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POLIO 2001 – AN OVERVIEW

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Acute Poliomyelitis

Because of effective immunization programs, acute poliomyelitis has become a rare occurrence in most of the world. The last major epidemics in the developed world occurred during the early 1950s.

Poliomyelitis is the result of a viral infection, which attacks the anterior horn cells of the spinal cord. The anterior horn cells control the skeletal muscle cells of the trunk and limbs. All of the anterior horn cells are affected with the acute infection. This accounts for the diffuse severe paralysis seen with the initial infection. A variable number of anterior horn cells survive the initial infection.

Acute polio is characterized by the sudden onset of paralysis accompanied by fever, acute muscle pain and often a stiff neck. Paralysis of the respiratory muscles is life threatening in the acute stage. When the shoulder muscles are involved, respiratory compromise should be suspected because of the close proximity of the anterior horn cells controlling each in the spinal cord. Mechanical support of ventilation may be required.

The treatment in the acute stage of the disease consists of providing the needed respiratory support, decreasing muscle pain, and performing regular range of motion exercises to prevent the formation of joint contractures.

Subacute Poliomyelitis

The subacute stage of polio is characterized by the recovery of a variable amount of muscle function. Mechanisms of regaining strength include 1) anterior horn cell survival; 2) axon sprouting; and 3) muscle hypertrophy. Although all of the anterior horn cells in the spinal cord were affected by the initial infection, some will survive. The average number of anterior horn cells to survive is 47 percent (Range 12 – 94 percent) as seen from post-mortem studies. The pattern of anterior horn cell survival in the spinal cord is random and does not follow anatomically continuous areas. The distribution of the paralysis is variable depending on which anterior horn cells were destroyed.

Additional muscle function is gained during the recovery phase by axon sprouting. One anterior horn cell innervates a group of muscle cells. When muscle cells are “orphaned” by the death of their anterior horn cell, a nearby nerve cell can sprout additional connections (axons) and “adopt” some of these muscle cells. A motor unit is defined as a nerve cell and all of the muscle cells it controls. The axon sprouting which occurs after polio can result in very large motor units.

The other mechanism by which peoples regain strength after polio is by muscle hypertrophy. The surviving muscle cells enlarge in an effort to provide additional needed

following onset, the emphasis is on preventing deformities and preserving function. Splinting and braces are often helpful to maintain joint position and supplement function.

Residual Poliomyelitis

It was during the residual stage that the orthopaedic surgeon traditionally became active utilizing surgical procedures to restore lost function and provide structural stability. If the person was still growing, it was important to prevent the formation of skeletal deformities resulting from the muscle imbalance.

The person with compromised function of the diaphragm was taught glossopharyngeal breathing in which air is swallowed into the lungs. This provides sufficient air exchange for light activities performed while sitting. The person often still required mechanical support of ventilation while sleeping.

Post-Polio Syndrome

With the last major polio epidemics in the United States occurring in the early 1950s, it has become common to see individuals who had polio as a child now returning complaining of increasing weakness. Many are concerned about loss of function. Polio has always been considered a static disease in that the paralysis is not progressive. The increasing weakness has been attributed to the overuse of muscles already weakened by the polio. Often muscles, which were thought unaffected, exhibit weakness at a later date. Studies have shown that a muscle must lose 30 to 40 percent of its strength for weakness to be detected using manual muscle testing. Gait laboratory studies have also demonstrated that activities of daily living require more muscle strength and stamina than were previously appreciated. Polio survivors have traditionally been encouraged to work harder to regain strength. The concept of “no pain – no gain” has proven detrimental to the polio survivor because it has encouraged chronic overuse of their muscles and resulted in further deterioration of function.

Most polio survivors begin to notice the deterioration approximately 30 years after the onset of the disease. The combination of symptoms varies slightly between people. The diagnosis of post-polio syndrome is made on the following clinical criteria:

- A history of poliomyelitis.
- A pattern of muscle weakness which is random and does not flow any nerve root or peripheral nerve distribution.
- A constellation of symptoms indicating decreasing strength and function.

There are no tests that are diagnostic for post-polio syndrome. Electromyography can demonstrate the presence of large motor units resulting from the previous axon sprouting. These findings are supportive but not diagnostic of polio.

- Increasing Muscle Weakness
- Severe Fatigue
- Muscle Pain
- Muscle Cramping
- Muscle Fasiculations
- Joint Pain or Instability
- Sleep Apnea
- Intolerance to Cold

Treatment of Post-Polio Syndrome

The treatment of post-polio syndrome is directed at preserving current muscle strength and preventing further weakness from occurring. Generally, it is not possible to strengthen a muscle that has been weakened by polio. Some gain strength can be seen when chronic overuse is corrected. The basic principles of treatment of post-polio syndrome are:

- Lifestyle modification to prevent chronic overuse of weak muscles.
- A limited exercise program incorporating frequent rest periods to prevent disuse atrophy and weakness.
- Lightweight orthotics support of limbs to protect joints and substitute for muscle function.
- Orthopaedic surgery to correct limb or trunk deformities.

Spine

A common complaint is back pain that usually results from postural strain. Excessive lumbar (low back) extension is used to substitute for weak or paralyzed hip extensors. Neck pain is frequently seen also. This, too, is from slowly increasing weakness. At times the neck muscles become tight from the strain and can actually press on the brachial plexus nerves of the arms causing numbness. This is called *Thoracic Outlet Syndrome*. It is treated with gentle stretching exercises of the neck. Both complaints can be treated by the use of external supports. It is important to relieve the excess workload from the muscles to prevent further deterioration. For neck pain tilting the seat of a chair backward 10 degrees is often sufficient to relieve the fatigue of the posterior cervical muscles from supporting the head. Patient education is essential since most individuals are reluctant to use braces that they long ago discarded.

Paralysis of the cervical (neck) spine musculature can result in the inability to maintain the head erect. This interfere with performing all other functions including walking. Fusion of the cervical spine can correct the problem.

Scoliosis (spinal curvature) is common secondary to paralysis and the resulting muscle imbalance. It can be further enhanced if a leg length discrepancy exists. External supports can be used to support the spine but these often interfere with breathing if the person is dependent on the use of accessory muscles for breathing. Spinal fusion may be needed to control the spine adequately. If fusion is needed, prolonged immobilization post-operatively is to be avoided. Prior to any surgery requiring general anesthesia or significant sedation, the vital capacity of the lungs should be assessed to determine the individual's needs for respiratory support.

Upper Extremity

Shoulder:

The shoulder is important for placing the hand in the desired position for use. The shoulder is totally dependent on muscle strength for active mobility. Weak muscles about the shoulder can be made more functional with the use of mobile arm supports for the wheelchair user. This allows a greater arc of motion with less muscle

strength. In the ambulatory person who requires upper extremity aids, shoulder stability is more important and a shoulder fusion may be helpful if there is sufficient strength of the chest wall muscles. Motion between the shoulder blade and the chest wall is maintained allowing use of the extremity for tabletop activities. Shoulder fusion does restrict the ability of the person to position the hand for bathroom hygiene so it is undesirable to fuse both shoulders.

Preservation of shoulder strength should be a priority of treatment since bracing and surgery of the paretic shoulder offer limited improvement. Shoulder weakness is found in 95 percent of individuals with post-polio syndrome and correlates closely with the amount of lower extremity weakness present. Rotator cuff tears are also common. Individuals with weak legs will use their arms to push up from a chair and pull themselves up stairs. They also lean heavily on upper extremity aids while walking. It is therefore important to remove as many unnecessary strains from the shoulders as possible. This can be done using elevated seats, motorized lift chairs, elevators or motorized stair chair glides, and optimal lower extremity bracing. In minimally or non-ambulatory individuals, an electric wheelchair or motorized scooter should be prescribed to prevent excessive strain on the shoulder muscles caused by propelling a manual wheelchair.

Elbow:

The elbow requires sufficient flexor strength to lift an object against gravity for function. A mobile arm support can maximize the effectiveness of the muscle strength for the person. Tendon transfers, such as the deltoid to the biceps, may also be useful in restoring active flexion.

Hand:

Opponens paralysis is common in the hand and results in a 50 percent loss of hand function. A splint used during the acute and recovery phases is useful to prevent an adduction contracture. Tendon transfer can restore Opponens function. The most muscle transferred is the superficial flexor of the ring finger.

Paralysis of the small intrinsic muscles of the hand interferes with function. A lumbrical bar orthosis will prevent hyperextension of the metacarpophalangeal joints and allow the long extensors to extend the fingers and open the hand. Surgical capsulodesis (tightening of the joint capsule) to limit metacarpophalangeal joint extension will accomplish the same result.

Paralysis of the finger flexors and extensors can be overcome with the use of a flexor hinge brace if wrist extensor function is present. Tendon transfers can provide the same result allowing the tenodesis effect to provide grasp and pinch functions.

Lower Extremities

Full range of motion of the hip and knee joints is needed for function. Contractures should be corrected when possible to permit more effective bracing. Iliotibial band contractures of the outer thigh are common. This causes the hip to assume a position of flexion, external rotation and abduction. The knee assumes a valgus (knock-knee) alignment and the tibia is externally rotated on the femur. Release or lengthening of the iliotibial band will correct the deformity.

An individual with flail lower extremities can stand using crutches and a knee-ankle-foot orthosis (KAFO) with the knees locked in extension and the ankles in slight dorsiflexion by hyper-extending the hips and utilizing the strong anterior hip capsule for support. Flexion contractures of the hips or knees prevents this alignment. If trunk support and upper extremity strength is adequate, the person could ambulate with a swing-through gait for short distances. This gait has high energy demands. With time the posterior knee joint capsule becomes stretched and the knee develops a recurvatum (back knee) deformity which is painful and can lead to arthritic degeneration of the knee. A knee-ankle-foot orthosis (KAFO) will protect the knee and provide improved stability for walking. If there is Grade 3 (FAIR) strength in the hip flexor muscles and passive full knee extension, then the knee joints can be left unlocked for walking. In this case a posteriorly offset knee joint is used to stabilize the knee and ankle dorsiflexion is limited to minus 3 degrees of neutral dorsiflexion to provide a hyperextension moment to the knee for stability.

Quadriceps (front thigh muscles) strength is not essential for ambulation. A strong gluteus maximus (buttocks) and good calf strength can substitute by keeping the knee locked in extension. If the calf strength is inadequate to control the forward motion of the tibia in mid to late stance, an ankle-foot orthosis (brace) is needed. It is not necessary to fix the ankle in mild plantarflexion to provide knee stability. This could result in a back knee deformity from the hyperextension push on the knee joint. An equines (toe down) position of the foot inhibits forward momentum and limits step length by preventing body weight from rolling over the forefoot prior to contact of the opposite leg with the ground. When good hamstring (back of thigh muscles) function is present, two of these muscles can be transferred forward to the quadriceps tendon to provide dynamic knee stability.

Muscle imbalances in the foot can lead to deformity. When muscle imbalances exist, tendon releases or transfers should be considered prior to the development of fixed deformities. Equinus (toe down) contracture of the ankle is very common. This results in genu recurvatum (back knee). The equines should be corrected by Achilles tendon (heel cord) lengthening. Accommodating the toe down posture by using an elevated heel on the shoe is not a good solution since this places excessive stress on the calf muscles to control the leg.

An Achilles tendon lengthening is frequently needed to correct an equines (toe down) contracture of the ankle to permit adequate bracing. When a cavus foot (very high arch) deformity is present, this causes forefoot equinus, which also limits bracing. If no fixed bony abnor-malities are present, then release of the plantar fascia ligament will be sufficient to correct the deformity. If the cavus deformity is caused by bony deformity, then a closing wedge osteotomy (cutting the bone) is needed to correct the deformity. A triple arthrodesis (fusion) of the heel can be used to correct deformities and provide a stable base of support.

The longstanding muscle imbal-ances, patterns of muscle substitution, and resulting joint and ligament strains often lead to degenerative arthritis. Total joint replacement can be performed but several special considerations are needed. Osteoporosis is common in the polio survivor because of prolonged lack of muscle

action on the bone. Joint contractures must be corrected at the time of surgery to prevent excessive forces on the artificial joint, which might lead to loosening. Weak muscles must be supported with the appropriate braces after surgery. The rehabilitation program will be longer than usual to regain joint motion and muscle function. Continuous passive motion devices and frequent joint range of motion must be used to gain joint mobility after surgery. Surgery can be expected to weaken the surrounding muscles. This must be taken into account before proceeding with total hip arthroplasty to prevent chronic dislocation.