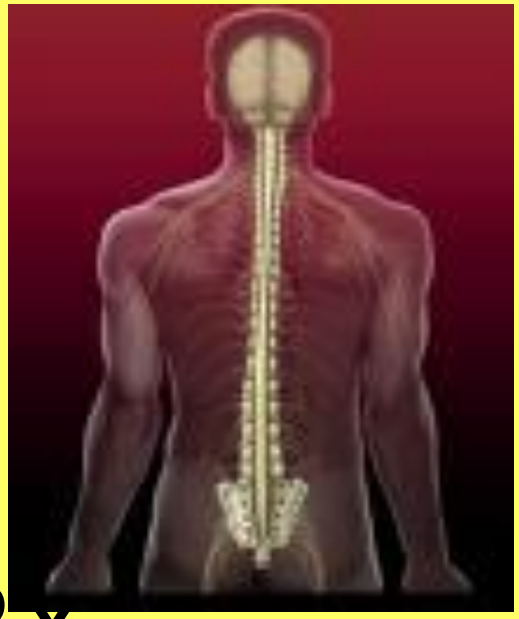
COMPLICATIONS AND PROGNOSIS OF SPINALCORD INJURY



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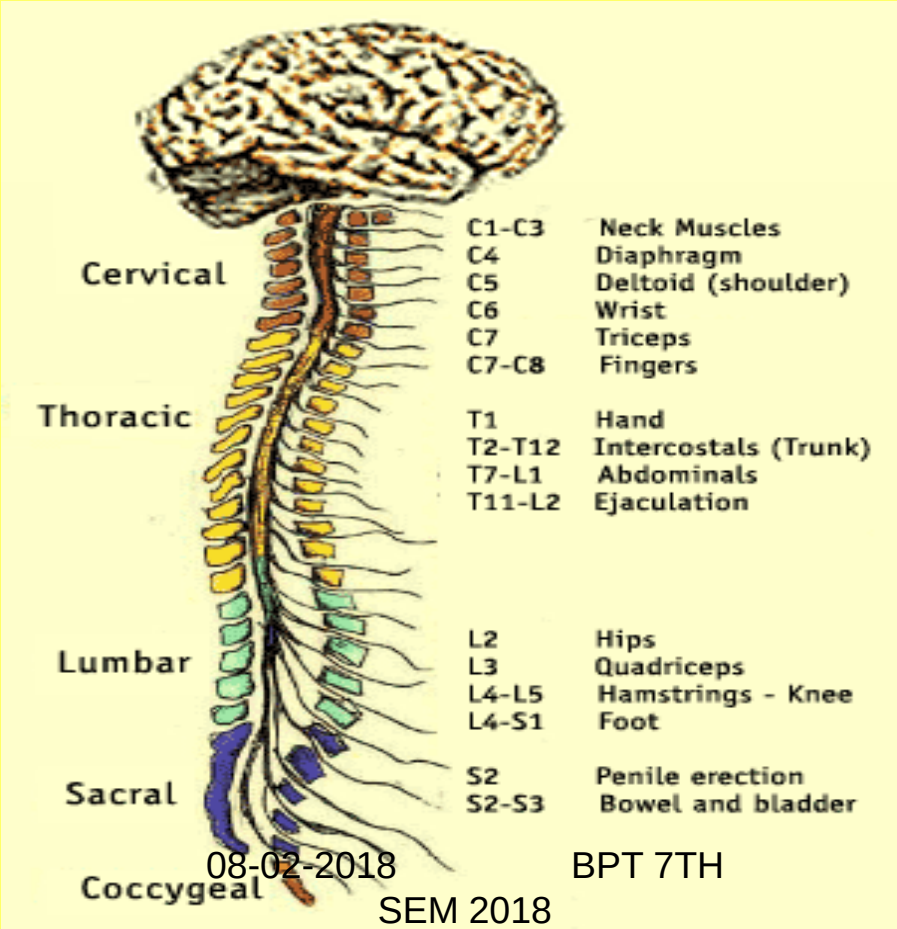
COMPLICATIONS AFTER SPINAL CORD INJURY



A.CARDIOPULMONARY →

1.Alelectasis
2.Pneumonia
3.Pneumothorax &chest injury
4.Pulmonary embolism
5.Empyema
6.Trapped lung
7.Aspiration
8.Ventilatory failure
9.DVT
10.Orthostatic hypotension
11.Apneic bradycardia

B.INTEGUMENTARY →

1.Pressure sore
2.Paronychia
3.Acne vulgaris
4.Seborrheic dermatitis
5.Chronic folliculitis
6.Nail changes

C.NEUROMUSCULAR → 1.Spasticity
2.Autonomic dysreflexia

D.MUSCULOSKELETAL → 1.Heterotopic ossification
2.Osteoporosis
3.Skeletal muscle atrophy
4.Contracture
5.Charcot spine
6.Iliolumbar syndrome
7.Iliopsoas abscess

E.GASTROINTESTINAL → 1.Constipation
2.Acute abdomen
3.Ileus
4.Gastritis& ulcer
5.Haemorrhoids
6.Cholelithiasis
7.Pancreatitis

F.VOIDING DYSFUNCTION ⟶

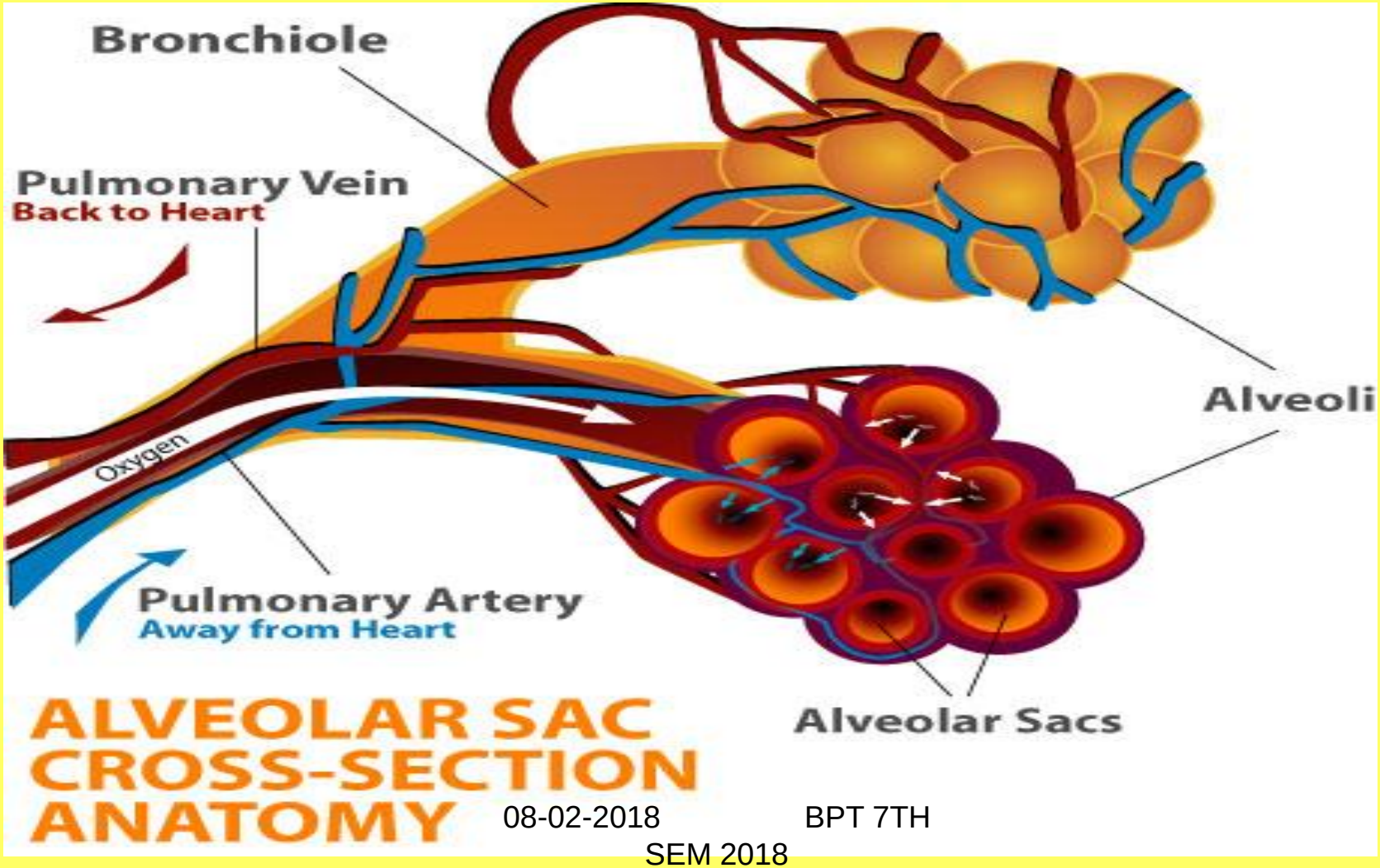
1.Urinary tract infection
2.Hydronephrosis
3.Vesico urethral reflux
4.Renal calculi
5.Renal deterioration
6.Bladder cancer

G.OTHERS ⟶

1.Pain
2.Post traumatic syringomyelia
3.Anemia
4.Obesity
5.Pregnancy complications
6.Depression

A leading cause of mortality in patients with tetraplegia and high paraplegia. Most frequently occur in high SCI between **C1 andC3**, phrenic nerve innervations. But this problem persist with different level of injury .**On examination**, breathing pattern is disturbed, increase in RR , inadequate chest expansion, reduction of m/s strength. Impairment lead to reduction of cough effectiveness, ability to expel secretions and thus susceptible to **retention of secretion and lead to infections**. Usually artificial ventilator or phrenic nerve stimulator is required to sustain life.

PULMONARY COMPLICATIONS



Normally ventilation is coordinated action of muscle on rib cage.

INSPIRATION

(Diaphragm, external intercostal, scalene, sternocleidomastoid, trapezius, pectoralis m/s)

↓

During inspiration, diaphragm contracts and descends, the intercostal elevates ribs and increases A-P &lateral diameters of thorax.

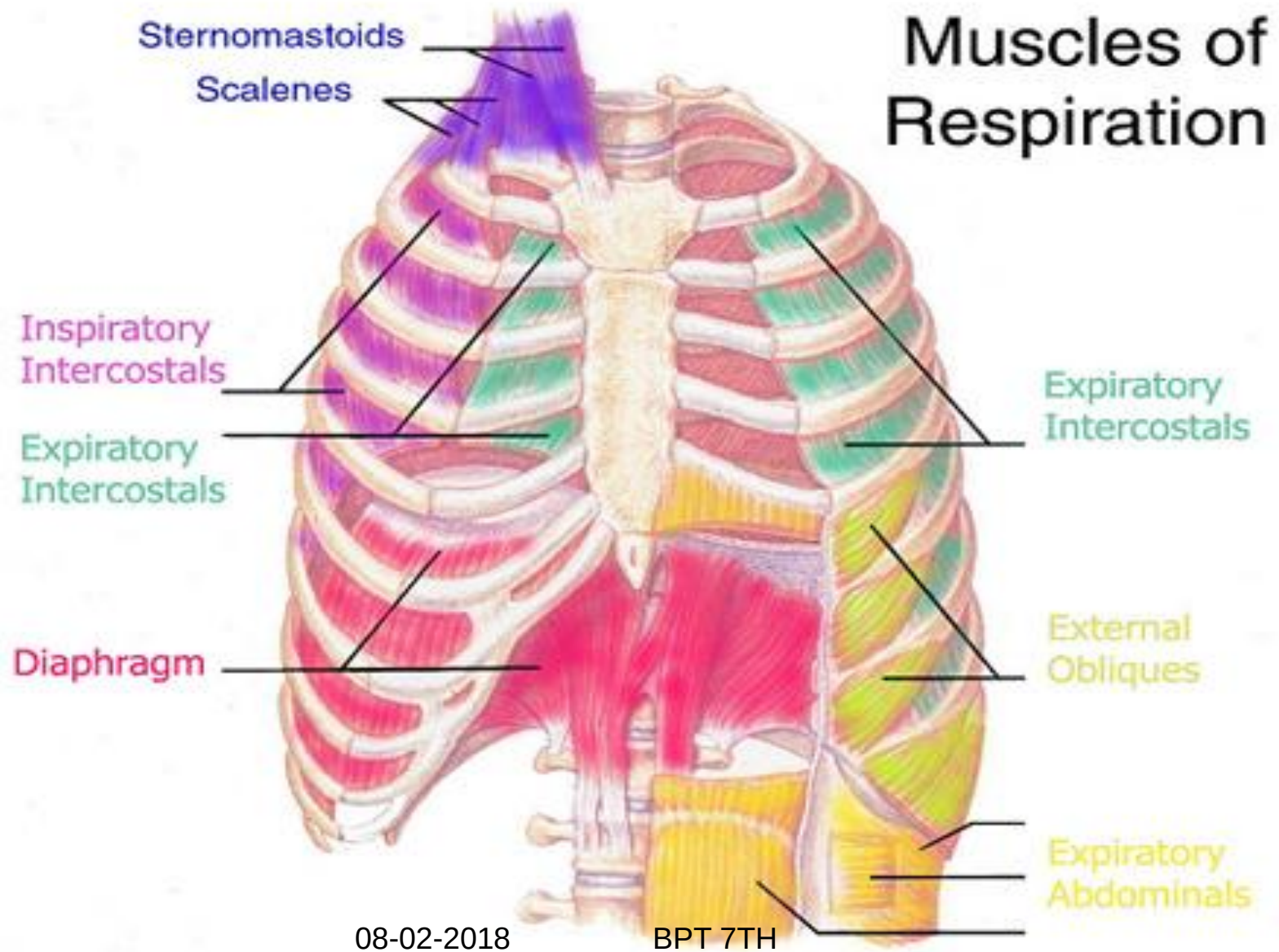
EXPIRATION

(abdominal, internal intercostal)

↓

During expiration, passive elastic recoil of lungs and ribcage, decreases IA pressure & increase IP pressure, air moves out. M/S support abdominal viscera, maintain diaphragm position. Intercostal depress rib and compress chest wall for forceful expulsion.

Figure 1.


Muscles of Respiration
Sternomastoids
Scalenes
Inspiratory Intercostals
Expiratory Intercostals
Diaphragm
Expiratory Intercostals
External Obliques
Expiratory Abdominals

1. Breathing pattern :-
    - Normally 2 chest: 2 diaphragm
    - C2 lesion:-4 neck breathing pattern.
    - C3,C4 lesion:-2 diaphragm,2 neck or 1 diaphragm and neck.
    - C6-C8 and T2-T4:- 4 diaphragm.
    - T6-T12 :- 2 chest, 2 diaphragm.

2. RR:- Increase in case of weakness of diaphragm to maintain ventilation.
3. VC:- C3-C4 --- 15% normal
   C6-C8 ---20% normal
4. Cough function
    - Function cough(T10-T12)
    - Weak (T4-T8)
    - Non functional (above T4)

PARALYSIS OF IC

Decrease in chest expansion &lowered inspiratory volume

HYPOVENTILATION

PARALYSIS OF ABDOMINALS

Diaphragm assume low position, lack abdominal pressure, decrease expiratory volume.

PARALYSIS OF EXTERNAL OBLIQUES

Lack of rib depre -ssion &chest wa -ll compression for forceful expulsion

Decrease cough effectiveness &ability to expel secretion.

RETENTION OF SECRETION
PULMONARY INFECTION

Due to above, the pulmonary complications include:-

1. Alelectasis
2. Pneumonia
3. Pleural effusion
4. Empyema
5. Abdominal complications.

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MANAGEMENT

Treatment methods focus on :-

- M/S strength improvement
- Improve chest mobility
- Bronchial hygiene

1.Muscle strengthening:-

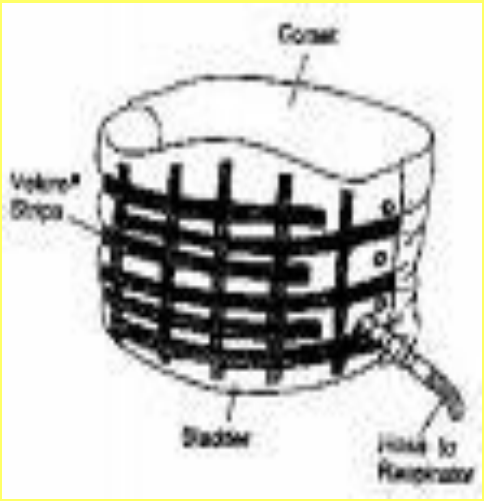
- Patient having **fair + strength** of diaphragm----

Progressive resistive exercises

Patient in supine lying, weight pan is placed between xiphoid process and umbilicus and allow full excurs -ion of diaphragm with epigastric rise. 5 pounds for 15 mins. is sufficient for patient having full diaphragm innervation.

•Patient having **fair strength** of diaphragm
15 degree head down inclined position where force of abdominal content acting against diaphragm.

•Patient having **poor strength** of diaphragm.
Pneumobelt (active assistive device can be used)



2. **Abdominal support:-**

•Pneumobelt:-

- use by patient having weak diaphragm.
- It is a corset with an inflatable bladder.
- Pt in sitting, inflation of the bladder cause exhalation by **pushing abdominal contents in and up pushing diaphragm into optimal ascended position** within the thoracic cavity.

- Corsets:-
- Used by pt. having weak abdominal.
- It helps to compensate for **weak or absent abdominal m/s** by supporting abd. **Contents &displace diaphragm to higher resting** position in the thoracic cavity.
- Lie just over last 2 floating ribs and cover iliac crest over ASIS b/l.

Estenne and DeTroyer found that ues of abd. Binder eliminate in RV that occur when pt. assume the seated position from supine lying.

NOT applied high ↘

- Reduce chest mobility
- Not able to support abdomen

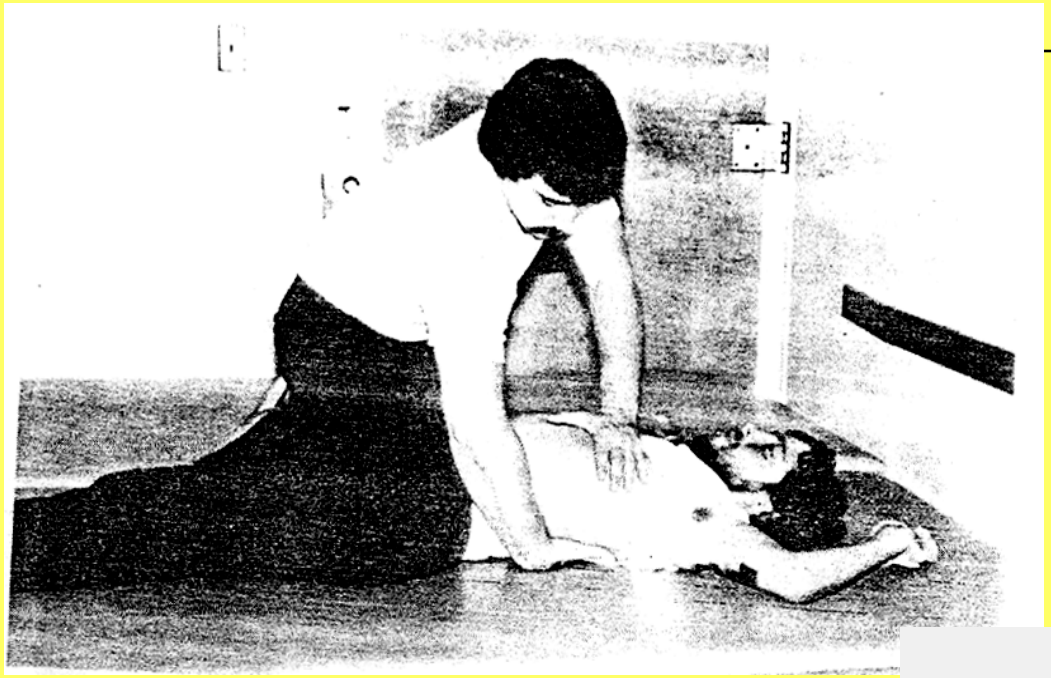
3. Chest mobility:-

- DBE:- Pt having voluntary chest expansion range of greater than 2 inches.
- Air shift :-
  - Pt having good chest wall ROM and intercostals weakness
  - Pt in supine lying, one hand on epigastric area &other hand on the upper part of the chest. The therapist ask to take deep breath and hold it and then tell pt. to suck the stomach in and move air to upper part of the chest.
  - Chest expansion increases from 0.5 to 2 inches.

- Manual chest stretching

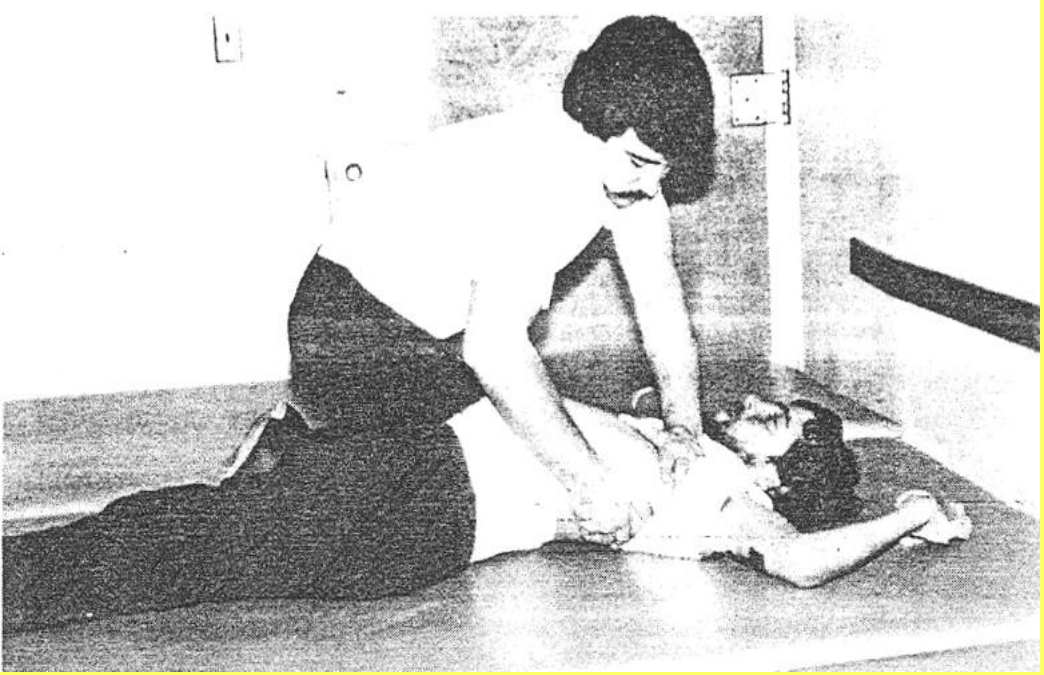
4.Bronchial hygiene :- Coughing techniques:

- Manual
- Self manual

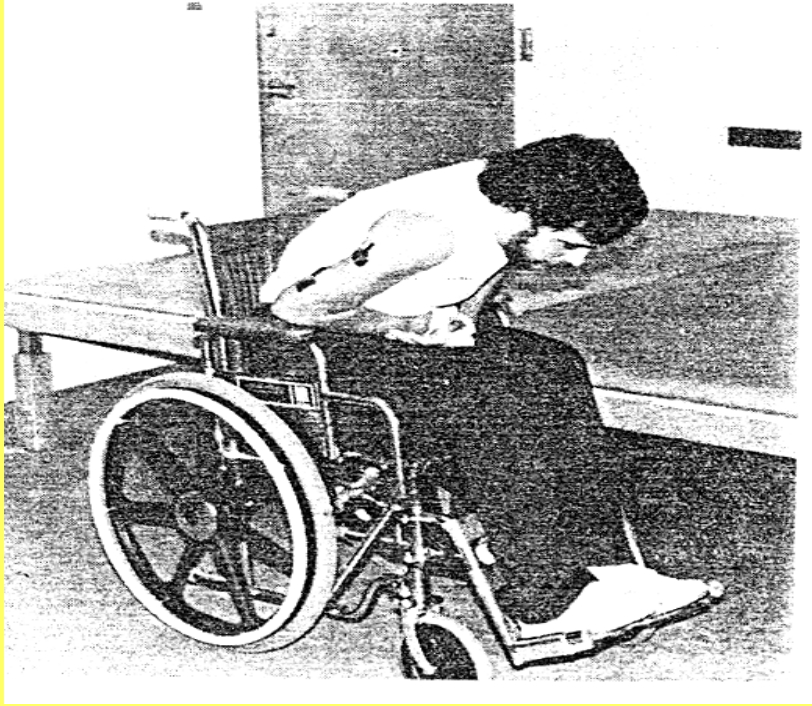


STARTING POSITION FOR MANUAL CHEST STRETCHING WITH ONE HAND UNDER PT.RIBS &OTHER ON TOP OF RIBS.

ENDING POSITION OF HANDS AFTER APPLYING A WRINGING-TYPE MOTION TO RIBS FOR MANUAL CHEST STRETCHING



LAST HAND POSITON AFTER PROGRESSING UP CHEST FOR MANUAL CHEST STRETCHING WITH TOP HAND JUST INFERIOR TO CLAVICLE



MANUAL COUGHING

08-02-2018 BPT 7TH SEM 2018

ATELECTASIS

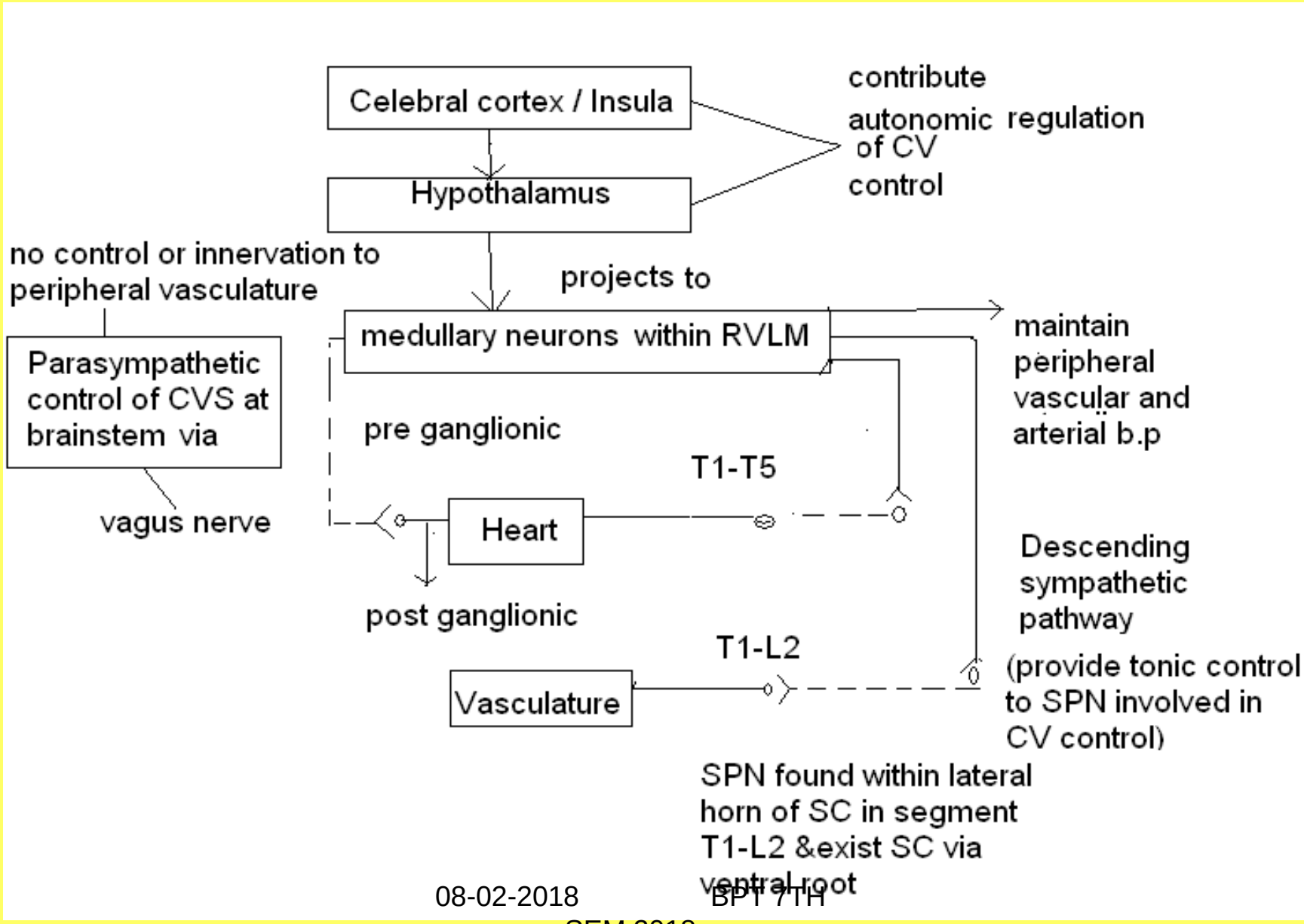
In study of patients received at a tertiary care hospital, it was found that 60% of the pt. admitted on ventilator had this problem. Effective methods for prevention includes use of CPAP with use of either a mask that seals around the nose or pillow like nasal tubes that fit into nares. TROMANS et al reported on efficacy of CPAP in preventing atelectasis during acute cases.

ROCKY MOUNTAIN REGIONAL SPINAL INJURY SYSTEM PROTOCOL for person with reduction of atelectasis.

A complication which is a challenge for managing the unstable blood pressure in SCI patient. There is a **decrease in systolic b.p of 20 mm** Hg/more or in diasystolic b.p of **10mm Hg/more** during assumption of upright posture from supine position. Usually occur in high level of injury and less in **below T6 or incomplete injuries** due to disruption of supra spinal control to splanchnic bed &major capacitance vessels. Pt. on assuming upright posture, c/o, light headiness, dizziness, nausea, syncope, numbness around face, pallor. This complication associated with fatigueness &cognitive deficit.

Orthostatic hypotension

Normally b.p. and h.r are controlled by coordinated input from sympathetic and parasymt.



Following SCI, disruption of descending spinal CV pathway, resulting in sympathetic hypoactivity (low resting b.p, disturbed reflex control). This may be due to:

- Loss of supraspinal control
- Reduced sympathetic tone below the lesion.
- Morphologic changes in sympathetic neuron. (atrophy of SPN)

Predisposing factor to orthostatic hypotension :

- Sympathetic NS dysfunction
- Altered baroreceptor sensitivity
- Lack of skeletal m/s pump
- CV disconditioning
- Altered salt and water balance

In able bodied pt. during tilting , there is decrease in bp that is sensed by aortic and carotid baroreceptors which causes increase in sympathetic outflow resulting in tachycardia and vasoconstriction.

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In SCI, due to interrupted efferent sympathetic activity, no increase in sympathetic outflow, rise in nor epinephrine & epi nephrine.
There is no or little rise in HR to counter balance rise in bp. Lack of m/s tone cause peripheral and splanchnic bed pooling, reduce cerebral flow and decreased VR to heart.

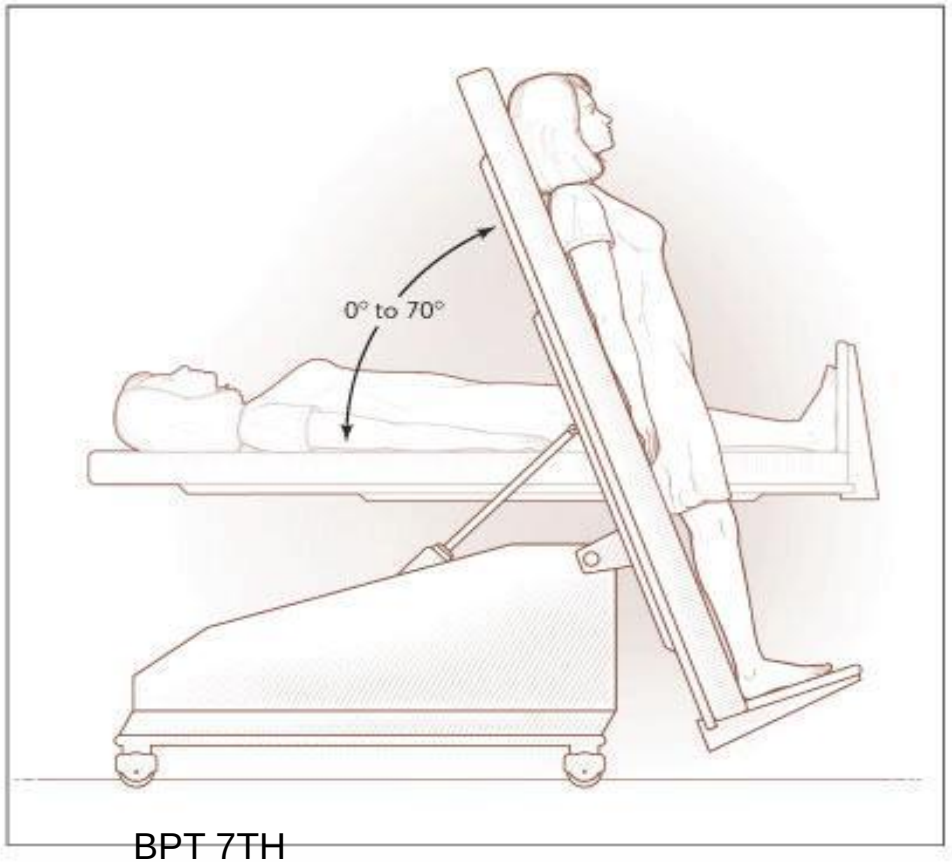
MANAGEMENT

Non-pharmacological

- Advice and avoidance of ppting factors.
- Increased salt and fluid intake.
- Abdominal compression bandage/support stocking.
- Sleeping with bed head raised by 10-20 degrees,then progress to reclining WC with elevating leg rests &use **TILT TABLE**.

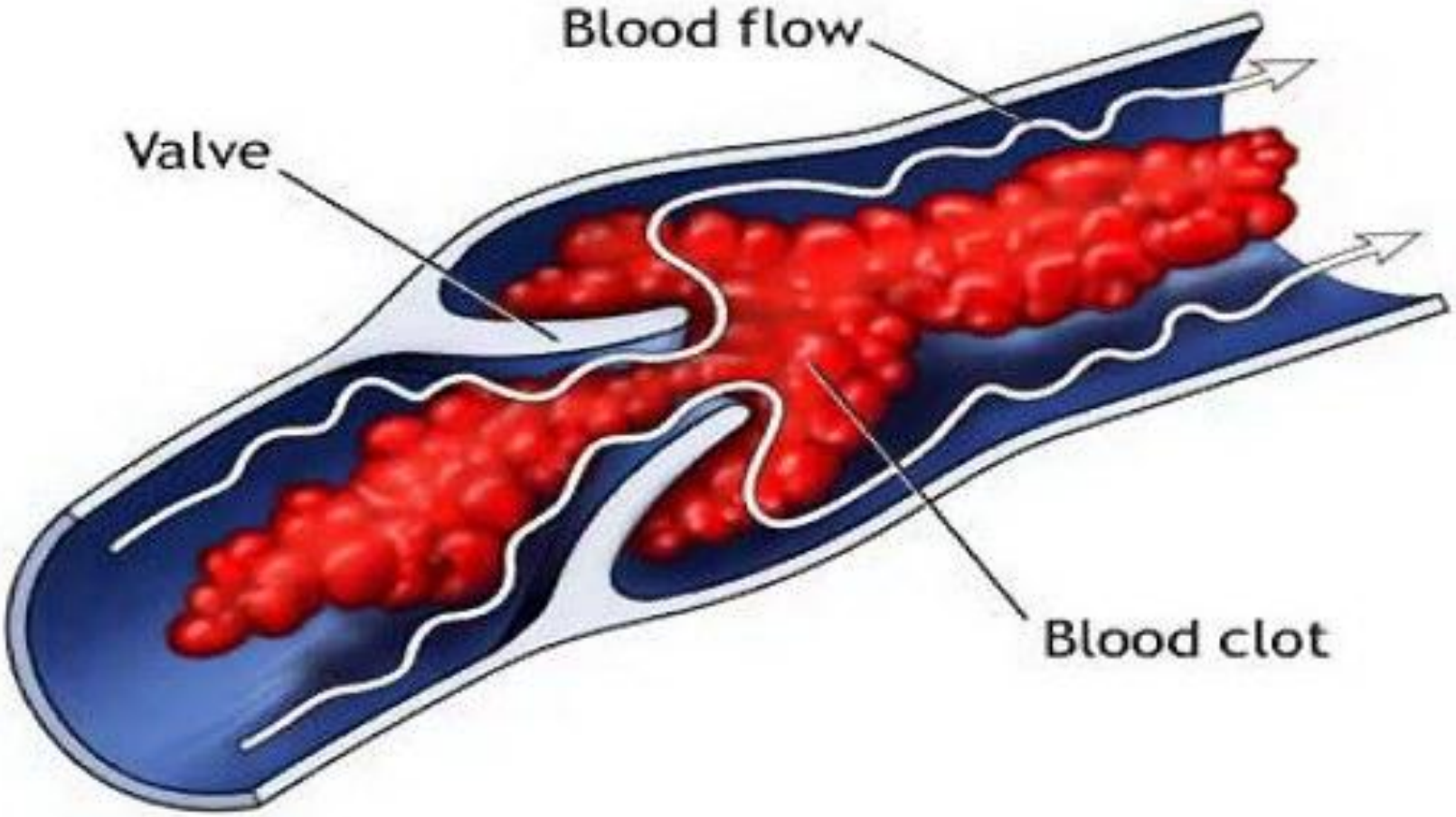
Pharmacological

- Fludrocortisone
- Midodrine



A life threatening complication occur within **1st two months** following injury. There is development of an abnormal blood clot i.e. thrombus within a vessel and cause death where clot dislodge and block pulmonary vessels. **On examination** there is swelling, erythma, heat pain, cyanotic discolorations of the affected limb. Often misleaded with early ectopic bone formation and long bone #. But venous flow studies, venography diagnose this complication.

DVT



Normally, the procoagulants (thrombin) formed are rapidly mixed with large quantities of blood and removed from liver.In case of SCI pt., due to loss of normal “pumping” mechanism provided by active contraction of LE musculature. This slows the flow of blood, thrombin in higher concentration develop in localized area, lead to thrombus formation.

Prolonged pressure, immobility, loss of vasomotor tone lead to DVT. Risk factors in SCI includes paralysis,prolonged immobility,trauma.

MANAGEMENT

This focus on prevention

Hull recently reviewed the literature & reported that greater than 70% of the total incidence of acute DVT was noted in acute SCI pt. who were not on prophylactic heparinization.

• Prophylactic anticoagulant therapy initiated following acute onset of injury and continued for 2-3 months or for up to 6 months.

•Turning program to avoid pressure over large vessels.

•Passive ROM ex. and early mobilization.

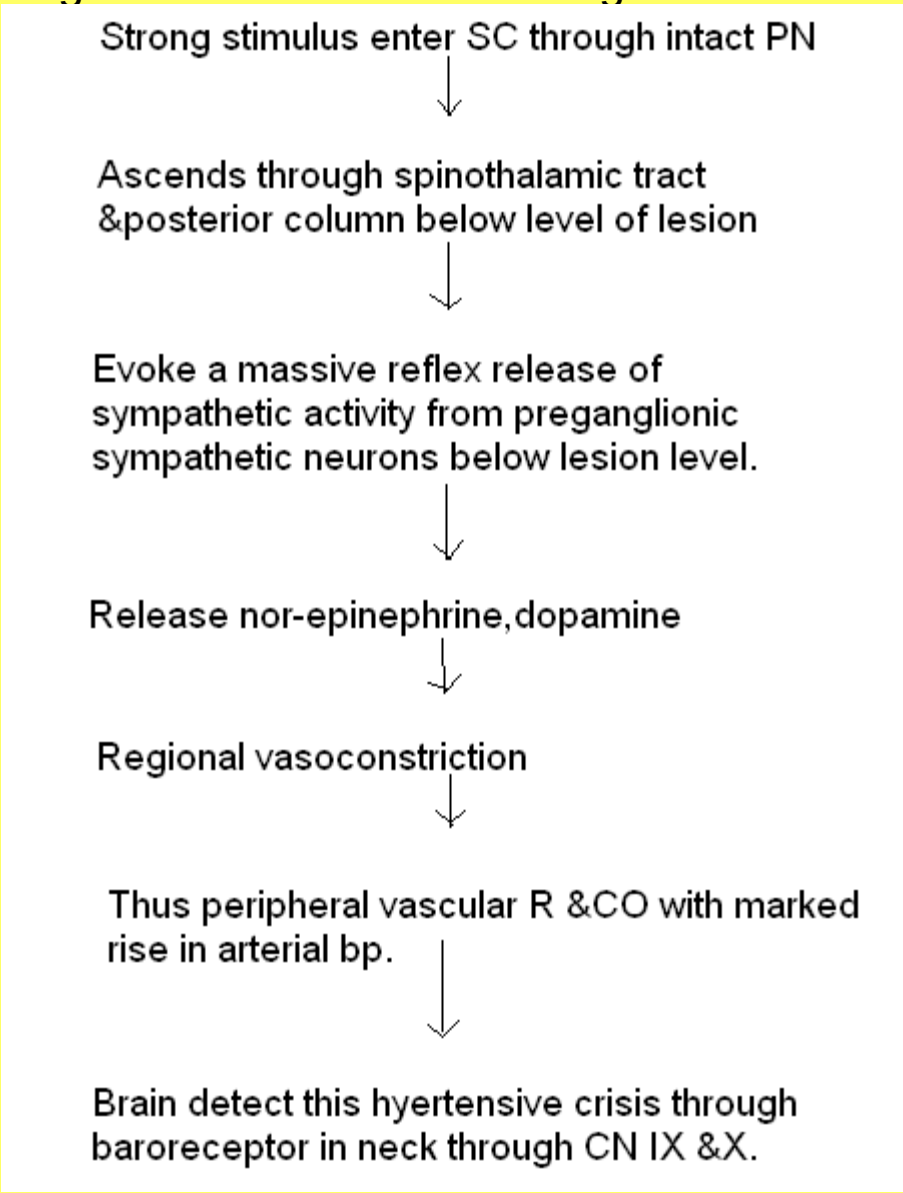
•Elastic support stocking.

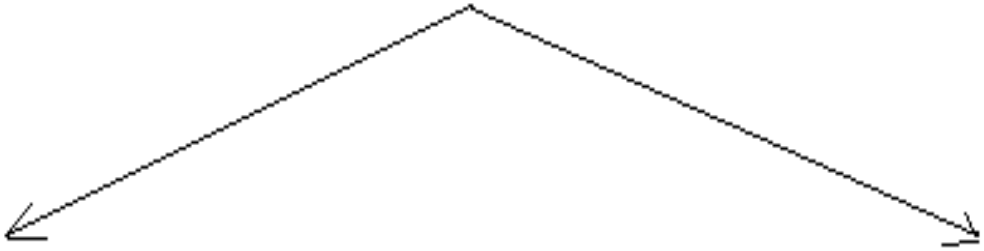
•Positioning of LE to facilitate venous return

A medical or clinical emergency unique to persons with spinal cord injury with **lesion at or above T6.Usually** may not occur until 2 or 3 years post injury. While doing exercises, patients c/o **head -ache, sweating** accompanied by **arterial b.p with bradycardia**. The main cause of this complications includes **bowel &bladder distension,** DVT, spasticity, ROM ex., pregnancy, labor, position changes etc. O/E there is systemic HT, bradycardia, anxiety, sweating, flushing, nasal congestion (above or around level of injury). Pallor, piloerection, bladder sphincter contraction (below the level of injury). Seen in both complete &incomplete lesion.

AUTONOMIC DYSREFLEXIA,HYPERREFLEXIA, SPASTICITY,CRISIS,MASS REFLEX,SYMPATHETIC HYPERREFLEXIA,PAROXYSMAL HYPERTENSION

Strong stimulus enter SC through intact PN



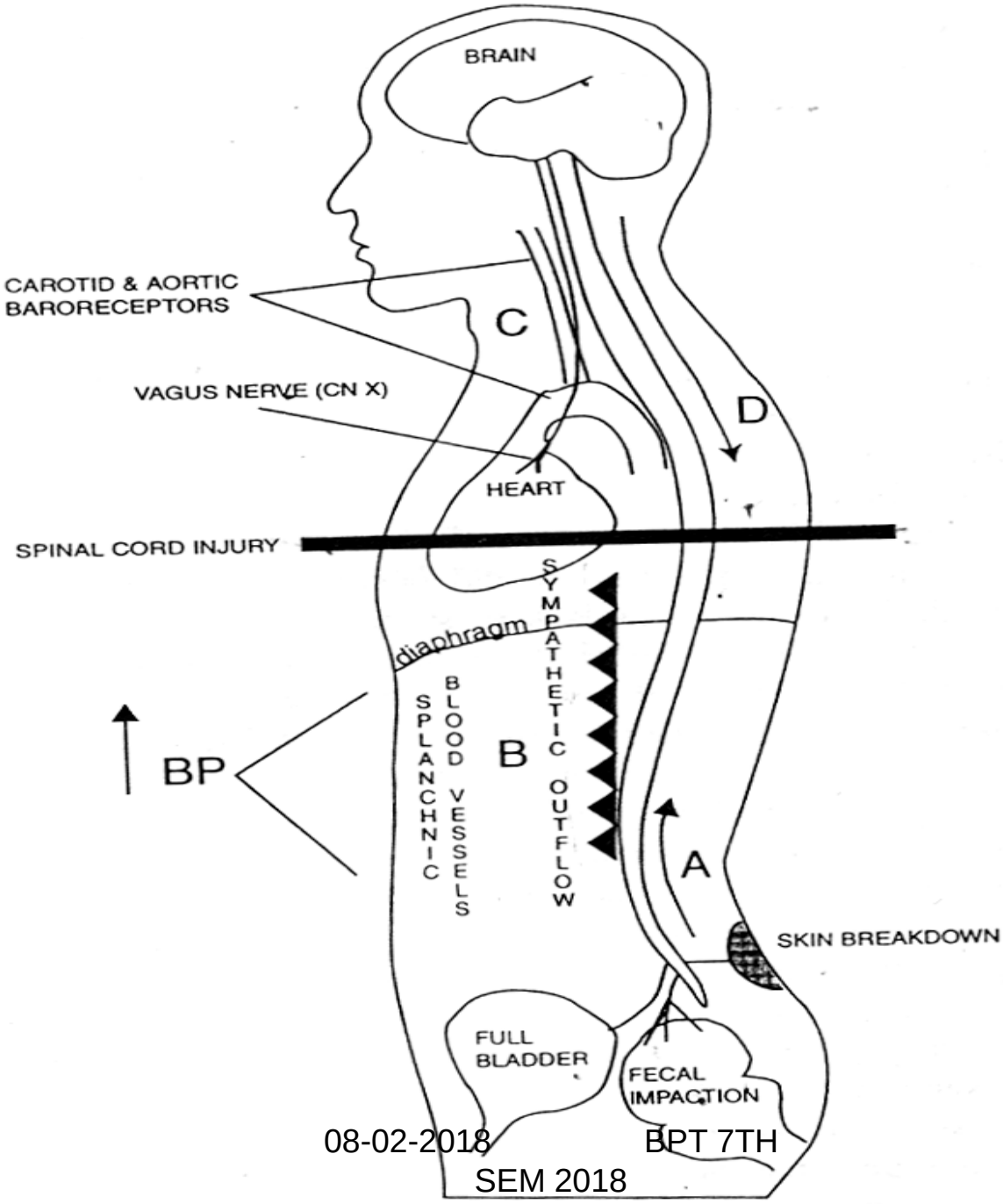


Brain attempt to shut down sympathetic surge by sending desending inhibitory impulse.

Brain attempt to bring down peripheral bp,hr slowing through intact vagus nerve.

But inhibitory impulses donot reach most sympathetic outflow level coz' of SCI at T6& above.

Compensatory bradycardia


BRAIN
CAROTID & AORTIC
BARORECEPTORS
C
VAGUS NERVE (CN X)
D
HEART
SPINAL CORD INJURY
SYMPATHETIC OUTFLOW
diaphragm
BLOOD VESSELS
SPLANCHNIC
B
BP
A
SKIN BREAKDOWN
FULL
BLADDER
FECAL
IMPACTION

MANAGEMENT

- Any symptoms suspected, **bp should be checked**.
- Patient should be upright, elevate head of bed to **45 degrees with maintained cervical tractions & body jacket** if any.
- Straighten all tubing &empty bag.
- If bladder is distended, apply **tapping over the bladder**, pulling pubic hair.
- If bowel distension, Nupercainal ointment inserted into the rectum(5min wait).
- Daily skin care, avoidance of infections.

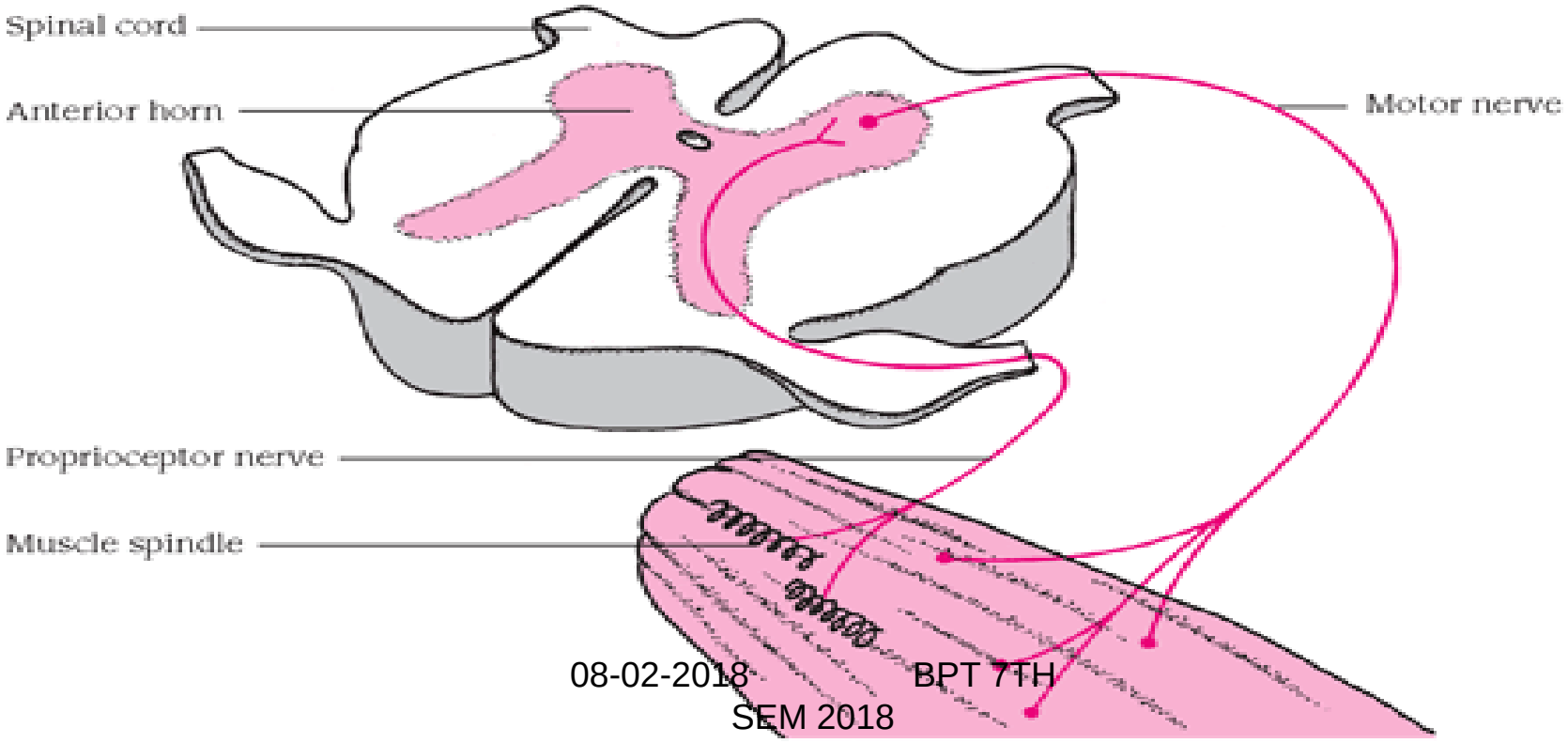
A person with SCI face this complication which occur during **1st 6months & plateau reached 1year after injury** after spinal shock phase. This complication may hinder the rehabilitation program to proceed but helps the patient to stand due to presence of this in lower limb. **On clinical examination** the part is positioned in abnormal pattern, increase m/s tone, increase tendon jerk with clonus. This result from release of intact reflex arc from CNS control in SCI patient. It is not a problem that is separated from SCI, it is inseparable from the problem of m/s weakness &is **part &parcel of UMN disease.**

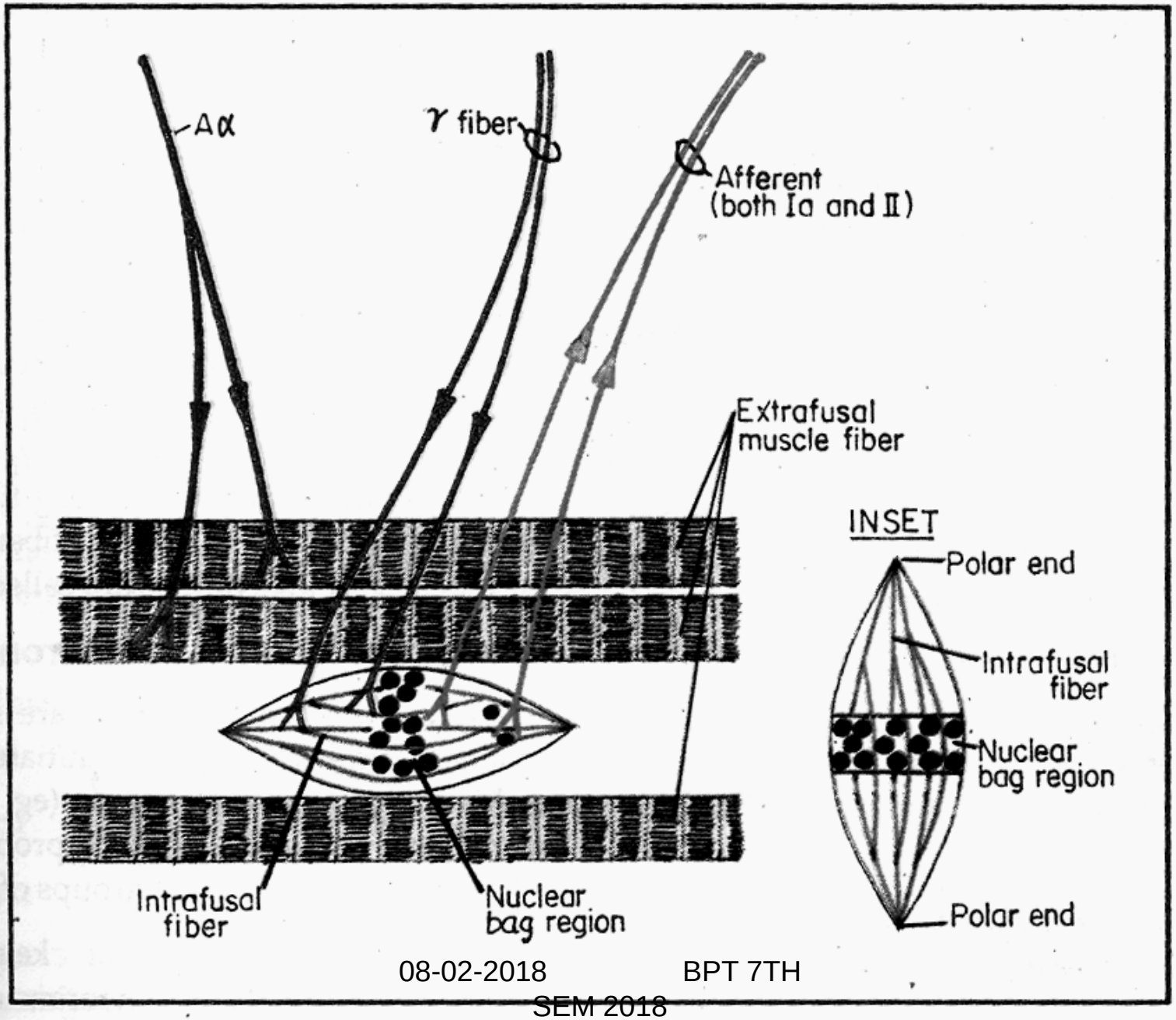
SPASTICITY

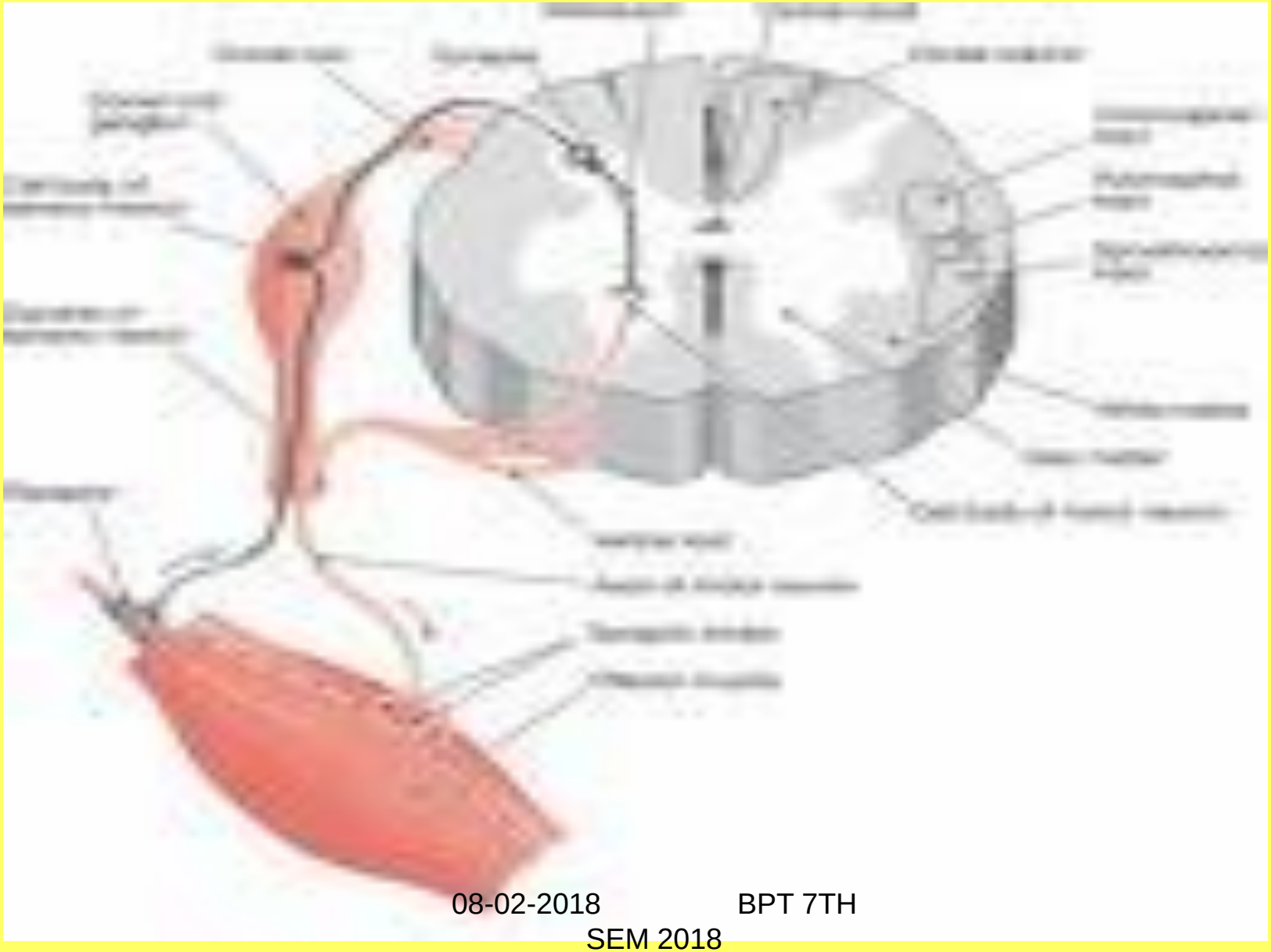
How spasticity develops

Motor activity is controlled by pyramidal and extrapyramidal tracts that originate in the motor cortex, basal ganglia, brain stem, and spinal cord. Nerve fibers from the various tracts converge and synapse at the anterior horn in the spinal cord. Together, they maintain segmental muscle tone by modulating the stretch reflex arc. This arc, shown in simplified form below, is basically a negative feedback loop in which muscle stretch (stimulation) causes reflexive contraction (inhibition), thus maintaining muscle length and tone.

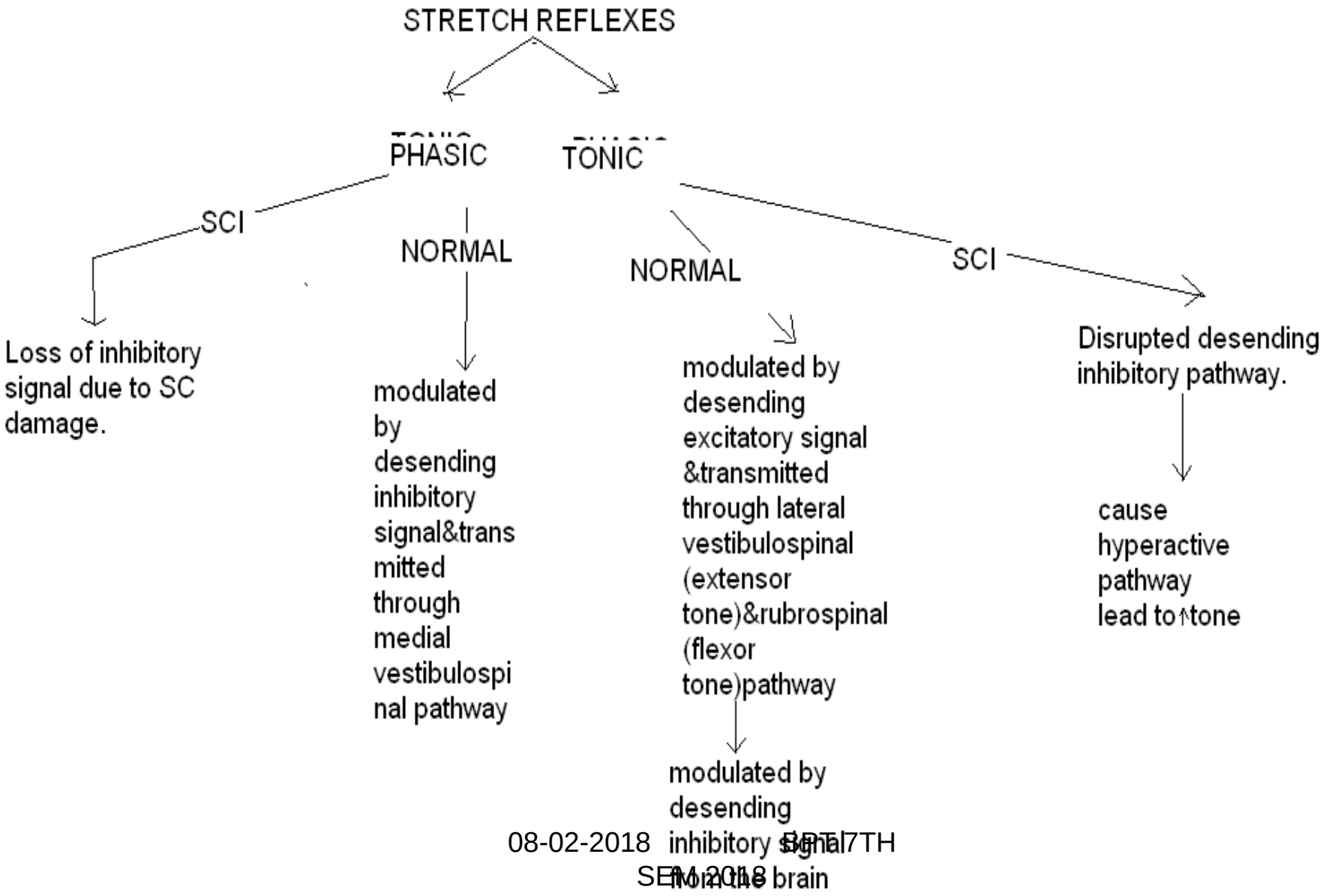
Damage to certain tracts results in loss of inhibition and disruption of the stretch reflex arc. Uninhibited muscle stretch produces exaggerated, uncontrolled muscle activity, accentuating the reflex arc and eventually resulting in spasticity.




Aα
γ fiber
Afferent
(both Ia and II)
Extrafusal
muscle fiber
INSET
Polar end
Intrafusal
fiber
Nuclear
bag region
Polar end
Intrafusal
fiber
Nuclear
bag region



STRETCH REFLEX



MANAGEMENT

- **POSTURE&POSITIONING:**

To maintain m/s length, tone reduction, prevention of contracture.

- **PROLONGED STRETCHING**:

To prevent contracture, cause temporary reduction in intensity of m/s contraction in reaction to m/s stretch, may cause plastic changes within CNS &mechanical changes at m/s, tendon ,soft-tissue level.

•WEIGHT BEARING

It modulate influence from cutaneous & joint receptors input to spinal motor neurons, resulting in decreased excit -ability.

•COLD & HEAT APPLICATION

COLD: •Slow NC
•Decrease in sensitivity of cutaneous receptors
•Alteration of CNS excitability
(study on rabbit shown that intramuscular temp. maintain -ed at 30 degrees cent. Effect upto 30-60mins.J SC 2002)

HEAT: •Facilitate uptake of released neurotransmitter.
•Return of calcium to sacroplasmic reticulum
(Lehmann&deLateur suggested that heating secondary afferent m/s spindle nerve endings &GTO could be a way in which inhibitory influence applied to motor neuron to diminish m/s excitation.

•**ELECTRICAL STIMULATION**:

Mild spasticity: may activate inhibitory network within SC.

Severe spasticity: stimulate dorsal root of upper lumbar cord segment.

- Stimulation of antagonist m/s augment reciprocal inhibition of spastic m/s.
- Repetitive tetanic stimulation of spastic m/s,fatigue of m/s due to repetitive tetanic stimulation

A condition usually occur due to inactivity or disuse of extremities of patients following SC lesion. There is **formation of bone meta plasia of soft tissue surrounding peripheral joints or ectopic** site below level of injury .Usually develop between **1 to 4** months after injury. On examination clinically loss of JROM, pain is affected region (sensation preserved), joint swelling, affect transfer, routine bowel bladder care &other ADL.X-ray&ultrasonography to rule out bone fracture, DVT. On blood test serum ALP(1week to 4months),ESR,C-reactive protein &CPK may be abnormal in acute case.

HETEROTOPIC OSSIFICATION

- Exact cause is not known.
- Extra articular & extra capsular.
- Develop in tendon ,CT between m/s, aponeurotic tissue peripheral aspect of m/s
- Several hypothesis like local trauma, spasticity ,tissue Hypoxia, completeness of SCI

MANAGEMENT

- **Medical**: NSAIDS, Disodium Etidronate
- **Radiation**: SAUTLER-BIHL et al found effectiveness of radiation therapy
- **Physiotherapy**: **1.**Inflammatory stage, no passive movement.
  2. After resolution a week), passive movt. to limb slowly & carefully 2-3 times only through full range.
  3. After 4-8 weeks, passive movt.& general activities are increased.
  4. Avoid vigorous stretching,ROM,forceful manipulation.
- **Surgery**: After 1 1/2 to 2 year for maximum maturation.
  Adv. Improve mobility &ADL.
  Prevent/ treat pressure ulcer.

Immobilization following SCI associated with decrease bone mass osteopenia, sometime leading to a condition with increased risk of #.Occur mostly during first 6months following SCI throughout entire skeleton except the skull. Bone loss initially around UE &trunk and between 4&16 months, bone loss continue in LE and maximum reached at 16 months. Immobility &lack of stress placed on skeletal system through dynamic weight bearing activities are major contributing factors. This condition is usually asymptomatic disorder unless pain due to # occur. On investigation total plasma protein &plasma albumin decreases, loss of vertical height of vertebrae.

OSTEOPORSIS

Normally there is a dynamic balance between the bone resorption activity of osteoclasts &role of osteoblasts in laying down new bone.Following SCI there is a net loss of bone mass coz' rate of resortion is greater than the rate of new bone formation.

MANAGEMENT:

Studies showed that early preventive pharmacologic intervention in SCI pt. should begin 1 year post injury.

•Dietary consideration:-----1.Calcium restricted food.
2.Vigorous hydration.
3.Milk,ice cream,diary product avoided.
4.High protein diet like egg, whole grain

.

•Early &continuing weight bearing.

Following SCI the most prominent adaptation in skeletal m/s below level of lesion which undergo marked changes in morphological, metabolic &contractile properties. FES is used to restore motor function in patient with UMNL following SCI.

MUSCLE ATROPHY

Denervation atrophy

- M/s innervated from zone of injury &LL m/s affected by cauda equina injury.
- Recovery through motor axon Sprouting.
- ES partially can reverse denervation atrophy.,but long duration,high intensity stimuli arenot tolerated.
- May interfere with reinnervation.

Disuse atrophy

- Affect all weakened m/s innervated below level SCI. develop weeks &affect type1 m/s fibre& conversion to type2 at 1-8mths.
- ES beneficial as short duration,medium intensity used to depolarize motor axons.

By 24 weeks of injury after traumatic complete SCI the average cross-sectional area of m/s of leg was 45% to 80% of matched able bodied controls. Study showed **the relative cross-sectional area of type1 fibers did not changes but type 2a decreases**. The exact cause is unknown. Animal studies showed that immobilization of LE for different period results is m/s atrophy ranging from 15% to70% of original m/s occur in first 7 days of immobilization.

MANAGEMENT

FES has been used to restore motor function in patient with UMNL causing SCI. This activate NM junction either directly or by going through PN &effect restoration of m/s strength.

Kagaya et al reported that cross-sectional area of m/s , m/s torque &force were increased at 25 weeks after TES in chronic paraplegia.

Kagaya et al reported that cross-sectional area of m/s , m/s torque &force were increased at 25 weeks after TES in chronic paraplegia.

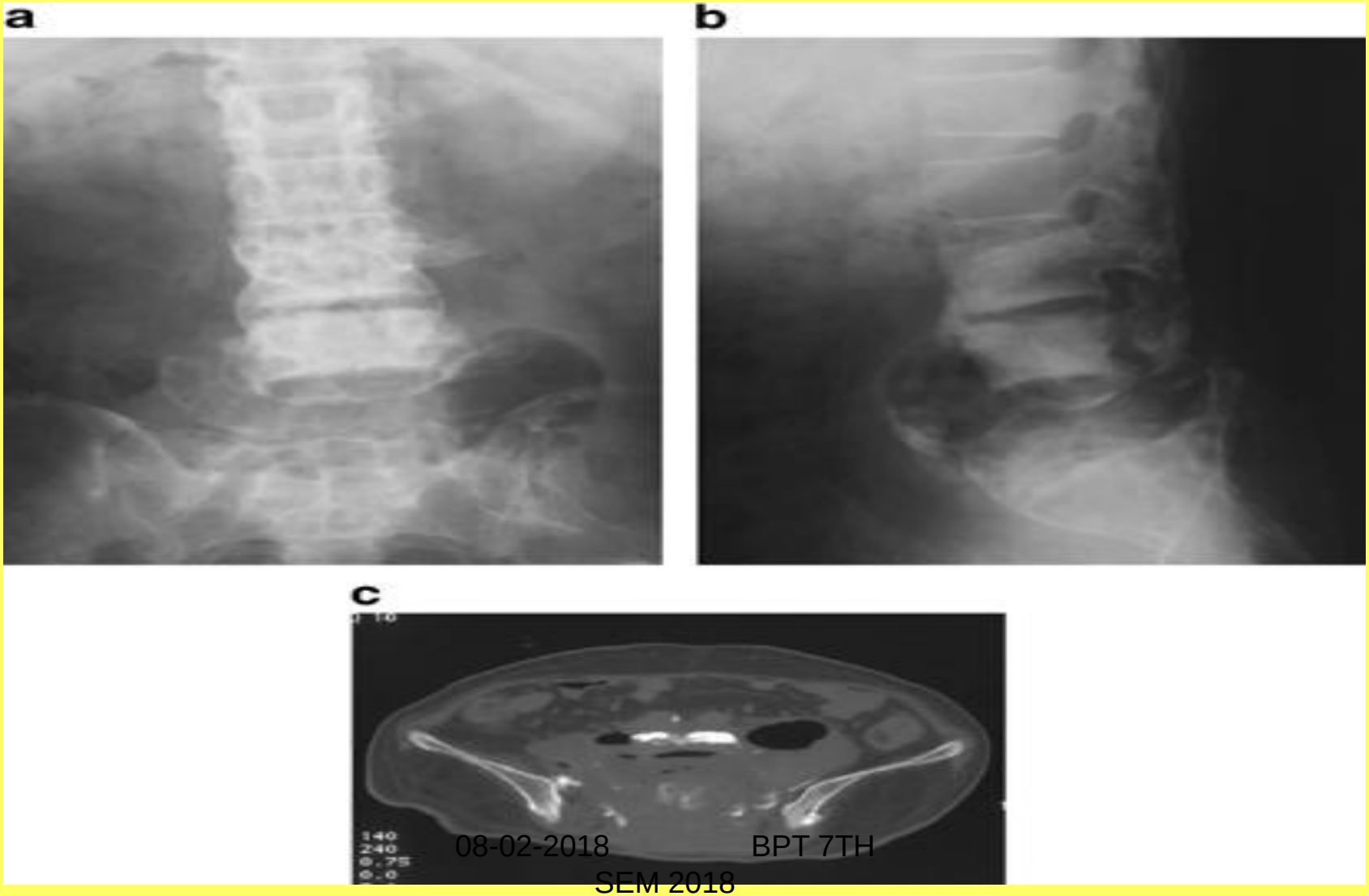
Study shows that there is progressive decrease in fiber diameter(type2 atrophy in early stage &type 1 atrophy in late stage). High frequency TES in acute phase SCI more effective reducing atrophy for type 2 fiber than in low frequency

Magunaga et al suggested that high frequency had advantage in closed loop control used for reducing m/s fatigue there is rapid response of m/s contraction there is strong contraction force.

So,type2(fast m/s) at high frequency (30-60Hz)& type1 can be stimulated at low frequency(10-20Hz)

A late complication of traumatic SCI, otherwise known as NEUROPATHIC SPINAL ARTHROPATHY. Patient c/o pain -less spinal deformity ,spinal instability, decreased sitting balance. On X-ray finding there is destruction of articular facets along with disc space narrowing, extensive osteo -phytes.

CHARCOT SPINE



Unknown trauma causes inflammation/direct bony injury

↓

With continued motion

↓

Progressive synovial inflammation

↓

Articular cartilage degeneration &bone exposure

↓

Erosion &fracture

↓

Loss of sensation lead to failure of protective m/s contraction associated With ligamentous &capsular stretch.

↓

Further repetitive trauma ,#,progressive instability

↓

Sclerosis of underlying bone occur,resulting in massive osteophytosis

MANAGEMENT

Non-surgical---------Body jacket immobilisation

Surgical---------Anterior & posterior fusion

A complication develop secondary to prolonged shortening of structure across & around a joint due to incorrect positioning, inadequate physiotherapy, spasticity resulting in limitation in motion. It is irreversible form of adaptative shortening.

CONTRACTURE

Lack of m/s function eliminate normal reciprocal stretching of m/s group &surrounding structures as the opposing m/s contracts.

Spasticity results in prolonged unopposed m/s shortening in static position.

Flaccidity results in gravitional forces maintaining a consistent joint position.

Faculty posture,heterotopic ossification, edema, imbalance in m/s pull contribute specific direction &location of contracture development.

MANAGEMENT

1. Passive movement
2. Prolonged passive stretching
3. Active exercises
4. Splinting
5. Positioning

LBP is a frequent compliant in SCI patient. Causes are not always known. Patient c/o Pain localized to one side. On prolonged sitting/standing/getting out of bed which may/may not be associated with trauma. On X-ray of LS spine, which is normal but show ossification of ilio-lumbar ligament.

ILIOLUMBAR SYNDROME

(CASE REPORT)

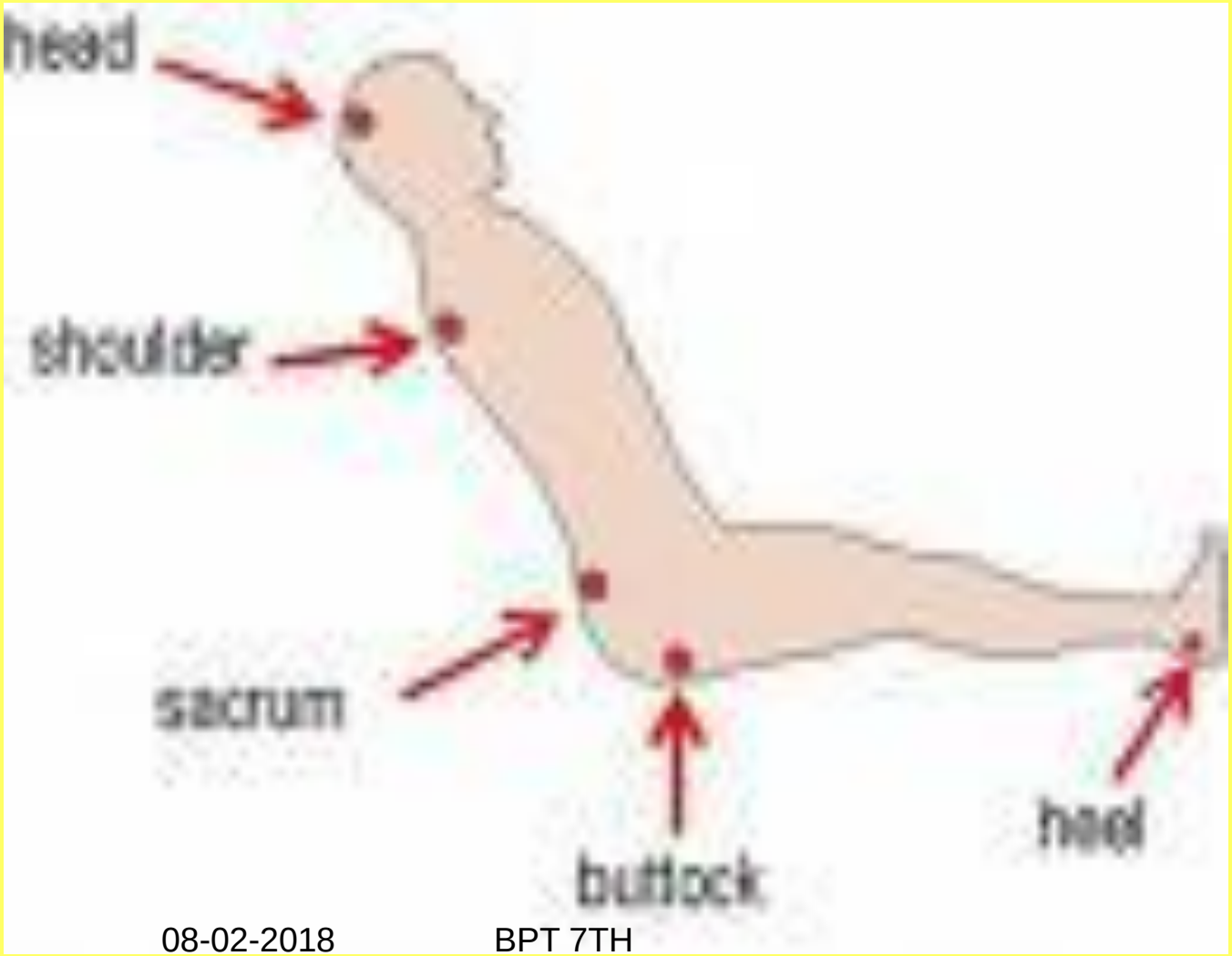
An infrequent complication in the treatment of SCI patient which is occuring in conjunction with deep extensive pressure ulcers involving hip joint. On physical examination confused with septic arthritis but psoas sign in diagnostic sign. Patient present with hip flexion deformity with external rotation & quadriceps wasting.

ILIOPSOAS ABSCESS

(CASE REPORT)

A complication prevalent & potientially a serious medical problem,particularly among a population of patients whose skin is more prone to breakdown. It is first & second most common problem following complete &incomplete SCI. **According to NPUAP**, it is defined as an area of unrelieved pressure over a defined area usually over a bony prominence resulting in ischemic, cell death,tissue necrosis. **On clinical examination there is breakdown of epidermis or dermis or subcutaneous fat or sometime bone.**This complication is due to immobilization & due to this patient is bedridden for prolonged period. This had negative consequences on patients **psychological physical rehabilitation** due to increases the cost of treatment & puts financial burden on family members of the patient.

PRESSURE SORE



PRIMARY FACTORS: 1.Pressure
2.Shear&Friction
3.Temperature
4.Age

(Study shown 100mm Hg P for 4 hours, there is ischemia &necrosis .P↑ ,less time require to get pressure sore.4mm of P for 1hour,there ↑chance of sore

CONTRIBUTING FACTORS: 1.Nutrition
2.Edema
3.Anemia
4.Endocrine

MANAGEMENT

A.**EDUCATION**:1.Etiology &Pathology

2.Risk factor

3.Proper positioning

4.Maintenance of optimum nutrition which body in +ve nitrogen balance,thus increase wound healing.

5.Daily inspection of part atleast twice daily particularly cutaneous site overlying osseous prominence.

6.Equipment like long handled flexible mirror to assist skin inspection.

7.Skin care.

8.Turning at regular interval of 2-3 hourly.

B. **Positioning and Equipment:-**

1.Pillows used to assist is maintaining desired position proximally and distally placed (bridging).

2.Low air loss and air fluidized beds should be used in case of multiple pressure ulcer with stage 3 or 4.

3.Weight shifting anterior, lateral every 15 to 30 mins for 30 secs when patient is sitting.

4.Forward leaning with chest towards thighs reduced P over IT to near or below 32mm Hg.

(Study shown this is better than 35 &65 degrees backward)

C. Nutrition:-

1.Total caloric need is 1.25-1.5 g/kg/day.

2.Forr wound healing Vit.C, E, Zn are required.

D. Medical complications contribute to poor wound healing:-

-Cardiac diseases

-DM

-Vascular diseases

-Malignancies
-Psychosis
-Pulmonary disease
-immune deficiency
-Collagan vascular disease

E. Medical management:-

-Cleaning

-Debridement chemical
Mechanical
Autolytic
Surgically

.

-

-Dressing

Wet dressing Dry Hydrocolloid occlusion

F. Surgical management:-

-Stage 3 and 4 need surgery

-Skin grafting

-Musculocutaneous flaps, skin flap

G. Physiotherapy:-

1.HVPGS

-Frequency:-100 Hz
-Polarity- -ve
-Intensity- below m/s contraction
-Duration- 30-40 min daily
-Effect-Induce fibroblast (DNA and protein synthesis)
-Proliferate and migrate epithelial cells and CT, so tissue contract easily and healing occur.

2.Zn iontophorosis:-

-Polarity=+ve electrode
-Intensity=0.1-0.2 mA/cm2
-Duration=15 mins

3.Ultrasound:-

-frequency=3 MHz/1 MHz
-Intensity= below 0.5 W/cm2 (non- thermal effect), 0.5-1 W/cm2 (thermal effect)
-Duration:-1minute to area of 0.75 cm2 at wound margin.

Effects:-

1.Thermal ⟶ ↑metabolic activities
↑blood flow
↑collagen extensibilities

2.Non thermal ⟶ ↑cell membrane permeability.
↑tensile strength elasticity of scar tissue
↑Ca 2+ uptake by fibroblast

Dyson and pond (1970) is a faline study applied US at a dose of frequency 3 MHz intensity 0.25 W/cm2, pulsed 1:4, 2 to 10 minutes application daily to the surrounding area of the sore applying liquid paraffin and coupling cream. After the growth was noted the application schedule changed to 3 times weekly. Results obtained showed US increases rate of growth of tissue at wound site. Paul et al (1960) in his clinical observation found US was markedly effective is relieving congestion, cleaning, necrotic area and promote healing.

.

4.LASER:-

-Frequency= 16 Hz
-Mode scanning at a distance= 30cm
-frequency =3 times/ week
-Effects – fibroblast causes stimulation of production of collages following irradiation causing wound healing.

Jan S Kana; MD (1981) studied effects of laser on wound healing is rats and found He-Ne laser irradiation had a statistically significant stimulating effect on collagen synthesis in the wounds.

5.IRR:-

-For stage 1 and 2.
-60-70 cm distance
-750-1000m lamp for 15 mins

Effects:-
- ↑metabolic rate
- collagen extensibility
thus accelerate healing and repair.

6.UVR:-

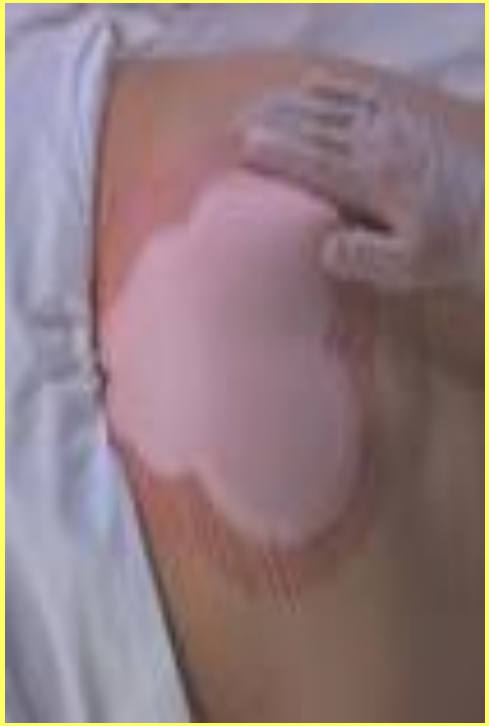
E4 dose

Effect:-Kill surface bacteria

-**High and High** (1983) found that radiation from kromayer was successful in killing bacteria and reduce colony numbers

-**Doddetal** (1989) have shown that skin oxygenation is increased for some 48 hrs after treatment.

7.Hydrotherapy- Assist debridement



8.Vaccum-assisted therapy / subatmospheric pressure therapy: placing open foam into wound sealing the site with adhesive drape &apply subatm.P 125mmHg)

9.Normothermia-(Use radiant heat dressing) (study shown to↓ mean wound SA by 61% over 4weeks period)

10.Constant tension approximation

Other skin complications following SCI:-

1.Paronychia-

-during 1st yr after injury
-Inflammation of toe nail involving folds of tissue surrounding the nail
-caused by Candida

2.Acne vulgaris-

-During 1st several mths after injury
-Seen is face and upper trunk
-Cause:- -Less vigorous bathing, skin hygiene
-Alteration is skin gland function

3.Seborrheic dermatitis-

-Due to inadequacy of facial cleaning and hair washing.

Wilson and Walshe studied the incidence is pt. during acute phase of SCI and found 65% of recently injured tetraplegia.

4.Chronic folliculitis

5.Cellulitis

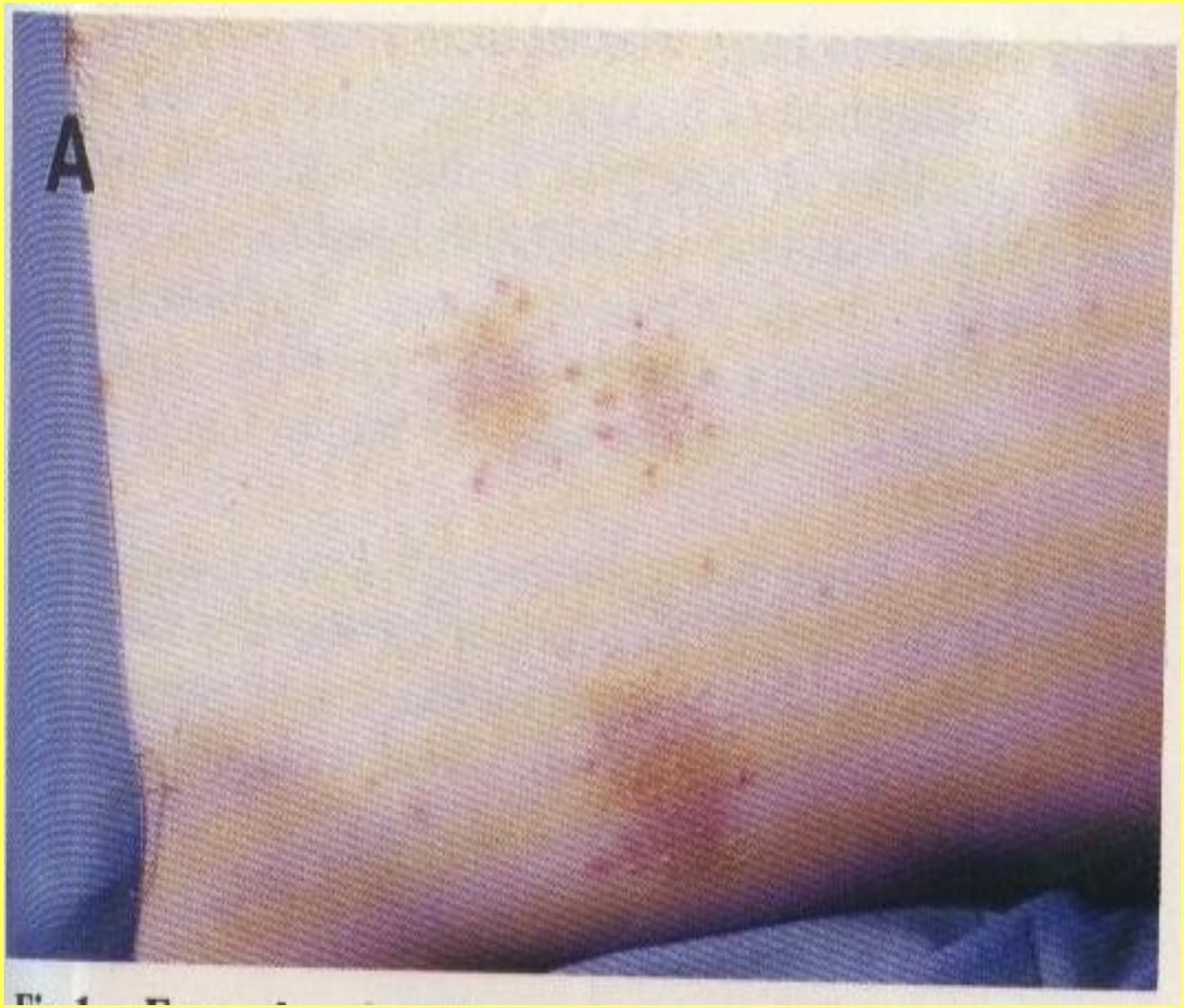
6.Osteomyelitis with deep draining ulcer.

7.Skin thickening below neurological level of injury.

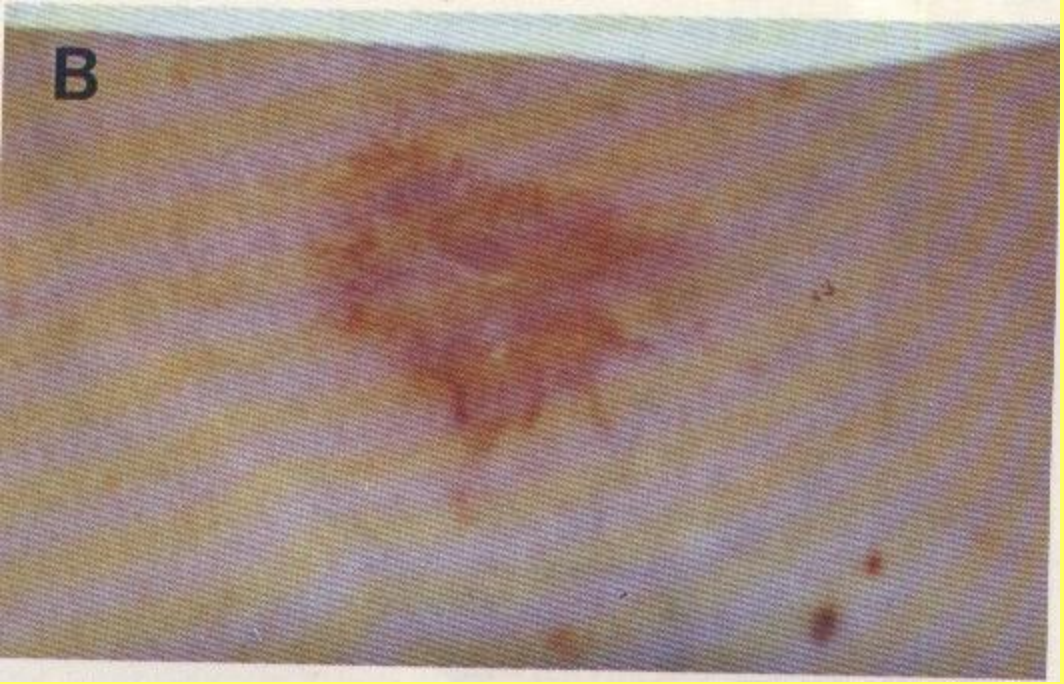
8.Nail hypertrophy due to repeated trauma to growing nail bed (para> tetra).

9.Onychogryphosis(hypertrophy)

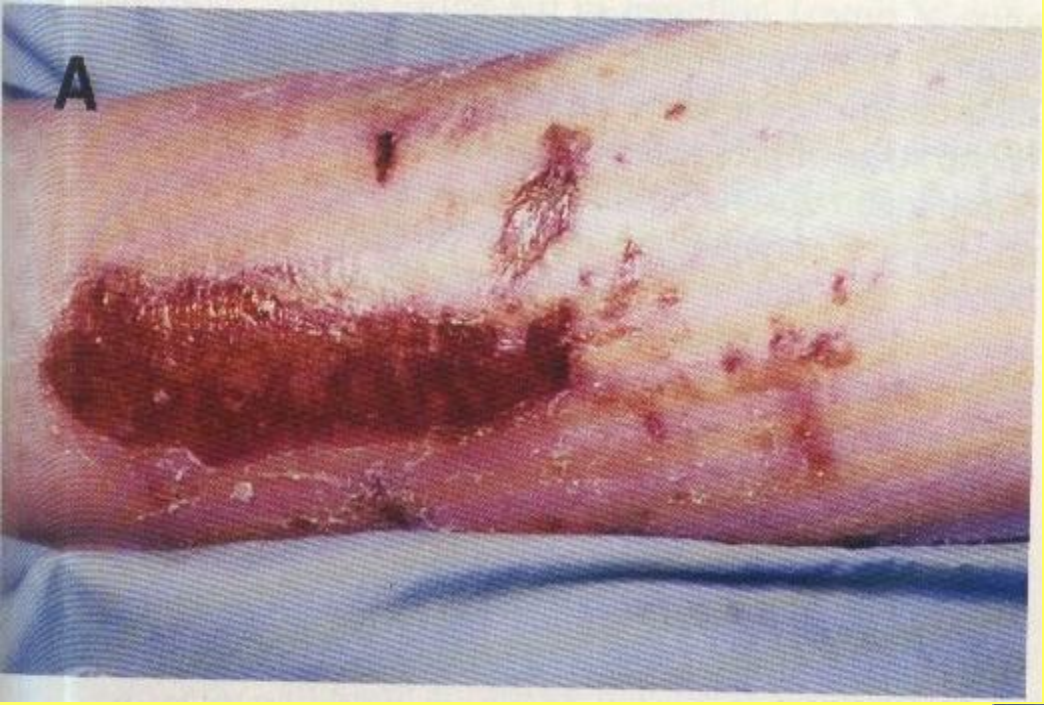
10.Onychomycosis(fungal infection of nail plate)



HYPERPIGMENTED SCALY LESIONS WITH SURROUND -ING PAPULES(Undiagnoed skin lesions.)

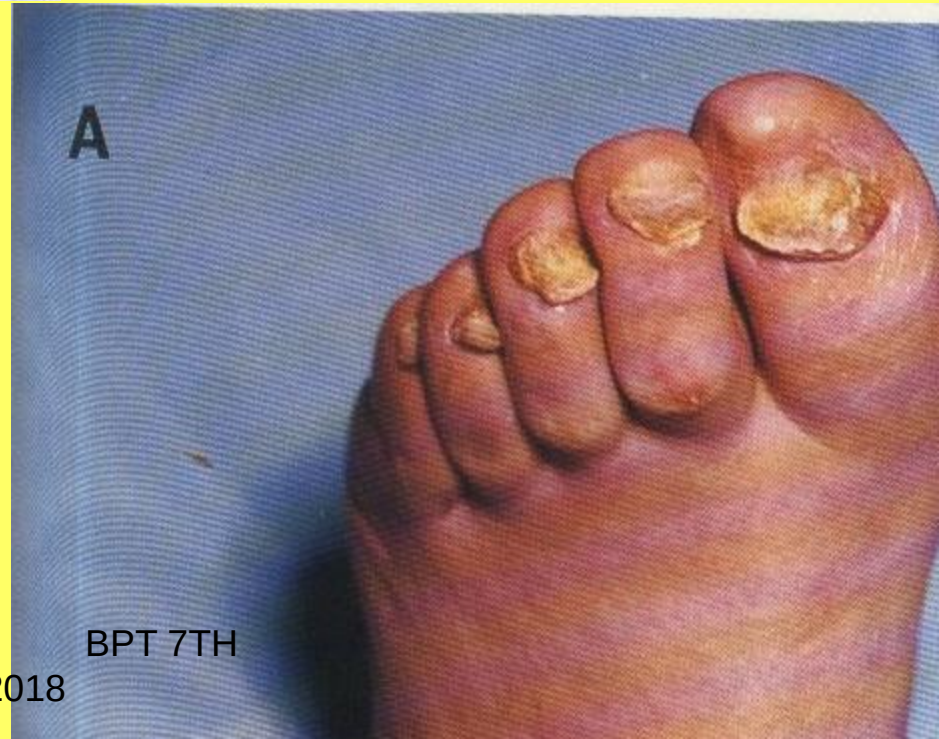


SUPERFICIAL AREA OF GRANULATION OVER STERNUM WITH LASTED ALMOST 1 YEAR & HEALED WITH SCARR -ING(Undiagnosed skin lesions)

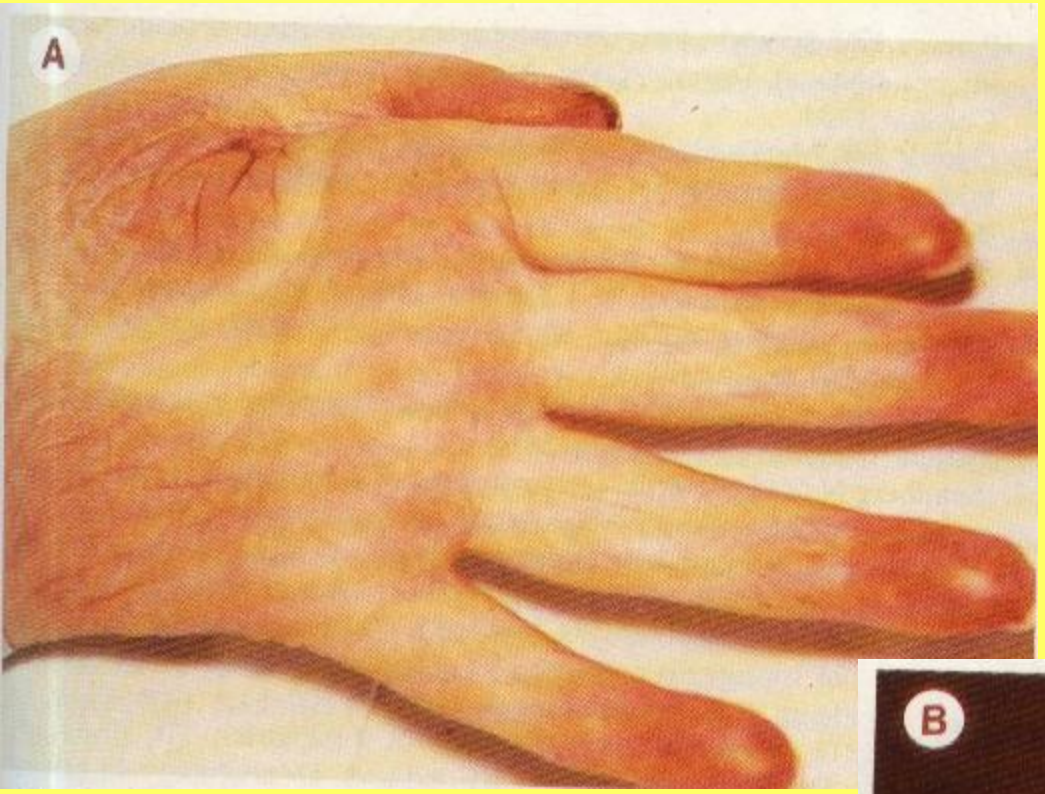


CHRONIC ULCERS OVER LATERAL LOWER LEG WITH EVENTUAL DIAGNOSI -S OF UNDERLYING OSTEO -MYELITIS OF FIBULA

NAIL HYERTROPHY (ONCHOGRYPHOSIS)
All nails on foot involved

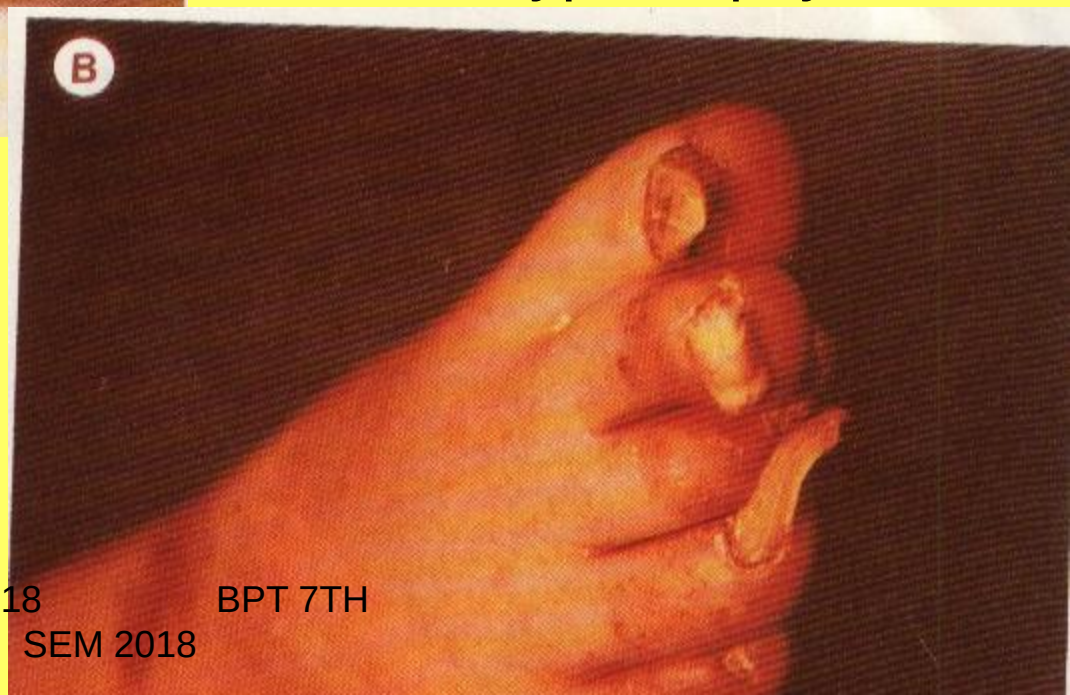


PERIUNGUAL & PALMAR ERYTHEMA



In hand without fingernail hypertrophy.

In feet with toe nail hypertrophy.



Management:-

-cleaning of face, interdigital spaces and groin thoroughly with soap and water.
-Drying with disposable paper towel/ different towel for each foot and each side of groin.
-Antifungal agents.

A common dysfunction which lead number of complications in persons with SCI. Bacteriuria is a common problem in patient with their dysfunction where patient c/o Fever with chill, dysuria, frequency, elevated serum WBC. The spinal integration centre for this is the conus medullaris. The lesion above and below both causes abnormal response.

VOIDING DYSFUNCTION

Normally when bladder is sufficiently full the stretch receptors within the wall of the bladder are stretched→afferent impulses set up→the impulses travel via parasympathetic fiber of pelvic splanchnic nerves→impulses terminate in spinal centers(**S2-S3**) →efferent impulses arise from this spinal centers→travels via parasympathetic fibers of same pelvic splanchnic nerves →impulses terminate on the detrusors m/s→contraction of the destrusor m/s →micturition

From brainstem both inhibitory (from medulla)& facilitatory (pons) fibers descends to terminate on spinal Centers.

Complete lesions above the sacral cord interrupt descending axons that normally control bladder function but don't interrupt sacral level reflexive control of the bladder. This result in hyper -tonic, hyper reflexive bladder with reduced bladder capacity. As reflex circuit for bladder emptying is intact, reflexive emptying may occur automatically whenever bladder is stretched. if sphin -cter is hypertonic flow of urine is functionally obstructed & kidney can be damaged.

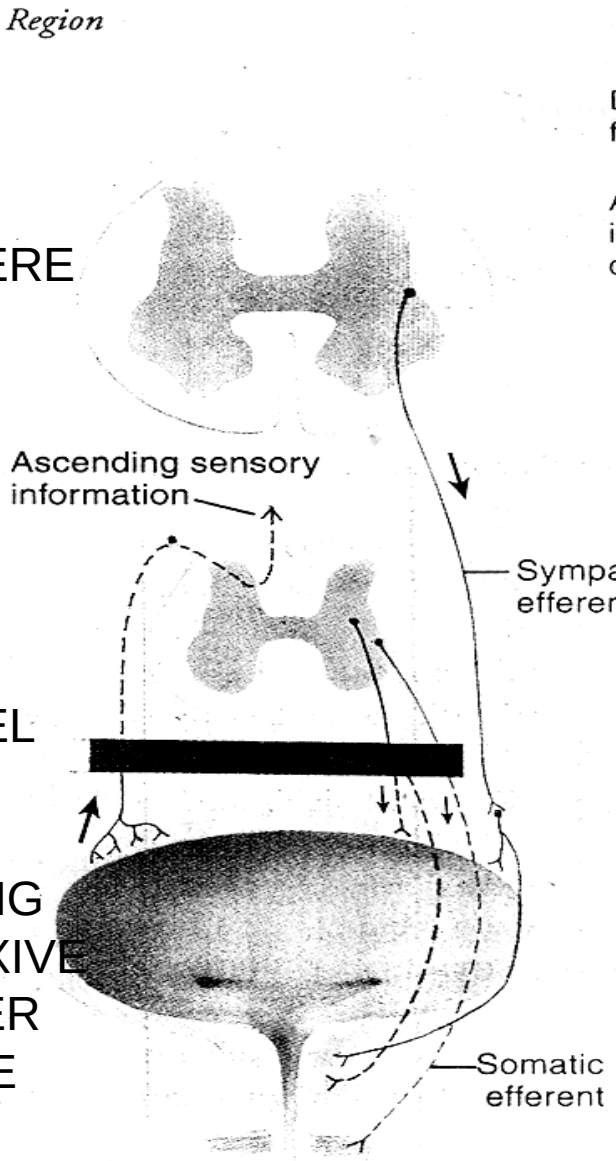
Complete lesions at S2-S4 SC or afferent or parasympathetic efferent produce flaccid ,paralyzed bladder This lead to bladder overfills with urine &bladder cannot stretch any further, urine dribbles out.

BLADDER DYSFUNCTION

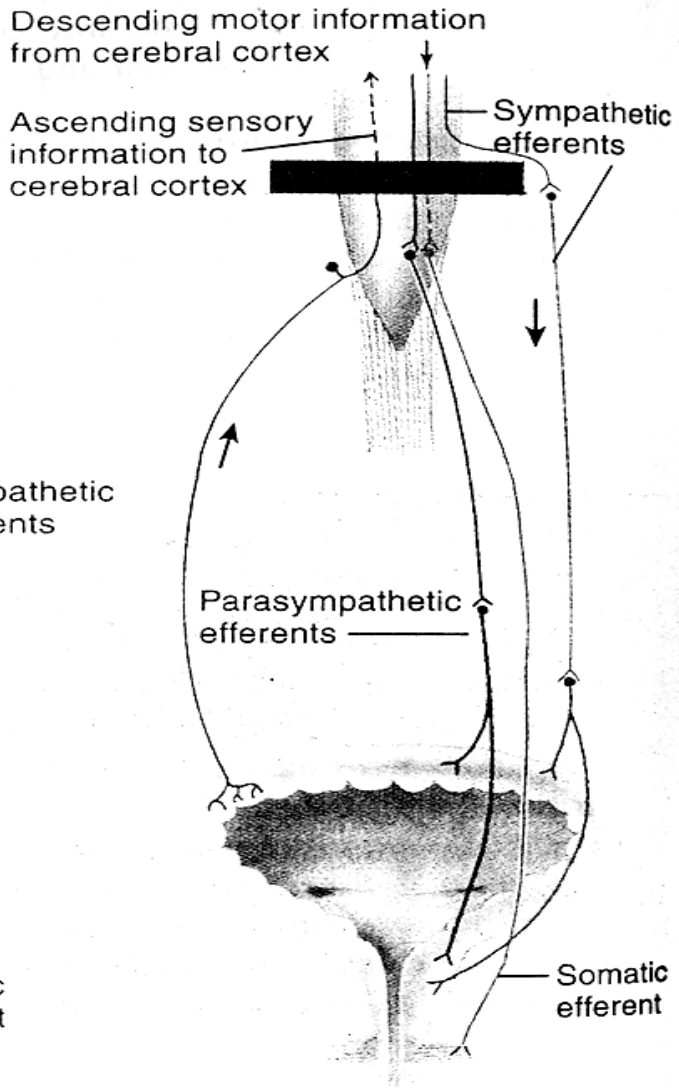
DOTTED LINE INDICATE NEURAL PATHWAYS IN -TERRUPTED&CONVEY INFORMATION.

A.FLACCID BLADDER WHERE ALL PATHWAYS INTERRT. EXCEPT SYMAP.&REFLE -XIC BLADDER EMPTYING INTERUPTED.

B.HYPERTONIC BLADDER WHERE COMMUNICATION BET. BRAIN &SACRAL LEVEL PARASYM. NEURONS CONTROLLING BLADDER INTERRUPTED,PREVENTING VOL. CONTROL.AS REFLEXIVE CONNECTION BET.BLADDER &SC INTACT SO REFLEXIVE EMPTYING OCCUR.



A



B

Normally when the urine comes to bladder, detrusor contract and IVP>uretheral R, their voiding occur and sphincter contract.

But following SCI, following bladder types are there:-

- SD+SS
- SD+FS
- FD+SS
- FD+FS

Above causes following complications like:-

-UTI
-DSD
-Renal calculi
-Bladder cancer

MANAGEMENT:-

1.Assymtomatic UTI:

Bladder program

REFLEX (Intermittent catheterization)	NON REFLEX (timed voiding program)
• fluid intake is monitored at 150-180ml/d from morning until evening. intake is stopped late in the day to reduce the need for catheter -isation during night.intially pt. is catherised every 4hrs.pt. void prior to catherisation with manual stimulation.catheter inserted and RV drained. As bladder emptying effective,RV decreases.	•First pt pattern of incontinence established.RV ic checked.Once Pattern established compare with Intake pattern. bladder gradually Trained to empty at regular, predicted interval. as incontinence decreases schedule readjusted to expand interval between voiding.at night intake avoided. Stimulation tech. can be given

2.Symtomatic UTI

- 7days course of antibiotics.
- Indwelling Foley's catheter in place during IV or oral fluid hydration to decompress bladder.

Following SCI, consequences like loss of bowel sensation, voluntary control of defecation, alteration of large bowel motor activity this results in abnormal transit time. Patient with Paraplegia below T10 and above L2 excluding conus and cauda lesions less prone to this complication than tetraplegic patient.

CONSTIPATION

Bowel control is similar to bladder control. The signal to empty The bowels is stimulation of stretch receptors in wall of rectum. Afferent fibers transmit the information to lumbar &sacral cord. The information is conveyed to the brain &if if appropriate ,the efferent signal is sent to relax sphincter.

Person with SC lesion above sacral cord is unaware of rectal stretch &no voluntary control of sphincter Yet rectal stretch can elicit reflexeic emptying of lower bowel coz' reflexic lower bowel emptying circuit is intact. If bowel emptying reflex Circuit is interrupted by lesion of S2-S4 or parasympathetic connections to S2-S4 the parasympathetic influence on peristalsis &reflex emptying of the bowel is lost.

MANAGEMENT:-

1. Stool softeners.
2. Bulk formers (psyllium)
3. Laxatives.
4. Diets and nutrition
    - Fibre rich food
    - Fluid intake

Reflexic complete SCI

-One bisacodyl supposition every other day.

-Two seena concentrate tablets every other day.

-100mg docusate Na twice a day.

-Prune juice,40z everyday.

Areflexic complete SCI

-Mg(OH)2 suspension 10z twice a day and repeat flat plate Xray is 3doup.

-100mg docusate Na twice a day.

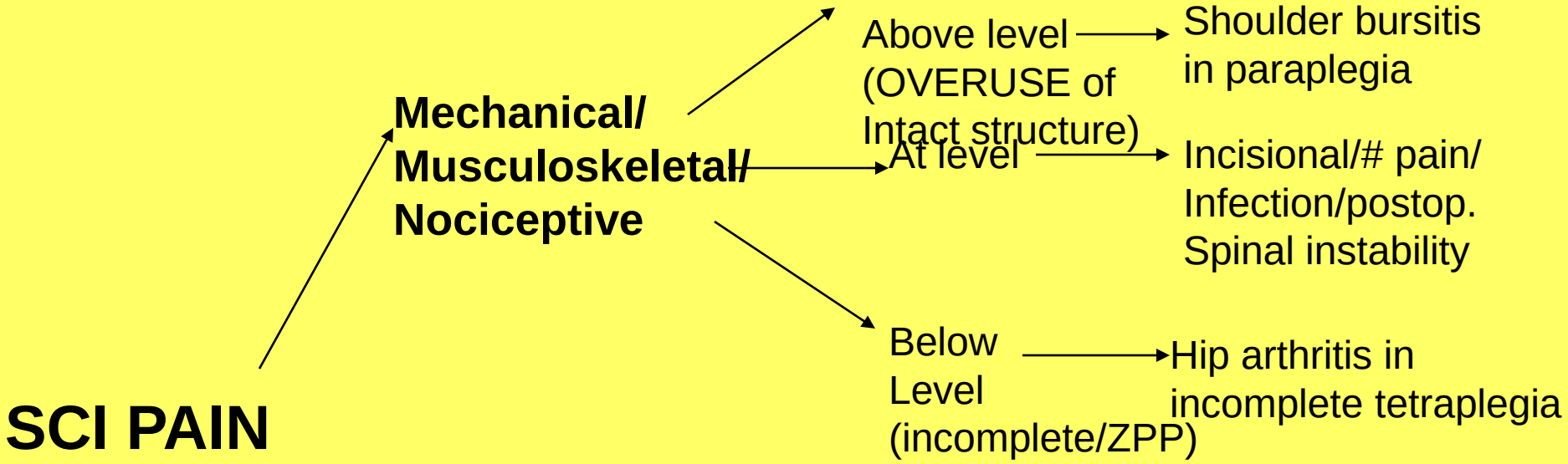
-Prune juice,40z everyday.

-1 tablet senna concentrate every other day.

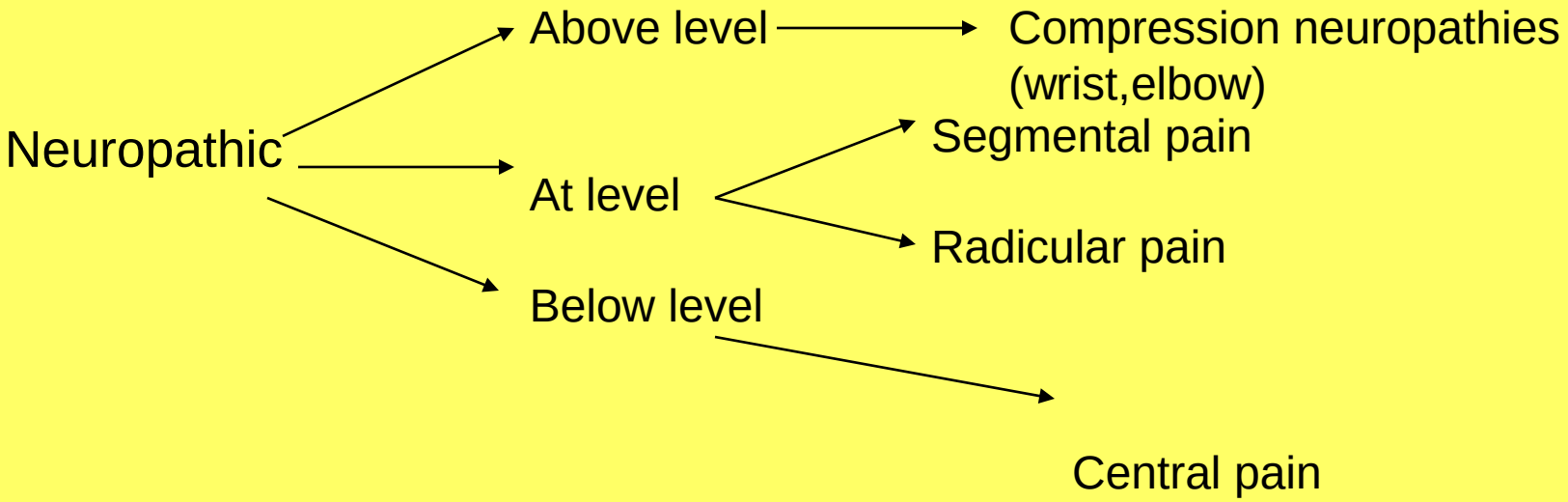
-Manual evacuation and strain everyother day.

The complication of the SCI patient which is common occurrence but the exact etiology of this is often poorly understood.
This occur due to direct trauma, damage to nerve roots, overuse of structures like bones, ligaments, m/s, IVD, facet joints. Higher in patient with thoracolumbar and incomplete SC Lesions.
Inactivity, stress, weather change, over activity are aggravating factors. This itself often has a great effect on the ability of the person with SCI to regain ambulatory function, return to work.

PAIN



- Pain normally or partially from innervated tissues.
- Only pain etiology easily identifiable.
- Sharp in nature (radiating/non radiating) or dull arching pain (non radiating)
- Relief with rest, bracing (TLSO), treating underlying cause with antibiotic, drainage of infection or surgery in vertebral instability.

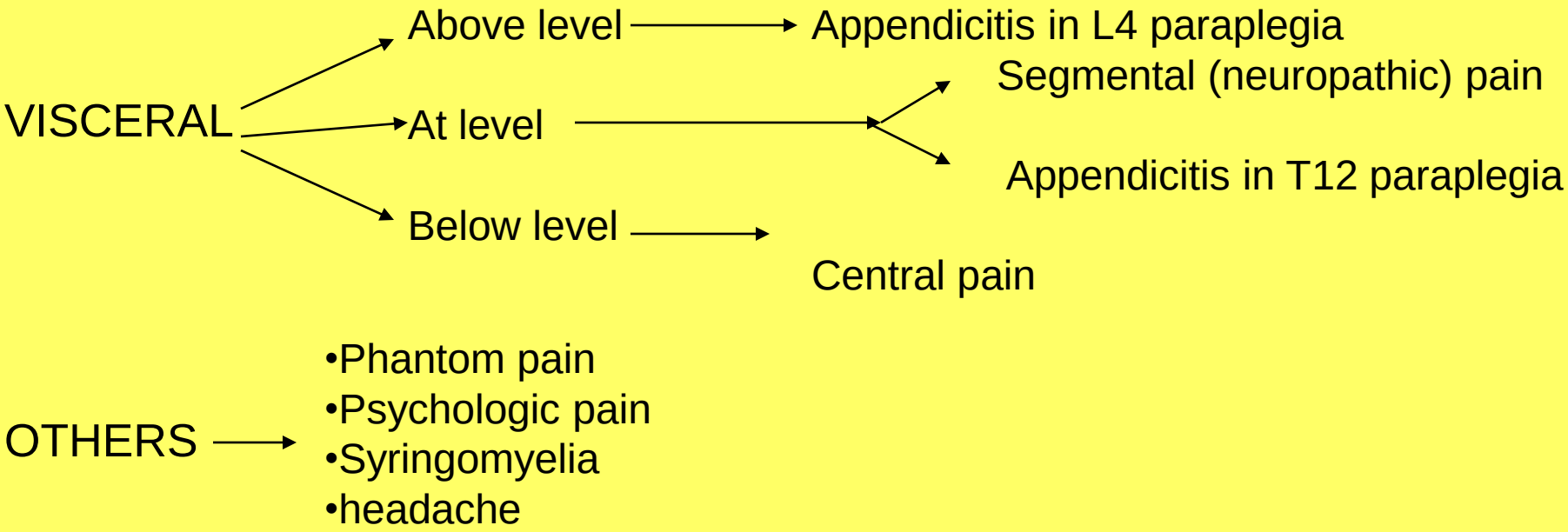


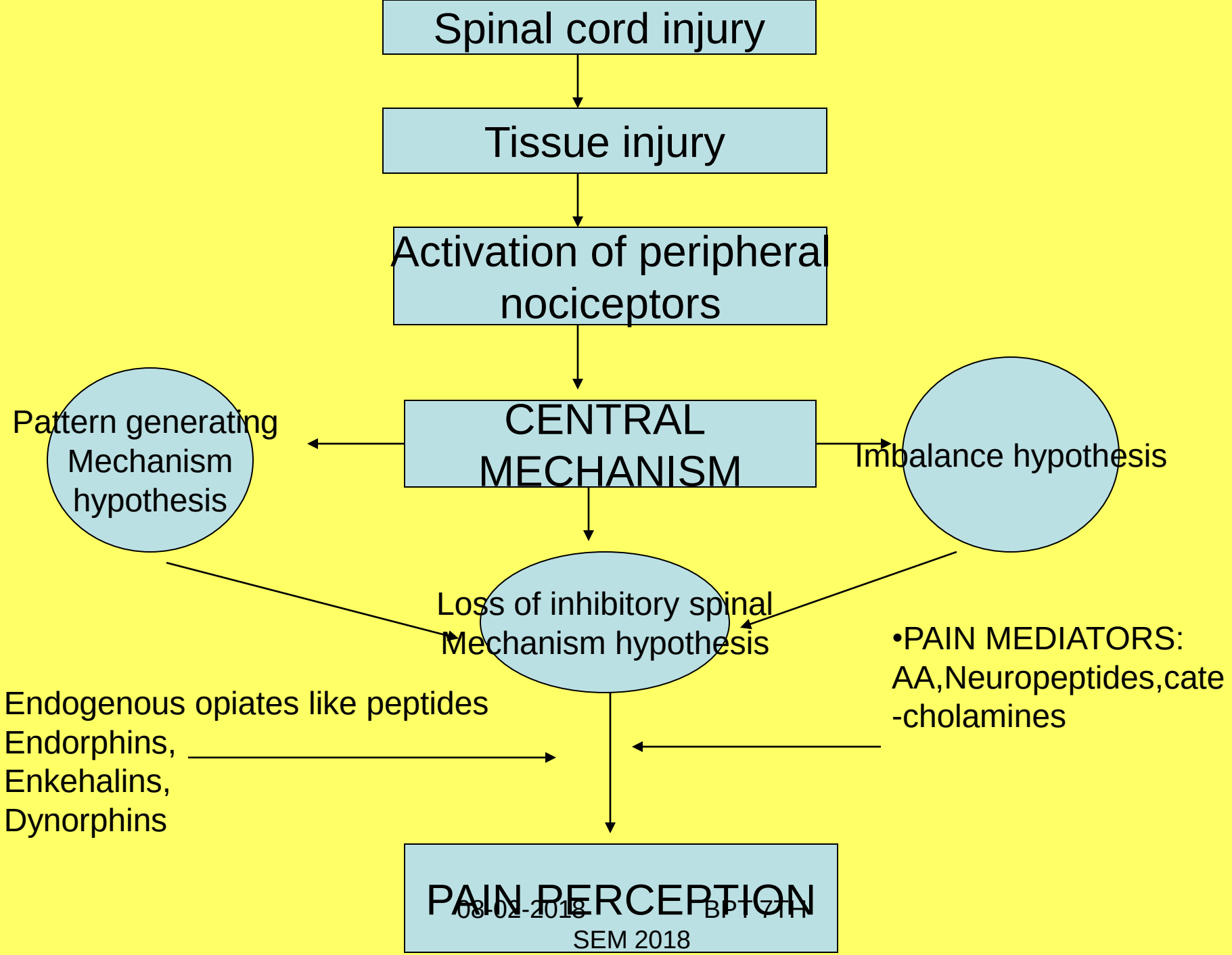
•Compression over route of nerve at the site i.e. ,median ,ulnar nerve due use of Cane, crutches ,wt.shift in UE,WC transfer &mobility.

•Segmental pain due to injury to gray matter of dorsal horn &hyperactivity of no -ciceptor cells located in grey matter. distinguished from radicular as typically b/l distribution through 1 or more adjacent dermatomes, vary with level of injury.

•Radicular pain due to root involvement arising from direct root damage,spinal column instability after spinal # or ligamentous injury, impingement of disk material, inflammation. usually burning, stabbing, shooting or electric shock like associated with allodynia or hyperesthesia.

•Central pain is burning ,aching, tingling,shooting.stabbing,pressure,cold,numb-ness, pin&needle, electric sensation,m/s cramp.It is diffuse,assymetrical,patchy perceived coming from specific body part or region below level of injury.May develop after injury,gradually over course of weeks to mths or years after injury.




Spinal cord injury
Tissue injury
Activation of peripheral nociceptors
CENTRAL MECHANISM
Pattern generating Mechanism hypothesis
Imbalance hypothesis
Loss of inhibitory spinal Mechanism hypothesis
•PAIN MEDIATORS: AA,Neuropeptides,cate -cholamines
Endogenous opiates like peptides Endorphins, Enkehalins, Dynorphins
PAIN PERCEPTION

Type of pain	Feature of pain	Causes of pain/Pathophysiology	Treatment
1. Musculo-skeletal pain	-Dull, aching, Worse with Activities, eased by rest.	Pain arises from damage or overuse in structure such as bones, ligaments, m/s, IVD, facet joints. This pain identified by location i.e. at or above lesion level in complete SCI. Occur due to faculty positioning inadequate ROM ex tightening of the capsule and surrounding soft tissue structures.	Regular ROM Ex
2. Visceral Pain	**Dull, poorly localised, Cramping related to Visceral function Or pathology**	**Identified by location (abdomen)**	Symptomatic treatment
3.Neurapathic pain (At level) pain **Central Radicular** Below level pain	**Burning, Stabbing, Shooting, Presence of Allodynia. Follow dermatomal pattern** Burning,tingling, Aching,shooting, stabbing	Damage to nerve roots at site of cord damage **Acute compression or tearing of nerve roots, Secondary to spnral instability, periradicular scar Tissue and adhesion formation.**	**Surgical Intervention i.e. Neurectomy, Posterior rhizotomies**

4.Spinal cord Dysesthesias (phantom pain)	Burning, Numbness, Pins and Needles, Tingling feeling	**Below level of lesion where sensation tend To be diffuse and not follow dermatomal Distribution. Abnormal propioceptive Sensation, causing individual to perceive Limb is other than actual position. It is due To perceive limb in other than actual Position. It is due to scarring at distal end of severe disc.**	**Pharmacotherapy Using carbamozepine. Gentle handling of pt's Limb and careful Positioning frequently Make pain more Tolerable.**

A common of worsening myelopathy after SCI and devastating because it may cause new disability after a person with SCI has successfully completed rehabilitation from their injury. Patient c/o burning or aching at site of original injury or radiate to neck or UE and worsen with coughing, sneezing, sitting. O/E there is dissociated sensory loss with impaired pain and temperature sensation. Usually occur two month to decades after injury. On investigation showed intramedullary cyst with well defined margin.

POST TRAUMATIC SYRINGOMYELIA

After SC trauma, there is accumulation of blood inside SC, gradually blood resolves and leaves some cavity and lead to tube formation within spinal cord.

Arachnoid scarring or adhesion of pia and arachnoid causes obstruction to absorption of CSF, leakage to near by ventral canal and form tube.

Due to kyphotic deformity, cord tethering occur with ↑ed cord tension on spine flexion and impaired CSF flow in subarachnoid space. (**Study** show pt. with SCI with less than 15degrees of residual by kyhosis and less than 25% canal are half as likely to develop post traumatic syrinx.)

MANAGEMENT:-

Surgical

Conservative

1.Activity restriction that might transmit pressure to subarachnoid space by avoiding high force exercise avoiding valsalva and crede and coughing.

1.Syringo peritoneal shunting (frequently used reported by Williams& Phili -ps)

2.Syringotomy

2.Percutaneous drainage by tapping syrinx with a needle under CT guidance.

3.Alter fluid dynamic in SC by keeping head of bed elevated to 20 degrees at night to lessen rostral slosh of fluid in syrinx.

This complication is defined as a deficiency of red blood cells secondary to multiple trauma. immobilisation, malnutrition even in the absence of detectable blood loss. This is associated with ↑ed fatigability and thus can affect ability to participate in rehabilitation program and functional capability. **Claus walker and Dunn** found significantly lower mean Hb level and a lower erythropoietin level in people with complete quadriplegia than in people with complete paraplegia is late phase (8 to 28 weeks after injury). By **1 year of injury** this complication improves in most patient with SCI.

In **chronic stage** this may be associated with chronic inflammat-ory complications such as pressure ulcers and UTI. On investigation found serum Fe, TIBC, transferin low. This complication is associated with UTI, decubitus ulcer, respiratory infection.

ANEMIA

Anemia in SCI

ACUTE->Blood loss due to bony, soft tissue/ visceral injury, GI hemorrhage, surgery 25% of persons had abnormally low serum erythro -poietin levels.

CHRONIC->Anemia of chronic disease involves characteristic disturbance of Fe metabolism ed production of RBC , RBC survival associated with chronic inflammatory , infectious or neoplastic illness.

Immobilization for at least 5 weeks lead to decline slowly of RBC mass in association with ↓ Fe turnover is plasma and in RBC, a ↓ed reticulocyte count, ↓urinary excretion of erythropoietin suggestive of ed production of this hormone.

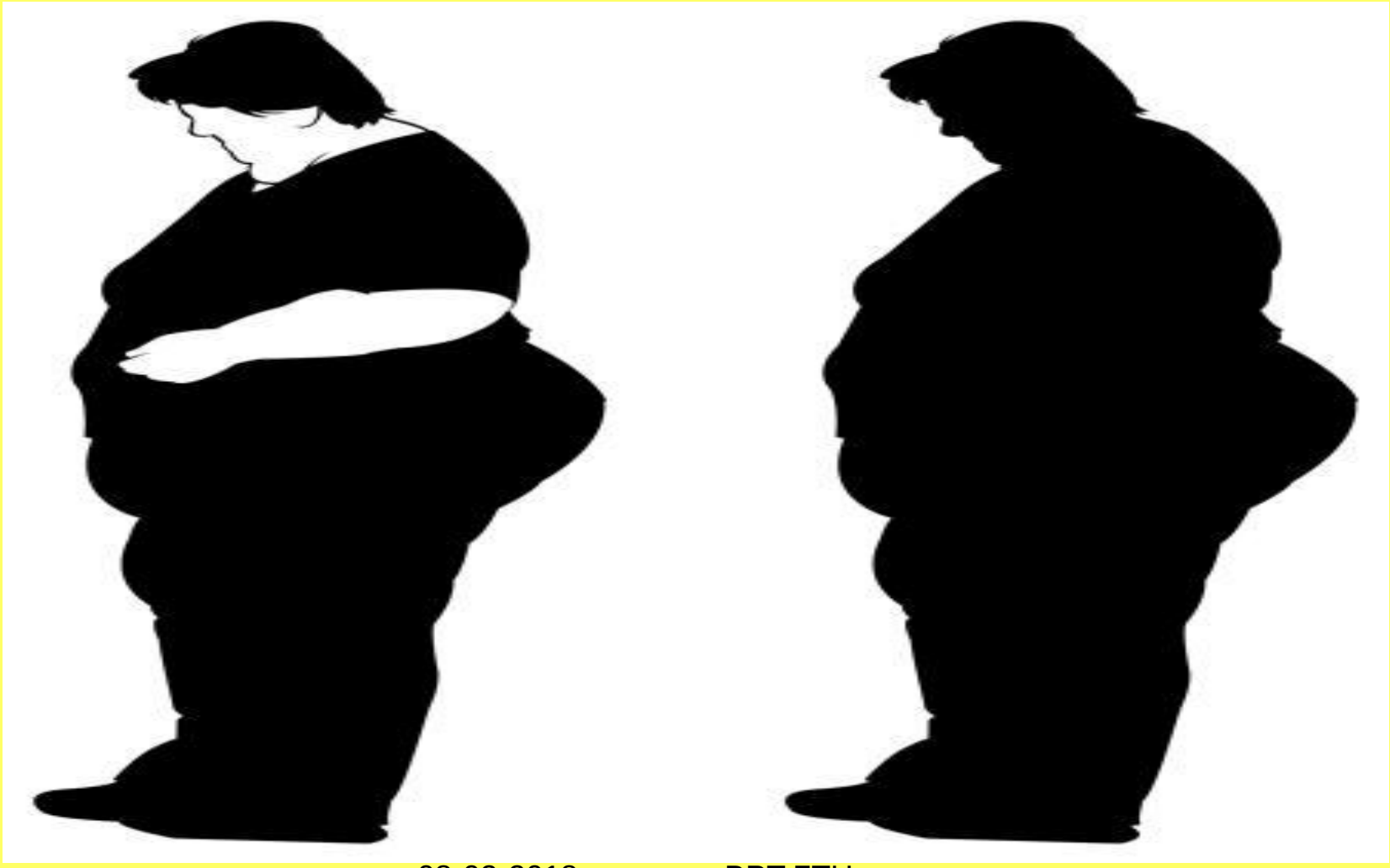
.

Malnutrition, i.e. protein deficiency causes depression of erythropoiesis, ↓ed erythropoietin production

.
Normochronic, normocytic anemia was the most frequent type of anemia reported by
Perkash and Brown, 11% patients suffered from hypochromic, microcytic anemia, 14%
patients suffered from normochromic, microcytic anemia.
Fe supplementation is given is case of chronic case.

A complication which itself is not dangerous to the patient but lead to other impairment like premature death, diabetes, hypertension, atherosclerosis and itself might interfere with their eventual functional outcome. This complication can be defined as a body mass index (kg/m_2) or BMI is 85^{th} percentile or higher. The 85^{th} percentile is 27.8 for males and 27.3 for females. This may occur due to sedentary lifestyle/bedridden due to prolonged immobilization . Sedentary lifestyle is a risk factor for CHD & it may contribute to lipid & carbohydrate abnormalities. Changes in body composition & inactivity predispose to lipid disorders& impair -ed glucose tolerance or diabetes mellitus.

OBESITY



Disorders of oral carbohydrates tolerance are more prevalent in persons with SCI than in the able bodied population. In persons with SCI who have a disorder in glucose uptake may be demonstrated. **Yalow and calleagues** reported **higher plasma insulin concentration is obese individuals.**

Obese individuals

↓

Adipose tissue

↓

Release cytokines,TNF- into circulation.

↓

Depress insulin receptor's Tyrosine kinase activity

↓

Reduced insulin action at peripherial tissue.

Brenes & associates suggested that eventual changes in the lipid profile predict 60% **increase in risk of a heart attack**.

Complications due to obesity:-

1.Respiratory compromise
2.Compressive mononeuropathies
3.Back injuries
4.Depression
5.CV disorders (CO, SV,bv ↑,hypertension, IHD)
6.CHD

Management:-

-Dietary control
-Exercises

SCI by no means contraindicates this issue, but it can present a challenge to the pts.and physicians. This itself under close medical supervision is possible for many SCI women but many complications may arise from this in women with SCI. Immediately after SCI, amenorrhea occurs in 85% of women with cervical and high thoracic injuries and 50% to 60% of woman have return of menstruation. The average deviation is 4.3 months (1week-24 months). After that it is possible and fulfill the loss of control over one's life and surrounding.

PREGNANCY RELATED COMPLICATIONS

Female sexual responses follow a pattern related to location of lesion. In **UMNL**, reflex arc will remain intact but psychogenic response will be lost and vice-versa in **LMNL**. Women with **complete, UMNL** have reflexogenic response whereas those with **incomplete SCI**, may have both reflexogenic and psychogenic response.Women with **complete LMNL** have psychogenic respon -se but not reflexogenic response whereas those with **incomplete LMNL**, may have both responses.

(As psychogenic responses are medicated through sympathetic system via hypogastric plexus(T11-L2) whereas reflexogenic responses by para sympathetic system (S2-S4).
Fertility is not considered a problem after SCI, a number of potential prenatal,peronatal, postnatal issues exit.

PRENATAL

- Constipation
- UTI
- ↓VC
- DVT
- Pressure sore
- ↑spasticity
- Autonomic dysreflexia

PERINATAL

- Premature delivery
- Autonomic dysreflexia
- DVT
- Pressure sore

POSTNATAL

- Episiotomy skin break down
- DVT

1.URINARY TRACT INFECTION: (Bacteriuria)

Cause:- -Incomplete bladder emptying
-Presence of foreign bodies such as catheters or calculi.

Management:- Eradicate asymptomatic bacteria by
-Minimizing residual volume.
-Avoiding use of indwelling catheter.
-Use of antibiotics when needed.
-Good fluid intake
-Extra care to prevent kinking of the catheter tubing

2.Anemia:-

By itself, anemia doesn't appear to have major impact on pregnancy. But persons with SCI considered to be at ↑ed risk of development of decubitus ulcer when anemia. Parturition causes significant blood loss and risk of requiring a transfusion is increased by anemia preceding delivery.

MANAGEMENT:
Enteral Fe supplementation is required but gives judiciously to a woman with SCI in whom it may cause difficulty in bowel evacuation.

3.SPASTICITY:

Increase is spasticity during pregnancy is tetraplegic pt. have been reported. Increased spasticity has been seen as a symptom of labor and autonomic hyperreflexia.

Management:-Medication

4.DECIBUTUS ULCER:-

Causes-During pregnancy, increased burden of weight decreases independence in ambulance and transfer and may contribute to this .

Management:

-Skin examination periodically.

-Use of larger WC to prevent abrasions and pressure

-Use of pressure relief technique.

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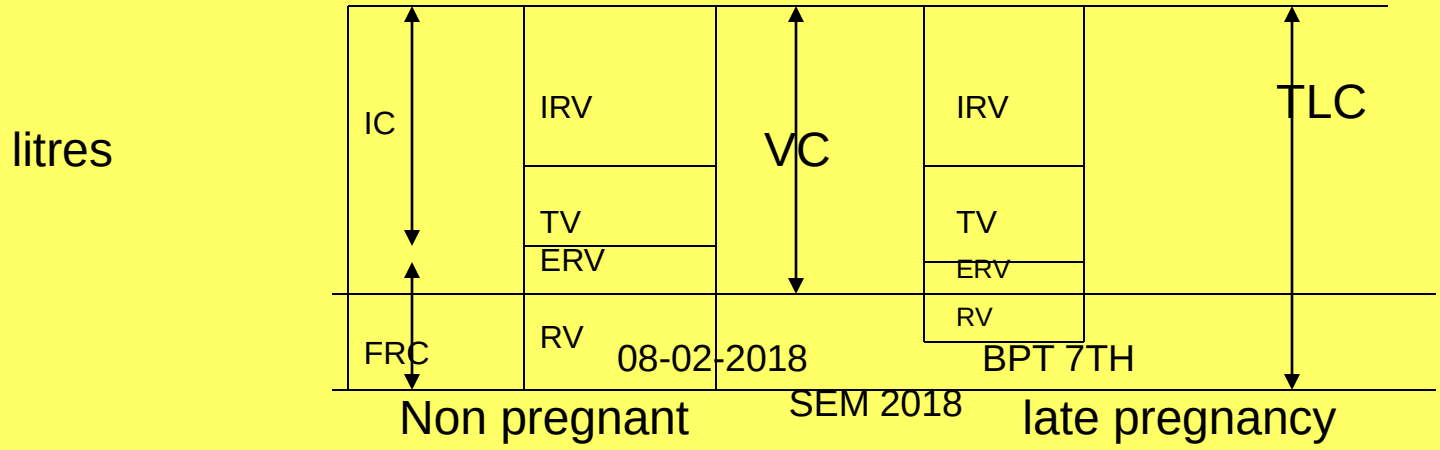
5.Pulmonary function:



Normally in pregnancy:

-↓FRC

-↑ed O_2 Consumption

-↑ed A-a O2 gradient which maternal O2 reserve

Management: Mechanical ventilation is needed if VC falls below 15 ml/kg.

6.DVT&Pulmonary embolism

Causes:-Restricted venous return from the growing uterus in combination with the persons restricted mobility.

Management:

1.Evaluation of edema in lower extremity.

2.Heparin(it doesn't cross the placenta)

7.AUTONOMIC HYPERREFLEXIA:

Occur patients with lesions above T5-T6 labor may be accompained this.
Management:-Removal of noxious stimuli, Pharmacological therapy, regional and general anesthesia.
-Avoid hypotension.
-Avoid prolonged labor.

8.NEUROGENIC BOWEL AND BLADDER:

Increased incontinence and worse constipation during pregnancy
Management:
Same strategy before pregnancy

Last but not the least complication which changes life of the patient physically, emotionally and vocational identity. It is a common form of psychosocial distress is SCI. This causes irritability, sadness, emptiness, hopelessness, helplessness, poor attention and concentration and causes suicidal tendency. Suicidal is leading cause of death within 4-5 yrs of injury .

DEPRESSION

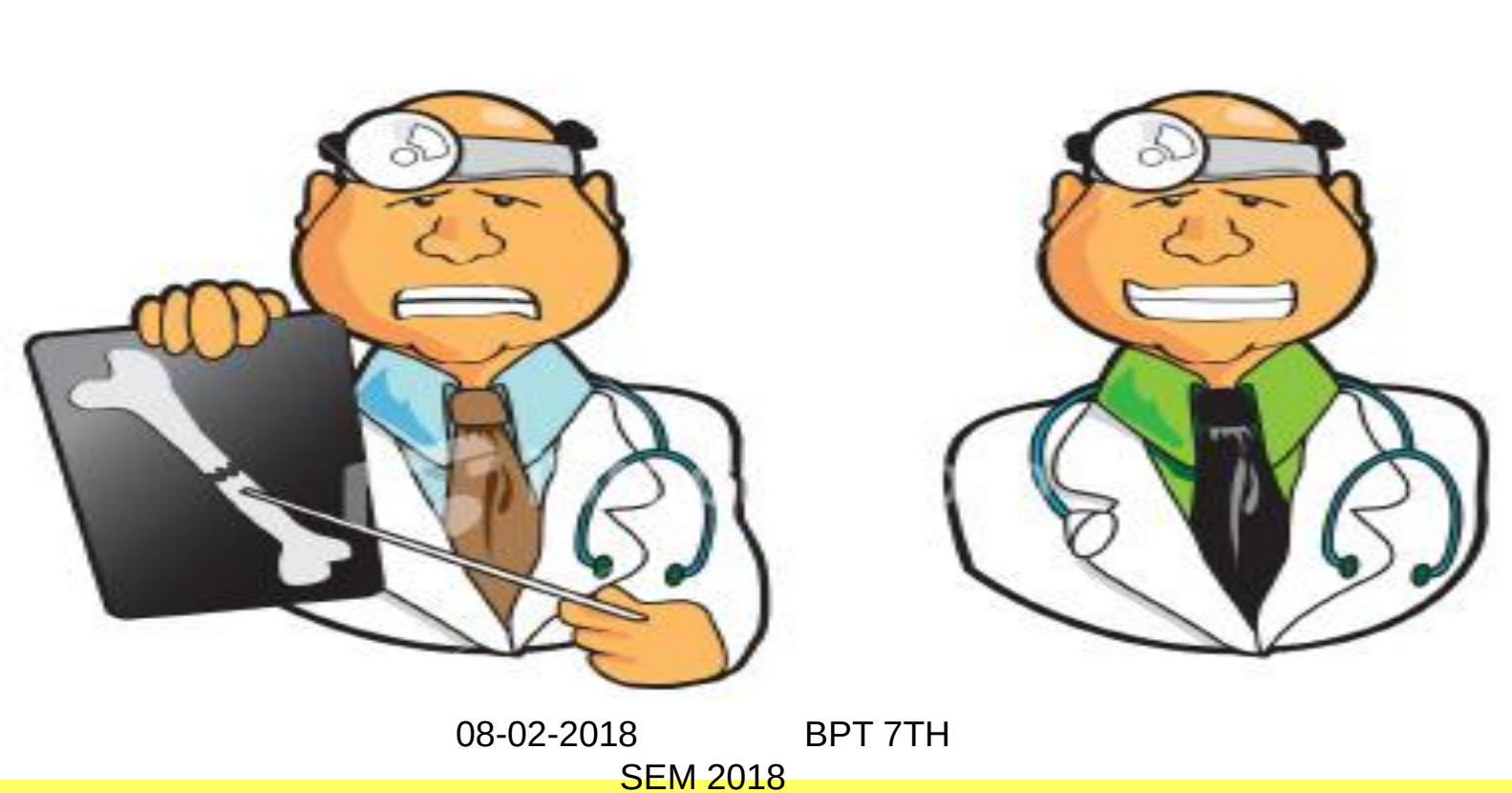
CAUSES:-

Biologic factors that include somatic effects of SCI such as fatigue
-sleep disturbances.
-Presence of general medical condition or medication.
-Due to self-body image.
-Lack of engagement in work.

MANAGEMENT:-

-Psychotherapy
-Counselling.
-Antidepressant

PROGNOSIS
OF
SPINAL CORD INJURY



IMPORTANCE: To develop realistic comprehensive rehabilitation program with limited resources & time.

DEPEND ON: Initial level of injury.
Initial strength of m/s.
Incomplete /complete
Age
Reflex recovery
Duration of injury
Rehabilitation program
Surgical intervention.

COURSE OF MOTOR RECOVERY IN ZPP IN SCI

Recovery at ZPP was better predicted by 72 hours MMT according to Brown and associates.

Ditunno & cowokers characterized recovery in ZPP as:

1.Conduction block of motor neurons due to edema, damage of myelin sheath which recover within 6 to 8 weeks.

2.Perpherial nerve sprouting of intact motor neurons occur after 2 months with m/s fiber hypertrophy.

- **COMPLETE TETRAPLEGIA**
- Patient regain 1 motor level from 1week to 1year(30%-80%).
- At 72hours-1week patient regain next motor level to at least 3/5 at 1year depend on initial strength & motor level.
  - 30%-40% of first 0/5 m/s.
  - 70%-80% of 1/5 or 2/5 m/s.
  - Presence of sensation increases chances of recovery at that level.
- At 1month,recovery at 1year
  - 95% of 1/5 or 2/5 recover to 3/5
  - 50%-60% of first 0/5 m/s recover to1/5
  - 25% of first 0/5 recover 3/5
  - Less than 10% of second 0/5 recover to 1/5
  - About 1% of second 0/5 recover to 3/5.
- Faster an initial 0/5 m/s starts to recover ,the greater prognosis for recovery.
- Most UE recovery during first 6 months with greatest rate changes during first 3 months.

INCOMPLETE TETRAPLEGIA

SENSORY

- Prognosis for motor recovery better if PIN PRICK sensation preserved rather than light touch.
- At 1 month may regain LE m/s recovery if B/L sacral pin sensation present.

MOTOR

- A greater % of key m/s recovery antigravity strength or better (>or=3/5) distal to NLI.
- Recovery is favorable :
  - 2/5 at 1 month recover to 3/5
  - 1/5 at 1 month recover to 3/5 at 1year(73%)
  - 0/5 at 1 month recover to 1/5 at 1year(54%)
  - 0/5 at 1 month recover to 3/5 at 1 year(20%)

MOTOR RECOVERY OF UE IN TRAUMATIC QUADRIPLEGIA

Harris & associates suggested that **recovery in UE** with complete Lesion at cervical level may be **more important than recovery in LE.**

Ditunno &associates in retrospective studies reported that **recovery of m/s strength in UE of zone of partial preservation could be predicted within 1 week after SCI**. They demonstrated that ECR motor power recovered to 3/5 by 8 mths after SCI if bic -eps strength was at least 2/5 at time of injury.

Reconstructive surgery for improving UE function is another intervention recommended by several authors to be performed only after 1 year & plateau of neurological reco -very.

COMPLETE PARAPLEGIA

- 80% with NLI at or below T9 : Some return of LEM function i.e., hip flexor & knee extensor.
- 55% with NLI at or below T12: LEM recovery higher.
- Presence of intact abdominal m/s initially: hip flexor recovery at 1year.
- Presence of hip flexors :knee extensor recovery i.e.,68% with hip flexors
  - 1/5 or2/5---3/5.
  - hip flexors 1/5 initially---1/5 knee extensor.

INCOMPLETE PARAPLEGIA

- Sensory recovery reached a plateau at 3 months, earlier than motor recovery.
- 80% of patient regain hip flexors &knee extensor to 3/5 at 1year.
- 55% of patient 0/5 m/s---1/5 at 1 month.
- 26% of patient 0/5 m/s---3/5 at 1year.
- 86% of patient 1/5 m/s---3/5 at 1 year.

COMPLETE TO INCOMPLETE STATUS

Within 1-3 months----better prognosis for motor recovery.

SENSATION EFFECT ON MOTOR RECOVERY:

Patient with sacral pin &normal pin sensation between level of Injury &sacral dermatomes----Greater motor recovery.
(Brown et al found that **presence of pin sensation at C5 within 1 week of injury, was highly predictive of C6 motor recovery to 3/5 or more in C4&C5 motor complete patient**.)
Study shown that absence of pin prick sensation predicts **poor bladder** recovery in pt. with T12-L1# &SCI (Arch.Phy.Med.Reh 2003,84)

REFLEX RECOVERY

- The **continued presence of the DPR(L5-S1) has poor prognostic significance for neurological recovery**.
- Duration of persistence of DPR has prognostic significance for walking.
- Holdsworth reported as segment reflexes recover from rostral SCI & accompanying recovery of voluntary movement is observed, then the prognosis for ultimate functional recovery is extremely poor.
- The **DPR was always seen in severe injuries & less frequently &of shorter duration(1day) in ASIA D pt. but was associated with poor prognosis for walking when persisted longer than 2 days in ASIA A,B C pt.**

- BC reflex absence or presence define LMN or UMN lesions.
- **SCI with associated spinal shock has worse prognosis than does the same degree of injury without spinal shock**

AMBULATION:

•Complete tetraplegia---- donot regain LE movement.

•Incomplete tetraplegia(within 1week)-sensory:sufficient motor recovery to walk.

----motor: better for ambulation (46% ambulate 1 year)

•Incomplete injuries if younger 50years----excellent prognosis for walking.

older than 50years---40% chance of walking if slight m/s power after post injury.

Retrospective study shown presence of preserved **pin appreciation** below zone of injury had **excellent prognosis** to regain functional ambulation.

SURGERY

The current literature shown variable results, increases risk of neurological deterioration with early surgical intervention. Others has shown that early surgery may decrease hospitalization & enhance recovery. **Retrospective study (Arch Phys Med Rehab 2004)** shows that early spinal surgical intervention (<72hrs after injury) was associated with earlier transition from acute care hospitalization & ↓ed the overall LOS in hospital, relative to surgery after 72 hours. Costs were higher in late surgery groups. But no difference in neurological or functional changes bet. surgical groups. Pulmonary complications frequent in late surgical intervention.

Schlegel et al reported fewer medical complications in pt. who underwent decompressive surgery less than 72 hours after injury compared with those who had surgical treatment after 72 hours.

Nonetheless ,spinal surgical procedures carry risk such as neurological deterioration ,bleeding, infection and dysphasia. Additionally, several studies have noted the beneficial effects of surgical intervention for earlier patient mobilization, which contrib -ute to earlier transition to rehabilitation, shorter hospital LOS, de -creased hospital costs.

Experimental studies have indicated that **olf -actory ensheathing glia may support partial neurological recovery following SCI (mechanism include axonal regeneration local sprouting& remyelination.), fetel allograft are also helpful.** (J.Spinal cord 2006)

Rehabilitation to promote neurological recovery

Rehabilitation is a complex interdisciplinary process requiring appropriate dose &timing of multiple intervention to achieve optimal patient outcome. much rehabilitation addresses adaptive equipment, functional training, behavioral substition,pt/family education, psychological adjustment to disability. Superimposed on these general rehabilitation interventions is the potential on neurological recovery.

INCOMPLETE SYNDROME

CCS: Favorable prognosis.
Recovery in sequence of LE---Bladder function---UE (proximal)---Intrinsic hand function

BSS: Ipsilateral proximal extensor----distal flexors.
UL weaker>LL, more likely to ambulate at discharge.
Recovery of bowel &bladder function.

ACC: 10%-20% Chances of recovery.

DIAGNOSTIC TECHNIQUES

- No hemorrhage (incomplete injury)----GOOD prognosis for motor recovery in UE&LE.
- NCV Loss of motor potential of median &ulnar nerves---- no active hand function.
- MRI: Hemorrhage (complete injury)----POOR prognosis Loss of tibial &peroneal nerves----No LE recovery.

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