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# Knee Hyperextension in Stance Phase of Gait in Hemiparesis Factors and Their Management: An Exploratory Case Series

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## ABSTRACT

**Objective:** To understand the various factors that lead to genu recurvatum post stroke and their management and gait rehabilitation for stroke patients.
**Method:** This is an exploratory study. Five patients were recruited for the study based on inclusion criteria. Pre and post-test examination were taken by Wisconsin Gait Scale (WGS) and on standardized assessment for genu recurvatum along with pre and post- radiographs of hyper extended knee. A customized physiotherapy program was given to each patient.
**Results:** There is no statistical analysis because of different factors contributing to genu recurvatum post stroke and their intervention is customized accordingly. As a result, only depend on Wisconsin Gait Analysis and Roentgenogram. The results showed that the degree of knee hyperextension changes occur in pre and post analysis.
**Conclusions:** The findings of this study indicate that a customized physiotherapy program may be more effective in hyperextension of knee in stance phase of gait in hemiparesis

***Keywords:*** Stroke, Genu recurvatum / Knee Hyper extension

## INTRODUCTION

Stroke is a disease with high morbidity and disability rate. After Stroke, slower walking velocity, shorter Step length, higher cadence, larger Step width, and longer stance phase duration can be apparently observed. Additionally, these abnormal gait parameters eventually lead to knee hyperextension, known as genu recurvatum. Walking with such knee hyperextension usually induces knee pain and joint lesions, leading to cumulative damage and degenerative changes and further decreasing the standing phase's stability. Genu recurvatum is one of the physical impairments that can affect functional recovery and independence after stroke.

### Genu recurvatum Presentations

Morris M.E (1992) hyperextension of the knee during the stance period is called dynamic recurvatum which may influence the entire gait pattern. It affects between 40-68% of hemiparetic stroke patients. From a biomechanical point of view, genu recurvatum occurs during the stance phase. It is characterized by a ground reaction force vector that passes well in front of the knee.

Nielsen JP, 1988-1995 concluded that there are three presentations of genu recurvatum which are referred to as external rotary deformity recurvatum (ERDR) implies an elevated heel with the forefoot pointing inwards and foot remaining in an equinovarus position while walking. Internal rotary deformity recurvatum (IRDR) occurs when the forefoot rotates outwards, forcing the patient to overextend the knee. Non-rotary deformity recurvatum (NRDR) implies abnormal positioning of the knee, with foot and ankle functioning normally.

Genu recurvatum commonly occurs after stroke due to several causes which will affected in stance phase of gait. By assessing its different aetiologies and its possible cause effected tailored program design could made customized protocol for prevent genu recurvatum in stance phase of gait as a cause-effect relationship between impairments of stroke and gait pattern cannot yet be determined in order to guide training programs.

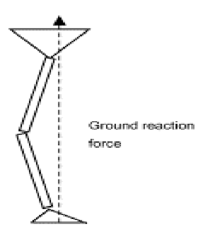



**Figure 1: - Showing Genu Recurvatum and Ground Reaction Vector Passes in Front of Knee Joint**

## Causes of Genu Recurvatum

1. ***Weakness of The Knee Extensors*–** Bleyenheuft et al, 2010 described that weakness of the knee extensors. In biomechanical terms, the patient keeps the knee in hyperextension so as to keep the ground reaction force in front of the knee, which prevents the latter from collapsing.
2. ***Spasticity of The Knee Extensors (The Vastus Muscles)*–** Usually, at the start of the stance phase, one observes discrete knee flexion in an eccentric movement, which helps dampen the impact of foot-ground contact. In the event of knee extensor spasticity during the stance phase, this normal knee flexion turns into abnormal extension.
3. ***Weakness of The Gluteal Muscles*–** P. Hansan et al, 2010 concluded that weakness of the gluteal muscles leads to forward pelvic tilt and lumbar hyperlordosis, excessive hip flexion and compensatory knee hyperextension. Fiziksel tip 2002 concluded that hip extensor weakness may contribute to genu recurvatum.
4. ***Weakness of The Knee Flexors (The Hamstring Muscles)*** – Nadeau S, (1999) concluded that weakness can also be iatrogenic in cases of over-extension of the hamstring muscles. In the stance phase, contraction of the knee flexor muscles is needed to control knee flexion– especially if the extensors are spastic. Conversely, spasticity of the knee flexors will generate excessive, permanent knee flexion. During the deceleration part of the swing phase, without the hamstring to slow down the swinging forward of the lower leg, the knee will snap into extension.
5. ***Limited Ankle Dorsal Flexion*–** As a result of spasticity and/ or retraction of the muscles in the posterior compartment of the leg, limited dorsal ankle flexion occurs. In this case, the knee is positioned in hyperextension due to the patient's inability to move the tibia forward during the stance phase, as a result of ankle stiffness. If the patient wishes to avoid genu recurvatum (and as long as the knee

flexor are sufficiently strong), patient will have to adopt an equinus gait Pattern.

6. ***Spasticity of Ankle Planter Flexors***– Moseley et al. 1993 described that ankle plantar flexor weakness and knee joint hyperextension during the mid-stance phase of gait. The gastrocnemius muscles produce knee flexor activity during mid-stance that acts to prevent knee hyperextension so weakness of this muscle could allow the knee to hyperextend.
7. ***Hip Flexion Contracture*** – Fiziksel tip (2002) concluded that hip flexion contractures or abnormal postural flexion at the hip can cause the ground reaction force anterior to the knee, and produce hyperextension Forces.
8. ***Proprioceptive Disorders*** – Proprioceptive deficit can also be one of the possible causes for knee hyperextension. Proprioceptive deficit causes decreased awareness of knee joint position. Reduced knee joint proprioception associated with the other etiologies, can increase the chances of knee hyperextension.

Some of the Individuals with stroke will be undergoing physiotherapy and some may not be undergoing physiotherapy. There is little literature available on the treatment guidelines prescribed to prevent or treat knee hyperextension in this population. The treatment methods adopted by physiotherapist to prevent and treat knee hyperextension may not have addressed all the possible causes of knee hyperextension. So it is necessary to assess different aetiologies that lead to knee hyperextension would be assessed in this study and customized physiotherapy program would be applied to patients with genu recurvatum post stroke.

## MATERIAL &METHODOLOGY

### Subjects and Assessment

The stroke patients who visited study center and further met the inclusion criteria selected as participant during specified schedule. A total of 5 stroke patients who were diagnosed either ischemic or hemiplegic who can walk 10 meters with or without support were purposively chosen from the outpatient or inpatient department at MVG University were selected as subjects for the Study. After necessary instructions and information about the study, the subjects had explained about the complete study procedure in his/her own language and his/her willingness to participate in the study had recorded in a consent form dually signed by participants. The study was approved by Institutional Ethical Review Board (IRB). Ultimately of all selected patients were available for case description study. The study group was analyzed for various parameters in order to evaluate various factors that contribute their genu recurvatum so that a customized physiotherapy program would be applied to patients with genu recurvatum post stroke.

### Subject Group

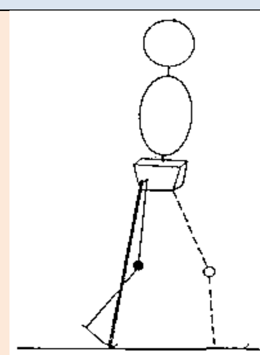
All subjects were males, 40–60 years of age, and who can walk 10 meters with or without support. All subjects also underwent for evaluation of their ambulatory performance by Wisconsin Gait Scale (WGS). Further a standardized assessment which include various causes of genu recurvatum were assess and pre x-ray of knee by radiologist through digital imaging and communications in medicine (DICOM®) format in order to lateral view in hyperextension of knee of patient in standing position (weight bearing position on affected limb) for calculation of angulation post stroke and those findings will not be discussed in detail, other than to mention that all imaging was interpreted clinically knee hyperextension not within normal limits and subject had a major physical impairments that affect their independence after stroke specially difficulty in walking

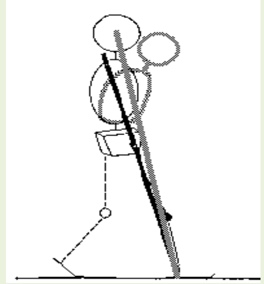
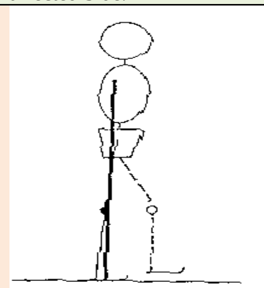
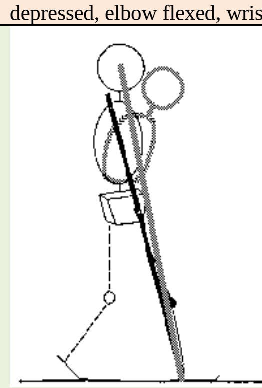
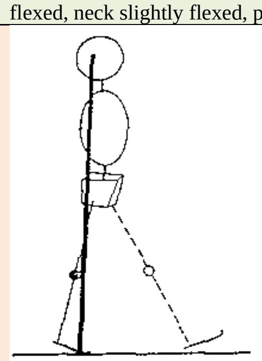
The Study Group (n=5) had analyzed for various parameters in order to evaluate ambulatory activity by Wisconsin Gait Scale (WGS). Each patient received treatment protocol for 20 days that includes customized physiotherapy individually tailored framework. The total time of instructed physiotherapy was 20 hours and together with customized physiotherapy program practiced for 20 hours. Per session includes passive stretching for the spasticity or tight muscles, gait training for problems related to gait deviation with/ without aids, static and dynamic strengthening exercises through wt. cuff according to patient tolerance for weak muscles, joint compression exercise and stroking has to be given for sensory deficit patients. Further graded exercise progression will be achieved by increasing the difficulty of the exercise protocol; adding weights; in case of muscle weakness; increasing the number of repetitions. Home protocol was designed for all participants including gait training and stair climbing training along with self- stretching and strengthening exercises. After 20 days re-assessment was performed including post Wisconsin Gait Score, post x-ray of knee by radiologist through DICOM format in order to lateral view the knee of patient in standing position (weight bearing position on affected limb) for calculation of post treatment change in angulation of genu recurvatum.

## CASE DESCRIPTION

Table 1 explores the description of five consecutive patients referred to physical therapy outpatient department at MVG University with a diagnosis of CVA/ stroke was screened for the eligibility criteria were selected for study. Each session describes how cases were examine and treatment is processes and discharge planning is done for stroke survivor, thus provided the benefits of a specialized rehabilitation tailored framework protocol would contribute to a positive patient experience overall.

**Table 1: - Shows Exploratory Case Study Framework**

| Case series | Observatory findings of various factors that could lead to genu recurvatum revealed | Gait observation (stance phase on affected side) | Intervention |
|---|---|---|---|
| **Patient 1: -**<br>• 44 yrs. /M<br>• Left CVA<br>• 1yr post stroke duration | • Weakness of ankle dorsiflexors.<br>• Tightness in the knee extension and ankle plantar flexors.<br>• Reduce active knee flexion range.<br>• Pronated and externally rotated foot.<br>• Proprioception loss in fingers and toes.<br>• Knee and ankle reflexes were exaggerated.<br>• Tone was increased in plantar flexors and knee extensors. | <br>Right knee hyper extended with hip maintained in extension and ankle is pronated and everted, shoulder drooped, arm hang on affected side. | • Passive stretching of knee extensor, ankle plantar flexor, evertors.<br>• Static AFO is removed as it was inhibiting dorsiflexor function.<br>• Strengthening exercises for knee flexors with weight cuff.<br>• Joint compression exercise.<br>• Gait training.<br>• Stair climbing training.<br>• Home protocol was designed for self- stretching and strengthening exercises. |

| | | | |
|---|---|---|---|
| **Patient 2: -**<br>• 64 yrs. /M<br>• Rt. CVA<br>• 5 yrs. Post Stroke | • Weakness in hip extension.<br>• Limited ankle dorsiflexors.<br>• Tightness in knee flexors and hip flexor.<br>• Increased tone in ankle plantar flexor, knee extensors.<br>• Proprioception loss in fingers and toes.<br>• Knee and ankle reflexes were exaggerated.<br>• Tone was increased in plantar flexors and knee extensors. | <br>Left knee hyper extended with hip maintained in extension and ankle in everted with flat foot, Shoulder drooped, elbow slightly flexed, Wrist dropped, finger dropped while trunk flexed on affected Side. | • Passive stretching of knee flexors and hip flexors, knee extensors, ankle plantar flexors.<br>• Strengthening exercise for hip extension, ankle dorsiflexors.<br>• Joint compression exercise.<br>• Gait training without walking aids.<br>• Stair climbing training.<br>• Home protocol was designed for self-stretching and strengthening exercises. |
| Patient 3: -<br>• 40 yrs. /M<br>• LEFT CVA<br>• 3 months Post Stroke | • Weakness in knee extensors.<br>• Limited ankle dorsiflexion.<br>• Tone was increased in ankle plantar flexor.<br>• Reduce knee flexion. | <br>Right knee hyper extended with hip maintained in extension and ankle in prorated, shoulder depressed, elbow flexed, wrist flexed position. | • Strengthening exercise for knee extensors, Hip flexor, Ankle dorsiflexors.<br>• Stretching exercise for ankle plantar flexors.<br>• Gait training without walking aids.<br>• Stair climbing training.<br>• Home protocol was designed for self-stretching and strengthening exercises. |
| Patient 4: -<br>• 46 yrs. /M<br>• Left CVA<br>• 4yr Post Stroke | • Weakness of knee flexor, hip extensors.<br>• Limited ankle dorsiflexors.<br>• Tightness in knee extensors and hip flexors, Ankle plantar flexors.<br>• Increased tone in ankle plantar flexor, Knee extensors.<br>• Knee and ankle reflexes were exaggerated. | <br>Right knee hyper extended with hip in flexion, foot in neutral, trunk slightly forward flexed, shoulder drooped, elbow severely flexed, wrist flexed, neck slightly flexed, protruded chin. | • Strengthening exercise for knee flexion, hip extension, ankle dorsiflexors.<br>• Stretching exercise for ankle plantar flexors, knee extensors, hip flexors.<br>• Gait training.<br>• Stair climbing training.<br>• Home protocol was designed for self-stretching and strengthening exercises. |
| Patient 5: -<br>• 56yrs. /M<br>• Left CVA<br>• 5yrs. Post Stroke | • Proprioception loss in elbow, wrist, finger, knee, ankle, toes.<br>• Weakness in ankle plantar flexors.<br>• Compensation of contralateral leg swing duration is reduced so that double support is achieved early. | <br>Right knee hyper extended with hip in flexion and ankle in neutral and foot also neutral, shoulder drooped, elbow flexed, wrist neutral, hand neutral, head neutral, neck neutral. | • Joint compression exercise for elbow, wrist, finger, knee, ankle, toes.<br>• Strengthening exercise for ankle plantar flexors.<br>• Gait training.<br>• Stair climbing training.<br>• Home protocol was designed for strengthening exercises. |

# RESULTS

## Sample characteristics

Table 2 summarizes the demographic age, side of hemiplegic involved, duration post stroke. Measurement is taken around the knee at 5cm above the level of patella for muscle wasting. Evidence of a decline in muscle mass girth in affected limb after the stroke.

**Table 2: Participant Demographic**

| Variable | Study Group (n=5) |
|---|---|
| Age | 40-65 yrs. |
| Left Stroke | 80% |
| Right Stroke | 20% |
| Duration Post stroke | 3 mon. – 5 yrs. |
| Quadriceps Muscle Girth | Reduction of Girth on Affected Side |

**Patient 1:** *Pre Wisconsin gait scale score* was 34. 6, and *Pre-x-ray of knee* revealed 19° (161°--180°) of hyper-extension and the *post Wisconsin gait scale score* was 29.7 and *post-x-ray of knee* revealed 14° (166°-180°) of hyper- extension.

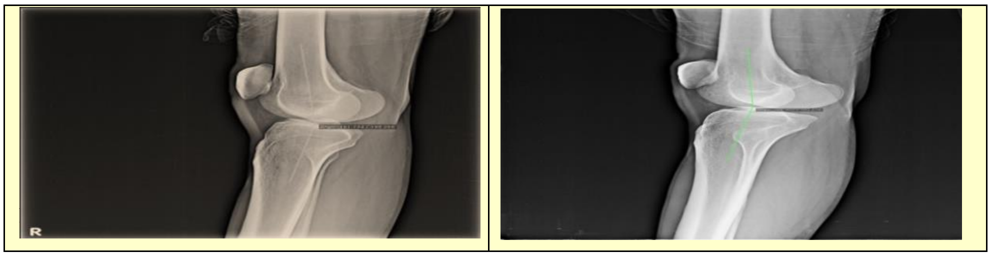
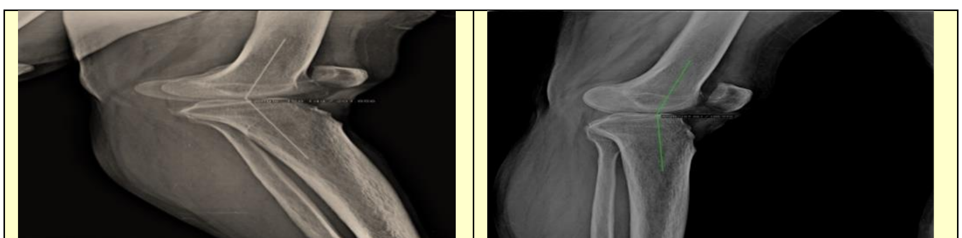


**Figure 2: - Shows Patient 1Pre vs. Post Therapeutic X-ray of Right Knee**

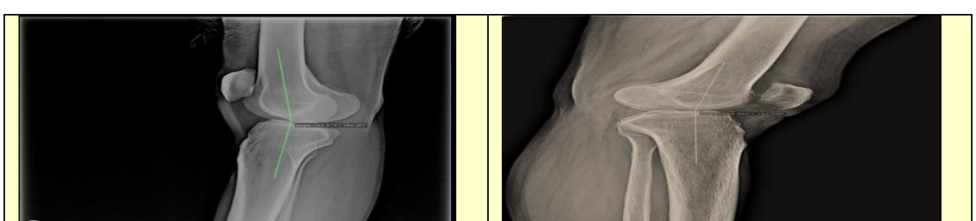
**Patient 2:** *Pre Wisconsin gait scale score* was 32.45, and *Pre-x-ray of knee* revealed 22°(158°-180°) of hyper-extension and the *post Wisconsin gait scale score* was 28.5 and *post-x-ray of knee* revealed 17°(163°-180°) of hyper- extension.



Pre therapeutic x-ray of Left knee | Post therapeutic x-ray of Left knee

**Figure 3: - Shows Patient 2Pre vs. Post Therapeutic X-ray of left Knee**

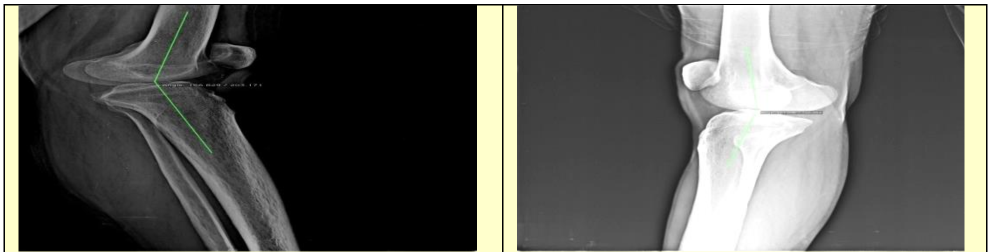
**Patient 3:** *Pre Wisconsin gait scale score* was 28, and *pre-x-ray of knee* revealed 15°(165°-180°) of hyper-extension and the *post Wisconsin gait scale score* was 25 and *post-x-ray of knee* revealed 12°(168°-180°) of hyper-extension



Pre therapeutic x-ray of Right knee | Post therapeutic x-ray of Right knee

**Figure 4: - Shows Patient 3 Pre vs. Post Therapeutic X-ray of Right Knee**

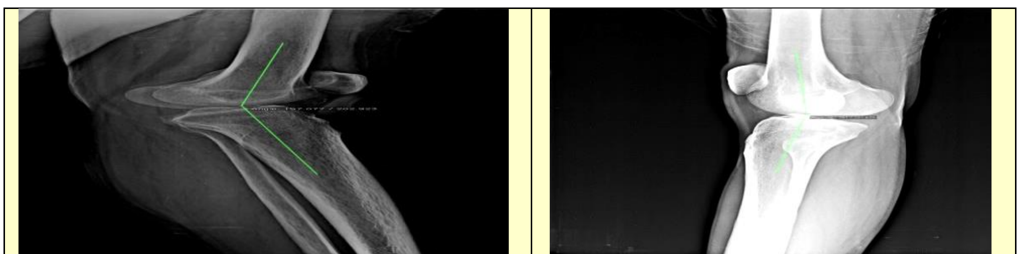
**Patient 4:** *Pre Wisconsin gait scale score* was 28.6, and *pre-x-ray of knee* revealed 24° (156° - 180°) of hyper-extension and the *post Wisconsin gait scale score* was 24 and *post-x-ray of knee* revealed 19° (161°-180°) of hyper- extension.



Pre therapeutic x-ray of Right knee | Post therapeutic x-ray of Right knee

**Figure 5: - Shows Patient 4 Pre vs. Post Therapeutic X-ray of Right Knee**

**Patient 5:** *Pre Wisconsin gait scale score* was 16, and *pre-x-ray of knee* revealed 23°(157°-180°) of hyper-extension and the *post Wisconsin gait scale score* was 14 and *post-x-ray of knee* revealed 18° (162°-180°) of hyper- extension.



Pre therapeutic x-ray of Right knee | Post therapeutic x-ray of Right knee

**Figure 6: - Shows Patient 5 Pre vs. Post Therapeutic X-ray of Right Knee**

## DISCUSSION

The objectives of this explorative study were to provide summary findings on "*various factors of knee hyperextension in stance phase of gait in hemi-paresis patients with stroke and their management*". WGA indicated that all Patients improved their gait after intervention. In contrast to the results of the Copenhagen Study (Jorgensen et al. 1995c), which detected no further improvement of gait function in hemiparetic patients three months post-stroke, gait improvements in patients more than six months post-stroke were obtained. When sufficient time is spent in active and focused exercise, even stroke patients can obtain beneficial results. While a number of processes have been identified as playing a role in neurological recovery following stroke, the role each play is not completely understood. Recovery from stroke is often attributed to resolution of edema and return of circulation within the ischemic penumbra (Dombovy 1991).However, spontaneous recovery can be prolonged well past the resolution period of acute structural changes caused by the stroke, with recovery occurring 4-6 weeks post stroke (Brodal 1973).Furthermore, animal and human trials have indicated that the cerebral cortex undergoes functional and structural reorganization for weeks to months to years following injury with compensatory changes extending up to 6 months to years in more severe strokes (Green 2003). Recovery is grouped into two categories: Local CNS processes (Early Recovery); CNS reorganization (Later Recovery). The ability to manipulate specific neuronal pathways and synapses has important implications for physiotherapeutic clinical interventions that will improve Health.

Promising therapies like specific exercise training and other physical neuro-rehabilitation can enhance brain and neuromuscular adaptation. PT for neurological patients is a comprehensive process that intends to teach, guide, and promote brain plasticity, thus reducing the threats for any functional and cognitive variations. As 20 days of customized physiotherapy program significantly improved the gait improvement abilities among stroke patients thus the WGA score shows difference from their pre and post intervention score. The total time of instructed physiotherapy was 20 hours and together with customized physiotherapy program practiced for 20 hours. However, specific gait characteristics were important to design specific program for individual gait pattern. Improvement in walking function is the goal most often stated by stroke patients. Therefore, a significant portion (25-45%) of physiotherapy time is spent addressing gait dysfunction, Independence, Quality of Life and participation. Many rehabilitation approaches to improving post-stroke gait exist and the following discussion will focus on the evaluation of various factors of knee hyperextension in stance phase of gait in hemi-paresis patients with stroke and their management.

Weakness of the knee extensors according to Perry J. (1992) in biomechanical terms, the patient keeps the knee in hyperextension so as to keep the ground reaction force in front of the knee, which prevents the latter from collapsing. With mild to moderate weakness, the knee is extended at or prior to heel-strike and knee flexion is eliminated or reduced. At times, this movement into full extension can be quire forceful, snapping the knee back. When normal plantar flexor and hip extensor strength is present, knee extension can be maintained by use of the hip extensors acting in a closed kinetic chain and/or by increased plantar flexor activity, which shifts the COP forward on the foot, in turn moving the GRF line in front of the knee.

Spasticity of the knee extensors (The vastus muscles) according to Perry J. (1992) usually, at the start of the stance phase, one observes discrete knee flexion in an eccentric movement, which helps dampen the impact of foot-ground contact. In the event of knee extensor spasticity during the stance phase, this normal knee flexion turns into abnormal extension. Knee is maintained in extension, during the swing phase or throughout the gait cycle, due to contracture of the rectus femoris, vastus medialis, vastus lateralis, vastus intermedius and/ or the hamstrings and may also be caused by hip flexor weakness or calf muscle spasticity. Toe drag is likely during the early swing phase. Circumduction of the involved limb, hiking of the pelvis, and/or contralateral limb vaulting may occur as a result.

Weakness of the gluteal muscles according to P. Hansan et al, (2010); Fiziksel Tip (2002) weakness of the gluteal muscles leads to forward pelvic tilt and lumbar hyperlordosis, excessive hip flexion and compensatory knee hyperextension. With weakness, several compensatory patterns are observed. Walking speed is slowed to reduce forward momentum (often early strategy when weakness is bilateral and affects both hip and back extensors). The trunk COM is moved relatively posterior by increasing lumbar extension or posterior trunk lean. This allows the GRF line to pass close to or posterior to the hip, allowing gravity to assist in maintaining joint stability. When weakness is isolated to gluteus maximus, there is a backward thrust or throwing of the trunk at heel strike, which moves the trunk posteriorly. To reduce any tendency for the hip to move into flexion, there is a reduction in knee flexion and the limb is maintained in a more extended position.

Weakness of the knee flexors (The hamstring muscles) according to Nadeau S., (1999), Bohannon and Andrews (1990) weakness can also be iatrogenic in cases of over-extension of the hamstring Muscles. In the Stance Phase, contraction of the knee flexor muscles is needed to control knee flexion – especially if the extensors are spastic. Conversely, spasticity of the knee flexors will generate excessive, permanent knee flexion. During the deceleration part of the swing phase, without the hamstrings to slow down the swinging forward of the lower leg, the knee will snap into extension. Knee is maintained in extension, during the swing phase or throughout the gait cycle, due to weakness of hamstrings and may also be caused excessive hip extension and ankle dorsiflexion and reduce plantar flexion during stance to pass COG anterior to knee joint. Toe drag is likely during the early swing phase. Circumduction of the involved limb, hiking of the pelvis, and/or contralateral limb vaulting may occur as a result.

Limited ankle dorsal Flexionas a result of spasticity and/or retraction of the muscles in the posterior compartment of the leg, limited dorsal ankle flexion occurs. In this case, the knee is positioned in hyperextension due to the patient’s inability to move the tibia forward during the stance phase, as a result of ankle stiffness. If the patient wishes to avoid genu recurvatum (and as long as the knee flexors are sufficiently strong), Patient will have to adopt an equinus gait. With mild to moderate weakness, this motion is poorly controlled (restrained) leading to "Foot Slap", which is best observed as walking speed increases. Lateral ankle stability may be reduced (remember, the dorsiflexors also evert or invert), increasing the risk of sprains and injuries. When weakness is severe, heel-strike may be absent entirely because of an inability to dorsiflexthe foot during swing. During swing phase toe clearance is reduced. This functionally lengthens the swing phase limb. A “Steppage Gait” (increased hip and knee flexion) is typically adopted to supply the necessary clearance.

Spasticity of ankle plantar flexors according to Moseley et al., (1993), Allison Cooper et.al., (2011) Ankle plantar flexor weakness and knee joint hyperextension during the mid-stance phase of gait causes the gastrocnemius muscles produces knee flexor activity during mid-stance that acts to prevent knee hyperextension so weakness of this muscle could allow the knee to hyperextend. In Stance phase, ankle plantar flexion spasticity prevents smooth forward movement of the trunk, making it difficult to "step" through and complete a normal gait cycle. At heel-strike, a plantar flexion spasticity will result in an absent heel strike and floor contact either flatfoot or with the forefoot depending on the severity of the contracture. Floor contact with the foot plantar flexed moves the center of pressure well anterior to its usual location in early stance. This moves the GRF vector anterior to the knee, resulting in inappropriate knee extension or hyperextension during the loading response.

Several patterns of gait abnormalities and compensatory strategies can be seen with plantar flexor spasticity. When no other problems are present, healthy individuals will often simply walk on the Forefoot (“Toe Walking”). This requires good strength and the ability to walk at reasonable speeds since inertia is used to facilitate the progression of' the trunk up and over the stance limb. More typically, plantar flexion spasticity occurs in combination with changes in muscle tone, strength, and voluntary control or with other joint abnormalities. In this context, the ability to compensate is more limited. In these Patients, the plantar flexed foot moves the GRF vector anteriorly far earlier in the gait cycle than normal. This results in early and prolonged knee extension (or hyperextension), often persisting though stance. At times, this can result in a rather forceful and rapid snapping of the knee into extension, the so called “Extensor thrust”.

When the plantar flexed foot prevents or severely limits forward rotation of the tibia, it becomes difficult for the trunk to progress forward over the stance limb. Increasing forward trunk Lean moves the COM over the stance phase limb that remains extended at the knee and plantar flexed at the ankle. As long as the COM does not move beyond the base of support and there is adequate proximal muscle strength to control the trunk motion, this strategy allows upright posture to be maintained. It is associated, however, with very short step lengths (“Step To” gait), slow walking speeds, and is usually seen in moderate to severely disabled Patients.

Weakness of ankle plantar flexors according to Moseley et al., (1993) Ankle plantar flexor weakness and knee joint hyperextension during the mid-stance phase of gait causes the gastrocnemius muscles produces knee flexor activity during mid-stance that acts to prevent knee hyperextension so weakness of this muscle could allow the knee to hyperextend. When weakness is present, excess anterior sagittal plane tibial-rotation (i.e., Dorsiflexion) is presented in mid and late stance (i.e. the foot remains dorsiflexed and heel rise is lost or attenuated). The rapid forward rotation of the tibia in stance moves the knee forward, prolonging the time during which the GRF line passes behind the knee. This increases stance phase knee flexion and the muscular demands on the quadriceps. The beneficial feature of the increase in knee flexion is to slow (but not prevent) trunk advancement over the stance phase leg. As the trunk continues its forward progression over the stance leg, the COM move further forward of the ankle joint, increasing the moment (torque) that is normally countered by the plantar flexors. This leads to a potentially unstable situation requiring than the stance limb be quickly unloaded to prevent dorsiflexion collapse. The contralateral leg step length (swing Duration) is reduced so that double support is achieved early.

Hip flexion contractures according to Fiziksel Tip, (2002) hip flexion contractures or abnormal postural flexion at the hip can causes the ground reaction force anterior to the knee, and produce hyperextension forces. When a hip flexion contracture is present, abnormalities will initially be seen during the latter half of stance, when maximal extension range is needed. When extension range is lacking, the pelvis must flex forward. Without any compensatory motion, this would force the trunk into a forward leaning position, moving the GRF anterior to the hip and increasing the hip extensor muscle torque required to stabilize the trunk. The most common compensatory strategy used by patients is to increase lumbar spine extension (i.e., Lordosis) to allow the trunk to remain vertically oriented. Lumbar spine extension can effectively compensate for hip flexion contractures up to about 15 degrees.

When hip flexion contractures exceed 15 degrees (a common occurrence) or there is limited lumbar spine extension range available (also common) the patient is forced to adopt a forward trunk tilt in terminal stance in order to complete the step. An alternative strategy used by some patients to compensate for limited hip extension is increased knee hyperextension and ankle plantar flexor (genu recurvatum). This strategy is uncommon because it ends to be very fatiguing and may increase pain in patients with hip joint arthritis.

Proprioceptive disorder according to Loudon JK, (2000) Proprioceptive deficit can also be one of the possible causes for Knee hyperextension. Proprioceptive deficit causes decreased awareness of knee joint position. Reduced knee joint proprioception associated with the other aetiologies can increase the chances of knee hyperextension. There is little evidence on the role of proprioceptive functions in the hyperextended knee in individual with stroke. Therefore, it is important to find the association between the knee hyperextension and knee joint proprioception. Further, Importance to proprioception training can be given during rehabilitation, to minimize the effect of proprioception deficit in hyperextended knee joint. As with proprioceptive deficit, hyperextension enables safe step-to-step transition, without the risk of poor knee control and subsequent collapse can occur.

## CONCLUSION

Findings based on some of the reviewed articles and our own clinical experience indicates Aetiology-Specific Treatment "*A Customized Physiotherapy Programme* Strategy" for genu recurvatum Patients. This Program could help confirm the clinical indications and identify the most appropriate treatment for each patient. The study could be continued in future with more sample size for genu recurvatum post stroke cases and also for different locations of lesion.

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