete injuries
- The presence of pressure sores, UTI, and pneumonia
- Age > 30 years



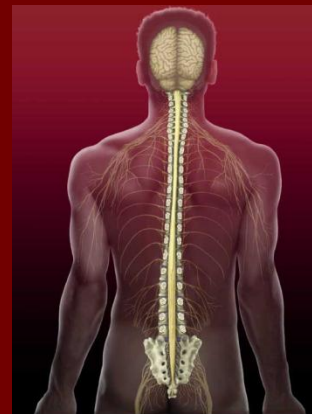
Joint Involvement

- SCI

- Hip>knee>shoulder>elbow

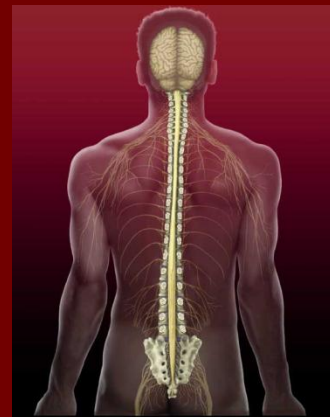
- TBI

- Hip>elbow=shoulder>knee



Pathophysiology

- Not entirely clear
- It appears from animal studies that certain osteoinductive factors stimulate differentiation of soft tissue mesenchymal cells into osteoblasts



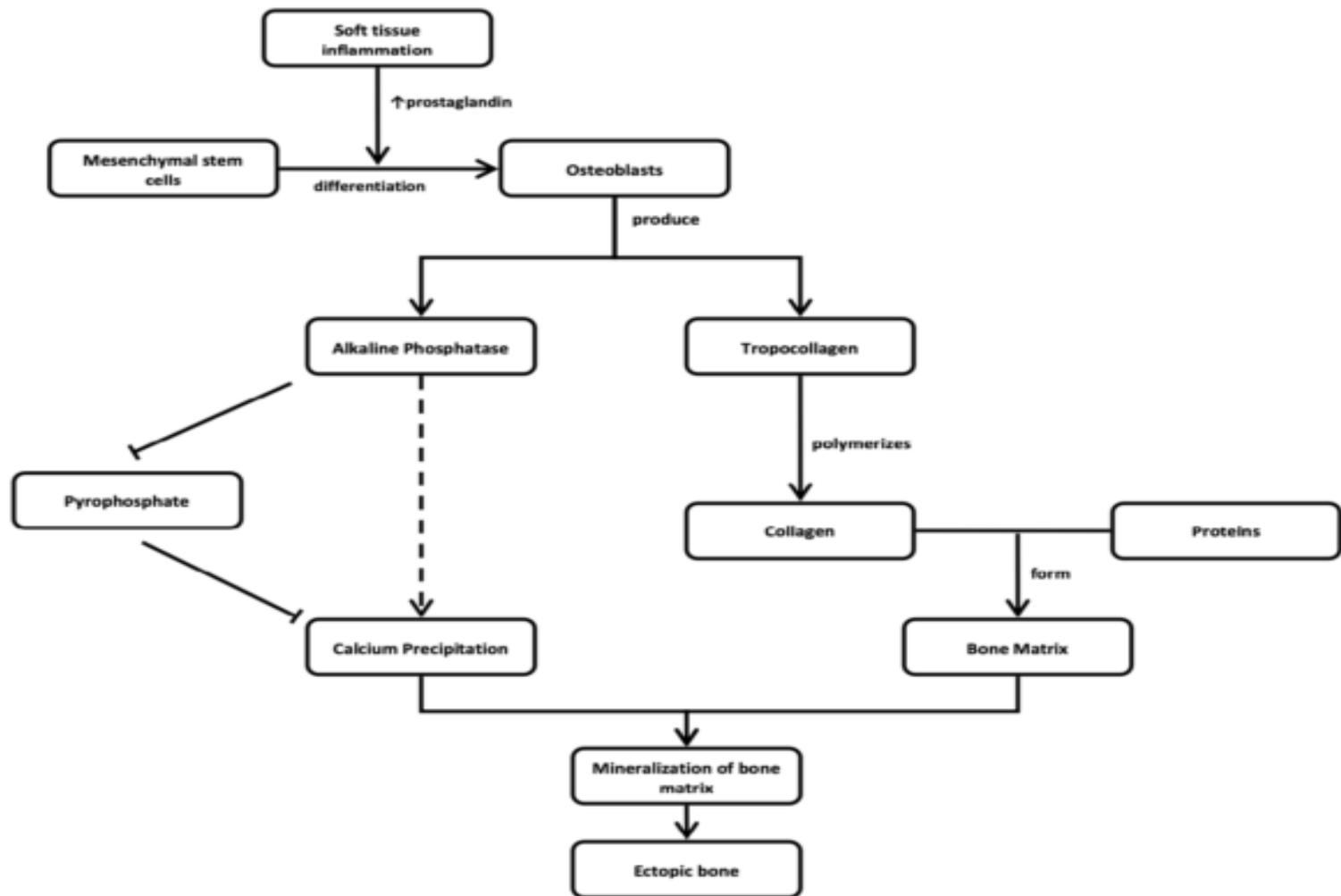
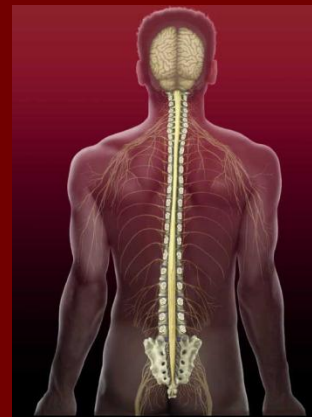


Figure 1. Mechanism for development of heterotopic ossification

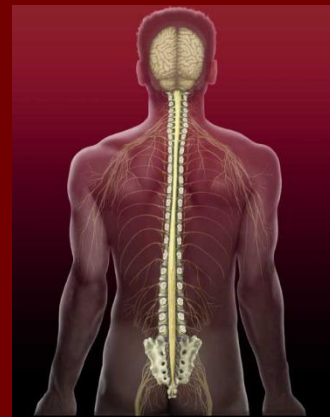
Clinical Presentation

- Most common presentation is painless loss of ROM found on routine exam or during stretching
 - “You know, I think Mr. Smith’s hip is getting tighter – he’s losing some range there.”
- Pain can be reported
- Tissue swelling
- Fever



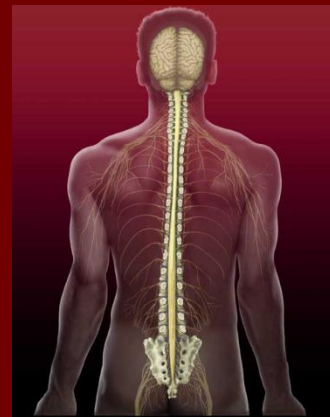
Differential

- DVT
- Cellulitis
- Septic arthritis
- Acute monoarticular arthritis
- Trauma
- Fracture
- Bone tumor



Investigations

- Lab tests are commonly used but are non-specific
 - Serum alkaline phosphatase
 - Serum creatine phosphokinase (CK)
 - Serum ESR/CRP



Investigations

- Serum alkaline phosphatase
 - Rises at 2 weeks, exceeds normal values at 3 weeks and peaks at 10 weeks
 - Returns to normal levels after the HO matures
 - Relatively sensitive but non-specific for HO
 - Levels are also elevated with trauma and fractures
 - Not elevated in acute HO in many patients



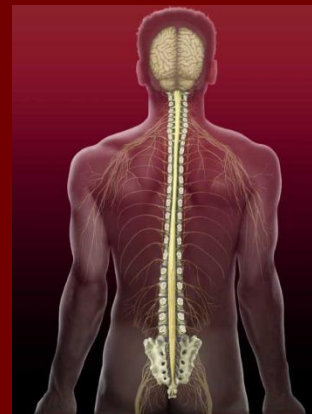
Investigations

- Serum ESR and CRP
 - The serum concentration of CRP correlates better than the ESR with the inflammatory activity of HO after SCI
 - The normalization of CRP is accompanied by a resolution of inflammation in periarticular soft tissue
 - Usefulness of CRP for predicting HO following SCI has not been conclusively established



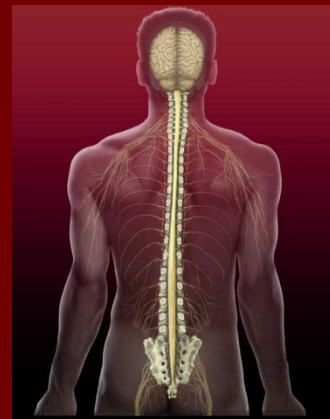
Serum creatine phosphokinase (iKa CK):

- 18 patients with SCI
 - 14 had normal CK; only one developed HO
 - 4 had elevated CK; all developed HO
 - Elevated levels may predict more severe HO



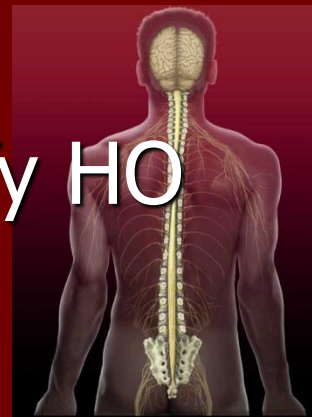
Imaging studies

- Bone scan and U/S are helpful in early diagnosis
- In later stages of bone formation, when ossification has occurred, plain XR's are most often used
- CT and MRI offer more precise delineation of ectopic bone
 - Used in pre-op planning

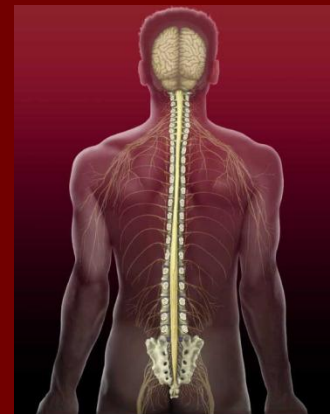
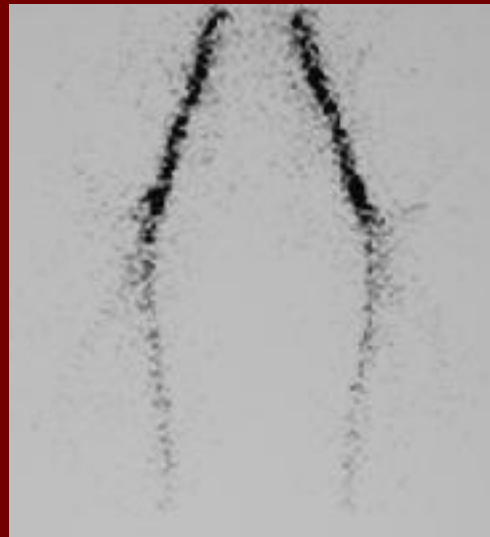


Triple phase bone scan

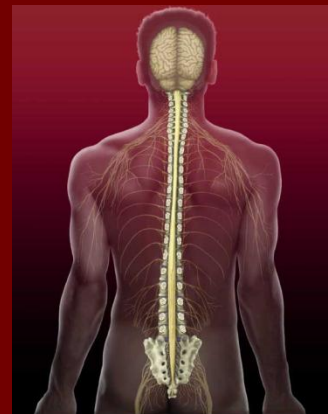
- TPBS will pick up abnormalities within the first 2-3 weeks of bone formation
- A positive bone scan, together with typical clinical presentation, is usually used to confirm the diagnosis
- Xrays become positive 3 weeks after a positive bone scan
- Ultrasound is quicker and can identify HO



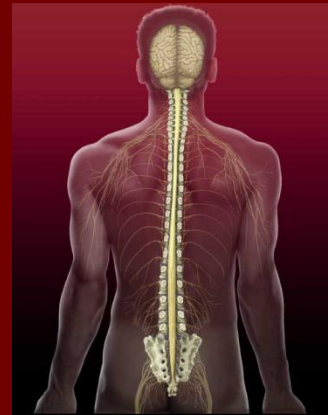
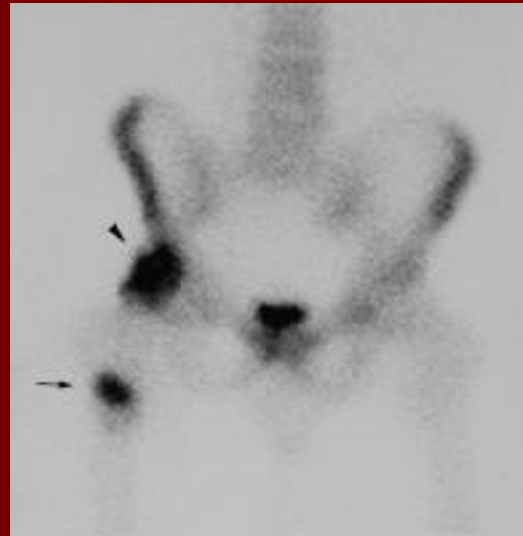
■ Blood flow (Phase 1)

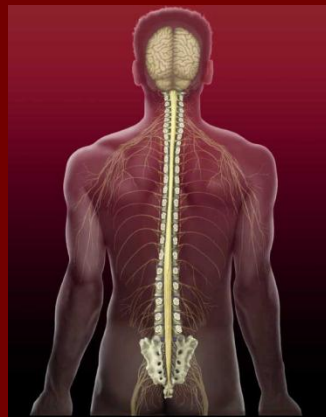


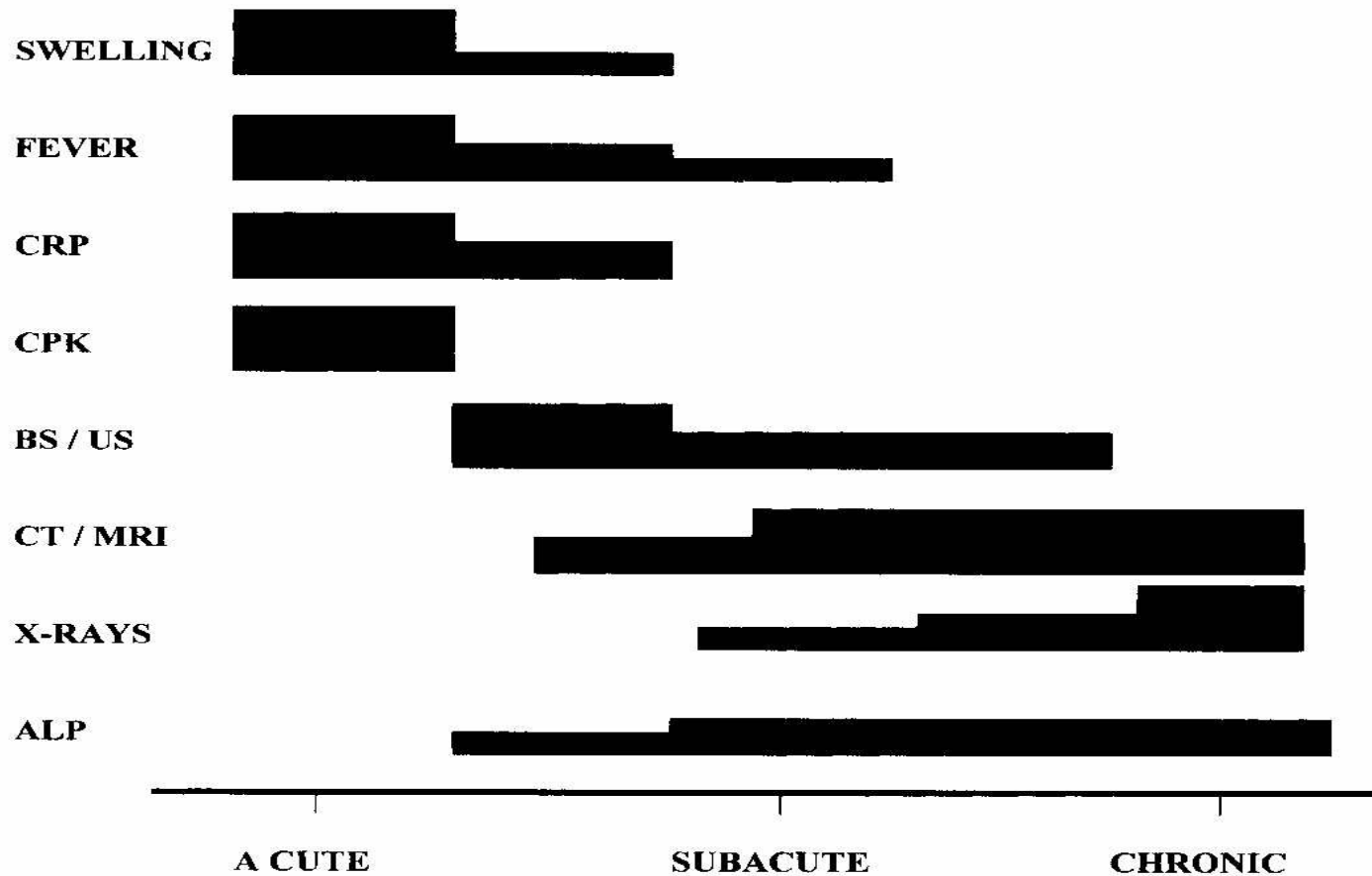
- Blood pool (Phase 2)



- Bone uptake (Phase 3)







STAGE AFTER SCI

Figure 3. Development of clinical findings in HO. BS, bone scintigraphy; US, ultrasonography.



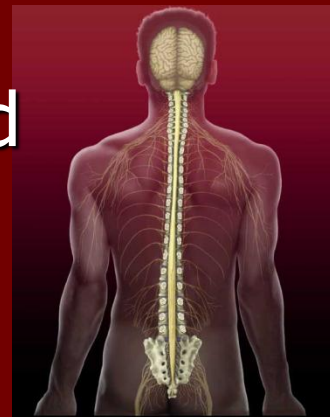
Therapies for prophylaxis

■ NSAIDS

- Short 2-3 week courses started about 3 w post SCI were effective in several studies

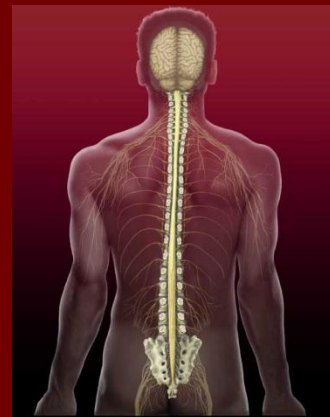
■ Limited evidence

- Warfarin
- Radiation therapy
- Pulse low intensity electromagnetic field therapy (PLIMF)therapy



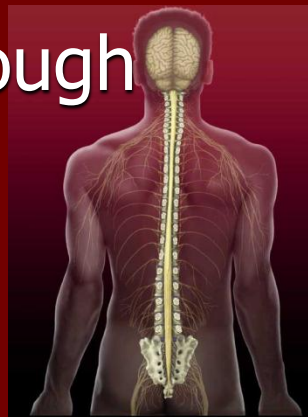
Treatment

- Why do we treat?
- Complications:
 - Pressure sores
 - Joint ankylosis/seating and positioning problems
 - DVT
 - Peripheral nerve entrapment
 - Spasticity
 - Autonomic dysreflexia
 - Pain
 - Fractures



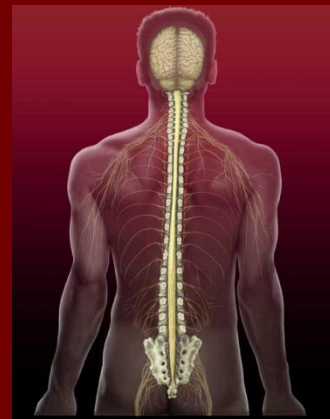
Conservative treatment

- Should not be overlooked
- Stretching and ROM exercises to maintain joint mobility
- ROM does not seem to increase or accelerate the degree or rate of bone formation as once believed although some still feel this is controversial
- There is a theoretical risk of fracture, although no good studies to support this



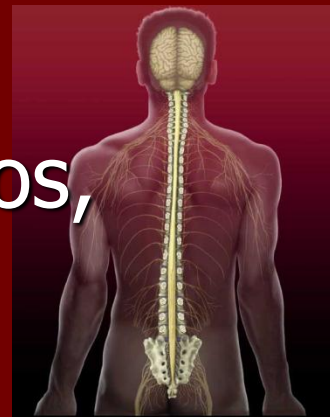
Modalities

- Modalities, particularly cold, may be helpful in symptomatic management
- Heat should probably not be used as it could theoretically increase the rate of bone formation by increasing blood flow to the area



Surgery

- Only proven effective treatment
- About 5% of SCI patients with HO will require surgery
- Timing:
 - after the bone has “matured”
- Goal: to de-bulk the area
- High rate of complications:
 - Bleeding and infection, recurrence
- Secondary prevention post op: bisphos, NSAIDS or radiation, PT/ROM



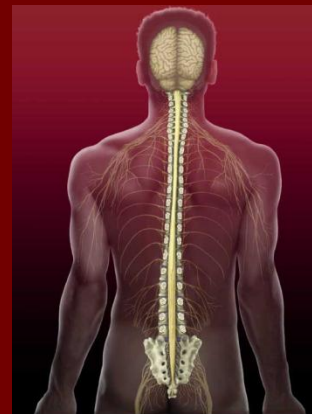
Pharmacologic Treatment

- Limited evidence supports bisphosphonates for treatment of HO in SCI.
 - Potentially effective if started **before** radiographic HO is evident
 - Inhibits growth of HO by inhibiting the transformation of amorphous calcium phosphate into crystalline hydroxyapatite



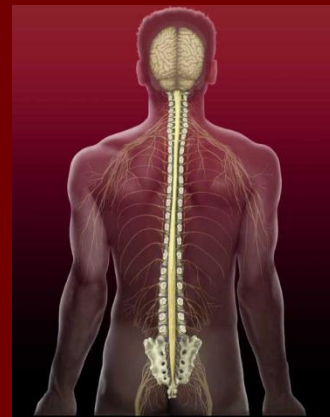
Treatment summary

- The end goal is to limit bone formation and prevent secondary complications
- Effectiveness of current bisphosphonate regimes have not been confirmed
- Primary prevention would be ideal
 - NSAIDs have been studied but aren't used often in our practice



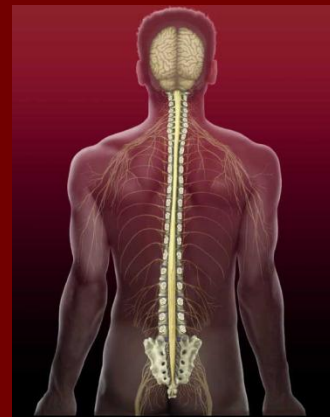
Summary

- HO is a poorly understood phenomenon that consists of the formation of mature bone in the periarticular soft tissues
- It is important in physiatry because it is common after TBI, SCI and after hip arthroplasty and because it can have important functional implications
- Complications are significant



Summary: HO

- Treatment consists of conservative interventions, continuous dose bisphosphonates +/- NSAID's +/- surgical excision
- The goal of treatment is to limit the progression of bone formation and limit functional loss and secondary complications



Thank-you!!

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