Musculoskeletal complications in patients with hemiplegia/hemiparesis following a stroke

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## Introduction

According to The World Health Organization (WHO), cardiovascular disease is the leading cause of death in the world, with approximately 6.7 million people losing their lives due to stroke in 2012.[1] The Center for Disease Control (CDC) states that stroke is one of the leading causes of long-term disability in the United States.[2] There are approximately 795,000 annual occurrences, with 185,000 of these being recurrent.[2] Hemiplegia is a common condition that occurs after stroke, referring to paralysis on one side of the body. Hemiparesis refers to weakness on one side of the body and is a less severe form of hemiplegia. Though these clinical disorders may be considered neurological, they can lead to a number of musculoskeletal complications. These musculoskeletal complications are common in patients who have experienced a stroke and can have a large impact on their quality of life.

The purpose of this paper is to discuss the different causes and possible treatment options for some of the common musculoskeletal complications that may occur in patients with hemiplegia post-stroke. Common musculoskeletal problems that occur in individuals post-stroke include: spasticity, muscle contractures, gait abnormalities, osteoarthritis, and shoulder pain or subluxation. In patients with hemiparesis due to stroke, they may experience muscle stiffness, spasms, pain, and spasticity in the affected limb(s).[3] This may lead to abnormal positioning of the affected

side, making it difficult to perform daily activities and increasing the risk of them developing muscle contractures, osteoarthritis, and muscle atrophy.[3]

In general, the majority of patients experience the most functional recovery during the first four weeks of treatment following their stroke.[4,5] However, after this initial recovery, only about 60% of individuals with hemiparesis are able to independently walk short distances or perform simple activities of daily living.[4] Though previous research reports that patients do not experience any functional recovery between three and six months post-stroke[4], a recent study found that individuals showed significant functional improvement, though to a lesser extent, up to six months after stroke.[5] A study by Patel found that the severity and accumulation of deficits following stroke affect patient outcomes, suggesting that rehabilitation interventions should have a focus toward impairments that are observed rather than the stroke diagnosis.[6] Rehabilitation interventions should be individualized, and should also address the common musculoskeletal complications that can occur after a stroke. This includes performing a thorough assessment looking at motor weakness, sensory deficits, functional limitations, and biomechanical alignments that may lead to additional musculoskeletal impairments.[7]

## Spasticity and Muscle Contractures

Spasticity is defined as “a velocity-dependent hyperactivity of tonic stretch reflexes”.[7(e129)] Though spasticity may be considered a neurological condition, it can lead to a number of musculoskeletal complications. Spasticity is one of the most common impairments that takes place after stroke, and may lead to impaired function

and harmful effects including: pain, muscle contractures, poor skin hygiene, difficulty with voluntary movements, and problems with gait.[7,8] Prevention treatments for spasticity include stretching, exercise, splinting, and proper positioning.[9] Other options for the treatment of spasticity include: botulinum toxin injections, oral anti-spasticity drugs, baclofen, and phenol peripheral nerve blocks.[9]

Patients with hemiparesis and spasticity have an increased risk of developing muscle contractures, which can lead to severe functional impairment if left untreated.[7] Muscle contractures restrict movement or range of motion, can be extremely painful, and may hinder the patient's rehabilitation, leading to a poor outcome.[7] Prevention of muscle contractures includes: proper positioning, range of motion exercises, stretching, and splinting.[7] Treatment of muscle contractures should take place early on to prevent disability and may include: splinting, serial casting, or surgical correction.[7] In order to prevent muscle contractures, spasticity should also be addressed.

Treatment of spasticity should begin with the least-invasive interventions and progress to the more invasive interventions as needed. Interventions such as proper positioning, passive stretching, and passive range of motion exercises should be performed throughout the day for patients with spasticity.[7] Oral medications for the treatment of spasticity include: tizanidine, baclofen, dantrolene, and diazepam.[7] Current evidence for the use of these medications is fair to poor, and though patients may show improvement in spasticity and pain, there doesn't seem to be any improvement in function.[7] These medications may also cause side effects including: sedation, hypotension, muscle weakness, hallucinations, drowsiness, and fatigue.[10] Patients taking these medications should be monitored for side effects in therapy.

There is limited evidence for the use of phenol or alcohol neurolysis for the treatment of spasticity.[7,11] Though it may be effective in reducing spasticity, it is an invasive procedure with the potential for side effects.[7] Botulinum toxin injections have been shown to be safe and effective in treating spasticity.[8,12,11] A study looking at the combination of splinting and botulinum toxin injections versus splinting or botulinum injections alone in the upper extremity showed improvement in spasticity in all of the groups, but report that the functional effects of botulinum toxin injections may be more beneficial when combined with splinting.[9] A case report by Sun et al also found botulinum toxin to be effective in improving spasticity and function of the upper limb when combined with constraint induced movement therapy (CIMT).[13] Intrathecal baclofen infusion is another option for the treatment of spasticity, where a liquid form of baclofen is infused into the spinal fluid by use of a pump, and was shown to be effective in reducing spasticity in patients with chronic stroke (>6months post-stroke).[14,11] It should be noted that this study had a small sample size, but they report that the patients tolerated the medication well and did not demonstrate any side effects.[14] Though pharmacological intervention may help in reducing spasticity, it does not necessarily lead to an increase in function.[7] Therefore, clinical judgment should be utilized and the use of pharmacological treatments should be combined with other rehabilitation interventions to help improve independence with functional activities.

A study by Moreno et al investigated the utilization of dry needling to treat spasticity, pressure sensitivity, and plantar pressure in patients post-stroke.[15] They report that a single-session of dry needling helped decrease spasticity and pressure pain sensitivity in patients post-stroke.[15] However, further research is required in order

to determine whether these changes would be maintained long-term. Another study by Gomez et al also looked at the effects of dry needling combined with conventional rehabilitation for reducing spasticity in the scapular muscles.[16] The inclusion of dry needling did not have a significant effect on spasticity, but they found that it was effective in decreasing pressure sensitivity and improving shoulder range of motion.[16] Finally, the most invasive treatment for spasticity includes neurosurgical procedures such as a selective dorsal rhizotomy or a dorsal root entry zone lesion.[7] Selective dorsal rhizotomy has primarily been studied in the pediatric population and there is limited research in the population of interest. Dorsal root entry zone lesioning is a procedure in which the surgeon enters the spinal cord via the dorsal root and destroys sensory nerves that relay pain messages to the brain.[17] There are significant risks associated with these procedures including surgical complications and the possibility of spinal cord damage.[7]

Spasticity may also cause equinovarus deformity in patients post-stroke, inhibiting their ability to walk.[18] Shortening of the triceps surae may occur, causing the forefoot to hit the ground prior to the rearfoot, which leads to “improper loading of the limb”.[18(pg. 736)] This can cause pain in the foot and ankle and also lead to musculoskeletal injuries in the knee by forcing the knee into genu recurvatum.[18] Though botulinum toxin injection may help to decrease spasticity, the equinovarus deformity may still be present and require additional physical therapy treatment. Physical therapy should focus on increasing flexibility and strength in the triceps surae in addition to gait training.[18] Serial casting may also be effective in lengthening the triceps surae by allowing for a prolonged stretch following botulinum toxin injection.[18] A systematic

review by Borges et al found that resisted exercise training helps to improve overall motor function in individuals post-stroke, though it did not help in decreasing spasticity.[19] Current evidence reports that the current treatments for spasticity do not necessarily help improve overall function in adults with hemiplegia post-stroke; therefore clinical judgment and the adjunct use of therapeutic exercise interventions should be utilized when choosing treatment options for spasticity.

Changes in Gait

Patients with chronic stroke often demonstrate gait abnormalities due to weakness and spasticity, which leads to further difficulty with independence in activities of daily living. Hemiparetic gait patterns are variable depending on the severity of the stroke, the recovery of muscle function, and development of spasticity.[20,21] Individuals who have experienced a stroke seem to initially demonstrate abnormal gait patterns due to decreased muscle activation or weakness.[20,21] In a study by Mulroy et al, they performed a gait analysis on individuals with hemiparesis post-stroke immediately following stroke and 6 months post-stroke.[21] In the early stages, they found that there was a decreased activation of the soleus, tibialis anterior, quadriceps, and hamstrings muscles on the paretic side following stroke.[21] This seems to result in gait deviations or abnormalities during both the stance and swing phases of gait.[21]

Around 6 months post-stroke, gait velocity seems to increase, but there is still significant weakness in the triceps surae muscles, leading to compensatory gait patterns depending on the level of strength in the quadriceps, hamstrings, and gluteal muscles.[21] Those who demonstrate only mild weakness in their triceps surae and

quadriceps muscles may be able to increase their velocity by increasing the amount of ankle dorsiflexion, which helps increase their stride length.[21] Patients who demonstrate greater weakness in the hamstrings and gluteals than in the quadriceps will likely demonstrate excessive flexion at the hip, knee and ankle joints.[21] Appropriate intervention for this group may include an ankle foot orthosis that includes a dorsi-flexion stop combined with strengthening for the hip extensor muscles.[21] The patients who demonstrate greater strength in their hamstrings and gluteal muscles than their quadriceps will likely compensate at the knee by snapping into genu recurvatum.[21] Patients who demonstrate decreased activation of the plantar flexor muscles may either collapse into knee flexion or snap back into genu recurvatum.[21]

Genu recurvatum occurs during the stance phase of gait and is observed in approximately 40%-68% of patients with hemiplegia due to stroke.[22] There are many possible causes of genu recurvatum, and determining the cause is essential in order to appropriately treat these patients.[22] Some of the causes include: weakness in knee extensor muscles, spasticity of the knee extensors, weakness in gluteal muscles, weakness of the hamstrings, decreased ankle dorsiflexion, and proprioceptive disorders.[22] There are multiple reasons why genu recurvatum should be treated: genu recurvatum causes asymmetric gait, it increases energy cost of walking, and most importantly, it may cause pain due to the increased stress that is placed on the ligaments and the posterior capsule of the knee.[22]

Interventions should treat the underlying cause, meaning that clinicians need to perform a thorough gait analysis to determine the underlying cause of genu recurvatum before considering treatment options.[22] If the patient demonstrates knee extensor

weakness, they may compensate by keeping the knee in hyperextension to move the ground reaction force (GRF) in front of the knee, thus preventing the knee from buckling (see image in appendix).[22] Treatment may include strengthening of the knee extensors or the use of a hinged knee orthosis that will allow the patient to move into slight genu recurvatum during the stance phase, without over hyper-extending the knee.[22] Another option is a full leg brace to keep the leg in extension, however this brace has some disadvantages that should be considered including: difficult to don/doff, uncomfortable in sitting, and being heavy and bulky.[22] If the hamstrings are weak, strengthening and the use of an ankle foot orthosis (AFO) limiting plantar-flexion may help prevent genu recurvatum.[23,22] This type of AFO may also help address spasticity of the knee extensors or the posterior leg muscles, and may improve the patient's gait pattern.[23]

Decreased muscle activity in the tibialis anterior will prevent the amount of ankle dorsiflexion needed during mid-swing.[22] If there is spasticity or a muscle contracture in the posterior leg muscles, the patient may hyperextend their knee in stance due to inability to dorsiflex their ankle.[22] If they try to avoid genu recurvatum, they are likely to demonstrate equinovarus gait pattern[22], leading to equinovarus deformity as previously mentioned.[18]

Osteoarthritis

Gait deviations and altered biomechanics secondary to: abnormal muscle tone, decreased sensation, and motor paralysis may lead to osteoarthritis (OA) in individuals with hemiplegia post-stroke.[3,24] In a study that looked at the impact of lower extremity pain in patients post-stroke, they found that 51% of the individuals with lower extremity

pain was due to osteoarthritis.[25] Previous research has shown that an asymmetric gait pattern in the amputee population leads to an increase in OA.[26] Individuals post-stroke also demonstrate a similar asymmetrical gait pattern with increased stance time on the non-paretic limb compared to the paretic limb, that often continues even after rehabilitation and gait training.[27] A recent study by Patterson et al found that the prevalence of OA in patients post-stroke is 53% higher when compared to individuals who have not had a stroke.[3] This is likely due to the increased demand on the non-paretic limb, leading to damage of the articular cartilage.[3,27]

Knee OA may lead to a decreased functional capacity and a significant impact on quality of life due to pain and decreased range of motion.[25] OA can lead to joint pain, restricted range of motion, a decrease in muscle strength, and motor impairment[28], which may impair recovery from stroke.[29] The presence of pain may also lead to decreased movement and lower activity levels, which may complicate rehabilitation programs and have a negative effect on patient outcome.[3] OA should be managed early on to help improve patient outcomes and prevent further disability in this population. Though the prevalence of OA in the stroke population is high, there is little evidence addressing treatment in this patient population. Therefore, the use of clinical judgment and current treatment options for OA should be utilized.

According to the practice guidelines set forth by the European League Against Rheumatism (EULAR) task force, treatment of hip or knee OA should be tailored to each individual.[30] Since increased weight is a risk factor and will increase contact force[31], patient's who are overweight should receive be educated about weight loss.[30] A referral to a dietician may also be necessary to discuss nutrition and safe dieting.

Patients should also be educated about the importance of participating in regular, low-impact physical activity. Physical therapists should look at the patients' footwear to determine if there is a way to decrease mal-alignment or decrease contact forces by adding cushioning and shock absorption.[30] Treatment should also include protection of the articular cartilage through protected mobilization, utilizing the fluid film lubrication method to help nourish and protect the articular cartilage.[31] Joint mal-alignments should be addressed in order to decrease the overall contact pressure through increasing the contact area and decreasing the contact pressure, preventing further damage to the articular cartilage.[31] Increasing the contact area can be accomplished by addressing mal-alignments through the use of orthoses, bracing, or an assistive device.[30,31] The contact force may be decreased through attenuation of the ground reaction force, or finding a way to quiet the soft tissue forces.[31] Since patients post-stroke have one-sided weakness, early intervention and prevention is of the upmost importance to prevent OA.

Shoulder pain and subluxation

Approximately 72% of patients will experience shoulder pain in the first year following their stroke.[7] There are several possible causes of shoulder pain after stroke including: shoulder trauma, adhesive capsulitis, heterotrophic ossification, rotator cuff tear, tendonitis, complex regional pain syndrome, traction or compression neuropathy[7], and shoulder subluxation.[32] Different treatment interventions include: electrical stimulation, steroid injections, exercise, shoulder positioning, use of sling, ice, heat, mobilization, and soft tissue massage.[7] Shoulder subluxation is one of the most common musculoskeletal complications following stroke.[32] Though the use of supportive

devices such as slings and wheelchair attachments are commonly utilized to prevent shoulder subluxation, there is insufficient evidence to support this intervention.[32] Electrical stimulation of the shoulder muscles may help to increase muscle tone, which helped increase shoulder stiffness, however, there is not sufficient evidence to determine whether it is effective in reducing shoulder pain, reducing spasticity, or improving motor recovery.[33,34] However, electrical stimulation to the shoulder muscles is safe and may help reduce the severity of shoulder subluxation.[33,34]

The literature has found a strong correlation between shoulder pain in the paretic limb and restrictions in passive external rotation.[35] Koog et al found that an improvement in shoulder pain does not seem to be related to a reduction in shoulder subluxation or spasticity, but instead with an increase in passive external rotation.[35] Though more research is needed, treatment interventions that focus on increasing shoulder external rotation in patients post-stroke may help to reduce shoulder pain. This same study also found aromatherapy, slow-stroke back massage, and intramuscular neuromuscular electrical stimulation to be effective in reducing shoulder pain, however, intramuscular botulinum toxin injection and intraarticular triamcinolone acetonide injection were not helpful.[35] A systematic review by Berreca et al looked at treatment interventions for the upper extremity post-stroke and found that: protection, electrical stimulation, use of a sling, gentle range of motion, and avoiding the use of pulleys may reduce or prevent pain in the upper extremity.[36] Strengthening of the shoulder may help to improve upper limb strength and overall function without causing an increase in pain or spasticity.[37]

Therefore, treating post-stroke shoulder pain in the paretic limb should incorporate several interventions. Hospital staff and caregivers should be educated about proper positioning of the shoulder to help prevent subluxation or other trauma.[7] A shoulder sling may be used to help with positioning immediately following stroke when the muscles are flaccid.[7,32] Electrical stimulation may be helpful in preventing shoulder subluxation by increasing muscle tone and stiffening the shoulder.[33,34] Patients should receive range of motion, stretching, and gentle mobilization specifically focused on increasing shoulder external rotation and abduction to prevent shoulder pain or adhesive capsulitis.[7,35] Overhead pulleys should be avoided to prevent further pain and subluxation.[7,36] Modalities may be used to help control pain, and shoulder strengthening may help increase overall function without causing further pain.[37]

Conclusion:

In conclusion, there are many musculoskeletal complications that may occur post-stroke that can impact the functional independence and quality of life of these individuals. Rehabilitation interventions for patients with hemiparesis post-stroke should be individualized, and should also address the common musculoskeletal complications that may occur. A thorough assessment and movement analysis should be performed to determine: motor weakness, sensory deficits, functional limitations, and biomechanical alignments that may lead to additional musculoskeletal impairments.[7]

**Appendix:**

**A:** Genu Recurvatum[22]

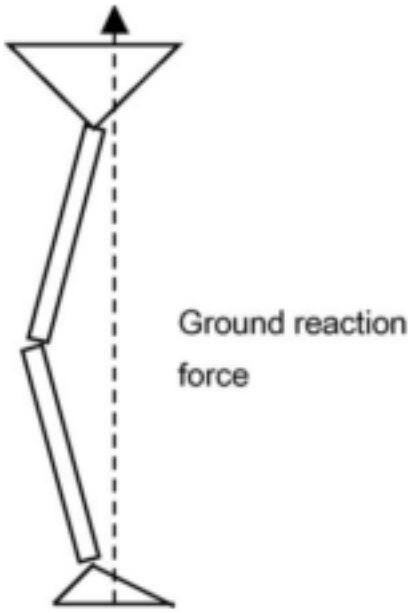


Fig. 1. Genu recurvatum and the ground reaction force. Representation of the patient's leg in the stance phase. The ground reaction force vector (dotted line) is well forward of the knee.

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