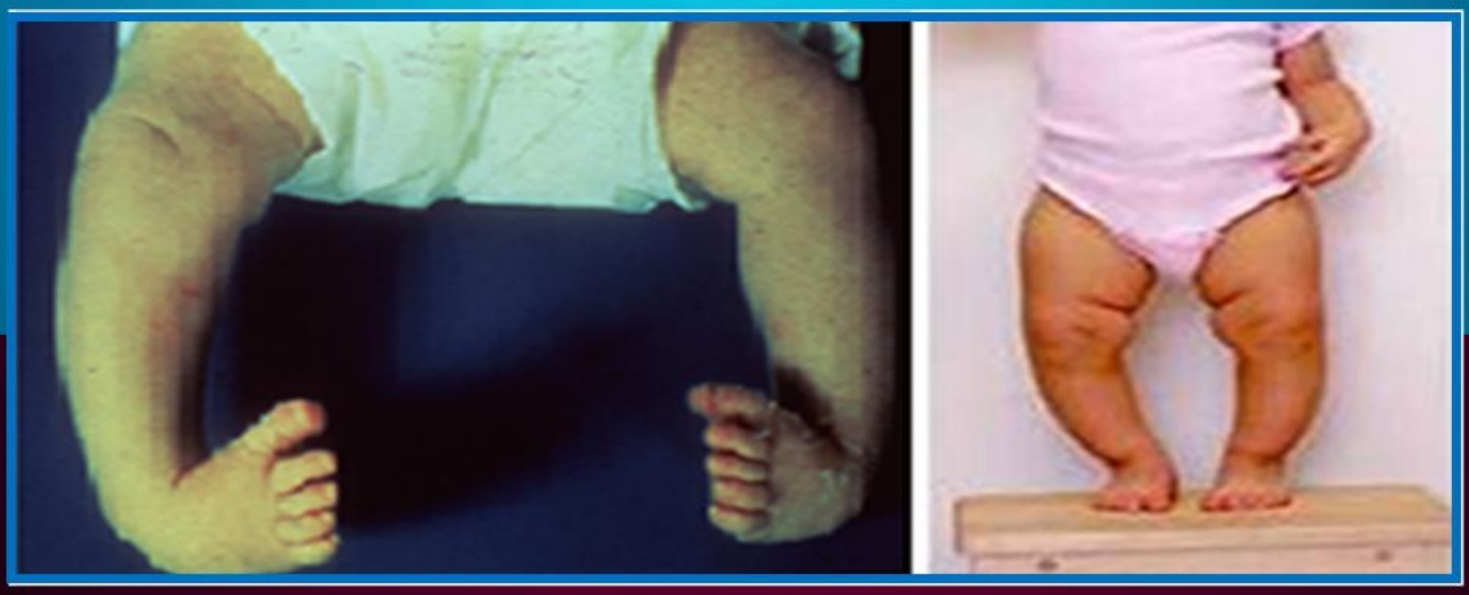
Journal of Neonatal Research and Pediatrics Care

Essential Mechanisms in Orthopedic Pediatric Rehabilitation



Ass. Prof. Dr. Ahmed Azzam

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Essential Mechanisms in Orthopedic Pediatric Rehabilitation

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Egypt

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Author Definition

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Dr. Ahmed Mohamed Azzam is an Egyptian assistant professor in pediatric rehabilitation from Mansoura city in the heart of the Niles Delta. Currently, he is Ass. Professor in Department of physiotherapy for developmental disturbance and pediatric surgery, Faculty of physical therapy, Cairo university, Egypt. Dr. Azzam has been awarded his PH.D degree in pediatric rehabilitation in Faculty of physical therapy, Cairo university, Egypt. He is holding a master degree in pediatric rehabilitation from Faculty of physical therapy, Cairo University, Egypt. After 10 years of hard work, he finally issued his series of rehabilitation (essential mechanisms of neurological pediatric rehabilitation, essential mechanisms of orthopedic pediatric rehabilitation and practical guide to occupational therapy). Dr. Azzam aimed at making pediatric rehabilitation interesting and of direct application to physiotherapy and occupational therapy. In our books text is linked with graphs, tables and diagrams to enable physiotherapy, occupational therapy students and readers to grasp real mechanisms that are essential for safe pediatric rehabilitation practice. Dr. Azzam sincerely hope that your reading of pediatric rehabilitation and occupational therapy books not only profitable to you but also stimulate your permanent interest in the fascinating subject of physiotherapy.

Preface

This book is a compact and accessible guide to the wide range practice of physical and occupational therapy. I feel too much gratitude to our professor Emam El negmy for his deep, continuous guidance, support, advices and consultation from time to time. Also too much gratitude to all our professors in department of physiotherapy for growth and development disturbance and pediatric surgery for their support and advices underlying mechanisms necessary to be awarded in pediatric rehabilitation are discussed as they are presented to the clinician giving background information and guidance. The main aim of this book is introducing understanding mechanisms used to be a base of evaluating the level of gross and fine motor skills, balance, tone disturbance, coordination, gait deviation, muscle flexibility, facilitatory techniques and inhibitory techniques should be included in the treatment program. This book is divided into 5 main chapters. The first is about Congenital anomalies mechanisms present in pediatric rehabilitation. In chapter 2 the reader learns the underlying mechanisms necessary in understanding of orthopedic disease in pediatric rehabilitation. In chapter 3 the reader learns about underlying mechanism of Trumatology in pediatric rehabilitation face physical therapist in pediatric rehabilitation. Chapter 4 demonstrated the underlying mechanisms of different type, evaluative and treatment techniques in Posture deviation. Chapter 5 learns the reader about underlying mechanisms of rehabilitation of soft tissue disorder referred to rehabilitation clinic pediatric. This book is useful for physical and occupational therapist especially pediatric physical therapists and pediatric occupational therapists.

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Contents

Abstract

The main aim of this book is introducing understanding mechanisms used to be a base of evaluating the level of gross and fine motor skills, balance, tone disturbance, coordination, gait deviation, muscle flexibility, facilitatory techniques and inhibitory techniques should be included in the treatment orthopedic pediatric rehabilitation. This book is divided into 5 main chapters. The first is about Congenital anomalies mechanisms present in pediatric rehabilitation. In chapter 2 the reader learns the underlying mechanisms necessary in understanding of orthopedic disease in pediatric rehabilitation. In chapter 3 the reader learns about underlying mechanism of Trumatology in pediatric rehabilitation face physical therapist in pediatric rehabilitation. Chapter 4 demonstrated the underlying mechanisms of different type, evaluative and treatment techniques in Posture deviation. Chapter 5 learns the reader about underlying mechanisms of rehabilitation of soft tissue disorder referred to rehabilitation clinic pediatric. This book is useful for physical and occupational therapist especially pediatric physical therapists and pediatric occupational therapists.

Keywords: Pediatric rehabilitation; Mechanisms; Orthopedic

Abbreviations: CA: Chromosomal Abnormalities; NTD: Neural Tube Deformity; CDH: Congenital Dislocated Hip; DNA: Deoxyribonucleic Acid; mRNA: Messenger Ribonucleic Acid; CSF: Cerebrospinal Fluid; ADL: Activities of Daily Living; SCM: Sternocleidomastoid; TENS: Transcutaneous Electrical Nerve Stimulation; AMC: Arthrogryposis Multiplex Congenita; JRA: Juvenile Rheumatoid Arthritis; LL: Lower Limb; ROM: Range Of Motion; GTOs: Golgi tendon organs; SMI: Sub Occipital Muscles Inhibition; RA: Rectus Abdominis; EO: External Obliques; GAE: Graduated Active Exercise.

Chapter 1

Congenital Anomalies

Introduction

Underlying mechanism of risk factors leading to congenital anomalies

1. Pollution (Environmental toxical substances)**:** Drinking water is often a vessel through which harmful toxins travel. Studies have shown that heavy metals, elements, nitrates, nitrites, fluoride can be carried through water and cause congenital disorders.
2. Repeated exposure to X ray: Chromosomal abnormalities and genetic mutation.
3. Repeated exposure to electromagnetic waves: Chromosomal abnormalities and genetic mutation.
4. Uncontrolled drugs (Hypnotic and antiemetic): The most typical disorder induced by thalidomide was reductional deformities of the long bones of the extremities (*phocomelia*), otherwise a rare deformity, which therefore helped to recognize the teratogenic effect of the new drug. Among other malformations caused by thalidomide were those of ears, eyes, brain, kidney, heart, digestive and respiratory tract. 40% of the prenatally affected children died soon after birth. As thalidomide is used today as a treatment for multiple myeloma and leprosy, several births of affected children.
5. Continuous fever to the mother: Meningitis or encephalitis to the fetus.
6. Placental injury: Different attacks of infection.
7. Partial lesion of umbilical cord: Reduced blood o_2 to fetus brain leads malformation.
8. +Consanguinity: Consanguinity is important factor which was found to increase the risk of CA .In Lebanon, as in Middle Eastern communities, consanguinity is high. This study reports a significant positive association between the consanguinity and CA in the Lebanese population. Many studies have demonstrated male predominance amongst congenital malformed babies.
9. Deficiency of vitamin B complex special folic acid: Responsible for formation of neural tube in 1^{st} 3 months of pregnancy For example, a lack of folic acid, a vitamin B, in the diet of a mother can cause cellular neural tube deformities that result in spina bifida. Congenital disorders such as a neural tube deformity (NTD) can be prevented by 72% if the mother consumes 4 milligrams of folic acid before the conception and after 12 weeks of pregnancy. Folic acid, or vitamin B_{12}, aids the development of the foetal nervous system.
10. Intra- uterine mal position: CDH (congenital dislocated hip)
11. Mal-nutrition: Deficiency of essential vitamins and substances→congenital malformation
12. Genetic factor: Genetic causes of congenital anomalies include inheritance of abnormal genes from the mother or the father, as well as new mutations in one of the germ cells that gave rise to the fetus. Male germ cells mutate at a much faster rate than female germ cells, and as the father ages, the DNA of the germ cells mutates quickly. If an egg is fertilized with sperm that has damaged DNA, there is a possibility that the fetus could develop abnormally.
13. Fetal alcohol exposure: The mother's consumption of alcohol during pregnancy can cause a continuum of various permanent birth defects like crano-facial abnormalities brain damage, intellectual disability, heart disease, kidney abnormality, skeletal anomalies, ocular abnormalities.
14. Toxic substances**:** Substances whose toxicity can cause congenital disorders are called "teratogens", and include certain pharmaceutical and recreational drugs in pregnancy as well as many environmental toxins in pregnancy.
15. Paternal smoking**:** Paternal smoking prior to conception has been linked with the increased risk of congenital abnormalities in offspring. Smoking causes DNA mutations in the germ line of the father, which can be inherited by the offspring. Cigarette smoke acts as a chemical mutagen on germ cell DNA. The germ cells suffer oxidative damage, and the effects can be seen in altered mRNA production
16. Infections: A vertically transmitted infection is an infection caused by bacteria, viruses or, in rare cases, parasites transmitted directly from the mother to an embryo, fetus or baby during pregnancy or childbirth. It can occur when the mother gets an infection as an inter-current disease in pregnancy.
17. Physical restraint**:** External physical shocks or constrainment due to growth in a restricted space, may result in unintended deformation or separation

of cellular structures resulting in an abnormal final shape or damaged structures unable to function as expected.

18. Socioeconomic status**:** A low socioeconomic status in a deprived neighborhood may include exposure to "environmental stressors and risk factors. Socioeconomic inequalities are commonly.
19. Father's age: Fathers contribute proportionally more DNA mutations to their offspring via their germ cells than the mother, with the paternal age governing how many mutations are passed on. This is because, as humans age, male germ cells acquire mutations at a much faster rate than female germ cells. Maternal age is an important parameter in the birth of a congenitally malformed fetus. Older mothers (35 years of age or older) have anomalous births, giving birth malformed fetuses.
20. Unknown or multi-factorial: Although significant progress has been made in identifying the etiology of some birth defects, approximately 65% have no known or identifiable cause. These are referred to as sporadic, a term that implies an unknown cause, random occurrence regardless of maternal living conditions, and a low recurrence risk for future children. For 20-25% of anomalies there seems to be a "multi-factorial" cause, meaning a complex interaction of multiple minor genetic anomalies with environmental risk factor

Spina Bifida

Underlying mechanism of different types of spina bifida

a. Spina bifida occulta (A-symptomatic type): congenital absence of the posterior vertebral arch (both lamina, spinous and transvers process of the vertebrae).

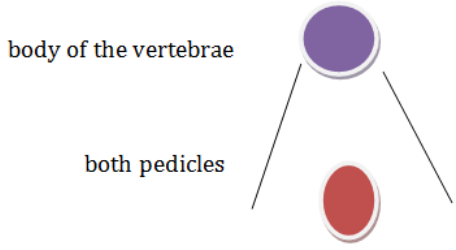

Figure 1: Spina bifida occulta.

b. Meningocele: Congenital absent of posterior vertebral arch with displacement of meninges outside spinal canal appears as a sac filled with CSF.

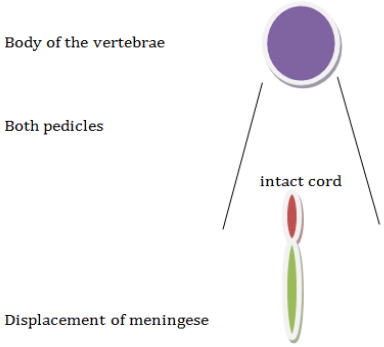

Figure 2: Meningocele.

c. Meningomyelocele: Congenital absent of posterior vertebral arch (both lamina, spinous and transvers process of the vertebrae) + displacement of the cord+ displacement of meninges filled with CSF.

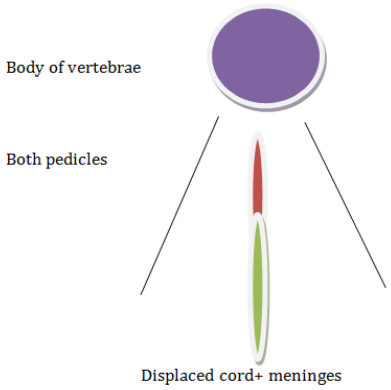

Figure 3: Meningomyelocele.

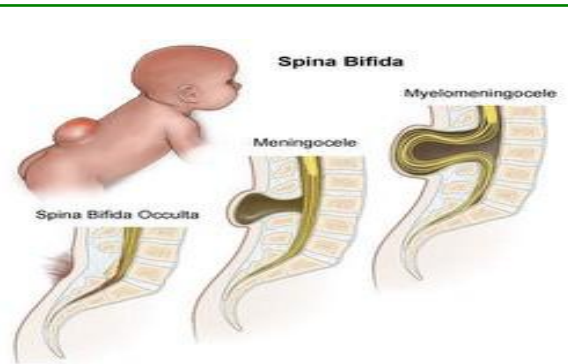

Figure 4: Different types of spina bifida.

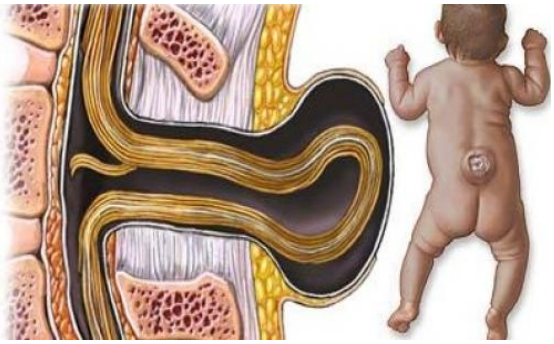

Figure 5: Meningomyelocele.

Underlying mechanism of bad prognosis in spina bifida

a. Physiotherapy results depend mainly on sensory feedback as a base of pyramid we build in till we reach to the gaining skill.
b. Spina bifida suffering from sensory loss due to compression of meningomyelocele sac on nerve root leads complete loss of sensation in both LL and urine and bowel in continence.

Underlying mechanism of physical problems of spina bifida

a. Loss of sensation due to nerve root compression
b. Flaccid paralysis of LL muscles
c. Uncontrolled urine and stool
d. Deformity
e. Hypotonia
f. Hypo-reflexia
g. Tightness of hamstring, hip adductor and calf muscle
h. Associated with CDH or hydrocephalus or both
i. Delayed mile stone due to weakness and loss of postural reaction
j. Weakness of trunk and upper limb muscles
k. Atrophy of LL muscles
l. Poor balance reaction
m. Poor ADL activities

Underlying mechanism of treatment of spina bifida

1. Facilitatory techniques (exteroceptive stimulation, proprioceptive stimulation and vestibular stimulation).
2. Faradic stimulation for treatment of atrophy and prevent more atrophy.
3. GAE for trunk muscles.
4. Balance training program.
5. Strengthing ex. for UL depressors and extensors for facilitation of crutches ambulation.
6. Gait training in closed environment.
7. Proprioceptive training (approximation, weight bearing).
8. Bowel and bladder hygiene.
9. Treatment of deformity by determining weak and tight side.
10. Mild, decent and gentle stretch for tight muscles.
11. Facilitation of mile stones.
12. Facilitation of ADL activities (feeding, dressing, bathing and transferring).
13. Using of orthoses for assist movement and prevent deformity.

a. Hip-knee-ankle foot orthoses was used if there paralysis of quadriceps.
b. If there is paralysis of hip extensors we use pelvic band.
c. If quadriceps is intact we use short leg brace.
d. Elbow crutches were used for more bases of supports.

Developmental hip dysplasia (Congenital dislocated hip)

Underlying mechanism of tests of hip instability

a. Telescoping test: Perform unilaterally, flexion hip 90 and knee 90 also perform distraction and compression we hear an audible click.
b. Ortolani test: Perform bilaterally by making abduction of both hips we find limitation of abduction on affected side.
c. Barollows test: Performed unilaterally by making in and out movement of head of femur we sense a palpable click.

Underlying mechanism of physical problems in CDH

a. The weakest muscles in CDH are (hip extensors, hip abductors and quadriceps).
b. The tightness muscles in CDH are (hamstring, hip adductors and hip flexors).
c. Shortening of the limb (leg length discrepancy).

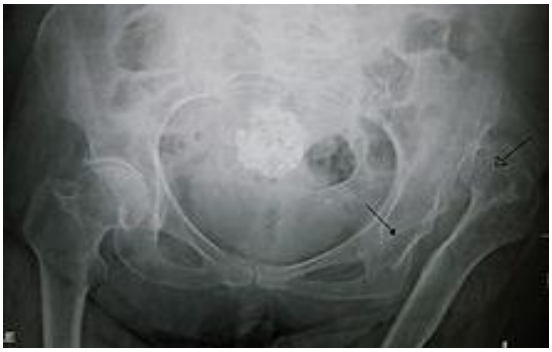

Figure 6: Developmental hip dysplasia (congenital dislocated hip).

Underlying mechanism of treatment of CDH

a. Splinting and positioning in flexion, abduction, external rotation (closed packed position).
b. Graduated active ex. for gluteus maximus and gluteus medius, quadriceps.
c. Faradic stimulation for gluteus maximus and medius, quadriceps.
d. Mild, gentle and decent stretching of tight muscle (iliopsoas, hip adductor, hamstring).
e. Gait training by using long leg brace with pelvic bands.
f. Post operative patients: Need Strengthing ex for gluteus maximus, gluteus medius, quadriceps.
g. Hydrotherapy.
h. Facilitatory techniques on the weak muscles (quick stretch, vibration and faradic stimulation to gluteus maximums and gluteus medius and quadriceps).
i. Proprioceptive training (static and dynamic training).

N.B packed positions of hip and shoulder joints.

a. Closed packed position of hip is abduction and external rotation.
b. Loosed packed position of shoulder is abduction and external rotation.
c. Closed packed position of shoulder adduction and internal rotation.
d. Loosed packed position of the hip is adduction with internal rotation

Foot deformities

Underlying mechanism of different types foot deformities

Characteristics	Talipus equino varus	Talipus equino valgus	Talipus Calcaneo varus	Talipus Calcaneo valgus	Flat foot (absent arch)	Pes-cavus (high arch)
Weakest muscle	Perineous tertius	Tibialis anterior	Peroneous longus	Tibialis posterior	Weakness of intrinsic muscles of foot+ absent of medial longitudinal arch of foot	Has less effect on body alignment
Shorted muscle	Tibialis posterior	Peroneous longus	Tibialis anterior	Perineous tertius	Types: Congenital (absent of arch in both weight bearing and non weight bearing position while acquired one absent in weight bearing only	
Position of deformity	Plantar flexion+ inversion	Plantar flexion+eversion	Dorsiflexion+ inversion	Dorsiflexion+ eversion	Walking on medial side of foot	
Support used in medical shoes	Wedge on lateral aspect of shoes (at lower side of the foot)	Wedge on medial aspect of shoes (at lower side of the foot)	Thermoplastic rounding on dorsum of foot for correction with posterior splint	Thermoplastic rounding on dorsum of foot for correction with posterior splint	Medial cushion Support inside shoes	
Splint	Wedge on lateral aspect of splint (at lower side of the foot)	Wedge on medial aspect of splint (at lower side of the foot)	Splint with Thermoplastic rounding on dorsum of foot	Splint with Thermoplastic rounding on dorsum of foot	Splint with Medial cushion Support inside shoes	
Treatment	-stretch of tibialis posterior (put foot in dorsi-flexion and eversion) -all facilitatory techniques to pereneous tertius (faradic stimulate on one electrode under head of fibula and other lateral to ant. tibial group - balance training for facilitation of weak muscle, relaxation of contracted muscle-	-stretch of peroneus longus (put foot in dorsi-flexion and inversion) -all facilitatory techniques to tibialis anterior (faradic stimulation one electrode under head of	-stretch of tibialis ant.(put foot in plantar flexion+eversion) -all facilitatory techniques to pereneous longus (faradic stimulation one electrode under head of fibula and other lateral to anterior tibial group	-stretch of peroneus tertius (put foot in plantar flexion+inversion) -all facilitatory techniques to tibialis posterior As quick stretch - balance training for facilitation of weak muscle, relaxation of	-all facilitatory techniques can be used on sole of foot As -faradic stimulation one electrode on base of toes and other on heel -walking on rough surface -strengthening of intrinsic muscles of sole of the foot	

	mobilization of the ankle and foot joints	fibula and other on anterior tibial group - balance training for facilitation of weak muscle, relaxation of contracted muscle - mobilization of the ankle and foot joints	- balance training for facilitation of weak muscle, relaxation of contracted muscle - mobilization of the ankle and foot joints	contracted muscle - mobilization of the ankle and foot joints	- balance training for facilitation of weak muscle, relaxation of contracted muscle - mobilization of the ankle and foot joints	

Table 1: Underlying mechanism of different types foot deformities.

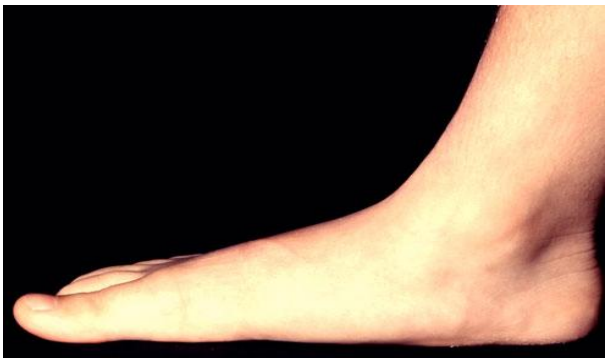

Figure 7: Flat foot.

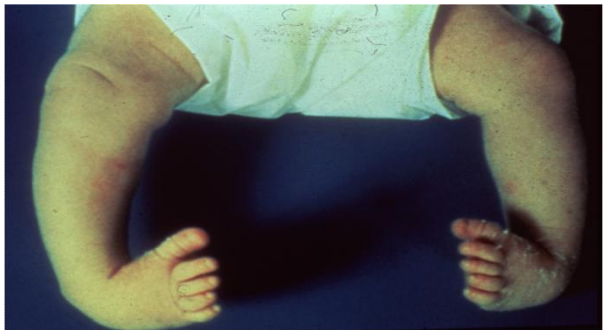

Figure 8: Club foot.

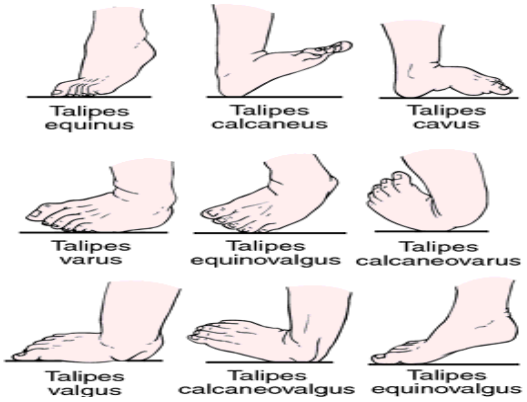

Figure 9: Different types of feet deformity.

Underlying mechanism of functional muscle test to ankle and foot muscles

All tibialis make inversion-all perenii make eversion.

Charactaristics	Tibialis posterior	Tibialis anterior	Pereneous tertius	Pereneous brevis	Pereneous longus
Primary movement	inversion	dorsiflexion	eversion	eversion	eversion
Secondary movement	Plantar flexion	inversion	dorsiflexion	Mid-position	Plantar flexion
Position of functional ms. test	Test primary movement Put subtalar joint in eversion and ankle in plantar flexion	Test primary movement Put ankle joint in plantar flexion and subtalar in inversion	Test primary movement Put subtalar joint in inversion with Dorsiflexion in ankle joint	Test primary movement Put subtalar joint in inversion with mid-position in ankle join	Test primary movement Put subtalar joint in inversion with plantar flexion in ankle join
Position of tactile stimulation	Tactile stimulation on tibialis post. tendon around medial	Tactile stimulation on tibialis ant. tendon on tendons in a mid way	Tactile stimulation on pereneous tertius tendon around lateral	Tactile stimulation on pereneous brevis tendon	Tactile stimulation on pereneous longus tendon around lateral malleoluse3-5

	malleoluse3-5 times followed by inversion and make another one and show the response -functional (as normal) -subfunctional (below normal) -zero (completely paralysed)	between both malleoli 3-5 times followed by dorsiflexion and make another one and show the response -functional (as normal) -sub functional (below normal) -zero (completely paralysed)	malleoluse3-5 times followed by eversion and make another one and show the response -functional (as normal) -sub functional (below normal) -zero (completely paralysed)	around lateral malleoluse3-5 times followed by eversion and make another one and show the response -functional (as normal) -sub functional (below normal) -zero (completely paralysed)	times followed by eversion and make another one and show the response -functional (as normal) -sub functional (below normal) -zero (completely paralysed)

Table 2: Underlying mechanism of functional muscle test to ankle and foot muscles.

Torticollus

Underlying mechanism of etiology of Torticollus

Etiology

1. Congenital

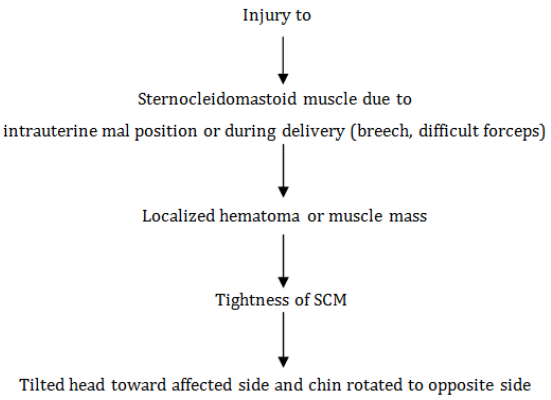

Figure 10: Etiology of Torticollus.

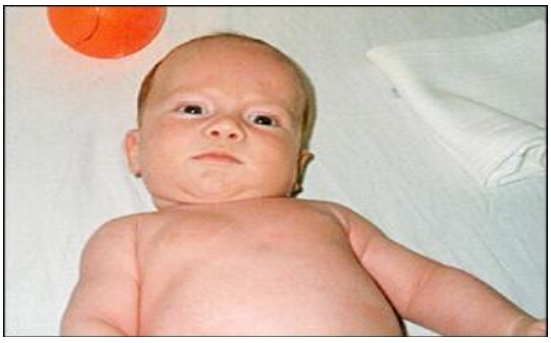

Figure 11: LT Torticollus.

2. Compensatory due to trauma
3. Infection due to muscular abscess
4. Neoplasm: cervical cord tumor
5. Neurogenic: As in poliomyelitis and dystonia

Underlying mechanism of treatment of Torticollus

1. Passive stretching for tight SCM (the muscle has two movement lateral flexion to the same side and rotation to opposite side). When make passive stretching to sternocleido mastoid we must stretch each part of muscle (lateral flexor part move to opposite side 20 second stretch then 20 second relaxation) the rotator

part (rotate to the same side 20 second stretch then 20 second relaxation).

2. Graduated Active EX. for weak SCM muscle by activating righting reaction training on opposite side of tightness, activation of neck muscles movement by scratch followed by movement on weak side then active assisted and active free.
3. Balance training for facilitation of weak SCM by activating right in, equilibrium and protective reaction on ball.
4. Pulsed US on mass for 1 minute (avoid irritation of trachea).
5. Myo-facial release on tight muscle.
6. Hot packs on tight muscle.

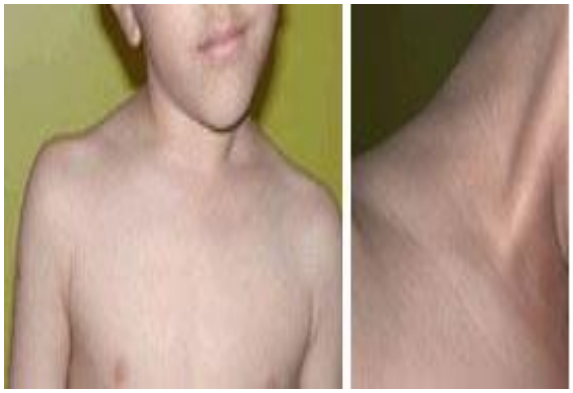

Figure 12: RT Torticollus.

N.B follow up of Torticollus occurred by round measurement to detect the decrease in muscle mass.

Knee Deformity

Underlying mechanism of knee deformity

Characteristics	Genu varum	Genu valgum	Genu recurvatum	Flexion knee deformity
Knee position	bow leg	knock knee	hyperextension knee	Loss of knee extension
Stretched side	Lateral compartment	Medial compartment	weakness of quadriceps (cannot locked the knee) make locking by the hand -weakness of hamstring lead to powerful quadriceps contraction -imbalance between quadriceps and hamestring	-weakness of quadriceps and ant. tibial group -
Contracted side	Medial compartment	Lateral compartment		shortenining of hamestring and gastrocnemius muscles
Foot position	Inversion subtalar joint with weight bearing on lateral side of foot	Eversion subtalar joint with weight bearing on medial side of foot		Weight bearing on toes and walking by toe gait
evaluation	-Observed from A-P view-measure distance between both knees by tape	-Observed from A-P view-measure distance between both feet	-nonweight bearing on plinth and make hyperextension knee-weight bearing make observation from lateral view	-flexibility tests(for hamstring and gastrocnemius)
treatment	On medial compartment -hot packs -passive stretching -myo facial release On lateral compartment All facilitatory techniques can be used	On lateral compartment -hot packs -passive stretching -myo facial release On medial compartment All facilitatory techniques can be used	-treat the cause Using facilitatory techniques specially electric stimulation and is ometric ex for gaining strengthen	-passive stretching to hamstring and gastrocnemius -graduated active ex. to quadriceps and ant tibial groups
Splint and medical shoes	Wedge on lateral side of foot	Wedge on medial side of foot	-Ankle foot orthosis -knee cage -posterior knee splint	Posterior knee splint

Table 3: Underlying mechanism of knee deformity.

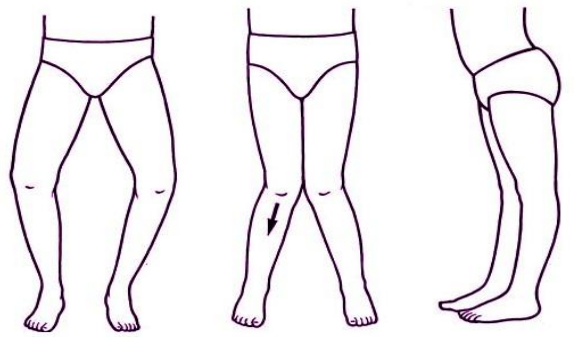

Figure 13: Different types of knee deformity.

Hydrocephalous

Underlying mechanism of hydro-cephalous

The functions of CSF include

1. Buoyancy: The actual mass of the human brain is about 1400 grams however the net weight of the brain suspended in the CSF is equivalent to a mass of 25 grams. The brain therefore exists in neutral buoyancy, which allows the brain to maintain its density without being impaired by its own weight.
2. Protection: CSF protects the brain tissue from injury when jolted or hit.
3. Chemical stability: CSF flows throughout the inner ventricular system in the brain and is absorbed back into the bloodstream, rinsing the metabolic waste from the central nervous system through the blood–brain barrier. This allows for homeostatic regulation of the distribution of neuroendocrine factors, to which slight changes can cause problems or damage to the nervous system.
4. Prevention of brain ischemia: The prevention of brain ischemia is made by decreasing the amount of CSF in the limited space inside the skull. This decreases total intracranial pressure and facilitates blood perfusion.

Figure 14: Hydrocephalus.

CSF circulation

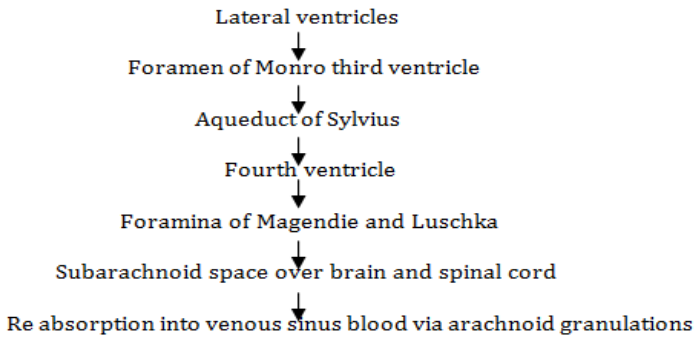

Figure 15: Circulation of Cerebrospinal fluid.

Any defect in this circulation as over production from the choroid plexus in lateral ventricles or obstruction of the passage of circulation as Arnold chiari syndrome or mal absorption of CSF by the villi and venous system. Hydrocephalus can be caused by congenital or acquired factors. Congenital causes include Spina Bifida, Arnold–Chiari malformation, cranio-synostosis, Dandy–Walker syndrome, and Vein of Galen malformations. Acquired causes include hemorrhage, meningitis, head trauma, tumors, and cysts.

Two types of hydrocephalus are commonly described non-communicating hydrocephalus and communicating hydrocephalus, although there is evidence that communicating forms can lead to obstruction of CSF flow in many instances. The fluid flows through the inter-ventricular foramen (of Monro) into the third ventricle, is augmented by fluid formed by the choroid plexus of this ventricle, and passes through the cerebral aqueduct (of Sylvius) to the fourth ventricle, which also possesses a choroid plexus. The CSF from all these sources, as well as any formed in the central canal of the spinal cord, escapes from the fourth ventricle into the subarachnoid space through the median aperture (of Magendie) and lateral aperture (of Luschka). The CSF then circulates through the freely communicating subaracchnoid cisterns at the base of the brain. From the cisterns, most of the CSF is directed upward over the cerebral hemispheres and smaller amounts pass downward around the spinal cord.

Chapter 2

Orthopedic Disease in Rehabilitation

Hemophilia

Underlying mechanism of hemophilia

Is a disease that prevents blood from clotting properly, so a person who has it bleeds more than someone without hemophilia does. It's a genetic disorder, which means it's the result of a change in genes that was either passed from parent to child or happened as a baby was developing in the womb. Hemophilia usually is inherited. "Inherited" means that the disorder is passed from parents to children through genes. People born with hemophilia have little or no clotting factor. Clotting factor is a protein needed for normal blood clotting. There are several types of clotting factors. These proteins work with platelets (PLATE-lets) to help the blood clot.

Platelets are small blood cell fragments that form in the bone marrow-a sponge-like tissue in the bones. Platelets play a major role in blood clotting. When blood vessels are injured, clotting factors help platelets stick together to plug cuts and breaks on the vessels and stop bleeding. The two main types of hemophilia are A and B. If you have hemophilia A, you're missing or have low levels of clotting factor VIII. About 8 out of 10 people who have hemophilia have type A. If you have hemophilia B, you're missing or have low levels of clotting factor IX. Rarely, hemophilia can be acquired. "Acquired" means you aren't born with the disorder, but you develop it during your lifetime. This can happen if your body forms antibodies (proteins) that attack the clotting factors in your bloodstream. The antibodies can prevent the clotting factors from working.

When most people get a cut, the body naturally protects itself. Sticky cells in the blood called platelets go to where the bleeding is and plug up the hole. This is the first step in the clotting process. When the platelets plug the hole, they release chemicals that attract more sticky platelets and also activate various proteins in the blood known as clotting factors. These proteins mix with the platelets to form fibers, and these fibers make the clot stronger and stop the bleeding. Our bodies have 13 clotting factors that work together in this process (numbered using Roman numerals from I through XIII). Having too little of factors VIII or IX is what causes hemophilia. A person with hemophilia will only lack one factor, either factor VIII or factor IX, but not both.

There are two major kinds of hemophilia

a. Hemophilia A, which accounts for about 80% of cases, is when factor VIII is lacking.
b. Hemophilia B is when factor IX is lacking. Hemophilia is classified as mild, moderate, or severe, based on the amount of the clotting factor in the person's blood. If someone produces only 1% or less of the affected factor, the case is called severe. Someone who produces 2% to 5% has a moderate case, and someone who produces 6% to 50% of the affected factor level is considered to have a mild case of hemophilia.

Hemophilia facts Hemophilia is one of a group of inherited bleeding disorders that cause abnormal or exaggerated bleeding and poor blood clotting. Hemophilia A and B are inherited in an X-linked recessive genetic pattern, so males are commonly affected while females are usually carriers of the disease. Hemophilia A is caused by a deficiency of clotting Factor VIII, while hemophilia B (also called Christmas disease) results from a deficiency of Factor IX. Hemophilia varies in its severity among affected individuals. Symptoms include excessive bleeding from any site in the body; long-term damage to joints from repeated bleeding episodes is characteristic. Treatment involves coagulation factor replacement therapy. The formation of inhibitors to the treatment factor concentrates is a significant complication of treatment.

Gene therapy treatments are a source of active research and hold promise for the future. Hemophilia A and B are inherited in an X-linked recessive genetic pattern and are therefore much more common in males. This pattern of inheritance means that a given gene on the X chromosome expresses itself only when there is no normal gene present. For example, a boy has only one X chromosome, so a boy with hemophilia has the defective gene on his sole X chromosome (and so is said to be hemizygous for hemophilia). Hemophilia is the most common X-linked genetic disease. Although it is much rarer, a girl can have hemophilia, but she would have to have the defective gene on both of her X chromosomes or have one hemophilia gene plus a lost or defective copy of the second X chromosome that should be carrying the normal genes. If a girl has one copy of the defective gene on one of her X chromosomes and a normal second X chromosome, she does not have hemophilia but is said to be heterozygous

for hemophilia (a carrier). Her male children have a 50% chance of inheriting the one mutated X gene and thus has a 50% chance of inheriting hemophilia from their carrier mother.

The process of blood clotting involves a series of complex mechanisms, usually involving 13 different proteins classically termed factors I through XIII and written with Roman numerals. If the lining of the blood vessels becomes damaged, platelets are recruited to the injured area to form an initial plug. These activated platelets release chemicals that start the clotting cascade, activating a series of 13 proteins known as clotting factors. Ultimately, fibrin is formed, the protein that crosslink with itself to form a mesh that makes up the final blood clot. The protein involved with hemophilia A is factor VIII (factor 8) and with hemophilia B is factor IX (factor 9).

Signs and symptoms of hemophilia

Hemophilia can vary in its severity, depending upon the particular type of mutation (genetic defect). The degree of symptoms depends upon the levels of the affected clotting factor. Severe disease is defined as <1% factor activity, 1% to 5% factor activity is moderate disease, and greater than 5% factor activity constitutes mild disease. The extent of bleeding is dependent upon the severity (the amount of factor activity) and is similar for hemophilia A and B. With severe hemophilia (A or B), bleeding begins at an early age and may occur spontaneously. Those with mild hemophilia may only bleed excessively in response to injury or trauma. Female carriers of hemophilia have variable degrees of factor activity; some may have near normal levels and do not show any bleeding tendencies, while some may have less than the predicted 50% reduction and may bleed more often than non-carrier females.

In severe hemophilia, bleeding episodes usually begin within the first 2 years of life. Heavy bleeding after circumcision in males is sometimes the first sign of the condition. Symptoms may develop later in those with moderate or mild disease. The bleeding of hemophilia can occur anywhere in the body. Common sites for bleeding are the joints, muscles, and gastrointestinal tract. Specific sites and types of bleeding are discussed below. Hemarthrosis (bleeding into the joints) is characteristic of hemophilia. The knees and ankles are most often affected. The bleeding causes distension of the joint spaces, significant pain, and over time, can be disfiguring. Over time, joint destruction occurs, and joint replacement surgeries can be required. Bleeding into the muscles may occur with hematoma formation (compartment syndrome). Bleeding from the mouth or nosebleeds may occur. Bleeding after dental procedures is common, and oozing of blood from the gums may occur in young children when new teeth are erupting. Bleeding from the gastrointestinal tract can lead to blood in the stool. Bleeding from the urinary tract can lead to blood in the urine (hematuria). Intracranial hemorrhage (bleeding into the brain or skull) can lead to symptoms such as nausea, vomiting, and/or lethargy. Increased bleeding after surgery or trauma is characteristic of hemophilia.

Signs and symptoms of spontaneous bleeding include

a. Unexplained and excessive bleeding from cuts or injuries, or after surgery or dental work
b. Many large or deep bruises
c. Unusual bleeding after vaccinations
d. Pain, swelling or tightness in your joints
e. Blood in your urine or stool
f. Nose bleeds without a known cause
g. In infants, unexplained irritability

Emergency signs and symptoms of hemophilia include

a. Sudden pain, swelling and warmth in large joints, such as knees, elbows, hips and shoulders, and in your arm and leg muscles
b. Bleeding from an injury, especially if you have a severe form of hemophilia
c. Painful, prolonged headache
d. Repeated vomiting
e. Extreme fatigue
f. Neck pain
g. Double vision

Physical problems in hemophilia

a. Hemophilic arthropathy is characterized by chronic proliferative sinusitis and cartilage destruction. If an intra-articular bleed is not drained early, it may cause apoptosis of chondrocytes and affect the synthesis of proteoglycans. The hypertrophied and fragile synovial lining while attempting to eliminate excessive blood may be more likely to easily rebleed, leading to a vicious cycle of hemarthrosis-synovitis-hemarthrosis. In addition, iron deposition in the synovial may induce an inflammatory response activating the immune system and stimulating angiogenesis, resulting in cartilage and bone destruction
b. Painful, prolonged headache

c. Neck pain
d. Extreme fatigue
e. Pain and swelling
f. Tightness in muscles
g. Stiffness in joints

Treatments of Hemophilia

a. Hot packs on tight muscles or stiff joint very mild heat for prolonged time
b. Ice application in swelling joint or muscles (very mild prolonged time)
c. Mild, decent, gentle passive stretch for tight muscles
d. Applications of TENS for reliving joint pain
e. Faradic stimulation on weak muscles(opposite to tight muscles)
f. Mild ,decent, gentle mobilization of stiff joint
g. Medical treatment the mainstay of treatment is replacement of the blood clotting factors.

People who have mild cases of hemophilia A are sometimes treated with the drug desmopressin, also known as DDAVP. This drug stimulates release of more clotting factor by the body. Pain relievers may be prescribed for symptom relief, but pain relievers other than aspirin or non-steroidal anti-inflammatory medications (such as naproxen, ibuprofen) must be used, since these types of drugs further inhibit the blood's ability to clot.

Arthrogryposis

Underlying mechanism of arthrogryposis

Arthrogryposis, or arthrogryposis multiplex congenita (AMC), comprises nonprogressive conditions characterized by multiple joint contractures found throughout the body at birth. Arthrogryposis is a general or descriptive term for the development of nonprogressive contractures affecting one or more areas of the body prior to birth (congenitally) describes congenital joint contractures in two or more areas of the body. It derives its name from Greek, literally meaning "curving of joints" (arthron, "joint"; grȳpōsis, late Latin form of late Greek grūpōsis, "hooking"). Children born with one or more joint contractures have abnormal fibrosis of the muscle tissue causing muscle shortening, and therefore are unable to perform passive extension and flexion in the affected joint or joints.

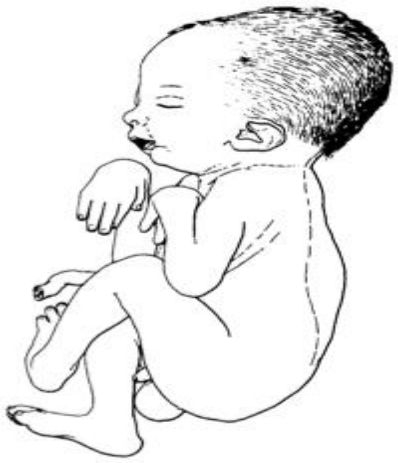

Figure 1: Arthrogryposis.

In addition to joint abnormalities, other findings occur with greater frequency in individuals with AMC. These include abnormally slender long bones of the arms and legs and cleft palate, a condition in which the roof of the mouth fails to fuse together leaving a groove across the top of the mouth. In males, the testes may fail to descend into the scrotum (cryptorchidism). Intelligence may or may not be affected. Approximately one-third of individuals with AMC may have structural or functional abnormalities of the central nervous system.

Causes

Arthrogryposis could be caused by genetic and environmental factors. In principle, any factor that curtails fetal movement of the fetus can result to congenital contractures. The exact causes of Arthrogryposis are unknown yet.

Extrinsic factors: The malformations of arthrogryposis can be secondary to environmental factors such as: decreased intrauterine movement, oligohydramnios (low volume or abnormal distribution of intrauterine fluid), and defects in the fetal blood supply. Other causes could be: hyperthermia, limb immobilization and viral infections. Myasthenia gravis of the mother leads also in rare cases to arthrogryposis. The major cause in humans is fetal akinesia.

Intrinsic factors: Arthrogryposis could also be caused by intrinsic factors. This includes molecular, muscle- and connective tissue development disorders or neurological abnormalities. There are more than 35 specific genetic disorders associated with arthrogryposis. Most of those mutations are missense, which means the mutation

results in a different amino acid. Other mutations that could cause arthrogryposis are: single gene defects (X-linked recessive, autosomal recessive and autosomal dominant), mitochondrial defects and chromosomal disorders Muscle- and connective tissue development disorders.

Loss of muscle mass with an imbalance of muscle power at the joint can lead to connective tissue abnormality. This leads to joint fixation and reduced fetal movement. Also muscle abnormalities could lead to a reduction of fetal movement. Those could be: dystrophy, myopathy and mitochondrial disorders. This is mostly the result of abnormal function of the dystrophin-glycoprotein-associated complex in the sarcolemma of skeletal muscles.

Neurological abnormalities

70-80% of the cases of arthrogryposis are caused by neurological abnormalities. Most of these result from an underlying genetic syndrome, or are due to environmental factors. The underlying aetiology and pathogenesis of congenital contractures, particularly arthrogryposis and the mechanism of the mutations remains an active area of investigation. Because identifying these factors could help to develop treatment and congenital finding of arthrogryposis.

Physical problems in arthrogryposis

A contracture: Is a condition in which a joint becomes permanently fixed in a bent (flexed) or straightened (extended) position, completely or partially restricting the movement of the affected joint. When congenital contractures occur only in one body area, it is not referred to as arthrogryposis but rather an isolated congenital contracture.

Stiffness: In most cases, affected infants have contractures of various joints. The joints of the legs and arms are usually affected; the legs are affected more often than the arms. The joints of the shoulders, elbows, knees, wrists, ankles, fingers, toes, and/or hips are also commonly affected. In addition, the jaws and back may also be affected in individuals with AMC. The shoulders may be internally rotated and drawn inward (adducted), the elbows are usually extended, and the wrists are usually flexed. In most affected individuals, the fingers are flexed and stiff. Although in most reports, the distal joints (i.e., those joints farthest away from the center of the body) are usually more severely affected, the shoulders and hips (which are proximal joints) often have significant contractures.

A tightness: The muscles of the affected limbs are shorted and may be underdeveloped (hypoplastic), resulting in a tube-shaped limb with a soft, doughy feeling. Soft tissue webbing may develop over the affected joints.

An atrophy: Occur due to prolonged stretch on weak muscles (opposite to tight muscles)

Delaying in ADL skills

Delaying in fine motor skills

Treatment of arthrogryposis includes

Physical therapy: The primary long-term goals of these treatments are increasing joint mobility, muscle strength and the development of adaptive use patterns that allow for walking and independence with activities of daily living. Standard physical therapy, which can improve joint motion and avoid muscle atrophy in the newborn period, is beneficial. Gentle joint manipulation and stretching exercises may also be beneficial.

a. Mild, decent, gentle stretch for tight muscles.
b. Mild, decent, gentle mobilization to stiff joints.
c. Hot packs on tight muscles and stiff joints.
d. Graduated active ex. for weak muscles.
e. Faradic stimulation on atrophied muscles.
f. Continous ultrasonic on tight muscles (on bulky of muscles away from metaphysic or bone ends).
g. Infra- red can be used on tight muscles and stiff joint.
h. Removable splints for the knees, wrist, elbow and feet that permit regular muscle movement and exercise are also recommended.

Occupational therapy: Facilitation of hand functions and ADL skills

Surgery

Juvenile Rheumatoid Arthritis

Underlying mechanism of juvenile rheumatoid arthritis

Is the preferred term for non infective inflammatory connective tissue disease in children less than 16 years, persistent arthritis that lasts for more than 6 weeks can be diagnosed as JRA if other causes of joint pathology have been ruled out.

Joint symptoms: Swelling, heat, loss of motion and pain when motion is attempted.

a. Most children with JRA have a negative blood test for rheumatoid factor (RF-).
b. Begin as a systemic illness (accounting for about 20% of cases).

c. Or with polyarthritis involvement (accounting for about 15% of cases).
d. Or in a pauciarticular manner involving less than four joints (accounting for about 60% of cases).
e. The aetiology is unknown and the role of genetic factors is as yet undetermined in the development of the disease it starts commonly between the ages of 3and 5 years and is uncommon in the first year of life, the incidence rises to just before puberty it is, in general, slightly more prevalent in girls the systemic onset variety is characterized by a high swinging fever a macular or maculopapular rash, eneralized, often massive lymphadenopathy and splenomegaly in about half of the cases. Ultimately the majority of these children develop arthritis usually in knees, wrist and ankles. The polyarticular variety predominantly affect girls and present with symmetrical arthritis of the small joints of the hands and fingers with characteristics spindling of the interphalangeal joints whilst in some cases the cervical spine is affected. Pauciarticular onset tends to involve one or two large joints often knees but rarely the hips except in older boys when ankles may be affected.

Physical problems in juvenile rheumatoid arthritis

a. Tenosynovitis: Tendon sheath inflammation commonly occurs in tendons of extrinsic muscles of wrist and fingers.
b. Disuse atrophy: Due to reflex spasm of muscle due to joint pain with long run lead to atrophy (protein muscles catabolism) leaving the joints less protected from external forces as weight bearing.
c. Arthritis pain.
d. Limitation of ROM.
e. Bony growth disturbance lead to deformity.
f. Retardation of general physical development.
g. Marked osteoporosis and rarefaction.
h. Delaying in bone growth due to premature fusion of the epiphysis.
i. Compression fracture and collapse of the cervical vertebrae.
j. Erosions is a rare feature except in sacroiliac joint.
k. Loss of joint surface congruinty, adhesions and osteophytes or bone spurs.
l. Tightness of muscles as tensor fascia latae, hamstring, gastrocnemius.
m. Postural deformity as scoliosis, genu valgum due to muscle imbalance with abnormal weight bearing.
n. Leg length discrepancy due to increased blood flow near to knee joint cause overgrowth of femur or tibia leading to shortening of the limb or due to joint destruction lead to closure of adjacent epiphysis.
o. Gait deviation due to pain and abnormal weight bearing.
p. Poor ADL activities: Difficulty in dressing, feeding, toileting and transferring.

Treatment of juvenile rheumatoid arthritis

a. Static and isometric ex: Especially antigravity muscles of the lower extremity (gluteus maximus, medius, quadriceps and ant. tibial. these ex. decrease pain by avoiding joint motion during muscle contraction. by using slight resistance or by using theraband.
b. Functional electrical stimulation for treatment of atrophy via stimulation protein synthesis inside muscles and increase numbers of sarcomere.
c. Facilitation of functional activity as swimming in a heated pool, using of swings as balance training.
d. Facilitation of ADL activities as feeding, dressing, toileting.
e. Facilitation of hand functions as using large circumference pencil for easy grasp.
f. Superficial heat as IR for improves circulation deep heat diathermy and US is forbidden to avoid destruction of epiphyseal plate.
g. Paraffin bath is a good way for treatment of tenosynovitis.
h. Mild, decent, gentle stretch for tight muscles.
i. Gait and balance training.
j. TENS can be used for relieving pain.
k. Splint: night splint used for correction, dynamic splint can be used to assist function.

Rickets

Underlying mechanism of rickets

A case of nutritional vita-D deficiency rickets due to dietary lack or underexposure to sunlight, malabsorbtion or combinations of these. Vita D itself is inactive. When the skin exposed to sunlight (UV supply) conversation to active metabolites which function as hormone takes place first in liver by 25 hydroxylation to form 25 hydroxy cholecalciferol and then in kidney to give1, 25 - dihydroxy cholcalciferol. Inadequate mineralization of growing bone due to lack of vita D or its active metabolites, but it may also be caused by sever calcium deficiency or by hypophosphateamia.

C/P and physical problems

a. Slight decrease of height due to deformities.
b. Enlargment of the head (increase of skull circumference).
c. Delayed closure of anterior fontanels.
d. Box shaped skull frontal and parietal bossing.
e. Delayed teething with tendency to develop caries.

f. Enlargment of lower radio-ulnar epiphysis, bowing of forearm, genu valgum, genu varum, genu recurvatum, coxa vara curvature of the neck of the femur, ant-lateral bowing of femur, pigeon chest due to sterna protrusion and funnel chest, green stick fractures (fracture of cortex on one side and intact cortex on other side), broadening of epiphysis especially in wrist and ankle.
g. Weakness of paravertebral muscles and laxity of spinal ligaments lead to scoliosis, hyperlordosis and kyphosis (apparent: appear in sitting and disappear when child held suspended from shoulder-smooth-in dorsolumbar region).
h. Constipation due to intestinal muscles Hypotonia.
i. Delayed motor milestones (sitting, standing, crawling, walking).
j. Pelvis is contracted inlet and outlet leading to obstructed labour later on.

Treatment of rickets

a. Repeated exposure to sunlight to get U.V to activate vita D.
b. Supplementation of the diet with daily requirement of vita D (liver, eggs, fortified milks, cod liver oil)
c. Increased of oral calcium intake.
d. Treatment of deformity
   i. Superficial heat on tight side and faradic stimulation on weak side.
   ii. Strength of the weak side.
   iii. Stretch of the tight side.
   iv. Maintain improvement by splint.
e. Faradic stimulation for treatment and avoid atrophy.
f. Balance training to improve postural reaction.
g. Strengthing ex. to back muscles by graduated active ex. and by functional activity as in swimming.
h. Facilitation of delayed milestone.

Chapter 3

Trumatology in Pediatric Rehabilitation

Introduction

Axial skeleton

Skull cranium and facial bones, hyoid bone (anchors tongue and muscles associated with swallowing), vertebral column (vertebrae and disks) and thoracic cage (ribs and sternum). Axial skeleton supports and protects organs of head, neck and trunk.

Appendicular skeleton

Pectoral girdle (clavicles and scapulae), upper limbs (arms), pelvic girdle (coxal bones, sacrum, coccyx) and lower limbs (legs). Appendicular skeleton- bones of limbs and bones that anchor them to the axial skeleton.

Articulation- where joints are formed

22 bones in skull, 6 in middle ears, 1 hyoid bone, 26 in vertebral column, 25 in thoracic cage, 4 in pectoral girdle, 60 in upper limbs, 60 in lower limbs and 2 in pelvic girdle (206 bones in all skeletons).

The skull

8 sutured bones in cranium, Facial bones: 13 sutured bones, 1 mandible, Cranium, encases brain, attachments for muscles and sinuses.

Vertebral column

7 cervical vertebrae, 12 thoracic, 5 lumbar, 1 sacrum (5 fused), 1 coccyx (4 fused) Vertebrae vary in size and morphology.

Thoracic cage

Ribs, thoracic vertebrae, sternum, costal cartilages. True ribs are directly attached to the sternum (first seven pairs). Three false ribs are joined to the 7th rib and two pairs of floating ribs. Clavicles and scapulae help brace shoulders.

Attachment sites for muscles

Bones of upper limb: Humerus (upper arm), Radius, ulna Carpals, metacarpals, phalanges.

Bones of lower limb: Femur, patella, tibia, fibula, tarsal, metatarsals and phalanges Joints. Immovable (synarthoses) bones sutured together by connective tissue as skull. Slightly movable (amphiarthoses): connected by fibrocartilage or hyaline cartilage as vertebrae, rib/sternum joint, pubic symphysis. Freely movable (diarthrosis): separated ligaments hold bones together tendons- muscle to bone lined by synovial membrane.

Types of freely movable joints

Saddle: Carpal and metacarpal bones of thumb.

Ball and socket: Shoulder and hip joints.

Pivot- rotation only: Proximal end of radius and ulna.

Types of movement and examples (with muscles)

Hinge- up and own movement in one plane: Knee and elbow.

Gliding- sliding and twisting: Wrist and ankle.

Condyloid- movement in different planes but not rotations: Between metacarpals and phalanges. Flexion (move lower leg toward upper), extension (straightening the leg abduction (moving leg away from body), adduction (moving leg toward the body), rotation (around its axis), supination (rotation of arm to palm-up position), pronation (palm down), circumduction (swinging arms in circles), inversion (turning foot so sole is inward), eversion (sole is out), elevation and depression (raising body part up or down).

Aging and bones

Both bone and cartilage tend to deteriorate: Cartilage chondrocytes die and cartilage becomes calcified. Osteoporosis; bone is broken down faster than it can be built. Bones get weak and brittle tend to fracture easily.

Risk factors for osteoporosis

Inadequate calcium, little weight-bearing exercise, drinking alcohol, smoking, being female: decreased estrogen secretion, after menopause.

Skeleton and other systems

a. Skin makes vitamin D which enhances calcium absorption.
b. Skeleton stores calcium for muscle contraction, nervous stimulation, blood clot formation.
c. Red marrow- site of blood cell formation.
d. Calcium levels regulated by parathyroid hormone and calcitonin.
e. Kidneys (can help provide vitamin D).
f. Digestive system (can release calcium into blood).
g. Growth hormone regulates skeletal growth by stimulates cell division in epiphyseal disks in long bones. Growth stops when epiphyseal disks are converted to bone. When excess growth hormone is produced in childhood leads to gigantism and in adulthood leads to acromegaly.

When muscle contracts, it shortens and causes movement. Skeletal muscles attached to bones by tendons, Insertion- attachment to more movable bone, Origin- less movable. Flexors and extensors act on the same joint to produce opposite actions.

Functions of bone (skeleton)

Support and protection, Blood cell formation, Mineral storage (calcium especially), Site for muscle attachment leads body movement.

Bones classified by shape: Long, short, flat, irregular, round. Bone enclosed in periosteum which contains blood vessels, continuous with tendons and ligaments, and epiphysis on both ends. Spongy bone contains red marrow. Compact bone medullary cavity- contains yellow marrow (fat), lined with endosteum (squamous epithelium). Compact bone osteocytes within lacunae arranged in concentric circles called lamellae this surround a central canal; complex is called haversian system canaliculi connect osteocytes to central canal and to each other.

Prenatal development: Skeleton is mostly cartilaginous cartilage cells and then osteoblasts start to deposit minerals. Cartilaginous disk (epiphyseal disk) remains in epiphysis Cells eventually stop dividing. Adults continually break down and build up bone. Osteoclasts remove damaged cells and release calcium into blood, osteoblasts remove calcium from blood and build new matrix. They become trapped forms Osteoclasts.

Pediatric Fracture Patterns

The mechanisms of fracture change as children age. Younger children are more likely to sustain a fracture while playing and falling on an outstretched arm. Older children tend to injure themselves while playing sports, riding bicycles, and in motor vehicle accidents. Also, because a child's ligaments are stronger than those of an adult, forces which would tend to cause a sprain in an older individual will be transmitted to the bone and cause a fracture in a child. Caution should therefore be exercised when assessing a young child diagnosed with a sprain.

Plastic Deformation

a. A force produces microscopic failure on the tensile/convex side of bone which does not propagate to the concave side. The bone is angulated beyond its elastic limit, but the energy is insufficient to produce a fracture.
b. No fracture line is visible radiographically.
c. Unique to children.
d. Most commonly seen in the ulna, occasionally in the fibula.
e. Bend in the ulna of < 20° in a 4 year old child should correct with growth.

Buckle fracture

a. Compression failure of bone that usually occurs at the junction of the metaphysic and the diaphysis.
b. Commonly seen in distal radius.
c. Inherently stable.
d. Heal in 3-4 weeks with simple immobilization.

Greenstick fracture

a. Bone is bent and the tensile/convex side of the bone fails.
b. Fracture line does not propagate to the concave side of the bone, therefore showing evidence of plastic deformation.
c. If the bone undergoes plastic deformation, it is necessary to break the bone on the concave side to restore normal alignment, as the plastic deformation recoils the bone back to the deformed position.

Complete fracture

Fracture completely propagates through the bone. Classified as spiral, transverse, or oblique, depending on the direction of the fracture line.

a. Spiral fractures
   i. Created by a rotational force.
   ii. Low-velocity injuries.
   iii. An intact periosteal hinge enables the orthopedic surgeon to reduce the fracture by reversing the rotational injury.
b. Oblique fractures
   i. Occur diagonally across the diaphyseal bone at 30° to the axis of the bone.

ii. Unstable, therefore alignment is necessary.
iii. Fracture reduction is attempted by immobilizing the extremity while applying traction.

c. Transverse fractures
i. Created by a 3-point bending force.
ii. Easily reduced by using the intact periosteum from the concave side of the fracture force.

Physeal fractures

a. Fractures to the growth plate can be caused by
i. Crushing,
ii. Vascular compromise of the physis or
iii. Bone growth bridging from the metaphysis to the bony portion of the epiphysis.
b. Damage to growth plate may result in progressive angular deformity, limb-length discrepancy or joint incongruity.
c. The distal radial physis is the most frequently injured physis.
d. Most physeal injuries heal within 3 weeks. This rapid healing provides a limited window for reduction of deformity.

Differences between pediatric and adult fracture healing

Fracture Remodeling

a. Process that occurs over time as a child's bone reshapes itself to an anatomic position.
b. The amount of remaining bone growth provides the basis for remodeling. Thus, the younger the child, the greater remodeling potential, and the less important reduction accuracy are.
c. Occurs over several months following injury.

Factors affecting amount of remodeling

Age: Younger children have greater remodeling potential.
a. Location: fractures adjacent to a physis undergo greatest amount of remodeling.
b. Degree of deformity.
c. Plane of deformity with respect to adjacent joint: remodeling occurs more readily in the plane of a joint than with deformity not in the plane of the joint.

Overgrowth

a. Caused by physeal stimulation from the hyperemia associated with fracture healing.
b. Prominent in long bones (ex. Femur).
c. Growth acceleration is usually present for 6 months to 1 year following injury.
d. Does not present a continued progressive overgrowth unless complicated by a rare arteriovenous malformation.
e. > 10 years of age, overgrowth is less of a problem and anatomic alignment is recommended.

Progressive Deformity

a. Injuries to the physis can be complicated by progressive deformities with growth.
b. The most common cause is complete or partial closure of growth plates.
c. Deformities can include angular deformity, shortening of bone, or both.
d. The magnitude of deformity depends on the physis involved and the amount of growth remaining.

Rapid Healing

a. Pediatric fractures heal more quickly than adult fractures due to children's growth potential and a thicker, more active periosteum.
b. The periosteum contributes the largest part of new bone formation around a fracture.
c. As children reach their growth potential, in adolescence and early adulthood, the rate of healing slows to that of an adult.
d. There is one downside to rapid healing, however refractures.

Common fractures in pediatric

Cause	Pathology	Presentation	Investigation
Birth Injury	Potential complication of forceps delivery - Complication of cephalo-pelvic disproportion and shoulder dystocias	Clavicular fractures - Humeral fractures Femoral fractures	X Ray - Cephalohematoma at the site of injury and calcification around fracture will help indicate if fracture occurred during birth or after
Rickets	Lack of vitamin D and metabolite disturbances →	No specific fracture pattern	Measure 25-hydroxyvitamin D

	defective mineralization of bone matrix - Similar to osteomalacia in adulthood	- May present with bowed limbs, "rickety rosary", craniotabes	levels
Osteomyelitis	Acute or chronic infection of bone → lesions of the metaphyses	- Long bones	Upon suspicion, immediate referral to orthopedic surgeon for body fluid tests, aspiration of bone and imaging
Child abuse	30-50% of children seen by orthopedic surgeons are the victims of non-accidental injury	-supra-condylar fracture(humerus) -Femur Distal femoral metaphyseal corner -Posterior rib -Scapular spinous process -Proximal humeral	Diagnosis requires multiple factors: history, clinical presentation, behavioural and physical observation

Table 1: Common Fractures in Pediatric.

Types of bone breaks

a. Simple- skin is not pierced.
b. Compound- skin is pierced.
c. Complete- bone is broken in half.
d. Partial- broken lengthwise but not into two parts.
e. Greenstick- incomplete break on outer arc.
f. Comminuted- broken into several pieces.
g. Spiral- twisted.

Fracture repair

Hematoma- blood clot in space between edges of break

↓

Fibrocartilage callus- begins tissue repair

↓

Bony callus- osteoblasts produce trabeculae (Structural support) of Spongy bone and replace fibrocartilage

↓

Remodeling- osteoblasts build new compact bone, osteoclasts build new medullary cavity

Figure 1: Fracture Repair.

Fracture pathology and diagnosis

a. Single traumatic incident: direct (tapping or crushing) or indirect (bending or twisting).
b. Repetitive stress (crack occur in bone due to repetitive stress) as in metatarsal fatigue fracture.
c. Abnormal weakening of the bone.

Types of load applied on bone

Tension force, compression force, bending force, shearing force, torsion force, combined loading.

Types of fracture according to load applied

Complete fracture

a. Avulsion fracture: Due to tension force.
b. Buckle, wedge, crush fracture: Due to compression force.
c. Butterfly, comminuted, compound, intra fracture.
d. Articular fracture: Due to non specific mechanism.
e. Depressed fracture: Due to shering force.
f. Spiral fracture: Torsion force.
g. Stellate fracture: Due to shering force.
h. Transverse fracture.
i. Oblique fracture.

Incomplete fracture

a. Greenstick fracture: Due to compression or bending force.
b. Compression fracture: In canecellous bone.
c. Stress fracture: Repetitive minor trauma.

Fracture healing process

a. Hematoma
b. Inflammation
c. Callus
d. Consolidation
e. Remodeling

Causes of non union

a. Distraction and separation of the fragment.
b. Interposition of soft tissue between fragments.
c. Excessive movement of fracture line.
d. Poor local blood supply.

Causes of malunion

Fragment joins in wrong position

a. Failure to reduce fracture adequately.
b. Failure to hold reduction while healing process occurs.

Causes of delayed union

a. Inadequate blood supply.
b. Infection.
c. Insufficient splintage.
d. Excessive traction.

Factors affecting on bone healing

a. Type of bone involved (canecellous heal faster cortical).
b. Type of fracture (transverse fracture takes longer time than spiral fracture).
c. State of blood supply
d. Patient's general constitution (healthy bone heals faster).
e. Patient age (healing is twice as fast in children as in adult).

Fracture indicated to surgery

a. When closed reduction failed.
b. When large articulare fragment needs accurate positioning.
c. When fragment are held apart.

Indications of internal fixation

a. Fracture that cannot be reduced except by operation.
b. Unstable fracture.
c. Poor united fracture.
d. Pathological fracture.
e. Multiple fractures.
f. Patient with difficult nursing (paraplegic).

Indication of external fixation

a. Sever soft tissue damage.
b. Nerve or vessel damage.
c. Unstable fracture.
d. Pelvis fracture.
e. Infected fracture.

Physical problems post – plaster cast and post-operative fixation

a. Limitation in joint range of motion due to accumulation of adhesions inside joint.
b. Atrophy of stretched muscles during cast or fixation due to destruction of actin and myosin inside muscles.
c. Weakness of muscles around fixated joint.
d. Tightness of muscles due to accumulation of adhesions inside muscles.
e. Mal – union or delayed union or non union of fracture (continuous pain and limitation).
f. Myositis ossificans due to overstretch on muscles lead to hematoma inside muscles lead to calcification inside muscle.
g. Volkmann's ischemic contracture (tight cast lead to ischemia).
h. Sudeck's atrophy.
i. Pain.

Treatment of post – plaster cast and post-operative fixation

a. Hot packs on tight region and callus formation (15 minutes).
b. Ultrasonic applied on and around callus away from joint and (plates, screws, intermedullary nails).
c. Graduated exercises started with static contraction then active assistance till resisted ex.
d. Auto-passive stretching ex. On tight muscles to avoid myositis ossificans.
e. Static stretching by positioning as quadruped and hand weight bearing.
f. Splinting (static as night splint to maintain range of motion, maintain functional position and dynamic for assist normal movement).
g. Home routine program (hot packs+ resisted ex).
h. Post- operative children (static and isometric ex) plus other methods with precautions.
i. Crutches and walkers can be used for gradual weight bearing ex.
j. Paraffin bath can be used for relive pain and decrease of tenosynovitis.

Cartilage Disorder

Structure and function of cartilage:

Cartilage is a connective tissue consisting of a dense matrix of collagen fibres and elastic fibres.

Types of cartilage:

a. Hyaline cartilage
b. Fibro cartilage and
c. Elastic cartilage. Elastic cartilage exists in the epiglottis, the eustachian tube and external ear.

The functions of elastic cartilage tissue

Elastic cartilages provides support to surrounding structures and helps the define and maintain the shape of the area in which it is present, e.g. the external ear.

The functions of fibrocartilage tissue

Fibrocartilage is permanently present in following locations in the body

a. The intervertebral disks of the spine,
b. As a covering of the mandibular condyle in the temporomandibular joint,
c. In the meniscus of the knee,
d. Callus,
e. Symphysis pubis,
f. Part of tendon.

Fibrocartilage tissue provides support and rigidity to attached/surrounding structures and is the strongest of the three types of cartilage.

Hyaline cartilage, is the most abundant of three types it is found in

a. Bronchi; Bronchial Tubes; Costal Cartilages; Larynx Nose; Trachea.
b. Covering the surface of bones at ends of the long bones, and also the anterior ends of the ribs.
c. Embryonic skeleton (i.e. in the fetus).

The Functions of hyaline cartilage tissue

Hyaline cartilage tissue provides smooth surfaces, enabling tissues to move/slide easily over each other, e.g. facilitating smooth movements at joints. It is also provides flexibility and support.

Articular Cartilage and Meniscus Composition

There are two major phases of articular cartilage and meniscus

a. A fluid phase containing water and electrolytes, and
b. A solid phase containing collagen (type I in meniscus and type II in articular cartilage), protoeglycans, glycoproteins and chondrocytes. Chondrocytes are the cells that produce cartilage matrix.

Physical problems in meniscus tear

Twisting joint injury, Physical problems

a. Pain
b. Swelling
c. Weakness
d. Atrophy
e. redness and hotness
f. loss of movement
g. poor balance
h. difficult ADL activities
i. poor proprioception
j. instability of joint and subluxation
k. hemiarthrosis in severe injury
l. locking of the knee joint due to present of loose body
m. knee instability

Treatment of mild cases by physiotherapy

Acute stage

a. RICE (rest-ice application-compression-elevation).

b. Splinting to put the joint in functional position allow muscle strain for repair.

Recovery stage

a. Electrotherapy modalities.

b. Superficial heat (dry or moist heat).

c. Electric stimulation for quadriceps muscles to avoid atrophy and for treatment of atrophy via stimulation of protein synthesis inside muscle.

d. LASER scanned and probe laser can be used to stimulate the proliferation of ATP leading to improvement of healing of soft tissues around knee.

e. Myofascial release on site of lesion to avoid fibrosis of soft tissue around.

f. GAE for stretched muscle to strength muscles around knee (start with static ex. and progressed to isometric ex.).

g. Balance training to improve postural reaction and stability.

h. Proprioceptive training via joint compression, weight bearing and vibration.

i. Mobilization of the joint to increase ROM without pain.

j. Splinting to put the joint in function position to prevent hyperextension knee allowing for sprained ligament healing (knee immobilizer and ankle foot orthosis to prevent hyperextension knee plus knee cage to assist in walking.

k. Anti-inflamatory drugs (systemic and localized).

l. In moderate and sever injury need surgical repair.

Post operative physiotherapy treatment

The same treatment as above except

a. If swelling is persisted we use ice bag instead of superficial heat till the swelling subsides.

b. Mild decent gentle stretch for tight muscles.

c. Ultrasonic on fleshy part of tight muscles.

d. Gait training closed then open.

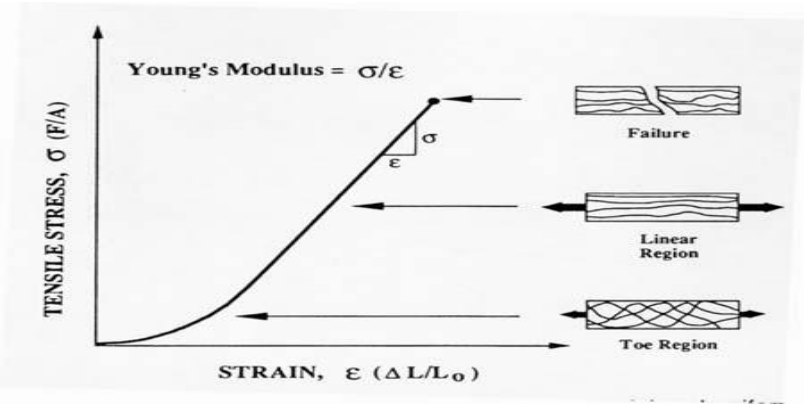

Figure 2: Stress-strain curve.

Chapter 4

Posture Deviation

Types of Posture Deviations

Normal Alignment of the trunk is (Anterior pelvic tilt, Lumbar extension and Thoracic extension). When feet under knees, anterior pelvic tilt and trunk extension are enhanced upper trunk rotation with lower trunk stability while reaching for clothing during dressing. Normal control requires the ability to dissociate (separate) different parts of the body from each other. High mobility, low articulation the reason why upper limb more effected than lower limb.

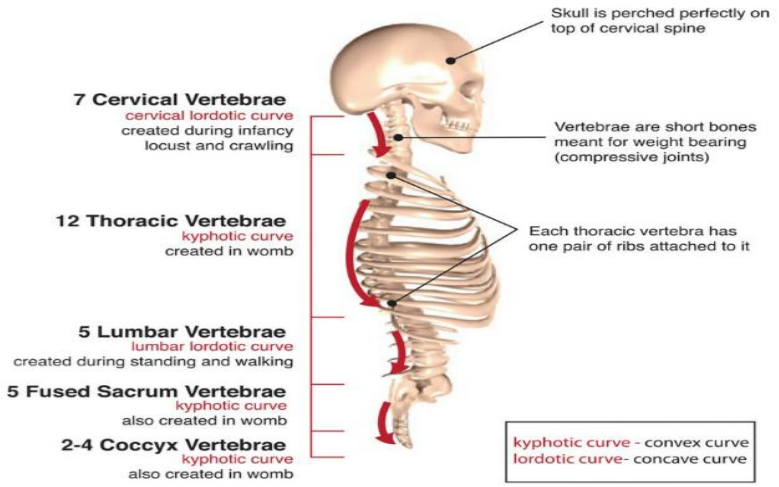

Figure 1: Kyphotic and lordotic curves in vertebral column.

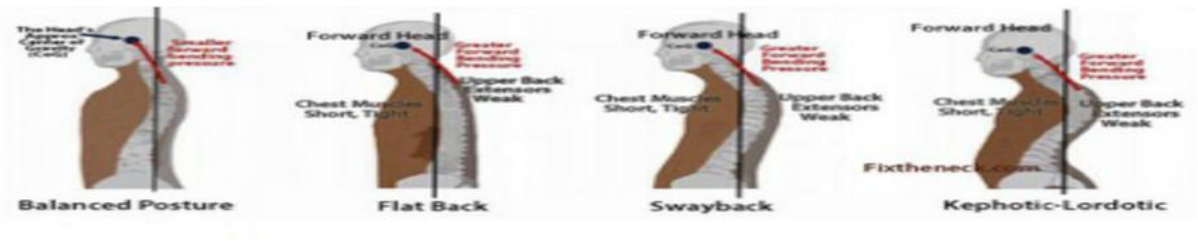

Figure 2: Types of Posture Deviations.

The force of gravity interacts with the force generated from muscle contraction to assist in the musculoskeletal development of the infant's thorax. Once the trunk muscles are strong enough, they can work with gravity (gravity assisted) or against it (gravity resisted) throughout all developmental activities. The usual interaction between the musculoskeletal system of the trunk and gravity result in normal development of the thorax. However, impaired muscles motor control which produce a subnormal motor response despite a maximal contraction of the muscle will not have the same ability to balance gravity influence thus altering the important relationship between these two forces.

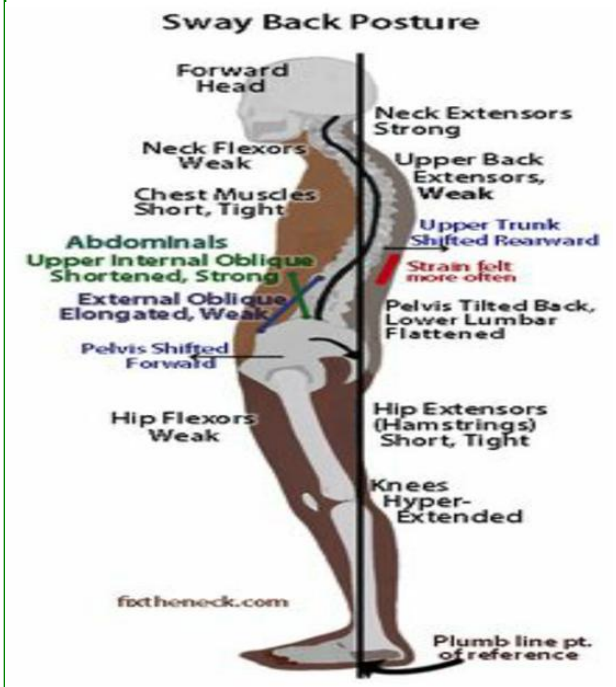

Figure 3: Sway Back posture.

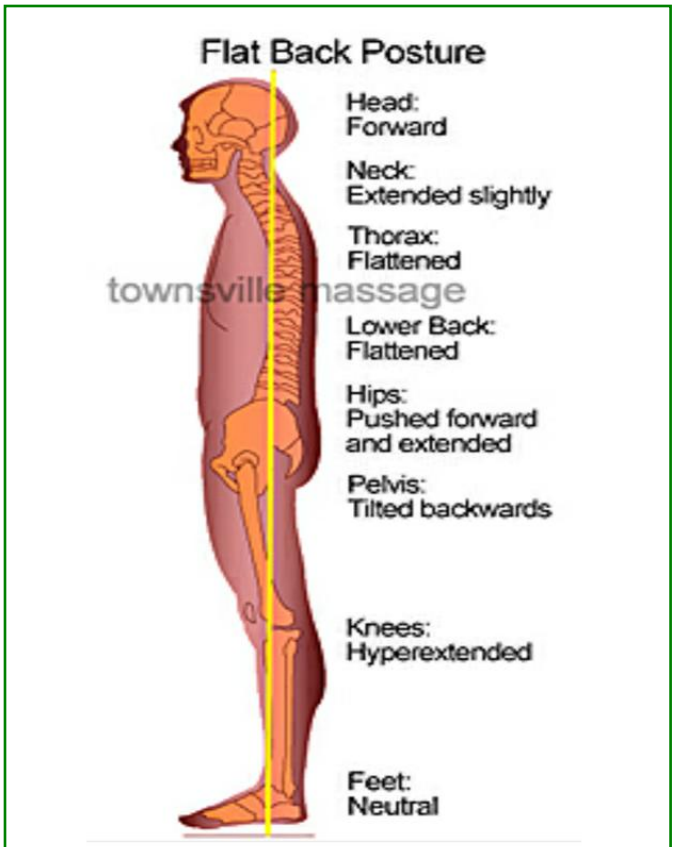

Figure 4: Flat Back posture.

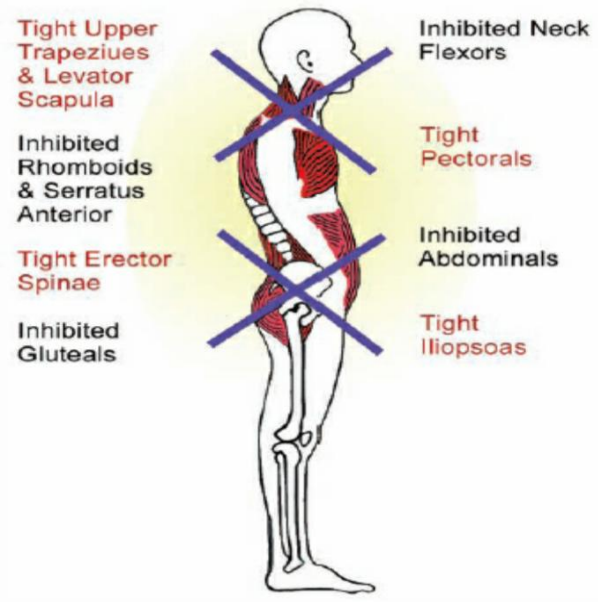

Figure 5: Kyphotic-lordotic posture.

Thus these impaired muscles motor control may be detrimental to the development of the chest, limiting indirectly the child pulmonary functions. Intercostals muscles have not yet developed an optimal length-tension relationship to produce a motor force adequate to significantly move the chest wall or head. Thus the newborn is without mechanical resources to expand their chest effectively in all three planes of ventilation particularly the anterior plane of upper chest.

Muscles that Control Pelvic Tilt

The deviation of pelvic alignment in the standing position is a common problem in children with CP. Such children retain an anterior pelvic tilt due to the contracture of the iliopsoas muscle as well as weakness in the trunk flexors and hip extensors. Problems associated with anterior pelvic tilt include femoral antetorsion and medial shift of the patella to the sagittal plane bisection of the knee joint. Therefore, a major goal of movement training is reciprocal control of the pelvis by improving interplay among the abdominal obliques, rectus abdominalis, quadrates lumborum and lumbar extensor muscles. Excessive anterior pelvic tilting which is common in spastic diplegic and quadriplegic cerebral palsy will produce a hamstring shift in conjunction to an apparent knee flexion tightness .However in children with crouch gait hamstring length is frequently normal or even long. There is 1 degree of excessive pelvic lordosis in relation to 2 degree increase of knee flexion. A hamstring shift of 20 degree is usually indicative of excessive anterior pelvic tilt

either from tight hip flexors musculature or weak abdominal muscles or weak hip extensors.

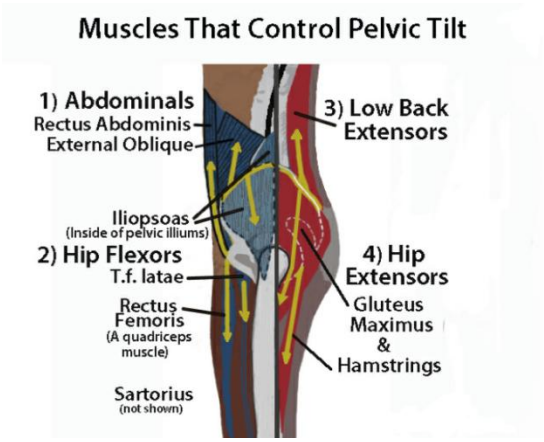

Figure 6: Muscles that Control Pelvic Tilt.

Children with cerebral palsy commonly have excessive femoral anteversion especially with Hypotonia and/or laxity of ligament. Common compensations of excessive femoral anteversion to cover femoral head include internal rotation of the femur and anterior pelvic tilt. This in turn promotes posture of internal limb rotation and excessive lumbar lordosis during gait that is common in cerebral palsy children. Hip and trunk muscles form force couples about the pelvis the gluteus maximus and hamstring working together with abdominal muscles to produce posterior pelvic tilting. The iliopsoas and rectus femoris working together with erector spinae to produce anterior pelvic tilting.

In particular, anterior pelvic tilt is performed by active force-coupling between the hip flexors and low-back extensors. In contrast, children with CP exhibit passive anterior pelvic tilt owing to contracture of the iliopsoas muscle and weakness of the trunk flexors and hip extensors. Therefore, pelvic tilt exercises for children with CP should enhance the contraction of these muscles, especially the force-coupling generation between the trunk flexors (i.e., RA and obliques externus abdominis) and hip extensors (i.e., GM and hamstrings) increased activation of the hip extensors, trunk flexors and diminished activation of the hip flexors ,erector spinae are the first choice in treatment.

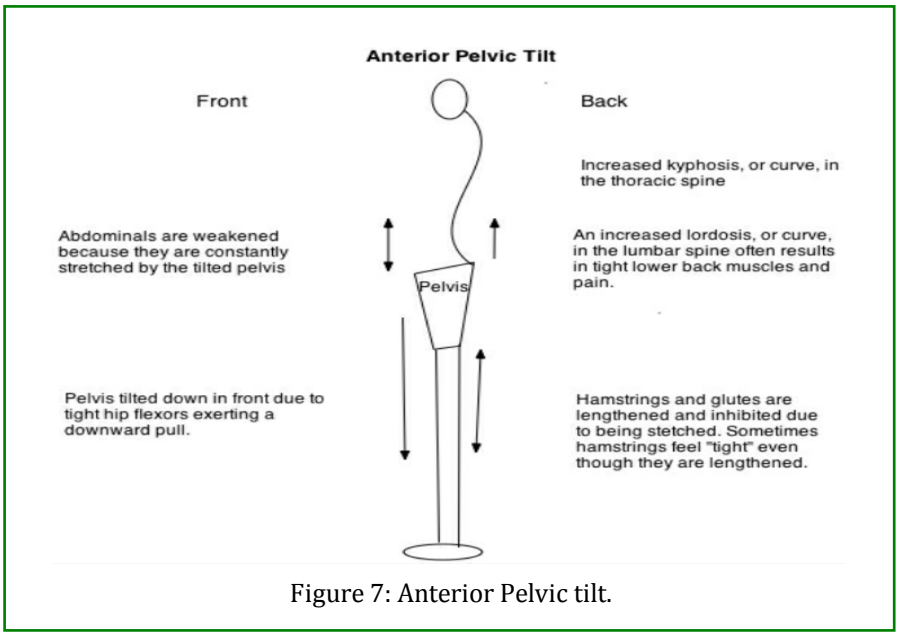

Figure 7: Anterior Pelvic tilt.

Anterior pelvic tilt→ lumbar extension→ thoracic extension→ scapular adduction→ humerus external rotation lead to supination of forearm lead to increased hand functions and grip in addition to in children who have very low tone and an extremely anteriorly tilted pelvis, e.g. in this position it is very difficult to expire forcefully as the abdominal muscles are stretched and the abdominal contents are not providing adequate support to the diaphragm. Abdominal muscles are unable to assist effectively with expiration as they are stretched in this position, making contraction difficult. Due to the mechanics of this position, full expiration may be difficult which may make activities such as effective coughing difficult. Lower ribs have limited ability to expand, so the ribs must expand laterally (bucket handle) and increase movement at the top of the rib cage to compensate.

Flaring or winging anteriorly of the bottom of the ribs due to lack of 'pull' from abdominal muscles may be noted during the mat evaluation.

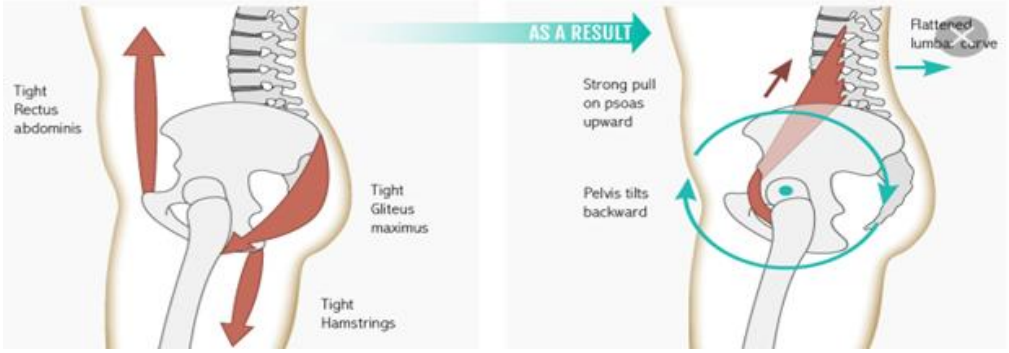

Figure 8: Posterior pelvic tilt.

Posterior pelvic tilt→ lumbar flexion→ thoracic flexion→ scapular abduction→ humerus internal rotation lead to forearm pronation lead to loss of supination lead to thumb and fingers faced downward lead to impaired hand functions and hand grip in addition to the abdominal contents will push up into the ribcage causing the lungs to have limited expansion capability. If you imitate the position in the picture you will be able to feel how it affects your ability to take a deep breath, increased energy requirements can lead to respiratory muscle weakness if energy requirements are not met adequately.

Ribs are limited with ability to expand forward (pump handle) so must move laterally (bucket handle) to compensate. Abdominal organs limit the diaphragm from moving downwards during inspiration. Due to the mechanics of this position full inspiration may be difficult which may decrease lung volumes and increase risk of lung collapse and difficulty completing an effective cough. Where breath/swallow coordination is impaired, possible increased risk of aspiration. If neck extension is present, it may open the airway, which can impair airway protection and lead to aspiration in neuromuscular diseases, a chronically slumped posture, the result of collapsing forces, can cause a multitude of postural deficiencies including:

a. A thoraco-lumbar kyphol-scoliosis which compresses the anterior rib cage, often causing a mid trunk fold at the xiphoid process, thus restricting breathing mechanics,
b. A compensatory forward head position on top of the thoracic kyphosis which compromises swallowing mechanics thereby increasing the risk of aspiration and mechanically compromising the recruitment of accessory muscles for increased lung volumes,
c. A compensatory upper quadrant position including protracted scapula and humeral internal rotation, impairing shoulder mechanics as well as chest wall muscle recruitment for breathing, and
d. A posterior pelvic tilt with excessive hip external rotation thus further compressing forces at the mid trunk and pelvic floor further impairing the diaphragm's mechanical advantage.

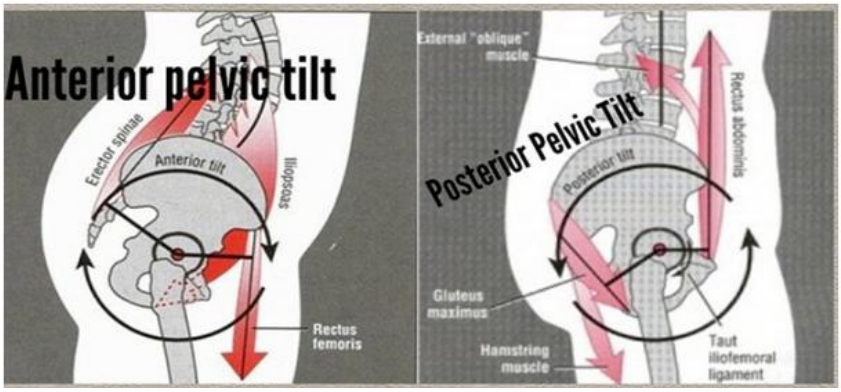

Figure 9: Anterior and posterior pelvic tilt.

It has been reported that children with spastic cerebral palsy has decreased chest mobility. This inefficient chest mobility is reported due to various secondary factors like over activity of trunk muscles like intercostals, diaphragm and rectus abdominis, barrel shaped chest, inefficient stabilization by abdominals. Along with this there occurs adaptive soft tissue tightness or shortening at the chest wall in response to the altered position of the chest (e.g. barrel shaped chest). Added to the inefficient expansion mechanism of the chest, this tightness of the muscles, fascia and the skin overlying the chest reduces the overall excursion of the chest thus all the more reduced expansion of the chest.

Exercises focusing upon postural muscle development, i.e. a multitude of muscles like The important role of abdominal muscles in establishing effective ventilation can be explained biomechanically as at the end of expiration diaphragm is forced upward, thus stretching it to an optimal length tension relationship. As a consequence muscle is more prepared to initiate a more forceful contraction at the next inspiratory cycle. Also upright positioning along with growing abdominal muscles pulls and rotates the ribs especially lower ribs downwards, widening the intercostals spacing and therefore achieving an ideal position for intercostals function in inhalation and exhalation maneuver. Specifically, the intercostals can now function to

a. Stabilize the chest wall during the negative thoracic pressure created during inhalation,
b. Increase the lateral and anterior dimensions of the chest wall during inhalation, and
c. Compress the chest wall during forceful exhalation. Thus, an improvement in abdominal muscle activation as a result of trunk stabilization exercises improves the biomechanical advantage for better activation and functioning of the respiratory muscles leading to an improvement in chest expansion.

Pleural dome connect with c6, c7, t1 and 1st rib by (transverse pleural, pleura-vertebral and costo-pleural ligaments). The lungs are attached to their surrounding structures by a suction system and the suspensory, lung and interpleural ligaments. The suction system is created by the negative pressure within the thoracic cavity, which forces the lung to be always flattened against the lining. The suction is localized at the periphery of the lungs and makes possible thoracic expansion. The Suspensory Ligament of the Pleural Dome The suspensory ligament attaches the pleural dome to the skeleton.

It consists of muscular fibers of the scalenus minimus (sometimes mixed with fibers of the anterior and medial scalene muscles), plus the fibrous fascicule. This ligament is not directly inserted into the parietal pleura, but rather into the intrathoracic fascia. This fascia forms a "connective tissue dome" at the level of the top of the lungs, where it and the elements of the ligament form a partition. This partition, which is anatomically independent of the parietal pleura, is firmly attached to the nearby skeleton and is called the cervicothoracic fibrous septum. In the physiology of movement, this septum is the link between the superior lobe of the lung and the cervicothoracic junction. The lung ligament is usually said to be formed from reflected folds of pleura under the pulmonary hilum. In fact, the fold does not stop at the pulmonary hilum, but continues as far as the diaphragm. Overall, the line of reflexion has the form of a tennis racquet, with the web-like part surrounding the pulmonary hilum in the front, behind and above, while the handle is represented by the lung ligament, which is connected to the thorax like a mesentery. Both strips of this "mesentery" are joined together.

The lung ligament is linked to the esophagus by means of surrounding fascia. Another link between the two lungs is the interpleural ligament, which is formed by the joining of the two interazygos cul-desacs lung tension lead to pleural tension lead to ligaments tension lead to pull on the cervical plexus lead to limitation of cervical and chest movement and decrease their stability. In C.P there is repeated lung infection and decrease of its elasticity which lead to limitation of chest and cervical movement so we can use osteopathic visceral manipulation to produce lung, pleural and ribs released followed by neuro-developmental approach.

During forced inhalation, this is only an exaggeration of normal inhalation. Each hemithorax increases in volume, followed by the pleura and lung. This increase is made possible by the mobilization of the supple structures of the hemithorax. The diaphragm (and diaphragmatic pleura) descends, the rib cage undergoes an anterior and lateral expansion and the costal pleura follow the action of the rib cage. Therefore, the expansion of the hemithorax, and thus the lung, takes place because of the lowering of the diaphragm and costal expansion. The pleuromediastinal wall is fixed. Because the superior diaphragm of the thorax is essentially made up of stringy structures, the pleural dome is fixed. These fixed points are necessary so that a structure may be stretched. This means that the lungs are subject to forces that pull in opposite directions, albeit on the same axis: traction (force) in one direction must always be linked to a countertraction (tension). The lung is submitted to a force F on the costal pleura, but also to a tension T on the mediastinal pleura, which prevents the entire lung from moving laterally. This tension, which balances the lateral costal expansion, is created via the lung ligament. The tension which balances the downward expansion caused

by the diaphragm muscle is created by the suspensory ligament. The movement of the thorax is the sum of the movements of each costovertebral unit (a thoracic vertebra and its pair of ribs).

The mobility of the lung during inhalation is an external rotation of the parenchyma on a vertical axis for the upper lobes and on an obliquely inferolaterally directed axis for the lower lobes. Pulmonary expansion is possible owing to the tension of the lung ligament, the left bronchus (which fixes the visceral pleura to the mediastinum) and the suspensory ligament (which fixes this dome at the top). During inhalation, the ligaments of the lung and bronchi exert an isometric tension on the lungs so that they do not move laterally as a block. One force (F1) resulting from the expansion of the right hemithorax and lung, is counteracted by an isometric tension (T1), created by the pulmonary ligament and the primary bronchus at the visceral pleura of the right lung. Similar forces F2 and T2 are applied to the left lung. Forces T1 and T2, being of equal force and in opposite directions, cancel each other out. The interpleural ligament, which is the union of the cul-desacs of both the aorta and esophagus, connects the right and left mediastinal parietal pleura. Forces F1 and F2 balance each other across the mediastinum and should remain in equilibrium.

Role of Sub-Occipital Muscles in Postural Control

The sub occipital muscles are comprised of eight muscles total (one set of four on either side).The upper cervical spine has been shown to be the most concentrated area of mechano-receptors (joint position receptors) in the body. The sub occipital muscles have been also shown to have a very dense number of muscle spindle cells and GTOs (Golgi tendon organs) monitoring joint position as it relates to the muscle. The fact that the SMI technique could increase the flexibility of the LL muscles may be because the superficial back line was relaxed through relaxation of the sub occipital muscles. The sub occipital muscles are the “proprioceptor monitors” that contribute significantly to regulation of head posture, and they have the most muscle spindles in the human body. Among them, in particular, the rectus capitis posterior minor muscle, which has 36 muscle spindles per gram, is known to contribute greatly to regulation of posture and the degree of tension.

A crucial area of the myofascial meridian called the Superficial Back Line (SBL). The SBL is a ribbon of facial and muscular continuity that begins with the plantar fascia and short toe flexors beneath the arches of the foot, wrapping around the heel to the soleus and gastrocnemii of the lower leg. The gastrocs interlock with the hamstrings, which are in turn continuous with the sacrotuberous ligament, which feeds into the sacral fascia. The sacral fascia is the fascial anchor for the back muscles that traverse the spine, and it is the very top muscles of this group to which we will turn our attention in this column. The SBL, however, continues on beyond the occipital ridge, up along the top of the skull with the epicranial fascia, including both the occipitalis and frontalis muscles, until it attaches at its other end, the brow ridge just above the eye socket.

While the sacral fascia may be the structural center of the SBL halfway between the foot's sole and eyebrow, the functional center is really just beneath the skull in the suboccipital muscles. Generally, the muscles that span the spine can be divided into the erector spinae, which are more superficial and cover many segments of spinal movement, and the transversospinalis muscles, which are shorter and deeper and are generally used more for orientation and stability rather than brute strength. Our focus is a set of four muscles at the very top of the transversospinalis tiny, hard to reach and differentiate, but mighty in their effect on posture and movement. The deepest layers of muscles (the suboccipital "star") are crucial to opening up and obtaining the most integrated function in the entire SBL. The high number of stretch receptors in these tissues, and their connection from the eye movements ensure their central role in postural control. Disturbances in the brain, spinal cord, nerves, muscles and skeleton may hinder overall motor function and gait. In children with spastic hemiplegia due to cerebral palsy (CP), a non uniform distribution of lower limb weight bearing, balance and proprioception deficits are observed in gait. This results in asymmetry, which is the most prominent characteristic of the gait in hemiplegic children. The body weight is supported mostly by the non-involved lower extremity. The weight transfer to the involved side is brief and incomplete.

Rectus capitis posterior minor has been noted to have a fascial bridge into our dura mater (the outer layer of the meninges which surrounds our spinal cord and brain). A potential role that rectus capitis posterior minor is to try and regulate dural folding, or movement of dura towards the spinal cord which occurs during head extension. It has been inferred that individuals may experience muscle tension, headaches and pain when the rectus capitis posterior minor muscle acts inappropriately on dura. The fascial system is important not only because it can passively distribute tension in the body muscles when mechanically stimulated, but also because it contains mechanoreceptors and possesses an autonomous contractile ability that influences the tension of the fasciae. The stimulation of intrafascial mechanoreceptors

(mostly interstitial and Ruffini endings) causes the vegetative nervous system and the CNS to change the tension in intrafascial myofibroblasts and regulate fascial pre-tension. These tensions are transmitted along the MFC (myofascial chains), thereby influencing the posture of the entire body. Connective tissue bridges were noted at the atlanto-occipital joint between the rectus capitis posterior minor muscle and spinal dural matter .The perpendicular arrangement of these fibers appear to restrict dura matter movement toward spinal cord. The ligamentum nuchea was found to be continous with posterior cervical spinal dura and lateral portion of the occipital bone. The dural-muscular and the dural ligamentous connection in upper cervical spine and occipital area provide anatomic and physiologic answers to cause tension regulation. Nuchal-dural-adhesion theory stated that increased tension within sub-occipital muscles may produce abnormal traction on the cranial dura stimulating dural nociceptive fibers leading to tension changes.

Rectus capitis posterior minor has been shown to have a high density of muscle spindles, which may indicate its role in movement is not as important as its role in proprioception of both the head and cervical spine. For this reason, atrophy of the rectus capitis posterior minor that may occur following injury or trauma can lead to diminished proprioception and balance. Rectus capitis posterior minor atrophy displayed a decrease in standing.

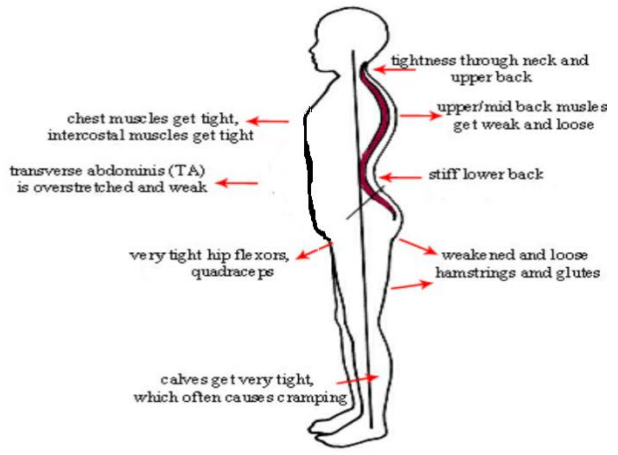

Figure 10: Role of Sub-Occipital Muscles in Postural Control.

The basic element of the correlation between small sub-occipital muscles and human posture is the existence of muscle-fascial chains (MFC). Fasciae are dense, fibrous connective tissues that interpenetrate and surround the human body to protect, nourish and hold organs in place. Three layers of fasciae exist: superficial, deep and visceral.Deep fasciae surrounds muscles, bones, nerves and blood vessels and is densely populated with myofibroblasts and several types of receptors (nociceptors, proprioceptors, mechanoreceptors, chemoreceptors, thermoreceptors). Myofibroblasts are fascial cells that are created as a response to mechanical stress and actively contract in a smooth, muscle-like manner.

MFC (muscle-fascial chains) is a group of muscles that are connected through the fasciae and are longitudinally positioned in the human body. They run in the same direction and overlap in a continuous chain, like tiles on a roof, which efficiently conducts tension. All of the muscles in the chain are mutually dependent and behave as if they were a single muscle. The existence of MFC may explain why disorders of the MM functions such as chewing and swallowing, can be transmitted to distal musculature, because of the connections within the fascial system, change in any part of the body may create a disorder in another. For example, a contracted masseter muscle transmits its tension to homolateral SCM, and such connections may explain the influence of the SCM on mandibular movements. The MFC may also explain why an anterior cruciate ligament injury influences muscular electromyography activity of masseter, anterior temporalis, posterior cervicals, sternocleidomastoid, and upper and lower trapezius. The sub occipitals are also partly “antagonized” (balanced) by the jaw muscles. This is an odd arrangement. Generally speaking you’ve got one muscle or group of muscles that pulls one way, and then muscles on the other side of the joint that pull the other way. But the jaw muscles do not affect the spinal joints, and cannot directly work against/with the sub occipitals to balance the head. Nevertheless, they do: muscle studies have shown that the jaw muscles behave much like they would in a more normal push-pull relationship with the sub occipitals. They function together and dysfunction together. Both of these muscle groups routinely harbour trigger points that cause headaches (among other things), and together they are the source of most tension headaches.

Underlying Mechanism of Sub Occipital Muscles Inhibition and Restoration of Posture Aligment

The SMI technique is a method of inducing relaxation of the fascia by applying soft pressure to the sub occipital area of the patient while child is lying comfortably, and it can be easily applied by a therapist.Increase in calf and hamstring elasticity occur due to the presence of Myodural Bridge connecting rectus capitis posterior minor muscles to the dura mater. Therefore, the SMI

technique to the sub occipital area and compared the effects on the flexibility of the calf, hamstring muscles and functional walking capacity.

There are several hypotheses for the positive result of this treatment:

a. The connection to dura mater: This plays to the continuity of the nervous system and how it links everything together.
b. Postural control: The sub occipitals play a role in postural control and will affect the coordinated movement of muscles down the chain.
c. Myofascial chains: Both the sub occipitals and the hamstring musculature are included in the superficial back line. Addressing any of the structures in the superficial back line may have a positive effect of the entire line itself.

Rectus capitis posterior minor is directly linked to the dura mater in the atlanto-occipital joint region. However, the authors likewise demonstrated that pressure applied intra-operatively on the posterior zone of the dura mater triggers pain in the sub occipital region. Since the myodural bridge has a direct influence on the pain-sensitive dura mater, a possible link between cervical muscles and headaches is postulated. On the basis of these findings, it may be hypothesized that the bridge between rectus capitis minor und dura mater is stretched in a whiplash trauma, which would explain the chronic symptoms of such patient's attachments of the ligamentum nuchae to the dura mater as well as to the posterolateral part of the occipital bone. There is anatomical relation between rectus capitis posterior minor and dura mater.

Dural vascular irritation leads to activation of the neck and jaw muscles; clinically, these points towards a relation to headaches and facial neuralgia. Hyper tonicity of the rectus capitis posterior minor causes permanent tension of the dura mater. This entails irritation of the meningeal vascular systems and, in due course, hyperactivity of the jaw and neck muscles. Thus improving the tone of the rectus capitis posterior minor may normalize dural blood flow and hence the tone of the jaw and neck muscles. Change in short neck muscle tone entails a change of posture. Thus the function of the Short neck muscles is largely proprioceptive and less a locomotor function. An "anterior type", which corresponds to a pattern with anterior orientation with respect to both posture (pelvic anteversion, hyperextension of Iower extremity, lumbar hyperlordosis) and jaw (mandibula anterior). The "posterior type" presents the opposite picture (pelvic retroversion, flexion of Iower extremity, increased kyphosis of dorsal spine and mandibula posterior).The utilization of suboccipital stretching to help inhibit calf and hamstring tightness and improve functional walking capacity. Suboccipital Muscle Inhibition Technique to evaluate the effect that "releasing" the subbocipital muscles would have on muscle elasticity. This technique consisted of the therapist placing an upward pressure on the posterior arch of the atlas (C1) of the supine patient for approximately 2 minutes, until tissue relaxation had been achieved. Following this technique, the subjects in the suboccipital muscle inhibition technique group saw greater improvements in finger-floor distance test, straight leg raise, and popliteal angle test compared to the control group (who received a placebo manual therapy technique).

Clinical significance of small sub-occipital muscles with high spindle density

a. Kinesthetic information from the sub-occipital muscles may be handled in more complex ways, as evidenced by convergence of vestibular, oculomotor, visual and neck proprioceptive inputs at various levels of neuroaxis.
b. Proprioceptive inputs from the cervical musculature are important in headeye co-ordination and postural orientation. The sub-occipital muscles in humans have extremely high spindle content.
c. Altering the afferent input from the upper cervical region can result in disturbances of gait, dizziness, loss of balance, ataxia, etc.
d. Known causes of altering the afferent input from the upper cervical region include whiplash injuries, altered cervical blood flow, and disturbances of cervical sympathetic tone.
e. Cervical proprioceptive afferent inputs, vestibular afferent inputs (labyrinthine), and afferent inputs from the extra ocular muscles converge at the vestibular nuclei and affect the thalamus and the cerebral cortex.
f. The muscle spindles of the cervical per vertebral muscles are the major proprioceptors of the neck, not the joint capsules.
g. Other muscles with high spindle density are found in the hand and foot.
h. Human sub occipital muscles have an extreme high spindle density, far greater than 50-100/gm, and far greater than other human muscles.
i. The highest spindle density is in the inferior oblique muscle which crosses atlanto-axial joint and handles the greatest proprioceptive input.
j. The proprioceptive input from the sub-occipital joints is from sensing joint position and movements of craniovertebral joints.
k. Most importantly, sub-occipital muscles proprioceptive input (along with vestibular organ and oculomotor afferent input) do much more than monosynaptic excitation of alpha-motorneurons. They send afferent input that integrates at numerous regions of the

neuroaxis, including the vestibular nucleus, visual relays in the mesencephalon (where the periaqueductal gray lives), the thalamus, and the cortex.

A common site of tightness, in these C.P children, is the gastrocnemius and soleus complex (the calf) contributing to abnormal foot mechanics during stance phase of gait. The tightness of the calf musculature in CP children results, in most cases, in equinus gait. This posture of the foot interferes with the first heel contact with the ground, thus disrupting controlled forward progression during stance phase. Equinus gait eliminates the normal foot mechanics. The tightness of gastrocnemius-soleus complex influences the relative movement of the foot and knee in the stance phase of gait. The goal of that sub-occipital muscles decompression techniques is to provide a sufficient dorsi-flexion allowing for normal "heel to toe" progression via inhibit and relax the gastrocnemius-soleus complex with the aim of restoring functional walking capacity.

Gait in children with CP is characterized by a slower walking speed, a shorter-stride length, and more time spent in double support. Due to the above mentioned problems, a general decrease in physical activity as well as walking capacity has been observed in children with CP. Children with spastic hemiplegic cerebral palsy had a longer gait cycle, slower walking speed, and longer support phase than did healthy children. The support phase was longer than the swing phase in children with spastic hemiplegic cerebral palsy. There were significant differences in the angles of the hip, knee, and ankle joint between children with spastic hemiplegic cerebral palsy and healthy children at the moment of touching the ground and buffering, and during pedal extension. Children with spastic hemiplegic cerebral palsy had poor motor coordination during walking, which resulted in a short stride, high stride frequency to maintain speed, more obvious swing, and poor stability.

The gait cycle has two phases: stance and swing. The duration is defined in percentage, in which 100% represents the total cycle time. Stance phase occurs when the foot contacts the ground and accounts for 60% of the total cycle. In swing phase – 40% of the cycle – the foot "leaves" the ground and moves forwards. In normal gait these phases are symmetric in duration (if you count double support) and repeat in each successive cycle.

We believe there are three basic prerequisites for normal movement: range of motion within normal limits, adequate force production, and normal motor control. If any one of these is deficient, the quality of the motor function is compromised. Perhaps the major problem in the children with minimal motor involvement was centrally mediated muscle imbalance that produced weakness secondarily, although they demonstrated fairly good selective motor control. Increasing force production of the ant. tibial and quadriceps femoris muscles in these children as a result of calf and hamstring relaxation with decreased prolonged stretch on anterior tibial and quadriceps muscles from tight calf and hamstring appeared to produce spontaneous improvements in their gait. In the children with lack of functional walking capacity, their lack of selective motor control may have interfered with their ability to utilize these muscles for functional activities. More effective remediation of the motor deficits seen in CP must address motor control problems as well as static contractures. Impaired motor control in cerebral palsied children lead to desynchrony between abdominal muscles/diaphragm and thorax muscles because the abdominal muscles are activated subconsciously (spastic), whereas thorax muscles tend to be used mainly as an accessory. This means that when one group of muscles is working on inhaling, others may be working on exhaling. This leads to smaller and more rapid and disjointed breaths and can impair breath/ swallow coordination Can result in a flatter upper rib cage, depressed sternum, flaring of lower ribs and an overall smaller rib cage leading to decreased chest expansion.

Role of Trunk Stability in Postural Control

Trunk stability has been defined in terms of a coactivation of global and local muscles. So, specific training that promotes the function of these muscles is needed to achieve co activation. Exercises for this purpose have been termed lumbar stabilization or core stabilization exercises. Although no formal definition of lumbar stabilization exercises exists, this approach is aimed at promoting the neuromuscular control, strength, and endurance of muscles that are central to maintaining dynamic stability of the spine and trunk.

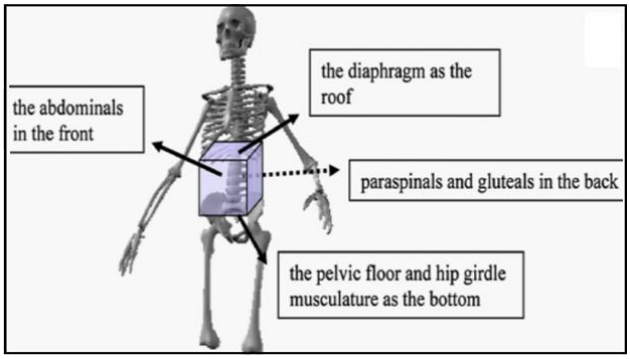

Figure 11: Role of Trunk Stability in Postural Control.

In general, the contraction of "antigravity" muscles is

primarily responsible for maintaining the body in an upright position both in both static and dynamic postures. The hip, knee, trunk, and neck extensors are considered major antigravity muscles. Other related muscles include the neck flexors and lateral benders, trunk flexors and lateral benders, hip abductors and adductors, and ankle pronators and supinators From a functional anatomy perspective, trunk muscles can be classified as either global or local muscles. The global muscles, such as the rectus abdominis (RA) and external obliques (EO), produce torque and transfer the load directly between the thoracic cage and the pelvis. The local muscles, such as the transverse abdominis (TrA) and lumbar multifidus (MF), have more direct or indirect attachments to the lumbar vertebrae. They are associated with the segmental stability of the lumbar spine during whole-body movements and postural adjustments. So, the functions of local muscles are necessary to enhance segmental stability of the spine.

The "core" is comprised of several groups of muscles including the transversus abdominus, multifidus, diaphragm and pelvic floor muscles. These muscles work together to produce maximum stability in the abdominal and lumbar (lower) back region, as well as coordinate the movement of the arms, legs, and spine. Fascia has been viewed clinically as a potential source of dysfunction. As several muscles are connected through the same fascia, myofascial chains may contain restrictions and dysfunction in one area that influence a remote area. Because of its lack of extensibility and its intimate relationship with the muscular system, fascia may limit free movement of joints, facilitating further dysfunction. These fascial layers help connect muscle throughout the region, creating myofascial chains. The abdominal fascia attaches to the external oblique, internal oblique, TrA, pectoralis major, and serratus anterior. It contains the links that form the diagonal muscle sling among the external oblique, pectoralis major, and serratus anterior. As the neuromotor system is released from dysfunction via myofascial release, the fascia is stressed by appropriate and orderly movement causing the collagen to lay down in the direction of the stress. A combination of increased level of ground substance with more orderly arrangement of the fibers therefore causes break down of the cross linkage and increases the extensibility of the muscle thus improving the chest expansion.

Treatment of Posture Deviations

Underlying mechanisms of treatment of posture deviation

The aim of this program is to correct and overcome muscular imbalance and motor control alterations.

Strength training used to be avoided in children with CP because it could lead to increased spasticity and reduced range of motion.

a. Postural control exercise for the trunk
b. Functional activating ex of inhibited abdominal and oblique abdominal muscles.
c. Myofascial Release was given to anterior chest wall: diaphragm, rectus abdominis, pectoralis major, intercostals and erector spinae muscles
d. Passive stretching ex. For tight anterior chest and erector spinae muscles
e. Hand function training as reaching in various directions as in sitting, upright position, kneeling, kneel sitting, sit to stand with support coordination, physical conditioning as well as motor learning
f. Chest expansion training ex which include maximum inspiration and expiration, will prevent stiffening of costovertebral joints and shortening of respiratory musculature.
g. PNF Activity of various upper limb patterns (upper extremity flexion, abduction, and external rotation) activates serratus anterior, both accessories respiratory muscles, and may possibly have an effect on the increase in respiratory muscle activity.
h. Appropriate activities such as swimming also have a positive effect on trunk motor control and pulmonary functions
i. Mat activities (bridging),unilateral bridging and sit to stand training
j. Weight transfer lateral and anteroposterior weight shifts to from anterior to posterior and left to right and finally reaching in all 3 planes within base of support; and dynamic reaching- requiring active trunk movements beyond the base of support
k. Pelvic tilting reciprocal control: anterior pelvic tilt is linearly associated with a force-coupling between the hip flexors and low-back extensor muscles. Modified trunk-hip activation exercise would produce a greater improvement in pelvic tilt motion during standing with diminished activation of the hip flexors and back extensors as well as increased activation of the hip extensors and trunk flexors.
l. Abdominal breathing, and pursed-lip breathing Individuals who experience difficulty breathing tend to compensate by excessive use of accessory respiratory muscles, including the PM and SCM during inspiration, and RA, EO, and internal oblique (IO) during expiration.the major respiratory muscles (the diaphragm and intercostal muscles) are activated together with the accessory respiratory muscles the PM and serratus anterior which helps expand the chest and improve breath in.
m. Facilitation of trunk and neck muscles coupled with posture exercises

n. Adaptive seating systems may improve posture, diminish the obstruction of the upper airway because of bad posture and improve respiratory function
o. Breathing exercises will be helpful in achieving effective inspiration and expiration, in improving coordination and in elimination of abnormal breathing patterns the breathing exercises were primarily encouraged using toys predominating expiratory maneuvers like blowing bubbles, whistles.
p. Upright posture activation (scapular retractors, spinal extensors, abdominals, glutei, etc.) due to trunk control is a part of postural control leading to
i. An improved breathing mechanics,
ii. Decreased chances of aspiration and improved recruitment of accessory muscles for increasing lung capacity
iii. Better chest wall recruitment for breathing and
iv. Improving onto diaphragm's mechanical advantage.
q. Balance training
i. Reactive postural adjustments or equilibrium reactions, which are flexible and varied responses to perturbations from the environment, can Improvement in motor behaviour -self initiated movements, or a moving support surface, towards expected perturbations.
ii. Load transfer and weight distribution patterns of core.
r. Soft Tissue Ideas of sub occipital treatment.

Indication of sub-occipital muscle inhibition (SMI) technique in pediatric

a. In dealing with C.P with extremely tight hamstrings, that attempting to stretch them is painful, or not possible or lead to strain.
b. During times of injury or after surgical release, when it would not be advised to passively take the hamstrings through a stretch
c. There are several safe treatment options for the sub occipital muscles inhibition
i. Compression
ii. Friction (be aware of your hand position for this)
iii. Positional Release

Trying to get the patient to relax and get comfortable is paramount over everything else when doing neck work (or any work for that matter!). This is not an area that we should be aggressive in, and appropriate touch and palpation is critical to a safe and effective treatment.

Compression & friction: Applying compression or friction to these muscles can be done so at the base of the occiput and even on the posterior tubercle of C1 and the spinous process of C2. Staying near the spinous process will ensure that you are not in the suboccipital triangle and you are away from the vertebral artery. Obviously if you feel a pulse under your fingers, change the position of your palpation. Take your time to slowly investigate these structures for trigger points and ischemia. Additionally, it helps to remember that the suboccipital muscles lie deep to other muscles, so don't be in a hurry to slam through all the superficial tissue. Take your time to appropriately assess the tissue for tone and tension, and ensure that the techniques are not producing pain and the client is comfortable enough for you to work deeper. When you find trigger points that are referring their common pattern (up into the head and behind the eyes) just hold that point for 8-12sec until the referral dissipates, and then move on to investigate the tissue next to it, keeping in mind that trigger points often form in clusters.

Positional release techniques: It is important to remind the therapist that the goal is to try and get all of your movement (contraction and then the passive movement to the next barrier of resistance) from the Atlas (C1) and Axis (C2). A good way to do this is to place one hand behind the head of the supine client, and then other hand on top of their forehead. This will help you move the head into flexion, stretching the sub occipitals, without getting too much contribution from the lower spine (C3-7). Additionally, in some acute situations, where there is pain upon isometric contraction (extension) of the sub occipitals, you can try and have the client contract the deep neck flexors (nodding their chin towards their Adam's apple and placing an isometric contraction into your hand which is on their forehead) to try and allow the sub occipitals to relax via reciprocal inhibition before taking them to their next stretch barrier. With the patient supine, the therapist sat at the head of the table and places the palms of hands under the subject's head, pads of therapist's fingers on the projection of the posterior arch of the atlas which is palpated between the external occipital protuberance and spinous process of axis vertebra. The therapist locates with the middle and ring fingers of both hands the space between the occipital condyles and the spinal process of the second cervical vertebra. Then, with the metacarpophalangeal joints in 90º flexion, therapist rests the base of the skull on hands. Pressure was exerted upward and toward the therapist. Treating the tight muscles for increasing length such as local site stretching techniques may cause aggravation of the local inflammatory response and may cause further muscle spasm and guarding. A different approach i.e. cervical spine treatment that might avoid compressing or stretching irritable structures but still produce an increase in joint range of motion and tight muscles extensibility. The change in the extensibility of muscle following application of cervical isometrics contract relaxes technique. Significant increase in remote joint range of motion.

Chapter 5

Rehabilitation of Soft Tissue Disorder

Ligaments and Tendons Disorder

Structure and function of ligaments and tendons

Tendons

A. Anatomy

a. Tendons contain collagen fibrils (Type I).

b. Tendons contain a proteoglycan matrix.

c. Tendons contain fibroblasts (biological cells) that are arranged in parallel rows.

B. Basic functions

a. Tendons carry tensile forces from muscle to bone.

b. They carry compressive forces when wrapped around bone like a pulley.

Ligaments

A. Anatomy

a. Similar to tendon in hierarchical structure.

b. Collagen fibrils are slightly less in volume fraction and organization than tendon.

c. Higher percentage of proteoglycan matrix than tendon.

d. Fibroblasts Ligaments and tendons exhibit both nonlinear and visco elastic behavior even under physiologic loading.

Stress-strain curve

Non linear elasticity: There are three major regions of the stress strain curve

a. The toe or toe-in region

b. The linear region and

c. The yield and failure region in physiologic activity.

Viscoelasticity: Viscoelasticity indicates time dependent mechanical behavior.

There are two major types of behavior characteristic of Viscoelasticity. The first is creep. Creep is increasing deformation under constant load.

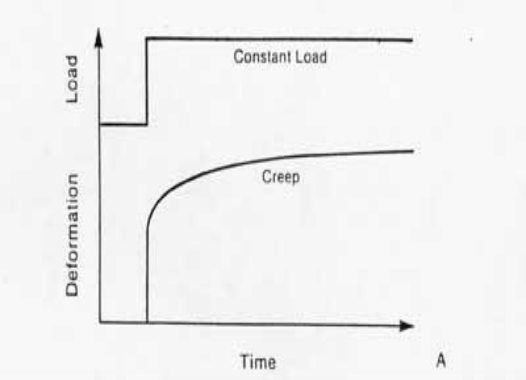

Figure 1: Creep (First significant behavior of Viscoelasticity).

The second significant behavior is stress relaxation. This means that the stress will be reduced or will relax under a constant deformation.

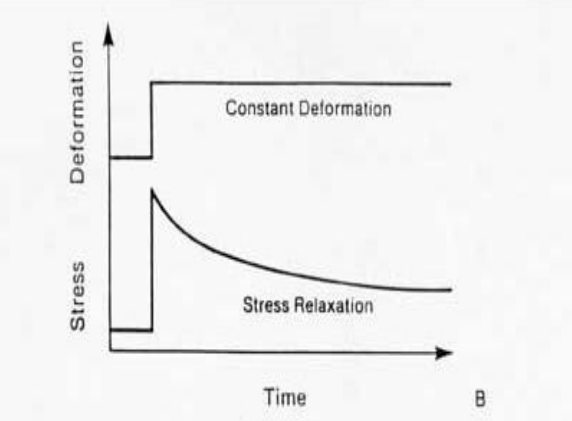

Figure 2: Stress Relaxation (Second significant behavior of Viscoelasticity).

Types of joints

a. Fibrous immovable: held together by ligament as tibio femoral joint.

b. Cartilaginous joint partially movable: articulated by cartilage as between vertebrae.

c. Synovial freely movable: contain synovial capsule.

Injuries to ligament and tendon caused by twisting or shifting force

a. Sprain ligament: due to twisting force in abnormal position cause some fiber cut.
b. Ruptured ligament: either complete cut or avulsion fragment.
c. Subluxation or dislocation: complete cut with partial or complete displacement.

Sprain

Physical problems of sprain

a. Pain
b. Swelling
c. Weakness
d. Atrophy
e. Redness and hotness
f. Loss of movement
g. Poor balance
h. Difficult ADL activities
i. Poor Proprioception
j. Instability of joint and subluxation

Treatment of sprain

Acute stage

a. RICE: Rest-ice application-compression-elevation.
b. Splinting to put the joint in functional position allow muscle strain for repair.

Recovery stage

a. Electrotherapy modalities
i. Superficial heat (dry or moist heat).
ii. Electric stimulation for stretched muscles to avoid atrophy and for treatment of atrophy via stimulation of protein synthesis inside muscle.
iii. Phonophoresis via using ultrasonic on site of sprained ligament away from the epiphyseal plate (joint surface) to prevent fibrosis of ligament by increase ligament fiber elasticity and improve healing.
iv. LASER scanned and probe laser can be used to stimulate the proliferation of ATP leading to improvement of healing.
b. Myofascial release on site of lesion to avoid fibrosis.
c. GAE for stretched muscle to strength muscles (starts with static ex. and progressed to isometric ex).
d. Balance training to improve postural reaction and stability.
e. Proprioceptive training via joint compression, weight bearing and vibration.
f. Mobilization of the joint to increase ROM without pain.
g. Splinting to put the joint in function position allowing for sprained ligament healing.
h. Posterior slap for sprained ankle or wrist is the best choice.
i. Anti-inflammatory drugs (systemic and localized).

Cruciate Ligament Tear

Twisting joint injury

Physical problems

a. Pain
b. Swelling
c. Weakness
d. Atrophy
e. Redness and hotness
f. Loss of movement
g. Poor balance
h. Difficult ADL activities
i. Poor Proprioception
j. Instability of joint and subluxation
k. Hemarthrosis in severe injury
l. Giving way of the knee joint
m. Knee instability

Treatment of mild cases by physiotherapy

A. Acute stage

a. RICE: Rest-ice application-compression-elevation.
b. Splinting to put the joint in functional position allow muscle strain for repair.

B. Recovery stage

a. Electrotherapy modalities
i. Superficial heat (dry or moist heat).
ii. Electric stimulation for quadriceps muscles to avoid atrophy and for treatment of atrophy via stimulation of protein synthesis inside muscle.
iii. LASER scanned and probe laser can be used to stimulate the proliferation of ATP leading to improvement of healing of soft tissues around knee.
a. Myofascial release on site of lesion to avoid fibrosis of soft tissue around.
b. GAE for stretched muscle to strength muscles around knee (start with static ex. and progressed to isometric ex.)
c. Balance training to improve postural reaction and stability.
d. Proprioceptive training via joint compression, weight bearing and vibration.
e. Mobilization of the joint to increase ROM without pain.
f. Splinting to put the joint in function position to prevent hyperextension knee allowing for sprained ligament healing (knee immobilizer and ankle foot orthosis to prevent hyperextension knee plus knee cage to assist in walking.
g. Anti-inflammatory drugs systemic and localized.
h. In moderate and sever injury need surgical repair.

Post operative physiotherapy treatment

The same treatment as above except

a. If swelling is persisted we use ice bag instead of superficial heat till the swelling subsides.
b. Mild decent gentle stretch for tight muscles.
c. Ultrasonic on fleshy part of tight muscles.
d. Gait training closed then open.

Muscular disorder

Types of muscles: skeletal muscle or striated muscle, responsible for locomotion, flight, cardiac muscle, which has a vital role and is able to function for a century or more, without ever taking a break, and smooth muscle or involuntary muscle which lines the walls of the arteries to control blood pressure, or controls the digestion of food by causing movement of the intestine.

Muscle contraction: The contraction cycle would thus involve the following events:

a. Liberation of Ca++ by the sarcoplasmic reticulum and its binding to Troponin, which abolishes the repulsion between the actin-troponin-tropomyosin system and the "head" of the HMM, with the ATP molecule bound to it.
b. Attachment of the HMM globule to "actin" which activates it as ATPase.
c. Splitting of the ATP into ADP and Pi. This splitting makes the HMM S2 shed its water envelope, whereupon the dehydrated thread curls up, shortens. The shortening of the HMM S2 pulls the thick and thin filaments alongside one another, causes them to slide.

Classification of muscular contraction: Static, concentric, eccentric, isometric, Isokinetic, isotonic.

Motor unit composed of

a. Motor neuron and all of the muscle fibers innervated by it.
b. Numbers of motor neuron supplied determine the degree of control.
c. Eye muscles and small muscles of the hand has great nerve supply so has great control.

Spasm

Pain, myositis and abnormal functional position of joint are the most common causes. Long run spasm leads to weakness

Complication of muscle spasm

Calf muscle spasm with long run	Secondary hyperlordosis
Cervical muscle spasm	Vertebro-basilar insufficiency
Lumbar muscles spasm	hyperlordosis
Piriformis muscle spasm	Clinical picture as sciatica
Pronator -teres spasm	Compression on posterior interosseous nerve lead to thenar atrophy
Stapedius muscle spasm	tinnitus
Spasm of paraspinal muscles	scoliosis
Spasm of hamestring	Knee and hip flexion
Spasm of scalp muscles	headache
Spasm of bronchial muscles	Asthma
Spasm of smooth GIT muscles	colics
Spasm of renal tubes	Renal colic
Spasm of coronary arteries	Angina pectoris

Table 1: Complication of muscle spasm.

Treatment of spasm

a. Superficial heat dry or wet.
b. Myofascial release of spasmed muscles.
c. After acute stage: mild, decent, gentle passive stretch.
d. Ultrasonic on fleshy part away from joint epiphyseal plate.
e. Splinting as a support and maintain new range.

Tightness of Skeletal Muscles

Deformity, post-plaster cast, post-operative, bedridden, associated with hypertonia, on opposite side of weakness are the most common causes.

Complications of muscle tightness

Tightness of culf muscles	Mechanical spinal pain
Tightness of hamestring	Flexion knee deformity and toe gait
Tightness of iliopsoas	hyperlordosis
Tightness of hip adductor	Hip subluxation
Tightness of rectus femoris	Flexion hip-extension knee
Tightness of sartorius	Flexion abduction external rotation with mild flexion knee
Tightness of tibialis posterior	Plantar flexion with inversion
Tightness of peroneus longus	Plantar flexion with eversion
Tightness of peroneus tertius	Dorsiflexion with eversion

Tightness of pereneous brevis	Eversion from mid position
Tightness of tibialis anterior	Dorsiflexion with inversion
Tightness of medial compartment of knee	Occur in bow leg
Tightness of lateral compartment of knee	Occur in knock knee
Tightness of quadrates lumborum	Lateral pelvic tilting
Tightness of back muscles	Hyperlordosis lumbar curve
Tightness of cervical extensors	Hyperlordosis cervical curve
Tightness of cervical flexors	Flattening of cervical curve
Tightness of abdominal muscles	Flattening of lumbar curve
Tightness of pectoralis major	Round shoulder
Tightness of periscapular muscles	Limitation of scapular movement and shoulder movement above 90
Tightness of subscapularis	Porter tip hand
Tightness of pronator	Loss of supination
Tightness of biceps brachii	Flexion elbow deformity
Tightness of wrist flexors	Wrist flexion deformity
Tightness of piriformis	Piriformis syndrome(squeeze sciatic nerve)
Tightness of pronator teres	Pronator teres syndrome(squeeze posterior interosseous nerve)
Tightness of tensor fasciolata	Flexion-abduction internal rotation of the hip with flexed knee
Tightness of lumbricals muscles	Dupuytren contracture
Tightness of thumb flexors	Trigger finger
Tightness of shoulder posterior capsule	Limitation of flexion
Tightness of shoulder ant. capsule	Limitation of extension

Table 2: Complications of muscle tightness.

Treatment of tight muscles

a. Superficial heat moist or dry to increase elasticity and improve circulation.
b. Mild, decent, gentle passive stretch to tight muscles.
c. Strengthening of opposite weak side by facilitatory techniques +resisted ex.
d. Maintain improvement of the ROM by splinting.
e. Home routine program.

Weakness

Prolonged over stretch on muscles lead to weakness, post operative cases, deformity, post- plaster cast, prolonged spasm, LMNL, abnormal functional position of joint.

Complications of muscle weakness

Weakness of gluteus maximum	Lurching gait
Bilateral Weakness of gluteus medius muscle	Waddling gait
Weakness or paralysis of quadriceps	Hand to knee gait
Weakness of pelvis muscles	Limbing gait
Weakness or paralysis of anterior tibial group muscles	High steppege gait
Unilateral weakness of gluteus medius	Trendlenburg gait
Weakness of abdominal muscles	Lumbar hyperlordosis
Weakness of back muscles	Flattening of lumbar curve
Weakness of cervical flexors	Cervical hyperlordosis
Weakness of cervical extensors	Flattening cervical curve
Unilateral weakness of paraspinal muscles	scoliosis
Weakness of triceps	Elbow weight bearing during creeping
Weakness of wrist extensors	Drop wrist
Weakness of tibialis posterior	Eversion +dorsiflexion
Weakness of tibialis anterior	Eversion+plantar flexion

Weakness of pereneous tertius	Inversion+plantar flexion
Weakness of pereneous longus	Inversion+dorsiflexion
Weakness of pereneous brevis	Inversion +midposition
Weakness of hamestring	Genu recurvatum
Weakness of rectus femoris	Genu recurvatum

Table 3: Complications of muscle weakness.

Treatment of weak muscles

a. Resisted ex. as isometric, eccentric, isotonic ex.
b. Functional activity as in swimming and walking in sand and theraband.
c. Facilitation of muscles by facilitatory techniques if the patient cannot perform active ex.
d. Graduated active ex. used when patient can perform active ex.
e. Maintain functional joint position to avoid over stretch on muscle which is the main cause of weak.

Atrophy

Underlying mechanism of atrophy

Destruction of actin and myosin leading to decrease numbers of sarcomeres leading to protein catabolism leading to decreased muscle contour .occur either due to

a. Pain lead to spasm and long run spasm lead to weakness and long run weakness lead to atrophy lead to deformity.
b. Tightness of muscles lead to over stretch on antagonist lead to weakness and long run weakness lead to atrophy lead to deformity.
c. Peripheral nerve lesion leading to interruption of reflex arch leading to loss of contractile function of muscle leading to loss of pumping action of muscles decreasing blood supply to muscles and bone leading to muscles atrophy and shortening of bone.
d. Disuse atrophy in UMNL.

Treatment

a. Neuromuscular electrical stimulation is used as prophylactic method to avoid atrophy and used to stimulate protein synthesis inside muscles to treat atrophy.
b. Isometric ex. used with NMES to overcome atrophy.
c. Splinting to maintain improvement.

Strain

Over stretch occur on muscles after trauma or overuse mechanism of injury

Physical problems of strain

a. pain
b. swelling
c. weakness
d. atrophy
e. redness and hotness
f. loss of movement
g. poor balance
h. difficult ADL activities
i. poor Proprioception

Treatment of muscle strain

Acute stage

a. RICE: Rest-ice application-compression-elevation.
b. Splinting to put the joint in functional position allow muscle strain for repair.

Recovery stage

a. Electrotherapy modalities
i. Superficial heat (dry or moist heat).
ii. Electric stimulation to avoid atrophy and for treatment of atrophy via stimulation of protein synthesis inside muscle.
iii. Phonophoresis via using ultrasonic on site of strain away from the epiphyseal plate joint surface to prevent Fibrosis by increase muscle fiber elasticity and improve healing.
iv. LASER scanned and probe laser can be used to stimulate the proliferation of ATP leading to improvement of Healing.
b. Mild, decent, gentle stretch of strained muscles to avoid fibrosis.
c. Myofascial release on site of lesion to avoid fibrosis.
d. GAE for strained muscle to strength muscles (start with static ex. and progressed to isometric ex.)
e. Balance training to improve postural reaction and stability.
f. Proprioceptive training via joint compression, weight bearing and vibration.
g. Mobilization of the joints to increase ROM without pain.
h. Splinting to put the joint in function position allowing for muscle healing.
i. Anti-inflammatory drugs systemic and localized.